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Editorial

*"Early detection of neonatal sepsis:
Way to reduce the neonatal mortality"*



Bangladesh Institute of Child Health



Dhaka Shishu (Children) Hospital

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DHAKA SHISHU (CHILDREN) HOSPITAL JOURNAL

CONTENTS

VOLUME 30

NUMBER 1

JUNE 2014

EDITORIAL

- 1 Early detection of neonatal sepsis: Way to reduce the neonatal mortality
MAK Azad Chowdhury

LEADING ARTICLE

- 3 Pediatric High Dependency Care(PHDC) in Bangladesh: Situation Analysis
Mohammed Reaz Mobarak, Manzoor Hussain, AKM Tajuddin Bhuyian, Md. Rafiqul Islam,
Ferdousi Begum, Nabila Akand, Tanzila Farhana

ORIGINAL ARTICLES

- 9 Correlation of Birth Weight with Foot Length of Neonates
Md. Aynal Hoque, Hossain Sahid Kamrul Alam, Md. Abu Sayeed
- 14 Evaluation of Hematological Scoring System (HSS) for Early Diagnosis of Neonatal Sepsis
Mir Mohammad Yusuf, Md. Jahangir Alam, Manzoor Hussain, MAK Azad Chowdhury
- 20 Concavo Convex Oblique Anastomosis in The Treatment of Jejunoileal Atresia – Our Experience in a
Tertiary Paediatric Hospital in Bangladesh
Sabbir Karim, Sultana Parvin, Kazi Md. Noor-ul Ferdous, S.M. Mushfiqur Rahman,
Ahsanul Kabir, Nazmul Islam, S.M. Mahmud, Mejbah Uddin
- 25 Prediction of Neurodevelopmental Outcome by Cranial Ultrasound in Preterm Neonates in
a Tertiary Care Hospital
Mirza Md. Ziaul Islam, M Monir Hossain, Naila Zaman Khan
- 33 Cancer in Children – A Situation Analysis at Dhaka Shishu (Children) Hospital
Sarabon Tahura, Md Selimuzzaman
- 37 Evaluation of EEG In ASD (Autism Spectrum Disorders) And Other Mental Health Disorder Attending
A Mental Health Clinic (MHC) in Dhaka Shishu Hospital
Humaira Rafiq Quaderi, Farzana Islam, Selina Husna Banu, Asma Begum Shilpi, Naila Zaman Khan
- 43 Improved Sleep and Behavioral Pattern after Adenotonsilectomy in Children with
Sleep Disordered Breathing (SDB)
Syed Hasan Imam Al-Masum, Ali Jacob Arsalan, Md. Jahangir Alam, Tanveen Ishaq

REVIEW ARTICLES

- 50 Trends in Breastfeeding Practices in Bangladesh, 1993-2014
Probir Kumar Sarkar, Abu Tayab, Nital Kumar Sarkar
- 55 Constipation in children - A Review
Rabi Biswas

CASE REPORTS

- 64 Sirenomelia: The Mermaid Baby - Report Of Rare Case
Mahmuda Hassan, Marium Begum, Nasim Jahan, Abdul Mannan,
Kona Choudhury, SM Zabrul Haque, Hamidur Rahman
- 67 A Tale of Early Age of Onset of Guillain-Barre Syndrome
Mir Mohammad Yusuf, Md. Jahangir Alam, MAK Azad Chowdhury
- 71 **Abstract from Current Literature**
- 73 **Dhaka Shishu Hospital (DSH) News**
- 74 **Bangladesh Institute of Child Health (BICH) News**
- 75 Postgraduate courses/training in paediatrics and child health
- 76 Students qualified from Bangladesh Institute of Child Health
- 77 Seminars, Symposiums, Workshop, CME / CPD
- 78 Instructions for Authors
- 81 Subscription form
- 82 Editor's Address

EDITORIAL

Early detection of neonatal sepsis: Way to reduce the neonatal mortality

MAK Azad Chowdhury

Neonatal sepsis is one of the three major causes of newborn mortality in our country- others being perinatal asphyxia and low birth weight. Despite advancement in diagnostic tools and availability of newer drugs, sepsis mortality remains high and many of the preterm and asphyxiated babies ultimately die of infection. At present neonatal mortality in developing countries is 11-68 per 1000 live births, in Bangladesh at present, it is 28 per 1000 live births. In developed countries, it is about 3-5 per 1000 live births. A recent systemic review estimated that the incidence of neonatal infection globally in 2012 was between 5.5 and 8.3 million and average case fatality was 9.8%.

An early diagnosis of neonatal septicaemia helps the clinician in instituting antibiotic therapy at the earliest, thereby reducing the mortality rate in the neonates. An early identification of an infected neonate also helps in avoiding the unnecessary treatment of a non- infected neonate.

Despite recent advances in the management of neonatal sepsis, including use of more potent antibiotics and an array of sophisticated biomarkers to diagnose sepsis, timely and early identification continue to be a frequent and challenging problem in the management of the newborn or high-risk neonate in the NICU.

The complex interaction between the functionally immature immune system of the newborn and the developing premature neonate linked with a wide spectrum of potential infecting organisms, ranging from hospital-acquired infections to those acquired from the mother transplacentally or during the birth process. Factors that delay diagnosis and initiation of therapy include lack of specific clinical features, as the infant often presents with subtle and nonspecific clinical signs and symptoms. The definitive conventional diagnosis of sepsis rests

upon isolation of pathogenic bacteria in blood cultures, which has low sensitivity and delay in reporting to influence initiation of antibiotic therapy. Further diagnostic limitations of the blood culture method include a higher incidence of false negative results, due to low blood volume drawn for culture and prior antibiotic use. As a result, early antibiotic therapy is frequently initiated for presumed infection or delayed due to uncertainty increasing disease risk.

Different parameters to detect sepsis have different sensitivity and specificity. Typical diagnostic parameters depend on conventional laboratory tests that are routinely serum based, such as white blood count (WBC), absolute neutrophil count (ANC), immature/total neutrophil (I/T) ratio.

Among the hematological parameters, combination of each parameter have more sensitivity than individual one. Neutropenia, I/T ratio, and low platelet count have better predictive value for sepsis. However, recent information has suggested that the diagnostic accuracy of WBC, ANC, and I/T parameters may better predict sepsis when using age-specific ratio normograms rather than fixed normal ranges.

Among the acute phase reactants, CRP, produced in the liver, is a frequently used laboratory test for the diagnosis of neonatal sepsis. This has a half-life of 24–48 hours and takes 10–12 hours to respond after an infection. Its changing patterns or continuous decreased levels are useful to monitor progress or guide clinicians in decisions related to duration of antibiotic treatment. But it is not detected so early in blood and a variety of noninfectious neonatal conditions can also increase CRP such as meconium aspiration syndrome, perinatal asphyxia, hemolysis, tissue injury, surgery, glucocorticoid etc.

Procalcitonin (PCT), another acute phase reactant, produced by monocytes and hepatocytes, increase early-within 2–4 hours, after an exposure to a bacterial pathogen during the acute stage of sepsis. Levels peak at 6–8 hours and remain elevated for the next 24 hours with a half-life of 24–30 hours. The sensitivity and specificity of it is more and the response of PCT to early infection is more rapid than that of serum CRP levels. However, PCT reliability as a single biomarker of neonatal infection is limited by its nonspecific elevations in healthy neonates over the first 48 hours of life. Furthermore, it can be falsely elevated in other non-infective conditions such as intracranial hemorrhage, birth asphyxia, and conditions associated with neonatal hypoxemia. The important aspect of positive PCT is that when it is used in conjunction with CRP, it gives more sensitivity to diagnosis of bacterial infection. Moreover its concentration remains high for long time than other parameters in blood.

Besides the above mentioned parameters, detection of cytokines, chemokines (IL6, IL8, TNF- alpha) in blood and uses of molecular technology (FISH, PCR for detection of DNA, RNA of organisms) are also promising to detect sepsis early but with limited uses in our country.

So for clinicians, in absence of definite organism in the culture, clinical suspicion along with the hematological and biological markers, an early diagnosis of neonatal sepsis can be made reliably in most cases. So by early diagnosis and prompt management, sepsis mortality in neonates can be reduced significantly which will be a step forward to achieve the Sustainable Developmental Goal (SDG) by 2030 in Bangladesh.

References

1. National neonatal health strategy Bangladesh. Ministry of Health and Family Welfare Government of the People's Republic of Bangladesh 2009.
2. Hoque M M, Ahmed ASM N U, Ahmed S S, Chowdhury MAK A. Clinical manifestation and bacteriological profile of septicemia in preterm neonates : Experience from a tertiary level paediatric hospital. *Bangladesh J Med Sci.* 2004;**10(1)** : 29-33
3. Vergnano S, Sharland M, Kazembe P, Mwansambo C, Health PT. Neonatal sepsis : an international perspective. *Arch Dis Child Fetal Neonatal Ed.* 2005; **90** : 220-22.
4. Seale AC, Blencowe H, Manu AA, et al; Psbi investigator Group. Estimates possible severe bacterial infection in neonates in sub-Saharan African south Asia and Latin America for 2012: a systemic review and meta analysis. *Lancet Infect Dis.* 2015;**14**:713-41.
5. Sucilathangam G, Amuthavalli K, Velvizhi G, Ashihabegum M.A, Jeyamurugan T, Palaniappan N. Early Diagnostic Markers for Neonatal Sepsis: Comparing Procalcitonin (PCT) and C-Reactive Protein (CRP). *Journal of Clinical and Diagnostic Research.* 2012 ;**6(4)**: 627-631.
6. www.dovepress.com , Mally P, Xu J, Hendricks-Muñoz KD. Biomarkers for neonatal sepsis: recent developments. Accessed on 4/8/16.
7. D'Alquen D, Kramer BW, Seidenspinner S, et al. Activation of umbilical cord endothelial cells and fetal inflammatory response in preterm infants with chorioamnionitis and funisitis. *Pediatr Res.* 2005;**57(2)**:263–9.
8. Ng PC, Lam HS. Diagnostic markers for neonatal sepsis. *Curr Opin Pediatr.* 2006;**18(2)**:125–31.
9. Newman TB, Puopolo KM, Wi S, Draper D, Escobar GJ. Interpreting complete blood counts soon after birth in newborns at risk for sepsis. *Pediatrics.* 2010;**126(5)**:903–9.
10. Ng PC, Li K, Leung TF, et al. Early prediction of sepsis-induced disseminated intravascular coagulation with interleukin-10, interleukin-6, and RANTES in preterm infants. *Clin Chem.* 2006;**52(6)**:1181–9.
11. Ng PC, Li K, Wong RP, Chui KM, Wong E, Fok TF. Neutrophil CD64 expression: a sensitive diagnostic marker for late-onset nosocomial infection in very low birthweight infants. *Pediatr Res.* 2002;**51(3)**:296–303.
12. Tang BM, Eslick GD, Craig JC, McLean AS. Accuracy of procalcitonin for sepsis diagnosis in critically ill patients: systematic review and meta-analysis. *Lancet Infect Dis.* 2007;**7(3)**:210–17.
13. Luzzani A, Polati E, Dorizzi R, Rungatscher A, Pavan R, Merlini A. Comparison of procalcitonin and C-reactive protein as markers of sepsis. *Critical Care Medicine.* 2003;**31(6)**:1737–41.
14. Chiesa C, Panero A, Rossi N, et al. Reliability of procalcitonin concentrations for the diagnosis of sepsis in critically ill neonates. *Clin Infect Dis.* 1998;**26(3)**:664–72.
15. Pavcnik-Arnol M, Hojker S, Derganc M. Lipopolysaccharide-binding protein in critically ill neonates and children with suspected infection: comparison with procalcitonin, interleukin-6, and C-reactive protein. *Intensive Care Med.* 2004; **30(7)**:1454–60.

LEADING ARTICLE

Pediatric High Dependency Care(PHDC) in Bangladesh: Situation Analysis

Mohammed Reaz Mobarak¹, Manzoor Hussain², AKM Tajuddin Bhuyian³, Md. Rafiqul Islam⁴, Ferdousi Begum⁵, Nabila Akand³, Tanzila Farhana³

Abstract

Background: Pediatric high dependency care(PHDC) is the provision of close monitoring and management of children who are physiologically unstable beyond the capacity of general pediatric care. PHDC is a level-2 care in between pediatric intensive care unit (PICU), level-3 or above care and pediatric observation unit (POU), level-1 care.

Objective: This analytical survey was conducted to investigate the availability, facility, bed- strength, functional characteristic, working force, standard of care, cost-distribution, service-monitoring and needs of PHDC including its role to reduce case fatality rate (CFR) and achieve Sustainable Development Goals (SDG) 3.2.

Methods: This study was carried out by the PHDC of Dhaka Shishu (Children) Hospital (DSH) during the period of 1st August to 30th October, 2015. The web-sites of the major hospitals in Bangladesh were included in the study. Direct visiting of certain hospitals and cross-sectional telephone-survey were conducted by a trained investigator with an open-ended questionnaire.

Results: Out of 29 government medical college hospitals, only 7 have intensive care units (ICU), 80 beds in total, of which only 3 have PICUs, 1 at Dhaka, 1 at Chittagong and 1 at Mymensingh. In total, 26 hospitals (both government and non-government) have PICU, of which 8 have PHDU. In 201 PICU and PHDU beds (76.92% of beds, in Dhaka city and 23.07%, outside), only 40 are PHDU beds. The total number of beds in these 26 hospitals is 11903 in which PICU beds are 1.68 % and PHDU beds are 0.7%. The bed to ventilator ratio is 3:1, 20 % of these hospitals have patient to nurse ratio 1:1, 30%, 2:1, and 50% 3:1 respectively, doctor patient ratio at day time is 1:3 but at night time 1:5. The approximate cost of PICU in a government hospital is BDT 1000 (12\$) per night, in an autonomous hospital, BDT 5000 (62\$) per night respectively. The cost of PHDU is BDT 3000 (38\$) per night in an autonomous hospital. In a private hospital the cost of PICU, is between BDT 8000 to BDT 25000 (100\$ to 313\$) per night and PHDU, BDT 6000 to 12000 (75\$ to 150\$) per night.

Conclusion: The cost of both PICU and PHDC are high both in government and private hospital for general people. There is negligence in monitoring quality of service, cost and data-recording of PHDU and PICU. Every major hospital should have a provision of a 10 bed PHDU. We need more children physicians, skilled nurses and paramedics as well as advanced equipment. PICU and PHDU should be closed units.

Key words: pediatric high dependency care and SDGs

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Introduction

Bangladesh is a highly populous country (estimated population, 159 million),¹ 42% of which is under 17. A few of the essential health indicators of the country has been shifting towards positive direction e.g. infant mortality rate (IMR), 35 per 1000 live birth, neonatal mortality rate (NMR), 28 per 1000 live birth, under 5 mortality rate, 51 per 1000 live birth, crude birth rate (CBR), 19.2 per 1000 live birth, natural growth rate (NGR), 1.37%, population per hospital bed 1860, population per physician 3297, and population per nurse 5720.² The *National Sustainable Development Strategies (NSDS), Targets 3.2 by 2030*, targets to end preventable deaths of neonates and children under 5, aiming to reduce neonatal mortality as low as 12 per 1000 live births and under 5 mortality to as low as 25 per 1000 live birth.³ Among the huge health care seeking people (50% of which are children), only 30% are fortunate enough to get hospital care. In a government hospital usually 2 patients compete for 1 bed. The case fatality rate (CFR) among children in government medical college hospitals is 1 in 9 and in regional district hospitals, 1 in 40 respectively.⁴

The PHDC is a provision of close observation, monitoring and therapies to children who are potentially physiologically unstable and demand management beyond the capacity of general pediatric care.⁵ It is a level-2 care located between PICU which is a level-3 or above care and POU which is a level-1 care. The general pediatric ward is a level 0 care.⁶

Background of PHDU and PICU

In 1852, Florence Nightingale first mentioned on high dependency care (HDC). She advocated the benefits of having a designated area in the hospital where post-operative and other patients requiring close-observation can be monitored. Such rooms were introduced in the 1940's as post-operative recovery rooms which significantly reduced the morbidity and mortality of post-operative patients. The intensive care unit (ICU) came into light during polio epidemic of 1950s, when ventilator support for respiratory failure became essential.⁷ In the UK the first ICU was established in 1967.⁸ A few years' later HDU was established.⁹ In 1988, the first attempt was made to define HDU in a report by the association of anesthetists. The department of health¹⁰, UK, in 1996 produced a guideline on admission and discharge from ICUs and HDUs.

In Bangladesh, the first ICU was established in 1980 at the National Institute of Cardiovascular Disease (NICVD) for the adults only.¹¹ Since then the number of ICUs have grown steadily countrywide, mostly in Dhaka. The first PICU at Dhaka Shishu Hospital (DSH) was established in 1992.¹²

Model of HDU

A single model of HDU will not fit in a search model has its advantage and limitation. The ideal model of HDU should be a model which will be affordable by general patients and which will satisfy them¹³. The aim of HDU is to provide a level of care between that of ICU and usual ward care. This means providing service with advanced monitoring equipment with skilled and adequate number of nurses and access to skilled medical staff.¹⁴

Three structural models described as 'Freestanding', 'Parallel' and 'Integrated' by Cheng et al,¹⁵ which have their advantages and limitations. Two other models of PHDCs have been described by Crawford and Powell.¹⁶

Peripheral PHDU

It is managed by a pediatrician as a specialist care unit separate from PICU.

Central PHDU

It is placed adjacent to PICU and is managed at 3 levels

General pediatric subspecialty approach

Combined approach with intensivist and general pediatrician

Intensivist in consultation with subspecialist

For each shift ensure presence of a nurse with specialized training on A (APLS-advanced pediatric life support /PALS- pediatric advanced life support), H (HDC module) and C (critical care course). A portion of this service is for critically ill children whose care-needs exceed the capacity of a general ward as defined by reaching pediatric critical care minimum dataset (PCCMDS) level 2 and does not meet the criteria for PICU as defined by reaching PCCMDS level 3 and above. Pediatric HDU services will be available to all critically ill children from the point of discharge from maternity or a neonatal unit until their 16th birthday. Interdependence of PHDU depends on the site and specialty with other services e.g. general pediatric

ward, PICU, pediatric critical care unit (PCCU), neonatal intensive care unit (NICU), anesthesia, ENT etc. In each hospital there should be a 24 hour service covered by a consultant pediatrician, a consultant anesthesiologist, and a nominated senior pediatric nurse with critical care qualification or at least five years' experience at acute pediatric care. A clinician should be available 24 hours a day for PHDU for initiating appropriate treatment and effective communication with children and their families. The nurse to patient ratio for PHDU should be 1:2. All staff should have training on infection control, handling infected and critically ill patients, fire precaution and risk assessment.

There are 2 types of ICU models operating world wide, 'open' and 'closed model'. Only one-third of the ICUs in the USA are operating on closed model, mainly because of a dearth of certified intensivists. In Bangladesh all of the ICU models are closed-type. In the 'open type', the consultant physician directs the care of the individual patient with the assistance of a house-staff who provides round-the-clock on-the-spot patient care. The physicians are not specially trained or experienced at critical care. An intensivist, if available, is not involved directly in the care of most of the patients. In a 'closed model' the patient care is co-managed by a trained intensivist. The patients are in effect placed under the charge of an intensivist who delivers round-the-clock cover through suitably trained physician staff.¹⁷

Methods

This surveillance was carried out by PHDU of DSH, during August to October 2015. We included all major government medical college hospitals, district hospitals with more than 100 beds, and renowned autonomous and private hospitals of Bangladesh. We cruised through the web-sites of these hospitals, census report of Bangladesh Bureau of Statics (BBS), report of Ministry of Health and Family welfare (MH&FW), Directorate General of Health services (DGHS), Health Bulletin 2014, survey report of UNICEF, main newspaper survey of Bangladesh, other national and international publications, direct visiting of many hospital PICU's & PHDU's. Direct visiting of certain hospitals and cross sectional telephone interview of hospital heads were conducted by a trained investigator. An open-ended questionnaire was used.

Results

In Bangladesh, out of 29 government medical college hospitals, only 7 have intensive care facility of which

3 can provide pediatric intensive care support (1 at Dhaka, 1 at Chittagong and 1 at Mymensingh). The rest of them are running with adult set-up and by anesthesiologists. There are only 80 intensive care unit (ICU) beds in total¹⁸. Cumulatively, 26 hospitals (both government and non-government) have PICU facility and the number of PICU beds of these hospitals is 201. In total, 76.92% of PICU beds are in Dhaka city and 23.07% beds are outside. Among these 26 hospitals, only 8 have PHDU facility. The number of beds of these PHDU is only 40. The total number of beds in these 26 hospitals is 11903 in which PICU beds are 1.68 % and PHDU beds are 0.7%. Unfortunately there is no PICU facility available at Rangpur, Rajshahi and Barisal divisions of Bangladesh where approximately 60% of the population reside. As a whole in government and private set-ups there are 61 NICU facilities available in Bangladesh, among which 33 have bed statements (415 beds in total).

In our study of the PICUs and PHDUs the bed to ventilator ratio was found to be 3:1. Twenty percent of these hospitals have patient to nurse ratio 1:1; 30%, 2:1; and 50% have 3:1 respectively (Table 1), doctor-patient ratio at day time is 1:3 but at night time, 1:5. Arterial blood gas (ABG) analyzers are available in 80% PICUs. Eighty five percent of the PICUs have access to 12 lead ECG, 70% have CPAP, 30% have defibrillator, 50% have infusion pumps. All the PHDUs and PICUs of Bangladesh are close type, which means they are separated from the general pediatric ward.

Table-I
Patient Nurses ratio of 26 hospitals

No Hospital	Patient: Nurse	%
5	1:1	20
8	2:1	30
13	3:1	50

The approximate cumulative expenditure of PICU for patients in government hospitals is BDT 1000 (9£) per night, and in autonomous hospitals, BDT 5000 (45£) per night and PHDU expenditure, BDT 3000 (27£) per night. In private hospitals PICU, this cumulative expenditure ranges between BDT 8000 (70£) to BDT 25000 (218£) and PHDU expenditure ranges between, BDT 6000 (54£) to 12000 (108£).

Table-II
Comparison of cost of PICU, PHDU in Bangladesh with Leeds Hospital UK¹⁹

Service description	Govt.Hospital Bangladesh (per night)	Autonomus Hospital Bangladesh (per night)	Private Hospital Bangladesh (per night)	Leeds Teaching Hospital UK (per night)
PICU	1000BDT/9£	5000BDT/45£	8000-25000BDT (70-218)£	1614.58£
PHDU	No data	3000BDT/27£	6000-12000BDT/54-108£	547.76£

In Bangladesh we do not have any national authority to monitor the standard and quality of service, admission criteria, and cost set-up. The data recording is also poor.

DSH: Critical Care Pediatrics

Dhaka Shishu Hospital is the largest tertiary care children hospital in the country which was established in 1974. At present the hospital has 640 beds. The hospital has 15 bed PHDU, 14 bed PICU, 7 bed pediatric cardiac intensive care unit (PCICU), 6 bed pediatric critical care nephrology unit (PCCNU), 10 bed NICU, 20 bed special care baby unit (SCABU). Besides this, there is a 10 bed post-operative care unit. In the year 2014, total 2,29,599 patients attended the OPD and emergency department and 35,990 patients were admitted in the in-patient department. Around 10,000 patients failed to be admitted due to scarcity of beds and financial constraints. From January to October 2015 a total of 465 patients got admitted in the PICU and 3 times of these patients were in the queue from different units.²⁰

Discussion

In Bangladesh, during the last decade there has been overwhelming growth of private hospitals and clinics. But, most of these hospitals and clinics have very scanty facility. Very few of these hospitals have ICUs and HDUs. It is proposed that a well-equipped clinical laboratory is essential on 24 hours' basis to provide basic hematological tests, biochemistry, blood gas and toxicological analysis in each hospital. Portable chest radiograph is also important which plays an important role in decision making of critically ill patients and this leads to therapeutic alterations in 66% of intubated patients and 23% of non-intubated patients. In our survey, we found that 100% of PICUs have 24 hours' routine laboratory facility including blood gas analysis and 60% have portable X ray facility.

In Bangladesh, there were 11463 general beds in 14 government medical college hospitals during the year

2014, the admitted pediatric patients were 140,127 of which 9,500 died (average case fatality ratio, 14:1). There were 9,500 beds in 62 district hospitals and 2,94,259 pediatric patients were admitted during the same year i.e. 2014, among which 6,798 died (case fatality ratio, 43:1). Approximately 1400 pediatricians (having at least one post graduate degree in pediatrics) are now working along with another 3000 who have 6-12 months' post graduate training on pediatrics²¹. Three hundred physicians took PALS training from Dhaka Shishu (Children) Hospital by the North American Medical Associations during 2012 and 2014. About 2000 nurses have pediatric training after diploma. These figures are small in comparison to 70 million pediatric population of Bangladesh

It is recommended that all the nurses of PICU and PHDU should have basic advanced life support (BALS) and PALS training. All major medical college hospitals, district hospitals and large private hospitals should establish at least 10 beds PHDU as well as PICU.

PHDU: The International Perspective

Inadequate information is available concerning the need and appropriateness of critical care and therefore, it is difficult to compare PHDU's in different countries.²²

In 1999, only 10% of the hospitals in the UK had PHDUs²³ and it was noticed that the development of HDU was unplanned and haphazard.²⁴ The cost of PICU and NICU per bed per night in the UK was £1702, PHDU, £584, and SCABU, £353 respectively.²⁵

The USA spends more than 1% of its gross national product (GNP) to provide ICU's and on the other hand the UK spent 0.05%.^{25,26} The average number of beds in a critical care unit of the USA was 11-12, while the average number of beds in the UK was 6.²⁷ The HDU in the USA is known as intermediate care areas (ICAs), or step up and step down care.^{28,29} The cost of pediatric intermediate care bed in the

USA was more than a usual ward bed (\$534±\$60 Vs \$381±\$42), but less expensive than that of a PICU (\$764±\$99)³⁰. In 95% of pediatric ICAs the registered nursing staff to patient ratio was 1:2 or 1:3.

The ICAs of the USA provide level- 2 care. In a survey, 501 hospitals were found to have PICU (2001) of which 141 had ICAs with a total 1342 ICA beds, and 31 (22%) had e" 15 ICA beds. The number of ICA beds per child varied between region to region, from 1 per 34,269 in the mountainous states to 1 per 81,656 in the New England.³¹ The number of hospitals in the USA with specialist pediatric critical care facility was 337.³² A cross-sectional telephone survey across 51 states of the USA and 12 provinces of Canada, identified 181 hospitals which have 2 or more pediatric wards or more than 50 acute pediatric beds.³³ Among these hospitals each had a PICU and 55% had an ICA and 47% of these ICAs were located within the PICU reach. The median number of ICA beds was 4 (ranging from 2 to 7).

In the then West Germany, definite guidelines regarding HDC beds were developed in 1974. However with the increasing need of ICU, the concept of short term ICU or HDC was realized with the advent of post-anesthesia care unit (PACU) which is a subdivision of high dependency care unit(HDCU).³⁴

In Greece, recommendations were made for the creation of new HDUs in peripheral hospitals with less than 150 beds and in areas that had a high incidence of accidents. Unfortunately no information was available regarding staffing and their training on ICUs in European countries.³⁵

The joint faculty of Australian and New Zealand College of Anesthetists and Royal Australasian College of Physician published minimum *Standards for ICU's and Recommendation on Standard for HDU's seeks accreditation for training on intensive care*. At least 300 admissions, irrespective of ventilator status, were required to meet the criterion of the guidelines. A nurse to patient ratio of 1:1 for mechanically ventilated patients and greater for patients requiring complex management is necessary. In 2005 the rate of admission for PICU was 1.4 per 1000 children. The percentage of ventilated patients admitted in PICU ranged from 9.7% to 85%.

Conclusion and Recommendation

This study is a quick attempt to assess the availability of PHDC as well as PICU, and their facilities e.g. bed strength, working force, standard of care, cost-

distribution, and needs in Bangladeshi hospitals. The cost of both PICU and PHDC are high both in government and private hospital for general people. There is negligence in monitoring quality of service, cost and data-recording of PHDU and PICU. This is also an initiative to look at the areas where improvements need to be addressed and stressed.

We need at least 50 PHDUs and more PICUs of 5 to 10 beds in each of 20 government medical college hospitals and 30 PHDUs and more PICUs in each 150 to 250 bed district hospital so that all critically ill children can be referred within 1 to 2 hours'. It is recommended that all the nurses of PICU and PHDU should have BLS and PALS training. We need more children physicians, skilled nurses and paramedics as well as advanced equipment. PICU and PHDU should be closed units.

References

1. BIDS Report; 2015.
2. Statistical pocket book Bangladesh; 2013, Bangladesh bureau of statistics, statistics division, ministry of planning, Dhaka; 2014.
3. National Sustainable Development Strategies 4 (NSDS).
4. DGHS, Health Bulletin; 2014.
5. NHS England NHSCB/E7b p-1-15.
6. Intensive Care Society (UK): ICS Level of Care; 2003.
7. Oh T. The development, utilization and cost implication of intensive care medicine: Strategies for the future. In: Tinker J, Brown D, Sib bald W. (eds) Critical care- Standard, Audit and ethics. Edward Arnold. London. 1996.
8. British Medical Association planning Unit (1967). Report of the working party on intensive care. British Medical Association. London.
9. Sheppard M, Wright M. Principles and practice of High dependency Nursing. Bailliere Tindall. Edinburgh; 2000.
10. Department of health. Guidelines on admission to and discharge from Intensive Care and High Dependency Units. London 1996.
11. Faruq MO, Ahsan AA, Fetema K. An audit of intensive care services in Bangladesh. *ibrahim Med Coll J* 2010; 4(1): 13-16.
12. Dhaka Shishu(children) Hospital, souvenir, December, 2011.
13. Timmermann AM. Nurse staffing and training, Europe In: Tinker J, Browne DRG, Sibbald WJ.

- Critical care, Standards, Audit and Ethics. Arnold. London;1996.
14. Gerber, Watcher, Structural models for intermediate care areas: One size does not fit all. *Critical care Medicine* 1999; **27**(10):2321-23.
 15. Cheng DCH, Byrick RJ, Knobel E. Structural models for intermediate care areas. *Critical care medicine*1999; **27**(10):2266-71.
 16. Crawford N, Powell C. Pediatric high dependency Care. *Current Pediatrics* 2004; **14**(3):197-201.
 17. www.archivehealthcare.financialexpress.com/201004/knowledge.org.shtml.
 18. ICU facilities scanty at government hospitals of Bangladesh, the daily star, Dhaka, front page; 2015 [cited 2015 September 16]. Available from: <https://www.the-dailystar.net/frontpage/icu-facilities-scanty-143884>.
 19. The leedsTeaching Hospitals NHS Trust SLA monitoring report as at period 3 financial year 2007/08. LTHOO Trust Summary.
 20. Dhaka Shishu (Children) Hospital, Souvenir, December 2015,20-22.
 21. www.bpabd.org/life-member.php#list-of-general-member.php.
 22. Wild C, Narath M. Evaluation and planning ICUs: methods and approaches to differentiate between need and demand. *Health Policy*. 2005; **71**:289-301.
 23. Garfield M, Jeffrey R and Ridley S, An assessment of the staffing level required for a high dependency unit. *Anesthesia* 2000; **55**:137-43.
 24. Comprehensive critical care. A review of adult critical care services. Department of Health. London; 2000.
 25. Rush forthPediatric High Dependency Care in west, North and east Yorkshire, September 2008, p25-26.
 26. Singer M, Myers S, Hall G, Cohen SL, Armstrong RF. The cost intensive care: a comparison on one unit between 1988 and 1991, *Intensive care medicine*. 1994;**20**:542-5.
 27. McPherson K. Safer discharge from intensive care to hospital wards. *British Medical Journal* 2001;**322**:1261-62.
 28. Jacobs's p, RapoportJ, Edbrook, Economies of scale in British intensive care units and combined intensive care/high dependency units. *Intensive care medicine* 2004; **30**:660-4.
 29. Popovitch J. Intermediate care unit. Graded care options. *Chest*.1991; **99**(1):4-5.
 30. Junker C, Timmerman JE, Alzoa C, draper EA, Wagner D. A Multicenter description of intermediate care patients: Comparison with ICU low-risk monitors patients. 2002; **121**(4):1253-1261.
 31. Lawless S, Zaritsky A, Phipps J, Riley lawless K. Characteristics of pediatric intermediate care unit in pediatric training programs. *Critical care medicine* 1991;**19**(8):1004-07.
 32. Randolph AG, Gonzales CA, Cortellni L, Yeh TS. Growth of pediatric intensive care units in the United States from 1995 to 2001. *The Journal of pediatrics* 2004;**144**:792-8.
 33. Odetola FO, Clark SJ, Freed GL, Bratton SL, Davies MM. National Survey of Pediatric Critical Care resourcesin the United States. *Pediatric* 2005; **115**(4):382-6.
 34. Vendenburg SD, Hutchinson JS, CS, and the Paediatric Early Warning system Investigation. A Cross-sectional Survey of Levels of Care and Response mechanisms for Evolving Critical illness in Hospitalized Children. *Pediatrics* 2007;**119**(4): 940-6.
 35. PerinT.High dependency units: reducing the cost of intensive care without loss of quality. *Europian journal of Anesthesiology* 1988;**15**:753-755.
 36. Timmerman AM.(1996).Nurse Staffing and Training, Europe in: Tinker J, Browne DRG, Sibbaid WJ. *CriticalCare,Standards,Audit and Ethics*. Arnoled. London.

ORIGINAL ARTICLE

Correlation of Birth Weight with Foot Length of Neonates

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Abstract

Background: Birth weight is the index of nutritional status for a community. The measurement of birth weight is important for monitoring health of the child. Recognizing the importance of birth weight measurement, 34th World Health Assembly in 1981, recommended it to be one of the 12 global indicators for monitoring of health of the community. Birth weight is an indicator for predicting the immediate or later outcome of a newborn child. In a developing country like Bangladesh where maximum delivery occurs in rural community and are mostly attended by Traditional Birth Attended (TBA) or relatives, birth weight cannot be recorded due to paucity of suitable weighing scale. To overcome the problem associated with weighing the newborn, it was considered justified to find out other simpler measurements that could be used as substitute of weighing in order to identify low birth weight babies.

Objective: To assess the validity of foot length of a newborn in estimating the birth weight and thereby to be used for screening of low birth weight babies.

Methods: A cross sectional study was carried out during the period of 1st July, 2006 to 31st December, 2006 in Dhaka Shishu Hospital, Dhaka and Maternal and Child Health Training Institute, Azimpur, Dhaka, Bangladesh. A total of 500 children both male and female aged less than 12 hours who had no congenital anomaly nor chromosomal abnormality were analyzed. Weight of new born babies were recorded by electric weight machine and foot length were measured by non-elastic, flexible GOLD FISH BRAND SUPERIOR TAILORING RULE (ordinary tailoring tape) of left foot.

Results: Birth weight $\geq 3500\text{gm}$ correlates with foot length of $\geq 9.70\text{cm}$; $\geq 3000\text{gm}$ with foot length of $\geq 8.80\text{cm}$; $\geq 2500\text{gm}$ with foot length of $\geq 7.89\text{cm}$; $\geq 2000\text{gm}$ with foot length of $\geq 7.10\text{cm}$; $\geq 1500\text{gm}$ with foot length of $\geq 6.5\text{cm}$.

Conclusion: Present study shows simple measurement of foot length at birth can be an alternative of birth weight recording as a measure of fetal growth and identifying newborns with low birth weight where recording of weight is not feasible.

Keywords: Foot length, birth weight

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Introduction

Bangladesh is a developing country with a population of approximately 160million¹. Majority (>80%)² of the population live in the rural areas where illiteracy, poverty and malnutrition are widely prevalent. Health service facilities even for the neonate, the most vulnerable group is not satisfactory till today in rural community. In these circumstances in Bangladesh majority 75% of the deliveries are conducted there, either by Traditional Birth Attendants (TBA) or by relatives.³

Birth weight is an indicator of future health and survival of the child. It is known to be an important factor which determines the readiness with which the newborn baby adjusts to its surroundings. Low birth weight is associated with a very high neonatal mortality, mainly due to adverse environmental influences, proneness to infections and difficulties in maintaining adequate nutrition.⁴ Birth weight is the factor that determines the ability of the infant to adopt to its new environment and to develop normally.⁵ The incidence of low birth weight babies in our countries quite high like other developing countries. Recording of birth weight is universal in developed countries, where as it is almost impossible in developing countries specially in rural communities where most of the deliveries are conducted.

The reasons for this lingers are as follows.⁶ People in general and specially in rural areas do not consider weight recording as an important point in the care of newborn. Most of the home deliveries are conducted by Dais or TBAs who do not possess weighing as a part of their delivery kits. There are taboos in the community which do not favor the measurement of birth weight because of the fear of bad effects of evil eye and the adverse effects of any words of praise. The practice of confinement after delivery restricts outsiders from entering the room where the mother and the baby are; thus reducing the chances of any help from a literate person to the TBA in recording the birth weight. As the measurement of birth weight in our community is not feasible, it is urgently felt that a means has to be devised to identify low birth weight babies. It is difficult to provide expensive weighing scales and its handling in the community set up. However, extreme accuracy which is needed in research work is not required for day to day decision making concerning special care at home or

referral to hospital. Therefore, some simple, practicable, quick, reliable, valid and cost-effective alternative to birth indentifying low birth weight in the community needs to be developed and introduced.

Methods

A cross sectional study was carried out during the period of July 2006 to December 2006 in Dhaka Shishu Hospital, Dhaka and Maternal and Child Health Training Institute, Azimpur, Dhaka, Bangladesh. A total of 500 new born babies constituted study population. Only the live born babies of both sexes were selected for the study. Seriously handicapped baby with major congenital malformation, seriously ill and baby of diabetic mother were excluded from the study. Babies of mothers having serious obstetrical or medical problem were also excluded. Babies were examined within first 12 hours of birth either born in hospital or outside. The following research equipments were used in the study: Weighing scale in Maternal and Child Health Training Institute, Azimpur, Dhaka (the high sensitive machines of DONG IL company, China) for dispensing goods up to 5 kg weight with a fraction of up to 20 gm was used. The error was corrected before using the machine. Electric weight machine in Dhaka Shishu Hospital, displaying grams of weight. Measuring tape (golden brand superior tailoring rule, China made). Techniques and tools used for data collection: A data sheet was developed to collect data which was checked and verified.

A pre test was done. Based on the findings, modifications were made and the requisite number of proforma were photocopied. Data was collected by the researcher himself in all the places. The following methods were used for data collection: Face to face interview. (Transfer of information from case sheets. Measurements of newborn babies. Weight measurement: Weight of new born babies were recorded by electric weight machine and baby scale within 12 hours of delivery. The baby scale was able to record weight fraction of a minimum value of 5 gm accurately and the electric weighing measured up to the exact grams. Foot length measurement: Foot length was measured to the nearest 0.1 cm in babies with non-elastic, flexible gold fish brand superior tailoring rule (ordinary tailoring tape). Measurement was taken by placing the tape along the sole of foot from the tip of the

heel to the tip of the great or second toe (whichever was longer) ensuring that the toes fully extended.

Results

During the study period total 500 cases according to the inclusion criteria were analyzed. Out of them, 220(44%) from Dhaka Shishu Hospital and 280(56%) from Maternal and Child Health Training Institute, Azampur, Dhaka.

Gestational age weeks	Weight in gm		Total
	Low birth weight (%)	Normal birth weight (%)	
Preterm <37	159 (31.8)	09 (1.8)	168
Term 37-42	15 (03)	151 (30.2)	166
Post term >42	02 (0.4)	164 (32.8)	166
Total	176	324	500

One hundred seventy six babies were low birth weight, out of them 159 (31.8%) were pre term, 15(03%) were term and 02(0.4%) were post term. One hundred and fifty one(30.2%) of normal birth weight of term babies (gestational age 37-42 wks), and 164(32.8%) of post term babies (gestational age >42

wks) and 9(1.8%) of pre term babies (gestational age <37wks) were normal birth weight.

Birth weight (gm)	Number of cases	Percentage
<1500gm	31	6.2
1500-1999gm	53	10.6
2000-2499gm	92	18.4
2500-2999gm	146	29
3000-3499gm	102	20.4
≥ 3500gm	76	15.2
Total	500	100

In the study 31(6.2%) cases were found to have birth weight <1500gms; 53(10.60%) cases were 1500-1999gms; 92(18.4%) cases were 2000-2499gms 146(29.2%) cases were 2500-2999gms; 102(20.4%) cases were 3000-3499gms and 76(15.2%) cases were >3500gms. Majority cases were 2500-2999gms.

Mean birth weight irrespective of male was 2741.90±747.44gms and female was 2607.67±650.23gms. The mean foot length (cm) of male was 8.65±1.22 and female was 8.57±1.14.

Foot length	5-6.5cm	6.6-7.5cm	7.6-8.6cm	8.7-9.5cm	>9.5cm	Total
Number of cases	42	41	93	210	114	500

Sex of cases	No	Mean ± SD	Weight of babies (gm)	
			Range	
			Minimum	Maximum
Male	268	2741.90 ± 747.44	1100	3995
Female	232	2607.67 ± 650.23	1000	3995
			Foot length	
Male	268	8.65 ± 1.22	05.4	10.0
Female	232	8.57 ± 1.14	5.5	9.9

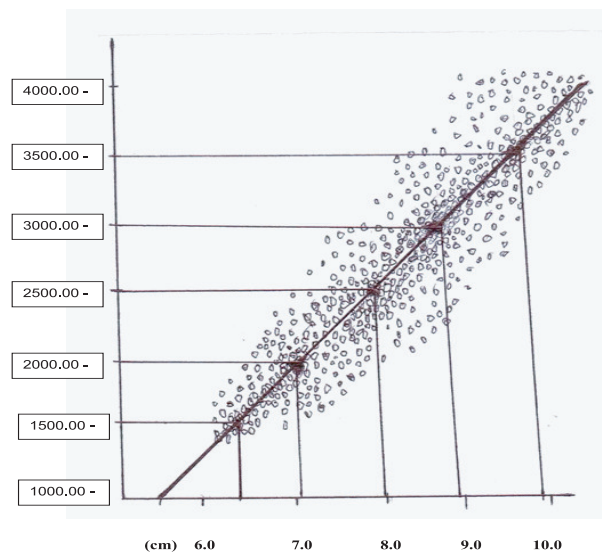
Table V
Gestational age wise mean anthropometric parameters of study cases.

	Gestational age			
	Preterm<37	Term 37-42	Post term >42	Total
	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD
Birth weight	1728.6 ± 647.49	2824.98 ± 573.83	3390.38 ± 361.29	2679.62 ± 706.51
Foot length	6.89 ± 1.35	8.19 ± 0.86	9.34 ± 0.27	8.15 ± 0.18

Mean birth weight of pre term babies were 1728.63±647.49gms, term were 2824.98±573.83gms and post term were 3390.38±361.29gms. Foot length preterm babies were 6.89±1.35cm, terms were 8.19±0.86cm and post term were 9.34±0.27cm.

Table VI
Relationship between birth weight and foot length

Birth weight	Foot length					Total n (%)
	5-6.5cm n(%)	6.6-7.5cm n(%)	7.6-8.5cm n(%)	8.6-9.5cm n(%)	>9.5cm n(%)	
<1500gms	29(93.5)	2(6.5)	0	0	0	31(100)
1500-1999gms	13(24.5)	39(73.5)	1(2)	0	0	53(100)
2000-2499gms	0	0	92(100)	0	0	92(100)
2500-2999gms	0	0	0	146(100)	0	146(100)
3000-3499gms	0	0	0	64(62.7)	38(37.2)	102(100)
>3500gms	0	0	0	0	76(100)	76(100)
Total	42	41	93	210	114	500(100)



Corresponding values of foot length:- e3500gms to e9.70cm, e3000gms to e8.80cm, e2500gms to e7.89cm, 2000gms to 7.10cm, e1500gms to 6.5cm.

Fig 1 Regression line of birth weight on foot length weight (gram)

Corresponding values of foot length:- ≥3500gms to ≥9.70cm, ≥3000gms to ≥8.80cm, ≥2500gms to ≥7.89cm, 2000gms to ≥7.10cm, ≥1500gms to 6.5cm.

Table shows foot length 5-6.5cm of birth weight <1500gms were 29(93.5%) and birth weight 1500-1999gms were 13(25.5%). Foot length 6.6-7.5cm of birth weight <1500gm were 2(6.5%) and birth weight 1500-1999gms were 39(73.5%). Foot length 7.6-8.5cm of birth weight 1500-1999gm were 1(2%) and birth weight 2000-2499gms were 92(100%). Foot length 8.6-9.5cm of birth weight were 2500-2999gm were 146(100%) and birth weight of 3000-3499gm were 64(62.7%). Foot length >9.5cm of birth weight 3000-3499gm were 38(37.2) and birth weight >3500gm were 76(100%).

Discussion

It is quite relevant here to discuss few findings and facts, before going into details about the observed correlation of anthropometric measurement of the newborns. The anthropometric measurement of newborn babies were considered in this study discussed below: (a) Birth weight – The mean birth weight of newborn in this study was 2679.62SD±706.51gms irrespective of sex of baby. The mean weight in male cases was 2741.90±747.44gms and 2607.67±650.23gms in female cases. Mean birth weight irrespective of

preterm <37wks was 1728.63±647.49, term 37-42wks 2824.98±573.83 and post term >42wks 3390.38±361.29. The mean value of birth weight as a whole in this study is influenced by the inclusion of good number of normal birth weight. The value of term babies (2824.98±573.83gm) similar with Islam and Ali (1983)⁶ study where mean birth weight was 2720gms in Rangpur Medical College Hospital and with Kalam and Talukder (1982)⁷ study where the mean birth weight was 2800gms. Present study findings also similar with birth weight in the study conducted by Das and Khanam (1992).⁸ Hossain et al. (1997)⁹ found the mean birth weight for newborn 2.9kg, and Islam et al. (1998)¹⁰ found birth weight as 2806gms which is slightly higher than the mean value of term their counterpart. These findings are similar with the present study. In Bangladesh, several researchers e.g. Islam and Ali,⁶ Kalam and Talukder,⁷ Nahar N,¹¹ Das and Khanam⁹ studied mean birth weight of male and female babies. All these studies showed that male babies had higher mean value of birth weight than their counterpart. This finding is similar with the present study. There are some studies about the foot length of neonates. These are Suzanne Panfold et al(2014).¹², Satarupa Mukherjee et al(2013),¹³ Holambe V.M. et al(2012),¹⁴ Hirve SS et al(1984),¹⁵ HoTY et al(2009)¹⁶ and their cut off value of birth weight were 8cm, 7.85cm, 7.63, 7.63cm and 7.90cm respectively. All these studies are similar with present study. In this study cut off value of foot length for birth weight ≤2500gms was ≥7.89 cm. This result corresponds with all above mentioned studies. The mean foot length of newborn in this study was found to be 8.15±0.18cm. The mean foot length for male and female newborn was found to be 8.65±1.22cm and 8.57±1.14cm respectively. The foot length in preterm, term and post term babies in the present study was 6.89±1.35cm, 8.19±0.86cm and 9.34±0.27cm respectively. Present study shows cut off value of low birth weight is 7.89cm.

Conclusion

Present study shows that foot length measurement at birth can assume the birth weight, where recording of weights not feasible and thereby identifying low birth weight.

References

1. Rahman M.F. 2012 statistical year book of Bangladesh 25th ed. Dhaka; B.B.S. Ministry of planning, Government of the Peoples Republic of Bangladesh.2001; 28.

2. Kumar V and Datta N. Birth weight as an indicator of health. *Indian Pediatric* 1984;**21**:113-8.
3. Shahidullah M, Begum NA, Hassain MQ, Kawser CA, Mannan MA, Islam N. Foot length as Surrogate for birth weight in the newborn-A study of 500 cases. *Bangladesh J Child Health* 2004;**28**(1):18-22.
4. Pratindhi AK, Shrotri AN, Shah U, Bodhani N., D. Domiciliary care of low birth weight neonate. *Indian J. Pediatric* 1986; **53**(1): 87-92.
5. Public health aspect of low birth weight, third report of the expert committees on material and child health. WHO, Geneva, 1961.
6. Islam MN and Ali MY. Anthropometric measurement of the newborn (A study of 248 full term babies). *Bangladesh Pediatric* 1984;**8**(1,2):9-12.
7. Kalam MA and Talukder MQK. Anthropometry of 432 term new born. *Bangladesh Pediatric* 1982;**6** (3,4): 123-8.
8. Das JC, Afroze A, Khanam ST, Paul N. Mid-arm circumference: An alternative measure for screening low birth weight babies. *Bangladesh Med Res Counc Bull* 2005; **31**(1):1-6.
9. Hossain MT, Begum MA, Huq MN. Anthropometric parameters of the neonates. *Dhaka Shishu (Children) Hospital Journal* 2000;**16**(1):1-21.
10. Islam MS et al (1988) Neonatal resuscitation: report on the field testing of a tube and mask device at home, health centre and small hospital(WHO) May-1984.
11. Nahar N, Afroza S, Hossain M. Incidence of low birth weight in three selected communities of Bangladesh. *Bangladesh Med Res Counc Bull* 1998;**24**(2): 49-54.
12. Suzanne Panfold et al. The reliability of a new born foot length measurement tool used by community low birth weight or premature babies born at home in Southern Tanzania. *BMC Public Health* 2014; **14**:859.
13. Satarupa Mukherjee et al. Measuring new born foot length to identify small babies in need of extra care , a cross sectional hospital based study. *Iran J Pediatr* 2013;**23**(5):508-12.
14. Holambe VM, Kakrami VA, Godale LB. Role of neonatal foot length as alternative predictor of low birth weight. *International Journal of Recent Trands in Science and Technology*. ISSN 2277 2812 E-ISSN 2249-8109;**5**(2):2012;PP 91-3.
15. Hirve SS, Ganatra RR. Foot tape measure for identification of low birth weight newborns. *Indian Pediatr* 1984;**30**(6):333-6.
16. HoTY et al. Assessment of growth from foot length in Taiwanese neonates. *Pediatr Neonatol*. 2009; **50**(6):287-90.

ORIGINAL ARTICLE

Evaluation of Hematological Scoring System (HSS) for Early Diagnosis of Neonatal Sepsis

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Abstract

Background: Neonatal sepsis is a common occurrence and major health problem in Bangladesh and other developing countries, a devastating cause of morbidity and mortality during first 28 days of life.

Objective: The study was carried out to evaluate and highlight the importance of hematological scoring system (HSS) in the early detection of neonatal sepsis.

Methods: This prospective study was done between June 2014 to December 2014 (Six months) consisting of 218 neonates admitted in Intensive Care Unit (ICU) Dhaka Shishu Hospital who were clinically suspected to have septicaemia. A total of 128 neonates were enrolled. The neonates with major congenital anomaly, inborn error of metabolism, severely jaundiced due to blood group incompatibilities or RDS were excluded. The hematological parameters were measured in all cases. Patients were scored according to hematological scoring system (HSS), incorporating increase or decrease in leucocytes count, absolute neutrophil count, immature neutrophil count (e.g. band form), presence of degenerative changes in neutrophil (e.g. toxic granulation, vacuolation). CRP estimation and blood culture were also performed. Blood culture is considered as gold standard for diagnosis of sepsis. Fourteen (11%) neonates out of 128 had culture proven sepsis. They were predominantly preterm and very low birth weight. Score 3 was considered as positive.

Results: On evaluation of various hematological parameters the sensitivity of HSS 71% and specificity of 73%, positive predictive value 24%, negative predictive value 95%. Considering the high sensitivity, specificity, negative predictive value, this study implies that score 3 were reliable as a screening tool for early neonatal sepsis.

Conclusion: HSS is a simple, quick, readily available effective tool to detect early neonatal sepsis. Thereby it provides a guideline in making decisions regarding judicious use of initial antibiotic therapy.

Key words: Neonatal sepsis, Blood culture, Hematological Scoring System

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Introduction

Neonatal sepsis is defined as systemic inflammatory response of the body to an infection during first 28 days of life diagnosed either on microbiologic cultures or strong clinical evidence of an infection. Upto 25% of children in intensive care units have sepsis.¹ Worldwide neonatal sepsis is responsible for 1.6 million deaths annually, most of them occur in developing countries² where mortality rate is between 11-68 per 1000 live birth and in Bangladesh rate is 23.3 per 1000 live birth.³ Were as in developed countries it is about 5 per 1000 live birth.⁴ Group B streptococcal (GBS) disease is the most important cause of neonatal sepsis in Europe, but there is preponderance of gram negative organism in tropical and developing countries.⁵ It has been seen that gram negative organism are leading pathogen in Bangladesh.⁶ Apart from mortality, morbidity associated with neonatal sepsis includes sensorineural hearing loss, visual disturbances, seizures, and neurodevelopmental issues.⁷ This life-threatening condition is treatable if diagnosed early but unfortunately, the early signs and symptoms are often nonspecific and confusing which makes it difficult to establish an early clinical diagnosis.⁸ As a result of this uncertainty antibiotics are often started on the slightest clinical suspicions of sepsis. This approach is effective in fighting against the acute infections, but increases the risks of antibiotics side effects and the emergence of drug resistant organisms in neonatal units.^{8,9} Blood culture is considered as gold standard for diagnosis of sepsis.¹⁰ A drawback of culture based diagnosis is the assay time of up to 36 hours.¹⁰ After 36 hours of incubation period specificity are 100%.¹¹ Measures of acute phase proteins, cytokines, cell surface antigens, and bacterial genomes have been used, either alone or in combination, for early diagnosis of neonatal sepsis. Some of these markers are sensitive and specific, but sophisticated or expensive so impractical for developing countries like ours.⁸ Early diagnosis of neonatal sepsis is still a great challenge. Many attempts have been made to develop a set of screening tests, which can rapidly diagnose infected neonates. So the ideal diagnostic test should have quick results, inexpensive and adequate sensitivity, specificity, positive predictive value and negative predictive value to reliably exclude sepsis and avoid unnecessary antibiotic therapy. For early diagnosis of neonatal sepsis a hematological scoring system

(HSS) of Rodwell are preferable because it includes all parameters. Hematological parameters should accurately predict the presence or absence of infection and be reliable.¹² Hematological scoring system (HSS) that we studied includes blood complete picture including white blood cell count, total neutrophil count, immature Polymorphonuclear leucocyte (PMNL) count, presence of toxic granule, platelet count and measure of CRP which is quick and cost effective. There are two exception an abnormal neutrophil count is assigned a score of 2 rather than 1 if no mature neutrophils are seen on the blood smear¹³ and another is CRP estimation.

Methods

This prospective study was conducted at neonatal intensive care unit of Dhaka Shishu Hospital during the period of June 2014 to December 2014. As per our operational definition and inclusion criteria, all the newborns presented with history of premature rupture of membranes, maternal intrapartum fever 101⁰F or clinical signs of sepsis as fever 101⁰F,¹⁴ poor feeding and lethargy or depressed neonatal reflexes were included in the study. Neonates who presented with major congenital anomaly, severely jaundiced due to blood group incompatibilities or RDS (clinical evaluation and CXR) were excluded from this study. After taking a careful history specified questionnaire was designed and the details information was recorded by the investigator. With all aseptic precaution 2 ml blood was withdrawn from suspected sepsis within 24 hours of admission. 1ml sample was anticoagulated with EDTA and using automated hematology analyzer, values of total leucocyte count (TLC), differential leucocyte count and platelet count were noted. Peripheral blood smears were stained by Leishman method. 1ml of blood was inoculated aseptically into conventional blood culture bottle sent to department of microbiology, Dhaka Shishu Hospital for culture and sensitivity. Patient was scored according to hematological scoring system (HSS), incorporating increase or decrease in leucocyte count, absolute neutrophil count, immature PMNs count and platelet count. Degenerative changes in PMNL (toxic granulation and vacuolation) which have seen with Giemsa stained slides. CRP estimation was also performed. Score of 3 was considered as positive.

Table I
Hematological Scoring System

Hematological test	Abnormality	Score
Increased or decreased WBC count	At birth $\leq 5,000 \text{ mm}^3$ or $\geq 25,000/ \text{mm}^3$, 12-24 hrs = $30,000/\text{mm}^3$, Day2 onwards = $21,000/\text{mm}^3$	1
Increased or Decreased total ANC	≤ 1800 or $\geq 5400, 14000, 5400$ at birth, 12- 48 hrs and 48 hrs onwards respectively	1
Immature PMN count (Band form)	$\geq 10\%$	1
Degenerative changes in PMN (Toxic granulation)	Present (≥ 3)	1
CRP	Raised $> 5 \text{ mg/dl}$	1
Platelet count	$\leq 150,000/\text{mm}^3$	1

CRP= C-reactive protein, ANC= Absolute Neutrophil count (neutrophils % x total WBC count). PMN =Polymorphonuclear leuco cyte

The sepsis work up included complete blood counts, absolute neutrophil counts (ANC), immature neutrophil count (e.g. band form), degenerative changes in PMN (e.g. toxic granulation, vacuolation), platelet count, C-reactive protein (CRP) measurements and blood culture. Findings of HSS were recorded in a proforma and later compared with results of blood cultures.

Interpretation of Hematological Scoring System:

Score	Interpretation
≤ 2	Sepsis (very unlikely)
≥ 3	Sepsis (considered positive)
> 4	Sepsis (very likely)

Results

The study was conducted on 128 neonates presenting with clinical features of suspected of neonatal sepsis. Out of 128 neonates, there were 65.6% male and 34.4% female. Nearly half of the newborns (35.1%) in this study were preterm. Hematological scoring system (HSS) was used as a screening test for the detection of neonatal sepsis. On presentation HSS was applied on all neonates with suspected neonatal sepsis. The results obtained were that out of 128 neonates 32% had positive HSS results and 68% had negative HSS results. Raised CRP was seen in 32% followed by low platelet count 25% (Table II).

Table II
Hematological Screening Score (HSS)

Variable	Frequency	Percentage
White blood cell count High Low	85	62.5
Absolute neutrophil count High low	35 3	27.3
Immature PMN count(band form)	13	10.15
Degenerative changes in PMN (toxic granule)	2	1.56
Raised C Reactive protein	41	32.03
Low Platelet count	32	25.00

Table III
HSS and Blood Cultures 2 x 2 Table

		Neonatal Sepsis on Blood Culture		
		Positive	Negative	Total
Neonatal Sepsis on HSS	Positive	10	31	41
	Negative	4	83	87
	Total	14	114	128

Sensitivity = 71%, Specificity = 73%, Positive Predictive Value = 24%, Negative Predictive Value = 95%

All neonates after assessment on HSS underwent blood culture. On blood culture neonatal sepsis was seen in 11% of cases and negative in 89% of cases. On applying the formula for calculation, sensitivity of HSS was found to be 71% and specificity 73%. The positive predictive value of HSS was 24% and negative predictive value was 95% (Table III).

Discussion

Despite the advances in neonatal care, early onset neonatal sepsis remains a serious and potentially life-threatening disease. The definitive diagnosis of septicemia is made by a positive blood culture, which requires a minimum of 48-72 hours, yields a positive result in 10-60% of cases.¹² For early diagnosis of neonatal septicemia a hematologic scoring system (HSS) of Rodwell was introduced in the past.¹³ The HSS assigns a score of 1 for each of seven hematologic findings and shown to be significantly ($P < 0.005$) associated with sepsis.¹³ The early diagnosis of neonatal septicemia is primarily based on clinical evaluation after which empiric antibiotics are started till the result of blood culture is available. But it requires time and chances of positive result are variable.¹⁵ In this study 10.9% neonates were considered as proven sepsis by blood culture. However suspected sepsis groups comprises a difficult diagnostic group and could not be ignored, because fatal infection had been reported in the presence of negative blood culture.¹⁶ Among the included cases of suspected sepsis, the predominance of male (65.6%) may be due to the factors regulating the synthesis of a globulin are situated on the X chromosome leading male gender less immunologically protected than the females. Premature rupture of membrane (PROM) has to be an important risk factor in neonatal septicemia because PROM poses of ascending infection to the

fetus. As no single individual haematological parameter is superior in comparison to another in predicting neonatal sepsis, a combination of these parameters in the form of HSS has been recommended. Hematologic scoring system (HSS) should improve the efficiency of the CBC as a screening test for sepsis until a reliable diagnostic test is available. The HSS has practical advantages; it is applicable to all infants, including those who have received antibiotic therapy prior to evaluation and simplifies the interpretation of hematologic profile. In this study with a cut off score ≥ 3 we observed a sensitivity of 71%, specificity of 73%, PPV 24%, NPV 95%. These results were consistent with other studies.^{17,27} Many studies performed to evaluate the haematological scoring system for the early diagnosis of neonatal sepsis revealed variable results.¹⁸⁻²³ Ghosh et al and Narasimha et al reported that Immature PMN count and Immature:Total (I:T) PMN ratio is sensitive indicator of neonatal sepsis. Degenerative changes in the PMNs made no significant contribution in the diagnosis, in their study.^{23,24} Presence of toxic granules indicates the production of unusual PMNs during infection and stress induced leucopoiesis. They are never seen in healthy babies. Their presence invariably indicates sepsis, but their count is not always increased. Thrombocytopenia was frequently associated with sepsis and indicated poor prognosis. This is thought to be due to increased platelet destruction, sequestration secondary to infections, failure in platelet production due to reduced megakaryocytes or damaging effects of endotoxin.^{25,26} Higher the score on HSS, more are the chances of sepsis. The simplification and standardization of the interpretation of this global test is still required. Variety of other rapid detection methods of

microorganisms, like DNA probes, automated blood culture system and fluorometric detection systems are also available globally, but HSS can still be used as a screening test for diagnosing sepsis and to differentiate infected neonates from the non-infected ones. Furthermore, the sensitivity and the specificity of the test are also high, with certainty of sepsis increasing with the score.²⁷ Murphy et al reported on 100% sensitivity and 100% negative predictive value of two normal white blood cell counts (WBC) within 8 to 12 hours and a negative blood culture at 24 hours for ruling out early-onset sepsis in the neonate.²⁸ Measurement of immature neutrophil granulocytes has been considered to be a helpful early indicator of various infectious conditions and has a long clinical tradition in the diagnosis of bacterial sepsis in neonates.^{29,30} CRP is one of the most widely available; most studied, and most used laboratory tests for neonatal bacterial infection. It is well known that it provides limited sensitivity when determined during the early phases of the disease, especially at the initial presentation, but provides very high negative predictive values and is thus useful for identifying infants unlikely to be infected or monitoring the response to treatment.^{31,32} Use of CRP in neonatal sepsis is complicated by a nonspecific rise that starts shortly after birth.³³⁻³⁵

Conclusion

Haematological scoring system is a simple, quick, readily available effective tool with high sensitivity and specificity in the early diagnosis of neonatal sepsis.

References

1. Wynn J, Cornell TT, Wong HR, Shanley TP. The Host Response to Sepsis and Developmental Impact. *Pediatrics* 2010;**125**:1031–41.
2. Darmstadt GL, Saha SK, Choi Y, Arifeen SE. Population-based incidence and etiology of community-acquired neonatal bacteremia in Mirzapur, Bangladesh: An Observational Study. *J Infect Disease* 2009;**200**:906–15.
3. Rasul CH, Hassan MA, Habibullah M. Neonatal sepsis and use of antibiotic in a tertiary care hospital. *Pakistan J Med Science* 2007;**23**(1) : 78-81.
4. Vergnano S, Sharland M, Kazembe P, Mwansambo C. Neonatal sepsis : an international perspective. *Arch Dis Child Fetal Neonatal Edition* 2005;**90**: 220-2.
5. Dawodu A, Urman ALK, Danso TK. A case control study of neonatal sepsis: Experience from Saudi Arabia, *Journal tropical pediatrics* 1997; **43**: 84-8.
6. Chowdhury AMAK, Rahman M, Karim AQMR. Characteristics of septicemia in newborn in Dhaka Shishu Hospital. *Dhaka Shishu (Children) Hospital Journal* 1998;**14**:9-12.
7. Stoll BJ. Infections of the Neonatal Infant. In: Kliegman RM, Behrman RE, Jenson HB, Stanton BF, editors. Nelson textbook of Pediatrics. 18th ed. Saunders. 2007;794-809.
8. Shirazi H, Riaz S, Tahir R. Role of the Hematological Profile in Early Diagnosis of Neonatal Sepsis. *Ann Pak Inst Med Science* 2010;**6**(3):152-6.
9. Edgar JDM, Gabriel V, Gallimore JR, McMillan SA. A prospective study of the sensitivity, specificity and diagnostic performance of soluble intercellular adhesion molecule, highly sensitive C-reactive protein, soluble E-selectin and serum amyloid A in the diagnosis of neonatal infection. *BMC Pediatrics* 2010;**10**:22-5.
10. Haque KN. Neonatal Sepsis in the Very Low Birth Weight Preterm Infants:Part 2: Review of Definition, Diagnosis and Management. *J Med Science* 2010;**3** (1):11-27.
11. Mishra UK, Jacobs SE, Doyle LW, Garland SM. Newer approaches to the diagnosis of early onset neonatal sepsis. *Arch Dis Child Fetal Neonatal Edition* 2006; **91**:208–12.
12. Fowlie PW, Schmidt B. Diagnostic tests for bacterial infection from birth to 90 days-A systematic review. *Arch Dis Child Fetal Neonatal* 1998;**78**:92-8.
13. Rodwell RL, Leslie AL, Tudehope DI. Early diagnosis of neonatal sepsis using a hematologic scoring system. *J Pediatrics* 1988;**112**:761-7.
14. Saleem M, Shah KI, Cheema SM. Hematological scoring system for early diagnosis of neonatal sepsis. *Journal of Rawalpindi Medical College* 2014;**18**: 68-72.
15. Hussein AB, Khaled MAR. CRP in neonate with suspected septicemia. *Rawal Med Journal* 2007; **32**(1):24-7.
16. Draz NI, Taha SE, Abou-Shady NM. Comparison of broad range DNA PCR to conventional blood culture for diagnosis of sepsis in the newborn. *Egyptian J Med Human Genetics* 2013;**14**(4):403-11.
17. Manucha V, Rusia U, Sikka M, Faridi MMA. Utility of hematological parameters & c-reactive protein in the detection of neonatal sepsis. *J Paediatr Child Health* 2002;**38**:459-64.

18. Polin RA, Parravicini E, Regan PA. Bacterial sepsis and meningitis. In: Robert MK, Hal BJ, editors. *Avery's disease of the new born*. 8th ed. *WB Saunders: Philadelphia* 2005;551-70.
19. Aulia D, Sanjaya AI, Timan IS. The use of immature to total neutrophil (IT) ratio to detect bacteraemia in neonatal sepsis. *J Lab Med Quality Assurance* 2003;**25**(2):237-42.
20. Shankar M J, Agarwal R, Deorari AK. Sepsis in the new born. *Indian J Pediatric* 2008;**75**(3):261- 70.
21. Tripathi S, Malik GK. Neonatal sepsis: Past, present and future. *Internet journal of Medical Update* 2010. (Cited on 10 August 2013).
22. Nandy M, Dutta S, Ganguly S, Paul DK, Bandhopadhyay M. Changing spectrum of neonatal septicemia. *Child New Born* 2007;**11**(1):3-6.
23. Ghosh S, Mittal M, Jaganathan G. Early diagnosis of neonatal sepsis using a hematological scoring system. *Indian J Med Science* 2001;**55**:495-500.
24. Narasimha A, Kumar ML. Significance of Hematological Scoring System (HSS) in early diagnosis of neonatal sepsis. *Indian J Hematol Blood Transfusion* 2011;**27**:14-7.
25. Eissa DS, El-Farrash RA. New insights into thrombopoiesis in neonatal sepsis. *Platelets* 2013;**24**(2):122-8.
26. Arif SH, Ahmad I, Ali SM, Khan HM. Thrombocytopenia and bacterial sepsis in neonates. *Indian J Hematol Blood Transfusion* 2012;**28**(3):147-51.
27. Khair KB, Rahman MA, Sultana T, Roy CK, Rahman MQ, Shahidullah M. Role of hematologic scoring system in early diagnosis of neonatal septicemia. *BSMMU Journal* 2010;**3**:62-7.
28. Murphy K, Weiner J. Use of leukocyte counts in evaluation of early-onset neonatal sepsis. *Pediatr Infect Dis Journal* 2012;**31**:1-4.
29. Buttarello M, Plebani M. Automated blood cell counts: state of the art. *Am J Clinical Pathology* 2008;**130**:104-16.
30. Chiesa C, Panero A, Osborn JF, Simonetti AF, Pacifico L. Diagnosis of neonatal sepsis: a clinical and laboratory challenge. *Clinical Chemistry* 2004;**50**:279-87.
31. Hengst JM. The role of C-reactive protein in the evaluation and management of infants with suspected sepsis. *Advanced Neonatal Care* 2003;**3**: 13-6.
32. Hofer N, Müller W, Resch B. Non-infectious conditions and gestational age influence C-reactive protein values in newborns during the first 3 days of life. *Clin Chem Lab Medicine* 2011;**49**:297-302.
33. Turner MA, Power S, Emmerson AJB. Gestational age and the C reactive protein response. *Arch. Dis. Child. Fetal. Neonatal Edition* 2004;**89**:272-5.
34. Chiesa C, Natale F, Pascone R, Osborn JF, Pacifico L. C- reactive protein and procalcitonin: reference intervals for preterm and term newborns during the early neonatal period. *Clin Chim Acta* 2011;**412**: 1053-9.
35. Hofer N, Zacharias E, Muller W, Resch B. An update on use of C-Reactive Protein in early onset neonatal sepsis: Current insights and new tasks. *Neonatology* 2012;**102**:25-36.

ORIGINAL ARTICLE

Concavo Convex Oblique Anastomosis in The Treatment of Jejunoileal Atresia – Our Experience in a Tertiary Paediatric Hospital In Bangladesh

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Abstract

Background: Jejunoileal atresia is a serious congenital anomaly and one of the leading cause of neonatal intestinal obstruction. It needs operative correction. There are different methods of anastomosis and each method has its merits and demerits.

Objective: The object of the study was to evaluate the outcome concavoconvex oblique anastomosis in jejunoileal atresia.

Methods: This prospective observational study was carried out in the Department of Pediatric Surgery, Dhaka Shishu (Children) Hospital during the period of July 2013 to June 2014. Total 21 patients with Type I, II and IIIA jejunoileal atresia were included in the study. Patients with jejunoileal atresia Type IIIB, IV and atresia complicated by perforation with generalized peritonitis were excluded from the study. Total 21 patients were operated by concavoconvex oblique anastomosis.

Results: Among the study subjects 13 were male, 8 were female with the mean age of presentation 3.23 ± 2.17 days. 7 patients were less than 2 kg during admission and 14 were more than 2 kg. Three (14.28%) developed anastomotic leakage, Two (9.52%) developed functional obstruction and One (4.76%) patient developed wound infection. Three (14.28 %) patients died.

Conclusion: This technique of concavoconvex oblique anastomosis results in wide and early functioning anastomosis which reduced the mortality and morbidity due to fewer angulations, better alignment of adjacent bowel loop, and wider functional anastomotic area resulting in linear flow of effluent.

Key words: Jejuno-ileal atresia, Concavoconvex oblique anastomosis

Introduction

Jejunoileal atresia (JIA) is a serious congenital anomaly of the gut and a common cause of intestinal obstruction in neonates occurring one in approximately 5000 live births.¹

It is widely accepted that an ischemic insult to the developing midgut at some stage during fetal development causes jejunoileal atresia.² Intestinal atresia is the third most common cause of intestinal obstruction after Hirschsprung's disease and

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meconium ileus³ and is found in approximately one fifth of the patients who require major operations in the first two weeks of life.⁴ Atresia refers to a congenital obstruction caused by complete occlusion of the intestinal lumen. This involved 50% in jejunoileal region, 44 % in duodenal and 6% in colonic region.⁸ In Bangladesh, jejuno-ileal atresia is the most common type of intestinal atresia.⁵ Prompt recognition of intestinal atresia is essential for appropriate management to be instituted.^{6,7,8} Decision for surgical option in a stable case of jejunoileal atresia without any complication is towards resection and anastomosis which include different methods from conventional end to back, end to oblique or concavoconvex oblique anastomosis.⁹ In the presence of gangrene and perforation of the proximal intestinal segment in jejunoileal atresia it is often difficult to select better surgical option between primary anastomosis and enterostomy. Immediate postoperative outcome seems to be satisfactory after enterostomy, but subsequent care of stoma and maintenance of nutritional status is so difficult, that most of the survivors not able to reach the next surgical procedure of stomal closure.¹⁰

Other procedures practiced in the treatment of jejunoileal atresia has high mortality and morbidity that's why we decided to do concavoconvex oblique anastomosis in the treatment of non complicated jejunoileal atresia Type I, II and IIIA and observed the outcome.

Methods

This prospective observational study was conducted in the department of Paediatric Surgery, Dhaka Shishu (Children) Hospital, Dhaka during the period of July 2013 to June 2014. Patients were included in the study by pre and per operative findings. We excluded the patients with jejunoileal atresia Type IIIB, IV and atresia complicated by perforation with generalized peritonitis from the study. Total 21 patients with uncomplicated Jejunoileal atresia were operated by concavoconvex oblique anastomosis technique using 5/0 polyglactine (vicryl). After getting written consent from the parents in a preformed consent form, Data were collected by general questionnaire, clinical examination, evaluating preoperative management, operative findings,

postoperative management & follow up. Collected data were arranged in systematic manner and statistical analysis was made.

Pre-operative management

After admission the patients were prepared for operation and initial management given by keeping warm, nothing per oral, gut decompressed by both continuous and intermittent nasogastric suction with IV glucose containing half strength normal saline and antibiotics (ceftazidime, metronidazole, Amikacin / Gentamycin) for covering both Gram Positive , Gram Negative and anaerobic organisms. Injection Vitamin K were given once daily for consecutive 3 days. Plain X-ray abdomen erect posture and baseline laboratory investigations including blood grouping, complete blood count, blood urea, serum creatinine, serum electrolytes and serum total protein with albumin were done. Serum bilirubin (total, direct and indirect) was measured if the baby seems to be icteric.

Operative technique

Laparotomy was done by right transverse supra-umbilical incision. Proximal dilated and distal constricted bowel segments were identified. Proximal distended segment was deflated and 5–10 cm of distended bowel was resected. Margin was cut obliquely starting from antimesenteric border towards mesenteric border making cut margin concave so that the 1 cm difference created between antimesenteric and mesenteric border preserving mesenteric vascular supply. Distal atretic bowel wall was also cut obliquely from mesenteric towards antimesenteric border making cut margin convex creating 1 cm difference between antimesenteric and mesenteric border (Fig 1). Anastomosis was performed in single layer interrupted by 5-0 polyglactine (vicryl). Two or three interrupted stitches were taken commencing from mesenteric border and anastomosis was progressed toward antimesenteric border from opposite direction, thereby completing anastomosis. Last two to three anterior stitches were taken full thickness Lembert, which gives better serosa to serosa approximation. Finally, mesenteric defect was closed. We first approximate mesenteric window of adjacent bowel and closed by inverting interrupted sutures.

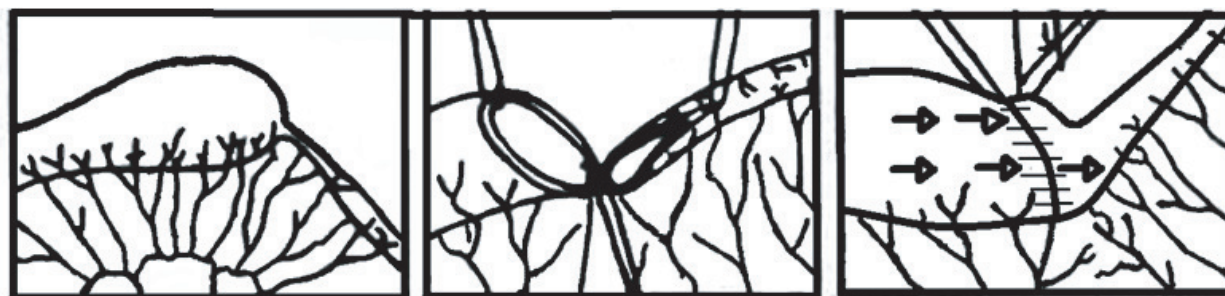


Fig 1 Conventional end to back anastomosis

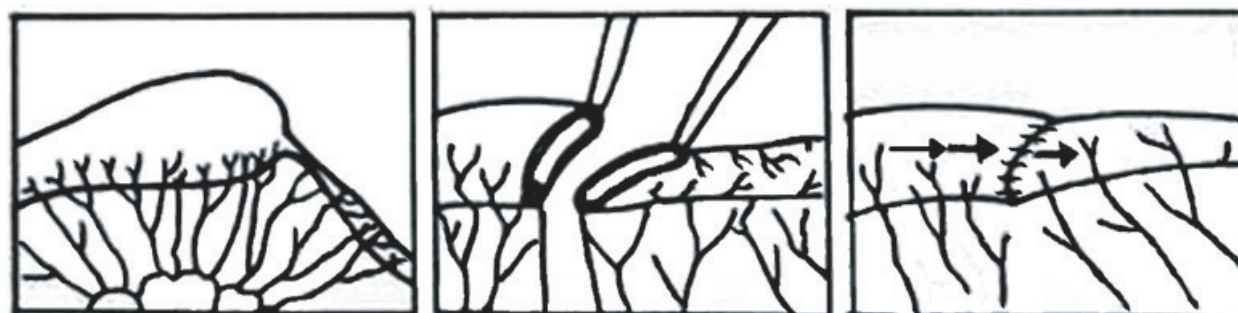


Fig 2 Concavoconvex Oblique anastomosis

Post operative management and follow up

Postoperatively nothing per oral, nasogastric suction, maintenance of IV fluid and antibiotics were continued. Hb% and serum electrolyte were checked. Potential nutritional supplement in the form of amino acid, fatty acid, albumin or blood transfusion was given in some patients. Antibiotics were continued postoperatively for 7 days and in selective cases up to healing of the wound. Measured feed was tried in all cases from 6th POD through NG tube. If the baby tolerated feed then gradually increased and intravenous fluid was reduced. In patients with bilious vomiting or abdominal distension feed was abandoned and kept on nothing per oral and feed was tried again after 24 hours.

Results

A total of 21 patients with Jejunoileal atresia (Type-I,II and IIIA) were included in the study and operated using concavoconvex oblique anastomosis technique. Demographic data (Age, sex, gestational age and weight) of the patients is shown in the Table -1.

Other associated anomalies found in 4 (19%) patients. Among them 1 patient had gastroschisis along with Type II jejunal atresia. 2 patients had B/L TEV and 1 patient had congenital cardiac anomaly (Large ASD). Gastroschisis patient died after operation. Mean day of oral feeding started at 7.12 days (SD: 2.17; range: 6-11days).

Data of post operative complications and mortality listed in the Table-2.

Table I
Demographic Data of the Patients

Variables	Numbers
Age : Range	1- 10 days
Mean \pm SD	3.23 \pm 2.17 days
Sex : Male	13 (61.9%)
Female	08 (38.1%)
Ratio	1.63 : 1
Gestational Age: Preterm	06 (28.6%)
Term	15 (71.4%)
Weight : < 2kg	07 (33.3%)
>2 kg	14 (66.7%)

Table II
Overall complications of the patients

Complication	Number (%)
Anastomotic leakage	3 (14.28%)
Functional Obstruction	2 (9.52%)
Wound Infection	1 (4.76%)
Persistent Jaundice > 2 wks	1 (4.76%)
Mortality	3 (19.04%)

After establishment of feeding two patients developed functional obstruction which was manifested by one to two episodes of bilious vomiting and treated with nothing per oral, I/V fluid ($\frac{1}{2}$ strength normal saline with 10 % dextrose) for transient period of 24 hours. Patients who developed anastomotic leakage, 2 of 3 died and other case of anastomotic leakage was treated with ileostomy. Total mortality was 3 (14.28%) in this series.

Discussion

Though factors affecting the survival of patients with jejunoileal atresia are gastrational age, age at presentation, birth weight, maturity of the baby, types and site of atresia, pneumonia, septicaemia, availability of NICU and finally type of treatment offered were the variables correlated with the short term out come and survival of the patients with jejuno-ileal atresia.¹¹

Majorities of child birth still occurred at home in Bangladesh means that, delay in presentation and surprising miss interpretation of passage of mucous as meconium by the parents and even by the primary health workers-is inevitable, which found to be a significant factors for mortality. Mean age of entire study group was 3.23 ± 2.17 days where as 4 patients who died; their mean age was 5.32 ± 1.68 days. This findings support our observation that delay in presentation increases the mortality rate. Our finding is very much similar with the result of Shahjahan et al. 2013.¹²

The more delay to present to hospital and diagnosis, the graver the prognosis would be. Mortality was also found to be associated with location and types of atresia. The more distal the lesion, the more are the chances of survival.¹¹

Many anastomotic techniques have been introduced for intestinal atresia. The procedures can be classified into two types: (1) widening of caliber of the diminutive distal bowel and (2) reducing the caliber of the large proximal bowel. End to back, end to side, and end to end oblique anastomosis that is described belong to first type, and tapering enteroplasty¹³ followed by end to end anastomosis into the second type.

End-to-back anastomosis^{2,14} shows neither technical problems nor post-operative anastomotic functional obstruction if the caliber ratio between the proximal and distal atretic bowels is not large. However, as

the caliber ratio increases longitudinal axis deviation between proximal and distal bowels gradually becomes close to 90° , resembling end to side anastomosis, which easily results in functional obstruction. It seems to be very difficult to perform functional end-to-back anastomosis in cases where caliber ratio is more than 4.²⁰

Tapering jejunoplasty¹⁵ of the proximal dilated segment followed by construction of end to end jejunostomy has been reported to be useful.^{16,17,18} for large caliber difference. However, this method has disadvantage of bowel dismotility and axial deviation. Another technique described by Patil et al in which linear anastomosis was done excising a circular disc of proximal dilated bowel and anastomosed to narrowed distal bowel in single layer, but in this case there was luminal disparity in proximal and distal segment causing leakage at anastomotic site and increased morbidity and mortality rate.¹⁹

In concavoconvex technique, we cut proximal bowel in concave fashion after 5–10 cm bowel resection and distal bowel in convex fashion and performed anastomosis in interrupted manner with vicryl 5-0 (round body). The resultant anastomosis is oblique and patulous one without bowel angulations. Furthermore, flow of intestinal content is linear one; hence there is less shearing force at anastomotic site. Also, this technique does not require Cheatle's slit. A Cheatle slit is a longitudinal incision into the antimesenteric border of the small intestine. This incision ultimately allows for a wide-caliber elliptical anastomosis to be performed.⁸

Conclusion

This technique results in wide and early functioning anastomosis. The mortality and morbidity rate is reduced. We believed that improved results are due to fewer angulations, better alignment of adjacent bowel loop, and wider functional anastomotic area resulting in linear flow of effluent. So, we recommend this method of bowel anastomosis in cases of jejunoileal atresia as a technical advancement.

References

1. Waldhausen JH, Sawin RS. Improved long-term outcome for patients with jejunoileal apple peel atresia. *J Pediatr Surg* 1997; **32**: 1307-9.

2. Nixon HH and Tawes R. Etiology and treatment of small intestinal atresia: Analysis of a series of 127 jejunoileal atresias and comparison with 62 duodenal atresias. *Surgery* 1970;**69**:41-51.
3. Touloukian RJ. Diagnosis and treatment of jejunoileal atresia. *World J Surg* 1993;**17**:310-7.
4. Spencer R. The various patterns of intestinal atresia. *Surgery* 1968;**64**:661-8.
5. Jafor MA. Congenital intestinal atresia and stenosis: management and outcome, MS thesis, University of Dhaka, 2000;4-80.
6. Adeyemi D. Neonatal intestinal obstruction in a developing tropical country: patterns, problems and prognosis. *J Trop Pediatr* 1989;**35**:66-70.
7. De Lorimier AA, Fonkalsrue EW and Hays DM. Congenital atresia and stenosis of the jejunum and ileum. *Surgery* 1969;**65**:819-27.
8. Grosfeld JL. Jejunoileal atresia and stenosis. In:., O' Neill, J.A., Rowe, M.I., Grosfeld, M.I., Fonkalsrud, E.W., and Coran, editors. *Pediatric Surgery*, 6th Ed. Mosby Year Book, St Louis, 2006;**2**:1059-71.
9. Almoutaz AE. Different Surgical Techniques in Management of Small Intestinal Atresia in High Risk Neonates', *Annals of Pediatric Surgery* January 2009; **5**(1):31-5.
10. Prasad TKS and Bajpai M. Intestinal atresia. *Ind J Pediatr* 2000;**67**:671-8.
11. Shakya VC, Agrawal CS, Shrestha P, Poudel P, Khanya S, Adhikary S. Management of jejunoileal atresias; an experience at estern Nepal. *BioMed Central Surgery* 2010; 10:35. doi:10.1186/1471-2482-10-35. A.G. Mosby, St Louis,
12. Shahjahan M, Ferdous KMN, Mitul AR, Islam K. Management of Jejunoileal Atresia : Our 5 year Experience. *CMOSHM CJ* 2013;**12**(3):52-5.
13. Gray SW, Skandalakis JI. Embryology for surgeons. Philadelphia: Saunders; 1972. p. 151.
14. Gambee LP. A single layer open intestinal anastomosis applicable to the small as well as large intestine. *West J Surg Obstet Gynecol* 1951;**59**:1-8.
15. Howard ER, Othersen HB. Proximal jejunoplasty in the treatment of jejunal atresia. *J Pediatr Surg* 1973;**8**:685-90.
16. Thomas CG, Carter JM. Small intestinal atresia: The critical role of a functioning anastomosis. *Ann Surg* 1974;**179**:663-70.
17. Grosfeld JL, Ballantine TV, Shoemaker R. Operative management of intestinal atresia and stenosis based on pathological finding. *J Pediatr Surg* 1979;**14**:368-75.
18. Patil VK, Kulkarni BK, Jiwane A, Kothari P, Poul S. Intestinal atresia: An end to end anastomotic technique. *Pediatr Surg Int* 2001;**17**:661-3.
19. Evans CH. Atresia of gastrointestinal tract. *Surg Gynecol Obstet* 1951;**92**:61-5.
20. Zaheer H, Gangopadhyay A.N. Punit S, Mohammad Akhtar H. Concavo-convex oblique anastomosis technique for jejunoileal atresia. *J Indian Assoc Pediatr Surg* 2009;**14**(4):207-9.

ORIGINAL ARTICLE

Prediction of Neurodevelopmental Outcome by Cranial Ultrasound in Preterm Neonates in a Tertiary Care Hospital

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Abstract

Background: Preterm infants are at risk for adverse neurodevelopmental outcomes. Cranial ultrasound has been proposed as a means of predicting neurodevelopmental outcomes in this population.

Objective: The present study attempted to correlate cranial ultrasound findings in preterm neonates with neurodevelopmental outcome.

Methods: This prospective cross-sectional comparative study was conducted in Dhaka Shishu (Children) Hospital from October 2011 to March 2012. Total 103 preterm infants were assessed for the associations between cranial ultrasound abnormalities and the risk of neurodevelopmental impairments during neonatal period and at 3 months of age.

Results: Abnormal cranial ultrasound findings (IVH, cerebral edema, ventricular dilation) found in 45.63 percent preterm neonates were predictive of the abnormal neurodevelopmental domains (sensitivity 93.62%, specificity 87.5%, PPV 13.73%, NPV 5.77%) during neonatal period. Among the preterm infants at discharge, 39.8 percent had abnormal primitive reflexes, 40.7 percent had gross motor abnormalities, 31.05 percent had fine motor abnormality, 38.8 percent had vision abnormality, 31.06 percent had hearing abnormality and 38.8 percent had speech abnormality with abnormal cranial USG findings. Again at 3 months of age, 47.5 percent infant had gross motor abnormality, 18.4 percent had fine motor abnormality, 8.7 percent had vision abnormality, and 15.6 percent had hearing abnormality which was strongly correlated with cranial ultrasound abnormalities (sensitivity 93.62%, specificity 89.29%, PPV 12% and NPV 5.66%).

Key words: Preterm; Neonates; Neurodevelopmental assessment; Cranial Ultrasound

Introduction

Preterm infants are at risk for different neurodevelopmental impairments.¹ Advances in the perinatal and neonatal care during the last two decades have improved the prognosis of infants² who are born prematurely. An estimated 20% of infants

born prematurely in Bangladesh and 30% have low birth weight.³ With a total population of >160 million, including >20 million children <5 years of age,⁴ large unrecognized populations may be at risk for neurodevelopmental morbidity particularly considering that 85% of the deliveries occurred at

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home, often with no care; only 7% of births are ever registered; and primary health care services do not include screening for the neurodevelopmentally delayed child. In an epidemiologic survey of disabilities among 7 to 9 years old children in Bangladesh, an estimated 60 to 1000 had some form of disabilities related to motor, vision, hearing, cognitive disabilities and seizure disorder.⁵ With fast declining infant and child mortality rates, smaller family sizes, and increasing emphasis on enrollment into primary school, the issue of “quality of survival” is gaining ground. Methodological issues remain as to how to identify younger children with impairments and disabilities. Yet, there is a growing awareness among parents of the presence of developmental problems early in a child’s life.⁶ Recent reviews of early intervention studies in the high risk populations have demonstrated the potential to improve long-term developmental activities in children across low and high income countries.^{7,8} Without these services, most children with neurodevelopmental impairments are likely to progress to more permanent functional limitation, disabilities and handicaps.⁹ In Bangladesh, this reflected in the rise in the prevalence of children who are at risk for disabilities.^{6,10} Clinicians routinely need to provide parents and caregivers with the prognostic information for their vulnerable infants, and most do this with the aid of some form of neuro-imaging. Cranial ultrasound (CUS) is cheap, safe and can be performed at the cot side by the attending neonatologist or pediatric radiologists. MRI is less widely available, more expensive and requires transportation to an imaging unit.¹¹ It was hypothesized in a study¹² that in infants with normal cranial ultrasound at term age, conventional MRI adds marginally clinical information. This implies that ultrasound can be used as screening method to identify infants with low risk of severe disability and thereby reduce the number of MRI. Cranial ultrasound is the most commonly used brain imaging technique in the neonatal intensive care of preterm infants. It reliably detects major intracranial lesions such as intraventricular hemorrhage (IVH), parenchyma hemorrhagic infarctions or cystic periventricular leukomalacia, all strongly predictive for the development of cerebral palsy (CP) and severe cognitive impairment.^{13,14,15} Some studies¹¹

showed that a normal ultrasound scan provide considerable confidence that an infant will have normal neurodevelopment. Khan NZ, et al,¹⁶ developed the Rapid Neurodevelopmental Assessment (RNDA) to determine functional status in the following domains: primitive reflexes, gross motor, fine motor, vision, hearing, speech, cognition, behavior and seizures.

The objective of the RNDA is to determine a comprehensive assessment procedure for ascertaining neurodevelopmental status of children aged 0 to 24 months for use by multidisciplinary professionals in a developing country. The purpose of this study was to predict neurodevelopmental outcome in preterm infants by cranial ultrasound. This study maybe of paramount importance in early identification and intervention to mitigate neurodevelopmental impairments in large population that live in developing countries where professional expertise is sparse.

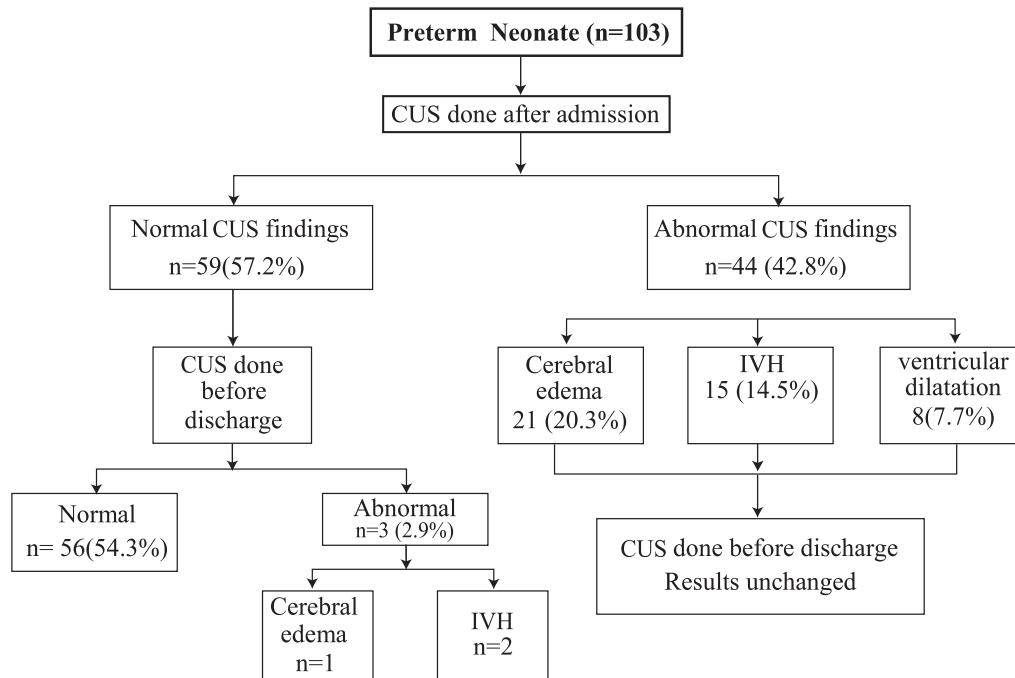
Methods

This prospective cross-sectional study was carried out in Dhaka Shishu (Children) Hospital from October 2011 to March 2012. Preterm neonates (<37 weeks of gestational age) admitted in the neonatal unit were included in the study. Neonates having perinatal asphyxia, RDS, sepsis, congenital anomalies, birth trauma were excluded from the study. All the neonates underwent cranial ultrasound (CUS) scanning after admission and before discharge. Control and cases were sorted out on the basis of normal and abnormal CUS findings respectively and both underwent rapid neurodevelopmental assessment (RNDA). Again both control and cases were followed up at their 3 months of age and RNDA were done. Finally, the findings of CUS were correlated with initial and 3 months age findings of neurodevelopmental assessment by RNDA.

Results

One hundred and three preterm neonates who were admitted in the neonatology unit of Dhaka Shishu (Children) Hospital during the study period from October 2011 to March 2012.

All the enrolled preterm neonates underwent cranial ultrasound (CUS) scanning immediately after admission and before discharge; the results are shown in the algorithm below:



So, the final CUS findings were: normal 56(54.3%), Cerebral edema 22(21.3%), IVH 17(16.5%) and ventricular dilatation 8(7.7%)

Table-I

Distribution of age, gestation age and weight in both groups (n=103)

	Normal cranial USG findings n=56	Abnormal cranial USG findings n=47	P value
Age in days (Mean±SD)	7.67±6.68	7.23±5.75	0.73
Gestational age in weeks (Mean±SD)	31.94±2.03	31.85±1.91	0.82
Weight gm (Mean±SD)	1.83±0.20	1.81±0.17	0.54

The mean age (in days) of control (infants with normal CUS findings) was 7.67 and case (infants with abnormal CUS findings) was 7.23. Mean gestational age (in weeks) in control was 31.94 and in cases was 31.85 and mean weight (in grams) in control was 1.83 & in case was 1.81.

Table- II

Rapid neurodevelopment assessment (RNDA) findings of the preterm neonates (n=103) during neonatal period.

Domain		Disability grade	No. of Preterm neonates
Primitive reflexes	Normal		62 (60.1%)
	Abnormal	Mild	7 (6.7%)
		Moderate Severe	14 (13.5%) 20 (19.4%)
Gross motor	Normal		61 (59.12%)
	Abnormal	Mild	6 (5.7%)
		Moderate Severe	16 (15.3%) 20 (19.7%)
Fine motor		Normal	
	Abnormal	Mild	3 (2.8%)
		Moderate Severe	10 (9.7%) 19 (18.4%)
Vision		Normal	
	Abnormal	Mild	5 (4.8%)
		Moderate Severe	15 (14.8%) 20 (19.4%)
Hearing		Normal	
	Abnormal	Mild	3 (2.8%)
		Moderate Severe	10 (9.7%) 19 (18.4%)
Speech		Normal	
	Abnormal	Mild	6 (5.7%)
		Moderate Severe	13 (12.6%) 21 (20.3%)
Cognition		Normal	
	Abnormal		3 (2.8%)
Behavior	Normal		100 (97%)
	Abnormal		3 (2.8%)
Seizure	Absent		100 (97%)
	Present		3 (2.8%)

One hundred and three preterm neonates underwent RNDA during neonatal period. Among these, abnormal domains found in no. of neonates are as follow: Primitive reflex- 41(39.7%), gross motor- 42 (40.7%), fine motor -32(31%), vision-40 (38.7%), hearing-32 (31%), speech-40 (38.7%), cognition- 3 (2.8%), behavior- 3 (2.8%), seizure 3 (2.8%).

Table-III

Association between neurodevelopmental findings (individual domain) at neonatal age and cranial USG findings (n=103)

		Normal cranial USG findings n=56	Abnormal cranial USG findings n=47	P*
Primitive reflexes	Abnormal	2	39	0.000
	Normal	54	8	
Gross motor	Abnormal	2	40	0.000
	Normal	54	7	
Fine Motor	Abnormal	2	30	0.000
	Normal	54	17	
Vision	Abnormal	2	38	0.01
	Normal	54	9	
Hearing	Abnormal	2	30	0.000
	Normal	54	17	
Speech	Abnormal	2	38	0.01
	Normal	54	9	
Cognition	Abnormal	2	1	0.56
	Normal	54	46	
Behavior	Abnormal	2	1	0.56
	Normal	54	46	
Seizure	Present		3	0.15
	Absent		100	

* χ^2 test

Abnormal primitive reflexes (P<0.05), gross motor (P<0.05), fine motor (P<0.05), vision (P<0.05), hearing (P<0.05) and speech (P<0.05) at discharge are strongly associated with abnormal cranial ultrasound findings.

Table-IV

Association between neurodevelopment outcome at neonatal age and cranial USG findings (n=103)

Neurological deficit	Normal cranial USG findings n=56	Abnormal cranial USG findings n=47	P*
Present	7	44	0.000
Absent	49	3	

* χ^2 test

Neurological deficit found by RNDA at discharge is strongly associated (P<0.05) with abnormal cranial USG findings.

Table- V

Accuracy of cranial USG findings in preterm neonates for prediction of neurodevelopmental abnormality at neonatal period (n=103)

Abnormality in cranial USG findings	Neurodevelopmental impairment		Total
	Present	Absent	
Present	44	3	47
Absent	7	49	56

Calculated sensitivity of cranial USG in prediction of neurodevelopmental impairment is 93.62%, specificity 87.5%, PPV 13.73% and NPV 5.77%.

Table-VI

Neurodevelopmental findings of preterm infants at 3 months of age (n=103)

Domain		No. of infants	Percentage of infants
GrossMotor	Abnormal	49	47.5
	Normal	54	52.5
FineMotor	Abnormal	19	18.4
	Normal	84	81.6
Vision	Abnormal	9	8.7
	Normal	94	91.3
Hearing	Abnormal	16	15.6
	Normal	87	84.4
Speech	Abnormal	15	14.5
	Normal	88	85.5
Cognition	Abnormal	6	5.8
	Normal	97	94.2
Behavior	Abnormal	11	10
	Normal	92	90
Seizure	Present	3	2.9
	Absent	100	97.1

One hundred & three enrolled preterm infants came for follow up at their 3 months of age underwent rapid neurodevelopmental assessment and the number of infants with abnormal domains are as follow: abnormal motor reflex- 49(47.5%), fine motor- 19(18.4%), vision- 9(8.7%), hearing- 16(15.5%), speech- 15(14.5%), cognition- 6(5.8%), behavior- 11(10%) and 3(2.9%) infants had seizure.

Table-VII

Association between neurodevelopmental findings (individual domain) at 3 months age and cranial ultrasound findings (n=103)

		Normal cranial USG findings n=56	Abnormal cranial USG findings n=47	P*
Gross motor	Abnormal	5	44	0.000
	Normal	51	3	
Fine Motor	Abnormal	4	15	0.001
	Normal	52	32	
Vision	Abnormal	2	7	0.04
	Normal	54	40	
Hearing	Abnormal	2	14	0.000
	Normal	54	33	
Speech	Abnormal	2	13	0.001
	Normal	54	34	
Cognition	Abnormal	5	1	0.14
	Normal	51	46	
Behavior	Abnormal	5	6	0.37
	Normal	51	41	
Seizure	Present	3	0	0.15
	Absent	53	47	

* χ^2 test

Strong association between neurodevelopmental findings at 3 months age and cranial ultrasound findings was found in following domains: gross-motor (P<0.05), fine motor (P<0.05), vision (P<0.05), hearing (P<0.05), speech (P<0.05).

Table-VIII

Association between neurodevelopmental outcome at 3 months age and cranial USG findings.

Neurological deficit	Normal cranial USG findings n=56	Abnormal cranial USG findings n=47	P*
Present	6	44	0.000
Absent	50	3	

* χ^2 test

Neurological deficits found by RNDA at 3 months of age is strongly associated (P<0.05) with abnormal cranial USG findings.

Table-IX

Accuracy between cranial USG findings in preterm infants for prediction of neurodevelopmental abnormality at 3 months of age (n=103)

Abnormality in cranial USG findings	Neurodevelopmental impairment		Total
	Present	Absent	
Present	44	3	47
Absent	6	50	56

Calculated sensitivity of cranial USG in prediction of neurodevelopmental impairment is 93.62%, specificity 89.29%, PPV 12% and NPV 5.66%.

Table-XI

Association between different cranial USG findings and neurodevelopmental outcome at 3 months of age (n=103)

		Neurological deficit present n=56	Neurological deficit absent n=47	P*
IVH	Present	16	1	0.001
	Absent	40	46	
Cerebral edema	Present	20	2	0.000
	Absent	36	45	
Ventricular dilatation	Present	8	0	0.02
	Absent	42	53	

* χ^2 test

Strong association is found neurological deficits with intraventricular hemorrhage ($P<0.05$), Cerebral edema ($P<0.05$) and ventricular dilatation ($P<0$).

Discussion

Children born prematurely are at risk for major and minor neurodevelopmental disabilities. Prediction of outcome is of significance in the neonatal period, when therapeutic intervention is introduced. A detailed neurologic examination in infancy is a valuable tool for predictive outcome, and neuroimaging assessment likewise can be critical in predicting outcome. Cranial ultrasound plays an important role as a bedside tool that can assess the different brain lesions in the preterm neonates. This study was performed in order to predict the neurodevelopmental outcome at 3 months of age in preterm neonates by cranial ultrasound (CUS). This study was conducted in order to correlate cranial ultrasound findings during neonatal period with neurodevelopmental impairment during neonatal period at 3 months of age in order to predict neurodevelopmental outcome by CUS

In this study, the mean age (in days) of the control (infants with normal CUS findings) was 7.6 and that of case (infants with abnormal CUS) was 7.23. Mean gestational age (in weeks) in control was 31.94 and in case it was 31.85. The mean weight (in grams) in control was 1.83 and in case was 1.81. We found more male (62.1%) than female (37.9%) in the study population ($n=103$). In one study,¹⁷ gestational age (in weeks) was 29 ± 0.9 which is lower than those of this study. The explanation may be that prompt referral to larger hospital with better management of preterm neonates in comparison to our health facilities may lead to survival of large number of very preterm neonates with lower birth weight.

Out of 103 preterm neonates, 54.3% (56) had normal CUS findings and 45.63% (47) had abnormal CUS findings. Among the CUS findings, 21.36% (22) had cerebral edema, 16.5% (17) had intraventricular hemorrhage (IVH) and 7.77% (8) had ventricular dilatation. In one study,¹³ CUS lesions were detected in the majority of infants (92%) born <32 weeks of gestation. Another study¹⁷ suggested that the incidence of IVH in preterm infants of less than 1500 gms, ranges from 20% to 40%. Other study¹⁸ conducted in 129 high risk preterm infants, 51.63%

(66) had abnormal CUS findings of which 41.08% (53) had IVH, 9.09% (6) had ventricular dilatation.

Among the 103 preterm neonates who underwent rapid neurodevelopmental assessment (RNDA) at discharge, number of infants having abnormal domains are as follow: primitive reflexes 41(39.8%), gross motor 42 (40.7%), fine motor 32(31.05%), vision 40(38.8%), hearing 32(31.06%), speech 40(38.8%), cognition 3 (2.9%), behavior 3 (2.9%) and 5 (4.85%) had seizure.

For each neurodevelopmental domain, we computed the proportion of preterm neonates with CUS findings (both normal and abnormal). Significant association of abnormal cranial ultrasound was found with following neurodevelopmental domains at discharge: Primitive reflex ($P<0.000$), gross motor ($P<0.000$), fine motor ($P<0.000$), vision ($P<0.01$), hearing ($P<0.000$) and speech ($P<0.001$). Tests for accuracy of cranial ultrasound findings were done for prediction of neurodevelopmental abnormality which revealed sensitivity of CUS 93.6%, specificity 87.5%, PPV 13.73% and NPV 5.77%. We have found that CUS abnormalities are more strongly associated with abnormal developmental domains at discharge (neonatal period). We could not compare our results with those of other studies due to paucity of study.

Follow up neurodevelopmental assessment was done of the 103 preterm infants by RNDA at 3 months of age. Among them, number of infants having abnormal developmental domains are: gross motor 49(47.5%), fine motor 19(18.4%), vision 9(8.7%), hearing 16(15.5%), speech 15(14.5%), cognition 6(5.8%), behavior 11(10%) and 3(2.9%) had seizure. These results are comparable to some studies conducted on preterm low birth weight babies of their neurodevelopmental status at their early infancy.^{20,21}

Number of infants having single neurodevelopmental domain affected are as follow: gross motor 6(5.8%), fine motor 3(2.9%), Vision 1(0.97%), hearing 2(1.9%), speech 2(1.9%). More than one domain affected in 36(34.9%) infants. The single largest category of NDI was also gross motor abnormality which is greater at 3 months of age than previously in neonatal period (at discharge). In contrast, other developmental domains abnormality have come down to lower level. Some studies^{19,20,21} showed that motor performance of children born prematurely is affected in 20%-

40%, which is comparable to our study. In an old study,²² NDA assessments at 20 and 30 months of age disclosed significantly lower Bayley Motor development scores in preterm infants.

For correlation of cranial ultrasound findings with neurodevelopmental domain at 3 months of age, we did Chi-square test. We found, strong association between abnormal cUS findings with the following abnormal neurodevelopmental domains: gross motor ($P < 0.000$), fine motor ($P < 0.001$), vision (< 0.04), hearing ($P < 0.000$), speech ($P < 0.001$). Again, no association between CUS finding and cognition, behavior and seizure was found at 3 months of age as those at discharge (neonatal period).

Accuracy test of CUS for prediction of neurodevelopmental abnormalities at 3 months of age showed: sensitivity 93.62%, specificity 89.2%, PPV 12% and NPV 5.66%. This result also reflected the same predictive value of CUS in predicting neurodevelopmental impairment (NDI) at neonatal period in preterm infants. Finally, individual cUS finding was correlated with presence or absence NDI at 3 months of age which showed strong predictive value of IVH ($P < 0.001$), cerebral edema ($P < 0.000$), ventricular dilation ($P < 0.02$).

One study²³ showed that ultrasound abnormalities were strongly associated with delayed psychomotor development than delayed mental development. The finding of ventriculomegaly and the diagnosis of periventricular hemorrhagic infarction were associated with more modest increases in risk at 2 years of age.

Some other studies²² also have found that ultrasound abnormalities are associated with a two to fourfold increase in the risk of low BSID-II scores and mental retardation.

Another study²⁷ revealed that major abnormalities on CUS predict the development of cerebral palsy (CP) and neuromotor delay at variable ages of follow up.

Although these comparisons are not directly relevant to our study, but can support this study to some extent in predicting NDI at 3 months of age by CUS at perinatal period. Hopefully, future studies in early months of life could be of comparable to our study.

Data from this study states that cranial ultrasound imaging in preterm neonates has strong predictive

value in future neurodevelopmental outcome in preterm neonates which in the near future may be validated by other studies.

The strength of the study includes prospective nature and having two groups for comparison. The limitations of the study include single centre, short period, small sample size and not comparing with other modalities like CT scan, MRI, etc.

Conclusion

Preterm neonates having abnormal cranial ultrasound findings like intraventricular hemorrhage (IVH), cerebral edema, ventricular dilation have strong association with abnormal neurodevelopmental domains such as primitive reflex (during neonatal period), gross motor, fine motor, vision, hearing, and speech both at neonatal period and at 3 months of age. Our findings suggest that the identification of early cerebral abnormalities with the use of cranial ultrasound should offer a valuable complement to other neonatal and psychological risk factors in improving identification of preterm infants at risk for subsequent neurodevelopmental impairment.

References

1. El-Dib M, Massaro AN, Bulas D. Neuroimaging and neurodevelopmental outcome of premature infants. *Am J Perinatal* 2010;**27**(10):803-18.
2. Kstra RE, Ferra TB. Survival and Long Term Neurodevelopmental Outcome of Extremely Premature Infants Born at 23-26 Weeks Gestational Age at a Tertiary Care Centre. *Pediatrics* 2004;**113**:e1
3. Saving Newborn Lives. State of the World's Newborn. Washington DC: Save the Children Federation; 2001: 1-4
4. United Nations Children's Fund. State of the World's Children: Childhood Under Threat. New York, NY; *United Nations Children's Fund*; 2005.
5. Khan N, Durkin M. Framework: Prevalence. In: Zinkin P, Mc Conachie H, editors. *Disabled Children and Developing Countries: Clinics in Development Medicine*, London, United Kingdom: Mackeith Press; 1995:1-9.
6. Khan NZ, Darmstadt GL. Neuro-developmental Outcomes of Preterm Infants in Bangladesh. *Pediatrics* 2006;**118**:280-9.
7. Gregar GMC S, Cheung-YB S, Cueto S. Developmental potential in the first 5 years for children in developing countries. *Lancet* 2007;**369**:60-70.

8. Maulik PK, Darmstadt GL. Community-based interventions to optimize early childhood development in low resource settings. *J Perinatal* 2009;**29**:531-42.
9. International Classification of Functioning, Disability and Health. Geneva, Switzerland: World Health Organization; 2001.
10. UNICEF. Monitoring Child Disability in Developing Countries: Results From the Multiple Indicator Cluster Surveys. New York, NY: United Nations Children's Fund, Division of Policy and Practice; 2008.
11. Nongena P, Ederies A, Azzopardi DV. Confidence in the prediction of neurodevelopmental outcome by cranial ultrasound and MRI in preterm infants. *Arch Dis Child Fetal Neonatal Ed* 2010;**95**:38.
12. Horsch S, Skiold B, Hallberg B. Cranial ultrasound and MRI at term age in extremely preterm infants. *Arch Dis Child Fetal Neonatal Ed* 2010;**95**:310-14.
13. De Vries LS, Haastert V, Rodemaker KJ. Ultrasound abnormalities preceding cerebral palsy in high-risk preterm infants. *J Pediatr* 2004; 144: 815-20.
14. O' Shea TM, Kuban KC, Allred EN. Neonatal cranial ultrasound lesions and developmental delays at 2 years of age among extremely low gestational age children. *Pediatrics* 2008;**122**:662-9.
15. Maalonf EF, Duggan PJ, Counsell SJ. Comparison of the findings on cranial ultrasound and magnetic resonance imaging in preterm infants *Pediatrics* 2001;**107**:719-27.
16. KHAN nz. Validation of Rapid Neurodevelopmental Assessment for under-two-year old children in Bangladesh.
17. Van de Bor M, Ouden LD, Guit Gerard L. Value of Cranial Ultrasound & MRI in Predicting Outcome in Preterm infants, *Pediatrics* 1992;**90** ;196.
18. Hankaran S, Bauer CR, Bain R. Prenatal and prenatal risk and protective factors for neonatal intracranial hemorrhage. *Arch Pediatr Adoles Med* 1996;**50**:491-96.
19. W Baets, M Meradji. Clinical ultrasound in preterm infants: long term follow-up. *Arch Child* 1985;**60**:702-05.
20. Hack M, Taylor HG, Drotar D. Chronic conditions, functional limitations, and special health care needs of school aged children born with low-birth weight in the 1990s. *JAMA* 2005;**194**:318-25.
21. Foulder-Huges LA, Cook RW. Motor, cognitive, and behavioral disorders in children born very preterm. *Dev Med Child Neurol* 2003;**45**:97-103 .
22. Jangmans M, Mercuri E, de Vries L. Minor neurological signs and perceptual motor difficulties in prematurely born children. *Arch Dis Child Fetal Neonatal Ed* 1997;**76**:9-14.
23. Graziani LZ, Posto M. Cranial ultrasound and clinical studies in preterm infants. *The Journal of Pediatrics* 1985;**106**:269-76.
24. Bohr BR, Weight LL, Dusick AM. Neurodevelopmental and functional outcomes of extremely low birth weight infants in the Neonatal Institute of Child Health and Human and Human Development. Neonatal Research Network, 1993-94. *Pediatrics* 2000;**105**:1216-26.
25. Whitaker AH, Feldman JF, Van, Rossem. Neonatal cranial ultrasound abnormalities in low birth weight infants: relation to cognition out comes at six years of age. *Pediatrics* 1998;**98**:719-29.
26. Van Bel F, den Ouden L, Van de Bor. Cerebral blood flow velocity during the first week of life of preterm infants and neurodevelopment at two years. *Dev Med Child Neurol* 1989;**31**:320-28.
27. Ancel PY, Livinee F, Larroque B. Cerebral palsy among preterm children in relation to gestational age and neonatal ultrasound abnormalities: the EPIPAGE Cohort study. *Pediatrics* 2006;**117**:828-35.

ORIGINAL ARTICLE

Cancer in Children – A Situation Analysis at Dhaka Shishu (Children) Hospital

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Abstract

Background: Childhood cancer burden is increasing day by day worldwide and now a day it is an emerging concern in developing countries also. Bangladesh, a developing country, has achieved appreciable reduction in infant and under-5 mortality but incidence of cancers in children is increasing at startling rate. But unfortunately there is very limited published data on the frequency and distribution of childhood cancers at national level in our country.

Objective: This study was undertaken to determine the current pattern of cancer among children at Dhaka Shishu (Children) Hospital.

Methods: It was a prospective study conducted at department of Pediatric Hematology and Oncology at Dhaka Shishu (Children) Hospital from January 2011 to December 2013. All children (0 day to 14 year of age) were included in the study when the diagnosis of malignancy was established. Consideration was given to total number of cases, age at presentation, sex distribution and type of cancers.

Results: Total 528 children (133,173 and 222 during the year of 2011, 2012 and 2013 respectively) were diagnosed as cancer during the study period. Among them male were 332(62.87%) and female were 196(37.13%) with a male female ratio of 1.7:1. Eighty percent of the total childhood cancer was Leukemia and 20% was solid Tumors. Most common cancer was Acute Lymphoblastic Leukemia (69.7%). Neuroblastoma was found the most common (4.5%) solid tumor during the study period.

Conclusion: The number of children diagnosed with cancers at Dhaka Shishu (Children) Hospital is increasing radically. ALL is found the commonest cancer among children along with Neuroblastoma is the most common solid tumor.

Key words: Cancer, children, Leukaemia, Neuroblastoma

Introduction

Childhood cancer encompasses, in general, the cases of cancer found in the cohort of 0–14 year-old children; however, some registers also include those found in the 15–19 year-old cohort.¹ Worldwide, the

incidence of childhood cancer is generally between 100–180 per 1,000,000 children/year.² But childhood cancer burden is increasing³ and over 80% of children who develop cancers each year live in low and middle income countries.⁴ Geographic differences in

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childhood cancer incidence rates suggest genetic and environmental influences on disease susceptibility.⁴ Bangladesh, a developing country, has achieved appreciable reduction in infant and under-5 mortality but incidence of cancers in children is increasing at alarming rate. But unfortunately there is very limited published data on the frequency and distribution of childhood cancers at national level. In India cancer is the 9th common cause for the deaths among children between 5 to 14 years of age.⁵ The proportion of childhood cancers relative to all cancers reported by Indian cancer registries varied from 0.8% to 5.8% in boys, and from 0.5% to 3.4% in girls.⁶ A population-based cancer registry is very much crucial for ascertaining the number of childhood cancer cases that develops within a population. But we have no network of cancer registries among children at national level. Current and locally relevant demographic data are also very important factors for development, implementation and evaluation of cancer control strategies.

Objectives

The aim of the present study is to find out the recent pattern of cancer among children seen at Dhaka Shishu (Children) Hospital, the largest referral tertiary care hospital in Bangladesh.

Methods

It was a prospective study carried out for a period of three years from January 2011 to December 2013 at Department of Pediatric Hematology and Oncology of Dhaka Shishu (Children) Hospital, the largest teaching and referral pediatric hospital in Bangladesh. All children (0 day to 14 years of age) were included in the study when the diagnosis of malignancy was established. Diagnosis of cancer was confirmed either by bone marrow aspiration morphology in the case of Leukemia or by histopathological study of tissue biopsy for solid tumors. Immuno-histochemistry and molecular studies were not routinely available. Demographic profiles (name, age, sex) of all children were noted. All the data was recorded on a proforma, and the data entered and analyzed for frequency, percentages and means on SPSS version 12.

After enrolment, all the data related to bone marrow study and tissue biopsy, total number of cases of childhood cancer, age at presentation, sex distribution and type of cancer were noted in a preformed datasheet with structured questionnaire.

Results

Total 528 children were diagnosed as cancer during the study period. Among them male were 332(63%)

and female were 196(37%) with a male female ratio of 1.7:1(Fig.-1)

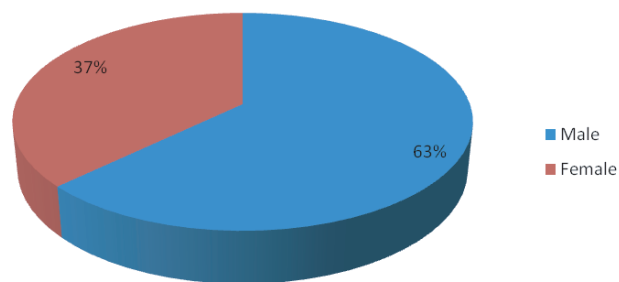


Fig 1 Sex distribution of children having Cancer (n=528)

The age distribution of patients is shown in Table 1. Acute Leukemias (ALL and AML) were commonest in the 5-10 year age group (226/422; 53.8%). We found total two cases diagnosed as infant Leukemia. One of them was diagnosed during neonatal period (22 days old) and he was the youngest patient in the series.

Diagnosis	No	Age range		
		0-4 yr	5-10 yr	>10 yr
Leukemia	422	130	226	66
Solid Tumors	106	56	33	17

Among the total children diagnosed as cancer in DSH Leukemia was found in 80% (422 children) cases and Solid Tumors were in 20% (106 children) cases during the study period. (Fig -2)

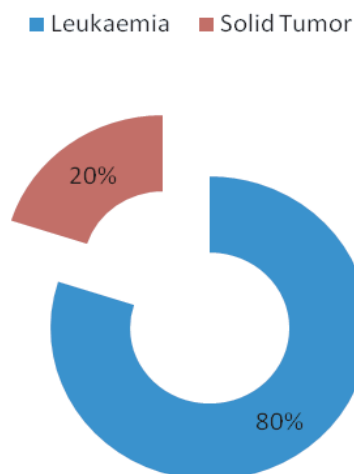


Fig 2 Types of Cancer in children (n=528)

The number of children diagnosed with cancers (specifically Leukemia) at Dhaka Shishu (Children) Hospital was increasing during the study period. Leukemia was found among 105, 136 and 184 children during the year of 2011, 2012 and 2013 respectively whereas the number of diagnosed children with Solid Tumors were 28, 37 and 38 respectively during these three years. (Fig-3)

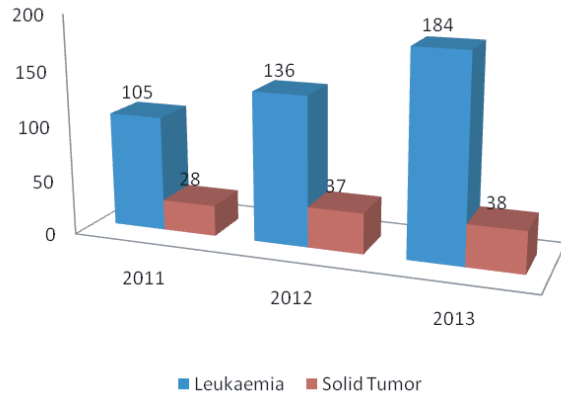


Fig 3 distributions of Leukaemias and Solid Tumors in DSH

Acute Lymphoblastic Leukemia (ALL) was the commonest Childhood Leukemia found during the study period followed by Acute Myeloid Leukemia (AML) and Chronic Myeloid Leukemia (CML) (Fig-4).

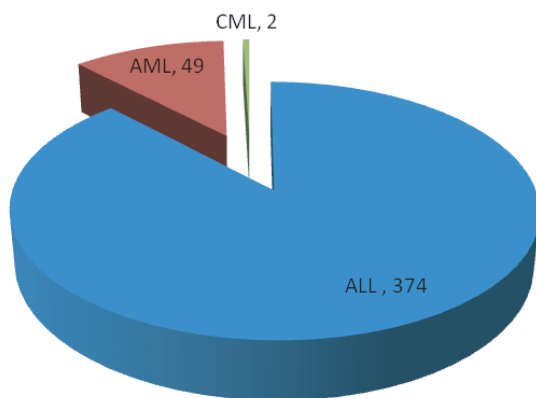


Fig 4 Types of Leukemia in children (n=103)

In DSH Neuroblastoma was found the most common Solid Tumor (29% of total Solid Tumors) in children during the study period. (Table-II)

Table-II
Pattern of Solid Tumors in children (n=103)

Solid Tumor	n=103(%)
Neuroblastoma	29 (22.64)
Wilms Tumor	22 (18.87)
Non-Hodgkin Lymphoma	21 (11.32)
Hodgkin disease	11 (9.43)
Histocytosis	8 (7.55)
Nephroblastoma	5 (1.89)
Retinoblastoma	3 (1.89)
Rhabdomyosarcoma	2 (1.89)
Germ cell Tumor	2 (1.89)

Discussion

Bangladesh, a developing country, lacks a population based childhood cancer registry. In this present study we attempted to determine the present status and recent trends of childhood cancer in our set-up. However, it does not give a true prevalence in the total population. But Dhaka Shishu (Children) Hospital is the largest referral pediatric hospital in Bangladesh. Hence, hospital based data such as ours is an important sources of epidemiologic information for education, planning and further research about childhood cancer in our country.

Over the three years of period, we diagnosed more than five hundred new cases of cancer and our observations, there was obvious gradual increase in number of childhood cancer over the study periods and interestingly, the rate of diagnosis of Leukemia, particularly ALL was increasing radically than solid tumors. It may be due to either a true increase in incidence and/or improvements in clinical and laboratory diagnosis of ALL.

We found that childhood cancers were more common in male children. This finding is similar in India observed by satyanarayana et al⁷ and by Sebefia et al⁸ in Ghana. The M/F ratio observed in our series were consistent with the values reported for other countries.^{1,2,9}

Our data showed that the occurrence of Leukemia was peak among the school age group whereas; in industrialized countries¹⁰ they observed a peak in the incidence of ALL in the preschool age group. In our series only 15.6 % children were older than 10 years who were diagnosed as Leukemia but solid tumors were more common in preschool (0-4yr old) age group.

In this study, the most common childhood cancer was Leukemia. This finding is similar to that reported by satyanarayana et al⁷ in India. For other continents, the data are similar for the children of Denmark, Australia, Hongkong and Singapore² but Sebefia et al⁸ found Lymphoma, predominantly Burkitt's Lymphoma as the commonest cancer in children. Other common cancers found in DSH were Neuroblastoma, Wilms Tumor, Non- Hodgkin Lymphoma and Hodgkin disease in order. CNS tumors are the second commonest childhood tumors in developed countries^{10,11} but in our series Neuroblastoma was the second commonest cancer in children and CNS tumors remained uncommon in our set up most probably due to under diagnosis.

This study has certain limitations. We have not enough facilities for immune-histochemistry and molecular studies to better characterize the tumors that were diagnosed, follow up data of the patients were also not obtained and finally it is a single center study. Further a large, multicenter and population-based study is needed in order to expound the recent trends and true incidence of childhood cancer in Bangladesh.

Conclusion

In Dhaka Shishu (Children) Hospital, ALL is found the commonest cancer among children along with Neuroblastoma is the most common solid tumor. The study observed that the number of children diagnosed with cancers is increasing day by day. So, it needs to give priority for developing a national program to establish a network for detailed and mandatory childhood cancer registry for the whole of the country. It can aid in the planning of future health policies in Bangladesh.

References

1. Ries LAG, Eisner MP, Kosary CL, Hankey BF, Miller BA, Clegg L, Mariotto A, Feuer EJ, Edwards BK, Eds: SEER Cancer Statistics Review, 1975–2002. 2005 [http://seer.cancer.gov/csr/1975_2002/]. Bethesda: National Cancer Institute based on November 2004 SEER data submission, posted to SEER web site 2005.
2. Parkin DM, Kramárová E, Draper GJ, Masuyer E, Michaelis J, Neglia J, Qureshi S, Stiller C. International incidence of childhood cancer Volume II. IARC Scientific Publication No. 144. Lyon: IARC; 1998. (11).
3. Mellstedt H. Cancer initiatives in developing countries. *Ann Oncol* 2006; 17 Suppl 8:viii 24-viii31(1).
4. Howard SC, Metzger ML, William JA, Quintana Y, Pui CH, Robison LL, Ribeiro RC. Childhood cancer epidemiology in low-income countries. *Cancer* 2008;**112**:461-72.
5. Summary- Report on Causes of Death: 2001-03 in India. Available from:URL:[http://censusindia.gov.in/VitalStatistics/Summary Report Death 01 03.pdf](http://censusindia.gov.in/VitalStatistics/SummaryReportDeath_01_03.pdf). (Accessed September 24,2013).
6. Three year report of the population based cancer registries 2009-2011: Report of 25 PBCRs; National Cancer Registry Programme, Indian Council Medical Research, Bangalore2013. Available from:URL: [http://ncrpindia.org/Reports/PBCR 2009 2011.aspx](http://ncrpindia.org/Reports/PBCR_2009_2011.aspx) (Accessed 24th September 2013).
7. Santyanarayana L, Asthana S, Labani SP. Childhood Cancer Incidence in India: A Review of Population-Based Cancer Registries. *Indian Pediatr* 2014;**51**: 218-20.
8. Segbefia CI, Renner LA, Dei-Adomakoh Y, Welbeck J. Changing Pattern of Childhood Cancers at Korle BU Teaching Hospital ACCRA Ghana. *Postgraduate Med J Ghana* 2013;**2**:65-7.
9. Demeocq F, Freycon F, Gembara P, Goubin A, Le Gall E, Pillon P, Sommelet D, Tron I, Lacour B: Cancer incidence among children in France, 1990–1999. *Pediatr Blood Cancer* 2004;**43**:749-57.
10. Li J, Thompson TD, Miller JW, Pollack LA, Stewart SL. Cancer incidence among children and adolescents in the United State, 2001-2003. *Pediatrics* 2008;**121**:1470-77.
11. Peris- Bonet R, Salmeron D, Martinez-Beneito MA, Galceran J, Marcos-Gracer R, Felipe S, Gonzalez V, Sanchez de Toledo Codina J. Childhood cancer incidence and survival in Spain. *Ann Oncol* 2010; **21**: 103-10.

ORIGINAL ARTICLE

Evaluation of EEG In ASD (Autism Spectrum Disorders) And Other Mental Health Disorder Attending A Mental Health Clinic (MHC) In Dhaka Shishu Hospital

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Abstract

Introduction: A proportion of children having EEG abnormalities without any overt seizure may present with behavioral, speech-language and communication disorder. Early detection of the underlying cause is our aim to address the condition effectively and immediately.

Objectives: To evaluate the EEG findings in children with newly diagnosed Autism Spectrum Disorder (ASD) and other mental health disorders attending the MHC.

Methods: A retrospective review of EEGs was done between June to September 2012. Two major diagnostic groups were those with ASDs and those with non-ASD disorders which included ADHD, learning deficit, phobia and anxiety disorders. In both categories EEGs were conducted when there were signs of excessive irritability, hyperactivity, sleep complaints, and any other paroxysmal events. Those with overt seizure disorders were excluded from this study.

Result: A total of 70 children were included, with a male female ratio of 3:1. Overall EEG abnormality revealed in 23% (44% localized epileptiform discharges, 25% generalized epileptiform discharge, 19% focal epileptiform discharge and 12% non-specific dysfunction). Within the two diagnostic categories 14% ASDs showed EEG abnormalities compared to 40% in the non-ASD categories.

Conclusion: The children with behavioural and cognitive and speech difficulties can cause Epileptiform discharges without overt clinical seizures. EEG is a helpful tool for the management of mental health disorders in children.

Key words: EEG, ASD, ADHD, Learning deficit

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Introduction

EEG refers to the recording of the brain's spontaneous electrical activity over a period of time.¹ Electroencephalography was introduced by the psychiatrist Hans Berger of Jena, Germany, in 1929, primarily to study brain dysfunction in mental illnesses.² Nevertheless, it continues to have value in the diagnosis of epilepsy, brain damage, delirium, and dementia and in differentiating brain disease from primary psychiatric disorders.^{3,4} It has been documented that epilepsy is associated with a variety of cognitive and behavioural manifestation.⁵ Some interictal behavioural disturbances may actually represent unrecognized seizures.⁶ In this view EEG is indicated in patients with new-onset psychosis, conditions characterised by rapid changes in mood or behaviour, or conditions characterized by fluctuating or progressive cognitive impairment.⁷ These observations result in the hypothesis that epileptiform discharges without clinical seizures could cause behavioural, cognitive and language impairments in children with autism and other mental health disorder.⁸ Autism is defined in ICD-10 by qualitative impairments in reciprocal social interaction, qualitative impairments in communication and stereotyped/repetitive patterns of interest and activity.⁹ ADHD is defined as a persistent and severe impairment of psychological development, characterised by early onset; a combination of overactive, poorly modulated behaviour with marked inattention and lack of persistent task involvement; and pervasiveness over situations and persistence over time of these behavioural characteristics.⁹ Since the time autism was first described in 1943 by Dr. Leo Kanner, many reports have linked ASD and epilepsy. Seizures are common and occur in approximately 20–30% of patients diagnosed with the more symptomatic subset of individuals with ASD.¹⁰⁻¹² Another studies showed The documentation of epileptiform EEG abnormalities varies from 10.3–72.4% of patients with ASD and in some studies 4–42%.¹¹ The diagnosis of seizure activity in autistic individuals is made more difficult because the behavioural abnormalities associated with complex partial and/or absence seizures (e.g., staring and non-responsiveness with or without repetitive motor behaviours) can all be attributed to the autism and recently, there have also been reports of high rates of epileptiform EEGs in children with autism without a history of seizures or epilepsy.^{13,14} Patterns of EEG

abnormalities include diffuse or generalized, multifocal and focal discharges, unilateral or bilateral and localized to many different brain areas.^{13,15-17} Some studies suggest that temporal abnormalities may be more common,^{13,18} but others do not support this view.¹⁴ The estimated incidences of paroxysmal EEG in ADHD vary between 12-15%¹⁹ to approximately 30%. Note that these individuals did not present with convulsions and thus did not have a clinical diagnosis of epilepsy, but simply exhibited a paroxysmal EEG in the absence of convulsions.²⁰ Another study showed 6.1% of ADHD children have abnormal electroencephalograph (EEG) results, compared to only 3.5% of healthy children.²¹ The prevalence of epilepsy is estimated to lie between 15 and 30% among people with learning disabilities.²² So From this view this study is done with the objective to evaluate the EEG findings in children with newly diagnosed ASD and other mental health disorder at the CDC, Dhaka Shishu Hospital.

Methods

This retrospective observational study was conducted at Paediatric Neuroscience Department of Dhaka Shishu Hospital (DSH) over a period of four months from June 2012 to September 2012. Children were attending at paediatric Neuroscience outpatient department (OPD) of DSH due to some mental health problem like sudden change of behaviour, aggressiveness, hyperactivity, inattention, anxiety, sleep disturbance etc were referred to Mental Health clinic of Child Development Center (CDC). At Mental Health Clinic general developmental assessment includes details history and physical examination was done by consultant physician trained on adolescent and child mental health disease. Children were classified according to ICD 10 criteria like ASDs, ADHD, learning deficit and anxiety disorders.⁹ For diagnostic purposes different screening tools were used like HOW and WHY, M-chat (Modified Checklist for Autism), PIA-CV (The parent interview for Autism-Clinical Version), and SDQ (The Strengths and Difficulties Questionnaire). EEGs were conducted when there were signs of excessive irritability, hyperactivity, sleep complaints, and any other paroxysmal events. Routine electroencephalogram (EEG) (a minimum of 30 minutes including both sleep and awake state with photic stimulation and hyperventilation where possible) was recorded by technicians using a digital EEG machine and reported by paediatric neurophysiologist.

Socioeconomic profile of families was defined as lower income if the monthly earnings were less than 5000 taka, middle income if it was between 5000- <20000 taka, and higher income if it was more than >20000-60000 taka.²³ All these information were kept in a record book at Mental Health clinic (MHC) for each patient. For this study we had searched record from record book and purposively selected the cases who were performed EEG. Subjects were divided into two major diagnostic groups like ASD and Non ASD disorders which include ADHD, learning deficit and anxiety disorder. Those who had history of overt seizure disorders were excluded from this study. Data was analyzed in SPSS version 21.

Results

A total of 70 patients were included during this period, male female ratio was 4:1. Maximum patients came from higher society (n=46) and most of them were <5 yrs (Table I). Among the two major diagnostic group 48 patients (68.6%) were ASD and Non ASD were found in 22 patient(31.4%). Again in Non ASD group 13 patients had ADHD, 6 had learning difficulties, 3 had Anxiety (Fig 1). Overall 16 patients (23%) revealed EEG abnormality. Among

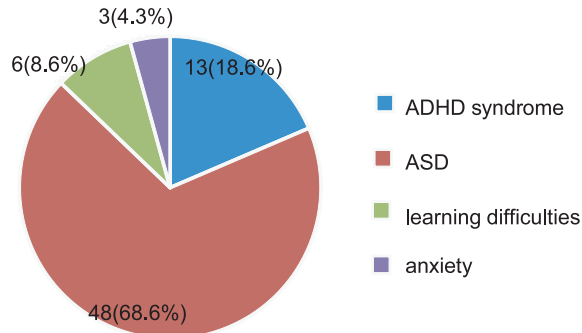


Fig 1 Clinical Diagnosis of study patient

Variable	Clinical Diagnosis		Total (n=70)	Percentage %
	ASD (n=48)	Non ASD (n=22)		
Gender				
Male	37	17	54	77
Female	11	5	16	23
Male:Female	3:1			
Age				
1-5yrs	40	4	44	63
> 5-15yrs	8	17	25	36
>15yrs	0	1	1	1
Family income				
Poor	3	1	4	6
Middle	14	6	20	28
High	31	15	46	66

them 7 patient from ASD, 5 patient from ADHD and 4 from learning difficulties (Table III). localized epileptiform discharges was found in 7 patients (44%), generalized epileptiform discharge 4patients(25%), focal epileptiform discharge 2patients(12%), non-specific dysfunction 3patients (19%) (Fig 2). Within the two diagnostic categories 14% ASDs showed EEG abnormalities compared to 40% in the non-ASD categories (Table-II). Most of the patient from learning difficulties were found EEG abnormalities (66%), compared to others where EEG abnormalities in ASD and ADHD patients were 14% and 38% respectively (Table-II).

Table II
Correlation between clinical diagnosis and EEG findings

Clinical diagnosis	Number n=70	EEG findings		Percentage%
		Normal	Abnormal	
ASD	48	41(86%)	7(14%)	68.6%
ADHD	13	8(62%)	5(38%)	18.6%
Learning difficulties	6	2(34%)	4(66%)	8.6%
Anxiety	3	3(100%)	0(0%)	4.2%
T0tal	70	54(77%)	16(23%)	100%

Table III
EEG findings in study patient

Clinical diagnosis	EEG findings				Total epileptiform discharge N=16
	Localized epileptiform discharge	Generalized epileptiform Discharge	Focal epileptiform discharge	Non specific dysfunction	
ASD	4	0	0	3	7
ADHD	1	3	1	0	5
Learning difficulties	2	1	1	0	4
Anxiety	0	0	0	0	0

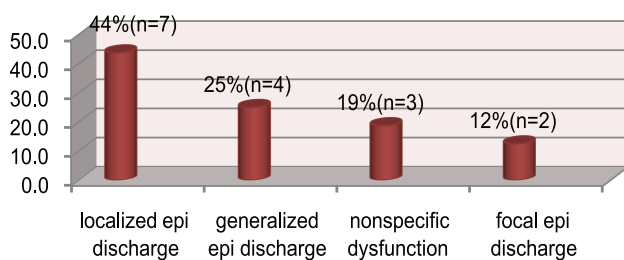


Fig 2 *Types of epileptiform discharge*

Discussion

In sociodemographic profile of this study we found significant gender difference, that is male is more than female with a male female ratio was 3: 1. It is similar to a recent study reported that ASD occurs more commonly in males than females with a ratio of 3 or 4: 1.²⁴ Most of the patient diagnosed as ASD was between 1-5 year of age and mean age was (12 +6) months. This differs from a study of Saudi Arabia where mean age was 8.2 ± 2 with a range from 4 to 11 years.²⁵ In both the diagnostic group most of patient came from high class family, which is similar to a study conducted among individuals with autism found a preponderance of parents from high social class backgrounds, as defined by their occupation, education, or intellectual level.^{26,27} Recent investigations have not observed any association between higher social class and autism.²⁸

Recent reports have suggested that the incidence of autism and related spectrum disorders (ASDs) is substantially higher than previously recognized. The incidence of autistic disorders was 38.9 per 10,000.^{29,30} In this study proportion of ASD was 68.6% (Fig 1) of the total subject. This difference may be due to small sample size of the study which is the limitation of the study. EEG abnormalities were found in 14 patients (23%) in this study, which is

almost similar to a retrospective observational study of clinical EEG in acute psychiatry from 2006, abnormal EEG was identified in 17% of the patients.³¹

Again among total epileptiform EEG (n=16), Localized abnormalities was found in 7 patient (ASD=4, ADHD=1, Learning difficulties=2). This result is similar to a study where Hashimoto et al.³² examined the EEGs of 86 children with ASD. Among them 37 cases had epileptic discharges. Of these 37 patients, 27 had localized spikes.

In this study 14% ASD patient showed EEG abnormalities. This result differs from other study where the authors found epileptiform EEG abnormalities in ASD varies from 10.3–72.4% and in some studies to 4–42%.¹⁴ But the result of this study was close to a study where rossi et al.³³ hughes et al,¹⁵ found 20% of individuals with ASD show epileptiform discharges at rest, typically without the presence of clinical seizures.^{5,33}

In non ASD group we found 38% patient of ADHD and 68% learning difficulties had EEG abnormalities. The result of this study does not support a recent study where the retrospective analysis of 1198 EEG recordings in prepubescent students with LD and ADHD, 218 (18%) had EEG abnormalities.³⁴ where as Jhon and hughes et al,²⁰ found 6.1% of ADHD children have abnormal electroencephalograph (EEG) results, compared to only 3.5% of healthy children. In this study, a large number of patient having learning difficulties and ADHD had EEG abnormalities and for this reason EEG may be a promising diagnostic and assessment tool for these conditions.

Conclusion

Epileptiform discharges without clinical seizures could cause behavioral, cognitive and speech

difficulties. Where there are brief and stereotypical changes of behaviour in patients with psychiatric disorders, epilepsy should be suspected. So EEG is indicated in patients with new-onset psychosis, conditions characterised by rapid changes in mood or behaviour, or conditions characterised by fluctuating or progressive cognitive impairment. EEG is a helpful tool for management of mental health disorders in children.

Limitation

It is a single centre study. Sample size is small.

References

- Niedermeyer E, Da Silva FH. *Electroencephalography: Basic Principles, Clinical applications, and Related Fields*. 5th ed. Lippincott Williams & Wilkins 2004.
- Berger H, Uber das Elektro enkephalogram des Menschen. *Arch Psychiatr Nervenkr* 1929;**87**:527–70.
- Fink M. EEG and behavior: association or disassociation in man? *Integ Psychiatr* 1993; **9**: 108-23.
- Niedermeyer E, Da Silva FH. *Electroencephalography: Basic Principles, Clinical Applications, and Related Fields*. 4 th ed. Baltimore, Williams & Wilkins, 1999.
- Cornaggia CM, Beghi M, Provenzi M, Beghi E. Correlation between cognition and behavior in epilepsy. *Epilepsia* 2006;**47**:34–9.
- Canitano R, Luchetti A, Zappella M. Epilepsy, electroencephalographic abnormalities and regression in children with autism. *J Child Neurol* 2005; **20**: 27-31.
- Sand T, Helene M, Vaaler AE. Is EEG a useful test in psychiatry. *Tidsskr Nor Legerforen* nr2013;**133**:1200–04.
- Tuchman R. Treatment of seizure disorders and EEG abnormalities in children with autism spectrum disorders. *J Autism Dev Disord* 2000;**30**:485-88.
- World Health Organization. *The ICD–10 Classification of Mental and Behavioural Disorders: Diagnostic Criteria for Research*. WHO,1993.
- Kagan-Kushnir T, Roberts W, Snead O. 3rd Screening electroencephalograms in autism spectrum disorders: evidence-based guideline. *J Child Neurol* 2005; **20**:197-206.
- Giovanardi Rossi P, Posar A, Parmeggiani A. Epilepsy in adolescents and young adults with autistic disorder. *Brain Dev* 2000;**22**:102–6.
- Canitano R, Luchetti A, Zappella M. Epilepsy, electroencephalographic abnormalities and regression in children with autism. *J Child Neurol* 2005;**20**:27–31.
- Chez MG, Chang M, Krasne V, Coughlan C, Kominsky M, Schwartz A. Frequency of epileptiform EEG abnormalities in a sequential screening of autistic patients with no known clinical epilepsy from 1996 to 2005. *Epilepsy Behav* 2006;**8**:267–71.
- Kim HL, Donnelly JH, Tournay AE, Book TM, Filipek P. Absence of seizures despite high prevalence of epileptiform EEG abnormalities in children with autism monitored in a tertiary care center. *Epilepsia* 2006;**47**:394–98.
- Hughes JR, Melyn M. EEG and seizures in autistic children and adolescents: further findings with therapeutic implications. *Clin EEG Neurosci* 2005;**36**:15-20.
- Canitano R, Luchetti A, Zappella M. Epilepsy, Electroencephalographic abnormalities, and regression in children with autism. *J Child Neurol* 2005;**20**:27–31.
- Gabis L, Pomeroy J , Andriola MR. Autism and epilepsy: cause, consequence, comorbidity, or coincidence? *Epilepsy Behav* 2005;**7**:652–56.
- Baird G, Robinson RO, Boyd S, Charman T. Sleep electroencephalograms in young children with autism with and without regression. *Dev Med Child Neurol* 2006;**48**:604–08.
- Hemmer SA, Pasternak JF, Zecker SG, Trommer BL. Stimulant therapy and seizure risk in children with ADHD. *Pediatr Neurol* 2000;**24**:99-102.
- Hughes JR, Leo AD, Michelle A. The Electroencephalogram in Attention Deficit–Hyperactivity Disorder: Emphasis on Epileptiform Discharges. *Epilepsy & Behavior, Elsevier* 2000; **1**:271–77.
- Richer LP, Shevell MI, Rosenblatt BR .Epileptiform abnormalities in children with Attention deficit -hyperactivity disorder. *Pediatr Neurol* 2002;**26**:125–29.
- National Institute for Health and Clinical Excellence (NICE), 2012. *The epilepsies: The diagnosis and management of the epilepsies in adults and children in primary and secondary care. [CG137]*. London: NICE.

23. The United Nations *Children's* Fund. Statistic- State of the children of the world. UNICEF, 2007.
24. Fombonne E. Epidemiological surveys of autism and other pervasive developmental disorders: an update. *J Autism Dev Disord* 2003;**33**:365–82.
25. Mostafa A, Waleed A, Hatem H. Sociodemographic factors in Arab children with Autism Spectrum Disorders. *Pan Afr Med J* 2012;**13**:65.
26. Creak M. Families of psychotic children. *Child Psychol Psychiatry* 1960;**1**:156-75.
27. Victor L. Epidemiology of autistic conditions in young children: Some characteristics of the parents and children. *Social Psychiatry* 1966;**1**:124–37.
28. Thomas A, Christina D, Per F. Autism in 3–6 year old children in a suburb of Goteborg, Sweden. *Autism* 1997;**1**:163–71.
29. Gillian B, Emily S, Andrew P. Incidence of the autism spectrum in a population cohort of children in South Thames: The Special Needs and Autism Project (SNAP) *Lancet* 2006;**368**:210–15.
30. Hazel G, Áine M, Howard M. Mental health of children and young people in Great Britain, London: Office for National Statistics, Palgrave Macmillan (01256 329242), 2005.
31. O'Sullivan SS, Mullins GM, Cassidy EM et al. The role of the standard EEG in clinical psychiatry. *Hum Psychopharmacol* 2006; **21**:265 – 71.
32. Hashimoto T, Sasaki M, Sugai K, Hanaoka S, Fukumizu M, and Kato T. Paroxysmal discharges on EEG in young autistic patients are frequent in frontal regions. *J Med. Invest* 2001;**48**:175-80.
33. Rossi PG, Parmeggiani A, Bach V, Santucci M, Visconti P. EEG features and epilepsy in patients with autism. *Brain Dev* 1995;**17**:169–74.
34. Boughton B. EEG may Be Useful for Diagnosis, Assesment of Children with ADD and Learning Disabilities. <http://www.medscape.com/view article/728632> (acssesed September 15, 2010).

ORIGINAL ARTICLE

Improved Sleep and Behavioral Pattern after Adenotonsilectomy in Children with Sleep Disordered Breathing (SDB)

Syed Hasan Imam Al-Masum¹, Ali Jacob Arsalan², Md. Jahangir Alam³, Tanveen Ishaq⁴

Abstract

Background: Sleep disordered breathing (SDB) is common in preschool and early school-aged children and is generally perceived as a neurodevelopmental condition and thereby maltreated resulting in a number of abnormal behavioral complications.

Objective: The present study was intended to evaluate whether adenotonsilectomy performed for sleep-disordered breathing in children is associated with improvements in behavior, cognitive function, and quality of life.

Methods: This prospective interventional study of pre-test post-test design was conducted in Dhaka Shisu(Children) Hospital, Sher-E-Bangla Nagar, Dhaka from January 2008 to December 2011(4-years). The study population was children from 3-8 years undergoing adenotonsilectomy for sleep disordered breathing (SDB) with attention-deficit hyperactivity disorder (ADHD). A total of 80 children fulfilling the inclusion criteria were included in the study.

Results: The participating children were primarily Mean age of the children were 5.6+ 1.6 with a female predominance (58%). Urban and rural residents were fifty-fifty. The lower middle class comprised 40%, followed by poor and middle class (each comprised 24%). Very few were rich and upper class. Snoring, upper airway resistance syndrome, obstructive hypoventilation and Obstructive Sleep-Apnoea Syndrome (OSAS) all demonstrated their significant presence before adenotonsilectomy, which after intervention reduced to minimum ($p < 0.001$). The major behavioral problems in children before intervention were attention-deficit hyperactivity disorder (82%), problematic behaviors on awakening (86%), night-to-night sleep duration variability (78%) and headache (80%). The minor behavioral problems were nocturnal enuresis and (36%) and day time sleepiness (14%). All the major problems responded well following adenotonsilectomy ($p < 0.001$). Among the minor problems incidence of enuresis reduced to less than half ($p = 0.027$). Learning disabilities and poor academic performance showed commendable improvement following adenotonsilectomy. Nearly half (46%) of the children had poor and 52% average quality of life as reported by their parents. After intervention 76% of the children had good quality of life. The difference in quality of the children before and after intervention is statistically significant ($p < 0.001$).

Conclusion: The study concluded that children diagnosed as having SDB experience improvement in both sleep and behavior after adenotonsilectomy.

Key words: Sleep-disordered breathing, attention-deficit hyperactivity disorder, adenotonsilectomy

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Introduction

Sleep disordered breathing (SDB) is a spectrum of sleep related breathing disorders (SRBDs) that includes primary snoring, upper airway resistance syndrome, obstructive hypoventilation, and Obstructive Sleep Apnoea Syndrome (OSAS). SDB is a spectrum of SRBDs that includes primary snoring, upper airway resistance syndrome, obstructive hypoventilation, and OSAS (the most severe aspect of the spectrum of SDB). In children, the term SDB is used more frequently than OSAS because the former term covers a wide range of sleep related breathing disorder. Although the prevalence of OSAS has been reported to range from 0.7% to 3%, the prevalence of snoring and clinical suspicion of SDB in children may approach 11%.^{1,2}

The impact of SDB on childhood development and behavior specifically, hyperactivity and inattention has been well published.³⁻⁷ Even though SDB is not more likely to occur among children with marked symptoms of attention deficit hyperactivity disorder (ADHD), it is highly prevalent among children with mild hyperactive behavior.⁵ SDB is substantially more likely to be found among children with ADHD and it has been suggested that treatment of SDB may eliminate ADHD in a subset of children if their habitual snoring and SDB were alleviated.⁶⁻⁸ In addition to hyperactivity, children with snoring and SDB have been shown to have neuro-cognitive impairment and poor school performance.¹⁰⁻¹³ Children without clinically significant hypoxia, as measured by pulse oximetry, but with habitual snoring have been shown to have poor academic performance than those who do not snore.¹² Sleep-disordered breathing is also associated with nocturnal enuresis, learning disabilities, daytime sleepiness, and somatic complaints such as headaches.¹ Behavioral and emotional difficulties have been found in children with SDB before intervention, and improvements have been reported after adenotonsillectomy.¹⁴

Attention-deficit hyperactivity disorder is a common neurodevelopmental disorder that has been estimated to affect approximately 5.3 % of children and adolescents worldwide¹⁵ and to persist into adulthood in approximately two-thirds of patients.^{16,17} The prevalence of sleep disturbances in individuals with ADHD is reported to be in the range 25–55 %.¹⁸⁻²¹ In a recent Australian study, 62% of children with ADHD had moderate to severe sleep problems and 22% took sleep medications during the one week observation period.²² Indeed, high nocturnal activity and disordered sleep were

defining characteristics of 'hyperkinetic reaction in childhood' or 'attention-deficit disorder' in earlier versions of the DSM.^{23,24}

Attention-deficit hyperactivity disorder is generally perceived as a neurodevelopmental condition potentially resulting from the interaction of multiple genetic and socio-environmental factors exerting via multiple pathways.²⁵⁻²⁸ As such, co-morbidity is not unlikely, and indeed 59–87% of children diagnosed with ADHD have at least one co-morbidity, and 20% have three or more co-morbid conditions or symptoms.²⁹ However, the relationships between sleep and attention-deficit hyperactivity disorder (ADHD) are complex and are routinely overlooked by practitioners. Studies are, therefore, required to investigate the relationship between sleep disordered breathing and its relationship with adenoid and tonsillar enlargement as well as to evaluate whether adenotonsillectomy performed for SDB results in change in behavior, cognitive function, and quality of life. The proposed study is, by far, the first study to address this issue in the context of our country. The data generated from the study is, therefore, of immense significance in the management of children with SDB.

Methods

This prospective interventional study of pre-test post-test design was conducted in ENT Department of Dhaka Shishu Hospital, Sher-e-Bangla Nagar, Dhaka over a period of 4-years from January 2008 to December 2011. The study population was children from 3-10 years undergoing adenotonsillectomy for sleep disordered breathing (SDB) with attention-deficit hyperactivity disorder (ADHD). However, children with SDB with congenital anomalies were excluded from the study. Having permission from the Ethical Committee of Dhaka Shishu Hospital, the study commenced with data collecting from the parents of the selected children (n = 80). The outcome variables were sleep disordered breathing, ADHD, cognitive function and quality of life. The follow up was made between 3-6 months after adeno'tonsillectomy. The test statistics used to analyze the data were descriptive statistics and Chi-square (χ^2) Test. Data presented on categorical scale were compared between children before and after intervention using McNemar Chi-square (χ^2) Test. The level of significance was set at 0.05 and $p < 0.05$ was considered significant.

Results

The present study demonstrated that nearly three-quarters (74%) of the children were 6 or > 6 years

old. Females were a bit higher (58%) than their male counterparts (42%). Half of the patients were rural residents (50%), one-third (34%) urban and the rest 16% were urban slum dwellers. In terms of socioeconomic status lower middle class comprised the main bulk (40%), followed by poor and middle class (each comprised 24%). Very few were rich and upper class (table I).

Table I
Distribution of respondents by their demographic characteristics (n = 50)

Demographic characteristics	Frequency	Percentage
Age (Years)*		
3-6	13	26
≥6	37	74
Sex		
Male	21	42
Female	29	58
Residence		
Rural	25	50
Urban	17	34
Urban slum	8	16
Socioeconomic status		
Poor	12	24
Lower middle class	20	40
Middle class	12	24
Upper middle class	4	8
Rich	2	4

*Mean age = (5.6 ± 1.6) years; range = (3 – 10) years

Snoring, upper airway resistance syndrome, obstructive hypoventilation and obstructive sleep-apnoea syndrome (OSAS) all demonstrated their significant presence before adenotonsilectomy, which after intervention reduced to minimum ($p < 0.001$ in each case) (Table II).

The major behavioral problems in children before intervention were attention-deficit hyperactivity disorder (82%), problematic behaviors on awakening (86%), night-to-night sleep duration variability (78%) and headache (80%). The minor behavioral problems were enuresis (36%) and day time sleepiness (14%). All the major problems responded well following adenotonsilectomy ($p < 0.001$). Of the minor problems incidence of enuresis reduced to less than half ($p = 0.027$) (table III). Cognitive function like learning disabilities and poor academic performance showed commendable improvement following adenotonsilectomy ($p < 0.001$) (table IV). There was dramatic improvement of quality of life after adenotonsilectomy (Fig. 1).

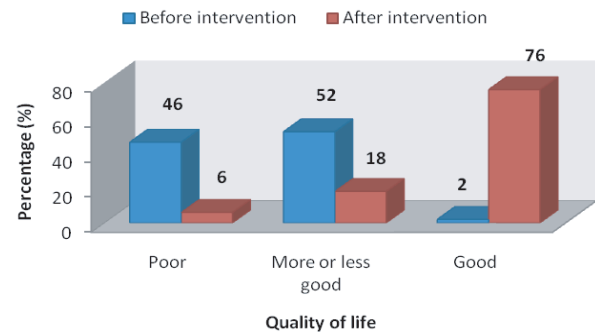


Fig 1 Bar graph showing good quality of life after adenotonsilectomy

Table II
Pattern of sleep breathing in children before & after intervention (n=50)

Pattern of sleep breathing	Group		P-value
	Before intervention (n = 50)	After intervention (n = 50)	
Snoring	43(86.0)	11(22.0)	<0.001
Upper airway resistance syndrome	45(90.0)	10(20.0)	<0.001
Obstructive hypoventilation	43(86.0)	7(14.0)	<0.001
Obstructive sleep apnea syndrome	40(80.0)	8(16.0)	<0.001

Figures in the parentheses indicate corresponding %;

*MacNemar Chi-squared Test (c^2) was done to analyzed the data.

Table III
Behavioral problem between before & after intervention (n=50)

Behavioral problem	Group		P-value
	Before intervention	After intervention	
	(n = 50)	(n = 50)	
Attention-deficit hyperactivity disorder	41(82.0)	9(18.0)	<0.001
Enuresis	18(36.0)	7(14.0)	0.027
Day time sleepiness	7(14.0)	10(20.0)	0.607
Problematic behaviors on awakening	43(86.0)	7(14.0)	<0.001
Night-to-night sleep duration variability	39(78.0)	6(12.0)	<0.001
Headache	40(80.0)	10(20.0)	<0.001

Figures in the parentheses indicate corresponding %;

*MacNemar Chi-squared Test (c^2) was done to analyzed the data.

Table IV
Cognitive function in children before & after intervention (n = 50)

Cognitive function	Group		P-value
	Before intervention	After intervention	
	(n = 50)	(n = 50)	
Learning disabilities	43(86.0)	6(12.0)	< 0.001
Poor academic performance	42(84.0)	7(14.0)	< 0.001

Figures in the parentheses indicate corresponding %;

*MacNemar Chi-squared Test (c^2) was done to analyzed the data.

Discussion

The children of SDB mainly presented (74%) to us at early school age (at 6 or > 6 years) with mean age being 5.6+₋1.6 years. A female predominance was observed in the series. Snoring, upper airway resistance syndrome, obstructive hypoventilation and obstructive sleep-apnoea syndrome (OSAS) all demonstrated their significant presence before adenotonsilectomy. After intervention a substantial proportion of these symptoms reduced ($p < 0.001$ in each case) indicating that adenotonsilitis was the main reason of these symptoms. Poor sleep in children may have negative effects on their daily functioning, mood, behavior and school performance.³⁰⁻³² Moreover, sleep problems have adverse effects on quality of life. In some cases, sleep problems may be a symptom of attention-deficit hyperactivity disorder.^{33,34} The major behavioral problems in children of this study before intervention

was attention-deficit hyperactivity disorder (82%), problematic behaviors on awakening (86%), night-to-night sleep duration variability (78%) and headache (80%). The minor behavioral problems were nocturnal enuresis and (36%) and day time sleepiness (14%). All the major problems responded well following adenotonsilectomy and reduced to bare minimum ($p < 0.001$). Of the minor problems incidence of enuresis reduced to less than half ($p = 0.027$). Cognitive function like learning disabilities and poor academic performance showed commendable improvement following adenotonsilectomy. Nearly half (46%) of the children had poor and 52% average quality of life as reported by their parents. After intervention over three-quarters (76%) of the children had good quality of life which is considered significant and is believed to be result of adenotonsilectomy ($p < 0.001$). Consistent with these findings Julie and associates³⁵ found significant

correlations between sleep and behavior before and after adenotonsillectomy bearing consistency with findings of the present study.

Although polysomnography (PSG) is considered the gold standard for evaluation of OSAS, for select patients PSG testing may not be necessary prior to adenotonsillectomy. There are no clinical practice guidelines regarding the use of PSG and candidacy for adenotonsillectomy, and most otolaryngologists make decisions or recommendations based on medical history that is suggestive of SDB, as well as on physical examination demonstrating adenoid and/or tonsillar hypertrophy. However, predictive accuracy of clinically suspected OSAS may be as low as 30% when PSG testing was performed as well.^{36,37}

It was seen in children with adenotonsillar hypertrophy that adenotonsillectomy is potentially curative for the whole spectrum of obstructive sleep disorders, including primary snoring and upper airway resistance syndrome, which may not show abnormalities on standard PSG.³⁸ In addition to clinical suspicion of SDB, using an instrument such as the Pediatric Sleep Questionnaire (PSQ) may help to identify patients who are appropriate candidates for adenotonsillectomy or those who may have residual SDB or associated symptoms that fail to improve after surgery.

It has been demonstrated that predictive accuracy for SDB is much higher when parental report of multiple measures of behavior is combined with medical history of snoring.³⁸ Therefore, PSQ could be used as a comprehensive parental questionnaire for assessing quality of sleep and the Conners' Parent Rating Scale–Revised Short Form (CPRS-RS) for assessing child behavior. The Conners' scales are the most widely used instruments for assessing child behavior. The CPRS-RS can be used for research, screening, or monitoring treatment effects over time and can also be used as a diagnostic tool in conjunction with other information, such as the short versions of the Conners' Teacher Rating Scale–Revised. Using both teachers' and parents' reports would certainly provide more information regarding the global behavior of the child.

This was a prospective observational study that has demonstrated associations between adenotonsillectomy and improvement in sleep and behavior. Although this study supports a cause-and-effect relationship between SDB and development

of behavioral problems, because it is not randomized or controlled as an intervention trial, this relationship cannot be proven. Without a control group that did not undergo adenotonsillectomy, we cannot prove definitively that the surgical intervention was the cause of the change in behavior following adenotonsillectomy. However, the PSQ and CPRS have been used together in a 4-year prospective cohort study³⁸ demonstrating that symptoms of SDB actually precede development of hyperactivity. Such findings lend validity to our data, which shows that treatment of SDB can lead to amelioration of behavior and sleep problems.

The findings of the present study strongly suggest adenotonsillectomy performed for sleep-disordered breathing in children is associated with improvements in quality of life, behavior, and cognitive function, but large, randomized, controlled studies are needed to provide definitive evidence of the benefits of this commonly performed surgical procedure in the general population.

Conclusion

The sleep disordered behavior (SDB) as, primary snoring, upper airway resistance syndrome, obstructive hypoventilation, and OSAS are not uncommon in paediatric population. Everybody should aware of that the hidden cause of these problems is adenotonsillitis. This study has proved that children diagnosed as having SDB experience significant improvement in both sleep and behavior after adenotonsillectomy.

References

1. De Serres LM, Derkay CSK. Impact of adenotonsillectomy on quality of life in children with obstructive sleep disorders. *Arch Otolaryngol Head Neck Surg* 2002;**128** (5): 489-96.
2. Tran KD, Nguyen CD, Weedon J. Child behavior and quality of life in pediatric obstructive sleep apnea. *Arch Otolaryngol Head Neck Surg* 2005;**131** (1) 52-7.
3. Chervin RD, Archbold KH, Dillon JE et al. Inattention, hyperactivity, and symptoms of sleep-disordered breathing. *Pediatrics* 2002;**109**(3): 449-56.
4. Gottlieb DJ, Vezina RM, Chase C. Symptoms of sleep-disordered breathing in 5-year old children are associated with sleepiness and problem behaviors. *Pediatrics* 2003;**112**(4):870-7.

5. +O'Brien LM, Holbrook CR, Mervis CB. Sleep and neurobehavioral characteristics of 5-7 years old children with parentally reported symptoms of attention-deficit/hyperactivity disorder. *Pediatrics* 2003;**111**(3):554-63.
6. Golan N, Shahar E, Ravid S. Sleep disorders and daytime sleepiness in children with attention-deficit/hyperactive disorder. *Sleep* 2004;**27** (2) 261-6.
7. Huang YS, Chen NH, Li HY. Sleep disorders in Taiwanese children with attention deficit/hyperactivity disorder. *J Sleep Res* 2004;**13**(3):269-77.
8. Chervin RD, Dillon JE, Bassetti C et al. Symptoms of sleep disorders, inattention, and hyperactivity in children. *Sleep* 1997;**20**(12):1185-92.
9. Weatherly RA, Ruzicka DL, Marriott DJ. Polysomnography in children scheduled for adenotonsillectomy. *Otolaryngol Head Neck Surg* 2004;**131**(5):727-31.
10. Blunden SL, ushington K, Kennedy D. Cognitive and behavioural performance in children with sleep-related obstructive breathing disorders. *Sleep Med Rev* 2001;**5**(6): 447-61.
11. Gozal D. Sleep-disordered breathing and school performance in children. *Pediatrics* 1998;**102** (3):616-20.
12. Gozal D, Pope DW. Snoring during early childhood and academic performance at ages thirteen to fourteen years. *Pediatrics* 2001;**107**(6):1394-9.
13. Urschitz MS, Guenther A, Eggebrecht E. Snoring, intermittent hypoxia and academic performance in primary school children. *Am J Respir Crit Care Med* 2003;**168**(4):464-8.
14. Goldstein NA, Post C, Rosenfeld RM. Impact of tonsillectomy and adenoidectomy on child behavior. *Arch Otolaryngol Head Neck Surg* 2000;**126**(4):494-8.
15. Polanczyk G, de Lima MS, Horta BL, Biederman J, Rohde LA. The worldwide prevalence of ADHD: a systematic review and metaregression analysis. *Am J Psychiatry* 2007;**164**:942-8.
16. Spencer T, Biederman J, Wilens TE, Faraone SV. Adults with attention-deficit/hyperactivity disorder: a controversial diagnosis. *J Clin Psychiatry* 1998;**59**:59-68.
17. Wender PH. Attention-deficit hyperactivity disorder in adults. *Psychiatr Clin North Am* 1998;**21**:761-74.
18. Corkum P, Tannock R, Moldofsky H. Sleep disturbances in children with attention-deficit/hyperactivity disorder. *J Am Acad Child Adolesc Psychiatry* 1998;**37**:637-46.
19. Hodgkins P. Management of ADHD in children across Europe: patient demographics, physician characteristics and treatment patterns. *Eur J Pediatr* 2013;**172**:895-906.
20. Owens JA. The ADHD and sleep conundrum: a review. *J Dev Behav Pediatr* 2005;**26**:312-22.
21. Sung V, Hiscock H, Sciberras E, Efron D. Sleep problems in children with attention-deficit/hyperactivity disorder: prevalence and the effect on the child and family. *Arch Pediatr Adolesc Med* 2008;**162**:336-42.
22. Efron D, Lycett K, Sciberras E. Use of sleep medication in children with ADHD. *Sleep Med* 2014;**15**:472-5.
23. Sadeh A, Pergamin L, Bar-Haim Y. Sleep in children with attention-deficit hyperactivity disorder: a meta-analysis of polysomnographic studies. *Sleep Med Rev* 2006;**10**:381-98.
24. Spruyt K, Gozal D. Sleep disturbances in children with attention-deficit/hyperactivity disorder. *Expert Rev Neurother* 2011;**11**:565-77.
25. Xu X, Mill J, Sun B, et al. Association study of promoter polymorphisms at the dopamine transporter gene in attention deficit hyperactivity disorder. *BMC Psychiatry* 2009;**9**:3.
26. Williams HJ, Owen MJ, O'Donovan MC. Schizophrenia genetics: new insights from new approaches. *Br Med Bull* 2009;**91**(1):61-74.
27. Faraone SV, Perlis RH, Doyle AE. Molecular genetics of attention-deficit hyperactivity disorder. *Biol Psychiatry* 2005;**57**:1313-23.
28. Floet AMW, Scheiner C, Grossman L. Attention-deficit/hyperactivity disorder. *Pediatr Rev* 2010;**31**(2):56-69.
29. Rowland AS, Lesesne CA, Abramowitz AJ. The epidemiology of attention-deficit hyperactivity disorder (ADHD): a public health view. *Ment Retard Dev Disabil Res Rev* 2002;**8**(3):162-70.
30. Owens JA, Fernando S, Mc Guinn M. Sleep disturbance and injury risk in young children. *Sleep Med* 2005;**3**(1):18-31.
31. Gaina A SM, Hamanishi S, Chen X, Wang H, Yamagami T, Kagamimori S. Daytime sleepiness and associated factors in Japanese school children. *J Pediatr* 2007;**151**(5):518-22.

32. Harriet H, Canterford L, Ukoumunne OC, Wake M. Adverse Associations of Sleep Problems in Australian Preschoolers: National Population Study. *Pediatrics* 2007;**119**(1):86-93.
33. Teng A. Sleep-related breathing disorders (Identifying and managing). *MJPCH* 2010;**16**:5.
34. Reine LLR, Waumans Rc, Van den Berg G, Gemke R. Sleep habits and sleep disturbances in Dutch children: a population-based study. *Eur J Pediatr* 2010;**169**(8):1009-15.
35. Wei JL, Mayo MS, Smith HJ, Reese M, Robert A. Weatherly. Improved Behavior and Sleep After Adenotonsillectomy in Children With Sleep-Disordered Breathing FREE. *Arch Otolaryngol Head Neck Surg* 2007;**133**(10):974-9.
36. Wang RC, Elkins TP, Keech D. Accuracy of clinical evaluation in pediatric obstructive sleep apnea. *Otolaryngol Head Neck Surg* 1998;**118** (1) 69-73.
37. Montgomery-Downs HE, O'Brien LM, Holbrook CR et al. Snoring and sleep-disordered breathing in young children: subjective and objective correlates. *Sleep* 2004;**27**(1):87-94.
38. Chervin RD, Ruzicka DL, Archbold KH. Snoring predicts hyperactivity four years later. *Sleep* 2005;**28**(7):885-90.

REVIEW ARTICLE

Trends in Breastfeeding Practices in Bangladesh, 1993-2014

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Abstract

Appropriate feeding practices are essential for the nutrition, growth, development and survival of infants and young children. Exclusive breastfeeding (EBF) means that the infant receives only breast milk for the first six months of life after birth. Breast milk is the gold standard of infant nutrition and breastfeeding is the gold standard of infant feeding. Bangladesh Breastfeeding Foundation (BBF), UNICEF and several other organizations are working in the country for the promotion of healthy feeding practices. This article presents the trends of breastfeeding practices in the country from 1993–2014, using data from Bangladesh Demographic and Health Surveys (BDHS). In Bangladesh, the prevalence of EBF remained largely unchanged for nearly two decades from 1993 to 2007, and the prevalence was about 45%. However, in 2011 a prevalence of 64% was reported, an increase by 21 percentage points from BDHS 2007. In BDHS 2014, 55% of infants under six months were exclusively breastfeed. This proportion is lower than that reported in the 2011 BDHS but still 5 percentage points higher than the HPNSDP target of 50% of EBF by 2016. Though considerable improvement has been made in Bangladesh, this is far below the widely accepted “universal coverage” target of 90% coverage. Bangladesh’s achievements seems especially impressive when compared with the low EBF average in South Asia for infants under six months, languishing at 45 percent. Child nutrition programmes worldwide continue to require investments and commitments to improve infant feeding practices in order to have maximum impact on children’s lives.

Key Words: *Exclusive breast feeding*

Background

The word nutrition is originated from the word ‘nutricus’ meaning to suckle at the breast. The three determinants of good nutrition- food security, disease control and caring are contained in the single activity of breastfeeding. Breast milk is the gold standard of infant nutrition and breastfeeding is the gold standard of infant feeding.¹ Breastfeeding provides survival, optimal growth, nutrition and development for infants and young children.² Perhaps no single public health intervention will be saving more lives and promote health of children as do breastfeeding.^{1, 3}

Breastfeeding is a well established and recommended intervention for the improvement of child nutrition. Studies have demonstrated that it reduces deaths in infants and young children. It is one of the most important factors for growth and development of infants and is globally endorsed as being the best for any neonate.⁴ The World Health Organization (WHO) recommends the practice of exclusive breastfeeding (EBF) of infants for the first six months of life after birth. EBF means that the infant receives only breast milk. No other liquids or solid are given, not even water, with the exception of oral rehydration salt solution, or drop/syrups of vitamins, minerals or

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medicines. There are two other breastfeeding categories according to recent WHO guidelines. Predominant breastfeeding means the infant's predominant source of nourishment had been breast milk. However, the infant may also have received water and water-based drinks like tea and local herbal drops and partial breastfeeding means the infant's feeding included non-breast milk foods such as animal/powdered/condensed milk and/or solid/semi-solid food (i.e. cereals, vegetables, fruits, lentils or meat).^{5, 6, 7, 8}

Throughout the world, in developed and developing countries alike, inappropriate feeding of infants leading to their poor nutrition is a significant problem affecting socio economic progress in general. Suboptimal breastfeeding was responsible for 11.6% of all child deaths in 2011.⁷ Universal (90%) coverage of breastfeeding is estimated to prevent around 13% of all deaths among children under five years of age in low and middle income countries.⁸ However, globally only half of infants under one month of age and 30% of infants age 1-5 months are exclusively breastfeed.^{9, 10}

In the late 1980s, an organization set up, 'Campaign for the Protection and Promotion of Breastfeeding (CPPBF)' in the country to provide a forum for

professionals, UN agencies and government bodies to work on the formulation and implementation of programs for the promotion of breastfeeding. CPPBF later on evolved into the Bangladesh Breastfeeding Foundation (BBF). BBF, along with UNICEF and its other regional partners, has developed multifaceted strategies and programs aiming to optimize breastfeeding and complementary feeding practices in the country. The baby friendly hospital initiative was established by WHO and UNICEF in 1991 as a hospital-based intervention to increase breastfeeding rates. In baby friendly hospitals breastfeeding is supported, practiced, protected and promoted.^{11, 12}

This article presents the trends of breast feeding practices in the country from 1993–2014, using data from Bangladesh Demographic and Health Surveys (BDHS) (Table 1). Thereby, the developments that have occurred in these practices have been assessed in an attempt to guide national health policies and interventions. Data in Bangladesh Demographic and Health Surveys of the following years were studied: 1993–94 (BDHS 93–94), 1996–97 (BDHS 96–97), 1999–00 (BDHS 99–00), 2004 (BDHS 04), 2007 (BDHS 07), 2011 (BDHS 11) and 2014 (BDHS 14).¹³⁻¹⁹ The 2014 BDHS collected data on infant feeding from the youngest children under two who were living with their mother using a 24-hour recall period.

Table I
Summery of surveys

Title of survey	Conducted by	Coverage	Sample size (no. of households)	Sampling method
Bangladesh Demographic and Health Survey (BDHS), 1993-94	National Institute of Population Research and Training (NIPORT), Mitra and Associates and Macro International Inc.	National	9681	Two-staged stratified sample
Bangladesh Demographic and Health Survey, 1996-97	(NIPORT), Mitra and Associates and Macro International Inc.	National	9099	Two-staged stratified sample
Bangladesh Demographic and Health Survey, 1999-2000	(NIPORT), Mitra and Associates and ORC Macro	National	10,268	Two-staged stratified sample
Bangladesh Demographic and Health Survey, 2004	(NIPORT), Mitra and Associates and ORC Macro	National	10,811	Multi-staged stratified sample
Bangladesh Demographic and Health Survey, 2007	(NIPORT), Mitra and Associates and ORC Macro International	National	10,819	Two-staged stratified sample
Bangladesh Demographic and Health Survey, 2011	(NIPORT), Mitra and Associates and ICF International	National	18,000	Two-staged stratified sample
Bangladesh Demographic and Health Survey, 2014	(NIPORT), Mitra and Associates and ICF International	National	18,000	Two-staged stratified sample

Results

Exclusive Breastfeeding under six months

Though the breastfeeding tradition in Bangladesh is without a doubt among the strongest in the world and Bangladesh is one of the few countries where most women have never abandoned the Innocenti Declaration's ideal of breastfeeding for two years or beyond. The prevalence of exclusive breastfeeding under six months shows an unsatisfactory improvement until the year 2007, with no statistically significant improvement over the years. However, it considerably improved during the period of recent BDH surveys. The estimates are: 45.9% (BDHS 93–94), 45.1% (BDHS 96–97), 46.1% (BDHS 99–00), 36.1% (BDHS 04), 42.9% (BDHS 07), 64.1% (BDHS 11) and 55.03% (BDHS 14).

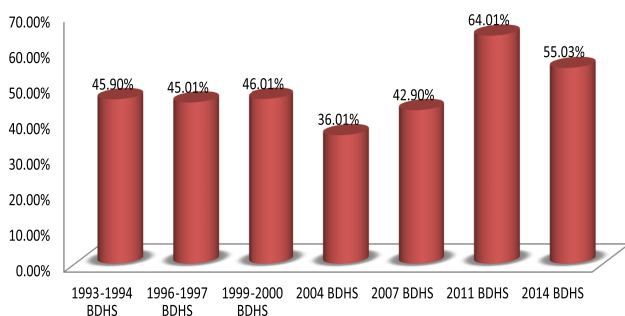


Fig 1 Trends in exclusive breastfeeding practices among children under six months

Early initiation of breastfeeding

Early initiation of breastfeeding and exclusive breastfeeding of children below six months are considered the two most decisive indicators for assessing breastfeeding practices in infants [10]. The proportion of infants in whom breastfeeding was initiated within one hour after birth is as follows: 8.6% (BDHS 93–94), 13.2% (BDHS 96–97), 16.5% (BDHS 99–00), 23.7% (BDHS 04), 41.7% (BDHS 07), 47.1% (BDHS 11) and 57% (BDHS 14). The first survey refers to last-born children in the three preceding years while the last survey refers to last-born children in the two preceding years. BDHS 96–97, BDHS 99–00 and BDHS 04 report the rate among all children born in the last five years. BDHS 07 reports early initiation only of the last-born child in the preceding five years. The figures show a statistically significant improvement in the rates of early initiation, with each survey.

Continued breastfeeding at one year

The prevalence of continued breastfeeding in children aged 12–15 months is high (above 94%) in all the surveys and changes in it have not been found to be statistically significant. The estimates are as follows: 95.5% (BHDS 93–94), 97.0% (BDHS 96–97), 95.1% (BDHS 99–00), 95.9% (BDHS 04), 94.5% (BDHS 07), 95.0% (BDHS 11) and 96% (BDHS 14).

Continued breastfeeding at two years

The proportion of children between 20–23 months of age who were continuing to breastfeed did not display a significant trend over the years, with values at 86.4% (BDHS 93–94), 89.7% (BDHS 96–97), 87.2% (BDHS 99–00), 90.3% (BDHS 04), 91.0% (BDHS 07), 89.6% (BDHS 11) and 87.3% (BDHS 14).

Predominant breastfeeding under six months

In BDHS 93–94 and BDHS 96–97, the data available were insufficient to estimate the prevalence of predominant breastfeeding (infants predominantly fed breast milk, but also receiving water, water-based drinks and fruit juice [12]) in infants less than six months of age. Estimates based on the other four surveys are 39.0% in 1999–2000, 37.2% in 2004, declining to 16.7% in 2007, 12.5% in 2011 and 13.7% in 2014.

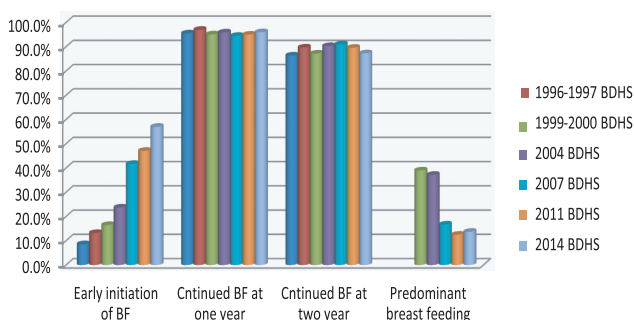


Fig 2 Trends in prevalence of common indicators, Bangladesh, 1993-2014

Discussion

Although considerable improvement have been made in some regions, the prevalence of exclusive breastfeeding far too low in many areas of the developing world. Global data suggests that the prevalence of EBF among infants younger than six months in developing countries increased from 33% in 1995 to 39% in 2010. The prevalence increased in almost all regions in the developing world, with the biggest improvement seen in West and Central

Africa where the prevalence of EBF more than doubled from 12% in 1995 to 28% in 2010. Eastern and Southern Africa also realized improvements with an increase from 35% in 1995 to 47% in 2010. But more modest improvements were observed in South Asia i.e. 40% in 1995 to 45% in 2010.²⁰

Exclusive breastfeeding is the best recommended infant feeding method for the first six month of life and has a protective effect against child morbidity and mortality. But it has not yet been universally practiced and reduction in the rate of EBF is taken as a serious problem, especially in developing countries. In Bangladesh 55% (BDHS 2014) of infants less than six months are exclusively breastfeed. This proportion is lower than that reported in the 2011 BDHS (64 percent). There was a sharp increase in exclusive breastfeeding from 43 percent to 64 percent between the 2007 BDHS and the 2011 BDHS. Intensive mass media campaigns for a couple of years prior to 2011 survey could have impacted the status i.e. the influence of a national media campaign that started in December 2010 and reached peak intensity in February 2011. Additionally, there is possible effect of several intensive programs that focus on maternal and newborn care and child health, including improved feeding that has been implemented 1-2 years before the survey and cover about 25 percent of the country's population. The 2013 Utilization of Essential Service Delivery Survey and the 2012-13 Multiple Indicator Cluster Survey reported lower EBF rates of 60 percent and 56 percent respectively.

In spite of the decline in exclusive breastfeeding between 2011 and 2014, the prevalence of EBF of infants up to six months in 2014 is 5 percentage points higher than the Health Population and Nutrition Sector Development Programme (HPNSDP) target of 50 percent of exclusive breastfeeding by 2016.¹⁹ Although considerable improvement has been made in Bangladesh, this is far below the widely accepted "universal coverage" target of 90% coverage.²⁰

Conclusion

The prevalence of exclusive breastfeeding remains far too low in many areas of the developing world. In spite of the well recognized importance of exclusive breastfeeding, the practice is not widespread in the developing world and increase in the global level is still very modest with much room

for improvement. So the child nutrition programmes worldwide continue to require investments and commitments to improve infant feeding practices in order to have maximum impact on children's lives.

References

1. Bilkis B, Khurshida K. Effects of educational level of father and mother on perceptions of breastfeeding. *J Enam Med Col* 2012;2(2):67-73.
2. Talukder MQK. How paediatricians can promote, protect and support breastfeeding. *Bangladesh J Child Health* 2011;35(3):79-83.
3. Talukder MQK. Saving lives of women and children through breastfeeding. Bangladesh breastfeeding Foundation, June 2005.
4. Prakash CJ, Mirak RA, Sumon KD, Shahnawaz A, Abu Syed GF, Tahmeed A. Prevalence of exclusive breastfeeding and associated factors among mothers in rural Bangladesh. *International Breastfeeding Journal* 2014;9:7.
5. Breastfeeding-exclusive breastfeeding. [http://www.who.int/elena/titles/exclusive_breastfeeding/en/].
6. WHO. Indicators for assessing infant and young child feeding practices: Conclusion of a consensus meeting held 6-8 November 2007 in Washinton D.C. USA, 2007.
7. Labbok MH, Belsey M, Coffin CJ. A call for consistency in defining breastfeeding. *Am J Public Health* 1997; 87(6):1060-61.
8. Manjeswori U, Ram KC, Lotta M, Prakash SS, Tor AS. Infant feeding practices in Bhaktapur, Nepal: a cross sectional, health facility based survey. *International Breastfeeding Journal* 2012; 7:1.
9. Black RE, Victora CG, Walker SP, Bhutta ZA, Christian P, de Onis M, Ezzati M, Grantham-McGregor S, Katz J, Martorell R, Uavay R. Maternal and under nutrition and overweight in low-income and middle-income countries. *Lancet* 2013; 382:427-551.
10. Jones G, Stekette RW, Black RE, Bhutta ZA, Morris SS, Bellagio Child Survival Study Group. How many child deaths can we prevent this year? *Lancet* 2003; 362:65-71.
11. Bangladesh Breastfeeding Foundation: protection, promotion and support breastfeeding. [<http://www.bbf-bd.org/home.>].
12. Talukder MQK. Protection, promotion and support of breastfeeding in Bangladesh. *Regional Health Forum* 1996;1(1):18-24.

13. National Institute of Population Research and Training (NIPORT), Mitra and Associates and Macro International Inc. Bangladesh Demographic and Health Survey (BDHS), 1993-94.
14. National Institute of Population Research and Training (NIPORT), Mitra and Associates and Macro International Inc. Bangladesh Demographic and Health Survey (BDHS), 1996-97.
15. National Institute of Population Research and Training (NIPORT), Mitra and Associates and Macro International Inc. Bangladesh Demographic and Health Survey (BDHS), 2001.
16. National Institute of Population Research and Training (NIPORT), Mitra and Associates and Macro International Inc. Bangladesh Demographic and Health Survey (BDHS), 2004.
17. National Institute of Population Research and Training (NIPORT), Mitra and Associates and Macro International Inc. Bangladesh Demographic and Health Survey (BDHS), 2007.
18. National Institute of Population Research and Training (NIPORT), Mitra and Associates and ICF International. Bangladesh Demographic and Health Survey (BDHS), 2011.
19. National Institute of Population Research and Training (NIPORT), Mitra and Associates and ICF International. Bangladesh Demographic and Health Survey (BDHS), 2014.
20. Xiaodong C, Tessa W, David WB. Global trends in exclusive breastfeeding. *International Breastfeeding Journal* 2012;7:12.

REVIEW ARTICLE

Constipation in children - A review

Rabi Biswas¹

Abstract

Constipation in children is a universal problem, occurring in 0.7-28% of the population. The exact aetiology is unknown, but the majority of children have a functional, rather than organic aetiology. Symptoms associated with constipation include abdominal pain, a poor appetite and faecal incontinence, all of which interfere with the quality of life of the child and his or her family. Early intervention with appropriate management is necessary to prevent ongoing sequelae. Once an organic cause has been excluded, a programme of intervention should be implemented, namely evacuation of any faecal mass present, followed by regular maintenance therapy to encourage evacuation of a daily soft stool for at least 2-3 months, prior to gradual withdrawal. Emotional support, exercise and dietary modification are linked to the therapy and will ensure a successful outcome. Failure to implement the protocol may result in ongoing problems in up to 50% of children as they enter adulthood.

Introduction

Constipation is a symptom rather than a disease and often constitutes a major problem for the child and his family. Unfortunately, no universal accepted definition for chronic constipation is available.¹ A group of pediatric gastroenterologists and pediatricians with an interest in gastrointestinal motility² reached agreement on defining childhood functional defecation disorders. It was also decided to adopt the term fecal incontinence instead of the terms encopresis and soiling. With 84% of constipated children suffering from this complaint,³ the involuntary passing of fecal material in the underwear is one of the major features of childhood constipation.

It is important to distinguish between constipation with or without fecal incontinence and functional non-retentive fecal incontinence (FNRFI). It is indicated that different patho-physiological mechanisms are involved.² Both entities can result in fecal incontinence, but in children with FNRFI, there are no signs of constipation. In many studies, constipated children with fecal incontinence and

children with FNRFI are grouped into one sample because they share the complaint of fecal incontinence. It is known that about 80% of the children with fecal incontinence experience chronic constipation.^{3,4}

The pathophysiological mechanisms underlying childhood constipation are undoubtedly multifactorial, and are not well understood. In 90% of all patients, no specific organic cause can be found.⁵ Fearful reactions to defecation and stool-withholding behavior are common in children with constipation.⁶⁻¹¹ Retained stools become progressively more difficult and painful to evacuate, leading to the development of fear and consequently persistent constipation.^{2,12}

This vicious cycle can be described as learned behavior.

Based on clinical experience, constipated children are traditionally treated by pediatricians combining medical and behavioral approaches like a toilet sitting regimen and education.^{13,14} Laxative therapy usually consists of a series of enemas to disimpact

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the child, dietary suggestions and daily dosages of laxatives. The goal of treatment is to promote daily, soft painless stools preventing re-accumulation of feces.¹⁵

Epidemiology

Constipation represents the main complaint in 3% of pediatric outpatient's visits.⁵ This percentage increases to 25–45% in specific pediatric-gastrointestinal motility clinics.^{15,19} To date, the worldwide prevalence of constipation in children varies widely and is estimated to range between 0.7 and 29.6%.^{10,20,21} The large range is likely due to variations in sample size and different methods of data collection. Van den Berg et al.²² found no evidence that constipation occurs less in non-Western societies. Higher rates are reported in girls than in boys, but a 1:1 ratio is also reported.²² A convincing explanation for this difference in gender ratio is absent.

Normal stooling pattern

As constipation is defined by the number of stools per week, it is important to correlate stool frequency with age. At one week of age, four stools (a range of 1-9) are passed per day, decreasing to three per day at four months of age, two stools per day at four years of age, and three per week at 10-15 years of age.

Definition of Constipation

Stool frequency reduces progressively in early childhood, from more than four stools a day to 1.2 a day at age 4 years,²³ by which age 98% of children are toilet trained. Constipation is typically characterized by infrequent bowel evacuations, large stools, and difficult or painful defecation. Attempts have been made to define terms and diagnostic criteria more precisely.

Constipation is defined subjectively a feeling of unsatisfactory evacuation. The other accompaniments could be passage of too small stool, too hard stool, too difficult to expel, too frequent and incomplete evacuation, but the objective and well accepted definition of constipation is passage of stools twice or less per week.

Based on the symptomatology certain criteria have been used in literature to define constipation. The guidelines of the North American Society of Paediatric Gastroenterology, Hepatology and Nutrition defines constipation as a delay or difficulty in defecation present for 2 or more weeks and sufficient to cause significant distress to the patient. Loening Baucke criteria²⁴ also called as Iowa criteria (Table-I) to define constipation has been used widely in various randomized controlled studies.

The Loening-Baucke criteria²⁴ of Paediatric acute constipation

At least two of the following criteria must present less than eight weeks:

1. Defecation frequency less than three times a week
2. Two or more encopresis episodes per week
3. Periodic passage of very large amounts of stool once every 7-30 days
4. A palpable abdominal or rectal mass at physical examination

Soiling and encopresis are terms that lack precision and are sometimes used interchangeably. Soiling can occur in the absence of constipation and may be voluntary or involuntary. Encopresis is usually used for the passage of normal stools in socially unacceptable places. These terms have largely been replaced by the term incontinence. The Paris Consensus on Childhood Constipation Terminology (PaCCT) Group has proposed a simplified terminology that more clearly defines the criteria for chronic constipation (Table-II),²⁵ which informs the recently published Rome III criteria for diagnosis of functional constipation (Table-III).^{26,27}

Terminology recommended by PaCCT Group²⁵

Chronic constipation—The occurrence of two or more of the following characteristics during the past eight weeks:

- Frequency of bowel movements less than three per week
- More than one episode of faecal incontinence per week
- Large stools in the rectum or palpable mass on abdominal examination
- Periodical passing of stools so large that they may obstruct the toilet
- Display of retentive posturing and withholding behaviours
- Painful defecation

Faecal incontinence- Passage of stools in an inappropriate place. **Organic faecal incontinence-** Faecal incontinence resulting from organic disease (neurological damage or sphincter abnormalities, for example)

Functional faecal incontinence- Non-organic disease which can be subdivided into:

- Constipation associated faecal incontinence
- Non-retentive (non-constipation-associated) faecal incontinence.

Constipation associated faecal incontinence—Functional faecal incontinence associated with the presence of constipation

Non-retentive faecal incontinence—The passage of stools in an inappropriate place, occurring in children aged 4 years and older, with no evidence of constipation on history or examination.

Faecal impaction—Large faecal mass in either the rectum or the abdomen which is unlikely to be passed on demand. The faecal impaction can be shown by abdominal or rectal examination or other methods.

Pelvic floor dys-synergia—Inability to relax the pelvic floor when attempting to defecate.

Diagnosis of functional constipation in childhood

For diagnosis of functional constipation under the Rome III criteria^{26,27} symptoms must include at least two of the followings:

- Two or fewer defecations per week
- At least one episode per week of faecal incontinence after the child has acquired toileting skills
- History of excessive stool retention or retentive posturing
- History of painful or hard bowel movements
- Presence of a large faecal mass in the rectum
- History of stools with large diameter that may obstruct the toilet
- In infants and children up to a developmental age of 4 years, these symptoms must be present for at least one month; in children over 4 years old, symptoms should be present for at least two months, with insufficient criteria for the diagnosis of irritable bowel syndrome

Aetiology

There is a functional, rather than organic, cause, in over 90% of paediatric constipation cases. Diet plays a role, with lack of fibre, roughage and fluid thought to be factors. Lack of exercise and behavioural overlay accentuate it, particularly when potty training and new school attendance occur at the same time. The administration of antibiotics, a nappy rash and diarrhoea occur often with illness, and this will initiate or exacerbate the condition. Slow transition has been suggested as a contributory factor in up to two thirds of children.²⁸

Only 5-10% of childhood constipation has an underlying organic cause (Table-I). Surgeons are duty-bound, using a rectal biopsy, to exclude an underlying organic cause for the constipation which

could necessitate a surgical operation for cure (aganglionosis, dysganglionosis and Hirschsprung's disease). "Red flags" should warn of a possible organic cause in a child who presents with constipation (Table-III).

Table I
Organic causes of constipation

Anatomical malformation
Imperforate anus
Anal stenosis
Anterior displaced anus
Pelvic mass (sacral teratoma)
Metabolic or endocrine
Hypothyroidism
Hypercalcaemia
Hypokalaemia
Diabetes mellitus
Multiple endocrine neoplasia type 2B
Gastrointestinal
Cystic fibrosis
Coeliac disease or gluten enteropathy
Neuropathic conditions
Spinal cord abnormalities
Including tethered cord
Neurofibromatosis
Intestinal nerve or muscle disorders
Hirschsprung's disease
Visceral myopathies
Visceral neuropathies
Abnormal abdominal musculature
Prune belly syndrome (Eagle-Barrett) syndrome
Down's syndrome
Gastroschisis
Connective tissue disorder
Scleroderma
Systemic lupus erythematosus
Drugs
Opiates
Phenobarbitone
Antacids
Anticholinergics
Other
Heavy metal poisoning (lead)
Vitamin D intoxication
Cow's milk protein intolerance

Table II*Surgical versus idiopathic causes of constipation***Red Flags**

Delayed passage of meconium > 24 hours

Age of onset < 12 months

Failure to thrive

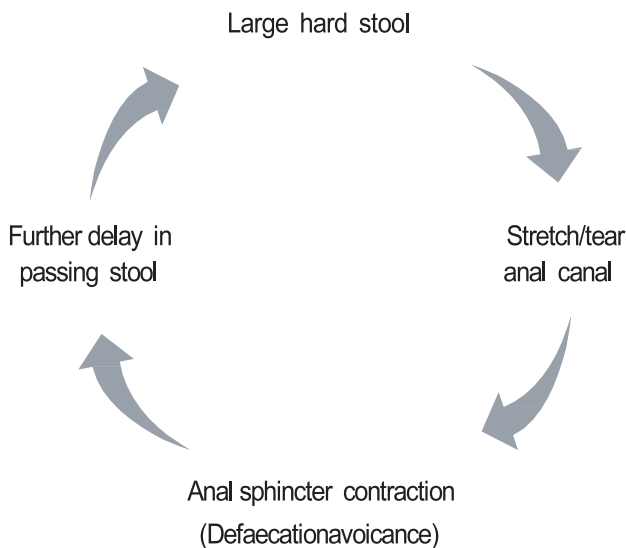
Distension

No soiling, stool withholding and large calibre stools

Associated with extraintestinal symptoms, cutaneous pigmentary abnormalities, urinary symptoms, enterocolitis and upper gastrointestinal symptoms

Pathophysiology of idiopathic constipation

The initiating factor is thought to be a perianal insult, either from a tear of the very sensitive anal canal lining (fissure in ano) caused by the passage of a large hard stool or perianal excoriation associated with ongoing loose stools. This damages the perianal mucosa and skin, causing pain with internal anal sphincter spasm, which leads to a reluctance to pass stools, stool retention, faecal desiccation and hard stools.²⁹ When the stool is passed, it again tears the anocutaneous junction, aggravating the condition (Figure 1). The vicious cycle continues unless treated, resulting in progressive and gradual distension of the rectum above the internal sphincter, with associated decreased prograde propulsive activity from the proximal dilated colon.

**Fig I** *Vicious cycle perpetuating constipation***Clinical assessment****History**

The history must include:

- A historical review, viz. the passage of the first meconium. (This should be within 24 hours in a term baby).
- The age at onset, and any possible relationship to the cessation of breastfeeding.
- The frequency, consistency and size of the stool.
- Associated pain or bleeding on defaecation.
- Incontinence and withholding behaviour.
- Dietary intake and medication.

Presentation

Other than the obvious decrease in stool frequency, constipation can present as abdominal pain (80%), soiling (80%), faecal impaction (70%) and stool withholding (over 90%), in children who are younger than three years of age. It is necessary to enquire about these associated symptoms as they already imply a significant effect, so mandate initiation of a therapeutic programme is needed to correct the progression of the constipation.

Examination

The examination must include:

- A percentile chart of the child's weight and height.
- A full general examination, excluding hypothyroidism.
- An abdominal examination, especially for distension and a palpable faecal mass.

Perineal inspection

The perineal inspection must search for:

- A fissure, fistula, excoriation and the presence of the stool on the perianal skin.
- Evidence of incontinence or abuse.

Rectal examination

The rectal examination must:

- Inspect the tone and size of the anal opening.
- Search for a palpable faecal mass, the consistency of the stool and other rectal masses.
- Exclude a pelvic mass. (Presacral tumours are rare causes of constipation).
- Check for explosive decompression on withdrawal of the finger in a small baby, as this suggests Hirschsprung's disease.
- Inspect the spine and sacrum.

Investigations

An abdominal X-ray can confirm the diagnosis and assess the extent of the problem, based on stool distribution throughout the colon and associated colonic distension.³⁰ Others question its use.³¹

Further investigations are only indicated if there are associated red flags (Table II), or in those with intractable symptoms. They may include a rectal biopsy to confirm the ganglion cells; a barium enema to identify strictures, an extrahernial mass or a transition zone; and X-ray transit studies, which may identify local rectosigmoid holdup and manometry to assess the state of the sphincters.

Treatment of constipation

Treatment of constipation is aimed at:

1. Treating the cause
2. Evacuation/disimpaction
3. Maintenance therapy.

Management of acute simple constipation

Acute constipation is usually mild and to treat proper diary should be maintained by the parents. Enough fluids and carbohydrate rich diet takes care of constipation in infants. At the same time the toilet training should also be imparted. This is very common in children.

Parents must be educated and reassured that it is not pathological. One has to eliminate the precipitating factor. Treat local causes like anal fissure, boil or dermatitis effectively. Procedures like enemas, finger evacuation/disimpaction, finger dilatation and frequent use of suppositories should be avoided. But encourage use of high fiber diet in terms of cereals, pulses, vegetables and fruits. Adequate fluid intake is advised to keep proper hydration. Initially laxatives can be used. Encourage toilet training simultaneously. Laxatives can be given for 7-10 days but prolonged use should be discouraged. If this is not properly treated, can result into chronic constipation.³²

Management of chronic constipation

The evidence for effectiveness of treatments is weak. Therapeutic trials have used a range of outcome measures; those of greatest clinical relevance are the number of defecations per week, use of laxatives, stool consistency, pain, difficulty in defecation, and number of soilings per month.

The Childhood Constipation Working Group of the British Society of Paediatric Gastroenterology,

Hepatology, and Nutrition recently reported that, on the basis of a systematic review of available treatments, there was insufficient evidence to allow any recommendations for practice and that guideline development would need to be based on a synthesis of clinical experience, evidence, and consensus.³³

Initial rapport

A critical first step is to manage the anxiety of both parent and child, to deal with attitudes of guilt or blame if they exist, and to develop a treatment plan. The child may be fearful of painful defecation and parents need to understand that coercive toilet training in this situation will be ineffective. In older children, faecal incontinence and its social consequences needs a non-accusatory, sympathetic management approach. A positive approach on the part of the clinician and a carefully explained management plan with the assurance of continued involvement over an extended period of time all contribute to an effective therapeutic relationship.

The objectives of treatment are to remove faecal impaction, to restore a bowel habit in which stools are soft and passed without discomfort, and to ensure self toileting and passing stools in appropriate places.

Disimpaction

The objective of disimpaction is to fully clear the rectum of retained faeces. High doses of mineral oil or polyethylene glycol 3350 (1-1.5 g/kg/day for three day³⁴ have been shown to be effective. Although many of the other available laxatives have also been used, evidence of their effectiveness is lacking. The use of suppositories, enemas, and manual evacuation is more contentious and a careful balancing of physical and psychological benefits and harms is necessary. Many paediatricians avoid rectal treatments if at all possible. Glycerol suppositories are suitable for infants and bisacodyl suppositories for older children. Phosphate, saline, or mineral oil enemas are effective; soap and water, and magnesium enemas are potentially toxic and should be avoided. In rare circumstances disimpaction under anaesthetic is indicated.

Osmotic laxatives

No randomised controlled trials have compared osmotic laxatives versus placebo in children. Two small randomised trials found no significant difference in stool frequency or consistency between lactulose and lactitol after two to four weeks in

children aged 8 months to 16 years, both having benefit.^{35,36} One of the trials found that lactulose increased abdominal pain and flatulence more than lactitol. A third randomised trial in non-breastfed constipated infants found no difference between different strengths of lactulose.³⁷

One randomised controlled trial has compared polyethylene glycol (PEG 3350) with lactulose in 100 children aged 6 months to 15 years, using a composite measure of success comprising defecation three or more times per week and encopresis once or less every week after eight weeks.³⁸ Treatment was significantly more successful in the PEG group than in the lactulose group (56% v 29%), and adverse effects were fewer. A second study confirmed the clinical and biological tolerance of PEG in children treated for three months and found it better than lactulose in respect of vomiting and flatulence side effects.³⁹

Stimulant laxatives

A Cochrane review (search date 2001) found no randomised controlled trials that adequately met the selection criteria, and it concluded that there is insufficient evidence on the use and effectiveness of stimulant laxatives for the treatment of childhood constipation.⁴⁰

Maintenance therapy

It is sensible to use laxatives over an extended period, which may be months or years, in order to establish a normal bowel habit and improve rectal awareness. This seems preferable to frequent attempts to wean off treatment, followed by the repeated need for disimpaction. Osmotic laxatives have the best evidence for effectiveness, and PEG is less likely to produce side effects than lactulose. The dose should be adjusted to achieve the passage of soft, formed stools. The chronic use of stimulant laxatives is contentious. They have been widely used in clinical practice, usually in combination with an osmotic laxative, though prolonged use can precipitate an atonic colon and hypokalaemia. As a result, intermittent use for avoiding a recurrence of impaction has been advocated. Adequate intakes of fluids and fibre should be encouraged, and specialist dietetic advice may be needed. The child and its parents can be asked to keep a bowel chart or diary.

Increased dietary fibre

No systematic reviews or randomised controlled trials of increasing dietary fibre in children with constipation have been reported.

Biofeedback and other psychological interventions

Two types of biofeedback have been widely studied, pressure biofeedback and electromyography biofeedback. In both, an audio or visual display is generated of the child's efforts to consciously contract and relax the muscles around the anus. One systematic review⁴¹ found higher rather than lower rates of persisting faecal incontinence after up to 12 months when biofeedback was added to conventional treatment (odds ratio 1.11, 0.78 to 1.58). One small trial of behaviour modification as an adjunct to laxatives found a significant reduction in soiling episodes at three and 12 months (odds ratio 0.20, 0.06 to 0.65).

Behaviour modification

Behaviour modification can be an important element of management and can be effectively delivered in a specialist, nurse led clinic. Regular toileting and unhurried time on the toilet should be encouraged. A reward system, especially one that is geared toward successful use of the toilet as opposed to clean pants is important. A diary of stool frequency can be helpful, and it can be linked to a system of reward as well as being a focus for positive reinforcement at surgery visits.

Dealing with incontinence

Both child and parents need a careful explanation of the involuntary process that leads to faecal incontinence as an essential first step. Rectal contractions occur regularly even in constipation, and are associated with transient relaxation of the internal sphincter. This allows loose or liquid stool in the vicinity to leak out. The child can be helped to focus on regular defecation and checking/changing of underclothes as positive actions to prevent the problem. Involving the school nurse can help with access to toilet facilities and make teachers aware of the child's problems. Though several studies have shown associations between encopresis, soiling, or incontinence and psychological and behavioural problems, good evidence of the effectiveness of psychological interventions in these children does not exist.

Prevention

Prevention of colonic dysfunctions have received much less attention but attending paediatrician can play important role by providing anticipatory counseling in terms of appropriate feeding advice, high fiber diet, interpretation of normal bowel habits, counseling life issues of the child and early detection of problem and intervention.

When to refer for specialist care

Assessment by a specialist with an interest in childhood constipation is necessary if an organic cause is suspected, if treatment is unsuccessful, or when management is complex. Treatment failure may be early, when attempts at disimpaction fail, or late, if there is difficulty maintaining remission. If an underlying problem is suspected, the general practitioner can instigate blood tests for inflammatory markers, hypothyroidism, hypercalcaemia and coeliac disease before the child attends outpatients.

Specialist follow-up typically takes place in a nurse led clinic at intervals of one to three months, depending on progress, with medical review as required. Families can be provided with a contact number in case they need help urgently. Multidisciplinary team meetings are particularly valuable for those children with associated family or psychological problems.

Surgery for functional constipation

In rare instances, continued failure to respond to treatment may require surgical intervention. Formation of a caecostomy and antegrade continence enemas can reduce frequency of soiling and abdominal pain in children with slow transit constipation, though stoma complications (stenosis, leakage, pain related to the catheter) are common.⁴² More recently, botulinum toxin has been used, with variable results, on the basis of the concept that some patients have a short aganglionic segment above the pectinate line, sometimes called "ultra-short Hirschsprung's disease".⁴³ Anal dilatation has no benefit in functional constipation.⁴⁴

Prognosis

Sixty per cent of children remit within a year, but up to 50% will relapse within five years. Thirty to 50% will have symptoms five years after the initial presentation, and 30% will suffer problems beyond puberty.^{45,46} A poor prognosis is predicted in the

event of an onset of less than one year, a history of more than one year and faecal incontinence on presentation.

Discussion

This literature review showed psychological and physical problems associated with constipation impose a large burden for the child and its parents, especially when complaints persist as children grow older. It is disappointing that research has not yielded a well-established treatment thus far for constipation. It seems crucial in the research field on functional defecation disorders in childhood to address differential effectivity across diagnostics subcategories (constipation with or without fecal incontinence vs. functional non-retentive fecal incontinence). Literature shows physical abnormalities only partially explain the persistence of constipation in childhood. Stool-withholding behavior and defecation anxiety seem of great importance in the development and maintenance of chronic childhood constipation. In addition, comorbid factors, i.e. behavioral problems, difficult temperament and parent-child interaction problems, are considered to be related with the onset or maintenance of constipation in children. This indicates a behavioral intervention program in addition to laxative treatment seems to be beneficial for constipated children and their parents in the pediatric setting. Referral to a mental health service might be useful for those constipated children who exhibit social withdrawal, a low self-esteem and depressive behavior due to the defecation disorder. In case of treatment resistance and family problems, referral is also preferred.

Conclusion

This article will hopefully serve as an extensive guideline to understand and utilize the knowledge in routine practice to treat constipated children. The literature review presents underlying theories from which the treatment techniques follow. By releasing the described protocolized behavioral intervention program and offering a theoretical framework, it will contribute to the implementation of an effective treatment for chronic constipation in childhood as well.

References

1. Benninga MA. Children with constipation: what happens to them when they grow up? *Scand J Gastroenterol* 2004;241:23-6.

2. Rasquin A, Di Lorenzo C, Forbes D, Guiraldes E, Hyams JS, Staiano A, et al. Childhood functional gastrointestinal disorders: child/adolescent. *Gastroenterology* 2006;**130**(5):1527-37.
3. Voskuijl WP, Heijmans J, Heijmans HS, Taminiu JA, Benninga MA. Use of Rome II criteria in childhood defecation disorders: applicability in clinical and research practice. *J Pediatr* 2004; **145**(2):213-7.
4. Loening-Baucke V. Functional fecal retention with encopresis in childhood. *J Pediatr Gastroenterol Nutr* 2004;**38**(1):79-84.
5. Loening-Baucke V. Chronic constipation in children. *Gastroenterology* 1993;**105**(5):1557-64.
6. Bernard-Bonin AC, Haley N, Belanger S, Nadeau D. Parental and patient perceptions about encopresis and its treatment. *J Dev Behav Pediatr* 1993;**14**(6):397-400.
7. Blum NJ, Taubman B, Nemeth N. During toilet training, constipation occurs before stool toileting refusal. *Pediatrics* 2004;**113**(6):520-2.
8. Cox DJ, Sutphen J, Borowitz S, Kovatchev B, Ling W. Contribution of behavior therapy and biofeedback to laxative therapy in the treatment of pediatric encopresis. *Ann Behav Med* 1998;**20**(2):70-6.
9. Loening-Baucke V. Constipation in early childhood: patient characteristics, treatment, and longterm follow up. *Gut* 1993;**34**(10):1400-4.
10. Loening-Baucke V. Prevalence, symptoms and outcome of constipation in infants and toddlers. *J Pediatr* 2005;**146**(3):359-63.
11. Partin JC, Hamill SK, Fischel JE, Partin JS. Painful defecation and fecal soiling in children. *Pediatrics* 1992;**89**(6):1007-9.
12. Di Lorenzo C. Childhood constipation: finally some hard data about hard stools! *J Pediatr* 2000;**136**(1):4-7.
13. Levine MD, Bakow H. Children with encopresis: a study of treatment outcome. *Pediatrics* 1976;**58**(6):845-52.
14. Baker SS, Liptak GS, Colletti RB, Croffie JM, Di LC, Ector W, et al. Constipation in infants and children: evaluation and treatment. A medical position statement of the North American Society for Pediatric Gastroenterology and Nutrition. *J Pediatr Gastroenterol Nutr* 1999;**29**(5):612-26.
15. Benninga MA, Voskuijl WP, Taminiu JA. Childhood constipation: is there new light in the tunnel? *J Pediatr Gastroenterol Nutr* 2004;**39**(5):448-64.
16. Destrée-Vonka, Onland-van Nieuwenhuizen AM. Onderzoeksprotocol voor de gedragstherapeutische behandeling van obstipatie bij kinderen van 4 tot 8 jaar en van 8 tot 18 jaar. Amsterdam: Psychosociale Afdeling, Emma Kinderziekenhuis/AMC;2001.
17. vanKuyk EM, Brugman-Boezeman AT, Wissink-Essink M, Severijnen RS, Festen C, Bleijenberg G. Defecation problems in children with Hirschsprung's disease: a biopsychosocial approach. *Pediatr Surg Int* 2000;**16** (5- 6):312-6.
18. Brugman A, Wissink-Essink ML, Severijnen RSV. De multidisciplinaire gedragstherapie van obstipatie en incontinentie. In: Looiaard N, Platenkamp J, van Velze-Boendermaker S, van Waarden-Koets A, editors. Waar is de uitgang? Een boekje open over anusatresie. Meppel: Drukkerij Giethoorn ten Brink; 2002; p.76-82.
19. Taitz LS, Wales JK, Urwin OM, Molnar D. Factors associated with outcome in management of defecation disorders. *Arch Dis Child* 1986;**61**(5):472-7.
20. Ip KS, Lee WT, Chan JS, Young BW. A community-based study of the prevalence of constipation in young children and the role of dietary fibre. *Hong Kong Med J* 2005; **11**(6):431-6.
21. Miele E, Simeone D, Marino A, Greco L, Auricchio R, Novek SJ, et al. Functional gastrointestinal disorders in children: an Italian prospective survey. *Pediatrics* 2004;**114**(1):73-8.
22. van den Berg MM, Benninga MA, Di Lorenzo C. Epidemiology of childhood constipation: a systematic review. *Am J Gastroenterol* 2006;**101**(10):2401-9.
23. Fontana M, Bianchi C, Cataldo F, Conti Nibali S, Cucchiari S, GobioCasali L, et al. Bowel frequency in healthy children. *Acta Paediatr Scand* 1989;**78**:682-4.
24. Loening-Baucke V. Chronic constipation in children. *Gastroenterology* 1993;**105**:1557-64.
25. Benninga M, Candy DC, Catto-Smith AG, Clayden G, Loening-Baucke V, Lorenzo CD, et al. The Paris consensus on childhood constipation terminology (PACCT) Group. *J Pediatr Gastroenterol Nutr* 2005;**40**:273-5.
26. Hyman PE, Milla PJ, Benninga MA, Davidson GP, Fleisher DF, Taminiu J. Childhood functional gastrointestinal disorders: neonate/toddler. *Gastroenterology* 2006;**130**:1519-26.
27. Rasquin A, Di Lorenzo C, Forbes D, Guiraldes E, Hyams JS, Staiano A, et al. Childhood functional gastrointestinal disorders: child/adolescent. *Gastroenterology* 2006;**130**:1527-37.

28. Wheatley JM, Hutson JM, Chow CW, et al. Slow transit constipation in childhood. *J Pediatr Surg* 1999;**34**(5):829-832.
29. Borowitz SM, Cox DJ, Tam A, et al. Precipitant of constipation during early childhood. *J Am Board FamPract*2003;**16**(3):213-18.
30. Blethyn AJ, Verrier-Jones K, Newcombe R, et al. Radiological assessment of constipation. *Arch Dis Child* 1995;**73**(6):532-3.
31. Pensabene L, Buonomo C, Fishman L, et al. Lack of utility of Abdominal X-ray in the evaluation of children with constipation: comparison of different scoring methods. *J Ped Gastroenterol Nutr* 2010; **51**(2):155-159.
32. Benninga MA, Voskuil WP, Taminiau JAJM. Childhood constipation: Is there new light in the tunnel? *J Pediatr Gastroenterol Nutr* 2004;**39**:448-64.
33. Price KJ, Elliott TM. Stimulant laxatives for constipation and soiling in children. *Cochrane Database Syst Rev* 2001;(3):CD002040.
34. Brazzelli M, Griffiths P. Behavioural and cognitive interventions with or without other treatments for defecation disorders in children. *Cochrane Database Syst Rev* 2006;(2): CD002240.
35. Pitzalis G, Mariani P, Chiarini-Testa MR, Virgili F, Gasparri R, Calvani L, et al. Lactitol in chronic idiopathic constipation of childhood. *Pediatr Med Chir* 1995;**17**:223-6.
36. Martino AM, Pesce F, Rosati U. The effects of lactitol in the treatment of intestinal stasis in childhood. *Minerva Pediatr* 1992;**44**:319-23.
37. Hejlp M, Kamper J, Ebbesen F, Hansted C. Infantile constipation and allomin-lactulose. Treatment of infantile constipation in infants fed with breast milk substitutes: a controlled trial of 2% and 4% allomin-lactulose. *UgeskrLaeger* 1990;**152**:1819-22.
38. Voskuil W, de Lorijn F, Verwijs W, Hogeman P, Heijmans J, Makel W, et al. PEG 3350 (Transipeg) versus lactulose in the treatment of childhood functional constipation: a double blind, randomised, controlled, multicentre trial. *Gut* 2004;**53**:1590-4.
39. Dupont C, Leluyer B, Maamri N, Morali A, Joye JP, Fiorini JM, et al. Double blind randomized evaluation of clinical and biological tolerance of polyethylene glycol 4000 versus lactulose in constipated children. *J Pediatr Gastroenterol Nutr* 2005;**41**:625-33.
40. BSPGHAN 2005 Newsletter. <http://bspghan.org.uk/news/news05.shtml>.
41. 24. Youssef NN, Peters JM, Henderson W, Shultz-peters S, Lockhart DK, DiLorenzo C. Dose response of PEG 3350 for the treatment of childhood fecal impaction. *J Pediatr* 2002;**141**:410-4.
42. Marshall J, Hutson JM, Anticich N, Stanton MP. Antegrade continence enemas in the treatment of slow-transit constipation. *J Pediatr Surg* 2001; **36**:1227-30.
43. Levitt MA, Pena A. Surgery and constipation: when, how, yes, or no. *J Pediatr Gastroenterol Nutr* 2005;**41**:58-60.
44. Keshtgar AS, Ward HC, Clayden GS, Sanei A. Role of anal dilatation in treatment of idiopathic constipation in children: long term follow-up of a double blind randomised controlled study. *Pediatric Surgery Int* 2005;**21**:100-5.
45. Van Ginkel R, Rietsma JB, Butler HA, et al. Childhood constipation: longitudinal follow-up beyond puberty. *Gastroenterology* 2003;**125** (2):357-63.
46. Sutphen JL, Borowitz SM, Hutchinson RL, Cox DJ. Long term follow up of medically treated childhood constipation. *Clin Paediatr (Phila)*.1995;**34**(11):576-80.

CASE REPORT

Sirenomelia: The Mermaid Baby - Report Of Rare Case

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Abstract

Sirenomelia also called "THE MERMAID SYNDROME" is found approximately 1:1,00,000 live approximately 300 cases reported in the literature, 15% of which are associated with twin and most often monozygotic. births¹ and is fatal within the first day because of agenesis or abnormal development of kidney and urinary system. It results from the failure of normal vascular supply from the lower aorta. Maternal diabetes mellitus has been associated with caudal regression syndrome and sirenomelia. We are reporting a rare case of mermaid baby delivered by a non-diabetic mother.

Key words: Sirenomelia , Mermaid syndrome

Introduction

Sirenomelia is a rare and fatal congenital anomaly. The malformation sequence consists of varying degrees of lower limb fusion bearing a resemblance to the mermaid of ancient Greek mythology. Severe malformations of the gastrointestinal, genitourinary, cardiovascular, musculoskeletal systems and single umbilical artery are usually present. Oligohydromnios secondary to severe renal dysplasia is universal. The Sirenomelia was first described in medicine by Rocheus and Polfyr in the sixteenth century. Duhamel in 1961 defined all the anomalies of mermaid syndrome and commented as the most severe form of caudal regression syndrome.^{2,3} Caudal regression is a rare syndrome which represents a spectrum of congenital malformations ranging from lumbosacral spine agenesis to the most severe cases of Sirenomelia with lower extremities fusion.⁴ Sirenomelia is thought to be a part of the caudal regression spectrum. This syndrome has

strong association with maternal Diabetes mellitus with relative risk 1;200-250 and 22% of the affected fetus will have diabetic mother.^{5,6} Intake of haloperidol antenatally is considered a cause for Sirenomelia. Other proposed teratogens are cadmium, lead, vitamin A, nutritional deficit and vascular hypoperfusion. Embryotoxic and teratogenic metal in a variety of animal species were studied but data from humans are limited.^{7,8}

Case report

A 26 year second gravid woman had no antenatal check up during her pregnancy period, USG was done just after arrival for fetal distress and it was nothing contributory except severe oligohydramnios and LUCS done due to less fetal movement at 36 wks of gestation and delivered a baby with fused lower limbs from the hip downwards and it was ended like a tip of a finger (sirenomelia apus). The baby cried immediately after delivery and there was no respiratory distress. APGAR score was 8/10 at 5

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minutes of age. Her first male child was 3 years of age and healthy. There was history of ingestion of tobacco leaves by the mother during antenatal period. There was no history of ingestion of other teratogenic agents, homeopathy, oral contraceptive medicine and mother was non-diabetic. Birth weight was 2.1kg with no obvious facial dysmorphism. But the baby had gross anomaly known as sirenomelia (Fig.1 and 2), characterized as fusion of lower limbs. It was not possible to identify the sex of the baby on physical examination as there was absence of external genitalia. Imperforated anus was also apparent, only a small dimple was present at the anal region. Father and other family members refused to admit the baby and not interested to do any investigations or interventions after seeing the baby, knowing the consequences and took the baby at home. We advised the party to give comfort in the form of general care and feeding .



Fig 1 Fused lower limb and no external genitalia



Fig 2 Arrow indicate a dimple over the anal region

Discussion

Sirenomelia is a rare congenital anomaly, till date approximately 300 cases have been reported in the literature. Most of these newborns were still born or died immediately after birth; death is usually due to renal agenesis, which is incompatible with life. It represents a broad spectrum of lumbosacral agenesis . In the most severe cases of sirenomelia both sacral agenesis and caudal regression syndrome are related. In caudal regression syndrome has variable spinal anomalies varying from partial sacral agenesis to complete absence of the lumbosacral spine.⁹

Sirenomelia is a lethal condition because of bilateral renal agenesis, which leads to severe oligohydramnios and lung hypoplasia.¹⁰

Sirenomelia is the most relentless condition of these syndrome. The term comes from “siren” or “mermaid” resulting from characteristic fusion of the lower extremities. The specific anomaly of sirenomelia is based on the presence of lower limbs fusion, associated with other skeletal and lumbar deformities (sacral agenesis). For this classical features, this condition is called mothshoconnya in Bengali language and also described in Bengali literature. Classification of sirenomelia from caudal regression syndrome is still debated¹¹ and there are different theories about its pathogenesis. Sirenomelia as a part of caudal regression syndrome has its own pathogenesis. The pathogenesis of sirenomelia has been proposed to be a vascular steal phenomenon with the single, aberrant, umbilical artery stealing blood supply from the lower torso and limbs.¹²

There are three different variant depending on the degree of fusion of the lower limbs.

1. **Symelia apus:** No feet are present and the limbs are completely fused into a single limb; one femur and one tibia are present. Our case had no foot, lower limb was rather ended like a tip of finger.
2. **Symelia unipus:** One foot is present (a partial fusion of both feet), 2 femurs, 2 tibiae and 2 fibulae.
3. **Symelia dipus:** Two feet are present giving the appearance of fins, hence the term ‘mermaid fetus’ for this condition. The fusion of the limbs extends only as far as the ankles.

The condition is seen in in 100-150 times more frequently in monozygotic than dizygotic twin or

singeltons and males are three times more affected than females.¹³

In sirenomelia third trimester ultrasonography for diagnosis is usually impaired by severe oligohydramnios related to bilateral renal agenesis, whereas during the early second trimester the amount of amniotic fluid may be sufficient to allow diagnosis and earlier less traumatic therapeutic abortion can be done.

Although sirenomelia has been described as a rare lethal anomaly, 9 mermaid syndrome cases have been reported surviving after reconstructive surgery.¹⁴ The most important characteristics of survival of the affected newborn is the presence of functional kidney.

Conclusion

Sirenomelia is a lethal congenital anomaly where neonates dies within 24 hours of age without any medical intervention. This rare fatal congenital malformation with severe visceral anomalies that can be detected among the survivors during the operative procedure and at autopsy of the dead babies.

References

1. Kallen,B;Castilla, E; Lancaster, P A; Mutchinnick, O; Knudsen, L B; Marrtinez-Frias, M L; Mas troiacovo, P; Robert. "the Cyclops and the mermaid; an epidemiological study of two types of rare malformation." *Journal of Medixal Genetics* 1992;**29**(1):30-5.
2. Carbillon L, Seince N, Largillière C, Bucourt M, Uzan M. First-trimester diagnosis of sirenomelia A case report. *Fetal Diagn Ther* 2001;**16**:284-8.
3. Duhamel B. From the Mermaid to Anal Imperforation: The Syndrome of Caudal Regression. *Arch Dis Child* 1961;**36**:152-5.
4. Dordoni D, Freeman PC. Sirenomelia sequence. *The Fetus* 1991;1:7553-9.
5. Aslan H, Yanik H, Celikaslan N, Yildirim G, Ceylan Y. Prenatal diagnosis of Caudal Regression Syndrome: a case report. *BMC Pregnancy and Childbirth* 2001;**1**:8.
6. Gonzalez-Quintero VH, Tolaymat L, Martin D, Romaguera RL, Rodriguez MM, Izquierdo LA. Sonographic diagnosis of Caudal Regression in the first trimester of pregnancy. *J Ultrasound Med* 2002;**21**:1175-8.
7. Maria Kippler, Fahmida Tofail, Renee Gardner, Anisur Rahman, Jena D. Hamadani,² Matteo Bottai, Marie Vahter. Maternal Cadmium Exposure during Pregnancy and Size at Birth: A Prospective Cohort Study. *Environ Health Perspect* 2012;**120**(2): 284-9.
8. Rothman KJ, Moore LL, Singer MR, Nguyen US, Mannio S, Milunsky A. Teratogenicity of high Vitamin A intake. *N England J Medicine* 1995;**23**(21):1396-73.
9. Jaffe R, Zeituni M, Fejgin M. Caudal regression syndrome. *Fetus Spinal Anomalies* 1991; **7561**:1-3.
10. Benaceraf BR. Caudal regression syndrome and sirenomelia in ultrasound of fetal syndrome, Churchill Livingstones, New York 1998;250-4.
11. Depraetere M, Dehawere R, Marien P. Severe axial mesodermal dysplasia spectrum in the infant of a diabetic mother. *Genet Couns* 1995;**6**:303-7.
12. Twickler D, Budorick N, Pretorius D, Grafe M. Caudal regression versus sirenomelia. *J Ultrasound Med* 1993; **12**:323-30.
13. Van Keirsblick J, Cannie M, Robrechts C, de Ravel T, Dymarkowski S, Va den Bosch T, et al. First trimester diagnosis of Sirenomelia. *Prenat Diagn* 2006;**26**:684-8.
14. Romano S, Esposito V, Fonda C, Russo A, Grassi R. Beyond the Myth: The mermaid syndrome from Homeus to Andersen A tribute to Hans Christian Anderson's bicentennial of birth. *Eur J Radiol* 2006;**58**:252-9.

CASE REPORT

A Tale of Early Age of Onset of Guillain-Barre Syndrome

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Introduction

Guillain-Barré syndrome (GBS) also known as Landry's ascending paralysis, a post infectious rapid onset immune mediated nerve dysfunction, a leading cause of acute flaccid paralysis worldwide, with an incidence between 0.6 and 4 per 100,000 per year.¹ French physician Jean-Baptiste Octave Landry first described the disorder in 1859.² In 1916, Georges Guillain, Jean Alexandre Barré, and André Strohl diagnosed the illness in two soldiers and described the key diagnostic abnormality, albuminocytological dissociation i.e. increased spinal fluid protein concentration but a normal cell count.³ Canadian neurologist C. Miller Fisher in 1956 described the variant.⁴ (1) Acute inflammatory demyelinating polyneuropathy (AIDP), in which sensory symptoms and muscle weakness, often with cranial nerve weakness and autonomic involvement occurs, demyelinating polyneuropathy most common in Europe and North America and has no clear association with antiganglioside antibodies. (2) Acute motor axonal neuropathy (AMAN): Isolated muscle weakness without sensory symptoms in less than 10%; cranial nerve involvement uncommon, axonal polyneuropathy is rare in Europe and North America, substantial proportion (30-65%) in Asia and Central and South America; sometimes called "Chinese paralytic syndrome", normal sensory action potential, association with antiganglioside antibodies (GM1a/b, GD1a & GalNac-GD1a). (3) Acute motor and sensory axonal neuropathy (AMSAN): Severe muscle weakness similar to AMAN but with sensory loss, axonal polyneuropathy, reduced or absent sensory action potential, association with antiganglioside antibodies (GM1, GD1a). (4) Pharyngeal-cervical-brachial variant: Weakness

particularly in the muscles of the throat, face, neck and shoulder girdle, sometimes axonal neuropathy in the arms and it is associated with antiganglioside antibodies (mostly GT1a, occasionally GQ1b, rarely GD1a). (5) Miller Fisher syndrome: Ataxia, eye muscle weakness, areflexia but usually no limb weakness, this variant occurs more commonly in men than in women (2:1). Cases typically occur in the spring and the average age of occurrence is 43 years old,⁵ association with antiganglioside antibodies (GQ1b, GT1a). Diagnostic criteria of GBS were developed in the late 1970s after the series of cases associated with swine flu vaccination. These were refined in 1990.⁶ Management of GBS with plasma exchange was first used in 1978 and its benefit confirmed in larger studies in 1985. Intravenous immunoglobulins were introduced in 1988, and its non-inferiority compared to plasma exchange was demonstrated in studies in the early 1990s.⁷ This devastating syndrome presents initially as numbness, tingling, and pain, alone or in combination. This is followed by weakness of the legs and arms that affects both sides equally and worsening in time even respiratory failure.⁸ In one in five people the weakness continues to progress for as long as four weeks.⁹ The muscles of the neck may also be affected, and about a half experience involvement of the cranial nerves, which supply the head and face; this may lead to weakness of the muscles of the face, swallowing difficulties and sometimes weakness of the eye muscles. In 8% the weakness affects only the legs (paraplegia or paraparesis).⁹ Involvement of the muscles that control the bladder and anus is unusual. In total, about a third of people with Guillain-Barré syndrome

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continue to be able to walk.⁵ Once the weakness has stopped progressing, it persists at a stable level (“plateau phase”) before improvement occurs. The plateau phase can take between two days and six months, but the most common duration is a week.⁹ Pain-related symptoms affect more than half, and include back pain, painful tingling, muscle pain and pain in the head and neck relating to irritation of the lining of the brain.⁹ Many people with Guillain-Barré syndrome have experienced the signs and symptoms of an infection in the 3–6 weeks prior to the onset of the neurological symptoms. This may consist of upper respiratory tract infection (rhinitis, sore throat) or diarrhea.⁸ Axonal variant of Guillain-Barre syndrome associated with *Campylobacter* infection in Bangladesh. It has been reported 69% of Guillain-Barre Syndrome had clinical evidence of a preceding infection.¹⁰ The virulence of *Campylobacter jejuni* is thought to be based on the presence of specific antigen in its capsule that are shared with nerves.¹¹ The level of consciousness is normally unaffected in Guillain–Barré syndrome. This life threatening scenario is complicated by other medical problems like pneumonia, septicaemia, autonomic dysfunction e.g. heart rate, blood pressure and perspiration to the point of clinical importance.¹² The nerve dysfunction in Guillain-Barré syndrome is caused by an immune attack on the peripheral nerves myelin sheath which are injured or degraded, the nerves cannot transmit signals efficiently resulting in devastating weakness of limbs.⁸ In those with severe weakness, prompt treatment with intravenous immunoglobulins or plasmapheresis, together with supportive care, will lead to good recovery in the majority. Some may experience ongoing difficulty with walking, painful symptoms, and some require long-term breathing support. Despite its low incidence, that has a high impact on prolonged hospitalizations with intensive care, high cost procedures and disability sequelae upto 20% of cases and mortality between 2% - 10%.¹³ Guillain-Barre Syndrome affects all ages but is rare in childhood, the risk is increased by 20% for every decade of life.¹⁴ In children, particularly those younger than six years old, the diagnosis can be difficult and the condition is often initially mistaken (sometimes for up to two weeks) for other causes of pains and difficulty in walking such as viral infections,⁹ or bone and joint problems. So therefore we are presenting a case of Guillain–Barré syndrome presents in early infancy.

Case Report

An eight months old female baby, only issue of a nonconsanguinous parents was admitted in intensive care unit of Dhaka Shishu Hospital on 14th April 2015 with the complaints of inability to move both lower limbs followed by respiratory difficulty for 4 days. She had an episode of gastroenteritis ten days prior to this incidence. She has no history of recent vaccination. Initially the young baby was admitted in a local private clinic of Dhaka city without improvement. On examination she was conscious with respiratory difficulty, endotracheal tube in situ and put on mechanical ventilation. Her pulse, blood pressure were normal. On respiratory system examination, bilateral crepitations were present. Capillary refill time was less than three seconds. On neurological examination the baby was alert cranial nerves were intact, muscle bulk in both upper and lower limbs was normal but hypotonic and areflexic. Other systemic examinations were normal. Relevant investigation were done, where normal complete blood count, random blood sugar, serum electrolytes and serum calcium. CRP was within normal limit. Blood culture showed no growth. Chest radiograph showed consolidation of upper zone of right lung field. Blood gas analysis showed respiratory acidosis. Initially she was managed in ICU with supportive medical treatment by IV fluid, Inj Ceftriaxon, Inj Vancomycin and IV immunoglobulin for three days. Four days after admission in ICU, patient was extubated, put on head box followed by O₂ in face mask. At twelve days of illness, lumbar puncture was done for CSF analysis showed albumino-cytologic dissociation (raised protein 200mg/dl and cell count 4/mm³). Considering history and clinical findings and supportive investigation (eg CSF study) the child was diagnosed as Guillian – Barre Syndrome.



Fig 1 Shows child in mechanical ventilation



Fig 2 Shows child in Head box



Fig 3 In ward with mother emotional-affectionate support

Discussion

Guillain-Barre Syndrome can be described as a collection of clinical syndromes that manifest as an acute demyelinating polyradiculoneuropathy with resultant ascending weakness and diminished reflexes. Difficulty with facial movements including swallowing and choking in saliva 25% people with Guillain-Barre Syndrome develops weakness of the breathing muscles leading to respiratory failure, inability to breathe adequately to maintain healthy level of O_2 and CO_2 in the blood.¹⁵ This may require intubation and breathing support of mechanical ventilation, generally on an ICU. This devastating disease may be associated with other abnormalities like autonomic dysfunction eg heart rate, blood pressure, perspiration and changes in the pupillary response. Vaccinations have been occasionally linked to Guillain-Barre Syndrome.¹⁶ Guillain-Barre Syndrome has been reported in all age groups, seems to be bimodal with a first peak in young adulthood (age 15-35y) and a second, higher one in middle-aged and elderly persons (ages 50-75y). Infants appear to have the lowest risk of developing Guillain-Barre Syndrome.¹⁷ In respect of investigation CSF analysis

is considered to best modality in the diagnosis of Guillain-Barre Syndrome, where raised protein and normal cell count reveals albumino-cytologic dissociation. Confirmed the diagnosis as early, proper intensive care including the following features play important role to minimize the morbidity and mortality.¹⁸ Respiratory and cardiac monitoring with proper therapy. Safe nutritional supplement. Treatment with IV immunoglobulin may hasten recovery.¹⁹ Physiotherapy to active muscle strengthen can be slowly introduced to improve their functional status.²⁰ The prognosis in our case seems better. Regular follow-up the child is essential because of any possibility of acute relapse after initial improvement or any neurologic sequale after treatment. Guillain-Barre Syndrome can produce long lasting changes in the physical and psychosocial status of patient and their families.²¹ Family education should be recommended on the illness, the disease process and the anticipated course to support physical well being of the child and psychosocial performance of her family.

Conclusion

This is the first case at such an early age with Guillain-Barre Syndrome successfully treated in our ICU. At this age confusions may arise with Spinal muscular atrophy, botulinism but Guillain-Barre Syndrome is a possibility. We should be aware about this.

References

1. McGpogan A, Madle GC, Seaman HE. The epidemiology of Guillain-Barre Syndrome, worldwide. A systemic literature review. *Neuroepidemiology* 2009;**32**(2):150-63.
2. Landry, Jean-Baptiste. *Gazette Hebdomadaire de Médecine et de Chirurgie* 1859;**6**: 472-4.
3. van Doorn, Pieter A; Ruts, Liselotte; Jacobs, Bart C "Clinical features, pathogenesis, and treatment of Guillain-Barré syndrome". *The Lancet Neurology* 2008;**7**(10):939-50.
4. Shahrizaila, N.; Yuki, N. "Bickerstaff brainstem encephalitis and Fisher syndrome: anti-GQ1b antibody syndrome". *Journal of Neurology, Neurosurgery & Psychiatry* 2012;**84**(5): 576-83.
5. Mori M, Kuwabara S, Yuki N "Fisher syndrome: clinical features, immunopathogenesis and management". *Expert Rev Neurother* 2012;**12**(1): 39-51.

6. Asbury, Arthur K.; Cornblath, David R. "Assessment of current diagnostic criteria for Guillain-Barré syndrome." *Annals of Neurology* 1990;**27**:21-4.
7. Walgaard, Christa; Jacobs, Bart C; van Doorn, Pieter A. "Emerging drugs for Guillain-Barré syndrome". *Expert Opinion on Emerging Drugs* 2011;**16**(1):105-20.
8. Yuki, Nobuhiro; Hartung, Hans-Peter "Guillain-Barré Syndrome". *New England Journal of Medicine* 2012;**366**(24):2294-304.
9. Van den Berg, Bianca; Walgaard, Christa; Drenthen, Judith; Fokke, Christiaan; Jacobs, Bart C.; van Doorn, Pieter A. "Guillain-Barré syndrome: pathogenesis, diagnosis, treatment and prognosis". *Nature Reviews Neurology* 2014;**10**(8): 469-82.
10. Islam Z, Jacos BC, Van Belkum A. Axonal variant of Guillian-Barre Syndrome associated with Campylobacter infection in Bangladesh. *Neurology* 2010;**74** (7):581-7.
11. Jacobs BC, van Doorn PA, Schmitz PI. Campylobacter Jejuni infection and anti-GM1 antibodies in Guillian-Barre Syndrome. *Ann Neurology* 1996;**40**(2);181-7.
12. Walgaard C, Lingsma HF, Ruts. prediction of respiratory insufficiency in Guillian-Barre Syndrome *Ann Neurol* 2010;**67**(6);781-7.
13. Van Doorn PA, Rub L, Jacobs BC. Clinical features, pathogenesis and treatment of Guillian-Barre Syndrome. *Lancet Neurology* 2008;**7**(10):939-50.
14. Govoni V, Gronieri E. Epidemiology of Guillian-Barre Syndrome. *Current Neurology* 2001;**14**(5):605-13.
15. Hughes RA, Rees JH. Clinical and epidemiologic features of Guillian-Barre Syndrome. *Journal of Infections disease* 1997;**176**(2):92-8.
16. Souayah N, Nasar A, Suri MF. Guillian-Barre Syndrome after vaccination in United States data from the centers for Disease control and Prevention / Food and Drug. Administration Vaccine Adverse Event Reporting System (1990-2005). *Journal of Clinical Neuromuscular Disease* 2009; **1**(I):1-6.
17. Evans OB, Vedanarayanan V. Guillian-Barre Syndrome. *Paediatric Review* 1997;**18**(1):10-6.
18. Bernsen RA, de Jager AE, Schmitz PI. Residual physical outcome and daily living 3 to 6 years after Guillian-Barre Syndrome. *Neurology* 1999;**53**(2): 409-10.
19. Hughes RA, Pritchard J, Hadeden RD. Pharmacological treatment other than corticosteroids, intravenous immunoglobulin and plasma exchange for Guillian-Barre Syndrome. *Cochrane Database Systemic Review* 2013;**2**:245-57.
20. Mullings KR, Alleva JT, Hudgins TH, Rehabilitation of Guillian-Barre Syndrome. *Dis Mon* 2010;**56**(15): 288-92.
21. Bersano A, Carpo M Allaria S, Long term disability and social status change after Guillian-Barre Syndrome. *Journal of Neurology* 2006;**253** (2):214-8.

ABSTRACT FROM CURRENT LITERATURE

Soft Drinks Consumption Is Associated with Behavior Problems in 5-Year-Olds

Shakira F. Suglia, Sara Solnick and David Hemenway

J Pediatr 2013;163:1323-8

Objective: To examine soda consumption and aggressive behaviors, attention problems, and withdrawal behavior among 5-year-old children.

Study design: The Fragile Families and Child Wellbeing Study is a prospective birth cohort study that follows a sample of mother-child pairs from 20 large US cities. Mothers reported children's behaviors using the Child Behavior Checklist at age 5 years and were asked to report how many servings of soda the child drinks on a typical day.

Results: In the sample of 2929 children, 52% were boys, 51% were African-American, 43% consumed at least one serving of soda per day, and 4% consumed 4 or more servings per day. In analyses adjusted for sociodemographic factors, consuming one (beta, 0.7; 95% CI, 0.1-1.4), 2 (beta, 1.8; 95% CI, 0.8-2.7), 3 (beta, 2.0; 95% CI, 0.6-3.4), or 4 or more (beta, 4.7; 95% CI, 3.2-6.2) servings was associated with a higher aggressive behavior score compared with consuming no soda. Furthermore, those who consumed 4 or more (beta, 1.7; 95% CI, 1.0-2.4) soda servings had higher scores on the attention problems subscale. Higher withdrawn behavior scores were noted among those consuming 2 (beta, 1.0; 95% CI, 0.3-1.8) or 4 or more (beta, 2.0; 95% CI, 0.8-3.1) soda servings compared with those who consumed no soda.

Conclusion: We note an association between soda consumption and negative behavior among very young children; future studies should explore potential mechanisms that could explain this association.

Budesonide versus Prednisone with Azathioprine for the Treatment of Autoimmune Hepatitis in Children and Adolescents

Marek Woynarowski, Antal Nemeth, Yaacov Baruch, Sibylle Koletzko, Michael Melter, Burkhard Rodeck, Christian P. Strassburg, Markus Prols, Małgorzata Woźniak, and Michael P. Manns

J Pediatr 2013;163:1347-53

Objective: To compare the effect of budesonide vs prednisone therapy both in combination with

azathioprine in pediatric patients with autoimmune hepatitis (AIH).

Study design: Forty-six patients with AIH (11 males and 35 females) aged 9-17 years were enrolled in a 6-month, prospective, double-blind, randomized, active-controlled, multicenter phase IIb study evaluating budesonide (n = 19; 3 mg twice or 3 times daily) vs prednisone (n = 27; 40 mg/day tapered to 10 mg/day), both with azathioprine (1-2 mg/kg/day), followed by a further 6 months of open-label budesonide therapy. The primary efficacy endpoint was complete biochemical remission (normal serum alanine aminotransferase and aspartate aminotransferase levels) without predefined steroid-specific side effects.

Results: We observed no statistically significant difference in the percentage of patients who met the primary endpoint between the budesonide (3 of 19; 16%) and prednisone groups (4 of 27; 15%) after 6 months, nor in the percentage of patients who experienced biochemical remission (budesonide, 6 of 19 [32%]; prednisone, 9 of 27 [33%]), lack of steroid-specific side effects (budesonide, 10 of 19 [53%]; prednisone, 10 of 27 [37%]). The mean weight gain was 1.2 _ 3.5 kg in the budesonide group and 5.1 _ 4.9 kg in the prednisone group (P = .006). A total of 42 patients received open-label budesonide treatment for another 6 months. After 12 months, 46% of these patients achieved complete remission.

Conclusion: Oral budesonide with azathioprine can induce and maintain remission in pediatric patients with AIH and may be considered an alternative therapy to prednisone. The treatment causes fewer side effects and does not lead to weight gain; however, it may be less effective than prednisone in inducing remission.

Endothelial Dysfunction in Children within 5 Years after Onset of Kawasaki Disease

Takamichi Ishikawa and Satoru Iwashima

J Pediatr 2013; 163:1117-21

Objective: To evaluate endothelial function in children within 5 years after the onset of Kawasaki disease (KD).

Study design: A total of 46 children were enrolled prospectively as follows: 9 patients with KD and coronary artery lesions composed group 1, 15 patients

with KD but without coronary artery lesions composed group 2, and 22 healthy age- and sex-matched children composed group 3. Flow-mediated dilatation (FMD) of the brachial artery, intima-media thickness of the common carotid artery, and biologic characteristics were compared among the 3 groups. Differences in the factors associated with endothelial function after KD were examined as well.

Results: The mean age of the study group was 6.5 ± 1.7 years. The patients with KD were studied at a median interval of 3.3 years (IQR, 2.0-4.4 years) from the onset of disease. The percent FMD (%FMD) was significantly lower in group 1 patients (median, 4.4%; IQR, 2.6%-5.7%) compared with both group 2 (median, 9.1%; IQR 6.6%-10.7%; $P < .01$) and group 3 (median, 11.1%; IQR, 10.1%-13.9%, $P < .01$). The %FMD was statistically significantly lower in group 2 compared with group 3 ($P < .05$). There were no significant differences in the intima-media thickness among the 3 groups. There was a significant negative correlation between %FMD and the total duration of fever ($r = 0.50$; $P = .013$).

Conclusion: The children with KD already had arterial endothelial dysfunction within 5 years after the onset of illness. The longer the duration of fever, the greater the risk of inflammation-induced endothelial dysfunction.

Oral Corticosteroids and Onset of Cardiomyopathy in Duchenne Muscular Dystrophy

Brent J. Barber, Jennifer G. Andrews, Zhenqiang Lu, Nancy A. West, F. John Meaney, Elinora T. Price, Ashley Gray¹, Daniel W. Sheehan, Shree Pandya, Michele Yang, and Christopher Cunniff

J Pediatr 2013;163:1080-4

Objective: To estimate the age when cardiomyopathy develops in boys with Duchenne

muscular dystrophy (DMD) and to analyze the effect of corticosteroid treatment on the age of cardiomyopathy onset.

Study design: We identified a population-based sample of 462 boys with DMD, born between 1982 and 2005, in 5 surveillance sites in the US. Echocardiographic and corticosteroid treatment data were collected. Cardiomyopathy was defined by a reduced fractional shortening ($<28\%$) or ejection fraction ($<55\%$). The age of cardiomyopathy onset was determined. Survival analysis was performed to determine the effects of corticosteroid treatment on cardiomyopathy onset.

Results: The mean (SD) age of cardiomyopathy onset was 14.3 (4.2) years for the entire population and 15.2 (3.4) years in corticosteroid-treated vs 13.1 (4.8) in non-treated boys. Survival analysis described a significant delay of cardiomyopathy onset for boys treated with corticosteroids ($P < .02$). By 14.3 years of age, 63% of non-treated boys had developed cardiomyopathy vs only 36% of those treated. Among boys treated with corticosteroids, there is a significant positive effect of duration of corticosteroid treatment on cardiomyopathy onset ($P < .0001$). For every year of corticosteroid treatment, the probability of developing cardiomyopathy decreased by 4%.

Conclusions: Oral corticosteroid treatment was associated with delayed cardiomyopathy onset. The duration of corticosteroid treatment also correlated positively with delayed cardiomyopathy onset. Our analysis suggests that a boy with DMD treated for 5 years with corticosteroids might experience a 20% decrease in the likelihood of developing cardiomyopathy compared with untreated boys.

DSH NEWS



Opening ceremony and Inauguration of NICU of Dhaka Shishu Hospital on 13th February 2014 and Doa with Our Honourable Chairman of the management Board National Professor Dr. Shahla Khatun along with our honourable Director Professor Manzoor Hussin and Professor Khaleda Banu Ex. Head of the Department of Neonatology and renowned surgeon Professor Golam Rasul



Celebration of World Heart Day on 12th April 2014 with rally started from Dhaka Shishu Hospital Compound with our honourable director Professor Manzoor Hussain and Academic Director Professor M.A.K. Azad Chowdhury

BICH NEWS

BICH is the academic wing of Dhaka Shishu Hospital. It was established in 30th January, 1983. It is affiliated with Dhaka University, Bangabandhu Sheikh Mujib Medical University (BSMMU) and Bangladesh College of Physicians and Surgeons (BCPS). It has been conducting different courses e.g. DCH, FCPS, MD Paediatrics, MS Paediatric surgery & B.Sc in Health technology. It also conduct different sub-specialty courses e.g. FCPS Neonatology, FCPS Haemato-oncology, FCPS Nephrology, MD Neonatology, MD Haemato-oncology and MD Nephrology. It conducts 3 months certificate course in Paediatrics and 15 days Intensive course for MCPS. It organizes IMCI training and Palli Shishu Rural Health Training. Apart from this, the Institute also run its regular academic activities. It has established Basic Science Department since 2006.

Diploma course of paediatric nursing has started from 1st January 2012 and Diploma in paediatric physiotherapy under process.

Library facilities

The library of BICH has a rich collection of updated medical texts and reference books and reputed Medical Journals of home and abroad. BICH has introduced Broad Band facilities which are open to all students, teachers/ consultants of hospital for 24 hours. Facilities of library are also improved by HINARI. Students can download 2230 Medical Journals & more than 50 Paediatric Journal.

Present News

A newly formed classroom in BICH has been named as Prof. Sultan Ahmed Chowdhury as a tribute to First Honorary Director of Dhaka Shishu Hospital.

Postgraduate courses/training in paediatrics and child health

1. FCPS in paediatrics : Twice in a year, in the months of January and July.
2. Recognized center by BCPS for training in FCPS (Paeditric surgery).
3. Recognized centre by BCPS for course and training in different subspeciality as: Neonatology, Pediatric nephrology, Paediatric haemato-oncology, Paediatric pulmonology & Paediatric neurology and development.
4. MD/MS in paediatrics : Part I: In the month of January every year; 2nd and 3rd part twice every year.
5. DCH course : Once in a year in the month of July.
6. Three months certificate course : The institute every year runs 3 months certificate course on paediatrics for general practitioners & other post graduate candidates e.g. MCPS.
(1st August – 31st October)
7. Training programme on IMCI (Integrated management of childhood illness), Essential Newborn Care for doctors and nurses. KMC (Kangaroo Mother Care) training, ETAT (Emmergency Triage, Assessment and Treatment) and CNCP (comprehensive newborn care package) training etc.

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Students qualified from Bangladesh Institute of Child Health

Undergoing Courses of BICH

Institution	Courses
Bangabandhu Sheikh Mujib Medical University	MD (Paediatric) MD (Neonatology) MD (Nephrology) DCH MS (Paediatric Surgery)
Bangladesh Collage of Physicians and Surgeons (BCPS)	FCPS (Paediatric) FCPS (Neonatology) FCPS (Haemato-Oncology) FCPS (Paed. Neurology & Development)
Dhaka University	BSc (Health Technology)

Students Qualified from BICH till January 2014

Name of Courses	Number
DCH	312
MD (Paediatrics)	81
MS (Paediatrics)	76
FCPS (Paediatrics)	20
MD (Neonatology)	9
MD (Paediatric Nephrology)	2
Total	500

Foreign Students Qualified from BICH till January 2014

Country of Origin	Course	Number
Nepal	DCH	23
	MS(Paed. Surgery)	2
	MD(Paed)	1
India	MD(Paed)	1
Iran	DCH	1
Iraq	DCH	1
Somalia	DCH	1
Sudan	DCH	1
Total		31

Present Students (January 2014)

Name of Courses	Number of Students
DCH	- 14
MD (Paediatrics)	Part-I 13
MD (Paediatrics)	Part-II 8
MD (Paediatrics)	Final Part 2
MS (Paediatrics)	Part-I 6
MS (Paediatrics)	Part-II 2
MS (Paediatrics)	Final Part 6
FCPS (Paediatrics)	Part-II 2
FCPS (Paediatric Nephrology)	- 1
Total	36

Seminar/Symposium & CME/CPD programs held at BICH (January to June, 2014)

Date	Topic	Presenter
23/03/2014	Wilson's Disease	Department of Paediatric Gastroenterology Hepatology & Nutrition, Dhaka Shishu (Children) Hospital
18/05/2014	Systemic Lupus Erythematosus (SLE) in Children	Department of Paediatric Rheumatology, Dhaka Shishu (Children) Hospital
08/06/2014	Scope of Minimal Invasive Surgery in Pediatrics	Department of Minimal Invasive Surgery, (SU-5), Dhaka Shishu (Children) Hospital
29/06/2014	Nephrotic Syndrome	Department of Paediatric Nephrology, Dhaka Shishu (Children) Hospital