

Osmani Medical Teachers Association Journal

Volume 14: Number 1
January 2015

Editorial

Use Of Botulinum Toxin In Different Neurological Disorders..... 3
Matiur Rahman and Mostafa Hosen

Original Articles

Randomized Controlled Trial of Baclofen and Diazepam in Improving Gross Motor Function in Cerebral Palsy..... 5
Singha J, Hossain M, Chowdhury T, Biswas P, Singha RK, Rahman ME, Shaha N, Begum S

Correlation of Preoperative Serum Level of CA 125 with Different Histopathological Types of Ovarian Carcinoma 11
Hossain MS, Chowdhury MA, Khan MAH, Hossain MA, Ansari MAS, Bhowmik DK, Chowdhury S

Comparative study of caudal anaesthesia - analgesia with general anaesthesia - opioid analgesia for lower abdominal surgery in paediatric patients..... 15
Dey GR, Dev PK, Miftahuddin S, Biswas P

Pearls and Pitfalls in the Management of Geriatric Trauma and Orthopaedic problems..... 19
Talukder DN, Fattah I, Miah AS, Hannan M.A, Islam F, Khan M, Das SR

Clinical Profile and Short Term Outcome of Acute Stroke: A Comparison between Diabetic and Non Diabetic Patients..... 24
Hossain ME, Ahammed F, Ray S, Abdullah AS, Karmaker G

An overview of 45 cases of abdominal hysterectomy in non-descended uterus.....30
Das SR, Chowdhury MA, Bhoumik DK, Nath S

Detection of Chlamydia trachomatis, Mycoplasma hominis and Ureaplasma urealyticum in patients with sterile pyuria by Polymerase Chain Reaction.....32
Siddiqui I, Islam KMS, Nehar N, Pervin S, Akter S

Prevalence of Multidrug resistant Tuberculosis (MDR- TB) in Sylhet Metropolitan City.....36
Naher BL, Rukan MBH, Alam MM, Uddin MB, Hossain A

Sonouroethrography in the evaluation of male anterior urethral abnormalities.....41
Bhuiyan MA, Islam S, Das SC, Paul NK

The Termination Pattern of the Anterior Interventricular Artery in the Hearts of Bangladeshi people..... 46
Quddus MA, Sultana Z, Akhter A

Case Reports

Joubert syndrome: Magnetic resonance imaging findings..... 48
Das SC, Bhuiyan MA, Paul NK, Rahman MMA, Singha PC, Azim M, Gope S

Craniofacial fibrous dysplasia: A case report 50
Zannat NE, Bhuiyan MA

First trimester spontaneous uterine rupture in previous lower segment caesarean scar 55
Begum J and Akter S

Information for the Contributors58

Osmani Medical Teachers Association Journal

EDITORIAL BOARD

PROF. OSUL AHMED CHOWDHURY- EDITOR IN CHIEF

DR. DILIP KUMAR BHOWMIK- EDITOR.

DEPUTY EDITORS

DR. ISHTIAQUE UL-FATTAH

DR. ENAYET HOSSAIN

DR. MD. SHAMSUR RAHMAN

DR. BEGUM LUTFUN NAHAR

DR. SHANKAR KUMAR ROY

DR. MD. ABDUS SAMAD AZAD

DR. MUJIBUL HAQUE

ADVISORY BOARD

DR. JAMIL AHMED

DR. N. K. PAUL

DR. LUTFE ELAHI FAROOQUI

DR. PROBHA RANJAN DEY

The Journal does not hold itself responsible for statements made by any contributor. Statements or opinions expressed in the Journal reflect the views of the concerned author (s) and do not represent official policy of the Teachers Association, Sylhet MAG Osmani Medical College, unless so stated. Although all advertising material accepted is expected to conform to legal requirements and ethical medical standards, acceptance does not imply endorsement by the Journal.



Teachers Association, Sylhet MAG Osmani Medical College

Executive Committee

President

Dr. Jamil Ahmed

Vice Presidents

Prof. Manajjir Ali

Prof. Zakia Sultana

General Secretary

Dr. Dilip Kumar Bhowmik

Treasurer

Dr. Ishtiaque Ul-Fattah

Joint Secretary

Dr. Md. Enayet Hossain

Organizing Secretary

Dr. Shankar Kumar Roy

Members

Dr. Lutfe Elahi Faruqui

Dr. N.K Paul

Dr. Subir Kumar Das

Dr. Probhat Ranjan Dey

Dr. Begum Lufun Nahar

Dr. Md. Abdus Samad Azad

Dr. Md. Shamsur Rahman

Dr. Mojibul Haque

USE OF BOTULINUM TOXIN IN DIFFERENT NEUROLOGICAL DISORDERS

C. botulinum elaborates eight antigenically distinguishable exotoxins (A, B, C₁, C₂, D, E, F and G). Type A is the most potent toxin, followed by types B and F toxin. Types A, B and E are commonly associated with systemic botulism in humans.¹ There are two serotypes of botulinum toxin that are used for human therapeutics: type A and type B. In the commercial market in the US, there are four marketed forms of botulinum toxin: three for type A and one for type B. Each of those toxins has different properties in terms of how they are manufactured, potencies, and dilution characteristic.² Botulinum Toxin A (BoNTA) is a purified neurotoxin complex produced from the fermentation of *Clostridium botulinum* type A. BoNTA inhibits acetylcholine release into the neuromuscular junction, resulting in reduction in muscular contractions. In Canada BoNTA is marketed in three distinct formulations, Botox, Dysport, and Xeomin. Myobloc is a botulinum toxin type B preparation.³

Botulinum toxins now play a very significant role in the management of a wide variety of medical conditions, especially strabismus and focal dystonias, hemifacial spasm, and various spastic movement disorders. Besides these, encouraging clinical reports have appeared for other uses such as headaches, hypersalivation and hyperhidrosis. In this article some important indications will be discussed.

Hemifacial spasm is characterized by unilateral involuntary contractions of muscles innervated by the facial. The usual cause is a vessel touching the facial nerve near its origin from the brain stem. Although it is a benign condition it can cause significant cosmetic and functional disability. It is a chronic disease and spontaneous recovery is very rare. The two treatments routinely available are microvascular decompression and Botulinum Toxin type A (BtA) muscular injections.⁴

Cervical dystonia is the most common form of focal dystonia. It is characterized by involuntary posturing of the head and frequently is associated with neck pain. Most cases are idiopathic, and generally it is a

life-long disorder. In recent years, botulinum toxin

type A (BtA) has become first line therapy for cervical dystonia. However, not all patients respond well to BtA, and 5 to 10% become resistant to it. Botulinum toxin B (BtB) is an alternative to BtA and offers the potential to help patients who do not respond to BtA. Adverse effects include neck weakness, dysphagia, dry mouth / sore throat and voice changes / hoarseness and are dose dependent. Indirect comparisons showed in general no differences between Dysport(r) and Botox(r) in terms of benefits or adverse events.⁵ At present there is no compelling theoretical reason why it should not be as effective as, or even more effective than, BtA2 AboBoNT and rimaBoNT-B and are established as safe and effective for the treatment of CD.

OnaBoNT-A and incoBoNT-A injections should be considered, and aboBoNT-A may be considered, as treatment options for blepharospasm.⁶

For focal manifestations of adult spasticity involving the lower limb that warrant treatment, onaBoNT-A and aboBoNT-A should be offered as treatment options.⁷

There is insufficient evidence to support or refute a benefit of incoBoNT-A or rimaBoNT-B for treatment of adult lower limb spasticity. OnaBoNT-A should be considered as a treatment option before tizanidine for treating adult upper extremity spasticity. OnaBoNT-A should be offered as a treatment option to patients with chronic migraine to increase the number of headache-free days and

should be considered to reduce headache impact on health-related quality of life. OnaBoNT-A should not be offered as a treatment for episodic migraine BoNT injection is probably ineffective for treating chronic tension-type headaches.⁷

Strabismus is a condition in which the eyes are out of alignment; Strabismus may develop in childhood or

may be acquired as an adult. Treatment options include eye therapy, glasses, prisms, occlusion, botulinum toxin or surgery, to reduce the deviation of the eyes. Currently there is no clear recommendation on the use of botulinum toxin in the treatment of strabismus.⁸

Myofascial pain syndrome (MPS) is described as distinct type of regional musculoskeletal pain complaint that is caused by myofascial trigger points (TrPs) within muscles or their fascia. The trigger is identified as the presence of a taut band in the muscle, tenderness on compression in a point of the band. Treatment of MPS involves treatment of TrPs and the removal of causative / perpetuating factors. Muscle stretch, TrP injection (such as injection of botulinum toxin, or anaesthetic), BoNTA is used off-label for the treatment of MPS.⁹

The use of botulinum toxin A (BoNTA) in the treatment of lower urinary tract dysfunction has expanded in recent years and the off-licence usage list includes neurogenic detrusor overactivity (NDO), idiopathic detrusor overactivity (NDO), painful bladder syndrome (PBS), and lower urinary tract symptoms resulting from bladder outflow obstruction (BOO) or detrusor sphincterdyssynergia (DSD). There are two commonly used preparations of BoNTA: Botox (onabotulinumtoxin A) and Dysport (abobotulinumtoxin A). The two preparations should not be used interchangeably.¹⁰⁻¹¹

The use of botulinum toxins has revolutionized the treatment of various ophthalmic spastic disorders, facial dystonias and periorcular wrinkles. A precise knowledge and understanding of the functional anatomy of the mimetic muscles is absolutely necessary to correctly use botulinum toxins in clinical practice. Adverse effects are usually mild and transient.

Matiur Rahman

Professor of Neurology, Sylhet MAG Osmani Medical College

Mostafa Hosen

Asstt. Professor, Neurology, Sylhet MAG Osmani Medical College

References

1. Münchau A, Bhatia KP. Uses of botulinum toxin injection in medicine today. *BMJ* 2000;320:161-5.
2. American Academy of Neurology. Clinical Practice Guidelines Process Manual, 2004 ed. St. Paul, MN: The American Academy of Neurology; 2004. Available at: <https://www.aan.com/>
3. Brin MF, Lew MF, Adler CH, Comella CL, Factor SA, Jankovic J, et al. Safety and efficacy of NeuroBloc (botulinum toxin type B) in type A-resistant cervical dystonia. *Neurology* 1999; 53:1431-8.
4. Cochrane Database of Systematic Reviews: Plain Language Summaries [Internet]. Botulinum Toxin type A (BtA) muscular injections are beneficial in treating hemifacial spasm. 2009.
5. Cochrane Database of Systematic Reviews: Plain Language Summaries [Internet]. A comparison of botulinum toxin type A versus botulinum toxin type B for involuntary positioning of the head, or cervical dystonia. This version published: 2009; Review content assessed as up-to-date: May 04, 2003.
6. Cochrane Database of Systematic Reviews: Plain Language Summaries. Botulinum toxin Type A (BtA) is effective and safe for treating people with cervical dystonia. 2009; Review content assessed as up-to-date: October 24, 2004.
7. Cochrane Database of Systematic Reviews: Plain Language Summaries. There is high level evidence to support the safety and effectiveness of Botulinum toxin-A (BoNT-A) as an adjunct to managing the upper limb in children with cerebral palsy. 2010; Review content assessed as up-to-date: August 17, 2008.
8. Cochrane Database of Systematic Reviews: Plain Language Summaries. Botulinum toxin for the treatment of strabismus. 2012; Review content assessed as up-to-date: December 05, 2011.
9. Anonymous. Botulinum Toxin A for Myofascial Pain Syndrome: A Review of the Clinical Effectiveness. Rapid Response Report: Summary with Critical Appraisal. Ottawa (ON): Canadian Agency for Drugs and Technologies in Health; 2014 Sep 22.
10. Contemporary management of lower urinary tract disease with botulinum toxin A: a systematic review of Botox (onabotulinum toxin A) and Dysport (abobotulinum toxin A) 2011.
11. Mangera A, Andersson KE, Apostolidis A, Chapple C, Dasgupta P, Giannantoni A, et al. Contemporary management of lower urinary tract disease with botulinum toxin A: a systematic review of Botox (onabotulinumtoxinA) and Dysport (abobotulinumtoxinA). *European J Urol* 2011; 60: 784-95.



Randomized Controlled Trial of Baclofen and Diazepam in Improving Gross Motor Function in Cerebral Palsy

JUGINDRA SINGHA¹, MONIR HOSSAIN², TANJINA CHOWDHURY³, PIYA BISWAS⁴, RAMANDRA KUMAR SINGHA⁵, M EKHLASUR RAHMAN⁶, NARAYAN SHAHA⁷, SULTANA BEGUM¹.

Abstract

Oral medications are commonly used to treat spasticity in children. For generalized spasticity oral baclofen is most commonly used drug among oral antispastic medication. The antispastic efficacy and tolerability of oral baclofen have not been well established in cerebral palsy cases. Diazepam has shown to be effective and tolerable in reducing spasticity in cerebral palsy, but there are insufficient data on its effects on function and its side effects profile. Both medication have been found to be effective in the treatment of spasticity of both cerebral and spinal origin among adults, but the use of oral medication in children with cerebral palsy has not been thoroughly studied.

The objective of the study was to find out comparative efficacy of oral baclofen and oral diazepam in improving gross motor function among children with cerebral palsy.

This was a randomized controlled trial done during July 2011 to June 2012. Total 72 patients with spastic cerebral palsy patients were enrolled in the out patients department (OPD) of paediatrics, Dhaka Medical College Hospital (DMCH) and Dept. of centre for Neurodevelopment & Autism in children, Bangabandhu Sheikh Mujib Medical University (BSMMU). 36 patients received baclofen (study group) and 36 Patients received diazepam

(control group) for six months. Outcome assessment were done using

The Gross Motor function was measured by Gross Motor Functional Classification System (GMFCS). Uniform physiotherapy was given to both groups by a qualified physiotherapist. The parents were advised for follow-up at 4 weekly till 6 months to assess efficacy of drugs, to note adverse effects if any and to check drug compliance. During follow-up visit all cases were again assessed through the above 4 scales to compare the effects of studied drugs.

Demographic characteristics of both the study groups are comparable according to sex and body weight ($P=0.334$, $P=0.147$) except age ($P=0.04$). Baseline characteristics are also comparable between groups gross motor function ($p=0.743$). Diazepam took 4 months for maximum improvement of gross motor function whereas Baclofen took five months.

Diazepam has superior efficacy in improving gross motor function over baclofen ($p=0.047$). No significant different side effects between the two groups ($p>0.05$). Diazepam is less costly than baclofen ($P<0.001$).

In conclusion, both baclofen and diazepam are effective in reducing spasticity among cerebral palsy patients. Diazepam has superior efficacy in improving gross motor function. Diazepam takes less time for significant improvement of gross motor function. Diazepam is less costly than baclofen.

[OMTAJ 2015; 14(1)]

Introduction

Cerebral Palsy (CP) describes a group of permanent disorders of the development of movement and posture, causing activity limitations that are attributed to non-progressive disturbances that occurred in the developing fetal or infant brain; The motor disorders of cerebral

1. Assistant Professor Department of Paediatrics, MAG Osmani Medical College, Sylhet.
2. Postgraduate student in Paediatrics, Dhaka Medical College
3. Resident Physician (Paediatrics), MAG Osmani Medical College Hospital, Sylhet.
4. Assistant Professor, Department of Paediatrics, Park View Medical College, Sylhet.
5. Assistant Professor of Psychiatry, MAG Osmani Medical College.
6. Professor, Department of Paediatrics, Dhaka Medical College.
7. Associate Professor of Child Neurology, Institute of Neuroscience, Dhaka.

palsy are often accompanied by disturbances of sensation, perception, cognition, communication, and behavior; by epilepsy, and by secondary musculoskeletal problems.¹ In a majority of cases, the predominant motor abnormality is spasticity.² Cerebral palsy is the most common childhood disability with a prevalence of 1.5 to 3 per 1000 live births.³ Survival rates for children with cerebral palsy are reported at 65% to 90% establishing the need for effective drug therapy to manage spasticity⁴.

The diagnosis of CP is based on a clinical assessment, and not on laboratory testing or neuroimaging. It is desirable to diagnose this condition as early as possible, it is often difficult to diagnose CP in the first half of infancy except in severe cases. The changing nature of symptoms and signs make the clinical classification difficult in the first year of life as the pattern of movement and tone may change completely.⁵

Spasticity is described as a velocity-dependent increase in tonic stretch reflexes with exaggerated tendon jerks, resulting from hyper excitability of the stretch reflex, as one component of the upper motor neuron syndrome. Children diagnosed with cerebral palsy have great difficulty in performing daily activities because of their inability to adequately control movement and posture or prevent involuntary movement. Restriction of spontaneous movement in an infant with cerebral palsy, spasticity retains the limbs in contracture position.⁶ Thus reduction of hypertonia and spasm is essential to optimize physical therapy and enhance motor development, improving in the performance, participation, and satisfaction in everyday activities of these children⁷. Treating the spasticity in a muscle during the period of accelerated bone growth also permits uniform rate of growth in the muscle, preventing contracture.⁸ Currently, the original or Modified Ashworth (MAS) and Tardieu Scales (TS) appear to be the most common tools used in clinical practice to detect and quantify spasticity in children with cerebral palsy.⁹

Once a decision is made to treat spasticity, a few oral drugs are available for consideration. These include benzodiazepines, baclofen, alpha-adrenergic agonists (tizanidine, clonidine), dantrolene sodium, and Gabapentine. No medication has been universally effective in the treatment of spasticity.¹⁰ The practice parameter recommended by the American Academy of Neurology (AAN) and the Child Neurology Society

(USA) for the management of spasticity in cerebral palsy has been published recently in the year 2010. This practice parameter committee has made an evidence-based review by thorough searching of published articles from 1966 to July 2008 using the AAN's 4-tiered classification scheme for therapeutic evidence to classify articles. The result of this review is that for generalized spasticity, diazepam is probably effective in reducing spasticity but there are insufficient data on its motor function and its side effect profile. There were insufficient data on the use of dantrolene, oral baclofen, and intrathecal baclofen. Their ultimate recommendation based on evidence (A, B, C, and U) was, for generalized spasticity that warrants treatment, diazepam should be considered for short-term treatment may be considered (Level B). There are insufficient data to support or refute use of oral baclofen (Level U). But in clinical context baclofen is widely used to treat CP.¹¹

Baclofen is a GABA agonist that is used to reduce muscle tone. Baclofen crosses the blood-brain barrier and binds at the GABA β receptors of the laminae I-IV of the spinal cord, where primary sensory fibers end.¹² Among side effects, baclofen can produce sedation when administered orally (that is dose-related); this may be minimized by initiating treatment at a low dose.^{12,13} This drug may also cause impairments of cognitive functions such as confusion, memory, and can also potentiates seizures; It may also cause orthostatic hypotension, dizziness, weakness, and ataxia.^{12,13 14} Most studies on baclofen have involved patients with spinal pathology or multiple sclerosis.^{15,16} The majority of MS patients showed a reduction in spasticity and in sudden and painful spasms.¹⁶ It has been shown to be safe in long-term use and to remain effective.¹⁷ There have been very few open studies investigating the effect of baclofen in spasticity of cerebral origin.¹⁸

Diazepam is the oldest antispasticity medication still in widespread clinical use. Its antispasticity effect is caused by its inhibitory effect at both the spinal cord and supraspinal level. The effect is GABA mediated and specifically related to GABA_A receptors. Adverse effect of diazepam is sedation, impaired memory and attention, ataxia, physical dependence and weakness. The sedative effect can be overcome by giving single daily bedtime dose.¹⁹ Diazepam has been used most extensively in patients with muscle over activity of spinal cord origin, and its antispastic efficacy has been demonstrated in these conditions by double-blind randomized controlled trial with a dose dependent antispastic effect.²⁰ In

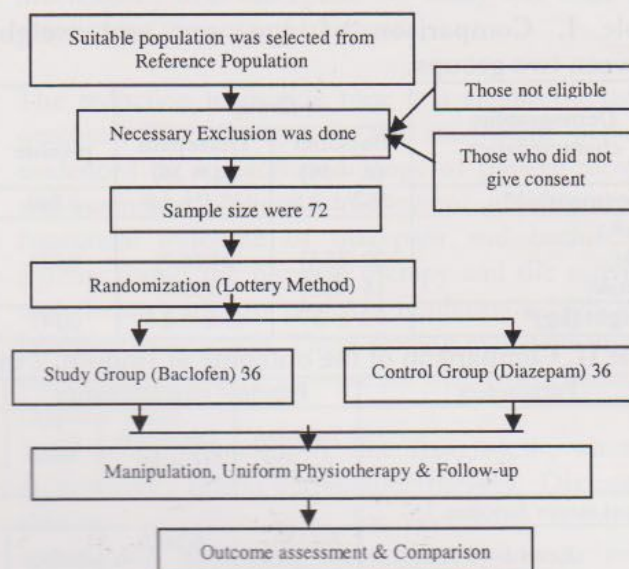
children with cerebral palsy, diazepam has also been demonstrated to have clinical efficacy for athetosis as well as against spasticity.²¹ The goal of this study is to determine the comparative efficacy of baclofen and diazepam in improving gross motor function in cerebral palsy.

Materials and Methods

This was a randomized controlled trial done during July 2011 to June 2012. This study was carried out in the Out Patient Department (OPD) of Paediatrics, Dhaka Medical College Hospital (DMCH) and Center for Neurodevelopment and Autism in Children (CNAC) of Bangabandhu Sheikh Mujib Medical University (BSMMU). All the spastic cerebral palsy patients seeking treatment in the above two centers were the reference population. After enrollment a detailed history was taken and a comprehensive clinical examination of all the reference population was done. From the reference population, spastic cerebral palsy cases fulfilling the selection criteria were enrolled as study population by systematic random sampling i.e. every second case had been enrolled. Necessary exclusion was done.

After taking written informed consent and counseling, enrolled cases were first assessed for Gross Motor Function and it was measured by Gross Motor Functional Classification System (GMFCS). level 1-3: In this scale patients walk without restrictions to limitations walking outdoors and in the community and level 4-5: Self-mobility with limitations to self-mobility severely limited even with the use of assistive technology. Baseline data was recorded in questionnaire sheet. Studied drugs were given according to dose schedule. Necessary instruction and demonstration was given to parents or care givers about the method of drug administration and necessary drugs were given till next follow-up (4 weeks later) to ensure drug compliance. Supervised physiotherapy was given uniformly to both groups by qualified physiotherapists following a specific protocol from the Department of Physical Medicine, Dhaka Medical college and BSMMU and showed and advised to mother to continue it through whole month at home. The parents were advised to come for 4 weekly follow-up till 6 months to assess efficacy of drugs, to note adverse effects if any and to check drug compliance. In each follow up patient got assessment, drugs and physiotherapy.

Flow Chart of the study:



Drug administration and titration:

After group allocation, study drugs were given according to following dose schedule.

Oral diazepam : At a dose of 1 mg given at bedtime for children under 8.5 kg body weight and 2 mg for children of 8.5 to 15 kg body weight for 6 months.²²

Diazepam for 4-week periods with intervening drug free periods of 2 weeks to delay the development of tolerance.

Oral baclofen: Baclofen was started with a very low dose (corresponding to approximately 0.3mg/kg a day) in two divided doses. The dose was increased 5mg weekly upto maximum dose.²³ Doses for maintenance therapy ranges between 0.75-2mg / kg / day two divided dose for 6 months.²⁴ After completion of the study, baclofen was tapered over one month to avoid acute drug withdrawal symptoms.

Physiotherapy:

Uniform physiotherapy was given to both groups by qualified physiotherapists following a specific protocol from the Department of Physical Medicine, Dhaka Medical college and BSMMU. One hour intervention continued daily for 5days weekly. Activities included in each session were body alignment and weight transfer in various positions, bimanual activities and facilitation sequences of movements. The treatment highlighted functional, meaningful task, in contexts appropriate for each child. Guiding the parents in their handling skills was integrated into the intervention. Parents were shown how to handle their children when dressing and undressing, and how to facilitate variation of movement pattern during play.²⁵

Results

Table I. Comparison of age, sex and weight between two groups.

Demographic characteristics	Group		p-value
	Baclofen (n = 36)	Diazepam (n = 36)	
Age (months)#	25.3 ± 2.8	37.4 ± 4.9	0.040
Sex*			
Male	20 (55.5)	24 (66.7)	0.334
Female	16 (44.5)	12 (33.3)	
Weight (kg)#	9.3 ± 2.7	13.0 ± 2.5	0.147

Demographic characteristics:

Table I demonstrates that the patients of Baclofen group were relatively younger compared to Diazepam group ($p = 0.040$). Males were predominant in both groups with no significant intergroup difference ($p = 0.334$). The mean weight was considerably higher in Diazepam group compared to that in Baclofen group ($p = 0.147$).

Table II. Comparison of the outcome at 1 month, 2 months, 3 months, 4 months, 5 months and 6 months.

Comparison of the outcome at month, 2 months, 3 months, 4 months, 5 months, 6 months, 5 months and 6 months.											
Parameters		Baseline characteristics			After 1 month			After 2 months			
		Baclofen (n=36)	Diazepam (n=36)	P- value	Baclofen (n=36)	Diazepam (n=36)	P- value	Baclofen (n=36)	Diazepam (n=36)	P- value	
Gross motor function											
Level 1—3		5(13.9)	6(16.7)	0.743	6(16.7)	8(22.2)	0.551	8(22.2)	14(38.9)	0.125	
Level 4-5		31(86.1)	30(83.3)		30(83.3)	28(77.8)		28(77.8)	22(61.1)		
After 3 months			After 4 months			After 5 months			After 6 months		
Baclofen (n=36)	Diazepam (n=36)	P-value	Baclofen (n=36)	Diazepam (n=36)	P-value	Baclofen (n=36)	Diazepam (n=36)	P-value	Baclofen (n=36)	Diazepam (n=36)	P-value
13(36.1)	19(52.8)	0.155	15(41.7)	25(69.4)	0.047	17(47.2)	25(69.4)	0.018	17(74.2)	25(69.4)	0.018
23(63.9)	17(47.2)		21(58.3)	11(30.6)		19(52.8)	11(30.6)		19(52.8)	11(30.6)	

Baseline characteristics:

Table II shows that level of gross motor function was also identically distributed between Baclofen and Diazepam group with most of the children exhibiting level 4-5 ($p=0.551$).

Outcome of children at month 1:

Outcome of children at month 1 shows that both groups Level of gross motor function did not respond well in either group ($p = 0.551$).

Outcome of children at month 2:

Gross motor function was considerably better in diazepam group compared to the baclofen group though the difference was not statistically significant ($p = 0.125$).

Outcome of children at month 3:

Evaluation of outcome at month 3 showed that the gross motor function was slow ($p = 0.155$).

Outcome of children at month 4:

At 4 months of intervention gross motor function maintaining significant difference from their baclofen counterparts ($p=0.047$)

Outcome of children at month 5:

No further change was noted in diazepam in terms of outcome variables. But baclofen group improved further. However, the differences between the groups were still significant ($p = p = 0.018$).

Outcome of children at month 6:

Gross motor function exhibited no further change ($p = 0.047$).

Table III. Comparison of side effects between two groups

Side effects	Group		p-value
	Baclofen (n = 36)	Diazepam (n = 36)	
Lethargy*			0.766
Yes	7(19.4)	8(22.2)	
No	29 (80.6)	28(77.8)	
Drowsiness*			0.466
Yes	5(13.9)	7(19.4)	
No	31(86.1)	29(80.6)	
Sedation*			0.781
Yes	9(25.0)	10(27.8)	
No	27(75.0)	26(72.2)	

Side effects:

The side-effects of the drugs reported by children's are illustrated in table IX. Commonly complained

side-effects were lethargy, drowsiness and sedation which were almost identically distributed between groups ($p > 0.05$).

Discussion

In this study, both the patients between the group were comparable according to sex and weight except age. Baseline characteristics are comparable. The patients of baclofen group were relatively younger compared to diazepam group. The mean age of the patients of baclofen group was of 25.3 ± 2.8 months whereas Adam and colleagues⁵² found mean age of 7.4 ± 2.3 years. The difference between this study and other study is that patient not coming to physician after 5 years due to socioeconomic condition & false belief.

Both Baclofen and Diazepam group experienced no significant change in gross motor function

upto 3 month. A substantial proportion of the children in both groups with level 4 – 5 gross motor function at baseline changed to level 1–3 at 6 months of evaluation. The difference between the two groups in terms of change in level of gross motor function was statistically significant.

Wagstaff and Bryson²⁶ found improvement in muscle tone occurred in 60 to 86% of diazepam recipients compared with 50 to 56% of baclofen.

Anna Mathew and Colleagues²² study randomized controlled trial in children with spastic CP weighing less than 15 kg to receive 1 of 2 doses of diazepam (0.5–1 mg vs 1–2 mg) or placebo at bedtime. Improvements 3 weeks after treatment included a dose-dependent reduction of tone as measured by the MAS, increased passive range-of-motion angles, and an increase in spontaneous movements, no functional outcome measures were reported.

Milla and Jackson¹⁸, a double blind crossover study in 20 patients, receiving at a dose of 10 to 60 mg/day, found a reduction of spasticity by means of the Asworth scale. In our study, patients are receiving at a of dose 0.75 to 2mg/kg/day in two to three divided doses.

Adam and colleagues²⁷ study double blind cross over oral baclofen with cerebral palsy using Modified Tardieu Scale, Goal Attainment Scale, and Pediatric Evaluation of Disability inventory which shows significant different between baclofen and placebo.

In adverse effect profile both diazepam and baclofen group show lethargy drowsiness and sedation. The adverse effects were almost identically distributed between groups. Regarding treatment cost, baclofen is

much costly than diazepam. No study was done before mentioning the cost of treatment.

The reduction in muscle tone is a significant technical outcome. The changes in the movement pattern, as evidenced by an increased range of passive movement and augmented voluntary movement, indicate a positive functional outcome of diazepam and baclofen. The parents found the physical therapy and the activities of daily living easier to perform rendering a high level of caregiver satisfaction. These finding may have important clinical implication for physician who treat children with cerebral palsy.

In a developing country like Bangladesh, where cost factors play a role in determining therapy, Diazepam is a safe and effective agent for decreasing stiffness and spasm and improving movement prior to initiating physical therapy in a young child with cerebral palsy. So diazepam should be used for the treatment of gross motor function in children with cerebral palsy. For further authentication of the research, multicentre larger sample size study should be done.

It is concluded from the study that both baclofen and diazepam were effective in reducing spasticity among cerebral palsy patients. Diazepam had superior efficacy in improving gross motor function. Moreover, diazepam is less costly than baclofen.

Recommendation

Diazepam may be considered in the management of gross motor function in patients with cerebral palsy. However, multicentre study including larger sample size should be carried out for validation.

Limitations:

1. Exclusion of a few conditions like leukodystrophy, galactosemia, epilepsy by clinical criteria only.
2. Double blinding could not be done.
3. Small sample size.

References

1. Rosenbaum P, Paneth N, Leviton A, Goldstein M, Bax M, Damiano D, et al. A report: the definition and classification of cerebral palsy. *Dev Med Child Neurol* 2007;109:8-14.
2. Thomas MO. Diagnosis, treatment and prevention of cerebral palsy. *Clin obst and gynaecol* 2008; 51: 816–28.

3. Surveillance of Cerebral Palsy in Europe (SCPE). Surveillance of cerebral palsy in Europe: a collaboration of cerebral palsy surveys and registers. *Dev Med Child Neurol*. 2000;42: 816-24.
4. Rapp CE, Torres MM. The adult with cerebral palsy. *Arch Fam Med* 2000; 9: 466-72.
5. Aneja S. Evaluation of a child with cerebral palsy. *Indian J Pediatr* 2004;71:627-8.
6. Hall DMB. Cerebral palsy In: Hall D.M.B. *The Child with a Handicap*. Blackwell Scientific Publications, Oxford. 1984; 303-55 Tal KC, Tal J, Aviva FV. Upper extremity function and occupational performance in children with spastic cerebral palsy following lower extremity Botulinum Toxin Injections. *J Child Neurol* 2010;25 :694-700.
7. Stanley F, Blair E, Alberman E. Epidemiological issues in evaluating the management of cerebral palsy. In *Cerebral Palsies: Epidemiology and Causal Pathways*. Clinics in Developmental Medicine. MacKeith Press, 2000; 151: 183-92.
8. Young RR, Emre M, Nance PW. Current issues in spasticity management. *Neurologist* 1997;3:261-75.
9. Mutlu A, Livanelioglu A, Gunel MK. Reliability of Ashworth and Modified Ashworth scales in children with spastic cerebral palsy. *BMC Musculoskelet Disord* 2008; 9:44
10. Katz RT. Management of spastic hypertonia after stroke. *J Neurol Rehabil* 1991;5 (suppl1):5-12.
11. Delgado MR, Hirtz D, Aisen M, Ashwal S, Fehlings DL, McLaughlin J. et al. Practice Parameter: Pharmacologic treatment of spasticity in children and adolescents with cerebral palsy. *Neurology* 2010;74 :336-43.
12. Young RR, Delwaide PJ. Drug therapy: Spasticity (second of two parts). *N Engl J Med* 1981;304:96-9.
13. Terrence CF, Fromm GH. Complications of baclofen withdrawal. *Arch Neurol* 1981;38:588-9.
14. Sawa GM, Paty DW. The use of baclofen in treatment of spasticity in multiple sclerosis. *Can. J Neurol Sci* 1979; 6:351-4.
15. McLellan DL. Co-contraction and stretch reflexes in spasticity during treatment with baclofen. *J Neurol Neurosurg Psychiatry* 1977; 40:30-8.
16. Feldman RG, Kelly-Hayes M, Conomy JP, Foley JM. Baclofen for spasticity in multiple sclerosis: double-blind crossover and three-year study. *Neurology* 1978; 28:1094-8.
17. Van Hemet JCJ. A double-blind comparison of baclofen and placebo in patients with spasticity of cerebral origin, in Feldman RG, Young RR, Koella (eds): *Spasticity: Disordered Motor Control*. Chicago, Year Book Medical Publishers, 1980.
18. Milla PJ, Jackson AD. A controlled trial of baclofen in children with cerebral palsy. *J Int Med Res* 1977;5:398-40
19. Patel DR, Soyode O. Pharmacologic interventions for reducing spasticity in children with cerebral palsy. *Indian J Pediatr*. 2005; 72 : 869-72
20. Corbett M, Frankel HL, Michaelis L. A double blind cross-over trial of valium in the treatment of spasticity. *Paraplegia* 1972; 10:19-22.
21. Wilson LA, McKechnie AA. Oral diazepam in the treatment of spasticity in paraplegia: a double blind trial and subsequent impressions. *Scott Med J* 1966; 11:46-51.
22. Mathew A, Mathew MC, Thomas M (late), and Antonisamy B. The Efficacy of Diazepam in Enhancing Motor Function in Children with Spastic Cerebral Palsy. *J Tropic Pediatr* 2005; 51:105-10
23. Gracies JM, Nance P, Elovic E, McGuire J, Simpson DM. Traditional pharmacological treatments for spasticity Part II: General and regional treatments. *Muscle Nerve* 1997; 6 (Suppl):S92-S120.
24. Rinaldis MD, Losito L, Gennaro L and Trabacca A. Long-term Oral Baclofen Treatment in a Child with cerebral Palsy: Electroencephalographic Change and Clinical Adverse Effects. *J Child Neurol* 2010; 25: 1272-4
25. Tordis U, stad, PT, Anne B, Soradh PT, and Ljunggren PT. Effects of Intensive Physiotherapy Newly Diagnosed with Cerebral Palsy. *Pediatr Phys Ther* 2009;21:140-1.
26. Wagstaff AJ, Bryson HM. Tizanidine a review of its pharmacology, clinical efficacy and tolerability in the management of associated with cerebral and spinal disorders. *Drugs* 1997;53;435-52.
27. Adam S, Kate H, Lawrence TL, Stephen OF. Oral baclofen in children with cerebral palsy: A double blind cross over pilot study. *J Pediatric Child Health* 2006;42;715-20.

Correlation of Preoperative Serum Level of CA 125 with Different Histopathological Types of Ovarian Carcinoma

Md Sabbir Hossain¹, Murshed Ahmed Chowdhury², Md Amjad Hossain Khan³, Md Abed Hossain⁴, MAS Ansari⁵, Dilip Kumar Bhowmik⁶, Shajia Chowdhury⁷.

Abstract

The present study was carried out with an aim to evaluate the correlation of preoperative serum carcinoembryonic antigen 125 (CA 125) level with different histopathological types of ovarian carcinoma. A comparative cross sectional study was done during the period of January 2010 to December 2010 in the Department of Pathology, Sylhet MAG Osmani Medical College (SOMC). Total 40 patients were selected from the inpatient department of Obstetrics and Gynaecology in SOMCH & Jalalabad Ragib-Rabeya Medical College Hospital on the basis of inclusion and exclusion criteria. Post surgical specimens were collected from the study subjects. Among total 40 cases of ovarian carcinoma, 30 (75%) cases were histologically diagnosed as serous cyst adenocarcinoma, 8 (20%) cases were mucinous cyst adenocarcinoma, 1 (2.5%) case was clear cell carcinoma and 1 (2.5%) case was malignant Brenner tumor. The mean value of preoperative CA 125 was 137.31 U/ml in serous cyst adenocarcinoma, 123.57 U/ml in mucinous cyst adenocarcinoma, 39 U/ml in clear cell carcinoma and 92.5 U/ml in malignant Brenner tumor. The mean value of preoperative CA 125 was highest in serous cyst adenocarcinoma. Phi (ϕ) coefficient correlation test (r_ϕ) was done for the CA 125 mean

levels and for the CA 125 positive cases with different histopathological types of ovarian carcinoma to see their correlation. The test was found significant in serous cyst adenocarcinoma where p value was <0.05 and the test was found insignificant in other types of ovarian carcinoma where p value was >0.05 . The preoperative assessment of serum CA 125 levels can predict the different types of ovarian carcinoma before operation, and the clinician can make a plan of treatment preoperatively.

[OMTAJ 2015; 14(1)]

Introduction

Ovarian carcinomas comprise a diverse group of neoplasms, exhibiting a wide range of morphological characteristics, clinical manifestations, genetic alterations and tumor behavior than any other organ in the body. Ovarian carcinoma accounts for the greatest number of deaths from malignancies of female genital tract and is the 5th leading cause of cancer fatalities in women¹. According to the annual report in 2005 of National Institute of Cancer Research and Hospital, Dhaka, the prevalence of ovarian cancers in Bangladesh were 3.8%.² WHO (World Health Organization) histological classification separates ovarian neoplasms according to the most probable tissue of origin. It is now believed that tumors of the ovary arise ultimately from one of three ovarian components: 1) surface epithelium, 2) the germ cells or 3) the stroma of the ovary. Of these different types, cancers of epithelial origin are the most common, comprising 90% of all ovarian malignancy. They are derived from the epithelium of the celom that normally covers the ovarian surface. This surface lining is multipotential and can differentiate into several types of epithelium, which explains why there is a wide variety of epithelial carcinomas in the ovary. Five major subtypes are included within the surface epithelial-

1. Assistant Professor, Department of Pathology, North East Medical College, Sylhet.
2. Professor, Department of Obstetrics and Gynecology, Sylhet MAG Osmani Medical College, Sylhet.
3. Professor, Department of Pathology, Sylhet MAG Osmani Medical College, Sylhet.
4. Professor, Department of Pathology, Jalalabad Ragib-Rabeya Medical College, Sylhet.
5. Associate Professor, Department of Pathology, Sylhet Women's Medical College, Sylhet.
6. Associate Professor, Department of Obstetrics and Gynecology, Sylhet MAG Osmani Medical College, Sylhet.
7. Associate Professor, Department of Pathology, Jalalabad Ragib-Rabeya Medical College, Sylhet.

stromal group. They are designated as follows: serous, mucinous, endometrioid, clear cell, and transitional cell (Brenner) type³.

With the blessings of modern sciences, we use tumor markers for the correlation of different types of ovarian carcinoma. At present serum CA 125 is the gold standard tumour marker for this disease⁴. Hence, types of ovarian carcinoma assume prime importance for patients who may receive chemotherapy or neoadjuvant therapy prior to resection of the tumor and in those who present with metastases. This study was done with an aim to evaluate the correlation of preoperative serum carcinoembryonic antigen 125 (CA 125) level with different histopathological types of ovarian carcinoma.

Material and Methods

This was a cross sectional comparative study carried out in the departments of Obstetrics and Gynaecology of SOMCH and JRRMCH during January to December 2010. Patients were enrolled on the basis of history, physical examination, inclusion and exclusion criteria that, admitted all women of ovarian carcinoma diagnosed by history, presentation, physical findings, and laboratory investigations and confirmed by histopathology. Exclusion criteria was the patient unfit for major surgical operation, inoperable ovarian tumor, ovarian tumor with pregnancy, patient not willing to participate in the study, patients with endometriosis, patients with other malignancies and menstruating women. In this study the clinical history of the patients were recorded in the predesigned questionnaire. The patients were examined thoroughly. All the findings, past history and investigations findings were recorded properly. Post surgical specimen was collected from the patient. The specimens were examined in the Department of Pathology, Sylhet MAG Osmani Medical College, Sylhet under proper daylight. Tumor mass was examined with particular emphasis on size, shape, number, consistency. On cut section, cystic fluids and solidity were examined meticulously. Three representative tissue blocks of 3-5 mm thickness were taken from the specimens. For microscopic examination routine paraffin sections were stained with haematoxylin and eosins staining method.

The estimation of serum CA 125 was done by ELISA method in the immunological laboratory by Immulite 1000, Sysmex, USA in the department of Microbiology, Sylhet MAG Osmani Medical College, Sylhet. CA 125 reagent used was manufactured by Jhonson, USA. Cut off value of CA 125 was 35 U/ml. The preoperative

serum was preserved in micro-centrifuged tube at -20°C for analysis. All data were analyzed by SPSS version 17. Quantitative data was analyzed by mean and standard deviation (SD). Qualitative data was summarized by ratio and percentage. Correlation between different types of ovarian carcinoma and levels of serum CA 125 were done by Phi (ϕ) coefficient correlation test (r_p). P value <0.05 was considered as significant.

Results

The age range of 40 patients was 21 to 80 years. Mean (\pm SD) age of the patients was 52.22 (\pm 11.49) years. The patients were divided into 6 age groups considering each decade as a single group. Maximum number of the patient 19 (47.5%) belonged to the age group of 51 to 60 years and the next 9 (22.5%) belonged to the age group of 41-50 years. So the incidence rate was higher among older age. Lower in early age because, the patient did not attend the physician early, also in extreme late age may be due to death of the patients associated with disease progression (Table-I).

Table-I: Distribution of patients according to their age group (n=40).

Age	No of Patients	Percentage
21-30 years	3	7.5
31-40 years	3	7.5
41-50 years	9	22.5
51-60 years	19	47.5
61-70 years	5	12.5
71-80 years	1	2.5
Total	40	100

Among the total 40 cases of ovarian carcinoma, 30 (75%) cases were histologically diagnosed as serous cyst adenocarcinoma, 8 (20%) as mucinous cyst adenocarcinoma, 1 (2.5%) as clear cell carcinoma and 1 (2.5%) as malignant Brenner tumor.

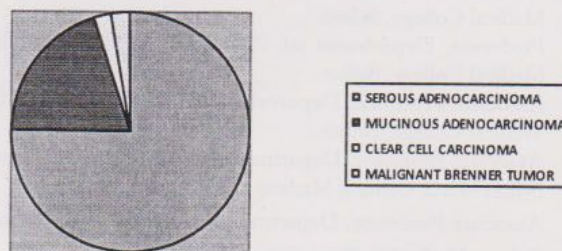
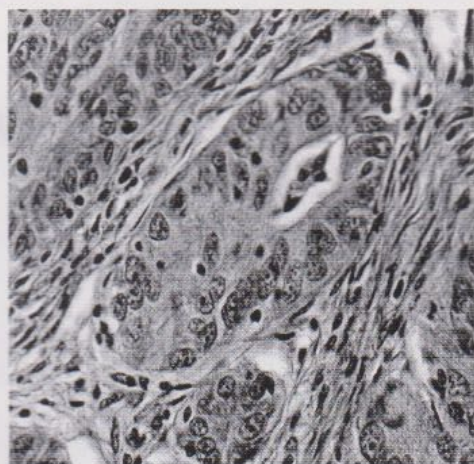
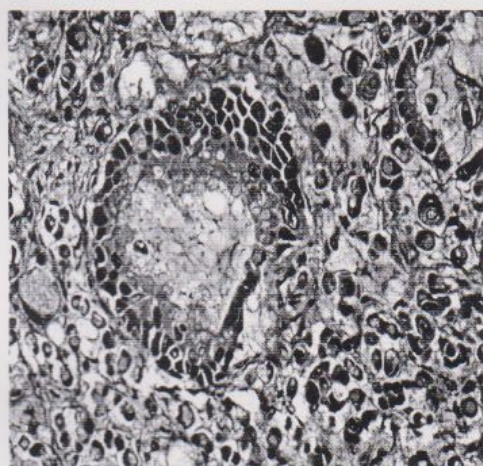


Figure-1: Histopathological types of ovarian carcinoma.**Figure-2:** Photomicrograph of serous cyst adenocarcinoma.**Figure-3:** Photomicrograph of mucinous cyst adenocarcinoma.

The mean value of preoperative CA 125 was 137.31 U/ml in serous cyst adenocarcinoma, 123.57 U/ml in mucinous cyst adenocarcinoma, 39 U/ml in clear cell carcinoma and 92.5 U/ml in malignant Brenner tumor. The mean value of preoperative CA 125 was highest in serous cyst adenocarcinoma (Table-II).

Table-II: CA 125 in different histopathological types (n=40).

Histopathological Diagnosis	Mean	Frequency	P Value
Serous cyst adenocarcinoma	137.31	30	0.013
Mucinous cyst adenocarcinoma	123.57	8	0.82
Clear cell carcinoma	39	1	0.82
Malignant Brenner tumor	92.5	1	0.82
Total	115.5	40	

Phi (ϕ) coefficient correlation test (r_ϕ) was done for the CA 125 mean levels and for the CA 125 positive cases with ovarian carcinoma to see their correlation. The p value was 0.013 in case of serous cyst adenocarcinoma which is <0.05 and 0.82 in other types of ovarian carcinoma which is >0.05 . The test was found significant in serous cyst adenocarcinoma and insignificant in other types of ovarian carcinoma.

Discussion

Cancer is a worse process, in maximum cases the ultimate fate is death. Almost the entire cancers end by death and a very few is left with permanent disability in adults. Approximately 90% of ovarian cancers are derived from the epithelium of the celom that normally covers the ovarian surface.

A variety of tumour markers have been tried to assist the diagnosis of epithelial ovarian carcinoma. At present serum CA 125 is the gold standard tumour marker for this disease. It helps in early detection, staging and assessment of adequacy of surgery and effectiveness of chemotherapy. It facilitates follow up and predicts subclinical recurrence, progression, prognosis and survival. Preoperative serum CA 125 levels have large bearing on prognosis because its level correlate well with body burden of tumor.

The present study was designed with the aim to evaluate the correlation of preoperative serum level of CA 125 with different types of ovarian carcinoma. Among the total 40 cases of ovarian carcinoma, 30 (75%) cases were histologically diagnosed as serous cyst adenocarcinoma, eight (20%) as mucinous cyst adenocarcinoma, one (2.5%) as clear cell carcinoma and one (2.5%) as malignant Brenner tumor. The highest was serous cyst adenocarcinoma which is similar to other studies.^{5,6,7}

In our study, the mean value of preoperative CA 125 was 137.31 U/ml in serous cyst adenocarcinoma, 123.57 U/ml in mucinous cyst adenocarcinoma, 39 U/ml in clear cell carcinoma and 92.5 in malignant Brenner tumor. The mean value of preoperative CA 125 was highest in serous cyst adenocarcinoma. These observations are consistent with the studies done by others⁸.

In conclusion, from present study it can be concluded that, the preoperative serum CA 125 levels correlate with different types of ovarian carcinoma but significantly increase with serous cyst adenocarcinoma. The preoperative assessment of serum CA 125 levels can predict the serous cyst adenocarcinoma before operation. The clinician can make a better treatment plan for the patient preoperatively.

References

1. Edyta C and Pirog. The female genital tract. In: Kumar V, Abbas AK, Fausto N and Astler JC eds. Robbins and Cotran pathologic basis of disease, 8th edition. Philadelphia, Elsevier. 2010; pp. 1039-52.
2. Department of Cancer Epidemiology, NICRH. Annual Report 2005, Dhaka. National Institute of Cancer Research & Hospital, 2005.
3. Crum C P. The female genital tract. In: Kumar V, Abbas AK, Fausto N and Astler JC eds. Robbins and Cotran Pathologic basis of disease, 7th edition Philadelphia, WB Saunders. 2004; pp. 1092-1104.
4. Gupta D, Lammersfeld CA, Vashi PG, Braun DP. Longitudinal monitoring of CA 125 levels provides additional information about survival in ovarian cancer. *J Ovarian Res* 2010; 3:1-8.
5. Bouanene H, Harrabi I, Ferchichi S, Limem HB, Miled A. Factors predictive of elevated serum CA 125 levels in patients with epithelial ovarian cancer. *Bull Cancer* 2007; 94: 18-22.
6. Thakur V, Anand AK, Mukherjee U, Ghosh D. Determination of cancer antigen 125 in ovarian carcinoma. *Indian J Clin Bio* 2003; 18: 27-33.
7. Fures R, Bukovic D, Hodek B, Klaric B, Herman R, Grubisic G. Preoperative tumor marker CA 125 levels in relation to epithelial ovarian cancer stage. *Coll Antro* 1999; 23: 189-94.
8. Tamakoshi K, Kikkawa F, Shibata K, Tonoda K, Obata NK, Wakahara F et al. Clinical value of CA 125, CA 19-9, CEA, CA 72-4 and TPA in borderline ovarian tumor. *Gyne Oncol* 1996; 62: 67-72.

Comparative study of caudal anaesthesia - analgesia with general anaesthesia - opioid analgesia for lower abdominal surgery in paediatric patients

Gopikesh Ranjan Dey¹, Parimol Kishore Dev¹, Syed Miftahuddin², Piya Biswas³

Abstract

Caudal anaesthesia in paediatric population is popular in the world for lower abdominal surgery. But in our country still general anaesthesia is used widely. This prospective randomized comparative study was carried out to compare the safety, cost-effectiveness and better postoperative pain control in caudal anaesthesia-analgesia than general anaesthesia-opioid analgesia in lower abdominal surgery in paediatric patients. Sixty male paediatric patients of age between 1-7 years, ASA physical status¹ scheduled to undergo lower abdominal surgery were included in this study. Patients were randomly allocated into two groups, each of thirty. In group I patients received caudal anaesthesia-analgesia were devoid of some adverse effects of general anaesthesia-analgesia. In group II patients received general anaesthesia-analgesia, 3(10%) patients developed difficulty in intubation; 2 (6.66%) patients developed laryngospasm and 3 (10%) developed delayed recovery. FLACC analysis (behavior pain scoring in children) were done to compare the postoperative pain control between two groups. In group I it was 1.30 and in group II it was 2.69, $p < 0.0001$ (significant). So group I had got better pain control. In case of cost, group II received general anaesthesia-analgesia was 13 times more costly than group I. Lastly it can be concluded that caudal anaesthesia - analgesia avoided hazards of general anaesthesia-opioid analgesia, cheap and safe.

[OMTAJ 2015; 14(1)]

Introduction

Lower abdominal surgery below the umbilicus such as Inguinal hernia(herniotomy), undescended testes (Orchipexy), scrotal surgery such as hydrocele(Repair of hydrocele) and penile surgery as hypospadias, epispadias (Repair and reconstruction) etc, are commonly done with general anaesthesia in our country. But it can be done with caudal anaesthesia with a dose of ketamine before to calm the patient. Single injection caudal block is one of the most popular and versatile paediatric regional anaesthetic techniques. It provides excellent perioperative analgesia. Caudal anaesthesia may be performed with the patient in the prone position, but the lateral position is usually more acceptable to the patient and easier in the anaesthetized paediatric patient¹.

Identification of the midline and performance of the block are less complicated in the paediatric patient making the lateral position clinically practical.² The word anaesthesia means absence of all sensation. General anaesthesia causes reversible loss of consciousness with suppression of reflexes. Surgical anaesthesia should be a state of harmless and reversible insensibility which allows operations of considerable magnitude to be carried out without hindrance to the surgeon or detriment to the patient³.

The aim of the study was to find out a safe, cost-effective technique with good postoperative pain control in lower abdominal surgery comparing with general anaesthesia in paediatric patients.

Materials and Methods

It was a randomized prospective case control clinical study. For this study, 60 male paediatric patients of ASA physical status I, aged 1-7 years, were selected and randomly divided into two groups. Each group consisted of 30 patients.

1. Assistant Professor, Department of Anaesthesiology, Jalalabad Ragib-Rabeya Medical College, Sylhet.
2. Assistant Professor, Department of Anaesthesiology, Sylhet MAGOsamni Medical College
3. Assistant Professor, Department of Paediatrics, Parkview Medical College

Exclusion criteria:

1. Acute pulmonary infection or disease.
2. Congenital heart disease.
3. Fasting less than 4 hours.
4. Poorly controlled seizures.
5. Central nervous system abnormalities such as hydrocephalous.

The study was performed in the department of anesthesiology and intensive care unit of North East Medical College hospital in the period from July 2008 to December 2009 and Jalalabad Ragib Rabeya Medical College hospital from August 2013 to December 2013.

Grouping of the subjects:

Group I: Consisted of 30 patients who received caudal anaesthesia and analgesia with ketamine.

Group II: Consisted of 30 patients received who general anaesthesia and opioid analgesia. Drugs used were: Ketamine, Bupivacaine 0.25%, Bupivacaine 0.125%, Diazepam, Atropine, fluid 5% Dextrose with 0.45% Sodium Chloride for group I patients. Inj fentanyl, thiopentone sodium, atropine, suxamethonium, neostigmine, vecuronium for group II patients. In group I cases, first of all an intravenous cannula of size 24 were given and 5% dextrose in 0.45% normal saline were started and were given 10ml/kg in running drops and the patients were placed in lateral position decubitus position. Ketamine 1mg/kg with diazepam 0.2 mg/kg, atropine 15µg/kg body weight were given very slowly intravenously to sedate the patient and make the patient calm and pain free. Antiseptic wash were given with povidone iodine and then with spirit over a wide area over the back of sacrum and thumb and middle finger were placed in the two posterior superior iliac spine, the index finger of same hand then palpates spinous process of sacral 4 vertebra, a 21 gauge needle were inserted first at 90 degree then 45 degree and finally at 30 degree through the sacral hiatus, until piercing the sacrococcygeal ligament which is continuous with the ligamentum flavum, correct placement of the needle were confirmed by the feel of the needle passing the ligament and the ease of injection. The absence of cerebrospinal fluid or blood were confirmed before giving drugs. Oxygen was given to every patient by nasal prong at 2L/min. 0.25% Bupivacaine was given in a dose of 1 ml/kg body weight. Caudal block can be done easily in paediatric patients because of better anatomic relationships and thus easier orientation and shorter time required for puncture. Perforation of sacrococcygeal ligament is more easily palpable and better distribution of injected anaesthetic than in adult.⁴ Surgical relaxation

was adequate. At the end of surgery, again caudal block were given in every patient with 0.125% Bupivacaine, half of the volume of the previous dose- which were used for surgery, for postoperative analgesia. Catheter technique for caudal analgesia became unpopular because of risk of infection.⁵ Postoperatively, analgesia was maintained mainly with the caudal given at the end of surgery for 1st postoperative day but five patients required one rescue dose for each of diclofenac sodium suppository 1mg/kg per rectally. They were discharged on 2nd postoperative day with advice of diclofenac sodium suppositories 1mg/kg twice daily for 4 days. In group II patients of general anaesthesia, pre-oxygenation were done with 100% oxygen, atropine 15µg/kg and fentanyl 1 µg/kg iv used as premedication, then induction were done with thiopentone 4 mg/kg iv, intubation with suxamethonium 1mg/kg, then muscular relaxation done with vecuronium 0.08 mg/kg and nitrous oxide, halothane were used for maintenance of anaesthesia along with oxygen. Reverse were done with neostigmine and atropine.

Postoperative analgesia were given with pethidine 1.5mg/kg body weight 8 hourly for the first day then diclofenac suppositories as 1mg/kg twice daily for 4 days. They were discharged on 5th postoperative day.

Monitoring: Pulse rate, blood pressure and saturation of oxygen which was monitored by pulse oximeter

Results

Observation of both groups was carried out and results evaluated for comparison.

Table-I: Demographic data of study subjects (n = 60)

Variables	Group I (n = 30) Average	Group II (n = 30) Average	P value
Age (years)	3.97	4.2	<0.91
Weight (kg)	15.13	15.2	<0.86
Duration of surgery(min)	53.77	56.97	<0.29
Baseline pulse rate (beats/min)	86.7	109.7	<0.0001
Systolic blood pressure(mm/Hg)	97.10	104.97	<0.0001
Diastolic blood pressure(mm/Hg)	58.87	73.03	<0.0001

The groups became statistically matched for age (P < 0.91), weight (P < 0.86), duration of surgery (P < 0.29),

and baseline pulse rate ($P < 0.0001$), Systolic blood pressure ($P < 0.0001$), Diastolic blood pressure ($P < 0.0001$)

Table-II: Comparison of hazards during anaesthetic procedure

	Group-I	Group-II	
1. Difficulty during intubation	0	3	10%
2. Laryngospasm	0	2	6.66%
3. Delayed recovery	0	3	10%

Table-III: Cost analysis between two groups (In taka)

	Group -I	Group-II	P value
Mean	91.40	1264.50	< 0.0001
Standard deviation	17.64	8.80	Statistically significant

FLACC⁶ pain scale was used to compare the postoperative pain in both groups.

Behaviour	0	1	2
Face	No particular expression or smile	Occasional grimace, frown, withdrawn, disinterested	Frequent to constant quivering chin, clenched jaw
Leg	Normal position or relaxed	Uneasy, restless, tense	Kicking or legs drawn up
Activity	Lying quietly, normal position, moves easily	Squirming, shifting back and forth, tense	Arched, rigid or jerking
Cry	No cry, (awake or asleep)	Moans or whimpers, occasional complaint	Crying steadily, screams, sobs, frequent complaints
Consolability	Content, relaxed	Reassured by occasional touching, being talked to, distractable	Difficult to console or comfort

Table-IV: Behaviour pain scoring in postoperative period between two groups (FLACC Scale)

	Group-I	Group-II	P value
Mean	1.30	2.69	< 0.0001
Standard deviation	0.65	1.04	Significant

Discussion

Lower abdominal surgery in infra-umbilical region in paediatric patients for hydrocele, undescended testes,

hypospadias, & epispadias can be done by both caudal and general anaesthesia.

In our study the group I (caudal group), had good haemodynamic control than that of group II (general anaesthesia). In group I, the mean systolic blood pressure, mean diastolic blood pressure and mean pulse rate were 97.10 mm of Hg, 58.87 mm of Hg and 86.7 beats/min respectively and whereas in group II it were 104.97 mm of Hg, 73.03 mm of Hg and 109.70 beats/min respectively.

Halothane used during general anaesthesia causes vasodilatation and decrease blood pressure but general anaesthesia cannot attenuate catecholamine, cortisol, growth hormone, glucagon and anti-diuretic hormone secretion like caudal, so ultimately causes tachycardia and increase blood pressure. Uguralp et al⁷ demonstrated better hemodynamic stability with caudal anaesthesia which relates to our study. But Kita et al⁸ compared caudal epidural anesthesia with general anesthesia in urological surgery in children and adolescent and found no significant differences and no hemodynamic and neurological sequelae.

Schrock et al and Gunter et al⁹ found that 0.175% Bupivacaine could produce adequate surgical anesthesia. In our study, we used 0.25% Bupivacaine that was good for surgical relaxation in caudal anaesthesia.

Patients received Caudal anaesthesia in group I was more safe than patients of group II of general anaesthesia because in that group 3(10%) patients developed difficulty in intubation, 2(6.66%) patients suffered from laryngospasm, and 3(10%) patients had delayed recovery.

G. Craven et al¹⁰ in his study between regional anaesthesia (spinal, epidural and caudal) and general anaesthesia for inguinal hernia repair found that regional anaesthesia had got lesser side effects than general anaesthesia which coincides our study.

We compared the cost of anaesthesia in both groups in table IV (in terms of taka) and found that general anaesthesia-opioid analgesia is 13 times more costly than caudal anaesthesia-analgesia. Schrock et al and Gunter et al¹² found that 0.175% Bupivacaine could produce adequate surgical anesthesia. In our study, we used 0.25% Bupivacaine, which were good for surgical relaxation.

In conclusion, caudal anaesthesia and analgesia for children were good for operation and postoperative period. Recovery and discharge were earlier and above all cost-effective, safe and were good for postoperative pain relief. It can be used safely for day care surgery.

References

1. Aitkenhead AR, Iain K. Moppett, Jonathan P. Thompson, Smith & Aitkenheads Textbook of anaesthesia, Sixth edition, New York, Churchill Livingstone Elsevier, 2013; p-534.
2. Brown DL. Atlas of Regional Anesthesia, New York, Elsevier Mosby Saunders; 2010, p-410.
3. Vickers M. Drugs in Anaesthetic and Intensive Care Practice. Eighth ed, London, Butterworth-Heinemann, 1999, p-121.
4. Jankovic D. Regional nerve blocks and Infiltration therapy: Textbook and color Atlas, 3rd edition, New York, Blackwell, 2004, p-390.
5. Kumar P, Rudra A, Pan AK, Acharya A. Caudal additives in paediatrics: a comparison among midazolam, ketamine and neostigmine co-administered with Bupivacaine. *Anaesth Analg* 2005; 101:69-70.
6. Zacharoff KL, Pujol LM, Corsine E. Pocket guide to pain management. 4th ed, Pub Inflexxion 2010, p-174.
7. Uguralp S, Mutus, Koroglu A, Gurbuz N, Koltuksuz U, Demican M. Regional Anesthesia is a good alternative to general anesthesia in pediatric surgery: Experience in 1,554 children. *J Pediatr Surg* 2002; 37:610-3.
8. Locatelli B, Ingelmo P, Sonzogni, Keta et al. Randomized, double blind phase-III, controlled trial comparing levo-bupivacaine 0.25%, ropivacaine 0.25% and bupivacaine 0.25% by the caudal route in children. *Br J Anaesth* 2005; 94:366-71.
9. Schrock CR, Jones MB. The dose of caudal epidural analgesia and duration of postoperative analgesia. *Paediatric Anaesth*: 2003 13: 403-8.
10. Craven PD, Badawi N, Henderson-Smart DJ, O'Brien M. Regional (Spina, epidural caudal) versus general anesthesia in preterm infants undergoing herniorrhaphy in early infancy. *Cochrane Database System Rev*: 2003; (3) CD 003669.



Pearls and Pitfalls in the Management of Geriatric Trauma and Orthopaedic problems

Dipankar Nath Talukder¹, IshtiaqueUl Fattah¹, Abdus Sabur Miah², M.A. Hannan³, Faruqul Islam³, Mohsenuzzaman Khan³, Shukla Rani Das⁴

Abstract

The geriatric trauma patient presents unique challenges to the emergency medical service provider. There are many ways to classify older adults; the term geriatric has been defined as individuals who are ≥ 65 years old. Aim of this observational study is to understand the pathophysiology of aging, pre-existing medical conditions affect the geriatric populations, the changes of aging populations to modifications of care for the elderly trauma patient, ability of the geriatric patient to compensate for shock and complications of treatment.

[OMTAJ 2015; 14(1)]

Introduction

The geriatric trauma patient presents challenges to the emergency health care provider. The term geriatric is defined as individuals who are ≥ 65 years old. People are living longer and are relatively healthier than their counterparts a generation ago, resulting in a rapid increase in both the number and percentage of people 65 and over. Estimates predict that by 2030 and 2050, one in five people in the United States and Indian subcontinent respectively will have reached age 65.¹

Geriatric trauma patients differ than younger patients due to associated physiological changes that occur with normal aging, multiple co-morbidities and prescription drug regimens that present prior to their traumatic event.^{2,3} Because of these age-related differences, geriatric trauma patient injuries from relatively minor accidents can have devastating

consequences—their response to bleeding, injury and shock differs greatly from their 18-year-old counterparts.

Normal changes of aging affect every pathophysiologic system.⁶ Aging is a progressive process. Physiologic reserve continues to decrease with age and eventually effects outcomes.^{2,4,5} Age-related changes such as weakness, unsteady gait, slowed reaction times and cognitive impairments, and changes in eyesight predispose older individuals to trauma. The cardiovascular system loses the ability to respond to compensatory mechanisms efficiently with aging due to loss of vasculature elasticity, atherosclerosis and reduced baroreceptor activity results in shock developing earlier in the elderly.⁷ Aging respiratory systems result in loss of alveolar elastic recoil, stiffening of the chest wall and an increase in flow resistance.⁸

Geriatric patients are more likely to have osteoarthritis, scoliosis and kyphosis, which impact normal physiology, mobility and function. Osteoporosis leads to increased potential to traumatic fracture which occur most commonly in the hip, distal radius, humerus and vertebral bodies.⁹ Only 2–3 non displaced rib fractures becoming a major source of pneumonia in the geriatric trauma patient.¹⁰

The changes in the central nervous system are both in structure, with decreasing numbers of neurons, and function, with subtle changes in vision, reaction time and cognitive function.¹¹ Shrinking brain mass also increases susceptibility to shearing forces and axonal injuries.^{2,3} 5% of geriatric patients exhibiting some degree of dementia.¹² Changes of aging also have an effect on pain perception.⁶

During the aging process, the skin loses the structural support of elastin fibers. Skin becomes more fragile,

1. Associate Professor of Ortho surgery, Sylhet MAG Osmani medical College
2. Director, Sylhet MAG Osmani Medical College Hospital
3. Consultant, Orthopaedic Surgery, Sylhet MAG Osmani medical College Hospital
4. Consultant, Obstetrics & Gynaecology, South Surma Upozila Health Complex, Sylhet

causing delayed wound healing and difficulty maintaining body temperature.^{2,6,8}

Pre-existing diseases are prevalent in the older adult. Just over 50% of older adults have underlying hypertension and > 30% have heart disease.¹³ Other conditions such as diabetes, previous cerebrovascular accident (CVA), chronic obstructive pulmonary disease (COPD), dementia and endocrine disorders are also more prevalent among the elderly. If a geriatric patient with a history of congestive heart failure is on a beta-blocker or an anticoagulant and suffers a traumatic event, the mortality rate is 5–10 times greater.¹⁴ Heart rates above 90 and systolic blood pressures (SBPs) > 110 mmHg are associated with increased mortality.¹⁵ When the patient is on anticoagulants or antiplatelet, relatively minor wounds can bleed more rapidly. The geriatric trauma patient is five times more likely to die from trauma than a younger patient who sustains a similar mechanism of injury.¹ Blunt trauma is more common with falls, accounting for nearly three quarters of all geriatric trauma.¹⁶ One out of five falls causes a serious injury such as broken bones or a head injury.¹⁷ Falls associated with blunt cerebral injury and long bone fractures have the greatest morbidity and mortality.¹⁸ About 25% of geriatric patients in MVCs suffer rib fractures and flail segments, which have more serious outcomes such as pneumonia and respiratory failure.¹⁹

The presence of a precipitating medical event in the older trauma patient may complicate assessment, response to treatment, and outcome.

Materials and Methods

This is an observational type of prospective study conducted in orthopaedic department of Sylhet MAG Osmani Medical College Hospital between September 2013 to August 2014, overaged patient ≥ 65 years and treated accordingly. Common geriatric trauma was hip related, ankle injury, proximal humeral fracture, comminuted fracture of humeral condyle, distal radius and spinal fracture. Common geriatric orthopaedic problems were cervical & lumbar spondylosis with radiculopathy, disabling frozen shoulder, osteoarthritis of knee, hip, ankle with disarrangement of mechanical axis, kyphosis, scoliosis, spondylolisthesis, Secondary metastatic diseases of spine, pelvis and proximal humerus and

plasmacytoma. Open fracture and morbid patients were excluded. Quantitative data was expressed as mean (SD) and qualitative data by percentage.

Results

Total number of study subject 834 of which trauma and orthopaedic cases was 65.1% and 34.9% respectively. Mean age was 79.4 ± 9.7 with range 65–94 years. Patient arrived at hospital within 1 day to 4 months. (Table - I)

Overall operative treatment was given in 50.6% cases. 69.9% trauma cases underwent operation where as 85.6% orthopaedic cases treated non operatively. (Table II, III, IV)

Most common complication was wound infection (3.5%) followed by implant failure (2.9%). Post operative bed sore also developed in 1.7% cases. (Table-V)

Mean hospital stay was 15.5 (4.3) days.

Table-I: Demographic information (n=834)

	Frequency	Percentage
Trauma cases	543	65.1
Orthopaedic cases	291	34.9
Male	437	52.4
Female	397	47.6
Age	Range(65- 94)	79.4 ± 9.7
Delay in hospital arrival	Range 1day-4months(trauma)	43.8 ± 11.2 (days)
Hospital stay	Range(10- 29days)	15.5 ± 4.3

Table-II: Distribution of patients according to treatment

	Conservative	Operative
Trauma	163(30.1%)	380(69.9%)
Orthopaedics	249(85.6%)	42(14.4%)
Total	412(49.4%)	422(50.6%)

Table-III: Patterns of operations (Trauma)

	Hemi	T Arthro	Screw	ORIF	CRIF	Ex. Fix	K wire
Hip related(126)	32	2	19	45	28	-	-
Humerus 98	-	1	-	20	-	2	9
Ankle 182	-	-	39	69	-	-	-
Radius 132-	-	-	-	80	-	8	24
Spine			2				

Table-IV: Patterns of treatment(Orthopaedics)

Disease		number	Non-operative	operative
Cervical & lumbar spondylosis		89	78	11
Frozen shoulder		109	107	2
Osteoarthritis	Hip	20	12	8
	Knee	35	29	6
	Ankle	11	6	5
Kyphosis		10	10	0
Scoliosis		3	3	0
Spondylolisthesis		2	2	0
Metastasis		10	1	9
Plasmacytoma		2	1	1

Table-V: Complications

Type	No	Percentage
Wound infection	29	3.5
Implant Failure	25	2.9
Bed sore	12	1.4
Neuropraxia	14	1.7

Discussion

We used pre-operative skeletal traction in all hip related fracture. Preoperative skin or skeletal traction is intended to decrease preoperative pain and assist with fracture reduction. There were no differences in pain with or without the use of traction on either the first or second postoperative day.²⁰ One trial indicated that analgesic use increased with pre-operative traction use.²¹

Pressure sores are common following hip fracture, with reported postfracture incidence rates ranging from 10% to 40%.²² We used air mattress to prevent pressure sores which increased risk of nosocomial infection and prolonged hospitalization. Two trials reported that foam and alternating pressure mattresses reduced the incidence of pressure sore development compared with usual care.²³

Despite numerous clinical trials regarding specific surgical techniques, best practices remain unclear, particularly for femoral neck fractures. A compression screw plate device is considered to be the standard of care for inter-trochanteric or extracapsular fractures. Surgical management of femoral neck or intracapsular fractures is dependent upon patient age, activity level, health status, and surgeon preference.²⁴

Suction wound drainage, routine practice in many hospitals, is implemented to promote postoperative wound healing by preventing large hematoma formation.²⁵ This technique, however, has an inherent

risk of increasing postoperative infection through the creation of a portal to deep tissues.

In 22 RCTs of adult patients with closed long bone fracture fixation, of which 16 trials were hip fracture patients, antibiotic prophylaxis decreased the incidence of deep wound infections and urinary tract infections²⁶

When patients sustain a foot or ankle injury, they also present with preexisting medical comorbidities. Combining their injuries and their medical problems often results in disproportionate stays in the intensive care unit or the hospital with greater risks of complications and mortality.² Withholding surgical intervention from the elderly patients who present with high-energy injuries can result in significant problems producing an increase in the socioeconomic burden from those injuries. This occurs despite a better understanding of the approaches and the techniques needed to manage these injuries, expected outcomes, and the resulting debilitating nature of injuries when they are treated nonoperatively.²

Proximal humerus fractures are common injuries, especially among older osteoporotic women. Restoration of function requires a thorough understanding of the neurovascular, musculotendinous, and bony anatomy. In the vast majority of cases, proximal humerus fractures may be treated non-operatively. In displaced fractures, however, surgical intervention may be pursued. While numerous constructs have been investigated, the proximal humerus locking plate is most widely used and effective. In our experience, with proper restoration of the medial calcar, even 3 and 4 part proximal humerus fractures may be effectively treated with ORIF. Arthroplasty is reserved for fractures that cannot be reconstructed, such as comminuted 4-part fractures, head-split fractures, or fractures with significant underlying arthritic changes. Reverse total shoulder arthroplasty is reserved for patients with a deficient rotator cuff, or highly comminuted tuberosities.²

Distal humerus fractures in the osteoporotic patient pose a treatment challenge. The surgeon is faced with technical difficulties such as poor screw purchase and comminuted fracture fragments in soft fragile osteoporotic bone. Although some authors still advocate formal osteosynthesis with plates and report good to excellent results. Pajarinen and Bjorkenheim found that age over 50, poor bone quality, and immobilization were poor prognostic factors for

success of ORIF. Another mode of treatment in severely comminuted, osteoporotic fracture is total elbow replacement (TER). Several authors have reported good to excellent results with elbow arthroplasty with none to mild pain postoperatively.

As the population in some countries continues to age, the number of DRFs will increase as well. Even though these fractures are among the most common injuries treated by orthopedic, trauma, and hand surgeons, the treatment options are variable and remain a topic of debate. There is still no consensus regarding the best treatment option for unstable DRFs in the elderly individuals. Stable and reducible Colles fractures, which do not re-displace in a cast in the first 10 days after reduction, are treated non-operatively with satisfactory radiologic and functional results.²

Chung et al systematically reviewed the current literature for the treatment options of DRFs in patients over the age of 60 years treated with 5 common techniques: volar locking plate system, non-bridging EF, bridging EF, percutaneous K-wire fixation, and cast immobilization. The authors concluded that despite worse radiographic results in the group with cast immobilization, functional results were no different from those in the surgically treated groups for patients above the age of sixty years.

The level of mobility after early ambulation is equal to the level of mobility after immobilization. Where immobilization may lead to complications, early ambulation was not associated with new complications or neurological damage. Based on these advantages, the treatment of elderly patients with a fracture involving both middle and anterior columns can be changed from immobilization to early ambulation. Therefore, the diagnosis of middle column damage by CT scan becomes less relevant in determining the treatment policy. Consequently, a diagnostic protocol with a limited role for CT imaging in elderly patients with a thoracolumbar spine fracture may be possible.

The MVA was still the most common mechanism of injury after which falls became the most common mechanism with the elderly also in our study.

We can conclude from our study that management of geriatric trauma and orthopaedic problem is difficult and a joint effort between orthopaedists, primary care physicians, anesthesiologist, nurses, orthotist, physiotherapist and social worker. Functional outcome is maximized by early fixation and mobilization in operative cases. Prevention is multifaceted: a fragility

fracture is the strongest predictor of a future fracture. So, with the increasing elderly population, orthopaedic surgeons must be proactive in secondary prevention of fragility fractures

References

1. Centers for Disease Control and Prevention. The State of Aging and Health in America 2013. Atlanta, Ga.: Centers for Disease Control and Prevention, US Dept of Health and Human Services, 2013.
2. American College of Surgeons: Advanced trauma life support for doctors: Student course manual, 8th ed. American College of Surgeons: Chicago, 2008.
3. National Association of EMTs: Prehospital trauma life support: Student course manual, 8th ed. Jones and Bartlett: Burlington, Ma., 2016.
4. Bonne S, Schuerer DJE. Trauma in the older adult: Epidemiology and evolving geriatric trauma principles. Clin Geriatr Med. 2013;29:137–50.
5. Victorino GP, Chong TJ, Pal JD. Trauma in the elderly patient. Arch Surg 2003;138:1093–8.
6. Nicole NH, Heuther SE: Structure, function, and disorders of the integument. In:McCance K, Heuther S (Eds.): Pathophysiology: The biologic basis for disease in adults and children. 6th ed. St. Louis, Mosby, 2010.
7. Brashers VL, McCance KL: Structure and function of the cardiovascular and lymphatic systems. In: McCance K, Heuther S (Eds.): Pathophysiology: The biologic basis for disease in adults and children. 6th ed. St. Louis, Mosby, 2010.
8. Brashers VL. Structure and function of the pulmonary system. In:McCance K, Heuther S (Eds.): Pathophysiology: The biologic basis for disease in adults and children. 6th ed. St. Louis, Mosby, 2010.
9. Crowther-Radulewicz CL: Chapter 41: Structure and function of the musculoskeletal system. In McCance K, Heuther S (Eds.): Pathophysiology: The biologic basis for disease in adults and children. 6th ed. Mosby: St. Louis, Mo., 2010.
10. Elmistekway EM, Hammad AA. Isolated rib fractures in geriatric patients. Ann Thorac Med 2007;2:166–8.
11. Sugerman RA: Structure and function of the neurologic system. In McCance K, Heuther S (Eds.): Pathophysiology: The biologic basis for disease in adults and children. 6th ed. Mosby: St. Louis, Mo., 2010.

12. Carpenter CR, DesPain B, Keeling TN, et al. The six-item screener and ADB for the detection of cognitive impairment in the geriatric emergency department patients. *Ann Emerg Med.* 2011;57:653–7.
13. Thompson HJ, McCormick WC, Kagan SH. Traumatic brain injury in older adults, epidemiology, outcomes and future implications. *J Am Geriatr Soc.* 2006;54:1590–5.
14. Ferraris VA, Ferraris SP, Saha SP. The relationship between mortality and preexisting cardiac disease in 5,971 trauma patients. *J Trauma* 2010;69:645–52.
15. Hefferman DS, Thakkar RK, Monahan SF, et al. Normal presenting vital signs are unreliable in geriatric blunt trauma victims. *J Trauma* 2010;69:813–20.
16. Labib N, Mouh T, Winocour S, et al. Severely injured geriatric population: Morbidity, mortality, and risk factors. *J Trauma* 2011;71:1908–14.
17. Centers for Disease Control and Prevention. (Sept. 21, 2015.) Important facts about falls. Available at: www.cdc.gov/HomeandRecreationalSafety/Falls/adultfalls.html. (Accessed on Sept. 24, 2014)
18. Stevens JA, Sogolow ED. Gender differences for non-fatal unintentional fall related injuries among older adults. *Injury Prev* 2005;11:115–9.
19. Lee WY, Cameron PA, Bailey MJ. Road traffic injuries in the elderly. *Emerg Med J* 2006;23:42–6.
20. Parker MJ. Pre-operative traction for fractures of the proximal femur. *Cochrane Database Syst Rev* 2003;(Issue 3).
21. Rosen JE, Chen FS, Hiebert R, Koval KJ. Efficacy of preoperative skin traction in hip fracture patients: a prospective, randomized study. *J Orthop Trauma.* 2001;15:81–5.
22. Allman RM, Goode PS, Burst N, Bartolucci AA, Thomas DR. Pressure ulcers, hospital complications, and disease severity: impact on hospital costs and length of stay. *Adv Wound Care.* 1999;12:22–30.
23. Nuffield Institute of Health, University of Leeds, NHS Centre for Review and Dissemination. How effective are pressure-relieving interventions for the prevention and treatment of pressure sores? *Effective Health Care Bull* 1995;2:1–15.
24. Audige L, Hanson B, Swiontkowski MF. Implant-related complications in the treatment of unstable intertrochanteric fractures: meta-analysis of dynamic screw-plate versus dynamic screw-intramedullary nail devices. *Int Orthop* 2003;27:197–203.
25. Parker MJ. Closed suction surgical wound drainage after orthopaedic surgery. *Cochrane Database Syst Rev.* 2001 Issue 4.
26. Gillespie WJ, Walenkamp G. Antibiotic prophylaxis for surgery for proximal femoral and other closed long bone fractures. *Cochrane Database Syst Rev.* 2001 Issue 1.
27. Growing R, Jain MK. Injury patterns and outcomes associated with elderly trauma victims in Kingston, Ontario. *Can J Surg.* 2007;50:437–444
28. Brauer CA, Manns BK, Ko M, Donaldson C, Buckley R. An economic evaluation of operative compared with nonoperative management of displaced intra-articular calcaneal fractures. *J Bone Joint Surg-Am.* 2005;87:2741–9
29. Boyle MJ, Youn SM, Frampton CM, Ball CM. Functional outcomes of reverse shoulder arthroplasty compared with hemiarthroplasty for acute proximal humeral fractures. *J Shoulder Elbow Surg.* In press.
30. John H, Rosso R, Neff U, Bodoky A, Regazzoni P, Harder F. Operative treatment of distal humeral fractures in the elderly. *J Bone Jt Surg Br* 1994;76:793–6.
31. Handoll HH, Madhok R. Withdrawn: surgical interventions for treating distal radial fractures in adults. *Cochrane Database Syst Rev.* 2009;3:1–3
32. Warwick D, Field J, Prothero D, Gibson A, Bannister GC. Function ten years after Colles' fracture. *Clin Orthop Relat Res.* 1993;295:270–4
33. Chung KC, Shauver MJ, Birkmeyer JD. Trends in the United States in the treatment of distal radial fractures in the elderly. *J Bone Joint Surg Am.* 2009;91:1868–73
34. Abudou M, Chen X, Kong X, Wu T. Surgical versus non-surgical treatment for thoracolumbar burst fractures without neurological deficit. *Cochrane Database Syst Rev.* 2013;6:CD005079.
35. Schwab CW, Kauder DR. Trauma in the geriatric patient. *Arch Surg.* 1992;127:701–6

Clinical Profile and Short Term Outcome of Acute Stroke: A Comparison between Diabetic and Non Diabetic Patients.

Md. Enayet Hossain¹, FerdausAhammed²,Subrata Ray³, Abu Sayeed Abdullah ⁴, Gobinda Karmaker³

Abstract

Diabetes mellitus (DM) and stroke are closely related as a part and consequence of metabolic syndrome. Diabetes mellitus is one of the most important risk factor of stroke and it's consequent morbidity and mortality.

Here we explored the clinical presentation of stroke and its outcome in both diabetic and non diabetic patient in this prospective comparative study.

The study was conducted on two groups that included total 164 subjects admitted in the Medicine and Neurology department of the Sylhet M A G Osmani Medical College Hospital during the study period from July 2014 to December 2014. One group contained 82 diabetic patients presented with acute stroke. Other group contained 82 patients of acute stroke without diabetes.

In this study the mean age of the patients of diabetic group was significantly lower than that of non-diabetic group ($p=0.023$). The sex difference between the patients of diabetic and non-diabetic group did not show any statistically significant difference ($p=0.151$). Ischemic stroke was significantly more in diabetic group than that of non-diabetic group ($p<0.01$) and hemorrhagic stroke in diabetic group was significantly less than that of non diabetic group ($p<0.01$). Stroke was severe in 39 (47.6%) patients of diabetic group and 20 (24.4%) patients of non-diabetic group. This study revealed that mortality was 14 (17.1%) in diabetic group and 3 (3.7%) in non-diabetic group at day 7 which was significantly

higher in diabetic group compared to non-diabetic group (OR=5.422; 95% CI=1.495-19.664; $p=0.005$). At day 7 poor functional outcome was significantly more in diabetic group compared to non-diabetic group of stroke patients (OR=2.748; 95% CI=1.386-5.447; $p=0.003$)

In conclusion, stroke in the diabetic patient is different from stroke in the non-diabetic patient in several aspects. The diabetic stroke patients are relatively younger. Ischemic stroke is more but hemorrhagic stroke is less frequent in diabetic individuals. Stroke severity at presentation, mortality rate and poor functional outcome are higher at day 7 in diabetic individual.

[OMTAJ 2015; 14(1)]

Introduction

After cardiac disease and cancer, stroke is the third most common cause of death. Diabetes mellitus is one of the major risk factors for stroke and it's consequent morbidity and mortality. This is probably due to the side effects of hyperglycemia in association with other risk factors, such as: hypertension, dyslipidaemia, obesity, etc. Here we tried to explore the clinical presentation and outcome of stroke in diabetic and non diabetic patient.

The Objective of study was to assess the clinical presentation of stroke and it's short term outcome in both diabetic andnon diabetic patient.

Materials and methods

This prospective comparative study was conducted on two groups that included total 164 subjects admitted in the Medicine and Neurology department of the Sylhet M A G Osmani Medical College Hospital during the study period from July 2014 to December 2014. One group contained 82 diabetic patients presented with acute stroke. Other group included 82 patients of acute stroke without diabetes.

1. Associate professor of Medicine, Sylhet MAG Osmani Medical College.
2. Lecturer (Pathology), Sylhet MAG Osmani Medical College.
3. Assistant registrar (Medicine), Sylhet MAG Osmani Medical College Hospital.
4. Junior Consultant (Medicine), Upazilla Health Complex, South Surma . Sylhet

The diagnosis of stroke was made based on clinical data and CT scan of brain.

Patients with history of transient ischemic attack and recurrent stroke were not included in the study.

Past medical and personal history for cigarette smoking, arterial hypertension and other associated disease condition were also sought.

Stroke severity at admission was determined using the modified National Institutes of Health Stroke Scale² and stroke was classified as mild (score 0-5), moderate (score 6-14), severe (score 15-31) on mNIHSS scale.

All those who had documented history of diabetes in past (treated with either insulin, oral hypoglycemic agents or not treated) or those who had random blood glucose level ≥ 11.1 mmol/liter (200 mg/dl) or fasting blood glucose level ≥ 7 mmol/liter (≥ 126 mg/dl) and HbA1C $\geq 6.5\%$, were included in diabetic group. Those who have no history of diabetes mellitus in past or random blood glucose level less than 200 mg/dl or fasting blood glucose level less than 126 mg/dl and HbA1c below 6.5% were included in non diabetic group.

Informed written consent was obtained from the patients or eligible guardians after full explanation of the details of the disease process and purpose of the study.

Results

The outcome of the study was as follows:

Table I: Age distribution of the patients

Age (years)	Study group		p value
	Diabetic (n=82)	Non diabetic (n=82)	
<40	2 (2.4)	1 (1.2)	*p=0.898
41-50	10 (12.2)	3 (3.7)	
51-60	41 (50)	36 (43.9)	
61-70	21 (25.6)	27 (32.90)	
>71	8 (9.8)	15 (18.3)	
Mean(+/-SD)	60.58 (SD 9.35)	63.78 (SD 8.45)	†p=0.023

Figure in the parenthesis indicates corresponding percentage

Table II: Sex distribution of the patients

Sex	Study group		*p value
	Diabetic (n=82)	Non diabetic (n=82)	
Male	54 (65.9)	45 (54.9)	p=0.151
Female	28 (34.1)	37 (45.1)	
Total	82 (100)	82 (100)	

Figure in the parenthesis indicates corresponding percentage

Table III: Smoking status of the patients

Sex	Study group		*p value
	Diabetic (n=82)	Non diabetic (n=82)	
Smoker	50 (61)	41 (50)	p=0.157
Non-smoker	32 (39)	31 (50)	
Total	82 (100)	82 (100)	

Figure in the parenthesis indicates corresponding percentage

Table IV: Distribution of the patients by status of arterial B.P.

Sex	Study group		*p value
	Diabetic (n=82)	Non diabetic (n=82)	
Hypertensive	54 (65.9)	44 (53.7)	p=0.037
Normotensive	28 (34.1)	38 (46.3)	
Total	82 (100)	82 (100)	

Figure in the parenthesis indicates corresponding percentage

Table V: Distribution of patients by severity of stroke on admission

Sex	Study group		*p value
	Diabetic (n=82)	Non diabetic (n=82)	
Severe	39 (47.6)	20 (24.4)	p=0.002
Mild to moderate	41 (52.4)	62 (75.6)	
Total	82 (100)	82 (100)	

Figure in the parenthesis indicates corresponding percentage

Table VI: Distribution of patients according to dyslipidaemia

Dyslipidaemia	Study group		*p value
	Diabetic group (n=82)	Non-diabetic group (n=82)	
Present	51 (62.2)	37 (45.1)	p=0.028
Absent	31 (37.8)	45 (54.9)	
Total	82 (100.0)	82 (100.0)	

Figure in the parenthesis indicates corresponding percentage.

Table VII: Distribution of patients according to type of stroke

Type of stroke	Study group		*p value
	Diabetic group (n=82)	Non-diabetic group (n=82)	
Ischaemic stroke	65 (79.3)	50 (61.0)	p<0.01
Haemorrhagic stroke	17 (20.7)	32 (39.0)	p<0.01
Total	82 (100.0)	82 (100.0)	

Figure in the parenthesis indicates corresponding percentage.

Table VIII: Mortality at day 7

Mortality	Study group		Odd Ratio (95% CI)	p value
	Diabetic group (n=82)	Non-diabetic group (n=82)		
Death	14 (17.1)	3 (3.7)	5.422 (1.495-19.664)	p=0.005
Survive	68 (82.9)	79 (96.3)		
Total	82 (100.0)	82 (100.0)		

Figure in the parenthesis indicates corresponding percentage. OR=Odd ratio; CI= confidence interval.

Table IX: Functional outcome of survivors at day 7

Functional outcome	Study group		Odd Ratio (95% CI)	p value
	Diabetic group (n=82)	Non-diabetic group (n=82)		
Poor (mRS, >2)	35 (51.1)	22 (27.8)	2.748 (1.386-5.447)	p=0.039
Good (mRS, ≤2)	33 (48.5)	57 (72.2)		
Total	68 (100.0)	79 (100.0)		

Figure in the parenthesis indicates corresponding percentage.

Discussion

Stroke is one of the leading causes of morbidity and mortality world wide. Diabetes mellitus (DM) is one of the well known risk factors for stroke. It has been suggested that stroke patients with DM have higher in-

hospital mortality rates and poorer outcome than those without diabetes.

Although strokes in patients with diabetes are associated with a worse outcome, there is no evidence to suggest that diabetes induces a larger area of cerebral infarction. Conversely, larger infarction volumes have been reported in those without diabetes, but with stress hyperglycemia, with the later appearing more likely to be a marker of severity.⁶

In this prospective comparative study, a total 164 patients with acute stroke of first attack were selected during the study period. Of them 82 patients with acute stroke having diabetes mellitus were enrolled in diabetic group and 82 stroke patients without diabetes mellitus were enrolled in non diabetic group.

In this study the age of the patients ranged from 35 to 90 years with the mean age of 60.58 (SD 9.35) years in diabetic group; whereas the age of the patients of non-diabetic group ranged from 38 to 85 years with the mean age of 63.78 (SD 8.45) years. The mean age of the patients of diabetic group was significantly lower than that of non-diabetic group (p=0.023). This result was supported by Jorgensen et al. that the mean age of the patients of diabetic group was significantly lower than nondiabetic group (71.5 ± 10.5 years vs 74.7 ± 11.2 years; p<0.001). But Zafaret al. found no age difference between diabetic and non-diabetic stroke patients (59.5 ± 11.82 years vs 60.4 ± 14.80 years; p>0.05).

In the present study there were 54 (65.9%) male and 28 (34.1%) female in diabetic group; whereas 45 (54.9%) male and 37 (45.1%) female in non-diabetic group. The sex difference between the patients of diabetic and non-diabetic group did not show any statistically significant difference (p=0.151). This result was correlated with the study of Zafaret al. that 28 (56%) male in diabetic group and 34 (68.0%) female in non-diabetic group. The sex of the patients both groups did not show any significant difference (p>0.05). Megherbiet al. found no sex difference between diabetic and non-diabetic stroke patients [female 50.9% vs 49.9%; p=0.580]. There was no significant difference between the sexes regarding different types of stroke suggesting that diabetes has the same impact on the cerebral vessels in both sexes.

This study revealed that 50 (61.0%) patients of diabetic group were smoker and 41 (50.0%) patients of non-diabetic group were smoker. The difference between the two groups was statistically not significant (p=0.157). Jorgensen et al. found that 41% of patients

of diabetics were smoker and 46% of patients of non-diabetics were non smoker ($p=0.20$). Zafaret al. also found that there was no significant difference of smoker between diabetic group and non-diabetic group [20 (40.0%) vs 16 (32.0%); $p=0.28$].

This study showed that 54 (65.9%) patients were hypertensive in diabetic group and 44 (53.7%) patients were hypertensive in non-diabetic group. The difference between the two groups was statistically significant ($p=0.037$). This result was supported by Sarkaret al. that stroke with diabetic patients significantly more hypertensive (70.9%) than that of non-diabetic patients (47.6%) ($p<0.001$). Jorgensen et al. also supported the present study that hypertension was significantly more common in the diabetic stroke patients (48%) than that of non-diabetic stroke patients (30.0%) ($p<0.001$). Martz et al. ²also found hypertension was significantly more common in the diabetic patients than that of non-diabetic patients [73 (83.9%) vs 26 (55.3%); $p=0.004$]. Several other studies showed that hypertension was significantly more common in diabetic patients. This strengthens the fact that diabetes and hypertension are closely related.

In the present study stroke was severe in 39 (47.6%) patients of diabetic group and 20 (24.4%) patients of non-diabetic group. The difference between the two groups was statistically significant ($p=0.002$). It is well established that blood glucose increases after the onset of acute stroke and the increase is related to the severity of the stroke. Smajlovic et. was in agreement with this result that diabetic patients with stroke had more severe strokes than non-diabetic patients (SSS 20.5 vs 39, $p<0.0001$). Martz et al. also supported the present study that patients with diabetes had more severe strokes (NIHSS on admission: 7.2 ± 6.6 vs. 3.7 ± 3.6 normoglycemia $p<0.001$). But Jorgensen et al. that there was statistically no-significant difference between the diabetic and non-diabetic group in respect to SSS51 score on admission was 24.2 points (SD, 19.8) in diabetic patients versus 23.4 points (SD, 18.1) in non-diabetic patients ($p=0.81$).

This study revealed that 51 (62.2%) patients of diabetic group and 37 (45.1%) patients of non-diabetic group had dyslipidaemia. The difference between the two groups was statistically significant ($p=0.028$). This result was supported by Sarkaret al. that stroke with diabetic patients significantly more hypercholesterolemia (30.9%) than that of non-diabetic patients (21.1 %) ($p<0.001$). But Martz et al. ²

found no significant difference in hypercholesterolemia of diabetic and non-diabetic stroke patients [39 (44.8) vs 23 (48.9); not significant]. In the current study ischemic stroke was in 65 (79.3%) and hemorrhagic stroke in 17 (20.7%) patients in diabetic group; whereas in non-diabetic group ischemic stroke was in 50 (61.0%) and hemorrhagic stroke in 32 (39.0%) patients. Ischemic stroke was significantly more in diabetic group than that of non-diabetic group ($p<0.01$) and hemorrhagic stroke in diabetic group was significantly less than that of non-diabetic group ($p<0.01$). This result was in agreement with the study of Sarkar et al. that ischemic stroke were higher in diabetic group (69%) as compared to non-diabetic group (45.8%) which was significant ($p<0.001$); whereas hemorrhagic stroke was higher in non-diabetic group (52.7%) than in diabetic group (30.4%). Jorgensen et al. found ischemic stroke were higher in diabetic group (68%) as compared to non-diabetic group (60%) but the difference was insignificant ($p=0.09$); while hemorrhagic stroke was higher in non-diabetic group (9%) than in diabetic group (1%) ($p=0.002$). Zafaret al. reported ischemic stroke was more common in diabetic patients [44 (88%) vs 29 (58.0%); $p<0.001$]; while hemorrhagic stroke was more common in non-diabetic patients [21 (42.0%) vs 6 (12.0%); $p=0.002$]. ⁶

This study revealed that mortality was 14 (17.1%) in diabetic group and 3 (3.7%) in non-diabetic group at day 7. Mortality was significantly higher in diabetic group compared to non-diabetic group (OR=5.422; 95% CI=1.495-19.664; $p=0.005$). This result was almost similar to the study of Hamidon and Raymond,

that DM was an independent risk factor for mortality, OR 4.88 (95% CI 1.25-19.1). Diabetic patients are generally prone to infection and are less immunocompetent, and usually have concurrent multiple end-organ damage that ultimately contributes to increased mortality. The detrimental effects of diabetes on the outcome of cerebrovascular disease are in keeping with previous reports that diabetics have poorer outcome after stroke and coronary artery disease than those without DM.

In this study the functional outcome of survivors at day 7 was poor (mRS, >2) in 35 (51.1%) and good in 33 (48.5%) patients in diabetic group; whereas it was 22 (27.8%) and 57 (72.2%) patients respectively in non-diabetic group. At day 7 poor functional outcome was significantly more in diabetic group compared to non-diabetic group of stroke patients (OR=2.748; 95% CI=1.386-5.447; $p=0.003$). A systematically

review of the existing data showed that in patients with no history of diabetes who have an ischemic stroke, even moderately elevated glucose levels are associated with both a 3-fold higher risk of short-term mortality and an increased risk of poor functional recovery compared with lower glucose levels.² Furthermore, two large studies demonstrated that glucose level at hospital admission was a significant predictor of mortality,² or poor functional recovery,²² after stroke independent of other prognostic factors. In addition, a study showed that persistent hyperglycemia was an independent determinant of infarct expansion and was associated with worse functional outcome.²

In conclusion, the mean age of the stroke patients of diabetic group was younger than non-diabetic group [60.58 (SD 9.35) versus 63.78 (SD 8.45); $p=0.023$]; but no sex difference between two groups [54 (65.9%) male versus 45 (54.9%) male; $p=0.151$]. Diabetic stroke patients were more prone to be hypertensive [57 (69.5%) vs 44 (53.7%), $p=0.037$] and dyslipidaemic [51 (62.2%) versus 37 (45.1%); $p=0.028$].

Stroke was severe in patients of diabetic group than that of non-diabetic group [39 (47.6%) versus 20 (24.4%); $p=0.002$]. Ischemic stroke was significantly more [65 (79.3%) versus 50 (61.0%); $p<0.01$] and hemorrhagic stroke was significantly less in [17 (20.7%) versus 32 (39.0%); $p<0.01$] diabetic group than that of non-diabetic group.

Mortality at day 7 was significantly higher in diabetic group compared to non-diabetic group [14 (17.1%) versus 3 (3.7%); OR=5.422; 95% CI=1.495-19.664; $p=0.005$]. Poor functional outcome at day 7 was significantly more in diabetic group compared to non-diabetic group of stroke patients [35 (51.1%) versus 22 (27.8%); OR=2.748; 95% CI=1.386-5.447; $p=0.003$].

In conclusion, stroke in the diabetic patient is different from stroke in the non-diabetic patient in several aspects. The diabetic stroke patient is younger. Ischemic stroke is more but hemorrhagic stroke is less frequent in diabetic individuals. Stroke severity at presentation, mortality rate and poor functional outcome are higher at day 7 in diabetic individual.

References

1. Dumitra B, Lan P, A. Babe.; Incidence and Type of Stroke in Patients with Diabetes. Comparison Between Diabetics and Nondiabetics; Rom. J Intern Med 2009; 47: 249-55.
2. Lyden PD, Lu M, Levine SR, Brott TG, Broderick J, NINDS rtPA Stroke Study Group. A modified National Institutes of Health Stroke Scale for use in stroke clinical trials: preliminary reliability and validity. Stroke 2001; 32: 1310-7.
3. Govan L, Langhorne P, Weir CJ. Categorizing stroke prognosis using different stroke scales. Stroke 2009; 40(10):3396-99.
4. Meyer BC, Lyden PD. The modified National Institutes of Health Stroke Scale: its time has come. Int J Stroke 2009; 4: 267-73.
5. Sweileh WM, Zyoud SH, Sawalha AF, Al-Jabi SW, Abu-Taha AS. Clinical characteristics, sex differences and in-hospital mortality among stroke patients with and without diabetes mellitus. Diabetologia Croatica 2011; 40: 41-6.
6. Idris I, Thomson GA, Sharma JC. Diabetes mellitus and stroke. Int J Clin Pract 2006; 60: 48-56.
7. Jorgensen HS, Nakayama H, Raaschou HO, Olsen TS. Stroke in patients with diabetes: the Copenhagen Stroke Study. Stroke 1994; 25: 1977-84.
8. Zafar A, Shahid SK, Siddiqui M, Khan FS. Pattern of Stroke in Type 2 Diabetic Subjects Versus Non Diabetic Subjects. J Ayub Med Coll Abbottabad 2007; 19: 64-7.
9. Megherbi S-E, Milan C, Minier D, Couvreur G, Osseby G-V, Tilling K, et al. Association between diabetes and stroke subtypes on survival and functional outcome 3 months after stroke. Data from the European BIOMED Stroke Project. Stroke 2003; 34: 688-94.
10. Hamidon B B, Raymond A A. The Impact of Diabetes Mellitus on In-hospital Stroke Mortality. J Postgrad Med 2003; 49: 307-10.
11. Sarkar RN, Banerjee S, Basu A. Comparative evaluation of diabetic and non-diabetic stroke--effect of glycaemia on outcome. J Indian Med Assoc 2004; 102: 551-3.
12. Matz K, Keresztes K, Tatschl C, Dachenhausen A, Brainin M, Tuomilehto J, et al. Disorders of glucose metabolism in acute stroke patients. Diabetes Care 2006; 29: 792-7.
13. Stearne MR, Palmer SL, Hammersley S, Franklin SL, Spivey RS, Levy JC, et al. Tight blood pressure control and risk of macrovascular and microvascular complications in type 2 diabetes. Br Med J 1998; 317: 703-13.
14. Christensen H, Boysen G. Blood glucose increases early after stroke onset: a study on serial measurements of blood glucose in acute stroke. Eur J Neurol 2002; 9: 297-301.
15. Smajlovic D, Salihovic D, Ibrahimagic O, Sinanovic O, Burina A. Stroke in patients with diabetes mellitus: a hospital based study. Med Arh 2006; 60(6 Suppl 2): 63-5.
16. Alvarez-Sabin J, Molina CA, Montaner J, Arenillas JF, Huertas R, Ribo M, et al. Effects of admission hyperglycemia on stroke outcome in reperfused tissue

- plasminogen activator-treated patients. *Stroke* 2003; 34:1235-41.
17. Williams LS, Rotich J, Qi R, Fineberg N, Espay A, Bruno A, et al. Effects of admission hyperglycemia on mortality and costs in acute ischemic stroke. *Neurology* 2002; 59: 67-71.
 18. NINDS t-PA Stroke Study Group. Generalised efficacy of t-PA for acute stroke: Subgroup analysis of the NINDS t-PA Stroke Trial. *Stroke* 1997; 28: 2119-25.
 19. Haffner SM, Lehto S. Mortality from coronary heart disease in subjects with type 2 diabetes and in non-diabetic subjects with and without prior myocardial infarction. *N Engl J Med* 1998; 339:229-34.
 20. Capes SE, Hunt D, Malmberg K, Patnak P, Gerstein H. Stress hyperglycemia and prognosis of stroke on nondiabetic and diabetic patients. A systematic overview. *Stroke* 2001; 32: 2426-32.
 21. Moulin T, Tatu L, Crepin-Leblond T, Chavot D, Berges S, Rumbach L. The Besançon Stroke Registry: an acute stroke registry of 2500 consecutive patients. *Eur Neurol* 1997; 38: 10-20.
 22. Bruno A, Biller J, Adams HPJr, Clarke WR, Woolson RF, Williams LS, et al. for the trial of ORG 10172 in acute stroke treatment (TOAST) investigators. Acute blood glucose level and outcome from ischemic stroke. *Neurology* 1999; 52: 280-4.
 23. Baird TA, Parsons MW, Phan T, Butcher KS, Desmond PM, Tress BM, et al. Persistent poststroke hyperglycemia is independently associated with infarct expansion and worse clinical outcome. *Stroke* 2003; 34: 2208-14.



Detection of *Chlamydia trachomatis*, *Mycoplasma hominis* and *Ureaplasma urealyticum* in patients with sterile pyuria by Polymerase Chain Reaction

Ishrat Siddiqui¹, KM Shahidul Islam², Najneen Nehar³, Shahanaz Pervin⁴ and Sonia Akter⁵

Abstract

Microbiological diagnosis of sterile pyuria is a challenge for the microbiologist as the causative organisms does not grow in conventional culture media. *Chlamydia trachomatis*, *Mycoplasma hominis*, and *Ureaplasma urealyticum* are some causes of sterile pyuria. This study had been designed to determine the occurrence of *Chlamydia trachomatis*, *Mycoplasma hominis* and *Ureaplasma urealyticum* in patients with sterile pyuria. During the period of July 2011 to June 2012 a total 280 urine samples were collected from clinically suspected patient of UTI from outpatient department of Dhaka Medical College Hospital. All urine samples were evaluated for significant pyuria by microscopic examination of centrifuged urine. Hundred and four (37.14%) samples with significant pus cells were cultured on MacConkey and Blood agar media to detect the presence of bacteria. Fifty six samples having significant pyuria (pus cell ≥ 10 / HPF) and negative for culture (showing no significant growth after 24 hr) were tested by multiplex PCR for *C. trachomatis*, *M. hominis*, and *U. urealyticum*. Fourteen (25%) *Chlamydia trachomatis* and 2(3.57%) *Ureaplasma urealyticum* were detected in culture negative urine samples by PCR. The rate of isolation of *C. trachomatis* was more (85.71) in female. PCR testing of sterile pyuria showed a significant number of *C. trachomatis* and *Ureaplasma* infections. Consequently, PCR is recommended for the detection of those microorganisms in the urine samples of sterile pyuria patients.

[OMTAJ 2015; 14(1)]

Introduction

Sterile pyuria is a condition in which white blood cells (WBCs) are present in the urine (≥ 10 /HPF) without bacteria growing in standard cultures. Sterile pyuria is associated with infective agents including viruses, fungi, *Mycobacterium tuberculosis* and atypical or fastidious organism such as *Chlamydia trachomatis*, *Mycoplasmas* and *Ureaplasmas*.²

One-third of women with dysuria and other symptoms of UTI have "sterile urine" with or without pyuria. Organisms such as *Chlamydia trachomatis* and *Ureaplasma urealyticum* should be considered in patients with pyuria and negative cultures.

According to the world health organization, approximately 89 million people are newly infected with *Chlamydia trachomatis* infections annually worldwide. Although many diagnostic modalities are present for detection of *Chlamydia trachomatis*, none are 100% sensitive.⁶ Polymerase Chain Reaction (PCR) is an accurate, rapid and reliable method for the detection of *Chlamydia trachomatis* in urine.

Although *M. hominis* and *U. urealyticum* can cause different urogenital diseases, these pathogens usually escape routine microbiological investigation of urine samples due to their slow or absent growth on standard media. Culture is considered the reference standard for detection of *M. hominis* and *U. urealyticum*, but it is expensive, needs specialized media and requires 2–5 days. Nucleic acid amplification techniques can detect infectious agents in less than 8 hours.

Molecular diagnostic approaches such as DNA hybridization and amplification techniques are being applied to the diagnosis of many infections. PCR are useful for the identification of microorganisms that are difficult to cultivate and for those that grow slowly.

The goal of the present study was to detect *C. trachomatis*, *M. hominis* and *U. urealyticum* in urine specimens of patients with sterile pyuria by PCR. It is worth mentioning that no previous studies have been

1. Lecturer, Dept. of microbiology. Sylhet MAG Osmani Medical College
2. Professor, Dept. of Microbiology. Dhaka Medical College
3. Lecturer, Dept. of Virology. Dhaka Medical College
4. Lecture, Dept. of Microbiology. Khulna Medical College
5. Assistant professor, Dept. of Microbiology. Ibn Sina Medical college

conducted on this topic in Bangladesh. This study is important in identifying the etiological agents for many of sterile pyuria cases and would be helpful in choosing the correct treatment.

Material and Methods

This study had been carried out in Department of Microbiology of Dhaka Medical College between July 2011 to June 2012. A mid-stream specimen of urine was collected from the patients of reproductive age having symptoms and signs compatible with urinary tract infection including dysuria, polyuria, and nocturia. All samples were tested for routine urinalysis. Culture on MacConkey's agar and blood agar were considered for those samples which had significant pyuria (e.i. pus cells ≥ 10 /HPF). Urine samples from which bacteria were isolated were tested for their antimicrobial susceptibility pattern and ESBL production. Samples negative for culture (showing no significant growth after 24 hrs) were subjected to PCR for detection of *Chlamydia trachomatis*, *Ureaplasma urealyticum* and *Mycoplasma hominis*.

Preservation of samples for PCR: Five ml of urine was centrifuged at 3000g (Eppendorf AG, 22331 Hamburg) for 30 minutes and after discarding the supernatant about one ml of deposit was transferred to eppendorf tube and kept at -20 C.

Polymerase Chain Reaction (PCR)

DNA extraction

For pellet preparation, one ml of preserved urine sample was centrifuged at 2000g for ten minutes and after discarding the supernatant, the deposit was taken as pellet. Hundred μ l lytic buffer (60.5mg Tris-HCl +1mg proteinase k+5 μ l Twin 20 in 10ml d/w) was added with the pellet and vortexed thoroughly. The mixture was incubated at 60 C for two hours. After incubation the tube was placed in a block heater (DAIHA Scientific, Seoul, Korea) at 100 C for ten minutes. Then it was immediately transferred on ice and kept there for five minutes. The solution was again centrifuged at 2000g (Eppendorf AG, 22331 Hamburg) for ten minutes. The supernatant was taken as DNA template.

Primers used in this study

Mixing of mastermix and primer with DNA template

Organisms	Primer sequence	Product size
<i>M.hominis</i>	My-ins (5'-GTAATACAT AGGTCGCAAGCGTTATC-3') MGSO-2-Bi(5'-CACCAT CTGTCACTCTGTAAACCTC-3')	520 bp
<i>C.trachomatis</i>	KL1, 5'- TCCGGAGCG AGTT ACGAAGA-3 KL2,5'-AATCAATGCCCGG GATTGGT-3'	241 bp
<i>U. urealyticum</i>	U4 ACGACGT CCATAAGC AACT U5 CAATCTGCTCGTGA AGTATTAC	429 bp

Primers were mixed with Tris-EDTA (TE) buffer according to manufacturer's instruction. For each sample a total 25 μ l of mixture was prepared by mixing 12 μ l of master mix (mixture of dNTP, taq polymerase, MgCl₂ and PCR buffer), one μ l of forward primer, one μ l of reverse primer (promega corporation, USA) for each organism i.e total 6 μ l primer, 3 μ l DNA template and 4 μ l of nuclease free water in a PCR tube. After a brief vortex, the tubes were centrifuged in a micro centrifuge for few seconds.

Amplification in thermal cycler (Eppendorf AG, Master cycler gradient, Hamburg, Germany)

PCR reaction consisted of initial denaturation at 94 C for ten minutes followed by 36 cycles each consisting of denaturation at 94 C for one minute, annealing at 55 C for 45 seconds and extension at 72 C for one minute 30 seconds and final extension at 72 C for 10 minutes.

Electrophoresis

PCR products were detected by electrophoresis on 1.5 % agarose gel. The gel was placed in the electrophoresis tank and 1X TBE buffer was poured in it. Five μ l of amplified DNA were loaded into wells of agarose gel submerged in TBE buffer in tank and was subjected to electrophoresis for 35 minutes at 100 volts. In each experiment a DNA ladder, a negative control (autoclaved distilled water) and a positive control of DNA of corresponding bacteria was loaded in three wells of agarose gel.

Visualization and interpretation of results

The agarose gel was stained with ethidium bromide. The gel was observed under UV transilluminater (Gel Doc, major science, Taiwan) for DNA bands. The DNA bands were identified according to their molecular size by comparing with the molecular weight marker (100 bp DNA ladder) loaded in a separate lane. Samples showing

the presence of corresponding bp band were considered positive for the presence of that organism.

Results

Two hundred and eighty patients of reproductive age of both sexes with symptoms suggesting of UTI were enrolled in this study, among which 104 samples were selected for culture. Bacteria were identified from culture positive samples (48) and tested for antimicrobial susceptibility pattern and ESBL production. The culture negative samples (56) were subjected to PCR for detection of *C. trachomatis*, *U. urealyticum* and *M. hominis*. *C. trachomatis* was detected in 14(25%) samples and *U. urealyticum* in 2(3.57%) samples. No sample was positive for *M. hominis*. Female of age group 15-25 were found mostly (42.86%) to be infected with *C. trachomatis*. Two (14.29%) *C. trachomatis* were found in male of age group 36-45 years. One (50%) *U. urealyticum* was isolated from male of age group of 26-35 years and one (50%) *U. urealyticum* was found in female of age group 36-45 years.

Table I: Results of multiplex PCR in culture negative urine samples (n=56).

Name of the bacteria	Total n (%)
<i>Chlamydia trachomatis</i>	14 (25)
<i>Ureaplasma urealyticum</i>	2 (3.57)
<i>Mycoplasma hominis</i>	0 (0)
No organism	40 (71.43)

Table II: Age and sex wise distribution of the study population in relation to isolated bacteria from urine by PCR.

Age group In years	DNA of bacteria					
	<i>C. trachomatis</i>		<i>U. urealyticum</i>		<i>M. hominis</i>	
	M n (%)	F n (%)	M n (%)	F n (%)	M N (%)	F n (%)
(15-25)	—	6 (42.86)	—	—	—	—
26-35	—	3(21.43)	1(50)	—	—	—
36-45	2(14.29)	3(21.43)	—	1(50)	—	—
46-55	—	—	—	—	—	—
56-65	—	—	—	—	—	—
Total	2(14.29)	12 (85.71)	1(50)	1(50)	0(0)	0(0)

Discussion

Sterile pyuria in patients with clinical symptoms consistent with UTI can be a diagnostic challenge and needs further investigation for detection of fastidious and atypical bacteria like *C. trachomatis*, *M. hominis*, and *U. urealyticum*. These bacteria are associated with various diseases of the genitourinary tract, but they are usually not detected by routine microbiological diagnosis. *C. trachomatis*, *M. hominis* and *U. urealyticum* are not screened

by routine examination of urine samples in health laboratories. Findings of this study showed that PCR testing of sterile pyuria could identify a significant number of causative bacteria and should demonstrate to the clinicians the advantage of detection of the fastidious bacteria in urine from the patients with UTI symptoms, when standard cultures fail to detect the microbial infection.

C. trachomatis infections are the most prevalent sexually transmitted bacterial infections among women and men worldwide. Traditionally, the gold standard for the identification of *C. trachomatis* is culture. However, culture is time-consuming and labor-intensive. It takes 3 to 6 days to complete, and it requires access to specialized facilities and trained personnel.² ELISAs have also been evaluated as screening tests for the rapid identification of infected individuals by using first-catch urine. ELISAs are relatively fast and easy to complete, but sensitivities of the tests for urine specimens remain relatively low.

In the present study, *C. trachomatis* infection was detected in 14(25%) cases of culture negative urine samples by polymerase chain reaction (PCR). Similar study from UK detected 15(21%) *C. trachomatis* infection in 71 sterile pyuria samples by PCR. This finding is in accordance with result of present study. When compared to the other studies from other countries, 10% infection

was reported in Palestine, 30.8% infection was reported in India and 31% infection with *C. trachomatis* was reported in Egypt by PCR.⁶ Variations of this prevalence between countries and studies could be due to several factors such as, study population, rate of infection in the study area, hygiene level and socioeconomic status of the study area, culture of the society whether it is open or conservative, and the technique and the DNA target of PCR used.

In the present study, *C. trachomatis* infection was found more (85.72%) in women. The reason for this may be due to the fact that in this study female were more (64.29%) than male in culture negative urine samples. Our results revealed that the occurrence of *C. trachomatis* was higher than the occurrence of the other bacteria detected in this study. These results suggest that *C. trachomatis* infection should be strongly considered in urine samples that show sterile pyuria.

At present, the main method of detecting *U. urealyticum* and *M. hominis* is by culture, but the organisms are difficult to isolate and requires special culture media. But these organisms can be detected rapidly and accurately by PCR which also avoids the problems associated with culture. In the present study *U. urealyticum* was found in

2(3.57%) culture negative samples and no sample was positive for *M.hominis*. When comparing to other studies, 22.1% infection with *U.urealyticum* and 11.6% infection with *M.hominis* was reported from Korea, 5% infection with *U.urealyticum* and 3% infection with *M.hominis* was reported from Palestine. These variations could be explained by differences in the study population and rate of infection in the study area.

Finally from these findings it can be concluded that, patient having symptoms suggesting of UTI with sterile urine in culture with pyuria should be consider for PCR to detect fastidious bacteria like *Chlamydia trachomatis* and *Ureaplasma urealyticum*. Furthermore broad spectrum study should be carried out to see the prevalence of these bacteria among high risk group like sexual worker or in STDs clinics. We also recommend to use PCR as a rapid and effective tool for the diagnosis of fastidious bacteria like Clamydia, Ureaplasma and Mycoplasma.

References

1. Nassar FA, Elamreen FH, Shubair ME, Sharif FA. Detection of *Chlamydia trachomatis*, *Mycoplasma hominis*, and *Ureaplasma urealyticum* by polymerase chain reaction in patients with sterile pyuria. *Adv Med Scien* 2008;53:80-6.
2. Tayal SC, Pattman RS. Sterile pyuria: consider chlamydial infection. *Brit J Clin Prac* 1996;50:166-7.
3. Head K. Natural approaches to prevention and treatment of infections of the lower urinary tract. *Alter Med Rev* 2008;13:227-30.
4. Simerville JA, William CM, Pahira JJ. Urinalysis: A comprehensive review. *Am F Phys* 2005;71:1153-61.
5. Rowe PJ. Reproductive tract infection. Annual Technical Report. Human Reproductive Programme. WHO;1998.186-90.
6. Mittal V, Agarwal J, Jain A, Verma AK. Prevalence of genital *Chlamydia trachomatis* in women using PCR on urine specimen. *Bio Res* 2010;21:301-4.
7. Mahony JB, Luinstra KE, Sellors JW, Jang D, Chernesky MA. Comfirmatory polymerase chain reaction testing for *Chlamydia trachomatis* in first void urine from asymptomatic and symptomatic men. *J Clin Microbiol* 1992;30:2241-45.
8. Daxboeck F, Zitta S, Stadler M, Iro E, Krause R. *Mycoplasma hominis* and *ureaplasma urealyticum* in patients with sterile pyuria. *J Infec* 2005; 51:54-8.
9. Waites KB, Katz B and Schelonka RL. Mycoplasmas and Ureaplasmas as neonatal pathogens. *Clin Microbiol Rev* 2005;18:757-89.
10. Gdoura R, Kchaou W, Keskes LA, Charkroun N, Sellemi A, Znazen A et al. Assessment of *Chlamydia trachomatis*, *Ureaplasma urealyticum*, *Ureaplasma parvum*, *Mycoplasma hominis*, and *Mycoplasma genitalium* in Semen and First Void Urine Specimens of Asymptomatic Male Partners of Infertile Couples. *J Androl* 2008; 29: 199-205.
11. Hislop J, Quayyum Z, Flett G, Boachie C, Fraser C and Mowatt G. Systematic review of the clinical effectiveness and cost-effectiveness of rapid point-of-care tests for the detection of genital chlamydia infection in women and men. *H Tech Assess* 2010; 14:14290.
12. Jaschek G, Gaydos CA, Welsh LE, Quinn T. Direct detection of *Chlamydia trachomatis* in urine specimens from symptomatic and asymptomatic men by using a rapid polymerase chain reaction assay. *J Clin Microbiol* 1993;31: 1209-12.
13. Chernesky MA, Mahoney JB, Castriciano S, Mores M, Stewart IO, Landis SF et al. Detection of *Chlamydia trachomatis* antigens by enzyme immuno assay and immunofluorescence in genital specimen from symptomatic and asymptomatic man and women. *J Infec Dis* 1986;154:141-8.
14. Jensen IP. A comparison of urine sample to urethral swab for detection of *Chlamydia trachomatis* in asymptomatic young men using two enzyme immunoassays. *Sex Trans Dis* 1992 ;19:165-9.
15. Basarab A, Browning D, Lanham S, O'Connell S. Pilot study to assess the presence of *Chlamydia trachomatis* in urine from 18-30 year old males using EIA/IF and PCR. *J Fam Plan and Rep HC* 2002; 28: 36-7.
16. Singh V, Salhan S, Das BC, Mittal A. Predominance of *Chlamydia trachomatis* serovars associated with urogenital infections in females in New Delhi, India. *Ind J Clin Microbiol* 2003; 41: 2700-2.
17. Mohamed N, Sharaf T. Evaluation of different techniques in diagnosing chlamydial endocervical infection among Egyptian females. *The Egypt J Hosp Med* 2001;2:138-47.
18. Kim SJ, Lee DS, Lee SJ. The prevalence and clinical significance of urethritis and cervicitis in asymptomatic people by use of multiplex polymerase chain reaction. *Kor J Uro* 2011; 52: 703-8.

Prevalence of Multidrug resistant Tuberculosis (MDR- TB) in Sylhet Metropolitan City

Begum Lutfun Naher¹. Md. Badrul Haque Rukan². Md. Mahbubul Alam³.
Md. Burhan Uddin⁴. Akter Hossain⁵

Abstract

A retrospective observational study was conducted among 2467 diagnosed tuberculosis (both pulmonary & extrapulmonary) patients with or without Multidrug-resistant Tuberculosis (MDR-TB) including both old & new cases in the Sylhet metropolitan city. The patients were selected irrespective of both age & sex during the period of June 2014- March 2016. MDR-TB detection programme started in sylhet chest disease hospital (CDC), only centre of Sylhet for detect MDR-TB. Patient's details were collected with the help of BRAC from their record book, who collected data from 5 Directly Observed Treatment (DOTs) corner in Metropolitan city. Patients usually admitted five DOTs corners and then referred chest disease hospital for detection of MDR-TB cases by Gene Expert. Here data represent by percentages. Among the MDR-TB suspected case 0.48% of MDR-TB were diagnosed as MDR-TB which is less than national study result.

[OMTAJ 2015; 14(1)]

Introduction

Tuberculosis (TB) is an infectious disease caused by *Mycobacterium tuberculosis*. It generally responds to anti-TB drugs. In some persons, these first line drugs are not effective as the causative organism becomes resistant to INH & Rifampicin. This condition is known as Multidrug-resistant Tuberculosis (MDR-TB).

Globally, an estimated 450,000 developed MDR-TB & at least 170,000 deaths were caused by the disease in 2012. Bangladesh ranks 10th among 27 high MDR-TB burden countries. The emergence of MDR-TB has become a major threat for TB control in Bangladesh.

According to world health organization (WHO) estimate, there are 10,000 MDR-TB patients in Bangladesh.

TB is an infectious disease that can affect any organ in the body, although the vast majority of cases involve only the lungs. Transmission of the disease occurs from person to person via the airborne route.

Beginning in the late 1940s antibiotics were developed that were effective in curing TB. However, mutant strains of TB that were resistant to one or more of these antibiotics began to be identified as early as 1956. Since then, the evolution of antibiotic resistance among TB has been growing problem that is now a major public health threat. Multiple drug resistant TB (MDR-TB) refers to strains of TB that are resistant to at least INH & Rifampicin.

TB is second only to HIV/AIDS as the greatest killer worldwide due to a single infectious agent. In 2012, 8.6 million people fell ill with TB & 1.3 million died of TB². MDR-TB is a major challenge to worldwide TB control. In 2013 a total of 55% the estimated MDR-TB were under detected & 29% of the diagnosed pts were not on treatment.

Bangladesh is one of the high burden countries for TB & has also been listed on the 27 high burden countries for MDR-TB by the WHO. Due to overall high TB burden in Bangladesh the proportion of patients with MDR-TB (1.4% & 29%, among the new & previously treated TB patients, respectively), poses a significant challenge for the national tuberculosis control programme. Several studies have reported that delay in TB treatment contributed to development of MDR-TB. TB remains a major public health problem in Bangladesh. The country rank 6th among 22 highest burden TB countries in the world. It is estimated that

1. Associate Professor, Department of Pharmacology, Sylhet MAG Osmani Medical College, Sylhet.
2. Assistant Professor, Department of Pediatrics, Park View Medical College Hospital, Sylhet.
3. Junior Consultant (Child), Chest Disease Hospital, Sylhet.
4. Associate Professor, Department of Pharmacology, Jalalabad Ragib Rabya Medical College.
5. Microbiologist, Sylhet RTRL.

about 70,000 people die every year due to TB. In 2009, 160,735 TB cases were notified to national TB control programme (NTP). Case notification rate of all forms of TB is low at 47%. MDR-TB is an emerging threat of Bangladesh. According to WHO estimates MDR-TB rate among all newly diagnosed cases is estimated at 2.2% & among previously treated cases at 15%⁶. TB is as old as the mankind⁷. TB is the most common cause of death due to a single infectious agents worldwide in adults⁸. According to recent estimation, one third of the human population (about 1.86 billion people) was infected with *Mycobacterium tuberculosis* worldwide in 1997⁹. New infections occur in about 1% of the population each year. In 2014 there were 9.6 million cases of active TB which resulted 1.5 million deaths. More than 95% of deaths occurred in developing countries. The world health declared¹⁰ TB a 'global health emergency' in 1993. Despite success TB control programme, MDR-TB in Bangladesh is increasing & currently MDR-TB rate is 3.6% in new case & 19% in retreatment case¹¹. MDR-TB is an increasing global problem, with most cases arising from patient non-compliance with prescribed treatment schedule.

Material and Methods

This retrospective observational study was conducted among 2467 diagnosed tuberculosis patients, including both pulmonary & extrapulmonary with or without MDR-TB irrespective of both age & sex during the period of June-2014 to March 2016.

Data collected from record book of BRAC office Sylhet, situated in Uposhohor who collected & preserved data of Tuberculosis patients in Sylhet division. The present study carried out in only Sylhet Metropolitan city. Patient's particulars were collected from 5 DOTs corner. For better service to TB patients metropolitan city divided into 5 area called DOTs corner.

5 DOTs corner named as-

1. Sylhet MAG Osmani medical college hospital DOTs corner
2. Uposhohor DOTs corner
3. Jalalabad Ragib-Rabeya medical college hospital DOTs corner
4. North-East medical college hospital DOTs corner
5. Prison DOTs corner.

From each DOTs corner presumptive MDR-TB patients referred to Sylhet chest disease hospital (CDC) for diagnosis MDR-TB by Gene Xpert machine. By Gene Xpert machine diagnosis only Rifampicin resistance. However, patients who are resistant to Rifampicin are generally also resistant to INH¹².

Results

Out of 438636 population in Sylhet Metropolitan city 184000 patients were presumptively suffering from tuberculosis but only 2467 patients diagnosed as tuberculosis by AFB test.

During study period a total of 2467 patients diagnosed as TB patients were included. Out of 2467 patients sputum sample from 933(37.82%) were positive on culture & 794(32.18%) negative on culture. Relapse cases were 110(4.46%), MDR-TB was 12(.48%). Of them 1406(57%) were male patients and 1065(43%) were female. New infections were diagnosed in 2467 (56%) patients.

Month-wise data collected from five DOTs centre were presented in Table-I to Table IV.

Table-1

Osmani medical college DOTs corner

Phases	Preumptive case	Case Identified	AFB (+)ve	AFB (-)ve	Relapse Case	M	F
July/14-Sep/14	1105	100	35	25	02	64	36
Oct/14-Dec/14	1115	101	36	27	03	52	49
Jan/15-Mar/15	1239	95	35	28	03	47	48
Apr/15-Jun/15	1150	105	33	30	07	55	50
Jul/15-Sep/15	1124	102	41	32	05	60	42
Oct/15-Dec/15	1252	104	36	44	03	50	54
Jan/16-Mar/16	1369	113	37	48	04	68	45
Total	8354	720	253	234	27	396	324

TableII

Uposhohar DOTs corner

Phases	Preumptive case	Case Identified	AFB (+)ve	AFB (-)ve	Relapse Case	M	F
July/14-Sep/14	573	96	41	20	02	56	40
Oct/14-Dec/14	573	91	37	17	05	54	37
Jan/15-Mar/15	337	78	30	19	06	46	32
Apr/15-Jun/15	478	88	27	21	03	52	36
Jul/15-Sep/15	549	98	34	34	04	57	41
Oct/15-Dec/15	566	93	36	27	03	58	35
Jan/16-Mar/16	565	104	41	41	07	63	41
Total	3641	648	246	179	30	386	262

Table-III**J.R.R. Medical College DOTS corner**

Phases	Presumptive case	Case Identified	AFB (+)ve	AFB (-)ve	Relapse Case	M	F
July/14-Sep/14	503	83	36	21	03	54	29
Oct/14-Dec/14	551	92	36	24	07	43	49
Jan/15-Mar/15	573	93	33	30	04	45	48
Apr/15-Jun/15	667	107	44	34	05	67	40
Jul/15-Sep/15	552	108	38	46	04	65	43
Oct/15-Dec/15	694	122	45	50	04	74	48
Jan/16-Mar/16	695	116	43	41	07	63	53
Total	4235	721	275	246	34	411	310

Table IV**North-East Medical College DOTS corner**

Phases	Presumptive case	Case Identified	AFB (+)ve	AFB (-)ve	Relapse Case	M	F
July/14-Sep/14	166	28	11	09	03	17	11
Oct/14-Dec/14	186	31	16	05	02	13	18
Jan/15-Mar/15	264	47	23	09	01	30	17
Apr/15-Jun/15	295	55	20	18	05	25	30
Jul/15-Sep/15	279	58	22	24	04	36	22
Oct/15-Dec/15	352	66	24	32	02	34	32
Jan/16-Mar/16	388	66	24	34	02	32	34
Total	1930	351	140	131	19	187	168

Table-V**Prison DOTS corner**

Phases	Presumptive case	Case Identified	AFB (+)ve	AFB (-)ve	Relapse Case	M	F
July/14-Sep/14	26	05	05			05	
Oct/14-Dec/14	28	02				02	
Jan/15-Mar/15	34	05	03	01		05	
Apr/15-Jun/15	36	03	03			03	
Jul/15-Sep/15	37	05	04	01		04	01
Oct/15-Dec/15	45	04	04			04	
Jan/16-Mar/16	43	03		02		03	
Total	249	27	19	04		26	01

Table VI**Summary of the data collected from five DOTS corner:**

Dots Corner	Presumptive case	Total case	AFB (+)ve	AFB (-)ve	Relapse Case	MDR-TB	M	F
Osmani Medical College Hospital	8354	720	253	234	27	5	396	324
Upashar	3641	648	246	179	30	3	386	262
North-East Medical College	1930	351	140	131	19	2	187	168
J.R.R. Medical College	4235	721	275	246	34	2	411	310
Prison	249	27	19	04			26	01
Total	184000	2467	933	794	110	12	1406	1065

Discussion

TB is a disease of global importance. The emergence of resistance to anti TB drugs & particularly MDR-TB has been identified as one of the major obstacles to global TB control¹³ WHO estimated that over 500,000 cases of MDR-TB occur annually worldwide & two million die of TB each year.¹⁴ The present study was undertaken to investigate the prevalence of MDR-TB in Sylhet Metropolitan city.

TB is a major health problem in Bangladesh. In 2008, The WHO ranked BD sixth among the world's 22 high burden countries & 9th among 25 high priority MDR & XDR TB countries. In this study MDR-TB was detected. 48% that was not consistent with Zaman et al. (2005)¹⁵ a community based survey in a rural & urban set up observed 5.5% MDR-TB.

A recent estimation made by WHO showed approximately 2.2% of new & 14.7% of previously treated pts suffer from MDR-TB in BD¹⁶. At present study observed sputum positive 37.82%, sputum negative 32.18%, male were 57%, female were 43% not in agreement with the observation of Sayera Banu et al (2012)¹⁷ who observed sputum positive 74%. sputum negative 26%, male were 75%, female were 25%.

One third of the world population has been infected with *Mycobacterium tuberculosis* with new infection occurring in about 1% of the population each year¹⁰ our observation (0.56) not consistent with this. About 80% of people in many Asian & African countries test positive in tuberculin test while only 5-10% of the US

population test positive not agreement in present study observed 37.82% test positive.

In this study MDR-TB prevalence is 0.48% compared to Sharma SK et al (2011) where out of 177 cases 12(1.33) cases of MDR-TB were detected, which also did not matched with this study.

Antituberculosis drug resistance surveillance between 1994 & 1997 documented the prevalence of resistance in 35 countries. Resistance to antituberculosis drug was found in all 35 countries surveyed suggesting that it is a global problem². In the US in 2000 the rate of primary MDR-TB was 1% of all cases of TB nationally.² This figure is closer to the present study where MDR-TB was detected in 0.48%.

Globally about 3% of all newly diagnosed patients have MDR-TB. The present study was not in agreement with S K Sharma et al(2004)²². 5% of all TB cases across the globe in 2013 were estimated to be MDR-TB cases, including 3.5% of newly diagnosed TB cases & 20.5% of previously treated TB cases.²

As of 2013, 3.7% of new tuberculosis cases have MDR-TB. Levels are much higher in those previously treated for tuberculosis-about 20%. WHO estimates that there were about 0.5 million new MDR-TB cases in the world in 2011.²

The extent of anti-TB drug resistance in Bangladesh is not well validated. No nationwide drug resistance survey was conducted in Bangladesh. However, limited survey conducted by the Damien Foundation & the International Centre for Diarrheal Diseases Research, Bangladesh (ICDDR, B) show an overall MDR-TB prevalence rate of 2%-5.5%. The MDR-TB rate varied between 5.6% & 15.4% in person who had received treatment for one month or more. WHO estimates 2.1% of MDR-TB among new TB cases & 28% among previously treated TB cases in the country.² MDR-TB is an increasing global problem.

In conclusion, present study found relatively low prevalence of MDR-TB. Data of one Metropolitan city may not reflect national prevalence rate. Many patients attending the private chambers or clinics were probably not covered by DOTs & BRAC report. Proper diagnosis may be one of the major obstacles for reliable data. Data on the prevalence of drug resistant are scarce & very limited. Proper counselling of TB patients & attention towards completion of the anti-TB treatment are needed. MDR-TB patients require to be detected early and referred to appropriate centers for specialized treatment and care and counselling. Sometimes due to delayed diagnosis & treatment initiation MDR-TB was

not recorded. Some patients who consult private practitioners may remain out of record. Those who received treatment from private practitioners should inform & be included in the data base. Delayed diagnosis and referred may hamper enlistment. All MDR-TB cases must follow proper reporting system specially those received treatment outside DOTs corner.

References

1. Zahid Al Alam ZA. Tackling drug resistant TB in Bangladesh. www.the-daily-star.com. 2015
2. Blackman C, Browning R, Kogut D, Young H. Multiple drug resistant tuberculosis. Boston university. Available at: [Sphweb.bumc.bu.edu/.../MDR-TB/ind](http://sphweb.bumc.bu.edu/.../MDR-TB/ind). (Accessed on 16 Dec 2013)
3. World Health Organization. Multidrug and extensively drug-resistant TB (MDR-TB). Global Report on Surveillance and Response. Geneva; 2010. Available at: <http://apps.who.int/iris/bitstream/10665/137094/1/9789241564809eng.pdf> (Accessed on 16 Dec 2013)
4. World Health Organization. Global Tuberculosis Report Geneva; 2014. Available at: <http://apps.who.int/iris/bitstream/10665/44286/1/9789241599191eng.pdf?ua=1&ua=1> (Accessed on 15 March, 2014)
5. He GX, WANG HY, Borgdorff MW, Van Soolingen D, Van der Werf MJ, Liu ZM, et al. Multidrug-resistant tuberculosis, People's Republic of China, 2007-2009. *Emerg Infect Dis* 2011;17:1831-8.
6. TBmhealth Gene xpert reporting. MDR-TB is an emerging threat in Bangladesh. TB care-II Project overview. Available at: www.tb-care.org/cp-bangladesh (Accessed on 15 Oct/2014)
7. Mohan A, Sarma SK. Epidemiology. In: Sharma SK, Mohan A, editors. Tuberculosis. New Delhi: Jaypee Brothers Medical Publishers. 2001; 14-29
8. World Health Organization. Tuberculosis fact sheet. Available at: <http://www.who.int/gtb/publications/factsheet/index.htm>. (Accessed on 1 July 2013)
9. Dye C, Scheele S, Dolin P, Pathania V, Ravignione MC. Consensus statement. Global burden of tuberculosis: estimated incidence, prevalence, and mortality by country. WHO Global Surveillance and Monitoring project. *JAMA* 1999; 282: 677-86.
10. Tuberculosis Fact sheet N 104. Geneva, WHO. 2014.

11. MS Flora,MN Amin,MR Karim,SAfroz,S Islam,A Alam,M Hussain.Risk factors of multi-drug-resistant tuberculosis in Bangladeshi population:a case control study. *BMRC Bull* 2013;39:34-41
12. National TB control programme DGOHS Bangladesh,First Bangladesh national tuberculosis drug resistance survey 2010-2011,Dhaka.2013.
13. Operational manual for the management of MDR-TB national tuberculosis control programme, Bangladesh 1st ed.2009.
14. Anti tuberculosis drug resistance in the world fourth global report.The WHO/IUATLD global project on antituberculosis drug resistance surveillance 2002-2007:WHO/HTM/TB/2008394. 2008.
15. Zaman K,Rahim Z,Ynus M,Arifeens S, Baqui A,et al. drug resistance mycobacterium Tuberculosis in selected urban & rural areas in Bangladesh. *Scand J Infect Dis* 2005;37:21-26
16. Multidrug & extensively drug resistant TB (M/XDR-TB), global report on surveillance& response,WHO/ HTM/ TB /20103.2010.
17. Banu S, Mahmud AM, Rahman MT, Hossain A, Uddin MKM, Ahmed T etal. Multidrug Resistant Tuberculosis in admitted patients at a Tertiary Referral Hospital of Bnagladesh, *PLoS ONE*: 2012; 7:e40545.doi.10.1371/journal.pone.0040545
18. Kumar V, AbbasdAK, Fausto N, Mutchell RN Robbins Basic Pathology (8thed.) Newyork, Saunders Elsevier.2007;pp. 516-522.
19. Sharma SK et al. prevalence of MDR-TB among newly diagnosed cases of sputum positive pulmonary tuberculosis.*Indian J Med Res* 2011 ; 133:308-11
20. Anti-tuberculosis drug resistance in the world. The WHO/IUATLD Global Project on Anti-tuberculosis Drug Resistance Surveillance: WHO/TB/97, 229. Geneva: World Health Organization:1997
21. Longo DL, Fauci ASet al. Tuberculosis. In: Harrison's Principles of internal Medicine. 18thed. New York: McGraw Hill. 2012. Pp. 1112-20
22. Sharma SK & Mohan A. Multidrug resistant tuberculosis. *Indian J Med Res* 2004; 120:354-76
23. Diagnosis and notification of multidrug-resistant TB (PDF). WHO MDR-TB Factsheet.Retrieved 9 May 2015.
24. WHO. Multidrug-resistant tuberculosis (MDR-TB) 2013 Update. Geneva, World Health Organization. 2013.
25. WHO. Global tuberculosis control.WHO Report 2011.

Sonouroethrography in the evaluation of male anterior urethral abnormalities

Mostaque Ahmed Bhuiyan¹, Shafiqul Islam², Swajal Chandra Das¹, NK Paul³.

Abstract

Urethral abnormalities are common problem affecting male anterior urethra. Retrograde urethrography (RGU) and micturating urethrography (MCU) has been the standard imaging study for evaluation of male anterior urethra which involves the use of radiation and contrast medium for visualized anatomy and gives limited information about periurethral structures. Sonourethrography (SUG) could diagnose anterior urethral stricture, measure its length, periurethral structures and extends of spongiofibrosis.

Purpose of the study was to determine the usefulness and to explore the abnormalities of male anterior urethra with the help of high resolution ultrasound.

Sixty male patients aged 10 to 70 years with a clinical diagnosis of lower urinary tract obstruction underwent sonourethrography between July 2013 to November 2014 in the Department of Radiology & Imaging, MAG Osmani medical college hospital, Sylhet.

Out of total 60 patients, SUG ruled out urethral lesion in 6 patients and 54 had definite sign of urethral abnormalities. Total 108 findings were demonstrated. 64 strictures were found in 54 patients. Single stricture was found in 44 patients and multiple strictures in 10 patients. Out of 54 patients 32 patients had short segment, 12 patients had long segment and 10 patients had multiple strictures. The most commonly involved site was bulbar urethra 29 (65.9%) followed by penile urethra in 15 patients (34.1%). Out of 54 patients diagnosed as urethral stricture disease, sonography revealed periurethral fibrosis in 34 patients (62%). Urethritis was seen in 18 patients, periurethral fistula in 11

patients, urethral diverticula in 6 patients, calculi in 3 patients and periurethral abscess in 2 patients. 6 patients had urethral injury well delineated by sonourethrography along with associated periurethral findings. Among these 2 patients had disruption of bulbar urethra associated with periurethral haematoma and collection.

[OMTAJ 2015; 14(1)]

Introduction

Sonourethrography is the visualization of urethra on ultrasound after instillation of normal saline into the urethra. Urethral pathologies especially anterior urethral strictures represent a significant part of workload of the urologist.¹ Thoughtful and satisfactory preoperative evaluation remains important achieving reasonable outcome. The appropriate choice of treatment modality for anterior urethral pathology depends on preoperative imaging and endoscopic techniques. The gold standard imaging technique is the Retrograde urethrography (RGU) and micturating cystourethrography (MCUG).² Both the techniques give two dimensional images, do not detect spongiofibrosis and gives limited information about periurethral structures. They also expose patients to ionizing radiation.

In 1988 McAninch et al.³ reported a new technique for imaging the male anterior urethra with high-resolution ultrasound (sonourethrography). The initial technique involved the use of a 5 MHz linear array transducer applied to the dorsal surface of the penis. Images were obtained during retrograde instillation of normal saline. In the last decades, the evaluation of anterior urethral abnormalities with SUG has made significant advances. Ultrasound of the anterior urethra offer a three dimensional study without exposure to radiation, it also accurately defines the length of strictures and detect spongiofibrosis.⁴ The use of sonourethrography in diagnosing patients with anterior urethral abnormalities is a valuable pre-operative evaluation technique. This study was therefore undertaken to explore the uses of high-resolution ultrasound in evaluating abnormalities of the male anterior urethra.

1. Assistant professor, Dept of Radiology & Imaging, Sylhet MAG Osmani Medical College.
2. Assistant professor, Dept of urology, Sylhet MAG Osmani Medical College.
3. Associate professor, Dept of Radiology & Imaging. Sylhet MAG Osmani Medical College.

Material and Methods

This study was carried out between July 2013 to November 2014 in the Department of Radiology & Imaging, Sylhet MAG Osmani Medical College Hospital, Sylhet. A total of 60 male patients aged 10 to 70 years with urinary complaints suggestive of urethral pathology were selected. Patients with symptoms suggestive of acute urethritis and recent instrumentation procedure were excluded.

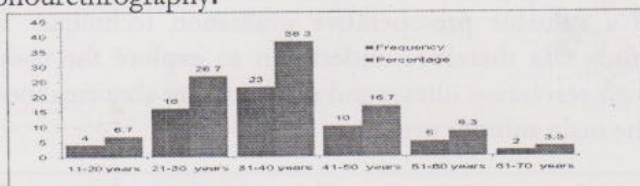
Sonourethrography was performed using I) GE LOGIC P5 and II) GE LOGIC 3 EXPERT ultrasound and color Doppler equipment with 5, 7.5 and 10 MHz transducers. With the patient in supine position the glans was disinfected and a Foley catheter was introduced such a way that the bulb laid in the fossa navicularis. The bulb was distended gently using 2 ml normal saline. About 20-30 ml normal saline was introduced maintaining continuous pressure. The penis was then cranially extended over the lower abdomen and ultrasonic gel was applied liberally to the ventral surface of the penis. Then penile urethra was visualized up to the penoscrotal junction with multiple longitudinal and transverse scans by placing the transducer on the ventral penile surface. Subsequently, the transducer was repositioned to visualize the proximal penile and distal bulbar urethra trans-scrotally and trans-perineally. The stricture length and diameter were determined using electronic caliper measurements. The duration of the procedure varied from 10– 20 min. panoramic reconstruction of the sonographic images was done for better understanding of urethral pathologies.

The informed verbal consent was taken from the patients and their confidentiality was maintained. The study protocol was reviewed by the Ethical Committee of Sylhet MAG Osmani Medical College.

Results

A total of 60 patients with symptoms of lower urinary tract obstruction underwent sonourethrography. Age of the patient ranged from 11 to 68 years with the mean age of 35.68 (SD 11.91) years. Maximum number of patients belonged to age group 31-40 years (Fig. 1).

Fig 1. Age distribution of the patients for sonourethrography.



The SUG ruled out urethral lesion in 6 patients (Fig. 2). Remaining 54 patients had definite signs of urethral abnormalities. A total 108 findings were documented on sonourethrography in 54 patients (Fig. 3).

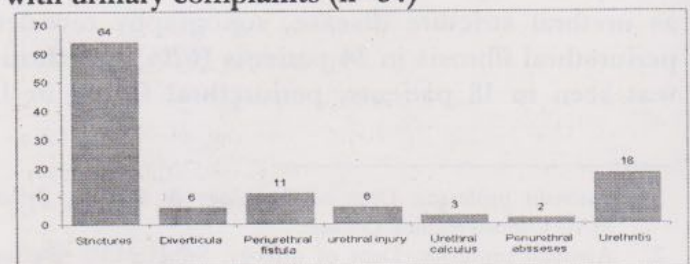
Fig2. Normal sonourethrography appearance of anterior urethra longitudinal view



Most common diagnosis was stricture urethra. Total 64 strictures were demonstrated in 54 patients. Out of 54 patients single stricture was found in 44 patients and multiple or complex strictures in 10 patients. Out of 54 patients, 32 patients had short segment stricture, 12 patients had long segment stricture and 10 patients had complex/multiple strictures. The most commonly involved site was bulbar urethra 29 patients (65.9%) followed by penile urethral 15 patients (34.1%) in cases of single stricture (Table-I). Out of 54 patients diagnosed as urethral stricture disease, sonography revealed periurethral fibrosis in 34 patients, urethritis in 18 patients, periurethral fistula in 11 patients, urethral diverticula in 6 patients, calculi in 3 patients and periurethral abscess in 2 patients. 6 patients had urethral injury well delineated by SUG associated with periurethral findings. Among these two patients had disruption of bulbar urethra associated with periurethral haematoma and collection.

During SUG, pain was experienced by one patient during inflation of the Foley's bulb in the fossa navicularis and bleeding per urethra in one patient.

Fig 3. Sonourethrography Findings in the patients with urinary complaints (n=54)



Number of total findings far exceeded the number of patients as few patients had multiple findings.

Table 1. Distribution of single urethral stricture according to length and location (n=44)

Strictures	Short segment	Long segment
Penile	8	7
Bulbar	24	5
Total	32	12

Discussion

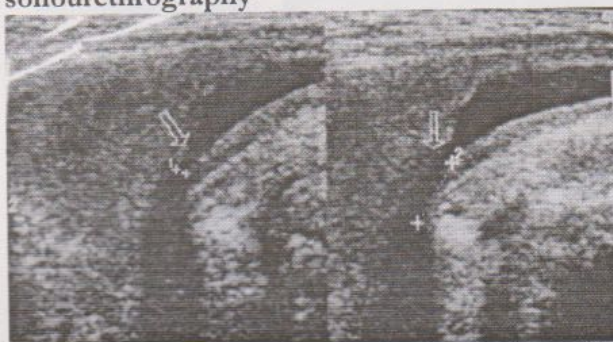
There are considerable numbers of patients with urethral problems and many of them required surgical procedures. Thus preoperative investigation should accurately demonstrate and locate the urethral pathology is a prerequisite for choosing surgical procedures for successful outcome. If such an investigation is radiation free and able to suggest the mode of treatment, then it virtually becomes indispensable. SUG is one such investigation. Although radiographic retrograde urethrography (RGU) has long been the gold standard for imaging the anterior urethra, inherent limitation persist.⁵ Limitation of RGU include variation in the appearance of strictures with positions of the patients and degree of stretch of the penis during the study, limited information about periurethral fibrosis and it has hazards of radiation expose to testes.^{6,7} Contrast material may extravasate into other areas of penis. In addition venous and lymphatic intravasation may occur.^{7,8} McAninch et al.³ began using ultrasonography to image the male urethra in the mid 1980s at San Francisco General Hospital to evaluate complex urethral strictures more precisely. Since then it has been routinely used urethral ultrasound to evaluate anterior urethral abnormalities.

In this study of 60 patients, most were belonged to age group of 31-40 years with the mean age of 35.68 year. Similar age distribution of patients with anterior urethral pathologies in several studies.⁸⁻¹²

During SUG the urethra distended with normal saline and appeared as homogenous echo-free band, 8-10 mm in diameter. Below the urethra an echogenic band was visualized, which was produced by dorsal acoustic enhancement and reflection from tunica albuginea. Strictures were described as segments of reduced distensibility on injection of saline. Total 64 strictures were demonstrated in 54 patients. Out of 54 patients single stricture was found in 44 patients and multiple or complex strictures in 10 patients. The accurate estimation of length of the stricture is important, as it is one of the factors that determine the selection of a suitable operative procedure. Earlier investigators, using standard radiographic imaging alone, proposed that only

strictures 1 cm or less be selected for excision therapy.¹³ Since sonographic measurements are often longer than the actual length, new ultrasonic criteria proposed indicated resection and end-to-end anastomosis for adult bulbar stricture measuring up to 25 mm. We categorized stricture length as short strictures less than 25 mm in length and long strictures, more than 25 mm in length. This classification is modification from the of classification proposed by Chiou et al.^{12,14} and Morey and Mc Aninch.⁴

Out of 54 patients, 32 patients had short segment stricture, 12 patients had long segment stricture and rest 10 patients had complex/multiple strictures. Involvement of the stricture were 29 (65.9%) in bulbar and 15 patients (34.1%) in penile urethra.

Fig-4: Patient with long bulbar urethral stricture on sonourethrography

Periurethral fibrosis was identified as regions of greater echogenicity in corpus spongiosum, and was classified as per the classification proposed by Chiou et al.^{12,14} Minimal spongiosal tissue involvement demonstrates either no identifiable spongy tissue involvement or a minimal abnormality. Moderate spongiosal tissue involvement shows definite areas of abnormal tissue beneath the urethral surface with sonographically normal tissue in the periphery. Extensive spongiosal tissue involvement consists of a near full-thickness involvement of the corpus spongiosum.

Out of 54 patients diagnosed with urethral stricture disease, sonography revealed periurethral fibrosis in 34 patients. Mild periurethral fibrosis was seen in 7 patients, moderate in 13 patients and extensive in 14 patients. The findings in present study were also similar with Nash et al.¹⁵ Periurethral fibrosis (spongiofibrosis) is a critical determinant of appropriate therapy and ultimate prognosis.¹⁶ Excessive fibrosis is said to be responsible for high recurrence rates.¹⁷

Other findings such as periurethral haematoma or collection in post trauma patients and periurethral abscesses or masses were well seen on SUG.

Sonourethrography definitely have upper hand in evaluation of periurethral pathologies.¹



Fig-5: Long penile urethral stricture with false tract on sonourethrography



Fig-6: Post traumatic disruption at bulbo-membranous junction with periurethral hematoma and false lumen as seen on sonourethrography.

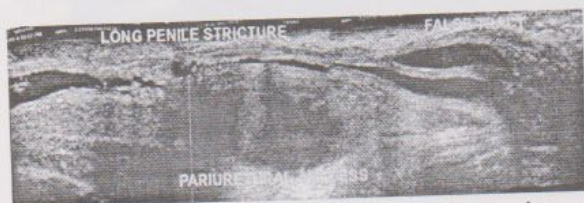


Fig-7: Long penile urethral stricture with periurethral abscess and false tract on sonourethrography.

In 3 patients sonography demonstrated luminal filling defects, diagnosed as urethral calculus (Fig-6). Bearcroft and Berman,⁸ studied 24 patients, 11 were normal, 5 patients demonstrated luminal filling defects: a mucosal tag, a flap, a urethral papilloma and two cases of debris within a diverticulum. Gluck et al.⁹ successfully demonstrated urethral strictures utilizing sonography. Although filling defect was not evaluated in their study, an intraluminal blood clot was well seen in one of their illustrations of straddle injury. Most of the previous studies underestimated these findings.

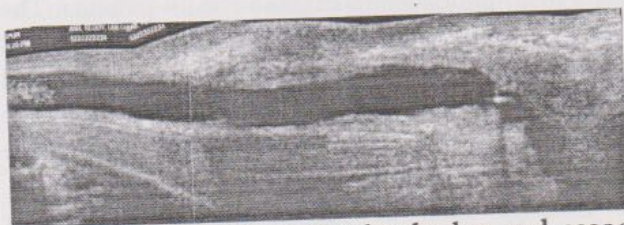


Fig-8: Patient with urethral calculus and associated short segment bulbar urethral stricture.

Total 6 cases of diverticula were diagnosed. Out of these, two wide-mouthed diverticula were post-traumatic. One shallow penile urethral diverticulum was seen in post-traumatic patient with bulbar urethral disruption. A case of syringocele was considered under the heading of diverticula as it has tendency to open in the urethral lumen to give appearance of diverticulum. Syringocele or cystic dilatation of cowper's gland duct is a lesion of uncertain origin even if it is probably congenital since it is found in young patients with negative clinical history. Syringocele can be easily diagnosed by SUG using transperineal scanning which shows a thin septation in the bulbar urethra dividing the distended lumen of the urethra in two parts. The one nearest the probe corresponds to the dilated cowper's duct (Fig-7).¹⁸

Bearcroft and Berman⁸ studied 24 patients: 3 cases demonstrated a wide mouth diverticula; however only two of these were seen on ultrasound. A shallow diverticulum was found in case of multiple strictures. Allan and Nurmi¹⁹ studied 16 male patients of which one patient showed diverticulum. Most of the previous studies underestimated these findings.



Fig-9: Patient with syringocele as appearing on sonourethrography with debris within.

Generalised mucosal irregularity was observed in 18 patients. Since most of these patients had history suggestive of chronic urethritis, they were presumed to be having urethritis (Fig-8).

Bearcroft and Berman,⁸ studied 24 patients of which 5 cases had generalized mucosal irregularity demonstrated on the sonogram. Most of the previous studies underestimated these findings.

Fig-10: Patient with penile urethral mucosal irregularity and bulbar stricture on sonourethrography.



Careful evaluation of the periurethral structures and proper transverse scanning is needed to demonstrate false passages on SUG. False passages were seen in 11 patients. 1 patient had urethrocuteaneous fistula, 4 patients had history of instrumentation for stricture and 3 patients had history of trauma. 3 patients had only false passage without any stricture and had history of trauma. Chiou et al.¹⁴ in their study, investigated 35 patients, eight patients had inadequate evaluation. Among 27 patients one patient had urethrocuteaneous fistula without stricture while another patient had false passage with stricture. Gupta et al.¹¹ were able to pick up 8 false tracts out of 10 on sonourethrography.

During SUG, pain was experienced by one patient during inflation of the Foley's bulb in the fossa navicularis and bleeding per urethra in one patient managed conservatively.

In conclusion, sonourethrography in experienced hand proves to be highly effective modality for diagnosis of anterior urethral strictures and periurethral fibrosis useful for determining the type of operative procedure. It is a simple portable, dynamic, multiplanar, easily available and cost effective technique for evaluating the male anterior urethra without radiation exposure.

References

1. Sandler CM. Questions and answers. *AJR Roentgenol* 1994; 163: 1263-7.
2. McAninch JW, Laing FC, Jeffrey RB Jr. Sonourethrography in the evaluation of urethral strictures: a preliminary report. *J Urol* 1988; 139: 294-7.
3. Pavlica P, Barozzi L, Menchi I. Imaging of male urethra. *Eur Radiol* 2003; 13: 1583-96.
4. Morey AF, McAninch JW. Ultrasound evaluation of the male urethra for assessment of urethral stricture. *J Clin Ultrasound* 1996; 24: 473-9.
5. Gupta N, Dubey D, Mandhani A, Srivastava A, Kapoor R, Kumar A. Urethral stricture assessment: a prospective study evaluation urethral ultrasonography and conventional radiological studies, *BJU international* 2006; 98: 149-53.
6. Mitterberger M, Christian G, Pinggera GM, Bartsch G, Strasser H, Pallwein L, et al. Gray scale and color Doppler sonography with extended field of view technique for the diagnostic evaluation of anterior urethral strictures. *J Urol* 2007; 177: 992-6.
7. Singh P, Choudhary S, Sundar E, Kumar S, Sahai A. A comparison of sonourethrography and retrograde urethrography in evaluation of anterior urethral strictures. *Clin Radiol* 2004; 59: 736-42.
8. Bearcroft PW, Berman LH. Sonography in the evaluation of the male anterior urethra. *Clin Radiol* 1994; 49: 621-6.
9. Gluck CD, Bundy AL, Fine C, Kevin RL, Jerome PR. Sonographic urethrogram: comparison to roentgenographic techniques in 22 patients. *J Urol* 1988; 140: 1404-8.
10. Gupta S, Majumdar B, Tiwari A, Gupta RK, Kumar A, Gujral RB. Sonourethrography in the evaluation of anterior urethral strictures: correlation with radiographic urethrography. *J Clin Ultrasound* 1993; 21: 231-9.
11. Samaiyar SS, Shukla RC, Dwivedi US. Role of sonourethrography in anterior urethral stricture. *Indian J Urol* 1999; 15: 146-51.
12. Chiou RK, Donovan JM, Anderson JC, Matamoros A Jr, Wobig RK, Taylor RJ. Color Doppler ultrasound assessment of urethral artery location: potential implication on visual internal urethrotomy. *J Urol* 1998; 159: 796-9.
13. Webster GD, Koefoot RB, Sihelnik SA. Urethroplasty management in 100 cases of urethral stricture: a rationale for procedure selection. *J Urol* 1985; 134: 892.
14. Chiou RK, Anderson JC, Tran T, Ptterson RH, Wobig R, Taylor RJ. Evaluation of urethral strictures and associated abnormalities using high-resolution and color Doppler ultrasound. *Urology* 1996; 47: 102-7.
15. Nash PA, McAninch JW, Bruce JE, Hanks DK. Sono-urethrography in the evaluation of anterior urethral strictures. *J Urol* 1995; 154: 72-6.
16. Jordan GH, Schlossberg SM, Devine CJ. Surgery of the penis and urethra. In: Walsh FC, Retik AB, Vaughan ED Jr, editors. *Campbell's Urology*. 7th ed. Philadelphia: WB Saunders. 1998; pp. 3318-94.
17. Merkle W, Wagner W. Sonography of the distal male urethra-a new diagnostic procedure for urethral strictures: results of a retrospective study. *J Urol* 1988; 140: 1409-11.
18. Merchant SA, Amonkar PP, Patil JA. Imperforate syringoceles of the bulbourethral duct: appearance on urethrography, sonography and CT. *Am J Roentgenol* 1997; 169: 823-4.
19. Alanen A, Nurmi M. Sonographic technique in diagnosis of urethral strictures in men. *Bildgebung* 1994; 61: 25-7.



The Termination Pattern of the Anterior Interventricular Artery in the Hearts of Bangladeshi people.

Md. Abdul Quddus¹, Zakia Sultana², Ayesha Akhter³

Abstract

This descriptive study was conducted to see the termination of the anterior interventricular artery. This study was carried out on 100 (One hundred) autopsied human hearts of different age groups from July 2005 to June 2006 in the Department of Anatomy, M.A.G Osmani Medical college, Sylhet. All the specimens were studied gross macroscopically by careful dissection. The artery was found to terminate before the apex in one case, at the apex in 14 (fourteen) cases and in the posterior interventricular groove in 85 cases. This study concluded that the termination of the anterior interventricular artery is variable.

[OMTAJ 2015; 14(1)]

Introduction

The anterior interventricular artery usually arises as a direct continuation of the left coronary artery^{1,2,3,4}. In some cases it may arise from the left posterior aortic sinus⁵. The artery appears on the sternocostal surface between the upper most part of the infundibulum and the left auricle. Then the artery passes down words over the sternocostal surface along the anterior interventricular groove. Some times deeply embedded on it and crossed by occasional bridges of myocardial tissue and by the great cardiac vein and its tributaries. From here its course and termination is variable. The artery may terminate either in the anterior interventricular groove before the apex or at the apex in about one third of cases or in the posterior interventricular groove after winding round the incisura apicis cordis. Here they usually anastomose with the posterior interventricular branch of the right

coronary artery at the junction of anterior one third and posterior two third of the groove^{2,3} or at the junction of anterior half & posterior half of the groove².

Knowledge regarding the termination of the anterior interventricular artery provides a quantity approach to the blood supply of the heart

Materials and Methods

The present study was performed on 100 (one hundred) human hearts of different age groups in both sexes of Bangladeshi people. The hearts with ascending aorta were collected from apparently fresh dead bodies that underwent medicolegal examinations from July 2005 to October 2005 in the morgue of the Department of forensic Medicine of M. A. G Osmani Medical College, Sylhet. The specimens were washed thoroughly with tap water and gently squeezed to remove the blood clots from the cavities of the hearts and from the lumina of the blood vessels as much as possible. Then the specimens were kept in formalin solution for fixation and preservation.

The formalin fixed specimens were kept in the tap water overnight to washout excess formalin to minimize the irritation of the eyes and nasal mucosa then the specimens were taken in a tray and the pericardium with the thymus, lymph nodes, nerves and other unwanted tissues were removed with the help of scissors and tooth dissecting forceps. The ascending aorta was exposed as per requirement.

The coronary arteries and the left circumflex branch of the left coronary artery were dissected as far as practicable, beginning from their origins. Fine scissors and fine plain dissecting forceps were used for the purpose. A self illuminating magnifying glass was used wherever magnification of the field was needed. Photographs were also taken as needed. The termination of the left circumflex artery was observed. Relevant ethical clearance was taken from the ethical review committee of Sylhet MAG Osmani Medical College.

1. Associate Professor of Anatomy, Jalalabad Ragib Rabeya Medical College, Sylhet
2. Professor, Dept. of Anatomy, Sylhet M.A.G. Osmani Medical College
3. Professor of Anatomy, Jalalabad Ragib Rabeya Medical College, Sylhet

Results

The anterior interventricular artery was found to arise from the division of left coronary artery in all cases. In 30 (thirty) cases, the artery was bridged by myocardium near the middle of their course in the anterior interventricular groove. The termination of the artery was variable 85 (85%) cases, the artery terminated in the posterior interventricular groove in 14 (14%) cases the artery terminated at the apex and in 1 (1%) case the artery terminated before reaching the apex.

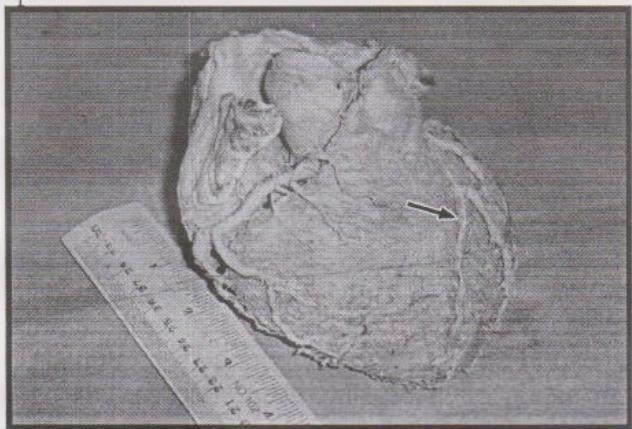


Figure 1: Anterior inter-ventricular artery, terminated at the apex.

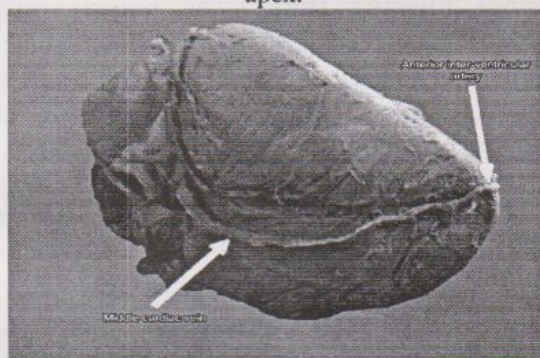


Figure 2: Anterior inter-ventricular artery terminated in the posterior interventricular groove and anastomosed with the middle cardiac vein

Table-I:

The termination of the Anterior Interventricular artery as recorded in different studies

Reference	Before the apex	At the apex	In the posterior interventricular groove
*Dragneff (1897)	---	---	100%
*Banchi (1904)	5%	79%	16%
*Gross (1921)	2%	38%	60%
*Craincianu (1922)	2%	20%	78%
James ¹ (1961)	---	17%	83%

*Paulin (1964)	7%	11%	82%
Baroldi and Scmazzone ⁸ (1967)	---	36.2%	63.8%
Bashir (1988) ⁶	---	20%	80%
Rahman (1989, cited by Sarker, 1996)	1.4%	27.14%	71.43%
Sarker ⁵ (1996)	---	15%	85%
present study (2006)	1%	14%	85%

* cited by Baroldi and Scmazzone (1967)

Discussion

The pattern of termination of the anterior interventricular artery differs from heart to heart. Regarding the variation in termination of the anterior interventricular artery. The finding of the other authors and those of the present study are compiled in the Table -I. It was found that in the most studies the majority of cases the artery terminated in the posterior interventricular groove which is in agreement with present study. However Branchi (1904) found the termination at the apex in the majority of cases. Dragneff (1897) found in the artery terminated at the apex in all cases (100%).

References

- James TN. Anatomy of the coronary arteries. New York: Paul B Hoeber, 1961: pp. 1-206.
- Williams PS, Warwick R, Dyson M, Banister HL, editors. Gray's anatomy. 37th ed. Edinburgh: Churchill Livingstone, 1989: pp. 696-734.
- Datta A.K. Essentials of human anatomy (Thorax and Abdomen): 5th ed. Kalkata: India, 2000:pp. 55-100.
- Snell RS. Clinical anatomy for medical students: 4th ed: Boston, Little Brown and Company, 1992: pp. 105-18
- Sarker MD. SA. "An anatomical study of the coronary arteries and the arch of the aorta in adult Bangladeshi people (M.Phil thesis)." Dhaka: University of Dhaka, 1996: 12-163.
- Bashir KE. A study of patterns and distributions of coronary arteries in adult postmortem human hearts in Bangladesh (Msc thesis). Dhaka: University of Dhaka, 1988: 1-51.
- Rahman ASMH. Anatomy of Coronary circulation and dimensions of adult postmortem human heart in Bangladesh (M Phil thesis). Dhaka: University of Dhaka, 1989.
- Baroldi G and Scmazzone G. Coronary circulation in the normal and pathological hearts. Office of the surgeon general, Washington: 1967, 1-96.

Joubert syndrome: Magnetic resonance imaging findings.

Swajal Chandra Das¹, Mostaque Ahmed Bhuiyan¹, N.K.Paul², MM.Ashiqur Rahman²
Poresh Chandra Singha¹, Maksudul Azim¹, Sudipta Gope³

Abstract

Joubert syndrome is a rare autosomal recessive disorder characterised with hypotonia, ataxia, mental & motor retardation, episodic tachypnea-apnea and oculomotor anomalies. Prognosis is poor in patients with hypotonia and severe growth retardation. Its characteristic imaging finding is hypoplasia of cerebellar vermis and 'molar tooth sign' in brainstem. Dandy walker malformation and Down syndrome are differential diagnosis. Clinical findings of Joubert syndrome are quite heterogeneous.¹ Thus determination of radiological findings are essential. In this paper, a boy of 17 years old who came to our centre for brain MRI with complains of ocular abnormalities, growth and mental retardation and diagnosed as Joubert Syndrome based on magnetic resonance imaging (MRI) findings.

[OMTAJ 2015; 14(1)]

Case report:

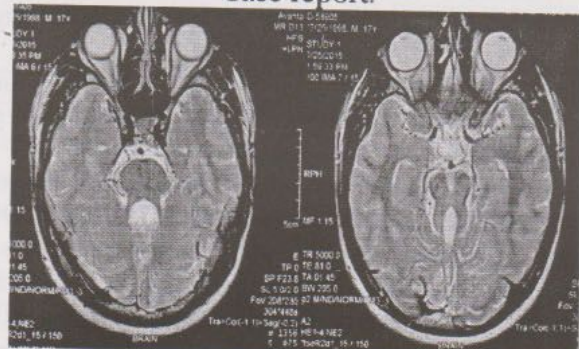


Figure 1: Molar tooth sign, elongated and thinned superior cerebellar peduncles.

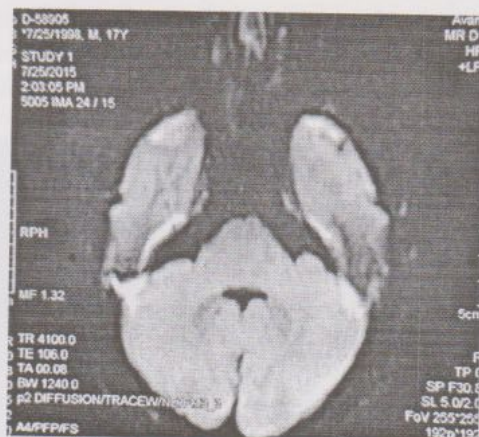


Figure 2: Batwing appearance of the 4th ventricle.

A 17 year old boy with motor and mental retardation, nystagmus and ataxic movements was referred to our department for a cranial magnetic resonance imaging study. The parents informed that he had abnormal eye movements and delayed neurologic development. An MRI study revealed vermian agenesis which resulted in median approach of the two cerebellar hemispheres. Isthmus was hypoplastic together with thickening and elongation of the superior cerebellar peduncles giving the "molar tooth" appearance (Figure 1). Also shape of the 4th ventricle had been changed into what described as "bat-wing" appearance (Figure 2). Size of the posterior fossa was normal with cerebrospinal fluid filling the ventricular space. All these MRI findings suggested "Joubert syndrome".

Discussion

Joubert syndrome first described by Marie Joubert and associates in 1969 in four siblings and one sporadic case that exhibited episodic hyperpnea, abnormal eye movements, ataxia and mental retardation with agenesis of cerebellar vermis². In the syndrome, midline structures of the brain-stem have both anatomic and functional defects. Neuropathological studies reveal agenesis of cerebellar

1. Assistant Professor of Radiology & Imaging, Sylhet MAG Osmani Medical College.
2. Associate Professor of Radiology & Imaging, Sylhet MAG Osmani Medical College.
3. Junior Consultant of Radiology & Imaging, Shahid Shamsuddin Ahmed Hospital, Sylhet.

vermis, malformations of several brainstem nuclei and dysplasia of structures at the ponto-mesencephalic junction. Extensive brainstem malformation could explain the oculomotor apraxia and hyperpnea; anomalies of the gracile nuclei and solitary tract are thought to contribute to the abnormal respiratory pattern³. The eye abnormalities observed in this disease include complete oculomotor apraxia in horizontal and vertical directions and ocular coloboma⁴. Hypotonia and mental retardation are nonvariable features of Joubert syndrome. Long-term follow-up of the children with this disease reveals majority having severe mental and motor developmental impairments⁵. The striking structural defects of Joubert syndrome in imaging studies are dysgenesis of the isthmus (part of the brainstem between pons and inferior colliculus) which is seen as elongation and thinning of ponto-mesencephalic junction, and deep interpeduncular fossa; thickening of superior cerebellar peduncles; hypoplasia of vermis characterized by incomplete lobulation and enlarged fourth ventricles; incomplete fusion of the halves of the vermis creating a sagittal cleft seen on coronal MRI planes. Combination of the first three features produces the characteristic "molar tooth sign" on axial MRI or CT planes⁶. Other syndromes like Arima, Senior-Loken and COACH which have posterior fossa malformations frequently lead to confusions in the diagnosis of Joubert syndrome. But molar tooth sign is virtually diagnostic of Joubert syndrome. Tectocerebellar dysplasia and Dandy-Walker

syndrome, other two situations which may be the source of confusion, also may accompany Joubert syndrome.⁷

References

1. Romano S, Boddaert N, Desguerre I, Hubert L, Salomon R. Molar tooth sign and superior vermis dysplasia: a radiological, clinical and genetic study. *Neuropediatrics* 2006;37:42-5.
2. Joubert M, Eisenring JJ, Robb JP, Andermann F. Familial agenesis of the cerebellar vermis. A syndrome of episodic hyperpnea, abnormal eye movements, ataxia and retardation. *Neurol* 1969;19: 813-25.
3. Yachnis AT, Rorke LB. Neuropathology of Joubert syndrome. *J Child Neurol* 1999;14: 655-9.
4. Tusa RJ, Hove MT. Ocular and oculomotor signs in Joubert syndrome. *J Child Neurol* 1999;14: 621-7.
5. Fennell EB, Gitten JC, Dede DE, Maria BL. Cognition, behavior and development in Joubert syndrome. *J Child Neurol* 1999;14: 592-6.
6. Quisling RG, Barkovich AJ, Maria BL. Magnetic resonance imaging features and classification of central nervous system malformations in Joubert Syndrome. *J Child Neurol* 1999;14:628-35.
7. Maria BL, Boltshauser E, Palmer SC, Tran TX. Clinical features and revised diagnostic criteria in Joubert syndrome. *J Clin Neurol* 1999;14: 583-90.

Craniofacial fibrous dysplasia: A case report

Nahar-E-Zannat¹ and Mostaque Ahmed Bhuiyan²

Abstract

Fibrous dysplasia is regarded as a developmental skeletal disorder characterized by replacement of normal bone with benign cellular fibrous connective tissue. This can weaken the affected bone and cause it to deform or fracture. In most cases, fibrous dysplasia affects only a single bone — most commonly the skull or a long bone in the arms or legs. This variety usually occurs in adolescents and young adults. People who have more than one affected bone typically develop symptoms before the age of 10. Fibrous dysplasia is a genetic disorder. Treatment, which may include surgery, focuses on relieving signs and symptoms. Here we report a case of craniofacial fibrous dysplasia of a 14 yrs old male patient.

[OMTAJ 2015; 14(1)]

Introduction

Fibrous dysplasia is a developmental disorder of growing bone. Etiology is unknown. The age of onset is usually between 10 and 30¹. It is of two types: polyostotic and monostotic. Polyostotic form involves several bones and accounts for 30%, on the other hand monostotic form involves a single bone and constitutes 70% of the case.²

Cranofacial involvement in fibrous dysplasia occurs in nearly 100% of the polyostotic and 30% of the monoostotic forms.³

The bones commonly involved are mandible (12%) and maxilla (12%), involvement of ethmoid, sphenoid, frontal and temporal bones are infrequent.⁴

These lesions cause expansion, thickening and sclerosis of the involved bones with resulted visual complications, hearing disturbances, facial asymmetry and tooth displacement depending on the bone involved. We report a case of craniofacial polyostotic fibrous dysplasia in a 14 years old male patient.

Case Report

1. Junior Consultant, Radiology & Imaging, South Surma Health Complex, Sylhet.
2. Assistant Professor, Radiology & Imaging, Sylhet MAG Osmani Medical College.

14 year old male patient presented with a swelling on the right side of face for 3 years, gradual enlargement of head, occasional headache and pain on the right side of face. There was no history of trauma. On clinical examination, a smooth bony-hard swelling was found on the affected site. Skin over the swelling was normal. There was no other swelling in the body and café-au-lit spots are not seen. Routine blood and urine investigation was normal. X-ray skull AP and lateral view showed a diffuse radio-opacity with radio-lucent areas cause bone expansion. [Figure-1]

Orthopantogram (OPG) showed radio-lucent area involving right side of the mandible adjacent to the root of premolar and first-molar teeth. [Figure-2]. CT scan of brain with bony window [Figure-3] showed a radio- dense mass involving frontal bone, right maxillary, sphenoid, ethmoid bone, right parietal, temporal, occipital and left temporal, parietal bone. Right side is more involved. It causes facial asymmetry with ground glass appearance and expansion of involved bones

MRI examination of brain [Figure-4] shows marked bony expansion involving frontal, right maxillary, sphenoid, ethmoid bone, right parietal, temporal, occipital and left temporal, parietal bone.

Heterogeneous signal intensity is noted in diploic space having cystic areas. Expansion of orbital bone causes narrowing of orbital space resulting proptosis of right eyeball. After intravenous contrast [Figure-5] some lesions shows heterogeneous and rim enhancement.

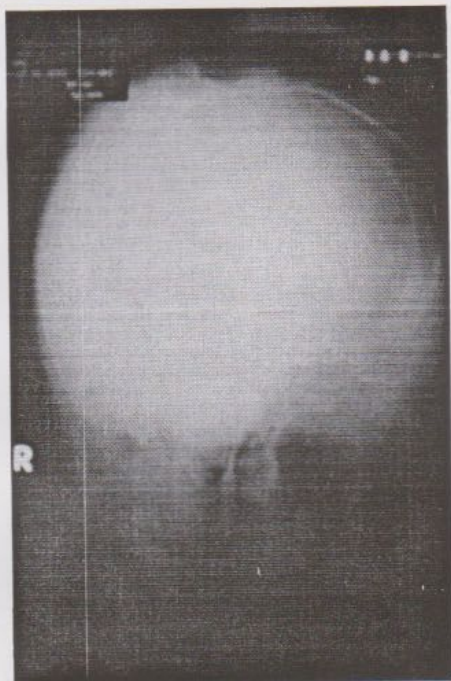


Figure-1

X-ray skull both view showing diffuse radio-opacity with radiolucent cystic areas causing expansion of diploic space.

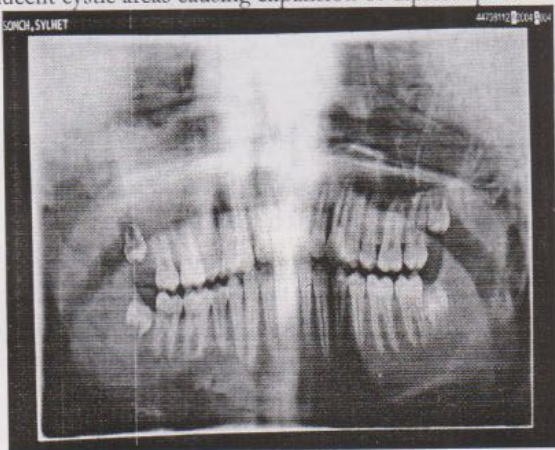


Figure-2

OPG shows radiolucent area involving right side of mandible adjacent to the root of premolar and first molar teeth.

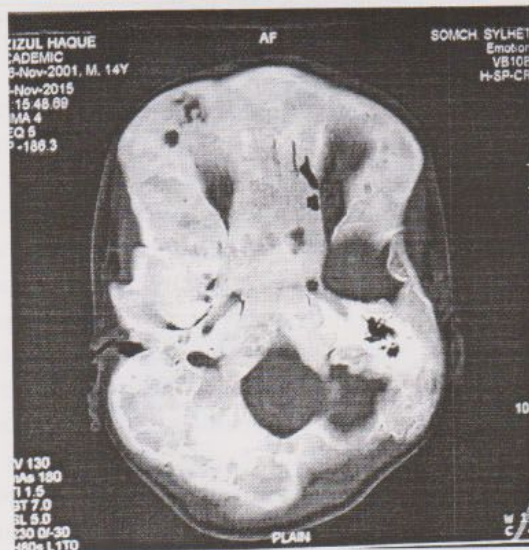


Figure-3 (a)

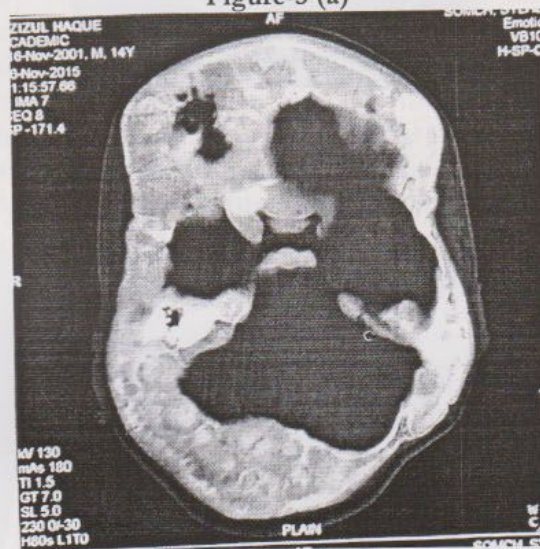


Figure 3 (b)

Axial CT image bony window (figure-3a, 3b) shows sclerosis with cystic areas causes marked expansion of frontal, right sphenoid, ethmoid, temporal and occipital bone.

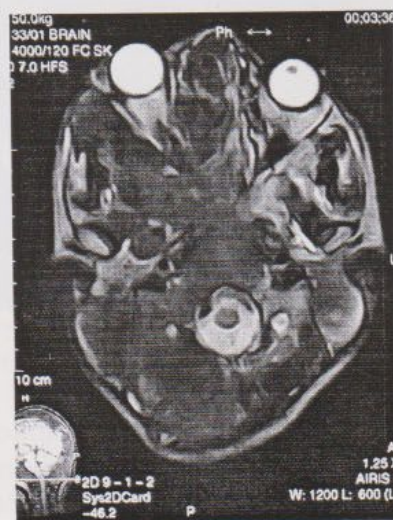


Figure- 4(a)

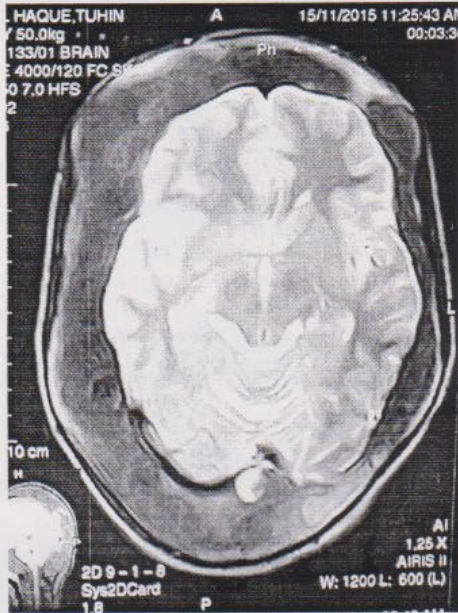


Figure-4(b)

MRI axial T2 WI (Figure 4a, 4b) showing expanded frontal, right sphenoid, ethmoid, temporal, parietal and occipital bone with heterogeneous signal intensity having few cystic areas.

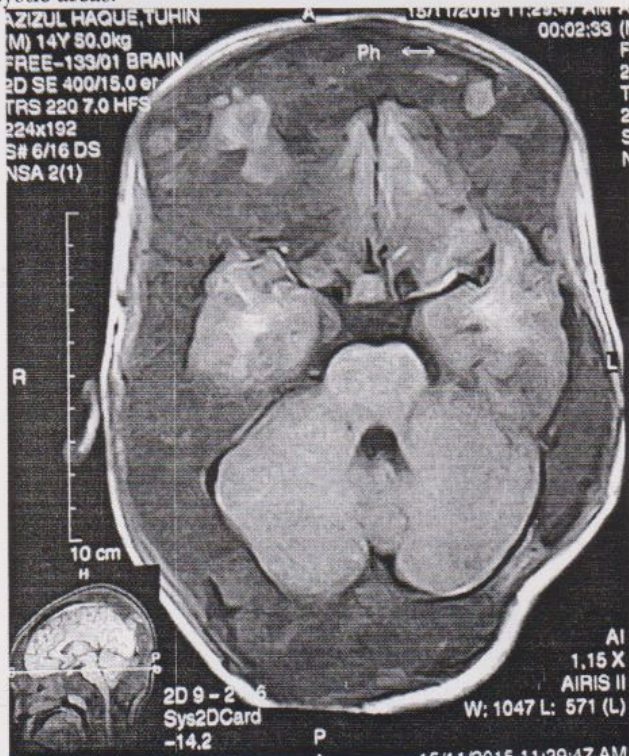


Figure-4(c)

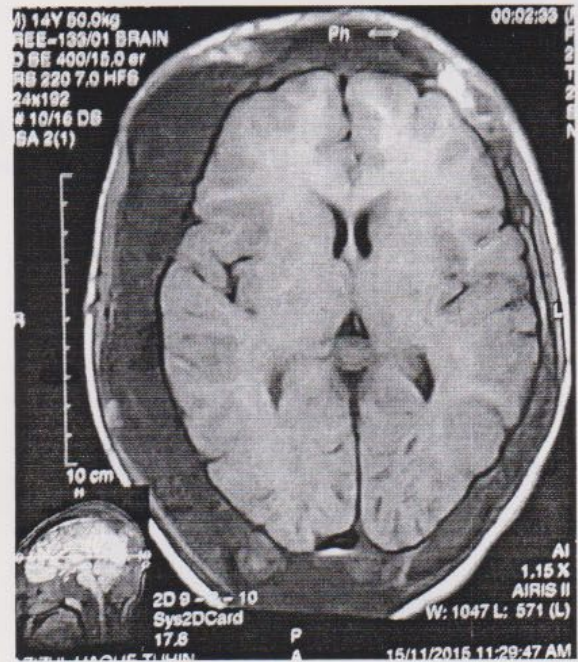


Figure-4 (d)

MRI axial T1 WI (Figure 4c-4d) showing expanded frontal, right sphenoid, ethmoid, temporal, parietal and occipital bone with heterogeneous signal intensity having few cystic areas.

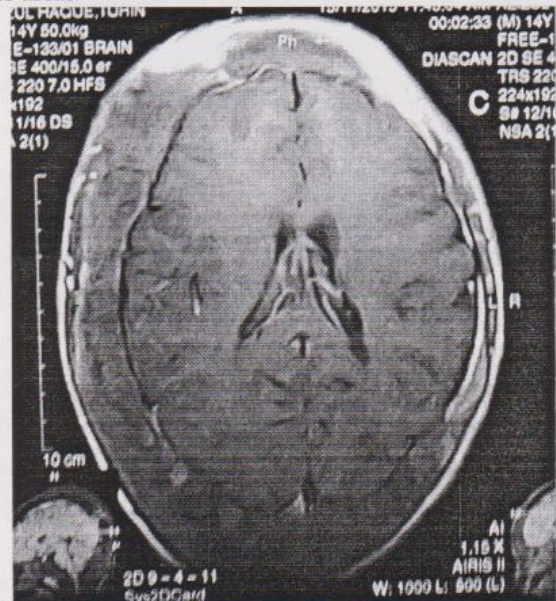


Figure-5 (a)

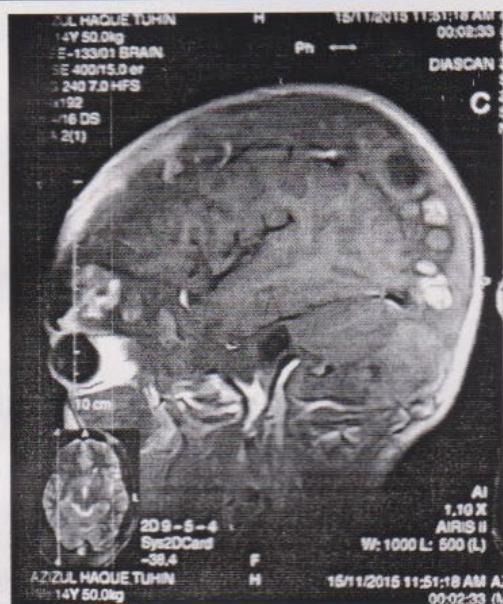


Figure-5(b)

MRI of brain T1WI with contrast (Figure 5a-axial, 5b-sagittal) image, showing heterogenous and rim enhancement of the lesion.

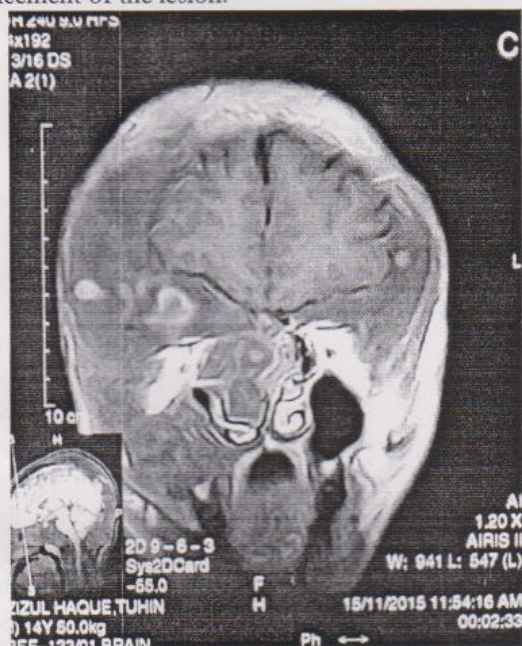


Figure-5(c)

MRI of brain T1 WI with contrast coronal image (Figure 5c) showing marked expansion of right sphenoid, ethmoid, parietal, temporal and maxillary bone with heterogenous and rim enhancement of the lesion. The diagnosis was confirmed by histopathology which shows, presence of excessive connective tissue with a characteristic whorled pattern and containing trabeculae of immature non-lamellar bone. The cyst containing serous fluid.

Discussion: In case of fibrous dysplasia medullary bone is replaced by well-defined areas of fibrous tissue

and cysts containing blood or serous fluid. Fibrous tissue then undergoes varying degrees of abnormal ossification so that some of the lesions shows an increasing density depend on the extent of ossification. This increasing density may be patchy giving a cottonwool appearance or homogenous giving a ground glass appearance.¹ In our case the lesion has a ground glass appearance. Craniofacial involvement in fibrous dysplasia is seen in both monoostotic and polyostotic forms. Monoostotic fibrous dysplasia has a different skeletal distribution from polyostotic disease and occurs most commonly in the femur followed by tibia, craniofacial bone and ribs³. In our case only craniofacial distribution was present. Clinical presentation of fibrous dysplasia varies with the primary bone involved and the extent of disease. Patient of craniofacial fibrous dysplasia presents with cranial asymmetry, facial deformity, nasal stuffiness, proptosis, visual impairment. In our case, patient presented with cranial asymmetry, facial deformity and proptosis. The disease has its onset during early life, usually in late childhood or early adolescence. Patients with polyostotic form of disease are mainly younger. In our case, the patient was 14 years of age. Radiographic features of fibrous dysplasia vary depending on the amount of bony and fibrous material within the lesion. It is sub-classified into three different pattern: pagetoid type 56%, sclerotic type 23%, radiolucent type 21%.⁵ The presented case was pagetoid type.

CT scan accurately established the diagnosis and the extent of bone involvement of optic canals, orbital fissures, frontonasal ducts and osteomeatal complex. CT scan characteristics of fibrous dysplasia include expansion of the involved bone with heterogenous pattern of CT densities associated with scattered island of bone formation. CT attenuation levels have been reported to range from 34 to 513 HU depending on the fibrous tissue and bone content.[6]. In our case, CT scan with bony window showed marked expansion of involved bones having sclerosis with cystic areas.

On MRI cyst will show features of fluid low on T1 WI, bright on T2WI, or STIR sequences. The greater the degree of mineralization the lower the signal, either homogenous or spotty. Hypocellular fibrous tissue is generally of low signal on T2 WI. After intravenous Gd-DTPA lesion show moderate to significant central contrast enhancement with some ring enhancement. The degree of contrast enhancement on T1 WI depends on amount and degree of bone trabeculae and collagen present.⁶ In

our case, MRI showed involved bones were grossly expanded having heterogeneous signal intensity on T1 WI and T2 WI. Cystic areas had low signal intensity on T1 WI and high on T2 WI. After intravenous contrast, heterogeneous and rim enhancement of the lesion is seen.

Complication of fibrous dysplasia is fracture, deformity, endocrine complication (Albright's syndrome, hyperthyroidism, Cushing's syndrome, gynecomastia and parathyroid enlargement), sarcomatous change. Endocrine complication is usually associated with polyostotic fibrous dysplasia.¹ In our case facial deformity and proptosis were present. No endocrine complication was seen.

In conclusion, typical radiological finding is helpful for the diagnosis of fibrous dysplasia and extent of the disease. X ray, CT, MRI all modalities are helpful for diagnosis of the case. Surgical treatment of fibrous dysplasia is conservative or radical excision with immediate reconstruction.

References

1. David Sutton. Text book of Radiology & Imaging. 7th edition vol: 2, New york, Elsevier, 2003;1130-4
2. Grabias SL, Campbell CJ. Fibrous dysplasia. *Orthop Clin North Am* 1997; 8:771-83
3. Hudson TM, Stiles RG, Monson DK. Fibrous lesions of bone. *Radiol Clin North Am* 1993; 31:279-97.
4. Araghi HM, Haery C. Fibro-osseous lesions of craniofacial bones. The role of imaging. *Radiol Clin North Am* 1993; 31:121-34.
5. Machida K, Makita K, Nishikawa J, Ohtake T, Olio M. Scintigraphic manifestation of fibrous dysplasia. *Clin Nucl Med*. 1986; 11:426-9.
6. Abdelkarim A, Green R, Startzell J, Preece J. Craniofacial polyostotic fibrous dysplasia: A case report and review of the literature. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2008;106: e49-55.

First trimester spontaneous uterine rupture in previous lower segment caesarean scar

Jahanara Begum¹ and Salma Akter²

Abstract

Uterine rupture is a potentially life threatening condition for both the mother & the baby that requires immediate surgical intervention.

Most of the cases has various risk factors & mainly occurs during the 2nd trimester. Spontaneous Uterine rupture in first trimester is extremely rare.

The initial impression may be an ectopic pregnancy. Intensive surgical method would be needed for acute diagnosis & immediate management. Uterine rupture has also been known in some women who have never had a caesarean section.

[OMTAJ 2015; 14(1)]

Introduction

Uterine rupture is one of the most dangerous condition carrying a increased risk of maternal & perinatal morbidity & mortality.^{1,2} Spontaneous rupture of the caesarean scar of the uterus during the first & second trimester is a rarity in obstetrics. It is most commonly seen in the last trimester, commonest cause being attempt of vaginal delivery especially with non-judicious use of oxytocics. Complete uterine scar rupture is a tear through the thickness of the uterine wall at the site of a prior caesarean incision. Fortunately, uterine rupture from a prior c- section with a low transverse scar is a rare event.³ Spontaneous uterine rupture of unknown cause during the early pregnancy is extremely rare. Few researchers have reported spontaneous uterine rupture without underlying cause during first trimester⁴. According to several information about uterine rupture most of the cases have various risk factors.² The single most important risk factors of uterine rupture is whether the uterus has previous scar or not⁵. The scar may arise

from the past injuries such as cesarean delivery, hysteroscopic resection of uterine septum, myomectomy are considered to be the causes of uterine rupture. It is reported that spontaneous rupture of unscarred uterus occurs in 1 in 1500.⁶ It may occur in a patient who has high parity, placenta increta or parcreta or in idiopathic causes. IVF, previous D&C & manual removal of placenta has been linked as risk factor.⁷ Uterine anomalies are one of the reasons for spontaneous unscarred uterine rupture in early pregnancy. Obstetrician must conceder these diagnoses when a pregnant patient presented with acute abdomen in early pregnancy.

We here reported a case of spontaneous uterine rupture in early pregnancy in a second gravida with history of previous one caesarean section.

Case Report

A 20 years old 2nd gravida, para - 1 with previous cesarean delivery was admitted in SOMCH on 5-11-2013 with history of severe lower abdominal pain at her ten weeks of gestation. Her menstrual history was regular. Her LMP was on 23-8-2013. She had a LSCS at term 6 month back due to fetal distress. The patient had same type of pain 3 days back & got admitted in a private hospital & treated conservatively. As her condition was not improving than she was referred to SOMCH.

On physical examination - she was in agony. There was pallor. Pulse was 130/min. B/P was not recordable. Abdomen was distended, tense & tender. Tenderness mostly found in pelvic region. Flank was full. Bowel sound was absent. With the suspicion of ectopic pregnancy per vaginal examination was done very gently. Uterus was found bulky. Cervix was soft, Os was closed, Pouch of doglus was full. There was no P/V bleeding. Then blood was sent for estimation. Sonography was done which showed uterus is enlarged contains single fetal outline with regular cardiac pulsation. CRL was 2.7 cm which corresponds to about 10 weeks of gestation. Huge pelvic collection

1. Associate Professor, Department of Obstetrics and Gynaecology, Sylhet MAG Osmani Medical College.
2. Resident Surgeon, Department of Obstetrics and Gynaecology Sylhet MAG Osmani Medical College

noted. Both tubes & ovaries appeared normal. Then diagnosis of haemoperitonium is made. Her blood report showed- Haemoglobin was 6gm/dl. Blood group was AB +ve, Random blood glucose was 6.5 mmol/L.

After taking informed written consent simultaneous resuscitation & laparotomy was done under general anesthesia. After laparotomy huge amount of blood found in the abdominal cavity. Due to previous C-Section bladder was adherent near the fundus of the uterus obscuring the previous scar. Both tubes & ovaries were found healthy. At first no bleeding point was visible. Still there was continuing collection of blood even after continuous removal with sucker tube. After exposing the lower segment by separating bladder from the uterus a rent was found on the previous LSCS scar which was bleeding profusely. Through this rent a sac along with a small fetus was protruding. the fetus along with placenta was removed. The rent was sutured by number 1 chromic catgut with continuous interlocking suture. Hemostasis was achieved. She was given 2 units of blood transfusion. Her post operative period was uneventful & was discharged on 6th postoperative day.

Discussion

Spontaneous rupture of the scar of the uterus during the first & second trimester is a rarity in obstetrics. It is most commonly seen in the last trimester & mostly during labour. Commonest cause being attempted vaginal delivery especially with non judicious use of oxytocics. Uterine rupture may mimic a large variety of conditions presented by acute abdomen or even may have minimal symptoms & signs despite what has really occurred. Neglecting this point may contribute to further irreversible catastrophes³. This condition can be confused with ruptured ectopic pregnancy or cesarean scar ectopic pregnancy. These patients often present with uterine rupture & shock. Etiology of cesarean scar pregnancy is unclear. Previous cesarean section, myomectomy, IVF, previous D&C & manual removal of the placenta has been linked as risk factors.⁵ In cesarean scar pregnancy the gestational sac is implanted in the myometrium at the site of previous cesarean section outside the uterine cavity & surrounded by myometrium & fibrous tissue of the scar.^{8,9,10} Maymen et al. reported an interesting association between cesarean deliveries for breech presentation & subsequent scar pregnancy. These were

also acknowledged by others. The hypothesis is that many cesarean deliveries for this indication (breech presentation) are elective & thus there is a poorly developed lower uterine segment. That may lead to faulty healing & consequently, implantation within the scar. Another factor contributing to the recently increasing incidence of this abnormal implantation may be the change in surgical technique for repairing the uterine incision. A single non inverting running suture, as commonly used today, may lead to impaired post operative healing & creation of defects within the scar.^{9,10}

Criteria for diagnosis of a cesarean scar ectopic pregnancy that is very likely to rupture in early pregnancy & commonly confused with scar rupture presented as acute abdomen due to haemoperitoneum⁹ are:-

1. Ultrasound appearance of an anterior bulging mass outside the contour of the uterus may be indicative.
2. Presence of trophoblast between bladder & anterior uterine wall.
3. No fetal parts in the uterine cavity.
4. Discontinuity of the anterior uterine wall in the surgical plane⁸.

In our case, patient had LSCS only six months back. She did not take abortion induction pill nor undergo MR or D&C. Same finding also found in a case report by visariya Nita et al. A case of a spontaneous unscarred uterine rupture in early pregnancy in a women with bicornuate uterus was reported by Kauffman & Linag. Where uterine anomalies are one of the risk factor for uterine rupture in early pregnancy.¹¹ Gurgen reported a case of uterine rupture at 14 weeks who conceive by IVF- ET with past history of fundal perforation during hysteroscopic synechiolysis for genital Tuberculosis. Spontaneous bilateral cornual uterine dehiscence early in the 2nd trimester after bilateral laparoscopic salphingectomy & IVF was reported by Inovary et al.¹² Liang et. al. reported a case of uterine rupture from placenta praevia in the first trimester.¹³

In Conclusion, the lesson learned from the case is that, although uterine rupture is very rare in the first & 2nd trimester of pregnancy. It should be taken into consideration in the differential diagnosis of acute abdomen especially if there is a predisposing factor like previous cesarean scar. Women having cesarean scar delivery should be advised not to conceive within months of the caesarian section.

References

1. Kong KY, Chang SK, Kim YJ, Lee JY, Chung JK. A clinical evaluation on the rupture of the gravid uterus. *Korean J Obstet Gynecol* 1993; 36: 1486 - 90.
2. Suner S, Jagminer L, Peipert JF, Linakis J. Fatal spontaneous rupture of a gravid uterus: Case report & literature review of uterine rupture. *J Emerg Med* 1996; 14: 181 - 5.
3. Nicette jukelevics, MA, ICCE/ revised sep.01, 2004. Uterine scar rupture - What is an uterine rupture? (Accessed on Nov 10, 2014 at WWW.belly belly. com. au/ birth/ uterine rupture.)
4. Yong- Joon Park, KiYong Ryu, Moon-11 park- Spontaneous uterine rupture in the first trimester: a case report. *J Korean Med Sci* 2005; 20: 1079 - 81.
5. Turner MJ. Uterine rupture. *Best Pract Res Clin Obstet Gynaecol*. 2002; 16:69-79.
6. Sallam AH, Preston J. Idiopathic uterine perforation in late pregnancy. *J Obstet Gynaecol* 2002; 22: 317.
7. Thomas A Molinaro M.D., Kurt T Barnhart, M.D., M.S.C.E. Ectopic pregnancies in unusual location. *Semin reprod Med* 2007; 25: 123-30.
8. Luce Tulpin, Olivier Morel, Cecile Malartic, Emanuel Barranger. Conservative management of a cesarean scar ectopic pregnancy: A case report. *Cases J* 2009, 2: 77- 94.
9. Rotas MA, Haberman S, Levгур M. Cesarean scar ectopic pregnancies - etiology, diagnosis & management. *American Coll Obstetr Gynecol* 2006; 107: 1373 - 81.
10. Ash A, Smith A, Maxwell D. Cesarean scar pregnancy. *BJOG* 2007; 114: 253 - 63.
11. Kauffman RP, Liang CC. Case report - First trimester Spontaneous uterine rupture in a young women With uterine anomaly. Hindawi publishing corporation. vol- 2014, Article page <http://dx.doi.org/10.1155/2014/967386>.
12. Inovay J, Marton T, Urbancsek J, et al. Bilateral cornual uterine dehiscence early in the second trimester after bilateral laparoscopic salpingectomy and in-vitro fertilization: case report. *Hum Reprod* 1999 ; 14 : 2471-3.
13. Liang HS, Jeng CJ, Sheen TC, et al. First-trimester uterine rupture from a placenta perceta. A case report. *J Reprod Med J Reproduct med* 2003;46:474-8.

INFORMATION FOR THE CONTRIBUTORS

THE OSMANI MEDICAL TEACHERS ASSOCIATION JOURNAL (OMTAJ) IS THE OFFICIAL ORGAN OF THE TEACHERS ASSOCIATION OF SYLHET M A G OSMANI MEDICAL COLLEGE AND IS PUBLISHED BI-ANNUALLY (JANUARY AND JULY EACH YEAR)

The guidelines are in accordance with the "Recommendation for the conduct, Reporting, Editing and Publication of Scholarly work in Medical Journals (ICMJE Recommendations)".¹

Subscription

The annual subscription rate for the non-members: medical students Taka 100/- and doctors Taka 200/- only.

Submission of manuscripts

The OMTAJ considers manuscripts for publication reporting original clinical or laboratory studies, reviews, case reports, medical progress and brief communications. Manuscript must not be longer than 2700 words. Please provide a word count excluding abstract and references.

Each manuscript must be accompanied by a covering letter from the corresponding author with a statement that the manuscript has been seen and approved by all authors and the material has not been previously submitted to or published elsewhere wholly or partially. A manuscript in duplicate together with tables and illustrations along with a copy in word 97/2000/word XP format in a 3.5" diskette/ CD should be sent to the Editor.

Letters to the Editor

Letters to the Editor are considered for publication (subject to editing and abridgement) provided they do not contain any material that has been submitted or published elsewhere.

Please note the following: *Your letter must be typewritten and triple spaced; *Its text, not including references, must not exceed 250 words, if it is in reference of a recent journal article, or 400 words in all other cases (please provide a word count). *It must have no more than five references and one figure or table. *It must not be signed by any more than three authors. *Please include your full address, office-time telephone (and/or mobile) number, and e-mail address.

Preparation of the manuscript²

All papers must be written in English. All sections of the manuscript should be typed double-spaced, with left alignment in MS Word documents and on one side of good quality bond papers of A4 size (21x 29.7 cm) with margins of at least 2.5 cm., beginning each of the following sections on separate pages: title page, abstract, text, acknowledgments, references, individual tables, and

legends for illustrations. Number pages consecutively, beginning with the title page.

Title page

The title page should contain: (1) the title of the article; (2) a short running head of fewer than 40 letter spaces; (3) name of the author (s); (4) institutional affiliation of each author; (5) name and address of the corresponding author.

Abstract

The second page should carry an unstructured abstract of not more than 150 words.

Text

The text of observational and experimental articles should be divided into sections with headings: Introduction, Materials & Methods, Results, and Discussion.

Acknowledgments

All acknowledgments including financial supports should be mentioned under the heading 'Acknowledgments' and not as a footnote on the first page or in the text.

References

Number references consecutively in the order in which they are first mentioned in the text. Identify references in text, tables, and legends by Arabic numerals (1, 2, 3....). Follow the form of references used in the *Index Medicus*, including the style of abbreviations. Try to avoid using abstracts as references: 'unpublished observations' and 'personal communication' may be inserted in the text.

Information supplied in the references section of any manuscript is not usually checked by the editorial staff, and hence, the concerned author(s) bear total responsibilities of the references.

Following are few examples of references:

1. Standard Journal Article: (List all authors when six or less; when seven or more, list only first three and add *et al*). Akhter A, Haque R, Kholil M, Sultana Z, Fakir MAH. Effects of Oral Garlic on Testicular Micro-architecture of Adult Rat. *Osmani Med Teachers Assoc J* 2002; 1(1): 1-3.

2. Corporate Author in Journal: Committee for Computer Application in Clinical Microbiology. Bacterial Antimicrobial Susceptibility Pattern, 1988. *J Infect Dis Antimicrob Agents* 1991; 8: 25-39.

3. Letter to Editor: Yagupsky P, MA Menegu. Intraluminal colonization as a source of catheter-related infection. *Antimicrob Agents Chemother* 1989; 33: 2025. (Letter)

4. Corporate Author in Book: World Health Organization. On being in charge: a guide to management in primary health care, 2nd ed. England: World Health Organization 1992.

5. Chapter in Book: Wenzel RP. Organization for infection control. In: Mandell GL, Douglas RG, Bennett JE, eds. *Principles and practice of Infectious Disease*, 3rd ed. USA: Churchill Livingstone Inc 1990: pp. 2176-80.

6. Thesis/ Dissertation: Kaplan SJ. Post-hospital home health care: the elderly's access and utilization [dissertation]. St. Louis (MO): Washington University 1995.

7. Formally published abstracts: Geesy GG, Costerton JW. Bacterial population adherent to submerged surfaces in a pristine mountain stream. *Abstracts of the Annual Meeting of the American Society for Microbiology* 1977: 235.

8. Articles from symposium volumes: Hamilton LD. Immunogenic polynucleotides. In: Beers RF Jr (ed). *Biological effects of polynucleotides: Proceedings of the symposium on molecular biology*. New York, Heidelberg, Berlin: Springer Verlag 1971: 107-28.

9. Insert from commercial product: Zyvox (linezolid). Peapack NJ: Pharmacia & Upjohn 2000 (package insert).

10. Web site: Division of tuberculosis elimination. Surveillance reports: reported tuberculosis the United States, 2000. Atlanta: Centres for Disease Control and Prevention, 2001. (Accessed June 27, 2001, at <http://www.cdc.gov/hchstp/tb/surv/surv2000>.)

11. On-line only Journal: Scientist JQ. 2 October 1998, Posting date. History of virology. *Am Virol J* 1998; 30-150. (*Page numbers may not be available*) [Online.] <http://cbxiou.pgr> (last accessed October 10, 1998)

12. Online version of print journal: Scientist JQ. History of clinical microbiology. *Clin Microbiol* 1999; 100: 123-345. [Online]

13. Online version of print books: Scientist JQ. 4 October 1998, Posting date. Culturing methods, 750-800 In: Gavier (ed). *Practical procedures for Laboratory*, 5th ed. [Online.] DEF Publishing Co. Boston, Mass [Http://cbxiou.pgr](http://cbxiou.pgr). (last accessed October 10, 1998).

Abbreviations

Except for units of measurements, abbreviations are discouraged. The first time an abbreviation appears, it should be preceded by the words for which it stands.

Drug name

Generic names should generally be used. When proprietary brands are used in research, include the brand name in parentheses in the methods section.

Permissions

Materials taken from other sources must be accompanied by a written statement from both author and publisher to the OMTAJ for reproduction.

Review and Action

Manuscripts are examined by editorial staff and usually sent to reviewers.

Rejected manuscripts will only be returned if accompanied by stamped and self-addressed envelope.

Letters about potentially acceptable manuscripts will be sent to the corresponding author after the review process is complete.

Copyright© 2015 Sylhet MAG Osmani Medical College Teachers Association.

¹ Recommendation for the conduct, Reporting, Editing and Publication of Scholarly work in Medical Journals (ICMJE Recommendations). International Committee of Medical Journal Editors. Available at www.icmje.org/icmje-recommendations

² Additional information regarding manuscript preparation and relevant editorial policy is available in the editorial office

Covering letter to the Editor for submission of manuscripts

To
The Editor
The OMTAJ
Sylhet MAG Osmani Medical College,
Sylhet-3100

Subject: Submission of manuscript

Dear Sir,

I/we am/ are submitting along with a manuscript for Original Article/ Case Report/ Review Article/ Medical Progress/ Occasional Note/ Others, having title:-----

----- for publication in the OMTAJ.

I/we mention that the manuscript had not been **submitted to**, or **accepted for publication**, or **published** in any form, in any other journal partially, or completely.

We also agree with the following orders of authorship to the manuscript, and we also certify that the authorship will not be contested by anyone whose name is not listed here.

Authors chronology

Signature

- 1.
- 2.
- 3.
- 4.
- 5.

Corresponding Author: -----Signature:-----

Address:-----