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ORIGINAL ARTICLE

The Osmani Medical Teachers Association Journal Volume 1 Number 2

One Stage Repair of Hypospadias - Experience in Sylhet MAG Osmani Medical College Hospital

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Abstract

We designed our study to perform successfully all types of hypospadias repair in children with the modern single stage reconstructive procedures. The aim of the study was to reduce the rate of urethrocutaneous fistula and to produce a straight penis with the meatus at the tip of glans allowing for a normal urinary stream & coitus. Between January 1998 and December 2001, 70 consecutive patients underwent primary hypospadias repair in the department of Paediatric Surgery. Of these children, only 25.71% (18 cases) were of 1-2 years old at the time of operations. The specific type of hypospadias repair techniques used included MAGPI in 8 cases, Snodgrass in 46, Mathieu in 2 cases, Onlay island flap in 6, Transverse preputial island flap technique (Duckett) in 07, and V. T. Joseph technique in one. Postoperatively a urethral stents were placed routinely into the bladder for all hypospadias patients except those undergoing a MAGPI procedure. Some 21 (30%) patients had a surgical complication requiring a secondary surgical procedure. Of them, urethrocutaneous fistula was developed in only 15 (21.43%) of cases. In 2 children significant meatal stenosis developed. In all of our patients, single stage repair was simpler to perform. Our results with one stage, techniques for hypospadias repair has led us to believe firmly that single stage repair of all hypospadias is essential in all young children to obtain satisfactory results.

[OMTAJ 2002; 1(2): 39-42]

Introduction

Hypospadias is one of the common congenital defects involving male genitalia. Abnormal meatus may open at the undersurface of penile shaft anywhere from the glans to perineum. It is found in 1 in 300 cases. Though more than 300 operative techniques with modifications are available, hypospadias is still a challenging problem for Paediatric surgeons. Basic elements for repair of hypospadias are: Glanulomeatoplasty, Orthoplasty, Urethroplasty, Skin cover & Scrotoplasty. Russell, in 1900, described a procedure for a one-stage repair of hypospadias where a flap was developed from the ventral surface of the penis.[1] Since then major milestones have been passed in single stage technique with contributions by Bevan,[2] Broadbent,[3] Horton & Devine,[4] Hodgson,[5] Asopa,[6] Duckett,[7] and VT Joseph [8] and their co-workers among others.

Today experienced paediatric urological surgeons routinely perform a single-stage hypospadias reconstruction in the vast majority of cases. Multistage repairs are still performed for complex or failed hypospadias problems. We applied to a variety of contemporary single stage hypospadias reconstructive procedures that allows us to successfully treat all types of hypospadias in one stage; with aims to reduce our rate of urethrocutaneous fistula and to produce a straight penis with the meatus at glands tip allowing for a normal urinary stream & coitus.

Methods

A total of 70 consecutive patients underwent primary hypospadias repair between January 1998 and December 2001 in a period 3 years. Children of all ages, who had not under gone previous operations for hypospadias, were included in this study.

No other surgical procedures were performed at the time of the hypospadias repair. The specific types of hypospadias repair employed included meatal advancement and glanuloplasty incorporated (MAGPI),[9] tabularized incised plate urethroplasty (Snodgrass),[10] Mathieu,[11] Onlay island flap,[12] Transverse preputial island flap technique (Duckett),[13] and V.T. Joseph technique.[14] All hypospadias repairs were performed with the aid of 2.5 optical loupe magnification and the operating microscope was not used. An artificial erection was performed in all cases to confirm complete chordee release, when needed.

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Fine non-crushing instruments, traction sutures and skin hook were used to minimize the surgical trauma to the genital skin. Intermittent application of a tourniquet and 1:100,000 dilution of adrenaline injection were used as a haemostatic aid as well as when necessary. Meticulous pinpoint haemostasis was achieved with bipolar electrocautery. Whenever necessary a single stage technique with vascularized preputial skin was used to form the neourethra. The various types of hypospadias reconstructive procedures were performed as described by the original authors.[9-14] Postoperatively, a urethral stint (6 Fr feeding tube for boys of 2 years) was placed routinely into the bladder for all hypospadias patients except those undergoing a MAGPI procedure. The urethral stent was sutured to the glans penis with 4/0 or 5/0 prolene sutures. This drainage tube was left in place for 7-10 days. All children with a urethral stent received antimicrobial medication postoperatively. A bio-occlusive compression dressing (Duoderm) was applied circumferentially to the penile shaft and glans. All patients were followed for 6 weeks to 4 years post-operatively.

Results

Among the 70 children, most of the patients (25.71%) were within 1-2 years old, followed by (21.43%) children between 3-4 years (Table-1).

Table 1. Age distribution of the patients (n= 70)

Age group in years	No. of cases	Percent
1 – 2	18	25.71
3 – 4	15	21.43
5 – 6	11	15.71
7 – 8	09	12.86
9 – 10	09	12.86
Above 10	08	11.43

In 44 patients, the meatus was in distal shaft (distal penile) and in 9 patients, it was located in the middle shaft. (Table 2).

Table 2. Types of Hypospadias found among the cases (n=70)

Sl no.	Types	Number of children	Percent
01	Glandular	06	8.57
02	Coronal	03	4.28
03	Distal shaft	44	62.86
04	Middle shaft	09	12.86
05	Proximal shaft	06	8.57
06	Penoscrotal	02	2.86
	Total:	**70**	**100**

Some 64 (91.43%) patients had chordee and 12.86% had associated with other Congenital anomalies (Table 3).

Table 3. Associated congenital anomalies among the cases (n=70)

Sl no.	Types	Number	Percent
01	Inguinal hernia	03	4.29
02	Congenital hydrocoele	01	1.43
03	Undescended testes	04	5.71
04	V.S.D	01	1.43
	Total:	**09**	**12.86**

A total of 21 (30%) patients had a surgical complication requiring a secondary surgical procedure following a single stage repair. An urethrocutaneous fistula was developed in only 15 (21.43%) of our patients (Table-4).

Table 4. Complications among the cases (n=70)

Sl no.	Complications	Number	Percent
01	Bleeding	01	01.43
02	Urethrocutaneous fistula	15	21.43
03	Stricture	02	02.86
04	Meatal stenosis	02	02.86
05	Sloughed flap	01	01.43
	Total:	**21**	**30.0**

Among the 15 patients with urethrocutaneous fistula, 5 (33%) were operated by Onlay island flap, and 6 (40%) by Transverse preputial island flap (for proximal shaft/ penoscrotal type) and 4 (27%) by Snodgrass procedure (Table-5). No child undergoing a MAGPI, Mathieu and VT Joseph technique had an urethrocutaneous fistula.

In 2 children significant meatal stenosis developed, which required a simple secondary meatoplasty. All urethrocutaneous fistula were closed as a simple secondary procedure. No other patient has had any complication requiring surgical correction till now.

Table 5. Surgical morbidity in different methods

Complications	No (%) of patients having morbidity in different techniques					
	MAGPI	Mathieu	Snodgrass	VTJ	OIF	TPIF
Bleeding (n=01)	-	-	-	-	01 (100)	-
Urethrocutaneous fistula (n=15)	-	-	04 (27)	-	05 (33)	06 (40)
Stricture (n=02)	-	-	-	-	01 (50)	01 (50)
Meatal stenosis (n=02)	01 (50)	-	01 (50)	-	-	-
Sloughed flap (n=01)	-	-	-	-	-	01 (100)

MAGPI (Meatal advancement glanduoloplasty incorporated), Mathieu (Mathieu Procedure), Snodgrass (Snodgrass Procedure), VTJ (V. T. Joseph Technique), OIF (Only Island Flap Technique), TPIF (Transverse Prepuceal Island Flap Technique).

Discussion

Single stage repair of hypospadias is well established in paediatric urologic practice. The challenge of achieving a perfect functional and cosmetic result is not illusory. Furthermore, the price one pays for accomplishing such a goal is not excessive. The results of hypospadias repair depend on the regularity of performing the surgery and the obsession of the surgeon in adhering to plastic reconstructive surgical principles. From the point of view of the optimal age for surgery we have found from our own experience that a full hypospadias repair can be done at the age of 1 year without undue technical difficulty. The primary justification for this approach has been the psychological advantage for the patients.

In addition, use of contemporary techniques achieves a primary single stage hypospadias repair in the vast majority of cases. In all of our patients, single stage repair was simpler to do. There is always adequate preputial skin both to create a neourethra and skin coverage as shown by Asopa *et al.*[6] In reviewing the complications of single stage repair of hypospadias in this series, we must bear in mind that several factors have to be taken into account: the degree of severity of the hypospadias, the condition of urethral plate & preputial skin, the technique used, the presence of postoperative complications such infection or haematoma formation and the experience of surgeon who carries out the operation.

Thus most of the fistulae occurred in this study had proximal shaft and penoscrotal hypospadias. By contrast the distal shaft and midshaft hypospadias had minimum fistulae. However, our overall fistulae rate (15/70, 21.43%) compares favourably with the experience of other workers.[15,16] It is interesting to note that in our series stricture occurred in 2 cases, one after the Onlay island flap and another after the TPIF (Duckett) repair for proximal shaft & penoscrotal hypospadias respectively. Nonetheless, the satisfactory cosmetic and functional results in the other children justify this acceptable morbidity. However, in our series, the bio-occlusive (Duoderm) dressings have been found easy to place and maintain for 7 to 10 days without slippage.

Our experience with one stage techniques for hypospadias repair has led us to believe firmly that single stage repair of all hypospadias is essential in all young children to obtain satisfactory results. Nevertheless, there is no one single method that can be employed in all cases[17] and the hypospadiologists need familiarity with a variety of surgical techniques which can be applied in individual circumstances.

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ORIGINAL ARTICLE

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Surgical Ascariasis In Children In Greater Sylhet Region

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Abstract

A retrospective study of 117 children, aged 3-12 years, revealed 29 cases that need surgical intervention due to ascariasis. The Surgical conditions found at laparotomy were intestinal obstruction (19, 65.5%), ileal perforation (5, 17.2%), appendicitis (4, 13.8%), and biliary ascariasis (1, 3.5%). The Study highlights the high incidence of surgical ascariasis among children with abdominal complain in an endemic area.

[OMTAJ 2002; 1(2): 43-44]

Introduction

Ascaris lumbricoides is one of the common helminthes found in human beings. They are known to cause a variety of medical and surgical problems.[1-7] This retrospective study was carried out to find the frequency of surgical ascariasis among paediatric patients admitted with abdominal complaints in greater Sylhet region.

Methods

From January 1999 to December 2001 records of 117 children, admitted with abdominal complaints in surgical wards of Sylhet MAG Osmani Medical College were studies retrospectively.

Results

From a total of 117 patients, 49 (41.9%) were treated conservatively and 68 (58.1%) needed surgical intervention (Table-1). Among surgical intervention, 29 has ascariasis and other diagnoses were appendicitis (11), congenital band obstruction (9), ileo-ileal intussusception (11), tubercular peritonitis (7) and liver abscess (1) (Table-2). The symptoms of 29 operated patients with ascariasis were abdominal colicky pain, abdominal distension, vomiting, constipation, recurrent right upper abdominal pain and jaundice (Table-3).

Eight patients developed symptoms after deworming, X-ray abdomen revealed multiple air and fluid level in 25 cases. Ultrasonography of the abdomen revealed dilated common bile duct with an echogenic linear shadow and a thick-walled gall bladder.

Table 1. Line of treatment provided

Sl no	Type of treatment	No (%) of the patients
01	Conservative	49 (41.9)
02	Surgery	68 (58.1)
	Total	117 (100)

Table 2. Causes of surgical intervention (n=68)

Sl no	Causes	No (%) of cases
01	Ascariasis	29 (42.6)
02	Acute appendicitis	11 (16.2)
03	Congenital band obstruction	09 (13.2)
04	Ileo-ileal intussusception	11 (16.2)
05	Tubercular peritonitis	07 (10.3)
06	Liver abscess	01 (1.5)
	Total	68 (100%)

Table 3. Symptoms of operated patients with ascariasis (n=29)

Sl no	Symptoms	No. of patients
01	Abdominal colicky pain	28
02	Abdominal distension, Vomiting, Constipation	21
03	Symptoms of appendicitis	8
04	Recurrent right upper abdominal pain	2
05	Jaundice	1

The operative findings were small bowel packed with worms with or without gangrene, appendicitis with intraluminal worms, intussusception, ileal perforation with extraluminal worms and common bile duct (CBD) dilated with dead worms (Table 4).

The operations performed were enterotomy and removal of worms, disimpaction of worms into ascending colon, appendicectomy, ileal resection and anastomosis, cholecystectomy and common bile duct exploration, peritoneal toileting and closure of ileal perforation and no definite surgery in two cases due to jejunitis (Table-5).

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Table 4. Operative findings (n=29)

Small bowel packed with worms		Appendicitis with Intraluminal Worms	Intussus-ception	Ileal perforation With extra-luminal worms	CBD dilated and dead worms
Gangrene	Without gangrene				
03	13	04	03	05	01

Table 5. Operative treatment performed (n=29)

Operative treatment	Number of cases
Enterotomy and removal of worms	11
Disimpaction of worms into ascending colon	03
Appendicectomy	04
Ileal resection and anastomosis	03
Cholecystectomy and CBD exploration	01
Peritoneal toileting and closure of ileal perforation	05
Operation not done	02

Discussion

Ascaris lumbricoides usually resides in jejunum and ileum (8:1) and 9% are static there.[8] However, they are known to migrate into the duodenum, hepatobiliary and pancreatic duct system[1] or even perforate the bowel.[4,6]

Bangladesh is an endemic area for ascariasis and more so in children. Data in this regard for greater Sylhet region are scarce. Wet soil, temperate climate, poor personal hygiene and sanitation are contributory factors encouraging development of larval stage of *A. lumbricoides*. Ascaris inside bowel can cause worm impaction, bullous obstruction with or without gangrene.[2,3,6] Worms are rarely found inside gall bladder and biliary tract.[1,2]

As eradication of ascariasis in our community is an unrealistic goal in the foreseeable future, prevention must be directed towards reducing the community worm load through various public health measures.[9]

Deworming periodically in endemic area, improvement of sanitation, proper excreta disposal safe drinking water can reduce the incidence of ascariasis and subsequently decrease the surgical problems.

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ORIGINAL ARTICLE

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Screening of Aetiological Agents of Abnormal Vaginal Discharge by Simple Microscopy

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Abstract

Almost 90% of the aetiological agents of abnormal vaginal discharge are infectious agents, namely *Candida albicans, Trichomonas vaginalis, Gardnerella vaginalis, Chlamydia trachomatis* and *Neisseria gonorrhoeae*. Most of them are identifiable by simple microscopy in a very simple laboratory set up. Keeping in mind the above background, this was a prospective study carried out among 112 women of different age groups complaining of abnormal vaginal discharge. Specimens of vaginal secretion and/or endocervical swab were collected and examined microscopically with various recommended preparations to screen for the aetiological agents. Among the study population, the highest (37.5%) found infected with Candidiasis, 20.54% with Trichomoniasis, 3.57% with Bacterial vaginosis and 1.78% with Gonococcal infection. Some 8.93% among the patients were found infected with both Candidiasis and Trichomoniasis. Ages of the enrolled patients were from 16 to 60 years with a mean age of 29.1 years. Almost all of the patients (94.64%) were housewives. All of them were married and a few (22.32%) had husbands working and living abroad. The highest number of patients (52/112, 46.45%) gave history of using combined oral pill as contraceptive measure. The findings of the present study carry no significant differences with other previous studies.

[OMTAJ 2002; 1(2): 45-47]

Introduction

Vaginal discharge is a very common problem among the women of our country. Various aetiological agents have been identified with the problem and vaginal discharges may originate both from the vagina and the cervix. Vaginal conditions include vulvovaginitis caused by *C. albicans*, *T. vaginalis* and trauma as well as bacterial vaginosis caused by *G. vaginalis* and other anaerobes. Cervical conditions include cerviciris caused by *C. trachomatis*, *N. gonorrhoeae* and other nonspecific causes as well as cervical neoplasm and IUCDs. Of the conditions among these two categories, the infections (*viz.* Candidiasis, Trichomoniasis, Bacterial vaginosis, Chlamydial and Gonococcal infections) estimate almost 90% of the problem and simply a pH of >6 is strongly predictive.[1, 2 In] a study in Bangladesh during 1993 to 95 among 150 cases attending Gynaecology and Skin VD departments of then IPGMR, monilial (*C. albicans*) infection was found as the highest (48%), Trichomoniasis the second (13%) and bacterial vaginosis (Clue cells) only in 4% cases.[3] *T. vaginalis* was identified in 11% of the patients by direct wet film examination in another study in Dhaka city.[4] Vulvovaginitis caused by *C. albicans* has also been reported as the most common cause of abnormal vaginal discharge in the UK, whereas *T. vaginalis* as the most common cause worldwide.[5] Estimates of the prevalence of bacterial vaginosis (BV) vary widely from 10-32% of women attending obstetric, Gynaecology and family planning clinics. However, half of the women with clinical criteria for BV have no symptoms.[7] The vaginal discharge of some women (5-10%) may not have any pathology, which are physiological and may be due to IUCDs application, use of oral contraceptives, and at the time of ovulation. There is no extensive study in our country with these problems. In the present study, most of the significant aetiological agents of abnormal vaginal discharge were identified with very simple easy-to-do test and conventional simple microscopy.

Methods

Study Population. Any patient complaining of vaginal discharge attending the Gynaecologist's chamber was enrolled into the study.

Specimen collection. Specimens of vaginal swab (from posterior fornix) in cases of non-purulent discharge and/ or endocervical swab in cases of purulent discharge were collected and transported to the laboratory within few minutes taking all aseptic precautions.

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Laboratory investigations. The following investigations were done:

a. Wet mount preparation: A wet mount preparation was made with physiological saline for each vaginal swab immediately and examined under microscope for characteristic movement of *T. vaginalis*, for clue cells and yeasts with pseudohyphae.

b. pH measurement: A suspension of the vaginal swab in normal saline is soaked to a pH paper and the pH was recorded by measuring resulting colour against a wide range (1-14) pH standard.

c. Amine test: A drop of 10% KOH was added to vaginal discharge on a glass slide and any characteristic fishy amine odour was recorded as positive finding of bacterial vaginosis.

d. KOH preparation: The 10% KOH preparation for amine test was examined under microscope irrespective of amine result to see any budding yeasts and pseudohyphae of *C. albicans*.

e. Gram's staining: The endocervical swabs and/or vaginal swabs were stained by Gram's method and examined under microscope to see any Gram-negative intracellular diplococci characteristic of *N. gonorrhoeae*, and other Gram-stained microorganisms.

Results

Ages of the patients were between 16 to 60 years with mean age 29.5 years. The highest numbers of patients (58/ 112, 51.79%) were enrolled among 21-30 age group (Figure 1). All among the enrolled patients were married, mostly living together with their husbands. A few (19/112, 16.96%) had husbands working and living abroad and some 2.68% (03/ 112) were widows. Almost all of the enrolled patients (106/112, 94.64%) were housewives (Table 1).

Table 1. Occupation of the patients (n= 112)

Occupation	Number examined (%)	Number found infected
House wives	106 (94.64)	58
Service	04 (3.57)	01
Commercial Sex Worker	02 (1.79)	02
Total:	112 (100)	61

The highest 46.43% patients gave history of using combined oral pill as contraception and some 18.75% using no contraceptive measure (Table 2).

Table 2. Contraceptive use among the patients (n= 112)

Method of the Contraceptive	Number of patients (users)	Percent
Combined Oral Pill	52	46.43
IUCDs	25	22.32
Others	14	12.50
None	21	18.75
Total:	112	100

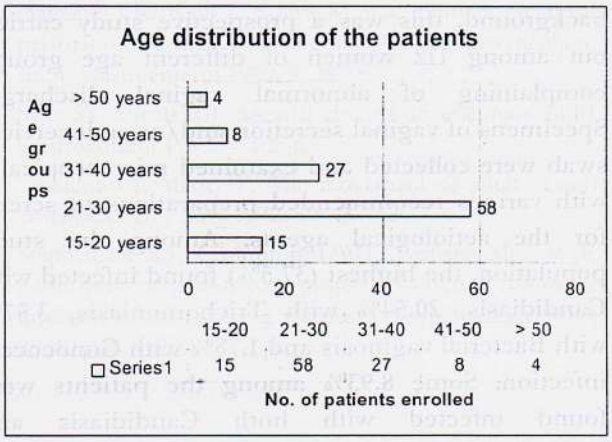

Figure 1: Age distribution of the patients

A total 61 (54.46%) of the patients with vaginal discharge were found infected with four agents identified by simple microscopy. The highest number among the patients complaining of white curd-like discharges (35/44, 79.55%) were found infected and the lowest (9/29, 31.03%) found infected among them complaining purulent/ mucopurulent discharges (Table 3). All of the cases having identified infecting agent had pH of vaginal discharges above>4.5 (Table 4).

Among the enrolled patients, 42 (37.5%) were found infected with Candida, 23 (20.54%) with Trichomonas. Clue cell was identified in 4 (3.57%) and finally gonococci were identified in 2 (1.78%) of the patients. Both *T. vaginalis* and *C. albicans* were found in 10 (8.93%) of the patients (Table 5).

Table 3. Physical finding of the vaginal discharges and corresponding infection among study population

Type of discharge	No. of patients (%)		Causative agents found				
	Examined	Infected	CA	TV	CA+TV	GV (BV)	GC
Watery discharge	39 (34.82)	17 (43.59)	01	08	05	03	00
Purulent/ Mucopurulent discharge	29 (25.89)	09 (31.03)	02	03	02	00	02
White, curd-like discharge	44 (39.29)	35 (79.55)	29	02	03	01	00
Total:	112 (100)	61 (54.46)	32	13	10	04	02

Key: CA- *Candida albicans*, TV- *Trichomonas vaginalis*, GV- *Gardnerella vaginalis*, BV- Bacterial vaginosis, GC- *Neisseria gonorrhoeae*

Table 4. pH of vaginal secretions examined and their relation with aetiological agents identified

pH range	No. (%) of specimens	
	Examined	Found infected (n=61)
Up to 4.5	21 (18.75)	00 (00%)
5.0 to 6.5	34 (30.36)	16 (26.23%)
> 6.5	57 (50.89)	45 (73.77%)
Total:	112 (100%)	61 (100%)

Table 5. Aetiological agents of abnormal vaginal discharge identified by microscopy

Name of the agents		No. of patients infected	Percent (n= 112)
C. albicans:	Single	32	28.57%
	Total	42	37.5%
T. vaginalis:	Single	13	11.61%
	Total	23	20.54%
Clue cells (Bacterial vaginosis)		04	3.57%
Neisseria gonorrhoeae		02	1.78%
Double infection (*C. albicans* + *T. vaginalis*)		10	8.93%

Discussion

Most of the aetiological agents of abnormal vaginal discharge have been identified by simple microscopy in this study. Findings of the present study have no significant difference with the previous studies; hence the methodology applied is supposed to be justified. Yet only 31.03% among the patients complaining of

purulent/ mucopurulent discharges were identified having causative agents, which suggest that most of the agents in these cases remain unidentified. This may be d or immunochromatography are all expensive and require considerable expertise. Taking into consideration of the cost-effectiveness, easy availability and simplicity to perform, the conventional simple microscopy can be used for diagnosis of individual cases as well as in epidemiological works.

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ORIGINAL ARTICLE

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Azithromycin in the Treatment of Chlamydial Urethritis in Men

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Abstract

This was a randomized un-blinded clinical trial to evaluate the efficacy of single dose of oral azithromycin in the treatment of chlamydial urethritis in male. The study also compared azithromycin with doxycycline in the treatment of chlamydial urethritis in men. Patients were evaluated by clinical and laboratory tests for *Chlamydia trachomatis* infection before therapy and 3 weeks after therapy. The patients were enrolled from Skin and Venereal Diseases Out Patients Department of Sylhet MAG Osmani Medical College Hospital as well as from a private chamber in Sylhet. A total of 40 male patients were diagnosed as chlamydial urethritis by both clinical and laboratory examinations. The major outcomes were clinical resolution of symptoms and signs of chlamydial urethritis, laboratory proven cure of chlamydia trachomatis infection and occurrences of adverse reactions to drugs. Of the 40 patients enrolled, 18 in the azithromycin-treated group and 17 in the doxycycline-treated group were evaluable for clinico-laboratory evaluation 3 weeks after starting treatment. Overall clinical cure rate was 88.9% in the azithromycin-treated group and 70.6% in the doxycycline-treated group. On the other hand, laboratory proven cure rate was 94.4% in azithromycin-treated group and 88.2% in doxycycline-treated group. *C. trachomatis* was eradicated in 94.4% in azithromycin group and 94.1% in doxycycline-treated group. Total clinical and laboratory proven cure rate was 83.3% in azithromycin-treated group and 70.6% in doxycycline-treated group. For empirical treatment **of chlamydial urethritis in male a single oral dose of azithromycin was as effective as standard 7-day course of doxycycline in achieving overall clinical and laboratory proven cure. Further more, it may offer advantage of better compliance to drug.**

[OMTAJ 2002; 1(2): 48-51]

Introduction

Chlamydia trachomatis is now considered as one of the most common sexually transmitted pathogens worldwide causing substantial morbidity in men women and infants.[1] .It is prevalent in both industrialized as well as developing countries.[2] In USA, more than 4 million cases were estimated in 1986. Some studies have carried out in Bangladesh in different times and settings, and showed the incidence of chlamydial infection as one of the most common sexually transmitted infection.[3,4] Regular reporting and documentation are lacking.

Urethritis manifested by urethral discharge, dysuria, or itching at the end of urethra is a response to inflammation of any etiology. Physical finding is urethral discharge, and laboratory findings include increased numbers of polymorphonuclear (PMN) leukocytes on Gram stain of urethral smear or in the sediment of first voided urine. Urethritis is called gonococcal when *Neisseria gonorrhoeae* is detected and non-gonococcal (NGU) when *N. gonorrhoeae* cannot be detected. *C. trachomatis* and *Ureaplasma urealyticum* are the most common causes of NGU. *C. trachomatis* contribute to 15-40% of NGU cases.

After an incubation period of 7-21 days, patients of chlamydial urethritis may present with dysuria and mild to moderate clear, purulent or mucopurulent urethral discharge. In 40% of cases, there may be few or no symptoms.[5]

High prevalence of genital chlamydial infection is partly due to lack of proper diagnostic facilities, because of mild or no symptoms, lack of familiarity of the clinicians and of proper treatment plan.

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Organisms can be cultured from urethral discharge. Chlamydial antigen or antibody detection can be done by immunochromatography and other methods. PCR has newly been introduced in the diagnosis. But all these are costly and not readily available in most places. Complications of chlamydial infection in men include acute epididymitis, Reiter's syndrome, persistent or recurrent urethritis and sometimes sterility. It may also facilitate transmission of HIV infection.[6] Thus early recognition and effective treatment of such infection may contribute to reduce the incidences of such dangerous complications.

Less emphasis has been placed on new therapeutic options since effective regimens are available and since antimicrobial resistance has not emerged. But all currently available therapeutic options (tetracycline, erythromycin, fluoroquinolones and doxycycline) are 7 days treatment and often cause mild but bothersome side effects. Thus the compliance especially in an asymptomatic patient may be poor using these drugs. Consequently infection may persist in this situation. The possibility of using a single dose treatment would be a significant improvement in managing these infections from a public health standpoint.

Azithromycin, a new azalide antibiotic has recently been demonstrated to be effective for treatment of chlamydial urethritis in men when given as a 1 gm single dose orally. It has a unique pharmacokinetic profile characterized by excellent bioavailability and sustained high tissue levels with minimal side effects of gastrointestinal symptoms.

With the advantages of effective and well-tolerated single dose therapy for chlamydial urethritis in men, we undertook this study to compare the efficacy and safety of 1 gm single dose of oral azithromycin with standard therapy, a 7-day course of 100 mg of doxycycline taken orally twice daily.

Methods

The study was carried out at skin and venereal diseases outpatients department of Sylhet MAG Osmani Medical College Hospital and a private chamber in Sylhet. A total of 40 patients of urethritis were selected for the study. All were male of at least 17 years of age and older who presented with typical symptoms of urethritis (discharge and dysuria). All were required to have an observable urethral discharge on examination. Gram stain of urethral smear with ≥5 PMNs/oil immersion field (OIF) and no microscopic evidence of gonococci, and urethral specimen or urine with positive chlamydial antigen test by immuno-chromatography. Those having ≥10 PMNs/high power field (HPF) of first voided centrifuged urine sample were also considered as one of the Laboratory criteria of diagnosis of chlamydial urethritis. Patients were excluded from the study if they had Gram stain evidence of gonococcal infection; other significant systemic disease; a urological abnormality; known HIV infection; allergy to azithromycin or doxycycline necessary for treatment with other antibiotic; treatment with an investigational drug in the last 30 days; antibiotic therapy in previous 2 weeks or a gastrointestinal condition that would likely affect drug absorption.

All participating patients signed written informed consent forms. Subsequently patients underwent a medical history and a physical examination. First voided urine was collected for routine microscopic examination and chlamydial antigen test, urethral swab was collected for Gram stain. Patients were advised to refer their sexual partners for evaluation and treatment, and were advised to abstain from sexual intercourse or use condoms for sexual intercourse before the completion of study.

Patients were randomly allocated to receive either azithromycin 1gm single dose or doxycycline 100 mg twice daily for 7 days. Follow up visits were scheduled 3 weeks after starting treatment and patients were evaluated again for the same clinico-laboratory parameters to achieve the cure. Patients were asked about potential adverse experiences at the follow-up.

Clinical efficacy was assessed at follow-up visit using objective evidence of urethritis (i.e., urethral discharge, dysuria) as the principal criterion to define clinical outcomes. Persistent symptoms of dysuria and observable urethral discharge were considered clinical failure. Urethral leukocytosis i.e., ≥5 PMNs/OIF of

Gram stain, ≥10 PMNs/HPF of first voided centrifuged urine sample and positive chlamydial antigen test were assessed at follow-up, and were the principal criterion to define laboratory outcome. Both the clinical and laboratory cure criterion were considered as total cure outcome.

Result:

The mean ages of the patients were 24 years ±4 in azithromycin-treated group and 25 years ± 4 in doxycycline-treated group. Majority of the patients were unmarried in both group. Highest numbers of patients were of higher education level. Students were maximum in both groups. Some 30% of patients in azithromycin-treated group and 25% of patients in doxycycline treated group reported past history of STD. As much as 55% in azithromycin-treated group and 45% in doxycycline-treated group had multiple sexual exposures. All of the patients in azithromycin-treated group and 75% in doxycycline-treated group reported sexual exposure with professional sex worker.

Table I. Socio-demographic profile and risk behavior of two treatment groups.

Characteristics	No (%) of patients in two treatment groups (n=20 in each group)	
	Azithromycin	Doxycycline
Mean age (years) ± SD	24 ± 4	25 ± 4
Marital status:		
Married	02 (10.0)	04 (20.0)
Unmarried	18 (90.0)	16 (80.0)
Occupation:		
Student	07 (35.0)	06 (30.0)
Labour	06 (30.0)	06 (30.0)
Service	03 (15.0)	03 (15.0)
Business	04 (20.0)	05 (25.0)
Academic qualification:		
Literate	01 (5.0)	01 (5.0)
Primary	06 (30.0)	06 (30.0)
Secondary	08 (40.0)	08 (40.0)
Higher	05 (25.0)	05 (25.0)
Past history of STD	05 (25.0)	05 (25.0)
No. Of sexual partner:		
Single	08 (40.0)	11 (55.0)
Multiple	12 (60.0)	09 (45.0)
Type of sex-partner:		
Regular	00 (00)	04 (20.0)
Casual Professional	20 (60.0)	15 (75.0)
Casual non-professional	00 (00)	01 (5.0)

Of the 40 patients enrolled in the study, 18 in the azithromycin-treated group and 17 in the doxycycline-treated group were evaluable for clinico-laboratory evaluation at 3 weeks after starting treatment.

Table II. Overall outcome of two treatment groups 3 weeks after treatment

Outcome	Azithromycin No. cured/ no. evaluable (%)	Doxycycline No. cured/ no. evaluable (%)
Clinical cure	16/18 (88.9)	12/17 (70.6)
Laboratory cure	17/18 (94.4)	15/17 (88.2)
Combined	15/18 (83.3)	12/17 (70.6)
Drug reaction	07/18 (38.9)	06/17 (35.3)

Overall clinical cure rate was 88.9% in azithromycin-treated group & 70.0% in doxycycline-treated group. Overall laboratory proven cure rate was 94.5% in azithromycin-treated group and 88.2% in doxycycline-treated group. Combined clinical and laboratory proven cure rate was 83.3% in azithromycin-treated group and 70.6% in doxycycline-treated group. Eradication of *C. trachomatis* (proven by chlamydial antigen detection by immunochromatography) was 94.4% in azithromycin-treated group and 94.1% in doxycycline-treated group. Mild to moderate degree of adverse drug reactions were observed in 38.9% patients in azithromycin-treated group and 35.3% patients in doxycycline-treated group, which did not require any treatment interruption.

Discussion

In our routine practice, the NGU patients diagnosed as chlamydial urethritis are treated empirically, generally with either doxycycline 100mg twice daily for 7 days or Tetracycline 500mg or Erythromycin 500mg 6 hourly daily for 7 days. For the advantage of dose schedule, it is important to ascertain whether azithromycin 1gm single dose given orally to treat chlamydial urethritis is effective, which was previously demonstrated in some study.[6,7] Our study results demonstrate that a single 1gm oral dose of azithromycin is as effective as the standard 7 days doxycycline therapy in achieving both clinical as well as laboratory proven cure. Both drugs were found equally effective in eradicating *C. trachomatis*. Both the regimens were equally well tolerated except for some mild to moderate degree of adverse drug-reactions like

nausea, headache, diarrhea and abdominal pain, but required no treatment interruption. The potential advantage of single dose azithromycin therapy must be weighed against its increased cost. But success of treatment depends on patient compliance with the drug administration schedule. Particularly, those patients who are asymptomatic are unlikely to comply with a long course of antibiotic. So, in these cases where compliance is likely to be of concern, the use of single dose azithromycin may be justified.

Our study was done with a small sample size in both treatment groups. Further study on larger sample sizes representative of chlamydial urethritis with long-time follow up should be done to establish the advantage of single dose azithromycin in the treatment of chlamydial urethritis. Also further approach is required to resolve the problem of persistent urethral inflammation evidenced by persistent urethral discharge and dysuria, which occurred despite the eradication of *C. trachomatis*.

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ORIGINAL ARTICLE

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Role of Histopathological Examination For the Diagnosis of Skin Diseases in Dhaka City

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Abstract

To find out clinico-histopathological correlation for the diagnosis and confirmation of various skin diseases in Dhaka city, 35 patients were randomly selected for biopsy irrespective of age, sex and skin disease, considering patients acceptance and need for biopsy. Reports of two separate biopsy specimens were collected for the same disease from two clinically similar lesions. Inspite of clinico-histo-pathological variations in various dermatoses, histopathological examinations were found helpful for establishment of diagnosis of skin diseases in Dhaka city. Reports from histopathologists, who are known to be more experienced in reporting dermato-histopathology, are found more acceptable in their reported cases. Better utilization of dermato-histopathological facilities, with its further development is discussed.

[OMTAJ 2002; 1(2): 52-54]

Introduction

Microscopic examination of skin tissue is the most important single diagnostic ancillary technique used in the diagnosis and management of a patient's skin disorder.[1] The correlation of clinical findings with the histological appearances is not only of direct benefit to individual patients but has led to the recognition of many new skin disorders.[1] The science and art of dermatopathology had its beginnings in early 19th century in Europe with the writings of pioneer dermatologists like Simon, Unna and Gans. Tradition of dermatologists writing about histopathological aspect of skin disease was carried on by individuals such as F. Pinkus, A. Civatte, J. Darier and W. Lever. In this century major contributions to the discipline have been made by British dermatopathotogists.[1,2]

Close co-operation between the clinical dermatologist and the diagnostic dermatopathologist is not only desirable but also essential. The spectrum of skin disease is huge and although in many conditions the histological features are pathognomic of a particular skin disorder, in others, the changes may be characteristic but not specific for one disease. Only by a close liaison between the disciplines of clinical dermatology and histopathology can the usefulness and limitations of skin biopsy examination be appreciated.[3] The clinician who reviews the histology of a biopsy carried out by himself or herself appreciates the problems of interpretation of an inadequate biopsy, a biopsy from an inappropriate or unrepresentative lesion and the effect and artifact caused by undue trauma at the time of biopsy. The pathologist intern can learn with experience that features once signed out as "non-specific dermatitis" are in fact specific for certain disorders.[4,5] For determination of clinico-histopathological correlation in the diagnosis and confirmation of various skin diseases, a study was carried out in Skin and VD department of Dhaka Medical College Hospital and Bangabandhu Sheikh Mujib Medical University during the year 2001. Here we will describe our results with conclusions and implications.

Methods

The patients were randomly selected for biopsy irrespective of age, sex and skin diseases whenever feasible (considering patients' acceptance and need for biopsy). From new patients, two separate biopsy specimens were collected from similar lesion or same lesion and sent to two histopathologists for histopathological examination. The patients who have had a previous histopathological examination report, only one biopsy specimen was taken and sent for reporting. Histpathologists were also requested to supply the slide for academic interest. Details of clinical information, including provisional and differential diagnosis was supplied to histopathologists, as far as

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possible. Both punch and scalpel biopsies were done, considering requirement and patient's compliance. No histopathologists gave any comment about inadequacy of biopsy specimen or insufficiency of clinical information.

Results

It was found that inspite of clinico-histopathological variations in various dermatoses, histopathological examination is found helpful for establishment of diagnosis of skin diseases in Dhaka city (Table-1).

Table-1: Result of histopathological examination in 35 cases

Sources	No. of cases	Results
Known to be more experienced	16	Helpful
Known to be less experienced	6	Helpful
Both	4	Helpful
Both	9	Not helpful

Among 35 cases, histopathological report differs from one another in 27 cases, but it was found that histopathologists, who have more experience in reporting dermatological conditions, made more acceptable and appreciable histopathological reports.

Out of 35 cases, histopathological reporting was found helpful in 26 cases (16 by experienced, 6 by less experienced and 4 by both). Histopathological reporting helped us for the diagnosis of six uncommon diseases. Namely

1) Connective tissue nevus
2) Subcorneal pustular dermatosis
3) Subacute cutaneous lupus erythematosus
4) Extensive hypertrophic seborrhoeic keratosis.
5) PKDL with only hypopigmentation.

We also failed to establish extensive macular atrophy, eruptive xanthoma, extensive psoriasis vulgaris, hypotrophic lichen planus and some other conditions by histopathology (Table-2).

Table-2: Clinico-Histopathologial correlation in 35 dermatological cases.

Case No.	Clinical diagnosis	Histopathological diagnosis	
		1st Histopathologist	2nd Histopathologist
1	Psoriasis	Subacute Dermatitis	Psoriasis
2	Eczema	Lichenplanus	Lichenplanus
3	Scleroderma	Scleroderma	Scleroderma
4	Post-kala-azar dermal leishmanoid	Tuberculosis	Post-kala-azar dermal leishmanoid
5	Tuberculosis verrucosa cutis	Chronic Nonspecific Dermatitis	Tuberculosis verrucosa cutis
6	Connective tissue nevus	Normal skin	Connective tissue nevus
7	Seborrhoeic Dermatitis	Seborrhoeic Dermatitis	Nonspecific Dermatitis
8	Subacute cutaneous lupus erythematosus	Nonspecific Dermatitis	Subacute cutaneous lupus erythematosus
9	Haemangioma	Haemangioma	Haemangioma
10	Lichenplanus	Nonspecific Dermatitis	Lichenplanus
11	Lichenplanus	Nonspecific Dermatitis	Lichenplanus
12	Lichenplanus	Nonspecific Dermatitis	Lichenplanus
13	Hypertrophic Seborrhoeic keratosis	Hypertrophic Seborrhoeic keratosis	Acrokeratosis verruciformis of Hopf
14	Tuberculosis verrucosa cutis	Viral wart	Tuberculosis verrucosa cutis
15	Lupus Vulgaris	Foreign body granuloma	Lupus Vulgaris
16	Tuberculoid leprosy	Nonspecific Dermatitis	Tuberculoid leprosy
17	Subcorneal pustular dermatosis	Subcorneal pustular dermatosis	Nonspecific Dermatitis
18	Tuberculosis verrucosa cutis	Tuberculosis verrucosa cutis	Benign Lichenoid Keratosis
19	Prurigo simplex	Prurigo Simplex	Nonspecific Ulcers
20	Post-kala-azar dermal leishmanoid	Post-kala-azar dermal leishmanoid	Chronic Nonspecific Dermatitis
21	Dermatitis herpetiformis	Dermatitis herpetiformis	Psoriasis Vulgaris
22	Leukoplakia	Pseudocarcinomatous hyperplasia	Nonspecific Dermatitis
23	Lichen sclerosus et atrophicus	Lichen sclerosus et atrophicus	Lichen sclerosus et atrophicus
24	Xanthoma	Xanthoma	Xanthoma
25	Lichenplanus	Lichenplanus	Lichenplanus
26	Lichenplanus	Lichenplanus	Lichenplanus
27	Macular Atrophy	Scleroderma	Lichen sclerosus et atrophicus
28	Prurigo	Lichenplanus	Nonspecific Dermatitis
29	Lichenplanus	Nonspecific Dermatitis	Prurigo simplex
30	Psoriasis	Pityriasis rosea	Pityriasis rosea
31	Dermatitis herpetiformis	Nonspecific Dermatitis	Disseminated infundibulofolliculitis
32	Dermatitis herpetiformis	Seborrheic keratoses	Acrokeratosis verruciformis of Hopf
33	Porphyria cutanea tarda	Nonspecific Dermatitis	Acrokeratosis verruciformis of Hopf
34	Hypertrophic lichen planus	Psoriasis	Acrokeratosis verruciformis of Hopf
35	Eruptive Xanthoma	Nonspecific Dermatitis	Acne vulgaris

Discussion

People differ; this is true of diseased individuals in terms of their lesions, both macroscopic and microscopic. It is also true of clinicians and histopathologists in terms of their observation and interpretations of these lesions. The study reported here provides an attempt to assess the magnitude of such differences as they affect the diagnosis of various dermatological conditions. What is important is to consider the nature and trends of disagreements in order to be able to apply these results for the improvement of diagnosis of different skin diseases.

Close co-operation between the clinical dermatologist and the histopathologist is in fact very poor in Dhaka city. Actually no specialized dermatopathologist is available in our country. But still those histopathologists, who worked more in dermatopathlogy, are reporting better and helping clinical dermatologist in establishing the diagnosis of various dermatological conditions. An urgent need for improvement of close co-operation among dermatologists and histopathologists those who are reporting histopathology of skin are beyond discussion. We think better utilization of histopathologists, known to be more experienced in dermatopathology is above all essentials. Histopathologists, who are known to be less experienced, can be made more experts by providing further planed-training in dermatopathology and thereby we can utilize their highest potentiality. Appropriate training for dermatologists in dermatopathology can also improve clinicohistopathological correlation. Last of all, establishment of dermatopathology as a separate subject and appointment of those specialists in close collaboration with dermatologists will be very much helpful. Establishment and better utilization of improved techniques like histochemical staining, polaroscopic examination, immunofluorescence testing, immuno-histopathology, electron microscopy etc. will be undoubtedly very much essential.

It is hoped that increasing number of clinicians and pathologists will become interested and expert in the discipline of histopathology of the skin and discover it to be both exciting and fun.

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ORIGINAL ARTICLE

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Drug resistance of *Escherichia coli*-Experience in Chittagong Medical College

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Abstract

Escherichia coli isolated from urine and pus was tested for antimicrobial susceptibility against commonly used antibiotics. About 90% of urine isolates and 100% of pus isolates were resistant to ampicillin. Least resistance was found against ceftriaxone (30% in urine isolates & 40% in isolates of pus). Resistance against other antibiotics was found varying from 40% to about 75%.

[OMTAJ 2002; 1(2): 55-56]

Introduction

Drug resistance is a nightmare of modern medical science. The microbes are quickly developing resistance and a drug is remaining freely usable for only a few years. Wide and inappropriate use of antibiotics facilitates this resistance.[1]

E. coli is the most common organism isolated from different samples. This study shows the resistance pattern of *E. coli* against commonly used antibiotics.

Methods

Samples used were Urine and Pus sent to the Department of Microbiology, Chittagong Medical College, Chittagong. Isolation and identification was done as per standard methods.[2]

Media used for Antibiotic susceptibility was Mueller-Hinton agar.[3,4]

Antibiotics used were: Ampicillin, Cotrimoxazole, Gentamycin, Nitrofurantoin, Nalidixic Acid, Ciprofloxacin, Ceftriaxone and Cephalexin. Antibiotic discs were procured from Oxoid Ltd., England.

Control Organism used was *E. coli* ATCC25922.[3,5]

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The method followed for antimicrobial susceptibility test was NCCLS (National Committee for Clinical Laboratory Standards) disk diffusion method (a modified Kirby-Bauer Method).[3,4] Briefly the method is as follows: Similar 2 to 3 colonies were suspended in sterile normal saline and the turbidity was adjusted to match with that 0.5 McFarland standard. Using a sterile swab stick, inoculation was given over Mueller-Hinton Agar. Then the above-mentioned antibiotic disks were placed on the media appropriately and incubated at 37°C for 18-24 hours. The diameter of zone of inhibition of control organism as well as test organism was compared with respective standard table for zone diameter. The result was interpreted as Sensitive (S), Intermediate (I), and Resistant (R).

Result

A total of 52 *E. coli* strains were isolated from specimens of urine and pus, of which 41 were isolated from urine and 11 from pus.

Among urine isolates, 90.2% of the *E. coli* strains were resistant to Ampicillin, 73.2% to Co-trimoxazole and 70.7% to Nalidixic acid. The lowest resistance was found against Nitrofurantoin (29.3%), followed by Ceftriaxone (30.7%). Ciprofloxacin was resistant against 56.3% isolates (table-1).

Table-1: Antimicrobial resistance pattern of *Escherichia coli* isolated from urine

Drugs	Sensitive	Intermediate	Resistant
Ampicillin (N=41)	4 (9.8)	0 (0)	37 (90.2)
Cotrimoxazole (N=41)	10 (24.4)	1 (2.4)	30 (73.2)
Nitrofurantoin (N=41)	24 (58.5)	5 (12.2)	12 (29.3)
Nalidixic acid (N=41)	10 (24.4)	2 (4.9)	29 (70.7)
Cephalexin (N=7)	2 (28.6)	2 (28.6)	3 (42.8)
Ceftriaxone (N=13)	6 (46.2)	3 (23.1)	4 (30.7)
Ciprofloxacin (N=32)	12 (37.5)	2 (6.3)	18 (56.3)
Gentamycin (N=12)	7 (58.3)	0 (0)	5 (41.7)

Note: S= Sensitive, I = Intermediate, R = Resistant. Figures in parenthesis indicate percentage

Among isolates of pus, all (100%) of the *E. coli* were resistant to Ampicillin, 67% to Cephalexin and 64% to Co-trimoxazole. The lowest resistance was found against

Ceftriaxone (40%) and Gentamycin (40%). Resistance to Ciprofloxacin was 60% (table-2).

Table-2: Antimicrobial resistance pattern of *Escherichia coli* isolated from pus

Drugs	Sensitive	Intermediate	Resistant
Ampicillin (N=11)	0(0)	0 (0)	11 (100)
Cotrimoxazole (N=11)	4 (36.4)	0 (0)	7 (63.6)
Cephalexin (N=9)	1 (11.1)	2 (22.2)	6 (66.7)
Ceftriaxone (N=10)	6 (60)	0 (0)	4 (40)
Ciprofloxacin (N=10)	4 (40)	0 (0)	6 (60)
Gentamycin (N=10)	6 (60)	0 (0)	4 (40)

Note: S= Sensitive, I = Intermediate, R = Resistant. Figures in parenthesis indicate percentage

Discussion

Drug resistance develops as result of mutation of microbial genes. The role of drugs in origin of drug resistance is only to eliminate the sensitive bacteria favouring the growth of resistance ones.[1] Widespread use and abuse of the drugs has lead to the survival and spread of drug resistance by microbes.[6] This study shows the situation of drug resistance in Chittagong Medical College and Hospital. As *E. coli* is the commonest organism isolated from different samples, its resistance pattern will reflect the situation of drug resistance in the institution. In this study, 100% of pus isolates (*E. coli*) and 90% of urine isolates (*E. coli*) were found resistant to ampicillin. So, this drug virtually has become useless against this organism. In case of Cephalexin, Cotrimoxazole and Nalidixic acid the resistance was varying from 40 to 74% in case of both urine and pus isolates. Even the latest two groups of drugs, the fluoroquinolones (e.g. Ciprofloxacin) and third generation Cephalosporins (e.g. Ceftriaxone) are on the face of great challenge by the microbes (resistance varies from 55-60% in case of Ciprofloxacin and from 30-40% in case of Ceftriaxone). Similar pattern of resistance was reported by Hossain[7] in Mymensingh Medical College except that resistance against Ceftriaxone was found in 18% of 25 *E. coli* studied. Another study, Carried out in a hospital of Dhaka city, shows that 70% of 310 *E. coli* isolated from urine were resistant to Ampicillin, Cephalexin, Doxycycline, Mecillinum and Nalidixic acid; and about 30-40% were resistant to Ceftriaxone, Gentamycin, Ceftazidime and Ciprofloxacin.[8] In study of Karim et el[9], carried out in tertiary hospital of Dhaka city, very high incidence of resistance was noted in *E. coli* against Cephalexin (84% in urine and 96% in pus isolates), Ceftriaxone (86% in pus isolates) and Ciprofloxacin (80% in urine and 90% in pus isolates).

So, it now becomes evident that very tough days are coming when eradication of pathogens with sensitive antibiotics might become difficult. We should be very rational in antibiotic use from right now. Antibiotics should be used only whenever necessary, and with the help of culture and sensitivity test. Following are several measures that can be adopted to prevent emergence of drug resistance:[10] (1) Maintain sufficiently high level of the drug in tissue to inhibit both the original population and 1st step mutants. (2) Administer two drugs simultaneously (that do not give cross-resistance), each of which delays the emergence of mutants resistant to other drug, and (3) Avoid exposure of microorganisms to a particularly valuable drug by limiting its use especially in hospitals.

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REVIEW ARTICLE

The Osmani Medical Teachers Association Journal Volume 1 Number 2

Dengue Virus : A Review

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Introduction

The reemergence of dengue viruses has been very dreadful in recent times. The term "Dengue" has its origin in Zanzibar, where the disease was called 'Denga' during 1870 epidemic.[1] There are four entities which comprises the Dengue syndrome. These are Undifferentiated Fever (UF), Dengue Fever, Dengue Haemorrhagic Fever (DHF) and Dengue Shock Syndrome (DSS).[2]

So far there are four virus serotypes (Type I, II, III, IV) and they are grouped under the family Flaviviridae. Dengue viruses share many characteristics with other flaviviruses. These viruses are RNA viruses having capsid and envelope. The virion is 50nm in diameter. Infection in human by one serotype produces life-long immunity against re-infection by the same serotype, but only temporary and partial protection against the other serotypes.[3,4]

Dengue fever is marked by a sudden onset of high fever, severe headache, and pain behind the eyes and myalgia /arthralgia. The symptoms and signs may be very similar to other viral infections. It occurs in epidemic form in most countries of Asia and other Pacific islands. Children below 15 years are the common susceptible victims. Two types of Aedes mosquitoes are the vector of dengue virus, which are *Aedes aegypti* and *Aedes albopictus*. *A. aegypti* is the vector of urban and later one is a rural vector. Aedes mosquitoes breed in clean still stagnant water. Usually discarded tins, water tanks, flowerpots are the ideal places for their breeding. Dengue epidemic in developing countries is due to many reasons. The disposal of sewage, method of water storage and most importantly nutritional status of general population are important reasons for these viral infections.[4,5,6]

Bangladesh at the moment has experienced this viral infection in a most horrific manner. This review paper highlights some events within the country. So far, in Bangladesh, only sporadic cases were diagnosed through small-scale surveys that actually failed to unearth the real situation in Bangladesh. The first scientifically designed survey was conducted in Chittagong in 1996-97. The positive rate was 7.1% among the selected patients.[6,7] This survey shows an average ELISA positive rate of 17.5% that warrants public health warning in Bangladesh. Chittagong being the most industrialized city, showed 34.3% of all reactive samples.[8,9] Khulna is another industrial area presented the second highest number of reactive sample (31.45%).[8]

Historical Aspects

The classical form of Dengue has been known for more than a century in the tropical South East Asia, and Western Pacific Regions.[1] Dengue Haemorrhagic fever was reported as a new disease for the first time in the Philippines in 1953. Serotypes 2, 3 and 4 were isolated in 1956. Multiple infections were followed in 1958 in Thailand, in 1970 in Myanmar and finally in India in 1963. In 1965 there was an outbreak of Dengue and 'Chikungunga' virus infection called 'Dhaka fever' which was the first documented out-break of Dengue in Bangladesh.[10] A WHO sponsored small scale survey also detected Dengue Haemorrhagic fever cases in 1982. It is difficult to predict why this virus was reactivated in Bangladesh. Probably seasonal occurrence such as monsoon-rain is ideal for breeding. The best environment conditions for mosquitoes breeding prevail during pre- and post-monsoon periods in the tropical zones. Aedes eggs can survive in dry condition for a year.[11]

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WHO currently estimates that there may be 50 million to 100 million cases of dengue infections worldwide every year. Two fifth of world's population are at risk of infection. In Bangladesh last year experienced the worst dengue outbreak, and the majority of cases being in the capital. It was estimated 5,500 people were infected with 98 deaths. In Dhaka since 1995, high percentages of population in Dhaka and Chittagong have antibodies against type 3 and to a lesser extent to types 1 and 2. Reports show that there are about 500,000 cases of dengue haemorrhagic fever (DHF) each year, which require hospitalization. Over the last 10-15 years, Dengue Fever and Dengue Haemorrhagic Fever has become a leading cause of hospitalization and death among children in South East Asian regions followed by Diarrhoeal diseases and Acute Respiratory infections.[12]

Social and Environmental Aspects

Diseases control unit of the Health Services of Bangladesh has documented that there are 5,575 cases of Dengue infection and 90 deaths have been reported.[13, 14] In comparison to Malaria, Tuberculosis, Leprosy, Filariasis, Diarrhoeal diseases, Leishmaniasis, there is no significant differences with Dengue infection in Bangladesh.[15] Dengue infection involves mostly the affluent section of the society indicating it is an urban disease. Usually there is negative correlation between the infection and the under nutrition.[16]

The peculiarity of the vector has close link with human habitation. Female Aedes mosquitoes are the vector of the virus and are peridomestic in nature. The tropical zone of the world between 35^{0}N and 35^{0}S latitude and area not over 1,000 ft. above sea level is the usual habitat, the area are marked by monsoon-rains. The breeding of the mosquitoes is highest during pre- and post-monsoon periods.[17]

Aedes breeds in clean, still, and stagnant water usually discarded tyres, water tanks and storage appliances are the ideal sites for breeding. Aedes is a voracious bloodsucker, which helps more virus transmission during blood meal. Biting occurs throughout the day especially marked at 8:00 a.m. to 13:00 p.m. and between 15:00 p.m. to 17:00 p.m. Therefore, late risers and late evening sleepers are more susceptible to mosquito bites. The mosquito sucks blood many times and therefore, it can infect many persons.[18,19]

Like all vector-borne diseases, Dengue also needs some conducive predisposing conditions for endemicity and outbreaks. The countries of South-East Asia share common features like large populations, rapid urbanization, development activities and monsoon-rains.[20] Urban human populations now constitutes the natural reservoir, travelers are the only disseminating factor of the viruses from one country to another. The spreading mechanisms of the outbreaks are related to the dramatic increase of travels and the variable susceptibility of natural Aedes population to the virus. It has been found that number of female *Aedes aegypti* per person is a very significant household risk factor.[21]

A survey in Bangladesh, Dhaka city showed that independent houses were most likely to have high densities of Aedes mosquitoes. It appears that rooftops concrete water container are one of the main breeding sources in independent houses.[22,23] In one study in the city of Chittagong from September 1996 to June 1997 among 255 positive cases shows a seasonal variation in Dengue Patients (Table 1).[24]

Table-1: Seasonal occurrences of positive cases

Season	Percentage of patients
PRE-MONSOON	28.5 %
MONSOON	25.7%
POST-MONSOON	45.7%

PATHOLOGY/PATHOGENESIS

There are generalized haemorrhagic manifestations in skin, subcutaneous tissues, mucosa of gut, heart and liver, but cerebral or subarachnoid area are rarely involved. The mechanism involved is increased vascular permeability that gives rise to loss of plasma from vascular compartment. As a result there is haemo-concentration, low pulse pressure and other signs of shock. Secondly, there is disorder in haemostasis leading to thrombocytopenia.[24]

There is remarkable depression of complement system like C3 and C5 level. Platelet defects may be both

qualitative and quantitative. Therefore, patients with platelets count greater than 100,000 per mm^3 may still have a prolonged bleeding time. The virus replicates in macrophages by heterotypic antibodies. The other serotypes produce cross-reactive antibodies, which ultimately fail to neutralize the virus. As a result, the infected monocytes increase in number.[24]

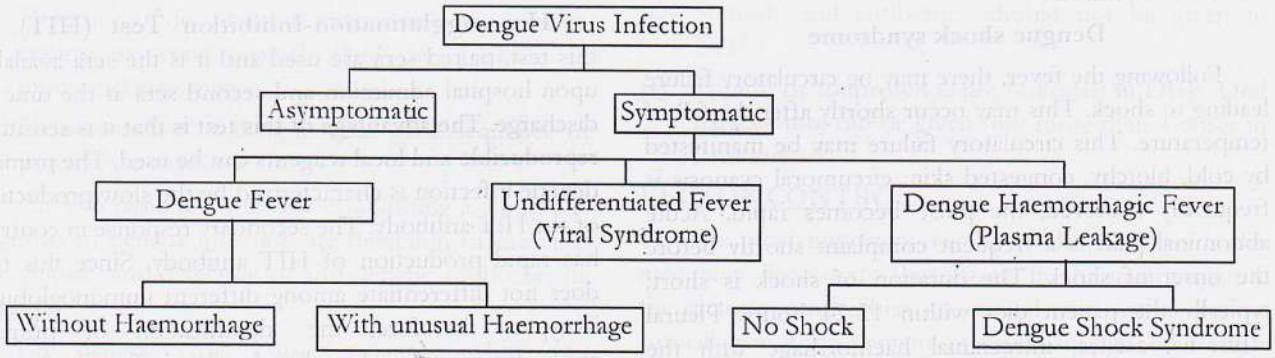
Clinical Diagnosis

The dengue virus can manifest itself in four ways. They are (1) undifferentiated fever, (2) Dengue fever, (3) Dengue haemorrhagic fever, and (4) Dengue shock syndrome.

It can be asymptomatic as well. It is most common in older children and adults.

The clinical and laboratory criteria for diagnosis of Dengue haemorrhagic fever were developed by the children's hospital, Bangkok, which have been adopted by the WHO for worldwide distribution since 1975 and are based on the presence of major manifestation in order of appearance as follows: [5]

1. High continuous fever for 2-7 days.
2. Positive tourniquet test.
3. Enlargement of liver
4. Circulatory disturbance- Shock
5. Thrombocytopenia ≤ 100,000 cells/ mm^3
6. Increased haematocrit by 20% (Packed cell volume)
7. Plasma leakage: Pleural effusion, Ascites.



Common differential diagnoses of dengue depending upon type of presentation and syndromes are: Influenza, Japanese-B encephalitis, Rubella, Malaria, Leptospirosis and Hepatitis. All these diseases are common in our country. Following an infection, a person develops immunity for that particular type of virus. In subsequent re-infection the patient develops spontaneous bleeding. This serious condition is called Dengue haemorrhagic Fever.[11]

The other bleeding manifestations are: bleeding gum, haematemesis, epistaxis, malena, subconjuntival haemorrhage (48.29%), cutaneous bleeding (44.83%).[24]

Dengue Haemorrhagic Fever (DHF)

DHF is not a complication of Dengue fever (DF), rather it can start from the very onset of the disease. The turning point between DF and DHF is an afebrile period, when DF progresses to remit but DHF presents its morbid manifestations. One cannot differentiate between DHF and DF at the very beginning.

Dengue Shock Syndrome is a complication of DHF. So, it is preferable to encompass all the entities as Dengue syndrome (DS) till differentiating manifestations are surfaced.[25]

There are four cardinal signs, which help the diagnosis of this variety.

(a) High fever
(b) haemorrhagic phenomena
(c) Hepatomegaly
(d) Circulatory failure

Haematocrit value is the clue for the diagnosis and management of the dengue haemorrhagic fever. If there is leakage of plasma, the haematocrit value is elevated resulting into haemoconcentration, effusion or hypoproteinaemia. These clinical conditions differ markedly from Dengue fever.[3, 5]

In children, the temperature rises above 39°C and remains high for 2-7 days. Febrile convulsion may occur in infants. Some 96.8% of patients have infected pharynx and sore throat. Rhinitis and maculopapular rash /myalgia comprise about 12.0%. [7]

The most common haemorrhagic phenomenon is positive tourniquet test. This test is performed by inflating a blood pressure cuff on the upper arm to a point midway between the systolic and diastolic pressure for about 5 minutes. A test is positive when 20 or more petechiae per 2.5 cm^2 are observed.[8]

The liver is usually palpable early in the febrile stage and is tender and jaundice is usually absent. The spleen may be prominent on the x-ray investigations.[6]

After 7 days, the fever falls followed by sweating and cool extremities.

Dengue shock syndrome

Following the fever, there may be circulatory failure leading to shock. This may occur shortly after the fall of temperature. This circulatory failure may be manifested by cold, blotchy, congested skin; circumoral cyanosis is frequently observed, the pulse becomes rapid. Acute abdominal pain is a frequent complaint shortly before the onset of shock. The duration of shock is short; typically the patient dies within 12-24 hours. Pleural effusions, ascites, intracranial haemorrhage with the development of metabolic acidosis are the manifestations of Dengue shock syndrome. Intracranial haemorrhage with electrolyte disturbances can also be the important findings of Dengue shock syndrome.[6]

Good prognostic signs are adequate urine output and the return of appetite.[6]

Laboratory Diagnosis

a. Isolation of virus: During the period of viraemia, the dengue virus can be isolated in the following way.

Inoculation into mosquito. Antigen can be detected by immunofluorescence from mosquito.

Cell culture. Detection of antigen by antibody staining, cytopathic effects, and plaque formation.

Inoculation into suckling mice. Sign and symptom of encephalitis develop.

Antigen detection in fixed tissue. They can be detected in peripheral blood leukocytes especially during the febrile phase of illness. Dengue antigen can also be found in liver and lung at autopsy.

b. Serological tests:

Reverse Transcriptase - PCR amplification of Dengue genotype RNA. The advantage is that during convalescence, when circulating antibodies interfere in the diagnosis, the dengue virus RNA genotypes in the blood circulation can be identified. But the test is highly prone to false positive results due to contaminations.[18]

Mac ELISA. This test helps in diagnosis of primary and secondary dengue infection, where the haemagglutination inhibition antibody is not confirmed. In addition, the detection of anti-flaviviral IgM in cerebrospinal fluid can be recognized. It is important that antiflavivirus IgM can be produced due to other virus such as West Nile virus. Therefore, there can be cross-reactivity. Mac-ELISA is more sensitive test and the disadvantages are very little.[18]

Haemagglutination-Inhibition Test (HIT). In this test, paired sera are used and it is the sera available upon hospital admission and second sera at the time of discharge. The advantage of this test is that it is sensitive, reproducible and local reagents can be used. The primary dengue infection is characterized by the slow production of the HIT antibody. The secondary response in contrast has rapid production of HIT antibody. Since this test does not differentiate among different immunoglobulin isotypes, therefore, the identification of primary antibody response is based on low level of antibody titre. The closely related flavivirus such as Dengue virus, Japanese encephalitis virus, and West Nile virus; can not be differentiated by this HIT.[18]

Both Mac ELISA and HIT techniques are used in Dengue antibody detection. HIT is the gold standard test. It is rather difficult to perform. It has to be done in the well-set laboratory. It needs two sera specimens (at least 10-14 days apart) for making diagnosis. The definitive diagnosis is four-fold rising in the antibody. The ELISA test is easy to perform. There are many rapid slide tests (ELISA kit), which takes only 5-10 minutes for the results. The diagnosis is rising IgM antibody to more than 40 U [personal communication with WHO consultant in Bangkok].

Neutralization test. The most sensitive and specific test is the serum dilution, virus constant, and plaque reduction tests. In early convalescence, specific neutralizing antibodies are detected, after primary infections. In secondary dengue infection, high titre neutralizing antibody is produced against all four dengue virus serotypes and other flaviviruses.[18]

Dot-blot Immunoassay. This is a new technique and at least one blot immunoassay for dengue antibodies is available.[18]

Complement fixation test. It is the least sensitive than other tests. Complement fixing antibody typically appears after IgM or HI antibody and it is usually more specific. The advantage is that it helps in the diagnosis in late dengue infection from paired serum.[18]

SUMMARY OF LABORATORY DIAGNOSIS:

The dengue fever can be established under the following guidelines:

(a) The dengue virus can be isolated from serum and autopsy.

(b) The detection of IgM or IgG titre (four fold greater) in paired serum samples.

(c) Detection of dengue virus antigen in autopsy tissue, serum or cerebrospinal fluid by ELISA, or immunofluorescence.

(d) Demonstration of dengue virus gene sequence by polymerase chain reaction.

The two basic methods for establishing a laboratory diagnosis of dengue infection are detection of the virus (e.g. culture) and detection of anti-dengue antibodies (in serum).[6]

The dengue virus causes viraemia, which last roughly 2-7 days. The virus infects the peripheral blood mononuclear cells. The antigen staining is usually 1-10 infected cells per 10,000 cells. The corresponding antibodies appear after several days of fever. IgM is the main isotype, which can be detected by Mac-ELISA. 80-90% of IgM can be identified within 5-10 days. Anti dengue IgG appears shortly afterwards. Both IgM and IgG antibodies neutralize dengue virus. The neutralizing antibodies rapidly increase as fever subsides and can therefore interfere with isolation of the virus from serum.[7,8]

Management

The management of Dengue haemorrhagic fever is largely based upon fluid therapy (both oral and infusion) to which the Haematocrit value is the key guide to fluid and infusion.[9,10]

The general management of these patients needs teamwork and a separate ward. The ward should have mosquito net to prevent nosocomial Dengue transmission. The patient needs special laboratory investigation when they are high-risk subjects. Such individuals are young infants (<1 year old), DHF, overweight, encephalopathy, thalassemia, Glucose-6-Phosphate Dehydrogenase deficiency and congenital heart diseases.[4]

General guidelines in the treatment of Dengue Haemorrhagic Fever (DHF) are as follows:[9,10,11]

(a) Hourly clinical assessment of the patient of DHF should be done.

(b) Haematocrit determination and platelet count are important for the early diagnosis of DHF.

(c) If the haematocrit is increasing, fluid should be change to colloidal solution (Dextran). If the haematocrit is decreasing, the fresh whole blood transfusion is necessary.

(d) Oxygen can be administered for shock and sodium bicarbonate should be given for acidosis.

(e) Steroids and antibiotics should not be given in DHF.

(f) Aspirin or Ibuprofen is not indicated in DHF. Oral paracetamol can be given (not more than 4 doses in 24 hours).[18]

VECTOR CONTROL:

The most important vector of dengue virus is the mosquito *Aedes aegypti* that should be the main target of the vector control. There are several ways such as vector surveillance, improvement of water supply and storage solid waste management chemical controls. Besides DDT, organophosphate insecticides such as fenthion, malathion, can be used. Larvicidal agents can be used also in containers having drinking water. These chemicals are 1% temephos sand granules. All Larvicidal agents have extremely low biological toxicity.[25]

Conclusion

The prevention of dengue fever is largely based upon the identification of risk factors and awareness. Factors responsible are overpopulation, uncontrolled urbanization, inadequate waste management.[1]

Seroepidemiological survey showed that dengue infection has both primary and anamnestic infection. Studies have shown that the risk of developing of Dengue Shock Syndrome following anamnestic dengue infection with Dengue type-2 was 14 times greater than Dengue shock syndrome due to type 1, 3 and 4. The role of complement, cytokines and IgG 1 in the pathogenesis of Dengue shock syndrome is under investigation, particularly to type-2, which is of epidemiological importance.[5] Thus, series of experiments will aid in the development of an appropriate vaccine. It is important

to bear in mind that Aedes mosquito is also a vector for Yellow Fever. The main threat of Yellow fever is the periodic invasion of the virus to densely populated urban areas where it can be transmitted by human biting species.[26] Therefore, it can be anticipated the future impact of this tropical disease in Bangladesh. The foremost essential step regarding the prevention of this deadly dengue is the identification and mode of Aedes mosquito breeding and the method of spraying insecticide/larvaecide at the appropriate sites. [26,27]

Most Aedes mosquitoes breed within houses where the reach of government interventions is limited. The participation and cooperation of general people with government agencies is essential for Aedes control programs.

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CASE REPORT

The Osmani Medical Teachers Association Journal Volume 1 Number 2

Exfoliative Dermatitis Following Chemotherapy- A Case Report

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A 45 years old lady reported to Skin and venereal disease out patients department in June 2002 with generalized exfoliation of the skin lasting for three months with history of pain in lower abdomen for 10 days. Exfoliation started from both upper limbs with erythematous patch and than progressed to all over the body. She had a history of ovarian tumour operation 5 months back, which was diagnosed as a papillary serous cyst adenocarcinoma of the ovary. Following operation chemotherapy such as Inj. Cistplatin and Inj. Endoxan were administered. She had mild lower abdominal pain with amenorrhoea for 1 year.

The skin lesions were bilaterally symmetrical in distribution with generalized scaling, erythema, discrete skin thickening and hyper-pigmentation. Some of the scales were small and thin, and some were large sheet like. Both oral cavity and genitalia were free of lesion. Scaling was present on scalp with partial loss of hair. No palmoplanter involvement was present. Systemic examination revealed a solid mass in the lower abdomen.

Routine examination of blood and urine were done and found within normal limit except a high ESR of 40 mm. Hg in the 1st hour. Her random blood sugar, blood urea, serum bilirubin, and SGPT were normal. Chest x-ray was normal. Ultrasonography of whole abdomen showed a complex solid mass in pelvic cavity posterior inferior to the urinary bladder, with irregular outline. The patient was diagnosed as Exfoliative dermatitis & treated conservatively in the dermatology unit. She was given antihistamine such as Tab. Cetrizine 10 mg daily, topical steroid, like betamethasone with an emollient liquid paraffin. Satisfactory clinical improvement was observed within few days of starting treatment and then she was referred to Gynaecology department for further evaluation of her underlying malignant condition.

Exfoliative dermatitis or erythroderma is a clinical syndrome characterized by widespread erythema, fine or large scales, and desquamation of a significant portion of the body surface.[1] Various etiological factors responsible for developing exfoliative dermatitis have been described in literatures. In one study, in a series of 60 cases, it was found that most common causative factors were pre-existing dermatoses (58%) and Drugs (16%). [2] In 16% of cases no etiology of exfoliative dermatitis could be found and 11% of the patients were associated with internal malignancy.[2] One of the most common malignancies is cutaneous T-cell lymphoma.[3] Among the pre-existing dermatoses, atopic dermatitis, psoriasis, seborrhic dermatitis, PRP etc. are common. Drugs that may cause exfoliative dermatitis are many. Cisplatin, a chemotherapeutic agent is also included in the drug list.[4]

Our patient had no history of pre-existing dermatoses. But she had a history of ovarian tumour operation 5 months back, which was diagnosed as adenocarcinoma of the ovary. Following operation chemotherapy such as Inj. Cisplatin and Inj. Endoxan were given to her. After that she developed generalized exfoliation of the skin. So, in this particular case, the cause may lie with the drug, which was given to her in the post-operative chemotherapy, i.e., Inj. Cisplatin. Cisplatin induced exfoliative dermatitis has very rarely been reported in literature.[4] Furthermore the original picture of the exfoliative dermatitis might be influenced by the regeneration of the underlying malignant condition of the ovary. So further evaluation of her underlying malignant condition is also stressed.

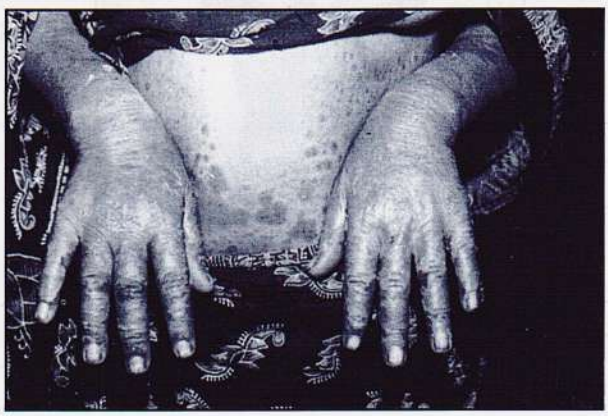

Figure 1: Scaling and exfoliation over abdomen and hands

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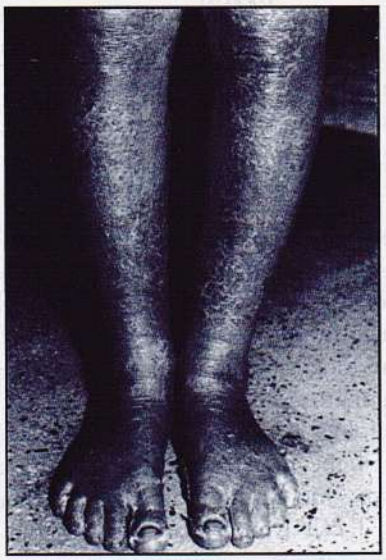

Figure 2: Scaling and exfoliation over legs

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CASE REPORT

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Myotonic Dystrophy-A Case Report

*MATIUR RAHMAN[1], ANISUR RAHMAN[2], IBRAHIM KHALIL[2], P K BHATTCHARRYA[3]

Introduction

Myotonic dystrophy (DM) is the commonest inherited muscular dystrophy affecting adults.[1,2] The incidence of myotonic dystrophy is about 1: 800 live births.[3] Myotonic dystrophy is an autosomal dominant disorder. This disorder is transmitted by a muta-tion consisting of an unstable expansion of CTG trinucleotide repeat sequence in a gene 19 q 13.3.[4,5] Evidence indicates that new mutation does not contribute to the pool of affected individuals. An Increase in severity of the disease phenotype in successive generations (genetic anticipation) is accompanied by an increase in number of trinucleotide repeats sequence. This disorder affects males and females equally.[6] The case presented here is one of the examples.

Case History

Mr. MA Wadud, a thirty five years man presented with complaints of weakness of both hands for three years, difficulty in walking for one year, and difficulty in talking for seven months. His symptoms were gradually aggravating. He first noticed the weakness when he could not carry the weight in his hands with occasional falling down of objects from his hands. After several months the weakness gradually became severe. He faced difficulties in doing fine tasks like buttoning the shirt, writing letter, eating, griping and pinching the objects. But he could raise his hands above the shoulders. He also complained about difficulty in walking for last one year. The patient also noticed that for last seven months he had difficulty in speech and his voice gradually became harsh in nature but he had no complaints regarding swallowing & breathing. Family history was non-contributory. On examination, he had normal intelligence. He had a "hatchet'-faced" appearance with frontal baldness (Figure 1). There was no ptosis, cataract, or ocular myotonia. Tongue, soft palate shows no abnormality. Examination of neck and upper limbs showed wasting of muscles. Wasting was more marked in neck flexors, sternocleidomastoids (Figure 2), flexors and extensors of wrists and fingers, intrinsic hand muscles. There was grip-myotonia as well as percussion-myotonia in both hands. There was no gynaecomastia or testicular atrophy. Examination of lower limbs also showed wasting of muscles along with foot drop. Wasting was more marked distally than proximally. All the jerks were reduced but there was no sensory loss. Haemoglobin concentration was 65%, CBC (complete blood count) was within normal limits, and ESR was 26 mm in first hour. Fasting blood sugar was 98 mg%. Chest X-ray, urine analysis, electrocardiogram were within normal limits. Serum CPK (Creatinine phosphokinase) was 423 mg% and serum aldolase level 56 mg%. Muscle biopsy showed features of muscular dystrophy. So, on the basis of clinical, biochemical and histopathological report, the patient was diagnosed as Myotonic dystrophy.

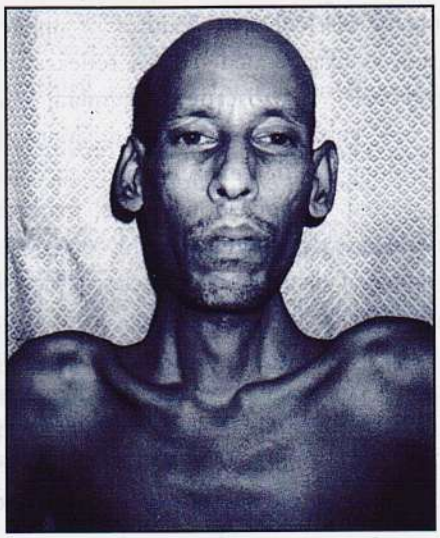

Figure 1: Frontal baldness with hatchet face

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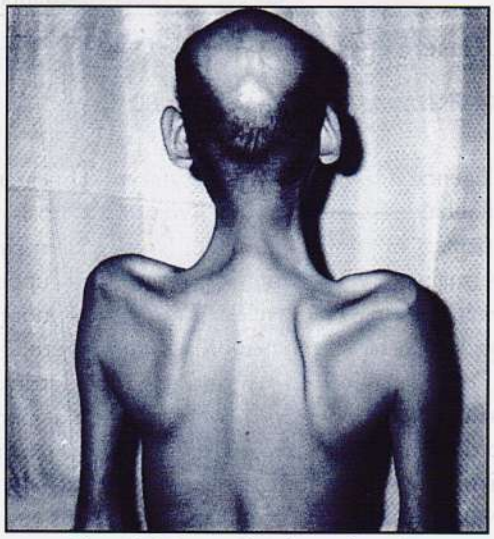

Figure 2: Wasting of neck muscles and winging of scapulae

Discussion

With the above presentation, the following differential diagnoses were considered: Motor neuron disease-gradual onset of weakness, dysphonia, progressive muscular wasting along with intact higher psychic function and sensory function suggested the diagnosis. But motor neuron disease usually occurs after the age of fifty. In motor neuron disease along with much wasting there should be fasciculation, features of upper motor neuron lesion as exaggerated tendon reflexes, extensor planter response.[7] Facioscapulo humeral muscular dystrophy was another consideration. Marked facial weakness resulting in a dull expressionless face, difficulty in speech, wasted sternocleidomastoids, marked weakness of neck muscles suggested the diagnosis.[8] But marked wasting of distal limbs muscles, typical hatchet face, absence of scapular winging, frontal baldness excluded the diagnosis. As there was a history of consanguineous marriage between his parents, limb girdles muscular dystrophy was alsó considered. But limb girdle muscular dystrophy affects primarily the pelvic and shoulder girdle muscles sparing the distal limb muscles.[9]

Finally, the case was considered as Myotonic dystrophy. Typical "hatchet face", frontal baldness, marked wasting of distal limb muscles, presence of grip myotonia as well as percussion myotonia, raised serum CPK, aldolase level, and suggestive histopathological report supported the diagnosis.[10] Due to some overlap feature of muscle wasting, sometimes other variety of muscular dystrophy can be confused with his myotonic variety. Proper history, careful examination for site of involved muscles can help us for clinical diagnosis of myotonic dystrophy.[11]

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Safe Motherhood-Bangladesh Perspective

*SHAMSUN NAHAR BEGUM HENA[1]

The 28th May was observed as safe motherhood day all over the world.

Idea of safe motherhood is creating environment within which a women is enabled to choose whether she will become pregnant, and if she does, ensure-

- Proper and effective care for prevention and treatment of pregnancy complications,
- Access to trained birth assistance,
- Access to emergency obstetric care when needed,
- Receives care after birth to avoid death or disability from complications of pregnancy and childbirth.

Pregnancy is a period of potential risk for serious obstetrical complications. Any pregnant woman can develop complications at any time during pregnancy, labour and in puerperium. About 585,000 maternal deaths occur annually all over the world (1990) and 99% of these deaths occur in developing country representing the widest disparity in world health between rich & poor (WHO 1985). In 1987 international "safe motherhood conference" at Nairobi, Kenya was launched with an aim to draw attention to the appallingly high maternal death in developing country and to mobilize immediate measure at national and international level to prevent the neglected tragedy. The main target was to reduce 50% of the maternal mortality rate (MMR) by the year 2000. This SMI (Safe motherhood initiative) gained additional strength at 1990 Children Summit, where reduction of mortality was listed as a major goal in the plan of action and was endorsed by 127 countries.

The estimated population of Bangladesh is 120.8 million. Of this 48.6% are female, 18% (23.6 million) of females are of reproductive age group (15-45yrs). Maternal mortality is a serious public health concern in Bangladesh. There are 18000 maternal deaths every year in Bangladesh (WHO/UNICEF- 2001).

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For each maternal death at least fifteen women experience a variety of morbidities like VVF, RVF, Vaginal stenosis, PID, genital prolapse, infertility leading to physical, marital and social problems. Vast majority of this death could be avoided if functional system were in place.

Bangladesh has a good comprehensive health care infrastructure for the delivery of maternal and related services. With substantial awareness of this problem, the government of Bangladesh along with the international agencies initiate activities with a target to reduce maternal mortality rate from5.5 to 2.5/1000 live birth by the year 2000.But with all efforts there is no substantial reduction of maternal mortality. And now the targeted goal suggested by the National Integrated Population and health program (NIPHP) is to reduce the maternal mortality rate from 4.5 to 3.0 per 1000 live births by the year 2005.

The factors making motherhood unsafe are-

A. **Direct/obstetrical causes: (75%)**

Resulting from complications of pregnancy, labour and puerperium. Hemorrhage (70%), sepsis, unsafe abortion, eclampsia and obstructed labour are the most important causes of maternal death.

B. **Indirect causes: (25%)**

Associated medical disorder complicating pregnancy like anaemia, heart disease, hepatic failure.

C. **Contributory causes:** (Social, environmental and organizational)

- Poverty, unemployment and female illiteracy.
- Gender discrimination, low valued placed on woman.
- Poor communication and transport facility.
- Early marriage.
- Too early, too many; too close and too late pregnancy.
- Poor contraceptive acceptance rate.
- Unsafe abortion practice.
- Poor perinatal (ante, intra and postnatal) care.
- Uneven distribution of health manpower- more physicians than midwives, most of them are urban based.
- Poor utilization of healthcare facilities due to lack of community and family awareness (ignorance, illiteracy and social prejudices).

- Constraints in health facility- shortage of trained personnel, inadequate logistics supply, defective obstetric care, inadequate transfusion facility.
- Lack of effective referral system between lower level health centers with the centers having higher facilities for emergency obstetric care.

The factors that delay the utilization of services at facility are depicted in the following (figure 1).

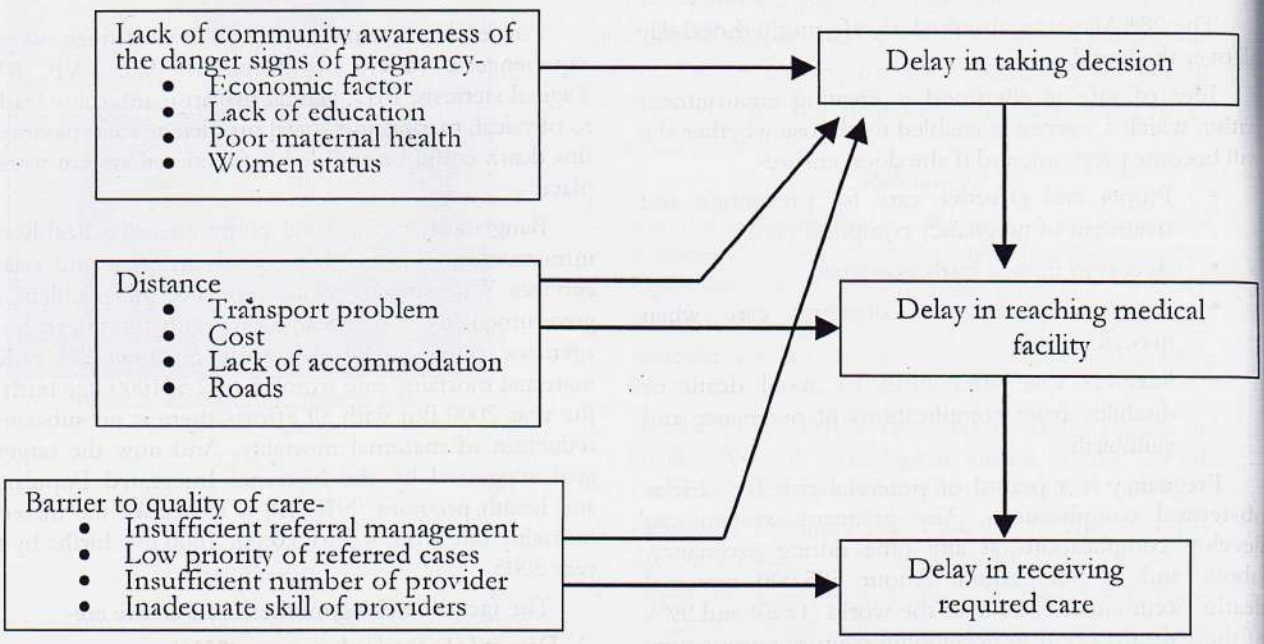

Figure 1: Three delays of utilization of services

Measures to be taken for safe motherhood-

As most of the factor responsible for maternal mortality and morbidity are known so a multipronged attack is needed to make motherhood safe.

The following pyramid represents three level at which interventions can work to bring about improvements in maternal health.

The intervention pyramid—

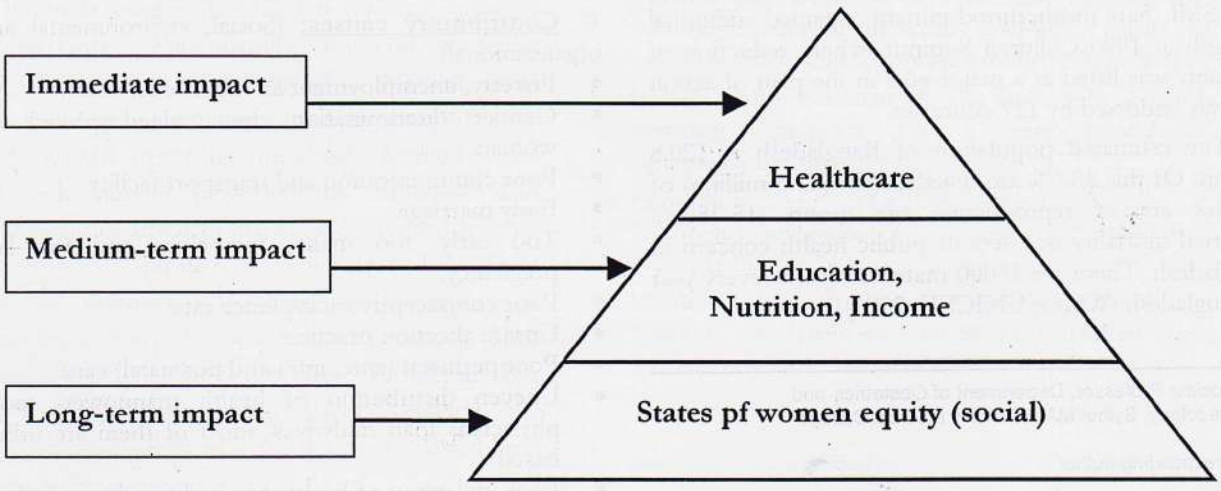

Figure 2: The intervention pyramid

There are four pillars of safe motherhood.

1. Family planning
2. Antenatal care
3. Clean/safe delivery
4. Essential Obstetric care

These four strategic interventions must be delivered through primary health care and rest on a foundation of greater equity for women.

The "four pillars" of safe motherhood:

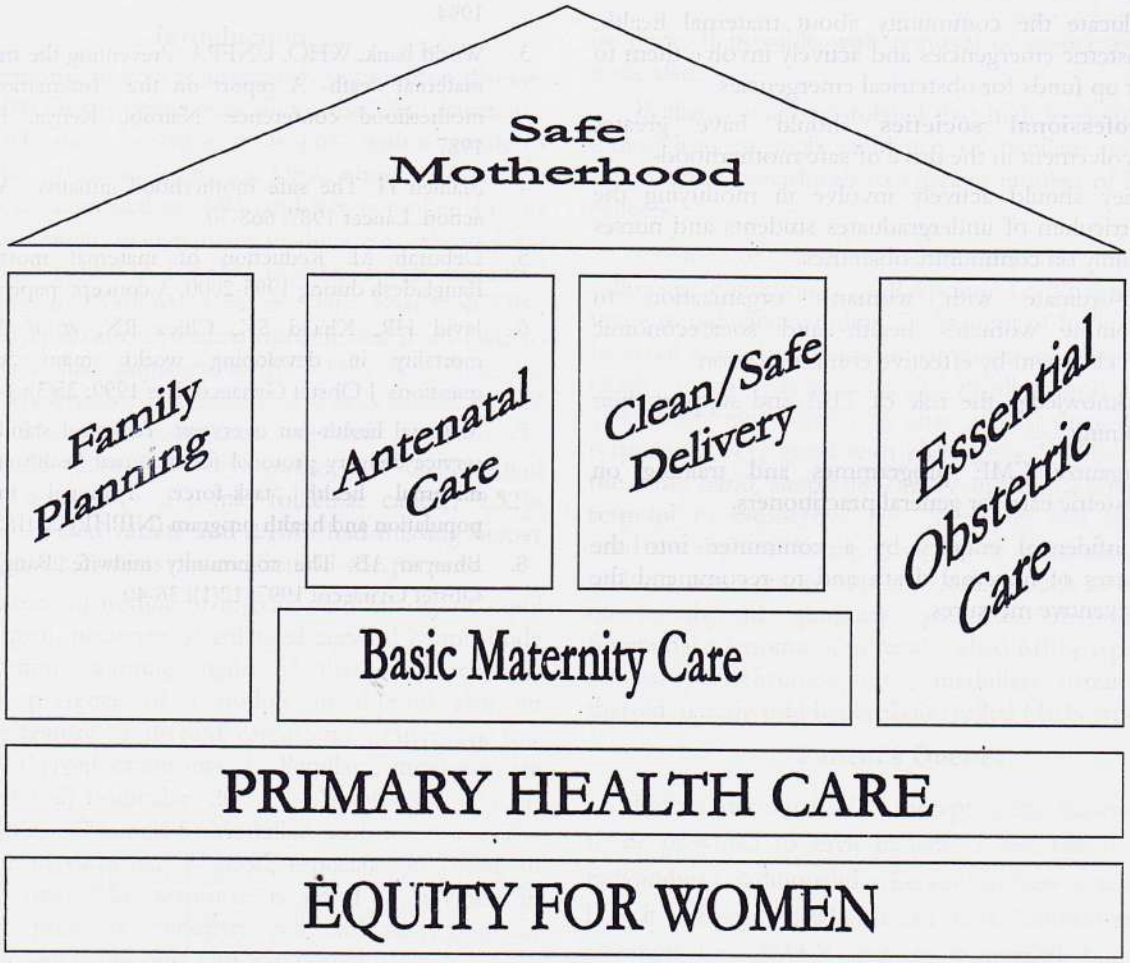

Figure 3: Four Pillars of Safe motherhood

- **Improved and affordable health care services**- all women should get adequate and effective antenatal care, tetanus immunization, iron & folic acid supplementation, screening of high risk pregnancy with appropriate intervention and referral, access to safe delivery practice, EOC services and post partum care.
- Developing **community based maternity care** in rural settings (as 90% deliveries occur at rural setting and 80% of the maternal death comes from unpredictable emergency situation).
- Development of **community midwife** with institutional training to serve the community they belong.
- **Wider availability of trained personnel** (midwife, trained birth attendant) -Training of health worker on safe delivery practices and in life saving skills for obstetrical emergency (primary PPH, prolong/obstructed labour, eclampsia).
- Health worker should have **backup support** at first referral level.

- Establishment of first referral center for **comprehensive obstetric care** at district or papilla level for obstetrical emergencies.
- **Community and family awareness**, awareness of pregnant women regarding optimal utilization of health facility, danger of unsafe delivery, **danger sign of pregnancy and birth planning.**
- Educate the community about maternal health, obstetric emergencies and actively involve them to set up funds for obstetrical emergencies.
- **Professional societies** should have greater involvement in the drive of safe motherhood-

  They should actively involve in modifying the curriculum of undergraduates students and nurses mainly on community obstetrics.

  Co-ordinate with woman's organization to promote woman's health and socioeconomic development by effective communication.

  Acknowledge the role of TBA and support their training.

  Organize CME programmes and training on obstetric care for general practitioners.
- Confidential enquiry by a committee into the causes of maternal death and to recommend the preventive measures.

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Treatment of Thyroid Cancer, Present Concept

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Introduction

Carcinoma thyroid is a relatively uncommon disease. About 400 deaths occur annually in the UK.[1] Incidence of thyroid cancer in Singapore is 4.6% with a female to male ratio of 3.6 to 1.[2] In the USA, about 17,000 new cases were diagnosed in 1998, and it was ranked 14th of all internal organ malignancies in the USA. About 1200 deaths occur among 135,000 thyroid cancer patients.[3] According to National Cancer Data Base (NCDB), 31,531 thyroid cancer patients were treated from 1985 to 1995, 85% had papillary cancer, 11% had follicular cancer, 3% Hurthle cell cancer, 4% had medullary cell cancer, 2% had anaplastic thyroid cancer.[4] In Bangladesh, Kabir F *et al* [5] found that 66.38% had papillary cancer, 19.3% had follicular cancer, 9.32% were with mixed variety and 2.16% had missing biopsy report in their 20 years observation on 461 patients.

Presence of nodule in thyroid with history of rapid growth, pain, presence of enlarged cervical lymph node are common warning signs of thyroid cancer. In children, presence of a nodule in thyroid also an alarming feature of thyroid carcinoma.[6] There are five types of thyroid carcinoma: 1) Papillary, incidence are about 70%, 2) Follicular- 20%, 3) Anaplastic- <5%, 4) Lymphoma- <2%, and 5) Medullary cell carcinoma-5%. Response to treatment is good, especially in young in papillary type. The response is good if resected in follicular, poor in medullary cell and very poor in anaplastic and lymphoma type carcinoma.[1]

Discussion

Treatment of thyroid carcinoma by radioactive iodine^{131} began about 40 years ago. Using beta energy of RAI^{131}, thyroid cancer cells are destroyed and recurrences are prevented.

After confirmed diagnosis of thyroid carcinoma by histopathological examination, total thyroidectomy is to be done, if possible with removal of associated lymph node also.

Williams *et al* [7] postulated that high levels of dietary iodine lead to high incidence of papillary cancer of thyroid, and lower levels to a greater number of follicular cancers.

Choice of dose schedule depends upon the following conditions. 1) Response of thyroid cancer tissue to radioactive iodine, 2) Amount of Iodine dose to be given considering amount of radiation absorbed dose (Rads) to the patient's 'body. Papillary and follicular carcinoma trap iodine and after a total thyroidectomy, response is very good with radioactive I^{131} therapy. On the other hand, anaplastic cancer and lymphoma do not respond to radioactive I^{131}. Medullary cell carcinoma needs total thyroidectomy; usually it is associated with multiple endocrine neoplasms (MEN), such as adenoma of parathyroid, pituitary, pancreas, thyroid, non-functional adenoma of adrenal (called MEN type 1) and adrenal pheochromocytoma, medullary carcinoma of thyroid, parathyroid hyperplasia (called MEN type 2).

Patient's Queries

Before starting RAI^{131} therapy some information is to be provided to each patient. These are: i) what is radioiodine/ radionuclide therapy. ii) how it works? iii) how it is given? iv) what it can treat? Patients are to be assumed that RAI^{131} acts as a normal biochemical substances and uses the normal human body biological processes. In fact, radionuclide therapy is used in a number of benign or malignant conditions: (a) Benign: Failure of medically treated rheumatoid arthritic joint pain, Benign enlargement of thyroid, Polycythaemia rubra vera, (b) Malignant: Thyroid cancer, Pheochromocytoma, Neuroblastoma, Carcinoid tumour, Paraganglioma. Radionuclide therapy is also used in patients with metastatic bone pain.

Logistic support

It is also important that the facility that will treat thyroid cancer patients should have some logistic support, like license to use radionuclides, qualified physicists and doctors and isolation room.

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Therapy protocol

1. Ablation therapy: There are three approaches to I^{131} therapy: a) Fixed dose method, b) Quantitative dosimetry, c) Blood dosimetry. Patients with uptake in tumour are routinely treated with fixed dose method.

a) Patients with lymph node metastasis but not large enough, treated with 100 to 175 mCi (3700-6475 Mbq). Cancer with distant metastasis is treated with 150-200 mCi (5550-7400 MBq). Cancer with distant metastasis is treated using 200 mCi (7400 MBq) or more. In Lung mets 175-200 mCi, in skeletal mets 200 mCi is usually given. Treatment can be started with low activity dose: 30 mCi, if thyroid cancer is diagnosed very early and no metastasis is found. This amount can be given repetitively as necessary.

   Thyroid replacement therapy should be stopped for 4 weeks. A tracer study should be performed prior to the therapy dose to ensure that RAI^{131} reaches the remnant thyroid and to measure the uptake (>50% at 24 hours).

   Up to 30 mCi (>1 GBq) dose can be given as outpatient and for more than that patients need to be admitted in a hospital with definite isolation. No visiting is to be allowed at least for 3 days.

b) **Quantitative method:** Dose calculated on the basis of tumour uptake. Maxon *et al* [8] and Brierley *et al* [9] advocate this method: a dose that will deliver 50,000-60,000 cGy to the residual normal tissue and 4000-5000 cGy to metastatic foci are likely to be effective. But in doing so it is important to consider the weight of the remnant thyroid tissue, 24 hours uptake, and effective half-life of RAI^{131}.

c) **Blood dosimetry:** Benua RS *et al* [10] advocate a dose delivery of 200 cGy to the blood as the maximum dose, with whole body retention less than 120 mCi (440 MBq) at 48 hours.

2. Recurrence Therapy: All replacement therapy should be stopped for 4 weeks. A whole body scan is to be performed and 5-7 GBq (135 to 190 mCi) of RAI^{131} should be used. The interval period between 1st dose and recurrent dose should not be less than 6 months for up to 5-10 treatments.

In case of lung metastasis, it is to be very cautious to avoid pulmonary fibrosis and to avoid bone marrow suppression in bone metastasis.

Follow up

Following therapy, patients must be hospitalized until the retained radioactivity is less than 39 mCi or the metered exposure rate from the patient is less than 5 mR/hr at one meter.

All patients are to be followed up for life, at 3 to 6 months interval. Serum free T_4, TSH and Tg levels are to be measured at every follow up. Whole body scans may be carried out if indicated. Replacement therapy with thyroxin is to be continued for life with doses depending on serum free T_4 and TSH level.

Contraindication of therapy

Absolutely in pregnancy and when breast feeding.

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Recent trends in the management of haemorrhoidal diseases

Haemorrhoidal disease is a very common medical problem. Since it is estimated that about 50% of population over 50 years of age has or has had haemorrhoidal symptoms.[1] Haemorrhoid and haemorrhoidal disease are not the same. Haemorrhoid is normal tissue consisting of vascular plexus form the anal cushion. The function of anal cushion is to protect the anal canal from injury by hard stool. When haemorrhoid produces symptoms then - it is called haemorrhoidal disease. Haemorrhoid divided into external and internal plexus. External plexus located under the skin of the margin of anus. They are primarily giving rise to painful thrombosis. The clots of the thrombus usually resolve within less than 2 weeks producing anal tag causing itching and soiling. Some times external haemorrhoid needs immediate evacuation of clot.

The internal haemorrhoidal plexus situated in the submucosal space above the dentate line, present mainly with bleeding and prolapses. But other less specific symptoms like pain, discomfort, soiling, itching and discharge associated with internal haemorrhoid.

The treatment of internal haemorrhoid based on several principles:[2] (i) The internal haemorrhoid are normal structure and treated only when produce symptoms. (ii) Treatment must leave anatomical structure as intact as possible. (iii) Haemorrhoidal disease is a benign condition and any treatment procedure should not worsen the prognosis.

Many factors still unknown in the pathophysiology of haemorrhoidal disease. As a result there are numerous theories and opinion regarding therapeutic option of haemorrhoidal disease. One of the pathogenesis process implicated in the acute internal haemorrhoid is rupture of anchorage of anal cushion leading to downward displacement of anal cushion causing stagnation and stasis of blood in the anal cushion.[3] It is demonstrated that stasis activates white cell to release inflammatory mediators resulting increased capillary permeability, fragility and necrosis of vessel wall of anal cushion. The anal cushions are therefore easily injured by passage of stool and bleeds.[3]

Current modalities of treatment of internal haemorrhoids are (i) Conservative treatment by regularization of intestinal function by use of stool softener, dietary advice (copious fluid and increased fiber in diet) and use of local topical agents. The topical agents act by lubricating the anal canal. But no study exists concerning their efficacy. (ii) Use of oral pharmacological drugs particularly phlebotonic drugs e.g., micronized purified flavonoids fractions (MPFF) which increases the venous return, increase venous tone, increases lymphatic drainage and prevent microcirculatory damage from inflammatory mediators like cytokines, leukotriens, oxygen free radical, platelets aggravating factors which is responsible for microcirculatory damage of anal cushion.[4] Recent study shows that MPFF has been shown to reduce the severity bleeding and prevent relapse in comparison to placebo. [5] This drugs are frequently used in several Asian and European countries with considerable efficacy.[6] (iii) Conservative instrumental out door treatment modalities are Injection Sclerotherapy (IS), Infrared Coagulation (IC) and Rubber Band Ligation (RBL). The aim of conservative instrumental technique is to produce sclerosis at the apex of internal haemorrhoid resulting adhesion of mucosa and submucosa to underlying muscle coat. The aim is not to destroy the haemorrhoidal tissue but rather to reposition them within the anal canal. Efficacy of instrumental treatment depends upon grade of internal haemorrhoid. Injection sclerotherapy (IS) and infrared coagulation (IC) give similar results.[7] RBL shows better result than IS and IC if the case is carefully selected. One of the important complications of RBL is pain persisting up to 72 hours after banding and patient cannot resume duty on the same day of treatment [6] (iii) Surgical treatments- The aim of surgical treatment is to remove the haemorrhoidal tissue. Surgery removes both external and internal haemorrhoid. The most widely used technique in surgery is open haemorrhoidectomy (Milligan and Morgan technique. Other method is closed haemorrhoidectomy and Cryohaemorrhoidectomy. Controlled study shows that there is no advantage was found in performing closed haemorrhoidectomy.[8] Cryohaemorrhoidectomy is not superior to surgical haemorrhoidectomy because it does not remove haemorrhoidal tissue but destroy it. Besides, cryohaemorrhoidectomy is painful and there is difficult to control the depth of tissue during cryo surgery.

It is important to note that about 90% to 95% of haemorrhoidal disease can be managed by non surgical approach particularly Injection Sclerotherapy (IS), Infrared Coagulation (IC) and Rubber Band Ligation (RBL).[9] Rubber Band Ligation (RBL) in grade I, II and early grade III is very useful out door procedure. Use of Micronized Purified Flavonoid Fraction (MPFF) is effective in stopping acute bleeding within 72 hours and reduces the risk of prolapses.[5,6] There is no trial on MPFF is carried out in Bangladesh until now. These needs further clinical trial in context of Bangladesh

before random use of MPFF. Surgery should reserve for late grade III and grade IV internal haemorrhoid.

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How to Write Original Article

*MEER MAHBUBUL ALAM[1]

Writing a bit different from delivering a lecture or give a statement because one can deny or contradict the statement, which is happening in our political speech. But by writing one is exposed to all the criticism for comments. That is why not many develop habit of writing easily. Writing needs extensive reading, thinking and taking opinion of others. The only two things one can write without any serious thinking- one is love letter and other is political speech. When we talk about medical writing, it is a bit serious than any other writing. That is one of the reasons why many among the well-known physicians and surgeons, including medical teachers, have no much-published work in their credit.

Like all other things, medical writing too has certain rules and one must follow them carefully. There are different types of medical articles, which will be discussed, in different issues systemically in this journal. This includes original articles, case reports, review articles, letters to editor, special communication, leading articles, dissertation, thesis, book review etc; each of the articles written with specific scientific purpose. Original article is the most common of all medical write-ups. It is written by conducting an original study or collecting data retrospectively. It may be inform of drug-trial or trial of an operation or evaluating of investigation or any aspect of problem. An original article needs extensive preparation, worthwhile material to report and knowledge about medical writing. It should have good style and should conform to a basic structure or format. A conventional format is title, abstract or summary, introduction, materials and method, results, discussion, acknowledgement and reference.

Title

Title should catch the eye and be accurate in telling the aspect of problem studied. It should be short, clear, descriptive and specific.

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* Corresponding Author

Abstract/Summary

This is usually written after rest of the paper although most journals begin the article with abstract or summary. Abstract and summary are more or less synonymous except some differences.

Abstract is the precise of the article. It should contain essence of introduction, the purpose of study, the materials and methods, statistical significance and main conclusion. It should be not more than 250 words.

Summary is the clear and precise statement of objective, main results and conclusion.

Introduction

Introduction should be brief and clear. State the problem that you likely to solve and give the reason why did you study the problem. It should not contain historical review of the subject or aspect of subject, which should not covered in the work that is being reported.

Materials and methods

Materials means-who is the subject of the study. Is it experimental model or patient? It should have criteria of selection and rejection.

Method means how you do the study. It should include experimental technique, study design, methods of assessment (e.g., statistical method) etc should be clearly mentioned.

Results

Here author should describe what did he find? Result communicates the observation of the study. Start with one easier to interpretative (age, sex) and give relevant data in the table, figure or as text.

Table: Tables are ideal for detailing numerical data on various group of patient. They should have proper legends and footnotes. Number of table should less and their place should be clearly marked in the text (3-5 tables are enough).

Figure: Figure should be suitable when data is not expressed any way. Figures are graph, pie chart, scatter diagram, line chart, etc.

Photograph: Clinical photograph should give with appropriate details.

Discussion

Here the author should describe what does the result mean and why? Main result should be summarized at the beginning. Only refer to the work of previous authors, which illuminates or illuminated by the present result. Final paragraph should firmly state the massage of the article and points out where gaps of knowledge could be filled.

Discussion should be end either with the intention of author to explore the gap further or giving the guideline for further research to other workers.

Acknowledgement

Acknowledge all those members who made the study possible including physician who referred the case, technicians who did the laboratory test, sponsoring organization who provide finance, statistician who help for statistical methods and those who provide secretarial help.

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References should be cited carefully and systemically as appeared in the text. Quote only those references, which the author has read and are retrievable. They should include the name of author(s) initials, title of the paper, name of the journal/book, year of publication, place of publication, name of publishers and first and last page of article.

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