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Original Article

Use of Internet among Rural Teenagers: Pattern of Usages, Awareness and Associated Health Hazards

*Jobayer M¹, Sultana R², Anwar SS³, Afroz Z⁴, Akter N⁵, Chowdhury M⁶, Rashed A⁷, Farhana N⁸, Islam LMT⁹, Hussain DA¹⁰

Abstract

Internet has become an essential tool for communication, information, and entertainment and use of internet is increasing among the teenage group. This cross sectional study was conducted among 385 teenagers from rural area of Narayanganj district in Bangladesh to evaluate the practice of internet use, their awareness about the beneficial and harmful effects and associated physical and psychological health hazards. The study reached its various findings from the view point of rural area of our country. The study population was chosen purposively; data were collected using a semi structured questionnaire regarding their knowledge and various practice of internet use relating to the objectives. Most of the respondents were students and came from lower middle class family. Majority of the study people recently started to use

internet and they were mostly light or moderate user in terms of time spending. Mobile phone was used as device by 84.7% people for their internet browsing. Teenagers used internet mainly for entertainment (42.1%) and social networking (35.6%) purpose and not much for learning or education. They were aware about the beneficial and the harmful effects of internet use especially abuses. Commonly experienced physical health hazards by them were headache (11.7%), straining of eyes or blurring of vision (10.7%) and skipping meals or change in eating behavior (9.1%), and among the psychological hazards, restlessness or agitation (8%) and depression (5.2%) were stated. It may be concluded that use of internet among teenagers of rural area of our country is at modest level associated with a few health hazards but the merits of internet based networking is yet to be revealed by them.

Key words: Use of internet, rural teenagers, health hazards.

INTRODUCTION

Internet has become an important tool for social interaction, sharing information and entertainment.¹ As internet has moved into homes, schools and businesses, there has been a rapidly growing public awareness of potential adverse effects arising from excessive, maladaptive or addictive internet usage.^{2,3} As an important period between childhood and adulthood, teenage is encompassed by alterations in physical, psychological and social development.⁴ Presence of relatively immature cognitive control makes this period a time of vulnerability and adjustment^{5,6} and may lead to a higher incidence of affective disorders and addiction among teenagers.^{7,8}

Total internet user in Bangladesh in 2016 is 21,439,070 which are 13.2% of population and 0.6% share of world internet users.⁹ Importance of internet is increasing here for communicative purposes among adults, but teenagers are also very frequent users. Teenagers are spending long hours on social networking sites and this has resulted in a significant drop in regular healthy social interaction and affecting school and home life. Scope of play out of doors is virtually non-existent for children and most of them spend time

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indoors and much of it is lonely time watching television or using internet. Passing time is a popular activity on Facebook as one can play games, chat, and network. Increased internet use results in less physical, psychological, and emotional outlets for stimulation necessary for development.

Misuse of Facebook by teenagers has become one of the most insidious menaces of the society. When a teenager spends too much time on the internet, their behavior patterns can be adversely altered as they spend less time on studies and household responsibilities. High-risk internet users have inappropriate dietary behavior which could result in stunted growth and development.¹⁰ Those who regard themselves as dependent on internet often report high levels of depressive symptoms.¹¹

Educating teenagers and parents is an important step for effective and safe use of internet. In recent studies, many reported that role of internet in completion of school projects is vital, and online resources have replaced need for library visits.^{12,13} Internet also plays an important role in how adolescents educate themselves about specific health matters.¹⁴ They regularly use internet to gather information on news, sports and other areas of personal interest. There is a tremendous range of information available online with respect to sexuality that ranges from information on sexual health resources to online pornography and some others misuses.¹⁵

Internet users in Bangladesh have increased much these days and the importance of internet is enormous especially to teenagers. They are spending long hours on internet. For these reasons, this study was carried out to reveal the real scenario of knowledge and practice of internet use among teenage people in rural area Bangladesh.

MATERIALS AND METHODS

This cross sectional study was conducted during November, 2016. It was carried out in rural area of Rupganj upazila under Narayanganj district. Six bangla medium schools with co-education (both male and female students) and two other common gathering places of the rural area of Rupganj were purposively selected for this study. Through non-probability sampling 385 respondents were selected. From each school 50 students of 13 to 18 years of age from different classes (class VII-X) who used internet by any device were included.

A self-administered questionnaire containing information on socio-demographic indicators, quality of life, internet usage and level of knowledge and awareness about internet were filled in. The following socio-demographic characteristics were obtained: age, gender, educational level

and self-reported family economy. Internet usage pattern was assessed by examining the frequency of internet use per day and week and the purpose of internet use. Collected data were classified according to characteristics and various statistical methods and 'Microsoft Excel' software were used for analysis.

RESULTS

Age of the respondents was between 13 to 18 years. Among them 211 (54.81%) were male and male to female ratio was 1:0.82. (Table-I)

Table-I: Age and sex distribution of the study population (n=385).

| Age (year) | Male | Female | Number n(%) |
|------------|------|--------|-------------|
| 13 | 14 | 15 | 29 (7.53) |
| 14 | 45 | 43 | 88 (22.84) |
| 15 | 53 | 45 | 98 (25.85) |
| 16 | 56 | 46 | 102 (26.49) |
| 17 | 21 | 19 | 40 (10.39) |
| 18 | 22 | 6 | 28 (7.27) |
| Total | 211 | 174 | 385 (100) |

About 85% of the teenagers used mobile phone as their device for the internet use and rest of them used laptop or desktop in home and cyber café. (Figure-1)

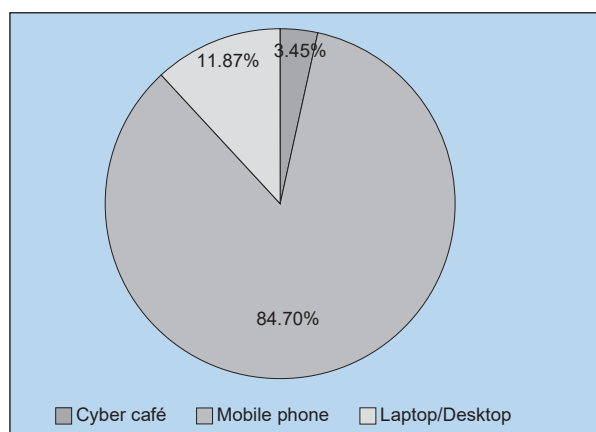


Figure-1: Pattern of devices for internet use (n=385).

Among the respondents 55.06% were 'moderate' users of internet in terms of time they spent daily or weekly. Overall 14% were 'heavy users' who spent more than four hours per day; among them 20.4% were male and 6.3% female and the difference between male and female heavy internet user was statistically significant. (Table-II)

Table-II: Distribution of study population according to frequency of internet use. (n=385)

| Category of user | Male n(%) | Female n(%) | Total n(%) | P value |
|--------------------------|-------------|-------------|-------------|------------------|
| Light (few times/week) | 51 (24.17) | 68 (39.08) | 119 (30.91) | P value- 0.00162 |
| Moderate (few times/day) | 117 (55.45) | 95 (54.60) | 212 (55.06) | P value- 0.8670 |
| Heavy (>4 hours/day) | 43 (20.38) | 11 (6.32) | 54 (14.03) | P value- 0.00007 |

Chi-square test was done to measure the level of significance.

Common purposes of internet use were entertainment (42.1%) and social networking (35.6%) whereas 9.9% teenagers used internet for educational purpose. (Table-III)

Table-III: Purpose of internet use (according to type of sites visited).

| Purpose | Male n(%) | Female n(%) | Total n(%) |
|-------------------|-----------|-------------|------------|
| Entertainment | 97 (45.9) | 65 (37.4) | 162 (42.1) |
| Social networking | 51 (24.2) | 86 (49.4) | 137 (35.6) |
| Gaming | 39 (18.5) | 09 (5.2) | 48 (12.5) |
| Education | 24 (11.4) | 14 (8.0) | 38 (9.9) |

According to the respondents, keeping communication (49.10%) and making new relationship (27.01%) were the best positive side of internet use and addiction to internet (52.99%) and waste of time (25.19%) were the common demerits for them. (Table-IV)

Table-IV: Perception of merits and demerits of internet among study population.

| Merits of internet use | Number | Percentage |
|---------------------------------|--------|------------|
| Communication | 189 | 49.10 |
| Making new relationship | 104 | 27.01 |
| Entertainment | 51 | 13.25 |
| Education | 28 | 7.27 |
| Others | 13 | 3.37 |
| Demerits of internet use | | |
| Addiction to internet | 204 | 52.99 |
| Waste of time | 97 | 25.19 |
| Less concentration in study | 62 | 16.11 |
| Others | 22 | 5.71 |

Headache was the most commonly (11.69%) experienced physical health hazard during internet use, followed by straining of eyes or blurring of vision (10.65%) and among psychological hazards restlessness or agitation (8.05%) and depression (5.19%) were most common. (Table-V)

Table-V: Different types of health hazard experienced by teenagers during internet uses.

| Health hazards | Male | Female | Total n(%) |
|---|------|--------|------------|
| Physical hazards | | | |
| Headache | 36 | 09 | 45 (11.69) |
| Straining of eyes or blurring of vision | 29 | 12 | 41 (10.65) |
| Skipping meal or change in eating pattern | 26 | 09 | 35 (9.09) |
| Less sleep or insomnia | 21 | 12 | 33 (8.57) |
| Less physical activity | 18 | 11 | 29 (7.53) |
| Psychological hazards | | | |
| Restlessness/ agitation | 19 | 12 | 31 (8.05) |
| Depression | 11 | 09 | 20 (5.19) |
| Loneliness | 07 | 11 | 18 (4.68) |
| Damaging relationship | 04 | 08 | 12 (3.12) |

DISCUSSION

While there are certainly concerns about the growth of technology and internet use among teenagers in Bangladesh and around the world, technology should not be necessarily negative. The fact is that technology has become an integral part of the life of the modern teenager.

Among the 385 rural teenage respondents most were students of different classes of secondary school level and maximum came from lower middle income group. They

started using internet in recent time and about 85% of them used mobile phone as their device for internet use and in majority cases (91%) their parents were the providers of the expenditure. The availability of mobile phone to the teenagers may have facilitated the use of internet in rural setting of our country as most of the rural area is not under cover of broad band networking of internet.

The teenagers were categorized as 'Heavy, Moderate and Light' user according to the time they spent daily or weekly on internet.¹⁰ Heavy users who spent more than four hours per day on internet comprised about 14% of the respondents; among them male were predominant and more than half of the respondents were moderate users of internet spending less than 4 hours but visited internet few times per day. Excessive internet use or uncontrollable use of internet is regarded as internet addiction or pathological internet use.^{16,17}

Main purpose of use of internet among teenagers in this study was entertainment (42.1%) and social networking; on the other hand less than one in ten teenagers used internet for their learning or educational purpose. Remarkably about half of the female respondents used internet for only social networking that means Facebook; whereas among male it was 24%. In contrast, a study in Canada showed that many of the young users searched internet for educational purpose like solving homework, completing assignments and answering questions or sometimes topics for health education.¹⁴ This inconsistency between the purpose of internet use probably is due to the differences between socio-economical statuses of two study groups.

The teenagers were aware about the beneficial and harmful affects of internet use especially internet abuses. They mentioned that keeping communication with friends and making new relationship were the most beneficial sides of internet for them whereas addiction to internet and wastage of time were the most damaging ones. It is not always a waste of time to hang out online and sometimes it is found that spending time online is good for young people to pick up social and technical skills they need to be competent citizens in the digital age.¹⁸

Time spent online affects psychological well-being and increment in this time decreases social integration which negatively affects psychological well-being.^{19,20} Teenage respondents were asked to mention any physical or psychological health hazards they had experienced during

time being online. The most commonly experienced physical health hazards they reported were headache, straining or blurring of eyes during internet use and occasional skipping of meals or change in eating pattern and behavior; among the psychological hazards restlessness or agitation and depression were most common. These symptoms were in accordance with findings of several studies who mentioned that heavy internet use is associated with mood disorders, such as depression and various adverse health outcomes like poor sleeping quality and lower level of physical activity.^{10,21} There is relationship between obesity and screen time, which is related to sedentary lifestyle and also snacking and decreased sleep time.²²

Technology is not a good or a bad thing. With proper usage and supervision from adults, teenagers can take advantage of many great learning opportunities on internet. If parents leave children unsupervised, it can result in internet addiction, depression, cyber-bullying, and unwanted predators taking advantage of unsuspecting teenagers. Parents should become more conscious and aware as well as responsible for monitoring online usage of their children. They should recognize that technology is now a normal part of many teenagers' lives.

Among some limitations of the study; data were collected from a small sample and as the study area was limited to seven villages, the result may not represent the whole rural teenage population of the country. The overall knowledge awareness and practice of internet among teenagers could not be addressed completely. Few persons of less or no schooling were involved so that the information did not reflect the real picture of rural average teenagers.

CONCLUSIONS

In the information age, internet use is becoming increasingly significant in acquisition of information and knowledge. Internet use is now considered to form a part of the culture of teenagers, and hence studying internet use and its merits as well as demerits is important to create a sound teenage culture. Teenagers of Bangladesh should be taken under cover of various awareness programmes for better understanding of the positive and negative aspects of internet use and abuses.

REFERENCES

1. Tsitsika A, Critselis E, Kormas G, Filippopoulou A, Tounissidou D, Freskou A, et al. Internet use and misuse: a multivariate regression analysis of the predictive factors of internet use among Greek adolescents. *Eur J Pediatr.* 2009; 168: 655-65.

2. Block JJ. Issues for DSM-V: internet addiction. *Am J Psychiatry*. 2008; 165: 306-7.
3. Cooney GM and Morris J. Time to start taking an internet history? *Br J Psychiatry*. 2009; 194: 185-7.
4. Ernst M, Pine DS, Hardin M. Triadic model of the neurobiology of motivated behavior in adolescence. *Psychol Med*. 2006; 36: 299-312.
5. Casey BJ, Tottenham N, Liston C, Durston S. Imaging the developing brain: what have we learned about cognitive development? *Trends Cogn Sci*. 2005; 9: 104-10.
6. Galvan A, Hare TA, Parra CE, Penn J, Voss H, Glover G, et al. Earlier development of the acumens relative to orbitofrontal cortex might underlie risk-taking behavior in adolescents. *J Neurosci*. 2006; 26: 685-92.
7. Steinberg L. Cognitive and affective development in adolescence. *Trends Cogn Sci*. 2005; 9: 69-74.
8. Grant JE, Potenza MN, Weinstein A, Gorelick DA. Introduction to behavioral addictions. *Am J Drug Alcohol Abuse*. 2010; 36: 233-41.
9. Internet Live Stats. Elaboration of data by International Telecommunication Union (ITU), World Bank, and United Nations Population Division. Available from: www.InternetLiveStats.com.
10. Kim JH, Lau CH, Cheuk KK, Hui HLC, Kan P, Griffith SM. Brief report: Predictors of heavy internet use and associations with health promoting and health risk behaviors among Hong Kong university students. *J Adolesc*. 2010; 33: 215-20.
11. Morrison CM and Gore H. The relationship between excessive internet use and depression: a questionnaire based study of 1319 young people and adults. *Psychopathol*. 2010; 43: 121-6.
12. Stahl C and Fritz N. Internet safety: Adolescents' self-report. *J Adolesc Health*. 2002; 31: 7-10.
13. Lenhart A, Simon M, Graziano M. The internet and education: Findings of the Pew Internet & American Life Project. [cited 2016 Dec 19]. Available from: http://www.pewinternet.org/pdfs/PIP_Schools_Report.pdf.
14. Media Awareness Network. Young Canadians in a Wired World-Phase II. [cited 2016 Jan 21]. Available from: http://www.mediaawareness.ca/english/research/YCWW/phaseII/upload/YCWWII_Student_Survey.pdf.
15. Kaiser Family Foundation Survey. Generation Rx.com: How young people use the internet for health information. [cited 2016 Dec 12]. Available from: <http://www.kff.org/entmedia/upload/Toplines.pdf>.
16. Widyanto L and Griffiths M. Internet addiction: a critical review. *Int J Mental Health Addict*. 2006; 4: 31-51.
17. Beard KW. Internet addiction: a review of current assessment techniques and potential assessment questions. *Cyberpsychol Behavior*. 2005; 8: 7-14.
18. Ito M. Hanging out, messing around and seeking out: kids living and learning with new media. The MacArthur Foundation. Cambridge, MA: MIT Press. 2008.
19. Van der Aa N, Overbeek G, Engels RCME, Scholte RHJ, Meerkerk GJ, Van den Eijnden RJJM. Daily and compulsive internet use and well-being in adolescence: a diathesis-stress model based on big five personality traits. *J Youth Adolesc*. 2009; 38: 765-76.
20. Weiser EB. The functions of internet use and their social and psychological consequences. *Cyberpsychol Behavior*. 2001; 4: 723-43.
21. An J, Sun Y, Wan Y, Chen J, Wang X, Tao F. Associations between problematic internet use and adolescents' physical and psychological symptoms: possible role of sleep quality. *J Addict Med*. 2014; 8: 282-7.
22. Fu K, Chan WSC, Wong PWC, Yip PSF. Internet addiction: prevalence, discriminant validity and correlates among adolescents in Hong Kong. *Br J Psychiatry*. 2010; 196: 486-92.

Original Article

Multimorbidity among Tuberculosis Cases: Bangladesh Perspective

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Abstract

Globally tuberculosis (TB) has become the leading cause of death from infectious diseases. Tuberculosis is a chronic infection and a person may suffer from tuberculosis and other chronic medical conditions at the same time. Co-occurrence of multiple chronic conditions in the same individual, known as multimorbidity (MM) is increasing worldwide. This cross-sectional study was carried out from January 2017 to December 2017 to reveal the extent of multimorbidity among tuberculosis cases. A TB case with multimorbidity was defined as TB with multimorbidity (TB-MM) subject. By convenient sampling, 227 tuberculosis cases from 8 Directly Observed Treatment, Short Course (DOTS) centers from Dhaka, Mymensingh and Netrokona districts were enrolled in this study. Among 227 tuberculosis cases 29 (12.8%) cases had multimorbidity (TB-MM subjects). Prevalence of multimorbidity was significantly higher in age group ≥ 40 years ($p < 0.001$), male cases ($p = 0.034$) and cases who had family income > 30000 BDT/month ($p = 0.001$), were currently smoker ($p = 0.028$) and whose BCG scars were not seen ($p < 0.001$). This study recommends that each TB case should be investigated for other chronic conditions to reveal the actual national magnitude of multimorbidity.

Key words: Tuberculosis, multimorbidity, co-morbidity.

INTRODUCTION

Tuberculosis (TB) has become the ninth leading cause of death worldwide and it has been the leading cause of death from a single infectious agent for last 5 years, which is now ranking above HIV/AIDS.¹ Despite the availability of affordable, effective treatment; it still affects and kills millions of people each year. In 2016, an estimated 10.4 million people were suffering from tuberculosis globally and 1.7 million died from the disease (including 0.4 million among people with HIV)¹, which represents an intolerable burden of human suffering.¹ Ending TB is not only a public health problem, but also a development challenge and opportunity. The WHO "End TB Strategy", aims to end the global TB epidemic with targets to reduce TB deaths by 90% and TB incidence rate by 80% by 2030.¹

In Bangladesh, tuberculosis (TB) is a major public health problem since long² and Bangladesh is one of the 30 high TB burden countries in the world.^{1,3} The goal of National Tuberculosis Control Program is to reduce morbidity, mortality and transmission of TB until it is no longer a public health problem.²

Tuberculosis is associated with weakened immune system and chronic infectious disease. Tuberculosis has been reported in association with acute lung infections (viral, bacterial, fungal), HIV infection, chronic helminth infestations, diabetes mellitus, chronic lung disease (e.g., COPD, pneumoconiosis), chronic kidney disease or end stage renal failure, gut malabsorption or gastric bypass surgery, skin disorders (e.g. psoriasis, alopecia areata), sarcoidosis, congenital and other immunodeficiencies, autoimmune diseases, solid organ transplants and cancers.^{9,10}

It is not uncommon that a tuberculosis case may also suffer from other long term conditions.⁹ With the increasing pace of globalization, improved life expectancy, ageing populations and the increase in long-term conditions indicate that "multimorbidity" (MM) or the coexistence of two or more chronic conditions in the same individual is rising.⁷

The consequences of multimorbidity include impaired functional status, decreased quality of life, increase in mortality, polypharmacy and complications of treatment, higher utilization of health care system and greater susceptibility to failures of care delivery and coordination and other safety issues.^{7,22} So, finding out the extent of multimorbidity (prevalence and associated factors) among tuberculosis cases is also very important. This study was expected to provide

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information about the extent of multimorbidity among tuberculosis cases in context of Bangladesh.

MATERIALS AND METHODS

This cross-sectional study was carried out from January 2017 to December 2017. Eight (8) DOTS centers (National Institute of Diseases of the Chest and Hospital (NIDCH), Dhaka; 250 Bedded TB Hospital, Shyamoli; Dhaka Medical College and Hospital, Upazila Nirbahi parishad, Mymensingh; Mymensingh Medical College and Hospital; Upazila Health Complex Trishal; Upazila Health Complex Nandail and Netrokona District Hospital) under National Tuberculosis Control Program from 3 districts (Dhaka, Mymensingh and Netrokona) under Dhaka and Mymensingh divisions were selected conveniently. By convenient sampling, 227 TB cases registered in selected DOTS centers were enrolled in this study. Available spot cases were interviewed at DOTS centers and a few listed cases (record review) who resided nearby the selected DOTS centers were contacted over phone whether they were available or not and then they were interviewed at their home. A pretested, semi-structured questionnaire was used for data collection. Data were collected by face to face interview, observation and medical records were reviewed for diagnosed chronic diseases. Those TB cases who had chronic conditions but medical records could not be reviewed, were excluded from this study. Informed consent was taken from the respondents and parents/legal guardians (in case of children). A TB case having two or more chronic medical conditions were considered as TB-MM subjects and “TB without MM subjects” if they had no MM.

Prior to commencement of the study, the research protocol was approved by the Ethical Review Board (ERB) of National Institute of Preventive and Social Medicine (NIPSOM). Permission was also taken from the selected DOTS centers. All ethical issues were maintained throughout the study.

Data were processed and analyzed using SPSS version 16.0. Descriptive data were expressed in frequency and percentage. Chi-square test was conducted to find out association of variables with multimorbidity.

RESULTS

A total of 227 TB cases were interviewed during 1 year study period. Mean age of the tuberculosis cases was 39 (± 17.01) years. About half (40.1%) of them belonged to 20-39 years age group. Regarding sex distribution, 57.7% were male and 42.3% were female. Higher number of cases (41.4%) completed their secondary education. More than half of the cases lived in urban area (57.7%) and 29.5% cases were housewives. Mean average monthly income of families of TB cases was 33731.28 (± 23381.09) BDT and half (50.2%) of them had their average monthly family income 15001-30000 BDT. Majority (81.9%) of the TB cases was new case and 18.1% were relapse cases. More than half (67%) of the cases suffered from

pulmonary tuberculosis and rest suffered from extra-pulmonary tuberculosis (33%). Regarding smoking habit, 19.4% TB cases were currently smoker and majority (80.6%) were found as non-smoker. BCG scar was seen in 55.5% cases (Table-I).

Table-I: Socio-demographic and health related characteristics of TB cases (n=227)

| Characteristics | Frequency (f) | Percentage(%) |
|-----------------------------|--------------------|---------------|
| Age (in years) | | |
| <20 | 27 | 11.9 |
| 20-39 | 91 | 40.1 |
| 40-59 | 64 | 28.2 |
| >59 | 45 | 19.8 |
| Mean±SD | 39±17.01 | |
| Sex | | |
| Male | 131 | 57.7 |
| Female | 96 | 42.3 |
| Educational status | | |
| Never attended school | 75 | 33 |
| Up to secondary | 94 | 41.4 |
| Above secondary | 58 | 25.5 |
| Occupation | | |
| Unemployed | 49 | 21.6 |
| Service holder | 42 | 18.5 |
| Business | 34 | 15 |
| Farmer | 25 | 11 |
| Laborer | 10 | 4.4 |
| Housewife | 67 | 29.5 |
| Family Income (BDT/ month) | | |
| Up to 15000 | 34 | 15 |
| 15001-30000 | 114 | 50.2 |
| >30000 | 79 | 34.8 |
| Mean±SD | 33731.28 ±23381.09 | |
| Area of residence | | |
| Urban | 131 | 57.7 |
| Rural | 96 | 42.3 |
| Types of Tuberculosis | | |
| Pulmonary | 152 | 67 |
| Extra-pulmonary | 75 | 33 |
| Types of Cases | | |
| New | 186 | 81.9 |
| Relapse | 41 | 18.1 |
| Smoking status | | |
| Currently smoker | 44 | 19.4 |
| Non-smoker | 183 | 80.6 |
| BCG scar | | |
| Seen | 126 | 55.5 |
| Not seen | 101 | 44.4 |

Among 227 TB cases, 29 (12.8%) cases were found as TB-MM subjects, where 20 had diabetes mellitus, 19 had hypertension, 7 had chronic lung diseases, 6 had arthritis, 6 had chronic kidney disease, 2 had cancer, 2 had Wegener's granulomatosis and 1 had chronic antral gastritis (Table-II).

Among 29 TB-MM subjects, 24 had two and 5 had three chronic diseases.

Table-III shows that age (years, $p < 0.001$), sex ($p = 0.034$), smoking status ($p = 0.028$), average monthly family income (BDT/month, $p = 0.001$) and BCG vaccination status ($p < 0.001$) were found to be significantly associated with multimorbidity status in TB cases.

Table-II: Chronic diseases among TB-MM subjects (n=29)

| Chronic diseases | Frequency (f) | Percentage (%) |
|--------------------------|---------------|----------------|
| Diabetes mellitus | 20 | 69 |
| Hypertension | 19 | 65.5 |
| Chronic lung diseases | 7 | 24.1 |
| Arthritis | 6 | 20.7 |
| Chronic kidney disease | 6 | 20.7 |
| Cancer | 2 | 6.9 |
| Wegener's granulomatosis | 2 | 6.9 |
| Chronic antral gastritis | 1 | 3.4 |

Table-III: Association between multimorbidity and health related characteristics of TB cases (n=227)

| Characteristics | | TB-MM subjects n=29 n (%) | TB without MM subjects n=198 n (%) | p value χ^2 |
|---------------------------|---------------------------|---------------------------------|--|---------------------|
| Age (years) | <40 | 2 (1.7%) | 116 (98.3) | <0.001* |
| | ≥40 | 27 (24.8%) | 82 (75.2) | |
| Sex | Male | 22 (16.8) | 109 (83.2) | 0.034* |
| | Female | 7 (7.3) | 89 (92.7) | |
| Educational status | Never attended school | 13 (17.3) | 62 (82.7) | 0.225 |
| | Up to secondary | 8 (8.5) | 86 (91.5) | |
| | Above secondary | 8 (13.8) | 50 (86.2) | |
| Occupational status | Unemployed-housewife | 11 (9.5) | 105 (90.5) | 0.290 |
| | Serviceholder-businessman | 13 (17.1) | 63 (82.9) | |
| | Farmer-laborer | 5 (14.3) | 30 (85.7) | |
| Family income (BDT/month) | Up to 15000 | 6 (17.6) | 28 (82.4) | 0.001* |
| | 15001-30000 | 5 (4.4) | 109 (95.6) | |
| | >30000 | 18 (22.8) | 61 (77.2) | |
| Area of residence | Urban | 20 (15.3) | 111 (84.7) | 0.189 |
| | Rural | 9 (9.4) | 87 (90.6) | |
| Type of tuberculosis | Pulmonary | 19 (12.5) | 133 (87.5) | 0.860 |
| | Extra-pulmonary | 10 (13.3) | 65 (86.7) | |
| Type of cases | New | 24 (12.9) | 162 (87.1) | 0.902 |
| | Relapse | 5 (12.2) | 36 (87.8) | |
| Smoking status | Currently smoker | 10 (22.7) | 34 (77.3) | 0.028* |
| | Non-smoker | 19 (10.4) | 164 (89.6) | |
| BCG scar | Seen | 4 (3.2) | 122 (96.8) | <0.001* |
| | Not seen | 25 (24.8) | 76 (75.2) | |

BCG= Bacillus Calmette-Guérin, BDT= Bangladeshi Taka; p value was extracted using chi square test *indicates statistically significant;

DISCUSSION

Over recent years, people living with multiple chronic conditions known as “multimorbidity” is rising in prevalence^{7,11}, both in developed¹² and developing countries.¹³ On the other hand, tuberculosis, a chronic infectious disease, is a major public health problem in Bangladesh for a long period of time. Tuberculosis can frequently co-occur with other long-term medical conditions.^{8,9,10} Therefore, present study was designed to find out the extent of multimorbidity among tuberculosis cases in our country and it should be stated that, a study was conducted on general rural elderly people to investigate multimorbidity in our country²⁴; another study was found regarding tuberculosis-diabetes comorbidity¹⁴ but studies regarding multimorbidity in similar tuberculosis population were very rarely found in Bangladesh. However, in Brazil, a study was conducted on all the reported TB cases registered in their National Notification System showed increased number of tuberculosis subjects presenting with multimorbidity (MM). MM increases the iniquity contributing to adverse clinical outcomes and treatment response in patients with TB.⁴

In this study, Mean age (including standard deviation) of the tuberculosis cases was 39 (± 17.01) years and most of the cases belonged to the 20-39 years age group (40.1%). More than half of the tuberculosis cases in this study were male (57.7%) and the rest were female (42.3%). Almost half (41.4%) of the tuberculosis cases completed secondary education. and half of the cases (50.2%) belonged to 15001-30000 taka income group. People from urban area (57.7%) suffered more from tuberculosis than rural area (42.3%). Regarding clinical characteristics of TB cases, more than half of the cases suffered from pulmonary tuberculosis (67%) and rest suffered from extra-pulmonary tuberculosis (33%). Majority of the cases (81.9%) were new cases and rest were relapse cases (18.1%). Tobacco smoking is associated with poor TB treatment results.^{15,16} In this study, 19.4% cases were found as currently smoker. BCG scar was seen in 55.5% of all TB cases. Malabika Sarker et al. conducted a study in order to measure the burden of TB-diabetes co-morbidity in Bangladesh, where the mean age (including standard deviation) of the tuberculosis cases was 39.9 (± 15.5) years, 38.7% were females, about half (53.1%) of them had a family income of 5000–10,000 BDT, 40.7% never attended school and more than half (59.2%) of the cases were from rural areas. About 82.2% had pulmonary TB where most of the cases (98.2%) were new cases.¹⁴

The proportion of multimorbidity among tuberculosis cases (TB-MM subjects) in this study was 12.8% where 24 (82.76%) TB-MM subjects had 2 and 5 (17.24%) had 3 chronic diseases along with tuberculosis. The finding is higher than the prevalence of multimorbidity in tuberculosis cases (1.14%) in Brazil.⁴ However, this difference might be due difference in sample size where that study was conducted on all the tuberculosis cases registered in the National Notification system in Brazil.

Multimorbidity and age has very strong and well-recognized association.^{4,20} The prevalence of multimorbidity increases substantially with age.⁷ In this study, proportion of multimorbidity among tuberculosis cases was significantly higher in age group ≥ 40 years ($p < 0.001$). This finding agrees with Reis-Santos et al study where 40-59 years (OR: 17.89; 95% CI, 5.71-56.03) and those ≥ 60 years (OR: 44.11; 95% CI, 14.09-138.06) were more likely to develop TB-MM.⁴

Gender is also one of the determinants of both TB²³ and MM.^{7,21} Generally, TB is more frequent in men²³ but women more often develop multimorbidity.²¹ In this study, sex of the tuberculosis cases was found to be significantly associated with multimorbidity ($p = 0.034$). In Brazil, simultaneous occurrence of TB and MM was lower for males (OR: 0.63; 95% CI, 0.52-0.76).⁴

Level of education can affect the probability of having tuberculosis and multimorbidity.⁴ Our study shows that higher proportion of multimorbidity was found in tuberculosis cases who never attended school ($p > 0.05$). The study conducted in Brazil revealed that higher proportion (1.7%) of TB-MM subjects were illiterate.⁴

In our study we found that urban tuberculosis cases had higher proportion of multimorbidity than rural cases ($p > 0.05$). Another previous study that showed TB-MM subjects were less likely to live in rural areas (OR: 0.63; 95% CI, 0.42-0.95).⁴ In this study, monthly family income was found significantly ($p = 0.001$) associated with multimorbidity in tuberculosis cases. It is known to us that poverty is one of the determinants of tuberculosis. A study conducted on multimorbidity in rural elderly people in our country showed that persons who were relatively better-off, suffered from more chronic conditions.²⁴

Type of tuberculosis and type of tuberculosis cases are important factors in disease diagnosis and treatment but no significant association was found between these factors and multimorbidity, which reflects the findings of the Brazilian study.⁴

Prevalence of multimorbidity was significantly ($p=0.028$) higher among currently smoker tuberculosis cases. Smoking prevalence is often high among TB cases. As a result, prevalence of other smoking-related conditions can be high as well in people with TB.^{15,16} A study conducted on multimorbidity in general population in China showed that smoker people were more likely to develop multimorbidity.¹³

In this study it was observed that proportion of multimorbidity was significantly ($p<0.001$) higher in tuberculosis cases without having BCG scar. However studies regarding TB vaccine efficacy and multimorbidity were very rarely found.

CONCLUSIONS

The current study showed increased number of TB-MM subjects in our country and multimorbidity among tuberculosis cases was significantly associated with age, sex, monthly family income, smoking habit and BCG vaccine scar. However, the study was conducted on a small scale. Moreover, tuberculosis cases who had other morbidities but medical records could not be reviewed, were excluded from this study. So, prevalence of multimorbidity in TB cases might have been underestimated. Subjects with MM may be prone to TB-drug intolerance, treatment failure and need a longer duration of treatment.⁴ Therefore; it is proposed that a systemic nationwide study should be conducted on each tuberculosis case to get the actual national scenario of multimorbidity.

REFERENCES

1. World Health Organization. Global tuberculosis report 2017 [Internet]. Geneva: World Health Organization; 2017 [cited 2017 Nov 8]. 262 p.
2. NTP. National Tuberculosis Control in Bangladesh. Annual Report 2016. National Tuberculosis Control Program, DGHS, Dhaka.
3. World Health Organization. "Global tuberculosis report 2016."
4. Reis-Santos B, Gomes T, Macedo LR, Horta BL, Riley LW, Maciel EL. Prevalence and patterns of multimorbidity among tuberculosis patients in Brazil: a cross-sectional study. *International journal for equity in health*. 2013 Dec;12(1):61.
5. Ording AG, Sørensen HT. Concepts of comorbidities, multiple morbidities, complications, and their clinical epidemiologic analogs. *Clinical epidemiology*. 2013;5:199.
6. Barnett K, Mercer SW, Norbury M, Watt G, Wyke S, Guthrie B. Epidemiology of multimorbidity and implications for health care, research, and medical education: a cross-sectional study. *The Lancet*. 2012 Jul 7;380(9836):37-43.
7. Mercer S, Furler J, Moffat K, Fischbacher-Smith D, Sanci L. Multimorbidity: Technical Series on Safer Primary Care.
8. Mori T, Leung CC. Tuberculosis in the global aging population. *Infectious Disease Clinics*. 2010 Sep 1;24(3):751-68.
9. Bates M, Marais BJ, Zumla A. Tuberculosis comorbidity with communicable and non-communicable diseases. *Cold Spring Harbor perspectives in medicine*. 2015 Nov 1;5(11):a017889.
10. Marais BJ, Lönnroth K, Lawn SD, Migliori GB, Mwaba P, Bates M, et al. Tuberculosis comorbidity with communicable and non-communicable diseases: integrating health services and control efforts. *The Lancet infectious diseases*. 2013 May 1;13(5):436-48.
11. Uijen AA, van de Lisdonk EH. Multimorbidity in primary care: prevalence and trend over the last 20 years. *The European journal of general practice*. 2008 Jan 1;14(sup1):28-32.
12. Barnett K, Mercer SW, Norbury M, Watt G, Wyke S, Guthrie B. Epidemiology of multimorbidity and implications for health care, research, and medical education: a cross-sectional study. *The Lancet*. 2012 Jul 7;380(9836):37-43.
13. Wang HH, Wang JJ, Wong SY, Wong MC, Li FJ, Wang PX, et al. Epidemiology of multimorbidity in China and implications for the health care system: cross-sectional survey among 162,464 community household residents in southern China. *BMC Med*. 2014;12(1):188.
14. Sarker M, Barua M, Guerra F, Saha A, Aftab A, Latif AM, et al. Double trouble: prevalence and factors associated with tuberculosis and diabetes comorbidity in Bangladesh. *PloS one*. 2016 Oct 31;11(10):e0165396.
15. World Health Organization. A WHO/The Union monograph on TB and tobacco control: joining efforts to control two related global epidemics. In: A WHO/the Union monograph on TB and tobacco control: joining efforts to control two related global epidemics 2007.

16. Murray JF, Pio A, Ottmani S. PAL: a new and practical approach to lung health. *The International Journal of Tuberculosis and Lung Disease*. 2006 Nov 1;10(11):1188-91.
17. Davenne T, McShane H. Why don't we have an effective tuberculosis vaccine yet?. *Expert review of vaccines*. 2016 Aug 2;15(8):1009-13.
18. Rafi W, Ribeiro-Rodrigues R, Ellner JJ, Salgame P. Coinfection-helminthes and tuberculosis. *Current opinion in HIV and AIDS*. 2012 May 1;7(3):239-44.
19. Zevallos K, Vergara KC, Vergara A, Vidal C, Garcia HH, Evans CA. Tuberculin skin-test reactions are unaffected by the severity of hyperendemic intestinal helminth infections and co-infections. *The American journal of tropical medicine and hygiene*. 2010 Aug 5;83(2):319-25.
20. Fortin M, Stewart M, Poitras ME, Almirall J, Maddocks H. A systematic review of prevalence studies on multimorbidity: toward a more uniform methodology. *The Annals of Family Medicine*. 2012 Mar 1;10(2):142-51.
21. Fortin M, Hudon C, Haggerty J, van den Akker M, Almirall J. Prevalence estimates of multimorbidity: a comparative study of two sources. *BMC health services research*. 2010 Dec;10(1):111.
22. Gijsen R, Hoeymans N, Schellevis FG, Ruwaard D, Satariano WA, van den Bos GA. Causes and consequences of comorbidity: a review. *Journal of clinical epidemiology*. 2001 Jul 1;54(7):661-74.
23. Lawn SD, Zumla AI. Tuberculosis. *Lancet*. 2011; 378(9785):57-72.
24. Khanam MA, Streatfield PK, Kabir ZN, Qiu C, Cornelius C, Wahlin Å. Prevalence and patterns of multimorbidity among elderly people in rural Bangladesh: a cross-sectional study. *Journal of health, population, and nutrition*. 2011 Aug;29(4):406.

Original Article

Anemia in Male with Type 2 Diabetes Mellitus

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Abstract

Diabetes mellitus (DM) is the leading cause of many chronic diseases. Anemias in men with diabetes mellitus greatly contribute to the pathogenesis and progression of cardiovascular disease and aggravate diabetic nephropathy and retinopathy. The present study was carried out to estimate the Hb level, to determine the total count of RBC and reticulocyte count to evaluate the anemia and FBG & HbA_{1c} to assess their glycemic status. The cross-sectional study was conducted in the Department of Physiology, Dhaka Medical College, Dhaka from January, 2011 to December, 2011. Total 90 male subjects were selected with the age ranging from 40 to 60 years. Among them 60 subjects were diabetic, 30 subjects were with controlled (B₁) and other 30 subjects with uncontrolled (B₂) type 2 diabetes mellitus. They were selected from Outpatient Department of BIRDEM Hospital by random basis. And the rest 30 age-matched, healthy non-diabetic male subjects were considered as control group (A) for comparison. They were selected from Dhaka City through personal contact. For statistical analysis unpaired Student's t-test was performed. The result was expressed as mean (±SD) among the groups. In this study, there are significant decrease in Hb level, total RBC count and increase

in reticulocyte count in study groups. The prevalence of anemia is high in patients with type 2 DM, which affects quality of life of diabetic patients and is associated with disease progression and co-morbidities that contribute significantly to the increasing risk of cardiovascular diseases.

Key words: Type 2 diabetes mellitus, HbA_{1c}, Hb. level, RBC count, reticulocyte count.

INTRODUCTION

Diabetes mellitus (DM) is a metabolic disorder which has a great impact worldwide. Diabetes mellitus is characterized by an elevation of blood glucose level caused by a relative or absolute deficiency of insulin. Type-2 DM may cause insulin resistance which can lead glucose accumulation in the circulation and consequently a hyperglycemic state, generating homeostatic and systemic imbalance.³

Studies from 91 countries it is found that in 2010 there will be 285 million people worldwide with diabetes, with considerable disparity between populations and regions. It is also found that the pattern of diabetes varies considerably according to countries' economic status. For developed countries, the majority with diabetes are aged over 60 years, whereas for developing countries most people with diabetes are of working age, between 40 and 60 years. This difference is likely to still be present in 2030. Population growth, ageing of populations and urbanization with associated lifestyle change is likely to lead to a 54% increase in worldwide numbers with diabetes by 2030.¹

Epidemiological data showed that in 2012 there were 371 million people affected with the disease in the world, and it is estimated that in the year of 2030 we will have about 552 million diabetics. This worldwide prevalence affects about 7% of the general population². In Bangladesh prevalence of diabetes was found 9.2% in the year of 2013.¹⁸

According to International Diabetes Federation (IDF) Diabetes Atlas Seventh Edition, it is estimated that about 415 million adults aged 20-70 living with diabetes and 5.0 million deaths were attributed to diabetes globally in 2015.¹⁹ According to WHO, diagnostic criteria of diabetes

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mellitus are fasting blood glucose ≥ 7.0 mmol/l, 2 hrs after blood glucose ≥ 11.1 mmol/l and HbA_{1c} $\geq 6.5\%$.¹⁴

DM is a common disease, caused by many etiological factors and oxidative damage is one of the risk factors to play a major role in the pathogenesis of this disease.⁴ High glucose level in diabetes inhibits G6PD expression and activity in endothelial cells, kidney, liver and RBCs, which leads to oxidative damage, cellular dysfunction and organ damage. This oxidative damage causes early destruction of RBCs which leads to anemia.⁵ High glucose concentrations increase the level of Reactive Oxygen Species (ROS). The ROS, generated by hyperglycemia, causes many of the complications of diabetes, such as nephropathy, retinopathy, and neuropathy. Recent evidence indicated that oxidative damage markedly rose in type- 2 diabetes mellitus.⁶

Hyperglycemia has a direct relationship with the development of an inflammatory condition showed by the increased expression of pro-inflammatory cytokines. The elevation of pro-inflammatory cytokines plays an essential role in insulin resistance and induces the appearance of cardiovascular complications diabetic micro and macro vascular disease, kidney disease and anemia.^{12, 21}

According to Escorcio et al. by increasing pro- inflammatory cytokines, anti-erythropoietic effect occurs, also promotes apoptosis of immature erythrocytes causing a decrease in the number of circulating erythrocytes and consequently causing a reduction of circulating hemoglobin.²² Anemia represents an emerging global health problem that negatively impacts quality of life.²³ The anemic framework promotes reduced exercise capacity, fatigue, anorexia, depression, cognitive dysfunction, decreased libido, and other factors, which increase cardiac risk patients and depress the quality and life expectancy of the same.²⁴

Some observer conducted a study to find out the association between anemias with new onset diabetes. They found that out of 1500 patients, 83 (5.5%) were anemic and 45 of the 83 patients were found to have identifiable cause of anemia.⁷ Again, some people (Meir (2003)) also observed the signs of hemolysis in diabetic subjects and they found decreased hemoglobin concentration, and increased reticulocyte count in them.⁸ Some researchers (Ranil et al. (2010)) observed that the prevalence of anemia among the type 2 diabetes subjects was 12.3%. Similar study was made by Craig et al. (2005). They found that 8 of the 45 male diabetic patients (17.8%) were classified as anemic.^{9, 10} A cross-sectional study was made by Thomas

et al. (2003) on 820 patients with diabetes mellitus. Among them, about 190 patients (23%) had anemia of unrecognized origin.¹¹

Under these circumstances, anemia in patients with diabetes must be treated once diagnosed, since it may contribute to the pathogenesis and progression of cardiovascular disease and serious diabetic nephropathy and retinopathy. Anemia in diabetic person has a significant adverse effect on quality of life and is associated with disease progression and the development of co-morbidities. The regular screening for anemia, along with other complications associated with diabetes, can help slowing the progression of vascular complications in those patients.^{12, 13}

MATERIALS AND METHODS

This cross sectional study was done in the department of Physiology in Dhaka Medical College Dhaka from January, 2011 to December 2011. Protocol of this study was approved by Ethical review committee of Dhaka Medical College and Diabetic Association of Bangladesh. For this study 60 male, age (40-60 years), diabetic subjects with FBG level ≥ 7.0 mmol/l and HbA_{1c} $\geq 6.5\%$ and duration of diabetes > 3 years were selected from BIRDEM hospital. All the study subjects were on oral hypoglycemic drugs. Thirty healthy adult male were considered as control group for comparison. After selection of the subjects, the nature, purpose and benefit of the study were explained to each subject in details and were encouraged for voluntary participation. They were also allowed to withdraw from the study whenever they feel like. Informed written consent was taken from the participants. Before taking blood, detailed family and medical history were taken. Anthropometric measurement of the subjects was done and blood pressure was measured. All the information's were recorded in a prefixed questionnaire. With aseptic precaution, 5 ml of venous blood was collected from ante-cubital vein by a disposable plastic syringe from each subject for estimation of HbA_{1c} and FBG level was estimated in the laboratory of the Biochemistry Department of BIRDEM hospital and the G6PD tests were done in the laboratory of the Department of chemical biochemistry, AFIP, Dhaka Cantonment, Dhaka. All the parameters were expressed as mean \pm SD (standard deviation). For statistical analysis was done by unpaired Student's t- test. P value < 0.05 was accepted as level of significance. Statistical analyses were performed by using a computer based statistical program SPSS (Statistical package for social science) Version 12.

RESULTS

A total number of 90 adult male subjects were selected for this study. Among them, 60 subjects were adult male with type 2 diabetes and 30 healthy subjects with same age range were selected as control group for comparison. The mean (\pm SD) FBG levels were 5.12 ± 0.76 , 7.48 ± 1.27 and 9.14 ± 2.10 mmol/L respectively in control subjects (group A), Controlled diabetes mellitus (group B₁) and Uncontrolled diabetes mellitus (group B₂). In this study, the mean (\pm SD) FBG level was significantly ($p < 0.0001$) higher in group B₁ and group B₂ in comparison to that of group A. Again, FBG level was significantly ($p < 0.0001$) higher in group B₂ than that of group B₁. The mean (\pm SD) HbA_{1c} levels were 5.83 ± 0.75 , and 9.98 ± 2.10 in group B₁ and B₂ respectively. In this study, the mean (\pm SD) HbA_{1c} level was significantly ($p < 0.0001$) higher in group B₂ than that of group B₁. (Table-I)

Table I. FBG level in different groups (n=90) and HbA_{1c} level in study groups (n=60)

| Parameters | Group A (n=30) | Group B ₁ (n=30) | Group B ₂ (n=30) |
|-----------------------------|--------------------------------|------------------------------------|----------------------------------|
| FBG (mmol/L) | 5.12 ± 0.76 (3.80-6.90) | 7.48 ± 1.27 (5.10 -9.90) | 9.14 ± 2.10 (6.00 -15.50) |
| HbA _{1c} (%) | | 5.83 ± 0.75 (4.30-6.90) | 9.98 ± 2.10 (7.30-15.10) |
| <i>Statistical analysis</i> | | | |
| Groups | FBG (mmol/L) (p value) | HbA _{1c} (%) (p value) | |
| A vs B1 | 0.0001*** | | |
| A vs B2 | 0.0001*** | | |
| B1 vs B2 | 0.0001*** | 0.0001*** | |

Results are expressed as Mean \pm SD & figures in parenthesis indicate the range.

The test of significance was calculated and p values < 0.05 was accepted as level of significance.

Group A : non- diabetes ns = Not significant
 Group B₁ : Controlled diabetes * = Significant at $P < 0.05$
 Group B₂ : Uncontrolled diabetes
 n = Number of subjects ** = Significant at $P < 0.01$
 *** = Significant at $P < 0.001$

The mean (\pm SD) hemoglobin levels were 14.24 ± 0.75 , 12.88 ± 1.16 and 11.72 ± 1.42 gm/dl in control non-diabetic

(group A), Controlled diabetes mellitus (group B₁) and Uncontrolled diabetes mellitus (group B₂) respectively. In this study, the mean hemoglobin level was significantly ($p < 0.0001$) lower in group B₁ and group B₂ in comparison to that of group A. Again, this value was significantly ($p < 0.001$) lower in group B₂ than that of group B₁. The mean (\pm SD) RBC count were 5.09 ± 0.50 , 4.86 ± 0.55 and 4.58 ± 0.77 $10^{12}/L$ in groups A, B₁ and B₂ respectively. In this study, the mean RBC count was lower in group B₁ and group B₂ than that of group A. The mean RBC count in group B₂ was lower than that of group B₁. In comparison to group A, it was significantly ($p < 0.003$) lower in B₂ but non-significant in B₁. The mean (\pm SD) reticulocyte count were 1.02 ± 0.53 , 1.49 ± 0.93 and 2.26 ± 0.99 % in group A, B₁ and B₂ respectively. In this study, the mean reticulocyte counts in different groups were within normal range. But the differences of these mean values were significantly lower in group B₂ ($p < 0.0001$) and in group B₁ ($p < 0.019$) than group A. Within the study group reticulocyte count was significantly ($p < 0.003$) higher in group B₂ than that of group B₁. (Table- II).

Table II: Hemoglobin conc., total count of RBC, Reticulocyte count in different groups (n=90)

| Parameters | Group A (n=30) | Group B1 (n=30) | Group B2 (n=30) |
|------------------------------|-----------------------------------|-----------------------------------|----------------------------------|
| Hb level (gm/dl) | 14.24 ± 0.75 (13.00-15.90) | 12.88 ± 1.16 (10.20-15.30) | 11.72 ± 1.42 (8.80-15.00) |
| RBC count ($10^{12}/L$) | 5.09 ± 0.50 (4.35-6.96) | 4.86 ± 0.55 (3.99-6.47) | 4.58 ± 0.77 (3.04-6.53) |
| Reticulocyte count (%) | 1.02 ± 0.53 (1.50-3.00) | 1.49 ± 0.93 (0.20-4.00) | 2.26 ± 0.99 (0.50-4.00) |
| <i>Statistical analysis</i> | | | |
| Groups | Hemoglobin (p value) | RBC count (p value) | Reticulocyte count (p value) |
| A vs B1 | 0.0001*** | 0.097ns | 0.019* |
| A vs B2 | 0.0001*** | 0.003** | 0.0001*** |
| B1 vs B2 | 0.001** | 0.108ns | 0.003** |

Results are expressed as Mean \pm SD & figures in parenthesis indicate the range.

The test of significance was calculated and p values < 0.05 was accepted as level of significance.

Group A : non- diabetes ns = Not significant
 Group B1 : Controlled diabetes * = Significant at $P < 0.05$
 Group B2 : Uncontrolled diabetes n = Number of subjects
 ** = Significant at $P < 0.01$ *** = Significant at $P < 0.001$

In this study, group B₁ (n=30), mean Hb level <13gm/dl was found in 15 (50.0 %) subjects and ≥13gm/dl was found in 15 (50.0 %) subjects. And group B₂ (n=30), mean Hb conc. <13gm/dl was found in 26 (86.7 %) subject and ≥13gm/dl was found in 4 (13.3 %) subjects. (Figure-I & II).

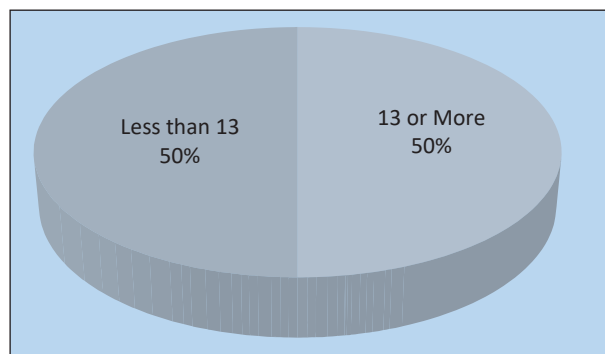


Figure 1. Distribution of study subjects (group B1 as controlled diabetes) by Hb level (n=30)

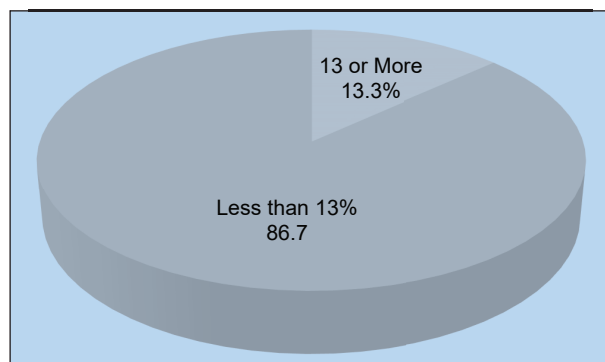


Figure 2. Distribution of study subjects (group B2 as uncontrolled diabetes) by Hb level (n=30)

DISCUSSION

The present study was undertaken to observe anemic status in type 2 diabetic male subjects. For this, fasting blood glucose (FBG) level and HbA_{1c} level were done to observe their glycemic status, and hemoglobin level, total count of RBC and reticulocyte count were also studied in all these subjects to find out their hemolytic status. Furthermore, FBG level, Hb conc., total count of RBC and reticulocyte count were also studied in apparently healthy persons to find out the base line data and also for comparison.

Hb conc. was significantly lower in diabetic males than that of the non-diabetic males. Similar findings were made some authors.⁷ In our country, Akhter et. al.¹⁴ showed Hb

conc. was significantly lower in diabetic females than that of the non-diabetic females.

Chronic disease such as DM, are accompanied by mild to moderate anemia, often called anemia of chronic disease.¹⁵ There are decreased values of Hb, hematocrit and red blood cells, which can be associated with a normocytic normochromic anemia, just like of an anemia of chronic disease. In mild to moderate anemia survival of red blood cells are decreases; it is about 80 days instead of 120 days. This phenomenon is attributed to hyperactivity state mononuclear phagocyte system, triggered by infectious, inflammatory, or neoplastic process, leading to early removal of circulating red blood cells.¹⁶

In the present study, early hemolysis is also observed in this study by decreased Hb%, RBC count and increased reticulocyte count. The reticulocytosis may be due to compensatory response to hemolysis. And HbA_{1c} test was done to assess their glycemic control, as HbA_{1c} express the average amount of glucose in the last three month.

CONCLUSIONS

In this study, there are significant decrease in Hb conc., total count of RBC and increase in reticulocyte count in study groups. Nevertheless mean value of RBC and reticulocyte count were within normal range, but Hb conc. was below the normal limit. The prevalence of anemia is high in patients with type 2 DM. This set of changes characterizes the anemia as chronic disease, which has a significant adverse effect on quality of life of diabetic patients and this is associated with the progression of the disease; the development of co-morbidities significantly contributes to the increased risk of cardiovascular disease.

REFERENCES

1. Shaw JE, Sicree RA, & Zimmet PZ. Global estimates of the prevalence of diabetes for 2010 and 2030. Diabetes research and clinical practice, 2010; vol.87, no.1, pp.4-14.
2. Pereira PF, Alfenas RDCG, & Araujo RMA, 'Does breastfeeding influence the risk of developing diabetes mellitus in children? A review of current evidence,' Journal de Pediatria, 2014; vol.90, no.1, pp.7-15.
3. Zhang X, cui X, Li F, et al. Association between diabetes mellitus with metabolic syndrome and diabetic microangiopathy. Experimental and therapeutic medicine. 2014; vol.8, no.6, pp.1867-1873.

4. Wan GH, Tsai C, &Chiu DTY. Decreased blood activity at glucose-6-phosphate dehydrogenase, associates with increased risk for diabetes mellitus. *Endocrine*, 2002; vol. 19, pp. 191-195.
5. Zhang Z, Liew CW, Handy DE, Zhang Y, Leopold JA, Hu J, Guo L, Kulkarni RN, Loscalzo J &Stanton RC. High glucose inhibits glucose-6-phosphate dehydrogenase, leading to increased oxidative stress and α -cell apoptosis. *The FASIB Journal*, 2010; vol. 24, no.5, pp. 1497-1505.
6. West CI. Radicals and oxidative stress in diabetes. *British Diabetic Association*, 2000; vol.17, pp. 171-180.
7. Pilona AP, Litonjua P, Devaraj S, Santa LA &Raskin, P. Anemia associated with new-onset diabetes: improvement with blood glucose control. *Endocrine practice*, 2002; vol. 8, no. 4, pp. 276-281.
8. Meir A, Kleinman Y, Rund D &Da'as N 2003. Metforming induced hemolytic anemia in a patient with glucose-6- phosphatedehydrogenase deficiency. *Diabetes care*, 2002; vol. 26, no.3, pp. 956-57.
9. Ranil PK, Raman R, Rachepalli SR, Pal SS, Kulothungan V, Lakshmipathy P, Satagopan U, Kumaramanickavel G & Sharma T. Anemia and diabetic retinopathy in type 2 diabetes mellitus. *J Assoc Physicians India*, 2010; vol.58, pp. 91-4.
10. Craig KJ, Williams JD, Riley SG, Smith H, Owens DR, Worthing D, Cavill I & Phillips AO. Anemia and diabetes in the absence of nephropathy. *Diabetes Care*, 2005; vol. 28, no.5, pp. 1118-23.
11. Thomas MC, MacIsaac RJ, Tsalamandris C, Power D, Jerums G. Unrecognized anemia in patients-with diabetes: a cross-sectional survey. *Diabetes Care*, 2003; vol.26, no. 4, pp.1164-9.
12. Angelouse A, & Larger E. Anaemia, a common but often unrecognized risk in diabetic patients: a review. *Diabetes & metabolism*, 2005; vol.41, no.1, pp.18-27.
13. Singh DK, Winocour P & Farrington K. Erythropoietic stress and anemia in diabetes mellitus. *Nature reviews endocrinology*, 2009; vol.5, no.4, pp. 204-210.
14. WHO. Anemia. 2012; World health organization.
15. Akhter N, Begum N, Ferdousi S. Glucose 6-phosphete dehydrogenase (G6PD) status in female type 2 diabetes mellitus and its relationship with HbA1C. *J Bangladesh Soc Physiol*, 2010; Dec; 5(2): 60-65.
16. Andrews M, & Arredondo M. Ferritin levels and hepcidin mRNA expression in peripheral mononuclear cells from anemic type 2 diabetic patients. *Biological trace Element Research*, 2012; vol. 149, no.1, pp. 1-4.
17. Barbieri J, Fontela PC, Winkelmann ER, Zimmermann CEP, Sandri YP, Mallet EKV, &Frizzo MN. Anemia in patients with type 2 Diabetes Mellitus. 2015; vol.2015, available from <http://dx.doi.org/10.1155/2015/354737>
18. Rahman MS, Akter S, Abe SK, Islam MR, Mondal NMI, Rahman JAMS, RahmanMM. Awareness, Treatment, and Control of Diabetes in Bangladesh: A Nationwide Population-Based study. *PLoS one* 2015; 10(2): e0118365.
19. IDF Diabetes Atlas Seventh Edition. Available at: <http://www.diabetesatlas.org/key-messages.html>.(accessed:1st April 2016).
20. Pérez B M, Díez IDLT, and Coronado ML. Mobile health applications for the most prevalent conditions by the World Health Organization: review and analysis. *Journal of Medical Internet Research*, 2013; vol. 15, no. 6, article e120.
21. Fava S, Azzopardi J, Ellard S, and Hattersley A. T. ACE gene polymorphism as a prognostic indicator in patients with type 2 diabetes and established renal disease. *Diabetes Care*, 2001; vol. 24, no. 12, pp. 2115–2120.
22. Escorcio CSM, Silva HF, Junior GBS, Monteiro MP, and Gonçalves RP. Evaluation of anemia treatment with EPO and oral and iv iron in patients with chronic kidney disease under hemodialysis. *RBSA*, 2010; vol. 42, no. 2, pp. 87–90.
23. MacCiò A and Madeddu C. Management of anemia of inflammation in the elderly. *Anemia* 2012; vol. 2012, Article ID 563251, 20 p.

Original Article

Diagnostic Accuracy and Validity of Magnetic Resonance Imaging in the Detection of Specific Type of Brain Tumour

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Abstract

Magnetic Resonance Imaging (MRI) is a widely accessible imaging technique for the detection of brain tumours and cancer, which are further confirmed by histopathological examination. Accurate detection of the tumours and its extent is very difficult. The present study attempted to evaluate the convenience of MRI in detection of different grades of astrocytomas, which are the most commonly occurring brain tumours. This cross-sectional study was conducted at the Department of Radiology and Imaging with the collaboration of Department of Neurosurgery and Department of Pathology at Sir Salimullah Medical College (SSMC & MH), Dhaka from January 2013 to December 2013 for a period of one year. The study population was all the diagnosed cases of intracranial astrocytoma patients regardless of their age and sex. The studied included 48 brain tumour (astrocytoma) patients, ages between 13 and 69 years old. All cases having no contraindication for MRI underwent MR examination followed by histopathological examination of the postoperative resected tissues. The findings of the MRI and histopathological examination were compared to find out the test validity of the MRI findings of the different grades of astrocytoma's. The highest sensitivity was found in grade III astrocytoma (90.5%) followed by grade II (85.7%) grade IV (75.0%) and grade I (60.0%). The highest specificity

was found in grade I astrocytoma (97.7%) followed by Grade III (96.3%), grade IV (92.5%) and grade II (91.5%). The highest accuracy was found in both grade I astrocytoma (93.7%) and grade III (93.7%) followed by grade II (92.5%) and grade IV (89.6%). As per the study findings it can be concluded that, MRI has a high diagnostic accuracy and validity for the detection of different grades of astrocytoma.

Key words: Magnetic Resonance Imaging; brain tumours; glioma; astrocytoma; validity test.

INTRODUCTION

Astrocytomas are the most common type of primary brain tumour within the group of brain tumours called gliomas. Plain radiograph was previously used in the past to detect such type of tumours. Cerebral angiography and pneumocephalography were also done; however, none of which was convincing. With the advent of CT and MRI there is revolutionary change in the detection of intracranial tumour. Diagnosing a brain tumor usually begins with magnetic resonance imaging (MRI), which if shows that, there is a tumor in the brain, the most common way to determine the type of brain tumor is to look at the results from a sample of tissue after a biopsy or surgery. MRI scan has made a significant impact on the differential diagnosis of intracranial tumours.³

MRI is more sensitive than CT for detection of intra-cerebral astrocytomas and for signifying their extent and spread. Compared with CT, MRI offers greater contrast resolution, including greater sensitivity for the detection of subacute and chronic haemorrhage in association with tumours and other lesions of brain⁴. MRI also has less ionizing radiation. Delineation of posterior cranial fossa soft tissue anatomy is better visualized with MRI than CT as because MRI lacks beam-hardening artefact⁵ and as it offers imaging from multiple planes⁶, thus accuracy of lesion localization is enhanced. MRI is advanced with providing important information such as, contrast material enhancement, peritumoural oedema, distant tumour foci, haemorrhage, necrosis, mass effect and so on, which are all helpful in characterizing tumour aggressiveness and hence tumour grade.⁷ In this context, this study addresses the diagnostic precision of MRI as a brain investigation tool to assess astrocytoma diagnosis and the tumour staging.

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MATERIALS AND METHODS

This study was designed as cross sectional study which was carried out in the Department of Radiology and Imaging with the collaboration of Department of Neurosurgery and Department of Pathology at Sir Salimullah Medical College (SSMC & MH), Dhaka from January 2013 to December 2013 for a period of one (1) year. A total number of 48 astrocytoma patients were evaluated by purposive sampling technique. Patients who were clinically suspected and CT scan diagnosed cases of intracranial astrocytomas referred to Radiology and Imaging department of Dhaka Medical College Hospital (DMCH) either from OPD or from indoor of DMCH for MRI of brain were included in this study. Prior to the commencement of this study, the research protocol was approved by the ethical committee (Local Ethical committee) of SSMC. All cases having no contraindication for MRI underwent MR examination. Patients were asked for or checked for any metallic or harmful. MR imaging was obtained with 0.3 Tesla machine (HITACHI). T1W image in axial, sagittal and coronal plane were obtained using short TR (500-800ms) and short TE (14-20ms). T2W image in axial and coronal plane were obtained using long TR (3500-4500ms) and long TE (80ms). FLAIR images were also taken. Contrast MRI studies using intravenous Gd-DTPA (Magnevist, 0.1 mmol/Kg) with axial, coronal and sagittal T1Wscanwere performed in all cases. The average time of examination was 45 minutes but ranges from 30-90 minutes. Slice thickness was 5-6 mm with a field of view 230x230 mm and pictures matrix was 256x256 or 192x256. The postoperative resected tissues were examined histopathological in the respective department. MRI scan findings were compared with histopathological reports. Then the collected reports were compared with findings of MRI. Data were collected using a preformed data collection sheet. Base line information was collected from the patient after exploration of different complaints and sign and symptoms. All information regarding clinical features and histo-

pathological results were recorded in a data collection sheet. Statistical analysis was performed by using window based computer software devised with Statistical Packages for Social Sciences (SPSS-17), 95% confidence limit was taken.

Results

Table 1: Distribution of Study Population according to MRI and Histopathological Diagnosis (n=48)

| MRI Diagnosis | Frequency | Percent |
|-------------------------------------|-----------|--------------|
| Pilocytic astrocytoma (Grade-I) | 4 | 8.3 |
| Low grade astrocytoma (Grade-II) | 15 | 31.2 |
| Anaplastic astrocytoma (Grade-III) | 20 | 52.1 |
| Glioblastoma multiformis (Grade-IV) | 9 | 16.7 |
| Total | 48 | 100.0 |
| Histopathological Diagnosis | Frequency | Percent |
| Pilocytic astrocytoma (Grade-I) | 5 | 10.4 |
| Low grade astrocytoma (Grade-II) | 14 | 29.2 |
| Anaplastic astrocytoma (Grade-III) | 21 | 43.8 |
| Glioblastoma multiformis (Grade-IV) | 8 | 16.7 |
| Total | 48 | 100.0 |

Table 1 shows the distribution of study population according toMRI and Histopathological Diagnosis. The MRI findings are as followed- Pilocytic astrocytoma (Grade-1) is found in 4(8.3%) cases. Low grade astrocytoma (Grade-II) was found in 15(31.2%) cases. Anaplastic astrocytoma (Grade-III) was found in 20(52.1%) cases. Glioblastoma multiformis (Grade-IV) was found in 9(22.1%) cases. According to histopathological diagnosis, Pilocytic astrocytoma (Grade-1) was found in 5(10.4%) cases. Low grade astrocytoma (Grade-II) is found in 14(29.2%) cases. Anaplastic astrocytoma (Grade-III) was found in 21(43.8%) cases. Glioblastoma multiformis (Grade-IV) was found in 8(16.7%) cases.

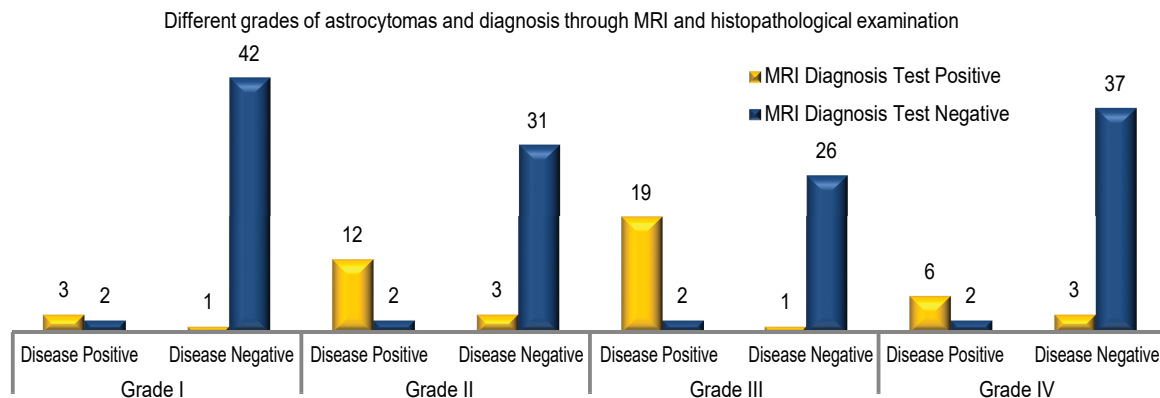


Fig. 1: Comparison of MRI findings with Histopathological Findings during Diagnosis of Different Grades of Astrocytoma (n=48)

The Figure 1 shows the comparison of MRI findings with Histopathological Findings during diagnosis of different Grades of Astrocytoma. In grade I, both histopathological and MRI positive astrocytoma case is found in 3 cases which indicate true positive. Both histopathological and MRI negative astrocytoma case is found in 42 cases which indicate true negative. Histopathological positive but MRI negative case is found in 2 cases which is known as false negative. Histopathological negative but MRI positive case is found in 1 case which is known as false positive. Similarly, in case of Grade II Astrocytoma, true positive cases were 12, 31 cases found to be true negative. False negative result found in 2 cases and in 3 cases found as false positive. In case of Grade III astrocytoma, 19 cases were true positive and 26 cases were true negative; 2 cases were false negative and 1 case was false positive. In case of Grade IV Astrocytoma 6 cases were true positive, 37 cases were true negative, 2 cases were false negative and 3 cases were false positive.

Sensitivity, specificity and accuracy of MRI for the diagnosis of different grades of astrocytoma

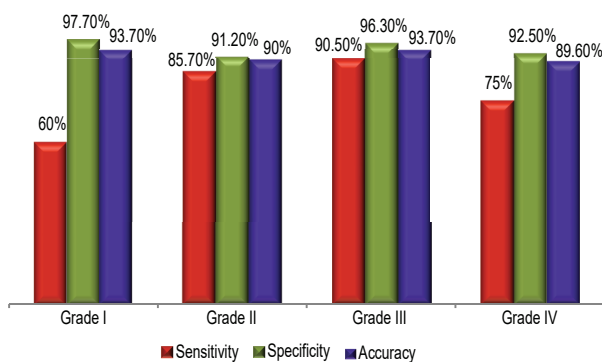


Figure 2: Comparison of Validity and Accuracy of MRI for the Diagnosis of Different grade of Astrocytoma

The sensitivity, specificity, positive predictive value, negative predictive value and accuracy of MRI for the diagnosis of astrocytoma grade I were 60.0% (95% CI 46.1-73.9%), 97.7% (95% CI 93.5-101.9%), 75.0% (95% CI 62.7-87.2%), 95.4% (95% CI 89.5-101.3%) and 93.7% (95% CI 86.8-100.6%) respectively. The sensitivity, specificity, positive predictive value, negative predictive value and accuracy of MRI for the diagnosis of astrocytoma grade II were 85.7% (95% CI 56.1-97.2%), 91.2% (95% CI 75.2-97.2%), 80.0% (95% CI 51.4-94.7%), 93.9% (95% CI 78.4-98.9%) and 90.0% (95% CI 81.5-98.49%) respectively. The sensitivity, specificity, positive predictive value, negative predictive value and accuracy of MRI for

the diagnosis of astrocytoma grade III were 90.5% (95% CI 68.2-98.3), 96.3% (95% CI 79.1-99.8%), 95.0% (95% CI 73.0-99.7%), 92.9% (95% CI 75.0-98.7%) and 93.7% (95% CI 86.8-100.6%) respectively. The sensitivity, specificity, positive predictive value, negative predictive value and accuracy of MRI for the diagnosis of astrocytoma grade IV were 75.0% (95% CI 62.7-87.2%), 97.7% (95% CI 85.5-99.9%), 75.0% (95% CI 53.4-80.0%), 95.4% (95% CI 88.7-101.1%) and 93.7% (95% CI 81.0-98.2%) respectively. The highest sensitivity was found in grade III astrocytoma (90.5%) followed by grade II (85.7%), grade IV (75.0%) and grade I (60.0%). The highest specificity was found in grade I astrocytoma (97.7%) followed by grade III (96.3%), grade IV (92.5%) and grade II (91.5%). The highest Accuracy was found in grade I astrocytoma (93.7%) and grade III (93.7%) followed by grade II (92.5%) and grade IV (89.6%).

DISCUSSION

The comparison of MRI findings with histopathological findings during diagnosis of Grade I Astrocytoma was recorded. Both histopathological and MRI positive astrocytoma case is found in 3 cases were true positive, 42 cases were true negative, 2 cases were false negative and 1 case was false positive. The validity of MRI during diagnosis of Grade I astrocytoma was recorded. The sensitivity, specificity, positive predictive value, negative predictive value and accuracy of MRI for the diagnosis of astrocytoma grade I are 60.0% (95% CI 46.1-73.9%), 97.7% (95% CI 93.5-101.9%), 75.0% (95% CI 62.7-87.2%), 95.4% (95% CI 89.5-101.3%) and 93.7% (95% CI 86.8-100.6%) respectively. In case of Grade II astrocytoma, true positive, true negative, false negative and false positive cases were 12, 31, 2 and 3 respectively. The sensitivity, specificity, positive predictive value, negative predictive value and accuracy of MRI for the diagnosis of astrocytoma grade II are 85.7% (95% CI 56.1-97.2%), 91.2% (95% CI 75.2-97.2%), 80.0% (95% CI 51.4-94.7%), 93.9% (95% CI 78.4-98.9%) and 90.0% (95% CI 81.5-98.49%) respectively. Similar findings are reported by Chishty et al⁷, who have recorded 100.0% sensitivity of MRI in detection of low grade gliomas. The study also recorded similar sensitivity of MRI findings to detect Grade III astrocytoma in correspondence to the study done by Chishty et al⁷; which for this study was, 90.5% and for the study of Chishty et al⁷ it was 95%. Law et al⁸ observed that PPV and NPV of MRI for determination of a high grade glioma were 86.1% and 44.1% respectively which was quite

consistent with the present study that is, where we found PPV and NPV of MRI for determination of grade IV astrocytomas the 70% and 95.4% accordingly.

The highest sensitivity of MRI for the diagnosis of different grade of astrocytoma was found in grade III astrocytoma (90.5%) followed by Grade II (85.7%) grade IV (75.0%) and grade I (60.0%). It is interesting that during comparison this result shows that sensitivity of the MRI for the detection of astrocytoma is decreased after increasing of grade of the tumour. Similar to the present findings Ellika et al⁹ has reported that sensitivity is 85.7% for different grading of astrocytomas with conventional MRI which is very close to the results of present study. Law et al⁸ found sensitivity of glioma grading ranging from 55.1% to 83.3%.

During comparing the specificity of MRI for the diagnosis of different grade of astrocytoma we recorded that, the highest specificity was found in grade I astrocytoma (97.7%) followed by Grade III (96.3%), grade IV (92.5%) and grade II (91.5%). These findings clearly show that the specificity of MRI for the detection of astrocytoma is very high and all are more than 90.0% which is very conclusive in diagnosis of astrocytoma. Law et al⁸ observed specificity of MRI in the diagnosis of high grade gliomas was 65% and in Riemann et al¹⁰ series it was 80.0% in diagnosing low grade gliomas which is in line with the present study. During diagnosis the specificity is very important for the detection of the disease state¹¹⁻¹².

During comparing the accuracy of MRI for the diagnosis of different grade of astrocytoma, the highest accuracy was recorded in both grade I astrocytoma (93.7%) and grade III (93.7%) followed by grade II (92.5%) and grade IV (89.6%). Accuracy of MRI for the detection of astrocytoma is very much effective and in all grade of tumor it was almost 90.0%. This indicates that MRI is a very effective diagnostic tool for the detection of different grades of astrocytoma. Similar to this result Chishty et al⁷ found 94.0% accuracy of preoperative MRI grading of intracranial astrocytomas. Again Riemann et al¹⁰ found 88.0% accuracy of contrast enhanced MRI for detecting intracranial astrocytomas which is almost similar to the present study. From the results of present study it is conclusive that MRI scan is a highly accurate and valid modality in the evaluation of different grades of brain tumours.

CONCLUSIONS

From the findings of this study it can be concluded that MRI is an effective tool for the diagnosis of different

grades of brain tumours, for this study which is astrocytomas. Sensitivity, specificity and accuracy of MRI for the diagnosis of astrocytoma are high in different grades of astrocytoma thus its validity is highly dependable.

REFERENCES

1. Leeds NE, Kieffer SA. Evolution of Diagnostic Neuroradiology from 1904 to 1999. *Radiology* 2000; 217(2):309-18.
2. Haaga JR, Kieffer SA, Chang JK, Lanzieri CF, Gilkeson RC. *Intracranial Neoplasms' Computed Tomography and Magnetic Resonance Imaging of the whole body*, Mosby, USA; 4th ed. 2003;1:59
3. Geets X, Daisne JF, Arcangeli S, Coche E, De Poel M, Duprez T, Nardella G, Grégoire V. Inter-observer variability in the delineation of pharyngo-laryngeal tumor, parotid glands and cervical spinal cord: comparison between CT-scan and MRI. *Radiotherapy and oncology*. 2005;77(1):25-31
4. Boss A, Bisdas S, Kolb A, Hofmann M, Ernemann U, Claussen CD, Pfannenberger C, Pichler BJ, Reimold M, Stegger L. Hybrid PET/MRI of intracranial masses: initial experiences and comparison to PET/CT. *Journal of Nuclear Medicine*. 2010; 51(8):1198-205
5. Allen CMC, Lueck CJ, Dennins M. *Davidson's Principles and Practice of Medicine*. 20th ed. Churchill Living Stone, India, 2006;236
6. Komotar RJ, Mocco J, Jones JE, Zacharia BE, Tihan T, Feldstein NA, Anderson RC. Pilomyxoid astrocytoma: diagnosis, prognosis, and management. *Neurosurgical focus*. 2005;18(6):1-4
7. Chishty IA, Rafique MZ, Hussain M, Akhtar W, Ahmed MN, Sajjad Z, Ali SZ. MRI characterization and histopathological correlation of primary intra-axial brain glioma. *JLUMHS*. 2010;9(02):64
8. Law M, Yang S, Wang H, Babb JS, Johnson G, Cha S, Knopp EA, Zagzag D. Glioma grading: sensitivity, specificity, and predictive values of perfusion MR imaging and proton MR spectroscopic imaging compared with conventional MR imaging. *American Journal of Neuroradiology*. 2003;24(10):1989-98
9. Ellika SK, Jain R, Patel SC, Scarpace L, Schultz LR, Rock JP, Mikkelsen T. Role of perfusion CT in glioma grading and comparison with conventional

- MR imaging features. *American Journal of Neuroradiology*. 2007;28(10):1981-7
10. Riemann B, Papke K, Hoess N, Kuwert T, Weckesser M, Matheja P, Wassmann H, Heindel W, Schober O. Noninvasive Grading of Untreated Gliomas: A Comparative Study of MR Imaging and 3-(Iodine 123)-L- α -methyltyrosine SPECT 1. *Radiology*. 2002;225(2):567-74
 11. Roth J, Roach ES, Bartels U, Józwiak S, Koenig MK, Weiner HL, Franz DN, Wang HZ. Subependymal giant cell astrocytoma: diagnosis, screening, and treatment. Recommendations from the International Tuberous Sclerosis Complex Consensus Conference 2012. *Pediatric Neurol* 2013;49(6):439-44
 12. Harrer JU, Parker GJ, Haroon HA, Buckley DL, Embelton K, Roberts C, Balériaux D, Jackson A. Comparative study of methods for determining vascular permeability and blood volume in human gliomas. *Journal of Magnetic Resonance Imaging*. 2004;20(5):748-57

Original Article

Association of Diabetic Retinopathy with Diabetic Foot in a Tertiary Care Centre

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Abstract

Diabetic retinopathy (DR) is one of the most common causes of blindness in developed countries. Early detection of diabetic retinopathy (DR) is crucial for preventing irreversible blindness. To measure the association of diabetic retinopathy with diabetic foot a cross-sectional study was carried out at Department of Ophthalmology and Surgery, BIRDEM General Hospital, Dhaka from January 2017 to September 2017. Patients were included, who were known diabetic. New cases of type-2 diabetes were also included because they might have complications at the time of diagnosis because of the nature of disease. Mean duration of diabetes was significantly higher in DFU with DR group. BUN and diabetes retinopathy were statistically significant ($p < 0.05$) between two groups. Mean HbA1c, pre-prandial glucose, C-peptide, cholesterol, triglyceride, HDL, LDL, hematocrit, creatinine, ABI, TBI and DM foot ulcer were not statistically significant ($p > 0.05$) between two groups. Most patients (33.3%) had a grade I ulcer in DF with DR group and 33.3% in DF without DR group. Medication taking was significantly high patients who were DF without DR. Combined agents was 11(36.7%) and 11(36.7%) in DF with DR and DF without DR group respectively. Insulin was taken 16(53.3%) in DF with DR group and 19(63.3%) in DF without DR group. Maximum patients had a grade I ulcer in diabetes foot ulcer. Medication taking was significantly high patients who were diabetes foot ulcer than without diabetes foot ulcer. Mean insulin, BUN and diabetes retinopathy was found significantly higher in diabetes with diabetes foot ulcer. Mean insulin BUN and diabetic retinopathy was significantly higher in diabetes with diabetes foot ulcer.

Key word : Diabetes foot ulcer, diabetes retinopathy, type 2 diabetes.

INTRODUCTION

Diabetes mellitus (DM) is the most common endocrine disorder in the world and is known to affect 8.3% of the population.¹ Diabetic retinopathy (DR) is one of the most common causes of blindness in developed countries.² Early detection of diabetic retinopathy (DR) is crucial for preventing irreversible blindness. The prevalence of retinopathy in diabetic inpatients was significantly higher than in an outpatient population and one quarter of inpatients with diabetes were noted to have previously undiagnosed retinopathy.³ Evidence indicates that with timely diagnosis and appropriate care, 50–70% of vision loss from diabetes can be prevented.⁴ Diabetes is one of the foremost causes of death in many countries and a leading cause of blindness, renal failure, and non traumatic amputation. Diabetes is also associated with numerous complications such as retinopathy, nephropathy, and neuropathy.⁵

MATERIAL AND METHODS

A cross-sectional study was carried out Department of ophthalmology and Surgery, BIRDEM General Hospital, Dhaka from January 2017 to September 2017. Patients were included, who were known diabetic. New cases of type-2 diabetes were also included because they might have complications at the time of diagnosis because of the nature of disease. For type-1 diabetics, only those patients were selected who have duration of diabetes of more than five years, because in type-1 diabetics complications usually starts after five years of duration, in accordance to the criteria of American Diabetes Association. Digital fundus photographs of the posterior pole were taken of each eye after pharmacological dilation. Presence, absence and severity of diabetic retinopathy and macular edema were graded on the basis of internationally accepted criteria. An investigator administered questionnaire and review of the medical record were used to obtain data about patient demographics, clinical characteristics and barriers to ophthalmic care. The association between these data and the presence of diabetic retinopathy was tested.

RESULTS

Out of 60 patients, the mean age was found 56.6 ± 9.3 years in DF (Diabetic foot) with DR (Diabetic retinopathy) group and 54.8 ± 10.1 years in DF without DR group. Male was predominant in both groups (73.3% vs 67.4% respectively).

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Mean duration of diabetes was significantly higher in DF with DR group ($p < 0.05$). In both groups majority patients had history of HTN (76.6% vs 73.3%). Mean systolic and diastolic blood pressure and BMI were not statistically significant ($p > 0.05$) between two groups. History of smoking was found 10(33.3%) in DF with DR group and 12(40%) in DF without DR group. Which was not statistically significant ($p > 0.05$) between two groups (Table 1).

Mean insulin, BUN and diabetes retinopathy were statistically significant ($p < 0.05$) between two groups. Mean HbA1c, pre-prandial glucose, C-peptide, cholesterol, triglyceride, HDL, LDL, hematocrit, creatinine, ABI, TBI and DM foot ulcer were not statistically significant ($p > 0.05$) between two groups (Table 2). Most patients (33.3%) had a grade I ulcer in DF with DR group and 33.3% in DF without DR group (Figure 1).

Most of the patients (86.7%) taking medication in DF with DR group and 30(100%) in DF without DR group. Medication taking was significantly high patients who were DF without DR. Combined agents was 11(36.7%) and 11(36.7%) in DF with DR and DF without DR group respectively. Insulin was taken 16(53.3%) in DF with DR group and 19(63.3%) in DF without DR group (Table 3).

Table I: Socio-demographic characteristics of the patients (n=90)

| Characteristics | DF with DR (n=30) | DF without DR (n=30) | P value |
|--------------------------|-------------------|----------------------|---------|
| Age (years) | 56.6±9.3 | 54.8±10.1 | 0.381 |
| Sex (Male) | 34 (72.3%) | 29(67.4%) | 0.612 |
| Diabetes duration (year) | 18.7±10.3 | 13.4±8.7 | 0.010 |
| History of HTN | 23 (76.6%) | 22(73.3%) | 0.799 |
| Blood pressure (mmHg) | | | |
| Systolic | 131.6±18.6 | 127.6±19.0 | 0.315 |
| Diastolic | 70.2±10.4 | 69.8±11.1 | 0.860 |
| BMI (kg/m ²) | 22.9±3.7 | 23.1±3.5 | 0.793 |
| History of smoking | 10(33.3%) | 12(40%) | 0.328 |

DF-Diabetic foot, DR-Diabetic retinopathy

P value was done from Student's t-test as shown mean±SD and Chi square test as shown categorical variables

Table II: Biochemical characteristics of the patients (n=60)

| Characteristics | DF with DR (n=30) | DFW without DR (n=30) | P value |
|-----------------------------|-------------------|-----------------------|---------|
| HbA1c (%) | 8.1±1.7 | 8.0±1.6 | 0.775 |
| Preprandial glucose (mg/dL) | 149.7±87.3 | 145.4±94.5 | 0.823 |
| C-peptide | 2.3±1.1 | 1.9±1.4 | 0.133 |
| Insulin | 19.7±12.0 | 33.2±23.7 | 0.001 |
| Cholesterol (mg/dL) | 150.5±42.3 | 152.3±36.7 | 0.830 |
| Triglyceride (mg/dL) | 136.7±98.7 | 137.3±66.3 | 0.973 |
| HDL (mg/dL) | 38.9±11.4 | 43.1±11.2 | 0.081 |
| LDL (mg/dL) | 81.6±32.9 | 82.2±33.1 | 0.931 |
| Hematocrit (%) | 33.7±5.8 | 35.9±5.2 | 0.062 |
| BUN (mg/dL) | 31.3±16.3 | 22.7±12.1 | 0.005 |
| Creatinine (mg/dL) | | | |
| ABI | 0.95±0.35 | 0.96±0.30 | 0.881 |
| TBI | 0.57±0.28 | 0.61±0.31 | 0.521 |
| DM foot ulcer | | | |
| Grade I | 10(33.3%) | 10(33.3%) | 0.515 |
| Grade II | 2(6.7%) | 5(16.7%) | |
| Grade III | 8(26.7%) | 7(23.33%) | |
| Grade IV | 9(30.0%) | 8(26.7%) | |
| Grade V | 1(3.3%) | 0 | |
| Diabetic retinopathy (DR) | | | |
| No DR | 0 | 7(23.3%) | |
| Mild NPDR | 0 | 5(16.7%) | |
| Moderate NPDR | 0 | 11(36.7%) | 0.001 |
| Severe NPDR | 0 | 7(23.3%) | |
| PDR | 30(100%) | 0 | |

DF-Diabetic foot, DR-Diabetic retinopathy

P value was done from Student's t-test as shown mean±SD and Chi square test as shown categorical variables

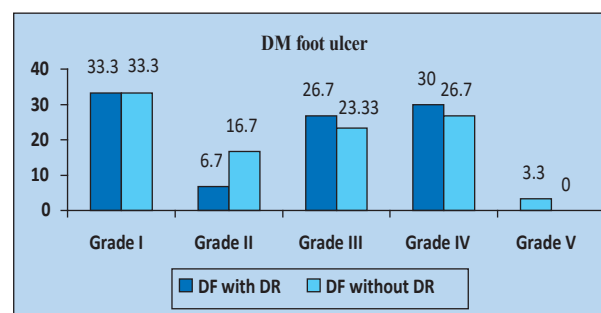


Figure 1: Bar diagram showing diabetes mellitus foot ulcer.

Table III: Methods of glycemic control (n=60)

| Methods of glycemic control | DF with DR (n=30) | DF without DR (n=30) | P value |
|-----------------------------|-------------------|----------------------|---------|
| No medication | 4(13.3) | | 0.015 |
| Medication | 26(86.7) | 30(100) | |
| Combined agents | 11(36.7) | 11(36.7) | 0.918 |
| Insulin | 16(53.3) | 19(63.3) | 0.357 |

P value was done from Chi square test as shown categorical variables

DISCUSSION

The demographics for the population studied are likely to differ from those of the general outpatient diabetic population in several ways. It is likely to be an older population with a greater duration of diabetes, poor diabetic control, medical comorbidities and lower socioeconomic status. This was a major motivation for us to undertake this work.

In our study it was observed that the mean age was found 56.6±9.3 years in DF (Diabetic foot) with DR (Diabetic retinopathy) group and 54.8±10.1 years in DF without DR group. In study of Hwang et al.⁶ observed that the mean age was found 66.7±8.8 years in DFU with DR group and 66.8±12.6 years in DFU without DR group. The difference was not statistically significant ($p>0.05$) between two groups. Girisha and Viswanathan¹ study showed the mean age of the population in our study was 58.28 years ± 11.36. AlGoblan et al.⁷ study also reported that the average age of the patients included in the study was 56 years (standard deviation [SD] ±9.7).

In current study observed that Male was predominant in both groups (73.3% vs 67.4% respectively). Mean duration of diabetes was significantly higher in DF with DR group ($p<0.05$). In this Hwang et al.⁶ male was found 75.0% in DF with DR group and 73.0% in DF without DR group. Mrozikiewicz-Rakowska et al.² study observed that male was found 77.0% in DR group and 54.0% in control group. Girisha and Viswanathan¹ study showed among the 145 cases studied, 95 (65.51%) were males and 50 (34.48%) were females with a male to female ratio of 1.9:1. Al Goblan et al.⁷ study revealed male found 59.3% and female 40.7%.

In this study mean duration of diabetes was found 18.7±10.3 years in DFU with DR group and 13.4±8.7 years in DF without DR group. Mean duration of diabetes

was significantly higher in DFU with DR group ($p<0.05$). Similar observation was found different studies Hwang et al. showed that the mean duration of diabetes was found 20.6±10.4 years in DFU with DR group and 15.8±10.3 years in DF without DR group. The difference was statistically significant between two groups ($p=0.022$). Mrozikiewicz-Rakowska et al.² study showed the mean duration of diabetes was found 16.97±9.2 years in DR group and 17.1±9.48 years in control group. AlGoblan et al.⁷ study on the length of the disease, 45% of the patients had diabetes for ,10 years, 38.6% for 10–20 years, and 16.4% for .20 years

In our study in both groups majority patients had history of HTN (76.6% vs 73.3%). Mean systolic and diastolic blood pressure and BMI were not statistically significant ($p>0.05$) between two groups. Mrozikiewicz-Rakowska et al.² study observed that the mean BMI was found 32.24±5.0 kg/m² in DR group and 30.24±5.42 kg/m² in control group. Hwang et al. study showed that history of HTN was found in 75.0% in DFU with DR group and 69.0% in DFU without DR group. Mean blood pressure and BMI were not statistically significant ($P>0.05$). History of smoking was found 10(33.3%) in DF with DR group and 12(40%) in DF without DR group. Which was not statistically significant ($p>0.05$) between two groups. Girisha and Viswanathan¹ study showed hypertension was the most common among the associated comorbid illness. AlGoblan et al.⁷ the mean body weight of the patients was 81 kg (STD ±13). BMI measurements showed that 12.9% patients had normal BMI, whereas 30.7% were overweight and 56.4% were categorized as obese with BMI .30. None of the patients included in the study had underweight BMI.

In present study the mean insulin, BUN and diabetes retinopathy were statistically significant ($p<0.05$) between two groups. Mean HbA1c, pre-prandial glucose, C-peptide, cholesterol, triglyceride, HDL, LDL, hematocrit, creatinine, ABI, TBI and DM foot ulcer were not statistically significant ($p>0.05$) between two groups. AlGoblan et al.⁷ study comparing patients with controlled highly elevated HbA1c, only 23% of patients had healed foot ulcers within 3 months, 28% between 3 and 6 months, and 48% > 7 months ($P=0.024$). A similar association was observed between the process of healing and HbA1c levels in our patients. While studying the healing process in relation to HbA1c levels, we observed that 68% of patients with normal HbA1c had completely healed foot ulcers, whereas 20% showed partial healing

and 4% had uncured foot ulcers. The remaining 8% of patients required graft placement. Among the patients with high HbA1c, 45.9% showed complete healing, whereas 33.8% had partial healing, 5.4% progressed to grafting process, and 14.9% had unhealed foot ulcers. Hwang et al.⁶ study observed in terms of DR, 90 patients (90%) had DR and 55 patients (55%) had proliferative diabetic retinopathy (PDR). Eight patients (8%) had mild non-proliferative diabetic retinopathy (NPDR) and 17 patients had moderate NPDR (17%). Severe NPDR was observed in 10 patients (10%).

In this study most patients (33.3%) had a grade I ulcer in DF with DR group and 33.3% in DF without DR group. Hwang et al. study observed that among 100 patients with DFUs, only one patient (1%) had a grade 5 ulcer. Girisha and Viswanathan¹ the number of patients with mild nonproliferative diabetic retinopathy were 95 (65.5%) and those with moderate nonproliferative diabetic retinopathy were 45 (31.03%). Severe nonproliferative diabetic retinopathy was seen in 5 patients who accounted for 3.44% of the total.

In this study Most of the patients (86.7%) taking medication in DF with DR group and 30(100%) in DF without DR group. Medication taking was significantly high patients who were DF without DR. Combined agents was 11(36.7%) and 11(36.7%) in DF with DR and DF without DR group respectively. Insulin was taken 16(53.3%) in DF with DR group and 19(63.3%) in DF without DR group. Hwang et al. was observed that no medication was found 10.0% in DFU with PDR group and medication used 90.0% in DFU with PDR group and 100% in DFU without PDR group. Combined oral agents was 35.0% in DFU with PDR group and 36.0% in DFU without PDR group. Insulin was 55.0% and 64.0% in DFU with PDR and DFU without PDR group respectively. The difference was not statistically significant ($p>0.05$) between two groups. Sharma et al.⁸ correlate the diabetic foot disease to the treatment history for the diabetes, the dreaded problem of diabetic foot was highest in patients who have not had any treatment (33.64%), followed by insulin users (21.99%), and then followed by oral hypoglycemic agents (14.43%). Surprisingly, another study has shown insulin to be a known risk factor diabetic foot disease.⁹ Perhaps untreated patients were left out in this study.

CONCLUSIONS

Maximum patients had a grade I ulcer in diabetes foot ulcer. Medication taking was significantly high patients

who were diabetes foot ulcer than without diabetes foot ulcer. Mean insulin, BUN and diabetic retinopathy was found statistically significantly higher in diabetes with diabetes foot ulcer.

REFERENCES

1. Girisha BS, Viswanathan N. In search of cutaneous marker for retinopathy in diabetic patients: A pilot study. *Clin Dermatol Rev* 2017; 1: 52-5.
2. Mrozikiewicz Rakowska B, Łukawska M, Nehring P, Szymański K, Agnieszka Sobczyk Kopcioł A, Krzyżewska M, et al. Genetic predictors associated with diabetic retinopathy in patients with diabetic foot. *Pol Arch Intern Med*. 2018; 128 (1): 35-42
3. Kovarik JJ, Eller AW, Willard LA, et al. Prevalence of undiagnosed diabetic retinopathy among inpatients with diabetes: the diabetic retinopathy inpatient study (DRIPS). *BMJ Open Diabetes Research and Care* 2016;4: e000164.
4. Klein R, Klein B. Vision disorders in diabetes. In: National Diabetes Data Group, ed. *Diabetes in America*. 2nd edn. Bethesda, MD: National Institutes of Health, National Institute of Diabetes and Digestive and Kidney Diseases, 1995:293-337.
5. Aziz KMA. Association between high risk foot, retinopathy And hba1c in saudi diabetic population. *Pak J Physiol* 2010;6(2):22-28
6. Hwang DJ, Lee KM, Park MS, Choi Sh, Park JI, Cho JH, et al. (2017) Association between diabetic foot ulcer and diabetic retinopathy. *PLoS ONE* 12(4): e0175270
7. AlGoblan AS, Alrasheedi IM, Basheir OH, Haider KH. Prediction of diabetic foot ulcer healing in type 2 diabetic subjects using routine clinical and laboratory parameters. *Research and Reports in Endocrine Disorders* 2016;6 11-16
8. Sharma R, Kapila R, Sharma AK, Mann J. Diabetic Foot Diseases-Incidence and risk factors: A Clinical Study. *J Foot Ankle Surg (Asia-Pacific)* 2016; 3(1): 41-46.
9. Boyko EJ, Ahroni JH, Stensel V, Forsberg RC, Davignon DR, Smith DG. A prospective study of risk factors for diabetic foot ulcer: The Scattle Diabetic Foot Study. *Diabetes Care* 1999; 22(7): 1036-4.

Original Article

In New Geon Extra Peritoneal Caesarean Section an Observational Study

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Abstract

This study was designed to find out the maternal and perinatal outcome among 80 women undergoing extraperitoneal cesarean section. This observational study was conducted at Institute of Child and Mother Health, Matuail, Dhaka from 1st March 2013 to 28th February 2014. Data were collected by structured questionnaire with the written consent of patients irrespective of routine and emergency cases where the procedure was successful in all patients. During operation, time was taken from open to close, incision to delivery of baby, total amount of blood loss during procedure, intraoperative complications, APGAR score of newborn were documented. Time required for return of bowel sound mobilization of patient within 24 hour and, duration of hospital stay were also included. Success rate of extra peritoneal CS was 100%, Time taken from incision to delivery was ≤6 minutes in 62.2% cases, time taken from incision to closure was between 31-45 min in 72.5% cases, blood loss ≤500 ml in 62% cases, return of bowel function between 5-8 hours in 52.5%, mobilization within 24 hours in 52.5%, neonatal one minute APGAR score ≥7 in 93.91%. As a surgical form of infection prophylaxis extraperitoneal cesarean section can be applied to avoid serious post-operative pelvic infections and its complications Extraperitoneal cesarean section would be a rational practice of cesarean section in the modern era.

Key words: Extraperitoneal, Caesarean section, Transperitoneal.

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INTRODUCTION

Due to caesarean section maternal mortality has been reduced drastically. Maternal morbidity like post operative pain, infection and post operative adhesions still present. Soiling of the peritoneal cavity may occur by infected amniotic fluid from the uterus, blood and handling of bowel which mainly result maternal mortality.

When the peritoneal cavity is opened by transperitoneal approach, organisms enters into the peritoneal cavity not only from skin and fascia but also from amniotic fluid especially when infected. Moreover, peritoneal cavity and intestines are subjected to dryness, and, chemical and physical injury by packs and tetra. As a results it is associated with paralytic ileus, wound sepsis, peritonitis and septicemia. All this occur especially in cases of prelabour rupture of membrane, prolonged labour. As a result there is prolonged hospital stay.^{1,2}

To reduce the morbidity and higher mortality of classical trans-peritoneal caesarean section in the pre-antibiotic era, the extraperitoneal approach was devised. It was first performed by Ferdinand Ritgen (1787-1867) of Gissen in 1821.³ The procedure was further modified by Latzko, Waters and Norton. This extraperitoneal approach reduced the mortality and morbidity to a significant extent.⁴

MATERIALS AND METHODS

This observational study was conducted in Institute of Child and Mother Health, Matuail, Dhaka. Data was collected by structured data sheet from 1st April 2013 to 28th March 2014.

Inclusion criteria: Elective and emergency extraperitoneal all cases of caesarean section.

Exclusion criteria :

- i. Placenta praevia
- ii. Previous abdominal surgeries
- iii. Prior surgeries on bladder
- iv. Women requiring caesarean section with bilateral tubal ligation

Operative technique : Spinal anesthesia or general anesthesia were given to the patients as indicated.

Catheterization of the bladder was done before operation. Skin incision was given either transverse supra-pubic or longitudinal. Transversally rectus sheath was incised and rectus muscles were separated. Transversals fascia was separated after separation of the recti. Urinary bladder covering the lower uterine segment was exposed.

By bilateral paravesical dissection, the peritoneal attachment to the dome of the bladder was isolated and by blunt dissection peritoneal fold was separated and was pushed upward to expose the lower uterine segment. Transverse incision was given to uterus and baby was deliver. After delivery of placenta and membrane, uterus was closed with number one chromic catgut in single layer.

Proper haemostasis was done. After ensuring no bladder injury and confirming the instrument and mop count, rectus muscles were approximated and rectus sheath was closed with 1/0 vicryl and skin closure was done subcutaneously by 2/0 vicryl.

Pre-operative antibiotic (Inj. 1 gm ceftriaxone) given to all patients before starting the procedure. In the post-operative word, patient's pulse, blood pressure, temperature, per vaginal bleeding were recorded daily. By mouth was given after appearance of bowel sounds. Four hour after operation patients were encouraged for early ambulation. Routine injectable antibiotics and analgesics were given for 24 hours. Catheter removal was done routinely after 24 hours, except in cases of obstructed labor.

Post-operative complications as pyrexia, pain, abdominal distention, wound infection, etc were taken into consideration and details reported. Post-operative pain was assessed using the visual analogue scale of 0 to 10 where '0' represents 'no pain' and '10' represents 'worst pain ever experienced'. Patients were discharged on third post-operative day if no other complication.

RESULTS

In the present study of 80 cases of extraperitoneal cesarean section. Analysis was done meticulously considering a wide range of parameters.

The maximum number of women belonged to the age group 18-26 years (73%). Most of the women in our study were primigravida (92%), 6% were second gravida and 2% third gravida. 89% of the women were term at delivery, 9% were preterm and 2% were post term. 95% of cases had a cephalic presentation, followed by breech (4%) and

transverse lie (1%). Majority of patients had ruptured membranes at admission (76%).

The most common indication for cesarean section was fetal distress (37%), followed by CPD (24%), oligohydramnios (14%), fetal growth restriction (11%), breech presentation (4%), transverse lie (1%), post term (3%) and DTA (3%). 3% of cases constituted other causes.

Of the 80 cases in which extraperitoneal cesarean section was tried, procedure was successful in 80 cases (100%).

Table 1: Success rate of extraperitoneal CS.

| | No. of cases | Percentage (%) |
|-------------|--------------|----------------|
| Cases tried | 80 | 100 |
| Success | 80 | 100 |

The time taken from skin incision to baby delivery was ≤6 minutes in 62.2% of the cases. In 4% of the cases, the incision-delivery interval was >10 minutes (Table 2). The duration of the surgery from skin incision to closure was between 31-45 minutes in 72.5% of the women. The procedure took >60 minutes in 3.7% of cases (Table 3).

The total blood loss during the procedure was ≤500 ml in 62% of the cases. Blood loss >1000 ml was present in 4% of the cases.

Table 2: Time taken - incision to delivery.

| Incision-delivery interval (minutes) | No. of cases | Percentage (%) |
|--------------------------------------|--------------|----------------|
| ≤6 | 50 | 62.2% |
| 6-10 | 26 | 32.2% |
| >10 | 4 | 5.6% |

Table 3: Time taken - incision to closure.

| Incision-delivery interval (minutes) | No. of cases | Percentage (%) |
|--------------------------------------|--------------|----------------|
| ≤30 | 12 | 15.4% |
| 31-45 | 58 | 72.5% |
| 46-60 | 7 | 8.4% |
| >60 | 3 | 3.7% |

Bladder injuries was not occur in this study. Maximum number of patients (80%) had pain scores between 0- 5. In 16.25% of the patients, pain scores were 6-7, and, in

3.75% of patients score was >8 (Table 4). 80% of the women did not require any additional analgesics.

Table 4: Post-operative pain.

| Pain scores | No. of cases | Percentage (%) |
|-------------|--------------|----------------|
| 0-5 | 64 | 80% |
| 6-7 | 13 | 16.25% |
| >8 | 3 | 3.75% |

Return of bowel sounds was present between 5-8 hours in 52.5% of the women. In 40% of the women, bowel sounds appeared at 4 hours (Table 5). Mobilization of the patient occurred within 24 hours in 52.5% of the patients, and, between 24-48 hours in 40% of the patients (Table 6).

Table 5: Appearance of bowel sounds.

| Appearance of bowel sounds (hours) | No. of cases | Percentage (%) |
|------------------------------------|--------------|----------------|
| 4 | 32 | 40% |
| 5-8 | 42 | 52.5% |
| 9-12 | 6 | 7.5% |
| >12 | - | - |

Table-6: Mobilization of patient.

| Mobilization of patient (hours) | No. of cases | Percentage (%) |
|---------------------------------|--------------|----------------|
| ≤24 | 42 | 52.5% |
| 24-48 | 32 | 40% |
| 48-72 | 6 | 7.5% |
| >72 | - | - |

Post operatively, pyrexia was present in 2% cases, urinary tract infection in 2% cases. No cases of wound infection, wound gaping, burst abdomen, sub-acute intestinal obstruction, sub-involution, peritonitis and genitor-urinary fistula were present in this study. Duration of hospital stay was ≤4 days in 88% of cases, 2% of cases had prolonged hospital stay >8 days. 67% of the babies born by extraperitoneal cesarean section in our study had birth weight between 2.5-3.5 kg. 23% cases had birth

weight ≤2.5 kg and in 10% cases, babies weighed >3.5 kg. The neonatal APGAR score was ≥7 in 93.91% cases. No cases of birth injuries was found in this study.

DISCUSSION

Caesarean sections which was done due to intra-uterine sepsis and after prolonged labour may cause various complications like wound sepsis, paralytic ileus, peritonitis, septicemia shock and even cause death of patients. Among all complications some of serious complications can be preventable by asepsis and appropriate antibiotics.

With this consideration the resurrecting extraperitoneal caesarean section from obstetric history, in the hope of reducing risks of sepsis associated with caesarean section.

Decrease risk of infectious complications in comparison to the conventional transperitoneal caesarean section, the women undergoing extraperitoneal cesarean section can be advocated. Fluids and feeding earlier in the post-operative period, and also ambulate early thereby reducing hospital stay. Extra peritoneal cesarean section can also ensure early feeding in the post operative period and also early ambulation. So their by reducing hospital stay duration.

If proper precautions are taken, complications like injury to the bladder and blood vessels can be avoided. Bangladesh like developing countries where obstetric sepsis is still a major cause of morbidity and mortality, any procedure that will prevent peritoneal contamination deserves consideration. Decreased infection rate, early mobilization and return of bowel function may weigh over the increased time taken for the procedure and technical difficulty.

REFERENCES

1. E. W. Cartwright. Extraperitoneal caesarean section: Physick-Sellheim principle. West J Surg. 1949; 57:509.
2. Derk Crichton. A simple technique of extra-peritoneal caesarean section. S Afr Med J. 1973; 47:2011.
3. Marshall CM. Extraperitoneal approach. In: Marshall CM, eds. Caesarean Section Lower Segment Operation. 1st ed. Bristol: John Wright; 1939.
4. Nanda SS et al. Int J Reprod Contracept Obstet Gynecol. 2014 Sep;3(3):724-727

Case Report

Neurological Complication of Chikungunya Fever: Guillain Barre Syndrome- A Case Report

*Hasan MN¹, Md. Rahman MA², Rahim MA³, Ahmed QMU⁴, Islam MS⁵**Abstract:**

Chikungunya fever has been known as reemerging disease since 2005. Its feature is more or less like dengue fever. Major outbreak occurred in Bangladesh in 2017. Lot of complications can occur in patient suffered from it. We report a Chikungunya case presenting with Guillain-Barré syndrome (GBS) with sensory involvement and bowel- bladder dysfunction who responded to plasma pheresis. Laboratory parameters and temporality support that GBS in the complication of Chikungunya.

Key words: Chikungunya, Guillain-Barré syndrome

INTRODUCTION

Chkungunya is an Alpha virus. It is transmitted to human by bite of Aedes mosquito¹. It was first isolated in 1952 in Tanzania and has been known as a reemerging disease since 2005². Major outbreak occurred in Reunion Island in 2004 which involved 38.2% of 785000 inhabitants³.

Chikungunya strain was isolated in India during the 2004-2006 outbreak⁴. In Bangladesh major outbreak occurred in 2017 in Dhaka city. Chikungunya fever is characterized by an abrupt onset of fever and moderate to severe arthralgia, myalgia, headache, rash, fatigue and nausea⁵. Unusual manifestation includes cardiovascular, ocular, gastrointestinal, renal, neurological features. Among the neurological features acute tranverse myelitis, encephalitis, stroke, psychosis Guillain Barre Syndrome are reported^{6,7}. We report a case of Chikungunya presenting with GBS with bowel bladder involvement who improved with plasma pheresis.

CASE REPORT

A 50 year old hypertensive man was admitted in the department on September 2017. He had history of high grade fever and multiple small and large joint pain. Four days later fever subsided but joint pain persisted. Twelve days later of fever he developed weakness of both lower limbs along with constipation and urinary retention. Urinary catheter was given to relieve retention. Within another two days he noticed weakness of both upper limbs. He had no shortness of breath, dysphagia or nasal regurgitation. On examination vital signs were stable. Neurological examination revealed muscle power of lower limbs 1/5 and 2/5 on upper limbs bilaterally. All the jerks of lower limbs were absent whereas in upper limbs were diminished. Plantar reflex absent bilaterally. Sensory functions including pain, touch, position and vibration were impaired without any definite sensory level. Fundoscopy was normal. Other system examinations were normal. On investigation (Table- 1) anti Chikungunya Ab IgM was positive, CSF study showed albuminocytological dissociation, NCS of limbs showed acute motor and sensory axonal neuropathy (AMSAN). MRI (Magnetic Resonance Imaging) of brain and dorsolumbar spine were normal. He was started with plasma pheresis. Muscle power improved from 1 to 3 in lower limbs and 2 to 4 in upper limbs, bowel and bladder function was static at the time of discharge. He was discharged with advice for regular physiotherapy at home.

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Table-1: Investigations

| CBC | Anti Chikungunya Ab(IgM+IgG) | S.electrolytes (mmol/L) | CSF study Physical- | NCS | MRI |
|---|------------------------------|---|---|---|--|
| Hb% 14.3g/dl ESR 05mm 1st Hour TC- WBC- 15×10 ⁹ /L RBC- 4.65×10 ¹² /L Platelet- 250×10 ⁹ /L DC- N84%, L 13%, M02%, E01% Hct- 0.43l/l | ICT for IgM positive | Na ⁺ 134 K ⁺ 4 Cl ⁻ 102 TCO2 15.9 | Colour watery Appearance clear Microscopic examination TC- no cell found Gram and ZN stain No organism found Glucose 3mmol/L Protein 287mg/dl ADA 1.6U/L | Sensory motor polyneuroradiculopathy: Acute motor sensory axonal neuropathy (AMSAN) | Dorsolumbar spine- Normal Brain- Normal |

ADA- Adenosine Deaminase

DISCUSSION

Chikungunya fever has a similar pattern of dengue fever. Some atypical cases were reported. Mortality in those cases were 10%.^{6,7} Neurological complications have been identified early in 1960 and still needs special attention.^{4,9} From the Reunion Island 12% Chikungunya cases had neurological manifestation in the form of GBS, seizure, cranial nerve deficit, altered consciousness and other neurological features.⁹ Our case presented with Chikungunya fever later on developed GBS within 12 days with atypical features like sensory and autonomic involvement in the form of bowel and bladder dysfunction. Limb weakness was ascending with bizarre sensory loss. There was no respiratory weakness. CSF (Cerebro-spinal Fluid) protein was very high whereas cell count was zero. Nerve Conduction Study (NCS) revealed Acute Motor and Sensory Axonal Neuropathy (AMSAN). On serological test for Chikungunya IgM was positive that confirms recent Chikungunya viral infection and establishes a temporal relation with the onset of GBS. For this reason we conclude that the Chikungunya virus was probably responsible for the GBS.

CONCLUSIONS

Finding Chikungunya case along with its complication such as GBS during major outbreak in Bangladesh is an epidemiological evidence that Chikungunya can cause GBS. Moreover laboratory parameters and temporality

support that GBS is the complication of Chikungunya fever in this case. This case report would create awareness among physicians that Chikungunya can cause neurological complications like GBS.

REFERENCES

1. National Guideline on Clinical Management of Chikungunya Fever. Dhaka: DGHS; 2017
2. Lahariya C, Pradhan SK. Emergence of chikungunya virus in Indian subcontinent after 32 years: a review. J Vector Borne Dis. 2006;43(4):151-60.
3. Lebrun G, Chadda K, Reboux A, Martinet O, Gaüzère B. Guillain-Barré Syndrome after Chikungunya Infection. Emerging Infectious Diseases. 2009;15(3):495-496.
4. Sá P, Nunes M, Leite I, Campelo M, Leão C, Souza J et al. Chikungunya virus infection with severe neurologic manifestations: report of four fatal cases. Revista da Sociedade Brasileira de Medicina Tropical. 2017;50(2):265-268.
5. Rajapakse S, Rodrigo C, Rajapakse A. Atypical manifestations of chikungunya infection. Trans R Soc Trop Med Hyg. 2010;104(2):89-96.
6. Lemant J, Boisson V, Winer A, Thibault L, Andre H, Tixier F, et al. Serious acute chikungunya virus infection requiring intensive care during the Reunion Island outbreak in 2005-2006. Crit Care Med. 2008; 36(9):2536-41.

7. Economopoulou A, Dominguez M, Helynck B, Sissoko D, Wichmann O, Quenel P, et al. Atypical Chikungunya virus infections: clinical manifestations, mortality and risk factors for severe disease during the 2005-2006 outbreak on Reunion. *Epidemiol Infect.* 2009;137(4):534-41.
8. Ludlow M, Kortekaas J, Herden C, Hoffmann B, Tappe D, Trebst C, et al. Neurotropic virus infections as the cause of immediate and delayed neuropathology. *Acta Neuropathol.* 2015;131(2):159-84.
9. Tandale BV, Sathe PS, Arankalle VA, Wadia RS, Kulkarni R, Shah SV, et al. Systemic involvements and fatalities during Chikungunya epidemic in India, 2006. *J Clin Virol.* 2009;46(2):145-9.

Case Report

Co-existence of Systemic Lupus Erythematosus and Ankylosing Spondylitis- A Case Report

*Nakarmi S¹, Ahmed S², Shahin MA³, Shazzad MN⁴, Mahmood⁵, Haq SA⁶

Abstract

Co-existence of systemic lupus erythematosus and ankylosing spondylitis is not common. In this report, we present a 24-year-old woman with both the entities diagnosed in the course of time. Initially she presented with malar rash, photosensitivity, oral ulcer and polyarthritis. Her ANA and anti-Smith antibody were positive, but anti-ds DNA was negative. With these features, she was diagnosed as a case of systemic lupus erythematosus. Few months later, she was diagnosed with ankylosing spondylitis on the basis of inflammatory back pain and bilateral sacroilitis.

Key words: Systemic lupus erythematosus, ankylosing spondylitis, sacroilitis

INTRODUCTION

Systemic lupus erythematosus (SLE) is a systemic autoimmune disease characterized by diverse multisystem involvement with symptoms ranging from mild skin involvement to life threatening organ disease.^{1,2} Ankylosing spondylitis (AS) is a chronic inflammatory disease which belongs to a group of heterogeneous conditions known as spondyloarthritis. Sacroilitis is the hallmark of AS, especially in earlier disease stages.³ Sacroilitis may be an infrequent manifestation of SLE itself^{4,18} or may rarely represent as coexistence of SLE with

AS⁵. However, peculiarities of sacroilitis in SLE are absence of typical back pain and HLA B27.⁴ So far, only 9 cases of coexistence of SLE and AS have been reported.⁵⁻¹³ In this report, we describe another case of such coexistence.

CASE SUMMARY

A 24-year-old woman was diagnosed with SLE on November 2016 on the basis of malar rash, oral ulcer, photosensitivity, polyarthritis, positive ANA and anti-Smith antibody. She was doing well with hydroxychloroquine and tapering doses of corticosteroid. Four months later, she experienced one episode of loss of consciousness, several occasions of vomiting along with 3 episodes of seizures, and at that time it was labeled as CNS involvement of SLE. She was then discharged with hydroxychloroquine, sodium valproate and prednisolone 7.5 mg daily. Two months later, she developed inflammatory back pain and severe pain in both hips (anterior). She did not have other features of SLE during this visit. Her SELENA SLEDAI score during admission was 2. Her hemoglobin was 7.1 gm/dl, reticulocyte count, bilirubin were normal, Coombs' test was negative, CRP-14.0 mg/dl, x ray SI joints showed sacroilitis grade II on right and grade III on left side with joint space reduction and periarticular sclerosis in both hip joints (image 1). MRI of both hip and sacroiliac joints revealed mild flattening of heads of femur bilaterally, T2WI fat suppression and STIR- mixed signal changes seen in femoral head of both sides with reduction of joint space of both hip joints (image 2). Joint effusion was noted in both hip joints. T1WI hypo, T2WI and STIR hyper intense signal changes were seen within subchondral marrow of both (more on left) sacroiliac joints with destruction of overlying articular cartilage resulting reduction of joint spaces (image 3). Ankylosing spondylitis was diagnosed on the basis of inflammatory back pain and bilateral sacroilitis. So, the final diagnosis of systemic lupus erythematosus, ankylosing spondylitis and bilateral avascular necrosis of femoral head was made. The cause of AVN was thought to be long term steroid therapy. Patient was then managed with NSAIDs and adjuvant analgesics and orthopedic consult was done for AVN.

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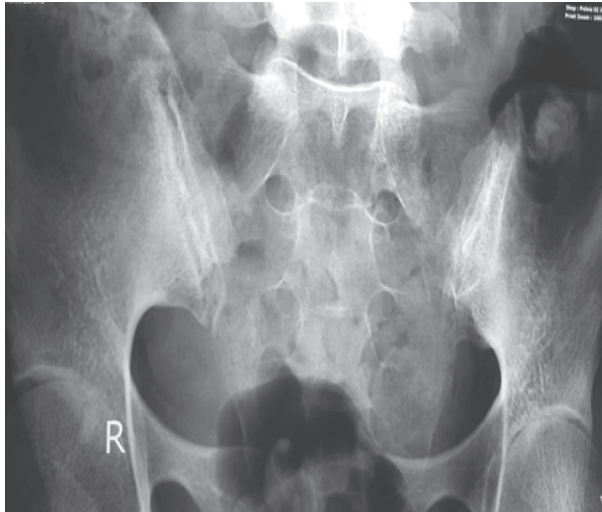


Figure 1: X ray pelvis showing bilateral sacroiliitis, joint space reduction in both hip joints

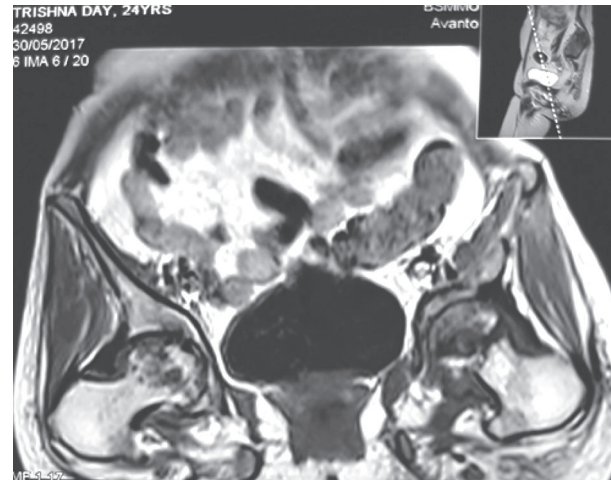


Figure 2: MRI of both hip and sacroiliac joints revealed mild flattening of heads of both femur, T2WI fat suppression and STIR- mixed signal changes seen in femoral head of both sides with reduction of joint space of both hip joints

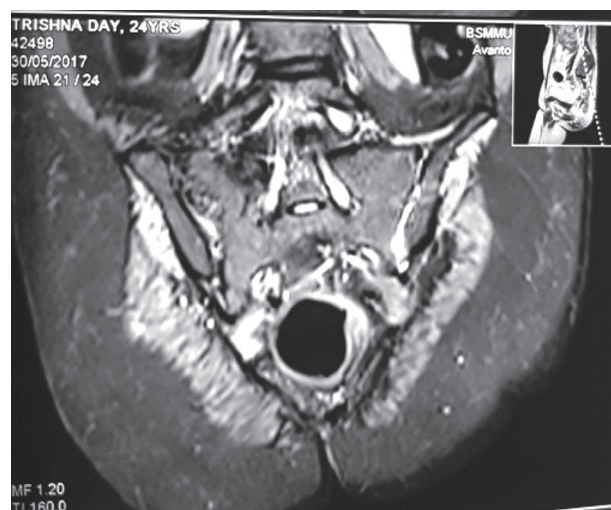
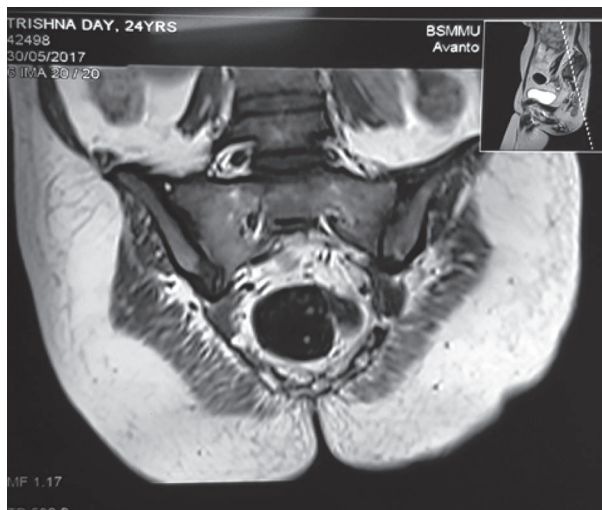


Figure 3: T1WI hypo (3a), T2WI and STIR (3b) hyper intense signal changes are seen within subchondral marrow of both (more on left) sacroiliac joints with destruction of overlying articular cartilage resulting reduction of joint spaces

DISCUSSION

SLE is predominant in women of reproductive age group with the F:M ratio of 8-15:1¹⁴, whereas AS has F:M ratio of 1:1.2.¹⁵ The coexistence of these two entities is very uncommon with only 9 case reports published in the literature. In our case, SLE preceded the features of AS by seven months. She fulfilled 1997 ACR criteria for SLE with presence of malar rash, photosensitivity, oral ulcer, polyarthritis, positive ANA, anti-Smith antibody and lupus anticoagulant. Six months later, she developed inflammatory back pain with bilateral sacroiliitis confirmed

by MRI. Thus additional diagnosis of AS was made as per modified New York criteria.¹⁶ Among the 9 cases mentioned above, 6 were female and 3 were male. ANA was positive in all the cases and anti-ds DNA in 8. In our case, ANA and anti-Smith antibodies were strongly positive, and anti-ds DNA was negative. The symptoms of SLE may or may not precede those of AS. In 3 cases including ours, symptoms of SLE preceded AS by few months to years (duration ranging from 4 months to 8 years).^{5,8} However, in 5 reported cases, symptoms of AS preceded those of SLE by several years, duration ranging from 4 years to 17 years.^{7,9,10,12,13}

Though sacroilitis may be a rare manifestation of SLE, its presence along with inflammatory back pain may be suggestive of an additional diagnosis. In a report by Nassonova et al, it was shown that 22 out of 43 male patients with SLE had radiological sacroilitis and 7 had ankylosis of sacroiliac joint. However, none had inflammatory back pain or positive HLA B27, and NSAIDs were not effective treatment for back pain.⁴ In our case, patient presented with inflammatory back pain which improved with NSAIDs treatment. Chandrashekhar et al discussed a case of 21-year-old female with SLE, dermatomyositis overlap with inflammatory low back pain and symptomatic bilateral sacroilitis. HLA B27 was negative. The severity and duration of back pain correlated with high disease activity of SLE.¹⁷ However, in our case, inflammatory back pain started when SLE was not active and patient's back pain responded to NSAIDs.

Similar to our case, Tarhan F et al has recently published one report of 55-year-old woman with SLE and subsequently developing inflammatory back pain, bilateral sacroilitis in MRI and positive HLA B27.⁵ MRI has been used as a diagnostic tool for sacroilitis by our team and Tarhan F et al. Similar case has been discussed by Kucuk A et al, where sacroilitis was diagnosed by CT scan.⁶ He reported a case of 33-year-old female with SLE and sacroilitis in CT, and gave history of inflammatory neck pain and back pain on query. Later she was found to be HLA B27 positive.

In previously reported cases, HLA B27 was positive in^{8,5,6,8-13} whereas it was negative in 1 case⁷ which is similar to ours. However the presence of other HLA genotypes was not searched in the previously mentioned case report⁷ and our case.

CONCLUSIONS

Coexistence of SLE and AS is a rare event. Though sacroilitis may be rarely present in SLE, if present with inflammatory back pain in the background of low disease activity of SLE, meticulous search for coexisting AS should also be done.

REFERENCES

1. Bertsias G, Cervera R, Boumpas DT. Systemic lupus erythematosus: pathogenesis and clinical features. In: Bijlsma J (ed). EULAR textbook on rheumatic diseases, London: BMJ Group, 2012, pp. 476–505.
2. COJOCARU M, COJOCARU IM, SILOSI I, VRABIE CD. Manifestations of Systemic Lupus Erythematosus. *Mædica*. 2011;6(4):330-336
3. Braun J, Sieper J. Ankylosing spondylitis. *Lancet* 2007;369(9570):1379-90
4. V. A. NASSONOVA, Z. S. ALEKBEROVA, M. Yu. FOLOMEYEV, N. M. MYLOV. Sacroiliitis in male systemic lupus erythematosus. *Scand J Rheumatol, Suppl* 52: 23-29, 1984
5. Figen Tarhan, Mehmet Argin, Gerçek Can, Mustafa Özmen, Gökhan Kecer. Coexistence of systemic lupus erythematosus and ankylosing spondylitis: another case report and review of the literature. *European Journal of Rheumatology* 2014; 1: 39-43
6. Adem Kucuk, Sinan Bagcaci, Murat Biyik et al. Ankylosing spondylitis in a patient with systemic lupus erythematosus: A Rare togetherness of two old diseases. *American Journal of Advances in Medical Science*. 2014; 2(3): 47-50
7. Kook MH, Yoo HG, Hong MJ, Yoo WH. Coexisting systemic lupus erythematosus and ankylosing spondylitis: a case report and review of the literature. *Lupus* 2012; 21: 348-9.
8. Mrabet D, Rekik S, Sahli H, Trojet S, Cheour I, Eleuch M, et al. Ankylosing spondylitis in female systemic lupus erythematosus: a rare combination. *Lupus* 2011; 20: 777-8.
9. Jiang L, Dai X, Liu J, Ma L, Yu F. Hypoparathyroidism in a patient with systemic lupus erythematosus coexisted with ankylosing spondylitis: a case report and review of literature. *Joint Bone Spine* 2010; 77: 608-10.
10. Singh S, Sonkar GK, Singh U. Coexistence of ankylosing spondylitis and systemic lupus erythematosus. *J Chin Med Assoc* 2010; 73: 260-1.
11. Korkmaz C. Delayed diagnosis of porphyria based on manifestations of systemic lupus erythematosus and ankylosing spondylitis. *J Nephrol* 2006; 19: 535-9.
12. Olivieri I, Gemignani G, Balagi M, Pasquariello A, Gremignai G, Pasero G. Concomitant systemic lupus erythematosus and ankylosing spondylitis. *Ann Rheum Dis*. 1990; 49: 323-4.
13. Nashel DJ, Leonard A, Mann DL, Guccion JG, Katz AL, Sliwinski AJ. Ankylosing spondylitis and

- systemic lupus erythematosus: a rare HLA combination. *Arch Intern Med* 1982; 142: 1227-8
14. Lu LJ, Wallace DJ, Ishimori ML, Scofield RH, Weisman MH. Male systemic lupus erythematosus: a review of sex disparities in this disease. *Lupus* 2010;19:119–29
 15. Nisha N Haroon,¹ J Michael Paterson,^{2,3,4} Ping Li,² Nigil Haroon^{5,6,7}. Increasing proportion of female patients with ankylosing spondylitis: a population-based study of trends in the incidence and prevalence of AS. *BMJ Open* 2014;4:e006634. doi:10.1136/bmjopen-2014-006634
 16. Van der Linden S, Valkenburg HA, Cats A. Evaluation of diagnostic criteria for ankylosing spondylitis. A proposal for modification of the New York criteria. *Arthritis Rheum* 1984; 27: 361-368
 17. Chandrasekhara PK, Jayachandran NV, Thomas J, Narsimulu G. Systemic lupus erythematosus and dermatomyositis with symptomatic bilateral sacroiliitis: an unusual and interesting association. *Mod Rheumatol* 2009; 19: 84-6

Case Report

Case study of Dyadic death in Bangladesh: by Hanging, Strangulation and Poisoning

* Sadek A¹, Halim KS²**Abstract:**

Here described two cases where mother is the main perpetrators of the event and the victims were their children in the age group of 1 year to 12 years. In 1st case 35 years lady killed her three daughters ages 12, 9, 1 by strangulation then hanged in south surma Sylhet city. In 2nd case A 32 years old married women killed her two sons (ages 5 & 8yrs) by poisoning and later she died by hanging in Sylhet city. Autopsy examination revealed ligature mark over neck consistent with that of antemortem hanging and ligature marks round the child's neck were strangulation case also poison found by chemical examination. So it's time to take awareness within community and take prevention such like notorious incident.

Key words: Dyadic death, homicide, hanging, strangulation & poisoning

INTRODUCTION

Dyadic deaths (homicide – suicide) is defined as a dramatic violent event in which an individual kills another and subsequently commits suicide immediately or after certain period of time that may range from hours to 1 week. There is however no standardized operational definition.¹ The Hanzlick-Koponen typology has the following special classifications, which can be divided into two broad categories: single victim and multiple victim events. And according to this, single victim events (dyadic deaths) include homicide - suicide or suicide pacts.² The most common type of dyadic deaths involve killing of intimate partners and perpetrators are male in most of cases.^{1,6} A woman who kills her child, according to is an evil trick of nature. The idea of striking an innocent and defenseless being is in itself unacceptable

according to our society; when the person performing the act of violence is the infant's parent, therefore the person who by definition is supposed to care for the child, it is all the more difficult to understand the situation. In this type of felony, the following categories can be distinguished: neonaticide, when the crime takes place on the same day in which the child is born; infanticide when the victim is less than a year old; pedicide if the minor's age is between 1 and 16. The present case reports are being reported with an attempt to evaluate these episodes so that preventive measures could be initiated or adopted. In the present report, in every case, homicide was followed by suicide of the perpetrator. All the perpetrators were mother and the victims were their children in the age group of 1 years to 12 years. All homicide-suicide episodes took place in urban area. Homicide-suicide episodes are complex phenomenon and multidimensional events associated with the additive or multiplicative effects of many circumstances including psychopathological, psychosocial, economical, cultural or environmental stressors.

CASE PRESENTATION

Case 1: A 35 years lady find dead along with three others child ages 12, 9, 1. Lady was found hanging from the ceiling of her tin-roofed hut while the bodies of her three children were found lying on the floor with nooses around their necks. Police have recovered the dead bodies from their house at south Surma in Sylhet city, Bangladesh and sent for autopsy. The female was a housewife and education wise she was under matriculate. The lower class family composed of husband; wife and three little daughter were living in rented tin-roofed hut. On that tragic day, when her husband had gone for work, the female strangulated her daughters with nylon rope and then hang herself with nylon rope to ceiling hook. When husband arrived at home, the room was locked inside. After peeping from window, the mother and daughter seems hanging. Accordingly police was informed and inquest was made. The cause of homicide-suicide episode was related to family dispute due to financial crisis. Autopsy examination revealed ligature mark over neck consistent with that of antemortem hanging and ligature marks round the child's neck were strangulation case.

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Table-1: observation table of two cases:

| Case no | Perpetrator | Relationship of victim with Perpetrator | Cause of death | |
|---------|-----------------|---|----------------|---------------|
| | | | Victim | Accused |
| 1. | Mother (35 yrs) | Daughters (12, 9 & 1 yrs) | Hanging | Strangulation |
| 2. | Mother (32yrs) | Sons (5 & 8yrs) | Hanging | Poisoning |

Case 2: A 32 years old married women killed her two sons (ages 5 & 8 years) by poisoning and later she died by hanging. The female was housewife also middle socio economic status. She lived in her own house 2nd floor. Husband was business man and lived away from home. On that shock day she didn't give her sons to Scholl. She gave them breakfast with poisons in morning. After died of two sons she hanged. Police brought 3 dead bodied for autopsy. Viscera are preserved and sent for chemical analysis also finds poison. Autopsy examination revealed ligature mark over neck in case of women consistent with that of ante mortem hanging. Here the cause of homicide-suicide episode was related to family dispute due to extramarital affairs.

DISCUSSION

Dyadic death episodes mostly seen in low socio- economic, less educated or illiterate families and included single victim or multiple victim events. Demographic characteristic of perpetrator and victim are more or less same as observed in various reports. Most episodes occurred at home and perpetrator is known to victim. Extra familial incidents are rare.^{7, 10} The cardinal feature is that all the perpetrators were mother and the victims were their children in the age group of 1 year to 12 years. In Bangladesh context, mother is placed next to God. Since conception she nurtures and cares for her child. She is considered as follower, a guide, a teacher, a companion, an advisor and so on. But in this contemporary period, with changing in pattern of life style and changing morals & principles, she is expected on one side, to help, earn and lend the family and on other side to take care of family with traditional household work. With continuing large-scale urbanization and increasing trend of nuclear families, the parents are always in stress especially the mothers since she have to take care of husband as well as her wards. When a phenomenon of homicide-suicide occurs involving mother as perpetrator, many questions arises need attention, many facts need consideration and many circumstances need evaluation. In developed countries, use of firearms is common method of homicide

in dyadic deaths .Other weapons associated with this includes knives, blunt objects, and, other methods of homicide have included strangling /asphyxiation, poisoning and physical assault or vehicular accidents. The phenomenon of dyadic deaths is different from suicides. In suicide episodes, an individual prefer to die alone whereas in homicide-suicide incidents the person kills other family members and then commit suicide. Why such differentiation occurs need thought. It may possible that the person believed that after his demise there would be no one to take care of his family members and therefore he would have acted in such hostile manner. The contrary may also be possible that if "I am not living then no one had right to live". It is difficult to interpret the thought retrospectively. Homicide-suicide episodes are complex phenomenon and multidimensional events associated with additive or multiplicative effects of many circumstances including psychopathological, psychosocial, economical, cultural or environmental stressors. Establishing a psychiatric diagnosis is difficult when both the perpetrator and victim are dead but in future psychological autopsy methodology could prove helpful to prevent such tragedies.

CONCLUSIONS

The social impact of homicide-suicide as phenomenon is larger than that of homicide and suicide cases taken into consideration individually. In Bangladesh scenario financial distress and family feud are main factor for dyadic death. Also jealousy, conflict over extramarital, sexual, love affairs, threat of separation or actual separation from intimate partner is important factors seen in spousal or consortial homicide. Financial burden and marital disharmony are the principle reasons for suicide in Bangladesh. So it's time to take awareness within community and take prevention such like notorious incident.

REFERENCES

1. Mazruk P et al. The epidemiology of murder –suicide, J Am Med Assoc 1992, 267, 31: 79-83.

2. Berman AL, Dyadic death: a typology, *Suicide Life Threat Behav.* 1996; 26, 4:342-50.
3. Milroy CM et al, Reasons for homicide and suicide in episodes of dyadic death in Yorkshire and Humberside, *Med Sci Law* 1995, 35: 213 -217.
4. Graser RR, Family murder in South Africa: some possible explanations, *Acta Criminology* 1992 ,5 (19): 75 -80.
5. Bossarte RM, Simon RT , Barker L , Characteristic of homicide followed by suicide incidents in multiple states 2003 -04, *Injury prevention* 2006,12: ii 33-ii38.
6. Morton E, Rynyon CW, Moracco KE et al. Homicide – suicide involving female homicide victim; population based study in north Carolina, 1988, 13:91- 106.
7. Gupta S, Vaishnav H, Dyadic Diwali .*J Indian Acad Forensic Med*, 2008 30:75-78.
8. Dhawane S G, Mukherjee A A , Bardale RV, Dyadic deaths : analysis of homicide suicide episodes .*Medicolegal updates*, 2007,7:11-12.
9. Gupta BD, Jani CB, Patel BJ ,Shah PH .Homicide – suicide deaths (dyadic) :Two case reports .*J Forensic Medicine Toxicol*;2000,17:31-37.
10. Gupta BD, Singh OG.A unique trend of murder – suicide in the Jamnagar region of Gujarat, India (A retrospective study of 5 years). *J Forensic Leg Med*; 2008;15 :250 -5.

Review Article

Obstructive Nephropathy in Children – A Review

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Abstract

Obstructive nephropathy is any affection of the urinary tract characterized by impairment of urine flow through the tract and which, if left untreated, will cause progressive renal damage. Causes in children are congenital or acquired. Congenital causes are pelvi-ureteric junction obstructions, posterior urethral valves (PUV), urethral atresia, phimosis and meatal stenosis. The acquired causes are calculi, post-traumatic and post-inflammatory strictures and meatal stenosis. There are some manifestations like prune-belly syndrome, hydronephrosis and renal failure. Diagnostic investigations include ultrasonography, intravenous urography, cystography and renography. Advanced technologies have impacted on the treatment of the different lesions such as utero vesico-amniotic shunt and endoscopic valve ablation for PUV and minimally invasive techniques for urolithiasis. Nephrectomy may be indicated in a unilateral damaged kidney. Sometimes treatment may fail because of pretreatment irreversible renal damage. Such as end-stage renal failure is an indication for renal transplantation. So, proper treatment is essential to prevent end-stage renal failure.

Key words: Obstructive Nephropathy, Renal damage, Renal failure

INTRODUCTION

Obstructive nephropathy is a hindrance of normal urinary flow, that leads to renal dysfunction^{1,2}. The kidney is an important organ for maintaining proper homeostasis. The

renal system plays an important role in hormogenesis, metabolism, detoxification & excretion of urine that bare zinjurious to the body. The kidneys excrete unwanted products of metabolism that depends on adequate flow of urine through urinary tract.³ Obstructive nephropathy initiates a complex sequence of events resulting in impaired renal function & it is a major cause of renal impairment in infants & children.^{4,5} End Stage Renal Disease (ESRD) is an indication for paediatric renal transplantation.⁵ Chronic urinary tract obstruction impairs renal growth & development during early development.¹ The approach should include – detecting the site of obstruction and finding out whether obstruction is complete or partial, unilateral or bilateral.⁶

Etiology: Urinary tract obstruction can be caused from congenital (anatomic) lesion or trauma, neoplasia, calculi, inflammation or surgical procedures⁶, although most childhood obstructive lesions are congenital.⁵ The obstructive lesion can occur from the calyces to the external urethral meatus.⁷ Most common causes of obstruction are shown here^{4,5}:

- Meatus: Stenosis, Phimosis, Prepuce adhesion.
- Anterior urethra: Diverticulum, Stricture, Valves, Polyps.
- Posterior urethra: Posterior urethral valve, Diverticulum, Stricture, Polyps.
- Bladder: Diverticulum, Bladder neck hypertrophy, Calculi, Neuropathic bladder.
- Ureterovesical junction obstruction.
- Ureter: Stricture, Calculi, Vascular obstruction, Ureterocele, Primary megaureter, Calculi.
- Pelviureteric junction: Intrinsic abnormalities, congenital pelviureteric junction obstruction, Kinks, Bands, Adhesion, Calculi, Aberrant vessels.
- Calyx: Infundibular stenosis, Tuberculosis, Calculi.

Pathophysiology: urethral obstruction resulting in dilatation of the proximal urethra¹. Bladder attempt to overcome the obstruction suffers hypertrophy showed by thickening of bladder wall, trabeculation, sacculation & hypertrophy of bladder neck. The increased intravesical

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pressure resulting in decreased emptying of ureter into bladder & reflux of urine into the ureter. Ultimately causing dilatation of ureter, which also undergoes hypertrophy.^{1,4,5} The intraureteric pressure increases & back pressure bring about hydronephrosis & thinning of renal parenchyma. Stasis of urine dilated urinary tract causes recurrent infections.^{1,4,5} The combined stasis, repeated infections & increased intraluminal pressure causes renal parenchymal scarring & progressive deterioration in renal function. The impairment of renal function is harmful to normal growth & development.^{4,5}

In acute obstruction (e.g. from a calculus) glomerular filtration stops & tubular transport is markedly decreased.⁶ If obstruction becomes prolonged, renal fibrosis & perpetual damage follows. In intrauterine period obstruction is more serious & leads to renal dysplasia.^{1,4,5} Renal damage is irreversible even if the obstruction is relieved & may headway to ESRD at an early age.^{1,4,5}

CLINICAL MANIFESTATIONS

Most of the clinical features are due to consequences of the obstruction.² Urinary tract obstruction causes hydronephrosis, which is typically asymptomatic in its early phase.⁵ An obstructed kidney due to a ureteropelvic junction or ureterovesical junction apparent as mass or cause upper abdominal or flank pain on affected side.^{1,4,5} Pyelonephritis can occur because of urinary stasis.^{1,5,8} Upper urinary tract stone causes abdominal or flank pain & hematuria with bladder neck obstruction, urinary stream may be poor & dribbling of urine. There may be straining, incontinence & incomplete voiding.^{1,4,5} Acute obstruction results in flank pain, nausea, vomiting. Chronic obstruction may be silent or can cause vague abdominal pain. In young infant pyelonephritis may cause sepsis.¹ Renal insufficiency can evident itself by failure to thrive, vomiting, diarrhoea or other non-specific features.⁸

Posterior Urethral Valve: Most common causes of childhood obstructive uropathy leads to renal failure.⁷ The reported incidence ranges from 1 in 3000 to 1 in 8000 boys.^{8,9} About 5-64% of these patient suffer ESRD during childhood or adolescence.¹⁰ Posterior urethral valves (PUV) are 3 types. Type-I is the commonest form, which radiate distally from the verumontanum & merge into each other to form anterior commissure. Type-II is usually undetectable, do not obstruct the flow of urine. Type-III are less common, consisting of a diaphragm with a central hole.¹

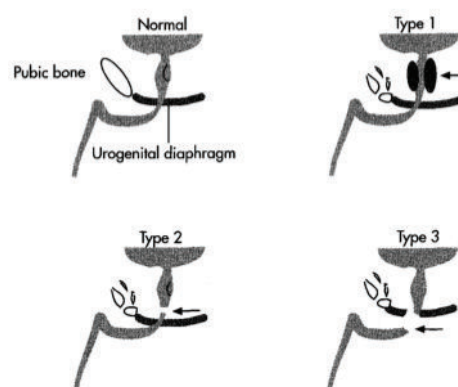


Figure:

PUV begins to express effects during developing urinary tract early in second trimester, the back pressure results in vesicoureteric reflux (VUR), hydronephrosis, renal dysplasia & impaired renal function.¹¹ It can be detected during antenatal ultrasound. The classical features are oligohydramnios, bilateral hydronephrosis & hydroureter, a thick-walled bladder & a dilated posterior urethra, renal dysplasia and pulmonary hypoplasia.^{12,13} The good prognostic factors are sodium <100 mEq/l, chloride <90 mEq/l & osmolality below 200 mosm /L.¹ Vesicoamniotic shunt has been described to overcome this to gain time for prolonging the pregnancy & protecting the developing kidneys & lungs.¹⁴ Treatment option is postnatal surgical valve ablation after treating UTI & correction of fluid & electrolyte abnormality. High loop ureterostomy is considered if the child is very sick, gross pyuria & if the trial of bladder drainage has not helped.¹¹

Pelviureteric Junction Obstruction: Pelviureteric junction (PUJ) obstruction is the functional obstruction of junction between the renal pelvis & ureter.¹¹ It is the most common cause of hydronephrosis with an incidence of 1 in 2000 children,^{15,16} with a male female ratio 3:1 & bilateral in 20-25% of cases.¹⁸ This may be due to intrinsic abnormality, muscular abnormalities of the ureter, ureteral polyps, ureteral folds, crossing vessels and rarely secondary to VUR.^{1,12}

Children usually present with flank mass, upper abdominal or recurrent flank pain, urinary tract infection (UTI), hematuria. Some may appear acutely with renal pain (Dietl's crisis).¹¹ Antenatally it is suspected in a fetus with hydronephrosis without ureteric dilatation & with a normal bladder and normal amniotic fluid volume.¹² Postnatally the majority will tend to deteriorate within the first 6 months of life as a result of maturational changes in

GFR.¹¹ By micturating cystourethrogram (MCU) & using diethylene triamine pentaacetic acid (^{99m}Tc DTPA) diuretic renogram with split renal function it can be diagnosed.¹² With grade 1 or 2 hydronephrosis, observation is usually appropriate.⁵ If the hydronephrosis is grade 3 or 4, diameter of renal pelvis more than 3cm, poor upper urinary tract drainage or poor differential renal function (35%) after diuretic renogram then surgery, Anderson Hynes pyeloplasty is recommended.^{5,17}

Vesicoureteric Reflux (VUR): VUR refers to the retrograde flow of urine from the bladder to the ureter & kidney. Reflux occurs when the submucosal tunnel between the mucosa & detrusor muscle is short or absent.¹⁸ About 38% of children with antenatal hydronephrosis it is seen.¹⁹ Reflux usually revealed during evaluation for a UTI. Around 80% are female and the mean age at diagnosis is 2-3 yrs.^{1,5} Reflux is present at birth in 25% of children with neuropathic bladder, 50% of boys with PUV, 15% with multicystic dysplastic kidney or renal agenesis, PUJ obstruction.^{1,7}

Reflux leads to pyelonephritis by facilitating the transport of bacteria from the bladder to the upper urinary tract. The inflammatory reaction may result in renal injury or scarring, renal insufficiency, ESRD.²⁰ Reflux severity is graded using the international reflux study classification is based on the appearance of the urinary tract on a contrast voiding cystourethrogram (VCUG).⁵ Grade-I: reflux into a nondilated ureter; Grade-II: reflux into the upper collecting system without dilatation; Grade-III: reflux into dilated ureter & blunting of calyceal fornices; Grade-IV: reflux into a grossly dilated ureter; Grade V: massive reflux with significant ureteral dilatation & tortuosity loss of the papillary impression.²¹

VUR is treated with antibiotic prophylaxis & follow up for grade I, II and surgical reimplantation & endoscopic correction for grade IV, V.²²

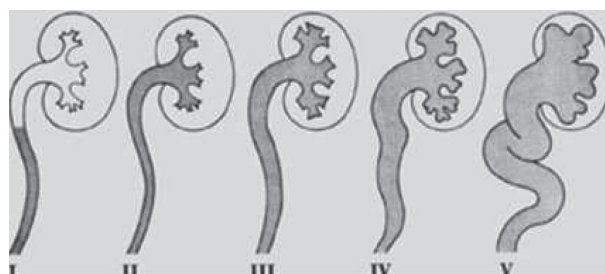


Figure:

Neurogenic Bladder: Neurogenic bladder means malfunction of the urinary bladder due to disease of the central nervous system or peripheral nerves that controls micturition.²³ The causes include dysraphism, open and closed spina bifida, sacral agenesis, spinal cord tumor, trauma, transverse myelitis and autonomic neuropathy.²⁴ Features lead to suspect are poor or impaired urinary stream, straining to pass urine, impaired / lack of bladder sensation, small urinary volumes, continual dribbling, infrequent micturition, impaired bladder emptying, recurrent UTI, abnormality of the spine, abnormality of lower limb & associated constipation.^{23,24}

For diagnosis USG of abdomen, MCU for VUR and dimercaptosuccinic acid (^{99m}Tc DMSA) scintigraphy for renal scars can be made.²⁵ Management done with bladder expression (the crede manoeuvre), bladder straining, clean intermittent catheterization, and anticholinergic drugs. Surgery, like vesicotomy, urinary diversion, sphincterotomy, reimplantation of ureters, endoscopic reflux correction is also recommended.²⁴

Urolithiasis: Approximately 7% of urinary calculi occur in children less than 16 years of age.²⁶ Renal stones are composed of calcium salts (70%), uric acid, magnesium ammonium phosphate or cystine. Metabolic abnormalities seen with these children like hypercalciuria, hyperoxaluria, hyperuricosuria, UTI, cystinuria & urinary tract anomalies.^{27,28} Medical management such as thiazides for hypercalciuria, alkali administration in renal tubular acidosis, allopurinol in hyperuricosuria. Surgical measure like extracorporeal shock wave lithotripsy, percutaneous nephrolithotomy or open surgery also done.²⁸

Urethral Stricture: Urethral stricture are common in boys. It can be congenital or acquired. Congenital stricture urethra is not common. Acquired stricture occurred due to infection, trauma.⁵ Child may manifest with dribbling of urine or poor urinary stream associated with straining, palpable bladder and occasionally palpable kidneys. The diagnosis can be made by a intravenous urography (IVU) or retrograde urethrography, ultrasonography. But endoscopy is confirmatory.⁵ Definitive treatment is surgery, which includes urethrotomy, urethral dilatation, urethroplasty.^{2,3}

Phimosis: Phimosis is inability to retract the prepuce. It is physiologic at birth. But with time the adhesions between

the prepuce, glans lyse & the distal phimotic ring loosens.²⁹ At the end of the first year of life retraction of foreskin behind the glandular sulcus is possible in only about 50% of boys, this rises to approximately 90% by the age of 3 years.^{1,35} In true phimosis dispose to smegma collection which causes recurrent balanoprophitis^{1,30}. If physiological phimosis persist with dysuria corticosteroid cream is given.^{1,31} Circumcision is needed in true phimosis.

Prune Belly Syndrome: Prune belly syndrome also known as triad syndrome or Eagle Barrett syndrome⁷ occurs in approximately 1 in 29000 to 40000 male births.³² Urinary tract abnormalities, abdominal muscle deficiency and undescended testis are the components.³³ Cardiac (ASD, VSD, TOF), pulmonary hypoplasia, gastrointestinal tract abnormality (Malrotation of Gut, Volvulus, Omphalocele, Imperforate Anus), musculoskeletal anomaly (limb anomaly, scoliosis, hip dislocation, pectus excavatum, talipes equinovarus) are often associate.³⁴



Figure:

Temporary bladder drainage procedures such as vesicotomy, orchidopexy & abdominoplasty with antibiotic prophylaxis are the treatment options. Renal transplantation also brings good results.^{5,12}

Hydronephrosis: Hydronephrosis can be defined as dilatation of the renal pelvis and or calices. It is detected by routine antenatal USG with an incidence of 0.5 to 1.³⁶ Commonly detected between 18 and 20 weeks of gestation at the routine anomaly scan.¹⁵ More than 5mm diameter is considered abnormal.^{15,38} It is usually unilateral but may be bilateral in 15-30% cases.⁴²

Society of fetal urology grading of hydronephrosis⁵.

| Grade | Central renal complex intact | Renal Parenchymal Thickness Normal | Ultra sound scan |
|-------|---|------------------------------------|------------------|
| I | Intake | Normal | |
| II | Slight splitting of pelvis | Normal | |
| III | Evident splitting of pelvis and calices | Normal | |
| IV | Wide splitting of pelvis and calices | Normal | |
| V | Further splitting of pelvis and calices | Normal | |

Prenatal intervention is placing a vesicocamniotic shunt^{37,39}. Postnatally with hydronephrosis, evaluation of VUR & antibiotic prophylaxis recommended. At 4-6 weeks of age MCU, diuretic renography could be done. Surgery is required in 28% cases. PUV account for 1-4%, where definitive treatment is surgery.³⁷

INVESTIGATION AND IMAGING MODALITIES FOR EVALUATION

Urine R/M/E, Culture & sensitivity test, Complete blood count, Serum creatinine, electrolyte, bicarbonate, calcium, phosphorous, parathyroid hormone.

Urinary tract dilatation, renal cortical thickness, calyx size, diameter of pelvis, ureter, thickness of bladder, tumor and calculi can be obtained by renal USG. Doppler USG needed for evaluation of vascular anomalies.^{1, 40} Plain x-ray KUB for urinary tract calculi.¹

Radionuclide studies includes ^{99m}Tc DTPA, ^{99m}Tc DMSA, ^{99m}Tc MAG3 (Mercaptoacetyl triglycine), ^{99m}Tc GHA (Glucuheptonate) for renal scarring and VUR.¹

Radiodiagnostic studies includes IVU for hydronephrosis, calculi, ureterocele; MCU for bladder and urethral obstruction, VUR; retrograde urethrography for urethral obstruction.¹

Endoscopy for direct visualization of lesion e.g PUV.

Magnetic resonance urography may be carried out for determination of cause, level and degree of obstruction in poorly functioning kidneys.⁴¹

COMPLICATIONS

Congenital obstructive nephropathy is the most common cause of chronic kidney disease (CKD) and ESRD in children.¹ Symptomatic and asymptomatic UTI are both common. Hypertension is common in scarred kidney with proteinuria. Fluid and electrolyte abnormalities, systemic acidosis (distal RTA) occurs due to insufficient urinary acidification.¹⁶ Growth retardation due to bony abnormalities and failure to thrive are also common.^{1,42}

TREATMENT

After diagnosing obstructive nephropathy therapy emphasizes on the rapid restoration of normal urine flow either by medical or surgical interventions.

There are some newer surgical interventions for VUR as conventional, open ureteric re-implantation increasingly replaced by alternative techniques:

*Sub-ureteric transurethral injection (STING) of dextranomer/hyaluronic acid co-polymer (Dx/HA) or Deflux procedure.

*Laparoscopic procedure

*Robot assisted procedure

Bladder drainage, vesicostomy, valve ablation and high diversion surgery and VUJ stents are indicated in PUV.^{42,43}

Initially patient may need acute renal replacement therapy to remove toxins and maintain fluid, electrolytes & acid base balance.⁴³ Some patients may experience enough recovery of renal function and compensatory function dialysis once established.¹²

In patients with CKD hyperkalemia and hyperphosphatemia can be compensate with low potassium, low phosphorus diets with phosphate binders.¹² Anemia is corrected with iron supplementation or human recombinant erythropoietin. Production of 1,25-dihydroxy

vitamin D is reduced, requiring supplementation. Nutritional supplementation and recombinant growth hormone is necessary to maintain normal growth.⁴⁴

PROGNOSIS

16.3% of pediatric obstructive nephropathy patients goes into ESRD requiring renal transplantations.⁴⁹ Long term renal function is variable depends on underlying pathology and associated complications. Prognosis is worse if UTI remains untreated.²² Earlier diagnosis and skilled intervention by pediatrician and pediatric urologists can give a better outcome.

REFERENCES

1. Srinivas M, Bhatnagar V. Obstructive Nephrology. In Pediatric Nephrology 5th edition edited by Srivastava RN, Bagga A Jaypee Brothers Medical Publishers Ltd. New Delhi 110002, India. 2011; 433-450.
2. Eke N, Elenwo SN. Obstructive uropathy in childhood: A Review. Port Harcourt medical Journal. 2007; 1: 137-144.
3. Chevalier RL, Peters CA. Obstructive uropathy In Pediatric Nephrology 6th edition edited by Avner ED, Harmon WE, Niaudet R, Yoshikawa N. Springer-Verlag Berlin Heidelberg. 2009; 1337-1376.
4. Mehta k. Approach to obstructive uropathy. Pediatric oncall. 2000; 1: 212-234.
5. Elder JS. Obstruction of the urinary tract in Nelson textbook of Pediatrics 19th edition edited by Kliegman RM, Stanton BF, Schor NF, St Geme JW, Behrman RE. Elsevier Inc. PA 191032899; 1838-1847.
6. Zeidel ML. Obstructive uropathy, Renal and Urology. 2008; 776-780.
7. Hodges SJ, Patel B, Mcclonie G etal. Posterior urethral valves. Scientific world journal. 2009; 9 : 1119.
8. Woolf AS, Triruchelvam N. Congenital obstructive uropathy: Its origin and contribution to end stage renal disease in children. Adv Ren Replace Ther. 2001; 8: 157-163.
9. Yohannes P, Hanna M : Current trends in the management of posterior urethral valve in the pediatric population. Urology. 2001; 60:947.
10. Roth KS, Carter WH, Chan JC. Obstructive nephropathy in children: Long term prognosis after

- relief of posterior urethral valve. *Pediatrics*. 2001; 107:1004.
11. Postlethwaite RJ, Dickson A. Common urological problems in *Clinical Pediatric Nephrology* 3rd edition edited by Nicholas JA, Robert JP. Oxford University press New York, USA. 2003; 227-258.
12. Becker A, Baum M. Obstructive uropathy. *Science direct*. 2006; 82:15-22.
13. Heikkila J, Christer H, Kyllonen L, Rintala R, Taskinen S. Long term risk of end stage renal disease in patient with posterior urethral valve. *The journal of urology*. 2011; 186, 2392-2396.
14. Eke N, Elenwo SN. Obstructive uropathy in childhood: A review. *Port Harcourt Medical Journal*. 2007; 1: 137-144.
15. Woodward M, Frank D. Postnatal management of antenatal hydronephrosis. *BJU Int*. 2002; 89: 149-256
16. Chevalier RL. Perinatal obstructive nephropathy. *Semin perinatal*. 2004; 28: 124-31.
17. Shokeir AA, Nijman RJ. Antenatal hydronephrosis changing concepts in diagnosis and subsequent management. *BJU Int*. 2000;85:987-94.
18. Elder JS. Vesicoureteral reflux In *Nelson textbook of Pediatrics* 19th edition edited by Kliegman RM, Stanton BF, Schor NF, St Geme JW, Behrman RE. Elsevier Inc. PA 191032899;1834-1838.
19. Mourmouris PI, Chiras T, Papatsoris AG. Obstructive Uropathy: From Etiopathology to Therapy. *World Journal of Nephrology and Urology*. 2014; 3(1): 126.
20. Woodward M, Frank d. antenatal renal problems: management in postnatal period In *Clinical Pediatric Nephrology* 3rd edition edited by Nicholas JA, Robert JP. Oxford University press New York, USA. 2003; 269-286.
21. Radmayr C. Congenital Obstructive Uropathy – Diagnostics for optimal treatment. *African Journal of Urology*. 2015; 21, 87-95.
22. Hari P, Srivastava RN. Urinary tract infection In *Pediatric Nephrology* 5th edition edited by Srivastava RN, Bagga A Jaypee Brothers Medical Publishers Ltd. New Delhi 110002, India. 2011; 273-300.
23. Elder JS. Voiding dysfunction In *Nelson textbook of Pediatrics* 19th edition edited by Kliegman RM, Stanton BF, Schor NF, St Geme JW, Behrman RE. Elsevier Inc. PA 191032899;1847-1852.
24. Borzyskowski M. Neuropathic bladder: Identification, investigation and management In *Clinical Pediatric Nephrology* 3rd edition edited by Nicholas JA, Robert JP. Oxford University press New York, USA. 2003; 179-195.
25. Kanitkar M. Disorders of micturition In *Pediatric Nephrology* 5th edition edited by Srivastava RN, Bagga A Jaypee Brothers Medical Publishers Ltd. New Delhi 110002, India. 464-489.
26. Elder JS. Urinary lithiasis In *Nelson textbook of Pediatrics* 19th edition edited by Kliegman RM, Stanton BF, Schor NF, St Geme JW, Behrman RE. Elsevier Inc. PA 191032899;1864.
27. Thomas DFM. Upper tract obstruction. In: Thomas DFM, Duffy PG, Rickwood AMK, editors. *Essentials of paediatric urology*. 2nd ed. London: Informa Healthcare. 2008; 73–92.
28. Bagga A. Urolithiasis In *Pediatric Nephrology* 5th edition edited by Srivastava RN, Bagga A Jaypee Brothers Medical Publishers Ltd. New Delhi 110002, India. 451-463.
29. Elder JS. Anomalies of the penis and urethra In *Nelson textbook of Pediatrics* 19th edition edited by Kliegman RM, Stanton BF, Schor NF, St Geme JW, Behrman RE. Elsevier Inc. PA 191032899;1852-1858.
30. Siddiqui MM, McDougal WS. Urologic assessment of decreasing renal function. *Med Clin North Am*. 2011;95(1):161-168.
31. Leclair MD, Gundeti MS, Heloury Y, Duffy P, Mushtaq I. PUJ obstruction and lower pole crossing vessels: further experience with the laparoscopic vascular hitch. *J Pediatr Urol*. 2009; 5(1):44.
32. Gundeti MS, Reynolds WS, Duffy PG, Mushtaq I. Further experience with the vascular hitch (laparoscopic transposition of lower pole crossing vessels): an alternate treatment for pediatric ureterovascular ureteropelvic junction obstruction. *J Urol*. 2008;180: 1832–6.
33. Leeners B, Sauer J, Schefels J, Cotarelo CL, Funk A. Prune belly syndrome: Therapeutic option including

- in utero placement of a vesicoamniotic shunt. *J Clin Ultrasound*. 2000; 28: 500-507.
34. Strand WR. Initial management of complex pediatric disorders: Prune belly syndrome, Posterior urethral valve. *Urol Clin North Am*. 2004; 31: 399-415.
35. Olsen LH, Rawashdeh YF, Jorgensen TM. Pediatric robot assisted retroperitoneoscopic pyeloplasty: a 5-year experience. *J Urol*. 2007;178(5):2137-41.
36. Sidhu G, Beyene J, Rosenblum ND. Outcome of isolated antenatal hydronephrosis: A systemic review and metaanalysis. *Pediatr Nephrol*. 2006; 21: 218
37. Bagga A, Gulati A. Disease of the newborn In *Pediatric Nephrology* 5th edition edited by Srivastava RN, Bagga A Jaypee Brothers Medical Publishers Ltd. New Delhi 110002, India. 494-524
38. Yiee J, Wilcox DT. Ureteropelvic junction obstruction. In: Wilcox DT, Godbole P, Koyle MA, editors. *Pediatric urology: surgical complications and management*. London: Wiley-Blackwell. 2008; 58-66.
39. Tseng TY, Stoller ML. Obstructive uropathy. *Clin Geriatr Med*. 2009;25(3):437-443.
40. Hewitson TD. Renal tubulointerstitial fibrosis: common but never simple. *Am J Renal Physiol*. 2009;296:1239-1244.
41. Sen KK, Mohan C, Verma BS. Magnetic resonance urography in obstructive uropathy. *MJAFI*. 2008; 64: 145-147
42. Feder MT, Blitstein J, Mason B, Hoenig DM. Predicting differential renal function using computerized tomography measurements of renal parenchymal area. *J Urol*. 2008;180(5):2110-2115.
43. Andreoli SP. Acute renal failure in newborn. *Semin Perinatol*. 2004; 28: 112-23.
44. Alvarez-Prats A, Hernandez-Perera O, Diaz-Herrera P, Ucero AC, Anabitarte-Prieto A, Losada-Cabrera A, Ortiz A, et al. Combination therapy with an angiotensin II receptor blocker and an HMG-CoA reductase inhibitor in experimental subtotal nephrectomy. *Nephrol Dial Transplant*. 2012; 27(7):2720-2733.

Letter to the Editor

Practice Points for Insulin in Diabetes Mellitus

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Diabetes mellitus is a global epidemic including Bangladesh. In the management of diabetes, the ultimate treatment is insulin therapy.

Abstract

Insulin is a type of parenteral anti-diabetic agent. There are several types of insulins classified according to onset and duration of action. Insulin is the main modality of treatment in cases of uncontrolled diabetes, those cases not controlled by oral agents and in patients with comorbidities. Insulin regimens are chosen depending on patient preference, meal pattern and risk of hypoglycaemia. The main limitation of this drug is hypoglycaemia and weight gain. Apart from these it is a safe and effective choice for treatment of diabetes.

Indications of insulin¹

1. Type 1 diabetes
2. Pregnancy, lactation
3. Acute metabolic complications
4. Severe acute illness
5. Comorbidity
6. Major surgery
7. Poor glycaemic status at onset of diabetes (very high blood sugar, HbA1c > 9.5%)
8. In case of oral anti-diabetic drug failure

Target blood glucose for optimizing glycaemic control

Target should be individualized. It depends on age, duration of diabetes, presence of comorbidities and complications, risk of hypoglycaemia. Approach should be patient centered².

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| Glycaemic parameter | Level |
|-----------------------------------|--|
| For non pregnant adult | |
| Fasting blood glucose (FBG) | 4.4-7.2 mmol/L |
| Post prandial blood glucose (PPG) | <10 mmol/L |
| HbA1c | 7% |
| | (6.5% for young patients without complications and low risk of hypoglycaemia; 8% for elderly patients, with comorbidities and risk of hypoglycaemia) |
| For children | |
| HbA1c | 7.5% |
| For pregnant woman | |
| HbA1c | 6-6.5% |

Ref: ADA 2018

Types of Insulin

Different types of insulin preparations are available in the market¹.

| Class | Name | Time with meals Conventional/Human |
|--|---|---------------------------------------|
| Short acting | Regular | 30 minutes before meal |
| Intermediate acting | NPH Lente | 30 minutes before meal |
| Analogue | | |
| Rapid acting analog | Glulisine Aspart Lispro | 10 minutes before meal |
| Long acting analog | Detemir Fixed time Glargine Degludec | Fixed time No specific time |
| Premixed Conventional: short/ intermediate acting (30/70, 50/50, 25/75) | | 30 minutes before meal |
| Analogs: biphasic aspart, degludec+aspart | | 10 minutes before meal |

Guidelines for the use of Insulin¹

- **Insulin mixtures / twice daily regimen**

1. It is convenient with good compliance, hence more widely used.
2. Requires rigid meal plan and more challenging to achieve good glycaemic control.
3. Premixed conventional / analogue insulin is given before breakfast and dinner.
4. In case of human insulin, 2/3rd of the total insulin dose is given before breakfast and 1/3rd before dinner.
5. Dose should be adjusted weekly by doing self blood glucose monitoring (SMBG).

- **Basal bolus regimen / multiple daily injections**

1. Good for those with flexible meal plan, requiring good glycaemic control and with danger of hypoglycaemia.
2. It is inconvenient with poor compliance.
3. Intermediate acting / long acting insulin analogue at bedtime is given as basal insulin. Short acting / rapid acting insulin analogue before each meal is given as bolus insulin.
4. Dose should be adjusted weekly by doing self blood glucose monitoring (SMBG).

- **Insulin can be used with oral anti-diabetic drugs. However, sulphonylurea should be avoided when this insulin regimen is given.**

Insulin administration technique¹

1. Insulin devices include syringe and vial and pen devices. Insulin pumps are also available in our country.
2. Insulin injection sites are the abdomen, upper arm and thigh.
3. It is given subcutaneously
 - pinch the skin and insert the needle at a right angle in case of pen device. At 30-45° in case of syringe.
 - do not give injections repeatedly at one site
4. Regular insulin and rapid acting analogs can also be given intramuscularly and intravenously.

Advice to patients on Insulin

1. Insulin may cause hypoglycaemia if given inappropriately, if there is missed meal or undue exercise.
2. Educate patient regarding hypoglycaemia symptoms, treatment and prevention.
3. Educate the patient about sick day management.
4. Educate the patient about insulin technique and self monitoring of blood glucose (SMBG).

References

1. Holt RIG, Cockram C, Flyvbjerg A, Goldstein BJ, editors. Textbook of Diabetes. Singapore: Wiley-Blackwell Publishing; 2010.
2. American Diabetes Association. Glycaemic targets: standards of medical care in diabetes. Diabetes Care. 2018; 41 (1): S55-S64.

Obituary news September-2017

BMA would like to express deep condolence on deaths of the following notable physicians in recent past:

| Sl. No. | Name | Age | Name of District | Date of Death |
|---------|--|-----|------------------|---------------|
| 1 | Dr. Moshir Rahman | 75 | Dhaka | 05/06/2017 |
| 2 | Dr. K.M. Nazrul Islam Ex- Professor of DMC | - | Dhaka | 11/6/2017 |
| 3 | Dr. Akhter Nehal Ex- Professor of Sher-E-Bangla Medical College | | | 15/6/17 |
| 4 | Dr. Md. Shah Alam Talukder | 65 | Dhaka | 16/6/2017 |
| 5 | Freedom Fighter Dr.Abu Siddique | - | Dhaka | 22/6/2017 |
| 6 | Prof. (Dr.) M.A. Majed Ex-President, BMA. | 85 | Dhaka. | 08/7/2017 |
| 7 | Dr. Bishnupada Pathi | 90 | Tangail | 22/7/2017 |
| 8 | Dr. Paritosh Chandra Bhaumik | 65 | Kishorgonj | 22/7/2017 |
| 9 | Dr. Khademul Islam Bakul | 65 | Dhaka | 06/8/2017 |
| 10 | Freedom Fighter Dr. Makbul Hossain | 65 | Mymensingh | 14/09/2017 |

May Allah bless the departed souls. Our heartiest commiseration to the deceased's family, our prayers are with them during this difficult moment of their life.