

Vol. 1, No. 1  
January 2016

# Tairunnessa Memorial Medical College Journal

Peer Reviewed Journal

TMCC Journal, January-June 2016, Volume 1, Number 1

Content	Page
<b>Editorial</b>	
<b>Ebola Virus Disease: an Update</b> <i>Prof Dr Asma Kabir</i>	9
<b>Original Article</b>	
<b>HBeAg Negative Chronic Hepatitis in Bangladeshi Patients</b> <i>Majid F, Moben AL, Jahan M, Hussain D, Tabassum S</i>	11
<b>Pattern of Victims in Fatal Road Traffic Accident</b> <i>Kabir MJ, Parvez S, Ahamed BU, Salam F, Hossain ME</i>	18
<b>Learner Based Education</b> <i>Kabir J</i>	22
<b>Common Skin Disease Among Primary School Children in a Selected Rural Area of Bangladesh</b> <i>Hasan MM, Riya S, Das S, Ahmed SS, Kabir A</i>	27
<b>Review Article</b>	
<b>Arsenic Contamination of Drinking Water and Health Effects: Bangladesh Perspective</b> <i>Kabir A, Riya S, Chowdhury AA</i>	32
<b>Autism: A Global Challenge</b> <i>Riya S, Kabir A</i>	36
<b>Case Report</b>	
<b>An experience on the Treatment of Relapse of a Kala-azar Patient with Liposomal Amphotericin-B</b> <i>Islam MS, Azad MAK, Kader MA</i>	41
<b>Systemic Sclerosis Presenting as Raynaud's Phenomenon: A Case Report</b> <i>Sultana GA, Mohammed ZRB. Md AA</i>	45



Official Journal of  
Tairunnessa Memorial Medical College

### ***Chief Patron***

Mrs. Jahanara Hoque, Chairman, Governing Body, TMMC

### ***Editorial Board***

Chairman : Prof. Dr. Dilruba Rahman, Principal, TMMC  
Editor in Chief : Prof. Dr. Asma Kabir, Vice- Principal, TMMC  
Executive Editor : Ms. Jackie Kabir, Director, Students Affairs, TMMC  
Associate Editors : Prof (c.c.) Dr. Md. Zafor Sadeque  
Prof (c.c.) Dr. Fatma M. Khan  
Dr. Sayeda Riya  
Dr. Farjana Majid

### ***Assistant Editors***

Dr. Magfura Pervin  
Dr. Farida Yeasmin  
Dr. Mohammad Mehedi Hasan

### ***Advisory Board***

Prof. Dr. Abdul Khaleque Akond  
Prof. Dr. Nahid Sultana  
Prof. (c.c.) Dr. Md. Abdul Kader  
Prof. (c.c.) Dr. Md. Abbas Uddin Khan  
Prof. (c.c.) Major (Rtd.) Dr. Sheikh Firoj Kabir

## **INTRODUCING TAIRUNNESSA MEMORIAL MEDICAL COLLEGE**

The college is housed in a 10 storied building. It has a floor space of 1,07,550 Sq-ft which accommodates 8 departments with laboratories and 6 lecture galleries filled with multimedia facilities. Due importance was given for Anatomy Dissection Hall and Museum which is situated on the second floor of the academic building. Total number of full time teachers in the college is 140; 11 professors, 14 Associate Professors and 17 Assistant Professors. Academic activities of the college began in the session 2003-2004. So far 201 students have been graduated from TMMC. Total numbers of students in MBBS course accounts 520 in TMMC. A state of art medical library is present on the 5th floor with 4000 books, 1500 journals and magazines. A rich computer laboratory, 10 computers with internet connection to facilitate students and faculties, is located next to the library. Para clinical students undertake field visits round the year as demanded by the curriculum. A 500-bed hospital is situated within 200 yards of the college. Gynecology and Obstetrics, Medicine and Surgery departments run with their allied subjects in this premise. In each department 2 beds are reserved for poor patients. On Mondays all specialists provide free service for poor patients and poor patients also receive discount on investigation cost. Free camping from the departments of Ophthalmology, Gynecology and Surgery are arranged on regular basis. The EPI (Expanded Programme on Immunization) is conducted in the premises on Monday and Thursday. The cafeteria 'Niloy' situated on the ground floor next to the college building managed by the college authority ensures availability of snacks and small meals for all in TMMC&H and it buzzes with students during their break. A 250 separate bedded girls' hostel situated within the campus, very close to the college building. A 250 bedded boys' hostel owned by Tairunnessa Memorial Medical College is located just one kilometer away from the campus. The hostels are taken care of by superintendents as assigned from the faculties. Students commute between the college and hostel by bus operated by the college management.

### **Location**

The College is situated on Dhaka Mymensingh Highway at Konia, Board Bazar. It is 20 km from Hazrat Shahjalal International Airport, Dhaka.

### **Objective**

The objective of the Tairunnessa Memorial Medical College Journal is to produce world class physicians who through their discourse will attempt to produce a forum for the medical teachers and administrators to share their creativity and ideas which can in future envision the future of the health care system.

## CHAIRMAN'S MESSAGE

It is indeed a great pleasure to declare the successful launching of the Tairunnessa Memorial Medical College Journal. With deep respect we dedicate the first issue to the memory of Md Shamsul Haque (1943-2013), Honorable Founder Chairman of the Tairunnessa Memorial Medical College & Hospital. His inspiration and motivation was instrumental in the establishment of this institute which has already earned reputation in ensuring quality teaching of students and patient care. Blessing of his departed soul is our strength to take the institute to further higher level. Publication of the Tairunnessa Memorial Medical College Journal is a part of the process to boost the standard of the institute and create opportunity for its faculties to conduct research and contribute to the journal. We thank and extend deep gratitude to all who worked hard to bring out the very first issue of the journal and start the long journey ahead.

Dr. Dilruba Rahman  
Principal  
Tairunnessa Memorial Medical College



### **Consideration of Manuscripts**

- All submitted manuscripts are subject to double blind peer review.
- All submitted articles are received on the fact that it represents an original contribution and they are not previously published or are being considered for publication else where.
- On acceptance of a manuscript submitted for publication it implies transfer of the copyright from author to publisher.

### **Ethical aspects**

Manuscripts based on human studies should be in accordance with ethical standards laid down in the Helsinki Declaration of 1975 revised in 2000.

In manuscripts where human subjects are involved in studies, must be approved by appropriate ethical committee.

### **Manuscripts preparation**

The guide lines given below are requested to be followed by the authors on submission of manuscript to TMMC for publication.

1. A cover letter addressed to the Editor of the Journal.
2. Typing on a email instead of CD. The e- mail address is [tmmcj.asma@gmail.com](mailto:tmmcj.asma@gmail.com)
3. Title Page
4. Abstract
5. Introduction
6. Materials and Methods
7. Results/Tables and legends / illustrations
8. Discussion
9. Conclusion
10. Acknowledgement (if any)
11. References

### **Cover letter**

A letter listing (i) full name with surname of principal author in bold font (ii) full address and affiliation of the principal author and co-authors to whom proofs will be sent should be given on e-mail account number (iii) All authors must sign on submission of manuscripts stating that they are the sole authors.

### **General information**

- Word limit for different types of articles: Original articles - upto 3000 words, review articles - 4,500 words and case report - 1700 words. Letter to the Editor - maximum 1000 words
- Manuscript submitted should be concise and written in English
- Manuscript must be prepared in accordance with the guidelines laid down by the International Committee of Medical Journal Editors ([www.icmje.org/index.html](http://www.icmje.org/index.html)).
- Text in double spaced throughout, justified and 2.5 margins
- Body text typed in Times New Roman and Font size 12

### **Title Page**

- Includes title of the article with, name of the institution/department where the study was conducted.
- Name of all authors with their designation and institutional designations.
- Name of the corresponding author with contact address telephone number, E mail address: [tnmcj.asma@gmail.com](mailto:tnmcj.asma@gmail.com)
- Disclosure of sources of funding or sponsor
- A short running head
- Number of tables given
- Number of figures given
- Word count of abstract

### **Abstract**

- Should not exceed 250 words
- Should state precisely and concisely about the work done the main findings and interpretation of the work
- Should be structured having background objectives, materials and methods, results and conclusion.
- A non structured abstract is suggested for review articles and case report.
- Below the abstract three to five appropriate key words relevant to the article should be mentioned.

## **Introduction**

The introduction will acquaint the readers the purpose and rationale of the article and should include.

- Strictly pertinent references.
- Brief review of the subject excepting date and conclusion.
- Should be very clear mentioning study design, place and period.

## **Materials and Methods**

This section of the study should be very clear and should describe:

- The study design, place and period.
- The selection criteria of the study population including controls (in any).
- The methods and apparatus used in the research in sufficient details to allow other researchers to reproduce the results.
- The drugs and chemicals used precisely including generic name, dose and route of administration.
- Statistical procedure should be briefly and comprehensively addressed.

## **Results**

- The results should be presented in logical sequence in text tables, and illustrations.
- Described without comment.
- Supplemented by concise textual description of the data presented in tables and figures where it is necessary.

## **Tables and Legends**

The following principles should be followed during preparation of tables.

- Should be simple, self explanatory and supplement, not duplicate in text.
- Each table should have a title and typed in double space.
- Each table should be numbered consecutively in Roman and printed in separate page.
- Do not use internal horizontal and vertical rules.

## **Illustration**

- All illustration must be numbered consecutively in Arabic numerals as cited in the text.
- Print photograph of each illustration along with its electronic file should be submitted.
- Figure number, title of manuscript, name of the corresponding author, and arrow indicating top should be written on a sticky label affixed on the back of each illustration.
- Original drawings, graphs, charts and letterings should be prepared on an illustration board or high grade white drawing paper by an experienced medical illustrator.

## **Legend**

- Must be typed in a separate page.
- Photo micrograph should indicate the magnification internal scale and the method of staining

## **Discussion**

The discussion section should reflect

- Comprehensive analysis of the results
- Emphasis should be made on new and important aspects of the study and the conclusions derived thereof.
- Repetition in detail data or other material given in the introduction or results section should be avoided.
- Describe the implications of the findings and their limitations, including, implications for future research.
- Relate the observations to other relevant studies.

## **Conclusion**

- Link the conclusion with the goals of the study
- Avoid unqualified statements and conclusions which completely do not support your data.
- Include recommendation when appropriate

## **Acknowledgement**

- Contributions that need acknowledgement but do not justify authorship should be specified.
- Individuals institution sponsor, organization for technical help, financial and material support can be acknowledged.

## **References**

- Reference should be written in modified Vancouver style and should follow the ICMJE guidelines (<http://www.icmje.org>).
- References should be numbered consecutively in the order in which they are first mentioned in text.
- Names of 6 (six) authors can be given followed by et,al if author number is more than six.

## **Stand journal article**

### **Example**

- Choudhury S, Chowdhury T A Laparoscopic assessment of tubal functions in sulfentility. Bang J Obstet Gynaecol 1992; **17**: 9-16.
- Journal articles with organization as author World Health Organization. NHO laboratory manual for the examination and processing of human semen 5th ed. Jereva: World Health Organization Press 2010 P 17.
- Standard Journal article on the Internet <http://www.unicef.org/bangladesh /child and Maternal Nutrition %281%29.pdf> accessed on 18th April 2014.

**TMMC JOURNAL**  
Volume 1, Number 1, January 2016

<b>Content</b>	<b>Page</b>
<b>Editorial</b>	
<b>Ebola Virus Disease: an Update</b> <i>Prof Dr Asma Kabir</i>	9
<b>Original Article</b>	
<b>HBeAg Negative Chronic Hepatitis in Bangladeshi Patients</b> <i>Majid F, Moben AL, Jahan M, Hussain D, Tabassum S</i>	11
<b>Pattern of Victims in Fatal Road Traffic Accident</b> <i>Kabir MJ, Parvez S, Ahamed BU, Salam F, Hossain ME</i>	18
<b>Learner Based Education</b> <i>Kabir J</i>	22
<b>Common Skin Disease Among Children in a Selected Rural Area of Bangladesh Primary School</b> <i>Hasan MM, Riya S, Das S, Ahmed SS, Kabir A</i>	27
<b>Review Article</b>	
<b>Arsenic Contamination of Drinking Water and Health Effects: Bangladesh Perspective</b> <i>Kabir A, Riya S, Chowdhury AA</i>	32
<b>Autism: A Global Challenge</b> <i>Riya S, Kabir A</i>	36
<b>Case Report</b>	
<b>An experience on the Treatment of Relapse of a Kala-azar Patient with Liposomal Amphotericin-B</b> <i>Islam MS, Azad MAK, Kader MA</i>	41
<b>Systemic Sclerosis Presenting as Raynaud's Phenomenon: A Case Report</b> <i>Sultana GA, Mohammed ZRB. Md AA</i>	45

## EBOLA VIRUS DISEASE: AN UPDATE

**Prof Dr Asma Kabir**

Ebola virus disease (EVD), formerly known as Ebola haemorrhagic fever, is a severe, often fatal illness in humans. The average EVD case fatality rate is around 50%. Case fatality rates have varied from 25% to 90% in past outbreaks. The first EVD outbreaks occurred in remote villages in Central Africa, near tropical rainforests, but the most recent outbreak in west Africa has involved major urban as well as rural areas. Ebola virus disease (EVD) first appeared in 1976 in 2 simultaneous outbreaks, one in Nzara, Sudan, and the other in Yambuku, Democratic Republic of Congo. The latter occurred in a village near the Ebola River, from which the disease takes its name. The current outbreak in West Africa, (first cases notified in March 2014), is the largest and most complex Ebola outbreak since the Ebola virus was first discovered in 1976. On 8 August 2014 the WHO Director-General declared this outbreak a Public Health Emergency of International Concern<sup>1</sup>. Since discovered Ebola has proven to be a stable one with a relatively constant mutation rate. The Ebola virus samples from this outbreak are 97% similar to the virus that first emerged in 1976<sup>2</sup>. It is thought that fruit bats of the Pteropodidae family are natural Ebola virus hosts. Ebola is introduced into the human population through close contact with the blood, secretions, organs or other bodily fluids of infected animals such as chimpanzees, gorillas, fruit bats, monkeys, forest antelope and porcupines found ill or dead or in the rainforest. Ebola then spreads through human-to-human transmission via direct contact (through broken skin or mucous membranes) with the blood, secretions, organs or other bodily fluids of infected people, and with surfaces and materials

(e.g. bedding, clothing) contaminated with these fluids. Health-care workers have frequently been infected while treating patients with suspected or confirmed EVD. Burial ceremonies in which mourners have direct contact with the body of the deceased person can also play a role in the transmission of Ebola. Ebola is not spread through the air, by water, or in general, by food. However, in Africa, Ebola may be spread as a result of handling bushmeat (wild animals hunted for food) and contact with infected bats. There is no evidence that mosquitoes or other insects can transmit Ebola virus<sup>1,2</sup>. WHO has estimated a mean incubation period for the first 9 months of the current West African outbreak as 11.4 days, with an upper limit (95% confidence) of 21 days<sup>3</sup>. First symptoms are the sudden onset of fever, fatigue, muscle pain, headache and sore throat. This is followed by vomiting, diarrhoea, rash, symptoms of impaired kidney and liver function, and in some cases, both internal and external bleeding (e.g. oozing from the gums, blood in the stools). Laboratory findings include low white blood cell and platelet counts and elevated liver enzymes. It can be difficult to distinguish EVD from other infectious diseases such as malaria, typhoid fever and meningitis.

Supportive care-rehydration with oral or intravenous fluids- and treatment of specific symptoms, improves survival. There is as yet no proven treatment available for EVD. However, a range of potential treatments including blood products, immune therapies and drug therapies are currently being evaluated. No licensed vaccines are available yet, but 2 potential vaccines are undergoing human safety testing.

People remain infectious as long as their blood and body fluids, including semen and breast milk, contain the virus. Those who have recovered from the disease can still transmit the virus through their semen for up to 7 weeks after recovery from illness. Good control on outbreak relies on applying a package of interventions, namely case management, surveillance and contact tracing, a good laboratory service, safe burials and social mobilization. Community engagement is key to successfully controlling outbreaks. Raising awareness of risk factors for Ebola infection and protective measures that individuals can take is an effective way to reduce human transmission.<sup>1,3</sup>

## References

1. Ebola virus disease. Fact sheet. Sept 2014. WHO. <http://www.who.int/media/centre/factsheets>
2. Ebola virus. <http://www.virology.ws/2014>
3. Transmission. Ebola hemorrhagic fever/ CDC. <http://www.cdc.gov/vhf/ebola/transmission>

## HBeAg NEGATIVE CHRONIC HEPATITIS B IN BANGLADESHI PATIENTS

Majid F<sup>1#</sup>, Moben AL<sup>2</sup>, Jahan M<sup>3</sup>, Hussain D<sup>4</sup>, Tabassum S<sup>3</sup>

<sup>1</sup>Department of Microbiology, Tairunnessa Memorial Medical College, Gazipur; <sup>2</sup>Department of Medicine, Shaheed Suhrawardy Medical College Hospital, Dhaka; <sup>3</sup>Department of Virology, Bangabandhu Sheikh Mujib Medical University, Dhaka; <sup>4</sup>Department of Physiology, Kumudini Women's Medical College, Tangail;

### Abstract

Hepatitis B virus (HBV) causes a spectrum of liver diseases including acute hepatitis, chronic hepatitis, liver cirrhosis, and hepatocellular carcinoma. Hepatitis B e antigen negative chronic hepatitis (e-CHB) with detectable levels of HBV DNA in serum has been reported in cases from Asia. Outcome and treatment also influenced by the HBeAg status among these patients. The present study was designed to evaluate the HBeAg status of CHB patients. A total of 200 serologically diagnosed CHB patients were enrolled for the study. Data were analyzed by SPSS for windows. Among the total study subjects, HBeAg positive CHB patients were 74 (37%) and negative patients 126 (63%). Of the HBeAg negative patients viral load was significantly lower and they were significantly older. The mean viral load of HBeAg positive and HBeAg negative was  $6.40 \pm 2.042$  [ $\log_{10}(\text{copies/ml})$ ] and  $2.83 \pm 2.55$  [ $\log_{10}(\text{copies/ml})$ ] respectively ( $p < 0.001$ ). HBV DNA found to be more reliable indicator of the presence of virus than HBeAg, and was detected in 98.65% (73/74) HBeAg positive carriers, and in 66.67% (84/126) HBeAg negative patients. HBeAg negativity is more prevalent among the CHB patients in Bangladesh.

**Key Words:** HBV DNA, CHB, HBeAg, Real time PCR.

### Introduction

Globally, over 2 billion people are infected with Hepatitis B virus (HBV) and 370 million people are living with chronic HBV. Among them, around 660,000 die annually due to consequences of this infection.<sup>1</sup> Bangladesh is a densely populated country with intermediate endemicity for chronic hepatitis B (CHB) infection.<sup>2</sup> Studies have shown that HBV is responsible for 31.25% cases of acute hepatitis, 76.3% cases of chronic hepatitis, 61.15% cases of cirrhosis of liver and

33.3% cases of hepatocellular carcinoma in Bangladesh.<sup>3-5</sup> The hepatitis B surface antigen (HBsAg) positivity among the healthy adult population of Bangladesh was 7.2%-7.5%.<sup>6-7</sup> The natural history of chronic hepatitis B is dependent on the age of acquiring hepatitis B infection. Serologic assays for HBV is the mainstay diagnostic tool for HBV infection. However, the advent of molecular biology-based techniques has added a new dimension to the diagnosis and treatment

### #Address for Correspondence

Dr Farjana Majid, Associate Professor (CC), Department of Microbiology, Tairunnessa Memorial Medical College, Konia, Board Bazar, Gazipur-1704. E-mail: farjana\_dr28@yahoo.com.



of patients with chronic HBV infection.<sup>8</sup> Viral load tests that quantify HBV in peripheral blood i.e., serum or plasma are currently the most useful and most widely used. High-sensitivity molecular assays are clearly important for the diagnosis of HBeAg negative CHB and occult HBV, where viral loads can be quite low.<sup>9</sup>

HBeAg-negative CHB is recognized as an important form of chronic hepatitis, where HBeAg negativity is due to mutations in pre-core and core-promoter regions.<sup>10</sup> In HBeAg-negative Asian CHB patients, 45-57% have pre-core mutations and 41-70% have core promoter mutations.<sup>11,12</sup> Most studies believe that in Asian carriers, precore mutations are not responsible for disease progression,<sup>13</sup> whereas, core promoter mutations may have some role in the development of cirrhosis-related complications.<sup>13,14</sup> Around 50% and 70% of patients clear HBeAg within 5 years and 10 years of diagnosis respectively.<sup>15</sup> In general, patients who clear HBeAg have a better prognosis than patients who remain HBeAg positive for prolonged periods of time.<sup>16</sup> It was thought that seroconversion from HBeAg to HBeAb is accompanied by cessation of HBV replication and remission of liver disease. The prevalence of HBeAg negative CHB were 39.7 % to 51.3% from Bangladesh,<sup>17,18</sup> whereas, in Iran, Hong Kong and Korea the prevalence were 65%, 69% and 19.6% respectively.<sup>19,11,20</sup> In Europe, the prevalence of HBeAg negative variants in patients with CHB were 72%- 90% from France, Italy and Greece.<sup>21-23</sup> The present study was undertaken to evaluate HBsAg status of CHB patients and determine viral count in the plasma.

### Materials and Methods

This cross sectional study was carried out among chronic HBV infected patient during the period of July 2010 to June 2011. The study population consisted of 200 serologically diagnosed chronic hepatitis B patients. Collection of specimens and laboratory work was carried out in the Department of Virology, Bangabandhu Sheikh Mujib Medical

University (BSMMU). The subjects were recruited by non probability convenience sampling method. Blood sample was collected using aseptic venipuncture technique. Approximately 5 milliliter venous blood was collected and taken into a EDTA containing tube. The HBV DNA was quantitated with a commercially available Kit (Robo Gene HBV DNA Quantification Kit, Lot-009, Germany) following manufacturer's protocol. Samples were screened for HBeAg using ELISA Kit (Bio-Quant, Inc. UK). Results were expressed as mean $\pm$ standard deviation or number (percentage) as appropriate. HBV-DNA value was expressed as log<sub>10</sub> conversion value. Fisher's Exact and unpaired Student's t-test were performed to calculate statistical difference between groups. Statistical analyses were carried out using software SPSS for Windows Version 17.0. A p value <0.05 was considered as level of significance.

### Results

In the present study 200 CHB patients were included and of them male female ratio was 3.76:1. Mean $\pm$ SD age (yrs) was 32.05 $\pm$ 12.99 (range: 7 - 65). Among the total subjects 74 (37%) were HBeAg positive and 126 (63%) HBeAg negative. Of the total subjects HBV DNA (by Real time PCR) level was 4.15 $\pm$ 2.6 and ALT (U/L) was 83.63 $\pm$ 146.23 in the two groups (Table 1).

The study subjects were subdivided into two groups on the basis of age  $\leq$  40 and  $>$  40 years. Among the total CHB patients 153 (76.50%) were below 40 years of age. Of them, 63 (41.18%) were HBeAg positive and the remaining 90 (58.82%) negative. Among the total CHB subjects 47 (23.50%) were above 40 years. In this group, majority 36 (76.60%) were HBeAg negative, while 11 (23.40%) HBeAg positive. The HBeAg status in relation with age was shown in table 2. HBeAg positivity in younger age group was relatively higher than older age group which was statistically significant ( $p < 0.05$ ).

HBV DNA and ALT levels in relation to HBeAg status was shown in table 3. Among the total study subjects, HBeAg positive CHB cases were 74 (37%) and negative 126 (63%). Among all the HBeAg positive and negative patients, mean viral load was  $6.40 \pm 2.04$  [ $\log_{10}$  (copies/ml)] and  $2.83 \pm 2.55$  [ $\log_{10}$  (copies/ml)] respectively. Viral load of HBeAg positive cases was significantly higher compared to the HBeAg negative cases ( $p < 0.00$ ). The mean ALT level was  $134.35 \pm 193.03$  (U/L) for HBeAg positive patients and  $53.83 \pm 99.33$  (U/L) for HBeAg negative patients. Overall, HBV DNA and ALT level were significantly higher in HBeAg positive patients ( $p < 0.05$ ).

**Table 1:** Baseline characteristics of study population

Variables	Values
Number of patients	200
Age (yrs)	$32.05 \pm 12.99$
Gender (male: female)	3.76 : 1
HBeAg (Positive/Negative)	74/126
Serum ALT (U/L)	$83.63 \pm 146.23$
HBV DNA load [ $\log_{10}$ (copies/ml)]	$4.15 \pm 2.93$

Data were expressed as mean  $\pm$  SD, number and ratio as appropriate.

**Table 2:** Relation between age and HBeAg status

Age Group	Subjects with CHB N (%)	HBeAg status		P value
		Positive N (%)	Negative N (%)	
$\leq 40$ yrs	153 (76.5)	63 (41.18)	90 (58.82)	0.037
$> 40$ yrs	47 (23.5)	11 (23.40)	36 (76.60)	
<b>Total</b>	200 (100)	74 (37)	116 (63)	

Data were expressed as number (percent). Fisher's Exact test was performed.  $P < 0.05$  was taken as level of significance.

**Table 3:** HBV DNA by Real time PCR and ALT levels on the basis of HBeAg status

Variable	HBeAg status		P value
	Positive (n = 74)	Negative (n = 126)	
HBV DNA [ $\log_{10}$ (copies/ml)]	$6.40 \pm 2.04$	$2.83 \pm 2.55$	$< 0.001$
ALT (U/L)	$134.35 \pm 193.03$	$53.83 \pm 99.33$	$< 0.001$

Data are expressed as Mean  $\pm$  SD. Unpaired Student's t-test was performed to evaluate statistical difference between groups.  $P < 0.05$  was taken as level of significance.

## Discussion

An estimated 350 million individuals in the world have chronic HBV infection. Although most of them are HBeAg negative. HBeAg positivity is highly prevalent only in younger age groups of HBsAg carriers.<sup>24,25</sup> In a recent community-based studies from different parts of the world, the prevalence of HBeAg negativity in chronic HBV infection has been found to range between 70% and 100%.<sup>26-30</sup> The loss of HBeAg is usually associated with biochemical and histologic remission of hepatitis and with significant suppression in HBV replication.<sup>24,31</sup> Thus, the great majority of HBeAg-negative subjects have normal ALT levels and undetectable serum HBV DNA by the classic hybridization methods. However, with very sensitive techniques as the polymerase chain reaction (PCR) and the nested PCR assay, residual amounts of HBV DNA can be detected in the serum of most HBeAg-negative subjects.<sup>32</sup>

The present study observed a significantly higher age among HBeAg negative patients (Table 2). Another study shows that, the age of patients with HBeAg-negative CHB ranges between 40 and 55 years.<sup>33</sup> A study from the Department of Hepatology, BSMMU also reported similar findings.<sup>2</sup> Although previous

studies reported that only a few countries had more HBeAg-negative than HBeAg-positive CHB patients,<sup>24</sup> It is now apparent that there is a worldwide increase in the prevalence of HBeAg-negative CHB. In Italy, 41% of patients with CHB were HBeAg negative during the period 1975 to 1985, but in the last decade this increased to 90%.<sup>34</sup> Due to mutation in core promoter and precore regions, HBeAg negativity occur in CHB patients, which decrease or prevent the synthesis of HBeAg but do not impair viral replication.<sup>35</sup> In the present study, prevalence of HBeAg negative cases was 63% (Table 2). A study from the Department of Virology, BSMMU in 2010 done found 88.57% of HBeAg negative CHB. Studies from the Department of Hepatology, BSMMU found 51.3% HBeAg negative CHB<sup>36</sup> and 39.7% HBeAg negative CHB.<sup>37</sup> These results indicate a significant increase of HBeAg negative CHB among Bangladeshi population. This probably indicates that the majority of HBV positive patients in our region has been infected for a long time and has developed mutations in the pre-core region.<sup>38</sup>

The mean viral load of HBeAg positive and HBeAg negative patients in the present study was  $6.40 \pm 2.04$  [ $\log_{10}$  (copies/ml)] and  $2.83 \pm 2.55$  [ $\log_{10}$  (copies/ml)] respectively. Previous studies show that in comparison to HBeAg positive CHB patients, HBeAg negative patients have lower serum HBV DNA and have more advanced disease as evidenced by liver histology<sup>39</sup>. Similar result was also observed in the present study. A study from Iran showed that HBV DNA levels were higher in HBeAg-positive patients, where 87% patients were negative for HBeAg.<sup>40</sup> In a study from Korea, it was observed that the median serum HBV DNA for HBeAg negative patients was approximately two log lower than HBeAg positive patient, regardless of ALT level.<sup>41</sup> Lower alanine aminotransferase levels was reported among

HBeAg negative patients from Italy.<sup>42</sup> A 6 years study conducted by the Department of Hepatology, BSMMU showed, ALT and HBV DNA levels were significantly lower in HBeAg negative subjects.<sup>2</sup> Another study from Bangladesh showed high DNA load in 96% HBeAg positive patients compared to only 54.1% among HBeAg negative patients.<sup>43</sup> In the study, HBV DNA and ALT level were also significantly lower in HBeAg negative patients (Table 3). Another study showed the viral load in HBeAg-positive patients was higher than in HBeAg-negative individual<sup>38</sup>. The mean ALT was significantly higher in HBeAg-positive than in HBeAg-negative patients, which could be due to a higher degree of inflammation.

## Conclusions

The present study demonstrated that, HBeAg negativity is more prevalent among the CHB patients in Bangladesh. Careful evaluation for the histological activity of HBV, functional status of the liver and presence of cirrhosis or hepatocellular carcinoma in HBeAg negative patient may be helpful for their further treatment. Patients who were HBeAg negative had lower viral load and were significantly older. ALT level was raised in patients with detectable HBV DNA. However, this study was limited by lack of determining the frequency of precore/core promoter mutation among HBeAg negative CHB patients. This aspects need to be evaluated further in future studies with large number of CHB patients.

## References

1. CDC (2008). Recommendations for Identification and Public Health Management of Persons with Chronic Hepatitis B Virus Infection. National Notifiable Diseases Surveillance System, [www.nastad.org/ Docs/ Public/... ./20081013\\_Beckett %20 Testing.pdf](http://www.nastad.org/Docs/Public/.../20081013_Beckett%20Testing.pdf)

2. Alam S, Ahmad N, Mustafa G, Alam K, Khan M. Characteristics of treatment naive chronic hepatitis B in Bangladesh: Younger populations are more effected; E antigen negatives are more advanced. *Saudi J Gastroenterol*. 2008; **14**:15-19.
3. Mahtab MA, Rahman S, Karim MF, Khan M. Epidemiology of hepatitis B virus in Bangladeshi general population. *Hepatobiliary Pancreat Dis Int*. 2008; **7**: 595-600.
4. Afroz S, Mahtab MA, Rahman S, Khan M. Hepatitis B virus is the leading cause of cirrhosis of liver in Bangladesh. *Hepatol Int*. 2007; **1**: 120.
5. Khan M, Zaki KMJ, Ahmed KU. Clinical profile: Prognostic index in hepatocellular carcinoma. *BMRC Bull*; XVII 1991: 49-62.
6. Islam MN, Islam KM, Islam N. Hepatitis-B virus infection in Dhaka, Bangladesh. *BMRC Bull* 1984; **10**: 1-6.
7. Khan M, Ahmad N. Seroepidemiology of HBV and HCV in Bangladesh. *Int Hepatol Comm*. 1996; **5**: 27-29.
8. EASL Clinical Practice Guidelines. Management of chronic hepatitis B. *J Hepatol*. 2009; **50**: 227-242.
9. Alexandra V. Molecular Testing in the Diagnosis and Management of Chronic Hepatitis B. *Clin Microbiol Rev*. 2007; **20** **3**: 426-439.
10. Brunetto MR, Stemler M and Schodel F. Identification of HBV variants which cannot produce precore derived HbeAg and may be responsible for severe hepatitis. *Ital J Gastroenterol*. 1989; **21**: 151-4.
11. Chan HL, Leung NW, Hussain M, Wong ML, Lok AS. Hepatitis B e antigen-negative chronic hepatitis B in Hong Kong. *Hepatology*. 2000; **31**: 763-768.
12. Yuen MF, Sablon E and Yuan HJ. Relationship between the development of precore and core promoter mutations and hepatitis B e antigen seroconversion in patients with chronic hepatitis B virus. *J Infect Dis*. 2002; **186**: 1335- 1338.
13. Yuen MF, Tanaka Y and Mizokami M. Role of hepatitis B virus genotypes Ba and C, core promoter and precore mutations on hepatocellular carcinoma: a case control study. *Carcinogenesis*. 2004; **25**: 153-1598.
14. Liu CJ, Chen BF and Chen PJ. Role of hepatitis B virus precore core promoter mutations and serum viral load on noncirrhotic hepatocellular carcinoma: a case-control study. *J Infect Dis*. 2006; **194**: 594-599.
15. Fattovich G. Natural history and prognosis of hepatitis B. *Semin Liver Dis*. 2003; **23**: 47-58.
16. Merican I, Guan R, Amarapuka D, Alexander M, Chutaputti A, Chien R, *et al*. Chronic hepatitis B virus infection in Asian countries. *J Gastroenterol Hepatol*. 2000; **15**: 12, 1356-1361.
17. Ahmad N, Alam S, Mustafa G, Adnan AB, Baig RH, Khan M. e-antigen-negative chronic hepatitis B in Bangladesh. *Hepatobiliary Pancreat Dis Int*. 2008; **7**: 379-382.
18. Mahtab MA, Rahman S and Karim F. Clinical and histopathological characterization of asymptomatic hepatitis B virus positive subjects in Bangladesh. *Acta Hep Japonica*. 2008d; **49**: 98.

19. Yosefirad M, Malekzadeh R and Khatibian M. Prospective controlled trial of interferon alpha-2b in Iranian patients with chronic hepatitis B. *Gastroenterol.* 1997; **112**: 1420.
20. Yoo BC, Park JW, Kim HJ, Lee DH, Cha YJ, Park SM. Precore and core promoter mutations of hepatitis B virus and hepatitis B e antigen-negative chronic hepatitis B in Korea. *J Hepatol.* 2003; **38**: 98-103.
21. Zarski JP, Marcellin P, Leroy V, Trepo C, Samuel D, Ganne-Carrie N. Characteristics of patients with chronic hepatitis B in France: predominant frequency of HBe antigen negative cases. *J Hepatol.* 2006; **45**: 355-360.
22. Lampertico P, Del Ninno E, Manzin A, Donato MF, Rumi MG, Lunghi G. A randomized, controlled trial of a 24-month course of interferon alfa 2b in patients with chronic hepatitis B who had hepatitis B virus DNA without hepatitis B e antigen in serum. *Hepatology.* 1997; **26**: 1621-1625.
23. Papatheodoridis GV, Manesis E and Hadziyannis SJ. The longterm outcome of interferon-alpha treated and untreated patients with HBeAg-negative chronic hepatitis B. *J Hepatol.* 2001; **34**: 306-313.
24. Hadziyannis SJ. Hepatitis B e-antigen negative chronic hepatitis B: from clinical recognition to pathogenesis and treatment. *Viral Hepat Rev* 1995; **1**: 7-36.
25. Lok AS, Lai CL, Wu PC, Leung EK, Lam TS. Spontaneous hepatitis B e-antigen to antibody seroconversion and reversion in Chinese patients with chronic hepatitis B virus infection. *Gastroenterology* 1987; **92**: 1839-1843.
26. Knoll A, Rohrhofer A, Kochanowski B, Wurm EM, Jilg W. Prevalence of precore mutants in anti-HBe-positive hepatitis B virus carriers in Germany. *J Med Virol* 1999; **59**: 14-18.
27. Chan HL, Leung NW, Hussain M, Wong ML, Lok AS. Hepatitis B e antigen-negative chronic hepatitis B in Hong Kong. *Hepatology* 2000; **31**: 763-768.
28. Chowdhury A, Santra A, Chaudhuri S, Ghosh A, Banerjee P, Mazumder DN. Prevalence of hepatitis B infection in the general population: a rural community based study. *Trop Gastroenterol* 1999; **20**: 75-77.
29. Madzime S, Adem M, Mahomed K, Woelk GB, Mudzamiri S, Williams MA. Hepatitis B virus infection among pregnant women delivering at Harare Maternity Hospital, Harare, Zimbabwe, 1996 to 1997. *Cent Afr J Med* 1999; **45**: 195-198.
30. Minuk GY, Orr PS, Brown R, Macdonald S, Chaudhary RK, Temple P. Pre-core mutant infections in the Canadian Inuit. *J Hepatol* 2000; **33**: 781-784.
31. Chu CM, Liaw YF. Natural history of chronic hepatitis B virus infection: an immunopathological study. *J Gastroenterol Hepatol* 1997; **12**: S218-S222.
32. Oketani M, Oketani K, Xiaohong C, Arima T. Low level wild-type and pre-core mutant hepatitis B viruses and HBeAg negative reactivation of chronic hepatitis B. *J Med Virol* 1999; **58**: 332-337.
33. Hadziyannis SJ and Vassilopoulos D. Hepatitis B e Antigen-Negative Chronic Hepatitis B. *Hepatology.* 2001; **34**: 617-623.



34. Rizzetto M, Volpes R and Smedile A. Response of pre-core mutant chronic hepatitis B infection to lamivudine. *J Med Virol.* 2000; **61**: 398-402.
35. Tran TT. Hepatitis B in Asian/Pacific islands: Overview and call to action. *Adv Stud Med.* 2007; **7**: 469-475.
36. Mahtab MA, Rahman S and Karim F. Clinical and histopathological characterization of asymptomatic hepatitis B virus positive subjects in Bangladesh. *Acta Hep Japonica*; 2008b; **49**: 98.
37. Ahmad N, Alam S, Mustafa G, Adnan AB, Baig RH, Khan M. E-antigen-negative chronic hepatitis B in Bangladesh. *Hepatobiliary Pancreat Dis Int.* 2008; **7**: 379-382.
38. Tonetto PA, Neiva SL, Fais V, Aline G, V Eduardo SL, Gonçalves Jr et al. Hepatitis B virus: molecular genotypes and HBeAg serological status among HBV-infected patients in the southeast of Brazil. *BMC Infect Dis.* 2009; **9**: 149
39. Pungpapong S, Kim WR and Poterucha JJ. Natural History of Hepatitis B Virus Infection. An Update for Clinicians. *Mayo Clin Proc.* 2007; **82**: 967-975.
40. Ganji A, Esmaeilzadeh A, Ghafarzadegan K, Helalat H, Rafatpanah H, Ali M. Correlation between HBsAg quantitative assay results and HBV DNA levels in chronic HBV. *Hepat Month* 2011; **11**: 342-345.
41. Heo J, Baik TH, Kim HH, Ha Kim HG, Kang HD. Serum Hepatitis B Virus (HBV) DNA Levels at Different Stages of Clinical Course in Patients with Chronic HBV Infection in an Endemic Area. *J Korean Med* 2003; **18**: 686-90.
42. Sagnelli E, Stroffolini T, Mele A, Imperato M, Almasio PL. Chronic Hepatitis B in Italy: New Features of an Old Disease- Approaching the Universal Prevalence of Hepatitis B e-Antigen-Negative Cases and the Eradication of Hepatitis D Infection. *CID.*
43. Mahtab MA, Rahman S, Kamal M, Khan M. Occult Hepatitis B Virus Related Decompensated Cirrhosis of Liver in Young Males: First Report of Two Cases from Bangladesh. *Hepatitis Monthly.* 2008c; **8**: 147-150.

## PATTERN OF VICTIMS IN FATAL ROAD TRAFFIC ACCIDENT

Kabir MJ<sup>1</sup>, Parvez S<sup>2</sup>, Ahamed BU<sup>3</sup>, Salam F<sup>4</sup>, Hossain ME<sup>1#</sup>

<sup>1</sup>Department of Forensic Medicine & Toxicology, Tairunnessa Memorial Medical College & Hospital;

<sup>2</sup>Department of Anatomy, University Dental College & Hospital; <sup>3</sup>Department of Forensic Medicine and Toxicology, Dhaka Community Medical College; <sup>4</sup>Department of Forensic Medicine and Toxicology, Green Life Medical College and Hospital

### Abstract

Road traffic accident (RTA) is the commonest manner of death which is increasing in all developed and developing countries. This retrospective study, from January 2012 to December 2012, was carried out at the mortuary of Dhaka Medical College to know the patter of victim involved in fatal Road traffic accidents. During this period, total 2547 cases were autopsied and out of this, 136 cases were death were due to RTA. Female deaths were only 14.0% and the remaining 86.0% male. Highest percentage of deaths were found in the age group of 31-40 yrs (26.5%). Regarding the types of occupants, pedestrian were highest percentage (60%) and the occupant of offended vehicle (12.8%). In 12.8% cases the types of occupants had not been mentioned.

**Key Words:** Victims, Vehicles, RTAs

### Introduction

Dhaka, the capital city of Bangladesh, is the most vulnerable city both in terms of total number of accidents and accident rates. Fatal accidents and motor collisions are decreasing while injury accidents are increasing. Most traffic accidents: cause fatality (69%) and hit a pedestrian (60%). I was observed that 54% of the accidents occurred during daytime. The paper recommends improvement measures for the road accident data collection and management system in Dhaka, Bangladesh. That includes the training need for the police staff, need for a geo-referenced database for data storage and management and involvement of multiple agencies in the process.<sup>1-4</sup>

### Materials and Methods

Among the total autopsy cases done at the mortuary of Dhaka Medical College, only victims of road traffic accidents were taken as the study specimen. This study was carried out from 1st January to December 2012, a period of one year, Deaths due to road traffic accidents usually within the region of Dhaka referred to the mortuary to Dhaka Medical College. So, it reflects the deaths due to RTA in Dhaka region. All the deaths, due to RTA were autopsied carefully with specially considering the age group, sex, types of vehicle occupants and the victim is known case or unknown case.

### #Address for Correspondence

Dr Mohammad Emran Hossain, Professor, Department of Forensic Medicine and Toxicology. Tairunnessa Memorial Medical College and Hospital, Konia, Board Bazar Gazipur-1704; E-mail; jubaidul.kabir@yahoo.com.

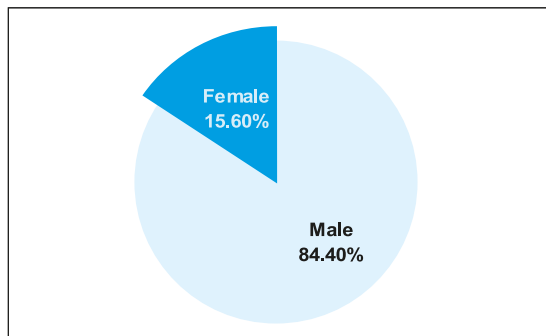
## Result

A total number of 2547 autopsies were performed in Dhaka Medical College in the year 2012. Of the total cases 136 (5.3%) deaths were due to road traffic accident (Table 1).

**Table 1:** Number of RTA cases among total autopsies done in Dhaka Medical college in 2012

Year of Autopsy	Total autopsy done	Death due to RTA	Percentage
2012	2547	136	5.3%

Of the total autopsized cases in 2012 in Dhaka Medical College 84.4% deaths due to RTA victims were male (Figure I).



**Figure I:** Male female ratio of the RTA victims autopsized in Dhaka Medical College in 2012.

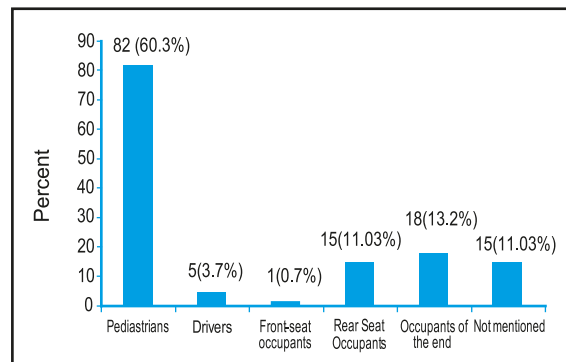
The types of the RTA victims were shown in the figure II. Of the 136 RTA victims 60.3% were pedestrian. About 11.03% were rear seat occupants, 3.7% drivers, 13.2% occupants of the end. In 11.03% of cases passengers sitting position was not mentioned (Figure II).

Among the RTA victims 16 (11.8%) had age up to 20 years. Of the 136 subjects 102 (75%) were between age group 21-50. In the higher age group >50 (yrs) the number was 18 (13.3%) (Table 2).

**Table 2:** Distribution of number of deaths due to RTA on the basis of age

Age group	Number	Percentage
0 - 10	4	2.9%
11 - 20	12	8.9%
21 - 30	35	25.7%
31 - 40	36	26.5%
41 - 50	31	22.8%
51 - 60	8	5.9%
61 - 70	10	7.4%
<b>Total</b>	<b>136</b>	<b>100%</b>

Data were expressed as number (percent)



**Figure II:** Distribution of the RTA victims on the basis of the types of the subjects affected.

## Discussion

The study was undertaken to know about the pattern of victims which are responsible for deaths in RTAs autopsied at the Dhaka Medical College mortuary. The highest incidence of RTAs are due to the fault of the victims, drivers, vehicles, bad road condition and/or wrong traffic signaling.<sup>4</sup>

Not surprisingly our study shows the overwhelming majority of the deceased (84.4%) were males. It is due to greater male exposure on urban streets and similar higher incidence of traffic accidents among males has been found by many other researchers.<sup>5-10</sup> The maximum 79.6% victims were within the age of 21 to 30 years



(30.4%) as they had to face RTAs for their daily works such as school, college, office, business and other activities which is similar to the result of Rahman *et al.*<sup>11-14</sup> Rear & Back seat occupant are more affected as they don't use seat belt and the forehead may strike the back side of the top of the front seat and the knee may strike the back of the front seat.<sup>4</sup> As it is Muslim country Muslim people are mostly affected (93.6%).

The study also showed that the pedestrians are the most common (60%) victims. These may be due to lack of knowledge of traffic rules, and addiction or visual defect of the victims and poor road condition, wrong signaling and addiction and reckless driving by the driver.

Of all these, factors related to the responsibility of the driver and the pedestrian victims have been included under the preview of the present discussion. Apart from the lack of driving skill and knowledge of the mechanism of the vehicle, some other defects or short comings of the driver of the offender vehicle may be best considered by a doctor. These are - whether the driver was intoxicated, because alcohol adversely affects the driving capacity. Due to its effect visual acuity, assessment of the position of another vehicle or the road turnings may be wrong. Due to loss of acuity of color vision signals on roads may be wrongly interpreted. Due to diminished alertness, the driver may not act immediately or properly when, say for example, all on a sudden he faces an acute curve or another vehicle in front. Blurring of the sensory perception may result in over-pressing of the accelerator and over-oscillation of the steering wheel. If the reflex response is delayed, then the driver cannot react in time in emergency circumstances. Safe driving needs quick judgment & action. Over confidence makes the driver reckless and rough & rash driving is the result which increases the risk of accident. For drivers, different countries have specified upper permissibly limit of blood alcohol. But in our country there is nothing specific in this regard. To assess the blood level of alcohol of drivers, road side test of expired air of driver is

possible with the help of breath analyzer or alco meter. These factors also act in pedestrian victims who meet with an accident. Hence, in resolving a case of compensation, or in the criminal side of a vehicular accident case, the role of a doctor is definitely more than conduction of post mortem examination in a perfect way.<sup>4</sup>

No significant variation was evident in the incidence of fatal vehicular accidents by days of a week in our study. This pattern differs from earlier study conducted in Delhi according to which highest numbers of accidents were on Saturdays. In the study conducted in Nepal highest numbers of vehicular accidents were observed on Sundays and lowest on Mondays.<sup>15,16</sup> National injury Mortality Surveillance System (2004) reported that most of the transport related deaths occurred on Saturday (20.8%) followed by Sunday (17.1 %).<sup>17</sup>

Maximum number of fatal accident took place in November months (11.04%) in present study. In Nepal, maximum numbers of cases were reported in July followed by January.<sup>16</sup> In the earlier studies conducted in Delhi reported maximum numbers of victims were seen in January month.<sup>15,18</sup> National Crime Record Bureau (2005) has reported higher incidence of road accidents during May (10.3%) and March (9.3%) in India with the peak time between 3 PM to 6 PM<sup>19</sup>. In the studies conducted in Mangalore and Katmandu (Nepal) most of the accidents had taken place during the afternoon and evening hours. In our study maximum incidence of vehicular accidents are reported in evening hours.<sup>20,20</sup> This difference in the peak is quite suggestive that fatal vehicular accidents had different temporal correlation with time, day and month as compared to nonfatal vehicular accidents.

## Conclusions

It is concluded that male is the frequent victim of the road traffic accidents and 84% of the cases belong to age group 21-50 years. Pedestrians are relatively common (60%) victims to RTA.

### Recommendations

1. Have to improve the road condition and signaling systems.
2. Have to use seat belt by the driver and occupants and periodic follow-up system for the fitness of vehicle as well as the drivers.
3. Finally the government has to aware the people on traffic education through visual and audiovisual medias and by inclusion of traffic education in school level syllabus.

### References

1. Mallik CC. A short textbook of Medical Jurisprudence. 3<sup>rd</sup> ed. Calcutta; 1993. 513-18.
2. Matheharam K, Patnaik AK. MODI's Medical Jurisprudence and Toxicology. 23<sup>rd</sup> ed. New Delhi: Lexis Nexis Butterworths; 2005. 783-93.
3. Mallik CC. A short textbook of Medical Jurisprudence. 3<sup>rd</sup> ed. Calcutta; 1993. 513-18.
4. Nandy A. Principles of Forensic Medicine. 1st ed. Calcutta: Apurba Nandi, 1995, 286-90.
5. Sagado MSL, Colombage SM. Analysis of fatalities in road accidents. *Forensic Sci Int* 1998; **36**: 91-96.
6. Sahdev P, Lacqua MJ, Singh B, Dogra TD. Road Traffic fatalities in Delhi: causes, injury patterns and incidence of preventable deaths. *Accid Ann Prev* 1994; **26**: 377-84.
7. Friedman Z, Kungel C, Hiss J, Margovit K, Stein M, Shapira S. The abbreviated injury scale-valuable tool for forensic documentation of trauma. *Am J Forensic Med Pathol* 1996; **17**: 733-38.
8. Henriksson EM, Ostrom M, Eriksson A.. Preventability of vehicle-related fatalities. *Accid Ann Prev* 2001; **33**: 467-75.
9. Sharma BR, Harish D, Sharma V, Vij K. Road Traffic accidents - a demograph and topographic analysis. *Med Sci Law* 2001; **41**: 266-74.
10. Jha N, Agrawal CS, Epidemiological Study of Road Traffic Accident Cases: A Study from Eastern Nepal. *Regional Health Forum WHO South-East Asia Region*, 2004; **8**: 15-22.
11. Rahman MA, Chowdhury MAS, Ahmed M, Siddiqua SNA. Pattern of injuries in death due to road-traffic accidents (RTA) autopsied at Mymensingh Medical College. *Mymensingh Med J* 2000 January; **9**: 12-14.
12. Masson JK. The Pathology of violent injury. 1st Eed. New York; 1978. 5-10.
13. Reddy KSN. The essentials of Forensic Medicine & Toxicology. 12th ed, Hyderabad: K. Suguna Devi. 1994. 129-32.
14. Knight B. Forensic Pathology. 1st ed. London: Edward Arnold; 1996. 252-53.
15. Mehta SP. An epidemiological study of road traffic accident cases admitted in Safdarjang Hospital, New Delhi. *Ind Med Res* 1968; **56**: 456-66.
16. Jha N, Agrawal CS, Epidemiological Study of Road Traffic Accident Cases: A Study from Eastern Nepal. *Regional Health Forum WHO South-East Asia Region*, 2004; **8**: 15-22.
17. A profile of fatal injuries in South Africa. 6th Annual Report of the National Injury Mortality Surveillance System 2004. <http://www.mrc.ac.za/crime/national2004.pdf>. Accessed on 15 May 2014.
18. Singh R, Bhatnager M, Singh HK, GP Singh, Kumar Y. An epidemiological study of victims of road traffic accidents cases: a study from national capital region (Ghaziabad), India. *Indian J Prev Soc Me* 2011; **42**: 28-33.
19. NCRB (2005). National crime records bureau, Ministry of Home Affairs, Govt. of India.
20. Menon A, Pai VK, Rajeev A. Pattern of fatal head injuries due to vehicular accidents in Mangalore. *J For Leg Med* 2008; **15**: 75-7.
21. Banthia P, Koirala B, Rauniyar A, Chaudhary D, Kharel T, Khadka SB. An epidemiological study of road traffic accident cases attending emergency department of teaching hospital. *J Nepal Med Assoc* 2006; **45**: 238-43.

## LEARNER BASED EDUCATION

Kabir J

*Department of English, Tairunnessa Memorial Medical College & Hospital*

---

### Abstract

Teaching methodology is one of the most important aspects of teaching. Traditionally we have an education system which is teacher based where the teacher is the focus of all the activities. The pedagogy in the 1990s came up with the idea of learner based education. In this kind of education learner is the main focus and all attention is concentrated on the learner. The subjects that are taught are as complex as life and also larger than life. The teacher needs to create a space where he or she can teach and thus communicate with the learner and the subject. In order to do so a teacher must be fully equipped with the knowledge of the subject which can never be complete. In my paper I will try to show how this problem can be solved by introducing learner based education model where students participate in such a way that they become the resource and share their knowledge with each other.

Teaching is an art which is of vital importance in grooming up children to become good and competent human beings. So that they are ready to encounter the ever changing world with pride and confidence. There are three important components which are intimately correlated in teaching, these are the teacher, the learner and the subject that is being taught. Teaching and learning are crucial for our individual and collective survival.

The subject that is being taught is as large and complex as life itself. So the knowledge about them can never be complete, and the students are even larger than lives. To bring about coordination among all of these factors is a task a teacher has to do on a day to day basis. Teaching is something that comes from within. The act of teaching requires an inner journey of the being intellectually, emotionally and spiritually. None of which can be ignored nor can they be used independently. For, if teaching is reduced to intellect, it becomes an abstraction, if it is reduced to emotions it becomes narcissistic, if it is, on the other hand, reduced to spiritual then it loses its root to the world. A good teacher means a lot of things put together. According to Dr Parker J Palmer, an educationist

'Good teaching comes from within, we teach who we are, and teaching holds a mirror to our souls.' He also mentions that the knowledge on the subject depends mainly on self knowledge. Once a teacher knows himself he is surely to know his students and the subject he teaches. A good teacher joins himself and his students along with the subject in the very fabric of life.

---

### Address for Correspondence

*Jackie Kabir, Assistant Professor, Department of English, Tairunnessa Memorial Medical College and Hospital.*

## Introduction

Traditionally our system of education is teacher centered where the teacher is an expert who delivers a set of ideas to his students or learners. Learners in turn learn these ideas and replicate them. This makes the teaching very predictable and often learners lose heart in grasping what they are taught. Hence the teacher also loses heart, as he can't get through to his learners. Our education system is full of paradoxes, paradoxes that govern our teaching learning methodology and this gives the lifeless results; for example

When the head is separated from the heart the result is that minds do not know how to feel and hearts do not know how to think. When facts are separated from feelings, it results in bloodless facts that make the world distant and remote. Similarly theory separated from practice results in theories that have little to do with real life. When we separate teaching from learning we get teachers who talk but do not listen and students who listen but do not talk. What we need to do here is an integrated teaching- learning method so that we embrace a view of the world in which opposites are joined, so that the whole picture is seen clearly.

However there is another method, which is available in recent times where the students are put at the center of their own learning. So that they carry out the discussion, research and come to a conclusion by themselves. The teacher acts as a facilitator in this model. This puts the responsibility of learning on the students. Here students are engaged in creating, understanding and exercising their control over their learning. All that the teacher does here is the planning and assessing of the students and assigning them the tasks according to their capabilities. Hence- in this kind of education system the teacher becomes a co-learner rather than the sole instructor.

This model of learner-centered education is not a new one. Emphasis on the learners have been put by

two most prominent educators of ancient times. They were Confucius and Socrates. Confucius stressed on good citizenship and character whereas Socrates put the stress on the individual. Educators like John Locke (1632-1774) and Jean Jacques Rousseau (1776-1778) talked about experience based education. For them, the child's mind is like a clean slate where the taught material would be imprinted. A century later, nineteenth century educator Colonel Francis Parker brought this method to America where he demonstrated learner-centered techniques to the teachers. By replacing drill with inquiry activities, Parker replaced memorization of facts with understanding. Twentieth century educator Jhon Dewey introduced this learner based education into a new program called 'constructivism' which is a way of learning in which the learners activities construct or build new concepts or ideas. So, it is understood that learner based education has been developing for the last five thousand years.

The main aspects of learner based education are that in this kind of education teaching is based on experience. John Locke believed that the only way an individual can learn is by doing it in practice. There is an old Chinese proverb which says

'Tell me and I will forget,

Show me and I will remember

Involve me and I will understand.'

This is what really the learner centered education is all about. John Dewey is known by his expression 'learning by doing.' Some students will learn very quickly and others will take a lot of time to do the same task. So in this kind of education the teacher has to assess the students before planning the curricula. So the task of the teacher involves personalizing the topic to meet each student's need. This Harvard based American psychologist introduced the theory of multiple intelligence which puts emphasis on identifying individual talents and aptitude.

The learner based education requires the teacher to make an effort to identify each students' past experience. If that is taken into account while the teacher plans the curricula, the students feel confident about learning. However, if the teacher humiliates the student about their past mistakes then the student feels discouraged to seek help in future work. Consequently, teachers who makes no effort to adapt their instructional strategies to the knowledge and experiences of their students are implicitly dismissing what learners can contribute in the learning process.

According to Locke, curiosity is the engine to learning. In learner-based education the learners' curiosity is fed and nurtured. He also advised that teachers should answer the learners queries and that the teacher should listen to the student's thoughts not to his words.

It is worth mentioning the learning cycle here. It shows that there are three stages of learning

Information in → 'Information storage → ' Information out

Once the information is given to the learner it should be stored so that it can be used to perform a task and to solve problems.

### Techniques used by Learner Based Education

Good teaching cannot be reduced to technique; good teaching comes from the identity and the integrity of a teacher. By the word identity it is mostly meant the surrounding where the teacher grew up, his genetic makeup, the culture he lived in. Identity lies in the intersection of diverse forces that make up his life. Integrity is the combination of those forces which bring about the wholeness in life rather than fragmentation and death.

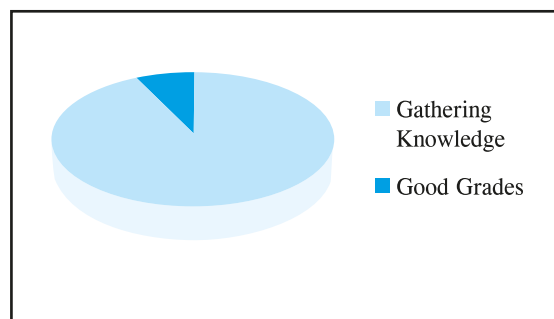
But there is a need to have some planning as to how a teacher is to carry out his lessons. Some of the techniques used by the learner-centered education are:

1. Role play
2. Pair work
3. Individual student's research and discovery
4. Brainstorming
6. Mind-mapping
7. Making use of open-ended questions
8. Cooperative learning groups

Through a learner-centered educational practice, each learner can construct his or her own understanding by tying new information to prior experiences while in the teacher-centered education, students merely become passive listeners and automated responders. It was found that when students discussed among themselves about a problem, they were able to talk to each other through to the solutions.

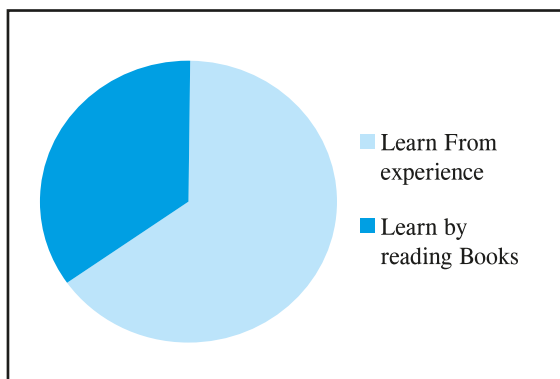
I carried out a survey on 100 students to find out what they thought about the learner focused education system.

The results were like this: 69.23% students preferred lecture to discussion, 76.92% opted for demonstration to theory, 65.38% believed that students' experience in learning is more effective than reading books, 92.31% believed that gathering knowledge is more important than getting good grades. In case of classroom scenario they the students divided as 57.7% wanted their classrooms to be that of formal and 42.31% wanted it to be informal classroom. On the issue of teacher being a resource person 50% of the students supported while the other 50% opined that they should be co learners.

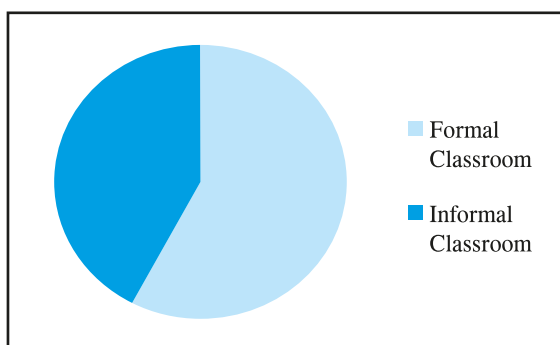


In this pie chart we see that 69.23% of the students preferred discussion to lecture.

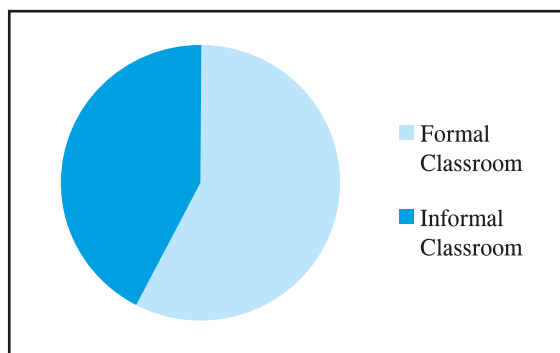




Regarding students studying for gathering knowledge is about 92.31% whereas only 7.69% study to get good grades.



In this Pie chart we see that 65.38% students think that learning from experience is more effective than learning by reading books.



57.7% students prefer formal classrooms whereas 42.31% prefer informal classrooms.

At first when students were asked about it they opted for teacher-focused classroom, as they are more familiar with that kind of classrooms. But as I explained the situation to them they thought that it would be a good idea to have more student friendly environment in classrooms. Most of the students however maintained that initially they depend on lecture based and gradually they become self-dependent learners.

While talking to teachers I found that most of them want to have a learner based teaching system while keeping the lecture part intact. According to some teachers students would not be going in the right direction if they were not introduced to the topic by the expert himself. Then the students can form groups of six or seven to discuss about a certain topic and then come to a conclusion.

In Bangladesh there are some hindrances in following the learner based education system as the number of students are too large sometimes up to 50 or even 60 in each class. Then in some class rooms the furniture are fixed; making it impossible to form groups. Lack of infrastructure in a common problem in Bangladeshi class rooms, the duration of class. Everyday five to six classes, each of which may be of 40 to 45 minutes duration may not suffice to impart lessons in the prescribed learner based education. The attitude which are feelings, beliefs or values which influence the way a student behaves is also important in introducing this kind of pedagogy. Even then we can say that independent learning is being used in many institutions but it is being done gradually.

### Conclusion

To be a learner centered teacher one has to focus the attention on the student and the learning process: how the student is learning, what the student is learning. The distinction

between teacher centered learning and student centered learning is that the spot light is shifted from the teacher to the student. This approach now features how the student accepts, builds and cultivates the ultimate responsibility of learning. Content is the vehicle to develop learning skills and strategies. Teachers must be well equipped to develop both general and specific contents for a given class.

In order to make this process work teachers must develop an integrated, coherent philosophy of education. Teachers need to have an approach not just a set of practices. One must take the trial and error approach. A realistic goal must be set. Teachers must also develop a deeper and more accurate self-knowledge. Feedback from students, colleagues and expert must be sought. So, in reality a lot of hard work and effort is needed to implement this kind of education otherwise it would just be a hypothesis.

### Work Sited

1. Palmer J Parker. The Courage To Teach. California: Jossey-Bass Inc, Publishers. 1998.
1. Weimer Maryellen. "Overview of Learner centered Teaching." Center for teaching, Learning and Faculty Development 2000. 15.10.09
2. [http:// www.ferris. edu/ fctl/ Teaching\\_and Learning\\_Tips /Learner-Centered % 20 Teaching / LCOverview.htm](http://www.ferris.edu/fctl/Teaching_and_Learning_Tips/Learner-Centered%20Teaching/LCOverview.htm)
3. "Learning Sciences and brain Research." 2004-2007. 15.10. 09 [www.ceri-forums.org](http://www.ceri-forums.org).
4. Tiny 08. "Learner Based Education." Socy Berty. Nov 2008. 13. 10.09 [http:// socyberty.com/education/learner-centered-education/](http://socyberty.com/education/learner-centered-education/)
5. "Learner Centered Psychological Principles" Nov 1997. 10.10.09 [www.apa.org/ed/ cpse/LCPP.pdf](http://www.apa.org/ed/cpse/LCPP.pdf)
6. Survey on Tairunnessa Memorial Medical College.

## COMMON SKIN DISEASE AMONG PRIMARY SCHOOL CHILDREN IN A SELECTED RURAL AREA OF BANGLADESH

Hasan MM<sup>1,#</sup>, Riya S<sup>1</sup>, Das S<sup>2</sup>, Ahmed SS<sup>1</sup>, Kabir A<sup>1</sup>

<sup>1</sup>*Department of Community Medicine, Tairunnessa Memorial Medical College, Gazipur*

<sup>2</sup>*MOMCH, Banskhal Upazilla Health Complex, Chittagong*

### Abstract

The study on common skin diseases among the primary school children in a selected rural area of Bangladesh was carried out in Kaligonj upazilla in Gazipur district on 120 children, during the period from July 2013 to December 2013 by non probability purposive sampling technique by using a structured questionnaire. Prevalence of common skin diseases was found to be 41.67% while infestations were more common among which 46% was pediculosis and 26% scabies. Amongst the infective dermatosis, boil 14%, herpes 6%, taenia capitis 4%, taenia corporis 2% and impetigo 2%. Frequent systemic surveys need to be carried out in rural parts of Bangladesh to unmask the problem of the rural children and help them combat the condition.

**Key Words:** skin disease, school children, personal hygiene.

### Introduction

Skin diseases are a major health problem affecting a high proportion of the population and causing distress and disability<sup>1</sup>. They are more frequent among primary school children in both developing and industrialized countries<sup>2</sup>. Skin disorders affect 20-30% of the general population at any time. Prevalence of skin disorders amongst children in various parts of world range from 4.3% to 49.1% in school based surveys<sup>4</sup>.

Skin diseases are a common cause of morbidity, especially among school children, worldwide. Although skin disease is rarely lethal, it can have a

significant impact in terms of treatment cost, days absent from school, and psychological distress<sup>5</sup>. It has been estimated that skin diseases account for about 30% of all pediatric consultations. Identification of skin disease in this age group by a trained dermatologist is very important. A world Health Organization (WHO) review of prevalence studies done on skin diseases among children reported an overall prevalence ranging from 21% to 87%<sup>6</sup>. Reports state that skin diseases account for 6% - 24% of all visits to the pediatric clinic. The school environment makes children vulnerable to cross transmission of communicable skin diseases among themselves and their families.<sup>7</sup>

### #Address for Correspondence

*Dr Muhammad Mehedi Hasan, Senior Lecturer, Department of Community Medicine, Tairunnessa Memorial Medical College, Konla, Board Bazar, Gazipur-1704. Email: drtito1979@gmail.com*



This study was conducted with the objectives to assess and address the dermatological problems among primary school children. It will be helpful for developing preventive health measures, health educational program and primary health care policy.

### Material and Methods

This descriptive cross sectional study was conducted among primary school children during the period from July to December 2013 to find out the prevalence of common skin disease. A 120 school children were selected from a primary school situated in Tumulia union of Kaligong Upazilla, Gazipur by purposive sampling. After taking written permission from school authority data were collected by face to face interview using a structured questionnaire regarding socio-demographic characteristics, personal hygiene. Every respondents were examined and check list was used to record different types of skin disease.

### Results

Among 120 respondents, majority of the children (66.66%) were male with a male female ratio of 2:1. Of the children 81.66% were in 5-10 years age group. Mean age of the respondents was 7.58 years. Majority (87.05%) of the mothers were house wife. among the mothers 50% had primary education with a mean monthly income of 6833.34 taka. (Table-1). Of all the respondents 68.33% used soap during bath, 17.05% sometimes used soap, while 14.17% did not use soap at all (Table 2).

According to table 3, sharing of towel with other family members was common (54.17%). Among 120 children, 50% washed cloth daily, 31% once a week while the rest 19% washed cloth twice a week (table 4).

Of all the respondents 58.33% were free from skin disease while the rest 41.67% had skin

disease (figure-1). The majority of dermatosis belongs to infestation group among which pediculosis was the most common (46%) followed by scabies (26%), Boil (14%), Herpes (6%), Impetigo (2%) and Taenia corporis (2%) and capitis (2%). Female children were mostly suffering from pediculosis while male children were victims of scabies.

**Table 1:** Distribution of the respondents (n=120) by socio-demographic characteristics

Variables	Frequency	Percent
<b>Age (yrs) Group</b>		
< 5	10	8.33
5 - 10	98	81.66
> 10	12	10.01
<b>Sex</b>		
Male	80	66.66
Female	40	33.34
<b>Education of mother</b>		
Illiterate	15	12.5
Primary	60	50
Secondary	35	29.17
HSC & higher	10	8.33
<b>Occupation of mother</b>		
House wife	105	87.05
Service	05	04.03
Business	02	01.06
Others	08	06.06
<b>Monthly family income (Taka)</b>		
< 5,000	38	31.00
5,000 - 10,000	60	50.00
> 10,000	22	19.00
Total	120	100

Data were expressed as number (percent).

**Table 2:** Distribution of the respondents according to daily use of soap during bath

Soap use Pattern	Frequency	Percent
Daily	82	68.33
Sometimes	17	14.17
No Soap use	21	17.05
Total	120	100

**Table 3:** Distribution of respondents on the basis of sharing of towel among family members

Sharing of towel	Frequency	Percent
Yes	65	54.17
No	55	45.83
Total	120	100

Data were expressed as number (percent).

**Table 4:** Distribution of respondents on the basis of practice of washing of cloths

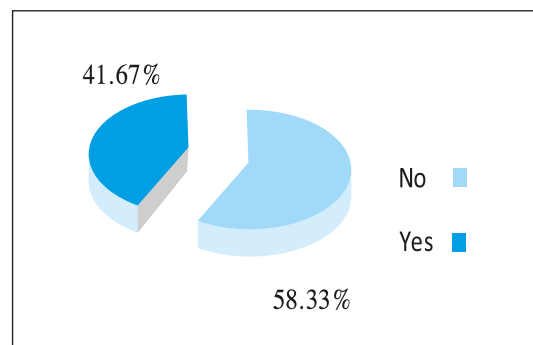
Washing of cloth	Frequency	Percent
Daily	60	50.00
Once a week	38	31.00
Twice a week	22	19.00
Total	120	100

Data were expressed as number (percent).

**Table 5:** Distribution of the respondents according to pattern of skin diseases

Disease	Number			Percent
	Male	Female	Total	
Infestation				
Pediculosis	5	18	23	46
Scabies	9	4	13	26
Infection				
Boil	6	01	07	14
Herpes	2	01	03	06
Impetigo	01	-	01	02
Taenia capitis	2	-	02	04
Taenia corporis	-	01	01	02
Total	25	25	50	100

Data were expressed as number (percent).

**Figure 1:** Distribution of the respondents with or without skin disease

## Discussion

This cross sectional study was conducted among primary school children in a rural area of Bangladesh to find out the presence of different skin diseases of the school children. The study period was from July 2013 to December 2013. Total number of children examined was 120 and data were collected from the children by face to

face interview using a structured questionnaire and check list and physical examination.

Socio-demographic characteristics play a important role in determining the pattern of skin disease in school children. This study shows that majority of the children (81.66%) were in the 5-10 years of age, 66.66% were males, 66.67% were Hindus and majority (50%) had monthly family income of 5000 - 10,000 taka, 50% of the mothers had primary education while 45.83% of fathers had secondary education, 87% of the mothers were housewives while 29.2% of the fathers were service holders.

In comparison Khalifa et al in their study involving primary school children in Baghdad, Iraq found that commonest age group was 12-15 years, mostly female, the percentage of illiterate were more among fathers than mother while mothers had primary education mostly.<sup>1</sup>

It was demonstrated that 68.33% used soap daily during bath. Of the total 54.17% of the children share towel with other family members but it was found 44.9% in other study<sup>5</sup>. It was found that 50% of the children washed their clothes daily.

Of the total children 41.67% had skin disease of which 46% had pediculosis, 26% scabies, 14% Boil, 6% Herpes, 4% Taenia capitis and minority 2% had Impetigo and 2% Taenia corporis. Suman et al reported over all prevalence of skin disorder to be 42.3%<sup>3</sup> while Khalifa et al found the prevalence of skin diseases to be 40.9% in Iraq.<sup>1</sup>

Saeed et al conducted one study in Al Khobar city, KSA involving female school children and reported that most common condition was pigmentary disorders 91.6% then dermatitis eczema 26.7%.<sup>7</sup> The findings of the present study, however, found to be consistent with one study which demonstrated presence of mostly superficial infection (fungal, bacterial and viral),

eczema as dermatosis and infestations (scabies) and pediculosis<sup>5</sup>. Scabies was found 26% in the present study. Although it was 15.16% and 13.14% in studies by Sarker et al<sup>8</sup> and Yousuf et al<sup>9</sup> respectively.

## Conclusions

The prevalence of skin disorders was high among the primary school children in the study area. Regular examination of school children by qualified doctors with the help of school authorities will help in reducing the prevalence of skin disorders in the society.

## Recommendations

1. Regular examination of school children by experienced doctors with the help of school authorities will help in reducing the prevalence of skin disease in school children.
2. Ensuring the routine school health check up with due importance to skin disease should be given.
3. Regular health education or awareness program of both parent and children regarding personal hygiene and paediatric skin disease, should be arranged.

## References

1. Khalifa K. A., Hadithi S A, et al. prevalence of Skin disorders among primary school children in Baghdad governorate, Iraq. EMHJ. 2010; **16**: 209-212.
2. Abolifotouh MA, Bahamadan K. Skin diseases among blind and deaf male students on South Western Saudi Arabia. Ann Saudi Med. 2000; **20**: 161-9.
3. Suman H, Savita C, Devesh M. A School survey of dermatological disorders and associated socio economic factors in Lucknow. Egypt Dermatol J 2012; **8**: 4.

4. Rea JN, Newhouse ML, Halil T. Skin disease in Lambeth: a community study of prevalence and use of medical care. *Br J Prev Sol Med.* 1976; **30**: 107-14.
5. Amin TT, Ali A, Kaliyadan F. skin disorders among male primary school children in Al Hassa, Saudi Arabia: Prevalence and socio - demographic correlates-a comparison of urban and rural populations. *The International Electionic J Rural Remote Res.* 2011; **1**: 1517.
6. Rao C, Rao R. A cross sectional study of Dermatological problems among differently Abled children. *Indian J Dermatol.* 2012; **57**: 35-37.
7. Al -Saeed W Y, Al -Dawood K M, Bukhari I A, Bahnassy A A. Prevalence and pattern of skin disorders among female school children in Eastern Saudi Arabia. *Saudi Med J.* 2006; **27**: 227-233.
8. Sarker SK, Islam AKMS, Sen KG, Ahmed ARS. Pattern of skin disease in patients Attending OPD of Dermatology Department at Faridpur Medical College Hospital, Bangladesh. *Faridpur Med Coll J.* 2010; **5**: 14-16.
9. Yousuf AKM, JoarderY, Akter A, Hoq MN, Rahman M, Akter F, Begum H et al. Pattern of skin disease in patients attended OPD of Dermatology and Venereology in a tertiary care teaching hospital in Bangladesh. *Bang J Physiol Pharmacol* 2013; **29**: 8-11.

## ARSENIC CONTAMINATION OF DRINKING WATER AND HEALTH EFFECTS: BANGLADESH PERSPECTIVE

Kabir A<sup>1</sup>, Riya S<sup>1</sup>, Chowdhury AA<sup>2</sup>

<sup>1</sup>Department of Community Medicine, Tairunnessa Memorial Medical College, Gazipur-1704

<sup>2</sup>Deputy Program Manager, DGHS, Mohakhali, Dhaka

### Abstract

Ground water in Bangladesh reported to be contaminated with inorganic arsenic above WHO recommended safe dose ie, 0.05 mg/l in 59 districts. Diabetes mellitus, HTN, chronic bronchitis, anemia, skin cancer, neuropathy were reported from several studies in Bangladesh. Significant association was observed between arsenic exposure and adverse pregnancy outcomes like still birth, neonatal and infant death in Bangladesh. Millions of people are at risk. Health education regarding awareness among peoples about arsenic and its deleterious effect and also support from both government and non- government organizations are necessary to combat the problem.

**Key Words:** Arsenic, Drinking water, Health effect.

### Introduction

Bangladesh is grappling with the largest mass poisoning of a population in history because ground water used for drinking has been contaminated with naturally occurring inorganic arsenic. It is estimated that of the 125 million inhabitants of Bangladesh between 35 million to 77 million are at risk of drinking contaminated water.<sup>1</sup> Bangladesh an alluvial and deltaic land of 147, 570 km<sup>2</sup>, is prone to various natural disasters like cyclones, floods, and droughts. In recent time environmental catastrophe pollution of ground water arsenic has been reported in Bangladesh.<sup>2</sup> It was found that Indian state of West Bengal also face this problem.<sup>3</sup> Ground water with elevated concentration of arsenic has been recognized as a problem of global concern. Arsenic contamination

of ground water is one of the principal pathways of human exposure to inorganic arsenic. High concentrations of arsenic has been reported from several regions of the world.<sup>4</sup> Inorganic Arsenic is recognized as a known carcinogen and toxicant. Many studies conducted in Taiwan, Chile, and Argentina has shown elevated risk of cancers among population chronically exposed to inorganic arsenic in drinking water. Significant dose response relationships were observed between arsenic levels in well water and cancers of bladder, kidney, skin, lung and liver in both male and female.<sup>5</sup> The toxicity of arsenic depends on many factors such as chemical and physical form of the compound, route of administration, dose and duration of exposure, dietary level of the

### Address for Correspondence

Dr Asma Kabir, MBBS, MPH, M. Phil (PSM), Professor, Department of Community Medicine, Tairunnessa Memorial Medical College, Konia, Board Bazar, Gazipur-1704. Email: drasma5000@gmail.com

interacting elements, nutritional status, age and sex of the exposed individuals.<sup>6</sup> According to WHO, drinking arsenic rich water over a long period results in various health effects.<sup>7</sup> It has been proved in scientific study that chronic arsenic exposure through drinking water is associated with an increase in the mortality rate. The health effects can endanger the security of the country in the long run.<sup>8</sup> In Bangladesh most of the people resides in village, there are 8000 villages where 80 percent of all tube wells are contaminated. Almost one in five wells is not providing safe drinking water. About 20 million people in Bangladesh are using tube wells with more than 50 ppb of arsenic.<sup>9</sup> This has necessitated world agencies to undertake immediate mitigation measures to tide over this crisis. The successful mission towards elimination of this health hazard rests on three pillars, diagnosis, prevention, and management.<sup>10</sup>

### Statistics of arsenic scenario in Bangladesh

Total number of districts in Bangladesh: 64

- WHO Arsenic drinking standard: 0.05 mg/l
- Population at risk: 75 million.<sup>11</sup>
- The DPHE (Department of Public Health Engineering) 2006 reported that arsenic contamination in tube well water is present in 62 of 64 districts of Bangladesh.
- Cumulative number of arsenic patients in Bangladesh up to the year 2010 was 56,758 detected under the National Arsenic Program of DGHS.<sup>12</sup>

**Arsenicosis case definition:** as per WHO working group-Chronic health condition arising from prolonged ingestion (not less than 6 months) of arsenic above a safe dose, usually manifested by characteristics skin lesions with or without involvement of internal organs.<sup>13</sup>

### Diagnosis of arsenicosis

Characteristics clinical and laboratory criteria for diagnosis of arsenicosis

Clinical cutaneous manifestations

- **Melanosis:** Fine freckled or spotted pigmentation (Rain drop pigmentation)  
Diffuse or generalized hyper pigmentation
- **Keratosi:** Mild-minute papule (<2mm) with slight thickening of palm and sole  
Moderate multiple keratotic papule (2-5mm)  
Severe large discrete papule (>5mm)
- **Malignant/ premalignant lesion:** Bowen's disease
- Laboratory criteria for establishment of exposure to arsenic
- **Water (for at least 6 months):** >0.05 mg/l.
- Hair and Nail (evidence of past exposure with in 9 months): >1 mg/kg of dry hair >1.5 mg of nail
- **Urine (evidence of recent exposure):** >50 microgram/l.<sup>14</sup>

### Health effects of chronic exposure

Respiratory effects has been found among exposed subjects was 2.9 (95% CI 1.6%-5.4%). The risk for chronic bronchitis among women was six times higher than men<sup>15</sup>. A study conducted in BSMMU, found that 37 (74%) out of 50 arsenicosis patients were anaemic. There was a positive correlation between grade of arsenicosis and haemoglobin level ( $p < 0.05$ ).<sup>16</sup> The prevalence of type 2 diabetes among the exposed person was 9% (95% CI 7%-11%). An increased risk for arsenic exposure over 50 microgram/l having double the risk of type 2 diabetes (OR=1.9, 95% CI 1.1-3.5).<sup>17</sup> In another study, the crude prevalence of diabetes mellitus among exposed person was 4.4% (95% CI 2.5-7.7%). The prevalence of diabetes mellitus has a dose-response pattern with a significant



trend in relation to exposure category ( $p < 0.001$ ).<sup>18</sup> Long term arsenic ingestion has been associated with increased cardio-vascular mortality. A prevalence study conducted in Bangladesh demonstrates a dose- response relationship between inorganic arsenic exposure from drinking water and risk of HTN.<sup>19</sup> The overall prevalence ratio of HTN for men was 2.1% (95% CI 0.7 to 6.5) and for women was 1.5% (95% CI 0.7-3.4) among the exposed persons.<sup>20</sup> In a study conducted in Comilla district in Bangladesh, among the arsenic exposed villagers, 60.24% reported with neuropathy<sup>21</sup>. In another study, 2006 pregnant women chronically exposed to a range of naturally occurring concentration of arsenic in drinking water in three upazilas in Bangladesh, there was a small but statistically significantly association found arsenic exposure and birth defects (OR=1.005, 95% CI 1.001-1.010)<sup>22</sup>. Drinking tube-well water with more than 50 microgram/l of arsenic during pregnancy significantly increased the risk of foetal loss (relative risk=1.14, 95% CI: 1.04, 1.25) and infant death (relative risk=1.17, CI: 1.03, 1.32). There was a significant dose response of arsenic exposure to risk of infant death ( $p=0.02$ ).<sup>23</sup> Excess spontaneous abortion, still birth and preterm birth rates among women with chronic arsenic exposure were first reported in Bangladesh in 2001. Exposure to arsenic concentration of 50 microgram/l in drinking water showed the odds ratios were 2.5(95% CI=1.5-4.3) for spontaneous abortion, 2.5(1.3-4.9) for still birth and (0.9-3.6) for neonatal death.<sup>24</sup> Prevalence of skin cancer in Bangladesh among arsenicosis patients 2.6% found to develop skin cancer.<sup>25</sup>

### Prevention and Control

The most important action in affected communities is the prevention of further exposure to arsenic by the provision of a safe water supply for drinking, food preparation and irrigation of food crops. In 2004 an Arsenic

Policy Support Unit and Implementation Plan for Arsenic Mitigation guided by national committee was created. In 2009 the National policy was revised and new initiatives taken.

- Awareness raising in rural area only to use those tube wells which are confirmed as arsenic safe.
- Sand filtered water from ponds for drinking water purpose.
- Provision of arsenic removal devices at household or community level.
- Drilling of deep hand -pump tube wells in the coastal zone.
- Construction of piped water supply scheme from arsenic safe source.<sup>26</sup>

### Conclusion

Long term ingestion of arsenic causes various adverse effects. We should give emphasis regarding awareness about health problems caused by arsenic, health education and community participation to overcome the problem.

### References

1. Smith AH, Lingas EO, Rahman M. Contamination of drinking water by arsenic in Bangladesh: a public health emergency. Bulletin of the World Health Organization, Bull WHO 2000; 78.
2. Ahmad SA, Sayed MHS, Barua S et al. Arsenic in drinking water and pregnancy outcomes. Environmental Health Perspectives. June 2001; **109**: 629-31.
3. Caldwell BK. Smith WT, Lokuge K et al. Access to drinking-water and Arsenicosis in Bangladesh. J Health Population Nutr. 2006; **24**: 336-345.
4. Kapaj S, Peterson H, Liber K et al. Human Health Effects From Chronic Arsenic Poisoning- A Review. J Environ Sci Health Part A. 2006; **41**: 2399-2428.

5. Milton AH, Hasan Z, Shahidullah SM et al. Association between nutritional status and arsenicosis due to chronic arsenic exposure in Bangladesh. *Inter J Environ Health Research*. 2004; **14**: 99-108.
6. Rahman MH, Sikder MS, Islam AZMM. Spirulina as food supplement is effective in arsenicosis. *J Pak Assoc Dermatol*. 2006; **16**: 86-92.
7. Bangladesh: Arsenic - free drinking water by 2013? [www.irinnews.org](http://www.irinnews.org).
8. Alam I. Arsenic poisoning in Bangladesh. Is it a security issue? [www.isn.cth2ch/Digital-library/publication/details](http://www.isn.cth2ch/Digital-library/publication/details)
9. Arsenic Mitigation in Bangladesh. UNICEF Report. Oct 2008.
10. Year Das NK, Sengupta SR. Arsenicosis: Diagnosis and treatment. *Indian J Dermatol Venereol Leprol*. 74.
11. Safiuddin M, Karim MM. Ground water Arsenic Contamination In Bangladesh: Cause, Effects And Remediation. 7th Annual paper meet of 1st IEB International Conference. Nov 2001.
12. Arsenic in groundwater: Mitigation program of the DGHS, *Health Bulletin* 2012, MIS, DGHS, Chapter 11, pg-131.
13. Arsenicosis Case -Detection Management and Surveillance. WHO Report of a Regional Consultation. June 2003.
14. Caussy D. A field Guide for Detection of Arsenicosis Cases. WHO Report. New Delhi. 2005.
15. Milton AH, Hasan Z, Rahman A et al. Chronic Arsenic poisoning and Respiratory Effects in Bangladesh. *J Occu Health* 2001; **43**: 136-140.
16. Sikder MS, Shakya NB, Rahman MH et al. Association between anemia and grading of arsenicosis. *J Pak Assoc Dermatol*. 2008; **18**: 202-206.
17. Islam MR, Khan I, Hasan SMN et al. Association between type 2 diabetes and chronic arsenic exposure in drinking water: A cross sectional study in Bangladesh. *Environmental Health* 2012; **11**: 38.
18. Rahman M, Tondel M, Ahmad SA et al. Diabetes Mellitus Associated with Arsenic Exposure in Bangladesh. *Am J Epidemiol* 1998; 148.
19. Islam AKMM, Majumder AAS. Hypertension in Bangladesh: a review. *Indian Heart Journal* 6403, 2012: 319-323.
20. Rahman M, Tondel M, Ahmad SA et al. Hypertension [http:// hyper.ahajournals.org/](http://hyper.ahajournals.org/) May 2, 2013.
21. Ahmad S, Sengupta MK, Mukherjee SC et al. An Eight - Year Study Report on Arsenic Contamination in Ground water and Health Effects in Eruani Village, Bangladesh and an Approach for Its Mitigation. *J Health Popul. Nutr* 2006; **24**: 129-141.
22. Kwok RK, Kaufmann RB, Jakaria M. Arsenic in Drinking -water and Reproductive Health Outcomes: A Study of Participants in the Bangladesh Integrated Nutrition Programme. *J Health Popul. Nutr* Jun 2006; **24**: 190-205.
23. Rahman A, Vahter M, Ekstrom EC et al. Association of Arsenic Exposure during Pregnancy with Fetal Loss and Infant Death: A Cohort Study in Bangladesh. *Am J Epidemiol* 2007; **165**: 1389-96.
24. Hasnat MA, Wayne S, Bayzidur R et al. Chronic Arsenic Exposure and Adverse Pregnancy Outcomes in Bangladesh. *Epidemiology* 2005; **16**: 82-86.
25. Alam M, Hazari SKS, Alam ASMT. Prevalence of Skin Cancer In Chronic Arsenicosis In Chittagong Medical College Hospital. *JCMCTA* 2010; **21**: 23-29.
26. Tuinhof A, Kemper K. Mitigation of Arsenic Contamination in Drinking Water-Supplies of Bangladesh- the Case of Chapai Nawabganj. World Bank Report Oct 2010.



## AUTISM: A GLOBAL CHALLENGE

Riya S<sup>1</sup>, Kabir A<sup>2</sup>

<sup>1</sup>Associate Professor and <sup>2</sup>Professor, Department of Community Medicine, Tairunnessa Memorial Medical College & Hospital, Gazipur

### Abstract

Autism is a developmental disorder of the human brain characterized by impairments in social interaction and communication, restricted and repetitive behavior, that first shows signs during infancy or before a child is three years old and follows a steady course without remission or relapse. This review focuses causes of autism as being multifactorial such as antenatal, perinatal, postnatal, neonatal and early childhood factors. It is important to differentiate autism from other conditions like communication disorder, learning disability, Asperger's syndrome and childhood disintegrative disorder. Several management techniques are being used to handle the condition to reduce family burden. However the condition is on the rise locally in Bangladesh and globally. Perhaps future studies may help to identify children at risk for these disorders earlier and lead to more effective interventions to enhance the quality of life for individuals with their families.

**Key Words:** Autism, ASD, Stereo type behavior, PDD NOS

### Introduction

Autism means to be on one's own. Autism spectrum disorder (ASD) and autism are both general terms for a group of neuro developmental disorders defined by social and communication deficits and repetitive behaviors that are typically detectable in early childhood, continuing throughout life.<sup>1</sup> Autism involves abnormalities of brain development and behavior which become apparent before a child is three years old and have a steady course with no remission. It is part of a larger family called the Autism Spectrum disorders.<sup>2</sup> Autism or classical ASD is the most severe form of ASD while other conditions along

the spectrum include a milder form known as Asperger's syndrome and Pervasive developmental disorder not otherwise specified (PDD NOS). Though ASD varies significantly in character and severity, it occurs in all ethnic and socio economic groups and affects all age groups. A number of preventable nutrition related disorders may happen between conception and delivery and cause irreversible neurological damage. Neurological development begins in the foetus and continues until the child is about 36 months old, this development depends mainly on four factors: maternal nutrition before, during

### Address for Correspondence

Dr Sayeda Riya, MBBS, MPH(CM), M.Phil(PSM), Associate Professor, Department of Community Medicine, Tairunnessa Memorial Medical College & Hospital, Gazipur. E-mail: drriya11@gmail.com

and after the pregnancy and the child's nutrition after breast feeding is over. The rapidly ensuing malnutrition can cause irreversible brain damage in as little as 5 to 10 days.<sup>3</sup>

Autistic children have 3 distinct features: lack of socialization, failure to communicate and stereotyped behavior. Heritability contributes about 90% of the risk of a child developing autism but the heritability of autism is complex and typically it is unclear which genes are responsible.<sup>4</sup> A substantial fraction of autism may be highly heritable but not inherited that is the mutation causes ASD is not present in the parental genome.<sup>5</sup> Risk factors for autism include parental characteristics such as advanced both maternal and paternal age.<sup>6</sup> The genetic quality of sperm as well as its volume and motility all typically decrease with age. Autism is a lifelong disease that ranges in severity from mild cases in which the patient can live independently to severe forms in which the patient requires social support and medical supervision throughout his or her life. Several studies on prenatal, perinatal, neonatal and early childhood factors in ASD show that there is association between several prenatal, perinatal and neonatal complications and ASD.<sup>8</sup>

Antenatal factors like having a job, mental stress, anaemia, threatened abortion, twin or multiple pregnancy, pre eclamptic toxemia of pregnancy, gestational diabetes, measles during pregnancy, taking infertility drugs, exposure to x rays, working on computer and watching television, may all contribute to development of autism.<sup>9</sup> Perinatal factors include mode of delivery, complications during labour and delivery like obstructed labour, malpresentation and cord round the neck and birth weight > 2.5 kg.<sup>10</sup> Post natal factors show mainly complications of the new born namely birth asphyxia, birth injury and neonatal jaundice. Early childhood factors showed MMR vaccination, measles infection, ear infection and gastro intestinal infection, head

injury, febrile convulsion, breast milk intake and drug intake like anti biotics and anti pyretics in the first 2 years after birth.<sup>11</sup>

ASD may begin at birth or within first two years of life.<sup>12</sup> It may vary widely in severity and may be accompanied without intellectual disability. It is a life long developmental disability and affected persons fail to communicate, develop friendship with peers, fail to understand other peoples feelings and thoughts and have difficulty in making sense of the world around them.<sup>13</sup> Hence the disease burden is heavy. Due to significant increase in the reported prevalence of ASD the centre for disease control and prevention (CDC) has recognized autism as an urgent public health concern.<sup>14</sup>

### **Autism: Global Situation**

For several years ASD remained a rare condition with a prevalence of only 1-2 per 1000 children worldwide. Nearly 83 million of the world's populations are estimated to be mentally retarded with 41 million having long term or permanent disability.<sup>15</sup> ASD is the second most common developmental disability next to mental retardation. According to CDC prevalence in USA: 1 in 110, England: 1.7/1000, China: 1.1/1000, Pakistan 1 in 500, India: 4 in 1000. ASD is more prevalent in upper socio economic class and four times more in boys than in girls.

### **Autism: Local Situation In Bangladesh**

There is gross lack of knowledge about autism even among doctors. Very often children are misdiagnosed and given antipsychotic drugs by psychiatrists. It is estimated by the ministry of social welfare that the total number of persons with autism could be as high as 1.4 million of whom only a few hundred have been diagnosed.<sup>16</sup> The prevalence is 1 in 500. Present activities with autism in Bangladesh include :diagnosis and assessment of autistic children, vocational training and special education, patient

counseling, teacher, training programme, seminars, workshops, sponsorship to autistic children from low income families. Some of the organizations in Bangladesh which have been working for autism are: Autistic welfare foundation, Bangladesh protibondhi foundation, Society for the welfare of autistic children, Child development centre: ICMH, Matuail, Dhaka .Child neurology clinic: pediatric unit DMCH, Shishu Bikash kendro: Dhaka shishu hostital,child neurology clinic: pediatric unit BSMMU, Beautiful mind: utara.

### Differential Diagnosis of Autism:

Deafness,Communicationdisorder,Learningdisability,Asperger'ssyndrome,Childhood disintergrative disorder.<sup>17</sup>

### Prognosis

1. Between 10-20% of children with childhood autism begin to improve between ages of 4-6 years, and are eventually able to attend an ordinary school and obtain work.
2. A further 10-20% can live at home but cannot work and need to attend a special school or training centre and remain very dependent on their families and or support services.
3. Remainder 60% improve little and are unable to lead an independent life, may need long term residential care
4. Those who improve or continue show language problems, emotional coldness and odd behaviors.
5. A substantial minority develop epilepsy in adolescence.

### Assessment

1. Cognitive level.
2. Language ability.
3. Communication skills, social skills and play.
4. Repetive/abnormal behavior.
5. Associated medical conditions.

### Management of autism

There is no cure for autism. Therapies and behavioral interventions are designed to remedy specific symptoms and can bring about substantial improvement. Earlier the intervention the better.<sup>18</sup> Doctors often prescribe antidepressants, anti psychotics and anticonvulsants to handle symptoms.

1. Management of abnormal behavior: by parents at home, instructed and supervised by a clinical psychologist.
2. Education and social services: Special schooling is needed for most autistic children. In severe condition residential schooling may be necessary.
3. Help for the family: parents may find it helpful to join a voluntary organization where common problems may be discussed with other similar parents.
4. Biomedical management: dietary restrictions may include all gluten and casein free diets. Gluten is rich in all wheat and flour products. Rice or potato flour may be used. Casein is rich in all milk products including yogurt. Soya milk may be used instead. Sugar, jaggery, dates and honey should be eliminated.
5. Applied Behavior Analysis:(ABA) is the oldest and most fully researched treatment specifically developed for autism. It is a very intensive system of reward based training which focuses on teaching particular skills.
6. Speech therapy: Almost all people with autism have issues with speech and language. Autistic individuals are often non verbal or use speech very poorly. Sometimes the issues relate not to articulation or grammar but to speech pragmatics (use of speech to build social relationships)
7. Sensory Integration Therapy: This is a form of occupational therapy offered by specially

trained occupational therapists involving specific sensory activities (swinging, bouncing, brushing etc) intended to help the patient regulate his or her sensory response.)

8. Social Skills Therapy: As there is gross lack of social and communication skills, social skill therapists help set up and facilitate peer based social interaction.

9. Physical Therapy: Many autistics have gross motor delays and low muscle tone. Physical therapy can build up strength, coordination and basic sports skills.

10. Play therapy: Autistic children need help learning to play and play can also serve as a tool for building speech, communication and social skills.

11. Behavioral Therapy: Behavior therapists figure out what lies behind negative behaviors and recommend changes to the environment and routines to improve behavior. As autistics suffer from hypersensitivities to sound, light and touch no wonder they sometimes act out.

12. Developmental Therapy: Building a child's own interests, strengths and developmental level to increase emotional, social and intellectual abilities such as shoe tying, tooth brushing etc.

13. Visually Based Therapies: Picture based communication systems such as PECS (picture exchange communication), video modeling, video games and electronic communication systems also tap into autistic people's visual strength to build skills and communication.

## Conclusion

Autism is a pandemic condition in the world today.<sup>19</sup> ASD'S are complicated conditions in which genetic, prenatal, social, developmental, nutritional and environmental factors play major roles.<sup>20</sup> Larger studies are needed to determine optimum multifactorial treatment plans involving nutrition, environmental control, medication, behavioral, educational, speech and physical therapies.

## References

1. Zhang X, Cong L, Tian J, Mio R, Weixi I, Picciotto I, Lihong Q et al. Pre natal and perinatal Risk factors for Autism in child, *J Autism Dev Discord*, 2010; **40**: 1311-1321.
2. Psychiatry oxford core text publishers, 3<sup>rd</sup> edition, oxford medical publication 292, Gelder M, Harrison P, Cousen P, Shorter textbook of psychiatry, 5<sup>th</sup> edition 671-5.
3. Timothy B, Alberti A, Pirrone P, et al. Supplement article, Evaluation, Diagnosis and treatment of Gastro intestinal disorders in individuals with ASD's: A consensus report *pediatric's vol. no 125, Supplement 2010*; **359**: 1-518.
4. Wakefield AJ, Antony A, Cornish E et al. CDC: autism spectrum disorders common. *JAMA*. 2007, **297**: 940.
5. Levyse M, Ittenbach R, Mulberg A et al, *Biol psychiatry*, 2007 Feb 15, **61**: 492-7
6. Reichenberg A, Sandler R, Brent L et al. Advancing paternal age and autism, *Arch Gen psychiatry*, 2006; **63**: 1026-1032.
7. Rutter M, Incidence of ASD: Changes over time and their meaning, *Acta Paediatr* 2005, **94**: 2-515.
8. Zimmerman J, Bilder D, Bakian A et al, Sociodemographic risk factors associated with ASD and intellectual disability. *Autism Research*, 2011; **Dol**: 10.1002.
9. Gardener H, Spiegelman D, Buka S et al. Prenatal risk factors for autism: comprehensive meta analysis, *Br J Psychiatry*, 2009; **195**: 7-14.
10. Glasson EJ, Bower C, Petterson B. Perinatal factors and the development of autism: a population study, *Arch Gen psychiatry* **61**: 618-27.

11. Teresa A, Jane A, Teasdale A. Incidence of pre, peri and post natal birth and developmental problems of children with SPD and children with ASD. *Front Integr Neuro sci.* 2009; **3**: 31.
12. James B, Stephen M, Grandin T, Rimland B. Infantile Autism: the syndrome and its implications for a neural theory of behavior, *J Am Physicians Surg.* 2003; **8**: 58-60.
13. Kidd PM, Autism, an extreme challenge to integrative medicine part 1: the knowledge fase. *Altern med,* 2002; **7**: 292-316.
14. Rutter M, Incidence of ASD, s: changes over time and their meaning, *Acta paediatr,* 2005; **94**: 2-15.
15. Durkin M, Maenner MJ, Newsschaffer CJ et al. Advanced paternal age and risk of ASD. *Am J Epidemilo.* 2008; **168**: 1268-76.
16. Rahman G, Rashid A, Kabir BG et al. Factors associated with nutritional status of children in children in Bangladesh: a multivariate analysis, *Demography India* 2008, **37**: 95-109.
17. Bailey A, Le courteur A, Gottesman I. Autism as a strongly genetic disorder, Evidence from a British twin study. *Psycho Med.* 1995; **25**: 63-77.
18. Elbaz F, Ismael NA, Noru El. Risk factors for Autism, an Egyptian study. *Sgypt J Med Hum Genet* 2001; **34**: 324-657.
19. Levy SE, Mandell DS, Schultz RT et al. Autism. *Lancet* 2009, **374**: 1627-1638.
20. Dietert JM, Wangs F, Chapman k et al. Enviromental risk factors for ASD, *Emerg Health Threat J.* 2011; **4**: 7111.

## AN EXPERIENCE ON THE TREATMENT OF RELAPSE OF A KALA-AZAR PATIENT WITH LIPOSOMAL AMPHOTERICIN- B

Islam MS<sup>1</sup>, Azad MAK<sup>1</sup>, Kader MA<sup>2</sup>

<sup>1</sup>Department of Medicine, Bangabandhu Sheikh Mujib Medical University (BSMMU), Shahbag, Dhaka, Bangladesh; <sup>2</sup>Department of Medicine, Tairunnessa Memorial Medical College & Hospital, Gazipur

### Abstract

We report a case of 23-year-old male from Ghatail, Tangail who presented with pyrexia of unknown origin for one month. He had pancytopenia with splenomegaly. The diagnosis was established by visualisation of amastigotes in bone marrow biopsy and by detection of antibodies to *Leishmania* spp. in blood. The infection was treated intravenously with liposomal amphotericin B for six days. The patient was afebrile after the first infusion. No relapse was reported.

### Introduction

*Leishmania* spp. is obligatory intracellular protozoans, which cause a wide range of clinical manifestations: visceral (VL, kala-azar), cutaneous and mucocutaneous leishmaniasis. Encountered in subtropical and tropical regions leishmaniasis has a prevalence of 12 million cases and an approximate incidence of 0.5 million cases of VL and 1.5 million cases of cutaneous leishmaniasis<sup>1</sup>. Disseminated VL is fatal if left untreated. The typical incubation period of VL varies from 3 to 8 months (longer periods up to several years have been reported). Over 90% of VL worldwide occurs in five countries across three continents: north eastern India, Bangladesh, Nepal, Sudan and north eastern Brazil. Leishmaniasis causes 2.4 million disability-adjusted life years and around 70,000

deaths per year.<sup>2,3</sup> As visceral Leishmaniasis mainly occurs in poor and remote areas where access to medical care is very limited, advances in treatment, diagnosis and vector control could help to break the vicious circle of poverty and disease. An intact cellular immunity is necessary to prevent the disease from affecting the entire reticulo-endothelial system.

An intact cellular immunity is necessary to prevent the disease from affecting the entire reticulo-endothelial system. Therefore malnourished and immune-compromised persons are the most affected. Even in southern Europe, where potent antiretroviral therapy is provided, VL is a common in AIDS-related infection. Interestingly 90% of HIV-associated VL represents reactivation of prior subclinical infection<sup>3,4</sup>.

### Address for Correspondence

Md Abdul Kader, Professor & Head, Department of Medicine, Tairunnessa Memorial Medical College & Hospital, Gazipur.

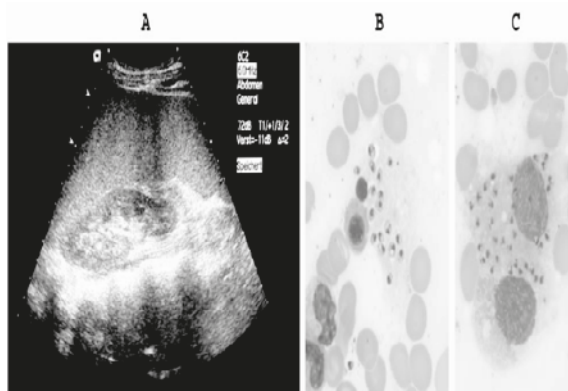


Common clinical manifestations are: fever (intermittent, remittent with two daily temperature spikes, or even continuous), cachexia, hypergammaglobulinemia, pancytopenia, hepatomegaly and splenomegaly. Most patients die due to intercurrent bacterial infection or tuberculosis. The gold standard of diagnosis remains direct microscopy (visualization of the parasite in affected tissues, mostly in bone marrow aspirate).<sup>4,5</sup> Agents with efficacy against VL include amphotericin B, pentavalent antimonial drugs, paromomycin (a parenteral aminoglycoside), and miltefosine. During the past decade, liposomal amphotericin B has been used with increasing frequency to treat visceral leishmaniasis (VL). In Africa and Asia, the VL disease burden is high and drug access is poor; liposomal amphotericin B is available only through preferential pricing for nonprofit groups in Asia. Clinical trials and experience demonstrate high efficacy and low toxicity for liposomal amphotericin B (total dose, 20 mg/kg) in immunocompetent patients with VL.<sup>4</sup>

### Case Description

We report a 23-year old -male from Ghatail, Tangail presented with fever for 11 months which was intermittent in nature with highest recorded temperature was 103 of not associated with chills and rigor. He was treated as a suspected case of kalaazar on the basis of clinical presentation and relevant lab reports from local Upazilla health complex with cap. miltefosine 50 mg 12 hourly for 30 days. His compliance was good and no adverse effect was occurred during the course of the treatment. His symptoms were subsided. But within 3 months he again became febrile. In last 6 months, he had about 6 kg weight loss in spite of good appetite. He also had easy fatigability. For that he was admitted in BSMMU. On examination the patient looks

emaciated with a dry, rough, dark skin. He was pale; there was no jaundice, cyanosis or lymphadenopathy. Abdominal examination revealed splenomegaly which was 10 cm from left costal margin, firm in consistency, non tender. Examination of other system was unremarkable. Initial lab investigation shows Hemoglobin: 8.9 g/dl; hematocrit: 0.3 l/l; erythrocyte count:  $3.71 \times 10^{12}/L$ ; WBC count:  $03 \times 10^9/L$  cells/mm<sup>3</sup> (54% PNL, 40% lymphocytes, 4% monocytes, 2% eosinophil); platelet count:  $120 \times 10^9/L$ ; ESR: 80 mm in the 1st hour. Peripheral blood film showed pancytopenia. RBS was 6.5 mmol/L, ALT 26 U/L, AST 53U/L, alkaline phosphatase 112U/L. Serum creatinine was 1.2 mg/dl, albumin 30 gm/L, Na<sup>+</sup> 136 mmol/L, K<sup>+</sup> 3.4/L, Cl<sup>-</sup> 98mmol/L. ICT for kala-azar was positive. Ultrasonogram of whole abdomen revealed splenomegaly (Fig: 1-A). Chest X-ray was normal. Examination of the bone marrow aspirate revealed presence of *Leishmania donovani* (Fig: 1-B, C). The patient was treated with injection Liposomal Amphotrecin B (fungisome - each ml of fungisome contains 1 mg of amphotericin BUSP, 45 mg of lecithin and cholesterol). Patient's weight was 40 kg and he was treated with total dose was 600 mg in 6 divided doses in 6 days. After the initiation of therapy, easy fatigability was reduced, appetite was improved, his discoloration of skin gradually disappeared and spleen size was progressively reduced. Laboratory investigation revealed- Haemoglobin 11.9 g/dL; hematocrit 0.36 l/l; erythrocyte count,  $3.9 \times 10^{12} /L$ ; WBC count,  $10 \times 10^9 /L$  cells/mm<sup>3</sup> (40% PNL, 46% lymphocytes, 4% monocytes, 10% esonophil); platelet count,  $390 \times 10^9/L$ ; ESR 20 mm in 1st hour. RBS was 5.6 mmol/L, ALT 34 U/L, serum creatinine 1.4 mg/dl, serum bilirubin 6 umol/L, albumin 34 gm/L, Na<sup>+</sup> 141mmol/L, K<sup>+</sup> 3.5/L, Cl<sup>-</sup> 99 mmol/L. LD body was absent in follow up bone marrow.



**Figure-1:** Ultrasound of splenomegaly (~ 18 cm) [A], bone marrow biopsy revealed numerous protozoan parasites (*Leishmania* spp.) extracellularly and within bone marrow macrophages [B,C]

## Discussion

Current options for the treatment of Kala Azar are antimony salts, amphotericin B and its lipid formulations, pentamidine, miltefosine and paromomycin<sup>6</sup>. Pentavalent antimony drugs are associated with adverse reactions and require long treatment periods (30 days). Antimony-resistant VL, especially in Bihar in India (45% of the world's cases) makes treatment even more difficult<sup>5</sup>. Miltefosine (the first effective oral treatment, testing still in progress) and the aminoglycoside paromomycin, which has shown good results in India and is currently being evaluated in East Africa, could provide a therapeutic alternative to antimony worldwide<sup>7</sup>. But, In our patient, disease was relapsed after treatment with Miltefosine. Although antimony drugs remain effective in Europe (cure rate of about 90%)<sup>8</sup>, most patients are now treated with liposomal amphotericin B, which is safe and very effective in a short time<sup>7</sup>. Reduction of the toxic effects by using lipid formulations allows the infusion of higher doses of amphotericin B. So far in Bihar 476 cases have been published (in six studies: two of which were comparative, three dose-finding, and one non-comparative) in

whom liposomal amphotericin were highly effective, associated with high cure rates even at very low doses<sup>9</sup>. In practical terms, the choice of which of these agents to use frequently comes down to cost and availability. In patients with impaired immunity and re-treating antimony failures liposomal amphotericin B (AmBisome®) might be the drug of choice. Various studies demonstrated that low dose and single dose liposomal amphotericin B were effective in the treatment of VL<sup>10</sup>. A single dose treatment of 5-7.5 mg/kg show cure rates of about 92% while a total dose of 2 mg/kg/day for 5 days can achieve a cure rate of roughly 99 % .Other liposomal preparations, including amphotericin B colloidal dispersion (ABCD) and amphotericin B lipid complex (Abelcet) have also been used in VL, although there is less experience with these compounds<sup>9,10</sup>. Our patient was treated with injection Liposomal Amphotrecin B (fungisome) 15 mg/ kg body weight in 6 divided doses and showed complete cure of the disease without any significant adverse effect.

## Conclusion

The Number of resistant Kalaazar with miltefosin is increasing day by day in Bangladesh. Conventional amphotericin B deoxycholate has high antileishmanial efficacy but is associated with high risk of renal toxicity and other side effects. Though this is our first experience with use of injectable Liposomal Amphotericin-B in a treatment failure Kala-azar patient, the management of this patient was uneventful with excellent recovery.

## References

1. WHO. The World Health Report 2000. Geneva: WHO, 2000.
2. Desjeux P. Leishmaniasis: current situation and new perspectives. *Comp Immunol, Microbiol and Infectious Diseases* 2004; **27**: 305-18.

3. Murray HW, Berman JD, Davies CR, Saravia NG. Advances in Leishmaniasis. *Lancet* 2005; **366**: 1561-77.
4. Olliaro PL, Guerin PJ, Gerstl S, Haaskjold AA, Rottingen JA, Sundar S. Treatment options for visceral leishmaniasis: a systematic review of clinical studies done in India, 1980-2004. *Lancet Infect Dis* 2005; **5**: 763-74.
5. Sundar S. Drug resistance in Indian visceral leishmaniasis. *Trop Med Int Health* 2001; **6**: 849-54.
6. Guerin PJ, Olliaro P, Sundar S, et al. Visceral leishmaniasis: current status of control, diagnosis, and treatment, and a proposed research and development agenda. *Lancet Infect Dis*. 2002; **2**: 494-501.
7. Sundar S, Mehta H, Suresh AV, et al. Amphotericin B treatment for Indian visceral Leishmaniasis: conventional versus lipid formulations. *Clin Infect Dis* 2004; **38**: 377-83.
8. Sundar S, Jha TK, Thakur CP et al. Low-dose liposomal amphotericin B in refractory Indian visceral leishmaniasis: a multicenter study. *Am J Trop Med Hyg* 2002; **66**: 143-6.
9. Drugs for Parasitic Infections. *Med Lett Drugs Ther*; August 2004. Online: [www.medletter.com/freedocs/parasitic](http://www.medletter.com/freedocs/parasitic).
10. Sudar S, Agrawal G, Rai M, Makharia MK, Murray HW. Treatment of Indian visceral leishmaniasis with single or daily infusions of low dose liposomal amphotericin B: randomized trial. *BMJ* 2001; **323**: 419-22.

## SYSTEMIC SCLEROSIS PRESENTING AS RAYNAUD'S PHENOMENON: A CASE REPORT

Sultana GA <sup>1</sup>, Mohammed ZRB<sup>2</sup>, Md AA<sup>3</sup>

<sup>1</sup>Department of Dermatology, Tairunnessa Memorial Medical College & Hospital, Gazipur, <sup>2</sup>Department of Dermatology, Cox's Bazar Medical College & Hospital, Cox's Bazar. <sup>3</sup>Department of Hepatology, Cox's Bazar Medical College & Hospital, Cox's Bazar

### Abstract

Raynaud's Phenomenon is a cause of significant morbidity in patients with systemic sclerosis (SSc). Both the small and large digital arteries are involved causing perfusion defects leading to ischemia. Microvascular disease causes intimal proliferation and luminal narrowing of small digital arteries, macrovascular disease causes narrowing or occlusion of larger digital arteries. Immediate clinical evaluation and treatment are mandatory at the onset of critical digital ischemia to prevent digital loss. This is a case report of a 31 year old female suffering from systemic sclerosis who presented with Raynaud's Phenomenon.

**Key Words:** Systemic sclerosis, Ischemia, Microvascular, Macrovascular, Raynaud's phenomena

### Introduction

Systemic sclerosis (systemic scleroderma) is a chronic connective tissue disease of unknown etiology that causes widespread microvascular damage and excessive deposition of collagen in the skin and internal organs.<sup>1</sup> The disorder is characterized by three features: tissue fibrosis, small blood vessel vasculopathy and a specific autoimmune response associated with autoantibodies.<sup>2</sup> Scleroderma is classified into two major subsets, diffuse and limited cutaneous sclerodermas, that are distinguished by the extent of skin thickening. Diffuse scleroderma is characterized by widespread skin thickening involving distal and proximal body regions; rapid onset (within 1 year) of skin and other features following appearance of Raynaud's

phenomenon; significant visceral involvement; high scores on disability and organ damage indices secondary to extensive fibrosis of tissues associated with antinuclear antibodies; and the absence of anticentromere antibody.

Limited scleroderma shows limited skin thickening, slow progression of disease and late visceral involvement, with unique features of isolated pulmonary hypertension and digital amputations associated with anticentromere antibody.<sup>2</sup> Systemic sclerosis presenting as Raynaud's phenomenon is sufficiently a rare presentation that merits being a subject of case report. We report a case of young Bangladeshi female presenting with Raynaud's Phenomenon in systemic sclerosis.

### Address for Correspondence

Dr. Gazi Asma Sultana, Associate Professor, Department of Dermatology, Tairunnessa Memorial Medical College & Hospital, Gazipur, Dhaka, Bangladesh. e-mail : keka3307@gmail.com

## Case Report

A 31 years old nonsmoker female patient from Gazipur with remote history of systemic sclerosis and recurrent hospital visits presented to the dermatology outdoor of Tairunnessa Memorial Medical college & hospital with history of skin dryness, cold sensitivity of fingers and tightness of the face. She had generalized body weakness also complained heartburn and dysphagia. The patient denied fever, chills, nausea, vomiting or any loss of blood. She reported irregular menstruation for the past three years and for the last six months she did not experienced any menstruation. She denied history of shortness of breath, recurrent sore throat and any chronic illnesses like hypertension, diabetes mellitus or bronchial asthma. There is no history of similar illness in her family. Before five years she developed gangrene in the little finger of the left hand and was admitted to the hospital. Along with full



**Fig. 1:** Ulceration on the tip of the left index finger.



**Fig. 2:** Tightness of the face.

investigations and management diagnosis was established as limited cutaneous scleroderma and was discharged home with medication, which patient couldnot mention. She had been following at the dermatology, cardiac and infectious clinics of the hospital with the periodically given medications. Physical examination revealed a chronically sick looking severely emaciated young woman afebrile with digital gangrene at left index and ring finger and right index finger and respiratory distress. The pulse was weak with normal blood pressure. She had vesicular breath sounds, mild hepatomegaly and extremities were mild edematous and cold with prolonged capillary filling. On cutaneous examination there was skin thickening, hyperpigmented patches with mask like face, erythematous macular lesions on the palmar and plantar areas along with hardening and cyanosis on the tip of the fingers with gangrene at the right index and ring finger and also index finger of right hand. The laboratory studies included a complete blood cell count, serum chemistry (e.g. serum total protein and albumin, electrolytes, liver, and kidney function, muscle enzymes and acute phase reactants) and urine analysis. All of the results were normal except for the lactate dehydrogenase (elevated to 785 U/L) and erythrocyte sedimentation rate (elevated to 88 mm/hr). Immunoglobulin and complement assays demonstrated elevated levels of IgG (2,340 mg/dL), IgE (932.0 IU/mL), C4 (58.52 mg/dL). However, there were normal levels of IgA, IgM and C3. Diagnostic laboratory profiles for rheumatological disease were performed. A speckled antinuclear antibody (ANA) pattern was observed with a very high titer of 1:320 and the level of anti-RNP antibody was elevated to 310.9 U/mL. However, the other autoantibodies were negative such as anti-Sm antibody, anti-double-stranded DNA antibody, anti-SS-A (Ro) antibody and anti-centromere antibody. In



addition, the RA factor and antiphospholipid antibody were also negative. The serology data and clinical characteristics supported the diagnosis of systemic sclerosis. According to the symptoms patient had no symptoms involving any specific organ, further studies to exclude suspected MCTD was done, which included chest x-ray, electrocardiography, echocardiography, pulmonary function testing, upper GIT endoscopy and kidney ultrasonography. The results of all of these studies were normal. To treat the Raynaud's phenomenon, slow release nifedipine was initially prescribed. But the symptoms did not improve with the nifedipine or with enalapril. Instead treatment with the steroid (Prednisolon) significantly decreased the frequency and severity of the RP and healed the ulcer on the tip of the fingers after a few months. In addition, the anti-RNP antibody decreased to 178 U/mL.

## Discussion

Scleroderma is a connective tissue disorder with multiorgan involvement. Numerous symptoms in scleroderma such as Raynaud's phenomenon, digital tip necrosis and angina are related to ischemic changes attributable to intimal thickening of medium and small arterial vessels in the absence of histopathologic changes of vasculitis.<sup>4</sup> Systemic sclerosis varies in severity and progression, ranging from generalized cutaneous thickening (systemic sclerosis with diffuse scleroderma) to a form distinguished by restricted skin involvement (often just fingers and face) and slow progression, often several decades, before full manifestation of characteristic internal involvement. This latter form is termed limited cutaneous scleroderma or CREST syndrome (calcinosis cutis, Raynaud's phenomenon, esophageal dysfunction, sclerodactyly, and telangiectasia).<sup>3</sup> The most common initial complaints in systemic sclerosis

are Raynaud's phenomenon manifested by episodic pallor followed by cyanosis and/or rubor of the distal portions of the digits after exposure to cold.<sup>4</sup> Raynaud's phenomenon often predates other manifestations in the limited subtype and is often found concurrently in diffuse systemic sclerosis.<sup>4-6</sup> The skin is almost always involved in systemic sclerosis. Induration is symmetric and may be confined to the fingers (sclerodactyly) and distal portions of the upper extremities, or it may affect most or all of the body. As the disease progresses, the skin becomes taut, shiny, and hyperpigmented; the face becomes masklike; and telangiectases appear on the finger, chest, face, lips, and tongue. Subcutaneous calcifications may develop (calcinosis circumscripta), usually on the fingertips (pulp) and over bony eminences.<sup>3,4,7</sup>

Evaluation should ideally start with meticulous clinical examination, palpation of peripheral pulses and assessing for persistent discoloration (cyanosis or pallor), digital ulceration, extreme tenderness, or frank gangrene. Nailfold capillaroscopic changes may be predictive of the development of digital ischemia.<sup>5,8</sup> Arterial Doppler, and Ankle brachial pressure index estimation is also helpful.

Laboratory analysis for prothrombotic states including the antiphospholipid antibodies (lupus anticoagulant, anti-cardiolipin, and anti-beta2-glycoprotein I antibodies), anti centromere antibody should be performed in all patients. Prompt clinical evaluation and referral for treatment is critical to the prevention of progression to digital loss.<sup>9</sup>

Angiographic evaluation for digital occlusions include conventional angiography, magnetic resonance angiography (MRA) or computed tomography (CT) angiography. Conventional angiography is extremely sensitive for identifying stenosis, occlusion, aneurysm, or other vascular irregularities and is still considered gold standard.<sup>6,7,10</sup>



**Reference**

1. Hinchcliff M, Varga J Systemic sclerosis/scleroderma: a treatable multisystem disease. *Am Fam Physician* 2008; **78**: 961-8
2. Arunachlam R, Thiriavium K et al. Scleroderma with crescentic glomerulonephritis: a case report. *J Med Case Report* 2008 13; 2, **151**: 1-5
3. Systemic sclerosis. In *The Merck Manual of Diagnosis and Therapy*. 17 ed. Edited by Mark H, Robert B. Publisher Merck and Co. USA. 1999: 110-165
4. Eisenberg ME, Nguyen BY, Karnath BM. Clinical Features of Systemic Sclerosis. *Hops Physic* 2008; 33-38.
5. M. Sebastiani, A. Manfredi, M. Colaci, R. Damico, V. Malagoli, D. Giuggioli, and C. Ferri: Capillaroscopic skin ulcer risk index: a new prognostic tool for digital skin ulcer development in systemic sclerosis patients. *Arthritis Care Res*. 2009; **61**: 688-694,
6. Acharya S, Shukla S, Thakare R, Mahjan SN. Systemic sclerosis presenting with severe digital ischemia. A rare case report. *IOSR J Dental Med Sci (IOSR-JDMS)*. 2014; **13**: 11-13.
7. Wigley FM. Clinical practice. Raynaud's phenomenon. *N Engl J Med* 2002; **347**: 1001-8.
8. Kawai S, Fukuda Y, Okada R. Atherosclerosis of the coronary arteries in collagen disease and allied disorders, with special reference to vasculitis as a preceding lesion of coronary atherosclerosis. *Jpn Circ J* 1982; **11**: 1208-21.
9. Planchon B, Pistorius MA, Beurrier P, De Faucal P. Primary Raynaud's phenomenon: age of onset and pathogenesis in a prospective study of 424 patients. *Angiology* 1994; **45**: 677-86.
10. Landry GJ, Edwards JM, McLafferty RB, Taylor LM Jr, Porter JM. Long-term outcome of Raynaud's syndrome in a prospectively analyzed patient cohort. *J Vasc Surg* 1996; **23**: 76-85.

## Peer Reviewers of this Issue

Prof (CC) Dr Md Mizanur Rahman  
MBBS, MD  
Department of Medicine  
Tairunnessa Memorial Medical College  
Gazipur

Prof (CC) Dr Mohammad Jubaidul kabir  
MBBS, DFM  
Department of Forensic Medicine & Toxicology  
Tairunnessa Memorial Medical College  
Gazipur

Prof dr Asma Kabir  
MBBS, MPH, M.Phil  
Department of community medicine  
Tairunnessa Memorial Medical College  
Gazipur

Dr Gazi Asma Sultana  
MBBS, DDV  
Associate Professor  
Department of Dermatology & Venerology  
Tairunnessa Memorial Medical College  
Gazipur

Prof (CC) Dr Sayeda Riya  
MBBS, MPH, M.Phil  
Department of Community Medecine  
Tairunnessa Memorial Medical College  
Gazipur

Prof Dr Abbas Uddin Khan  
MBBS, MD  
Department of Paediatrics & Neonatology  
Tairunnessa Memorial Medical College  
Gazipur



## **Tairunnessa Memorial Medical College & Hospital**

Konia, Gazipur-1704, Bangladesh

College Hotline : 01787028828, 01929493646

Hospital Hotline : 01914213134

Fax: 880-2-8316332,

E-mail : [tmch@citechco.net](mailto:tmch@citechco.net)