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# JOURNAL OF BANGLADESH COLLEGE OF PHYSICIANS AND SURGEONS

**Vol. 26, No. 1, Page 1 - 57** **January 2008**

## CONTENTS

## *EDITORIAL*

# Treatment of Hepatitis C Virus Infection

The hepatitis C virus (HCV) can cause both acute and chronic hepatitis. The acute process rarely causes hepatic failure, but usually leads to chronic infection. In contrast, chronic HCV infection clearly linked to advanced liver disease, hepatocellular carcinoma (HCC), and has become the leading indication for liver transplantation. Treatment of chronic HCV is aimed at slowing disease progression, preventing complications of cirrhosis, reducing the risk of HCC, and treating extrahepatic complications of the virus. The natural history of hepatitis C is quite variable. Up to 85% of patients with acute HCV eventually progress to chronic infection. Of them, 15-20% of patients will develop cirrhosis within 15-20 years.[1]

Candidacy for therapy of hepatitis C includes persons who are 18 years of age or older, are willing to be treated, and do not have contraindications to treatment if they have detectable HCV RNA in serum and evidence of chronic hepatitis suggested by elevated serum alanine aminotransferase levels or the presence of considerable necroinflammatory activity and fibrosis on liver biopsy. At present, the recommended therapy for chronic hepatitis C is a combination of formulations of interferon alfa and ribavirin (RBV).[2] Interferon alfa is a cytokine that has an important function in the innate antiviral immune response.[3] It acts by attaching to cell-surface receptors that signal through the system of Janus-activated kinase and signal transducers and activators of transcription, leading to induction of multiple interferon-stimulated genes.[4] These genes include double-stranded RNases, inhibitors of viral protein translation, and proteins that destabilize viral messenger RNA. Interferon alfa also induces the expression of genes involved in the immune response, resulting in activation of natural killer cells, maturation of dendritic cells, proliferation of memory T cells, and prevention of T-cell apoptosis.[5] Ribavirin is an oral nucleoside analogue with broad activity against viral pathogens.[3] Its mechanism of action against HCV is not completely clear. Ribavirin appears to have minimal direct activity against HCV replication,[6] but it may lead to rapid and lethal mutation of virions or depletion of intracellular guanosine triphosphate, which is necessary for viral RNA synthesis.[7,8] Ribavirin also has immuno modulatory effects.

Definitions[9] of response to treatment are: rapid virological response (RVR) is defined as non-detectability of serum HCV RNA (<50 IU/mL) after 4 weeks of therapy, early virological response (EVR) is defined as undetectable HCV RNA (<50 IU/mL) or at least a 2 log decrease in serum HCV RNA from baseline level after 12 weeks of therapy, end-of-treatment virological response (ETVR) is indicated by non-detectability of HCV RNA at the end of therapy, sustained virological response (SVR) is defined as undetectable serum HCV RNA (<50 IU/mL) 24 weeks after the end of therapy. SVR has been shown to have the following beneficial effects: (i) fibrotic regression; (ii) substantially reduced rate of HCC; (iii) decreased rate of other complications, including liver failure and liver-related death; and (iv) improved quality of life. The most recent important advance in the treatment of hepatitis C was the development of a long-acting interferon, pegylated interferon (peginterferon), produced by the covalent attachment of polyethylene glycol to the interferon molecule. With its increased half-life, peginterferon can be given as a weekly dose.[10] Two peginterferon formulations are currently approved for the treatment of hepatitis C: alfa-2a (Pegasys, Roche) and alfa-2b (Peg-Intron, Schering-Plough). In two large trials of these agents, the rates of sustained virologic response to a 48-week course of peginterferon and ribavirin were 54 and 56%, as compared with 44 and 47% with standard interferon and ribavirin and only 29% with peginterferon alone.[11,12] Response rates were higher among patients with genotype 2 or 3 than among those with genotype 1. A subsequent trial of different regimens of peginterferon alfa-2a and ribavirin showed that patients with genotype 2 or 3 could be treated with a lower dose of ribavirin (800 mg rather than 1000 to 1200 mg daily) and that the rates of sustained virologic response after 24 weeks of therapy (81 and 84%) were similar to the rates after 48 weeks of therapy (79 and 80%).[13] The following have been shown to influence treatment outcome: (i) age; (ii) sex; (iii) virus genotype; (iv) virus load; and (v) stage of fibrosis, especially F3, F4.

There are few absolute contraindications for use of peg-IFN-a and ribavirin. They include- present or

past psychosis or severe depression, uncontrolled seizures, hepatic decompensation, pregnancy (RBV), renal failure (RBV), severe heart disease (RBV). The relative contraindications for IFN and ribavirin are history of depression, uncontrolled diabetes mellitus, uncontrolled hypertension, retinopathy, psoriasis, autoimmune thyroiditis or other active autoimmune disorders including autoimmune hepatitis, symptomatic heart disease or severe vascular disease (RBV), anemia/ischemic vascular disease (RBV). In addition to these contraindications special caution is required if IFN is administered in the circumstances like neutropenia (neutrophil count <1,500 cells/cmm), thrombocytopenia (platelet count <85,000/cmm), organ transplantation, history of autoimmune disease, presence of thyroid autoantibodies, age older than 65 years. Side-effects related to IFN include: cytopenia, abnormalities of thyroid function, depression, irritability, concentration and memory disturbances, visual disturbances, fatigue, muscle aches, headaches, nausea and vomiting, loss of appetite and weight, low grade fever and skin irritation, insomnia, hearing loss, tinnitus, interstitial fibrosis and hair thinning. Side-effects associated with ribavirin include hemolytic anemia, fatigue, itching, rash, cough, gastrointestinal upset, pharyngitis, gout and birth defects. Serious side effects of combination therapy occur in 1 to 2% of patients, and permanent injury and death can occur.[14] There are several areas of uncertainty in the treatment of hepatitis C like use in children, patients >65 years and/or with significant comorbidities, body mass index >30 and hepatic steatosis, persistently normal serum ALT, acute hepatitis C, patients with minimal histologic evidence of liver disease, genotype 4-6 infections, decompensated cirrhosis, failed to respond or have relapsed with prior HCV therapy, substance abuse disorders including alcoholics, HIV/HCV coinfection, renal disease including haemodialysis, history of solid organ transplantation.

Efforts have been made to develop new molecules for better treatment outcomes. Potential targets and approaches in the next 5 years are on long acting IFN, direct antiviral agents like polymerase and protease inhibitors, ribavirin analogues, immunomodulators and therapeutic vaccines. Peginterferon alpha and ribavirin represents the best current treatment available. Major limitations are cost and sensible side-effects. However SVR rates are promising that can even ensure hepatitis C a curable disease.

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***(J Bangladesh Coll Phys Surg 2008; 26: 1-2 )***

**References:**

1 Poynard T, Bedossa P, Opolon P. Natural history of liver fibrosis progression in patients with chronic hepatitis C. Lancet 1997; 349: 825-32.

2 National Institutes of Health Consensus Development Conference Statement: management of hepatitis C: 2002 — June 10-12, 2002. Hepatology 2002; 36: Suppl 1: S3-S20.

3 Feld JJ, Hoofnagle JH. Mechanism of action of interferon and ribavirin in treatment of hepatitis C. Nature 2005; 436: 967-72.

4 Sen GC. Viruses and interferons. Annu Rev Microbiol 2001; 55: 255-81.

5 Tilg H. New insights into the mechanisms of interferon á: an immunoregulatory and anti-inflammatory cytokine. Gastroenterology 1997; 112: 1017-21.

6 Lau JY, Tam RC, Liang TJ, Hong Z. Mechanism of action of ribavirin in the combination treatment of chronic HCV infection. Hepatology 2002; 35: 1002-9.

7 Maag D, Castro C, Hong Z, Cameron CE. Hepatitis C virus RNA-dependent RNA polymerase (NS5B) as a mediator of the antiviral activity of ribavirin. J Biol Chem 2001; 276: 46094-8.

8 Crotty S, Maag D, Arnold JJ, et al. The broad-spectrum antiviral ribonucleoside ribavirin is an RNA virus mutagen. Nat Med 2000; 6: 1375-9. [Erratum, Nat Med 2001; 7: 255.]

9 APASL hepatitis C working party. Asian Pacific Association for the Study of the Liver consensus statements on the diagnosis, management and treatment of hepatitis C virus infection. J Gastroenterol Hepatol 2007; 22: 615-633.

10 Glue P, Fang JWS, Rouzier-Panis R, et al. Pegylated interferon-á2b: pharmacokinetics, pharmacodynamics, safety and preliminary efficacy data. Clin Pharmacol Ther 2000; 68: 556-67.

11 Manns MP, McHutchison JG, Gordon SC, et al. Peginterferon alfa-2b plus ribavirin compared with interferon alfa-2b plus ribavirin for initial treatment of chronic hepatitis C: a randomised trial. Lancet 2001; 358: 958-65.

12 Fried MW, Shiffman ML, Reddy KR, et al. Peginterferon alfa-2a plus ribavirin for chronic hepatitis C virus infection. N Engl J Med 2002; 347: 975-82.

13 Hadziyannis SJ, Sette H Jr, Morgan TR, et al. Peginterferon alfa-2a and ribavirin combination therapy in chronic hepatitis C: randomized study of treatment duration and ribavirin dose. Ann Intern Med 2004; 140: 346-55.

14 Hoofnagle JH and Seef LB. Peginterferon and Ribavirin for Chronic Hepatitis C. N Engl J Med 2006; 355: 2444-2451.

## ORIGINAL ARTICLES

# Expectation of Patients from Doctors

MN ABSAR[a], MH RAHMAN[b]

**Summary:**

***Doctors are produced to meet the health need of the people. People, as a patient have many expectations from a doctor. Doctors often don't know those expectations. The objective of this study was to find out the expectations of the patients from doctors and the goal is to make the doctors aware of these expectations so that they give due attention to those expectations. Waiting patients in GPs' clinic and the patients waiting in the out patient department of the Upozilla Health Complex and Rangpur Medical College Hospital were interviewed to know their expectation from doctors. Focus group discussion was done with community people to know their expectation from doctors when they need health care. It was found that most people expects good treatment(70.70%), greetings from doctor(51.91%), good history taking(100%), attentiveness(78.02%),to be allowed to narrate their ailments without interruption(87%), privacy(75%), clarification of prescription(75.15%), referral if needed(98%), dietary advice(67%), information about the ailment(75.79%), partnership in decision making(72.92%) and confidentiality(46.49%).***

***(J Bangladesh Coll Phys Surg 2008; 26: 3-9)***

**Introduction:**

Patients are the clients of doctors. Health service provided by the doctors is the commodity which a patient receives as a client. It is being gradually recognized by the professionals that the service given to the patient should have the concordance with the expectation of the patients[1,2]. Satisfaction of the patient largely depends on their expectation and recognition of their concern and expectation is important, because expectation drives people to seek health care for their symptoms[3,4]. Satisfaction, moreover has positive impact on patients compliance keeping on follow up visit[2]. It is quiet impressive to observe that patients are more satisfied and shows considerable improvement in symptoms and functional states when their expectations are met properly[5,6].

Studies revealed the fact that patient's expectations are often ignored by physicians. It is a matter of concern in primary care practice that physicians, "rarely take into account their patients' most important expectations, even when they have been made aware of them"[7].

The explicit desire or request for health caring services are usually interpreted as expectations[6]. More precisely, "patients' expectation are anticipations that given events are likely to occur during or as a result of medical care"[1].

Dimensions of expectations are many. Richard and his colleagues studied expectations of internal medicine patient for care during office visit which revealed that most important expectations are as follows[8].

| | |
|---|---|
| History & Examination: | |
| Family history | 49% |
| Personal history | 35% |
| ENT examination | 55% |
| Lung auscultation | 65% |
| Abdominal palpation | 54% |
| Cardiac auscultation | 68% |
| Laboratory: | |
| Cholesterol test | 38% |
| Blood test | 52% |
| Radiology | 20% |
| ECG | 22% |
| Urine test | 31% |
| Medication: | |
| Pain pills | 13% |
| Antibiotic | 16% |
| Other medication | 32% |
| Counseling: | |
| Diet & exercise | 38% |
| Discussion about how to manage condition | 71% |
| Smoking | 18% |
| Provide prognostic information | 69% |
| Stress counseling | 40% |
| Referral: | 52% |

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Valori et al showed that despite socio demographic contrast patients' expectations are of almost identical components[9]. These are i). Explanation and reassurance, ii). Emotional support, iii). Investigation and treatment[9].

Patient's dissatisfaction is often rooted in unmet expectation; rational or irrational. Almost each patient comes to the physician with some perception, past experience and accumulated knowledge. These often influence their expectation[10]. There is almost universal agreement that one important responsibility of physician is to meet patient's needs and expectations.

Developing countries has started to reform their existing system of medical education in a way to make it more people oriented so that they really address the expectation of the community people. We may have to think in this context. So, we have to know our peoples expectation beforehand. This study is intended to know the expectations of our patients and people, both in community and health facilities.

**Method of Study:**

**a). Study design:**
It is a qualitative cross-sectional study.

**b). Study place:**
i). OPD of Rangpur Medical College Hospital
ii). OPD of Upozilla Health Complexes (UHC)of Rangpur District.
iii). Rural and Urban community of Rangpur District.

**c). Study population:**
i). Patients attending OPD of Rangpur Medical College Hospital.
ii). Patients attending OPDs of UHCs of Rangpur District.
iii). Community people of rural and urban residential areas of Rangpur District.

**d). Sample size:**
i). Patients attending OPDs = 314.
ii). Community people = 6 groups of 10 peoples.

**e). Data collection:**
i). Patient's interview; An open ended questionnaire was used to interview the patients waiting in OPDs. Patients had been interviewed before they met the doctor.
ii). Focus group discussion; A guide line was prepared for focus group discussion(FGD) in the community. Six groups consisting of 10 people in each group were discussed with according to the standard method of FGD.
Data was collected by one data collector who was trained before hand.

**f). Study period :**
June2001 to December2001.
Patients of all discipline except psychiatry were included in the study.
Data were analyzed manually.
Overall study design was consulted with expert group formed by WHO.

**Results:**

**Patients' interview;** Total number of patient was 314. 73% was male and 26% female. Their educational status and occupation are shown in Table-I. Mean age of the patients was 34.12±12.53 yrs.(Table-II). Expectation of patients from doctor which appeared more frequently in the response were, good treatment, cure from the ailment, good behavior, good medicine, good caring attitude, good advice, adequate examination and attention to the

**Table-I**

*Demography of interviewed patients (n = 314)*

| Category | No. (%) |
|---|---|
| Sex: | |
| Male | 231(73.6) |
| Female | 83(26.4) |
| Education: | |
| Illiterate | 50(15.9) |
| Up to HSC & equivalent | 212(67.5) |
| Graduate & equivalent | 31(9.9) |
| Post graduate | 21(6.7) |
| Occupation: | |
| Service | 56(17.8) |
| Business | 44(14) |
| Student | 50(15.9) |
| Other(house wife, cultivator, labour, etc.) | 164(52.2) |

**Table-II**

*Age of the interviewed patients (n= 314)*

| | |
|---|---|
| Maximum age | 80 yrs |
| Minimum age | 18 yrs |
| Mean age | 34.12 yrs |
| Std. Deviation | 12.53 |
| Median age | 32 yrs |
| Mode | 30 yrs |

patient(Table-III). Expectations in specific dimensions are shown in Table-IV . More than 50% patients want that they should be greeted by the doctors and should be offered seat. Attentiveness to patient's statement is expected by 78% patients. About 60% patients expected that they should have the opportunity to stay with the doctor for about 16.5min. in average. However, 40% said that stay time depends on the nature of the disease. About 87% patients desired to talk to doctor without interruption and talk about anything they want to. 48% patients desired to be examined privately in separate space. 75% patients wanted prescription to be made clear by the doctor by verbal narration. Almost all(99%) patients stated that laboratory investigation should be done if it is necessary for the diagnosis. 98% of the patients want that doctors should refer the patient to the next appropriate person when he fails to understand the problem. 67% patients are in opinion that dietary advice should be given to the patient and about 56% expect health advice from doctors. 75% patients expressed the expectation to know about their disease and prognosis. 72.92% patients felt that decision about treatment strategy should be taken jointly by doctor and patient. 46.49% patients are in opinion that their personal information should not be disclosed to anybody else. However 51.59% is in favor of disclosing information to others if necessary in the interest of the patient. Facilities expected in the waiting place are, cleanliness, sitting arrangement, fan, toilet facility, drinking water, light etc.(Table-V ). There is a big list of criteria of a good doctor in patients consideration which is shown in Table-VI .

**Table- III**

*General expectations of patients from doctor (Terms used are patients' own language) (n=314)*

| Expectations | No. of respondents(%) |
|---|---|
| Good treatment | 222(70.70) |
| Good medicine | 45(14.33) |
| Cure from ailment | 77(24.52) |
| Good behavior | 54(17.19) |
| Attention to patient | 12(3.82) |
| Adequate examination | 14(4.45) |
| Good caring attitude | 27(8.59) |
| Good advise | 26(8.28) |
| Low cost treatment | 03(0.95) |
| Appropriate diagnosis | 08(2.54) |
| Low consultancy fee | 02(0.63) |
| Cordiality | 02(0.63) |
| Availability | 05(1.59) |
| Adequate time for consultation | 01(0.31) |
| Sympathy | 02(0.63) |

**Focus group discussion (FGD):** On free discussion in focus group almost uniform opinion was found. The expectations expressed by the groups during discussion is compiled bellow. These, in their language are;

"1).We want to get doctors easily and at our arms length in need. 2). We want cheap treatment. 3). We want cordiality and good behaviour from our doctor. 4). Doctor should have up to date knowledge. 5). Doctor should see the patient taking more time. 6). Patient should be allowed to tell every thing he wants to say to his doctor. 7). Diagnosis should be correct. 8). Doctor's fee should be less. 9). Doctor should see the patient attentively and listen to the patient attentively and should not be engaged in other activities like talking to others, taking tea etc. 10). We want good treatment and care. 11). Doctor should pay equal attention to all patients irrespective of social status. 12). Doctor should reveal all facts about disease to his patient and involve patient in decision making. 13). If doctor fails to cure the patient he should refer the patient to appropriate person or place. 14). A doctor should not see more than 15 to 20 patients a day. 15). The staff of doctor should be well behaved.16). A doctor should be more humane and his attitude should not be business man like. 17). Doctor should greet the patient and be more sympathetic to patient. 18). A doctor should talk more with the patient. 19). Doctor should treat the patient

**Table-IV**

*Expectation in specific dimension (n=314)*

| Dimensions of expectation | No. of respondents (%) |
|---|---|
| First interaction: | |
| Doctor should greet the patient | 163(51.91) |
| Doctor should ask the patient to sit | 185(58.91) |
| Doctor should ask about ailments | 64(20.38) |
| History and clinical examination: | |
| Doctor should ask about what happened to me | 314(100) |
| Doctor should examine the patient | 91(28.98) |
| Doctor should ask for laboratory investigation | 20(6.36) |
| Doctor should listen attentively | 245(78.02) |
| Doctor should note down the ailments of the Patient | 23(7.32) |
| Stay time with doctor depends on disease | 126(40.12) |
| Doctor should not interrupt patient while narrating | 274(87.26) |
| Doctor may interrupt if feels things are not Relevant | 38(12.10) |
| Doctor should examine patient privately | 151(48.08) |
| Prescription should be narrated verbally | 236(75.15) |
| Verbal narration of prescription is not necessary | 16(5.09) |
| Investigations should be done only if necessary | 311(99.04) |
| If doctor do not understand the problem of the patient he should refer the patient to specialist/Hospital | |
| Other advice, patient expect from a doctor: | 309(98.40) |
| Dietary advice | |
| Health advice | 212(67.51) |
| Advice for next visit | 175(55.73) |
| Preventive knowledge | 15((4.77) |
| What else to be done apart from medicine | 22(07) |
| Family problem | 04(1.27) |
| Family planning advice | 01(0.31) |
| Spiritual healing | 06(1.91) |
| No additional advice is necessary | 01(0.31) |
| Information about nature and prognosis of the disease: | 64(20.38) |
| Nature and prognosis should be informed to patient | |
| Nature and prognosis need not be explained to patient | 238(75.79) |
| No response | 71(22.61) |
| Participation of patient in decision making: | 05(1.59) |
| Only doctor will decide about treatment strategy | |
| Only patient will decide the treatment strategy | 84(26.75) |
| Doctor and patient both will take part in decision making | 01(0.31) |
| Secrecy of patients private information: | 229(72.92) |
| Doctor should keep it secret | |
| May reveal to others in patient's interest | 146(46.49) |
| Don't care | 162(51.59) |
| 06(1.91) | |

**Table-V**

*Facilities expected in waiting place*
*(This is the overall extract of the expectations of patients)*

| | | | |
|---|---|---|---|
| 1. | Cleanliness | 12. | Air cooler |
| 2. | Sitting arrangement | 13. | Drinking water |
| 3. | Fan | 14. | Magazine |
| 4. | Toilet | 15. | News paper |
| 5. | Light | 16. | Attendant |
| 6. | Table | 17. | Water glass |
| 7. | TV | 18. | Locker for keeping patients' belongings |
| 8. | Bed for seriously ill patient | 19. | Toys for children |
| 9. | Adequate space | 20. | Telephone |
| 10. | Separate arrangement for male and female | 21. | Arrangement for food |
| 11. | Good ventilation | 22. | Quiet environment |

**Table-VI**

*Criteria of a good doctor in the opinion of the patients*
*(This is an overview of the criteria set by the patients)*

- Good behavior
- Honesty
- Politeness
- Sound professional knowledge
- Higher degree
- Attentive to patient
- Low consultancy fee
- Ability to cure patient
- Sympathetic to patient
- Available in need
- Give good treatment
- Give enough time to patient
- Diagnose correctly
- Smartness
- Reassure patient
- Good professional skill
- Cordiality
- Give good advice
- Examine patient properly
- Gives good medicine
- Gives correct medicine
- Punctuality
- Nonsmoker
- Enough experience
- Humane ness
- Give equal attention and care to every patient
- Patience
- Show honor to patient
- Healthy
- Pious
- Foreign degree
- Gives low cost treatment
- Non threatening attitude

considering them as his own family member. 20). Doctor should examine the patient properly. 21). Doctor should be a life long learner and he should do research. 22). Doctor should have a good degree(not specified). 23). Doctor should reassure patient. 24). Doctor should be skilled. 25). Doctor should not smoke. 26). Doctor should prescribe good medicine(medicine which works good) and should not prescribe medicine which is not available in the market and he should narrate properly how to take medicine. 27). In emergency doctor should take quick action and he may seek other's help if necessary. 28). Doctor should show patience and answer all queries of a patient. 29). Patients should be asked to come for follow up and doctor should write letter to the patient to come for follow up if patient does not come. 30). Doctor

patient relation ship should be very friendly. 31). Doctor should not hurt the patient during examination. 32). Doctor should have high moral character. 33). Doctor should advise about diet and other good health habits. 34). Unnecessary investigations should not be done.

**Discussion:**

Expectation of our patients as expressed during their interview and focus group discussion is almost same as the expectations of the patients elsewhere in the world. They want good behaviour, cordiality, sympathy, advise in different health aspects including diet and prevention, availability of doctor in need, caring attitude, proper history taking and clinical examination, correct diagnosis and treatment, degree, knowledge and skill of doctor, prompt and easily available and appropriate emergency service, information about disease , confidentiality , low cost treatment, referral, ownership in decision making. Referral advice is one important expectation of the patients often ignored[3,6,8]. Verbal narration of the prescription, health information and advice about the diet are the expectations frequently observed. Other studies also revealed that the patients give much importance on these expectations[6,8,11]. These expectations should be taken care of adequately. It is quite likely that doctors concentrate more on examining and diagnosing the ailments and feels free of any responsibility by giving only the written order(prescription) to the patient which contains only some medicine. They are likely to be careless about other necessary advice like diet, relevant health information and how to take drugs. In fact it needs talking to patients adequately and appropriately. This is an avenue of patient- physician communication. This also includes explanation about disease and it's prognosis. These are also expected by the patients frequently. This is observed in our study and in many other studies also[5,8,9,12,13,14]. Most patients expect to be involved in decision making along with doctors. This is our observation and other studies also have the same observation[11, 15,16]. In 1980 the supreme court of Canada suggested that physicians have a legal obligation to disclose and explain the treatment and letting the patients make their own choice[17]. Confidentiality is a major area of patients' expectation. In our study a good number of patients also expressed the view that the secret information may be revealed to others if necessary for patient's interest. In a study by Jung, Wensing and Grol, GPs and patients agreed equally that patients' information should be kept secret[11]. Investigation for diagnosis is advised frequently by doctors. Majority of the patients' expectation is that laboratory investigations should be done if it is necessary for diagnosis. In other studies also laboratory test emerged as a significant expectation of the patients[3,18,19].

Patients has to wait for a considerable time in the waiting room of the concerned doctor. Logically waiting facilities is a general concern of the clients. In our study patients have expected to have many items present in waiting place. It is a natural human instinct to get as much as possible and to demand it. However some of the facilities demanded by the patients need to be considered with importance. These are; adequate space, cleanliness, comfortable sitting arrangement, fan, toilet, safe drinking water, adequate illumination and ventilation, good manner of staffs and quiet environment.

The criteria of a good doctor is also remarkable. Good behavior, humaneness, knowledge, cordiality, honesty etc. are the attributes of a good doctor indeed.

Patients expect that the doctor will be attentive to the patient, allow the patient telling his ailment as much as the patient desires, charge minimum fee, have empathy to patient. Above all a doctor should be a good friend and should be trustworthy and he should be available in emergency situation. Apparently there are many expectations which can only be met if a doctor is a missionary. In real life situation a doctor is an ordinary human being. He has all the personal priorities for his family members. He is a professional rather than a missionary. But as he is a service provider he should take consideration of what his clients(patients) expect form him. However, we have to see what the doctors' attitude is towards the expectation of the patients. We have to look at the expectation and reality gap. Then we can see how this gap can be shortened if not eliminated.

**Conclusion:**

It is obvious from the above discussion that the expectations of our patients are almost same as that of others in different parts of the world. Expectations are

of many folds and dimensions. These expectations needs to be addressed adequately. As the satisfaction and expectation goes parallel, to give highest satisfaction to the patient, doctors should address as much expectation as possible. However we have to see how doctors can accommodate best with these expectations. So, we should create awareness among the doctors as regard to the expectation of the patient from doctors.

**Acknowledgment:**
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**References:**

1. Uhlmann RF, Inui TS, Carter WB. Patient requests and expectations. Medical care 1984; 22(7): 681-85.
2. Jackson JL, Kroenk K. Patient satisfaction and quality care. Military medicine 1997; 162(4):273-77.
3. Marphe RL, Kroenk K, Lucey CR, Wilder J, Lucas CA. Concerns and expectations in patients presenting with physical complaints. Arch Intern Medicine 1997;157(jul 14): 1482-88.
4. Uhlmann RF, Carter WB, Inui TS. Fulfillment of patient request in a general medicine clinic. American journal of public health 1984;74(3):257-58.
5. Jackson JL, Kroenk K. The effect of unmet expectations among adults presenting with physical symptoms. Ann Int Med 2001; 134(9):889-94.
6. Joos SK, Hickman DH, Borders LM. Patients' desire and satisfaction in general medicine clinics. Public health reports 1993; 106(6): 751-59.
7. Sanchez-Menegay C, Stalder H. Do physicians take into account patients' expectation? Journal of general internal medicine 1994; 9(jul): 404-6.
8. Kravitz RL, Cope DW, Bhrany V, Leake B. Internal medicine patients for care during office visits. J Gen Int Med 1994; 9(Feb): 75- 81.
9. Valori R, Wloshynowych M, Bellenger, Auvihare, Salmon P. The patient request form: A way of measuring what patients want from their general practitioner. Journal of psychosomatic research 1996;40(1):87-94.
10. Kravitz RL, Callahan EJ, Paterniti D, Antinius D, Dunhum M, Lewis CE. Prevalence and sources of patients' unmet expectation for care. Ann Intern Med 1996; 125(9): 730-37.
11. Juang HP, Wensing M, Grol R. What makes a good general practitioner: do patient and doctors have different views? British journal of general practice 1997; 47(December):805-9.
12. Deyo RA, Diehl AK. Patient satisfaction with medical care for low back pain. Spine 1986; 11(1): 28-30.
13. Gillette RD, Kues J, Harrigan JA, Franklin L. Does the family physician's role correspond to the patients' expectation? Family medicine 1986; 18(2):68-72.
14. Sancheg-Menegay C, Hudes ES, Cummings SR. Patient expectation and satisfaction with medical care for upper respiratory infections. J Gen Intern Med 1992; 7(July/August):432-35.
15. Strull WM, Lo B, Charles G. Do patients want to participate in medical decision making? JAMA 1984; 252(21): 2990-94.
16. Rubin HR, Gandek B, Rogers WH, Kosinski M, Horney CA, Ware JE. Patients' rating of outpatient visits in different practice setting. JAMA 1993; 270(7): 835-40.
17. Chefe SMJ. Legal obligation of physicians to disclose information to patients. Can Med Assoc J 1991; 144(6):681-5.
18. Broody DS, Miller SM, Lerman CE, Smith DG, Lazaro CG, Blum MJ. The relationship between patients' satisfaction with their physician and perception about interventions they desired and received. Medical care 1989; 27(11): 1027-35.
19. Jackson JL, Kroenk K, Chamberlin J. Effects of physician awareness of symptoms-related expectation and mental disorders. Arch Fam Med 1999; 4(Mar/Apr): 135-42.

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# Frequency of Hypomagnesemia in Hospitalized Diabetic Hypokalemic Patients

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**Summary:**

***Recent studies suggest that there is strong relationship between serum magnesium and diabetes. Low serum magnesium is one of the risk factors of diabetes mellitus and its complications, at the same time diabetes is one of the common causes of hypomagnesemia. Hypokalemia is also quite common in diabetic patient. When hypokalemia coexists with hypomagnesemia, the chance of 'complications of hypokalemia' increase significantly. In addition, the correction of hypokalemia becomes difficult. However, the exact frequency of hypomagnesemia in diabetic hypokalemic patient is not yet defined. Therefore, the objective of the current study was to find out the frequency of hypomagnesemia in hospitalized diabetic hypokalemic patient. Thirty consecutive diabetic patients with hypokalemia admitted under medical unit 1 BIRDEM were included in this study. There were 20 Female; and 10 male, mean age was 52.33±12.97 years, duration of diabetes was 1 - 20 years, mean serum potassium was 2.37± .36m mol/l, The mean ± SD of serum magnesium was 0.67 ±0.26 m mol/l. Hypomagnesemia was present in 19 patients (63.3%). Fifteen normokalemic diabetic patients were taken as control. Only one subject had Hypomagnesemia in control. Sample mean of serum magnesium has been found significantly lower than general population (P=.001) and control ((P=.034)***

***Key ward: Hypokalemia, hypomagnesemia, diabetes, frequency***

***(J Bangladesh Coll Phys Surg 2008; 26: 10-13)***

## Introduction:

The recent World Health Organization (WHO) report on diabetes prevalence alarmed that diabetes has posed a serious threat to entire population of the world . This trend observed two folds increase in the developed and almost three folds in the developing nations. The increasing prevalence of diabetes in underdeveloped countries is hypothesized that under-nutrition either in terms of trace element or micronutrient deficiency or of chronic energy deficiency (CED) may be a etiologic factor of diabetes in this population. Recent studies identified magnesium as the most important micronutrient associated with diabetes. The incidence of subclinical magnesium deficiency is common in diabetes and cardiovascular disorders. Magnesium deficiency has recently been related with age-related diseases through free-radical mechanism [1]. The existence of oxidative stress has been well documented in diabetes and late diabetic complications. The prevalence of hypomagnesemia has been found to vary widely, depending on the patient's clinical condition. In a general population, 6.9% of patients were shown to be hypomagnesemic.[2] In hospitalized patients on a medical-surgical floor, there was a prevalence of 11%,[3] while in the intensive care unit it was found to be 20%.[4] In a postoperative intensive care unit setting, the prevalence was 60%.[4] A study of diabetic patients established a prevalence of 25%.[5] In a recent study in Bangladesh, Khan LA showed that almost 70% of patients with MRDM had clinically defined hypomagnesemia. Of the patients who had type 2 DM not related to malnutrition, 42% exhibited hypomagnesemia. [6] Hypokalemia is also common in diabetic patients. Diabetes itself can cause hypokalemia or it can result from treatment of diabetes and its complications. Hypokalemia may present as a medical emergency, and in many case it coexists with hypomagnesemia, especially in diabetic

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patients. Hypomagnesemia is present in about 40% of diuretic treated hypokalemic patients.[7] When both exist together not only the correction of hypokalemia become difficult, the chances of acute complications also increase significantly.[8] Therefore, this study was carried out to evaluate the prevalence of hypomagnesemia in hospitalized diabetic hypokalemic patients.

Hypothesis

Prevalence of hypomagnesemia is significantly higher in hospitalized diabetic hypokalemic patients than normal population.

**Aims and Objectives:**

1. To find out frequency of hypomagnesemia in 'hospitalized diabetic hypokalemic patients.
2. To correlate the frequency of hypomagnesemia in 'hospitalized diabetic hypokalemic patients with that of normal population.

Subjects

Sample size 30

Place of study- medical unit-1, BIRDEM

Diabetic patients with general medical illness.

Inclusion criteria: Known diabetic patients with serum potassium less than 3mmol/l on admission, under medical unit 1 of BIRDEM Hospital during the period of August 2003 to January 2004' were recruited in the study.

Exclusion criteria: Patients admitted with diabetic ketoacidosis and hyperosmolar non-ketotic coma.

**Controls:**

Fifteen diabetic normokalemic age matched adult, both male and female patients admitted under medical unit-1 were randomly selected (eight from female ward and seven from male ward) as control.

**Methods:**

Laboratory procedures: All tests were done in BIRDEM biochemistry lab. For magnesium Roche Diagnostic GmbH, D-68298 Mannheim, Germany, supplied assay reagent. Reference range of Serum magnesium - 0.65-1.05 m mol/l. Serum potassium was assayed by ion probe method. Reference range of Serum potassium - 3.5-5.2 m mol/l

Statistical methods: Data were entered into and analyzed with 'Statistical Packages for Social Sciences' (SPSS) version 11.0 program. Value was expressed as frequency, percentage, ratio, mean ± standard deviation (SD), median, and range. Serum magnesium values were analyzed using The One-Sample T Test. Population mean of serum magnesium 0.85-m mol/l was used as specified constant [9].

**Results:**

Total 30 patients were included in this study. male female ratio were 1:2.minimam age was 30 years and maximum was 79years.the mean age was 52.33years(SD 12.97).Duration of diabetes were 1 to 20 years, mean 8.85 years (SD 5.13), four patients were only on diet control, seven patients were on oral hypoglycemic agent, and 19 patients were on insulin. Micro vascular complications were present in 22 patients. Diabetic retinopathy was in 18 patients, Diabetic nephropathy was in 14 patients and Diabetic neuropathy was in 8 patients (Table-1). Macro vascular complications were present in eight patients (Table-2). Hypertension was present in 16 patients (53.3%). ECG changes typical of hypokalemia was present in 17 patients (56%), most of them had hypomagnesemia.(Table-3) Out of 19 patients with hypomagnesemia, 14 had typical ECG changes (73.7%) whereas only three patients (27.3%) out of 11 with normal magnesium level had ECG changes. Only moderate to severe hypokalemic patients (serum potassium less than 3mmol/L) were included in this study. Mean potassium level of selected subjects was 2.37mmol/L with standard deviation of ± 0.36. Minimum and maximum values were 1.80mmol/L and 2.90mmol/L respectively. (Table-4)Seventeen patients (56.7%) were severely hypokalemic (serum potassium<2.5m mol/l) and 13 patients (43.3%) moderately hypokalemic (serum potassium <3.0mmol/l-2.5mmol/l). Magnesium level was checked in all cases. The mean ± SD was 0.67mmol/l ± 0.26.(Table-4) Hypomagnesemia was present in 19 patients (63.3%).(Figure-1) The lowest and highest values of serum magnesium level were 0.40 m mol/L and 1.40 m mol/L respectively(table-4). The sample mean was analyzed against population mean (0.85m mol/l) and controls mean by using one sample test and found significantly lower, (P =.001) and (P=.034) respectively(table-5,6). Serum magnesium level had no relationship with duration of DM, (P=0.211) (Figure - 2). In Hypomagnesemic patients, Mg level did not

correlate with K level (P=0.829).(Figure-3) Out of fifteen controls, eight were female and seven were male. The Mean ± SD of serum potassium & serum magnesium level of control were respectively 4.5±0.556 m mol/l and 0.78±0.120 mmol/l. The lowest and highest values of serum potassium level were respectively 3.9 m mol/l and 5.9 m mol/l. The lowest and highest values of serum magnesium level were 0.6 m mol/l and 1.1 m mol/l respectively. (Table-7) Hypomagnesemia was present in one subject in control.

**Table-I**

*Frequency of microvascular complications (n =30)*

| Microvascular complications | Frequency | Percent |
|---|---|---|
| Diabetic retinopathy | 18 | 60% |
| Diabetic nephropathy | 14 | 46.7% |
| Diabetic neuropathy | 8 | 26.7% |

**Table-II**

*Frequencies of macro vascular complications (n =30)*

| Macro vascular complications | Frequency | Percent |
|---|---|---|
| IHD | 5 | 16.7% |
| CVD | 4 | 13.3% |
| PVD | 0 | 0% |

**Table-III**

*ECG change (n =30)*

| Hypomagnesemia | | Frequency | Percent |
|---|---|---|---|
| Present | Yes | 14 | 73.7 |
| | No | 5 | 26.3 |
| | Total | 19 | 100 |
| Absent | Yes | 3 | 27.3 |
| | No | 8 | 72.7 |
| | Total | 11 | 100 |

**Table-IV**

*Serum potassium and serum magnesium in sample (n =30)*

| | Lowest value | Highest value | Mean | SD |
|---|---|---|---|---|
| Serum Potassium | 1.80 mmol/L | 2.90 mmol/L | 2.90 mmol/L | ± 0.36 |
| Serum Magnesium | 0.40 mmol/L | 1.40 mmol/L | 0.67 mmol/L | ± 0.26 mmol/L |

**Table-V**

*Correlation of serum magnesium between sample and general population*

| | Mean | |
|---|---|---|
| Sample(n=30) | 0.67mmol/l | (P=.001) |
| General population (n=15) | 0.85mmol/L | |

**Table-VI**

*Correlation of serum magnesium between sample and control*

| | Mean | SD | |
|---|---|---|---|
| Sample(n=30) | 0.67mmol/l | ± 0.26 | (P=.034) |
| Control(n=15) | 0.78mmol/L | ±0.120 | |

**Table –VII**

*Serum potassium and serum magnesium in control (n =15)*

| | Lowest value | Highest value | Mean | SD |
|---|---|---|---|---|
| Serum Potassium | 3.9 mmol/L | 5.9 mmol/L | 4.5 mmol/L | ± 0.556 |
| Serum Magnesium | 0.6 mmol/L | 1.1 mmol/L | 0.78 mmol/L | ± 0.120 mmol/L |

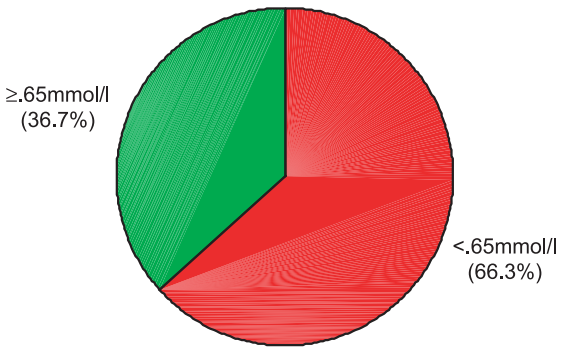


**Fig.-1:** *Magnasium status of study population*

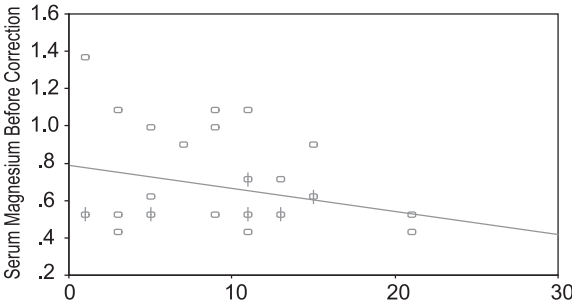


**Fig.-2:** *Correlation between serum magnesium &duration of diabetes*

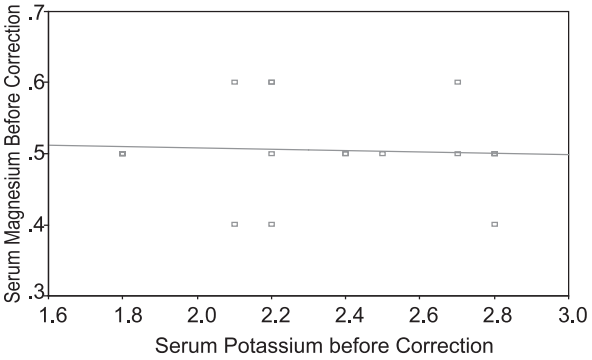


**Fig.-3:** *Correlation between serum magnesium & serum potassium*

**Discussion:**

Age and sex distribution in this study reflects age and sex distribution of patients admitted into medical unit 1 of BIRDEM. It has nothing to do with serum magnesium or serum potassium level. Mean diabetic duration of the study population was 8.85 years. Serum magnesium level has no direct relationship with diabetic duration if the DM is well controlled. Hypomagnesemia occurs in DM subjects due to loss of magnesium through urine because of glycosuria. In hypomagnesemic patients, no correlation was observed between serum potassium and serum magnesium level (Pearson's correlation co-efficient, r = -0.053, P=0.829). (Figure-3) As there is no other study on this topic, non-relationship observed here cannot be explained. However, there may be some non-linear relationship between them, which cannot be commented using Pearson's test. The frequency of hypomagnesemia is high in this study (63.3%) compared to other studies. However, subjects were different in those studies. So far no study has been found through journal search involving diabetic hypokalemic subjects. In a general population, 6.9% of patients were shown to be hypomagnesemic.[2] In hospitalized patients on a medical-surgical floor, there was a prevalence of 11%,[3] while in the intensive care unit it was found to be 20%.[4] In a postoperative intensive care unit setting, the prevalence was 60%.[4] A study of diabetic patients established a prevalence of 25%.[5] In a recent study in Bangladesh. Khan LA showed that almost 70% of patients with MRDM had clinically defined hypomagnesemia. Of the patients who had type 2 DM not related to malnutrition, 42% exhibited hypomagnesemia. [6] Sample mean of serum magnesium has been found significantly lower than general population (P=0.001) and control (P=.034). It is expected, as because, DM is a direct cause of hypomagnesemia and hypomagnesemia can be a cause of DM. Moreover, as the study subjects were all hypokalemic, many of these hypokalemia might have been the consequence of hypomagnesemia.

**Conclusion:**

Hypomagnesemia is a frequent finding in hospitalized diabetic hypokalemic patients & needs controlled clinical trial to see the 'effect of magnesium supplementation in correcting potassium level in diabetic hypokalemic patients' without checking magnesium level.

**References:**

1. Saito N, Nishiyama S. Aging and magnesium. Clin Calcium 2005; 15(11):29-36.
2. Whang R, Hampton EM, Whang DD.(Guerrero-Romero and Rodriguez-Moran 2006) Magnesium homeostasis and clinical disorders of magnesium deficiency. Ann Pharmacother 1994; 28:220-225
3. Wong ET, Rude RK, Singer FR. A high prevalence of hypomagnesemia in hospitalized patients. Am J Clin Pathol 1983; 79:348-352
4. Ryzen E, Elbaum N, Singer FR. Parenteral magnesium tolerance testing in the evaluation of magnesium deficiency. Magnesium 1985; 4:137-147
5. Mather HM, Nisbet JA, Burton JH. Hypomagnesemia in diabetes. Clin Chim Acta 1979; 95:235-242
6. Khan LA. Serum and urinary magnesium in young diabetic subjects in Bangladesh. Am J Clin Nutr 1999;69:70-3.
7. Kramer, B.K. and D. Endemann. Cardiac risks of hypokalemia and hypomagnesemia. Ther Umsch, 2000. 57(6): p. 398-9.
8. Hannedouche, T. and A. Delgado.Hypokalemia, hypomagnesemia and ventricular arrhythmia during diuretic treatment of arterial hypertension. Arch Mal Coeur Vaiss, 1988. 81(6): p. 819-24.
9. Wester Po. Magnesium, Am J Clin Nutr 1987; 45: 1305-1312

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# Dynamics of Spirulina In Promoting Health Benefits For Arsenicosis Patients

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**Summary:**

***A total number of 40 patients affected with arsenicosis were selected and clinically examined prior to feeding trials. The interrelatedness between improvement respondents following spirulina intake in respect of age, sex, nutrition and social condition of patients exposed to arsenic contaminated water was evidenced. Among sex-wise respondents about 62% females showed improvement in comparison to 58.3% males. The spirulina intake caused more improvements in age group 15-35 years (66.66%) than patients of 35 to 55 years (50%). The impact of spirulina improvement showed a different vulnerability of social taboos. It occurred 71.42% in middle class, while in poor class this was 69.29%. The greatly vulnerable poverty related malnourished arsenicosis patients responded to therapy equally as attained by well nourished patients. The overall response revealed that 60% patients showed improvement with spirulina treatment which was statistically highly significant (x2 = 8.64 at P< 0.01).***

***The viability of spirulina to offer health benefits to arsenicosis patients proved considerably satisfactory, because there was significant improvements of general health in all patients who received spirulina during the experimental period. Although 60 to 70% recovery has been recorded with spirulina intake in arsenicosis patients, but it is still interesting to note that the drug assisted remedy from malnutrition and might have boosted up the immune system. The present research study evidenced that arsenic, which induces cellular toxicity, could be prevented by treatment with known supportive treatment, such as spirulina along with other antioxidants.***

***(J Bangladesh Coll Phys Surg 2008; 26: 14-21)***

## Introduction:

With more than an estimated 20 million of its 125 million people assumed to be drinking arsenic contaminated water, Bangladesh is facing, what is perhaps the largest poisoning in history. According to WHO some 70 million people are alarmingly at risk of consuming arsenic contaminated water of tube wells, the main source of portable water of the 60 out of 64 districts [1]. The vibration 'arsenic engulfing Bangladesh' has recently been highlighted in the report of the United Nations University (UNU) in Tokyo and the Dhaka based Earth Identity Project prompted recognition of its major impacts affecting agriculture and other related industries, water management, public health and overall national economy [2, 3]

Arsenic acts as a silent killer which is undetectable in its early stage of arsenic poisoning and takes between 8 and 14 years to impact on the health depending on the amount of arsenic ingested, nutritional status and immune response of the individual[4, 5, and 6.7]. To date, several thousands of patients with arsenic related skin diseases have been found in the first limited surveys[2].Bangladesh Government is however promoting surveillance and helping to strengthen data collection and Bangladesh Arsenic Mitigation and Water Sanitation Project (BAMWSP) is focusing on risk reduction strategies Drugs used for chelating arsenic in chronic Arsenicosis are tried. Use of antioxidants has shown evidences of improvement and topical use of salicylic acid has been found effective in reducing pain and roughness of keratosis [8]. Recently *Spirulina*, blue-green algae developed by Bangladesh and French scientists has been found to have very good effects on people suffering from

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Arsenicosis [9 and 10]. Leading dermatologists of the country unanimously recommended *Spirulina* to treat arsenic patients [11].

The present research study undertaken focuses on the future use of *Spirulina* in the diet program for general maintenance, supportive treatment and nutritional improvement. The chief objective is to confirm whether arsenic patients given *Spirulina* show significant improvement and exhibit potential health benefits.

**Material and method:**

*Type of study*: The type of study undertaken was based upon randomized sampling and observational double blind trial

*Duration of study*: six months

*Study population*: A total number of 40 patients having suspected skin lesions of Arsenicosis were selected randomly during the period of January 2004 to July 2004 at the Dermatology Venereology Department of Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka, Bangladesh.

*Patients' selection*: Patients who attended the arsenic clinic belonging to the Department of Dermatology and Venereology of Bangabandhu Sheikh Mujib Medical University with typical clinical manifestation of chronic arsenicosis as per standard instruction and recommendation.

*Inclusion criteria*:

1. Patients who had given verbal consents and were willing to comply with this study process
2. Arsenicosis patients of both sex groups between 15 to 55 years
3. Patients who did not receive any treatment (either systemic or topical) prior to one month of the study
4. Patients having history of consumption of arsenic contaminated water
5. Patients who had hyper-pigmentation or leuko-melanosis in the trunk and extremities, keratosis in palms and soles and other typical signs and symptoms of chronic arsenicosis

*Exclusion criteria*:

1. Patients who refused to be included in the study
2. Patients having age below 15 years and above 55 years of age
3. Pregnant women and lactating mothers
4. Arsenicosis with complications and malignancy
5. Patients who are to take regular antibiotics or steroids for other major systemic illnesses.

Pre-feeding examination: Clinical examination of arsenic affected patients prior to feeding trials

Post-feeding examination: Clinical examination of arsenic affected selected patients periodically after spirulina feeding trials.

Source of samples: Clinical samples of the study were obtained from selected patients belonging to affected areas under investigation

Collection and transportation of samples: Arsenicosis patients were asked either to come to the hospital or they were visited as per schedule of the study. Clinical samples for the study were collected and processed as per standard recommendation.

Feeding of spirulina samples: Spirulina samples in the form of capsule or other means were fed to 20 arsenicosis patients. Capsules were prepared as per recommendation. Spirulina samples manufactured by Lifeline International under the supervision of BCSIR Li No. Sachi/ppp1 (314)/90/4737) may be given in the form of 10-gram powder, dissolved in water, and given daily in divided doses.

Feeding of placebo: Placebo prepared similar to spirulina were fed to other 20 arsenicosis patients

**Method:**

Selection of patients: A total number of 40 patients affected with suspected arsenicosis were selected for this study. They were placed under two equal groups. The patients and physicians were fully unaware of the above treatments intended for the study. The patients for this study were between 18 to 55 years of age.

**Data of information and history of pre-fed patients:** Data of information were collected from all arsenic exposed patients prior to the above feeding trials and recorded.

**Data of information and history of post-fed patients:** In post-fed group both placebo and spirulina treated patients were subjected to analogous study as mentioned earlier in prefed patients. Here the clinical examination was performed after every 15 days of feeding trials. Comparative nature and extent of clinical manifestations appearing and other

improvement occurring and not occurring were recorded. After every three months all postfed patients were examined to detect any changes in improvement. The duration of post-feeding was limited for one year.

*Advice to patients:* All 40 selected arsenic patients were advised to use strictly surface water for domestic works and take arsenic free drinking water during the total period of the study.

A checklist in the form questionnaire consisting of patients data was evaluated prior to feeding and after feeding. The questionnaire format is presented in fig 2.

*Grading of severity of chronic arsenicosis and assessment of response to spirulina:*

This is done and evaluated by calculating the total scores obtained from 'Questionnaire format'. Ascending total scores indicate severity of infection, while descending scores refer to less to no infection. The numerical 0 indicates no clinical signs or symptoms or no improvement from spirulina. Negative signs of numericals, such as - 1, - 2, - 3, - 4 and - 5 used indicate severity of infection as less severe, fairly severe, moderately severe, severe and highly severe respectively. Analogously the degree of improvement from spirulina treatment is calculated using the positive numericals, such as + 1, + 2, + 3, + 4 and +5 which stand for less, slight or partial, moderate, good and very good improvement. The total score allotted for 20 clinical features are 100, each feature having maximum of 5 scores. Improvement is noticed when the total score after treatment ranges between 1 to 100 and no improvement was recorded when the total score was 0 and the difference of evaluation before and after treatment stands between 0 and -100.

*Diagrammatic illustration of the research plan:* The plan of the research is presented below diagrammatically in figure 1.

Statistical analysis was done in which clinical scores were evaluated. Frequency distribution, Chi square test, Yate's correction and other software were employed for determining significant correlation between observations

*Response to treatment:* Clinical examination of arsenic affected patients prior to treatment trials was recorded. Patients were clinically examined for noticing any improvement that occurred after every two weeks. Nutritional status of patients was assessed using MAC (mid arm circumference) value.

**Result:**

The present study demonstrated the interrelatedness between improvement of respondents following spirulina intake and age, sex, nutrition and social condition of arsenicosis patients exposed to arsenic contaminated water. Table 1 represented that the impact of responses of patients to spirulina differed among male and female personnel. The sex-wise distribution of respondents to the treatment of arsenicosis with spirulina evidenced that about 62% females exhibited improvement in comparison to 58.3% males. None of the females got improvement with placebo treatment, whereas 20% males got some improvement.

**Table- I**

*Sex-wise respondents of chronic arsenicosis patients to spirulina treatment (n=40)*

| Treatment | Improvement (%) in males | Improvement (%) in females |
|---|---|---|
| Placebo (n=20) | 21.48 | 0 |
| Spirulina (n=20) | 58.33 | 62.50 |

It is evident from table 2 that the spirulina intake caused improvement in patients more in age group 15-35 years (66.66%) than patients of age group 35to 55 years (50%). The respondents to treatment with spirulina although was found about 16% more in younger age group, but with placebo treatment no obvious improvement occurred. The respondents with placebo treatment were 11.11% and 18.18 % in older and younger age groups respectively.

**Table-II**

*Age-wise respondents of chronic arsenicosis patients to spirulina treatment (n=40)*

| Treatment | Improvement (%) in age group 15 - 35 years (n=20) | Improvement (%) in age group 36- 55 years (n=20) |
|---|---|---|
| Placebo (n=20) | 18.18 | 11.11 |
| Spirulina (n=20) | 66.66 | 50.00 |

The impact of arsenicosis on the susceptibility of the poor to arsenicosis and vulnerability to social taboos represent a different phenomenon, when the patients were given treatment with spirulina. The result is presented in table 3. The spirulina improvement that occurred in middle class people was 71.42%, while in poor class this was 69.29%. In case of placebo treatment the socioeconomic class did not play any

role in the improvement of arsenicosis patients. The percentage distributions of improvement recorded in poor class and middle class arsenicosis patients were 14.28 and 16.66 respectively.

**Table-III**

*Relationship between Socioeconomic status of patients and spirulina intake (n=40)*

| Socio-economic status of patients | Improvement (%) after Placebo treatment (n=20) | Improvement (%) after spirulina treatment (n=20) |
|---|---|---|
| Poor class | 14.28 | 69.29 |
| Middle class | 16.66 | 71.42 |

The interrelatedness of nutritional status and spirulina intake as presented in Table 4 demonstrated that the nutritional level of arsenicosis patients contributed to the response of improvement. The greatly vulnerable poverty related malnourished 60% arsenicosis patients responded to improvement equally as shown by 60% well nourished patients.

**Table IV**

*Interrelatedness of nutritional status with spirulina intake of arsenicosis patients pas (n=40)*

| Nutritional status | Improvement (%) after Placebo treatment (n=20) | Improvement (%) after spirulina treatment (n=20) |
|---|---|---|
| Well nourished | 50 | 60 |
| Malnourished | 11.11 | 60 |

The overall improvement and response of 40 chronic arsenicosis patients to treatment with spirulina are presented in table 5. It is worthy to note that 60% patients showed considerable improvement with spirulina treatment, whereas 15% patients got improvement with placebo treatment. It is clearly evident from $x^2$ test that there is a strong association between spirulina intake and improvement status among patients with arsenicosis. That is spirulina intake certainly caused improvement and the result is found statistically highly significant ($x^2 = 8.64$ at $P < 0.01$)

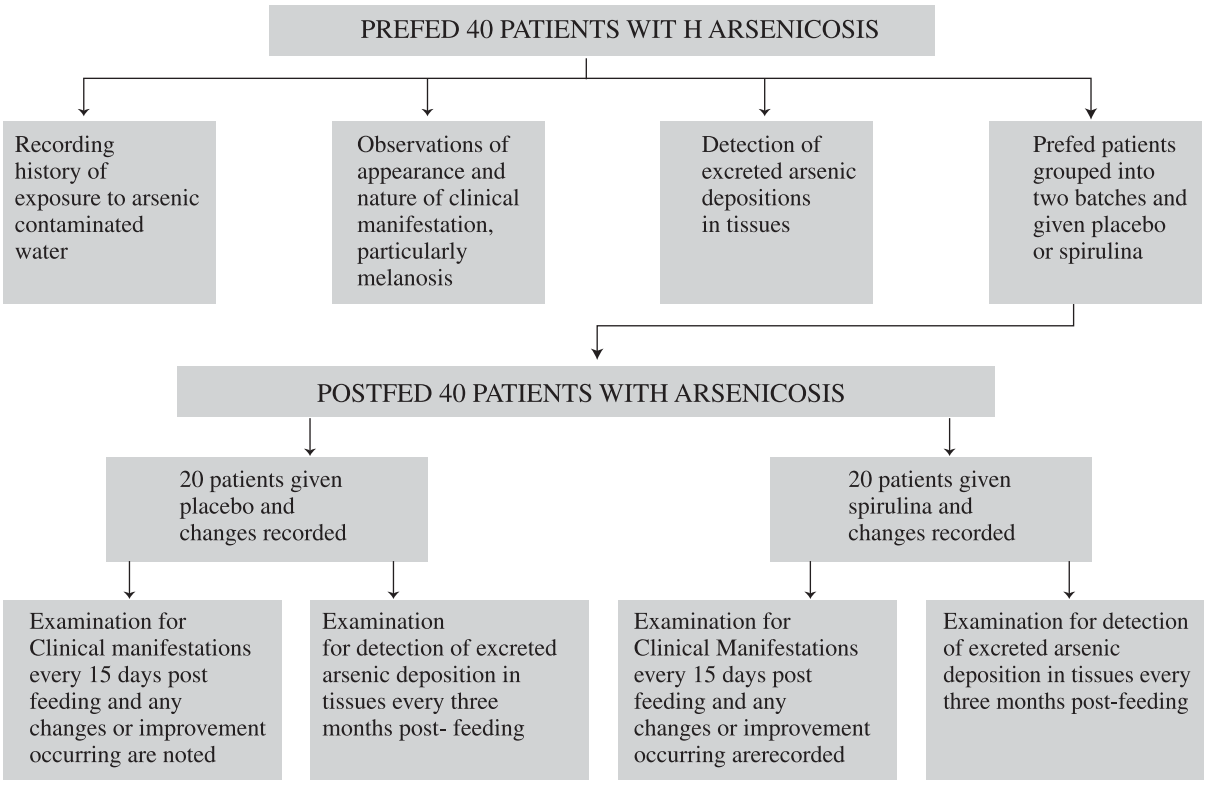


**Fig.1:** *Schematic illustration of the experimental study*

Name Age Sex Address

Source of drinking water F.U. for source of drinking water

(Arsenic contaminated)

Nutritional status: Wt. Malnourished Well-nourished

Socio-economic status: poor class Middle class High class

H/O past illness (if any):

Drug history:

Personal habit: Smoking Alcohol intake

Hyper-pigmentation: *Scoring* ① Diffuse over palm and sole Raindrop pigmentation or leucomelanosis of upper trunk ③ Raindrop pigmentation in whole trunk

Hyperkeratosis: *Scoring* ① Diffuse in palm and sole spotted keratosis, palm and sole

Marked spotted keratosis with fissuring and marked thickening

| Clinical symptoms | Evaluation before treatment | Evaluation after treatment |
|---|---|---|
| Weakness | | |
| Weight loss | | |
| Anorexia | | |
| Conjunctival congestion/ Burning eyes | | |
| Cough/ Dyspnea | | |
| Abdominal discomfort/ pain | | |
| Hyperhydrosis | | |
| Burning hands and feet | | |
| Headache | | |
| Palpitation | | |
| Tingling and numbness of limbs | | |
| Total Score | | |
| Clinical signs | | |
| Evaluation before treatment | | |
| Evaluation after treatment | | |
| Raindrop pigmentation | | |
| Hyperkeratosis | | |
| Anemia / Pallor | | |
| Edema | | |
| Jaundice | | |
| Nail changes | | |
| Hair changes | | |
| Cutaneous malignancy | | |
| Gangrene | | |
| Total score | | |

**Fig.-2:** *Questionnaire Format*

**Discussion:**

Extensive pollution of the major drinking water source (underground tube well water) with high levels of arsenic has recently been recognized as an important public health hazard in Bangladesh[12], as a result the incidence of morbidity and mortality associated with arsenicosis calamity is increasing day by day [13, 14 and 15]. Since specific and useful treatment of chronic arsenicosis is still not available, Bangladesh researchers are seeking for effective therapeutic strategies to find out new modalities of treatment [16]. Recent development in molecular biology and the state-of-the-art-knowledge indicate that application of antioxidants (selenium, vitamin C, vitamin E, β-carotene etc) in the treatment of arsenic toxicity may be useful. The present study evaluates and validates the therapeutic efficacy and health benefit of the β-carotene containing spirulina in the treatment of chronic arsenicosis victims.

Spirulina (A*rthrospira platensis)* is a microscopic spiral shaped, blue green vegetable alga is the richest source of beta-carotene and mixed carotenoids in the world [17, 18 and 19]. Scientific reports indicated that spirulina is nature's richest whole-food source of phytocyanin, an immune stimulant [8, 20 and 21]. It has been consumed by millions of people of all ages in the U.S. and more than 40 countries for over 20 years.

Spirulina is now available in tablets, capsules and powder. The dark green powder can be blended into fruit and milk drinks or added to recipes to boost nutritional value. The administration of spirulina in the present study showed significant improvement without any notable toxicity. Khan et al [22] led to opinions that the resultant clinical arsenicosis depends on chemical and physical form of the compound and the route by which it enters the body, the dose and the duration of exposure, age, sex and socioeconomic condition of the affected individuals. The result of this study evidenced that irrespective of age, sex, nutritional status and socio-economic category the spirulina caused about 60 to 70% improvement among individuals affected with chronic arsenicosis. Guha Mazumder [23] earlier found that the high protein diet could play role in the alleviation of symptoms of arsenic poisoning, because it enhanced the excretion of arsenic in urine by increasing methylation in the body. Many researchers also demonstrated that the protein of spirulina might be beneficial in promoting cell growth and repairing the damage of liver and Kidney tissue.

The study undertaken revealed that the treatment orally with spirulina has been associated with clinical regression of arsenic keratosis. This improvement is due to the fact that spirulina contains high amount of gamma linoleic acid (GLA), which is a precursor for the body's prostaglandin in master hormone that controls many body functions. Rabbani at al [12] demonstrated that the parameters of arsenic toxicity and endogenous antioxidants could be brought towards normal or favorable conditions much better by giving a recipe of vitamins, zinc and selenium. Since spirulina is the nature's richest food source of entire antioxidant spectrum, including beta-carotene, vitamin E, selenium, methionine, copper, manganese, zinc, B-vitamin etc., it has therefore clearly proved that this protein diet has fought against the oxidizing free radicals caused by pollution, malnutrition, stress and injury to arsenicosis patients[10, 24, 25,26 and27]

The detoxification goal of arsenic toxicity should be to neutralize the poison in the body or eliminate it from the body and repair the damage to organs and cells[12,28and 29].It is revealed from the present study that arsenicosis patients belonging to older age group, 36 - 55 years improved a little more (50%) than the younger patients of the group 15 – 35 years (33.33%) in the spirulina intake population. The explanation may be thought to be due to the fact that older people were more careful and responsible to take medicine and followed the health care instruction more appropriately than the younger. It may be believed that in the older age group the spirulina intake could have enhanced more bio-transformation of inorganic arsenic to less toxic organic arsenic, by rapid elimination of arsenic from the body and preventing tissue deposition and by increasing the ability of body's antioxidative defensive system

Studies on arsenicosis indicated that malnutrition increases the susceptibility to arsenicosis if the undernourished persons are exposed to arsenic contamination through drinking water. The therapeutic efficacy of spirulina in the treatment of arsenicosis patient suggested that malnourished patients showed marked improvement of their physical weakness and felt better after treatment. Similar improvement is found in reducing skin manifestations of arsenicosis patients who received

spirulina[10, 22, 25 and 27]. Recently the leading dermatologists of the country advocated for the beneficial effect of spirulina and recommended it as a great whole food alternative to isolated vitamins and minerals. It is already agreed by researchers [18, 19 and 30] that spirulina contains the most remarkable concentration of nutrients known in any food, plant, grain or herbs.

As regards the sex-wise relationship with the beneficial effect of spirulina intake by chronic arsenicosis patients, it is clearly demonstrated in this study that the improvement occurred after treatment did not find any such relationship remarkably. Although most of the arsenic affected victims are males of low income group was recorded by Watanabe et al[31] , but Ali et al[32] found more social awareness among females on arsenic contamination of water as assessed by Huq et al [33], which revealed that more females (32.58%) than male respondents (27.27%) could mention the cardinal signs of arsenicosis correctly. Interestingly more female personnel (70.99%) opined correctly that arsenic contamination is a problem of considerable length than the males (41.82%). This awareness among females' amounts to more mobilizing for avoiding the source of arsenic contaminated tube well water. The women after receiving treatment with spirulina used surface water for washing, cooking and other domestic works. The response of spirulina treatment was more or less same in both sexes, revealing the fact that low social status in the family or low education or low income of the females cannot be justified to be correlated with the improvement of treatment, rather the consciousness or awareness plays vital role in getting remedy from arsenicosis.

The result of the present study and reports available from earlier workers[10, 26, 33] evidenced that the use of spirulina showed significant symptomatic improvement without any notable toxicity. Moreover it can be consumed easily by mixing with rice or any other foodstuff. The in-vitro study of Chowdhury et al [26] has clearly shown that spirulina could act as a chelating agent when arsenic containing urine is passed through a column chromatography containing spirulina. Momtaj and Hussein [27] indicated a hospital based clinical trial with spirulina and demonstrated improvement in reducing skin manifestations of patients of arsenicosis who received 10 gm spirulina daily for four months. It was further found that higher dosage of such drug for outpatients and shorter recovery times might be possible.

Since spirulina intake has proved to offer health benefits for problems of arsenicosis, the present study also advocates that spirulina should be made available and blended into fruit or milk drinks or added to recipes to boost nutritional value, which will increase health and energy. In India spirulina known as "Spiru-Om" is well accepted by the children. It is given in extruded noodles, sweetened with sugar to preserve the beta-carotene[30]. In 1974 at the world food conference by the UN spirulina was declared to be the best food for tomorrow[34]. It is commercially produced all over the world including Mexico, California, India, Japan, Thailand, Brazil, and Vietnam. Considering the potential health benefit of the algae the BCSIR has successfully cultured Spirulina. The Government should think and give due attention to spirulina cultivation for utilization of the product locally by the malnourished as well as people suffering from arsenicosis and arsenicosis like problem.

**Conclusion:**

It may therefore be concluded that the use of Spirulina could reverse the conditions and restore the patients to normal life. However long term extensive studies are imperative to establish confidently the viability of spirulina for the treatment of arsenicosis. The mechanism of action of spirulina in the treatment and management of chronic arsenicosis if could be known in greater magnitude and could be driven at for setting up its potential benefit , then the present arsenicosis crisis in the form of calamity would be minimized in the near future.

**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.2000; 78(9): 1093-1103
2. Haque, MM. Panite Arsenic Tarale Garal (in Bengali) Arsenic in water, poison in liquid. Monograph, Published by Mrs. Nargis Akhter, House no. 92 (A-1), Road no. 11/A: Dhanmondi residential area, Dhaka 2002: pp 1-58
3. McLellan, F. Arsenic contamination affects millions of Bangladesh. Lancet. 2002; 358: 127
4. Abernathy, CO, Calderon RL, Chappal, WR 1997. Arsenic Exposure and Health Effects 1997; Chapman and Hall, London: Ist ed. pp. 1-429
5. Sikder, MS, Maidul, AZM, Ali, M. Rahman, MH. Socio-economic Status of Chronic Arsenicosis Patients in Bangladesh.Mymensingh Med. J 2005; 14(1): 50-53

6. Ahmad, K. Report highlights widespread arsenic contamination in Bangladesh. Lancet. 2001; 358: 133

7. Ahmed, M.F. An assessment of arsenic problems in Bangladesh: In International workshop on arsenic mitigation, Dhaka, 2002: pp. 15-20

8. Dillon, JC, Phuc, AP, Dubacq, JP. Nutritional value of the alga spirulina. World Rev. Nutr. Diet. 1995; 77, 32-46

9. Kabir, I. Effect of spirulina on induced chronic arsenicosis in rat. Thesis, 2000; BSMMU

10. Sikder, MS, Maidul, AZ.M, Khan, MAK, Huq MA, Choudhury, SAR, Misbahuddin Effect of Spirulina in the treatment of chronic arsenicosis.Bangladesh J. Dermatol. Venereol. Leprol. 2000; 17: 9-13

11. Islam, AZMM.Spirulina- Personal communication; 2003

12. Rabbani, GH, Das, HK Hossain, A, Ali, SMK, Nasir, M, Chowdhury, AKA Bangladesh Environmental Crisis: Mass arsenic poisoning through contaminated drinking water. 29th Annual Conference, Global Health Council, 2002; May 28-31, Washington, D.C. pp. A 48-A 49

13. Barkat, A, Maksud, AKM, Anwar, KS, Munir, AKM .Social and economic consequences of arsenicosis in Bangladesh. Bangladesh environment; 2002 vol. 1, BAPA, pp. 216-233

14. Jakariya, M, Chowdhury, AMR, Hossain, Z, Rahman, M, Sarker, Q, Khan, RI , Rahman, M. Sustainable community-based safe water options to mitigate the Bangladesh arsenic catastrophe – an experience from the upzilas. Current Sci. 2003. vol. 85, no.2, 141-146

15. Sikder, MS, Rahman, MH, Maidul, AZM, Khan, MSU, Rahman, MM. Study on the histopathology of Chronic Arsenicosis. Journal of Pakistan Association of Dermatologists. 2004: 14, 205-209

16. Ahmed, M.F, Minnatullah, KM, Shamsuddin, AL and Ahmed, SA Alternative water supply options. In: Arsenic mitigation in Bangladesh (Ed. Ahmed, M.F. and C.M. Ahmed); 2002: pp 81-174, LGD, LGRD&C, Dhaka

17. Annapurna, VV, Deosthale, G. Bamji, MS. Spirulina as a source of vitamin A. Plant foods Human Nutrition. In International Workshop of Arsenic Mitigation, January 14-16, 1991; 41(2): 125-134

18. Belay, A. and Ota, Y. Current knowledge on potential health benefits of SpirulinaJ. Appl. Phycol. 1993; 5: 235-241

19. Fox, R.D. Spirulina – production and potential. 1996; Published by Edisud, LaCalade, Aixen- Province, France

20. Kay, R.A. Microalgae as food and supplement In Critical Reviews on Food Science and Nutrition. 1991;30 (6): 555-571 Published by CRC Press, USA

21. Ciferri, O. Spirulina, the edible organism In Microbiological Reviews. 1983; 2, Dec.: pp 551-578

22. Khan, MAK, Choudhury, SAR. Misbahuddin, M., Maidul, AZM. Sahjahan, M. Effects of Spirulina in the treatment of Chronic Arsenic poisoning in Bangladesh Bang. J. med. sci. 2001; Vol.7, no.1. (Cited in the 'Research Studies on Health Impact of Arsenic Exposure', BMRC, 2002, p 223-231)

23. Guha Mazumder, DN. Treatment of arsenic toxicity as observed in West Bengal J. Int. Med. Asso. 1986 ; 94 (2) : 41-42

24. Hopenhayn-Rich, C., Biggs, ML, Smith, AH, Kalman, DA, Moore, LE Methylation study of a population experimentally exposed to arsenic in drinking water .Environ Health Perspective. 1996; 104: 620-628

25. Huq, IH, Sultana, N., Correll, R. Naidu, R. Arsenic in food chain; 4th International Conference on Arsenic Contamination of Ground water in Bangladesh: cause, effect and remedy. 2002; 12-13 Jan., Dhaka Community Hospital, Bangladesh and School of Environmental Studies, India (cited in 'Research studies on Health Impact of Arsenic Exposure', BMRC, 2002. p 317 : Bang. J. Physiol. Pharmacol. 16(1), 15-16

26. Chowdhury, UK, Biswas, BK, Chowdhury,TR, Samanta,G, Mandal, BK, Basu, GC, Chanda,CR, Lodh, D, Saha,,KC, Mukherjee, S, Roy, S, Kabir,S, Quamruzzaman, Q.Chakraborti, D. Ground water Arsenic Contamination in Bangladesh and West Bengal, India. Environmental Health Perspectives.2000; 108 (5): 393-497

27. Momotaj, H. and Hussain, AZM. Effect of Spirulina on Arsenicosis Patients in Bangladesh.Paper presented in the International Conference at the Columbia University, New York, 2001; November 26-27

28. Rabbani, GH and Saha, SK. Chronic arsenic toxicity through contaminated drinking water in Bangladesh: Magnitude of the problem, health effects and detoxification. The Orion. 2000; 11: 3-7

29. Saha, KC. Arsenicosis in West Bengal (Environmental problems and solution) Sadananda Prakashani, 2002; pp: 1-17

30. Seshadri, CV. Large scale nutritional supplementation with spirulina alga All India Coordinated Project on Spirulina, 1993 Shri Amm Murugappa Chettiar Research Center (MRCC), Madras, India

31. Watanabe,C, Inaoka,T, Kadono,T Nagano, M., Nakamura, S., Ushijima, K., Murayama, N., Miyazaki, K., Ohtsuka, R Males in rural Bangladeshi communities are more susceptible to chronic arsenic poisoning than females: analysis based on urinary arsenic .Environ Health Perspect. 2001; 109: 1265-1270

32. Ali, SMK, Edib, K, Pramanik, MMK, Alam, AMS,Rabbani, GH, Anwar, KS, Hossain, A, Nasir, M, Saha,SK Nutritional status of patients with arsenicosis in rural Bangladesh.Bangladesh Environment. 2002; vol. 1, pp: 167-184

33. Rahman, MH. Viability of Potential Health benefits of Spirulina in Arsenicosis Management. Dissertation FCPS Department of Dermatology and Venereology, BSMMU. 2005; pp:1-58

34. Henrikson, R. Earth food spirulina, 1997; 3rd ed. pp: 66-67 California, Ronore Enterprises Inc

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# Balloon Temponade to Prevent Primary PPH in Jaundice-A Prospective Study

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**Summary:**

***This prospective interventional study was carried out on 40 intrapartum jaundice patients admitted in the Department of Obstetrics and Gynaecology, Dhaka Medical College Hospital during January to December 2004. The purpose of the study was to evaluate the effectiveness of prophylactic intrauterine hydrostatic balloon/condom temponade in addition to other conventional methods to prevent and control postpartum haemorrhage in patients with jaundice, to detect the prevalence of different etiological agents responsible for jaundice and to assess the foeto-maternal outcome in this study group. Prophylactic intrauterine hydrostatic balloon temponade was inserted in all of the 40 cases which showed an excellent effectiveness in preventing postpartum haemorrhage in patients with jaundice. The study also showed increased prevalence of Hepatitis E virus(HEV) and a high proportion of perinatal deaths in the study population.***

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## Introduction:

Jaundice in pregnancy has always been a major obstetric problem in a developing country like Bangladesh. Unfortunately, the magnitude of this disease is yet to be explored in our country. Most of our female population of reproductive age are not vaccinated against the hepatitis B virus. Data from two teaching hospitals reveal that enterically transmitted hepatitis A and E are also endemic in the country[1], attaining an exaggerated proportion in rainy season, especially in flood-affected areas. Only a few laboratories in this country have the complete diagnostic facilities to detect all types of hepatitis viral infection, the cost of investigation is also high, so it is a financial burden for most of the patients.

Haemorrhage during the third stage of labour is a matter of grave concern in almost all pregnancies with jaundice. The bleeding is partly due to uterine atony, but mostly due to the deficiency of coagulation factors produced by the damaged liver. Excessive bleeding during the third stage usually precipitate hepatic pre-coma or coma and further deteriorate the condition of the patient. Prognosis of hepatic failure is extremely poor in this group of patients. Inj.Oxytocin, Inj.Ergometrine, per rectal use of tab.misoprostol along with gentle massage of the uterus are the important parameters for reducing third stage bleeding. Prophylactic use of intrauterine hydrostatic Balloon temponade not only tries to stop the bleeding effectively, it also decreases the chance of further post partum haemorrhage afterwards. Simultenous transfusion of fresh blood and fresh frozen plasma provides fresh clotting factors and decrease bleeding.

This prospective interventional study was therefore carried out with an aim to establish the effectiveness of prophylactic intrauterine hydrostatic balloon temponade in preventing postpartum haemorrhage hence improving the prognosis and survival rate in this study group to detect the prevalence of different types of viral hepatitis in intrapartum jaundice patients in Dhaka, Bangladesh. It was tired to find out any association of intrapartum jaundice with the age, parity or gestational age of the mother. Maternal and foetal outcome, especially the prevalence of perinatal deaths was assessed. Postpartum haemorrhage has always been a complication in this group of patients.

## Materials and Methods:

This prospective interventional study was carried out on 40 pregnant patients with jaundice admitted in the Department of Obstetrics and Gynaecology, Dhaka

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Medical College Hospital during the study period. The duration of the study was from January to December 2004. All the patients included in this study were in different stages of labor. Informed consent was taken from all the patients.

Special emphasis was given to control primary postpartum haemorrhage by using prophylactic intrauterine hydrostatic balloon temponade/catheter in addition to other conventional methods (which included Inj.Oxytocin, Inj. Ergometrine, per rectal use of tablet Misoprostol along with gentle message of uterus). Fresh blood and fresh frozen plasma were also transfused in appropriate cases.

Intrauterine hydrostatic balloon/condom temponade was inserted in all the patients as a prophylactic measure to prevent postpartum haemorrhage. A condom was used as a balloon and was tied at the tip of a plain rubber catheter. Other end of the rubber catheter was connected to an infusion set attached with 500 cc normal saline. Placing the patient in a dorsal position, a sims speculum was used to expose the cervix and the anterior lip was grasped by a sponge holding forcep. Tip of rubber catheter along with the condom was held by another sponge holding forcep and was introduced into the uterus. Initially, around 100cc fluid was introduced followed by packing the vagina with vaginal packs/pads to prevent the balloon temponade from escaping out of the uterus. Then remaining 200-300cc fluid was introduced. The rubber catheter was disconnected from the infusion set and was tied strongly to make it water seal and was finally attached with the inner aspect of the thigh of the patient by a piece of micropore. The Balloon temponade was removed between 36-48 hours after insertion and the amount of postpartum haemorrhage was observed. Maternal and Fetal outcome was also noted. Apart from this, 3 important viral markers (HBsAg, Anti-HCV and Anti-HEV IgM) by ELISA method were used to detect the etiological agent responsible for viral hepatitis in the study group. Inclusion criteria for HELLP syndrome were platelet count <1,00,000/cu mm of blood, Aspartate Transaminase(AST)>70 U/L and Lactate Dehydrogenase(LDH)>600U/L. All the patients with HELLP syndrome had pregnancy induced hypertension(PIH).

All the informations were collected in a pre-designed structured data collection sheet and were compiled on a master chart first. Then organized by using a scientific calculator and standard statistical formula. Percentages were calculated to find out the proportion of the findings. The results were presented in Tables, Figures, Diagrams etc.

**Results:**

Majority of the patients(52.5%) were from 21-25 age group.

Most of the patients were either primi(35%) or 2nd gravida(40%).

A total of 72.5% patients went into preterm in the study population.

Viral hepatitis happened to be the chief aetiological agent(Table-I) responsible for jaundice (85% cases) followed by HELLP syndrome(15%) cases in this study. The viral hepatitis group was further subdivided on the basis of serological markers by ELISA method. The study shows(Table-II) that 20.5% patients were infected with Hepatitis B virus (HBV), 56% with hepatitis E virus(HEV), 6% with Hepatitis C virus(HCV) and as high as 17.5% patients had both HEV and HBV.

**Table-I**

*Aetiological distribution of patients(n=40):*

| Aetiological agent | No. of patients | Percentage |
|---|---|---|
| Viral hepatitis | 34 | 85% |
| HELLP Syndrome | 06 | 15% |
| Others | 00 | 00% |
| Total | 40 | 100% |

**Table-II**

*Distribution of patients by specific viruses (n = 34)*

| Name of virus | No. of patients | Percentage |
|---|---|---|
| HBV | 07 | 20.5% |
| HEV | 19 | 56% |
| HCV | 02 | 06% |
| HEV+HBV | 06 | 17.5% |
| Total | 34 | 100% |

Out of the 41 babies (one patient had twins), this study shows a high proportion(18 cases,44%) of perinatal death(Table-III). There was intrauterine death (IUD) in 27% cases, stillbirths (SB) in 23% cases and early neonatal deaths(END) in 50%cases(Table-IV). Most of the early neonatal deaths were due to prematurity.

**Table-III**

*Distribution of foetal outcome (n = 41)*

| Foetal outcome | No. of foetuses | Percentage |
|---|---|---|
| Survives well | 23 | 56% |
| Perinatal death | 18 | 44% |
| Total | 41 | 100% |

**Table-IV**

*Distribution of Perinatal deaths(n = 18):*

| Type of Perinatal death (PND) | No.of foetus | Percentage |
|---|---|---|
| Intrauterine death (IUD) | 5 | 27% |
| Stillbirths (SB) | 4 | 23% |
| Early neonatal death (END) | 9 | 50% |
| Total | 18 | 100% |

34(85%) patients had vaginal delivery whereas caesarean section was done in 6(15%) cases due to other obstetric indications(Table-V).

**Table-V**

*Mode of termination of pregnancy(n = 40):*

| Mode of termination | No. of patients | Percentage |
|---|---|---|
| Vaginal delivery | 34 | 85% |
| Ceasarean section | 06 | 15% |
| Total | 40 | 100% |

There were 4 maternal deaths(10%) in this study(Table-IV). The deaths were caused by fulminant hepatic failure.

**Table-VI**

*Distribution of maternal outcome(n = 40):*

| Maternal outcome | No. of patients | Percentage |
|---|---|---|
| Improved well | 36 | 90% |
| Maternal death | 04 | 10% |
| Total | 40 | 100% |

Prophylactic intrauterine hydrostatic balloon temponade was inserted in all of the 40 cases. It shows an excellent effectiveness (90%, 36cases) of the temponade(Table-VII) in preventing and controlling postpartum haemorrhage in the study population. In 36 cases (90%); there was no postpartum haemorrhage as mentioned in table VII.

**Table-VII**

*Amout of PPH after removal of temponade(n = 40):*

| Results | No.of patients | Percentage |
|---|---|---|
| No PPH | 36 | 90% |
| Mild to moderate PPH | 04 | 10% |
| Total | 40 | 100% |

**Discussion:**

Jaundice in pregnancy is a high risk case because of its association with postpartum haemorrhage. Intrauterine hydrostatic ballon/condom temponade is now increasingly being used to control postpartum haemorrhage and is well supported by several studies[2,5]. But, this study showed the effectiveness(90% cases) of the balloon temponade in preventing and controlling postpartum haemorrhage in patients with jaundice. So along with other conventional methods, this cheap and easily available balloon temponade can be used satisfactorily in preventing postpartum haemorrhage in this group of patients. The remaining 4 patients in which the intrauterine temponade proved to be ineffective were managed conservatively along with transfusion of fresh blood and fresh frozen plasma. However all these 4 patients ultimately died from fulminant hepatic failure.(Table VI).

Though some papers are available regarding the etiological agents for jaundice in Bangladesh[3], no recent data are there about the etiological agents responsible for jaundice in pregnancy. This small study showed that HELLP syndrome was responsible for jaundice in 15% cases and viral hepatitis in 85%cases. Hepatitis E virus(HEV) was the key aetiological agent in as high as 56% cases followed by Hepatitis B virus (HBV) and Hepatitis C virus(HCV) in 20.5% and 6% cases respectively. Interestingly, 17.5% patients had both HEV and HBV as presented in table II.

**Conclusion:**

Jaundice in pregnancy is an increasing obstetric problem in Bangladesh. The study shows that it is mostly restricted to last trimester and is associated with preterm labour and significant perinatal death. It also indicates that there is increased prevalence of Hepatitis E virus infection in pregnant women in Bangladesh. Prophylactic use of intrauterine hydrostatic balloon temponade in addition to other conventional methods exhibited excellent effectiveness in controlling postpartum haemorrhage in the study group.

The study strongly recommends using prophylactic intrauterine hydrostatic balloon temponade/catheter in all cases of pregnancy with jaundice to prevent and control postpartum haemorrhage.

**References:**

1. Rahman MH: Disease pattern among the patients admitted in two teaching hospitals. Teach Assoc J. 1992 5:21-27
2. Akhter S, Rashid M, Begum R, Zabeen F, Kabir Z et.Al: Use of a condom in the control of massive postpartum haemorrhage. Med Genmed Obs/Gyn with women's health. Medscape General Medicine 5(3) 2003 Medscape p1-9
3. Khan WI, Sultana R, Rahman M, Akhter H, Haq JA, Mohsin MA, Khan AK: Viral hepatitis: recent experiences from serological studies in Bangladesh. Asian Pac J Allergy Immumol. 2000 Jun;18(2):99-103
4. Siddiqui M: A clinical trial on intrapartum jaundice in Dhaka Medical College Hospital. FCPS Dissertation 2005 p1-68
5. Sultana M: Intrauterine hydrostatic ballooning by condom is a method for control of postpartum haemorrhage. FCPS Dissertation 2004 p75-83
6. Khan M, Chowdhury S: Diagnosis and Management of Liver isease in Pregnancy. Bangladesh Journal of Obstet Gynaecology, 1992,Vol.7(2),p64-77
7. Sheikh A, Sugitani M, Kinukawa N, Moriyama M, Komiyama K, Arakawa Y, Ishaque S, Hasan M, Suzuki K: Hepatitis E virus infection in fulminant hepatitis patients and an apparently healthy population in Bangladesh. Am J Trop Med Hyg.2002;66(6):p721-724
8. Khan M, Chowdhury S, Khondoker MAK, Akhtar A: Fulminant liver failure in pregnancy. Journal of IPGMR, 1992 Vol.7(2) p51-56
9. Alan H.De Cherney, Lauren Nathan: Current Obstetric and Gynecologic Diagnosis and treatment, Lange Medical Book/ McGraw Hill, ninth edition.p438-440
10. S.S.Ratnam, Bhascor Rao, S.Arulkumaran: Obsterics and Gyne-cology for postgraduates, Orient Longman Limited, volume 1, second edition1999.p38-42
11. American College of Obstetricians and Gynecologists: Viral hepatitis in pregnancy. ACOG Technical Bulletin no.248, July 1998
12. D.Keith, Edmonds: Dewhurst's Textbook of Obstetrics and Gynaecology for Postgraduates.Blackwell Science.6 thedition. p330-41
13. Medhat A, El-Sharkawy MM, Shabaan MM, Ghaneima SE: Acute Viral Hepatitis in Pregnancy. Int J Gynaecol Obstet. 1993 Jan;40(1):25-31
14. SchellenbergJC: Management of primary postpartum haemorrhage. Br J Obstet Gynaecol 1997;104:275-277
15. Sarkar CS, Giri AK, Maity TK: Jaundice in Pregnancy: A clinical study. J Indian Med Assoc. 1992 May;90(5):117-8
16. Jaishal SP, Jain AK, Naik G, Soni N, Chitnis DS: Viral hepatitis during pregnancy. Int J Gynaecol Obstet. 2001 Feb;72(2):103-108
17. Khuroo Ms, Kamil S: Aetiology, clinical course and outcome of sporadic acute viral hepatitis in pregnancy. J Viral Hepat. 2003 Jan;10(1):61-9
18. Megafu U: Jaundice in pregnancy: aetiology, management and mortality at Enugu, Nigeria. East Afr Med J. 1981 Jul;58(7):501-9
19. Mukherji J, Ganguly RP, Saha S: Maternal mortality due to post partum haemorrhage. J Obstet Gynecol Ind vol 51(5).Sep2001.p130-5

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# Percutaneous Transluminal Coronary Angioplasty (PTCA) and Stenting – Study of 100 Cases

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**Summary:**

***This prospective ongoing study conducted in University Cardiac Center, BSMMU, Dhaka from July'2004 to April'2006. 100 patients (mean age 52.4±6.2 years) underwent Percutaneous Transluminal Coronary angioplasty and stenting (PTCA & stenting) were evaluated. This study was designed to evaluate the short term angiographic and clinical results of stentangioplasty during hospital stay. The study group of 100 patients consisted of 88 (88%) men and 12 (12%) women. About risk factors 36 (36%) had hypertension, 30 (30%) were smoker, 20 (20%) suffered from diabetes mellitus, 14 (14%) had family history of ischaemic heart disease. Average Left ventricular ejection fraction was 54.2±7. Target vessel PTCA were done on 130 vessels, intracoronary stent implanted in 124 vessels, direct stenting was done in 80 cases, failed PTCA were in 4 (4%) cases, and three patients had dissection. The native vessels had a mean reference diameter of 2.89 mm and their luminal diameter increased significantly after percutaneous coronary intervention (PCI). Thombolysis in myocardial infarction (TIMI) flow analysis showed most of the patients had TIMI-1 flow (95,73%) before the procedure and maximum patients achieved TIMI-3 flow (91, 70%) after the procedure with significant clinical improvement. All the patients were discharged by one to three days of the procedure with improvement of their clinical condition. So PTCA and Stenting is a safe and effective technique with high procedural success rate and good short-term (hospital) clinical results in the native coronary artery lesions.***

***Key words: Coronary artery diseases; PTCA and stenting.***

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## Introduction:

Percutaneous coronary intervention (PCI) is widely used to relieve angina and ischaemia because it is effective in this role. Since the introduction of coronary balloon angioplasty into the clinical practice in 1977[1] improvement in equipment design and operator experience have permitted this procedure to be applied to the treatment of a broad spectrum of coronary artery disease (CAD). Advances in coronary based interventions, especially the use of bare-metal stents (BMS) and drug eluting stents (DES), have improved the efficacy and safety profile of percutaneous revascularization observed for patients undergong PTCA. Coronary angioplasty (PTCA) as a primary method for establishing coronary patency in patients with acute myocardial infarction is the riding crest of a wave of enthusiasm that threaten to engulf us and carry us off into the uncritical adoption[2]. Primary percutaneous coronary angioplasty (PTCA) has recently been advocated as the treatment of choice for patients with acute myocardial infarction[3]. Direct PTCA has been shown to have a high primary success rate (90%-99%) with few procedural complications and a low in-hospital mortality. It can establish TIMI grade III flow in upto 95% of patients within two hours of hospital admission[4]. Thrombolytic agents can not achieve this. Angioplasty has a more rapid action and greater success because it can dislodge and mechanically disrupt thrombus as well as reduce any residual coronary stenosis caused by atheroma. These actions reduce the risk of recurrent ischaemia, reocclusion and reinfarction[4].

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With the combination of sophisticated equipment, experienced operators and modern drug therapy, PCI has evolved into an effective non- surgical modality of treatment. The efficacy of PTCA has recently improved by coronary stenting and adjunctive pharmacological therapy, such as aspirin[5,6,7], clopidogrel[8.9] and glycoprotein IIb/IIIa receptor blocker[10]. The outcomes of PCI are measured in term of success and complications and related to the mechanism of the employed procedure. The aim of the study was to evaluate the immediate (short-term) clinical and angiographic outcome following PTCA and stenting in patients with coronary artery disease. (CAD).

**Methods:**

**Study Patients**

Between July' 2004 to October' 2006, 100 consecutive patients underwent PCI at University Cardiac Center (UCC), Banghabandhu Sheikh Mujib Medical University, Dhaka. The study group of patients consisted of 88 (88%) men and 12 (12%) women. With mean age of the patients was 52.4±6.2 years. (Table-01). Clinical inclusion criteria were symptomatic coronary artery disease with angina class I to IV and potential indication for coronary artery bypass surgery.

**Stenting procedure**

PTCA was done by technique as described by Gruentzig through femoral arterial approach according to modified seldinger technique. Outer diameter of guiding catheter was 7 to 8 French and 0.014 J steerable guidewire were used and dilatation was attempted using balloon catheter with 20-30 x 2-3 mm balloon size and balloon mounted stents were implanted at the target site. The balloon inflation pressure ranged from 4 to 14 atmosphere. Predilatation was performed using undersized, conventional angioplasty balloon before stenting.

Stenting was not performed in a vessel with a diameter 2.5 mm, if the lesion was longer than 40 mm, in case of extreme vessel tortuousity, if a large thrombus was visible at lesion site.

**Medications, sheath removal and discharge**

All patients received aspirin (75 mg daily) indefinitely and clopidogrel (75 mg daily) 72 hours before the procedure up to six month to one year after procedure. Intravenous heparin 10,000 unit IV was given as bolus during procedure and APTT was monitored. Sheath was removed after 4-6 hours and uncomplicated patients were discharged on next day.

**Outcome:**

The outcome of PCl are measured interms of success and complication and are related to the mechanism of the deployed devices as well as the clinical and anatomical patient-related factors. Complications can be divided into two categories;

Those common to all arterial catheterization procedures and

Those related to the specific technology used for the coronary procedure. With increased operator experience, new technology and adjunctive pharmacotherapy, the overall success and complication rate of angioplasty have improved.

**Definitions of PCI Success:**

The success of a PCI procedure may have the following components- angiographic success, procedural success and clinical success.

Angiographic success- A successful PCI produces substantial enlargement of the lumen at the target site. Previously defination was the achievement of a minimum stenosis diameter reduction to <50% in the presence of grade 3 TIMI flow[11]. With the advent of advanced adjunct technology, including coronary stents, a minimum stenosis diameter reduction to <20% has been the bench mark of an optimal angiographic result.

Procedural success- A successful PCI should achieve angiographic success without in hospital major clinical complications eg. death, myocardial infarction, emergency coronary artery bypass surgery during hospitalization [11,12].

Clinical success- clinically successful PCI includes anatomic and procedural success with relief of signs and or symptoms of myocardial ichaemia after the patient recovers from the procedure[13,14].

Definitions of procedural complications. According to the 1998 coronary interventional document[13]

procedural complications are dovided into six basic categories: death, MI, emergency CABG, stroke, vascular access site complications and contrast agent nephropathy.

**Results:**

In this study 36 (36%) patients had history of hypertension, 30 (30%) were smokers, 20 (20%) were diabetic, 14 (14%) had family history of ischaemic heart disease. Average left ventricular ejection fraction (LVEF) was 54.2 ± 7 (Table-I). Among these cases coronary vessel involved in 147 cases, target vessels were 130, PTCA was done in 124 and intracoronary stent deployed in 124 patients (Table-II).

Most of the patients in study presented as a case of post MI angina (38%) followed by stable angina pectoris (18%) (Table-I)

Single vessel disease was 60 (60%), double vessel disease was 33 (33%). triple vessel disease was 7 (7%). (Table-II). Majority of the patient had Type A Lesion 81 (62.3%). 93 (93%) patients had clinical success, 95 (95%) patient had angiographic and procedural success (Table-VI).

All the stent implantation procedures were successful except[1] five. In particular, there was one stent displacement. Intracoronary stent implanted in LAD 71 (57.3%), (Fig 1.a.b) LCX 24 (19.4%), RCA 16(12.9%) (Fig-2, a,b). Diagonal 10 (8.07%) and Marginal 3 (2.4%) (Table-03). Failed PTCA were in five cases due to tortuousity of vessels or total occlusion not crossed by PTCA wire or balloon. Bere metal stents (BMS) were deployed in most of the patient (71.8%) (Table-IV). with mean diameters (mm) 2.89+02 and mean leangth of 14.68 mm+2. TIMI Grade 03 flow was in 91 patients (70%) (Table –V). Three patients developed coronary artery dissection during the procedures who needed immediate intracoronary stent deployment. Stentangioplasty were successfully done in 12 patients with CTO and failed in 3 (three) patient due to failure in crossing guide wire. Three patients developed ischaemic pain and two patients developed heart failure and six patients developed major arrhythmias (Table-VII).

**Table-I**

*Baseline charectersties of study population (n=100)*

| Demography/other features | N (% SD) |
|---|---|
| Mean age (year) | 52.4+6.2 |
| Male | 88 (88%) |
| Female | 12 (12%) |
| Risk Factors | |
| Smoking | 30 (30%) |
| Hypertension | 36 (36%) |
| DM | 20 (20%) |
| Dyslipidaemia | 13 (13%) |
| Positive F. History | 14 (14%) |
| Clinical Diagnosis | |
| Stable angina pectories | 28 (28%) |
| Unstable Angina | 18 (18%) |
| AMI (Ant and Inf) | 09 (09%) |
| Recent MI (Ant. Sept & Inf.) | 07 (07%) |
| Post MI Angi na. | 38 (38%) |
| Anierior MI | 24 (24%) |
| INferior MI | 08 (08%) |
| Combined | 06 (06%) |
| Ejection fraction (mean) | 54.20±7 |

**Table-II**

*Angiographic diagnosis of study population (n =100)*

| Vessels | Total Number | Percentage (%) |
|---|---|---|
| Total diseased vessels | 147 | - |
| Total target vessel | 130 | - |
| Single vessel disease | 60 | 60 |
| Double vessel disease | 33 | 33 |
| Triple vessel disease | 7 | 7 |

**Table-III**

*Site of stent deployment in the target vessel (n = 124)*

| Site | Total number | Percentage (%) |
|---|---|---|
| LAD | 71 | 57.3 |
| LCX | 24 | 19.4 |
| RCA | 16 | 12.9 |
| Diagonal | 10 | 8.07 |
| Marginal | 3 | 2.4 |

**Table-IV**

*Characteristic of deployed stents in the target vessels (n =124)*

| Parameters | Total no | Percentage % |
|---|---|---|
| **Types** | | |
| Bare metal | 89 | 71.8 |
| Drug coated | 32 | 25.8 |
| Drug eluting | 3 | 2.4 |
| **Diameter** | | |
| 2.5 mm | 35 | 28.2 |
| 2.75 mm | 26 | 21 |
| 3.00 mm | 45 | 36.3 |
| 3.5 mm | 15 | 12.1 |
| 4.0 mm | 3 | 2.4 |
| Mean diameter = 2.89 ±2 (mm) | | |
| **Length (mm) :** | | |
| Range ( 0-10) | 26 | 21 |
| Range (10-19) | 78 | 63 |
| Range (20-29) | 18 | 14.5 |
| Range (30-39) | 2 | 1.6 |
| **Meanlength = 14.68mm ± 2** | | |

**Table-V**

*TIMI flow of the target vessels (n= 130)*

| Parameter (TIMI flow) | Before procedure Number (%) | After procedure Number (%) |
|---|---|---|
| Grade -0 | 10(8%) | 6(4.6%) |
| Grade -1 | 95(73%) | 0(0%) |
| Grade -2 | 25(19%) | 33(25.4%) |
| Grade -3 | 0(0%) | 91(70%) |

**Table-VI**

*Result of stentangioplasty of study population (n =100)*

| Results | Total number | Percentage % |
|---|---|---|
| Clinical success | 93 | 93 |
| Angiographic success | 95 | 95 |
| Procedural success | 95 | 95 |

**Table-VII**

*In-hospital clinical outcome of study population (n = 100).*

| Parameters | Total number Patient | Percentage (%) |
|---|---|---|
| Dissection | 3 | 3 |
| Acute stent thrombosis | 0 | 0 |
| Acute MI | 3 | 3 |
| Acute LVF | 5 | 5 |
| Failed PTCA | 4 | 4 |
| Major arrhythmias (e.g. VT. VF) | 6 | 6 |
| Death | 1 | 1 |

**Discussion:**

Based on our result, we believe that all types of PTCA and intracoronary stenting can play an important role in the treatment of symptomatic coronary artery diseases. Our in hospital mortality rate was one (%) comparing favorably with the results obtained in the previous angioplasty trials[15,16,17]. The mean age of our study population was 52.4 ± 6.2 years, which is earlier age than other studies[16, 18.]

Early recurrent ischaemia occurred in 4 (4%) patients in our series. Grines LC et al and Moreno R et al showed early recurrent ischaemia after PTCA in 5.1% and 6.5%, cases in their series[15,18], which are very close to our study and other studies in our country[13,14]. Chest pain without changes on ECG just after PTCA occurred in cases which simulate the results of other study[15].

Two patients had reappearance of symptoms within 1 month. Check angiogram of them were done which showed normal functioning of the stents. Thrombus containing lesions have been considered as contraindication for stentangioplasty. Stents were successfully deployed in two patients containing thrombus in LAD (Proximal) and LCX (distal) after treatment with low molecular weight heparin. Studies have shown that presence of angiographicaly visible thrombus as a risk factor for subsequent stent thrombosis[16]. However, other have represented coronary stenting as safe and effective therapy for thrombus containing lesion[17,18].

Elective stenting was done in most (80%) of patients. Similar elective stenting have also been reported by Moussa et al, Colombo et al[20] and Kimura et al[21] (70%, 67% and 71% respectively). This study showed stenting as modality of treatment for suboptimal PTCA, acute vessel closure, dissection during PTCA and restenosis following PTCA similar to those reported as an indications for stenting by various authors[14,22,23,27]. Hence unlike PTCA, this success of intracoronary stenting is not influenced by lesion morphology.

Recently developed drug eluting stents have reduced the incidence of restenosis drastically to 8 to 10%. Ten years follow up of initial Cohort of patients treated PTCA revealed 89.5% survial rate (95% with single vessel disease, 81% in-patients with multivessel disease[24]). In patients undergoing with the 1985-1986 NELBL PTCA registry[25] a five year survival was 92.9% for double vessel disese and 86.5% for triple vessel disease. In patients with multivessel disease undergoing PTCA in BARI[26] 5 year survival was 86%. infarct free survival was 78.7%. Specifically 5 year survival was 84.7% with TVD, 87.6% with DVD (Double vessel disease).

Percutaneous intervention (PCI) of the chronic total occlusion (CTO) lesion present great challenge including reduced success rate, prolonged procedure time, large amount of contrast use, high occlusion rate, and costs. The goal of intervention in CTO is to penetrate the total occlusion and pass the wire in the true luman of the distal vessel without causing stent angioplasty intimal dissection, Accordingly we have successfully done stentangioplasty in 12 patients with CTO and failed in three patient due to failure of passing the wire.

**Limitation of the Study:**

This is single center observational prospective study to assess the safety and short-term clinical and angiographic outcome of small number of patients. Further randomized trial may be needed for the better result.

**Conclusion:**

Percutaneous coronary intervention (PCI) have revolutionized the effective management of coronary artery disease and their symptoms. It has been increasingly demonstrated to reduce the risk of adverse events in patients with acute coronary syndrome (ACS). Intracoronary stent implantation in coronary artery stenosis following PTCA is a valid strategy with good clinical and angiographic in-hospital results. This very study is an initial experience in a new center with small number of patients may serve as an impetus for future large scale study in home and abroad.

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**References:**

1. Gruenzig AR, Senning A, and Siegenthaler WE,: Non-operative dilatation of coronary artery stenosis percutaneous transluminal angioplasty. N Eng J Med 1979; 301:61
2. Vaikus P. Limitations of primary angioplasty in acute myocardial infarction. International Roundup. Br. Heart J 1995; 73: 409.
3. Boyle RM, Immediate angioplasty in the United Kingdom. International Roundup, Br Heart J 1995; 73:4113.
4. Experience of primary angioplasty in the United Kingdom, Br. Heart J 1995; 73:414.
5. Schomig A, Neumann FJ, Kastrati A, Schuhlen H, Blasini R, Hadamitzky M et al. A randomized comparison of antiplatelet and anticogulation therapy after the placement of coronary artery stent. N Engl J Med 1996; 334: 1084-1089.
6. Bertrand M, Legrand V, Boland J, Emanuelson H, et, al. Full anticoagulation versus ticlopidine plus aspirin after stent implantation. A randomized mulitcenter European study: the PANTASTIC trial (abstract). Circulation 1996; 99 (suppl-1): 1685.
7. Urban P, Macaya C, Rupprecht H-J, Kiemeneij F, Emanuelsson H, Fontanelli A et al, for the MATTIS investigators. Multicenter Aspirin and Ticlopidine Trial after Intracoronary stenting in high risk patients (MATTIS) (abstract). J Am Coll Cardiol 1998; 31 (Suppl A): 397 A.
8. Berger PB, Bell MR, Rihal Cs, Ting H, Barsness G, Garratt K et al. Clopidegrol versus ticlopidine after intracoronary stent placcement. J Am Coll Cardiol 1999; 34: 1891-1894.
9. Moller C, Bottner HJ, Petersen J, Roskamm H.A randomized comparison of clopidogrel and aspirin versus ticlopidine and aspirin after the placement of coronary artery stents, Circulation 2000; 101: 590-593.

10. Lincoff AM, Califf RM, Topol EJ, platelet glycoprotein IIb/IIIa receptor blockade in coronary artery disease. J Am Coll Cardiol 2000; 35: 1103-1115.

11. Guidelines for percurtaneous transluminal coronary angioplasty: a report of the American College of Cardiollgy /American Heart Association Task Force on Assessment of Diagnostic and Tehrapeutic Cardiovascular Procedures (Committee no Percutaneous Transluminal Coronary Angioplasty). J Am Coll Cardiol 1993 ; 22 : 2033 - 54.

12. Kent KM, Bentivoglio LG, Block PC, et al. Percutaneous transluminal coronary angioplasty report from the Registry of the National Heart, Lung and Blood Institute. Am J Cardiol 1982;49:2011-20.

13. Mommenuzzaman NAM, Hossain SM. Uddin MJ, Foreigan M. Rahman F. Islem KQ et al. Experience of percutaneous Transluminal Coronary Angioplasty (PTCA) and stenting study of 168 cases. Bangladesh Heart Journal 2001 ; 16(2): 71 – 75.

14. Udden MJ. Chowdhury AHK, Ali M, Majumder AAS Islam KD. Mondal R, Rahman F et al. Percutaneous Coronary Intenventions (PCI)- Result of 100 cases. Bangladesh Heart Journal 2002 ; 1812 : 109-115.

15. Grines CL, Browne KF, Marco J, et al. A comparson of immediate angioplasty with thrombolytic therapy for acute myocardial infarction. N Eng J Med 1993; 328: 673-679.

16. Saporito J, Rothberg M, Smestad G. Primary PTCA in a rural hospital. J Invas Cardiol 1996; 8: 249-251.

17. Gershlick HA. Acute Management of Myocardial Infraction. Infastion Medicine International 1997; 40 (11): 62-67.

18. Moreno R, Garcia E, Soriano J, Abeytua M, loez de Sa'E, Acosta J, Perez de Isla L. Rubio. R, Lopez-sendon JL. Early coronary angioplasty for acute myocardial infraction infasetion; angioplasty for acute myocardial infasetin; predictors of a poor outcome in a noon selected poputation. J Invas Cardiol 2001; 13: 202-210.

19. Moussa 1, Mario CD, Moses J, Reimers B, Francesco LD, Blengino S et al. Comparison of angiographic and clinical outcomes of coronary stenting of chronic total cocclusions versus subtotal occlusion. Am J Cardiol 1998; 81: 1-6.

20. Colombo A, Hal P, Nakamura S, Alamgor Y, Maiello L, Martini G et al. Intracoroncry stenting without anticoagulation accomplished with intravascular ultrasound. Circulation 1995;91: 1678-88.

21. Kimura T, Yokoi M, Nakagawa Y, Tainura T, Kaburagi S, Sawada Y et al. Three years follow up after implantation of metalic coronary artery stents. N Eng1 J Med 1996;334 – 56 561-6. –6.

22. Pepin CJ, Holmes DR, Block PC, Brinker JA, Mullins CE, Nissens SE et al. Coronary artery stents: Acc Expert consensus document. J Am Coll Cardiol 1996;18:7824-94.

23. Eeckhout E, Kappenberger L, Goy JJ. Stents for intracoronary placement : Current status and future directions. J Am Coll Cardiol 1996; 27: 757-65.

24. Fischman DL, Leon MB, Balm DS, et al. A randomized comparion of coronary stent placement and ballon angioplasty in the treatment of coronary artery disease; Stent Restenosis Study Investigatiors. N Eng L J Med 1994:331:496-501.

25. Homes DR, Jr. Kip KE, Kelsey SF, Detre KM, Rose n AD. Cause of death analysis in the Nhlbi Ptca Registry: results and considerations for evaluating long-term survival after coronary interventions. J Am Coll Cardiol 1997;30:881-7.

26. Comparison of coronary bypass surgery with angioplasty in patients with multivessel disease: the Bypass Angioplasty Revasecularization Investigation (BARI) Investigators. [Published erratum appears in N Eng1 J Med 1997;336:147.] N Eng1 J Med 1996;335:217-25

27. Platelet glycoprotein IIb/IIIa receptor blockade and low-dose heparin during percutaneous coronary revascularization: the EPILOG Investigators. N Eng1 Med 1997;336:1689-96

## REVIEW ARTICLES

# Bird Flu and Bangladesh: Review and Update

ASM NU AHMED

**Summary:**

***Bird flu is an infectious disease of birds caused by Influenza A (H5N1) virus. Although the virus usually do not infect humans, more than 200 confirmed human casess of Avian Influenza A have been reported worldwide. Almost all of them had caught it from birds. While H5N1 undergoes specific mutations and reassorting creating variations which can infect species not previously known to carry the virus, health experts fear the virus could trigger a pandemic if it mutates to form a strain that can transmit between humans. As bird flu confirmed in about 59 countries, it is extremely pathogenic, humans do not possess any immunity to it, currently no vaccine is available and sometimes H5N1 can become resistant to the antivirals, a global pandemic would kill millions of people. A human pandemic would be particularly catastrophic in developing countries where living conditions and malnutrition are likely to make people more vulnerable, health services are weak and vaccines and antivirals would be beyond reach. Recently bird flu has been detected in Bangladesh, which may lead to devastating consequences on the health of the people and economy of the country. So every effort should be taken to prevent its further spread, make people aware of the disease, training health personnel and stockpiling antivirals to face any human epidemic.***

***(J Bangladesh Coll Phys Surg 2008; 26: 32-38)***

**Introduction:**

The dreaded Avian Influenza virus, better known as 'bird flu', has been detected at Savar on the outskirts of the capital Dhaka. The Bangladesh government announced the detection of the Avian Influenza on Thursday, 22nd March 2007; samples were sent to the Bangladesh Livestock Research Institute and also to the National Institute of Animal Health in Bangkok, which confirmed the presence of the deadly H5N1 strain of the virus.[1] The virulent H5N1 virus has killed at least 169 people across the world since late 2003 through contact with infected birds, according to the World Health Organization (WHO).[2] Blood samples of a number of workers at the farms, where the deadly virus was confirmed among chickens, tested negative for the virus.[3] It is unclear how poultry became infected at the farm in Savar town, about 40 km. west of Dhaka. Bangladesh, home to hundreds of thousands of poultry farms, has already banned import of poultry products from more than 50 countries as part of a preventive measure to check bird flu from entering the country. Despite all the precautionary measures, its entry could not be stopped finally.

The authorities concerned have reportedly taken a few emergency steps to stop spread of the disease and advised the people not to be panicked by the news of the disease and continue consuming cooked poultry meat and boiled or fried eggs as usual. Since then bird flu has been reported in 12 districts both in organized and backyard poultry. To mitigate further spread the authorities, meanwhile, have culled more than 100,000 poultry in the affected regions.[4] Besides, decision has been taken to destroy all poultry birds within one kilometer radius of any affected area to contain the disease. The members of the armed forces have cordoned the risk zone to stop any attempt to smuggle out poultry birds from there. Other measures include health checks on all workers who may have come into contact with the infected birds.[3] So far there has been no human case of bird flu in Bangladesh.

**The Avian Influenza A (H5N1) virus:**

Avian Influenza is an infectious disease of birds caused by Influenza A (H5N1) virus,– also called 'H5N1 virus' – occurs mainly in birds, is highly contagious among birds, and can be deadly to them.[5] H5N1 is a subtype of the species *Influenza A virus* of the *Influenzavirus A* genus of the *Orthomyxoviridae* family. Like all other Influenza A subtypes, the H5N1 subtype is an RNA virus.[6] (Fig. 1) Infected birds shed Influenza virus in their saliva, nasal secretions, and feces; the virus can survive for considerable lengths of time outside of the host. Susceptible birds become infected when they have contact with contaminated secretions or

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excretions or with surfaces that are contaminated with secretions or excretions from infected birds. Domesticated birds, including chickens, ducks, and turkeys, may become infected with Avian Influenza virus through direct contact with infected waterfowl or other infected poultry, or through contact with surfaces (such as dirt or cages) or materials (such as water or feed) that have been contaminated with the virus.[5] Infection with Avian Influenza viruses in domestic poultry causes two main forms of disease that are distinguished by low and high extremes of virulence. The 'low pathogenic' form may go undetected and usually causes only mild symptoms (such as ruffled feathers and a drop in egg production). However, the 'highly pathogenic' form spreads more rapidly through flocks of poultry. This form may cause disease that affects multiple internal organs and has a mortality rate that can reach 90-100%, often within 48 hours.[7]

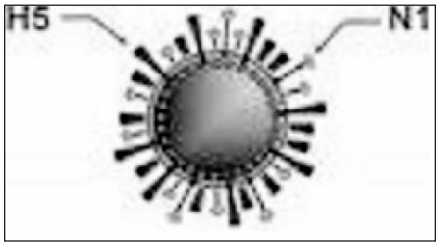


**Fig. 1:** *H5N1 virus*

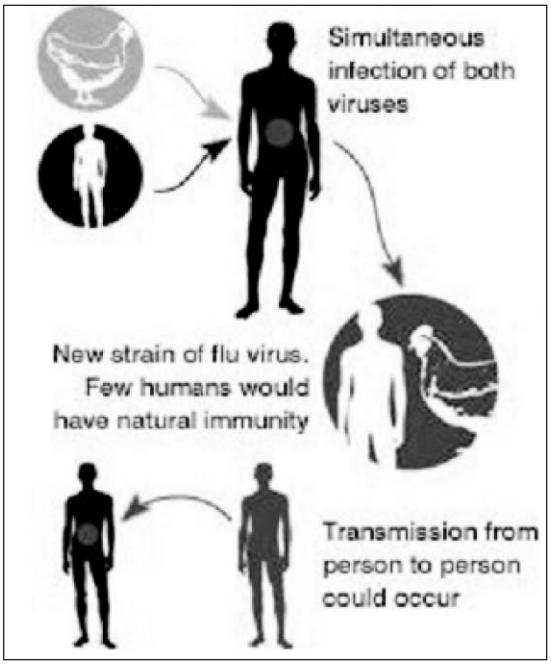


**Fig. 2:** *If a person were infected with avian and human flu viruses at the same time, they could swap genes creating a new strain transmissible between humans.*

Avian Influenza A (H5N1) virus that has been reported from Africa, Asia, Europe, and the Near East:

The disease, which was first identified in Italy more than 100 years ago, now occurs worldwide. Since December 2003, Avian Influenza A (H5N1) infections in animals have been reported in Asia, Africa, the Pacific, Europe, and the Near East. Outbreaks of Avian Influenza H5N1 occurred among poultry in eight countries in Asia (Cambodia, China, Indonesia, Japan, Laos, South Korea, Thailand, and Vietnam) during late 2003 and early 2004.[8] At that time, more than 100 million birds in the affected countries either died from the disease or were killed in order to try to control the outbreaks. By March 2004, the outbreak was reported to be under control.

Beginning in June 2004, however, new outbreaks of Influenza H5N1 among poultry and wild birds were reported in Asia.[9,10] Since that time, the virus has spread geographically. Reports of H5N1 infection in wild birds in Europe began in mid 2005. In early 2006, Influenza A H5N1 infection in wild birds and poultry were reported in Africa and the Near East.[11]

**How is Avian Influenza detected in birds:**

Tests for diagnosing all Influenza strains of animals are rapid and reliable. Many laboratories in the WHO global Influenza network have the necessary high-security facilities and reagents for performing these tests as well as considerable experience.

**What animals can be infected with Avian Influenza A (H5N1) viruses:**

In addition to birds, pigs, tigers, leopards, ferrets, and domestic cats can be infected with Avian Influenza A (H5N1) viruses.[12] In addition, in early March 2006, Germany reported H5N1 infection in a stone marten (a weasel-like mammal). The Avian Influenza A (H5N1) virus that emerged in Asia in 2003 is evolving and it is possible that other mammals may be susceptible to infection as well. Centers for Disease Control and Prevention (CDC), USA is working closely with domestic and international partners to continually monitor this situation and will provide additional information to the public as it becomes available.[13]

**Do bird flu viruses infect humans:**

Although Avian Influenza A viruses usually do not infect humans, the first documented infection of humans with an Avian Influenza virus occurred in Hong Kong in 1997, when the H5N1 strain caused severe respiratory disease in 18 humans, of whom 6 died.[14,15] The infection of humans coincided with an epidemic of highly pathogenic Avian Influenza, caused by the same strain, in Hong Kong's poultry population.[14] Since January 2004, the World Health Organization has reported human cases of Avian Influenza A (H5N1) in Asia, Africa, the Pacific, Europe, and the Near East. Since then more than 306 confirmed cases of human infection with Avian Influenza A (H5N1) viruses have been reported , more than half of those people reported infected with the virus have died.[10] Most cases have occurred in previously healthy adults and children, and some with chronic medical conditions and have resulted from direct or close contact with H5N1-infected poultry or H5N1-contaminated surfaces.

In general, H5N1 remains a very rare disease in humans. The H5N1 virus does not infect humans easily, and if a person is infected, it is very difficult for the virus to spread to another person. While there has been some human-to-human spread of H5N1, it has been limited, inefficient and unsustained.[16] For example, in 2004 in Thailand, probable human-to-human spread in a family resulting from prolonged and very close contact between an ill child and her mother was reported.[16,17] Most recently, in June 2006, WHO reported evidence of human-to-human spread in Indonesia. In this situation, 8 people in one family were infected. The first family member is thought to have become ill through contact with infected poultry. This person then infected six family members. One of those six people (a child) then infected another family member (his father). No further spread outside of the exposed family was documented or suspected.[5,11]

**What are the implications of Avian Influenza to human health:**

Thus two main risks for human health from Avian Influenza are 1) the risk of direct infection when the virus passes from the infected bird to humans, sometimes resulting in severe disease; and 2) the risk that the virus – if given enough opportunities – will change into a form that is highly infectious for humans and spreads easily from person to person.[18]

**Why H5N1 is of particular concern:**

Of the 15 Avian Influenza virus subtypes, H5N1 is of particular concern for several reasons. H5N1 mutates rapidly and has a documented propensity to acquire genes from viruses infecting other animal species.[19] Its ability to cause severe disease in humans has now been documented on two occasions.[2,10] In addition, laboratory studies have demonstrated that isolates from this virus have a high pathogenicity and can cause severe disease in humans. Birds that survive infection excrete virus for at least 10 days, orally and in faeces, thus facilitating further spread at live poultry markets and by migratory birds.

**What are the symptoms of Avian Influenza in humans:**

Published information about the clinical course of human infection with H5N1 Avian Influenza is limited to studies of cases in the 1997 Hong Kong and 2004 Thailand outbreaks.[10,14,20] The reported symptoms of Avian Influenza in humans have ranged from typical human Influenza- like symptoms (fever, cough, sore throat, and muscle aches) to eye infections, pneumonia (with chest radiograph changes), severe respiratory diseases (such as acute respiratory distress syndrome) leading to respiratory failure requiring intubation, and other severe and life-threatening complications.

**How is Avian Influenza detected in humans:**

Avian Influenza cannot be diagnosed by symptoms alone, so a laboratory test is required. Avian Influenza is usually diagnosed by collecting a swab from the nose or throat during the first few days of illness. This swab is then sent to a laboratory, where they will either look for Avian Influenza virus using a molecular test, or they will try to grow the virus.[21,22] Growing Avian Influenza viruses should only be done in laboratories with high levels of protection. If it is late in the illness, it may be difficult to find an Avian Influenza virus directly using these methods. If this is the case, it may still be possible to diagnose Avian Influenza by looking for evidence of the body's response to the virus. This is not always an option because it requires two blood specimens (one

taken during the first few days of illness and another taken some weeks later), and it can take several weeks to verify the results.[23]

**How is Avian Influenza in humans treated:**

Studies done in laboratories suggest that the prescription medicines approved for human Influenza viruses should work in treating Avian Influenza infection in humans. Four different Influenza antiviral drugs (amantadine, rimantadine, oseltamivir, and zanamivir) are approved by the U.S. Food and Drug Administration (FDA) for the treatment and prevention of Influenza.[24,25] All four have activity against Influenza A viruses. However, sometimes Influenza strains can become resistant to these drugs, and therefore the drugs may not always be effective. For example, analyses of some of the 2004 H5N1 viruses isolated from poultry and humans in Asia have shown that the viruses are resistant to two of the medications (amantadine and rimantadine).[26] Two other antiviral medications (oseltamivir and zanamavir) would probably work to treat Influenza caused by H5N1 virus, but additional studies are needed to demonstrate their current and ongoing effectiveness.

**Threat of a global pandemic:**

Nonetheless, because all Influenza viruses have the ability to change, scientists are concerned that H5N1 virus one day could be able to infect humans and spread easily from one person to another.[27,28] Because these viruses do not commonly infect humans, there is little or no immune protection against them in the human population. A human pandemic could start in two ways. If a person were infected with Avian and human flu viruses at the same time they could swap genes creating a new strain transmissible between humans.[18] (Fig. 2) In this case it would spread very rapidly. Alternatively, the virus could adapt more gradually, improving its ability to bind to human cells during repeated human infection. If H5N1 virus were to gain the capacity to spread easily from person to person, an Influenza pandemic (worldwide outbreak of disease) could begin.

No one can predict when a pandemic might occur. However, experts from around the world are watching the H5N1 situation in Asia and Europe very closely and are preparing for the possibility that the virus may begin to spread more easily and widely from person to person.

**Is there a vaccine to protect humans from H5N1 virus:**

There is currently no commercially available vaccine to protect humans against the H5N1 virus that is being detected in Asia and Europe. No vaccine is available because there is no way to predict what the pandemic strain will look like. It takes at least six months to make a new flu vaccine once the virus has appeared.[29] However, CDC continues to work with WHO and the National Institutes of Health (NIH) on development of a vaccine for Influenza A (H5N1). Research studies to test a vaccine that will protect humans against H5N1 virus began in April 2005, and a series of clinical trials is under way.[30]

**Is there a risk for becoming infected with Avian Influenza by eating poultry:**

There is currently no scientific evidence that people have been infected with bird flu by eating safely handled and properly cooked poultry or eggs. Even if poultry and eggs were to be contaminated with the virus, proper cooking would kill it.[31] In fact, recent studies have shown that the cooking methods that are already recommended by the U.S. Department of Agriculture (USDA) and the Food and Drug Administration (FDA) for poultry and eggs to prevent other infections will destroy Influenza viruses as well.[32]

So to stay safe, the advice is the same for protecting against any infection from poultry:

Wash your hands with soap and warm water for at least 20 seconds before and after handling raw poultry and eggs.

Clean cutting boards and other utensils with soap and hot water to keep raw poultry from contaminating other foods.

Use a food thermometer to make sure you cook poultry to a temperature of at least 165 degrees Fahrenheit. Consumers may wish to cook poultry to a higher temperature for personal preference.

Cook eggs until whites and yolks are firm.

**Is there travel restrictions to areas with known H5N1 outbreaks:**

CDC does not recommend any travel restrictions to affected countries at this time. However, CDC

currently advises that travelers to countries with known outbreaks of H5N1 Influenza avoid poultry farms, contact with animals in live food markets, and any surfaces that appear to be contaminated with feces from poultry or other animals.[33]

**Bird import ban:**

There is currently a ban on the importation of birds and bird products from H5N1-affected countries. The regulation states that no person may import or attempt to import any birds (Class Aves), whether dead or alive, or any products derived from birds (including hatching eggs), from the specified countries.

**Control measures:**

The quarantining of infected farms and destruction of infected or potentially exposed flocks are standard control measures aimed at preventing spread to other farms and eventual establishment of the virus in a country's poultry population.[34] Apart from being highly contagious, Avian Influenza viruses are readily transmitted from farm to farm by mechanical means, such as by contaminated equipment, vehicles, feed, cages, or clothing. Highly pathogenic viruses can survive for long periods in the environment, especially when temperatures are low. Stringent sanitary measures on farms can, however, confer some degree of protection.

In the absence of prompt control measures backed by good surveillance, epidemics can last for years. For example, an epidemic of H5N2 Avian Influenza, which began in Mexico in 1992, started with low pathogenicity, evolved to the highly fatal form, and was not controlled until 1995.[35]

**Is Bangladesh ready to face an epidemic:**

Bangladesh has started its preparedness to combat against bird flu from 2005. 'National Avian Influenza and Human Pandemic Influenza Preparedness and Response Plan Bangladesh 2006-2008' was approved by the Government of bangladesh in 2006 as a preparedness for any bird flu outbreack.[4] There is detailed plans for prevention and measures to be taken during any epidemic. Government has already started mass campign to increase public awareness about the disease, training health care personnel, stockpiled antiviral drugs and other supportive medications and equipments for management and prophylaxis. Vaccines for seasonal influenza are also being stockpiled for health care workers and initiative has been taken to set up a modern influenza referral laboratory.[4]

**Conclusion:**

The dreaded Avian Influenza virus has claimed many lives and played havoc with poultry industry in some countries of Asia and Europe, was also detected in neighbouring India and Myanmar only recently. Despite all the precautionary measures, its entry into our country could not be stopped finally. Health experts fear the virus could trigger a pandemic if it mutates to form a strain that can transmit between humans. The Asian Development Bank says even a mild pandemic could kill 3 million in Asia alone and cost the region $300 billion.[35] Its site has background and papers on the potential economic impact in Asia. A human pandemic would be particularly catastrophic in developing countries where living conditions and malnutrition are likely to make people more vulnerable, health services are weak and vaccines and antivirals would be beyond reach. Even if there is no pandemic, bird flu will threaten the livelihoods of millions of people in Asia and Africa as health officials carry out mass poultry culling and other countries ban imports.

The news about anyone being afflicted in our country with the virus might stir panic among the population and play havoc with the poultry industry in particular. Fortunately timely measures taken by the Government could halt the spread of the disease, saved the poultry sector and prevent any human casualty. To face any future epidemic we should have regular publicity in the media to make people aware of the disease, well-trained health care personnel and stockpiled enough antiviral drug oseltamivir, commercially known as Tamiflu, may improve survival prospects if taken within 48 hours of symptoms appearing.[36] Every effort must also be made to protect the poultry farms in Bangladesh. About 3.5 million people are employed in the poultry sector in Bangladesh that adds a value worth some Taka 50 billion or $833 million to the country's GDP. Thus, if the epidemic breaks out among poultry birds here, culling of a very large number of birds will be required and that would invite ruin to this budding

sector and leave considerable adverse effects on the economy. Furthermore, regular publicity needs to be done in the media to make people aware of the disease, to help prevent its spread.

## References:

1. Giasuddin M, Alam J, Samad MA, Al-Mamun M, Taimur JFA. Highly Pathogenic Avian Influenza: Bangladesh Situation. Bangladesh Livestock Research Institute Web site. (Accessed September 28, 2007, at http://www.blri.gov.bd/banner_detail.php?bannerid=1)
2. Beigel JH, Farrar J, Han AM, Hayden FG, Hyer R, de Jong MD, et al. "Avian influenza A (H5N1) infection in humans". N Engl J Med 2005; 353: 1374–85.
3. FluTrackers.com. Bangladesh says no human case of bird flu. (Accessed September 28, 2007, at http://www.flutrackers.com/forum/showthread.php?t=19511)
4. Rahman M. Preparing for the next Influenza Pandemic: Bangladesh Perspective. Journal of Bangladesh College of Physicians and Surgeons 2007; 25: 53–5.
5. Centers for Disease Control and Prevention. Key Facts About Avian Influenza (Bird Flu) and Avian Influenza A (H5N1) Virus. (Accessed October 25, 2005, at http://www.cdc.gov/flu/avian/gen-info/facts.htm)
6. Murphy BR, Webster RG: Orthomyxoviruses. In: Fields Virology 1996, (ed.): Fields BN, Knipe DM, Howley PM, Chanock RM, Melnick JL, Monath TP, Roizman B, Straus SE, 3rd ed., pp 1397–1445, Lippincott-Raven, Philadelphia, PA
7. Payungporn S, Chutinimitkul S, Chaisingh A, Damrongwantanapokin S, Nuansrichay B, Pinyochon W, et al. "Discrimination between Highly Pathogenic and Low Pathogenic H5 Avian Influenza A Viruses". Emerging Infectious Diseases 2006; 12: 700–1.
8. World Health Organization. Avian Influenza A (H5N1) – situation (poultry) in Asia as at 2 March 2004: need for a long-term response, comparison with previous outbreaks. Wkly Epidemiol Rec 2004; 79: 96–9.
9. Hien TT, Liem NT, Dung NT, San LT, Mai PP, Chau NV et al. Avian Influenza A (H5N1) in 10 patients in Vietnam. N Engl J Med 2004; 350: 1179–88.
10. Chotpitayasunondh T, Ungchusak K, Hanshaoworakul W, Chunsuthiwat S, Sawanpanyalert P, Kijphati R, et al. Human disease from Influenza A (H5N1), Thailand, 2004. Emerging Infectious Diseases 2005; 11: 201–9.
11. Webster RG, Govorkova EA. "H5N1 Influenza – Continuing Evolution and Spread". N Engl J Med 2006; 355: 2174–7.
12. Keawcharoen J, Oraveerakul K, Kuiken T, Fouchier RAM, Amonsin A, Payungporn S, et al. Avian Influenza H5N1 in tigers and leopards. Emerging Infectious Diseases 2004; 10: 2189–91.
13. Harder TC, Werner O. "Avian Influenza", In: Influenza Report 2006, (ed.): Kamps BS, Hoffman C, Preiser W. Paris, France: Flying Publisher. ISBN 3-924774-51-X.
14. Chan PK. Outbreak of Avian Influenza A (H5N1) virus infection in Hong Kong in 1997. Clin Infect Dis 2002; 34: S58–S64.
15. Claas EC, Osterhaus AD, van Beek R, De Jong JC, Rimmelzwaan GF, Senne DA, et al. Human Influenza A H5N1 virus related to a highly pathogenic avian influenza virus. Lancet 1998; 351: 460–1.
16. Ungchusak K, Auewarakul P, Dowell SF, Kitphati R, Auwanit W, Puthavathana P, et al. Probable person-to-person transmission of Avian Influenza A (H5N1). N Eng J Med 2005; 352: 333–40.
17. Centers for Disease Control and Prevention. Cases of Influenza A (H5N1) – Thailand, 2004. MMWR Morb Mortal Wkly Rep 2004; 53:100–3.
18. Reuters AlertNet - Bird flu: Threat of a global pandemic. 2007. (Accessed June 13, 2007, at http://www.alertnet.org/db/crisisprofiles/BIRDFLU.htm?v=in_detail)
19. The World Health Organization Global Influenza Program Surveillance Network. "Evolution of H5N1 Avian Influenza viruses in Asia". Emerging Infectious Diseases 2005; 11: 1515–21.
20. Yuen KY, Chan PK, Peiris M, Tsang DN, Que TL, Shortridge KF, et al. Clinical features and rapid viral diagnosis of human disease associated with Avian Influenza A H5N1 virus. Lancet 1998; 351: 467–71.
21. Lee M, Chang P, Shien J, Cheng M, Shieh HK. Identification and sub typing of Avian Influenza viruses by reverse transcription-PCR. J Virol Methods 2001; 97: 13–32.
22. Ng EKO, Cheng PKC, Ng AYY, Hoang TL, Lim WWL. Influenza A H5N1 detection. Emerging Infectious Diseases 2005; 11: 1303–5.
23. World Health Organization. Recommended laboratory tests to identify Influenza A/H5 virus in specimens from patients with an influenza-like illness. 2005. (Accessed September 2, 2005, at http://www.who.int/csr/disease/avian_influenza/guidelines/avian_labtests1.pdf.)
24. Govorkova EA, Leneva IA, Goloubeva OG, Bush K, Webster RG. Comparison of efficacies of RWJ-270201, zanamivir, and oseltamivir against H5N1, H9N2, and other Avian Influenza viruses. Antimicrob Agents Chemother 2001; 45: 2723–32.
25. Cooper NJ, Sutton AJ, Abrams KR, Wailoo A, Turner D, Nicholson KG. Effectiveness of neuraminidase inhibitors in treatment and prevention of Influenza A and B: systematic review and meta-analyses of randomized controlled trials. BMJ 2003; 326: 1235.

26. Belshe RB, Burk B, Newman F, Cerruti RL, Sim IS. Resistance of Influenza A virus to amantadine and rimantadine: results of one decade of surveillance. J Infect Dis 1989; 159: 430–5.

27. Webster RG, Walker EJ. "The world is teetering on the edge of a pandemic that could kill a large fraction of the human population". American Scientist 2003; 91: 122.

28. Allen PJ. The world awaits the next pandemic: will it be H5N1, the 'bird flu'? J Child Health Care 2006; 10: 178–87.

29. Schultz J. Bird flu vaccine won't precede pandemic. United Press International Web site. (Accessed February 4, 2006, at http://www.upi.com/ConsumerHealthDaily/view.php?StoryID=20051128-054641-9412r)

30. World Health Organization. Antigenic and genetic characteristics of H5N1 viruses and candidate H5N1 vaccine viruses developed for potential use as pre-pandemic vaccines. (Accessed August 18, 2006, at http://www.who.int/csr/disease/avian_influenza/guidelines/h5n1virus2006_08_18/en/index.html)

31. Mounts AW, Kwong H, Izurieta HS, Ho Y, Au T, Lee M, et al. Case-control study of risk factors for Avian Influenza A (H5N1) disease, Hong Kong, 1997. J Infect Dis 1999; 180: 505–8.

32. US Food and Drug Administration. What Consumers Need to Know About Avian Influenza? (Accessed October 19, 2006, at http://www.cfsan.fda.gov/~dms/avfluqa.html)

33. Centers for Disease Control and Prevention. Update: notice to travelers about Avian Influenza A (H5N1). July 29, 2005. (Accessed September 2, 2005, at http://www.cdc.gov/travel/other/avian_flu_ah5n1_031605.htm.)

34. World Health Organization. Avian Influenza - fact sheet. (Accessed January 15, 2004, at http://www.who.int/csr/don/2004_01_15/en/)

35. Asian Development Bank report: Bird flu could drastically impact Asian economies. (Accessed November 4, 2005, at http://www.birdfludefense.com/012963.html)

36. Ward P, Small I, Smith J, Suter P, Dutkowski R. Oseltamivir (Tamiflu) and its potential for use in the event of an influenza pandemic. J Antimicrob Chemother 2005; 55: Suppl 1:i5–i21.

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# Fluid and Electrolyte Homeostasis in Newborn Baby

Jagadish C Das

**Summary:**

***Assessment of fluid and electrolyte properly in neonate is very important but difficult. Fluid and electrolyte homeostasis during this period depends on some factors notably gestational age of baby, its postnatal age, pathological conditions and environmental situation. In fetus, water and electrolytes is constantly supplied from mother, which is cut-off by delivery of the baby. Extra-cellular fluid volume that is greater than intracellular fluid volume in fetus precipitously decreases after birth. Adaptation of fluid and electrolyte after birth is due to discontinuation of placental exchange, onset of insensible water loss, thermoregulation, autonomic renal regulation and intake of fluid and other nutrients. The adaptation course is divided into transition phase, intermediate phase and stable growth phase. Fluid and electrolyte therapy in neonate should be very judicious, because administration of minimum fluid and electrolyte may bring a maximum proportionate change of such environment. Fluid requirement in neonate after birth increases gradually by first few days. Preterm baby require more fluid than term baby during the first week of life due to high insensible water loss in the former. Electrolytes with intravenous fluid should be offered after ensuring initial diuresis, a decrease in sodium or at least 5-6% weight loss in neonates.***

***Key word: Fluid, electrolyte, homeostasis, newborn baby.***

***(J Bangladesh Coll Phys Surg 2008; 26: 39-45)***

## Introduction:

Fluid and electrolyte assessment during neonatal period is very important and difficult. This is because the transition from fetal to neonatal period is associated with major changes in water and electrolyte homeostatic control. The fetus has a constant water and electrolyte supply from mother across the placenta. After birth, the newborn must acquire responsibility for its own. fluid and electrolyte homeostasis in an environment where fluid and electrolyte availability and losses fluctuate widely. Proportion of various constituents of such environment varies normally depending on gestational age of neonate and even on postnatal age of baby. It also varies during pathological situations and environmental conditions. Again, relative small absolute changes in body water and electrolyte represent as large proportionate change in a neonate due to its small body size[1]. So careful fluid and electrolyte management is essential for the well being of sick neonate. Inadequate administration of fluids can result in hypovolemia, hypersomolarity, metabolic abnol-111alities and renal failure in neonate whereas excess fluid administration may results generalized edema and abnormalities of pulmonary function. Excess fluid in newborn infant is also associated with patent ductus arteriosis, congestive heart failure, intraventricular hemorrhage, necrotizing enterocolitis and bronchopulmonary dysplasia (BPD)[2]. Electrolyte abnormality in neonatal period is quite undesirable. Sodium is a permissive factor for growth and depletion of sodium in this period is associated with poor weight gain along with other abnormalities[2]. Though both Hyponatranlia and hypernatramia contribute to neurological morbidity in sick neonate[2], prevalence of hypematramia (7.1/100 000 term baby[3] or 274/100 000 term baby[4]), fluid and other electrolyte abnormality is not less even in developed nations[2]. Minerals are essential for healthy life, but its administration within few hours of life is associated with adverse effect on neonatal health[2]. So, fluid and electrolyte management in neonatal period should be very judicious and thus is ever encountered in medicine. The goal is not to maintain fluid and electrolyte status after birth, rather to allow the changes to occur appropriately. Shortage of understanding of health clinicians regarding fluid and electrolyte homeostasis of newborn baby will make an adverse effect on newborn health. Unfortunately, understanding of many physicians remained incomplete regarding such vital pediatric issue. The review is written to orient and update health personals particularly the clinicians regarding some fundamental aspect of such important topic to help

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neonate through allowing such vital changes to occur appropriately.

**Background:**

Total body water (TBW) decreases markedly from intrauterine life to adulthood. Water contributes to 90% of body weight in the 24 weeks old fetus, 75% in term neonates, and 50% in adults'. During intrauterine life water content decreases along with relative increase of fat mass particularly during third trimester of gestation[6]. Water turnover is high in neonates and decreases with increasing age[7,8]. Body water is divided into two compartments: intracellular fluid (ICF) and extracellular fluid (ECF)[9]. In the fetus, the ECF volume is larger than ICF volume, and ECF decreases with age. The ECF volume drops precipitously after birth in large part because of postnatal dieresis. By 1 year of age both fluid compartments come close to adult levels (Fig-1)[10]. The major ion of ECF is sodium (Na+). Blood volume in neonates is 85-100 ml/kg body weigh compared to 60-70 ml/kg body weights in adolescents and adults[11].

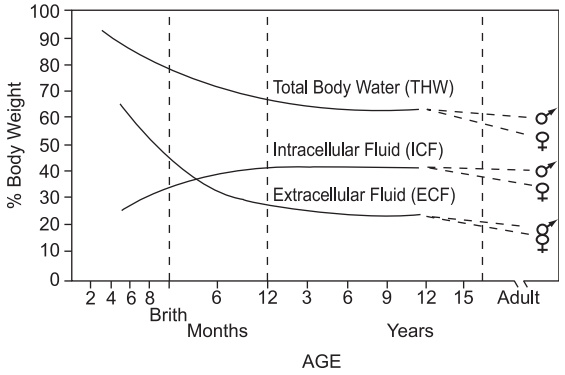


**Fig-1:** *Rearrangement of body fluid from intrauterine to extrauterine life.*

Immediate adaptation process after birth affects the metabolism of water and electrolytes as a result of discontinuation of placental exchange and the onset of considerable insensible water loss and thermoregulation. Subsequent adaptation includes the onset of autonomic renal regulation of fluids and electrolytes, and intake of fluids and other nutrients[12]. The time course of adaptation may be divided into three major phases[12].

*Phase-1 (Transition phase): The immediate postnatal phase is characterized by a relative oliguria[13] followed by a diuretic phase, during which body fluid compartments are rearranged by isotonic or hypertonic (i.e.hypernatraemic and hyperchloremic) contraction. Duration of this phase varies from hours to days. These changes are caused by considerable evaporative water loss via immature skin and by continuing natriuresis[14]. Phase 1 usually ends when maximum weight loss has occurred[12]. *Phase-II (Intermediate phase): This phase is characterized by diminished insensible water loss along with increasing cornification of epidermis, a fall in urine volume to less than 1-2 ml/kg per hour, and a low sodium excretion. Duration of this phase varies from 5 to 15 days[12]. *Phase-III (Stable growth phase): This is characterized by continuous weight gain with a positive net balance for water and sodium. Expected weight gain is 10-20gm/kg body weights per day[12].

The renal glomerular surface area available for filtration is small in neonates than that of in older infants and adults. In term neonate, glomerular filtration rate (GFR) increases significantly during the first week of life and continues to rise over the first two years of life[12]. Immaturity to the distal nephron with an anatomically shortened loop of Henle leads to reduced ability to concentrate urine[12]. Maximum urinary concentrations are up to 550 mosm/1 in preterm infants, and 700 mosm/1 in term infants, compared to 1200 mosm/1 in adults[15]. Neonates may be placed at risk for volume depletion when a high renal solute load cannot be compensated for by the ability to produce concentrated urine. Although hormonal factors i.e. renin-angiotensin-aldosterone system (RAAS), and arginin-vasopressin (AVP) is mature early in gestation, the effects are limited by renal immaturity[16]. A lower plasma oncotic pressure and higher permeability of the capillary wall in preterm infants compared to term infants and adults[12] enhances the shift of water from, intravascular to interstitial compartment, with an increased risk of edema especially under pathologic conditions[17].

Fluid and electrolyte assessment generally focuses on body water, serum sodium, potassium and calcium concentrations.

Body water and sodium: A weight loss of 5-10% in term[18] infants and 10-20% in preterm[19] infants is common during the first week of life. The net water and sodium loss is accepted as appropriate after

birth[20]. Assessment of degree of this water loss is complicated by a relatively large and highly variable insensible water loss[1]. The more immature the infant, the more pronounced the contraction of the extra cellular space and higher the insensible water loss. Both of these factors predispose to hypernatremia in the first few days of life.

Potassium. Serum potassium concentration rise in the first 24 to 72 hours after birth in moderately to markedly premature infants, even in the absence of exogenous potassium intake and in the absence of renal dysfunction[21]. This increase seems to be the result of a shift of potassium from the intracellular to extracellular space. The magnitude of this shift roughly correlates with the degree of immaturity[21]. Potassium load is excreted by the kidneys[1].

Calcium. Total calcium concentration in cord plasma increases with increasing gestational age and is significantly higher than maternal values[1]. With delivery of baby, plasma calcium falls, reaching a nadir at age 24-48 hour[22]. Serum parathyroid hormones (PTH) increase postnatally in response to this fall in plasma calcium concentration. This increase in PTH mobilizes calcium from bone, and plasma calcium concentration rises and subsequently stabilizes even in the absence of exogenous calcium intake. Clinically significant hypocalcemia occurs in premature infants, asphyxiated newborns, and infants of diabetic mothers. The etiology in all these circumstances is a sluggish response in PTH secretion to the postnatal fall in plasma calcium concentration.

Approximately 50% of total plasma calcium is bound (predominantly to albumin) and 50% is ionized. Ionized calcium is the best indicator of physiologic blood calcium activity. Changes in plasma ionized calcium concentration parallel those described above for total plasma calcium concentration[23]. Lower serum albumin concentration and acidosis, not uncommonly found in premature infants, result in a lower total plasma calcium concentration for a given plasma ionized calcium concentration. Changes in ionized plasma calcium mirror to total plasma calcium concentration. Again, larger sample volume is required in many laboratories to determine ionized calcium. Hence, calcium status is routinely monitored with total plasma calcium concentration[23]. In neonates, faecal sodium loss depends on gestational age. The loss is 0.1 mmol/kg/day in preterm and 0.02 mmol/kg/day in term babies[12]. Faecal potassium losses are about twice as high as sodium loss, but show no relation with gestational age[12]. Under pathological conditions (e.g. bowel obstruction, ileostomy, pleural fluid aspiration etc.) electrolyte contents of lost fluids cannot be predicted precisely. Here, it is wise to measure at least once the sodium concentration of such lost fluid in order to replace them. Chloride loss usually correlates with sodium loss and potassium loss is usually much smaller[12].

**Fluid and electrolyte management:**

Management of fluid and electrolyte in neonate should be based on background of such issue. It depends on baby's age, weight, associated pathological situation, environmental conditions and on phase that the baby is passing.

*Phase 1: Transition phase*

The objectives for fluid and electrolyte administration during this period are:

- To allow contraction of ECF with negative water balance of not more than 10% without compromising intravascular fluid volume and cardiovascular function.
- To allow a negative net balance for sodium of 2-5 mmol/kg per day for the first postnatal days, to maintain normal serum electrolyte concentration.
- To secure a sufficient urinary output and avoid oliguria (<0.5-1.0 ml/kg per hour) for longer than twelve hours.
- To ensure regulation of body temperature by providing enough fluid for transepidermal evaporation.

During phase 1, administration of fluid and electrolytes needs very caution. Clinician is to be judicious enough regarding fluid and electrolytes at the onset of diuresis and in polyuric patient specially in ELBW infant[12]. Fluid load during this phase in healthy preterm neonate range from 96 to 200 ml/kg/day from the third day of life[24], but rarely exceed 130 ml/kg per day. The fluid intake dependent on birth weight and increases daily (Table-1) Electrolyte administration during the first 3-5 days

also depends on maturity and birth weight[12]. Generally, sodium[25] and potassium[12] supplementation should be started after ensuring initial diuresis, a decrease of serum sodium or at least 5-6% weight loss in neonates[25].

Sodium intake should be restricted in very-low-birth-weight (VLBW) babies during the period of ECF contraction until a weight loss of approximately 6-10% has occured[26]. Fluid restriction reduces chance of patent ductus arteriosus, nacrotizing enterocolitis, death and tends to reduce the risk of bronchopulmonary dysplasia but increases risk of dehydration[27]. A restricted sodium intake has positive effects on oxygen requirements and the risk of later bronchopulmonary dysphasia[26]. Sodium restriction also induces higher risk of development hyponatraemia, and is associated with pontine (brain) myelinolyosis[12].

*Phase lI: Intermediate phase*

The objectives for fluid and electrolyte administration during phase ii are to:

- Replete the body for electrolyte losses, replaces actual water and electrolytes.
- Augmentation of oral feedings.

Phase lll: Stable growth phase -

The objectives for fluid and electrolyte administration during phase I I I are:

- To replace losses of water and electrolytes (maintaining water and electrolytes homeostasis).
- To provide extra water and electrolytes to build up new tissue at intrauterine growth rates.

The recommended fluid intakes in phase II (Table-2) are based on studies suggesting that a daily fluid intake equal to or higher than 170 ml/kg body weight per day is accompanied by high urinary excretion with negative sodium balance, even if Na+ intake is as high as 10 mmol/kg body weight per day[12]. Fluid therapy in extremely low birth weight (ELBW) in excess of 200/ml/kg/day does not maintain Na+ balance, regardless of the amount of NaCI provided. It is important to note that ELBW infants require more fluids than recommended during the first week of life for term infants, because of high insensible water loss[28]. Evaporation of water from upper respiratory passages accounts for approximately one third of net insensible water loss and reaches the level of 0.8-0.9 ml/kg-body weight per hour in premature infants and 0.5 ml/kg body weight per hour in term babies 12 Electrolytes requirements in preterm very low birth weight baby is less than that of preterm low birth-weight baby[12].

Fluid requirements during the phase III Table-3 re related to the expected weight gain[8]. Daily sodium requirement in term baby in this stage is -3 mmol/kg body weight in comparison to 3-5 mmol/kg body weight in preterm infant[12].

Urinary output may be as high as 6.0 ml/kg per hour of free water in the presence of a total urine production of 9.8 ml/kg per hour in preterm infants with birth weight 2000gm[28]. Water loss from stool is negligible in early life prior to establishing enteral feeding. When full enteral feeding is achieved, faecal losses of 5-10 ml/kg per day are usually assumed to balance metabolic water production[29]. Plasma Na+ concentrations are normal in infants with sodium intake of 1.1-3.0 mmol/kg body weight per day and

**Table-I**

*Parenteral fluid and electrolyte intake during the first postnatal week.*

Recommended fluid intake (ml/kg body weight per day)

| Days after birth | 1stday | 2ndday | 3rdday | 4thday | 5thday | 6thday |
|---|---|---|---|---|---|---|
| Term neonate | 60-120 | 80-120 | 100-130 | 120-150 | 140-160 | 140-180 |
| Preterm neonate >1500g | 60-80 | 80-100 | 100-120 | 120-150 | 140-160 | 140-160 |
| Preterm neonate <1500g | 80-90 | 100-110 | 120-130 | 130-150 | 140-160 | 160-180 |

Recommended Na+, K+, Cl" supply (mmol/kg body weight per day)

* Na+ = 0- 3 (5) ** K+ = 0- 3 Cl- = 0- 5

**Table-II**

*Parenteral fluid and electrolyte intake for newborn infants during the intermediate phase.*

| Gestational age Birth weight | Fluid intake (ml/ kg body weight/day) | Na+ intake (mmol/kg body weight/day) | K+ intake (mmol/kg body weight/day) | CI- intake (mmol/kg body weight/day) |
|---|---|---|---|---|
| Term neonate | 140-170 | 2.0-5.0 | 1.0-3.0 | 2.0-3.0 |
| Preterm neonate | | | | |
| > 1500g | 140-160 | 3.0-5.0 | 1.0-3.0 | 3.0-5.0 |
| < 1500g | 140-180 | 2.0-3.0(5) | 1.0-2.0 | 2.0-3.0 |

**Table-III**

*Potential fluid and electrolyte intake during the first month of life with stable growth*

| Gestational age | Fluid intake (ml/ kg body weight/day) | Na+ intake (mmol/kg body weight /day) | K+ intake (mmol/kg body wt/day) |
|---|---|---|---|
| Term neonate | 140-160 | 2.0-3.0 | 1.5-3.0 |
| Preterm neonate | 140-160 | 3.0-5.0(7.0) | 2.0-5.0 |

fluid intakes of 140-170 ml/kg body weight per day[12,30]. There is evidence that fluid intake lower than 140 ml/kg body weight/day, together with Nay intake of about 1 mmol/kg body weight per day, is adequate to maintain sodium balance in ELBW neonates[31,32]. There is no increase in morbidity among infants given less Na+ and less fluid[33]. A non-significant trend to higher incidence of patent ductus arteriosus and bronchopulmonary dysplasia is observed in infants given more Na+ and more fluid intake[12]. Breast-fed term babies need as little as 0.35 to 0.7 mmol/kg body weight per day of Na during the first 4 months of life to achieve adequate growth. A recommendation to provide 1.0 to 2.0 mmol /kg per day of NaCI should counter-balance incidental losses from skin or gastrointestinal tract. In preterm infants, a higher growth rate explains a higher sodium requirement[12].

Preterm infants retain about 1.0 to 1.5 mmol/kg body weights per day K+, which is about the same as foetal accretion. About 2 to 3 mmol/kg/day of potassium, which is similar to the amount provided in human milk, is usually recommended for young infant[12].

During fluid and electrolyte management the clinician has to consider about some important environmental factors, which can potentially influence, such vital issues notably insensible water loss.

* A double wall incubator reduce insensible water loss in VLBW neonate by about 30% when a humidity of 90% is used at thereto-neutral temperature. With maturation of the epidermal barrier it is possible to reduce ambient humidity step by step commonly after first 5 days of life[12]. * Use of waterproof coverings (such as plastic films, plastic blankets, bubble blankets) in addition to treatment in a double wall incubator leads to further reduction of insensible water loss by 30-60% [12]. * Use of radiant warmers or single wall incubators for VLBW care may increase water loss and impair thermoregulation[34]. * Use of emollient ointments decreases insensible water loss of up to 50% in open care conditions[35,36]. * Endotracheal intubations and mechanical ventilation using warmed and humidified air significantly reduces insensible respiratory water loss by 20 mm/kg body weights per day[12].

**Messages:**

- During delivery of baby the extracellular fluid volume is larger than the intracellular volume.
- Extracellular volume drops precipitously after birth mainly due to postnatal diuresis.
- Adaptation of fluid and electrolytes is due to discontinuation of placental exchange, onset of insensible water loss, thermoregulation, renal regulation and intake of fluid and nutrients.
- Adaptation course is divided into three phases-namely, transition phase, intermediate phase and stable growth phase.
- Electrolytes generally should be supplemented after ensuring initial diuresis.
- Some environmental factors e.g. incubator care, waterproof coverings, radiant warmers, use of emollient etc potentially influences fluid and electrolyte management.

**Conclusion:**

Fluid and electrolyte homeostasis in neonatal period is an important issue. Proportion of various constituents of this environment in neonate is very different from older children. Even in same neonate, this environment changes depending on some factors including postnatal age. A relative small absolute pathological change in any of the constituent of this environment may bring a deleterious effect on neonatal health. During delivery extracellular fluid volume is larger than the intracellular volume which drops precipitously thereafter. Adaptation of fluid and electrolytes is divided into three phases and is due to discontinuation of placental exchange, insensible water loss, thermoreguilation, renal regulation and intake of fluid and nutrients by neonate. After birth if needed, fluid without minerals is supplemented and minerals are added when initial diuresis has occurred. The objective of management of this environment is not to maintain the status after birth but to allow the changes to occur appropriately. Clinician is to be updated enough regarding this change. Proper understanding of fluid and electrolyte homeostasis of newborn baby will make problem related to such environment preventable with a favorable change in neonatal health.

**References:**

1. Shaffer SG, Meade VM. Sodium balance and extracellular volume regulation in very low birth infants. J Pediatr 1989; 115: 285-290.
2. Modi N. Fluid and electrolyte balance. In. Rennie JM. Roberton's Textbook of Neonatology 4th ed. Philadelphia. Elsevier Churchill Livingstone 2005; 335-351.
3. Laing IA, Wong CM. Hypernatraemia in the first few days: is the incidence rising? Archives of Disease in Childhood 2002; 87: F158-162.
4. Oddie S, Richmond S, Coulthard H. Hypernatraemic dehydration and breast feeding: a population study. Archives of Disease in Childhood 2001; 85: 318-320.
5. Fomon SJ, Haschke F, Ziegler EE. Body composition of reference children from birth to age 10 years. Am J Clin Nutr 1982; 35: 1169-1175.
6. Fusch C, Slotboom J, Fuehrer U. Neonatal body composition: dual-energy x-ray absorptiometry, magnetic resonance imaging, and three-dimensional chemical shift imaging versus chemical analysis in piglets. Pediatr Res 1999; 46: 465-473
7. Fusch C, Hangerland E, Scharrer B. Water turnover of healthy children measured by deuterated water elimination. Eur J Pediatr 1993; 152: 110-114.
8. Bernardi JL, Goulart AL, Amancio OM. Growth and energy and protein intake of preterm newborns in the first year of gestation-corrected age. Sao Paulo Med J 2003; 121: 5-8.
9. LinshawMA. Selected aspects of cell volume control in renal cortical and medullary tissue. Pediatr Nephrol 1991; 5: 653-665.
10. Greenbaum LA. Pathophysiology of body fluids and fluid therapy. In. Behrman RE, Kliegman RM, Jenson HB editors. Nelson Textbook of Pediatrics 17th ed. Philadelphia. Saunders; 2004: 191.
11. Nicholson JF and Pesce MA. Laboratory testing in infants and children. In. Behnnan RE, Kliegman RM, Tensor HB editors. Nelson Textbook of Pediatrics 17th ed. Philadelphia. Saunders; 2004: 2393-2505.
12. ESPGHAN. Guidelics on pediatric parenteral nutrition. JPediatr Gastroenterol Nutr 2005; 41: S33-S38
13. Modi N. Development of renal function. Br Med Bull 1988; 44: 935-956.
14. Modi N. Hutton TL. The influence of postnatal respiratory adaptation on sodium handling in pretei-n. neonates. Early Hum Dev 1990; 21: 11-20.
15. Chevalier RL. Developmental renal physiology of the low birth weight pre-tern newborn. J Urol 1996; 156: 714-719
16. Haycock GB Aperia A. Salt and the newborn kidney. Pediatr Nephrol 1991; 5: 65-70.
17. Jobe A, Jacobs H, Ikegami M. Lung protein leaks in ventilated lambs: effects of gestational age. J Appl Physiol 1985; 58: 1246-1251

18. Podratz RO, Broughton DD, Gustafson DH, Bergstralh EJ, Melton J. Weight loss and body temperature changes in breast-feed and bottle-feed neonates. Clin Pediatr 1986; 25:73-77.

19. Shaffer SG, Quimiro CL, Anderson JV, Hall RT. Postnatal weight changes in low birth weight infants. Peclicztries 1987; 79: 702-705.

20. Lorenz JM. Assessing fluid and electrolyte status in newborn. Clinical Chemistry 1997; 43(i). 205-210.

21. Sato K, Kondo T, Iwao H, Honda S, Ueda K. Internal potassium shift in premature infants; cause of non-oliguric hyperkalemia. JPediatr1995; 126: 109-113.

22. Tsang RC, Chen IW, Freidman MA, Chen L Neonatal parathyroid function: role of gestational and postnatal age. J Pediatr 1973; 83:728-730.

23. Wandrup J, Kroner J, Pryds O, Kstrup KW. Age-related reference values for ionized calcium in the first week of life in premature and full-teen neonates. Scand J Clin Lab Invest 1988; 48: 255-260.

24. Coulthard MG and Hey EN. Effect of varying water intake on renal function in healthy pceterm babies. Arch Dis Child 1985; 60: 614-620.

25. Hartnoll G, Betremieux p, Modi N. Randomized controlled trial of postnatal sodium supplementation in infants of 25-30 weeks gestational age; effects on cardiopulmonary adaptation. Arch Dis Child Fetal Neonatal 2001; 85: F29-32.

26. Hartnoll G, Betremieux P, Modi N. Randomized controlled trial of postnatal sodium supplementation on body composition in 25 to 30 week gestational age infants. Arch Dis Fetal Neonatal 2000: 82: F24-28.

27. Bell EF, Acarregui MJ. Restricted versus liberal water intake for preventing morbidity and mortality in preterm infants (Cochrane Review). In. The Cochrane Library, Issue I. Chichester, UK: John Wiley & Sons Ltd.; 2004.

28. Adamkin DH. Issues in the nutritional support of the ventilated baby. Clin Perinatol 1998; 25: 79-9G.

29. Catzeflis C Schutz Y, Micheli JL. Whole body protein synthesis and energy expenditure in very low birth weight infants. Pediatr Res 1985; 19: 679-687.

30. Polberger SK,Axelsson IA,Raitha NC.Growth of very low birth weight infants on verying amounts of human milk protein. Pediatr Res 1989; 25: 414-119.

31. Costarino AT, Gruskay JA, Corcoran L. Sodium restriction versus daily maintenance replacement in very low birth weight premature neonates: a randomized, blind therapeutic trial. JPedurtr 1992; 120: 99-106.

32. Ekblad H, Kero P, Takala J.Water, Sodium and acid-base balance in premature infants :therapeutical aspects. Acta Pediatr Scand 1987; 76: 47-53.

33. Asanto H, Taki M, Igarashi Y. Sodium Homeostasis in premature infants during the early postnatal period: results of relative low volume of fluid and sodium intake. Pediatr Nephrol 1987; 1: C38

34. Meyer MP, Payton MJ, Salmon A. A clinical comparison of radiant warmer and incubator care for preterm infants from birth to 1800 grams. Pediatrics 2001; 108: 395-401.

35. Lane AT, Drost SS. Effects of repeated application of emollient cream to premature neonates' skin. Pediatrics 1993; 92: 415-419.

36. Nopper AJ, Honrri KA, Sookdeo- Drost S. Tropical ointment therapy benefits in premature Infants. JPediatr 1996; 128: 660-669.

## CASE REPORTS

# Endoscopic Total Thyroidectomy: Report of Two Cases

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**Summary:**

***Two patients (one male and one female) recently under went total thyroidectomies using a standard laparoscope at the department of surgery, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka. Three ports (one mid line and two laterals) were employed and a harmonic scalpel was used for the dissection. To the best of our knowledge, there was no report of endoscopic total thyroidectomy from Bangladesh. Both patients were fed on the first post operative day. They were discharged between the 3rd and 4th post operative days. There was no hypocalcaemia but one patient developed temporary unilateral vocal cord palsy. Endoscopic thyroidectomy appears to be a technically feasible patient friendly modality of treatment for the selected cases of thyroid swelling in an experienced hand with excellent out come.***

***Key Words: Endoscopic; laparoscopic; thyroid surgery; total thyroidectomy.***

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**Introduction:**

Solitary thyroid nodule is one of the common problems encountered in surgical practice. About 15 % of the solitary thyroid nodules subsequently turned out to be thyroid carcinoma requiring total thyroidectomy and another 30-40 % to be follicular adenoma[1]. An operation on the thyroid gland in conventional method whether due to a benign or a malignant lesion requires a long incision in front of the neck and always matter of distress to the young patients who are the most common sufferers. In the recent years to address this problem lateral mini cervical incision was tried. However, endoscopic thyroidectomy through the thoracic or axillary routes was recently performed in some centre [2]. Demonstration of endoscopic excision of benign thyroid nodule, done recently at the live demonstration workshop of the Society of Laparoscopic Surgeons of Bangladesh by Dr. Shailesh Puntambekar, was the first endoscopic thyroid operation in Bangladesh. Subsequently, Endoscopic total thyroidectomy was performed using laparoscope in these two reported patients having papillary carcinoma of thyroid presented with solitary thyroid nodule.

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**Case Report 1:**

Mr. F 36 year's old male patient presented with a swelling in front of the neck which he noticed for two weeks. There was no feature of hypo or hyper thyroidism. However, he had history of weakness and weight loss for the last 2 years. On examination his pulse was 78 per minutes and regular in rhythm. Examination revealed a firm oval nodule of 3.0 X 2.8 cms in size arising from the left thyroid lobe. Rest of the gland was normal. His TSH, FT3, FT4 were within normal limit. FNAC revealed a papillary carcinoma. His serum calcium was normal but serum creatinine was 165 µmole / L, chest x-ray showed enlarged right Para-tracheal lymph nodes and CT scan of the chest showed mediastinal lymphadenopathy. Endoscopic total thyroidectomy was done through the thoracic route with 30 degree telescope. Even though, both his recurrent laryngeal nerves (RLN) were identified clearly and safeguarded, he developed hoarseness of voice due to temporary right RLN palsy which showed marked improvement within three weeks. Post operatively his serum calcium was normal.

He was referred to the nuclear medicine department for further management

**Case Report 2:**

Ms. P 16 years old female patient presented with a painless lump in front of the neck for 2 years. The swelling was gradually enlarging in size. There was no feature of hyper or hypothyroidism. On examination, she had solitary thyroid nodule about 3 X 2.5 cms in size arising from the left lobe of the

thyroid. There was no palpable cervical lymph node. Her TSH, FT3, FT4 were within normal limit. FNAC revealed papillary carcinoma. She also under went endoscopic total thyroidectomy via thoracic route using 30 degree telescope. She was offered oral liquid within 8 hours and solid diet from the 1st post operative day and she was discharged on the 3rd post operative day. Post operatively there was no hypocalcaemia and no recurrent laryngeal nerve palsy. She was also referred to the nuclear medicine department for further management

**Operative Procedure:**

Under general anesthesia each patient was placed supine with neck extended as it is done for the open thyroidectomy. Three ports were used, one 10 mm in the mid line 5 cms below the supra-sternal notch for the telescope and two 5 mm lateral ports 5 cms lateral to the midline on either side just below the clavicle for the working instruments. Initially, the sub platismal plane was created with a pair of hemostat and Tubb's dilator (the instrument used for closed mitral commissurotomy); later with a harmonic scalpel. This space- extended from the hyoid bone above to the clavicles below and laterally to the lateral borders of the sterno-cleido-mastoid muscles. The investing layer of the cervical fascia was divided in the mid line. Strap muscles retracted laterally to expose the gland. The lower pole of the involved thyroid lobe was pushed up and all vessels were divided with a harmonic scalpel. The recurrent laryngeal nerve was identified and lateral and posterior part of the gland was mobilized protecting the parathyroid glands and recurrent laryngeal nerve. The main trunk of inferior thyroid artery was preserved to maintain the blood supply to the parathyroid. Then the superior thyroid pedicle was divided with the harmonic scalpel. Once the involved lobe was completely mobilized, the other lobe was mobilized in the same way. The specimen was put in a plastic bag and removed through the 10 mm port which was stretched a bit to facilitate the delivery. The wound was irrigated with normal saline, hemostasis was checked and ports were closed after keeping a drain.

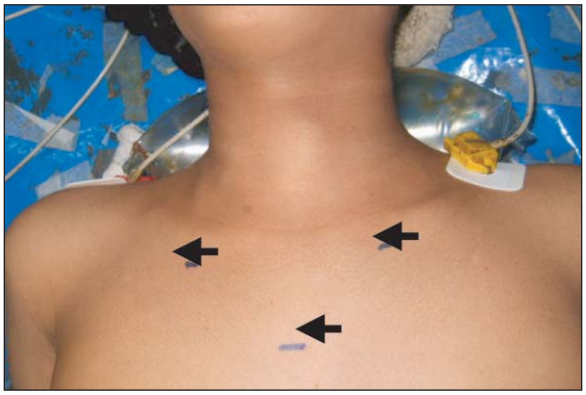

**Fig.-1:** *Neck position and port site marked. (in thyroid nodule)*

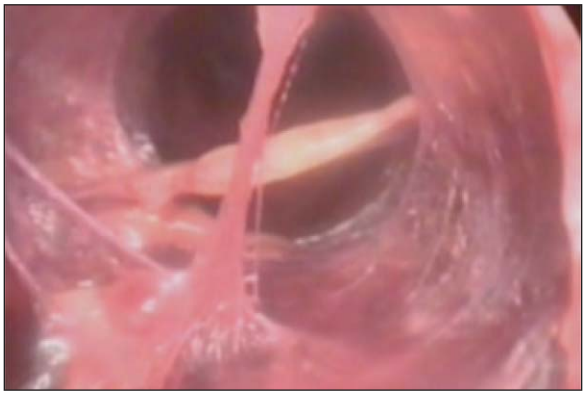

**Fig.-2:** *Creation of sub-platismal space*

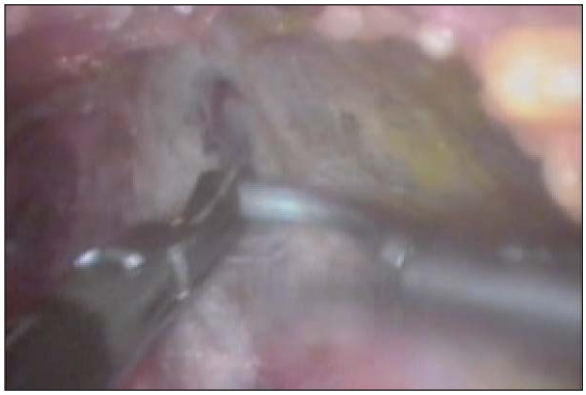

**Fig.-3:** *Sub platismal dissection*

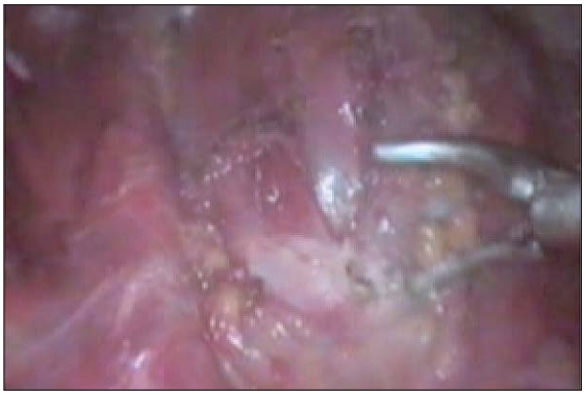

**Fig.-4:** *Strap muscles retracted.*

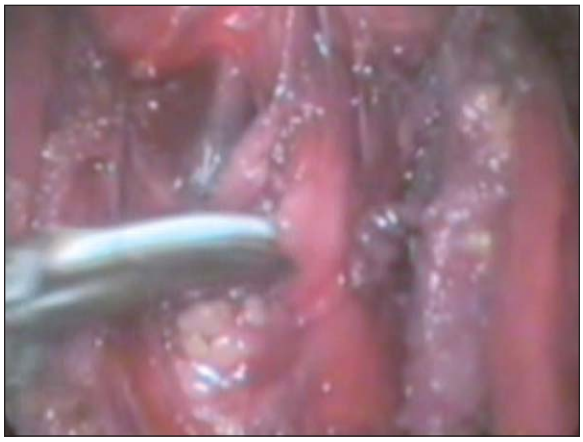

**Fig.-5:** *Inferior thyroid artery with parathyroid gland*

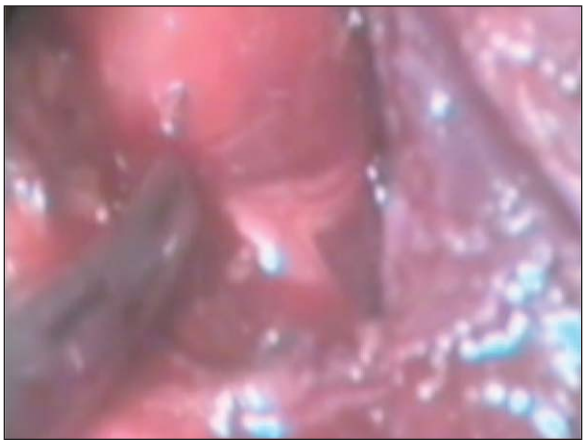

**Fig.-6:** *Left recurrent laryngeal nerve*

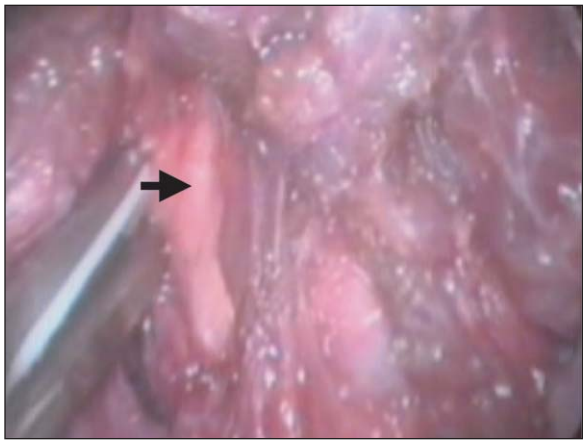

**Fig.-7:** *Right recurrent laryngeal nerve.*

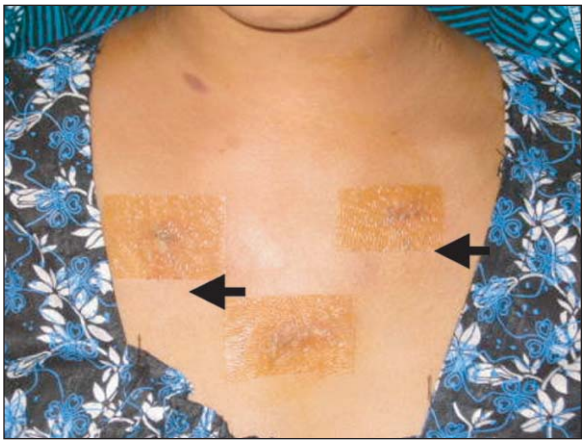

**Fig.-8:** *Port site after completion of operation.*

**Discussion:**

Endoscopic Thyroidectomy can be done through the cervical ports or via extra-cervical ports [2]. Both our patients had operation through the thoracic ports. In thoracic ports the scars are usually hidden under the garments. The sub –platismal plane is relatively avascular so creation of the sub-platismal space did not pose any difficulty. Moreover, the use of the Tubb's dilator and the harmonic scalpel made it much easier. Carbon-di -oxide insufflations at 5-10 mm of Hg pressure was used for the flap lifting and maintaining the working space. Although, skin traction could be used [3] but it was felt that it might give unnecessary scar in the neck. After the lifting of the flap, rest of the operation was essentially the same as the conventional open thyroidectomy.

It was felt that the magnified view obtained during the endoscopic surgery would make the identification of parathyroid glands and recurrent laryngeal nerves much easier. The main trunk of Inferior thyroid artery was protected to keep the blood supply of the parathyroid glands. The size of the thyroid nodules in both the patients were about 3 cms. In most of the series, endoscopic thyroidectomy was done for the selected group of thyroid patients where the size of the nodule was less than 3 cms, without any history of neck surgery, neck irradiation and evidence of thyroiditis [4.] Most of the authors reserved this for benign cases [2]. But both of the reported patients had malignancy. Here, total thyroidectomy was performed without any difficulty. Management of the lymph nodes in thyroid carcinoma may be an argument against endoscopic surgery but it was felt that an expert surgeon should be able to manage those cases endoscopically. Port site deposits may be another argument but to reduce that, the specimens

were removed within a plastic bag. Further study and long term follow up is needed to see whether this produce any significant problem or not.

The operation time for the first case was 240 minutes but for the second case 180 minutes. It is expected that the operation time would reduce further with experience.

None of the patients had any features of hypo-parathyroidism although the incidence of hypo-parathyroidism in total thyroidectomy [5] may be as high as 33 %. Even though, in every case both the recurrent laryngeal nerves (RLN) were identified during surgery, first patient had temporary RLN palsy which showed marked improvement with in three weeks of operation. The over all incidence of RLN palsy is quite low but the incidence varies quite widely with the type of surgery. In patients with total thyroidectomy the temporary and permanent RLN palsy may be as high as 13.6 % and 9% respectively [6]. Most important measure to preserve the RLN is to identify the nerve during surgery and where not possible intra-parenchymal dissection or subtotal excision is advocated by some authors [6].

In this series, none of the patients required any blood transfusion during surgery. Both patients required only one dose of pethedine. Both were satisfied with the post operative cosmetic appearance.

**Conclusion:**

Endoscopic total thyroidectomy for carcinoma of thyroid appears to be a technically feasible patient friendly modality. However, a dedicated surgical team and technical supports are essential. Long term follow up with a larger series is necessary to validate our present result.

**References:**

1. Krukowski ZH. The thyroid gland and the thyroglossal tract. In: Russell RCG, Williams NS and Bulstrode CJK (editors). Baily and Love's Short Practice of Surgery. 24th edition. London: Arnold, 2004; P, 776- 804.
2. Chowbey PK, Soni V, Khullar R, Sharma A, Baijal M. Endoscopic neck surgeryJ Min Access Surg 2007; 3: 3-7. .
3. Ambrosi A, Fersini A, Tartaglia N, Prete FP, Natale F, Lorusso G, Giannone N, Samele F , Neri V. Video – assisted thyroidectomy with minimally invasive central cervicotomy: initial experience in an endocrine surgery division. Chir Ital. 2006 Sept-Oct; 58(5): 549-56.
4. Sebag F, Palazzo FF, Harding J, Sierra M, Ippolito G, Henry JF. Endoscopic lateral approach thyroid lobectomy; safe evolution from endoscopic parathyroidectomies. World J Surg. 2006 May; 30(5): 802-5.
5. Islam KMT. Fluctuation of serum calcium level after thyroidectomy and rationality of calcium supplementation. Dissertation , BCPS, 2005; 120-130.
6. Avtac B, Karamercan A. Recurrent laryngeal nerve injuries and preservation in thyroidectomy. Saudi Med J. 2005 Nov; 26(11): 1746-9.

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# Loss of vision in a bleeding peptic ulcer patient following resuscitation - an unusual cause of non-arteritic anterior ischaemic optic neuropathy

MSH MAJUMDER

**Summary:**

***A 55-year old male, known to have peptic ulcer disease for 4 years, started haematamesis and melaena in a rural area of Bangladesh and was admitted into hospital with circulatory collapse. He was resuscitated with 8 units of whole blood transfusion and became stable. But he developed sudden loss of vision and fundoscopy revealed bilateral papilloedema but CT scan appeared normal. Clinical and ophthalmological evaluation was consistent with non-arteritic anterior ischaemic optic neuropathy.***

***Key words : optic neuropathy ; peptic ulcer***

***(J Bangladesh Coll Phys Surg 2008; 26: 50-54)***

## Introduction:

Peptic ulcer disease is very common in our population and with the availability of newer H2-blockers, proton pump inhibitors and antihelicobacter pylori regimens, the complications of peptic ulcer disease, like bleeding, perforation, obstruction etc. have not been reduced that much significantly. The important and fatal complication of peptic ulcer disease is bleeding from ulcer site. Bulk majority of upper gastrointestinal bleeding constitutes bleeding from peptic ulcer site and may bring to mortality if not managed properly. Severe bleeding leads to haemorrhagic shock and multiorgan failure, endangering the life [1-4]. Loss of vision which can be unilateral or bilateral, transient or persistent ; is a very rare consequence of severe bleeding. Severe haemorrhage can cause sudden bilateral loss of vision by ischaemia of optic nerve head called anterior ischaemic optic neuropathy. Anterior ischaemic optic neuropathy is classified as arteritic and non-arteritic form. Giant cell arteritis is considered as an important cause of arteritic form of anterior ischaemic optic neuropathy. Non-arteritic anterior ischaemic optic neuropathy is a partial or total infarction of the optic nerve head caused by occlusion of the short posterior ciliary arteries; leads to optic disc swelling and loss of vision [5-9].

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**Received:** 17 December, 2006 **Accepted:** 21 April, 2007

## Case report:

A 55-year old non-diabetic, normotensive, ex-smoker, known peptic ulcer disease for four years, started upper gastrointestinal bleeding, manifested as haematamesis and melaena in a rural area of Bangladesh. He was brought to hospital in a collapsed state - altered mental state, low volume rapid pulse, low blood pressure and urine volume supression. He was managed by oxygen inhalation, intravenous fluids and blood transfusion after blood grouping and cross matching, intravenous ranitidine and dopamine infusion. His haematamesis and melaena continued for four days and blood was also transfused simultaneously, a total of eight units of human whole blood. His Hb fell to 4g/dl, slight increase in urea, normal serum electrolytes, normal blood sugar and liver function tests. After settling general condition, he was undergone upper gastrointestinal endoscopy, revealing no oesophageal or gastric varices but gastric ulcer with evidence of recent bleeding and deformed duodenal bulb ( Fig. 1). The patient became stable and about to discharge the next day but unfortunately, he developed bilateral loss of vision. His wife was saying that, on awakening at morning, her husband asked her, why night was so long. She surprised, saying that it was not night but morning and came to know that he could not see anything because of loss of vision. Fundoscopy revealed bilateral papilloedema with retinal haemorrages ; and central white spots called Roth's spots (Fig. - 2). A CT scan of brain was advised immediately which revealed neither intracranial space occupying lesions nor abnormality of ventricular system (Fig. - 3). So,

local causes of optic disc swelling were thoroughly searched, like optic neuritis, central retinal vein occlusion, optic nerve tumour, lymphoma or leukaemic infiltration etc ; and his both orbital cavities on CT scan did not show optic nerve tumour, orbital tumors or any other retro-orbital masses (Fig. - 4). He was having no clinical features suggestive of giant cell arteritis or other collagen vascular diseases. A diagnosis consistent with non-arteritic anterior ischaemic optic neuropathy was made. Fluorescent angiography was advised but regreted because of financial constrains. His visual acuity showed no perception of light. He was given antiulcer drugs, antihelicobacter pylori regimens, methycobalamine, vitamin B complexes ; and advised follow up visit for fundoscopy. Fundoscopy after one month showed bilateral secondary optic atrophy with vascular sheathing and absorption of retinal haemorrhages ( Fig.-5 ) ; and visual acuity still showed no perception of light. The next follow up visit - 5 months after becoming blind ; showed improvement of vision with perception of movement of objects.

*Photograph of the patient*

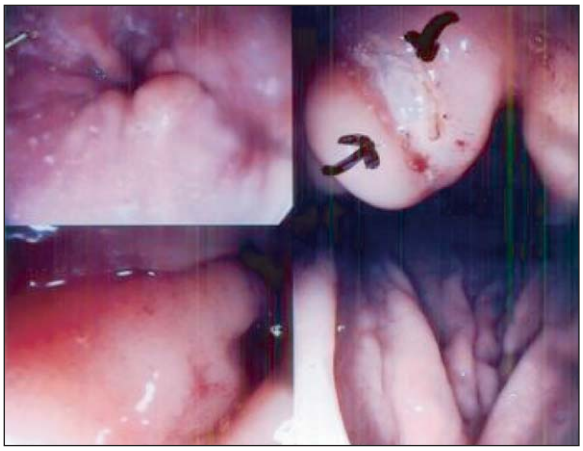

**Fig.-1:** *Upper G I Endoscopy showing gastric ulcer with evidence of recent bleeding and deformed duodenal bulb.*

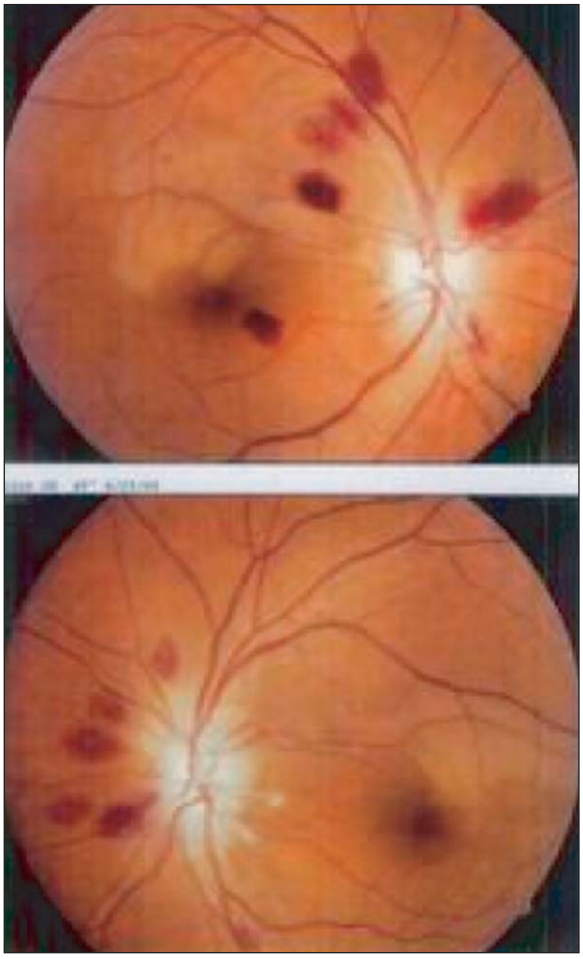

**Fig.-2:** *Bilateral papilloedema with retinal haemorrages*

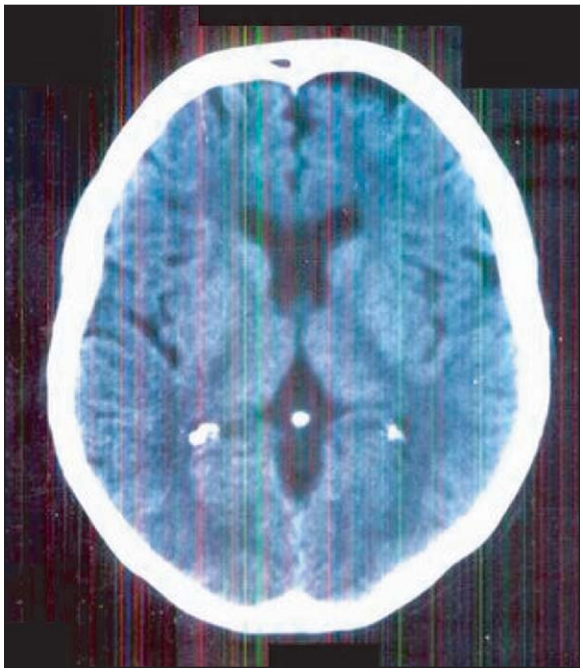

**Fig.-3:** *CT Scan of brain showing no intracranial space occupying lesions.*

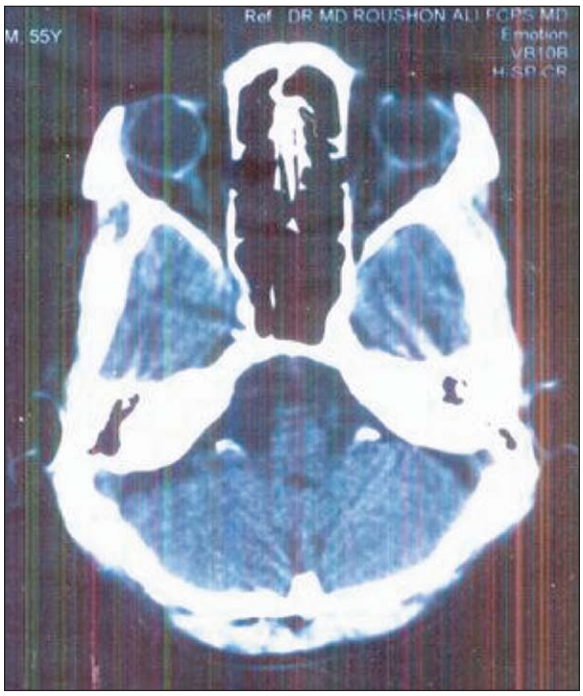


**Fig.-4:** *Normal orbit and retro-orbital structure*

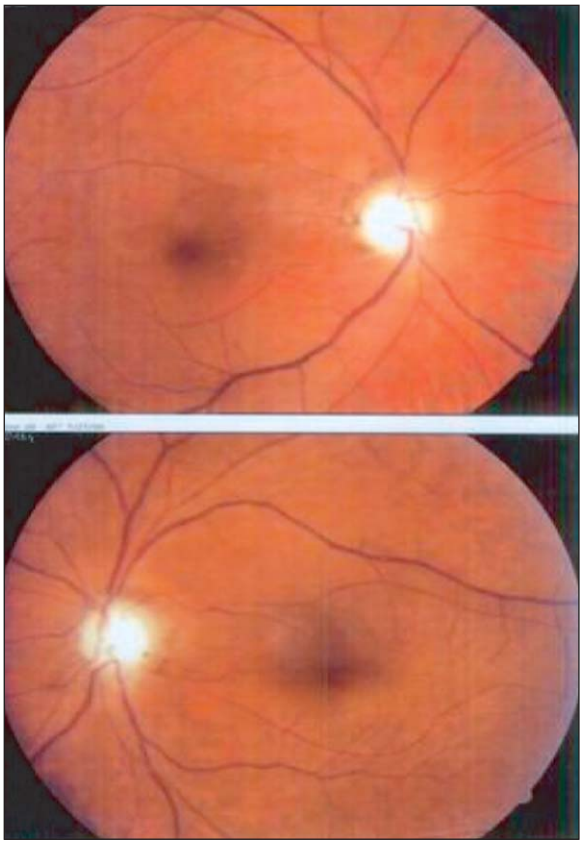

**Fig.-5:** *Bilateral secondary optic atrophy*

**Discussion:**

Bleeding peptic ulcer disease is a medical emergency and we usually concentrate to resuscitate the patient with blood transfusion, and also to find out the cause. We rarely consider blindness as a complication of bleeding peptic ulcer disease. In this patient, immediate fundoscopy following loss of vision showed bilateral optic disc swelling or papilloedema. Optic disc or optic nerve head swelling may be caused by a variety of conditions that are conviniently classified into papilloedema, pseudopapilloedema, and local causes. The clinician must be able to differentiate whether the swelling indicates serious life or sight threatening pathology, or is a coincidental finding of little significance. Papilloedema refers to optic disc swelling caused by raised intracranial pressure. Pseudopapilloedema refers to those benign conditions that resemble papilloedema either by the manner of presentation, or by the disc appearance,

causes include optic disc drusens or anomalous disc. Local causes of optic disc swelling include a variety of conditions for which careful examination of visual fields, fundus, vitrous and orbit usually provides a diagnosis. The two most common, anterior ischaemic optic neuropathy and demylinating optic neuritis, present with characteristics symptoms and signs [8-13].

Anterior ischaemic optic neuropathy refers to optic disc swelling and loss of vision caused by ischaemia in the territory of the short posterior ciliary arteries and subdivision into a non-arteritic form and an arteritic form serves a useful clinical functions. The arteritic form of AION (Anterior Ischaemic Optic Neuropathy) is mostly due to giant cell arteritis which can be diagnosed by characteristic features of headache, jaw pain and scalp tenderness over temporal artery region; high ESR and C-reactive protein; and characteristic temporal artery biopsy [8,9].

Non-arteritic anterior ischaemic optic neuropathy is a partial or total infarction of the optic nerve head, also caused by occlusion of the short posterior ciliary arteries, typically occurs between ages of 45 and 65 years with structural crowding of optic nerve head. Predisposing systemic conditions include smoking, hypertension,diabetis mellitus, hypercholesterolaemia, collagen vascular disease, hypotensive events and cataract surgery [8-10]. Presentation is with sudden, painless unilateral visual loss which is frequently discovered on awakening, suggesting that nocturnal hypotension may play an important role. Simultaneous bilateral non-arteritic ischaemic optic neuropathy is very rare, but may follow major haemorrhage. In the reported case, this was due to major haemorrhage or may be due to sustained hypotension. Major haemorrhage, whatever may be the cause, can lead to ischaemic optic neuropathy. Over a period of 6 months, 40 percent of affected eyes show a spontaneous improvement, whilst 20 percent deteriorate [9]. There is an increased incidence of cerebrovascular and cardiovascular disease. Decrease of blood pressure or arterial hypotension may be considered as a risk factor of non-arteritic anterior ischemic optic neuropathy ( NAION ), leading to a vascular insufficiency in the optic nerve head [14-17]. This patient had sustained arterial hypotension while he was having bleeding for four days.

**Conclusion:**

Severe haemorrhage is always considered as a life threatening condition and we would like to emphasize that it is not only life threatening but can also be sight threatening, even if life could have been saved. So, correction of underlying cause and adequate measures can prevent or minimize loss of vision.

**Acknowledgement:**

My deepest gratitude and gratefulness are extended to the patient Mr. Chand Mia and all his family members for their help and co-operation.

**References:**

1. Palmer KR, Penmar ID, Paperson S. Alimentary tract and pancreatic disease. In: Haslett C, Chilver ER, Hunter JAA, Boon NA, editors. Davidson's principles & practice of medicine. 19th ed. Edinberg : Churchill Livingstone; 2002. p. 764-6, 782-8.
2. Laine L. Gastrointestinal bleeding. In: Kasper DL, Fauci AS, Braunwald E,. Isselbacher KJ, Wilson JD, Martin JB,et al. editors. Harrison's principles of internal medicine. 16th ed. New York : McGraw-Hill Companies ; 2005. vol-1, p. 235-8.
3. McQuaid KR. Alimentary tract. In:Tierney LM, McPhee SJ, Papadakis MA, editors. Current medical diagnosis & treatment. 44th ed. New York : Lange Medical Books / Mc Graw Hill ; 2005. p. 533-6.
4. Abraham Bogoch. Bleeding from alimentary tract. In: Haubrich WS, Schaffner F, editors. Bockus gastroenterology. 5th ed. Philadelphia : W.B. Saunders Company ; 1995. vol-1, p. 62-84.
5. Pounder RE, Eraser AG. Diagnosis, medical management and complications of peptic ulcer disease. In: Haubrich WS, Schaffner F, editors. Bockus gastroenterology. 5th ed. Philadelphia : W.B. Saunders Company ; 1995. vol-1, p. 770-77.
6. Longmore M, Wilkinson IB, Rajagopalan SR. Oxford hand book of clinical medicine. 6th ed. Oxford : Oxford University Press ;2004. p. 224-6, 804-5.
7. Cello JP. Gastrointestinal haemorrhage. In: WynGaarden JB, Smith LH, Bennett JC, editors. Ceceil text book of medicine.19th ed. Philadelphia : W. B. Saunders Company ; 1992. vol-1, p. 742-6.
8. Kanski JJ. Clinical ophthalmology. 5th ed. London : Butterworth Heinemann ; 2003. p. 603-6.

9. Burdon MA, Saunder MD. Papilloedema, pseudopapilloedema and local causes of optic disc swelling. In: Easty DL, Sparrow JM, editors .Oxford text book of ophthalmology. Ist ed. 1999. vol-2 p. 830-7.

10. Anderson DR, Quigley HA. The optic nerve. In: Hart WM, editor. Adler's physiology of eye . 9th ed. Harcourt Brace & Company ; 1992: p. 616-24.

11. Targownik LE, Nabalamba A. Trends in management and outcome of acute nonvariceal upper gastrointestinal bleeding ; 1993-2003. Clin Gastroenterol Hepatol 2006 ; 11 (10) : p. 16 – 9.

12. Collins D, Worthley LI. Acute gastrointestinal bleeding : part 1. Crit Care Resusc 2001 ; 6 ; 3 (2) : p. 105-16.

13. Arber N, Tiomny E, Hallak A, Santo M, Moshkowitz M, Konikoff FM, et al. An eight year experience wih upper gastrointestinal bleeding : diagnosis, treatment and prognosis. J Med 1994 ; 25(5) : p. 261-9.

14. Bulboaca A, Nicula C. Arterial hypotension - risk factor in nonarteritic anterior ischemic optic neuropathy. Oftalmologia 2002 ; 53(2) : p. 52-5.

15. Tsai RK, Liu YT, Su MY. Risk factors of non-arteritic anterior ischemic optic neuropathy (NAION) : ocular or systemic. Kaohsiung J Med Sci 1998 ; 4 ; 14(4) : p. 221-5.

16. Sawle GV, James CB, Russell RW. The natural history of non-arteritic anterior ischemic optic neuropathy. J Neurol Neurosurg Psychiatry 1990 ; 10 ; 53(10) : p. 830-3.

17. Hayreh SS. Anterior ischemic optic neuropathy. Differentiation of arteritic from non-arteritic type and its management. Eye 1990 ; 4 (1) : p. 25-41.

# *COLLEGE NEWS*

*(J Bangladesh Coll Phys Surg 2008; 26: 55-57)*

## Schedule of Continuing Professionals Development Lectures during November to December, 2007

| Date | Time | Topic | Speaker | Chairperson / Moderator |
|---|---|---|---|---|
| 06-11-07 Tuesday | 11-00am to 11-50am | " Assisted Reproductive Technology-Technique (documentary) and limitations." | **Dr. Mosammat Rasida Begum** FCPS ( Obst. & Gynae) Assistant Professor of Obst. & Gynae DMC, Dhaka. MS ( Med.Edu,UK) M.Sc (Assisted Reproductive Technology, UK) | **Prof. Latifa Shamssuddin** (Chairperson) Prof. of Obst. & Gynae<br>**Dr. Rumana Shaikh** (Chairperson) Assoc. Prof. of Gynae<br>**Dr. Nahla Bari** (Moderator) Asst. Prof. Gynae |
| | 11-50 am to 12-10pm | TEA | | |
| | 12-10 pm to 1-00 pm | " Opthalmoscopic findings of Systemic disease" | **Prof. Dr. Md. Arif Mian** MBBS; D.O; F.C.P.S Professor and Head Dept. of Opthalmology SSMC & Midford Hospital, Dhaka. | **Prof. Md. Israfil (Opth.)** (Chairperson)<br>**Prof. HAM Nazmul Ahasan** (Chair Per.) Prof. of Medicine, DMC<br>**Dr. Md. Shahinur Rahman** (Moderator) Asst. Prof. of Ophthalmology |
| 13-11-07 Tuesday | 11-00 am to 11-50 am | " An update on rehabilitation in Spinal Cord Injury. | Prof. Dr. Sohely Rahman MBBS, FCPS (Physical Medicine) Prof. & Head, Dept. of Physical Medicine Begum Khaleda Zia Medical College & Shaheed Suhrawardi Hospital Sher-e-Bangla Nagar Dhaka-1207. | **Prof. Md. Moyeenuzzaman** (Chair P.) Prof. of Physical Medicine<br>**Dr. Md. Abdur Rashid** (Chair P.) Assoc. Prof. Physical Medicine<br>**Dr. Fahmida Hafez** (Moderator) Asst. Prof. Physical Medicine |
| | 11-50 am to 12-10 pm | "TEA | | |
| | 12-10 pm to 1-00 pm | " Sleep disorders" | **Dr. Md. Ashraf ali** Professor of Medicine Dhaka National Medical College Dhaka. | **Prof. A.K.M. Anisul Haque** (Chair P.) Prof. of Neuro-medicine<br>**Dr. A.K.M. Mosharraf** Hossain (Chair P.) Assoc. Prof. of Respiratory Medicine<br>**Dr. A.K.M. Shamsul Kabir** (Moderator) Asst. Prof. of Medicine |

| Date | Time | Topic | Speaker | Chairperson / Moderator |
|---|---|---|---|---|
| 20-11-07 Tuesday | 11-00am to 11-50am | " Anti- hypertensives in hypertensive disorders of pregnancy" | **Prof. Firoza Begum** Prof .obst.& Gynae BSMMU, Shahbag, Dhaka. | **Prof. Md. Jalaluddin** (Chairperson) Prof. of Cardiology (Retd.) **Prof. Sultana Razia Begum** (Chair. P.) Prof. of Gynae, BSMMU **Dr. Arzu Manth Ara Begum** (Moderator) Assoc. Prof. Gynae |
| | 11-50 am to 12-10pm | TEA | | |
| | 12-10 pm to 1-00 pm | " Criterion Based Medical Audit of Severe Acute Maternal Morbidity" | **Dr. Iffat Ara** Assoc. Prof. Obst. & Gynae Faculty Institute of Child & Mother Health (ICMH) | **Professor Rehana Begum** (Chair P.) Professor of Obst. & Gynae **Professor Rahima Begum** (Chair P.) Professor of Obst. & Gynae **Dr. Tarafdar Runa Laila** (Moderator) Asst. Professor of Obst. & Gynae |
| 27-11-07 Tuesday | 11-00am to 11-50am | " Management of Chronic Hepatitis 'C': An update" | **Dr. Col. Md Mokhlesur Rahman** Classified Medical Specialist & Gastroenterologist CMH, Dhaka Cantonment, Dhaka. | **Prof. Salimur Rahman** (Chair P.) Prof. of Hepatology, BSMMU **Dr. Md. Abdul Masud** (Chair P.) Assoc. Prof. of Gastroenterology **Dr. Md. Shahinul Alam** (Moderator) Asst. Prof. of Hepatology, BSMMU |
| | 11-50 am to 12-10pm | TEA | | |
| | 12-10 pm to 1-00 pm | " Basic Management of war injuries/ Mass casualties". | **Dr. (Major) Md Abdul Hannan** FCPS (Surgery) Surg Lt Commander BN Graded Specialist in Surgery BNS Upasham, Khulna. | **Prof. Brig. Gen. Anjan Kumar Deb** (Chair P.) Prof. of Surgery (Retd.) **Prof. A. N. M. Zia-ur-Rahman** (Chair P.) Prof. of Surgery, SSMC **Dr. Nishat Begum** (Moderator) Assoc. Prof. of Surgery, DMC |

| Date | Time | Topic | Speaker | Chairperson / Moderator |
|---|---|---|---|---|
| 04-12-07 Tuesday | 11-00am to 11-50am | " Techniques of Obstetric Fistula Repair-Past, Present & Future" | **Prof. Sayeba Akhter** Prof. of Obst & Gynae BSMMU, Dhaka. | **Prof. Kohinoor Begum** (Chair P.) Prof. & Head, Dept. of Gynae<br><br>**Prof. Firoza Begum** (Chair P.) Prof. of Gynae, BSMMU<br><br>**Dr. Meherun Nessa** (Moderator) Asst. Prof. Gynae |
| | 11-50 am to 12-10pm | TEA | | |
| | 12-10 pm to 1-00 pm | "Role of MR imaging in Backache" | **Dr. Nila Kanta Paul** Asst. Prof. & Head, Dept. of Radiology Sylhet MAG Osmani Medical College | **Dr. Brig. Gen. Chowdhury Abdul Gaffar** (Chair P.) Prof. of Radiology (Retd.), BMC<br><br>**Prof. Muhammad Mahbubur Rahman** (Chair P.) Prof. & Head, Dept. of Radiology, NICVD.<br><br>**Dr. Md. Salahuddin Al-Azad** (Moderator) Assoc. Prof. of Radiology, BSMMU |
| 11-12-07 Tuesday | 11-00am to 11-50am | "Fertility control A cohort study | **Dr. Rabeya Akther** Senior Medical Officer Bangladesh Bank Medical Centre Motijheel, Dhaka. | **Prof. A. K.M. Anowarul Azim** (Chairperson.) Prof. of Gynae, NMC&H<br><br>**Prof. Atika Begum** (Chairperson) Prof. of Gynae , S.S.H<br><br>**Dr. Syeda Farida Begum** (Moderator) Assoc. Prof. of Gynae, BMC |
| | 11-50 am to 12-10pm | "TEA | | |
| | 12-10 pm to 1-00 pm | " Surgical Infection" | **Dr. Kamrun Nahar** Asst. Professor, Colorectal Surgery BSMMU, Dhaka. | **Prof. Md. Abdul Awal** (Chairperson) Prof. of Surgery (Retd.)<br><br>**Prof. Syed Serajul Karim** (Chairperson) Prof. of Surgery, BSMMU<br><br>**Dr. Md. Kamrul Islam** (Moderator) Asst. Prof. (Transplant Surgery), NIKDU |