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# JOURNAL OF BANGLADESH COLLEGE OF PHYSICIANS AND SURGEONS

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## *EDITORIAL*

# Communication Skill in Medicine

Health care professionals including doctors need to communicate with patients or their parties in their day to day activities. It is an essential activity of doctors. In the present medical curriculum emphasis has been given to learn communication skill by the future doctors.

**How to improve communication between doctors and patients?**

Communication difficulties between doctors and patients have been looked at by researchers from several disciplines who have tried to explore why these occur. Mishler, for example, has argued that doctors and patients talk to each other in different voices. The voice of medicine is characterized by medical terminology, objective descriptions of physical symptoms. The voice of patients, on the other hand, is characterized by non-technical terminology about the subjective experience of illness within the context of social relationships and the patient's everyday world. Typically, doctors have more power than patients to structure the nature of the interaction between them. As a consequence, patients may feel that their voice is overridden, silenced, or stripped of personal meaning and social context. To improve communications between doctors and patients we also need to understand the nature of the decision making that is taking place in the consultation.

**Why is good communication important?**

The communication skill means 'better care for our patients'. There is considerable evidence to show that doctors who communicate well with patients are more likely to:

1. Make an accurate, comprehensive diagnosis.
2. Detect emotional distress in patients.
3. Have patients who are satisfied with the care they have received and who are less anxious about their problem.
4. Have patients who agree with and follow the advice given.

In countries where patients are less likely to sue their doctors, patients still express dissatisfaction about how doctors communicate and relate to them.

**Can communication skills be learned?**

Training to be a doctor involves the acquisition of knowledge, skills and appropriate attitudes. Like many aspects of medical education, it was assumed until fairly recently that students acquire good communication skills and appropriate attitudes by a sort of osmosis- by observing and modeling their behavior on that of their teachers.

In the 1970's a series of studies was carried out on medical students during their fourth-year clerkship in psychiatry. The study found that before training, students experienced difficulties in obtaining histories from patients. The difficulties which were highlighted included:

- Not obtaining all the necessary information from the patient.
- Forgetting to ask about the influence of the patient's problems on themselves and their family.
- Failing to notice and respond to verbal and non-verbal cues from the patient.
- Looking bored during the interview.

It was found that the students who had received feedback training were better at communicating with patients.

**Patient-Physician communication: Why and How?**

Patient-physician communication is an integral part of clinical practice. When done well, such communication produces a therapeutic effect for the patient, as has been validated in controlled studies. Formal training programs have been created to enhance and measure specific communication skills. Many of these efforts, however, focus on medical schools and early postgraduate years and, therefore, remain isolated in academic settings. Thus, the communication skills of the busy physician often remain poorly developed, and the need for established physicians to become better communicants continues.

The manner in which a physician communicates information to a patient is as important as the information being communicated. Patients who understand their doctors are more likely to acknowledge health problems, understand their treatment options, modify their behavior accordingly, and follow their medication schedules.

**Why Bother Communicating with patients?**

**A Reminder about the value of communication**

From obtaining the patient's medical history to conveying a treatment plan, the physician's relationship with his patient is built on effective communication. In these encounters, both verbal and nonverbal forms of communication constitute this essential feature of medical practice.

**How to Communicate with Patients**

**Reminder for the busy Physician**

Medical professionals debate the best strategies for the effective communication, as well as the ability of these strategies to be taught or evaluated objectively. Certainly, each physician must develop his or her own style of communication. At the same time, many professional and academic organizations have now also defined key elements of communications skills needed by physicians. For example, the Accreditation Council for Graduate Medical Education recommends that physicians become competent in five key communication skills: 1) listening effectively; 2) eliciting information using effective questioning skills; 3) providing information using effective explanatory skills; 4) counseling and educating patients; and 5) making informed decisions based on patient information and preference.

**Assess What the Patient Already Knows**

Before providing information, find out what the patient already knows about his or her condition. Many times, other physicians or health care providers have already communicated information to the patient, which can have the effect of coloring patient perceptions and perhaps even causing confusion when new information is introduced.

**Assess What the Patient Wants to Know**

Not all patients with the same diagnosis want the same level of detail in the information offered about their condition or treatment. Studies have categorized patients on a continuum of information-seeking behavior, from those who want very little information to those who want every detail the physician can offer. Thus, physicians should assess whether the patient desires, or will be able to comprehend, additional information.

**Be Empathic**

Empathy is a basic skill physicians should develop to help them recognize the indirectly expressed of their patients. Once recognized, these emotions need to be acknowledged and further explored during the patient-physician encounter.

**Slow down**

Physicians who provide information in a slow and deliberate fashion allow the time needed for patients to comprehend the new information. Other techniques physicians can use to allow time include pausing frequency and reinforcing silence with appropriate body language. A slow delivery of with appropriate pauses also gives the listener time to formulate questions, which the physician can then use to provide further bits of targeted information. Thus, a dialogue punctuated with pauses leads to deeper comprehension on both sides.

One study found that physicians typically wait only 23 seconds after a patient begins describing his chief complain before interrupting and redirecting the discussion. Such premature redirection can lead to late-arising concerns and missed opportunities to gather important data.

As a side note, patient satisfaction is also greater when the length of the office visit matches his or her pre-visit expectation. In situations involving the delivery of bad news, the technique of simply stating the news and pausing can be particularly helpful in ensuring that the patient and patient's family fully receive and understand the information. Allowing this time for silence, tears, and questions can be essential.

**Keep it Simple**

Physicians should avoid engaging in long monologues in front of the patient. Far better for the physician to keep to short statements and clear, simple explanations.

**Tell the Truth**

It is important to be truthful. In addition, it is important that physicians not minimize the impact of

what they are saying. Saying that a patient has 'gone' or has 'left us,' for an example, could be interpreted by an anxious family member as meaning that the patient has left his room to have a radiologic film taken or to undergo a test. Alternatively, physicians who use "D" words (eg, dying, died, dead) when appropriate, effectively communicate and minimize confusion.

**Be Hopeful**

Although the need for truth-telling remains primary, the therapeutic value of conveying hope in situations that may appear hopeless should not be underestimated. Particularly in the context of terminal illness and end-of-life care, hope should not be discouraged.

For example, in situations such as the imminent death of a patient, hope can be conveyed to the family by assuring them that therapy can be effective in allaying pain and discomfort. Thus, even when physicians must convey a grim prognosis to a patient or must discuss the same with family members, being able to promise comfort and minimal suffering has real value.

**Watch the Patient's Body and Face**

Much of what is conveyed between a physician and patient in a clinical encounter occurs through nonverbal communication.

For both physician and patient, images of body language and facial expressions will likely be remembered longer after the encounter than any memory of spoken words.

It is also important to recognize that the patient-physician encounter involves a two way exchange of nonverbal information. Patient's facial expressions are often good indicators of sadness, worry, or anxiety. The physician who responds with appropriate concern to these nonverbal cues will likely impact the patient's illness to a greater degree than the physician wanting to strictly convey factual information.

**Be Prepared for a Reaction**

Patients vary, not only in their willingness and ability to absorb information, but in their reactions to physician communications. Most physicians quickly develop a sense for the various coping styles of patients, a range of human reactions that has been categorized in several specific clinical settings.

In responding to any of these patient reactions, it is important to be prepared. The first step is for the physician to recognize the response, allowing sufficient time for a full display of emotions. Most importantly, the physician simply needs to listen quietly and attentively to what the patient or family is saying. Sometimes, the physician can encourage patients to express emotions, perhaps even asking them to describe their feelings. The physician's body language can be crucial in conveying empathic concern in these encounters.

**Conclusion:**

Simple choices in words, information depth, speech patterns, body position, and facial expression can greatly affect the quality of one-to-one communication between the patient and physician. To a large degree, these are conscious choices that can be learned and customized by the physician to fit particular patients in clinical situations. Avoiding communications pitfalls and sharpening the basic communication skills can help strengthen the patient-physician bond that many patients and physicians believe is lacking.

There is no shortcut for physician-patient interaction. We have to acknowledge the need of good physician-patient interaction for wide range of benefit out of it. It is our collective responsibility to teach our 'future doctors' this important but basic skill in medicine.

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***(J Bangladesh Coll Phys Surg 2007; 25 : 115)***

**Further reading:**

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2. Silverman J, Kurtz S and Drapper J (eds) (2005) Skills for Communicating with Patients. Radcliffe Publishing Limited, Oxford.
3. Lloyd M and Bor R (1996) Communication Skills for Medicine. Churchill Livingstone, New York.

# Evaluation of Iron Status by Bone Marrow Iron Stain and its Correlation with Serum Iron Profile in Chronic Kidney Disease (CKD)

MM RAHMAN[a], PK DUTTA[b], M HOQUE[c], MIH KHAN[d], D BANIK[e], AK DUTTA[f], MU HASAN[g], EB YUNUS[h], ABM YUNUS[i], MJ RAHMAN[J]

**Summary:**

***This observational study was done on 52 cases of pre-dialysis chronic kidney disease (CKD) patients with chronic anaemia. The aim of the study was to determine the tissue iron status, comparison of the tissue iron with serum iron profile and justification of giving iron in chronic kidney disease (CKD) patients on the basis of serum iron profile. Bone marrow iron stain was done in each case and compared with the serum iron profile. The mean age of the patients was 46.8 ± 12.6 years and the mean haemoglobin and serum creatinine levels of the study population were 9.36 ± 2.13 gm/dl and 8.0 ± 4.2 mg/dl respectively. Stainable iron deposits were present in 40 (77%) cases. The mean serum ferritin and transferin saturation (TSAT) of the 52 cases were found to be 412.9 ng/ml and 28.3% and that for the 12 iron deficient cases were 101.8 ng/ml and 23.8%. Over all normal (>100ng/ml <500ng/ml), increased (>500ng/ml) or low (<100 ng/ml) serum ferritin was found in 28 and 15 and nine cases respectively. On the other hand, normal (>20% <50%) and low (<20%) TSAT were found in 31 and 12 cases, and high TSAT (>50%) in only nine cases. Out of the 12 cases having no evidence of stainable iron in the marrow low serum ferritin and low TSAT were found in eight (66.6%) and six (50%) cases, and high TSAT and either normal or high serum ferritin in six (50%) & four (33.3%) cases respectively. Low TSAT was also found in six (15%) cases of those having iron deposits in the marrow. It is, therefore, concluded that absence of stainable iron in the bone marrow is a better evidence of iron depletion than the serum iron profile and that serum ferritin and TSAT correlate less well with the bone marrow iron status in patient with chronic kidney disease.***

***(J Bangladesh Coll Phys Surg 2007; 25 : 117-120)***

**Introduction:**

Iron deficiency whether absolute or functional is an important cause of anaemia in patients with chronic kidney disease [1]. Iron deficiency anaemia is treated either by oral or parenteral iron therapy, or by blood transfusion. Improvement of hemoglobin level in CKD improves performance status, reduces disease progression and decreases the rate of morbidity and mortality [2]. On the other hand, increased tissue iron can aggravate disease progression by precipitation of infection and generation of hyper-reactive free radical mediated tissue injury [3, 4]. Therefore, it is important to know the exact body iron status in CKD patients for the proper management of the patients. In normal healthy individual iron is stored in the liver, muscle and bone marrow. Bone marrow iron is contained mainly in the macrophages that release it to the developing erythron for the synthesis of haemoglobin.

Low serum iron, low serum ferritin, low TSAT and high concentration of transferin indicate iron deficiency. Serum ferritin level and TSAT are usually considered as corner stone for the diagnosis of iron deficiency. Though serum ferritin of less than 100 ng/ml and a STAT of less than 20% indicate iron deficiency in CKD, these two parameters fail to detect functional iron deficiency in all cases of CKD patients[5, 6, 7]. Increase in serum ferritin without actual

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changes in storage iron commonly occurs in a chronic disease[8]. Chronic renal failure (CRF) with occult infection may cause increased serum ferritin level irrespective of the actual iron storage. On the other hand, transferin saturation may also be misleading since nutritional status and loss in urine in nephrosis can affect transferin concentration in CRF [9, 10]. So, TSAT may decrease to a level consistent with iron deficiency yet serum iron may be normal or even elevated [7]. Tissue iron stain provides an important tool to evaluate the actual iron status in the body. In absolute iron deficiency no stainable iron can be detected in the tissue. Absence of stainable iron in the bone marrow is accepted as a gold standard for absolute iron deficiency [10]. In this study, the actual iron status in CKD patients was evaluated by detecting the stainable iron in the bone marrow and it was compared with biochemical parameters of serum iron.

**Materials and Methods**

This descriptive observational study was conducted in the Nephrology unit of Chittagong Medical College Hospital. A total of 52 CKD patients due to various causes with chronic anemia were included in this study (Fig-1). There were 34 male and 18 female patients. The age ranges of the patients were between 23 and 75 years. The inclusion criteria were pre-dialysis CKD patients with chronic anaemia having no history of active bleeding and who had never been treated with rHuEPO. CKD patients having a hemolytic disorder or primary hemosiderosis were excluded from the study. The characteristics of the patients are shown in Table-1.

The bone marrow was collected from the iliac crest with the help of a 'Salah' bone marrow aspiration needle under local anaesthesia. Subsequently, bone marrow iron was detected by 'Prussian blue' stain on smear prepared with definite marrow fragment whereby presence of siderotic granules in the macrophages appeared as either blue black dots or clumps. Scoring of stainable iron deposits in the marrow was done as 'absent' if such blue-black dot and/or clump were absent or as 'present' if such dots or clumps were present. At the same time, serum iron profile that includes the estimations of serum iron, total iron binding capacity (TIBC), serum ferritin and percent saturation of transferin (TSAT) were determined. Serum iron and TIBC were estimated by 'spectrophotometric' method (Photometer 5010, GMBH, Germany) where as Serum ferritin level was determined by 'chemiluminescence' method (IMMULYTE One System, DPC, USA). TSAT was calculated from the 'serum iron' and 'TIBC' in each case. The results were recorded in printed form and subsequently analyzed.

**Results:**

A total of 52 CKD patients were included in this study. There were 34 (65.4%) males and 18 (34.6%) females with a male to female ratio of 1.8:1. The mean age of the patients was 46.8 $\pm$ 12.6 years (range 23 – 75 years) and the mean haemoglobin and serum creatinine levels were 9.36 $\pm$ 2.13 gm/dl and 8.0 $\pm$ 4.2 mg/dl respectively. Out of these 52 patients stainable iron deposits were detected in 40 (77%) patients and the rest 12 (23%) patients had no stainable iron in the marrow. The mean serum iron, TIBC, serum ferritin and TSAT of the 52 cases were found to be 115.3 ug/dl, 421.9 ug/dl, 412.9 ng/ml and 28.3% respectively while in those of the 12 patients with no stainable iron in the marrow were found to be 114.5 ug/dl, 417 ug/dl, 101.8 ng/ml and 23.8% respectively (Table-II). Out of the 40 patients who had stainable iron deposits in the marrow, only one patient had low (<100 ng/ml) serum ferritin, 14 patients had high (>500 ng/ml) serum ferritin and 25 patients had serum ferritin level within the therapeutic range (100 – 500 ng/ml). On the other hand, out of the 12 patients with no stainable iron in the marrow eight patients had low serum ferritin, one patient had high serum ferritin and three patients had serum ferritin level within the therapeutic range. Low (<20%), high (>50%) and therapeutic range (20 – 50%) of TSAT were found in 6, 3, and 31 of those patients having detectable iron in the marrow where as low and high TSAT were found in equal number of 12 patients having no detectable iron deposits in the marrow (Table-III).

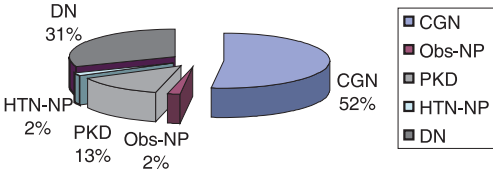


**Fig-1:** *Relative distribution of causes of CKD in 52 patients.*

**Table-I**

*Characteristics of 52 CKD patients*

| | |
|---|---|
| Total number of cases | 52 |
| Male | 34 (65.4%) |
| Female | 18 (34.6%) |
| Mean age | 46.8 ± 12.6 years |
| Mean haemoglobin | 9.36 ± 2.13 gm/dl |
| Mean serum creatinine | 8.0 ± 4.2 mg/dl |

**Table-II**

*Mean serum iron profile of 52 CKD patients*

| | All cases (n=52) | Iron deficient case (n=12) |
|---|---|---|
| S. iron (ug/dl) | 115.3 | 114.5 |
| TIBC (ug/dl) | 421.9 | 417.0 |
| S. ferritin (ng/ml) | 412.9 | 101.8 |
| TSAT (%) | 28.3 | 23.8 |

**Table-III**

*Comparison of serum ferritin and TAST with bone marrow iron status*

| Bone marrow iron status | Serum ferritin | | | TSAT | | |
|---|---|---|---|---|---|---|
| | <100 ng/ml | 100 - 500 ng/ml | >500 ng/ml | <20% | 20 – 50% | >50% |
| Bone marrow iron present n=40 | 01 | 25 | 14 | 06 | 31 | 03 |
| Bone marrow iron absent n=12 | 08 | 03 | 01 | 06 | 00 | 06 |
| n= 52 | 09 | 28 | 15 | 12 | 31 | 09 |

**Discussion:**

There are controversies prevailing about the best measure of the body iron status in CKD patients. In an otherwise healthy individual a TSAT of less than 16% and serum ferritin of less than 12 ng/ml indicates absolute iron deficiency. But in CKD patient, absolute iron deficiency is defined as having a serum ferritin level of less than 100 ng/ml and TSAT level of less than 20%[11]. The concept of functional iron deficiency further complicates the scenario. As both the serum ferritin and TSAT levels are affected by many factors besides body iron store, new parameters of iron status particularly in CKD patients undergoing haemodialysis and/or receiving rHuEPO have been evaluated [8, 9, 10, 12, 13, 14]. But absence of iron deposits in the bone marrow is still considered as gold standard for absolute iron deficiency[10].

NK/DOQI has recommended the therapeutic range of TSAT between 20% and 50% and serum ferritin between 100 and 500 ng/ml [11]. In this study of 52 non-dialysis patients it was found that 43 patients had serum ferritin either within or above this therapeutic range. 28 patients had serum ferritin between 100 and 500 ng/ml and 15 patients had more than 500 ng/ml. Only nine patients had serum ferritin below 100 ng/ml. On the other hand low TSAT (<20%) and high TSAT (>50%) were found in 12 and nine cases respectively. Therapeutic range of TSAT was found in 31 cases. The mean serum ferrtitin and TSAT level in these cases were 412.9 ng/dl and 28.3%. Thus, according to the NK/DOQI criteria, majority of study population were not actually iron deficient. These findings are in contrast with that of Gotloib et al who found mean serum ferritin and TSAT level of 235.9 ng/ml and 13.5% in a study of 47 CKD patients [15]. This contradiction justifies the concept that serum ferritin and TSAT are not absolute reliable parameters for the body iron status. Stainable iron deposits in the bone marrow were also evaluated in all of the cases. Out of 52 cases stainable iron deposits were found in 40 (77%) cases and absent in 12 (23%) cases. In one study, no evidence of stainable iron deposits was found in 46 out of 47 CKD patients with anaemia [15]. Other studies found diminished iron store in 29% cases of renal transplantation patients and 23% cases of CRF patients [16, 17]. Mean serum ferritin level in iron deficient CKD patients was found to be 70ng/ml in two studies that are in contrast to present findings [17, 18]. However, another study estimated diagnostic

threshold of ferritin for iron deficiency as 80-350 ng/ml [19]. They also found absence of iron in 76.4% patients on maintenance haemodialysis [19]. Here higher value of mean serum ferritin and TSAT level of 101.8 ng/ml and 23.8% were found in 12 iron deficient patients. There were also high TSAT and high or normal serum ferritin in six (50%) and four (33.3%) cases respectively of the 12 patients having no iron deposits and that low TSAT and low serum ferritin in six and one cases respectively of those 40 patients having iron deposits in the marrow.

Therefore, it is concluded that though iron deficiency is said to be an important cause of anaemia in CKD, many patients may not have iron deficiency. It is also concluded that serum iron profile correlate less well with the bone marrow iron status and that serum iron profile may be misleading in evaluating the actual status of the body iron store particularly in pre dialysis chronic kidney disease patients.

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# Effect of Supplementation on Weight Gain of Growth Faltered under 2 Children in A Rural Area of Bangladesh

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**Summary:**

***Effect of food supplementation for different duration on weight gain of growth faltered under 2 children was assessed in this study to identify an optimum duration for supplementation.***

***This was a longitudinal prospective study conducted among 510 children aged 6-23 months enrolled for food supplementation from 1st November 98 to 30th May' 99 under community based food supplementation program of Bangladesh Integrated Nutrition Project (BINP). The children were assigned in 3 groups of supplementation for 30, 60 and 90 days. All children were assessed for Graduation (weight gain 500 gm) at assigned duration of supplementation and followed for 90 days with monthly anthropometric monitoring.***

***The proportion of graduation was 21.7 percent (CI 15.8-28.7), 48.8 percent (CI 41.1-56.6) and 80.5 percent (CI 73.4-86.4) at 30, 60 and 90 days of supplementation respectively. There was no significant difference of mean weight gain between 60 and 90 days of supplementation. There was limited impact on weight gain once graduation was achieved.***

***As continuing supplementation will require more cost and graduated children could maintain weight gain with family diet it is recommended to assess for graduation after 60 days instead of existing practice of 90 days supplementation in the national nutrition program and consider 60 days supplementation as an optimum duration for growth faltered under 2 children.***

***(J Bangladesh Coll Phys Surg 2007; 25 : 121-125)***

## Introduction

Malnutrition has been declared as silent emergency by UNICEF[1]. Malnutrition is most prevalent among 6-23 months children as they are more prone to inadequate weaning practices[2]. In Bangladesh about 60 percent children between 6 & 71 months are malnourished with about 17% are wasted and 51.4% are stunted[3].

Supplementary feeding programs to improve nutritional status of malnourished children are in wide use in different countries[4] . The impact of such programs are inconsistent and vary according to variation in target population, timing of start of intervention and duration of supplementation[5,6]. Although the growth of malnourished child might continue with continued food supplementation there is chance of limited impact on growth once momentum of weight gain is reached[7].

To mitigate the problem of malnutrition among children Government of Bangladesh implemented a community based food supplementation program for growth faltered children aged 6 – 23 months through Bangladesh Integrated Nutrition Project (BINP) since 1995. The criteria of growth faltering under BINP program was weight loss or not gaining weight over last two months and weight gain less than 300 grams over three consecutive monthly growth monitoring and was accepted for this study[8] . The target for supplementation of BINP program was to achieve 500gm weight gain (graduation) after 90 days of supplementation and in the absence of such gain supplementation was continued for further 90 days.[8] The present study has been done to assess the effect of different length of supplementation on weight gain and to identify an optimum duration of supplementation for growth faltered children.

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**Methodology**

This was a longitudinal prospective study conducted during 1st November' 98 to 30th May'99 on 510 children aged 6-23 months enrolled for food supplementation under BINP program in Faridpur Sadar thana (sub district) of Bangladesh. This thana had 187 Community Nutrition Centres (CNC). All the CNCs were recruited and assigned to three groups randomly to provide supplementation for either 30, 60 (intervention group, A and B) and 90 days (control, group C). The control group CNCs followed existing program of supplementation and was required to compare the effects with intervention groups. The children at the particular CNC were enrolled consecutively. The supplement was a mixture of cereal consisting of rice 40 gms, pulse 20 gms, molasses 10 gms and oil 6 gms and yield approximately 150 Kcal to each child. The food was available in dry packets processed by trained community nutrition promoters (CNPs) and was monitored extensively for quality control. The dry material was prepared into semisolid by adding measured amount of water by CNP before being fed to the children at the CNC.

The graduation criteria in this study were weight gain of 500 gms at 30, 60 and 90 days of supplementation in group A, B and C respectively. Supplementation was discontinued among graduated children in group A and B after 30 and 60 days respectively, and in the absence of such weight gain supplementation was continued till 90 days. All children were followed up for 90days. The height and weight measurements were taken at enrolment and monthly by trained data collectors by using Salter scale with 100 gm precision and were done preferably in the morning before feeding. Height was measured by locally made wooden length board at the same time. If any child after graduation in respective group (30 and 60 days supplementation) becomes eligible for supplementation according to BINP criteria during follow up period they were free to enroll again in the supplementation program.

The ethical clearance was obtained from ethical review committee of Institute of Child and Mother Health. Using SPSS 7.5 software program statistical analyses were carried out. The nutritional status of the children expressed in Z scores.

**Results**

The Children in each group were comparable and did not differ in selected socio-demographic factors and anthropometric indices at enrolment (Table 1, 2). The distribution of drop out was 10, 3 and 8 in group A, B and C respectively mainly because of change of residence and 3 infants died. Proportion of graduation was 21.7 percent (CI 15.8-28.7), 48.8 percent (CI 41.1-56.6) and 80.5 percent (CI 73.4-86.4) after 30, 60 and 90 days of supplementation respectively (Figure –1). Mean weight gain at 90 days among the graduated children after 60 days supplementation was not different from those received supplementation for 90 days (p=0.92). Graduated children continue to gain weight without supplementation during 90 days follow up period (Table –3).

**Table-I**

*Socio Demographic Characteristics of Children at Enrolment (n=510)*

| Variables | Children group | | | |
|---|---|---|---|---|
| | A (n= 172) | B (n= 174) | C (n= 164) | P value |
| Age in months at enrolment | 12.81 ± 3.4 | 13.12±3.2 | 13.05 ±3.4 | 0.668 |
| Birth weight, kg (Mean ± SD) | 2.73 ±0.4 | 2.70± (0.4) | 2.8 ± 0.4 | 0.063 |
| SexBoy (%)Girl (%) | 45.354.7 | 46.054.0 | 46.353.7 | 0.0983 |
| No of Family members (Mean ± SD) | 5.27±1.7 | 5.60±2.0 | 5.46±2.1 | 0.030 |
| Mothers' education (Year ± SD) | 1.55±2.5 | 1.36±2.4 | 1.13±2.6 | 0.317 |
| Fathers' education (Year ± SD) | 2.22±3.3 | 2.07±2.4 | 2.01±3.3 | 0.829 |
| Monthly income | 2886.11±1153 | 2497.37±777 | 2686.75±958 | 0.250 |

P value reached from one way analysis of variance among 3 groups

**Table-II**

*Anthropometric Characteristics of Children at Enrolment (n=510)*

| Variables | Children Group | | | |
|---|---|---|---|---|
| | A (n= 172) | B(n= 174) | C (n= 164) | P value |
| Weight at enrolment, kg (Mean ± SD) | 6.88 ± 1.30 | 6.77 ± 1.28 | 6.9± 1.2 | 0.210 |
| Length at enrolment, cm (Mean ± SD) | 66.0± 6.0 | 66.0 ± 6.8 | 67.5± 6.1 | 0.048 |
| Weight for age[a] (Mean ± SD) | 69.61± 11.4 | 67.75± 10.3 | 66.78± 12.2 | 0.073 |
| Height for age[a] (Mean ± SD) | 87.41± 7.04 | 88.03 ± 5.8 | 89.07 ± 6.6 | 0.069 |
| W/A Z score | -3.10± 1.5 | -3.0± 1.35 | -2.79 ± 1.54 | 0.188 |
| H/A Z score | -2.70±1.0 | -2.9 ± 0.91 | -2.99± 1.08 | 0.029 |

a - expressed as percentage of NCHS reference value

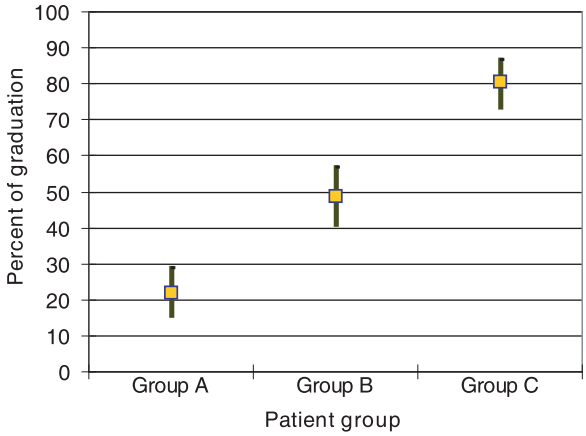


**Fig.-1:** *Graduation rate at completion of different duration of supplementation*

**Table-III**

*Sustainability weight gain*

| Characteristics | Mean weight gain | | | | |
|---|---|---|---|---|---|
| | Baseline | 30 d | 60 d | 90 d | P Value |
| Graduated & Supplementation for 30 days (n= 37) | 6.06 ±1.32 | 0.89 ± .69 | 1.1 ±0.92 | 1.33 ±0.93 | 0.21 |
| Graduated & Supplementation for 60 days (n= 83) | 6.35 ± 1.10 | 0.45 ±. 38 | 0.85 ±. 45 | 0.96 ±0.67 | 0.92 |
| Graduated & Supplementation for 90 days (n= 154) | 6.92 ± 1.28 | 0.33 ±.32 | 0.66 ±0.43 | 0.97 ±0.52 | 0.97 |

**Discussion**

The finding of this study suggests that food supplementation even for as short as 30 days could be beneficial in some children with poor nutritional status, and proportion of graduation increase with time. However, momentum of weight gain does not depend on duration of supplementation and increasing duration of supplementation from 60 to 90 days have insignificant effect on weight gain. The graduated children did not relapse back within 90 days observation period.

The study subjects were randomized appropriately as indicated by similar socioeconomic status and anthropometrics measurements. The data collectors were not aware of the study objectives and methodology. However, the investigators had no

control over enrolment of children for supplementation and there may be some bias as has been shown that about 20.4 percent were not underweight and 32.5 percent not stunted at enrolment [9]. Although the household food intake and morbidity pattern of study children was not controlled during assessment of graduation there was no significant difference among them. Mean age at enrolment (13.0± 3.2 months) in this study is consistent with the national prevalence of under weight reported as 40% and 65% among 6-11 months and 12-23 months respectively[10].

Study exploring the optimum duration of supplementation for growth faltered children consistent with similar context is scarce. In Guatemala, recovery from wasting among supplemented children aged 6-24 months increased as duration increased (attributable benefit 0.18 at 3 months to 0.25 at 6 months and 0.37 at 12 months) but larger effects were obtained during the first three months[6]. There is evidence that responsiveness of children to supplementary feeding with a higher yield of energy is greatest during the first 2 years with little benefit thereafter[4].

Study from Jamaica among children aged 3-36 months with moderate to severe malnutrition has found that children receiving supplementation for 3 months gained significantly more in weight but the advantage was lost once supplementation was ceased [7]. In a study from Indonesia, supplementary feeding over 90 consecutive days on infants aged 6 – 20 months shows accelerated rate of weight gain and motor development but did not have noticeable effect on length[11]. It is suggested that the changes in growth rate after supplementation are more rapid and usually more marked for weight than for recumbent length [12].

The Tamil Nadu Integrated Nutrition Program has shown that greater impact on prevalence of malnutrition was associated with longer duration of supplementation.[13] However, a study among Bangladeshi urban slum children has revealed that during the first 3 months of intervention, the monthly weight gain of supplemented children was significantly higher than the controls, but the change was not significant in subsequent 3 months of supplementation [14].

The target group in the BINP program is 6 to 24 months old and about half of children enrolled for supplementation had gain weight as per desired level of graduation within 60 days. Once graduated children could maintain weight gain with usual family diet, there was no significant difference of mean weight gain between 60 and 90 days of supplementation. However, behavior change communication component of the BINP program could have a positive synergistic effect on growth through education and awareness of parents on child care and nutrition.

**Conclusion and recommendation:**

Although the effectiveness of a supplementation program is assessed through nutritional impact produced and duration is a determining factor, the cost involvement is also a major concern especially for low resource countries like Bangladesh. If children are looked for graduation after 60 days instead of existing practice of 90 days and supplementation are withheld in graduated children the cost of the program can be reduced substantially without compromising the beneficial effect of supplementation. Considering the effect on weight gain it is recommended to consider 60 days supplementation as optimum duration for growth faltered under 2 children in Bangladesh.

**Acknowledgement**

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# Seroprevalence of Hepatitis C Virus Infection Among Health Care Workers

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**Summary:**

***Background and aims:*** *Parenteral route is the principal mode of transmission of Hepatitis C Virus (HCV). Health care workers are at risk of infection with HCV. Aim of study was to estimate seroprevalence of HCV amongst health care workers and identify possible risk factors of HCV infection.* ***Materials and Methods:*** *355 health care workers were selected from July 2005 to June 2006 working in different departments of Bangabandhu Sheikh Mujib Medical University, Dhaka, Bangladesh. Among them 43.5% were doctors, 32.1% nurses, 11.8% ward boys, 5.9% operation theatre staffs (OT staffs), and 6.8% others. Sera were tested for HCV antibodies by ELISA. Data analyzed by SPSS 10.0 version.* ***Results:*** *Mean age was 31.56±7.4 years. Males were 51.4% and females 48.6%. Anti-HCV was positive in 5(1.4%) cases out of 355. Most prone to HCV infection were nurses (3) followed by doctor (2). No ward boy or OT staff was affected. Previous surgical (80%) and dental procedures (60%) were the main risk factors than recipients of blood transfusion (20%), intravenous drug users (20%), and multiple sexual exposures (20%).* ***Conclusions:*** *Nurses are more prone to HCV infection. Surgical procedures are the main risk factors for acquiring HCV infection. Proper sterilization of surgical instruments is recommended.*

*(J Bangladesh Coll Phys Surg 2007; 25 : 126-129)*

## Introduction:

Hepatitis C virus infection continues to be a major disease burden all over the world. In 1999, WHO estimated a worldwide prevalence of about 3% with HCV affecting 170 million people worldwide.[1] In Asia the figure is 0.3%, in China the figure ranges from 0.5%-0.8%. [2] However, there is considerable geographical variation in the incidence and prevalence of HCV infection. Much of the variability between regions can be explained by the frequency and extent to which the risk-factors involved, drug use accounting for 60-80%,[2-5] transfusion and transplants 5-13%,[6] unsafe injection, other health care related procedure 2-18%,[7] occupational exposure 0-7% and perinatal transmission 0-40%.[8-11]

Generally, most studies of prevalence use blood donors to report frequency of HCV usually by anti-HCV antibodies and do not report follow up HCV testing. Incidence of HCV seroconversion after accidental needle stick exposure is uncertain, with reports ranging from 0-10%.[12-15] Whether health care workers have a higher prevalence of hepatitis C virus infection through percutaneous occupational exposure than the general population is unclear.[2,16-21] This study was done to estimate seroprevalence of HCV infection amongst health care workers and identify possible risk factors of HCV infection.

## Materials and Methods:

In this study 355 consecutive healthcare workers of different departments of Bangabandhu Sheikh Mujib Medical University Hospital were included for detection of Anti-HCV in their sera. This study was done in the Department of Hepatology, Bangabandhu Sheikh Mujib Medical University, Dhaka, Bangladesh during the period from July 2005 to June 2006. The study populations were of different categories like doctors, staff nurses, ward boys, OT staffs and other categories of staffs (others). They were of either sex with ages ranging from 18 to 56 years.

Before the commencement of the study a well formed questionnaire was prepared consisting of employee's name, sex, date of birth, location of employment, marital status, history of jaundice, blood transfusion, intravenous drug uses, dental procedure, hospital admission, multiple sexual exposures, history of surgery and hepatitis B virus vaccination. For each

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case, after enrollment, 3 ml of blood was collected in a sterile test tube without anticoagulant from anticubital vein with all aseptic precaution. Each case was assured that secrecy will be maintained in case of positive result.

Sera were screened for antibody to HCV by ELISA-3. Reagent used was diasorin (Italy). Reactive samples were considered to be positive.

Previous blood transfusion (recipient), dental procedure, surgical procedure were catagorized as the major risk factors and multiple sexual exposures, intravenous drug abuse, alcohol abuse, diabetes mellitus & tuberculosis as the minor risk factors.

**Results:**

Of the 355 health care workers studied, males were 183(51.4%) and females were 172(48.6%). Amongst them 148 (43.5%) were doctors, 109 (32.1%) were staff nurses, 40 (11.8%) were ward boys, 22 (5.9%) were OT staffs and 23 (6.8%) were others; 64.6% were married and 35.4% were unmarried (Table-1). Among the major risk factors- 46.6% had history of dental procedure, 31.9% had history of surgery, 6.8% had history of blood transfusion (Table-1) and 26.6% had history of jaundice. Among the minor risk factors-7.3% had multiple sexual exposures, 4% had I/V drug abuse, 6.2% had diabetes mellitus, 2.5% had history of tuberculosis and 1.7% had history of alcohol intake. Of the 355 health care workers, 159 (44.9%) were vaccinated against hepatitis B virus.

Of the 355 cases, five (1.4%) were anti-HCV positive. Among Anti-HCV positive cases, three were staff nurses (60%) and two were doctors (40%) {Fig.-1}. Of the Anti-HCV positive cases, three were females and two were males; four were married, four had past history of jaundice, four had previous

**Table-I**

*Demographic profile of study population (n=355).*

| Variables | Values | | |
|---|---|---|---|
| Age (Years) | 31.56 ± 7.41 (Mean ± SD) | | |
| Sex | Males | Females | |
| | 51.4% | 48.6% | |
| Job Status | Doctors | Nurses | Others |
| | 43.5% | 32.1% | 24.4% |
| Marital Status | Married | Unmarried | |
| | 64.6% | 35.4% | |
| H/O Jaundice | Present | Absent | |
| | 26.6% | 73.4% | |
| H/O Hospitalization | Present | Absent | |
| | 39.8% | 60.2% | |
| H/O Surgery | Present | Absent | |
| | 32.2% | 67.8% | |
| H/O Blood Transfusion | Present | Absent | |
| | 6.8% | 93.2% | |
| H/O Dental Procedure | Present | Absent | |
| | 46.6% | 53.4% | |
| H/O Multiple Sexual Exposure | Present | Absent | |
| | 7.3% | 92.7% | |
| H/O I/V Drug Abuse | Present | Absent | |
| | 4.0% | 96.0% | |

**Table-II**

*Demographic profile of Anti-HCV positive patients (n=5).*

| Variables | Values | |
|---|---|---|
| Age (Years) | 31.67± 4.51 (Mean ± SD) | |
| Sex | Males | Females |
| | 2 | 3 |
| Job Status | Doctors | Nurses |
| | 2 | 3 |
| Marital Status | Married | Unmarried |
| | 4 | 1 |
| H/O Jaundice | Present | Absent |
| | 4 | 1 |
| H/O Hospitalization | Present | Absent |
| | 3 | 2 |
| H/O Surgery | Present | Absent |
| | 4 | 1 |
| H/O Blood Transfusion | Present | Absent |
| | 1 | 4 |
| H/O Dental Procedure | Present | Absent |
| | 3 | 2 |
| H/O Multiple Sexual Exposure | Present | Absent |
| | 1 | 4 |
| H/O I/V Drug Abuse | Present | Absent |
| | 1 | 4 |

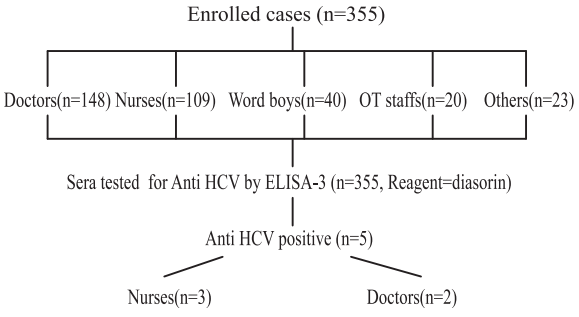


**Fig-I:** *Study design and outcome*

surgery, one had history of previous blood transfusion, three had previous dental procedure (Table-2), two had family history of jaundice, one had history of multiple sexual exposures and one had history of intravenous drug abuse. No OT staff or ward boy was affected. Mean age was 31.67±4.51(Mean ± SD) years. None of them had history of diabetes mellitus or tuberculosis.

In the present study, the prevalence of hepatitis C virus infection among the health care workers is 1.44%. In present study, history of surgery (80%) and history of dental procedures (60%) were two main risk factors to be anti HCV positive among the health workers. History of blood transfusion (20%), history of multiple sexual exposures (20%) and I/V drug abuse (20%) were the minimum risk factors.

**Discussion:**

Hepatitis C virus infection is the leading issue of concern in health care workers in Bangladesh and abroad. HCV is predominant cause of chronic hepatitis and cirrhosis worldwide and an important factor in the development of hepatocellular carcinoma. HCV infection appears to be endemic in most parts of the world with estimated overall prevalence of 3%, representing approximately 170 million of HCV affected person world wide.[1]

Dominant mode of transmission is blood to blood contact, the quoted transmission rate is 0-10% (average 1.8%) in the situation where health workers sustain a sharp injury from an affected patient[22,23]. While this is less than the comparable figure for hepatitis B virus (HBV) which has a transmission rate of 25-35%, it should be remembered that health care workers can be vaccinated against HBV but not HCV.

In several investigations on the possibility that HCV was a major occupational risk to dentists, it was concluded that nosocomial transmission of HCV in dentistry is possible but relatively unlikely.[23-26] Health workers who perform exposure prone procedures where injury to the workers may result in exposure of the patients open tissue to the blood of the workers are theoretically of increased risk of infection with blood borne viruses. According to UK health department guidelines, these occupations include surgeons, interventional physicians and intensive care unit and accident and emergency staffs.

A previous study shows seropositivity of 0% among voluntary blood donors in Dhaka; [27] this may reflect the average prevalence among general population. The current study shows seroprevalence of 1.44% among health care workers, which is higher than the general population. The high prevalence of HCV among health care workers may be due to their exposure to infected blood/blood products of patients with HCV infection. The exposure may in the form of surgical or dental procedures rather than the other routes like accidental needle- pricks, contact of cut skin surface with blood/blood products.

**Conclusion:**

Priority should be given to the primary prophylaxis against hepatitis C infection as there is no pre & post exposure vaccine against it. Different strategies are required to interrupt different patterns of HCV transmission. This study on randomly selected health care workers has helped in identifying predominant mode of transmission of the virus as being surgical/dental procedures in the studied population but require further study in this regard. Proper sterilization of surgical/dental instruments should be the main strategy to interrupt the transmission of hepatitis C virus infection.

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# The Outcome of Delayed One Stage Urethroplasty in Post-Traumatic Prostato-Membranous Urethral Stricture : A Retrospective Study of 50 Cases

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**Summary:**

***We have reviewed the result of delayed one stage urethroplasty through perineal approach in post traumatic prostato-membranous urethral stricture with associated pelvic fracture in a retrospective study. The place of study is Bangladesh Medical College Dhaka. The study period was from Nov'01 to Aug'04. The sample size was 50. Thirty two cases presented with post-traumatic stricture of urethra ( PTSU ) with suprapubic cystostomy (SPC). The rest 18 cases were complicated and presented with recurrent stricture with associated periurethral abscess, suprapubic and perineal urethral fistula or thigh urinoma. These cases were managed initially by suprapubic cystostomy (SPC) and adequate local care before re-admission for definitive procedure.***

***All cases were underwent delayed one stage urethroplasty through perineal approach after > 3 months of SPC and were followed carefully for > 06 months in each case. The outcome in 42 cases were excellent; required no treatment. In 06 cases the result was good requiring 01 - 03 sessions of optical internal urethrotomy (OIU). Poor result was observed in 02 cases, managed by > 3 sessions of OIU followed by intermittent self dilatation (ISD). No patient needed re-operation. The overall result is excellent.***

***(J Bangladesh Coll Phys Surg 2007; 25 : 130-138)***

## Introduction :

Post traumatic stricture of urethra (PTSU) is one of the common problem with increasing in incidence. The association between pelvic fracture and disruption of prostato-membranous urethra following blunt trauma, as a result of high speed motor vehicle or pedestrian-motor vehicle accident is well described[1]. Management of traumatic prostato-membranous urethral disruption or its subsequent complication is one of the most controversial and technically difficult problem in Urology[2]. Morbidity in the form of urethral strictures, impotence, incontinence may remain a source of life long misery[3]. The area of contention in the management of traumatic posterior urethral injury is centered on the argument that, primary urethral alignment is superior than conventional early SPC + delayed urethroplasty ? Which procedure decrease the need for further intervention ? However it is universally accepted that suprapubic cystostomy alone the best initial management. Further management depends upon the available facilities and experience of the surgeon[3].

In general the short term success of immediate urethral realignment is excellent. But the long term result is not rewarding. It is currently accepted that it is a procedure of choice in cases of widely separated urethral ends or having associated bladder neck and rectal injuries. The major advantage of delayed urethral reconstruction is that it can be done under controlled conditions with better long – term result[4]. The main drawback of delayed urethral reconstruction is that the patient has to accept urethral stricture and suprapubic catheter for longer period[5].

## Patients and methods :

This retrospective study was done in Bangladesh Medical College Dhaka. The study period was

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from Oct'01 to Sept'04. The selection criteria was traumatic prostato-membranous disruption of urethra with associated pelvic fracture and the length of stricture segment is < 2.5 cm. The sample size was 50. Thirty two patients presented with PTSU with SPC. The rest 18 patients were complicated by previous intervention. We managed them with SPC first with adequate local care and was readmitted > 03 months later. All cases were re-evaluated with histoty, physical examination and investigations ( to ensure operative fitness, normal renal function, sterile urine ). Primary assessment of urinary tract was done by ultrasound scan and contrast imaging technique ( Figure : 1, 2, 3. ) ; simultaneous combined antigrade cystourethrogram and retrograde urethrogram (RGU). All patients were consulted routinely with orthopedic surgeon. Adequate movements of the lower limbs were assessed in all cases to ensure proper positioning during operation.

Operative technique: After spinal anaesthesia patients were placed in lithotomy position. The extent of stricture segment was assessed finally with the help of antigrade suprapubic and retrograde urethral dilators before the inverted U / Y incision was made in the perineum. With meticulous and clean dissection the immediate distal healthy urethra was exposed first. Then the total length of the stricture segment along with surrounding fibrous tissue was excised. The distal bulber urethra and the proximal prostatic urethra was made pliable for tension free, spatulated, water tight anastomosis around a 14 Fr. foly's catheter by interrupted 08 - 10 stitches using 5'0' vicryl suture. Mobilization of distal corpus spongiosum is sufficient in most of the cases to get well alignment of urethral ends. To further assist urethral alignment we needed to separate the corporal bodies in the midline with diathermy under the symphysis pubis in 10 cases. We didn't need urethral re-rooting or inferior pubectomy in any of our cases. Absolute haemostasis was ensured and the wound was drained routinely before closure in layers.

**Result :**

The mechanism of injury ( Table : I ) in pelvic fracture and prostatomembranous urethral disruption was either blunt trauma to the lower abdomen or compression injury to the pelvis. In 76 % ( n – 38 ) of cases it was due to motor vehicle accident, in 12% ( n – 06 ) of cases it was the result of pedestrian motor vehicle accident and in 8% ( n - 4 ) of cases it results from crush injury and in 4% ( n – 2) cases it results from accidental fall from height. Thirty percent ( n – 15 ) of the patients were under went emergency laparotomy. Of them bowel repair with proximal enterostomy was done in 06 cases. Four cases were referred to us after closure of enterostomy. In rest 02 cases, enterostomy was closed after successful urethroplasty. Forty percent ( n – 20 ) cases needed emergency orthopaedic management for fracture of pelvis and limbs. All cases of pelvic fracture were managed by conservative treatment. Of them 76% ( n – 38 ) cases have high risk pelvic fracture ( Table : II ). The minimum age of the victim was 12, maximum 70 and average 32 years. The minimum period of time of accident and presentation to us was as low as 03 days and as high as 26 years ( Table : III ). Thirty two ( 64% ) cases presented with SPC. The rest of the cases were complicated following intervention ( Table : IV ), presented with suprapubic leakage following failed delayed urethroplasty ( 8%, n – 4 ), dense recurrent urethral stricture with thigh urinoma ( 6%, n – 3 ), proximal periurethral absess and fistula ( 22%, n – 11 ). These eighteen complicated patients were managed with SPC first and adequate local care (drainage of perineal absess, thigh urinoma and regular dressing of the wound.). Ten cases in our series developed secondary bladder stone. Of them 2 cases were under went suprapubic cystolithotomy two times and 8 cases for one time.

**Table-I**

*Nature of violence resulting urethral injury and pelvic fracture (n=50)*

| Nature of violence | Number |
|---|---|
| Motor vehicle accident | 38 |
| Pedestrian motor vehicle accident | 06 |
| Crush injury | 04 |
| Accidental fall from height | 02 |

**Table-II**

*Various types of associated pelvic fracture ( n - 50 )*

| | | Number |
|---|---|---|
| Low risk pelvic fracture | Single ramus ( pubic ) fracture | 12 |
| High risk | Diastasis (pubic symphisis) + multiple rami fracture pelvic fracture | 30 |
| | Diastasis (pubic symphisis ) + vertical displacement + multiple rami fracture | 08 |

**Table -III**

*Age, duration between the onset of injury and presentation( n - 50)*

| | Age of patient | Number | Time of injury and presentation | Number |
|---|---|---|---|---|
| Min. | 12 years | 04 | 03 days – 3 year | 48 |
| Max. | 70 years | 01 | 22 - 26 years | 02 |
| Mean | 32 years | | 1.5 years | |

**Table -IV**

*History of past urological intervention (n – 50)*

| | | Number |
|---|---|---|
| SPC | | 32 |
| Primary urethral | Dilatation & catheterization | 03 |
| re-alignment | Rail road method | 09 |
| | Primary Repair | 02 |
| Failed delayed perineal urethroplasty | | 04 |

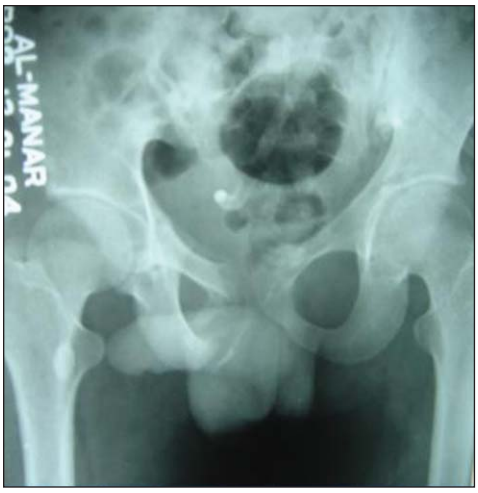


**Fig.-1:** *Control film : RGU + Antegrade cystourethrogram. (Healed mal-united pelvic fracture, suprapubic catheter in situ)*

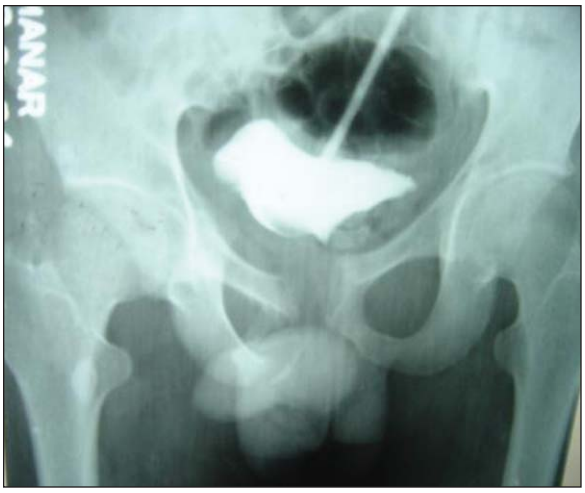


**Fig.-2:** *(same patient): Antegrade Cystourethrogram : Bladder is outlined, dye has not passed into the prostatic urethra due to bladder neck spasm.*

After urethroplasty we ensure adequate rest of bladder by continuous drainage of urine through suprapubic and penile catheter ( mainly to stent the anastomosis ) aided with oral uro-selective anti-cholinergic drug. In complicated and doubtful cases (n-12) peri-catheter retrograde urethrogram (Figure : 4) was done at the end of 3rd. week. Anastomotic leakage of contrast was seen in two cases ( Figure : 5) and the urethral catheter was continued for another 03 weeks ( Total : 3 + 3 weeks ). Repeat peri-cathter urethrogram appeared normal in both cases (Figure : 6). Urethral catheter was removed in 21st. POD in 48 cases, at the end of 6 week in 2 cases with clamping of suprapubic cather. Suprapubic catheter was removed after ensuring the normal flow of urine in all cases.

All patients were followed carefully ; monthly for three month and three monthly for one year, 6 monthly for one year then yearly afterwards. Minimum period of follow-up was 06 months[6] if there is evidence of excellent AUA symptom score. Post-operatively we assessed every patients with history using American Urological Association (AUA) symptom index[7] and clinical examination. We routinely observe the urine flow during every follow-up. In equivocal and doubtful cases we have done uroflowmetry ( n -15 ). All of our patients were continent( Table : V ). Forty two cases had excellent urine flow. Further intervention was required only in 08 cases ( 01 – 02 session of OIU in 06 cases, > 3 session of OIU + ISD in 02 cases ). We have taken history of penile erection and nocturnal tumescence in every cases before and after operation. Sixty eight percent ( n - 34) of our patient have excellent quality of erection, 32% ( n – 16 ) cases have sufficient penile turgidity. There was no significant change of quality of erection in our series following intervention.

**Table-V**

*Result of perineal anastomotic urethroplasty ( n – 50 )*

| | Number |
|---|---|
| Excellent ( require no treatment ) | 42 |
| Good ( require 01 – 02 session of OIU ) | 06 |
| Poor ( require > 3 session of OIU + ISD ) | 02 |
| Total failure | 00 |

**Table-VI**

*The overall Result of perineal urethroplasty ( n – 50 )*

| | | Number |
|---|---|---|
| Parameters | Post-operative State | 50 |
| Continence | Well preserved | 50 |
| Potency | Not influence by operation | 50 |
| Rec. stricture | Membranous / short | 08 |

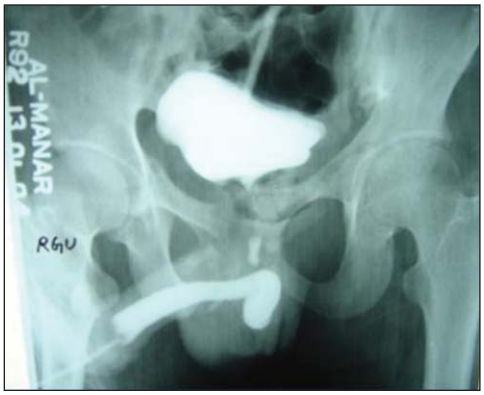


**Fig.-3:** *(same patient) : Combined ante-grade cysto-urethrogram and retrograde urethrogram : Bladder is outlined, prostatic urethra is partially visualized, anterior urethra is well outlined upto bulber urethra ; there is no communication between the anterior and posterior urethra.*

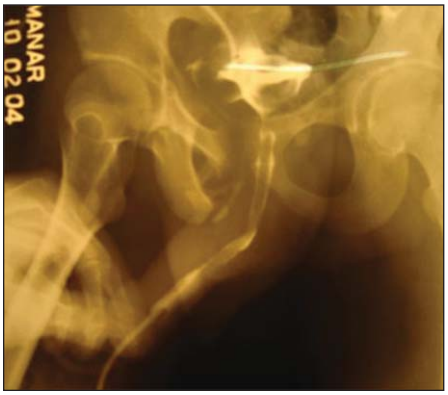


**Fig.-4:** *(same patient) : Post bulbo-prostatic perineal anastomotic urethroplasty. Retrograde peri-catheter urethrogram (21st. POD) : Dye has passed into the bladder and there is no anastomotic leakage. Urethral catheter could be removed with confidence.*

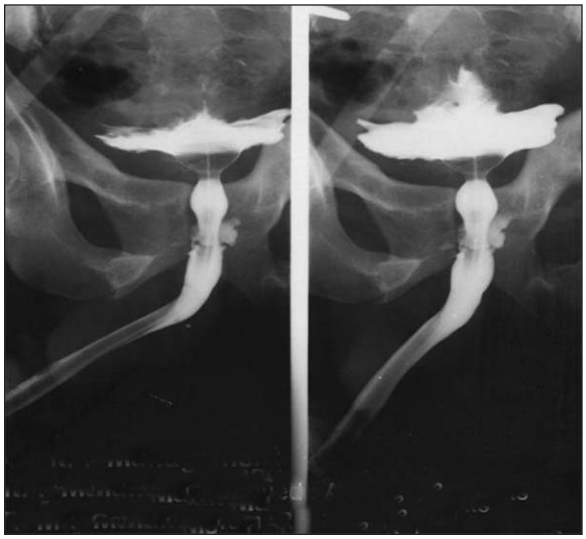

**Fig.-5:** *(Case - 2) : Post bulbo-prostatic perineal anastomotic urethroplasty. Retrograde peri-catheter urethrogram (21st. POD) : Dye has passed into the bladder and there is leakage in the anastomotic site. Urethral catheter was decided to continue for another 3 week.*

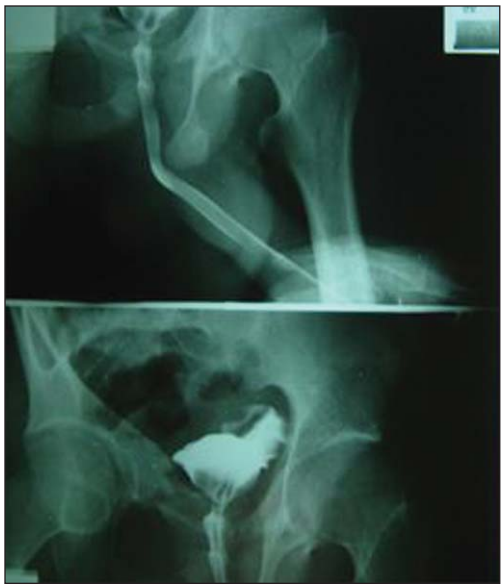

**Fig.-6:** *( Case - 2 : Post urethroplasty anastomotic leakage in 21st. POD. ) Repeat retrograde peri-catheter urethrogram ( 3 week later) : Dye has passed into the bladder and there is no leakage in the anastomotic site.*

**Discussion :**

Posterior urethral disruption usually occur after blunt rapid deceleration injury. Overall the risk of injury is greatly influenced by age and the types of pelvic fracture[8] Urethral injuries in association with pelvic fracture mostly due to indirect violence rather than direct trauma to the urethra by bony spicule[1]. High risk pelvic fracture and incidence of urethral injury is common in younger age group than in older patients[8]

As high as 3 to 25% of pelvic fracture and 10% - 20% of bladder injuries have associated posterior urethral injury[9]. Obviously pelvic fracture as they are intimately related to urethral injuries may be classified as[10] :-

No risk group :– isolated fractures of acetabulam, ilium and sacrum.

Low risk group :– single or ipsilateral pubic or ischiopubic rami fracture.

High risk group :– straddle and Malgaigne fractures. These types of fracture results sudden upward displacement of hemi-pelvis specially pubic rami results traction injury to the anterior and posterior urethral ligaments, sudden diastases of pubic symphysis also damage the posterior pubourethral ligament and urogenital diaphragm[1]. In our series most of the pelvic fracture ( n – 38 ) are of high risk group ( Table : II )

Initial evaluation of urethral injury was very difficult in most of the cases because of pain and co-morbidity. Patients who are unable to void with hematuria, blood at the urethral meatus, perineal hematoma or extravasation of urine with or without a high-riding prostate on digital rectal examination are signs that suggest a prostatomembranous urethral disruption[11]. In doubtful cases, retrograde urethrogram with gentle pressure using water soluble contrast will show urethral continuity with extravasation in case of partial urethral tear(34%) where as extravasation of contrast without urethral continuity is the feature of complete urethral disruption(66%)[12]. Emergency IVU may be done when severe injury is suspected. Cystogram phage of IVU may show extravasation of contrast in case bladder injury.

The extend of bladder elevation will give the idea of extent of urethral disruption or the volume of pelvic hematoma[12].

The aim of rational management of the posterior urethral disruption is to make the patient free of strictures, continent and potent[13]. Management of urethral injury and its complication has been evolved in the last decade. Suprapubic cystostomy alone is the best initial management. It is an effective temporary technique of urinary diversion that does not interfere with the management of associated life threatening conditions. SPC is safe, simple may be performed quickly under local anaesthesia even in haemodynamically unstable patient in emergency situation within limited setup[2]. In our study ( Table : IV ) majority of urethral injuries (n – 32, 64%) were managed by SPC in addition to laparotomy ( n – 15 ) for associated acute abdomen before referral to us. In rest of the case ( n – 18, 36% ) we did SPC first to manage the complications.

Primary urethral realignment using either railroad method, interlocking sound or endoscopic technique may be tried immediately or several days later after stabilization of associated injuries[14]. Though it is relatively easier technique, specially the endoscopic one but there may be chance of creation of false passage, introduction of infection, severe bleeding due to dislodgement of clot, pudendal nerve injury, difficulty in assessment of vitality of injured urethral ends[5]. In most patients who undergo primary realignment may develop stricture in the later period. In our study ( Table : IV) out of 18 failed cases 14 cases had history of primary urethral realignment. The subsequent morbidity of such recurrent strictures is directly related to different modalities of initial management. Urethroplasty may be easier in such cases because the strictures are usually short without lateral displacement of prostate[15]. Sometime it may also managed by optical internal urethrotomy with or without intermittent self urethral dilatation[16]. The need for multiple urethrotomies are proportional to the length of stricture segment ( > 2 cm.) or in presence of dense peri-urethral fibrosis. The success rate of urethroplasty after multiple urethrotomy is much less. So repeated OIU is only recommended for unfit and elderly patients having short life expectancy. In our series failure cases following primary urethral realignment further complicated by periurethal abcess, fistula and thigh urinoma so urethroplasty was much difficult in those particular cases.

Considering various limitations and high incidence of late complication it is currently accepted that there are special criteria[14] where immediate urethral realignment is a procedure of choice without primary suture[15].

Primary urethral realignment may be advantageous in cases of widely separated urethral ends or having associated bladder neck and rectal injuries. Widely separated urethral ends are associated with disruption of all facial attachment and is likely to be disorganized even after dissolution of haematoma[17]. The accurate diagnosis of bladder neck injury and its primary anatomical repair is very important for the preservation of continence because external sphincter is usually damaged following injury or urethroplasty. Recognition of associated rectal injury and its repair will prevent the devastating events[14].

Delayed recostruction is advocated 2 – 6 months after initial suprapubic cystostomy. By this time the inflammation subsides and the scar matures[18]. The major advantage of delayed urethral reconstruction after prostatomembranous disruption is that it can be done under controlled conditions when the patient has recovered from major associated injuries[4]. The main drawback of delayed urethral reconstruction is that the patient has to accept either incomplete or in most cases complete obliteration of urethra and also has to carry and care suprapubic catheter for longer period of time[1].

Delayed endoscopic management of thin diaphragmatous stricture or those with a narrow lumen, is easier with insignificant morbidity. However, endoscopic maneuvers for complete urethral obliteration remains controversial. As much as 61% of impassable strictures could be managed endoscopically. The immediate success

rate may vary from 54% to 100% in different series. But the long-term recurrence rate of available series are not encouraging (62% to 89% )[19]. Despite initial enthusiasm for the newer technology, laser urethrotomy appears to offer no advantage over conventional urethrotomy[5].

Delayed open urethral reconstruction may be done in single session or in two session. The most common two-stage repairs have been described by Johanson, Somervil et al, Schreiter and Noll. Two stage urethroplasty currently reserved in case of severe urethral damage, extensive urethral stricture or often after failed 1- stage repairs. The interval between two stages would allow for healing of the usually infected and damaged tissue before final reconstruction of the urethra[20].

The different route of approach in single stage urethroplasty includes perineal, transpubic or combined perineal and suprabic with or without free graft[21]. The success rate of delayed one stage urethroplasty through perineal approach in experienced hand is highly encouraging. It is more then 95%[18]. Much superior result (97%) was claimed in other study also[22]. We did delayed one stage urethroplasty through perineal approach in all cases with overall excellent result (Table : VI).

The assessment of exact site and extend is very important for operative planning. Conventional retrograde urethrogram is helpful to delineate the distal urethral margin. Antigrade cystourethrogram or MCU may fail to outline the prostatic urethra because of bladder neck spasm. The role of Color Doppler[23] and Sonourethrography[24] is useful for the assessment of anterior urethra strictures and local tissue vascularity better then conventional RGU. The narrow area of prostatomembranous stricture region is difficult to image accurately. Pelvic MRI though very expensive but is excellent in this regard for precise evaluation of distance and direction of disrupted prostatomembranous urethra[22]. Thus preoperative MRI has main clinical impact to determine the route of better approach, to justify the planning of total or inferior pubectomy in difficult situation. We did conventional RGU and antigrade cystourethrogram in all cases. We finally assess the stricture site under anaesthesia before incision using antigrade suprapubic and retrograde urethral dilators.

The American Urological Association (AUA) symptom index was introduced in 1992 and has gained worldwide acceptance for the assessment of symptomatic BPH and its treatment outcome[7]. The role of AUA symptom index for assessment of therapeutic response after urethroplasty is well established. If the symptom scores were failed to diminished after urethroplasty subsequent investigations will show the presence or recurrence of stricture. Following successful re-operation it was seen to decrease the symptom score in the same patient[25]. We have also used AUA symptom index during history taking for the assessment of post operative outcome in all cases when attended in follow-up.

The result of different modalities of managements in post traumatic prostato-membranous urethral disruption published in different journal from 1968 to 1990 were analyzed in a review study[26]. It was observed that some of the patients among immediate suprapubic cystostomy with delayed urethroplasty group ( total : n- 199 ) may develop normal flow of urine during the waiting period of operation, leading to conclusion that some of the cases of partial urethral rupture may heal completely without residual disability ( n – 9, 5.25%). No recurrence of stricture was seen in rest of the patient underwent delayed urethroplasty. Among the early urethral realignment group ( n- 304 ), 53.9% cases developed stricture. The result of the study is compatible to another review study of 237 patients of 5 different series[12]. In our series we did not find to establish the normal flow of urine in any of our patients while waiting for delayed urethroplasty. But we have seen to pass insignificant volume of urine in some of our cases ( n – 05 ). Eighty four percent ( n – 42 ) of our patients did not need any intervention after urethroplasty. Recurrent urethral stricture was seen in only 16%( n – 8 ) cases managed by OIU + ISD ( Table : IV). The overall result in our study is excellent and no patient needed re-operation.

Impairment of male sexual function after disruption of prostatomembranous urethra with

associated pelvic fracture is well known and it varies from 2.6 to 50%[27]. There was lot of controversies regarding the nature of intervention influencing the incidence of impotence and the incontinence. Morehouse et al claimed higher incidence of impotence and incontinence among the patients treated with immediate and early realignment technique then initial SPC and delayed urethroplasty. Webstar et al[12] supported the observation of Morehouse et al and recommend to prefer initial SPC and delayed urethroplasty over immediate or early urethral realignment unless there is specific indication.

The study of Morehouse et al was thoroughly analyzed and was challenged later in many other studies[14,26,27]. It was observed that Morehouse et al. dealt a group of patients, referred to them after failed attempt of immediate realignment[28]. Higher incidence of impotence and incontinence was observed among referred group and was attributed to the result of initial operative technique. Morehouse et al compared those group of referred patients with their own population of patients to whom elective initial SPC and delayed urethroplasty was done. Patterson et al[29] in their series had shown the excellent preservation of potency in early realignment group; even equal or better preservation of potency was claimed in some study[14]. Where as Zincke H et al reported poor preservation of potency in delayed urethroplasty group[30].

During delayed urethroplasty identification of neurovascular bundle may be impossible or at least very difficult because it lies more lateral or already entered in the corporal bodies. On the other hand it is difficult to explain the neurovascular injury during early urethral realignment by careful manipulation of only the trauma site, situated more anteromedial to the neurovascular bundle. Even then this maneuver is much specific by early endoscopic realignment technique[14].

The nervi erigentes; situated dorsolateral to the prostato-membranous urethra is more likely to get injured following associated pelvic fracture[14]. King et al reported 42% incidence of loss of potency if urethral rupture is associated with pelvic fracture and is only 05% when it is not associated with pelvic fracture. This study was supported by the study of Gibson et al[27]. There are also reported observation of regaining of potency following delayed urethroplasty[27]. It is also difficult to explain how delayed urethroplasty helps recovery of potency. Considering the current available literature it could be inferred that the outcome of management of post-traumatic prostate-membranous disruption in relation to potency and continence depends upon the result of initial injury to the neurovascular bundle supplying the corporal bodies and injury to the sphincter ( internal, or both external and internal ) rather then operative technique; provided it is done in good centre by expert hand[14]. In the present series so far preoperative and postoperative history concern operative technique did not influence the potency in any of our cases. All patients were continent postoperatively with intact internal urethral sphincter.

**Conclusion :**

Post traumatic prostatomembranous urethral disruption results from distraction force affecting the weakly supported membranous urethra may be associated with pelvic fracture. Every case is to be judged individually to select best options of available modalities. Suprapubic cystostomy alone is the best initial management. Further management depends upon the expertise of the surgeon and available facilities. Primary urethral realignment should be done only in selective group of patients. Two stage urethroplasty is currently reserved in case of severe urethral damage, extensive urethral stricture or often after failed 1- stage repairs. The best long-term result could be obtained by delayed one stage urethroplasty through perineal approach as seen in the present study.

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# Concepts in Rehabilitation of Burn Patients

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**Summary:**

***Burn is not the skin loss only. Functional and psychological outcome must be assessed and treated accordingly. Prevalence of burn injury is very high in Bangladesh though exact statistics is not available. Houses of poor people especially in the slum area, which are made of combustible materials, are vulnerable to set off fire. Chemical burns like throwing acids by culprits are common in our country. Hospitals are treating these patients without proper medical and psychological rehabilitation. This article will review the rehabilitation concepts of burn injury and provide some information to those physicians and surgeons who treat burn injury.***

***(J Bangladesh Coll Phys Surg 2007; 25 : 139-143)***

**Introduction:**

The human and economic cost of burn injury is enormous. Burn size and age are cardinal determinants of survival. Mortality is highest in the very young and elderly.[1.] Mortality is greater in female than male with comparable injury [2.]

Burn injury is not a new problem. Mankind has struggled with burn injuries since the discovery of fire. Documents as early as Papyrus Erbes in 1600 BC specify burn treatment techniques.[3]. Hippocrates stressed burn wounds with wine or water to avoid suppuration. With the advent of graft technique early closure of burn wound become possible significantly improving survival rates.

Modern burn treatment is the result of a long history of medical and technical advances.

The survival rates of seriously burned patient have increased dramatically, especially in the last three decade. More patients are returning to active lives at home, at work and in the community. The physiatrist is uniquely trained to manage the complex rehabilitation problem of burn injuries and to aid burn patients in returning to a full and productive life.

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**Rehabilitation:**

Once a patient has sustained a burn injury, the rehabilitation phase begins. It continues long after discharge. The goal of rehabilitation is to achieve the optimal level of functioning. The location, depth and distribution of burn injury are important in preventing complications. The extent of injury correlates with survival as well as with time required to return to independent functioning. Age, previous level of independence, premorbid medical condition and other injuries must be considered when assessing the burn patients. [4,]

During acute period goals include promoting wound healing and preventing complications by preserving joint function, strength, endurance and functional abilities. Goals are individualized according to the location and extent of burn injury and previous functional level. Goals are continuously assessed and modified as the patient improves.

**Positioning:**

Proper positioning is fundamental to burn rehabilitation. Positioning prevents contracture formation, controls edema and maintains tissues in an elongated state. Because of pain, burn patients assume primarily flexed and adducted position that inadvertently favors contracture development. The positioning program must be individualized in accordance with the sites of injury. Use of splints, pillows and foam wedges, can achieve proper positioning.

**Splinting:**

Splints are used to maintain anticontracture position and range of motion in joints at risk for development

of contracture. Prefabricated or custom splints can be used but proper fitting must be ensured. Splints should be easy to don and doff. The type of splint used is dependent on the area burned, the depth of injury, the patient's functional status and the patient's ability to participate in positioning and exercise program. The functional position of hand is the hand splinted in full interphalangeal extension, 60 to 80 degrees of metacarpophalangeal flexion, thumb abduction and wrist extension.[5, 6.]

**Exercise in burn rehabilitation**

Exercise is fundamental to maximizing patient function and overall outcome. In prescribing exercise, the extent, depth and location of the injury is considered. The risk of exercise disrupting wound healing requires regular wound inspection. Stretching can also disrupt already tenuous joint. The initial exercise program should focus on preserving ROM and maintain strength. Active forms of exercise are indicated for patients who are alert and able to participate. For critically ill patients, the slow controlled movement of passive ROM exercise is appropriate. Massage is a useful adjunct in burn treatment and rehabilitation.

**Ambulation and mobility**

Ambulation and mobility are important elements of a comprehensive rehabilitation program. Early ambulation maintains balance and decreases the risk of deep vein thrombosis. Ambulation should start as soon as possible after admission. Ambulation can be limited by medical status, the depth and extent of lower extremity burns and previous medical conditions such as peripheral vascular disease. Elastic wrap or stockings prevent venous stasis, control edema, and reduces the risk of local trauma and decrease pain induced by dependent position.

**Scar Rehabilitation:**

The risk of hypertrophic scar increases with the depth of injury and length of time required for healing. The risk is reported by some authors to be greater in more darkly pigmented areas.[7,8.] Pressure treated scars have better functional and cosmetic outcome. The application of continuous pressure through garments, orthoses and splints is the primary non-surgical modality used to control hypertrophic scarring. The mechanism by which pressure suppresses hypertrophic scarring is unclear, but it has been hypothesized that pressure causes decreased capillary perfusion and decreased tissue oxygenation, resulting in reduced cellular activity and collagen synthesis. [9.] The application of pressure should continue until the scar is mature. Patient education is essential in increasing pressure therapy compliance. Wounds expected to heal in less than three weeks often do not require early surgery and are less likely to develop significant scarring. [10,11]. Custom made elastic facemasks and transparent orthoses are available for controlling facial scarring. Silicone and other materials can be added to face masks to achieve better pressure application in regions such as nasolabial folds, [12.] Microstomia correction appliances may be dynamic or static. [13,14.]

The use of pressure garments on grafted burns or burns that take longer than 14 days to heal is considered standard care in most burn care centers, despite the question of its efficacy.[15, 16.] Topical application of silicone has also been employed to treat burn-related scarring. Much is known about the effect of topical silicone application, but to date, as with pressure garments, no randomized, controlled trials have conclusively demonstrated that such therapy minimizes hypertrophic scarring.[17, 18.] Available tools to modify the progression of hypertrophic scar formation are limited in number and effectiveness. These tools include scar massage, compression garments, topical silicone, steroid injections, and surgery. In some contractures over major joints, serial casting may be useful.[15]

**Hands rehabilitation**

Hands are the most common sites of burn injury [19]. Because of the highly specialized functions of the hand, the burned hand requires the care of burn specialists experienced in hand management. Treatment goals include edema control, early wound closure, rapid return of hand function and prevention of hand deformities. After assessing the hand for potential hypertrophic scarring and contracture formation, a well-designed program of exercise, and splinting should be prescribed. Exercise coupled with splint, when indicated can prevent hand deformities and restore hand function. When splints are used, they should maintain the hand in an anticontracture position, which will prevent

anticipated deformity. This injury requires the hand be splinted in the position of function. Once the wound is covered, passive ROM can be performed judiciously.

**Neuromuscular complications:**

Peripheral nerve injuries are common after burns. Neurological involvement includes focal nerve compression, multiple mononeuropathies and generalized polyneuropathies. Focal mononeuropathies commonly occur secondary to positioning, improperly applied splints or bulky dressings [17]. Burn injuries are intrinsically associated with generalized peripheral neuropathy. Approximately 15 % of the patients have the peripheral neuropathy generally occur in patients who have 20% total burn surface area. The etiology of peripheral neuropathy of burns has not been established, but neurotoxicity from antibiotics and the possibility of a circulating neurotoxin from the burn injury itself have been hypothesized.[20, 21.]

**Pediatric burns:**

Mortality rates are higher in infant compared with those in adolescence or adults. Children younger than 1 year are at greater risk for mortality than during subsequent school years.[2]. Children are often unable to cooperate with many aspect of therapy and do not understand long-term goals. Loss of function such as hand dexterity and ROM not only interferes with child's current development level but also can limit future academic and vocational success. Because of the growth, children require more frequent modification of splint and custom fitted pressure garments. It is important to monitor jaw development and dental alignment during the use of facemasks and orthoses to avoid malocclusion [22,23,24.] The combination of oxandrolone and exercise offers greater benefits in lean body mass, body weight, and physiologic parameters compared with oxandrolone alone, exercise plus placebo, and placebo alone in severely burned children.[25]

**Geriatric burns**

The geriatric patients can be at increased risk for burn injury because of premorbid mobility limitations, visual deficits, impaired sensation and cognitive problems. Knowledge about the patient's functional level prior to the burn injury is important in planning the rehabilitation program. Early mobilization is imperative because the effects of immobility occur more rapidly and more pronounced in elderly persons [26]. Well-designed exercise programs can assist geriatric patients in making significant gains in strength and endurance.

**Psychological adjustment**

A burn injury can be the most devastating traumatic event a person can experience. Preinjury psychological status is strong predictors of a patient's long-term emotional status after serious burn.[27] Patients who are previously well adjusted emotionally are likely to adjust the insult. Previous psychological dysfunction is likely to be accentuated by burn trauma. [28,29]. Delirium, adjustment disorders, major depression, and posttraumatic stress disorder are most common psychiatric disorders during recovery from burn injury[30]. Adjustment disorder is the second most frequently encountered disorder. It can persist longer in the presence of chronic conditions, such as disabling medical conditions or financial difficulties as a result of unemployment.[31,32.] Depression and posttraumatic stress disorders are major complication of burn rehabilitation.

**Work issues**

Returning to work is an issue of major importance to many burns patients. The presence of hand burns, the type of employment, and age significantly influence return to work. [33,34]. Special problems affecting return to work include skin fragility, heat and cold intolerance, altered sensation and impaired coordination and dexterity. Burn injuries often occur at work and affected individuals may have difficulty returning to the site of their injury. In the case of severe injury approximately 20 to 50% of the patients require change in occupation.

**Outpatient rehabilitation:**

Planning for discharge should begin as early as possible. Education of the patient and the family builds a foundation for all aspect of rehabilitation, including outpatient treatment. At discharge the patient need to be independent in all aspect of care in order to return home. The need for physical and

occupational therapy does not end at discharge. Ideally the patient should be referred to a therapist with previous experience in treating burn injuries. Therapy program need to focus on ROM, strengthening, endurance, mobility and gait. After discharge the patient faces the task of family and community reintegration. Self-esteem can be significantly altered by changes in the appearances and functional abilities. With post burn injury status; women and girls have lower self-esteem than men and boys who experiences comparable injuries. [35].

Social studies indicate that the greater the patient's social support from family and friends, the more positive is the body image and higher the sense of self esteem. Symptoms of depression occur less frequently in this group of patients [36,37]. Social support appears to be a key factor in a person's psychological adaptation to a burn injury. [37].

**Conclusion**

Burn is not the skin loss only. Functional and psychological outcome must be assessed and treated accordingly. Rehabilitation should begin at the time of admission. Serious burn injury results in multi system trauma and has implications far beyond skin loss. Collaboration between the physiatrist and burn surgeon managing the patient's acute medical and surgical care should begin immediately on the patient's admission to the burn unit. Integrating the expertise of the burn surgeon and physiatrist will ensure that all aspect of patient care are addressed and the patient will receive burn care services needed to achieve an optimum functional and cosmetic outcome. Success ultimately depends on the combined effort of the patient and the rehabilitation team.

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# Non Alcoholic Fatty Liver Disease- Is It Always Benign?

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**Summary:**

***Nonalcoholic fatty liver disease (NAFLD) is the most common liver disease observed in the clinical practice of hepatology affecting approximately 20% of the general population. It is increasingly apparent that non alcoholic steatohepatits (NASH) and NAFLD are not Western disease. There is evolution of Western-style life among the Asian population and NASH has increasingly been diagnosed in several regions in Asia. NASH is considered as a type of a larger spectrum of NAFLD that is a consequence of insulin resistance and other underlying factors with histological findings ranging from fatty change alone to fat plus inflammation, to fat plus ballooning degeneration, and to fat plus alcoholic hepatitis-like lesions including Mallory body and fibrosis, the latter two categories being considered as NASH. Although liver biopsy is currently the gold standard for diagnosis, there is a need for less invasive methods. Imaging by ultrasound, computerized tomography and magnetic resonance are all able to demonstrate fat. Ultrasound, although probably not the most reliable imaging method, has many advantages and, when positive, gives a high degree of certainty of the diagnosis depending on the prevalence of fatty liver in the population being studied. Unlike liver biopsy, none of these techniques is able to differentiate simple steatosis from non- alcoholic steatohepatitis. The ultimate goal of treating the patient with NASH is to prolong life by avoiding the end-organ diseases associated with insulin resistance and the metabolic syndrome. Treatment of patients with nonalcoholic fatty liver has typically been focused on the management of associated conditions as well as discontinuation of potentially hepatotoxic drugs. Weight loss and exercise improve insulin sensitivity. Bariatric surgery may improve liver histology in patients with morbid obesity. Insulin sensitising drugs showed promise in pilot trials as have a number of hepatoprotective agents. Further randomised, well controlled trials are required to determine the efficacy of these drugs.***

***In this article, we will review (1) various processes that are involved in the pathogenesis of NASH (2) the existing medical therapy for patients with nonalcoholic fatty liver, (2) the emerging and potentially useful medications.***

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## Introduction:

Nonalcoholic steatohepatitis (NASH) represents a part of a wide spectrum of non-alcoholic fatty liver disease (NAFLD), which ranges from simple steatosis and steatohepatitis to advanced fibrosis and cirrhosis.[1] Nonalcoholic fatty liver disease (NAFLD) is the most common liver disease observed in the clinical practice of hepatology.[2] NASH today is third most common cause of chronic liver disease in North America after alcoholics liver disease and hepatitis and the most common cause of raised transaminases more than six-months. Primary NAFLD is related to insulin resistance and thus frequently occurs as part of the metabolic changes that accompany obesity, diabetes and hyperlipidaemia. However, it is important to exclude secondary causes of hepatic steatosis by clinical assessment. Treatment of these conditions differs and revolves around correcting the underlying cause.[3]

## Methodology:

PubMed was searched in June 2006 for all English-language publications including the search terms "Non alcoholic fatty liver disease", "Non alcoholic steatohepatitis" from 1980 onwards. The full articles for selected identified records that were thought to be

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potentially relevant were obtained. The references in these selected articles were also checked for additional information that might be relevant.

Definition: Non-alcoholic fatty liver disease (NAFLD) represents a spectrum of liver diseases characterized mainly by macrovesicular steatosis that occurs in the absence of alcohol consumption in amounts considered injurious to the liver.[4] The hepatic histology can vary from isolated hepatic steatosis alone to steatohepatitis and are referred to as non-alcoholic fatty liver (NAFLD) and non-alcoholic steatohepatitis (NASH) respectively. In addition to predominantly macrovesicular steatosis, the diagnosis of steatohepatitis also requires the additional presence of varying combinations of findings including cytologic ballooning, Mallory's hyaline, scattered inflammation and pericellular fibrosis.[4] NASH is may be defined as a syndrome characterized by the association of fatty liver and lobular hepatitis and chronically elevated ALT plasma levels in patients with negligible alcohol intake.[5] The syndrome is mainly associated with the metabolic syndrome which is characterized by a constellation of findings including obesity, diabetes, hypertension and hypertriglyceridemia obesity, diabetes and dyslipidaemia. [4, 6] But a few patients are lean, have normal fasting glucose and glucose tolerance, and show no evidence of increased plasma lipids. Patients with fatty liver and hepatitis are identified as having NASH.[6]

Epidemiology: The last two or three decades have seen the evolution of Western-style life of near complete inactivity, energy-dense food choices and liberal fiscal resources to obtain them and other means to avoid physical activity. Moreover, what is increasingly apparent is that NASH and NAFLD is not a Western disease and many population groups in the Asia-Pacific region are particularly prone to type 2 diabetes. For this reason, NASH has increasingly been diagnosed in several regions in Asia including Indonesia, Malaysia, Philippines, Thailand and India.[7]

True prevalence of NASH is difficult to assess without large-scale epidemiological studies. Steatosis is the most common cause of raised transaminases and affects nearly 10-24% of general population.[8, 9] In india, only 2-3% in the general population have steatosis.

The most important risk factors for NAFLD are obesity, diabetes, hyperlipidemia and female sex. Prevalence of obesity in non alcoholic steatohepatitis ranges from 30-100% in various series of patients. NAFLD may occur in up to 75% of type 2 diabetes, although obesity may be a confounding variable.[1,4,10]

**Causes of non-alcoholic fatty liver disease**

Primary:
Obesity, glucose intolerance, hypertriglyceridaemia, low HDL cholesterol, hypertension.

Secondary:

Nutritional
Protein-calorie malnutrition, rapid weight loss, gastrointestinal bypass surgery, total parental nutrition, short bowel syndrome, small bowel bacterial overgrowth, e.g. small bowel diverticulosis.

Drugs
Glucocorticoids, oestrogens, tamoxifen, amiodarone, methotrexete, diltiazem, zidovudine, valproate, aspirin, tetracycline, cocaine, protease inhibitors.

Metabolic
Lipodystrophy, hypopituitarism, dysbetalipoproteinaemia, Weber-Christian disease. Lipid loss versus lipid deposition syndromes,

Toxins
*Amanita phalloides* mushroom, phosphorus poisoning, petrochemicals, *bacillus cereus* toxin.

Infections
Human immunodeficiency virus, hepatitis C.

Others
Chronic inflammatory disorders, e.g. rheumatoid arthritis and systemic lupus erythematosus.

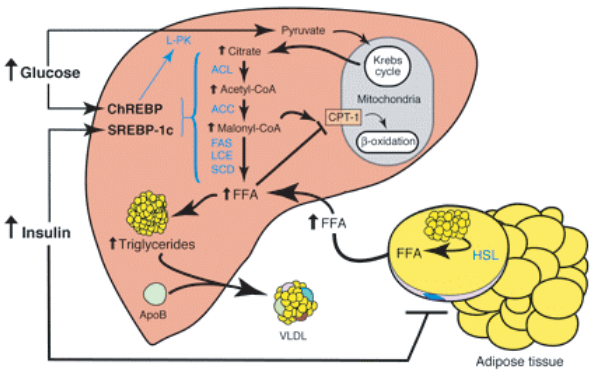
Pathogenesis (figure I):

The pathogenesis of NAFLD is not fully understood, however the finding that not all patients with steatosis develop hepatic inflammation and hepatocellular damage has led to the hypothesis that different pathogenic factors lead firstly to hepatic steatosis and secondly to hepatic damage ("the second hit").[10] Accumulation of hepatic fat is closely linked to

insulin resistance.[11] Insulin resistance is present in approximately 98% of individuals with NAFLD and over 80% of subjects with NAFLD meet minimal criteria for the metabolic syndrome.[4] Insulin resistance which increases lipolysis of peripheral adipose tissue with resultant increased fat influx into the liver in the form of free fatty acids. Insulin resistance also promotes de novo triglyceride synthesis within the liver and inhibits fatty acid oxidation thereby promoting triglyceride accumulation.[11] It is unknown what "second hit" leads to the development of liver damage, although several factors have been implicated including oxidative stress, mitochondrial abnormalities, and hormonal disturbances involving leptin and adiponectin.[12]

The recently discovered hormone resistin is linked to the development of insulin resistance, but direct evidence of resistin levels in humans with nonalcoholic fatty liver disease (NAFLD) is lacking. One study showed that NAFLD patients have increased circulating resistin and that increased levels are not correlated to either insulin resistance or BMI, but only to the histological severity of the disease.[13]

Figure I: Metabolic alterations resulting in hepatic triglyceride accumulation in insulin-resistant states.[11]




(Note: FFA: Free Fatty acid, HSL: hormone-sensitive lipase FAS: fatty acid synthase LCE: long-chain fatty acyl elongase, SCD: stearoyl-CoA desaturase, ACC: Acetyl-CoA carboxylase, Ch REBP: carbohydrate response element binding protein, SREBP-1c: Sterol regulatory element–binding protein-1c, ApoB: CPT-1: carnitine palmitoyl transferase-1).

Natural history

NAFLD exists as a histological spectrum of changes; simple steatosis refers to >5% hepatic steatosis in the absence of significant inflammation and hepatocellular damage whereas NASH demonstrates inflammation and hepatocellular damage and sometimes fibrosis.[14] NAFLD may be progressive resulting in cirrhosis that may be complicated by hepatocellular carcinoma and liver failure. Overall, about 5% of patients with NAFLD develop cirrhosis over an average of a seven year period with 1.7% dying from complications of liver cirrhosis.[15]

The high prevalence and chronic nature NAFLD subsequently translates to a significant health burden for the general community. In addition, subjects with a diagnosis of NAFLD have a higher risk of all cause mortality than the general population.[10] Many patients with nonalcoholic fatty liver disease have a relatively benign course, whereas in some others, the disease progresses to cirrhosis and its complications.[3] Histological features assist in stratifying patient risk of progressive liver disease.[10] Patients found to have pure steatosis on liver biopsy seem to have the best prognosis within the spectrum of nonalcoholic fatty liver disease, whereas features of steatohepatitis or more advanced fibrosis are associated with a worse prognosis.[3] Simple steatosis is comparatively benign with a 0%–4% risk of developing cirrhosis over a one to two decade period. In contrast, 5%–8% of patients with NASH may develop cirrhosis over approximately five years. Assessment of fibrosis stage is also valuable in prognosticating risk of developing liver related morbidity, with patients with advanced fibrosis (bridging fibrosis and cirrhosis) at most risk. Although these features aid in stratifying patients at risk, a significant proportion of patients will have all of these adverse prognostic markers but will not develop liver related morbidity or mortality. Thus accurate prediction of those patients who will benefit most from treatment is difficult.[10]

Clinical Features

Most patients with nonalcoholic fatty liver disease have no symptoms or signs of liver disease at the time of diagnosis.[3] Commonest symptoms are fatigue or malaise and a sensation of fullness or discomfort on the right side of the upper abdomen.[3, 4] Hepatomegaly is the only physical finding in most patients.[3] Other

features are pruritus, oedema, stigmata of chronic liver disease, acanthosis nigricans, obesity, diabetes, hypertension, dyslipidemia.[3, 4]

*Diagnosis of Nonalcoholic Fatty Liver Disease*
Presumptive Diagnosis:[21]

Elevated serum liver enzyme levels (AST, ALT, or -glutamyl-transferase) *or* imaging study with evidence of fat *and* minimal or no alcohol intake *and* negative test results for viral hepatitis, autoimmune disease (primary biliary cirrhosis), and congenital liver disease (e.g, Wilson disease). Definitive Diagnosis: Liver biopsy specimen with evidence of fat with or without inflammation or fibrosis *and* minimal or no alcohol intake.

The diagnosis of NASH can be done on the basis of the following criteria: [19]

(1) Intake of less than 20 g of ethanol per day,

(2) Biopsy proven steatohepatitis; steatosis, inflammatory infiltrates, and ballooning degeneration with or without Mallory bodies or pericellular/ perivenular fibrosis,

(3) Appropriate exclusion of other liver diseases.

*Identify cause of NAFLD*
In the presence of features of the metabolic syndrome, further evaluation of other causes for NAFLD is usually unnecessary. But detailed drug history is mandatory in all cases. The initial clinical evaluation should include an assessment of adipose tissue distribution to look for lipodystrophy. The initial laboratory evaluation should include a fasting lipid profile.[4]

*Laboratory abnormalities:*
Mildly to moderately elevated serum levels of aspartate aminotransferase, alanine aminotransferase (Usually < 250 u/l, or both are the most common and often the only laboratory abnormality found in patients with nonalcoholic fatty liver disease.[3, 4] The ratio of aspartate aminotransferase to alanine aminotransferase is usually less than 1, but this ratio increases as fibrosis advances.[3] Other abnormalities, including hypoalbuminemia, a prolonged prothrombin time, and hyperbilirubinemia, may be found in patients with cirrhotic-stage nonalcoholic fatty liver disease. Elevated serum ferritin levels are found in half the patients and increased transferrin saturation is found in 6 to 11 percent of patients.[3]

*Imaging studies:*
The diagnosis of NAFLD requires confirmation of hepatic steatosis by imaging or liver biopsy with clinical exclusion of excessive (>20 g/day) alcohol ingestion.[14] Ultrasound, computed tomography, or magnetic resonance studies can confirm the presence of hepatic steatosis with a comparatively high degree of accuracy.[16, 17] The presence of > 33% fat on liver biopsy is optimum for detecting steatosis on radiological imaging. Radiological modalities are unable to distinguish between NASH and other forms of NAFLD, a distinction that has important prognostic implications.[17]

Ultrasound is comparatively cheap and readily available but is less sensitive at detecting minimal (<30%) steatosis or among obese patients (BMI of 35-40 kg/m$^2$).[18] Thus a negative ultrasound does not necessarily exclude NAFLD. The sonographic features of NAFLD include increased hepatic parenchymal echotexture and vascular blurring. These findings are however also seen in those with any form of chronic liver disease and, although sensitive (85–95%), they are non-specific (positive predictive value 62%).[4] The severity of hepatic echogenicity is graded as follows:

Grade 0: normal echogenicity, grade I, slight diffuse increase in fine echoes in liver parenchyma with normal visualization of the diaphragm and intra hepatic vessel borders; grade 2, moderate, diffuse increase in fine echoes with slightly impaired visualization of intra hepatic vessels and diaphragm; grade 3, marked increase in fine echoes with poor or non-visualization of intra hepatic vessels borders, diaphragm and posterior right lobe of the liver.[17]

CT imaging: CT imaging of the liver provides a more specific method for the non-invasive diagnosis of NAFLD. Hepatic steatosis decreases the CT attenuation of the liver. While these features allow hepatic steatosis to be defined with a 76% positive predictive value[4] The severity of hepatic fatty infiltration is graded as follows: grade 0, normal; grade 1, liver attenuation slightly less than spleen; grade 2, more pronounced difference between liver and spleen and intra hepatic vessels not seen or

slightly higher attenuation than liver; grade 3, markedly reduced liver attenuation with sharp contrast between liver and intra hepatic vessels.[17]

MRI: MRI is even more sensitive than a CT scan for the assessment of hepatic steatosis[4] Despite minimal improvement in the interobserver concordance, MRI does not offer any additional advantages over CT or ultrasonogram in detecting the pathologic features that are important in establishing the diagnosis of NASH. The minimal advantages of MRI should be balanced against the wider availability and lower cost of ultrasonogram.[17]

Given the modest increase in diagnostic accuracy and marked increase in cost with CT imaging and MRI, a hepatic sonogram is the most commonly used imaging modality to diagnose a fatty liver. However, it is important to note that none of these methods can diagnose steatohepatitis or accurately assess the stage of the disease. [4]

*Liver biopsy:*

The detection of NASH is usually delayed and there are no serum surrogate markers for NASH,[19] Hence the precise diagnosis of NAFLD and the distinction between hepatic steatosis and steatohepatitis requires a liver biopsy.[4, 19] The presence of severe fibrosis, the most worrisome feature in liver biopsies in patients with NASH, has been noted in 15 to 50% of patients, whereas well established cirrhosis has been documented in 7% to 26% of patients at the time of diagnosis.[20]

So Liver biopsy is the gold standard for diagnosis and is the only investigation able to distinguish between simple steatosis and NASH or stage the degree of fibrosis.[17] Liver biopsy remains not only the best diagnostic tool for confirming NASH, but also the most sensitive and specific means of providing important prognostic information in patients with this condition.[20]

Given the large number of afflicted subjects, the invasive nature of a liver biopsy and the lack of effective treatment, there is often a reluctance to pursue this diagnosis with vigour and many patients are left with a diagnosis of suspected NAFLD. The need to perform a biopsy and make these distinctions continue to generate controversy.[4] There are three basic principles that determine the need and the aggressiveness with which one should try to evaluate any medical problem. First, one must consider the differential diagnosis and the potential possibilities that exist. Next, one must know the natural history of these conditions and consider which diagnoses, if missed, will have dire consequences for the individual patient. The answer to this question is linked to the critical final question: even if the diagnosis is made, is effective treatment available?[4]

The decision to perform a liver biopsy must be individualised and may be useful when there is diagnostic uncertainty or to provide prognostication regarding outcome. The clinical corollary is that individuals without risk factors for NAFLD are more likely to have an alternate cause for their abnormal liver enzymes.[4] Liver biopsy may also be performed in patients with risk factors of advanced fibrosis (diabetes, obesity, age >45, AST: ALT>1),[20] where a diagnosis of cirrhosis has implications for screening for varices and hepatocellular carcinoma.[10]

*Importance of NASH:*

NASH progresses to cirrhosis at a rate that is generally similar to other causes of chronic hepatitis. Therefore, conservative extrapolation from the NHANES III data suggests that 6.4 million US adults have NAFLD (69% of the 9.1 million individuals with cryptogenic aminotransferase level elevations), and 640 000 of these may have cirrhosis (assuming that cirrhosis develops in 10% of those with NASH). In comparison, population-based studies suggest that less than 2% of the US adult population (2 million individuals) are infected with chronic hepatitis C. If 20% of the chronic hepatitis C population develops cirrhosis, then chronic hepatitis C accounts for approximately 500 000 cases of cirrhosis, fewer than caused by NASH.[21] But compensated cirrhosis due to NASH is associated with a lower mortality rate compared with that due to HCV. It is also associated with a lower rate of development of ascites, hyperbilirubinemia, and hepatocellular carcinoma.[22] NAFLD is significantly associated with a moderately increased CVD risk among type 2 diabetic individuals. This relationship is independent of classical risk factors and is only partly explained by occurrence of metabolic syndrome.[23]

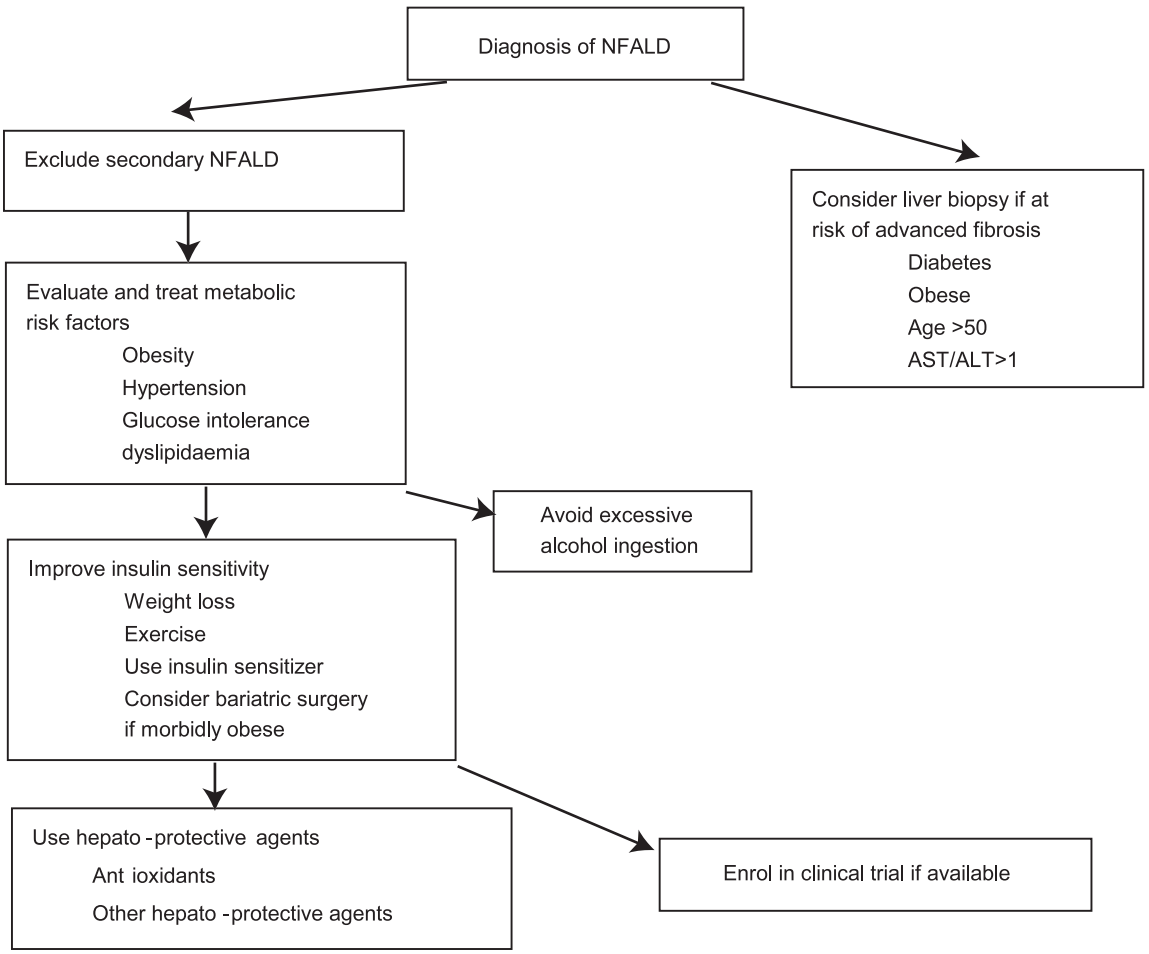
NAFLD and hepatitis C—risk factors and clinical implications:

Hepatitis C and NAFLD are the two most common liver diseases in the Western hemisphere. It is therefore natural that these conditions often co-exist in the same individual. Hepatitis C, especially genotype 3, is often associated with hepatic steatosis.[29] Steatosis influences chronic hepatitis C progression in a genotype specific way.[30] In subjects with genotype 3 infection, a sustained virologic response to treatment is associated with improvement in hepatic steatosis. The diagnosis of NAFLD in a subject with hepatitis C infection is based on the presence of hepatic steatosis. The presence of NAFLD in subjects with hepatitis C genotype 1 infection is most strongly associated with the presence of the metabolic syndrome and insulin resistance. The degree of hepatic steatosis correlates with the degree of hepatic fibrosis and the presence of concomitant steatosis is associated with more advanced fibrosis. Insulin resistance and hyperinsulinemia have been associated with increased collagen production by hepatic stellate cells. Subjects with hepatitis C and NAFLD are more likely to be virologic nonresponders following anti-HCV therapy. The value of treating insulin resistance and NAFLD prior to antiviral therapy remains to be experimentally verified.[29]

Treatment (Figure II):

There is currently no proven therapy for NASH.[21] Treatment strategies for NAFLD have revolved around (1) identification and treatment of associated metabolic conditions such as diabetes and hyperlipidaemia; (2) improving insulin resistance by weight loss, exercise, or pharmacotherapy; (3) using hepato-protective agents such as antioxidants to protect the liver from secondary insults.[10, 24]

Figure II Treatment algorithm for NAFLD [10]



The goals of treatment include (1) correction of the underlying risk factors, (2) avoidance of factors that promote progression of liver disease, and (3) specific treatment of non-alcoholic steatohepatitis.[25]

*Treatment of associated metabolic conditions:*
The features of metabolic syndrome are commonly present in subjects with NAFLD, with 67%–71% being obese, 12%–37% having impaired fasting glycaemia, 57%–68% having disturbed lipid profiles, and 36%–70% being hypertensive. Therefore, patients with newly diagnosed NAFLD should be screened for these conditions and appropriate treatment instituted in an effort to ameliorate the vascular risk as well as to improve NAFLD.[10] The ultimate goal of treating the individual is to prolong life by avoiding the end-organ diseases associated with insulin resistance and the metabolic syndrome. Thus, effective treatment of the metabolic syndrome is mandatory in patients with NAFLD and insulin resistance on general principles.[4]

*Weight loss and exercise:*
The most common risk factor for NASH is obesity.[4] Moderate amounts of weight loss as well as exercise are associated with improvement in insulin sensitivity and sustained improvement in liver enzymes and quality of life.[10, 26] The goal of weight management is to induce a negative calorie balance. This can be accomplished by diet and exercise. Reduction of dietary carbohydrates improve the lipid profile of overweight individuals. Saturated fat intake should also be restricted to prevent diabetes and coronary artery disease. The use of heart healthy diet designed to produce a calorie deficit of 500–1000 calories/day for those who are overweight or obese appears to be rational. Exercise is also proven to be beneficial for coronary artery disease, congestive heart failure, peripheral vascular disease, overall well being among others. Moderate to high-intensity exercise (30 min 3–5 times/week) is ideal for reduction of the risk of co-morbidities associated with obesity. Pharmacological treatment of obesity should be considered in those with a BMI>30kg/$m^2$ or a BMI>27kg/$m^2$ with associated obesity-related co-morbidities. There are currently two drugs approved for weight management: (1) Sibutramine and (2) Orlistat.[4]

*Insulin sensitising drugs:*
It is well established that insulin resistance is a common association with patients with NAFLD and plays an important part in lipid accumulation within the liver and perhaps its progression to NASH. In keeping with this, insulin resistance is predictive of the necroinflammatory form of NAFLD and conditions associated with insulin resistance such as obesity and diabetes are associated with the presence of advanced fibrosis among subjects with NASH.[10] Given the relationship between insulin resistance and NASH, an insulin sensitizer is preferred in those with diabetes and NASH although there are no published studies to support or refute this concept.[4] Insulin sensitising drugs such as metformin and the thiozoladinediones such as pioglitazone are being used in phase III clinical trial in patients with NAFLD.[10]

*Antioxidants:*
Subjects with NAFLD exhibit increased levels of oxidative stress and lipid peroxidation that may play a part in disease progression. Vitamin E is a potent antioxidant and has been evaluated among paediatric and adult patients with NAFLD. Two small pilot trials have shown reduction of ALT levels among adult and paediatric patients with NASH.[10, 27] But combination therapy with Vit E and Pioglitazone was found to be significantly better than vitamin E alone.[27] It is important to note that in two of the studies, there was at least instance each of hepatotoxicity requiring discontinuation of the drug.[4]

*Other hepato-protective agents:*
A variety of hepato-protective agents used in other liver disease have been evaluated in patients with NAFLD. Pentoxifylline inhibits TNF and has been shown to improve short term survival in severe alcoholic hepatitis. Early pilot trials have shown improvement in aminotransaminases in NAFLD patients with 1200–1600 mg/daily of pentoxifylline.

Ursodeoxycholic acid (UDCA) has anti-inflammatory, immune modulating and antiapoptotic properties and is widely used in chronic cholestatic liver diseases.[1] In a study a significant improvement in the liver enzymes and degree of steatosis was found at two years of treatment as compared with baseline; this significant improvement in liver

enzymes and steatosis was also seen in the placebo group. The improvement seen with UDCA treatment was not significantly better than that seen in the placebo group. Based on this study, UDCA is not recommended for the treatment of NAFLD.[4, 10,]

*Lipid lowering drugs:*

As hypertriglyceridaemia and low HDL cholesterol levels are a manifestation of insulin resistance and common among subjects with NAFLD, several investigators have used lipid lowering drugs to treat NAFLD.[10] A decline in transaminase levels and normalization of ultrasonographic evidence of fatty liver were observed on treatment with omega-3 fatty acids in patients with hypertriglyceridemia, with atorvastatin in those with hypercholesterolemia, and orlistat in overweight patients with hyperlipidemia.[28]

*Potential Treatments for Nonalcoholic Fatty Liver Disease[4, 21]*

Lifestyle-modifications to induce weight loss-
Caloric restriction
Physical activity
Insulin-sensitizing agents-
Biguanide (metformin)
Thiazolidinediones (rosiglitazone, pioglitazone)
Leptin (not available commercially)
Antioxidants-
Vitamin E
Betaine
Silymarin
Vitamin C
Hepatoprotective agents-
Ursodeoxycholic acid
Lipid-lowering agents-Fibrates such as gemfribrozil,
Statins
Anti TNF regimen
Pentoxyfylline
Adiponectin

**Conclusion**

Non-alcoholic fatty liver disease is increasingly being recognized as an important and common condition. NASH is not currently considered as a merely benign clinical entity, but is rather thought as a common disease with a variety of clinical sequelae including liver cirrhosis and even hepatocellular carcinoma. It is usually associated with the metabolic syndrome. Its definite diagnosis requires a liver biopsy. The need for a liver biopsy should be individualized and requires consideration of the likelihood of having alternate liver diseases as well as the likelihood of having advanced fibrosis and whether confirmation of the stage of disease will affect management. There is currently no established pharmacologic treatment of NAFLD. Treatment should be focused on correction of the underlying metabolic syndrome. The role of specific pharmacologic treatment continues to evolve. Several large clinical trials using a variety of agents are currently under way and should provide additional treatment option for those with NASH.

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## CASE REPORTS

# Metastatic Jaw Swelling as the First Manifestation of Adrenal Malignancy: A Case Report

SMA SADAT[a], SN RITA[b], M AHMED[c]

**Summary:**

***This case report is of a 65-year-old man with two large soft tissue swellings in upper & lower jaws for 20 days. Incision biopsy revealed metastatic carcinoma with possible primaries was – adrenal gland and kidney. Metastatic adenocarcinoma in left lung and primary mass in right adrenal gland were diagnosed by other relevant investigations. Due to unfavorable general condition, Chemotherapy could not be started. Patient died 20 days after the confirmation of diagnosis & during the course of radiotherapy.***

***(J Bangladesh Coll Phys Surg 2007; 25 : 153-156)***

**Introduction:**

About 1% of the malignant tumors of the body metastasize to the oral cavity[1]. Metastatic tumors in jaws and oral soft tissues are very rare. They represent about 1% of all oral malignancies[2,3,4]. These tumors usually come from lung, breast, genital organs, thyroid, prostate, kidney, bone and adrenals. The following report describes a case of right adrenal tumor with metastasis to the oral soft tissues of both upper and lower jaws, which is very rare. Although most of the lesions of adrenal glands are benign adenomas, adrenocortical carcinomas and metastases constitute 5% to 10% of all tumors[5]. Sometimes they are incidentally detected and can be confirmed by abdominal ultrasonography, computed tomography, magnetic resonance imaging and core biopsy. The purpose of this article is to report an additional example, which was recognized as the first manifestation of adrenal malignancy.

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**Case report:**

A 65-year-old man was referred from Chittagong Medical College to the department of Oral & Maxillofacial Surgery, Dhaka Dental College & Hospital, Dhaka, Bangladesh on 4th August 2005 for evaluation of two rapidly progressive swelling in right maxillary and left mandibular soft tissue growth. The patient noticed two small painless swelling in his right upper and left lower jaw 20 days back. The swellings were very rapidly growing and were associated with mastication problem & general weakness. Patient had not given any history of bleeding from the mass. The past medical history did not reveal any significant disease with no history of chest discomfort or abdominal pain or mass. He had moderate hyperacidity and occasional cough. Patient was habituated with cigarette smoking for last 30 years.

Physical examination showed a lean and thin, malnourished, old man with emaciated facies. He was a febrile and recorded blood pressure and pulse rate were 140/90 mm of Hg and 82/min respectively. Facial asymmetry was evident owing to the swelling in both right upper and left lower face (Fig 1). Intra-orally, right maxillary soft tissue swelling was sessile, firm, non-tender, smooth surface, normal color, measuring 6x5 cm (Fig 2). On the left lower jaw another swelling of 5x4 cm was evident with the same features as above (Fig 3). Both masses were in the molar regions displacing the involved teeth. Both sub-mandibular lymph nodes were enlarged, non-tender, free, firm in consistency with 1 cm in diameter. Other cervical lymph nodes were normal. Clinical examination of chest and abdomen revealed normal findings.

**Fig 1:** *Extra-oral Photograph*

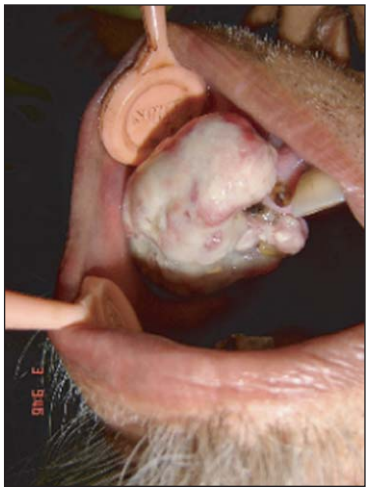

**Fig 2:** *Intra-oral Photograph Rt maxillary mass*

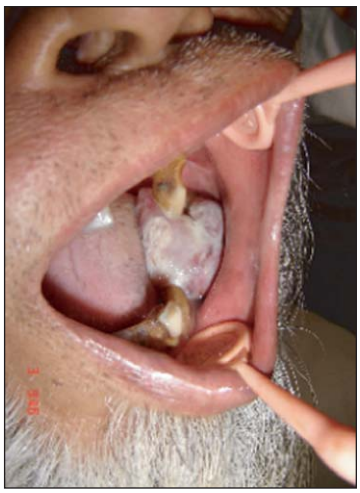

**Fig 3:** *Intra-oral Photograph Lt mandibular mass*

Panoramic radiograph showed soft tissue swelling in the above-mentioned areas with some pressure erosion to the underlying bone (Fig 4). All relevant hematological values were within normal range except ESR 70 mm in 1st hour. Incision biopsy of both lesions reported poorly differentiated malignancy suggesting of metastatic carcinoma with possible primaries of adrenal gland and kidney (Fig 6,7).

Ultra sonogram of whole abdomen revealed a fairly big (5.6x5.0 cm) solid mass above the right kidney well separated from kidney and appears to be an adrenal mass. Chest radiograph showed ill defined medium dense homogenous opacity in left upper and lower zones obliterating left costophrenic angle and left dome of diaphragm indicating the consolidation with pleural effusion due to bronchial neoplasm (Fig 5). CT guided FNAC of the bronchial mass reported metastatic adenocarcinoma. By this time patient was

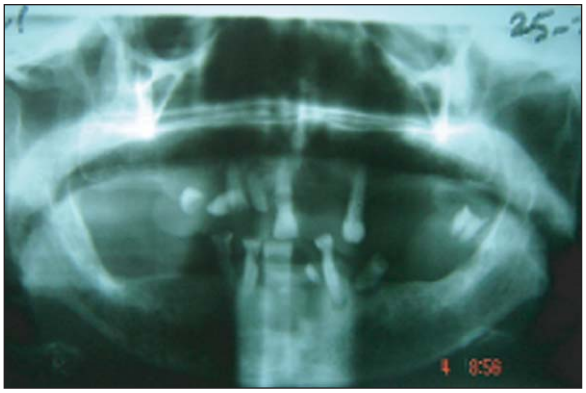

**Fig 4:** *Orthopantomogram of jaws*

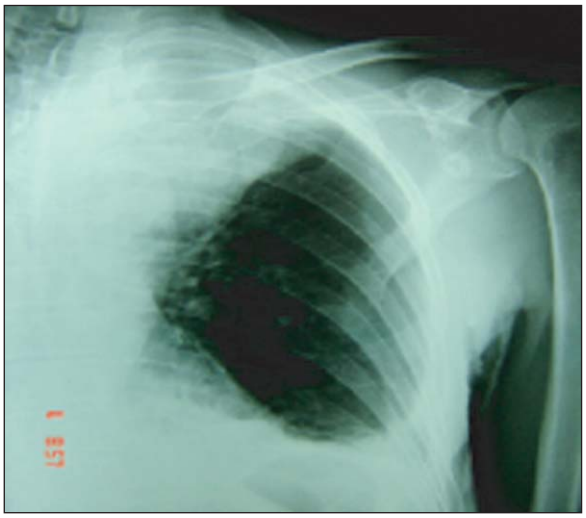

**Fig 4:** *Orthopantomogram of jaws*

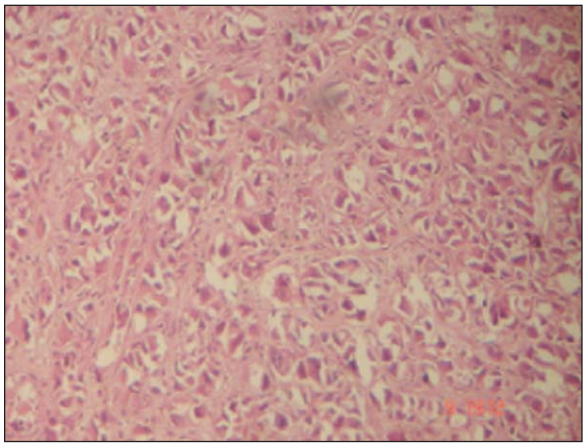

**Fig 6:** *Low power microscopic view*

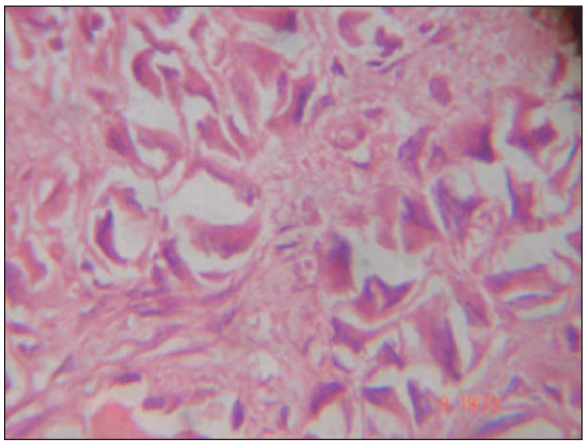

**Fig 7:** *High power microscopic view*

very weak and debilitated and so analysis of adrenal mass and bone scan were not performed. With all documents patient was referred to oncology department where radiotherapy started. Chemotherapy could not be planned due to unfavorable general condition. Patient died 20 days after diagnosis (during the course of radiotherapy).

**Discussion:**

Tumors that metastasize to the oral soft tissues are very rare, comprising only 0.1% of oral malignancies[6,7]. The majority of the oral metastasis occur in jaws (90%), and only about 10% occur in the soft tissues[1].Of the later, 5% are in the tongue, 4% in the gingiva and cheek and 1% in the elsewhere[1]. The most common sites of primary tumors in female are in breast (42%), adrenals (8.5%), genital organs (7.5%) and thyroid (6%)[8,9]. In men the most frequent sites are lung (22.3%), prostate (12%), kidney (10.3%), bone (9.2%) and adrenals (9.2%)[8,10]. In most of the cases malignant cells disseminate mainly to the premolar and molar regions of the mandible where, in a significant number of cases, they can be recognized as the first manifestation of a yet undiagnosed malignant tumor[4,11]. The report describes a case of right adrenal tumor with metastasis to the oral soft tissues of both upper and lower jaws, which is very rare and the presentation was limited to the oral cavity only. Simultaneously metastasis occurred in left lung which was diagnosed by CT guided FNAC. After Ultrasonographic evaluation, the adrenal mass can be diagnosed properly by CT guided FNAC or core biopsy. Adrenal core biopsy is a useful method for identifying and classifying adrenal tumorous lesions if sufficient biopsy specimens can be obtained. However, in clinical practice it remains to be shown whether the benefits of biopsy outweigh the risks of the procedure[5]. In this reported case it was not practiced due to deterioration of patients general condition. Snyder MB et al. reported a case of similar adrenal tumor metastasized to jaw and pulpal tissue in 71/2 - year-old boy[12]. Most of the time, duration of survival of patient becomes short after diagnosis of secondary metastasis. In this case patient died 20 days after the diagnosis. Surgical treatment of metastatic mass is not recommended. Combination Chemotherapy and Radiotherapy can often increase the duration of survival of patient. Due to poor general health, Chemotherapy could not be started in this reported case. Only Radiotherapy to the jaw lesions was applied for controlling the rapid growth of lesion. From the reported case the maxillofacial surgeons should be reminded that 1% of oral cavity malignancies represent metastatic disease, and this reinforces the necessity of maintaining a high index of suspicion for metastatic lesions, even in the most unlikely locations.

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# Ladd's Band - A Case Report

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**Summary:**

***We present here a 7 years' old boy presented with recurrent abdominal pain and vomiting causing severe growth failure since 6 months of age. He had some dysmorphism including fish like mouth, upturned nostrils, hypertelorism, low set posteriorly rotated ears, absence of left kidney and closing ventricular septal defect (VSD). Barium study revealed extrinsic obstruction in duodenal 3rd part with high up caecum. Laparotomy showed Ladd's band. Ladd's procedure was done upon this child, which led to resolution of his clinical abdominal problems.***

***(J Bangladesh Coll Phys Surg 2007; 25 :157-160)***

**Case Summary**

Joy, a boy of 7, from Dhaka was admitted in Bangladesh Medical College Hospital on 02.07.2005 with recurrent upper abdominal pain and vomiting with severe wasting since 6 months of age. His grandmother said that he was growing almost uneventfully up to 6 months of age. Each attack of abdominal pain and vomiting usually lasted for 4-5 days. The problem recurred in an almost similar pattern once in every 2-3 weeks. The pain was localized, moderate to severe occurring around the umbilicus, comes and goes, and aggravated by food intake. Occasionally he feels a dull aching pain in both loins. During each attack he vomits several times mostly after meals. Vomiting is not projectile, and contained only food particles, no blood, and no bile. He gets severely constipated during each episode. Joy did not have any urinary complaints. Parents were concerned, as their child was not growing rather getting stunted and wasted. He was admitted at least 20 times in different hospitals till to date. Once he was admitted in a hospital where he was investigated and suspected to be a syndromic child with congenital absence of left kidney, VSD and gastro-esophagial reflux disease (GERD). Joy was born full term normally at home. Mother had regular antenatal care and the pregnancy was uneventful. He cried just after birth and sucked breasts strongly. Feeding was otherwise normal. Joy was immunised. His growth was normal for the first 6 months. Developmental milestones were satisfactory.

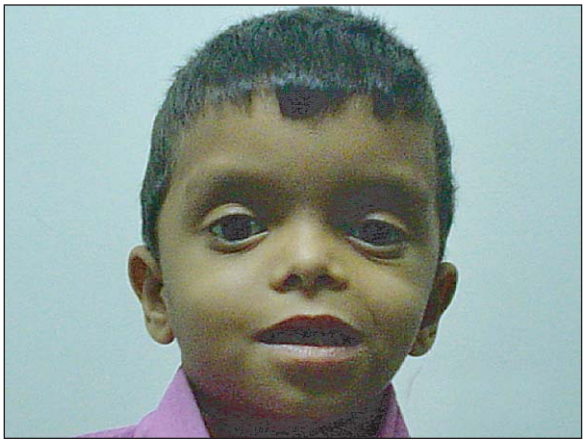

**Fig 1:** *Joy with dysmorphic face*

Joy is the only child of his nonconsanguineous parents. Other family members are reasonably well. Physically, Joy was looking grossly emaciated and stunted. His weight was 10 kg (35% of reference value of CDC; far

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below the 3rd centile), height 95 cm (71.2% of reference value of CDC, far below the 3rd centile), Occipitofrontal Circumference (OFC) 44 cm (reference value for 6 and half months according to CDC); and Mid Arm Circumference 14 cm. His psychomotor, visual, hearing and speech development was absolutely normal. Joy was looking dysmorphic with fish like mouth, upturned nostrils, hypertelorism, low set posteriorly rotated ears, looking pale but no jaundice, oedema or enlarged lymph nodes. Pulse was 68/min, temperature 98°F and breathing 16. He was alert and cooperative. Heart showed normal S1 and S2 with no murmur. Chest was normal. There was no abdominal distension, tenderness, nor any organomegaly. CNS examination revealed normal findings. Laboratory data following admission shows normal peripheral blood count and film study; normal liver functions, and urinalysis. Electrolytes and renal functions were normal. USG revealed no abdominal organomegaly with absence of left kidney. DTPA shows absence of left kidney with normally functioning right kidney. IVU shows non-visible left kidney with normal right kidney. Color Doppler echocardiography done earlier (2004) showed two small perimembranous VSD (almost closing), mild Tricuspid Regurgitation with normal pulmonary arterial pressure. Barium meal and follow-through showed eccentric narrowing of 3rd part of duodenum from outside with thicker mucosal pattern of upper jejunum and high up caecum.

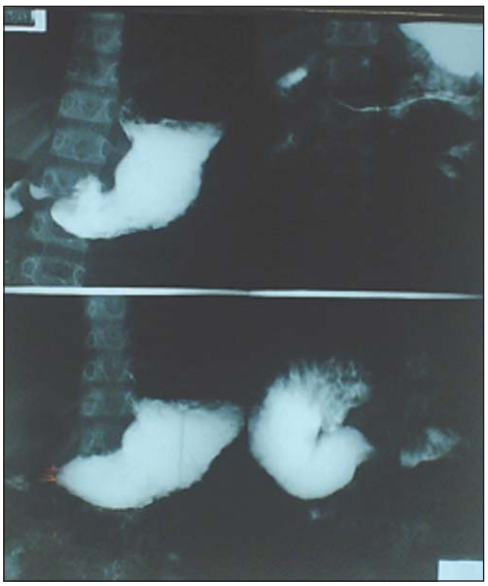

**Fig-2:** *Barium meal of upper GIT shows narrowing of the 3rd part of duodenum.*

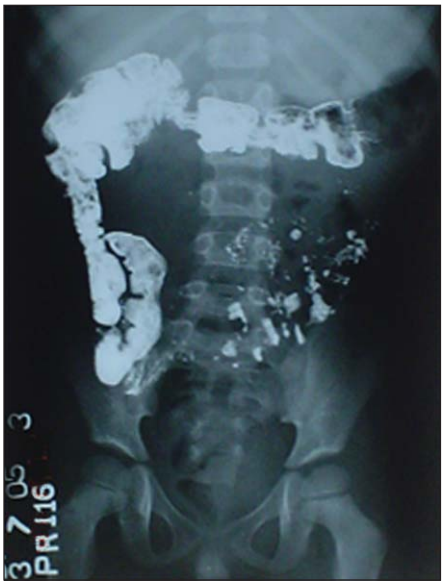


**Fig-3:** *Ba-meal and follow-through of upper GIT shows high up caecum.*

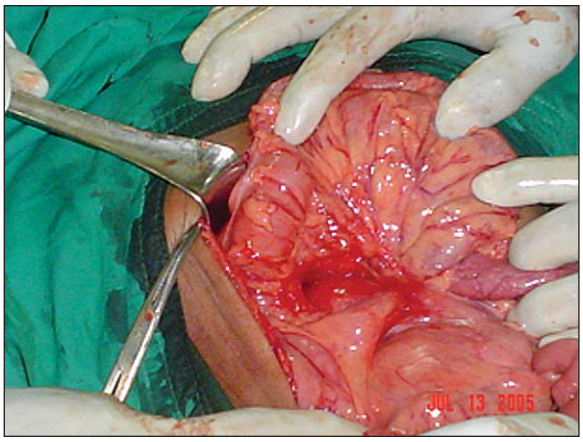


**Fig-4:** *Shows peroperative view of Ladd's band.*

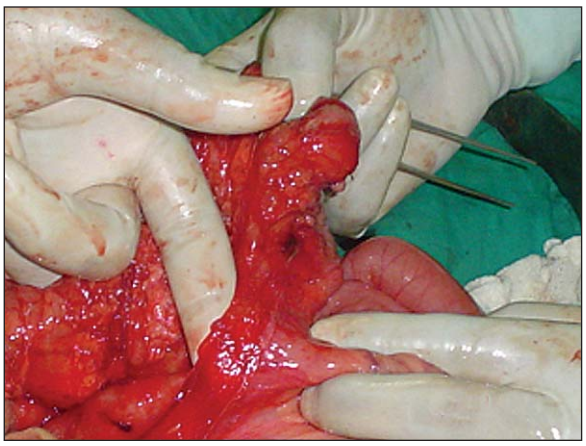

**Fig-5:** *Shows Ladd's band*

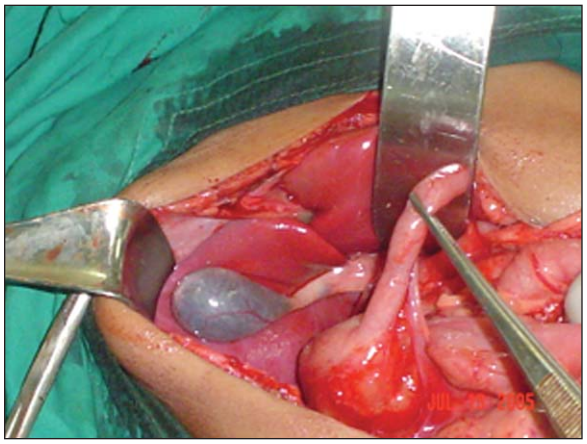

**Fig-6:** *Shows high up caecum almost under the gall bladder*

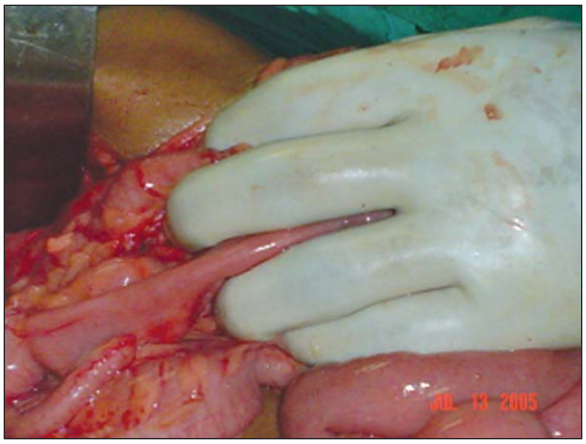

**Fig-7:** *Ladd's procedure is being done*

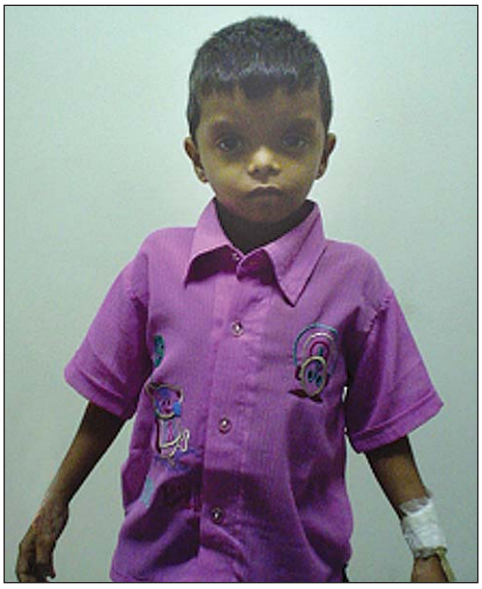

**Fig-8:** *Joy after corrective surgery*

Laparotomy was done and revealed normal stomach but dilated first and second duodenal parts. Caecum was high up, almost under the gallbladder. There were lot of bands between caecum and right paravertebral gutter. The band was carefully divided. Kocherization of duodenum done. The caecum was transferred to *left* iliac fossa and appendicectomy was carried out. Postoperative recovery was uneventful. He went back home with advice to take small frequent meals. Zinc, iron, and multivitamins were supplemented. Joy was advised to report after 2 weeks**.** Joy reported after 1 month with no abdominal complaints and gained a weight of 2 killograms.

**Discussion**

Extrinsic obstruction of the duodenum may be caused by congenital peritoneal band, intestinal malrotation, annular pancreas, preduodenal portal vein and duodenal duplication.[1,2,3] Peritoneal band is always associated with intestinal malrotation, which means an incomplete rotation of intestine during fetal development. There are 4 types of congenital peritoneal band. In type 1, caecum that lies abnormally in right upper quadrant of the abdomen, has a band, which extends across the 2nd and 3rd parts of the duodenum to the paravertebral gutter, this band is called Ladd's Band. Duodenal obstruction may result from either compression by Ladd's Band and/or from midgut volvulus. The type 2 band extends from the hepatic flexure of the colon across the 2nd part of duodenum to the right paravertebral gutter, causing duodenal compression at that site. The type 3 band is the hypertrophied hepatoduodenal ligament, which obstructs the duodenum at the junction of its 1st and 2nd parts. The type 4 band is a dense fibrous band which binds the distal portion of the 3rd part of the duodenum to the paravertebral fascia, causing extrinsic obstruction and is always associated with an incompletely rotated duodenum.[4] It seems that this case falls in the type 1 category.

Clinical diagnosis of congenital band with malrotation in older child is not always easy because of its non-specific presentation. Commonest complaints are intermittent colicky abdominal pain (100%), recurrent vomiting (89%), haematemesis and constipation (55%). Repeated episodes of bloody stool, diarrhoea and failure to thrive (44%).[5] In all cases of persistent vomiting specially biliary a contrast Ba-meal and

follow-through study should be done which is diagnostic if correctly interpreted. [6,7] It should be remembered that congenital peritoneal band is always associated with malrotation and this can cause volvulus of the midgut. In one series 31 out of 219 children had volvulus, 7 needing resection with one death.[8, 9] Therefore, once band is diagnosed Ladd's procedure should be routinely done. Nowadays, it can be done by laparoscopy having the advantage of short convalescence and low morbidity.[10, 12] The association between gastro-esophagial reflux (GER) and intestinal malrotation has well been described. Delayed or impaired gastric emptying is thought to be a contributing factor in the development of GER.[11] This child was having persistent periodic vomiting and got different brands of anti-reflux drugs with little benefit. This should have had raised the query to clinician's mind about the possibility of malrotation. Recurrent pancreatitis is also described in malrotation by some authors and this resolved spontaneously after Ladd's Procedure.[12] This child with some dysmorphic features having absent left kidney, closing VSD, recurrent abdominal pain and vomiting eventually developed severe growth failure and protein energy malnutrition. Single malformation in one system should warn the clinician to have a look for anomalies into other systems. A simple investigation like barium-meal and follow-through would have solved this child's problems much earlier. Any child with long standing periodic vomiting with recurrent abdominal pain causing growth failure an upper GIT barium study should be a routine procedure.

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4. Dr. Md.Ashraf Uddin Ahmed, internee doctor, Department of Paediatrics, Bangladesh Medical College Hospital

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# Device Closure of Ventricular Septal Defect with Amplatzer Muscular Occluder: A Case Report

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**Summary:**

***A four year old girl was diagnosed as a case of mid muscular Ventricular Septal Defect (VSD) since early infancy. She had history of failure to thrive (FTT) and recurrent chest infection or pneumonia. As her pulmonary artery pressure was almost normal she was planned for device closure on elective basis once device and technology would be available in cardiac centre of combined Military Hospital (CMH) Dhaka. Finally it was done on 21st August 2005 and patient was discharged after 72 hours observation period. Echocardiography on next morning showed complete occlusion of defect with no residual shunt.***

***(J Bangladesh Coll Phys Surg 2007; 25 : 161-163)***

**Introduction :**

Ventricular Septal defect (VSD) is the most commonly encountered lesion reported in most cardiac centres [1]. The incidence of VSD in all live birth is approximately 1.5 to 3.5 per 1000 term infants and 4.5 to 7 per thousands premature infants [2,3]. The lower prevalence in adults with congenital heart disease is in large part due to spontaneous closure of many Defects[2,3]. Surgical treatment of haemodynamically significant VSD`s has long been established. Transcatheter closure of muscular ventricular septal defects was first attempted on a compassionate basis using the larger Rashkind occluding device [4]. Though device closure of Atrial Septal Defect(ASD) and Patents ductus arteisosus(PDA) has wide acceptance , transcatheter closure of VSD remains challenging and controversial[5]. It is being available in a small number of centres world wide . Cardiac centre of combined military hospital is a pioneer for all kinds of intervention in newborn and children and first ever case of VSD device closure in Bangladesh was done here with good result which led writing this report.

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**Case Report :**

Miss T, a seven year old girl was diagnosed as a case of Ventricular Septal Defect (VSD), midmuscular type since early infancy. She had history of recurrent respiratory tract infection with failure to thrive. She had hospital admission with pneumonia several times in the past. She was on medical management and follow up evaluation with CXR, electrocardiography and echocardiography and colour doppler was repeated at 3 monthly interval. Her last follow up reports on July 2005 were as follows : ECG showed sinus rythm with left ventricular forces, CXR showed mild cardiomegaly and echocardiography showed a 6 mm size midmuscular VSD with normal pulmonary artery pressure. As VSD size was not decreasing and patient was not gaining weight, she was planned for VSD device closure which was performed on 21st Augest 2005 on elective basis.

Equipments required

1. Muscular VSD device
2. VSD device delivery system with sheath.
3. Terumo exchange wire
4. Arterial and venous sheath
5. JR cathter
6. Pigtail cathter
7. Snare catheter
8. Echocardiography machine with TFE probe.
9. Standard paediatric drape .

Procedure : A 6F sheath was introduced in right Jugular vein, 5F sheath to right femoral vein and 4F sheath to left femoral vein and artery . A pigtail

angiogram of left ventricle (LV) was performed to see the size of VSD. A JR 4 catheter was passed from RFA to LV and VSD was crossed with Terumo wire and JR catheter. Terumo wire was then placed to main pulmonary artery (MPA) and snared with a snare catheter passed through right Jugular vein to MPA. Terumo wire was than withdrawn through right Jugular vein with the help of snare. A long trans septal sheath of 7F size was than forwarded over terumo guide to RA to RV to LV.A VSD mid muscular device of 8x6 mm size was than loaded in the loader with the help of delivery cable. The loader was than attached to long sheath and cable forwarded through the sheath to LV. LV disc was released under TFE and flouroscopy guide. Delivery cable was then withdrawn to RV along with sheath and RV disc was released . A pigtail angiogram of LV was done to confirm complete closure. Sheath and pigtail was then removed. Patient was heparinized during procedure and Inj. Cefuroxime 15 mg\kg body weight IV was given after deployment of device. Cefuroxime was repeated at 8 hourly interval for 24 hours. Echocardiography repeated on next morning showed no residual shunt. Patient was discharged 3 days after the procedure. Follow op appointment was given at 1, 3, 6, 9, 12, 18, 24, months and yearly thereafter.

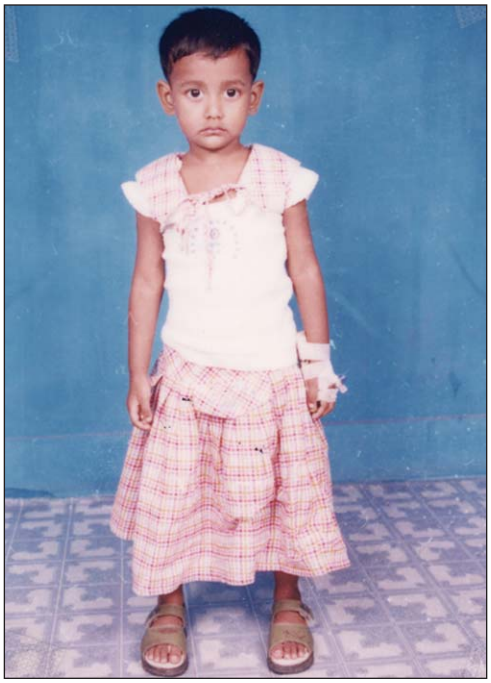

**Fig.-1.** *Miss T, first ever case of VSD device closure performed in Bangladesh.*

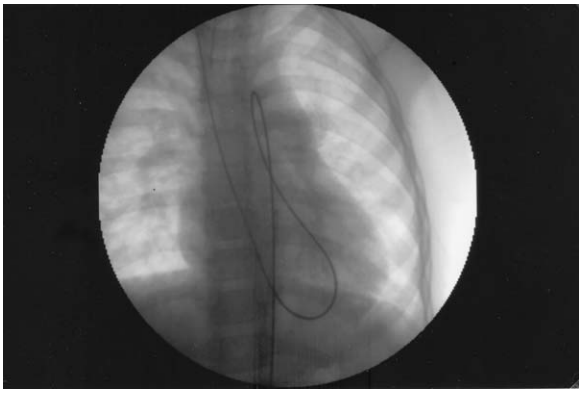

**Fig.-2:** *Showing guide wire and catheter which travelled through the heart from right jugular vein to right femoral artery.*

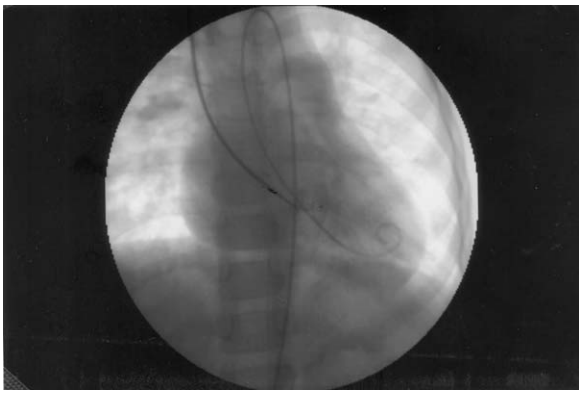

**Fig.-3:** *Showing VSD device attached to delivery cable.*

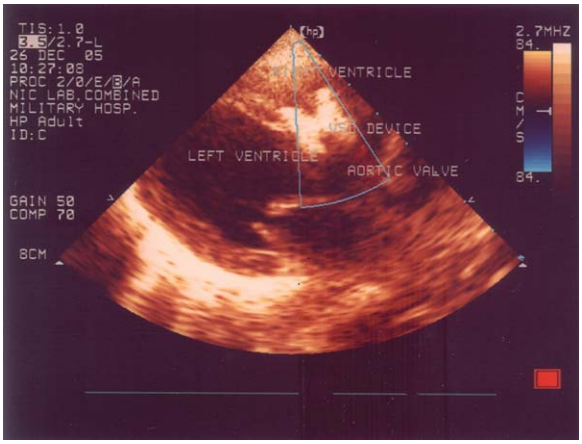


**Fig.-4:** *Chest X-ray showing VSD device in position.*

**Discussion :**

The earlist interventional procedure to be applied in the catheterization laboratory was balloon atrial septostomy[6]. Shortly thereafter, non surgical closure of a PDA was acheived and within ten years ASD had

been closed successfully with catheter device in animals [7]. Non surgical closure of VSD is much less well accepted and can only be regarded as an option for very selected cases, and available only in a few centres worldwide . The procedure for VSD was first attempted by lock et al in 1988 and devices originally designed for the closure of other intracardiac defects (Rashkind umbrella device, lock clamshell, cordioseal, coils, Sideris buttoned devices etc.)[8] were used with a variable success rate and residual shunt. Recently Amplatzer VSD Occluder and Sideris device are in use. The Amplatzer VSD occluder (muscular) has been undergoing clinical trail since 1988 after the animal experiments had shown 100% occlusion and complete endothelization at 3 months[8]. In our case we used Amplatzer muscular VSD occluder. No residual shunt was noticed after implatation.

To evaluate the feasibility, safety and efficacy of transcatheter closure of VSD, many studies were conducted. A study conducted on dog in Azabu university, Japan using coils for perimembranous VSD showed minimal residual shunting after implantation[9]. Another study conducted on ten patient with Amplatzer muscular occluder in Sao Paulo, Brazil proved this device feasible, safe and effective[10]. Immediate and intermadiate term follow up was analyzed in many studies and transcatheter closure of VSD was proved effective in selected cases of VSD`S[11,12,13,14].

Now a days intraoperative device closure of muscular VSD'S are practiced by surgeons [15] .Infants requiring operative Interventions with muscular VSD'S are difficult to manage and have an increased mortality and morbidity.

In some centres hydride procedures are practiced where the heart is exposed surgically to puncture directly and then intervention is performed. The opening in the ventricular wall then closed surgically [15].

Intraoperative VSD device placement could avoid ventriculotomy, division of intracardiac muscle bands, and is ideal for the neonate or infant.

**Reference :**


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# *COLLEGE NEWS*

*(J Bangladesh Coll Phys Surg 2007; 25 : 164-167)*

**Examination News:**

Result of FCPS Part-I, FCPS Part-II and MCPS Examinations held in July, 2007 are given below:

4949 candidates appeared in FCPS Part-I Examination held in July, 2007, of which 812 candidates came out successful, Subject- wise results are as follows:

**FCPS Part-I Examination:**

| SL No. | Name of the Speciality | No. of Candidates Appered | No. of Candidates Passed | % of Pass |
|---|---|---|---|---|
| 1 | Anaesthesiology | 68 | 3 | 4.41 |
| 2 | Biochemistry | 3 | 0 | 0.00 |
| 3 | Dentistry | 223 | 13 | 5.83 |
| 4 | Dermatology & Venereology | 104 | 14 | 13.46 |
| 5 | Family Medicine | 9 | 0 | 0.00 |
| 6 | Haematology | 31 | 2 | 6.45 |
| 7 | Histopathology | 17 | 1 | 5.88 |
| 8 | Medicine | 1584 | 193 | 12.18 |
| 9 | Microbiology | 16 | 1 | 6.25 |
| 10 | Obst. & Gynae | 1158 | 311 | 26.86 |
| 11 | Ophthalmology | 121 | 11 | 9.09 |
| 12 | Otolaryngology | 72 | 6 | 8.33 |
| 13 | Paediatrics | 540 | 88 | 16.30 |
| 14 | Physical Medicine & Rehabilitation | 31 | 12 | 38.71 |
| 15 | Psychiatry | 25 | 0 | 0.00 |
| 16 | Radiology & Imaging | 95 | 0 | 0.00 |
| 17 | Radiotherapy | 19 | 4 | 21.05 |
| 18 | Surgery | 830 | 153 | 18.43 |
| 19 | Transfusion Medicine | 3 | 0 | 0.00 |
| | Grand Total | 4949 | 812 | 16.41 |

737 candidates appeared in FCPS Part-II Examination in Different subjects, List of candidates who satisfied the board of examiners is as follows:

| Roll No. | Name of candidate | From where Graduated | Speciality |
|---|---|---|---|
| 071-7045 | Dr. A.S.M. Areef Ahsan | Dhaka Medical College, Dhaka | Medicine |
| 071-7090 | Dr. Enshad Ekram Ullah | Comilla Medical College, Comilla | Medicine |
| 071-7097 | Dr. Jamal Uddin Ahmed | Dhaka Medical College, Dhaka | Medicine |
| 071-7105 | Dr. M.M. Jahangir Alam | Mymensingh Medical College, Mymensing | Medicine |
| 071-7106 | Dr. M.M. Shahin-ul-Islam | Dhaka Medical College, Dhaka | Medicine |
| 071-7133 | Dr. Md. Hafizur Rahman | Dhaka Medical College, Dhaka | Medicine |
| 071-7173 | Dr. Mesbah Uddin Noman | Dhaka Medical College, Dhaka | Medicine |
| 071-7175 | Dr. Mohammad Abdul Malek | Dhaka Medical College, Dhaka | Medicine |
| 071-7193 | Dr. Mohammad Quamrul Hasan | Sher-e-Bangla Medical College, Barisal | Medicine |
| 071-7195 | Dr. Mohammad Razzak Mia | Dhaka Medical College, Dhaka | Medicine |
| 071-7017 | Md. Mohsin Hossain | Mymensingh Medical College, Mymensing | Cardiology |
| 071-7608 | Dr. Abu Daud Md. Shariful Islam | Rajshahi Medical College, Rajshahi | Surgery |
| 071-7609 | Dr. Abu Naser Md. Mozammel Haque | Rajshahi Medical College, Rajshahi | Surgery |
| 071-7641 | Dr. Mahmud Ekram Ullah | Dhaka Medical College, Dhaka | Surgery |

| Roll No. | Name of candidate | From where Graduated | Speciality |
|---|---|---|---|
| 071-7642 | Dr. Major Ahmed-Ul-Mursalin Choudh | Sir Salimullah Medical College, Dhaka | Surgery |
| 071-7653 | Dr. Md. Ahsan Habib | Dhaka Medical College, Dhaka | Surgery |
| 071-7688 | Dr. Md. Suhel Al Mujahid Reza | MAG Osmani Medical College, Sylhet | Surgery |
| 071-7689 | Dr. Md. Tarikul Islam | Sher-e-Bangla Medical College, Barisal | Surgery |
| 071-7693 | Dr. Md. Nazmul Huda | Dhaka Medical College, Dhaka | Surgery |
| 071-7697 | Dr. Mohammad Ashik Anwar Bahar | Chittagong Medical College, Chittagong | Surgery |
| 071-7712 | Dr. Muhammad Yasser Arfat | Rajshahi Medical College, Rajshahi | Surgery |
| 071-7714 | Dr. Nayeem-Al-Imaam | Mymensingh Medical College, Mymensing | Surgery |
| 071-7723 | Dr. S.M. Quamrul Akther | Mymensingh Medical College, Mymensing | Surgery |
| 071-7725 | Dr. Salma Anam | Sir Salimullah Medical College, Dhaka | Surgery |
| 071-7726 | Dr. Samia Mubin | Dhaka Medical College, Dhaka | Surgery |
| 071-7727 | Dr. Shafiqur Rahman | Sir Salimullah Medical College, Dhaka | Surgery |
| 071-7730 | Dr. Shakera Ahmed | Chittagong Medical College, Chittagong | Surgery |
| 071-7740 | Dr. Tajkera Sultana Chowdhury | Comilla Medical College, Comilla | Surgery |
| 071-7514 | Dr. Major Khaleda Akhter | Sir Salimullah Medical College, Dhaka | Paediatrics |
| 071-7521 | Dr. Md. Abdul Kadir | Dhaka Medical College, Dhaka | Paediatrics |
| 071-7536 | Dr. Md. Jamshed Alam | Rajshahi Medical College, Rajshahi | Paediatrics |
| 071-7542 | Dr. Md. Mashiur Rahman | Dhaka Medical College, Dhaka | Paediatrics |
| 071-7546 | Dr. Md. Rashedul Haque | Dhaka Medical College, Dhaka | Paediatrics |
| 071-7554 | Dr. Mohammad Shameem Hasan | Chittagong Medical College, Chittagong | Paediatrics |
| 071-7570 | Dr. Ranjit Basak | Dhaka Medical College, Dhaka | Paediatrics |
| 071-7574 | Dr. Sadeka Choudhury Moni | Dhaka Medical College, Dhaka | Paediatrics |
| 071-7273 | Dr. Anzuman-Ara-Begum | Mymensingh Medical College, Mymensing | Obst. and Gynae |
| 071-7275 | Dr. Begum Afzalunnessa Chowdhury | Sher-e-Bangla Medical College, Barisal | Obst. and Gynae |
| 071-7287 | Dr. Farzana Sharmin | Sir Salimullah Medical College, Dhaka | Obst. and Gynae |
| 071-7296 | Dr. Jahanara Arzu | MAG Osmani Medical College, Sylhet | Obst. and Gynae |
| 071-7302 | Dr. Jiban Krishan Sarker | Dhaka Medical College, Dhaka | Obst. and Gynae |
| 071-7316 | Dr. Mahabuba Khatun | Sir Salimullah Medical College, Dhaka | Obst. and Gynae |
| 071-7318 | Dr. Mahbuba Nargis | Sir Salimullah Medical College, Dhaka | Obst. and Gynae |
| 071-7320 | Dr. Mahenaz Afroz | Sir Salimullah Medical College, Dhaka | Obst. and Gynae |
| 071-7322 | Dr. Mahmuda Begum | Mymensingh Medical College, Mymensing | Obst. and Gynae |
| 071-7326 | Dr. Masooma Jalil | MAG Osmani Medical College, Sylhet | Obst. and Gynae |
| 071-7327 | Dr. Masuda Sultana | Sir Salimullah Medical College, Dhaka | Obst. and Gynae |
| 071-7358 | Dr. Naznin Rashid Shewly | Sher-e-Bangla Medical College, Barisal | Obst. and Gynae |
| 071-7367 | Dr. Rabeya Begum | Comilla Medical College, Comilla | Obst. and Gynae |
| 071-7370 | Dr. Rebeka Sultana | Sir Salimullah Medical College, Dhaka | Obst. and Gynae |
| 071-7373 | Dr. Riffat Rahim | Mymensingh Medical College, Mymensing | Obst. and Gynae |
| 071-7377 | Dr. Rozina Ahmed | Chittagong Medical College, Chittagong | Obst. and Gynae |
| 071-7379 | Dr. Rumana Sultana | Mymensingh Medical College, Mymensing | Obst. and Gynae |
| 071-7389 | Dr. Selina Akter | Dhaka Medical College, Dhaka | Obst. and Gynae |
| 071-7406 | Dr. Shirin Akhter | Sher-e-Bangla Medical College, Barisal | Obst. and Gynae |
| 071-7416 | Dr. Tazeen Afreen | Dhaka Medical College, Dhaka | Obst. and Gynae |
| 071-7418 | Dr. Umme Parveen | Rajshahi Medical College, Rajshahi | Obst. and Gynae |
| 071-7421 | Dr. Utpala Mazumder | Chittagong Medical College, Chittagong | Obst. and Gynae |
| 071-7459 | Dr. A.N.M. Nurul Huda | MAG Osmani Medical College, Sylhet | Otolaryngology |

| Roll No. | Name of candidate | From where Graduated | Speciality |
|---|---|---|---|
| 071-7468 | Dr. Md. Abdul Azim | Mymensingh Medical College, Mymensing | Otolaryngology |
| 071-7426 | Dr. Bebakananda Biswas | Rajshahi Medical College, Rajshahi | Opthalmology |
| 071-7446 | Dr. Md. Zahedur Rahman | Rajshahi Medical College, Rajshahi | Opthalmology |
| 071-7448 | Dr. Natasha Kajmina | Sir Salimullah Medical College, Dhaka | Opthalmology |
| 071-7451 | Dr. Quazi Mohammed Maniruzzaman | Chittagong Medical College, Chittagong | Opthalmology |
| 071-7594 | Dr. Julhash Uddin Ahmmad | Mymensingh Medical College, Mymensing | Psychiatry |
| 071-7003 | Dr. Md. Anisul Islam | Sher-e-Bangla Medical College, Barisal | Anaesthesiology |
| 071-7005 | Dr. Mahmudur Rahman Khondoker | Rajshahi Medical College, Rajshahi | Anaesthesiology |
| 071-7015 | Dr. Sayeed Mahmud Ali Reza | Sher-e-Bangla Medical College, Barisal | Anaesthesiology |
| 071-7598 | Dr. Kanta Das | Dhaka Medical College, Dhaka | Radiology & Imaging |
| 071-7600 | Dr. Nehar Ranjan Chakraborty | MAG Osmani Medical College, Sylhet | Radiology & Imaging |
| 071-7602 | Dr. Aliya Shanaz | Chittagong Medical College, Chittagong | Radiotherapy |
| 071-7603 | Dr. Shamsun Nahar | Mymensingh Medical College, Mymensing | Radiotherapy |
| 071-7591 | Dr. Md. Abu Taslim | MAG Osmani Medical College, Sylhet | Physical Medicine & Rehabiliation |
| 071-7456 | Dr. (Lt.Col.) Golam Mohiuddin Chowd | Dental College, Dhaka | Oral and Maxilofacial Surgery |
| 071-7263 | Dr. Jamal Pasha Chowdhury | Sir Salimullah Medical College, Dhaka | Microbiology |
| 071-7044 | Dr. Wasim Selimul Haque | Sir Salimullah Medical College, Dhaka | Histopathology |

245 candidates appeared in MCPS Examinations in different subjects. List of candidates who satisfied the board of examiners is as follows:

| Roll No. | Name of candidate | From where Graduated | Speciality |
|---|---|---|---|
| 071-9001 | Dr. Farzana Kalam | Chittagong Medical College, Chittagong | Anaesthesiology |
| 071-9003 | Dr. M.M. Shahidur Rahman | JMC, Kishoreganj | Anaesthesiology |
| 071-9014 | Dr. Mohammad Mohiuddin | MAG Osmani Medical College, Sylhet | Anaesthesiology |
| 071-9018 | Dr. S.M. Majibaur Rahman | Rajshahi Medical College, Rajshahi | Anaesthesiology |
| 071-9022 | Dr. A.K.M. Abdul Awal | Khulna Medical College, Khulna | Clinical pathology |
| 071-9028 | Dr. Md. Nazmul Hasan Khandker | DDC, Mirpur-14 | Dental Surgery |
| 071-9033 | Dr. Umma Habiba | DDC, Mirpur-14 | Dental Surgery |
| 071-9036 | Dr. Md. Humayun Kabir | Sir Salimullah Medical College, Dhaka | Dermatology and Venereology |
| 071-9038 | Dr. Rokeya Begum | Sher-e-Bangla Medical College, Barisal | Dermatology and Venereology |
| 071-9066 | Dr. Khandker Md. Nurus Sabah | MAG Osmany Medical College, Sylhet | Medicine |
| 071-9084 | Dr. Md. Quadrat-E-Elahi | Sir Salimullah Medical College, Dhaka | Medicine |
| 071-9117 | Dr. Anzuman Ara Boku | SZMC, Bogra | Obst. and Gynae |
| 071-9120 | Dr. Begum Ferdousy | Sher-e-Bangla Medical College, Barisal | Obst. and Gynae |
| 071-9124 | Dr. Dilshan ara Begum | Rajshahi Medical College, Rajshahi | Obst. and Gynae |
| 071-9127 | Dr. Fahmida Shahnaz | Rangpur Medical College, Rangpur | Obst. and Gynae |
| 071-9128 | Dr. Farhana Ahmed | Chittagong Medical College, Chittagong | Obst. and Gynae |
| 071-9130 | Dr. Farida Islam | Mymensingh Medical College | Obst. and Gynae |
| 071-9135 | Dr. Jasmine Akhter | Chittagong Medical College, Chittagong | Obst. and Gynae |
| 071-9137 | Dr. Kaji Md.Nasimuzzaman | MAG Osmani Medical College, Sylhet | Obst. and Gynae |
| 071-9140 | Dr. Kazi Taslima | Sir Salimullah Medical College, Dhaka | Obst. and Gynae |
| 071-9144 | Dr. Maya Rani Biswas | Rajshahi Medical College, Rajshahi | Obst. and Gynae |
| 071-9145 | Dr. Md. Abdus Salam | Chittagong Medical College, Chittagong | Obst. and Gynae |
| 071-9149 | Dr. Mohammed Kamal Hossain | Dhaka Medical College, Dhaka | Obst. and Gynae |
| 071-9152 | Dr. Mrinal Kanti Bandapadhaya | IPOMI, Russia | Obst. and Gynae |

| Roll No. | Name of candidate | From where Graduated | Speciality |
|---|---|---|---|
| 071-9153 | Dr. Mst. Anjuman Ara | Rajshahi Medical College, Rajshahi | Obst. and Gynae |
| 071-9155 | Dr. Mst. Shahana Pervin | Rangpur Medical College, Rangpur | Obst. and Gynae |
| 071-9157 | Dr. Muhamad Aminur Rahman | Chittagong Medical College, Chittagong | Obst. and Gynae |
| 071-9159 | Dr. Musammat Shamima Akter | MAG Osmani Medical College, Sylhet | Obst. and Gynae |
| 071-9162 | Dr. Nazma Begum | SZMC, Bogra | Obst. and Gynae |
| 071-9163 | Dr. Nazneen Naher | Rajshahi Medical College, Rajshahi | Obst. and Gynae |
| 071-9167 | Dr. Nusrat Jahan Khan | JMC, Kishoreganj | Obst. and Gynae |
| 071-9179 | Dr. Samia Sultana Lubna | JMC, Kishoreganj | Obst. and Gynae |
| 071-9181 | Dr. Shahanaj Sharmin | Chittagong Medical College, Chittagong | Obst. and Gynae |
| 071-9184 | Dr. Shammi Sultana Ferdousi | Rangpur Medical College, Rangpur | Obst. and Gynae |
| 071-9158 | Dr. Shamsun Nahar | Mymensing Medical College, Mymensingh | Obst. and Gynae |
| 071-9192 | Dr. Suraiya Parvin | Sher-e-Bangla Medical College, Barisal | Obst. and Gynae |
| 071-9195 | Dr.A.Q.M. Omar Sharif | Sher-e-Bangla Medical College, Barisal | Obst. and Gynae |
| 071-9196 | Dr. Amina Akhter | AMC, Pakistan | Opthalmology |
| 071-9199 | Dr. Golam Faruk Hossain | Mymensingh Medical College | Opthalmology |
| 071-9208 | Dr. Kazi Ashraful Islam | Rajshahi Medical College, Rajshahi | Paediatrics |
| 071-9223 | Dr. Muhammad Abdul kayum Shaikh | Mymensingh Medical College, Mymensingh | Psychiatry |
| 071-9225 | Dr. Md. Saiful Islam | Rajshahi Medical College, Rajshahi | Radiology & Imaging |
| 071-9237 | Dr. Nandan Kumar Majumder | Chittagong Medical College, Chittagong | Surgery |
| 071-9244 | Md. Atiqul Haque Sarder | Sir Salimullah Medical College, Dhaka | Surgery |

35 candidates appeared in Priliminary FCPS- II Examination in different subjects. List of candidates who satisfied the board of examiners is as follows:

| Roll No. | Name of candidate | From where Graduated | Speciality |
|---|---|---|---|
| 005-8002 | Dr. Mohammad Faizul Haque Khan | Sir Salimullah Medical College, Dhaka | Preli-Paediatrics |
| 005-8030 | Dr. Mohammed Rashedul Islam | Chittagong Medical College, Chittagong | Preli-Surgery |