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CONTENTS

EDITORIAL

Perspectives in the Prevention and Control of Non-Communicable Diseases (NCD)

Non-communicable diseases are defined as a set of chronic diseases of major public health importance, such as cardiovascular diseases, cancer, diabetes mellitus, lung diseases such as asthma and chronic obstructive pulmonary disease, the development of which is influenced by one or more common risk factors (e.g., smoking, diets rich in energy and fat, physical inactivity and stress)[1]. The literary meaning that diseases which are not communicable (infectious) are non-communicable diseases is also true. But, the diseases of major public health importance, which are considered eminently preventable and/or controllable, are the diseases included under the term NCDs.

The world is clearly witnessing a growing man-made epidemic of NCDs which is being greatly aggravated by a rapidly ageing global population. It has been estimated that by the year 2020 up to three-quarters of all deaths in the world will result from NCDs, and Ischaemic Heart Disease and Depression will top the list of major public health problems in the world, and that there will be a significant increase in the disease burden from lung cancer[2]. In 2002, non communicable chronic diseases were the leading cause of DALYs in all regions of the world except for Sub Saharan Africa. NCDs accounted for 47% DALYs and 53% worldwide deaths in 2002 and the prevalence of NCDs is projected to increase considerably over the next 20 years[2].

There is considerable diversity in the distribution of NCDs both between countries and regions of the world as well as differences within countries. The rise of NCDs in developing countries is inextricably linked to economic and cultural globalisation. Much of the projected rise in NCDs is preventable, particularly that due to smoking, poor diet, physical inactivity and obesity. Health systems in developing countries face both a growing need for prevention programmes and increasing numbers of individuals requiring treatment[3].

The conceptual basis for building a framework for prevention and control of non-communicable diseases can be discussed under the following headings[2]-

Health Determinant and Public Health Perspective

The health experience of an individual or a community is influenced by a variety of factors and conditions. The factors which have been found to have the most significant influence on health -for better or worse - are known as 'the determinants of health'. Broadly speaking, these health determinants cover people's genetic predisposition, lifestyles and other behavioural factors, social relationships with families, friends and community, and the powerful forces of the general socio-economic and cultural environment where they learn, play, work and live. These different determinants operate together to influence health and disease status at both the population and individual levels[3]. For public health policy to be optimal, it is important that we have a good understanding of the underlying factors that determine health.

A public heath approach which focuses on population and risk factors rather than on individuals' symptoms or diseases is important to achieve the goal of promoting health and preventing diseases, addressing the underlying factors that determine health, and increasing the effectiveness and efficiency of healthcare system[4].

Clustering of Risk Factors[4-6]

NCDs are attributed to the complex web of factors described above. Many of these diseases share common behavioural risk factors. For example, four of the most important NCDs are- diseases of the circulatory system, cancer, chronic respiratory diseases, and diabetes mellitus -share three major behavioural risk factors, namely smoking, physical

inactivity and unhealthy diet, which are mediated through common biomedical risk factors, notably excess weight, hypertension and adverse lipid profile. Preventive actions addressing these common behavioural risk factors will improve the community's health profile, which includes optimal body weight, blood pressure and lipid profile. The community will then be benefited from lower incidence of diseases and better health condition.

Life-course Approach

Individuals are influenced by factors acting at all stages of the life span and the risk of developing NCD accumulates with age. Life-course approach acknowledges such interactive and cumulative impact of social and biological influences throughout life, particularly the importance of early life factors predisposing to NCD in later years[7].

Utilising opportunities at each stages of life, it may be possible to have fewer disabilities and reduce premature deaths. The functional capacity, such as muscular strength and cardiovascular output, accumulates in childhood and peaks in adulthood, and then declines in older age. As the rate of functional capacity decline is largely determined by behavioural factors, adopting a healthy lifestyle will help maintain or prevent early decline in functional capacity during older age. For example, stop smoking at age 60, 50, 40 or 30 gains about 3, 6, 9 and 10 years of life expectancy respectively[8]. Thus, it is important to secure growth and development in early life, maintain the highest possible level of function in adult life as well as maintain independence and prevent disability in older life[9].

Preventive Strategy

The planning of NCD prevention and health promotion programmes is based on the three levels of prevention: primary, secondary, and tertiary[10-11].

Primary Prevention: Primary prevention is concerned with measures that prevent the onset of disease. Some of the important strategies under this category include health education, immunisation, environmental measures and social policy. The ultimate goal is to bring about a change in behaviour or factors affecting individuals so that diseases will be prevented from developing. This approach has contributed to some notable examples of successful intervention in public health.

Secondary Prevention: Secondary prevention refers to stopping the progression of a disease after its occurrence, by early detection and diagnosis followed by prompt and effective treatment. The prevention of relapse or recurrence of disease conditions through intervention or attention to lifestyle improvement measures, e.g. smokers to quit smoking after a heart attack is also grouped under this category. Screening, which is one form of secondary prevention, has been more accepted by the general public as a means to "prevent" diseases in recent years[12].

Tertiary Prevention: Tertiary prevention refers to the rehabilitation of patients with an established disease to minimise residual disabilities and complications and maximise potential years of enjoyable life, thereby improving the quality of life even if the disease itself cannot be cured[12].

Population-wide versus individual-based Approach

The distribution of health determinants and risks in a population has implications for successful prevention strategies. While a population-wide strategy for prevention targets at controlling the determinants of health in the population as a whole, an individual-based (also known as high-risk) strategy for prevention identifies high-risk susceptible individuals and offer them some individual protection[13].

The two approaches have their inherent pros and cons[14]. The population-wide approach seeks to promote healthy behaviour to achieve an overall lowering of the risk in the entire population. The potential gains are comparatively extensive but the effect on each participating individual may not be very significant. In contrast, the individual-based approach may appear more appropriate to the individuals. However, it only has a limited effect at a population level and it does not alter the underlying causes of illness. Such an approach also requires continuous and expensive screening processes to identify the high-risk individuals.

Cardiac rehabilitation programme, which is an example of individual-based approach for prevention, is known to be effective in reducing cardiac deaths. Patients are encouraged to exercise and change their

lifestyles after having a heart attack or other heart problems and they can be benefited from tailored lifestyle programmes. A systematic review reported that total cardiac mortality was reduced by 26% to 31% in the exercise only and comprehensive cardiac rehabilitation groups[15]. Another study showed that lifestyle intervention using such approach reduced the risk of people with impaired glucose tolerance in developing diabetes mellitus by 58% over 6 years[16].

With regard to effective interventions using population-wide approach, raising the duties on tobacco products has resulted in a large improvement in population health because fewer people smoke as the price of tobacco rises. Reducing the salt content of processed foods available for sale in the markets, either through legislation or self-regulation of the industry, has resulted in a corresponding reduction in age-specific and sex-specific mean systolic blood pressure[17]. When NCD are prevalent in the community, even modest changes in risk factor levels through population-wide approach will yield significant public health benefit[18].

There are many examples worldwide on successful mix of population-wide approach and individual-based approach for preventing and controlling NCD. Communities can make major gains once becoming involved in reducing health risk behaviours associated with many chronic diseases. Some of the most notable cardiovascular diseases prevention trials are the Stanford Three-Community Project, North Karelia Project, Stanford Five-City Project, Minnesota Heart Health Program and the Pawtucket Heart Health Program[16-18]. These projects have made known that cardiovascular diseases are preventable through modifications of established risk factors including cigarette smoking, elevated blood lipids, elevated blood pressure and sedentary lifestyle.

The basic premise for this work is that community-wide strategies lead to a reduction in disease rates through changes in individual and community risk factors. Each provides valuable models, diversified methodologies addressing awareness and education, skill-building and advocacy, and strategies for planning and implementing community-based/led programmes. These programmes are cost-effective, easily transferable and have dramatic impacts on health policy development[18].

Health Disparity

Disparity in health usually refers to a broad range of differences in health status between population subgroups. Although some disparities in health are inevitable because of genetic and biological make-up in individuals, health disparities are often attributed to differences in personal lifestyle, exposure to material resources and opportunity of receiving healthcare services[19]. For example in China, as the result of increasing affluence and the adoption of western diet, people living in the cities had a 2.7-fold increase risk of having diabetes mellitus than those living in poor rural area[20]. Striving to minimise the health gap between population subgroups has become a challenge in public health[2]. Thus, an important public health task is to identify the underlying health determinants attributable to health disparities and develop responsive policies for their reduction.

Health Literacy and Social Marketing

Health literacy is the ability to read, understand, and act on healthcare information. Study has indicated that poor health status is disproportionately high among people with low health literacy[21]. For enhancing the population health, therefore, the health literacy of the whole population needs to be increased. Social marketing, as an effective health promotion method, can motivate people to use health information and change behaviour in ways that promote and maintain good health. Over years, many places including Hong Kong have used social marketing campaigns for health promotion[22-23].

Setting Health Priority

Bangladesh has one of the most vulnerable economies, characterized by extremely high population density, low resource base, and high incidence of natural disasters. These have adverse implications for long-term savings, investment, and growth. There is never as much funding as is needed to address all important health problems, so priorities need to be set. Priority setting is imperative for the rational utilisation of resources for public health programmes in a community[24]. However, identification of priority health areas is not easy. Whether or not a particular disease or health condition should be focused and targeted for preventive activities depends on a number of factors.

Over the past two decades, some developed countries have gone through the process of identifying health

priorities and started working on the identified health priority areas. For example, Australia has selected seven National Health Priority Areas for action, including asthma, cardiovascular health, cancer control, injuries prevention and control, diabetes mellitus, mental health and arthritis and musculoskeletal conditions, while the United States (US) also views heart disease and stroke, cancer and diabetes mellitus the most important health problems[25]. We need to agree what priorities should be set in Bangladesh and what targets need to be met. Before selecting the priorities of NCDs for intervention we have to obtain the population based data through surveillance[26].

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ORIGINAL ARTICLES

Management of Bronchiolitis with or without Antibiotics –A Randomized Control Trial

MJU MAZUMDER[a], MM HOSSAIN[b], ARML KABIR[c]

Summary:

Background: There has been epidemics of bronchiolitis in the recent years in Bangladesh. Bronchiolitis is mostly (95%) a viral disease in infants and young children but being treated with antibiotics in 99% of cases in our situation. Antibiotic has little role in the management of bronchiolitis. Very few randomized control trials without antibiotics in the management of bronchiolitis have so far been done.

Objectives: To evaluate the outcome of bronchiolitis with or without antibiotics in a hospital setting.

Methods: A randomized control trial was done during one winter season of 2005 with all cases of bronchiolitis attending a teaching hospital of Dhaka, Bangladesh. Sample size was selected conveniently. One hundred twenty six consecutive cases (one month up to 2years) with clinical bronchiolitis (runny nose followed by wheeze, cough, breathing difficulty perceived by caregiver, chest indrawing and rhonchi on auscultation) who attended the hospital were enrolled in the study. Detailed history and clinical examination were done and the children were randomized into 3 groups: (1) parenteral antibiotic group, paren AB (30) treated with supportive management and IV ampicillin, (2) oral antibiotic group, oral AB (33) treated with supportive management and oral erythromycin and (3) no antibiotic group, no AB (63) treated with supportive management only. The children were managed both in indoor and outdoor but very sick patients particularly those having oxygen saturation <90% were admitted into the hospital or excluded from the study (if not agreed for hospitalization). Oxygen therapy was given to cases having oxygen saturation < 90% and IV fluid (10% dextrose in 0.225% NaCl) was given to severely distressed children. Tube feeding was given to children who were unable to take milk by mouth but not very sick deserving IV fluid. Antibiotic was given according to the protocol. All children were followed up for 23 parameters, hospitalized cases were observed 8 hourly and outdoor (OPD) cases twice in the morning and at noon. Outcome measures were breathing difficulty, feeding difficulty, social smile, fast breathing (R/R > 50/m), hypoxia (oxygen saturation <95%), wheeze, rhonchi and crepitation. Verbal consent of the parents was taken before the study. Whenever patients condition became worse with the given treatment, the children was taken out of the study and more intensive management was given. Parents were also at liberty to discontinue the treatment process whenever they wanted irrespective of the reasons.

Results: Out of enrolled 126 children with bronchiolitis 104 (82.5%) improved and were discharged safely. The improved children in different groups were as follows: paren AB 29(27.8%) , oral AB 32(30.7%) and no AB 43(41.3%). Total 22 cases were excluded from the study, o1 from paren AB, 01 from oral AB and 20 from no AB group. Among them 18 were OPD cases , did not turn out on regular follow up, 2 cases left hospital on DORB and 2 cases were excluded from no antibiotic group for persistence of breathing difficulty and crepitation in the lung and treated with antibiotics. There was no death. Mean TWBC count was around 8500/cmm in all the groups. The mean value of neutrophil and lyphocytes were 33% and 61% respectively. Radiologically about 70% cases had hyperinflation, 52% cases had hypertranslucency and 56% cases had streaky densities. Hundred percent children had breathing difficulty at the time of inclusion into the study in all the groups. The decrement of breathing difficulty was gradual in all the groups and on day 5 only 27% in paren AB , 25% in oral AB and 34% in no AB group had breathing difficulty (p o.66). About 50% children had feeding difficulty at the

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beginning of study in all the groups. The decrement of feeding difficulty was found rapid and similar in all the groups and there was no feeding difficulty on day-5 in all the groups. Only 34% children in paren and oral AB group and 30% in no AB group had social smile on day-1. On day-3 about 90% of children of all the groups started smiling in spite of having fast breathing and chest in drawing. About 91% children had tachypnea (RR >50/m}at the time of inclusion into the study. The decrement of fast breathing was gradual and similar in all the groups and on day five only about 10% children had fast breathing and it was equal in all the groups (p.05). About 54% children had hypoxia during inclusion in all the groups (p0 .49). The improvement of hypoxia was rapid and similar in all the groups and on day-5 only 6.7% had hypoxia. Hundred percent children of all groups had wheeze at the beginning of the study. The decrement of wheeze was gradual and similar in all groups. On day five total 15% children had wheeze and it was almost equal in all the groups (p.82). The decrement of crepitations in all the groups was also gradual. During inclusion into the study about 60% children had crepitations and it was almost equal in all the groups and on day five about 14% children had crepitations in all the groups (p o.97).

Conclusion: The recovery of bronchiolitis managed with supportive therapy alone was found similar to those treated with combined supportive therapy and antibiotics (either oral or parenteral).

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Introduction:

Bronchiolitis is a common acute contagious respiratory illness of infants and young children involving the lower respiratory tract. It is the most significant respiratory illness of young children[1]. It is a viral disease. The most important causative agent is respiratory syncitial virus (RSV) and it accounts for bronchiolitis in more than 50% cases. Other causative agents are parainfluenzae, influenzae,adenovirus, mycoplas-ma pneumonae, herpes simplex, human metapneumovirus and mumps virus[2,3]. There is no evidence of a bacterial cause for bronchiolitis, although bacterial pneumonia is sometimes confused clinically with bronchiolitis[3].

During an epidemic in Bangladesh RSV was found as the most common responsible agent for bronchiolitis. In a recent study in the different hospitals of Dhaka city in the month of January and February 348 cases were diagnosed as bronchiolitis and were found positive for RSV antibody (1gM or 1gM and Ig G) in 50% cases[4]. In the same study it was found that antibiotics were used in almost all cases (99%). But the fact is that antibiotics have been shown to be of no benefit in the treatment of bronchiolitis[5]. In Canada 57%-81% of infants with diagnosis of bronchiolitis were getting antibiotics, despite the fact that antibiotics have been shown to be of no benefit in the treatment of bronchiolitis. In addition, there is evidence that RSV infection does not predispose to bacterial superinfection regardless of radiologic finding[6]. Kuppermann et al showed in a prospective cohort study that none of the 156 patients with bronchiolitis had bacteremia[7]. Secondary bacterial infections appear uncommon in RSV bronchiolitis. The routine use of antibiotics has not been shown to influence the course of bronchiolitis and there is little rationale for their use. Only when there is evidence of secondary infection should antibiotics be considered[5]. In a standardizing care of bronchiolitis by penny et al they concluded that antibiotics are indicated if bacterial pneumonia is suspected e.g. high fever, toxic appearance, WBC> 15,000 and lobar infiltrate[8]. It has been repeatedly shown that the excessive and often inappropriate use of antibiotics promotes the development of resistant organisms[9]. The incidence of bronchiolitis has been documented in recent years in Bangladesh[10]. We have developed a consensus in the guidelines for the management of bronchiolitis both at home and hospital settings[11]. Bronchiolitis is a viral disease and the most common cause is RSV. It is a self-limiting disease. As bacterial infections are rare in bronchiolitis, antibiotics have little therapeutic value[12]and antibiotics are not recommended unless there is concern for complications such as secondary bacterial pneumonia[13]

Materials and Methods:

This was a randomized control trial conducted during January to July 2005 in a teaching hospital, Institute of Child and Mother Health(ICMH) Matuail, Dhaka. Total 126 consecutive cases (one month upto two years) with clinical bronchiolitis were selected conveniently. Diagnosis of bronchiolitis was made on the basis of following inclusion criteria : age one month up to 2 years, preceding/ existing runny nose, cough, breathing difficulty (as perceived by the

caregiver), lower chest in-drawing, wheeze and rhonchi on auscultation. The exclusion criteria were: child with atopic conditions (asthma, allergic rhinitis, allergic conjunctivitis, atopic eczema), congenital heart disease, high fever >102°F and toxic appearance. Children below 2 years with breathing difficulty attended at outpatient department were identified and included in the study on the basis of inclusion criteria. Parents/attending relations were briefed about the study, its implications, management, follow up, their options to discontinue. The children whose caregiver agreed, were included in the study. A structured questionnaire was filled up through face to face interview with the caregivers at the beginning of the study. Detailed history and clinical examination were done and the children were randomized into 3 groups: (1) paren antibiotics (AB) who were treated with IV ampicillin and supportive management,(2) oral antibiotics (AB) who were treated with oral erythromycin and supportive management and (3) no antibiotics (AB) who were treated with supportive management only. Randomization was done on the basis of odd and even number. All odd number cases were managed with antibiotics (oral and parenteral alternately) along with supportive management and even number cases were managed with supportive management only. A pulse oximeter was used to observe the level of oxygen saturation in blood immediately after inclusion into the study. The very sick patient who had significant hypoxia (O_2< 90%), feeding difficulty and those selected into paren AB group were admitted into the hospital. Among them whose parents did not agree to be hospitalized were excluded from the study. Oral AB group was given syrup erythromycin 30-50 mg/ kg/ day every 6 hours[14], the paren AB group was given IV ampicillin 100-200 mg/ kg/ dose every 6 hours[15] and no antibiotic was given to no AB group. The supportive therapy was given to all cases according to national guidelines for the management of bronchiolitis[16]. All hospitalized children were managed with salbutamol nebulisation 6-8 hourly (0.15 mg/kg/dose), oxygen therapy (when oxygen saturation < 90%), IV fluid 10% dextrose in 0.225% Nacl (incase of severely distressed children), NG tube feeding to children who were unable to take milk by mouth but not very sick deserving IV fluid, paracetamol for fever and oropharyngeal suction when needed. Hospitalized children were followed up 3 times in 24 hours (9: 30am, 2.00pm and 8:00pm) and OPD cases were followed up two times (9:30am and 2:00pm) up to seven days in a structured follow up sheet. All OPD cases were either in oral or no AB group and nebulised in the morning and at noon and advised to take salbutamol syrup at a dose of 0.2- 0.4 mg/kg orally at night. Total 23 parameters were followed up daily which were: cough, runny nose, breathing difficulty, feeding difficulty, socialsmile, restlessness, inconsolable cry, sleeping difficulty, nasal blockade, convulsion, wheeze, chest indrawing, nasal flaring, cyanosis, impairment of consciousness, temperature, respiratory rate, heart rate, liver, spleen, rhonchi, crepitation and arterial oxygen saturation by pulse oximeter. Outcome measures were breathing difficulty, feeding difficulty, social smile, fast breathing (R/R > 50/m), hypoxia (oxygen saturation <95%), wheeze, rhonchi & crepitation. Hematological profile (total and differential count of WBC, Hb%, ESR,CRP) and X-ray chest was done in all cases. Criteria for discharge were satisfactory feeding as per mothers confidence, return of social smile and no significant hypoxia (SaO^2 >90%) in room air. The improvement of individual feature were defined as marked improvement: improvement of the feature in about 90% of children, rapid recovery: marked improvement occurring within 4 days, very rapid recovery: marked improvement occurring within 2 days, gradual recovery: marked improvement occurring beyond day- 4, very gradual recovery: marked improvement occurring at the end of day -7. All data were checked for consistency and correctness and scrutinized by one of the authors. The data were cleaned and entered by data enterers into the Epi-info program and analyzed in the SPSS software program with the help of an epidemiologist. Data recorded in a pretested questionnaire in to computer and analyzed by using SPSS statistical software employing appropriate statistical test like Chi square and determination of p value. Ethical approval was obtained from the Ethical Committee of ICMH and informed consent was taken duly from the parents before enrollment.

Results:

Most of the babies (92.2%) were within first year of life, 67.3% were male and 32.7% were female. Out of

enrolled 126 children 104(82.5%) improved and discharge safely. The improved children (104) who were as follows: paren AB (29) treated with IV ampicillin, oral AB (32) treated with oral erythromycin and no AB(43) given no antibiotics. Total 22 cases were excluded from the study, o1 from paren AB, 01 from oral AB and 20 from no AB group. Among them 18 were OPD cases , did not turn out on regular follow up, 2 cases left hospital on DORB and 2 cases were excluded from no antibiotic group for persistence of breathing difficulty and crepitation in the lung and treated with antibiotics. Hematological profile was like that of bronchiolitis[4, 22] in all the groups. Mean TWBC count was 8500/cmm. The mean value of neutrophil and lymphocytes were 33% and 61% respectively. CRP was found <6 in75% cases, 12 in 11.5% cases and 24 in 12.5% cases. Radiologically, all cases had the similar features suggestive of bronchiolitis[18], hyperinflation in 70%, hypertranslucency in 52% and streaky densities in 56% cases. All the children (100%) had breathing difficulty at the time of inclusion into the study in all the groups. The decrement of breathing difficulty was gradual in all the groups. There was a tendency of early recovery in oral AB and paren AB in comparison to no AB group but the tendencies were not significant p 0.66. On day-5 about 27% paren AB, 25% oral AB and 34% no AB had breathing difficulty (Fig-1). About 50% children had feeding difficulty at the beginning of the study; paren AB 41.3%, oral AB 40.6% and no AB 58% p 0.23. The decrement of feeding difficulty was found rapid and similar in all the groups and on day 5 there was no feeding difficulty in all the groups (Fig-2). About 33% of children had social smile at the time of inclusion in to

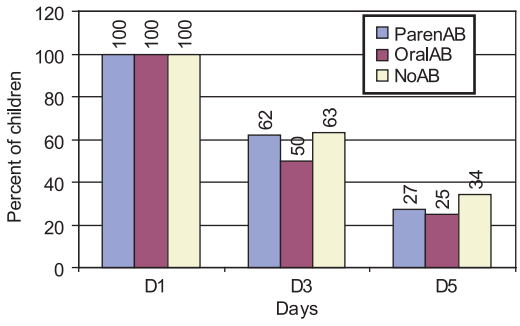

Fig.-1: *Gradual decrement of breathing difficulty in all the groups*

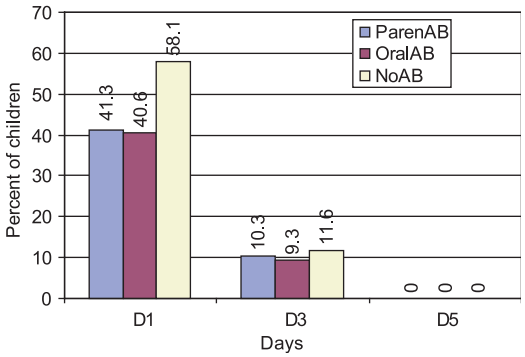

Fig.-2: *Rapid decrement of feeding difficulty in all the groups*

the study in all the groups, paren AB 34.5%, oral AB 34% and no AB 30% p0.90. There was rapid and similar improvement of social smile in all groups and on day 5 100% children in all groups developed social smile (Fig-3). About 90% children had tachypnea (RR>50 per minute) at the time of inclusion into the study, paren AB 86.9%, oral AB 93.7% and no AB 93%. The decrement of fast breathing was gradual and similar in all the groups and on day five total 10% children had fast breathing, paren AB 10.3%, oral AB 9.3% and no AB 11.6% p .05 (Fig -4). Fig 5 showed that about 54% children had hypoxia (SaO2 < 95%) at the time of inclusion in to the study, paren AB 62%, oral AB 46%, no AB 53.4% p 0.49. The improvement of hypoxia was gradual and similar in all groups and on day five total 6.7% had hypoxia paren AB 6.8%, oral AB 9.3%, no

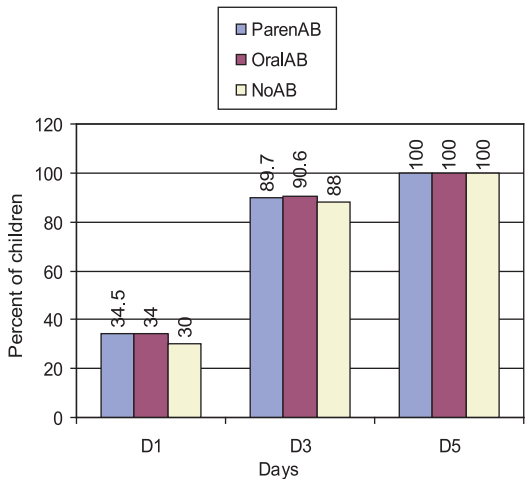

Fig.-3: *Rapid return of social smile in all the groups*

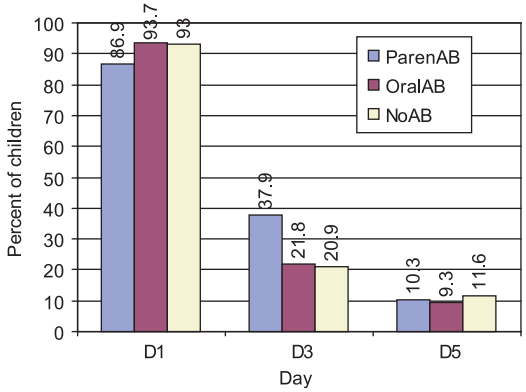

Fig.-4: *Gradual decrement of fast breathing (RR>50/min) in all the groups*

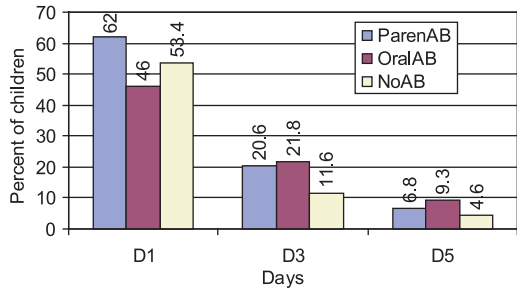

Fig.-5: *Gradual decrement of hypoxia in all the groups*

AB 4.6% . Hundred percent (100%) children had wheeze during inclusion into the study in all groups. On day 5 total 15% children had wheeze, among them paren AB 13.7%, oral AB 20.6%, and no AB 13.9% p.82 (Fig-6). On the day of admission 100%

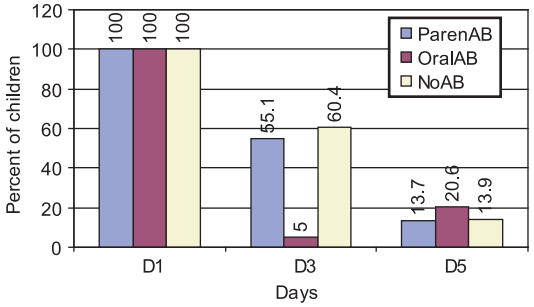

Fig.-6: *Gradual decrement of wheezing in all the groups*

children had rhonchi in all groups. On day 5 about one fifth of total children had rhonchi, paren AB 20.6%, oral AB 25% and no AB 20.9% p.89. About 60% children had crepitation during inclusion into the study. On day 5 total 14.42% children had crepitation, paren AB 13.7%, oral AB15.6% and no AB 14%. Liver and spleen were palpable in 53% and 22% cases respectively.

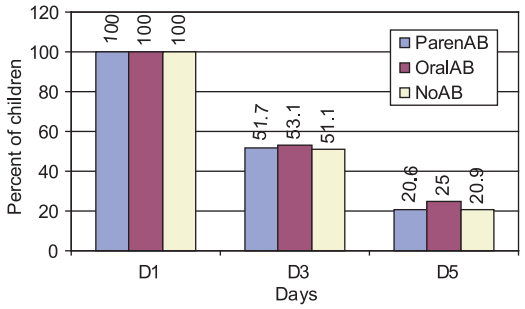

Fig.-7: *Gradual decrement of ronchi in all the groups*

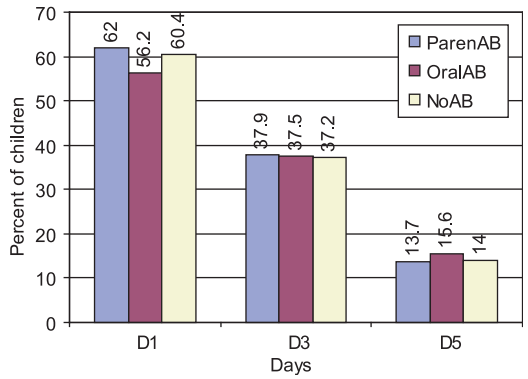

Fig.-8: *Gradual decrement of crepitation in all the groups*

Discussion:

This randomized control trial provides the opportunity to see the outcome of bronchiolitis with or without antibiotic in a hospital setting. We strictly followed the case definition of clinical bronchiolitis [13,17,18]. All children were one month up to 2 years with preceding/ existing runny nose, cough, breathing difficulty , lower chest indrawing, wheeze and rhonchi on auscultation. Hematological profile was similar to other studies[4,19] in all the groups, mean TWBC count was 8500/cmm. The mean value of neutrophil and lymphocytes were 33% and 61% respectively. Radiologically, all cases had the similar

features suggestive of bronchiolitis[13,19,20]. Hyperinflation in 70% and hyper translucency in 52% cases and streaky densities in 56% cases. Liver and spleen were palpable in 53% and 22% cases respectively because of downward displacement of these organs by the hyperinflation of the lungs. In this study the children were randomized into three groups according to odd and even number successively following enrollment into the study. The patients in three groups were as follows: paren AB (29). oral AB (32) and no AB (43).

Paren AB group was given intravenous (IV) ampicillin as was the recommendation of WHO for the treatment of severe pneumonia of hospitalized children in the Integrated management of Childhood Illness (IMCI) guideline[15]. The only non-viral cause of bronchiolitis is Mycoplasma pneumonae in 5% of cases. The median age of bronchiolitis in our situation was 3 months[4] and Chlamydia trachomatis is also a cause of afebrile pneumonia in infant of under 6 months of age [21,22] . Considering these two factors, oral erythromycin was chosen for another choice of antibiotic apart from IV ampicillin. All the children were managed with salbutamol nebulisation in addition to other supportive management whenever needed like IV infusion of 10% dextrose in 0.225% saline, oxygen therapy, NG tube feeding, oropharyngeal suction and paracetamol suspension for fever. All the children were treated free of cost to encourage the parents to stay in the hospital or to come on regular follow up in outdoor . In spite of all efforts twenty (15.8%) parents discontinued the treatment and excluded them from the study. All admitted cases were followed up 8 hourly and outdoor cases were followed up in the morning and at noon using a structured follow up sheet. The follow up features were 23 variables, 10 symptoms and 13 signs including estimation of oxygen saturation of blood by pulse oximeter. We were very liberal to address the ethical issue and the children whose condition deteriorated, were taken out of the study and further evaluation was done and treated accordingly. We were always vigilant on the child's condition in the hospital so that we could take out the child from the study group whenever needed, we also instructed the parents/caregivers who continued treatment at home to comeback immediately in the hospital when they felt that the condition of the child became precarious. Two cases were excluded from the study and given more intensive management as they were not improving by supportive management only. The parents were also at liberty to discontinue the treatment whenever they felt like. Twenty (15.8%) parents discontinued the management although initially agreed to be included in the study ,among them 18 parents (OPD case) did not come back for follow up and 2 parents left the hospital on risk bond during study and they were also excluded from the study. The limitation of the study was that we could not follow up all the cases for 24 hours as some cases (about one third) were outdoor cases and they were only followed up in the morning and at noon and number of dropout cases were very high (22%).Though all the cases were targeted for follow up for 7 days, some cases left the hospital after 5 days as parents felt improvement of their child and they were discharged on request and some OPD cases were also allowed to discontinue follow up on request as they registered significant improvement.. For this reason data was analyzed up to day 5. In this study it was found that the recovery was essentially similar in all the groups whether treated with IV antibiotic or oral antibiotic or no antibiotics. Recovery like social smile, feeding difficulty, breathing difficulty, chest indrawing and oxygen saturation in all the groups were almost similar. The improvement was rapid in the parameters of feeding difficulty and social smile in all the groups and it was on day 3 of hospitalization. The improvement was gradual in all the groups in the features like breathing difficulty, fast breathing, hypoxia, wheeze, rhonchi and crepitation. .It took 5 to 7 days for marked improvement.

One randomized control study so far conducted long ago on the use of antibiotic in bronchiolitis and it found no evidence to support the use of antibiotics for bronchiolitis[23] which corroborates present study results. Another randomized, double blind, placebo-control trial conducted and showed that clarithromycin had statistically significant effects on the clinical and laboratory findings in respiratory syncitial virus bronchiolitis. But the study was conducted with small sample size (21 infants)[24].

Conclusion:

The recovery of bronchiolitis managed with supportive therapy only, was found similar to those treated with antibiotics (either oral or parenteral) and supportive therapy. The recovery was rapid and similar in the features like feeding difficulty and social smile. The recovery was gradual and similar in other feature like breathing difficulty, tachypnea, hypoxia, wheezing, rhonchi and crepitation.

Recommendation: Further multi centre study with large sample size is needed to recommend or refute the role of antibiotic in bronchiolitis.

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Epidemiology, Clinical Profile and Outcome of Patients of Snake Bite in Mymensingh Medical College Hospital

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Summary:

Snake bite is a serious global health problem. Incidence is high in Bangladesh and mortality is identified to be one of the highest in the world. Most bites are occurred by non-poisonous snakes and as many as 40% bites inflicted by venomous snakes do not produce features of envenoming. They need supportive treatment only. Poisonous bites are treated with antivenin. But most people apply tight tourniquet and take useless and harmful treatment from traditional healers before getting admitted in hospital. Delay in diagnosis and treatment causes fatality in many cases. 46 patients admitted in department of Medicine, MMCH from April,07 to March,08 with snake bites were studied. Among them 35% were poisonous and 65% were nonpoisonous bites. Male and female ratio was 3.6:1. Mean age (years) was 34.9 ± 16.2 SD. Mean time of interval between bite and hospitalization (hours) was 7.8 ± 9.5 SD. 94% bites occurred in land and 7% in water. 100% patients applied multiple tourniquets in the affected limb. 24% patients received treatment from traditional healers with development of cellulitis in 64% of them. None received proper first aid management. Most incidences were in July – August. Snakes could not be identified in 50% poisonous and 77% non-poisonous cases. Among the identified poisonous snakes, kraits were 84%. Clinical features were also suggestive of krait bite in 88% poisonous cases. Among poisonous cases, 94% presented with neurological manifestation and ptosis was present in all of them. 75% poisonous snake-bite patients received antivenin and none of them developed anaphylaxis. Among 4 poisonous snake bite patients who did not receive antivenin, 2 survived. Mortality in poisonous cases was 44%. All of the non-poisonous cases improved with supportive treatment.

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Introduction:

Snake bite is one of the significant causes of global morbidity and mortality. It has been estimated that 5 million snake bite cases occur worldwide every year, causing about 100,000 deaths.[1]In Bangladesh adequate data is not available due to lack of systematic record keeping system and lack of information and awareness at community level. An epidemiological study estimated the incidence of snake bites in Bangladesh about 8000 per year with 22% mortality[2] which has been identified to be one of the highest in the world. Bangladesh supports approximately 80 species of snakes.[3-6] Among them only few are venomous. These are Cobra, Krait, Russel's viper, Saw scaled viper, green snakes, sea snakes. Most bites are occurred by non-poisonous snakes and as many as 40% bites inflicted by venomous snakes do not produce signs of envenoming. Bites usually result from an unfortunate accidental interaction between a snake and a human victim. It occurs mostly when the people are at work like cultivation, gardening, plantation, wood collection, watching the crops even during walking. However bites are fairly common when victims are at sleep. Snake bite is a horrifying experience for the victim. During the bite it is unlikely that people can identify the offending snake. They may think that every bite could result in fatality. Venomous snake bites can be presented with local or systemic features of envenoming—neurological, haematotoxicities, myotoxicities, organ failure and some nonspecific features. Frequently victims present with complication of treatment by traditional healers or self induced inappropriate application of tourniquet. As for centuries people are used to take treatment from traditional healers, ohzas who demonstrate a number of rituals which are useless and harmful. The mainstay of management is anti-snake venom which although

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effective, can cause anaphylaxis. So at primary level hospital, it is usually withheld despite indication for possible danger which is easy to manage with proper approach.[7,8] Only supportive treatment including tetanus prophylaxis and assurance is sufficient for non-poisonous bites. The interval between the bite and death is < 6 hours in most cases.[9] So delay in diagnosis and treatment causes fatality.

Materials and Methods:
This is an observational study done in the department of Medicine, Mymensingh Medical College Hospital from 1st April, 2007 to 31st March, 2008. Patients aged 13 years and above admitted with the suspicion of snake bite during the period were identified. Among them, those who saw snakes or in whom bite mark or scratch mark were present or those who developed features of envenomation were diagnosed as snake bite cases. Patients with local or systemic features of envenomation according to the 'National Guideline of Management of Snake Bite' were detected as poisonous snake bite cases and types of snakes were also suspected accordingly. 20 minutes whole blood coagulation test were done in all patients. Other investigations were done according to same guideline. Data were collected in a specific format and analyzed subsequently.

Results:
With suspicion of snake bite more than 250 patients were admitted during the study period. Only 46 (less than 18%) of them were identified as both poisonous and non-poisonous snake bite. Among them 16 (35%) were poisonous who had clinical features of envenomation and 30 (65%) were non-poisonous who did not have any feature of envenomation.

100% patients applied multiple tight tourniquets in the affected limb. Short term complication happened to none of them. 11 patients (24%) received treatment from traditional healers. Among them 7 developed cellulitis (64%). None of them received first aid management. 43 (94%) bites occurred in land, 16 were poisonous (37%) & 27 were nonpoisonous (64%). 3 (7%) were in water, all were nonpoisonous.

15 patients (94%) presented with neurological manifestation with ptosis in all of them (100%) and in 14 of them no other local or systemic features were present. 2 patients came with local features of envenomation, 1 associated with bleeding manifestation and the other with features of neurotoxicities.

12 patients (75%) received antivenin and none developed anaphylaxis due to antivenin.

All patients including non-poisonous bites received supportive treatment including tetanus prophylaxis.

Table-I

Characteristics of snake bite cases

Non-poisonous: Poisonous	1.9: 1
Male: Female	3.6: 1
Mean age (yrs)	34.9 ± 16.2 SD
Mean time of interval between bite & hospitalization (hrs)	7.8 ± 9.5 SD
Mortality in poisonous cases	44 % (approximately)

Table-II

Age distribution of snake bite patients (n=46)

Age group (yrs)	Poisonous (% -approximately)	Non-poisonous (%-approximately)	Total (%-approximately)
13-20	02 (04%)	09 (20%)	11 (24%)
21-30	04 (09%)	09 (20%)	13 (28%)
31-40	06 (13%)	02 (04%)	08 (17%)
41-50	02 (04%)	05 (11%)	07 (15%)
51-60	01 (02%)	02 (04%)	03 (07%)
>60	01 (02%)	03 (07%)	04 (09%)
Total	16 (35%)	30 (65%)	46 (100%)

Table-III

Gender variation in snake bite patients(n=46)

Sex	Poisonous (%-approximately)	Non-poisonous (%-approximately)	Total (%-approximately)
Male	12 (26%)	24 (52%)	36 (79%)
Female	04 (9%)	06 (13%)	10 (22%)
Total	16 (35%)	30 (65%)	46 (100%)

Table-IV

Time passed before admission of snake bite patients

Hour	Poisonous (n=16)		Non-poisonous (n=30)		Total (n=46) (%-approximately)
	Improved (%-approximately)	Died (%-approximately)	Improved (%-approximately)	Died (%-approximately)	
0-6	03 (19%)	01 (06%)	25 (83%)	00 (00%)	29 (63%)
>6-24	05 (31%)	06 (38%)	05 (17%)	00 (00%)	16 (35%)
>24	01 (06)	00 (00%)	00 (00%)	00 (00%)	01 (02%)
Total	09 (56%)	07 (44%)	30 (100%)	00 (00%)	46 (100%)

Table-V

Monthly variation in occurrence of snake bites (n=46)

Month	Poisonous (%-approximately)	Non poisonous (%-approximately)	Total (n=46) (%-approximately)
7-Apr	00 (00%)	03 (07%)	03 (07%)
7-May	00 (00%)	04 (09%)	04 (09%)
7-Jun	03 (07%)	01 (02%)	04 (09%)
7-Jul	03 (07%)	14 (30%)	17 (37%)
7-Aug	03 (07%)	05 (11%)	08 (17%)
7-Sep	02 (04%)	01 (02%)	03 (07%)
7-Oct	03 (07%)	01 (02%)	04 (09%)
7-Nov	01 (02%)	00 (00%)	01 (02%)
7-Dec	00 (00%)	00 (00%)	00 (00%)
8-Jan	00 (00%)	00 (00%)	00 (00%)
8-Feb	00 (00%)	00 (00%)	00 (00%)
8-Mar	01(02%)	01 (02%)	02 (04%)
Total	16 (35%)	30 (65%)	46 (100%)

Table-VI

Type of snakes identified by the victims

	Feature of envenomation (n=16)	No Feature of envenomation (n=30)
Krait	05 (31%)	00 (00%)
Cobra	01 (06%)	00 (00%)
Others	00 (00%)	07 (44%)
Unidentified	08 (50%)	23 (77%)
Not seen	02 (13%)	00 (00%)
Total	16 (100%)	30 (100%)

Table-VII

Clinical manifestations of poisonous snake bite cases (n=16)

Features	Died (% -approximately)	Improved (% -approximately)	Total (% -approximately)
Neurotoxic	07 (44%)	08 (50%)	15 (94%)
Haemotoxic	01 (06%)	00 (00%)	01 (06%)
Myotoxic	00 (00%)	00 (00%)	00 (00%)
Organ failure	03 (19%)	02 (13%)	05 (31%)
Non specific	04 (25%)	08 (50%)	12 (75%)
Local sign	01 (06%)	01 (06%)	02 (13%)
20 min WBCT positive	00 (00%)	01 (06%)	01 (06%)

Table-VIII

Treatment response with antivenin

Antivenin	Improved (% -approximately)	Died (%-approximately)
Given (n=12)	07 (58%)	05 (42%)
Not given (n=04)	02 (50%)	02 (50%)

Discussion:

Though many patients were admitted in hospital with suspicion of bitten by snakes, most of them were not at all cases of snake bite. Among the snake bite cases, most are nonpoisonous (65%) which is consistent with other studies [10] and need supportive and symptomatic treatment only. People of younger age group are affected in majority of cases. Males (79%) are mostly victimized. These are reflected in other studies also. Both are may be due to involvement of more outdoor activities of these groups. There was seasonal variation of the incidences of snake bites being mostly occurred in July-August and least incidences were in November-March which is almost similar to the result of study done in the northern area of Bangladesh.[10] This may be due to habitat of snake and environmental factors like rainy season, flood etc. In most cases snakes were seen but not identified which is not only true for our population but also for the Australian. [11] Probable cause may be, victims and

the bystanders remain in a panic state and may be most of the people have little knowledge about types of snakes. Studies about types of snake is inadequate but a survey of 10% of the country in 1988-9 reveals Cobra bites was 34% of all bites[12] and cobra had been found to be the commonest poisonous snake in Chittagong[13] also. In present study, among the 6 identified poisonous snakes, Kraits were the culprit in majority cases (83%) and clinical manifestations were also suggestive of krait bite in most cases (14 out of 16) reflecting the predominance of this species in this region. After the bites, all applied tight tourniquet to their limbs which were not done in appropriate method and may lead to serious complications to patients though fortunately none in this study population developed so. The incidence of taking useless and harmful treatment from traditional healers are not very much high in this study in comparison to other studies which is a good sign and reflects the awareness of people about it. But those who took it, most suffered from complication like cellulitis. The alarming fact is that none received any proper first aid management even in primary level hospital. All of the poisonous snake bite occurred in land and in case of bite in water, all were nonpoisonous, though the number of cases were too small to comment. Most of the poisonous cases (75%) came after 6 hours of bite and 38% died. Those who came within 6 hours (25 %) were mostly improved (19%). Only one patient whose features of envenomation appear after 24 hours and came after 2 days, survived without receiving antivenin. Analysis of features of envenomation shows neurotoxic features in 94% cases and ptosis was present in all of them which is higher than other studies.[10] And in 88% cases it was the only manifestation suggesting kraits bites and again indicating strongly high prevalence of kraits in this region. 2 patients presented with local features of envenomation, 1 associated with haematological features with positive 20 minute whole blood coagulation test indicating the presence of viper. Antivenin was given in 75% of patients and anaphylaxis was not developed in any case. 25% patients did not receive antivenin due to unavailability and unaffordibility. Surprisingly 2 of them (50%) survived which is yet to be explained. ICU facilities were utilized in 2 patients and 1 improved. All of the non-poisonous bite cases improved with supportive treatment. Mortality in this study is very high (44%) than other studies in Bangladesh[2,10] which may be due to delay in admission and initiation of treatment.

Conclusion:

Snake bites cases are still a serious health problem for us. Many patients are not aware of what to do instantly and not getting initial first aid management. They are spending valuable times before seeking treatment in hospitals and causing fatality. Though incidence of seeking treatment from traditional healers are declining, to adopt proper first aid management including application of tourniquet is still a problem. Serious adverse reaction to antivenin is not very common though the number of study population is too small to comment. Fear of giving antivenin should be alleviated. Larger and more studies are required for improving management of this important but neglected problem.

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Is Low Level of Serum Ionized Magnesium Responsible for Eclampsia?

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Summary:

Objective: *To determine the level of serum ionized magnesium in eclampsia patient before and 24 hours after giving the loading dose of magnesium sulfate and if low, to determine the relationship between levels of ionized magnesium in serum and occurrence of eclamptic convulsions.*

Methods: *Fifty eclampsia patients received a loading dose of 4 gm of magnesium sulfate, which was diluted with 12 ml of distilled water and then was given intravenously over a period of 10-15 minutes and it was followed by 3 gm of magnesium sulfate deep intramuscular injection in each buttock. Patient's venous blood samples were obtained before and 24hours after loading dose of magnesium sulfate and analyzed for ionized magnesium, sodium, potassium, and calcium level.*

Results: *Level in eclampsia women is 0.47 (0.15-1.04) m mol/L and 0.74 (0.20-2.00) m mol/L before and 24 hours after treatment with loading dose of magnesium sulfate, respectively .This change is significant.(P value is < 0.001) . Change in ionized magnesium level associated with change in diastolic and systolic blood pressure, mean arterial pressure and albuminuria .This changes are also significant (p<.001).*

Conclusions: *The evidence indicated that low level of ionized magnesium in serum may be the cause of eclamptic convulsion.*

Key word: *Ionized magnesium, Eclampsia, magnesium sulfate.*

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Introduction:

Eclampsia is one of the important cause of maternal and perinatal morbidity and mortality throughout the world. Though the cause of eclampsia is unknown but now a day's eclampsia is considered as a malnutrition related disease [1]. Eclampsia commonly seen in teenage pregnant woman who lives in slum area , devoid of both home care and antenatal care. In Bangladesh, eclampsia accounts five percent of total obstetric admission in our health facilities and sixteen percent of maternal death .[2]

Malnutrition is common in our country. Pregnancy imposes a great stress on the nutritional reserves. There is depletion of essential nutrients like Vitamin-B complexes, Vitamin-A, Folic acid, Iron and Calcium. Intake of this essential nutrient in poor income women is far below than recommended daily allowances. [3] Decreased protein intake causes decrease calcium absorption from the gut and stimulate parathyroid hormone secretion .Thereby enhance calcium reabsorption from bone and maintain calcium balance.[4] There is progressive decline in magnesium consumption from 475-500 mg/ day in 1900—1908 to 175-225 mg /day in 1990-2002 due to widespread consumption of processed foods and decreased consumption of fresh foods [5].Nuts and green leafy vegetables are good sources of magnesium.[5]

Eclampsia is primarily a convulsive state, despite extensive physiological, biochemical and anatomical changes that occur during pregnancy and may be local or systemic[6], serum electrolytes and ionized Ca^{2+} level remain normal [6-8] So present study has focused on ionized magnesium level . Magnesium is one of four cations that must be kept in balance in extra cellular fluid to regulate all body functions that require ATP. [9, 10]

In women with eclampsia, magnesium deficiency is suspected. Because during normal pregnancy plasma volume increases 50%, increased metabolic and endocrine activity of mother and fetus, estrogen and

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progesterone levels which increases as pregnancy advances [6,7] , elevates the body's demand for magnesium[11] and more and more magnesium are utilized and produce physiological hypomagnesaemia and symptoms of hypomagnesaemia. [4, 10-13] On the other hand, if pregnancy remain uncared, women have no ANC, decreased dietary supply of protein [3], vitamins and minerals ,preexisting protein energy malnutrition, low maternal age, .low income, low quality of life, these lead to severe micronutrient deficiency particularly magnesium deficiency that causes tissue hyperirritability and convulsion .[4,10-13]

Serum magnesium level is tightly regulated in a narrow range of approximately 0.7 to 1.0 mmol/l because only 1% of the total body content of magnesium is extra cellular.[9] When serum ionized magnesium level falls below 0.7 m mol/L are indicative of magnesium deficiency. But patient remain asymptomatic[9]. Symptoms appear when level falls below 0.5 m mol/L. This level indicate depleted total body magnesium stores because magnesium reside in side the cell, total body magnesium deficit is often~200 m mol(4800 mg) by the time serum level falls to<0.5m mol/L .[9] Magnesium is a natural calcium blocker [5]. Alteration of intra and extra cellular magnesium concentration may affect cell function through their effect on calcium handling. There is evidence that magnesium acts by opposing calcium dependent arterial vasoconstriction by antagonizing the increase in intracellular Ca^{+2} concentration caused by ischemia.[14]

Magnesium depletion causes impaired parathyroid hormone secretion and function [9, 14] and chronic magnesium deficiency causes damage to the cardio vascular, renal and neuromuscular tissue[10]. It enhance the contractility of several vascular beds in vitro, including coronary arteries, the mesenteric vasculature and umbilical vessels and may potentiate the contractile response to humoral agents such as nor adrenalin, prostaglandins and angiotensin II and leads to wide spread vasoconstriction [12,14]. Vasoconstriction causes increased resistance to blood flow and accounts for the development of arterial hypertension. Vasoconstriction induced ischemia may be sufficient enough to lower the epileptiform activity in affected region of brain [5, 15]

Methods:

This study is designed to estimate level of serum ionized Mg^{+2} in eclampsia women before and 24 hours after giving the loading of magnesium sulfate and to determine the relationship between low level of ionized magnesium and occurrence of eclamptic convulsion. This is a prospective study carried out in Dhaka Medical College Hospital, Dhaka, Bangladesh during the period of July 2003 to September 2004.The patients were selected randomly. All patients with a clinical diagnosis of eclampsia , not received magnesium sulfate before admission ,were eligible for the study irrespective of when or where fit had occurred, whether the baby was delivered or not. Follow up was done to the other ward, discharged from the hospital or until death (whichever came first).All patients received a loading dose of 4 gm of magnesium sulfate, which was diluted with 12 ml of distilled water and then was given intravenously over a period of 10-15 minutes and it was followed by 3 gm of magnesium sulfate deep intramuscular injection in each buttock. Patient was followed up at fifteen minutes interval ,points noted during follow up were respiratory rate more than 16 per minute, urine output more than 25ml per hour and presence of knee jerk and thereafter follow up was done 4 hourly. This dose schedule has been found out to be effective for Bangladeshi women in a well designed study.[2]

Two venous blood samples were taken before and 24 hours after giving the loading dose of $MgSO_4$. Two ml. of venous blood was taken with a plastic syringe from the antecubital vein. Blood was transferred to a test tube containing heparin for determination of serum electrolytes particularly ionized magnesium. The investigation was done at Biomedical Research Group, Bangladesh Institute of Research and Rehabilitation in Diabetes Endocrine& Metabolic Disorder**. NOVA-8** was used as analyzer instrument. Reference range of Serum magnesium 0.7-1mmol/L.[11] Ethical clearance was obtained from BCPS Ethical Review Committee for the study. Informed consent was obtained from each patient for publication and presentation.

Data Analysis

All collected data were compiled and appropriate statistical analysis were done using computer based

software (SPSS for windows, version 10.1). Results are expressed as median (Rang). For statistical comparison, non-parametric tests i.e. Wilcoxon Signed Ranks Test. Pearson's correlation were used. All tests were two sided, and statistical significance was inferred for P values less than 0.05.

Results:

For the purpose of this study, fifty eclampsia patients have been studied. Median age of the participant women is 21(17-34) years. Forty eight percent of them are less than 20 years old, forty two percent belong to age group 21-30 years and only 10 percent belong to age group 31-35 years. Seventy two percent of the patients are primi and remaining are 2nd, 3rd and 4th gravid. Seventy two percent of the women have no ante natal care. Only 28 percent of the women have regular ante natal care. Sixty eight percent of the patient developed ante partum eclampsia and sixteen percent women developed intrapartum eclampsia. Only fourteen percent women developed eclampsia before 34 weeks of gestation, thirty percent of the women developed eclampsia between 34-37 weeks of gestation and forty percent of women developed eclampsia after 37 weeks of gestation. Sixteen percent women developed postpartum eclampsia and all are term pregnancy. Ninety percent of women have monthly income less than 3500/taka .They (husband) are day labors. (Table I).

Table-I

Demographic characteristics of the patients in the study

Variables	Frequency
Age (mean)	21(17-34)yrs
Primi gravida	72 %
ANC- NO	72 %
Types of eclampsia: antepartum eclampsia	68 %
Intrapartum eclampsia	16%
Postpartum eclampsia	16 %
Duration of pregnancy	
< 34wks	14 %
34-37weeks	40%
> 37wks	16 %
Income (yearly)35000Taka	90%
Living condition (Slam dweller)	90%
Occupation (Day Labor & rick show puller)	90%

At the time of admission all patients had tachycardia, tachyponea, adequate urine output (100 ml/ 4 hour) and presence of knee jerk. Twenty percent of the women had severe SBP (more than 170 m mHg), twenty eight percent of the women had moderate SBP (150-170 m m Hg) and thirty two percent of the women had normal SBP(less than 140 m m Hg). Twenty four percent of the women had severe DBP (DBP more than 110 m m Hg), fifty four percent women had moderate DBP (100-110 m m Hg) and twenty two percent of women had normal DBP(less than 100m m Hg). Ten percent of women had no uinary albuminuria, twelve percent of women had one plus albuminuria, thirty eight percent of women had two plus albuminuria and forty percent of the women had three plus albuminuria.

Serum level in ecplampsia patients before giving $MgSO_4$ is 0.47(0.15-1.04) m m mol /L and 24 hours after giving the loading dose of $MgSO_4$ is 0.74(0.20-2.0) m mol/ L. After $MgSO_4$ infusion, level raises. This change is significant (P < .001). The changes in serum electrolytes level before and 24 hours after magnesium sulfate therapy are not significant (Table II). Change in ionized magnesium level associated with change in diastolic blood pressure, systolic blood pressure, mean arterial pressure and albuminuria. This changes are also significant (p<.001) (Table III).

Table-II

Serum electrolytes in eclamptic patient

Parameter	Before (mmol/L)	After (mmol/L)	P value
Mg++	0.47 (0.15-1.04)	0.74 (0.20-2.0)	0.001
Na+	143 (132-158)	141 (124-150)	0.320
K+	3.7 (1.7-8.3)	3.5 (1.9-8.2)	0.062
Ca++	0.75 (0.38-1.23)	0.73 (0.29-1.33)	0.948

In this study only four percent patients had previous history of pregnancy induced hypertension and eclampsia, fifty two percent of the patients had sudden onset of eclamptic convulsion, whereas thirty eight percent of the patients had premonitory symptoms and ten percent of the patients had pregnancy induced hypertension which progress to eclampsia.

Table-III

Change in Blood pressure and albuminuria in eclampsia patient

Parameter	Before	After	P value
Diastolic BP (mm Hg)	108 (70-130)	90 (70-120)	0.001
Systolic BP(mm Hg)	160 (110-220)	137 (120-190)	0.001
Mean Arterial Pressure (mm Hg)	123 (83-150)	106 (86-140)	0.001
Albuminuria (g/L)	2+(0-3)	1+ (0-3	0.001

Results are expressed as median (range). Unpaired t-test was done as a test of significance.

Discussion:

Availability and accessibility to antenatal care plays an important role in the prevention of eclampsia. There is evidence that, 90% eclampsia seen in poor, under privileged, malnourished, illiterate younger primi [3, 17 -19] who lived in urban slam or remote areas[21] devoid of health care facility.

Magnesium sulphate is an ideal anticonvulsant drug because it has rapid onset of action and it control convulsion effectively [6, 18, 19] .Though there are some risks of magnesium toxicity during treatment with $MgSO_4$, these can be overcome by close observation of the patient, and monitoring of some parameters and use of specific antidote injection calcium gluconate. Fortunately nobody developed such toxicity in this study.

In this study, serum ionized magnesium level in eclamptic patient before and after treatment with $MgSO_4$ are 0.47 (+ 0.18) m mol/L and 0.74 (+ 0.38 m mol /L), respectively. Abnormally low concentration of is found in the serum of the eclampsia patients and it is the possible independent determinant factor for developing eclampsia [4,10-13] in Bangladeshi women. As in the pathogenesis of eclampsia, ionized magnesium level is abnormally lowered, so the possible mechanisms of magnesium deficiency are:

Lack of ante partum care: Regarding ante partum care, seventy nine percent of women have no (39.6%) or irregular(39% of women have1-2 visits) antenatal care visit .This finding compatible with national statistics and other study [17,18,28] .Ante partum care identifies risk factors and complication and rectifies it. In our counrty, only 20% women received regular ANC and 40% have no ANC. Lack of dietary support & discriminatory food allocation [3,21- 23] causes malnutririon. Bangladeshi people are mainly rice eater. Protein and green leafy vegetable comprises only 6% and 3% of their diet respectively [23.] There was a strong association between primi gravida, low maternal age, low quality of life, illiteracy, poor ANC and eclampsia [3, 23-26]. There is a close relationship between protein intake and magnesium balance and malnutrition .Mother's nutrition begin from her intrauterine life. Malnutrition particularly chronic energy deficiency and anaemia, contribute to poor maternal health and adverse pregnancy outcome for both mother and her infant .Forty percent of the adolescent girls, forty six percent of the non pregnant and thirty nine percent of the pregnant women are anaemic. Ninety seven percent of the women are house wife. Thirty four percent of the women belong to 19-24 years age group and 27.7% women of 25-29 years age group having malnutrition. Ninety five percent house hold headed by male person and sixty percent house hold head never attend school[26]. Nausea, protracted vomiting during pregnancy, [6] Increased phosphorylation after 28 weeks of gestation both in mother and fetal cell [6] , all together causes magnesium deficiency .That is why seventy percent eclampsia seen in antepartum period.[7]

Mismanaged labor: In prolonged labor, patients have neither food nor fluid ,or hyper alimentation with magnesium or mineral free fluid [27]. Lack of nutritional support[4], increased metabolic and endocrine activity of stressful situation depletes body's magnesium stores[6] during labor. Anxiety, fear, and other emotional reaction may affect the rate and depth of respiration and consequently the CO_2 content of blood & acid-base balance [6]. These imbalance causes tissue irritability and convulsion [4, 10-13] in the absence of hypertension and albuminuria. Eighty five percent deliveries occur at home. Eighty

two percent of delivery is unattended or attended by unskilled person. Only 18%delivery attended by medically trained person. [28] Complication are more among attended labor and delivery. Findings of this study compatible with other studies.[18,19]

Mismanaged puerperium: Puerperium is a hypercatabolic condition. Lack of dietary support and fluid imbalance put the mother on risk of eclampsia .Only 21 percent women received postnatal care. 14.5 percent developed postpartum complication [28] .Extrinsic factors such as - Inadequate perinatal care, [28] illiteracy (47% women never attend school) and poor socio-economic condition [26] which affect outcome of pregnancy. Findings of the study have similarity with national statistics and other studies [18,19].

Low maternal age: Women at or below 20years, because of growth spurt and increased metabolic activity increases magnesium demand[6], In Bangladesh early marriage are common. Seventy two percent of 15-19 years girls are married and they contribute approximately 20% of total pregnancy [29]. They are physically and psychologically immature for reproduction. Forty six percent of the children under five years are under weight and 36.2% are stunted [28] Teen age (69.4% women became married before the age of 18 years) [26] pregnant women unable to bear the stress of pregnancy and labor. So pregnancy outcome among teen age mother are complicated .This findings are compatible with other studies [17,18].

In this study, eclamptic patient after receiving in hospital infused Hartsol solution and loading dose of $MgSO_4$. After stabilization, there was induction of labor with misoprostol tab, oxytocin drip or LSCS operation according to indication. Patients with high systolic BP > 180 mm Hg or Diastolic BP > 110 mm Hg, treated by giving $^1/_V$ inj. Hydralazine. In preterm patient injection Oradexon 2½ ampule $^1/_m$ for fetal lung maturity or injection Oradexon 1-2 ampule ^{1}/v in suspected cases of cerebral edema were given. Despite of these treatment serum electrolytes level before and after $MgSO_4$ infusion remain more or less same except significant change inlevel.

Dr. Monira Ahmed [18] Anwary S A[19] in their study showed that there is reduction in serum magnesium concentration in eclampsia patient before treatment with magnesium sulfate and level rises after therapy. They suggested that this lower level was partly due to hemodilution and partly due to hyper metabolic condition of pregnancy. But eclamptic patient had less intravascular volume [14,17] or they are volume depleted.

Hypertensive disorders of pregnancy, which account for approximately 15% of pregnancy related deaths [30], represent the 3rd leading cause of maternal morbidity and mortality in USA and in Bangladesh. The cause of eclampsia still remains elusive, but continued research provided hope with regard to screening, improved diagnosis and management. Treatment and management of the hypertensive disorder of pregnancy have not changed substantially in the past 50 years [30,31].

Eclampsia occur suddenly [15, 16, 31] . Worldwide interest is going in the prevention for eclampsia as a part of antenatal care or treatment strategies. Serum Electrolytes measurement is expensive, not easily available. So dietary correction, prepregnancy counselling , antenatal care and postnatal care can prevent the development of eclampsia[28].

The role of $MgSO_4$ in the treatment of eclampsia is clear because after administration of $MgSO_4$ pulse, Blood pressure, proteinuria, level of consciousness improved and no further convulsion occur. Role of in causation of eclampsia is not known but low level of is found in eclampsia patient compared to the same patients 24 hours after giving the loading dose of $MgSO_4$ .This study showing that the factors most predictive of eclampsia in pregnant women is possibly abnormal low concentration of in serum. On the basis of these findings predisposing factors, aetiopathogenesis and sign symptoms are also correlated.[16,18, 19]

Conclusion:

The present study shows that eclampsia is a malnutrition- poverty related disease and the association of low level of ionized magnesium with the occurrence of eclamptic fit. So, it is presumptive that abnormally low concentration of ionized magnesium in the serum is responsible for eclamptic convulsion.

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Laparoscopic Versus Open Appendicectomy – A Randomized Controlled Trial

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Summary:

Background: *The authors compare open and laparoscopic appendicectomy in a randomized fashion with an object to define benefit of laparoscopic procedure if any.*

Methods: *Patients of acute appendicitis were randomized to either laparoscopic(n=62) or open (n=58) appendicectomy . Operation time, per-operative findings, concomitant and or other pathological lesions, postoperative pain, rescue narcotic analgesia required, negative appendicectomy rate, hospital stay and complications were noted.*

Results: *No patient in the laparoscopic group required conversion to open. The mean operation time were 36.51±15.81 minutes and 31.62±19.61 minutes for the laparoscopic and open groups respectively (p=0.1368). But the operation time is low in LA group (mean 37.92±16.28 versus 62.55±20.04 minutes, p=.0080) when only high up retrocaecal types were considered. In the laparoscopic group 45 patients (72.58%) had acute appendicitis, 15 (24.19%) had other pathologies (appendix were histologically normal) and in 2(3.225%) appendix were normal. Post operative pain score was significantly low (p=0.037) in LA group after six hours but became insignificant after twelve hours (p=0.959) and twenty fours (p=0.114). The LA group required significantly less rescue narcotic analgesia (p=0.026). Hospital stay was significantly shorter in the laparoscopic group (29.935±8.995 versus 35.413±11.30 hours, p=0.0038). The wound infection rate is higher in open group (13.79% versus 6.45%)*

Patients who underwent LA have a shorter operation time in high-up retrocaecal type of appendicitis, significantly less pain and require less rescue narcotic analgesia in comparison to open operation.

Conclusions: *Laparoscopic method offers an excellent opportunity to detect concomitant other pathology and there by reduce incidence of missdiagnosis and negative appendicectomy rate. The authors consider LA to be the procedure of choice in patients with acute appendicitis.*

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Introduction:

Since its introduction by McBurney in 1894, appendectomy has been the treatment of choice for acute appendicitis[1]. Appendicitis is the most common intra-abdominal condition requiring emergency surgery, with a lifetime risk of 6%[2, 3]. For more than a century, open appendicectomy (OA) remained the gold standard for the treatment of acute appendicitis. Unfortunately the diagnosis of acute appendicitis is often difficult, mainly clinical and always challenging. An accepted negative appendicectomy rate for presumed appendicitis ranges from 15% to 20%, even higher in women of childbearing age (20% to 30%)[4,5]. Attempts were taken to reduce the negative exploration rate by lower abdominal CT scan and Ultrasonography (US) but result were frustrating. The highest positive predictive value for diagnosis of acute appendicitis by CT and US is 83.8% and 81.3% respectively[6]. Before the era of laparoscopic surgery single umbilical port diagnostic laparoscopy enabled to diagnose appendicitis as high as in 50% cases[7]. The advent of endoscopic surgery led to the idea of performing laparoscopic

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appendicectomy (LA). In 1983 Semm, a German Gynecologist performed the first LA[8]. More than 2 decades later, the benefits of LA are still controversial. Despite numerous case series and randomized clinical trials comparing LA versus OA, a consensus concerning the relative advantages of each procedure has not yet been reached[3,9,10,11]. The goal of the present study is to compare the effectiveness and or benefits of LA over OA based on a simple randomized clinical trial with the hypothesis that LA is beneficial.

Materials and Methods:

From July 2006 to June 2007, patients between 19 and 49 years diagnosed as acute appendicitis were offered entry into the study. The diagnosis of appendicitis was made if all six set criteria: history of right lower quadrant pain and or periumbilical pain migrating to the right lower quadrant, nausea and/or vomiting, temperature more than 38ºC and/or leukocytosis above 10,000 cells per cmm, right lower quadrant guarding and or tenderness on physical examination, lower abdominal US diagnostic to appendicitis or normal and normal kidney ureter bladder (KUB) region X-Ray were present. Patients with confirmed or suspected appenducular lump, ASA score III and IV, previous history of lower abdominal surgery was not included in the study. The qualifying patients were informed of the risk and benefits of each operation and asked to sign a detailed informed consent in "Bangla", approved by the institutional review board (IRB). All were informed that oral fluid will be started 6 hours after operation and if tolerated well will be followed by liquid and discharged from the hospital at or after 24 hours if not contraindicated. Baseline parameters were evaluated before randomization once the informed consent was signed. Patients were assigned randomly to receive either open or laparoscopic appendicectomy.

All operation were performed by first author experienced in open surgery (> 20 years) and advanced laparoscopic techniques (>7 years). Patients received 200 mg Ciprofloxacine every 12 hours and 500 mg Metronidazole intravenously from the time of diagnosis until oral fluid was well tolerated post-operatively. OA used a McBurney muscle-splitting incision 3-4cm, and extension as required in the right lower quadrant. The distal ileum was visualized to detect possible Meckel's diverticulitis. The skin incision was closed with 2-0 vicryl either intradermally or transdermal interrupted stitch.

LA was performed using 3 ports, 10-mm umbilical port initially used for telescope. One 5-mm ports inserted in the right lower quadrants and another 5-mm or 10 mm port in the midline suprapubically according to availability of 5-mm telescope. Telescope shifted to the suprapubic port. The foot end and right side of the OT table was elevated up to15^0. The abdominal cavity was explored to locate the appendix and rule out other possible diagnoses. Position of the appendix, adhesion if any was recorded. In case of adhesion adhesiolysis done using monopolar diathermy hook dissection by taking care to not to injure the gut or other structure. The base of the appendix was doubly ligated with no.1 silk by intracorporeal suture (Fig.2) after creating a window and divided in between. The mesoappendix was ligated by similar fashion or clipped and divided close to the appendix using monopolar diathermy (Fig.3**).** The right lower quadrant, the right colic gutter and the subhepatic space in the case of purulence were irrigated and the fluid was suctioned. The appendix was removed through umbilical port after taking in side the reducer sheath to avoid contamination or in a self made endo-bag- with sterile hand glove. Facial defects in the port sites were closed using 2-0 Vicryl suture. The skin incisions were closed in every case using 2-0 Vicryl.

In LA operation time was recorded from first port insertion to last port closure in minutes and in OA from skin incision to skin closure. 50 mg I.V. pethedine and 8mg Ondansetron (Onaseron Inj.) was used per-operatively in all cases. At the end of operation just after recovery 50 mg Diclofenac in the form of suppository was used and repeated 8 hourly for 48 hours.

Post-operatively pain was measured following Visual Analogue Scale (VAS) [12] every 6 hours and when it was >36mm rescue analgesia –in the form of pethedine-75 mg were administered intramuscularly and recorded. Bowel sounds were checked every 6 hours. Once present, the patients were started on a

clear liquid diet and advanced to regular diet when the liquid diet was tolerated and flatus observed. Patients were discharged when they tolerated a regular diet, had pain score <24 mm.

All resected specimen of appendix were submitted for histopathology. On discharge all the patients were advised to visit on 4th and 7th post-operative day if not indicated earlier due to pain, vomiting, fever or absolute constipation and any wound complication were recorded.

Statistical Methods

The sample size was calculated before the beginning of the trial based on an analysis of sample sizes required for each of the main parameters (pain score 40±15mm[11] and negative exploration rate 25%±5.6[4]) for 95% confidence interval ($\alpha = 0.05$) and a power of 80%. to ensure 20 % difference. A sample size of 5 6 was calculated to be sufficient to detect this difference.

An unpaired student's t test was used for parametric data. Fisher's exact test or the χ^2 test was used to compare 2 x 2 contingency tables. Analysis were performed using SPSS version 11.5, Chicago, Illinois, USA, Excel-analyze it 2007 and Graph pad Quick cal 2007.

Results:

There were 120 patients entered in to the study, 62 in LA group and 58 in OA group. Nine patients excluded from the study because of contraindication to creation of carbon dioxide pneumo-peritoneum. Of these 2 patients were ASA grade IV, 2 patients suffering from COPD, 2 patients with persistent hypertension (systolic > 200 mm Hg and diastolic > 110 mm Hg) even on antihypertensive treatment, 3 with previous lower abdominal surgery.

Base line characteristics of patients in both LA and OA group were similar except male female ratio, 19:43/32:26 (p= 0.0058) indicating female preponderance (Table I).The mean operative time in LA group was 36.51 minutes; for the OA group, 31.62 minutes (p=0.136) when

considered whole series and it is insignificant. But when the open and laparoscopic groups are divided into subsets based on appendix position, high up retrocecal (HUR) groups and analyzed separately, operation times detected significant (Table II). The HUR appendicitis in LA group required 37.92 minutes for the completion of surgery but it was 62.55 minutes in open group (p=0.008, highly significant). When HUR are excluded mean operation time is longer in LA group (35.57 minute versus 25.94 minutes, p=0.0083).

In the open group, 47 patients had acute appendicitis (81.03%), 9 (15.56%) had normal appendix and in 2(3.45%) other pathology were there, subsequent histopathology also revealed normal appendix. In the laparoscopic group, 45 patients had acute appendicitis (72.58%), 2 (3.23%) had normal appendix and in 15(24.19%) had different types of other pathology (Table II).

Post-operative pain analysis revealed that six hours after operation mean pain score is significantly higher in open group, 36.45±10.39 versus 32.77±8.72 (p=0.037). Required amount of rescue parenteral narcotic analgesia is also significantly higher, 28 versus 14(p=0.027) (Table II).

There were significant differences between the patients in the laparoscopic and open groups regarding time required to tolerate oral fluid (10.16±4.15 hs vs. 12.62±3.84 hs, p= 0.001) and normal diet (18.48±4.386 hs vs. 21.93±6.21hs, p= 0.00059) (Table II). The overall hospital stay was 29.94±8.99 hours(range, 24-72 hs) in the LA group and 35.41±11.3 hours (range 24-90 hs) in the total open group (p = 0.0039) (Table-II).

There were two intra-operative port site bleeding (right lower quadrant port) in the LA group, managed by diathermy coagulation and temporary all coat abdominal wall suture. In three cases in the LA group there was partial avulsion of parietal peritoneum due to use of 10 mm suprapubic port and forceful introduction. No active measures were taken and all recovered uneventfully. In one patient in the LA group there was per-operative bleeding due to loose ligature and ineffective diathermy coagulation of appendicular artery. The situation tackled effectively by intra-corporeal gauge compression followed by an application of clip. In another cases in OA group accidentally the appendicular artery was slipped and retracted, required wound extension to control the situation. In LA group one patient developed mild surgical emphysema, resolved spontaneously. Wound or port site infection is significantly higher in open group (p=0.019). Mortality rate was "0" in both groups. No patient in the LA group required conversion to open operation.

Table-I

Base line characteristics of laparoscopic and open appendicectomy group

	Laparoscopic	Open	p Value
No. of patients	62	58	
Mean age(yrs)	29.83±5.2(19-45)	31.05±6.157(19-49)	0.245(n.s.)†
Male:female	19:43	32:26:00	0.0058(s)‡
Mean BMI	23.27±1.85	23.92±1.66	0.045(n.s.)†
ASA class			
I	49	50	0.3(n.s.)‡
II	13	8	
No. attack			
Single	16	19	0.40(n.s.)‡
Multiple	46	39	
WBC preoperative(thousand/cmm)	11.13±0.65	11.18±0.7	0.67(n.s.)†

Values in the parentheses are range. ± indicates standard deviation; n.s., not significant, s. significant, † student t test, ‡, chi squre test.

Table II

Per-operative and post operative Clinical outcomes

	Assigned to LA	Assigned to OA	p
Mean operation time (min)			
Total series	36.51± 15.81(21-90)	31.62±19.61(17-107)	0.136(n.s)†
HUR type (no.13:9)	37.92±16.28(23-82)	62.55±20.04(42-107)	0.008 (s)†
Excluding HUR	35.57±14.28(21-85)	25.94±13.32(17-73)	0.0083(s)†
(n.49:49)			
Peroperative pathology			
Acute appendicitis	45	47	0.00421 (s)‡
Alternate pathology	14	2	
Normal appendix	3	9	
Histologically normal appendix	17(37.78%)	11(23.4%)	0.27391(n.s)‡
Post-operative pain score(mm,VAS)			
6 hours after operation			
12 hours after operatio	32.77±8.72	36.45±10.39	0.037 (s)†
18 hours after operation	17.59±6.88	17.62±8.68	0.96(n.s)†
4.45±6.09	2.72±6.	0.114(n.s)†	
Rescue narcotic analgesia required (no. of patient)	14	28	0.027(s)‡
Time to liquid(h)	10.16±4.15	12.62±3.84	0.001(s)†
Time to solid(h)	18.48±4.386	21.93±6.21	0.00059(s)†
Hospital stay	29.94±8.99	35.41±11.3	0.0039(s)†

Table-III

Alternate pathology detected peroperatively

Alternate pathology	Assigned to LA	Assigned to OA
Rt. Ovarian cyst	2	
PID	2	
Sulpingitis	3	
Adhesion	4	1
Ruptured ovarian follicle	1	
Ruptured luetine cyst	2	1

Table-IV

Complications

	Laparoscopic group (n=62)	Open group (n=58)	P
Port site bleeding	2	Not applicable	
Parietal peritoneal avulsion	3	not applicable	
Peroperative bleeding	1	1	
Port site infection	2	9	0.019(S)€
Port site erythema	2	5	
Subcutaneous Emphysema	1	not applicable	
Mortality	0	0	
Conversion to open	0	Not applicable	

S indicates significant, € Fisher's Exact test.

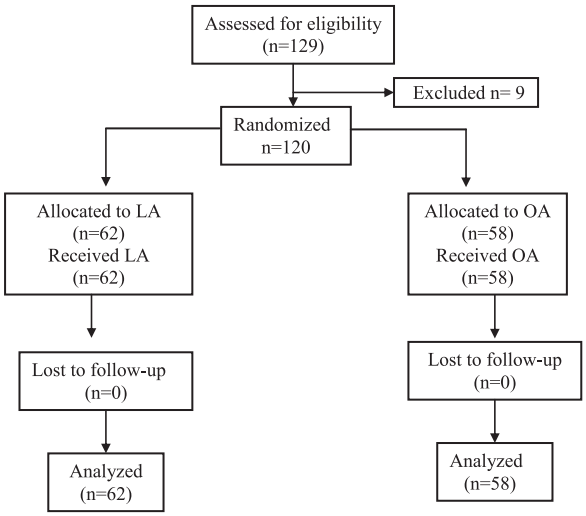

Fig.-1: *Flow diagram of trial participants*

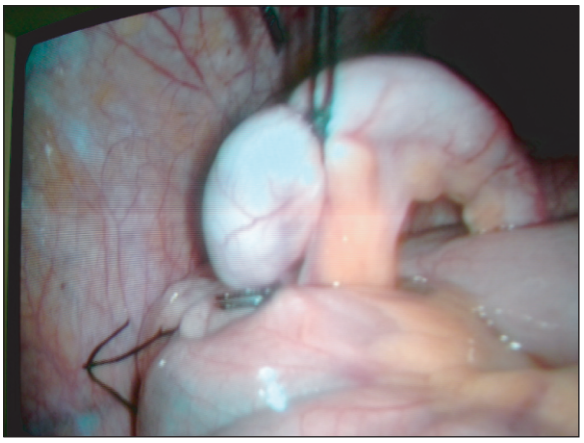

Fig.-2: *Shows intracorporeally ligated Appendix base*

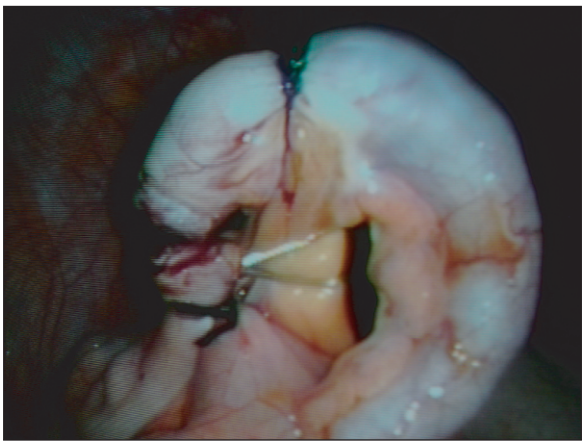

Fig-3: *Shows clipping of the mesoappendix*

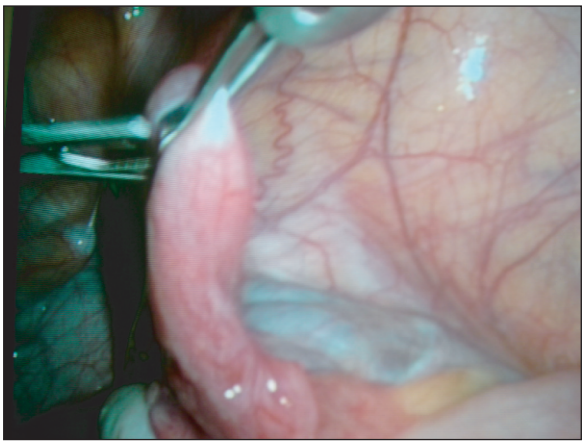

Fig-4: *Shows salphingitis as concomitant finding*

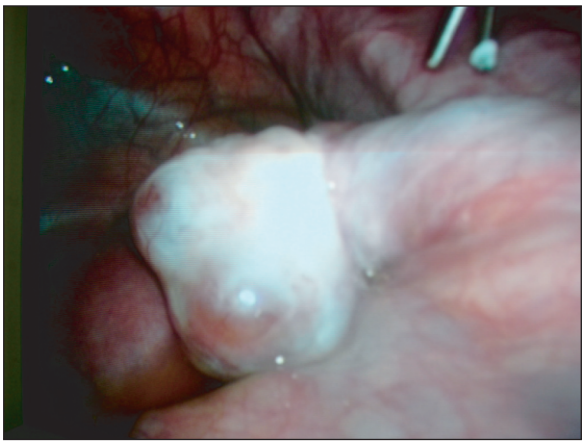

Fig-5: *Shows ovarian tumor as concomitant finding*

Discussion:

Laparoscopic Appendicectomy (LA) is relatively a new procedure as compared to laparoscopic Cholecystectomy (LC). A lot of analysis being performed through out the world regarding laparoscopic versus open appendectomy. Unlike LC, LA has not universally accepted as "Gold standard" because of controversy regarding exact benefit.

Despite the high success rate of conventional appendectomy, the most important drawback is negative appendicectomy rate, still in the range of20% to 30%[4, 13, 14]. The surgical technique for laparoscopic appendectomy is now well described, and several methods have been developed, involve single-port technique[15], two port[16] or standard 3- or 4-trocar technique. The base of the appendix can be divided by intra or extracorporeal suturing, endo-loop placement, clip application, stapling device or even without any clip or ligature by bipolar diathermy only[17].

On the basis of preliminary experience of 47 cases of LA as case series this prospective randomized controlled trial was undertaken to evaluate the time of operation, per-operative findings, concomitant and or other pathologies with negative appendicectomy rate, post operative pain, hospital stay, and incidence of complications.

Base line characteristics in both groups were same except (Table-I) predominant female sex in LA group. This is probably due to more consciousness of female patients regarding cosmatics and less pain explained during taking of informed consent. It may affect the per-operative findings.

The mean operation time in the laparoscopic group is longer (36.51minutes vs.31.62 minutes). But it is not statistically significant (p=0.136). This is relatively shorter (LA group) than the reported operative time of 40 to 50 minutes in maximum studies. In a Meta-analysis of 23 studies by Omer A et.al[18] mean operation time varies from 41to 72 minutes. However present finding is comparable to the result of Utpal D[19], found mean operative time of 28 minutes in laparoscopic group. In this study, operation time in laparoscopic and open group is almost same (36.52/31.62 min). In many studies during early nineties operation time in laparoscopic group was

longer, (61±4.8 minutes by van L et.al[4], 87 minutes by Richard C et.al[20])and also significantly more in comparison to open operation, (61 versus 46 minutes[4], 87 versus 65 minutes[20]). But in almost all recent studies the result failed to show a significant difference[18], even when it was performed by trainee surgeons (74±2.8 minutes /63±2.2 minutes in LA and OA group respectively[21]) like present study. This improvement is due to overcoming of learning curve and much technological advancement during last couple of years. In most of the series LA performed by a group of junior surgeons, but in present study the laparoscopic surgery was done by relatively experienced surgeon which might be the cause of relatively lower duration of operation.

When the open and laparoscopic groups are divided into subsets based on position of the appendix, the real advantage of laparoscopic group in terms of operation time become evident. The mean operation time for high up retrocecal (HUR) subset in open group is longer (62.55 minutes vs, 37.92 minutes; p= 0.0083). LA has the advantage to give clear and magnified visions of appendix with more space to maneuver irrespective of its position through a small hole. But in OA incision invariably needs to be extended, which is responsible for more operation time.

In LA group alternate pathology was found in 14 cases and in open group it was 2 (p= 0.00421). There were 3 normal appendix in LA group and 9 in open group. All 17 in laparoscopic group and all 9 in open group were histologically normal appendix. So the negative diagnosis is higher in LA group (37.78% versus 23.4%) though not significant statistically, p=0.27391. This is due to removal of normal appendix detected along with concomitant other pathologies. There is strong suspicion that undetected pathology along with normal appendix left out in OA group. Although the treatment of acute appendicitis is simple and straightforward, its diagnosis remains a challenge, and the negative appendectomy rate in large series ranges from 15% to 33%[6]. The incidence of perforated appendicitis in delayed cases is not less then 14%[22].The risk of two adverse outcomes, misdiagnosis and perforation of appendix must be balanced. Ultrasonography (US), CT scan and diagnostic laparoscopy as a method of investigation applied to reduce the incidence of negative or misdiagnosis. The positive predictive value for CT is 83.8%, and for US is 81.3%. The false-negative rates are 60% for CT and 76.1% for US[6]. In single port laparoscopy the appendix could only be visualized directly in as high as 10-34 % 0f cases[20]. The LA gives an opportunity to expose the whole abdomen without any extra effort facilitates alternate left out pathology, is the cause of high incidence of misdiagnosis in open appendicectomy. We performed appendicectomy in all cases. To do or not to do appendicectomy in case of detected alternate pathology or in normal appendix is a debate. Van LV et.al[4] suggested to do the appendicectomy if the detected pathology does not contraindicates appendicectomy. In another study by Steven LL et.al[6] appendicectomy were performed in all cases as incidental appendicectomy. In present study detected alternate pathology was managed laparoscopically either by the operating surgeon or with the help of gynaecological surgeons as demanded.

Post-operative pain 6 hours after operation is significantly low in laparoscopic group (32.77±8.72mm versus 36.45±10.39, p=0.037) and rescue narcotic analgesia required in open group is significantly high (in 28 cases versus 14 cases, p= 0.027) .But after 6 hours pain score is same in both groups. During open operation muscle splitting is responsible for more pain. The present result is comparable to the result of Van LV et.al[4], though their result was marginally insignificant (p=0.06) and study by Mustafa K[23]. A prospective randomized trial of 75 by Richard CF et.al[20] detected shorter duration of parenteral and oral analgesic use in laparoscopic group ($p<0.05$). But Namir K et.al[24] detected no difference of pain score between open and laparoscopic group even in early post-operative period.

Time to both liquid and solid is significantly lower in laparoscopic group (Table-III) are consistent with many studies[4, 5,14,20,24]. In laparoscopic surgery gut are not exposed to the external environment, there are minimum handling, are the cause of minimum impairment of gut function.

Hospital stay is significantly low in LA group (29.94±8.99 hs versus 35.41±11.3, p=0.0039). The

length of hospital stay in present study is short, is similar to many others studies[10, 18, 23, 24]. Few publications, particularly in the early nineties demonstrated hospital stay > 2 days[4, 20]. Perhaps this is one area where OA has caught up with the laparoscopic techniques. Duration of hospitalization from time of operation to discharge of patient was calculated. Prolonged hospitalization is an important factor and peoples[25] tried to evaluate the factors responsible for prolonged hospitalization after LA which are, nausea and vomiting, leukocytosis, gangrenous or perforated appendicitis and appendix position. Peroperative ondenosation was found as antiemitic routinely in all cases and only few number of patient required narcotic analgesia post-operatively may be the cause of very short hospital stay.

Overall complication and "0" mortality shown in Table IV are comparable to many studies[4,14,19,20,24]. Significantly low incidence of wound infection in LA (p= 0.019) is one the most important point in favor of LA. In LA appendix always removed in canula sheath or endo-bag. There is no question of contamination of wound. But in OA what ever may be the level of care always there is chance of wound contamination. A study of 175 LA by et.al[14] is comparable, though the study showed increased incidence of intra-abdominal abscess in laparoscopic group (LA 1.8% , OA.0.61%). But the complication was limited to gangrenous or perforated appendicitis and probably was related to vigorous irrigation of the peritoneal cavity.

In present study no patient in the LA group required conversion to open. It is consistent with very low conversion rate ranging from 0.6- 2% even some times in a hand of trainee's[26, 27, 28]. There were high conversion rate (2- 12%) in study done during early nineties[4, 20, 29]. In comparison to laparoscopic Cholecystectomy LA gained its acceptability very slowly due to many similar results both in open and laparoscopic technique. In many series operation were performed by junior surgical team may be cause of high conversion rate. The"0" mortality of the study consistent with many studies[4, 5,20,21,24].

Present study has some limitations. Cost and quality of life after operation were not analysed. Follow-up was limited to the first 1 week postoperatively. The aim was to detect operation time, pain after operation, concomitant findings and or negative exploration, early postoperative complications after hospital discharge.

Conclusion:

The study has clearly demonstrated that the operation time in laparoscopic group is though insignificantly high but it is significantly low when high-up retrocaecal group are analyzed separately. The laparoscopic technique provide an opportunity to detect concomitant pathology in the lower abdomen easily without any extra effort and thereby reduces real negative appendicectomy rate.

Pain score is significantly low in LA group during 6 hours after operation, and required dose of narcotic analgesia is more in OA group though it becomes similar after 6 hours. Laparoscopic group tolerated oral fluid and diet early and hospital stay is significantly low in LA group.

This study confirmed the benefits of laparoscopic appendicectomy over open operation. So it is concluded that laparoscopic appendicectomy should the procedure of choice.

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Juvenile Idiopathic Arthritis: Mast Common Rheumatic Disorder in Children- An Overview

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Summary:

Juvenile Idiopathic Arthritis (JIA) is the most common form of chronic arthritis in children and an important cause of both the short term and long term morbidities. JIA is the new terminology proposed by the International League of Associations of Rheumatologists (ILAR). Three separate systems are used currently to classify chronic arthritis in children. These are American College of Rheumatology (ACR) classification, The European League against Rheumatism (EULAR) classification and ILAR classification. The diagnosis of JIA remains a clinical one, and is essentially one of exclusion in addition to suspicion and recognition of patterns. There is no single test for the diagnosis of JIA. The treatment of JIA is rapidly changing. Aims of good management of JIA include: controlling pain and inflammation, preserving function and promoting normal growth and development. Remarkable advances have been made in the management of JIA with the advent of new modalities of treatment. Effective management of JIA needs a multidisciplinary team approach. Even after effective management, about one-third of JIA patients continue to manifest their disease activity into adulthood with serious morbidity and disabilities.

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Introduction:

Juvenile idiopathic arthritis (JIA) is an umbrella term referring to a group of disorders characterized by chronic arthritis[1]. JIA is the most common chronic rheumatic illness in children and is a significant cause of both the short and long term morbidity and disability[1,2]. Arthritis may be present in children in a number of conditions including infection, systemic diseases, and malignancies and as a part of auto-immune disease. But the prototype of childhood arthritis is JIA.

JIA is a clinical diagnosis made in a child less than 16 years of age with arthritis (defined as swelling or limitation of motion of the joint accompanied by heat, pain or redness) for at least 6 weeks duration with other identifiable causes of arthritis excluded[1].

The prevalence of JIA ranges from 8 to 150 per 100,000 children with an annual incidence of 1 to 22 per 100,000 [3,4]. Many factors contribute to the discrepancies between reported prevalence and incidence of JIA. Studies based truly in the community reported the highest prevalence and incidence[5].

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Twice as many girls as boys develop JIA. Among children with poly-articular onset, girls outnumber boys by a ratio of about 3:1. In striking contrast, systemic onset occurs with equal frequency in boys and girls. No race or geographical region is immune to JIA.

Classification of JIA:

The classification of juvenile arthritis is an evolving process which has not yet achieved its ultimate goal. The ultimate goal of classification is delineation of biologically distinct disease groups with prediction of outcome and responses to treatment[2]. It has been problematic for decades. The heterogeneity of these diseases was discussed by Diamont-Berger and Still in 1891 and 1896[6]. They recognised that many children with chronic arthritis had a disease that was unlike adult rheumatoid arthritis. However, subsequent workers have differed as to whether childhood arthritis should be grouped with adult rheumatoid arthritis or with spondylo-arthropathies.

In the 1970s, two sets of criteria were proposed to classify chronic arthritis in childhood:

1. Developed and tested by a committee of American College of Rheumatology (ACR) and the definitive Juvenile rheumatoid arthritis (JRA) criteria were published[7].
2. European League Against Rheumatism (EULAR)

proposed Juvenile chronic arthritis (JCA) and its criteria for diagnosis[8].

In the 1990s a third classification had been proposed by the paediatric task force of the International League of Associations for Rheumatology (ILAR)[9] . The ILAR classification and its revision were proposed by an international group of paediatric rheumatologists in Santiago and Durban with the aim of achieving as much homogeneity within categories as possible[2,10]. This was necessary because the terms JCA and JRA were not inter-changeable as their subgroups were different[2]. None of the classification system is perfect: some patients fulfill criteria for more than one subtype, whereas others are difficult to classify into any specific subgroups. In the ILAR system, these patients are classified as "other"[1]. All of these three schemata are shown in Table I and II.

Table-I

Summary of classification of chronic arthritis in children[1]

ACR (1977) JRA	EULAR (1978) JCA	ILAR (1997) JIA
Systemic	Systemic	Systemic
Polyarticular	Polyarticular JRA	Polyarticular RF negative Polyarticualr RF positive
Pauciarticular	Pauciarticular	Oligoarticular persistent extended
	Juvenile psoriatic	Psoriatic arthritis
	Juvenile ankylosing spondylitis	Enthesitis-related arthritis
		Others

Table-II

Summary of the differences among the three classification systems[1]

	ACR	EULAR	ILAR
Onset types	3	6	7
Age of onset	<16 years	<16 years	<16 years
Duration of arthritis	>6 weeks	>3 months	>6 weeks
Includes JAS, JpSA	No	Yes	Yes
Includes IBD	No	Yes	Yes
Includes course	No	No	Yes

Aetio-pathogenesis:

Although the causes of JIA still remain unclear, it seems to have a complex genetic background involving the effects of multiple genes related to immunity and inflammation[11]. Some hypotheses are there like, arthritis may be triggered by psychological stress, abnormal hormone levels, trauma or infection is a genetically pre-disposed individual. Certain HLA class I and class II alleles are associated with an increased risk of JIA[12]. Other genes conferring risk include cytokine production-regulating genes.

There is evidence of immune- dysregulation in JIA. Complement activation and consumption promote inflammation. Increasing serum levels of immune-complexes are found with active disease. Anti-nuclear antibodies (ANA) are found in approximately 40% of patients with JIA[13]. Approximately 5% to 10% patients are Rheumatoid factor (RF) positive[11]

The T lymphocyte-mediated immune response is involved in chronic inflammation and these are the pre-dominant mononuclear cells in the synovial fluid. Elevated serum levels of IL-6 IL 2R and soluble tumour necrosis factor (TNF) receptor correlate with inflammatory parameters in JIA patients[14]. Earliest change is swelling and congestion of the synovial membrane and the underlying connective tissues, which become infiltrated with lymphocytes, plasma cells and macrophages[15]. The synovitis is characterized by villous hypertrophy and hyperplasia with hyperaemia and oedema of sub-synovial tissue. Pannus formation occurs in advanced or uncontrolled diseases and result in progressive erosion of articular cartilage and adjacent bones[16]. Later on fibrosis or bony ankylosis may occur.

Clinical Presentation:

The ILAR classification of JIA includes seven subtypes. In order of frequency, the disease subtypes are oligoarticular JIA (50-60%), Polyarticular JIA (30-35%), systemic onset (10%-20%), psoriatic arthritis (2-15%) and Enthesitis related arthritis (1-7%)[1]. The subtypes are classified depending on the clinical features during the first 6 months of disease. Important clinical features other than arthritis include: presence of enthesitis, dactylitis, inflammatory lumbo-sacral pain, sacroilitis, psoriasis, nail pitting, fever, rash and serositis[1].

Oligoarticular JIA (OJIA)

It is diagnosed in patients with arthritis in fewer than five joints during the first 6 months of disease.

Usually there is involvement of large joints of the lower limbs such as knees and ankles. Oligo-articular patients, especially ANA positive girls, are at higher risk of developing uveitis, which is usually their most serious problem[1].

Arthritis that remains confined to four or less joints is designated as persistent oligo- articular JIA. A child who develops active arthritis of five or more joints after the first 6 months of disease is considered to have extended oligo articualr JIA. Extended disease confers a worse prognosis[17].

Polyarticular JIA

Patients with arthritis of five or more joints in the first six months of disease are diagnosed as polyarticular JIA. This subtype again includes children with RF negative disease (20%-30% of JIA patients) and RF positive disease (5%-10% of JIA patients)[11]. Common age of onset in this category is one to five years. Older teenage girls with polyarticular diseases often have a positive rheumatoid factor[18]. In polyarticular disease, usually small joints of the hands are involved symmetrically and large joints of both upper and lower limbs may also be affected. Chronic uveitis develops less frequently than in oligo articular disease.

Systemic onset JIA (SOJIA)

SOJIA is the only subtype of JIA without a strong age, gender or HLA association[1]. At onset extra articular manifestations including rash, fever, lymphadenopathy, hepato- splenomegaly and serositis predominate. The diagnosis remains a challenge in the absence of arthritis which may evolve over time. About 10 percent patients may not develop arthritis for many months. Children with SOJIA typically have 2 weeks of high-spiking fever, classically with two peaks daily (double quotidian). During episodes of fever, chills are common, and the child appears very toxic, but when a febrile, child appears well.

With the characteristic quotidian fever with an evanescent rash and other extra articular manifestations, diagnosis of probable SOJIA may be made, with confirmation of the diagnosis when persistent arthritis develops[19]. The arthritis associated with systemic onset JIA is usually polyarticular, affecting both small and large joints. Asymmetric, oligoarthritis is less common.

Enthesitis related arthritis (ERA)

Enthesitis related arthritis is much more common in boys than in girls. It is most common in boys older than 8 years of age[20]. Patient with juvenile ankylosing spondylitis and arthritis associated with inflammatory bowel diseases are included in the ERA subtype. The hallmarks of the disease are pain, stiffness and eventual loss of mobility of the back. Peripheral arthritis usually affecting few joints of the lower extremity precedes axial involvement and arthritis of the sacro-iliac joints may take years to develop.

Extra-articular manifestations include anterior uveitis, aortic insufficiency, aortitis, muscle weakness and low grade fever. Acute uveitis is common, often unilateral and recurrent. It may present as a red, photophobic eye[1].

Psoriatic Arthritis

Juvenile psoriatic arthritis is sometimes quite difficult to diagnose. The pattern of arthritis may be variable: asymmetric large joint involvement and the small joints of the hand and feet[21]. Interphalyngeal joints and the tendon sheath are often inflamed, resulting in the diffuse swelling of the digit known as ‘‘sausage digit”. Arthritis may develop many years before the skin rash. Other than rash, extra articular manifestations include nail changes (pitting, onycholysis, and oil-drop sign) and anterior uveitis.

Diagnosis of JIA:

The diagnosis of JIA is a clinical one made after identifiable causes of arthritis are excluded by a careful history and examinations along with appropriate radiographs and laboratory tests[1]. Important clinical features like systemic illness, preceding infection, duration of fever, rash, bleeding, injury and character of the arthritis help to differentiate JIA from other causes of arthritis[1]. The differential diagnosis of arthritis includes: reactive arthritis, inflammatory diseases, septic arthritis, acute rheumatic fever, multi-system diseases like SLE, malignancy and trauma. A number of laboratory tests and imaging studies are required to exclude all the differential diagnosis and to confirm the diagnosis of JIA[6].

Laboratory findings:
Complete blood count is by far the most important investigation, which classically shows: lower haemoglobin, neutrophillic leukocytosis, thrombocytosis and high ESR[16]. ESR and C - reactive protein (CRP) are always high in children with SOJIA and polyarticular disease, but is often normal in oligo arthritis and ERA[1,6].

Urine analysis should be done to exclude the possibilities of infection and SLE. Antinuclear antibody (ANA) is found in approximately 40% of all children with oligo articular or polyarticular JIA[13]. But this is always negative in systemic onset diseases. Rheumatoid factor (RF) is found in 5% to 8% cases of polyarticular JIA especially in older girls. RF positivity is usually associated with poor overall prognosis and eventual functional disability[6]. Anti-cyclic citrullinated peptide (Anti-CCP) antibody is a good serological marker for early rheumatoid arthritis which is highly specific for the disease[22].

Imaging studies:
Radiographs of the affected joints give information about soft tissue swelling, decreased bone density, joint space narrowing, joint erosion, deformity and fracture[15]. Ultrasonography is often the best way of identifying intra articular fluid, particularly in joints such as shoulder and hip, where it is difficult to identify clinically[6]. Magnetic resonance imaging (MRI) provides very detailed and sensitive information of both structure and physiology of cartilage, bone and other loco-motor tissue[16].

Management of JIA
Management of JIA is rapidly changing as the need for more effective treatment is regularly documented by different studies[23]. Objectives of the management of JIA are:

- Controlling pain and inflammation
- Preserving function
- Promoting normal growth
- Overall development and well being.

There are no therapies till date that have been demonstrated to achieve these results consistently. Treatment of JIA is even more challenging as because the aetiology of JIA is unknown, and the mechanisms of action of commonly used drugs are not clearly known. During past decades, a major transformation had occurred in the treatment of rheumatoid arthritis in terms of approach, termed the therapeutic pyramid, where conservative management was done with non-steroidal anti-inflammatory drugs (NSAIDs) for several years; disease modifying anti- rheumatic drugs (DMARDs) were withheld until clear evidence of erosions was seen[24]. This form of treatment had been replaced by early initiation of DMARDs and combination DMARDs therapy in patients with the potential for progressive disease. The idea of early intervention with DMARDs had been validated by several randomized trials[25, 26].

This paradigm shift is the result of unsatisfactory outcomes with the pyramid approach, and an increased awareness of the cost, lost productivity, morbidity and decreased life expectancy associated with JIA[24].

Non steroidal anti-inflammatory drugs (NSAIDs):
First-line therapy of JIA includes NSAIDs. In addition, long-acting intra-articular corticosteroid injections are safe and effective and may have beneficial effects on growth as well[27]. NSAIDs control pain and inflammation and are usually given to all types of JIA for 4 to 8 weeks before starting treatment with a DMARD (Fig-1).

Commonly used NSAIDS are aspirin, naproxen, ibuprofen, diclofenac and indomethacin[18]. Till now, there is no clear-cut consensus on the optimal NSAIDs for patients with JIA. Many clinicians choose NSAIDs on the basis of considerations, such as dosing schedule, patient preference or medication taste[27]. But most physicians use naproxen as a first choice in the majority of cases[16].

Disease-Modifying Anti-rheumatic Drugs (DMARDs):
The term "Disease-modifying antirheumatic drugs" (DMARDs) is limited to agents that retard radiologic progression of the disease[27]. These drugs include methotrexate, hydroxychoroquine, sulphasalazine, gold salts, leflunamide, cyclosporine, cyclophosphamide and azothioprine[28].

Methotrexate (MTX)
The introduction of MTX few decades ago redefined the treatment algorithm for JIA and MTX became the

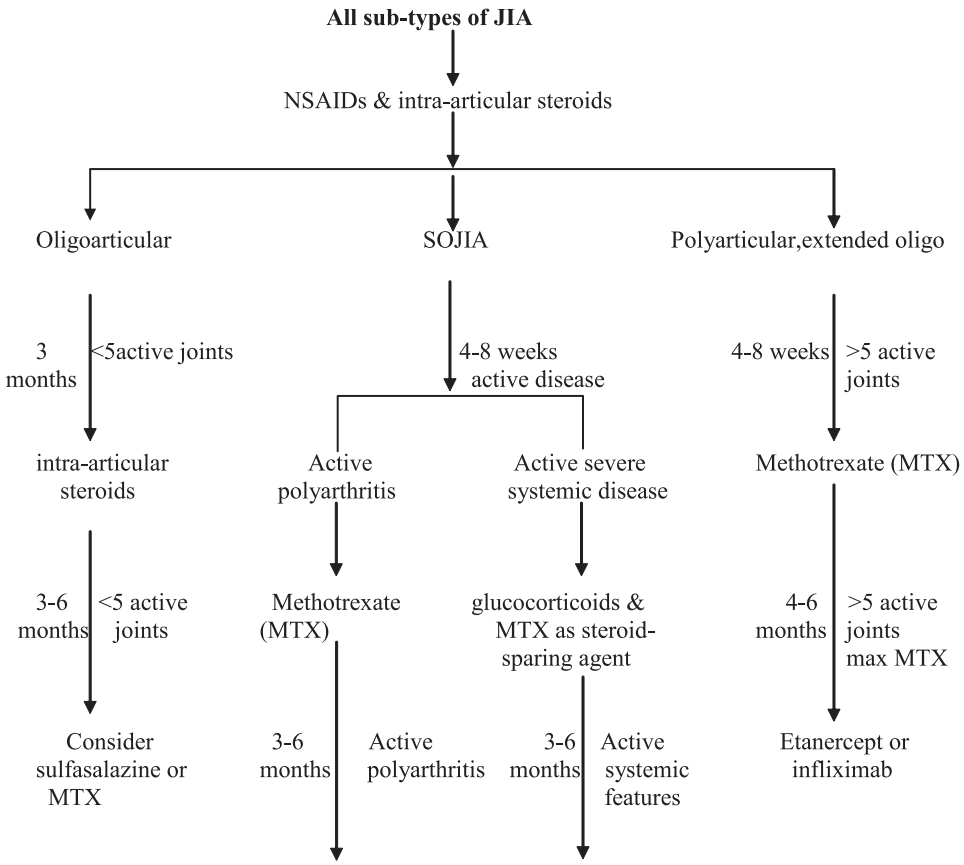

Fig.-1: *Suggested treatment algorithm for JIA1*

gold standard of therapy[28,29]. Methotrexate has proven to be an effective, safe and reliable option for treatment in all forms of JIA[28-31]. It has a major advantage that it can be administered as once weekly dose. Single weekly doses of MTX found to be effective in children with 0.3 – 1.0 mg/Kg/week (10-30 mg/m^2). These doses are much higher than weekly doses usually given to adult patients[23]. Sub-cutaneous use of MTX increases the bioavailability and efficacy. Supplementation with folic acid lessens the gastrointestinal and muco-cutaneous side effects without altering the therapeutic effect of MTX[32].

Liver enzymes and a complete blood count should be monitored every 1 to 2 months, although serious, irreversible liver disease is rare in children[1]. There is no doubt that MTX is currently the most useful drug for the treatment of JIA. But there is nothing to guide the clinical decision making regarding the duration of MTX treatment after remission is achieved[33]. Some authors did not find any influence on remission after prolonged MTX treatment[34].

Sulfasalazine

Sulfasalazine is an effective drug suppressing the disease activity of JIA patients[23]. However, drug toxicity is a problem. Headache, rash, elevated liver enzymes, leucopenia, hypoimmuno-globulinaemia and gastrointestinal problems are common side effects of Sulfasalazine.

Leflunomide

Leflunomide, an immunosuppressive agent, is approved for the treatment of adult rheumatoid arthritis and is currently being studied for use in JIA. Preliminary published results show that its efficacy is similar to that of MTX[34].

Corticosteroids

Intra-articular therapy: Intra-articular injection of corticosteroids in the treatment of JIA is well-established for mono or oligoarthritis, or alternatively as an adjunct in treating polyarticular disease[28]. Intra-articular therapy can effectively treat joint inflammation locally, for long periods of time, with excellent and rapid resolution of synovitis.

Parenteral corticosteroids: Parenteral high-dose corticosteroids used intermittently in 'pulse fashion' is a useful and very effective adjunct to therapy in SOJIA or severe polyarticular JIA[35]. It is also thought to minimize the cumulative steroid toxicity of continuous daily oral steroids.

Oral corticosteroids: The general approach to oral corticosteroid use is to avoid them if possible, and if required, to use the minimum dose. Commonest use of oral corticosteroid is while awaiting the desired effect of DMARDs therapy, and once effective, steroids should be weaned rapidly[28].

Biological agents:

The biologics (eternacept, infliximab, adalimumab, anakinra, abatacept and rituximab) have been demonstrated to be effective in treating inflammatory arthritis[23]. Their use in children poses special problems, including the increased risk of infections, possibilities of later malignancies or possible development of de-myelinating disease. The cases of re-activated tuberculosis have been particularly difficult.

An important issue with anti-TNF (Etanercept, infliximab and Adalimumab), anti IL-I (Anakinra) and the B-cell depletor (rituximab) is how and when to discontinue these powerful and effective treatments.

Autologous stem cell transplantation:

A number of studies have reported the use of autologous stem cell transplantation in very severe forms of JIA, resistant to all forms of treatment[36]. Encouraging results, including complete and long lasting remission induction have been reported.

Other issues in the management:

At present, remarkable advances in the treatment of JIA have been made with the advent of new DMARDs, and biologic therapy. Physical and occupational therapies are important adjuncts to medication because they help to maintain and improve range of motions, muscle strengths, and skills for activities of daily living. Splints may be used to prevent contractures or work to improve range of motion. Arthroplasty might be needed for patients with severe deformities[1]. So, effective management of JIA requires a multidisciplinary team approach.

Nutritional impairment is common in children with JIA. Growth may be affected by decreased total calorie intake, by active disease itself or by medication side-effects[28]. Localized disturbances in growth, such as leg-length discrepancy or jaw growth abnormalities may also occur. Delayed puberty is quite common in JIA patients. Attention to growth parameters including pubertal status is important.

Children with JIA are at increased risk for osteopenia and osteoporosis[1]. Low bone mineral density (BMD) has been associated with severe disease; younger age, lower body mass index, and lean body mass, decreased intake of calcium and vitamin D, and decreased physical activities. Appropriate calorie and calcium intake along with physical activities should be encouraged[1,28].

Uveitis remains as an important complication in some JIA patients. Regular screening of all children is needed for early detection and management of uveitis and prevention of blindness[28]. Attention to the psychological well-being of the patient with JIA is essential in the setting of this chronic painful and disabling condition. Early discussion with the patient when they reach early adolescence, along with their family is very important regarding the process of transition to adult health care services[37].

Counseling the family is most important for effective management of this chronic illness. The parents and if appropriate the child must be educated about the present state of knowledge of JIA, its outcome, and therapy. An optimistic attitude must be maintained[6].

Monitoring of progress:

Disease progress should be determined by a range of factors or outcome measures. These are critical objective parameters both for therapeutic trials and

for day-to-day practice to judge whether or not the patient has improved. These variables are now used in a combined fashion as a standardized outcome measures[38].

Outcome of JIA:

Traditionally, the teaching regarding the prognosis of JIA was over optimistic, such that most children 'grow out of it'. Realistically, and depending on types, JIA is not a benign disease. Once remission has been achieved, nearly 50% of the patients may have relapse at any time. Nearly one-third of patients have their disease activity into adulthood. Among them many live with considerable limitations of daily activities[28]. In general longer disease duration, poly-articular disease and systemic onset JIA have worst prognosis. Oligo-articular JIA is known to be as the most benign[1]. The mortality rate based on reports from the United States and Canada is reported as 0.29/100 patients. Most deaths occurred in patients with Systemic onset JIA[39].

Conclusion:

In spite of new insights into aetiology and considerable advances in the management, JIA remains an important cause of chronic pain and disability in children. Recognition of the need to treat this disease early, effectively and aggressively have resulted in increasingly better disease control and achievement of inactive disease in greater number of children. It is expected that these approaches will result in better quality of life allowing children with JIA to become adults leading normal or near normal lives.

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Small Bowel Phytobezoar Obstruction in a Post Gastrojejunostomy Patient – A Case Report

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Summary:

Small bowel phytobezoars are, though well-known, a rare cause of mechanical intestinal obstruction. It occurs mainly in the patients who had a previous gastric operation. We present a case of phytobezoar obstruction in a patient, who underwent gastrojejunostomy operation. We also report here a rare radiological finding of phytobezoar obstruction. The patient was treated surgically and had an uneventful recovery.

(J Bangladesh Coll Phys Surg 2009; 27: 99-102)

Introduction:

In hospital admission, small bowel obstruction accounts for 20% of the patients. The common causes are adhesions, strangulated hernia, malignancy, volvulus and inflammatory bowel disease. Phytobezoars are rare, accounting for only 0.4-4% of all intestinal obstruction[1]. Phytobezoars are concretion of poorly digested fruit and vegetable fibres that are found in the alimentary tract, particularly orange pith or pulp in patients with history of surgery[2]. Bezoars are long been a source of gastrointestinal problems. Classically, bezoars were first described as a cause of gastric bleeding, ulceration, perforation and peritonitis[3], but intestinal obstruction was not a frequent occurrence. However, since 1961, when Norberg first reported obstruction of small intestine by a bezoar in a patient in whom gastric surgery has been carried out, more and more cases have been reported[4]. Our experience, reported in the following section, is representative of this entity of small bowel obstruction from ingested plant matter in patients in whom gastrojejunostomy was performed.

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Case report:

A 60-year-old female was admitted in this hospital with the complaints of epigastric pain and discomfort for about 20 days. The pain was episodic but very severe and colicky in nature. It started about 10 minutes after taking meal. Pain was not radiating anywhere, but it was associated with nausea and vomiting. The bitter tasted vomitus contained the eaten food particle. Vomiting relieves the symptoms. The patient had no anorexia, but was afraid of taking food. She had constipation and mild weight loss. For these complaints, she was taken to a district hospital and treated by nil per oral; with nasogastric suction and intravenous fluid for 5 days and she felt well and could tolerate liquid feeding orally and hence discharged. After 15 days she suddenly developed bloating of abdomen, belching, pain, nausea and vomiting. Then she was referred to Khulna Medical College Hospital.

Nineteen years back, she underwent vagotomy and gastrojejunostomy operation for pyloric stenosis and 5 years back she had leprosy and was treated. She was convinced after the bypass operation that leafy vegetables would keep her physique alright. Hence, she used to eat more green leafy vegetables. She would take fish with average Bangladeshi meal thrice daily.

On examination, she was mildly cachectic, anaemic and dehydrated. Her pulse rate was 90 beats/ min and BP was 110/70 mm Hg. On examination of her abdomen, we found it scaphoid and visible peristalsis was noted. Neither any palpable mass nor any organomegaly was found. But her bowel sound was sluggish. Her rectum was found normal by digital examination. Examination of head, neck, chest and cardiovascular system was unremarkable.

Complete blood count and metabolic panel were normal. ECG, Chest X-ray and plain X-ray abdomen revealed no abnormality. Ultrasonography of whole abdomen was clueless. Barium meal X-ray reported emptying was delayed; visualized jejunal loops were dilated with intraluminal filling defects are noted at the end of the visualized contrast column, giving the impression of intussusception (Figure 1). Endoscopic examination reveals no abnormality in the stomach and duodenum.

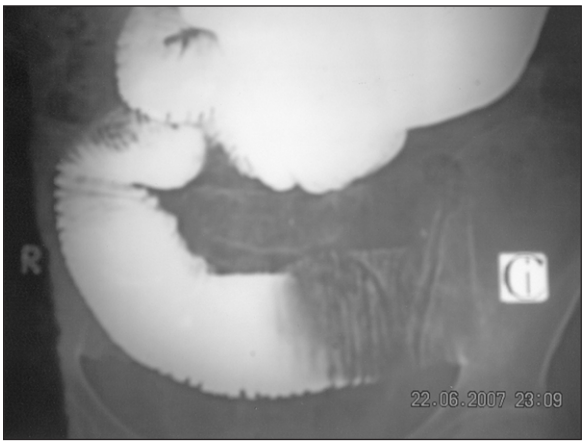

Fig.-1: *Barium meal study of the patient: giving impression of intussusception.*

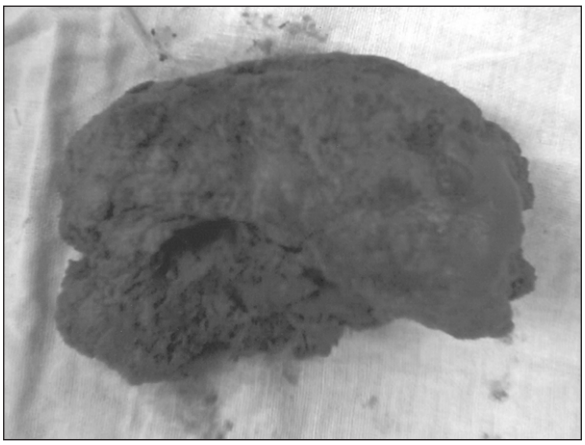

Fig.-2: *The removed phytobezoar.*

To manage the patient, a nasogastric tube was placed to at intermittent suction and intravenous nutrition was provided. After improving her general condition and with the preoperative diagnosis of upper gastrointestinal obstruction, we decided to operate. Peroperatively we found, both the loops of her gastrojejunostomy were patent. But, an obstructing mass, about 8 cm in length and 5 cm in diameter, was noted in jejunum, about 25 cm distal to the stoma. After giving a transverse incision in the jejunum the mass was found greenish in colour and was made up of fibres of leafy vegetables (Figure 2). Jejunum was closed and then wound was closed in layers. Her postoperative period was uneventful and she recovered from the illness completely. Later on, she can take food normally.

Discussion:

The term bezoars derived from the Persian, *padzahr*, which means counterpoison. Their therapeutic uses were first accredited to Aramzoar, a 12th century Arabian surgeon[5]. Bezoars are found in the stomachs of antelopes and goats and were highly prized for their magical healing powers[6].

There are 4 types of bezoars. Phytobezoars are the most common, and are composed of vegetable matter such as celery, pumpkin, grape skin, prune and persimmons and it contains large amount of nondigestable fibres such as cellulose, hemicellulose, lignin and fruit tannins. Trichobezoars are gastric concretion of hair fibres which usually presents in patients with history of psychiatric predisposition and in children with mental retardation. Pharmacobezoars consist of medication bezoars, which in bulk will adhere, such as cholestyramine, kayexalate resin, cavafate and antacids. Lactobezoars are milk curd secondary to infant formula, described in low birth weight neonated fed on highly concentrated formula within the first week of life[7].

Previous gastric resection or ulcer surgery such as partial gastrectomy or truncal vagotomy with pyloroplasty predisposes to bezoars, which are included in postgastrectomy syndromes. Incidence of postgastrectomy bezoar range between 5-12%[2] It is interesting to note that more than half of cases of phytobezoars had history of previous gastric surgery[8]. Our patient gave a history of laparotomy and gastrojejunostomy done 19 years ago.

Other predisposing factors are ingestion of high fibre foods, abnormal mastication, diminished gastric secretion and motilility, autonomic neruropathy in diabetic patients and myotonic dystrophy[9]. An

association between H2 blocker therapy in the elderly and phytobezoar formation is also reported, presumably related to lowered gastric secretion and reduced gastric emptying[10,11].

In a normal stomach, vegetable fibres, which cannot pass through the pylorus, undergo hydrolysis within the stomach, which softens them enough to go through the small bowel. After gastric surgery, the gastric motility is disturbed and the gastric acidity is decreased, and the stomach may empty rapidly with an increased possibility of bezoar formation. Normally found in the stomach, they may pass into the small bowel. Primary small bowel bezoar is very rare and is normally formed in patients with underlying small bowel disease such as diverticulum, stricture or tumour[12].

Clinical manifestations depend on the location of the bezoars. Gastric bezoars cause mostly non-specific symptoms (eg, epigastric pain, dyspepsia, occasional vomiting, and postprandial fullness). The most common clinical manifestations of an intestinal bezoar are complete or partial mechanical intestinal obstruction. They usually become impacted in the narrowest portion of the small bowel, the commonest site being the terminal ileum followed by the jejunum[13], as was found in our patient. In these patients, temporary relief with recurrence is named intestinal 'lucid interval' by some authors[14]. The mentioned patient supports this 'lucid interval' by her relief of symptoms in between her two-hospital admissions.

Barium studies characteristically show an intraluminal filling defect of variable size that is not fixed to the bowel wall. Barium filling the interstices gives a mottled appearance similar to that of a villous tumour[9]. H.C. Teng et al. claim to be the first in describing an interesting finding in the barium study of a patient with phytobezoar obstruction. That showed an intraluminal filling defect with a claw appearance giving the impression of an intussusception. The barium study of presented patient represents this recent finding[12] (Fig 2).

Small-bowel obstruction secondary to phytobezoars should be differentiated mainly from similar conditions secondary to adhesion. However, careful patient questioning and a high index of suspicion in patients with previous gastric surgery should, at least, suggest the diagnosis. Subsequently, gastroscopy may reveal a bezoar in the stomach. When the latter is found, an attempt should be made to break the bezoar into small pieces that could be removed endoscopically. Various proteolytic materials such as papain, cellulase, pancreatic enzymes, and bile salts have all been tried to disintegrate phytobezoars[15,16], with little success[17]. They are best treated surgically. At the time of surgery milking the bezoars into the cecum is the procedure of choice. If this is not possible, enterotomy may become necessary[18].

Conclusion:

Bezoar formation is rare in healthy subjects and majority develops in persons with predisposing factors. Prevention includes avoidance of high fibre foods, introduction of prophylactic medication to improve gastric emptying and psychological or psychiatric follow up in patients with psychiatric disease[7]. Proper counseling about food habit bears immense importance after gastric surgery to prevent this.

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Combined Carotid Endarterectomy and off Pump Coronary Artery Bypass Grafting : A Case Report

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Summary:
A 64 years male presented as a case of triple vessel coronary artery disease with bilateral asymptomatic carotid lesion. He underwent combined right carotid endarterectomy (CEA) and off pump coronary artery bypass grafting (OPCAB) with excellent outcome.

(J Bangladesh Coll Phys Surg 2009; 27: 103-106)

Introduction:

There are few areas in cardio-vascular surgery more controversial than the issue of combined carotid endarterectomy (CEA) and coronary artery bypass grafting (CBG). Although indications for both operations have been elucidated and techniques have been standardized, the results varies between centers[1].

Carotid artery disease is an important risk factor for stoke (CVA) after CABG[1,2]. Hemodynamically significant carotid artery stenosis are associated with as many as 30% of the stroke arising after CABG[1,2].

Presence or absence of a carotid bruit is poorly predictive of high grade stenosis even in the setting of known symptomatic carotid artery disease[3].

Carotid duplex Ultrasonogrpahy is currently the most widely used technique for preoperative screening to detected important carotid artery disease in patients undergoing CABG. If the preoperative carotid duplex study demonstrates high grade (>70%) stenosis and the patient's hemodynamic state is stable with no critically stenotic coronary arteries, CEA and CABG procedures are staged, performing the CEA first. If the patient is hemodynamically unstable, or if there is high grade left main coronary artery or proximal LAD disease, a combined CEA and CABG procedure is performed(4). This approach is justified because prophylactic CEA has been shown to be superior to conservative therapy for preventing stroke in symptomatic or asymptomatic patients with high grade carotid stenosis[5].

When a combined procedure is performed, CEA is done before cardiopulmonary bypass (CPB) is established[4].

Publications have shown CABG is safe and effective in patients with significant left main CAD when using off-pump (OPCAB) CABG techniques[6,7].

Case report:

A 64 years old male was admitted in the cardiac surgery ward of the National Heart Foundation Hospital with complains of chest pain on exertion and dyspnoea for 6 months. He was a known patient of hypertension and diabeties mellitus. He had also a history inferior MI, 15 years back.

On physical examination the patient was found anxious looking and of average built. All peripheral pulses were palpable and of moderate volume. On auscultation there was bilateral carotid bruit. Hematological and biological parameters were within normal limits. ETT was positive. Echocardiographic

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evaluation revealed anterior septal and apical wall moderately hypokinetic; inferior and basal posterior walls were echogenic and akinetic. Ejection fraction 50%. Duplex study of neck arteries revealed 70-75% right ICA and 35-40% left ICA lesion with flow disturbance.

Coronary angiogram showed left main coronary artery 60%, left anterior descending (LAD) 75%, diagonal (D) 50%, left circumflex (LCx) 90%, obtuse marginal (OM) 70%, right coronary artery (RCA) 100% stenosis. D and OM were narrow calibre, diffusedly diseased vessels.

Our final diagnosis was significant left main with triple vessel CAD and bilateral asymptomatic carotid artery lesion.

Combined right CEA and OPCAB grafting was planned.

We started midline sternotomy and harvesting of GSV as bypass conduit simultaneously. Then the right sided carotid endarterectomy was performed using CCA to ICA intravascular shunt. The distal CCA, bifurcation, proximal ICA and ECA origins were made clean of atheromatous plaques. Arteriotomy was closed using PTFE patch. After establishing satisfactory ICA/ECA flow and ensuring proper hemostasis, the neck wound was loosely approximated over a sponge.

Then OPCAB to LAD and PDA was performed. Due to diffusely diseased and narrow calibre, OM and D were not graftable. At the same time the LIMA with unsatisfactory flow was not used as a conduit.

Post operative recovery was smooth and unremarkable. On the 7th post-operative day stitches were removed, healing was by 1st intention.

At routine follow-up at 1 and 3 months, the patient was found in satisfactory condition. He was hemodynamically stable and without any chest pain or neurological symptoms.

Discussion :

Next to operative mortality, an irreversible perioperative cerebrovascular accident is the most dreaded perioperative complication of myocardial revascularisation, primarily because of the devastating consequences to the patient, and also because of the significantly increased cost of hospital and posthospital care[9]. In their study Puskas et a[9] noted 2.2% stroke after CABG. The stroke was associated with significantly more in hospital morbidity, longer length of stay and almost twice the hospital cost. Patients in that study who suffered a perioperative stroke has a 23% hospital mortality rate[9]. The most common cause of perioperative stroke is atherosclerotic or thrombotic embolic debris from the heart or major vessels. From a surgical point of view, of all the potential causes of perioperative stroke, carotid stenosis is the one artomatic situation about which the surgeon can routinely take action to remove the pathology.

Early studies related the presence of carotid stenosis to development of perioperative neurologic injuries using auscultatory evidence of carotid disease as the method of diagnosis. A number of investigators studied the relationship between carotid bruits and perioperative stroke[1,2,3]. Unfortunately carotid bruit are not reliable indicators of ICA stenosis, nor are they discriminatory as to the degree of carotid stenosis. Much better definition of the degree of stenosis obtained with noninvasive carotid testing using Doppler ultrasound techniques. the accuracy and reliability of these methods to quantify the degree of stenosis and provide visual images of the stenosis has made these methods the initial and in most of the cases sole method of choice is evaluating patients suspected of having carotid occlusive disease[10]. In our protocol of evaluation of CAD patients, it has because a standard and routine non-invasive procedure.

It is well established from the NASCET and ACAS trials that the long-term stroke risk of medical therapy is far higher than the risk of carotid endarterectomy in patients who have high grade carotid stenosis [5,10]. Therefore, if one was to improve the overall morbidity and mortality of patients with combined cardiac and carotid disease, one must certainly approach these patients with CEA in addition to CABG.

There continues to be a dilemma regarding the best means of surgical management of significant carotid artery disease in patients requiring coronary artery bypass surgery.

Most surgeons advocating a sequential operative approach to patients with severe combined disease usually do the CEA initially if the patient is hemodynamically stable and not ischaemic. However, the risk of a perioperative coronary ischaemic event remains a real threat.

Some cardiac surgeons have opted to perform an initial CABG followed by CEA for patients with unstable angina and asymptomatic carotid lesion. The principal risk with this approach is the potential for postoperative stroke.

Currently concomitant carotid and coronary bypass operations for virtually all patients with severe combined disease is advocated. The strategy of performing both operative procedures during one anesthetic is based upon the premise that only such an approach in patients with severe combined disease can minimize cardiac events that frequently complicate isolated CABG. Moreover, doing the two operative procedures together is more cost effective in terms of number of anesthesia and additional hospital stay.[10,11]

Recent publications have shown CABG is safe and effective in patient with critical left main stem stenosis when using OPCAB techniques[6,7]. It has been shown that the requirement for iinotropic support, prolonged length of stay, incidence of stroke and chest infection were significantly reduced in patients receiving OPCAB coronary surgery[6].

Taking all these into consideration we planned to perform combined CEA and OPCABG in our case. We followed the standard protocol and were meticulous in performing all steps of surgery. Postoperative management of our patient was not difficult from those patients having isolated CABG. We maintained a good coronary and cerebral perfusion pressure in the early postoperative hours.

Standard anticoagulation protocol consisting of aspirin within 6 hours of surgery was followed. Additional heparin was used because of prosthetic carotid patch.

Post operative period was smooth and unremarkable.

Conclusion :

The case is probably first time reported in Bangladesh. Symptomatic or asymptomatic high grade carotid lesion along with CAD are usual and frequent findings now-a-days, Non-invasive carotid evaluation, using duplex ultrasonography as a screening modality is an well established and time tested investigation. At the same time excellent randomized trials have established the safety and efficacy of CEA as the most appropriate treatment for both symptomatic or asymptomatic high grade carotid stenosis. A number of randomized studies have demonstrated the advantage of concomitant CEA and CABG over staged procedures. OPCAB procedure is also established as a safe, effective and patient friendly procedure. Considering all these we planned combined CEA and OPCAB coronary revascularisation. After this first successful case, we were confident to perform this procedure more in future.

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Skin Tuberculosis: A Case Report

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Summary:

A 17 year old boy presented with multiple non healing ulcers over the right lower limb and left upper limb for 13 years which were non responsive to varieties of treatment modalities used in his locality. There were also contracture deformities of the affected limbs. Clinically the diagnosis was quite confusing. but histopathology of specimen from skin lesion confirmed it as a case of skin tuberculosis. The patient was put on anti TB drugs and the ulcers responded well and started to heal rapidly. Some reconstructive surgery will be done for his contractures after completion of the anti TB drug regimen and proper healing of ulcers.

(J Bangladesh Coll Phys Surg 2009; 27: 107-111)

Introduction:

Tuberculosis of the skin is caused by *M. tuberculosis* , *M. bovis* and under certain conditions the bacillus Calmettee- Guerin (BCG), an attenuated strain of M. bovis.[1] With respect to Mycobacterial skin disease , the so called atypical mycobacteria may be the cause of skin disease more frequently than *M. tuberculosis*.[2]Cutaneous tuberculosis (TB) is essentially an invasion of the skin by *Mycobacterium tuberculosis,* the same bacteria that causes pulmonary TB.[3] Cutaneous TB is a relatively uncommon form of extrapulmonary TB. Even in countries such as India and China where TB still commonly occurs,cutaneous TB cases are rare(< 0.1%)[2]. Direct infection of the skin or mucous membranes from an outside source of mycobacteria results in a initial lesion called the tuberculous chancre. The chancres are firm shallow ulcers with a granular base. They appear about 2-4 weeks after mycobacteria enter through broken skin. The immune response of the patient and the virulence of the mycobacteia determine the type and severity of the cutaneous TB.The types are TB verrucosa cutis, Lupus vulgaris, Scrofuloderma, Miliary TB and Tuberculid TB. The diagnosis is usually made or confirmed by a skin biopsy. Typical tubercles are caseating epithelioid granulomas that contain acid-fast bacilli. Other tests that may be necessary include: Tuberculin skin test(Mantoux), sputum culture (It may take a month or longer for results to be reported), Chest X-ray and other radiological tests for extrapulmonary infections.Patients with pulmonary or extrapulmonary TB need to be treated with antitubercular drugs. This usually a combination of antibiotics(Isoniazid, Rifampicin, Pyrazinamide and Ethambutol) given over a period of 6 months.[4] Involvement of other organs along with skin may need treatment for several months and sometimes years. Patients with TB infection but no active disease must also be treated with antitubercular drugs to prevent development of active disease. Occasionally surgical excision of localized cutaneous TB is recommended.

Case Report:

The patient was a 17 year old boy from Norshingdi, got admitted in surgery unit II of Dhaka Medical College Hospital (DMCH) on 12th March, 2007 with the complaints of multiple ulceration over the right lower limb for last 13 years, left upper limb for 8-9 years and progressive stiffening of the joints of affected limbs for the last one year.Initially there was a very small nodule on the sole of the right foot wich ulcerated and gradually spread over the whole lower

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limb. At the age of 7 years after getting a cut injury over the base of left thumb same scenario began.These ulcers were non-responsive to different medications. It was associated with variable episodes of fever, loss of appetite and weight loss. During the whole period of illness he never had any history of cough and haemoptysis, significant bowel and bladder complaints or any other systemic problems.

He is also not a known diabetic patient. He gave no history of childhood skin disease. He received some vaccines but the exact nature was not clear. He also gave no history of contact with TB patient. On examination the patient's body build and nutritional status was found to be below average. He was anaemic and oedema was present on the affected leg with no significant lymphadenopathy. His right lower limb showed multiple painful ulcers of variable size with discharging pus, floors were covered with slough and bleeded after removing the slough. There were also some healing ulcers which had scabs. Same features were seen on the left upper limb but with more crust and slough formation. No nerve thickening was found. The skin was thickened, hyperkeratotic. All other systemic examinations revealed no abnormalities and testes were normal. On investigations, total WBC count was 9800/cu mm, differential count were neutrophil 66%, lymphocyte 31%, eosinophil 2%, monocyte 1%, Haemoglobin was 65%, ESR 70 mm in 1st hour, blood sugar 2 hours after breakfast was 4.8 mmol/l, serum total protein 88 gm/l, serum albumin 34 gm/l, serum globulin 54 gm/l, serum A:G was 0.6:1, serum creatinine 1.4 mg/dl, MT test was positive (30 mm in diameter), HIV screening test was negative. Chest X-ray, X-rays of the affected limbs, Ultrasonogram of whole abdomen were normal. Skin scrapping for fungal infection was also negative. Skin biopsy was taken and histopathology report revealed that this was a case of skin tuberculosis. Fig-1. After confirming the diagnosis antitubercular drug was started in the form of isoniazid, rifampicin, pyrazinamide and ethumbutol for the initial 2 months and isoniazid and rifampicin for next 4 months[4]. During that period the wound was closed regularly but kept it open. The patient is on his third month of treatment now and the ulcers have already healed a lot. Fig-2, Fig-3, Fig-4, Fig-5. After completion of this antitubercular drug

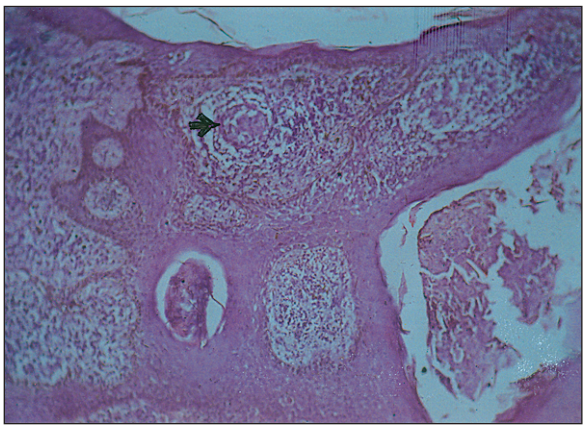

Fig.-1: *Photomicrograph of skin biopsy showing features suggestive of tuberculosis. Black arrow indicate granuloma.*

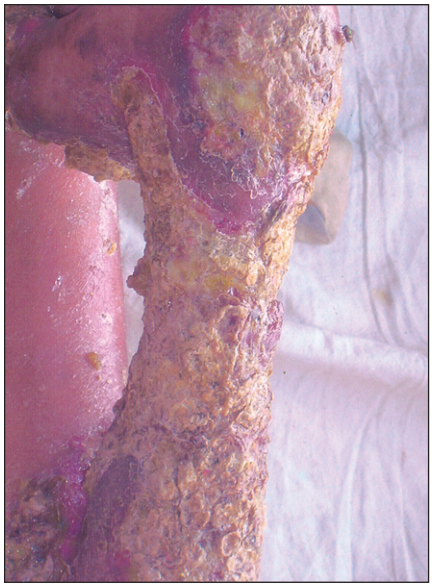

Fig.-2: *Lesion of the hand before starting anti TB drugs.*

Fig.-3: *Lesion of the foot before treatment with anti TB drugs.*

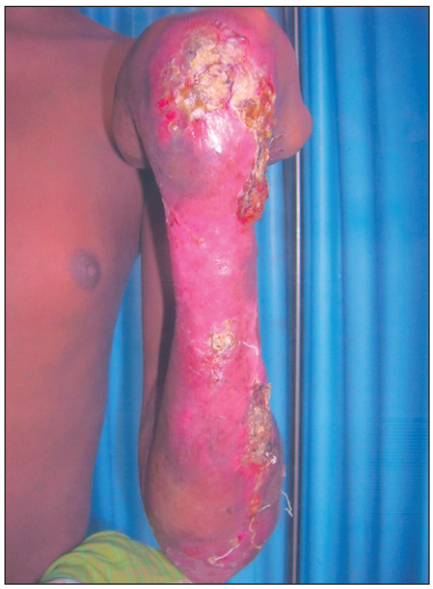

Fig.-4: *Lesion of the hand after 3 months treatment with anti TB drugs.*

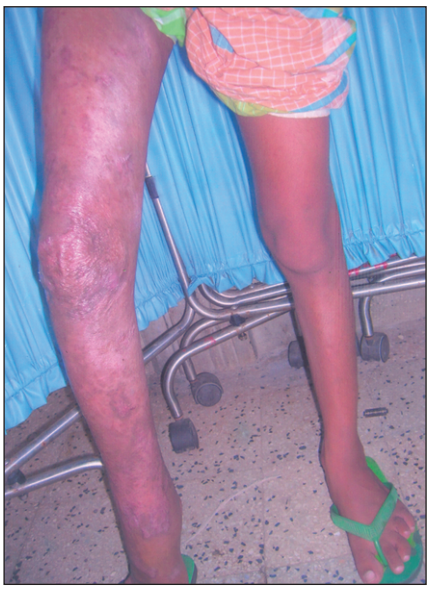

Fig.-5: *Lesion of the foot after 3 month treatment with anti TB drugs.*

regimen this patient will receive some form of reconstructive surgery to relieve his wound contracture.

Discussion:

Cutaneous tuberculosis is caused by *Mycobacterium Tuberculosis* and a member of a group of closely related organisms in the *M. tuberculosis* complex: *Mycobacterium africanum, Mycobacterium bovis, Mycobacterium microti,* and *M. tuberculosis* are also responsible. World incidence of TB increased with population density and urban development. During industrial revolution in Europe (1750), it was responsible for more than 25% of adult deaths. Indeed, in the early 20th century, TB was the leading cause of death in the United States[3]. Although 1 of 3 individuals globally is infected with tubercle bacillus, the incidence of cutaneous TB appears to be low. In areas such as India or China where TB prevalence is high, cutaneous manifestations of TB (overt infection or tuberculids) are found is less than 0.1% of individuals seen in dermatology clinics.In a 10 year (1983-1992) retrospective survey of patients seen in government dermatology clinics in Hong Kong, the detected Incidence of cutaneous TB among patients was 179 per 267,089 (0.07%). Among patients with cutaneous TB, 15% had classic cutaneous TB (approximately 5% each of lupus vulgaris, TB verrucosa cutis and scrofuloderma.) and 85% had tuberculids. In a tertiary care hospital in northern India, 0.1% of dermatology patients seen from 1975-1995 had cutaneous TB. Lupus vulgaris was the most frequent manifestation (55%), followed by scrofuloderma (27%), TB verrucosa cutis (6%), tuberculous gumma (5%) and tuberculids (7%). The incidence of patients with cutaneous TB seen from 1980-1993 in a hospital dermatology clinic in Madrid was 16 (0.14%)in

10,304. 160 cases of tuberculosis of the skin seen in the Government Dermatological Clinics in Hong Kong [5] are reviewed . Tuberculosis verrucosa was the commonest form and account for 46% of cases. The onset was in below the age of 20 in 77% of these patients. In a study of cutaneous tuberculosis took place in Morocco[6] two hundred and sixteen cases of cutaneous tuberculosis were identified and included. Men and women were equally affected. The mean patient age was 29 years. Major clinical types of cutaneous tuberculosis were scrofuloderma and gumma (72%), lupus vulgaris (12%), tuberculosis verrucosa cutis (7%), tuberculids (6%), orificial tuberculosis (1%) and tuberculous chancre (1%). Systemic involvement was seen in 35% of cases. Where performed (66%), 81% of subjects had

positive Mantoux skin test. Lesion biopsy for histopathologic study was performed in 81% of patients and showed classical tuberculous findings in 57%. *Mycobacterium tuberculosis* was isolated in culture from 9% of patients.

Primary inoculation TB results from direct introduction of mycobacteria into the skin or mucosa of an individual who was not previously infected with TB or was immunized with the *M bovis* strain Bacilli Calmette Guerin (BCG). Since mycobacterias do not penetrate intact skin, initiation of infection almost always follows an injury, usually in children. Common sites include the face and other exposed skin[3]. This presentation is quite similar to our case. But there are also some variations in development of skin tuberculosis such as a case published in a Turkey Journal.[6] where the authors reported a case of Tuberculosis verrucosa cutis in a patient with long standing generalized lichen planus of more than 20 years history and improvement of lichen after antitubercular polychemotherapy. The authors here tried to reflect a possible reciprocal causal relationship between two cutaneous conditions of different natures. In another case published in the American Journal of Nephrology [8] skin tuberculosis with atypical mycobacteria 8 years after combined pancreas- kidney transplantation where they report on a Mycobacterium marinum infection in a diabetic woman 8 years after undergoing a combined pancreas-kidney transplantation. This is, to their knowledge , the first case report on an isolated skin infection with atypical mycobacteria after simultaneous pancreas- kidney transplantation. A genetic probe categorization revealed an infection with *M. marinum.* Skin tuberculosis caused by *M marinum* is an uncommon complication in kidney or pancreas-kidney transplant recipients, hence the diagnosis can be delayed TB is an airborne communicable disease that occurs after inhalation of infectious droplets expelled from patients with laryngeal or pulmonary TB during coughing, sneezing or speaking. Each cough can generate more than 3000 infectious droplets. Droplets are so small (1-5 micro meter) that they remain airborne for hours. The probability that disease transmission will occur depends upon the infectiousness of the tuberculous patient, the environment in which the exposure takes place, and the duration of exposure [3] . But this is not always the same in skin tuberculosis. A rare case published in the American Journal of Tropical Medicine and Hygiene in 2004 reported a case of tuberculosis transmission from a patient with skin lesion and a negative sputum smear[9]. Here the author mentioned about a five-month- old girl who presented with miliary tuberculosis and multiple intracranial tuberculomas and her disease was actually transmitted from a 20 year old man presented with skin tuberculosis. *M. tuberculosis* isolates from the man and the infant matched exactly on both IS6110 restriction fragment length polymorphism analysis and spacer oligonucleotice typing. It has been estimated that patients with negative acid- fast smears cause 17 percent of new infections and present serious challenges to tuberculosis control. The common investigations that can be done for cases of skin tuberculosis are Chest X- ray, Tuberculin test(Mantoux), sputum smear and staining for acid fast bacilli, skin biopsy and histopathological examinations [1] were all done in this case and the results were also quite similar to other cases of skin tuberculosis. In presented case treatment was started with initial four drugs regimen comprising of isoniazid, rifampicin, ethumbutol, and pyrazinamide for two months and isoniazid and rifampicin for next 4 months. Though there is some mentioning about role of surgery in case of treating skin tuberculosis but this still

controversial and in presented case any surgery was not done for the wound. But the patient will receive some reconstructive surgery to relieve his wound contracture on a later period.

Conclusion:

Any longstanding ulcer not responding to conventional treatment needs assessment and treatment in expert hands before implicating their mutilating effects on human body.

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COLLEGE NEWS

(J Bangladesh Coll Phys Surg 2009; 27: 112-127)

Examination News:

Result of FCPS Part-I, FCPS Part-II and MCPS Examinations held in January, 2009 are given below:

3361 candidates appeared in FCPS Part-I Examination held in January, 2009, of which 501 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	54	5	9.26
2	Biochemistry	2	0	0.00
3	Dentistry	150	18	12.00
4	Dermatology & Venereology	90	8	8.89
5	Family Medicine	2	0	0.00
6	Haematology	19	2	10.53
7	Histopathology	10	2	20.00
8	Medicine	1069	151	14.13
9	Microbiology	7	2	28.57
10	Obst. & Gynae	798	141	17.67
11	Ophthalmology	90	8	8.89
12	Otolaryngology	71	9	12.68
13	Paediatrics	359	93	25.91
14	Physical Medicine & Rehabilitation	14	5	35.71
15	Psychiatry	10	2	20.00
16	Radiology & Imaging	52	3	5.77
17	Radiotherapy	24	11	45.83
18	Surgery	537	40	7.45
19	Transfusion Medicine	3	1	33.33
	Grand Total	**3361**	**501**	**14.91**

977 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
013-8502	Dr. Md Shahidul Islam	MAG Osmani Medical College, Sylhet	Urology
074-7002	Manisha Paul	MAG Osmani Medical College, Sylhet	Anaesthesiology
074-7004	Dr. Md masudul Alam Mazumder	Sher-E-Bangla Medical College, Barisal	Anaesthesiology
074-7007	Dr. Rabeya Begum	Rangpur Medical College, Rangpur	Anaesthesiology
074-7008	Dr. Nitai Chandra Sarkar	Rangpur Medical College, Rangpur	Anaesthesiology
074-7009	Dr. A.K.M. Faizul Hoque	Sir Salimullah Medical College, Dhaka	Anaesthesiology
074-7014	Jesmin Akhter Leena	Dhaka Medical College, Dhaka	Dermatology & Venereology
074-7016	Dr. Abu Jafar Md Shahidul Hoq	Chittagong Medical College, Chittagong	Dermatology & Venereology
074-7034	M. Mizanur Rahman	Sir Salimullah Medical College, Dhaka	Haematology
074-7039	Akhil Ranjon Biswas	Rangpur Medical College, Rangpur	Haematology
074-7040	Md. Iqbal Karim	Sir Salimullah Medical College, Dhaka	Histopatholoy

Roll No.	Name of candidate	From where Graduated	Speciality
074-7048	Dr. Abu Nayeem	Dhaka National Medical College, Dhaka	Medicine
074-7065	Dr. Md Khalilur Rahman	Rajshahi Medical College, Rajshahi	Medicine
074-7085	Dr. Mohiuddin Humayun Kabir Chowdhury	Chittagong Medical College, Chittagong	Medicine
074-7089	Dr. Md Abdur Rob	Mymensing Medical College, Mymensing	Medicine
074-7096	Nirmal Kanti Sarkar	Dhaka National Medical College, Dhaka	Medicine
074-7109	Mohammad Mahbubul Haque	Mymensing Medical College, Mymensing	Medicine
074-7110	Dr. Rokanuzzaman Bhuyian	Mymensing Medical College, Mymensing	Medicine
074-7118	Md. Kamal Hossain	Dhaka Medical College, Dhaka	Medicine
074-7133	Dr. Md. Mahabulbul Alam Khandker	Mymensing Medical College, Mymensing	Medicine
074-7138	Dr. Ratan das Gupta	MAG Osmani Medical College, Sylhet	Medicine
074-7139	Dr. Mohammad Shah Jamal	Rangpur Medical College, Rangpur	Medicine
074-7144	Rumana Habib	Mymensing Medical College, Mymensing	Medicine
074-7145	Quazi Md Anisujjaman	Dhaka Medical College, Dhaka	Medicine
074-7160	Dr. Mehruba Alam Ananna	Sir Salimullah Medical College, Dhaka	Medicine
074-7180	Shiek Md Abul Fazal	Dhaka Medical College, Dhaka	Medicine
074-7183	Iftikher Alam	Dhaka Medical College, Dhaka	Medicine
074-7204	Dr. Abu Ayub Md. Nazmul Huda	Sir Salimullah Medical College, Dhaka	Medicine
074-7210	Dr. Md. Enayet Hossain	Mymensing Medical College, Mymensing	Medicine
074-7211	Dr. Md. Noor Islam	Rangpur Medical College, Rangpur	Medicine
074-7213	Dr. Bakhtiare Mohammad Shoeb	Dhaka Medical College, Dhaka	Medicine
074-7219	Dr. Md. Abdullahel Kafee	Mymensing Medical College, Mymensing	Medicine
074-7229	Muhammed Arshad-UL-Azim	Chittagong Medical College, Chittagong	Medicine
074-7235	Muhammed Syedul Alam	Dinajpur Medical College, Dinajpur	Medicine
074-7236	Mohammed Habibur Rahman	Chittagong Medical College, Chittagong	Medicine
074-7240	Mohiuddin Ahmed	Shahid Ziaur Rahman Medical College, Bogra	Medicine
074-7241	Salahuddin Mohammed Ali Haider		Medicine
074-7254	Mohammad Yunus	Rajshahi Medical College, Rajshahi	Medicine
074-7262	Hari Bhushan Sarker	MAG Osmani Medical College, Sylhet	Medicine
074-7265	Sarmistha Biswas	MAG Osmani Medical College, Sylhet	Medicine
074-7270	Saleh Mohammad Shahedul Islam	MAG Osmani Medical College, Sylhet	Medicine
074-7278	Dr. Md Farhad Hussain	MAG Osmani Medical College, Sylhet	Medicine
074-7281	Dr. Md. Mizanur Rahman Khan	Dhaka Dental College, Dhaka	Medicine
074-7287	Dr. Fatema Ahmed	Chittagong Medical College, Chittagong	Medicine
074-7299	Tarek Mahmood	Mymensing Medical College, Mymensing	Medicine
074-7301	Mohammad Sayeed Hassan	Dhaka Medical College, Dhaka	Medicine
074-7303	Md. Mamunur Rashid	Rajshahi Medical College, Rajshahi	Medicine
074-7310	Golam Rahman Bhuiyan	Dhaka Medical College, Dhaka	Medicine
074-7324	Dr. Abul Makarrem Baruddin Safdar	Sir Salimullah Medical College, Dhaka	Medicine

Roll No.	Name of candidate	From where Graduated	Speciality
074-7341	Naimah Masood	Bangladesh Medical College, Dhaka	Obst and Gynae
074-7344	Farjana Yasmin	Dhaka Medical College, Dhaka	Obst and Gynae
074-7345	Fahmida Shireen	Rajshahi Medical College, Rajshahi	Obst and Gynae
074-7348	Tasnim Akter	Chittagong Medical College, Chittagong	Obst and Gynae
074-7356	Nasima Akther	Chittagong Medical College, Chittagong	Obst and Gynae
074-7358	Jannatul Ferdous	Khulna Medical College, Khulna	Obst and Gynae
074-7359	Chowdhury Shamima Sultana	Sir Salimullah Medical College, Dhaka	Obst and Gynae
074-7360	Munirunnessa	Mymensing Medical College, Mymensing	Obst and Gynae
074-7366	Shamim Jahan	Dhaka Medical College, Dhaka	Obst and Gynae
074-7368	Dr. Lima Shompa	Dhaka Medical College, Dhaka	Obst and Gynae
074-7380	Dr. Nahid Reaz Shapla	Dhaka Medical College, Dhaka	Obst and Gynae
074-7388	Monika Rani Mohanta	Rajshahi Medical College, Rajshahi	Obst and Gynae
074-7400	Dr. Most. Safura Khatun	Rangpur Medical College, Rangpur	Obst and Gynae
074-7407	Dr. Afsari Ahmad	Sher-E-Bangla Medical College, Barisal	Obst and Gynae
074-7411	Dr. Farhana Parveen	Chittagong Medical College, Chittagong	Obst and Gynae
074-7417	Dr. Nowshafreen Chowdhury	Sir Salimullah Medical College, Dhaka	Obst and Gynae
074-7430	Dr. Kazi Farhana Begum	Rangpur Medical College, Rangpur	Obst and Gynae
074-7455	Asifa Ali	Rangpur Medical College, Rangpur	Obst and Gynae
074-7463	Begum Shaira Sharifa	Sher-E-Bangla Medical College, Barisal	Obst and Gynae
074-7466	Most. Afroza Sarkar	Rajshahi Medical College, Rajshahi	Obst and Gynae
074-7485	Mafruha Jahan	Rangpur Medical College, Rangpur	Obst and Gynae
074-7508	Jinnatun Nur	Mymensing Medical College, Mymensing	Obst and Gynae
074-7509	Sabina Easmin	Dhaka Medical College, Dhaka	Obst and Gynae
074-7515	Sankar Kumar Basak	Dhaka Medical College, Dhaka	Obst and Gynae
074-7516	Kazi Salma Rezina	Dhaka Medical College, Dhaka	Obst and Gynae
074-7521	Dr. Nurjahan Begum	Rajshahi Medical College, Rajshahi	Obst and Gynae
074-7524	Dr. Mukti Rani saha	Faridpur Medical College, Faridpur	Obst and Gynae
074-7530	Dr. Salma Akhtar Walida	MAG Osmani Medical College, Sylhet	Obst and Gynae
074-7543	Dr. Tanzila Halim	Jahurul Islam Medical College, Bajitpur	Obst and Gynae
074-7547	Dr. Begum Mushaheda Annur	Mymensing Medical College, Mymensing	Obst and Gynae
074-7556	Dr. Tahmina Hossain	Rajshahi Medical College, Rajshahi	Obst and Gynae
074-7565	Dr. Shirin Akhtar	Comilla Medical College, Comilla	Obst and Gynae
074-7576	Mst. Amena Rahman	Comilla Medical College, Comilla	Obst and Gynae
074-7596	T.H. Johra	Mymensing Medical College, Mymensing	Obst and Gynae
074-7600	Dr. Md. Abdur Raquib	Rangpur Medical College, Rangpur	Opthalmology
074-7603	Dr. Md Abdur Rashid	Mymensing Medical College, Mymensing	Opthalmology
074-7608	Dr. Md. Shafiqul Alam	Dhaka Medical College, Dhaka	Opthalmology
074-7611	Md. Abdus Salam	Sir Salimullah Medical College, Dhaka	Opthalmology
074-7617	Dr. Nishat Parveen	Mymensing Medical College, Mymensing	Opthalmology

Roll No.	Name of candidate	From where Graduated	Speciality
074-7620	Dr. Nazmun Nahar	Sir Salimullah Medical College, Dhaka	Opthalmology
074-7621	Dr. Md. Yosuf Ali	Rajshahi Medical College, Rajshahi	Opthalmology
074-7623	Dr. Md. Masud Murshed Talukder	MAG Osmani Medical College, Sylhet	Opthalmology
074-7624	Nazneen Khan	Dhaka Medical College, Dhaka	Opthalmology
074-7627	Dr. Abu Noyeem Md Yousuf	Khulna Medical College, Khulna	Opthalmology
074-7631	Dr. Mahmood Mujtaba	Dhaka Medical College, Dhaka	Opthalmology
074-7647	Dr. Md Shazibur Rashid	Comilla Medical College, Comilla	Otolaryngology
074-7648	Md. Bashir Ahmed	Sir Salimullah Medical College, Dhaka	Otolaryngology
074-7671	Dr. Kanij Fatema	Dhaka Medical College, Dhaka	Paediatrics
074-7690	Dr. Shahed Iqbal	Institute of Applied Health Science Under USTC, Chittagong.	Paediatrics
074-7695	Dr. Reema Afroza Ali	Dhaka Medical College, Dhaka	Paediatrics
074-7698	Dr. Rafeza Khanam	Chittagong Medical College, Chittagong	Paediatrics
074-7708	Mohammad Jahirul Islam	Dhaka Medical College, Dhaka	Paediatrics
074-7715	Fahmida Begum	MAG Osmani Medical College, Sylhet	Paediatrics
074-7726	Dr. Nasima Akter	Sir Salimullah Medical College, Dhaka	Paediatrics
074-7730	Dr. A.S.M. Waliullah	MAG Osmani Medical College, Sylhet	Paediatrics
074-7735	Farah Naz Shoma	Dhaka Medical College, Dhaka	Paediatrics
074-7738	Kiswar Silvana	MAG Osmani Medical College, Sylhet	Paediatrics
074-7741	Mohammad Khaled Hossain	Dhaka Medical College, Dhaka	Paediatrics
074-7748	Laila Shamima Sharmin	Dhaka Medical College, Dhaka	Paediatrics
074-7759	Probir Kumar Sarkar	Dinajpur Medical College, Dinajpur	Paediatrics
074-7765	Mohammaed Zahir Uddin	Dhaka Medical College, Dhaka	Paediatrics
074-7781	Dr. Ferdousi Begum	Dhaka Medical College, Dhaka	Paediatrics
074-7794	Mohammad Moniruzzaman	Chittagong Medical College, Chittagong	Physical Medicine & Rehabilitation
074-7799	Dr. Mohammad zahidul Hoque	Chittagong Medical College, Chittagong	Physical Medicine & Rehabilitation
074-7801	Mohammad Monirul Islam	Rajshahi Medical College, Rajshahi	Psychiatry
074-7802	Dr. Avra Das Bhowmik	Sir Salimullah Medical College, Dhaka	Psychiatry
074-7803	Md. Hafizur Rahman Chowdhury	Sir Salimullah Medical College, Dhaka	Psychiatry
074-7804	Iffat Ara Begum	Rangpur Medical College, Rangpur	Radiology & Imaging
074-7807	Dr. Ashraf Uddin Khan	Chittagong Medical College, Chittagong	Radiology & Imaging
074-7808	Taposshi Sarker	Dhaka Medical College, Dhaka	Radiology & Imaging
074-7812	Dr. Md. Sadrul Amin	Mymensing Medical College, Mymensing	Radiology & Imaging
074-7816	Mohammad Ali Kabir	Rajshahi Medical College, Rajshahi	Radiology & Imaging
074-7820	Dr. Md. Mashiur Rahman	Sir Salimullah Medical College, Dhaka	Surgery
074-7836	Syeda Asmema Shashi	Medical College for Women and Hospital, Dhaka	Surgery

074-7858	Md. Ahad Ali Mahalder	Chittagong Medical College, Chittagong	Surgery
074-7881	Dr. Fatema Akter	Rajshahi Medical College, Rajshahi	Surgery
074-7914	Mohammad Khairuzzaman	Faridpur Medical College, Faridpur	Surgery
074-7920	Hafiz Ahmed Nazmul Hakim	Dhaka Medical College, Dhaka	Surgery
074-7939	Dr. Arif Salam Khan	Dhaka Medical College, Dhaka	Surgery

65 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
008-8002	Md. Shakib-Uz-Zaman Arefin	Sir Salimullah Medical College, Dhaka	Preli-Medicine
008-8024	Md. Abul Kashem	Sir Salimullah Medical College, Dhaka	Preli-Medicine
008-8026	Dr. Mohammad Shakhawat Alam	Rajshahi Medical College, Rajshahi	Preli- Paediatrics
008-8028	Dr. Fatema Saam	Mymensing Medical College, Mymensing	Preli- Paediatrics
008-8033	Sk. Nishat Abdullah	Rangpur Medical College, Rangpur	Preli- Surgery
008-8036	Dr. Sattar Mohammad Sumon	Moulana Bhasani Medical College, Dhaka	Preli- Surgery
008-8043	Ahmed Sharif	Armed Forces Medical College, Dhaka	Preli- Surgery
008-8047	Dr. Mohammad Hedayyet Ali Khan		Preli- Surgery
008-8049	Dr. Mohammad Nazrul Hossain	Sir Salimullah Medical College, Dhaka	Preli- Surgery
008-8052	Dr. Iftekhar Ibne Mannan	Mymensing Medical College, Mymensing	Preli- Surgery
008-8058	Debashis Sarkar	Rajshahi Medical College, Rajshahi	Preli- Surgery
008-8061	Syed Mosfiqur Rahman	Mymensing Medical College, Mymensing	Preli- Surgery
008-8065	Saif Ul Hoque		Preli- Surgery

327 candidates appeared in MCPS 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
074-9003	Mohammad Abu Taher	Sher-E-Bangla Medical College, Barisal	Anaesthesiology
074-9005	Reza Ershad	Mymensing Medical College, Mymensing	Anaesthesiology
074-9006	Abu Hena Mostafa Kamal	Rangpur Medical College, Rangpur	Anaesthesiology
074-9008	Mohammad Hasan Kibria	Dhaka National Medical College, Dhaka	Anaesthesiology
074-9009	Mohammad Sazzad Hossain	Sher-E-Bangla Medical College, Barisal	Anaesthesiology
074-9013	Abdul Motaleb		Clinical Pathology
074-9015	Mohammad Sowkat Hossain	Comilla Medical College, Comilla	Clinical Pathology
074-9018	Mohammed Nuruzzaman Bhuiyan	Sher-E-Bangla Medical College, Barisal	Clinical Pathology
074-9019	Dr. Farjana Sultana	Dhaka Dental College, Dhaka	Dental Surgery
074-9025	Dr. Mohammad Rahmat Ullah Siddique	Sher-E-Bangla Medical College, Barisal	Dermatology and Venereology
074-9029	Abu Reza Sayem Ahmed	Sher-E-Bangla Medical College, Barisal	Dermatology and Venereology
074-9030	Md. Zafrul Islam	Bangladesh Medical College, Dhaka	Dermatology and Venereology
074-9032	Meherun Kabir	Chittagong Medical College, Chitagong	Dermatology and Venereology
074-9033	Mst. Moriom Nessa	Rajshahi Medical College, Rajshahi	Dermatology and Venereology
074-9034	Md. Kamruzzaman	Sher-E-Bangla Medical College, Barisal	Dermatology and Venereology

Roll No.	Name of candidate	From where Graduated	Speciality
074-9038	Dr. Md. Abdus Salam	MAG Osmani Medical College, Sylhet	Family Medicine
074-9064	Dr. Muhammed Mustafizur Rahman Palash	Chittagong Medical College, Chitagong	Medicne
074-9072	Dr. Md Atiquel Islam Chowdhury	Chittagong Medical College, Chitagong	Medicne
074-9089	Manabendra Bhattacharjee	MAG Osmani Medical College, Sylhet	Medicne
074-9101	Jafrin Jahan	Jahurul Islam Medical College, Bajitpur	Medicne
074-9117	Md. Nazrul Islam	MAG Osmani Medical College, Sylhet	Medicne
074-9124	Md. Sirajum Munir	Rajshahi Medical College, Rajshahi	Medicne
074-9134	Dr. Md. Motlabur Rahman	Rajshahi Medical College, Rajshahi	Medicne
074-9147	Dr. Alifa Nasrin	Chittagong Medical College, Chitagong	Medicne
074-9154	Dr. Kalpana Rani Mpidha	Sher-E-Bangla Medical College, Barisal	Obst and Gynae
074-9155	Dr. Tanbirul Arafin	Sher-E-Bangla Medical College, Barisal	Obst and Gynae
074-9161	Dr. Kazi Sonia Munir	Sher-E-Bangla Medical College, Barisal	Obst and Gynae
074-9162	Dr. Monorama Sarkar	Rangpur Medical College, Rangpur	Obst and Gynae
074-9165	Dr. Syeda Razia Karim	Institute of Applied Health Science . Under USTC, Chittagong	Obst and Gynae
074-9167	Reefaat Rahman	Dhaka Medical College, Dhaka	Obst and Gynae
074-9172	Akterun Naher	MAG Osmani Medical College, Sylhet	Obst and Gynae
074-9176	Laila Noor	Dhaka Medical College, Dhaka	Obst and Gynae
074-9203	Yasmin Ara Begum	Chittagong Medical College, Chitagong	Obst and Gynae
074-9205	Rahnuma Hasan	Chittagong Medical College, Chitagong	Obst and Gynae
074-9206	Nadira Haque	Community Based Medical College, Mymensing	Obst and Gynae
074-9208	Raihana Musawwir	Dhaka Medical College, Dhaka	Obst and Gynae
074-9219	Dr. Ashrafun Nahar	Sir Salimullah Medical College, Dhaka	Obst and Gynae
074-9225	Dr. Salma Akhter	Chittagong Medical College, Chitagong	Obst and Gynae
074-9228	Dr. Nadia Rahman	Chittagong Medical College, Chitagong	Obst and Gynae
074-9229	Dr. Nazma Akter	Rangpur Medical College, Rangpur	Obst and Gynae
074-9232	Dr. Mst. Umme Hani	Sir Salimullah Medical College, Dhaka	Obst and Gynae
074-9239	Md. Ruhul Amin Khan	Sher-E-Bangla Medical College, Barisal	Opthalmology
074-9244	ABM Shawkat Hayat	Sher-E-Bangla Medical College, Barisal	Opthalmology
074-9245	Md. Zahidur Rahman	Rajshahi Medical College, Rajshahi	Opthalmology
074-9248	Dr. Md Asduzzaman	Mymensing Medical College, Mymensing	Otolaryngology
074-9250	Mohammed Imran Khan	Dhaka National Medical College, Dhaka	Otolaryngology
074-9258	Dr. Md Ahsanuzzaman Khan	Jahurul Islam Medical College, Bajitpur	Otolaryngology
074-9274	Fahmila Anjum	Jahurul Islam Medical College, Bajitpur	Paediatrics
074-9276	Dr. Mohammad Shafiullah	Rangpur Medical College, Rangpur	Paediatrics
074-9279	Shaker Uddin Ahmed	Chittagong Medical College, Chitagong	Psychiatry
074-9280	Dr. ATM Mostafizur Rahman	Sher-E-Bangla Medical College, Barisal	Radiology & Imaging
074-9290	Md. Abdullah As Sajjad Rahimi	Comilla Medical College, Comilla	Surgery
074-9300	Dr. Syed Md. Muhsin	Chittagong Medical College, Chitagong	Surgery
074-9322	Ahmad Seraji	Dhaka Medical College, Dhaka	Surgery

Election of the Councillors and Executive Committee:
The under mentioned Fellows of the Bangladesh College of Physicians and Surgeons are declared to have been elected Councilors of the Council of the College for a period of four years with effect from 01-03-2009

The newly elected Councillors are:

1. Professor T. I.M. Abdullah-Al-Faruq
2. Professor Abdul Kader Khan
3. Professor Md. Sanawar Hossain
4 Professor Kanak Kanti Barua
5. Professor Mohammod Shahidullah
6. Professor Nazmun Nahar
7. Professor Mohammad Saiful Islam
8 Professor Md. Abul Kashem Khandaker

Members of the Council of the BCPS

1. Professor Nazmun Nahar
2. Professor S.A.M. Golam Kibria
3. Professor Md. Sanawar Hossain
4. Professor Mohammod Shahidullah
5. Professor Mahmud Hasan
6. Professor Ava Hossain
7. Professor Kanak Kanti Barua
8. Professor Abdul Kader Khan
9. Professor Quazi Deen Mohammad
10. Professor Choudhury Ali Kawser
11. Professor Md. Ruhul Amin
12. Professor A.H.M. Towhidul Anowar Chowdhury
13. Professor Sayeba Akhter
14. Professor M.A. Majid
15. Professor T.I.M. Abdullah-Al-Faruq
16. Professor Mohammad Saiful Islam
17. Professor Md. Abul Kashem Khandaker
18. Professor A. K.M. Anowarul Azim
19. Professor Rashid-E-Mahbub
20. Professor A.K.M. Mahbubur Rahman

The Ministry of Health & Family Welfare, Government of the People's Republic of Bangladesh has nominated following four Fellows as Councillors.

1. Professor Waliullah (Deceased)
2. Professor Rashid-E-Mahbub
3. Professor A.K.M. Mahbubur Rahman
4. Professor Ava Hossain
5. Professor A. K.M. Anowarul Azim
   (in place of late Professor Waliullah)

Executive Committee:

President	:	Professor Nazmun Nahar
Senior Vice- President	:	Professor S.A.M. Golam Kibria
Vice-President	:	Professor Md. Sanawar Hossain
Treasurer	:	Professor Mohammad Shahidullah
Member	:	Professor Mahmud Hasan Professor Ava Hossain
Honorary Secretary	:	Professor Kanak Kanti Barua

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Examination Committee:

Professor S.A.M. Golam Kibria	Chairperson
Professor Mahmud Hasan	Member
Professor Latifa Shamssuddin	"
Professor Quazi Deen Mohammad	"
Professor Chowdhury Ali Kawser	"
Dr. (Major Gen.) A.K.M. Zafrullah Siddiq	"
Professor Ava Hossain	"
Professor Syed Atiqul Haq	"
Professor Md. Ruhul Amin Miah	"

Reference Committee:

Professor Nazmun Nahar	Chairperson
Professor A. K.M. Anowarul Azim	Member
Professor Rashid-E-Mahbub	"
Professor Md. Sanawar Hossain	"
Professor Md. Nazrul Islam	"
Professor A.K.M. Mahbubur Rahman	"
Professor T.I.M. Abdullah -Al-Faruq	"
Professor Md. Ruhul Amin	"

Finance & Tender Committee:

Professor Md. Sanawar Hossain	Chairperson
Professor Sayeba Akhter	Member
Professor A. N. M. Zia-ur-Rahman	"
Professor Md. Abul Kashem Khandaker	"
Professor Mohammad Saiful Islam	"
Professor Mohammad Shahidullah	"
Professor Md. Moyeenuzzaman	"
Professor M.A. Majid	"

Disciplinary Committee:

Professor A.H.M. Ahsanullah	Chairperson
Prof. A.H.M. Towhidul Anwar Chowdhury	Member
Professor Md. Tahir	"
Professor Rashid-E-Mahbub	"
Professor Md. Abdul Mobin Khan	"
Professor A.K.M. Mahbubur Rahman	"

Museum Committee:

Professor Humayun Kabir Chowdhury	Chairperson
Professor A.N.M. Atai Rabbi	Member
Professor Syed Mukarram Ali	"
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Dr. Kamal Sayeed Ahmed Chowdhury	"
Dr. (Lt. Col.) Md. Rafiquzzaman	"
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Professor Syed Serajul Karim	"
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Professor S.M. Mahbub Alam	"
Professor A.R.M. Luthful Kabir	"
Professor Fakhruddin Mohammad Siddiqui	"
Professor Md. Fazlul Kadir	"
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Dr. Dewan Golam Md. Akaiduzzaman	"
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Dr. Dipi Barua	"
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Professor Parveen Shahida Akhter	"
Dr. (Lt Col) Asadullah Mohammad Hossain Saad	"
Dr. Nila Kantha Paul	"
Professor (Brig. Gen.) Zuberul Islam Chowdhury	"
Professor Md. Mokles Uddin	"
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Prof. Syed Md. Akram Hussain	"
Professor Dr. Md. Enamul Haque	"
Professor Md. Iqbal Hossain	"
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Professor(Brig. Gen.) Faruk Ahmed	"
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Dr. Mohammad Golam Rabbani	"
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Brig. Gen. (Professor) A.K. Md. Mustafa Abedin	"
Professor Alamgir Kabir	"
Dr. Shahed Ahmad Chowdhury	"
Dr. (Lt. Col.) Md. Mohibur Rahman	"
Dr. Rukhsana Khanam	"
Dr. (Major) A.K.M. Abu Yousuf	"
Dr. (Major) Suraiya Akhtar	"
Dr. (Lt. Col.) Md. Mizanur Rahman	"
Dr. Niru Nazmun Nahar	"
Dr. Mohammad Humayun	"
Dr. Mohammed Mosleh Uddin	"
Professor Mohiuddin Ahmed Khan	Member secretary

Faculty of Surgery (Including Dentistry):

Professor Md. Abdul Awal	Chairperson
Professor M.A. Majid	Member
Professor Shamsuddin Ahmed	"
Professor A.N.M. Atai Rabbi	"
Professor Rashid-E-Mahbub	"
Professor Md. Shelim Bhuiyan	"
Professor M. Alimuzzaman	"
Professor Md. Sanawar Hossain	"
Professor S.A.M. Golam Kibria	"
Professor Abu Ahmed Ashraf Ali	"
Professor A.K.M. Shariful Islam	"
Professor Md. Khalilur Rahman	"
Professor Humayun Kabir Chowdhury	"
Professor Abdus Salam	"
Professor A.K.M. Mahbubur Rahman	"
Professor Mohammad Ali	"
Professor Shafiqul Haque	"
Professor Abdus Sobhan Pramanik	"
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Professor Syed Serajul Karim	"
Professor Hasan Md. Abdur Rouf	"
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Professor Abdul Haque	"
Dr. (Major Gen.) A.K.M. Zafrullah Siddiq	"
Professor Md. Shahid Hossain	"

Professor Mohammed Afzal Hossain	"
Maj. General (Dr.) Md. Ali Akbar (Retd.)	"
Professor Md. Abdul Gafur Miah	"
Professor Md. Mazibar Rahman	"
Professor Mohammad Saiful Islam	"
Professor Syed Mahmudur Rahman	"
Professor Moudud Hossain Alamgir	"
Professor Faruk Ahmad	"
Professor A.H.M. Shamsul Alam	"
Brig. Gen. (Dr.) Harunur	"
Professor Md. Shahidur Rahman	"
Professor Israil Biswas	"
Professor Md. Mizanur Rahman	"
Professor Golam Muhiuddin Akbar Chowdhury	"
Professor T.I.M. Abdullah -Al-Faruq	"
Professor Zahidul Haq	"
Professor Omar Faruque Yusuf	"
Professor A.M.S.M. Sharfuzzaman	"
Professor Mirza Mahbubul Hasan	"
Professor Md. Abdul Hannan	"
Professor Md. Shahid Karim	"
Brig. Gen. (Dr.) Bijoy Kumar Sarkar	"
Professor Syed Mahbubul Alam	"
Professor Feroze Quader	"
Professor (Brig. Gen.) Md. Saidur Rahman	"
Dr. Md. Mujibur Rahman Howlader	"
Dr. S.M. Anwar Sadat	"
Dr. A.Z.M. Mostaque Hossain	Joint Secretary
Dr. Md. Zulfiqur Rahman Khan	Member Secretary

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Professor Md. Quamrul Islam	Member
Professor Aminuddin Ahmed Khan	"
Dr. Md. Taslim Uddin	"
Dr. Md. Shahidur Rahman	"
Dr. Shamsun Nahar	"
Professor Sohely Rahman	"
Professor Md. Habibur Rahman	"
Dr. Md. Hilalul Islam	"
Dr. Abul Khair Mohammad Salek	"
Dr. Md. Moniruzzaman Khan	"
Dr. Md. Mahfuzur Rahman	"
Dr. Suzon Al Hasan	"
Dr. Md. Ahsnullah	"
Dr. Md. Shah Zaman Khan	"
Dr. Md. Abdus Shakoor	"
Prof. Md. Abdur Rashid	Member secretary

Faculty of Otolaryngology:

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Professor Md. Alauddin	"
Professor Pran Gopal Datta	"
Dr. Brig. Gen. (Retd.) Syed Ahsan Karim	"
Professor Nilkanta Bhattacharjee	"
Professor Md. Abdullah	"
Professor Md. Kamrul Hassan Tarafder	"
Professor Mohammad Zillur Rahman	"
Professor S.M. Khorshed Alam Mazumder	"
Professor Md. Monwar Hossain	"
Professor (Col) Md. Abdul Mannan	"
Professor Khabir Uddin Ahmed	"
Dr. Belayat Hossain Siddiquee	"
Professor Md. Ashraful Islam	"
Dr. Mir Hasan Shaheel Mahmood	"
Dr. Mahmudul Hassan	"
Dr. Md. Abu Hanif	"
Dr. Md. Azharul Islam	"
Brig.Gen. (Prof.) Muhammad Shahid Khurshid Alam	"
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Professor Shah Md. Bul Bul Islam	"
Professor Sk. Md. Abdul Mannaf	"
Professor A.S.M. Kamaluddin	"
Professor Ava Hossain	"
Professor Jamal Nizamuddin Ahmed	"
Professor Md. Hazrat Ali	"
Professor Syed Maruf Ali	"

Professor Md. Arif Mian	"
Professor Brig. Gen. (Retd.) Nazrul Islam	"
Brig. Gen. (Dr.) Md. Anwar Hossain	"
Professor Jalal Ahmed	"
Professor Md. Shahidul Alam	"
Professor Deen Mohd. Noorul Huq	"
Professor Md. Shamsul Haque	"
Professor Abu Ahmed Mohiuddin	"
Professor Md. Nazrul Islam	"
Dr. Md. Shahidul Islam (Faruque)	"
Professor Md. Hassan Shahid Suhrawardy	"
Dr. Ashraf Sayeed	"
Dr. Md. Abid Kamal	"
Dr. Md. Mizanur Rahman	"
Dr. Md. Shafiqul Islam	Member Secretary

Faculty of Psychiatry:

Professor Syed Kamaluddin Ahmed	Chairperson
Professor A.K.M. Nazimuddowla Chowdhury	Member
Professor M. A. Sobhan	"
Professor Md. Rezaul Karim	"
Professor Md. Nazmul Ahsan	"
Professor Saroj Kumar Das	"
Professor Md. Enayet Karim	"
Professor Abul Hasnat Mohammad Firoz	"
Professor Md. Shah Alam	"
Dr. Rezawana Quaderi	"
Professor Waziul Alam Chowdhury	"
Professor Mohammad Ahsanul Habib	"
Professor Md. Sayadul Islam Mullick	"
Dr. Jhunu Shamsun Nahar	"
Dr. A.H.M. Mustafizur Rahman	"
Dr. Mohammad Tazul Islam	"
Professor Mahmood Hasan	"
Dr. Md. Abdul Hamid	"
Dr. Md. Faruq Alam	"
Professor (Brig. Gen.) Md. Sajjadur Rahman	"
Dr. Abdullah Al-Mamun	"
Dr. Mohammad Delowar Hossain	"
Professor Md. Golam Rabbani	Member Secretary

Faculty of Obstetrics & Gynaecology:

Professor A. K. M. Anowarul Azim	Chairperson
Professor Abdul Bayes Bhuiyan	Member
Professor A.H.M. Towhidul Anowar Chow.	"
Professor Shahla Khatun	"
Professor M. Anwar Hussain	"
Professor Sultana Jahan	"
Professor Latifa Shamsuddin	"
Professor Sultana Razia Begum	"
Professor Rehana Begum	"
Professor Mahmuda Khatun	"
Professor Anowara Begum	"
Professor Kohinoor Begum	"
Professor Rahima Begum	"
Professor Sayeba Akhter	"
Professor Md. Shah Alam	"
Professor Nasima Begum	"
Professor Hosne Ara Begum	"
Professor Shamsun Nahar	"
Professor Merina Khanam	"
Professor Md. Azizul Islam	"
Professor Ameena Majid	"
Professor Sameena Chowdhury	"
Professor Laila Arjumand Banu	"
Professor Fatema Begum	"
Professor Parveen Fatima	"
Professor Atika Begum	"
Professor Firoza Begum	"
Professor Saleha Begum Chowdhury	"
Professor Farhana Dewan	"
Professor Maliha Rashid	"
Professor Farhat Hussain	"
Professor Rowshan Ara Begum	Member Secretary

Faculty of Basic Medical Sciences:

Maj. Gen. Md. Jalal Uddin	Chairperson
Professor Shah Md. Monir	Member
Professor Syed Mukarram Ali	"
Professor M.A. Hai	"
Professor A.K.M. Nurul Anowar	"
Professor S.A.R.Chowdhury	"
Professor Tehmina Hussain	"
Professor Md. Nazrul Islam	"
Professor Saleha Husain	"
Prof. Subhagata Chowdhury	"
Professor Md. Ruhul Amin Miah	Member
Professor Rokeya Begum	"
Professor Md. Zahurul Haque	"
Professor Iqbal Arslan	"
Professor Md. Sahadat Hossain	"
Professor Abdullah Akhtar Ahmed	"
Professor A. Khaleque Akond	"
Professor Badrul Islam	"

Professor Md. Ruhul Amin	"
Professor Naima Muazzam	"
Professor Mohammad Kamal	"
Professor Kh. Manzare Shamim	"
Professor Nadira Islam	"
Professor Md. Motahar Hossain	"
Professor Md. Aftabuddin Ahmed	"
Professor Shah Alam	"
Professor Shamim Ara	"
Professor Kh. Md. Shefyetullah	"
Professor Majbun Ara Begum	"
Professor Zinnat Ara Begum	"
Professor Afsana Karim	"
Professor Humaira Naushaba	"
Professor Faruk Ahmed	"
Professor Jalaluddin Ashraful Haque	"
Professor Selina Ahmed	"
Prof. Md. Mizanul Haque	"
Professor Abu Taher	"
Dr. (Brig. Gen.) Shazadi Nilufar	"
Dr. (Lt. Col.) Selina Akhtar	"
Dr. Zahedul Karim Ahmed	"
Professor Md. Mozammel Hoque	Member Secretary

Faculty of Paediatrics:

Professor Md. Fazlul Haque Nazir	Chairperson
National Prof. M. R. Khan	Member
Prof. M.Q.K. Talukder	"
Professor Md. Nurul Islam	"
Professor Md. Hamidur Rahman	"
Professor Md. Abdul Mannan Miah	"
Professor Md. Moazzam Hossain	"
Professor Md. Monimul Haque	"
Professor Kishwar Azad	"
Professor Md. Sirajul Islam	"
Professor Hosne Ara Begum	"
Professor Md. Abdul Halim	"
Professor Choudhury Ali Kawser	"
Professor A. F.M. Salim	"
Professor Naila Zaman Khan	"
Professor Khan Nizamuddin	"
Professor Md. Nurul Absar	"
Professor Mohammad Hanif	"
Professor A.S.M. Bazlul Karim	"
Professor Afiqul Islam	"
Prof. S.M. Shahnawaz Bin Tabib	"
Professor Md. Mizanur Rahman	"
Professor A.R.M. Luthful Kabir	"
Professor Ainun Afroze	"
Professor Choudhury Habibur Rasul	"
Professor Syed Zahid Hossain	"
Professor Soofia Khatoon	"
Professor Abdul Hannan	"
Professor Mohammod Shahidullah	"
Professor Syeda Afroza	"
Professor Md. Ruhul Amin	"
Professor Shahana Akhter Rahman	"
Professor Tahmina Begum	"
Professor Md. Nazrul Islam	"
Professor Md. Iqbal Bari	"
Professor Golam Muin Uddin	"
Professor Md. Abid Hossain Molla	"
Professor Mohammad Nurul Huq	"
Dr. Nazneen Akhter Banu	Joint Secretary
Professor Md. Ekhlasur Rahman	Member Secretary

Faculty of Family Medicine:

Professor Sultana Razia Begum	Chairperson
Professor Nazmun Nahar	Member
Professor S.A.M. Golam Kibria	"
Professor Md. Sanawar Hossain	"
Professor Mohammad Shahidullah	"
Professor Mahmud Hasan	"
Professor Ava Hossain	"
Professor Kanak Kanti Barua	"
Professor Abdul Kader Khan	"
Professor Quazi Deen Mohammad	"
Professor Choudhury Ali Kawser	"
Professor Md. Ruhul Amin	"
Professor A.H.M. Towhidul Anowar Chowdhury	"
Professor Sayeba Akhter	"
Professor M.A. Majid	"
Professor T.I.M. Abdullah-Al-Faruq	"
Professor Mohammad Saiful Islam	"
Professor Md. Abul Kashem Khandaker	"
Professor A.K.M. Mahbubur Rahman	"
Professor Rashid-E-Mahbub	"
Professor A. K.M. Anowarul Azim	"
Professor Abdul Bayes Bhuiyan	"
Professor A.H.M. Ahsanullah	"
Professor Shamsuddin Ahmed	"
Professor M. A. Majed	"
Professor Mobin Khan	"

Professor Mirza Mazharul Islam	“
Professor A.N.M. Atai Rabbi	“
Professor Tofayel Ahmed	“
Professor Md. Abul Faiz	“
Professor Emran Bin Yunus	“
Professor Nooruddin Ahmed	“
Professor Firoz Ahmed Quraishi	Member Secretary

Faculty of Medicine :

Professor Abul Khair Md. Rafique Uddin	Chairperson
National Professor Nurul Islam	Member
Professor Md. Nurun Nabi	“
Professor Firdous Ara J. Janan	“
Professor Md. Jalaluddin	“
Professor Mahmud Hasan	“
Professor Tofayel Ahmed	“
Professor Md. Fazlul Haque	“
Professor Mobin Khan	“
Professor Md. Zahangir Kabir	“
Professor Naseem Akhter Chowdhury	“
Professor Md. Habibur Rahman	“
Professor Kaniz Moula	“
Professor Hasina Banoo	“
Professor Dipti Chowdhury	“
Professor Md. Nazrul Islam	“
Professor Paritosh Kumar Baral	“
Professor Md. Abul Faiz	“
Professor Md. Abdul Jalil Chowdhury	“
Professor Zafar Ahmed Latif	“
Professor Khokan Kanti Das	“
Professor Md. Gofranul Hoque	“
Professor A.K.M. Anisul Haque	“
Professor Quazi Deen Mohammad	“
Professor A.K.M. Khorshed Alam	“
Professor Ghulam Mahmood	“
Professor Md. Abul Kashem Khandaker	“
Maj. Gen. (Dr.) Md. Abdul Moyeed Siddiqui	“
Professor Md. Mustafizur Rahman	“
Dr. (Brig. Gen.) Md. Rabiul Hossain	“
Professor Chandanendu Bhushan Sarker	“
Professor Syed Wahidur Rahman	“
Major Gen. (Professor) Md. Golam Rabbani	“
Professor Emran Bin Yunus	“
Professor Shamim Ahmed	“
Professor Quazi Tarikul Islam	“
Professor Mohammed Abu Azhar	“
Professor Md. Abu Bakar	“
Professor Syed Atiqul Haque	“
Professor Hasan Askari Md. Nazmul Ahsan	“
Professor Fakhruddin Mohammad Siddiqui	“
Professor Md. Azizul Kahhar	“
Professor Md. Rajibul Alam	“
Professor Md. Rashidul Hassan	“
Professor Md. Amirul Haque	“
Professor Md. Enamul Karim	“
Professor Nooruddin Ahmad	“
Professor Md. Muhibur Rahman	“
Professor Md. Fazlul Kadir	“
Professor Khwaja Nazim Uddin	“
Professor Khan Abul Kalam Azad	“
Professor Md. Abul Ahbab	“
Professor Md. Ali Hussain	“
Professor Syed Mainul Hasan Sadik	“
Professor Md. Zakir Hossain	“
Professor Taimur A.K. Mahmud	“
Professor A.R.M. Saifuddin Ekram	“
Professor Md. Abul Bashar	“
Professor Muhammad Rafiqul Alam	Member Secretary

Obituary

The following Fellows who died between January to May 2009

PROFESSOR WALIULLAH

Professor Waliullah, who died on 23 March, 2009, was a highly respected teacher and a pioneering and influential figure in the medical communities in Bangladesh.

He was a pioneer specialist in Medicine. In honour of his work in promoting medical education and teaching in Bangladesh, he was awarded fellowship (without examination) in 1974 from Bangladesh College of Physicians and Surgeons (BCPS).

He was punctilious, diligent and with a high moral sense. He will be remembered for his compassion and caring, integrity and high endeavor.

He was the Principal of Dhaka Medical College. He served the nations in various capacities.

He was the government nominated Councillor of BCPS from March, 2001 to March, 2003 and again he was nominated by the government as Councillor for four years from March,2009.

He had been suffering from many chronic disease for a long time.

PROFESSOR CHOWDHURY BADRUDDIN MAHMOOD

Professor Chowdhury Badruddin Mahmood was an outstanding personality and a leader in specialist of Paediatrics. He was a distinguished student and passed the Fellowship examination in Paediatrics on July, 1978 from Bangladesh College of Physicians and Surgeons (BCPS).

He loved the happy and friendly atmosphere in the Hospital. He was a man of quality and tireless energy, a careful, who paid always enormous attention on his duty. He was a great teacher and had the ability to make everyone in the team feel valued.

He died on 7th April,2009.

DR. DEBASHIS SAMADDAR

Dr. Debashis Samaddar died on 24 May 2009. He was started his service life from Dinajpur Medical College. He passed fellowship examination in Radiotherapy on July, 1999 from Bangladesh College of Physicians and Surgeons (BCPS). Afterwards he was out of the country for his service.

DR. NASIR AHMED

Dr. Nasir Ahmed was a brilliant student. He passed fellowship examination in Psychiatry on July, 1990 from Bangladesh College of Physicians and Surgeons (BCPS). After passing fellowship, he enjoyed his service life in Newzeland. He died on 26 May, 2009 by heart attack.