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The "Journal of the Medical College for Women & Hospital" is a project of the Medical & Health Welfare Trust, Uttara, Dhaka, Bangladesh.

The journal is a half yearly (January & July) multidisciplinary medical journal. High quality articles written in English, dealing with aspects of clinical, academic or investigative medicine or research will be welcome. Emphasis is given on matters relating to medicine & biomedical sciences in Bangladesh in particular, though papers are welcome from all over the world.

The journal publishes original articles, review articles, case reports, letters to the editor, editorials and short/special communications. The editorial board wishes to minimize delay between manuscript submission, decision and publication. Accordingly, authors are requested to follow carefully the instructions set out under "Information and instruction for Authors".

OBITUARY

Professor Dr. Md. Rafiqul Islam, MBBS (Dhaka), FRCS (Edinburgh)
Professor & Head, Department of Surgery, Medical College for Women & Hospital
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Professor Dr. Md. Rafiqul Islam, Founder Professor of Surgery & Head, Department of Surgery, Medical College for Women & Hospital (MCW&H), Uttara, Dhaka who will always be remembered as one of the prominent surgeons of Bangladesh and for his devotion and contribution in the field of surgery, died suddenly on 22 August 2003.

He was born in an aristocratic muslim family on 14 August 1935 in Pitalganj village under Hossainpur Upazilla in the district of Kishoregonj. He obtained his MBBS degree in 1959 from Dhaka Medical College (DMC), Dhaka under the University of Dhaka, Bangladesh and had a highly successful career in surgery.

He started his career in the Government Health Service at Chittagong Medical College Hospital (CMCH), Chittagong where he worked as an Assistant Surgeon (10 May 1960 to 23 October 1960), Emergency Medical Officer (24 October 1960 to 23 June 1962), Clinical Assistant (24 June 1962 to 09 April 1963) and Registrar (10 April 1963 to 26 September 1963). He earned excellent reputation while working at CMCH, Chittagong.

He then went to the United Kingdom and Ireland on deputation for higher studies from 27 September 1963 to 10 August 1967. He successfully obtained his Fellowship of the Royal College of Surgeons, Edinburgh (FRCS) in 1967 and returned to his homeland immediately and joined back to the Government Health Service at CMCH, Chittagong as Assistant Surgeon (16 August 1967 to 15 August 1968). He was soon promoted to the academic ranks in which capacity he served at various medical colleges of Bangladesh. He was Assistant Professor of Surgery at Rajshahi Medical College (RMC), Rajshahi (01 February 1968 to 10 May 1972); Associate Professor of Surgery at MAG Osmani Medical College (MOMC), Sylhet (11 May 1972 to 30 August 1977). He was soon given the post of Professorship and he served as Professor of Surgery at MOMC, Sylhet (30 August 1977 to 10 September 1979); Sher-e-Bangla Fazlul Huq Medical College, Barishal (16 September 1979 to 22 March 1985); CMC, Chittagong (23 March 1985 to 15 July 1990) and DMC, Dhaka (16 July 1990 to 12 August 1992). Finally, he went on leave preparatory to retirement (LPR) on 13 August 1992 and retired from the government health service on 13 August 1993.

With this brilliant academic background in the field of surgery, Prof. Dr. Md. Rafiqul Islam joined the Medical College for Women & Hospital (MCW&H) as the Founder Professor of Surgery and Head, Department of Surgery in 1992. He served the MCW&H with dedication for more than 11 years not thinking much for his financial gains from the MHWT, Uttara. He was a man with strong ethical standards, personality, sense of humour and belongingness. All of us would ever remember him for his devotion and contribution in surgery and tremendous love and affection for his colleagues and students alike.

A devoted family man, Prof. Islam was married to Dr. Rowshan Ara Begum who is also a medical graduate (MBBS). He is survived by his wife and four children (two sons and two daughters) who remember him as a caring and loving husband and father respectively.

May Allah, the most powerful and merciful, grant his soul eternal peace.

JOURNAL OF THE MEDICAL COLLEGE FOR WOMEN & HOSPITAL
INFORMATION AND INSTRUCTION FOR AUTHORS

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Title page : The title page must contain : 1) title of the article, 2) correct names of each author plus academic degrees for each, 3) each author's official academic and/or

clinical title and affiliation, 4) name and address of the institution(s), 5) a short-title and key words, 6) name and address, including telephone and fax numbers, of the author to whom correspondence should be sent. For case reports, it is recommended that the number of authors be limited to four or five.

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   Moxon ER (Editor). Modern Vaccines, 3rd edition; London: Edward Arnold; 2002

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ANNOUNCEMENT

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The Editorial board invites scientifically good quality articles/papers in medical sciences for publication in the future issues of JMCWH. Presently the JMCWH is published biannually in January and July. The articles may be submitted at any time as it is a continuous process. Please consult the "**Instructions for authors**" regarding **vancouver style** and submit the manuscripts to the following address:

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JOURNAL OF THE MEDICAL COLLEGE FOR WOMEN & HOSPITAL

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CONTENTS

Editorial

DENGUE VACCINE: WOULD IT BE AVAILABLE ?

The editorial board considers itself lucky for being able to welcome the readers to Vol 2, No 1, January 2004 issue of JMCWH. The editorial board received many interesting articles of medical importance of which ten have been published in this current issue of JMCWH. The leading article was "Clinical profile and treatment outcome of dengue fever- A prospective study".

Dengue fever (DF) is a mosquito-transmitted acute febrile infectious disease caused by an arbovirus called dengue virus belonging to the Flavivirus (genus) and Flaviviridae (family). Dengue virus containing single stranded ribonucleic acid (RNA) has four serotypes (DEN-1, DEN-2, DEN-3, DEN-4); infection with one serotype provides life-long homotypic immunity, but not against the other serotypes. During a dengue infection, the presence of heterologous antibodies either maternally acquired or from a previous dengue infection has been shown to increase the risk for severe disease, i.e. dengue haemorhagic fever (DHF) and dengue shock syndrome (DSS)[1,2,3]. World health organization (WHO) currently estimates that there are about 50-100 million cases of DF including 2.5-5.0 million cases of DHF/DSS and 30,000 deaths each year[4]. DF/DHF/DSS has dramatically emerged as a major public health problem globally including Bangladesh for which the reasons are complex and not well understood, although several important epidemiological factors have been identified[5,6]. DF/DHF/DSS is a zoonosis and is maintained by a forest cycle involving wild monkeys and jungle mosquitoes and such a cycle was demonstrated in Malaysia. Dengue viruses are transmitted from human to human by Aedes mosquitoes and A. aegypti is the most important epidemic vector, although other species such as A. abopictus have also been incriminated as a secondary vector[7,8]. **No mosquito bites, no dengue fever. So what can we do about it ?**

Regarding prevention and long term control measures, presently WHO recommendation emphasizes on sustainable, community-based, integrated mosquito vector (A. aegypti) control with the following approaches: environmental modification, environmental manipulation, personal protection with limited reliance on insecticides and increasing awareness in the community about DF/DHF/DSS and control measures of the mosquito that transmits it[8,9]. However, this mosquito is well adapted to urban environments because it breeds inside homes, and has proven expensive and very difficult to control even with stringent efforts [5]. As a result, current dengue control programmes have been unable to protect the millions of people particularly children at risk of infection and illness[9]. Secondly, no specific treatment for DF/DHF/DSS is yet available, although case-fatility rates for DHF/DSS can be reduced from ≥ 20 % to < 1.0 % by intensive supportive

therapy[8]. These points clearly hightens the urgent need for a protective and cost –effective dengue vaccine for prevention and long-term control of dengue infection. The results of a recent survey on policymakers' view in four South Asian countries (Cambodia, Indonesia, Philippines, Vietnam), in fact, suggested urgent need and favourable conditions for public and private sector markets for dengue vaccine[10].

Since the discovery of vaccination against smallpox by Edward Jenner in 1797 nearly 207 years ago, many vaccines have been developed, being used in routine immunization schedule and the world still needs many more new vaccines including one against DF/DHF/DSS [11,12,13]. The ultimate question that arises is **"Dengue vaccine: would it be available"?** The development of a vaccine offers the potential for effective prevention, long-term control and possible eradication of dengue infection. The good news is that there are several promising dengue vaccines under development including four live-attenuated chimeric vaccines generated by introducing premembrane (prM) and envelope (E) genes from dengue virus into full length cDNA of attenuated yellow fever virus or dengue vaccine viruses (panel)[14]. The first of these tetravalent vaccine developed at Mahidol University, Bangkok and licensed to Aventis Pasteur, Lyon, France, has been successfully tested through Phase-I and Phase-II clinical trials and met the serologic criteria required for an extended phase-III clinical trial [13]. In a separate effort at the Walter Reed Army Institute of Research, Silver Spring, MD, USA, many live-attenuated candidate vaccines have been developed, tested in phase-I clinical trials and the successful one is licensed to GlaxoSmithKline, Rixensart, Belgium and phase-II and III trials are planned for 2003-04[14,15]. At the Department of Medicine and the Centre for Vaccine Development, University of Maryland School of Medicine, Baltimore, MD, USA, sixteen formulations of a tetravalent live- attenuated dengue vaccine were developed and three of them proved to be sufficiently attenuated clinically justifying further testing in phase-II and phase-III clinical trials[16].

With the safety concerns about enhanced disease occurring in an incompletely immunized individual, can the promise of dengue vaccines be realized? To consider this issue, Rockefeller Foundation, USA; International Vaccine Institute (IVI), Soul, Korea; Paediatric Dengue Vaccine Initiative (PDVI) and WHO have been working together on a four –point programme, but with limited budgets. This initiative will certainly accelerate the development of dengue vaccine. Recently, on 9 September 2003, Bill & Melinda Gates Foundation, USA announced a **US$ 55 million grant** to the 1V1 to support the PDV1 to develop and conduct more clinical trials for Dengue vaccine in Southeast Asia and South America[17]. This would help to overcome financial constrains in conducting more clinical trails to find answers to certain fundamental questions such as immunogenicity, safety, durability of immunity and reactogenicity of the dengue vaccine for ultimate use. **Without valid answers to these fundamental questions, "A cost- effective, safe and protective dengue vaccine: would it be possible"?** By moving forward aggressively with both laboratory research and clinical trials, scientists can help prevent the sufferings of hundreds of thousands of children every year. There is a consensus that a window of opportunity now exists to bring an effective and safe dengue vaccine on stream quickly.

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ORGINAL ARTICLE

CLINICAL PROFILE AND TREATMENT OUTCOME OF DENGUE FEVER – A PROSPECTIVE STUDY

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ABSTRACT

This prospective study was carried out to evaluate the clinical profile and treatment outcome of 60 cases of Dengue fever. All the patients enrolled in this study were admitted in the Departments of Medicine and Paediatrics, Combined Military Hospital (CMH), Dhaka during the period of July to September 2002. Out of these 60 patients, 52 (86.7%) were male and 8 (13.3%) were female. Age of the patients ranged from 41/2 years to 55 years. 54 patients (90%) had classical dengue fever (CDF), 3 patients (5%) were documented to have Gr-I dengue haemorrhagic fever (DHF) and the remaining 3 (5%) had Gr-II DHF. Average duration of fever and hospital stay was 7.2 days and 7.1 days respectively. Fever and headache were the predominant symptoms in this series. 59 patients (98.4%) presented with fever and 48 patients (80%) had intense headache during admission. Various types of skin rash were present in 27 patients (45%), 18 patients (30%) had ascites, 17 patients (28.3 %) had hepatomegaly and 16 cases (26. 7%) were documented to have pleural effusion. Bleeding manifestations were documented in 16 patients (26.7%) in this series. Gum bleeding and sub conjunctival bleeding were the commonest bleeding manifestations and were present in 15% and 8. 3% of the patients respectively. Four patients (6. 7%) developed severe gastrointestinal bleeding and required fresh blood transfusion, 57 patients were treated symptomatically and had an uneventful recovery. Seventeen dengue patients (28.3 %) had low platelet count but only 3 (5%) cases required platelet concentrate as they developed severe symptomatic thrombocytopenia. There was no fatality in this series.

[J Med Coll Women Hosp 2004; 2(1): 4-9]

Indexing words : Dengue fever Dengue hemorrhagic fever

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INTRODUCTION

Dengue fever is a viral disease caused by any one of the four serotypes of the single stranded RNA containing dengue virus transmitted by Aedes aegypti mosquitoes. It is epidemic in the America, southeast Asia, Western Pacific and the Eastern Mediterranean[1,2,3]. Dengue fever has become an important public health problem in Bangladesh. The disease caused by dengue virus infection is classified into two clinical entities: dengue fever and dengue haemorrhagic fever; it is the latter that is divided into four stages[4]. The features of dengue fever and dengue haemorrhagic fever are similar at the onset of illness. Dengue haemorrhagic fever is characterized by abnormal capillary permeability, which usually develops as the fever subsides, with shift of fluid from intravascular to extravascular space.

This process leads to haemoconcentration and mild to moderate circulatory failure (dengue haemorrhagic fever grades I to III), which is manifested by profuse sweating, cold skin, increased pulse rate and decreased pulse pressure, blood pressure or both, of varying severity. Profound shock with very low blood pressure and undetectable pulse characterize grade IV dengue haemorrhagic fever, which is also called dengue shock syndrome[4,5].

The year 2001 and 2002 witnessed unprecedented dengue epidemic in Bangladesh and this outbreak caused many unwarranted deaths[6,7]. Early diagnosis of dengue is very important. Timely intervention can prevent unfortunate fatality. The purpose of this study was to determine the clinical pattern and treatment outcome of a cohort of dengue patients treated in the Combined Military Hospital, Dhaka.

MATERIALS AND METHODS

This nonrandomized study was conducted in the Departments of Medicine and Paediatrics, CMH, Dhaka from July 2002 to September 2002. A total of 60 cases of dengue fever were enrolled in the study. The composition of the patients were male and female of all ages and from all socio-economic classes. Signs, symptoms and laboratory data were documented on pre designed protocol. The diagnosis was made on the basis of history, clinical findings, antibody detection, platelet count, ultrasonogram and X-Ray chest. Assessment of the patient's condition included packed cell volume, liver function tests, electrolytes, prothrombin time and partial thromboplastin time etc. The cases were classified according to WHO guidelines[8]. The patients who were negative for antibodies to dengue virus (IgG or IgM class), in spite of clinical suspicion, were excluded from the study.

RESULTS

A total of 60 patients of Dengue fever of various grades were studied. Among these 52 (86.7%) were male and 8 (13.3%) were female. Male to female ratio was 6.5:1. Age range was 41/2 to 55 years (Table-I). Patients representing lower, middle and upper socio-economic classes were 53 (88.3%), 5 (8.3%) and 2 (3.2%) respectively. 54 patients (90%) had classical dengue fever (CDF), 3 patients (5%) were documented to have Gr-I

dengue haemorrhagic fever (DHF) and the remaining 3 (5%) had Gr-II DHF. Gr-III and IV dengue fever was not documented in this series (Table-II).

TABLE 1:Age and sex distribution of patients (N = 60)

Number of patients	60
Age range	
41/2-15 years	3(5%)
>15-35 years	44 (73.3%)
>35-55 years	13 (21.7%)
Male	52 (86.7%)
Female	8 (13.3%)
M : F	6.5:1

TABLE II: Classification of cases as per WHO grading (N = 60)

Classification of cases	No of patients (%)
CDF (Grade 0)	54 (90%)
DHF (Grade I)	3 (5%)
DHF (Grade II)	3 (5%)
DSS (Grade III)	00
DSS (Grade IV)	00

Average duration of fever and hospital stay was 7.2 days and 7.1 days respectively. Fever and headache were the predominant symptoms in this series. 59 patients (98.4%) presented with fever and 48 patients (80%) had intense headache during admission. Except fever and headache other important presenting symptoms include bodyache in 30 patients (50%), skin rash in 27 patients (45%), vomiting in 24 patients (40%) and bleeding manifestations in 16 patients (26.7%) (Table-III). Among the clinical signs ascites was present in 18 patients (30%), hepatomegaly in 17 (28.3%), pleural effusion in 16 (26.7%), pressure blanching in 15 (25%) and subconjunctival haemorrhage was noted in 5 patients (8.3%) (Table-IV).

TABLE III: Symptoms of patients (N = 60)

Symptoms	No of patients (%)
Fever	59 (98.4)
Headache	48 (80.0)
Bodyache	30 (50.0)
Skin rash	27 (45.1)
Vomiting	24 (40.0)
Bleeding manifestations	16 (26.7)
Retro orbital pain	13 (21.7)
Arthralgia	7 (11.7)
Cough/Sorethroat	6 (10.0)
Abdominal pain	5 (8.35)
Lacrimation/ photophobia	3 (5.0)
Rhinorrhoea	2 (3.4)

TABLE IV: Distribution of clinical signs observed (N = 60)

Clinical signs	No of patients (%)
Ascites	18 (30.0)
Hepatomegaly	17 (28.3)
Pleural effusion	16 (26.7)
Pressure blanching	15 (25.0)
Skin erythema	10 (16.7)
Subconjunctival haemorrhage	5 (8.3)
Positive torniquette test	3 (5.0)
Petechae / purpura	2 (3.3)
Echymosis	2 (3.3)
Jaundice	1(1.7)

Gum bleeding, subconjunctival haemorrhage and melaena were the common forms of bleeding manifestations in the present series and were present in 15%, 8. 3% and 6.7% of patients respectively (Table-V).

Leucopenia was documented in 19 patients (31.6%) and 17 (28. 3%) patients had low platelet count. 5 patients had impaired liver function tests but clinical jaundice was documented in only one patient (Table-VI).

TABLE V: Patterns of haemorrhagic manifestation (N = 60)

Bleeding manifestation	No of patients (%)
Gum bleeding	9 (15.0)
Sub conjunctival bleeding	5 (8.3)
Melaena	4 (6. 7)
Cutaneous bleeding	4 (6.7)
Epistaxis	3 (5.0)
Haemorrhagic gastritis	3 (5.0)
Haematuria	2 (3.3)
More than one bleeding manifestation	8(13.3)

TABLE VI: Haematological Parameters (N = 60)

Haematological Parameters	No of patients(%)	Remarks
Haemoglobin (>16 gm/dl)	4 (6.7)	Highest 17.8 gm/dl
Haematocrit (>45%)	8 (13.3)	Highest 56%
Leucopenia (<4 x 109/L)	19 (31.6)	Lowest 1x109/L
Leucocytosis (>11 x 109/L)	3 (5.00)	Highest 15 x 109/L
Neutrophil (>70%)	9 (15.0)	Height 80%
Neutrophil (<40%)	4 (6.7)	Lowest 22%
Lymphocyte (>40%)	6 (10.0)	Height 68%
Platelet (<100 x 109/L)	17 (28. 3)	Lowest <10x109/l
High ALT (>60 i u)	5 (8.3)	Clinical jaundice in 1 patient

DISCUSSION

Dengue viruses are found virtually throughout the tropics and cause an estimated 50-100 million illnesses annually,including 250000 – 500000 cases of dengue haemorrhagic fever-a severe manifestation of dengue and 24000 deaths[9]. Four dengue serotypes are recognized. Infection with one serotype is thought to produce lifelong immunity to that serotype but only a few months immunity to the others[9]. All four types can lead to any grade of dengue infection but in Bangladesh Den-2 and Den-3 are common[7].

Classic dengue is more commonly seen among older children, adolescents and adults. In our study, male female ratio was 6.5:1. This may not reflect the actual sex differences of dengue fever in the community because this study was conducted in an army hospital and the main bulk of the patients of the military hospital are male. Possibly for that reason males predominate over females in this series.

Bleeding manifestations were documented in 16 patients (26.7%) in this series. Gum bleeding and subconjunctival bleeding were the commonest bleeding manifestations and were present in 15% and 8.3% of the patients respectively. Among the clinical signs skin rash was present in 27 cases (45%), ascites was noted in 18 patients (30%) and hepatomegaly in 17 (28.3%). Enlargement and tenderness of the liver has been reported in up to 40% of patients in some recently published data[9,10]. carried out in Chennai. Fulminant hepatic failure have also been documented in dengue haemorrhagic fever[11].

Low platelet count with leucopenia are strongly associated with dengue infections[12,13,14]. In our series 19 patients (31.7%) had leucopenia and 17 patients (28.3%) had low platelet count. Clinical profile of the disease in this series can be compared to a similar study carried out in the same institute in 2000 by Hussain et al. Haemorrhagic manifestations were present in 19.86% cases in that study[7]. In our series it was 26.7% but it was found to be much lower than the study of Agarwal et al which found 54% and Wali et al 56.4% cases of haemorrhagic manifestations in India[15,16]. Gum bleeding was the commonest form of bleeding manifestation in this series.15% of patients had this bleeding problem. In the previous study of Hussain et al gum bleeding was found in 27.53% of cases. In that series subconjunctival haemorrhage was the commonest bleeding manifestation[7]. Incidence of bleeding gums was upto 27% in some recently published series conducted with Thai adults[14].

Low platelet counts do not predict clinically significant bleeding in dengue. It follows that platelet or blood transfusions should not be administered based upon platelet count alone[17]. Dengue haemorrhagic fever or dengue shock syndrome cases frequently have compensated consumptive coagulopathy that seldom requires treatment. Bleeding is most likely caused by activated platelets resulting from damaged capillary endothelium. Dengue haemorrhagic fever and dengue shock syndrome can be safely treated with just normal saline. Colloids should be immediately given to children presenting with a pulse pressure at or below 10 mm of Hg[17,18,19,20]. In our series we did not observe significant relationships with low platelet count and clinically significant bleeding. Only 3 patients (5.0%) required platelet transfusion. Mortality in dengue fever can be as high as 10-20% without early appropriate treatment but it is as low as 0.2% in hospitals with staff experienced in the disease [9,20,21]. In Bangladesh the toll of dengue as documented by the National Control Program was 5551, 2617 and 6104 cases in 2000,2001 and 2002 respectively with DHF frequency of 21.0%,16.9% and 7.1% and case fatality rate of 1.7%,1.6% and 0.9%[22]. All patients in this series were treated symptomatically with intravenous fluid, antibiotics with simple analgesics like paracetamol and the outcome was excellent. There was no fatality in this series. Overall mortality was 3.8% in Agarwal study and 10.91% in Wali study[15,16].

CONCLUSION

Although dengue fever is characterised by spontaneous recovery, dengue haemorrhagic fever may progress through the grades of severity unless treated appropriately. The only method currently available to prevent dengue infections is the control of aedes aegypti, the mosquito vector. This approach has proved expensive and mostly unworkable. As there are considerable overlapping of signs and symptoms of dengue fever with other febrile illnesses, diagnosis may be unduly delayed specifically, if encountered to the uninitiated physician. Awareness of the increasing incidence and escalation of the dengue battle is important both for the epidemiologist as well as the clinician in order to avoid diagnostic negligence,

unnecessary morbidity and some times unfortunate mortality. The diagnosis and management of dengue is fairly simple for most cases. If one adheres to the national guidelines enables one to suspect the condition at an early period and refrain from overdoing. Early detection and careful monitoring of symptoms and investigations are necessary and appropriate simple management may prevent unfortunate fatality.

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ORIGINAL ARTICLE

HEALTH PROBLEMS AMONG THE SENIOR CITIZENS OF UTTARA MODEL TOWN OF DHAKA CITY

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ABSTRACT

Objectives: To determine the pattern of health problems among the senior citizens of Uttara Model Town; **Methods:** The Cross sectional study include 104 elderly aged 60 years and above of both sex in Uttara model town. The study population was selected purposively. Data were collected by the researchers themselves using face to face interview with a structured pre-tested questionnaire and was processed manually using a scientific calculator; **Year and place of work:** During 1st September to 15th December 2001 at the Department of Community Medicine, Medical College for Women & Hospital, Uttara Model Town, Dhaka; **Results:** Out of 104 respondents interviewed, 54% were male and 46% were female. About 66% were <65 year age group and only 35% were > 65 years. Among them 68% of the male were literate but 54% of the female were illiterate. Nearly 13% of the female were widow and 18% of the male were single. Most of the respondents belongs to family income >5000 taka per month. Most of the respondents 62% were not regular in health check up. There was significant association between sex of the citizens and health checkup ($P<0.05$). Males were more regular than females. Out of 104 respondents, 20% suffered from arthritis 17% from diabetes, 14% from hypertension, 14% from ischemic heart disease (IHD), 9% from chronic bronchitis, 9% from peptic ulcer, 7% from urogenital problem and about 13% from depression. Depression was significantly associated with economic conditions of the respondents ($P<0.05$); **Conclusion:** The study results indicated that most of the senior citizens were suffering from different types of chronic diseases, but their awareness and knowledge about regular health cheek up were at a low level. Depression like mental illness gradually increased among them.

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Indexing words: Health problem Senior citizen Geriatric

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INTRODUCTION

Elderly People constitute a significant proportion of total population of Bangladesh. But under the existing socio-economic condition to the country most of them are living with poor health, economic hard ship and social insecurity. National income is unevenly distributed and about 75% of the people are living below the poverty line. In view of these, the rights of the elderly persons to live with honour and dignity should be upheld by providing care and support to enable them to pass their remaining life in happiness and satisfaction[1]. Arbitrarily, at the age of 60 years and beyond, people may be regarded an old or aged. The number of aged in Bangladesh is gradually increasing. At present they comprise about 6 percent of the total population because of relatively high rate of population growth in the country and of gradual development in health services, the number of elderly population is increasing at a faster rate. It is likely to be doubled by the year 2020[2,3]. The goal of health for all by the year 2000 has given much more attention to the elderly citizens in member countries of WHO. The movement emphasize that the people of the world have equal value, rich and poor, women and men, old and young and they have equal access to food, water, shelter, clothing and health care and rehabilitation also[4,5]. This study has been carried out among the elderly population living at Uttara model town with a view to find out their health problems. This will be helpful for further study in large population group and developing necessary health program for the senior citizen.

MATERIALS AND METHODS

The study was of descriptive cross-sectional type Data were collected from 104 elderly aged 60 years and above of both sex in Uttara model town during the period of September to December 2001. Study population was selected form accidental non probability sampling. Information on disease pattern were collected during the interview of the respondents. Data were collected through face to face interview by using a structured pre tested questionnaires. The study population were selected purposively. Data were processed manually using a scientific calculator. The statistical significance was evaluated by Chi-squared test.

FINDINGS

Majority of the respondents were in the age group less than 65 years. Among them 64% were male and 68% were female. About 34% were more than 65 years.60% were literate and 42% were illiterate. 13% of the female were widows and about 18% of the male were single. Most of the respondents belonged to family income of > TK 5000 per month (65%) (Table-1a &1b).

Table-1(a) : Socio-demographic characteristics of the respondents.

Variables				
	Male		Female	
Age (years)	Frequency	%	Frequency	%
<65	36	64	33	69
>65	20	36	15	31
Education				
Illiterate	18	32	26	54
Literate	38	68	22	46
Marital Status				
Married	38	68	42	87
Single	18	32	6	13
Total	**56**	**100**	**48**	**100**

Table 1(b) : Socio-demographic characteristics of the respondents.

Monthly Family income	Frequency	%
< TK 5000	36	35
> TK 5000	68	65
Total	**104**	**100**

About 25% of the female senior citizens were suffering from arthritis. On the other hand most of the male study groups were suffering from diabetes 17%, chronic bronchitis 12%, and urogenital problem 10% (Table-2).

About 16% of the females were suffering from depression. It was found that diabetes, arthritis, hypertension, IHD, were the main health problems among the study population and about 12% of the respondents had depression like mental illness (Table-2).

Tabel-2: Distribution of the respondents by their types of health problems.

Variables						
Health Problems	Male		Female		Total	
	Frequency	%	Frequency	%	No	%
Arthritis	8	14	12	25	20	20
Hypertension	8	14	6	12	14	14
IHD	6	10	8	16	14	13
Chronic bronchitis	7	12	2	4	9	8
Diabetes	10	17	8	16	18	18
Peptic ulcer	6	10	3	16	9	8
Urogenital & others	6	10	1	2	7	6
Depression	5	8	8	16	13	13
					104	100

χ^2=5.94, df=1, P<0.05

About 62% of females had not done regular health check up and 42% of males had done regular health check up (Table 3). Significant association was found between depression and economic status of the citizens (Table-2). And there was significant association between health checkup and sex of the respondents (Table-3).

Table-3: Distribution of the respondents according to their health check up.

Health Checkup	Male	%	Female	%
Done	24	43	18	38
Not Done	32	57	30	62
Total	56	100	48	100

χ^2=10.34, df=1, P<0.05

DISCUSSION

Ageing is a universal process. Old age is an incurable disease but most recently, Sir James Sterling Ross Commented 'you do not heal old age, you protect it, you promote it, you extend it.' Old age should be regarded as a normal inevitable biological phenomenon[6]. Elderly population (>65 years) is indicative of population at high risk. They are more susceptible to disease, disability and deformities. The study revealed that economic status influenced the health of the aged people, but higher status responsible for depression like mental illness. This finding is similar to the findings of Suraiya et al[1]. Arthritis, hypertension, diabetes, chronic bronchitis, peptic ulcer, IHD, depression were the main health problems in our study population. Similar findings were reported by Ibrahim et al in the urban area where they found that the main causes of hospitalization were pain and fever (18.3%), hypertension (11.8%), cataract (10.5%), peptic ulcer (8.5%), diabetes (7.8%), cardiovascular disease (17.2%) and chronic bronchitis (5.2%). Arthritis was more common in females than in male[7]. Majority of the respondents did not perform health checkup, although males were a bit regular than females. Our findings were similar as in the studies done by Suraiya Jaben and others [1,7].

CONCLUSION

The study results indicated that most of the senior citizens were suffering from a variety of chronic health problems and mental illness was also an emerging problem among them. They had very limited knowledge about problems and not very regular and particular about their health checkup. So, it demands special attention to solve the problems of the elderly population.

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ORIGINAL ARTICLE

COMPLEMENTS (C3, C4) IN PATIENTS WITH HEART DISEASES AT AL-WAHDA HOSPITAL, DERNA, LIBYA

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ABSTRACT

Patients with heart diseases (HD) (n=14; age range: 43-75 years; sex: 5 males, 9 females) together with normal control subjects (NC) (n=20; age range: 27-60 years; sex: 10 males, 10 females) were investigated, as a preliminary study, for serum levels of complements C3 & C4. Serum C3 level was significantly elevated in HD while C4 level was normal [HD vs NC C3 (mg/dl): 179.7 ± 83.2 vs 143.1 ± 30.3, P<0.05; C4 (mg/dl): 54.2 ± 16.1 vs 49.1± 11.8, P>0.2]. Raised serum C3 with normal C4 level was an indication of acute phase response perhaps to an unknown antigen. The complement system is a potent mechanism for initiating and amplifying inflammation and also for causing tissue damage through immune complexes and anaphylatoxins such as C3a and C5a. The various possibilities potentially responsible for damage to the heart tissues in HD were discussed.

[J Med Coll Women Hosp 2004; 2(1): 14-16]

Indexing words: Heart disease Complement C3 Complement C4

INTRODUCTION

Among the life-threatening diseases, the most prominent ones are the heart diseases and cancer. Coronary heart disease (CHD) is the largest cause of morbidity and mortality in the world. The disease develops by the interaction of a variety of environmental agents that can be favourably altered by adjustments to life style. Ischemic heart diseases (IHD) and acute myocardial infarction (AMI) are the consequences of CHD[1, 2, 3]. Immunological mechanisms have been implicated in the pathogenesis of many clinical situations involving the heart. Delayed hypersensitivity to group "A" hemolytic streptococci was known to be present in rheumatic fever [4]. In about half the cases of AMI, autoantibodies to human heart and deposition of inactivation products of C3 and C4 were demonstrated[4, 5, 6]. Recent

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studies have indicated that complement system is activated in the plasma of patients with AMI[7, 8, 9]. It is possible that complement system may be involved in the pathogenesis of many of the heart diseases including IHD and AMI. Literature review indicated that no work has been done or reported about the complement components in Libyan patients with heart diseases. We have therefore investigated, as a preliminary study, the complement 3 (C3) and complement 4 (C4) levels in the serum of Libyan patients with heart diseases and the results are reported in the present article.

MATERIALS & METHODS

Subjects : A total of 14 patients with heart diseases (HD) were obtained from Coronary Care Unit (CCU) and Intensive Care Unit (ICU) at Al-Wahda Hospital, Derna, Libya. The diagnosis of HD was based on both clinical and laboratory investigations as available in the hospital. A total of 20 normal healthy controls were also included in the study for comparison as normal controls (NC).

Serum Specimen : A 5ml aliquot of blood collected from each subject (HD & NC) was allowed to clot for 30 minutes at room temperature, centrifuged for 10 min at 2500 rpm and the separated serum was stored frozen at -20°C until required for analysis.

Assay of C3 & C4 Levels in Serum : Single radial immunodiffusion method of Mancini et al was used for quantitative estimation of C3 and C4 levels in serum[10]. The immunokits from Kallestad, USA and Diagnolab, Spain were used for C4 and C3 estimation respectively.

Statistical Analysis : The results were evaluated statistically by comparing mean ± SD values using Student's t-test[11].

RESULTS

Subjects : Among the 14 patients with HD (age range : 43 – 75 years; sex : 5 males, 9 females), 6, 3, 3 and 2 were with IHD, AMI, Chronic heart failure (CHF) and atrial febrilation (AF) respectively. Of the 20 NC subjects (age range: 27 – 60 years) 10 and 10 were males and females respectively.

Complement Levels : Serum C3 level was significantly elevated in HD compared to NC ($P<0.05$), although no significant difference was observed for C4 level ($P > 0.2$) (Table-1).

Table-1: Serum Complements (C3, C4) in HD and NC subjects and their statistical analysis

Subjects*	Complements (mg/dl)	
	C3	C4
HD (N=14)		
Observed range :	75.1 – 338.3	23.4 – 73.9
Mean ± SD :	179.7 ± 83.2	54.2 ± 16.1
NC (N=20)		
Observed range :	101.0 – 193.9	15.2 – 61.1
Mean ± SD :	143.1 ± 30.3	49.1 ± 11.8
Student's t-test* (HD vs NC) :		
t :	2.048	1.005
df :	32	32
P :	< 0.05	> 0.2

* HD : Patients with heart diseases; NC: Normal Control subjects N = Number of subjects; SD: Standard Deviation; $P \leq 0.05$: Significant; $P > 0.05$: Not Significant.

DISCUSSION

The present study showed that serum C3 level was mildly but significantly elevated in HD compared to NC ($P<0.05$). However, no significant difference was noted for serum C4 level between HD and NC ($P>0.2$). Most likely explanation of our findings could be that raised level of serum C3 in HD was due to acute phase response perhaps against an unknown antigen. The complement system is a potent mechanism for initiating and amplifying inflammation. Some of the breakdown products of complement proteins, notably C3a, is chemotactic for neutrophils and trigger degranulation of basophils and mast cells and hence the name anaphylatoxins. Heart muscle tissues contain a lot of mast cells and therefore, inappropriate production of anaphylatoxins leading to amplified inflammation may be potentially responsible for damage to the heart tissues in HD particularly in AMI[4, 5, 6, 7, 8, 9, 12]. The production of anaphylatoxins can be verified by analyzing the levels of complement breakdown products, such as C3a and C5a in serum. Therefore, our results need cautious interpretation until further studies including more HD – patients of homogenous nature and assay of serum anaphylatoxins are conducted.

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REVIEW ARTICLE

A POSITION PAPER ON A RATIONAL GUIDE TO PAEDIATRIC ASSESSMENT AND TREATMENT IN ATTENTION DEFICIT HYPERACTIVITY DISORDER

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ABSTRACT

Attention deficit hyperactivity disorder (ADHD) is best conceptualized as a neurodevelopmental disorder that impacts upon key areas of children's development. Children with ADHD are pervasively overactive, fidgety and generally disruptive that lead to parents to an unbearable awful situation. There is growing confrontation among the parents of ADHD children, health and educational services. Parents are harassed demanding their children are assessed for the condition and consequently the paediatric and child psychiatric clinics face an exponential increase in referrals requesting a "service for this condition". This position paper aims to establish a rational guide to paediatric assessment and treatment in ADHD. This has resulted a service to this condition requiring successful intervention, coordinated involvement of local health, education, family support and some time welfare agencies working together with sufferers of family often over several years. Thus many of them are now benefiting sophisticated therapeutic interventional programme.

[J Med Coll Women Hosp 2004; 2(1): 17-24]

Indexing words : Attention deficit hyperactivity disorder Pervasiveness,

INTRODUCTION

Attention deficit hyperactivity disorder (ADHD) was first described by Professor George Frederick Still, the same children's physician who described stills disease since nearly a century[1]. ADHD is best conceptualized as a neurodevelopmental disorder that impacts upon key areas of children's developments. The areas of dysfunction are overactivity and restlessness, inattentiveness and distractibility, impulsiveness and social disinhibition. There are many ways of conceptualizing the cause of ADHD, including the effects of diet, early childhood attachment problems, adverse

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family influences and over stimulation of young children in a modern frenetic society. Recent work involves functioning of the fronto-temporal regions. This area of the brain is concerned with conscience, planning and also inhibition of instinctive mid-brain impulses. ADHD is often associated with other disorders such as conduct disorder, sleep disorders, specific or general developmental delay, depressive disorder, delinquency, truancy or drug and alcohol abuse. A standard clinical assessment, including mental state, neurodevelopmental and brief physical examinations is required. It is also necessary to check for the Fragile-X condition in hyperactive children. The important considerations are the chronicity, pervasiveness and handicapping nature of the clinical features and also the resources of patients and their families. The most important consideration with respect to the treatment of children with ADHD is that it must involve and individually tailored multimodal approach commonly employing several strategies working in parallel. Drug therapy in particular should never be used as a sole treatment. This position paper reviews the recent literatures that has resulted successful intervention requires the coordinated involvement of local health, educational and sometimes welfare agencies working together with suffers and their families, often over several year.

Some cases may also benefit from treatment as adult.

CLASSIFICATION / CLINICAL FEATURES

Both of the major systems of classification for mental disorder, the Diagnostic and Statistical Manual (DSM-IV) and the International Classification of Diseases (ICD-10), identify the same central features of this syndrome[2,3].

DSM-IV CRITERIA

Attention Deficit / Hyperactivity Disorder

A. Either (1) or (2)

(1) Inattention : At least six of the following symptoms of inattention have persisted for at least six months to a degree that is maladaptive and inconsistent with developmental level :

(a) Often fails to give close attention to details or makes careless mistakes in schoolwork, work, or other activities.

(b) Often has difficulty sustaining attention in tasks or play activities.

(c) Often does not seem to listen to what is being said to him or her.

(d) Often does not follow through on instructions and fails to finish schoolowork, chores, or duties in the workplace (not due to oppositional behaviour or failure to understand instructions).

(e) Often has difficulties organising tasks and activities.

(f) Often avoids, express reluctance

engaging in tasks that require sustained mental effort (such as schoolwork or homework).

(g) Often loses things necessary for tasks or activities (e.g. school assignments, pencils, books, tools, or toys).

(h) Is often easily distracted by extraneous stimuli.

(i) Often forgetful in daily activities.

(2) Hyperactivity – Impulsivity : At least five of the following symptoms of hyperactivity – impulsivity have persisted for at least 6 months to a degree that is maladaptive and inconsistent with development level:

(a) Often fidgets with hands or feet or squirms in seat.

(b) Leaves seat in classroom or in other situations in which remaining seated is expected.

(c) Often runs about or climbs excessively in situations where it is inappropriate (in adolescents or adults, may be limited to subjective feelings of restlessness).

(d) Often has difficulty playing or engaging in leisure activities quietly.

(e) Is always "on the go" or acts at if "driven by a motor".

(f) Often talks excessively.

(g) Often blurts out answers to questions before the questions have been completed.

(h) Often has difficulty waiting in lines or awaiting turn in games or group situations.

(i) Often interrupts or intrudes on others (e.g. butts into others' conversations or games).

B. Some symptoms that caused impairment were present before age seven.

C. Some symptoms that cause impairment are present in two or more settings (e.g. at school, work, and at home).

D. There must be clear evidence of clinically significant impairment in social, academic, or occupational functioning.

E. Does not occur exclusive during the course of a Pervasive Development Disorder, Schizophrenia or other Psychotic Disorder, and is not better accounted for by Mood Disorder, Anxiety Disorder, Dissociative Disorder, or a Personality Disorder.

Both of the major systems of classification of mental disorder, the DSM-IV and the ICD-10, identity the same central features of this syndrome. But some important differences are that ICD-10 requires both hyperactivity and impaired attention whereas in DSM-IV the diagnosis can be made if there is either inattention or hyperacetivity & impulsiveness.

PREVALENCE

Taylor and his co-workers in the UK used the ICD-10 criteria for hyperkinetic disorder to establish a prevalence rate of 0.5 – 1.0%. Unfortunately, only a small fraction of the children in epidemiological research have been assessed in any detail and diagnosed accurately, and even fewer have received appropriate help[4]. American data suggest a much higher prevalence rate of 3 – 5%, which is usually attributed to the broader concept of ADHD in the USA.

AETIOLOGY

There are many ways of conceptualizing the cause of ADHD, including the effects of diet, early childhood attachment problems, advanced family influences and overstimulation of young children in a modern frenetic society. What is known is that the features of ADHD are overrepresented in children with developmental delays and those with brain damage. The evidence points to the main aetiological factor being a disorder of maturation and it has all the characteristics that are shared by other specific developmental disorders such as:

1. An overrepresentation of affected males (3 – 5:1)
2. A tendency for the features to ameliorate with time (especially motor restlessness)
3. A strong genetic influence (established by family and twin studies)
4. An improvement of symptoms in response to appropriate training and practice.
5. Co-morbidity with other specific developmental delays (specially reading retardation, clumsiness and nocturnal enuresis)
6. All the symptoms occur as part of normal development and follow a normal developmental course.
7. The symptoms become much worse following any emotional or physical stress.

Barkley conceptualizes ADHD as primarily representing a syndrome where the normal consequences of behaviour are not attended, resulting in delayed social learning[5].

Dietary factors are now thought to be a very rare cause of hyperkinesis. A recent study in the UK has suggested that parental accounts of behavioural changes following selected dietary challenges should be accorded clinical attention and supported approperiately[6].

ASSESSMENT

This starts with a standard clinical assessment, including mental state, neurodevelopmental and brief physical examinations. Measurements of blood pressure, height and weight are required before psychostimulants are prescribed. Any features that might be attributable to

epilepsy merit an EEG. It is also advisable to check for the Fragile-X condition in hyperactive children who are also globally developmentally delayed[5]. Currently there are many diagnosic instruments available to facilitate the diagnosis of ADHD, most of which have acceptable degrees of reliability and validity (e.g. the Connors' Questionnaries)[7]. Questionnaires and rating scales should never be allowed to replace the well conducted clinical assessment of the child in a range of different settings, but they can be useful as a screening instrument and to monitor progress. In clinical practice, the diagnostic guidelines of the DSM-IV and ICD-10 schemes are probably the most useful, and DSM-IV presents the key clinical features in order of frequency from factor analysis studies.

The important considerations are the chronicity, pervasiveness and handicapping nature of the clinical features and also the resources of patients and their families. In this context, reports that include diary-documented accounts covering the key symptom areas, from parents and school teachers and any other people in regular contact with the patient are invaluable Formal neuropsychological workup is not normally necessary (nor locally available) but might be required for more complex cases, particularly when there is evidence of past neurological problems or evidence of specific or global developmental delay.

DIFFERENTIAL DIAGNOSIS

ADHD can be confused with the following conditions :

1. The most common condition with which ADHD is confused is general developmental delay (mental retardation) : children who are significantly delayed may show several features
2. Children who are ill – disciplined and generally badly behaved primarily as a result of poor parental guidance and inadequate limit – setting. They may also be overactive, particularly when excited, but usually settle once the 'ground rules' have been established and implemented in a consistent manner.
3. Severely emotionally abused and neglected children may be overactive, disinhibited and impulsive in company : they may also seek inappropriate physical contact with unfamiliar adults.
4. Overtired children commonly become somewhat overactive and fractious prior to bedtime : appropriate treatment involves the inoculation of good sleep habits and the establishment of clear bedtime rules.
5. Paradoxical states of extreme transient hyperactivity associated with clouded consciousness may be seen in children given sedative medication. Other drugs such as sympathomimetic asthma drugs and some anticonvulsants can occasionally produce these symptoms.

6. Very rarely, parents may present a child with a spurious history in an attempt to obtain counselling or psychotropic medication vicariously. (It should be noted that there are no accounts of ADHD sufferers abusing their prescribed psychostimulants).

Treatment

The most important consideration with respect to the treatment of children with ADHD is that it must involve an individually tailored multimodal approach commonly employing several strategies working in parallel. Drug therapy, in particular, should never be used as a sole treatment. Successful intervention requires the coordinated involvement of local health, educational and sometimes, welfare agencies working together with sufferers and their families, often over several years. Some cases may also benefit from treatment as adults.

Individual Therapy

Dynamic psychotherapy with younger sufferers is generally unhelpful. Nevertheless, building up a therapeutic relationship over time whilst employing a generally firm but fair approach is to be encouraged. Sufferers should be encouraged to use problem-solving and specific 'stop and think' measures to control their impulsivity and to generally develop an internal critical dialogue with themselves.

Special educational measures

A high standard of communication and consistency of approach between home, school and clinic is essential. Any behavioural management programme will require a named person (class teacher, special needs coordinator, school nurse of classroom-based support worker) to ensure adequacy, consistency and to take responsibility for its implementation.

Parent and Training Support

At presentation, affected children's parents are often demoralized, angry and sometimes actively hostile towards their children and also towards potential therapists! Issued such as appropriate limit-settings, reduction of criticism and increasing praise should be focused on before psychostimulant therapy is considered. Children with ADHD and their families are currently inadequate and piecemeal being usually dependent upon the special clinical interests of local child psychiatrists and paediatricians for their existence[8,9].

Medication

Indeed, in server cases, psychostimulant treatment may be the only way of breaking into the vicious circle of inattention and disruptive behaviour. The most commonly used agent in this class is methylphenidate (Ritalin), and a straightforward dosage regimen is shown in **Box-1**.

Box-1: A methylphenidate protocol

Initial daily dose: 5mg
Incremental increase: 5mg every 2days
Usual effective dose: 60-80mg
Schedule: Normally twice or there times per day
Timing: After food to avoid nausea and anorexia
Last dose at least 4 h before bedtime

Despite being a controlled drug, methylphenidate can now be prescribed by any medical practitioner. Prescriptions have to be hand-written, stating the dosage, formulation (tables) and total number of tables in words and figures as well as the name and address of the patient and prescriber. The other drugs in the psychostimulant class are dexamphetamine and pemoline.

Monitoring

Given that severely affected cases commonly attract the attention of several health, educational, social service, and sometimes, voluntary agencies, it is salutory that the specialist clinician who might be involved with ADHD children over several years takes some responsibility (and, ideally, the lead role) for coordinating services **(Box-2)**. By ensuring regular family reviews and wider network meetings, information necessary to inform the direction of the overall treatment strategy can be discussed and implemented.

PROGNOSIS

When left without intervention or support, children with ADHD have been shown to be much more likely than matched non-ADHD children to become more oppositional and defiant during their mid-childhood, often becoming conduct-disordered in late childhood and delinquent in adolescence. Associated with this are the poor prognostic risk factors of progressive educational underachievement and especially literacy delays, poor self-esteem and later development of neurotic and depressive disorders. Sufferers' constitutional impulsivity and risk taking propensities make them more likely to engage in drug and alcohol abuse, criminal activity and also to become involved in chaotic personal relationships. In more than one-third of cases, the features of the handicapping syndrome (especially impulsivity and inattentiveness) will persist well into adulthood. Until very recently it was believed that even when treated with psychostimulants, clinically hyperactive children generally had a poor prognosis. Nevertheless, recent longitudinal, long-term, interventional programmes which also involve the use of long-term medication, can significantly improve the prognosis[8,9].

Box- 2: Outline of local service of ADHD patients

Setting

- Locally based outpatient clinic with access to day hospital assessment facilities
- Dedicated clinical sessions allowing at least 90 min for an initial assessment
- Facilities for whole-family interviews (big room with range or toys), physical examination (including height and weight measurement), indirect observation (one –way screens and video cameras), electrocardiography and phlebotomy

Staff

- Dedicated multidisciplinary team consisting of a specialist clinical (development pediatrician and/or child psychiatrist), community nurse, clinical psychologist, social worker, and a hospital pharmacist
- Close links with generic child health services and schools

Principles

- Joint planning with health, educational, welfare and lay agencies
- Early detection and treatment through assertive outreach
- Multimodal, family-based, individually tailored and closely monitored treatment programmes
- Provision of long-term care and follow-up
- Liaison and consultation services to colleagues ad other agencies
- Ongoing audit of practice
- To raise the awareness and facilities understanding of ADHD locality

CONCLUSION

ADHD is totally neglected in developing countries but the structured way of dealing with these patients has to be encouraged and made the aware to the society.

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REVIEW ARTICLE

LIPOPROTEINS AND ANTIOXIDANT MICRONUTRIENTS : IMPLICATIONS FOR CORONARY HEART DISEASE

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ABSTRACT

Coronary heart disease (CHD) is one of the major causes of death and disability worldwide. Oxidation of low-density lipoprotein–cholesterol (LDL-Ch) by oxygen free radicals (OFR) provides a molecular link to the development of atherosclerosis and hence risk of CHD. Free radical oxidation can usually be protected against by appropriate antioxidant micronutrients such as vitamin E, vitamin C and beta-carotene. Epidemiological data have shown that low plasma levels of these antioxidant vitamins are associated with increased risk of CHD. Recently immersed concept is that supplementation with the antioxidant micronutrients (vitamin E, vitamin C, beta-carotene) may be considered as higher intakes may prevent and hence reduce the risk of CHD. The probable implications of these antioxidant vitamins in the aetiopathogenesis of CHD were reviewed in the light of recent literature.

[J Med Coll Women Hosp 2004; 2(1): 25-30]

Indexing words : Lipoproteins Antioxidants Vitamin E Vitamin C Beta-carotene Coronary heart disease

INTRODUCTION

Coronary heart disease (CHD) is one of the major causes of death worldwide, although it varies in the incidence patterns in different parts of the world and one way or another atherosclerosis overweighs all other aetiologies in CHD[1,2,3]. Among the lipids, cholesterol rich low density lipoprotein (LDL), particularly oxidatively modified LDL, is the one most frequently implicated in the aetiopathogenesis, and molecular link to the development, of atherosclerosis leading to CHD[4,5,6]. However, hypertriglyceridemia with increased concentrations of very low density lipoprotein (VLDL) also appears to increase risk of CHD, but in contrast serum

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levels of high density lipoprotein (HDL) are inversely related to risk : the higher the level, the lower the risk[4,5,7]. Lipid peroxidation causing oxidative modification of LDL can usually be protected against by appropriate antioxidant micronutrients such as vitamin E, vitamin C and beta-carotene as reported by many investigators[8,9,10]. Recent attention therefore has focused on identifying agents that may prevent the oxidation of LDL and thus retard the progression of atherosclerosis and hence CHD[3].

MOLECULAR MECHANISM OF ATHEROSCLEROSIS

There is growing evidence that oxidation of LDL plays an important role in atherosclerosis and oxidized LDL is more atherogenic. Oxidation of LDL by OFR provides a molecular link to the development of atherosclerosis and hence CHD. Free radical oxidation of polyunsaturated fatty acids (PUFAs) of lipids (lipid peroxidation) leads to malondialdehyde (MDA / CHO-CH2-CHO) production which can cross-link with the protein moiety of LDL to form modified LDL. This modified (oxidized) LDL is avidly recognized by the macrophage scavenger receptor resulting in rapid cholesterol ester uptake causing fatty streaks (foam cells) formation leading to atherosclerosis[4,6,9]. The LDL-cholesterol (LDL-Ch) is a complex particle comprising a cholesterol ester core surrounded by phospholipids (including the PUFAs), protein (apoB100) and cholesterol molecules. In addition, the fat soluble vitamin E and beta-carotene, are also contained within the LDL-Ch complex[11]. It is the phospholipids part of the LDL-Ch which is more prone to be damaged by free radicals and hence, the balance of antioxidant vitamins in the LDL-Ch complex may be critical for its protection from free radical attack[8,9,10,12]. In fact, recent reports have raised exciting public awareness of the possibility that antioxidant micronutrients such as vitamin E, vitamin C and beta-carotene may slow the progression of atherosclerosis and hence may prevent and reduce the incidence of CHD[3,11,13,14].

ANTIOXIDANT MICRONUTRIENTS IN CHD

Vitamin C, vitamin E and beta-carotene are three important naturally occurring antioxidant micronutrients[10].The role of these antioxidant vitamins in the prevention of free radical damage of tissues and hence in the prevention of CHD has been reviewed[13, 14, 15]. Vitamin E is well known to be an antioxidant factor and lipid peroxidation of PUFAs in LDL starts only after endogenous antioxidants (vitamin E, vitamin C & beta-carotene) are consumed and vitamin E and vitamin C were shown to synergistically inhibit oxidation of LDL[16,17]. It has been reported that dietary supplementation of vitamin C and vitamin E synergistically lowers plasma lipids and

therefore may have a role in reducing the incidence of CHD[18]. Riemersma et al reported a significant negative correlation between plasma vitamin C, vitamin E, beta-carotene and risk of CHD, whereas no correlation was obtained with vitamin A[19]. Benzie and Strains showed that vitamin C and vitamin E supplementation to volunteers led to significant increase in total antioxidant (reducing) power and significant decrease in uric acid and lipid (cholesterol & triglyceride) concentrations. Thus increased intake of vitamin C and vitamin E may help to decrease risk of CHD by a combination of lipid and urate lowering effects, as well as by virtue of their antioxidant properties[20]. Recent epidemiological data have shown an impressive inverse correlation between both vitamin C and vitamin E levels and morbidity from ischemic heart disease [21].

The antioxidant that plays the most important role in controlling the process of LDL-Ch oxidation is vitamin E. Since vitamin E is able to form a close association with the PUFA molecules as proposed by Diplock, Lucy and Giasuddin[22,23], it is in prime position to break free radical initiated reactions[24]. Vitamin E, acting as an antioxidant in the lipid phase, arrests the propagation of the free radical damage by converting the peroxy fatty acid radical (ROO·) to a hydroxyl fatty acid (ROOH). The resulting vitamin E radical (vitamin E – O·) can stabilize another ROO· by converting itself to quinone form (vitamin E = O). Alternatively vitamin E – O· can be reconverted to active vitamin E (vitamin E–OH) by oxidation of vitamin C, or by reduced glutathione (GSH), which is able to block further action of lipid radicals[24,25]. Vitamin C is water-soluble and its antioxidant action in the aqueous phase is possibly through regeneration of vitamin E mainly[24], in addition to direct removal of reactive oxygen species (ROS) such as O_2^-, H_2O_2 and $^1O_2$[14]. Reduced form of vitamin C can be regenerated enzymatically as well as non-enzymatically[24]. It is therefore reasonable to suggest that this synergistic effect between vitamin E and vitamin C may be lacking in patients leading to atherosclerosis and hence CHD.

Regarding beta-carotene, it is thought that the sole function of it is to act as a precursor of vitamin A. Nevertheless substantial amounts of beta-carotene can be absorbed from the diet without being changed to vitamin A[11,24,26]. Beta-carotene has been shown to be capable of acting as an antioxidant in experimental animal studies[11,27,28]. The mode of action of beta-carotene as an antioxidant is probably through resonance stabilization of ROO. and quenching of $^1O_2$ at low oxygen tension and thus the chain breaking action of beta-carotene compliments that of vitamin E[27,28]. However, studies on beta-carotene are limited and detailed information are not

available about how resonance-stabilized carbon-centered radical of beta-carotene is finally metabolized[27]. Although its role in preventing CHD development is unclear, recent reports have suggested that beta-carotene may be able to protect against atherosclerosis[10,11,13,14,29] and hence CHD through its antioxidant action. Consequently, beta-carotene may have a vitamin E sparing effect or synergistic antioxidant action with vitamin E in the lipid environment. Therefore, low beta-carotene levels might also be responsible for accelerated oxidative modification of LDL-Ch and increased tendency for atherosclerosis and hence CHD[10,30,31]. However, it is important to note that only 70% LDL-Ch is metabolized through conventional LDL receptor-mediated mechanism. The remaining 30% of LDL-Ch is metabolized through other LDL receptor-independent mechanisms that needs to be accounted for in the overall mechanism of atherosclerosis leading to CHD[32]. Secondly, significant role for vitamin E, vitamin C and beta-carotene in maintenance and proper functioning of the immune system have been reported[26,33]. Immunological mechanisms and parameters are therefore important points to be considered also in CHD patients as proposed by many investigators[32, 34, 35, 36].

CONCLUSION

In conclusion, hyperlipoproteinemia particularly increased LDL-Ch is one of the major risk factors for atherosclerosis leading to CHD. Other risk factors of atherosclerosis such as smoking, hypertension and diabetes mellitus cannot however be ignored. Secondly, reduced level of antioxidant micronutrients (vitamin E, vitamin C, beta-carotene) that might be a consequence of excessive utilization due to increased oxidative stress in vivo or low dietary intake of fruits and vegetables, the sources for antioxidant micronutrients, may also be one of the prominent causes of atherosclerosis and hence CHD. Recently emerged concept has suggested that supplementation with antioxidant micronutrients (vitamin E, vitamin C, beta-carotene) may be considered as higher intakes may prevent CHD[3, 10, 11, 14]. However, vitamin E like other antioxidants, also have pro-oxidant actions, especially at very high concentrations. This may explain why, although studies have shown an association between high blood concentrations of vitamin E and a lower incidence of atherosclerosis, the effect of high doses of vitamin E have been disappointing[37]. One must therefore wait until more clear-cut beneficial results are available from detailed clinical trial studies. Also, further studies are warranted about the relationship among lipids, free radicals and other important antioxidant defense mechanisms and the mechanisms of LDL-receptor-independent LDL-Ch metabolism[32, 34, 35, 36].

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CASE REPORT

GIANT FIBROADENOMA OF THE BREAST : A CASE REPORT

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ABSTRACT

Giant fibroadenoma of the breast is a very rare pathology presenting in adolescents. Here we report such a case that was managed successfully by excision of the fibroadenoma with well preservation of the shape of the breast.

[J Med Coll Women Hosp 2004; 2(1): 31-34]

Indexing words : Breast Fibroadenoma

INTRODUCTION

Fibroadenomas are benign tumours composed of both glandular and fibrous tissues. Their size varies from smaller than 1cm in diameter to as large as 15cm in diameter; those with diameter exceeding 5cm and/or weighing>500g are known as giant fibroadinoma or juvenile fibroadenoma[1,2]. Fibroadenomas are found in 10% of all women (20% of black women)[3,4]. The highest incidence is in women from their teen years into their 20s and rarely develop after the age of 30 years. The prevalence is approximately 8%-10% in women older than 40 years[5,6]. After carcinoma, fibroadenoma is the second most common solid tumour in the breast and is the most common tumour in women under the age of 30 years[7].

CASE REPORT

A 20 years old unmarried girl presented with huge enlargement of her right breast, that has increased in size very rapidly, for one year and pain in the right breast for one week.

On examination, she was a young lady of average built and nutrition with no anaemia and jaundice. Her right breast was about

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two times larger than the left. The right nipple was retracted and much below the level of the left one. The overlying skin was stretched and showed engorged veins. There were about 5 palpable lumps in the right breast and 2 in the left. One of the lumps in the right breast was about 11cm in diameter with well defined margin, irregular surface, firm consistency, was non tender, with some fixation to the skin around the areola but freely mobile over the underlying structures. The other lumps were on average of 2cm in diameter with similar features.

The reports of all routine laboratory investigations were within normal limit. On aspiration, blood stained fluid was drawn and FNAC report was not conclusive. Then she was advised for surgery. On giving a submammary incision in the right breast a large, well-encapsulated, lobulated, firm mass of about 10cm X 9cm X 6cm was found which could be removed with minimum destruction of the breast tissue.

Two other similar lumps of about 2cm x 3cm and 2cm x 1.5cm were also removed from the right breast (Figure-1). Two smaller lumps in the right and two in the left breast were not removed and kept for observation in the subsequent follow-up. Retracted nipple was corrected by simple eversion procedure (Figure-2).

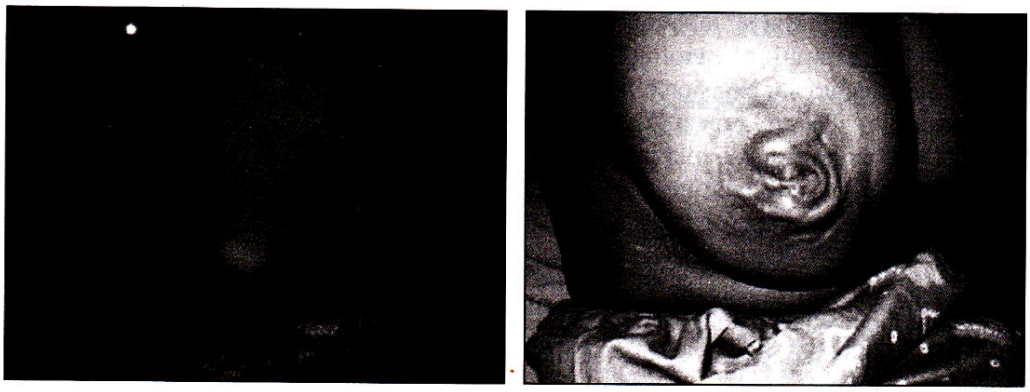
Post operatively the two breasts were almost similar in size and shape. The lump was histologically reported as fibroadenoma.

DISCUSSION

Giant fibroadenoma is predominant in Black adolescents and in the Oriental race[2,8]. Clinically it is characterized by massive and rapid enlargement of a breast mass. Fibroadenomas cannot be prevented but can be discovered early by regular breast self-examination. It normally has rubbery texture, smooth surface and moves easily under the skin. In our case, clinically there seemed to be some fixation to the skin due to huge size of the mass but at operation no adhesion was found. It may be painless, can be tender or even painful. Giant fibroadenomas can grow to immense proportion, resulting in congestion and ulceration of skin by centrifugal pressure. Such an enlargement of the breast can also be due to cystosarcoma Phyllodes or virginal hypertrophy. Fibroadenomas may be single or multiple in one or both breasts. Approximately 10%-15% of the tumours are multiple[2,3]. In approximately one half of women who receive cyclosporin A after renal transplantation, fibroadenomas develop, and these are often multiple and bilateral[2,9].

Fibroadenomas represent a hyperplastic or proliferative process in a single terminal ductal unit. Microscopically, the tumour is characterized by a rich cellular stroma and a prominent glandular epithelium. Their development is considered to be an aberration of normal development and involution (ANDI)[1]. The exact etiology is not known but is believed to be an end-organ hypersensitivity to normal level of gonadal hormones; genetic and environmental factors could also be involved[10].

The preferred diagnostic method is determined by the patient's age and clinical



Pre-operative view

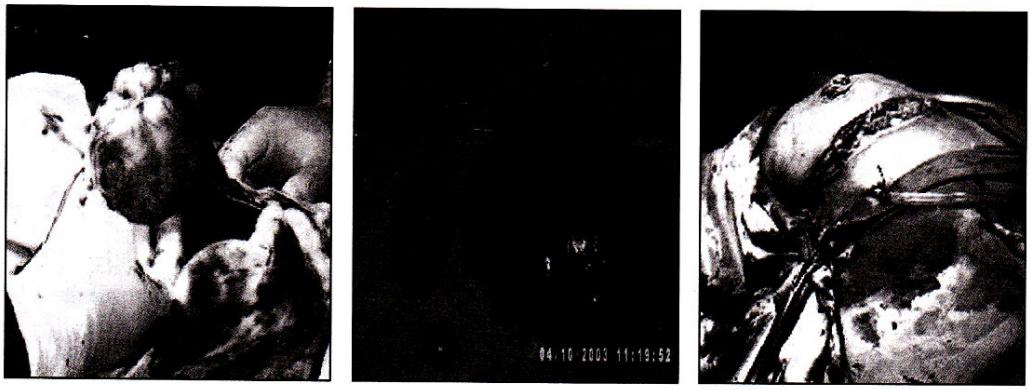


Figure-1 : Pre-operative view

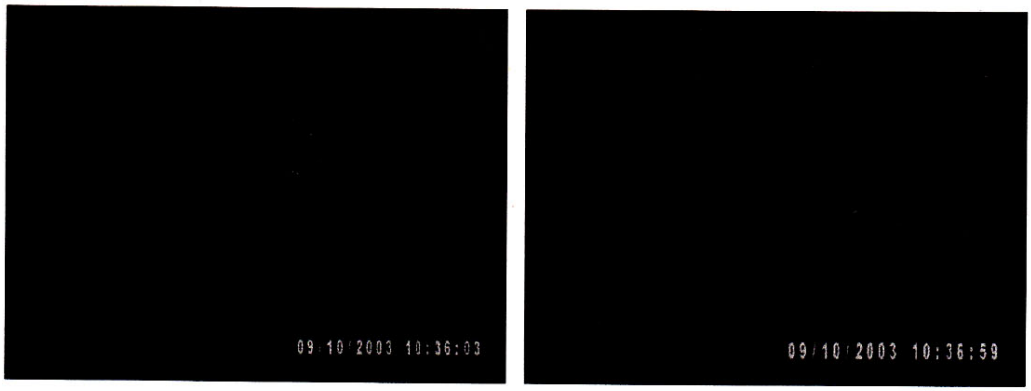


Figure-2 : Post-operative view

state. Ultrasonography, mammography, fine needle aspiration cytology, core biopsy, incision biopsy, excision biopsy, etc are advisable. The treatment is usually surgical and ranges from simple excision to subcutaneous mastectomy with reconstruction, depending on the size of the lump. In our case, though the lump was very large it could be managed well by simple excision. When a lump is removed it does not usually affect the shape of the breast, as the normal breast tissue will fill out and make up for it. After removal, the lumps recur in up to 20% of cases[3,4].

Fibroadenomas are benign lesion and are not considered to have malignant potential. However, because they contain epithelium a risk of neoplasia exists as in other location in the breasts. Women with complex fibroadenoma (those associated with fibrocystic changes, proliferative breast disease, sclerosing adenosis, calcifications, papillary apocrine changes) have a relative risk of cancer of about 2.2; those with a family history of breast cancer are also at an increased risk[5,6,9].

CONCLUSION

The pre-operative diagnosis of Giant fibroadenoma may be difficult due to its clinical presentation that may mimic that of malignant tumours e.g. huge size, ulceration over the skin etc. This diagnostic dilemma may lead to a decision of radical surgery like mastectomy, which is unnecessary for giant fibroadenoma. Usually the normal breast tissue remains compressed with size and weight of giant fibroadenoma but once the tumour is removed the compressed breast tissue expands and reach to normal size as compared to its counterpart, as we see in the 'post-operative view'. So, precise diagnosis and meticulous surgery can prevent many of the mutilating surgery. In benign breast disease, if surgery is indicated, one should have prime consideration to the aesthetic point of view.

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CASE REPORT

PERIODIC PARALYSIS : A CASE REPORT

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ABSTRACT

Hypokalaemia is frequently encountered in clinical practice. But hypokalaemic periodic paralysis is a rare condition. Although it is rare, it is not uncommon in clinical practice. It is usually overlooked and undiagnosed by the physician. A careful history and clinical examination along with electrolyte measurement can give a conclusive diagnosis. In this article we discussed a case of hypokalaemic periodic paralysis who was admitted in the medicine department of the Medical College for Women & Hospital, Uttara, Dhaka.

[J Med Coll Women Hosp 2004; 2(1): 35-37]

Indexing words : Periodic paralysis

INTRODUCTION

Periodic paralysis is a rare condition characterized by the episodic attack of flaccid paralysis or weakness of the body that occur in association with variation of plasma potassium level[1]. Main variety is hypokalaemic periodic paralysis but it may be hyperkalaemic or normokalaemic, although the latter two varieties are less common[2]. Hypokalaemic periodic paralysis presents with weakness in the lower extremities with progression to arm muscles but not to respiratory muscles[3]. Hypokalaemic attacks typically begin in late childhood or adolescence, usually occur at night, tend to be severe, and last a day or longer[4]. It usually occurs after rest or sleep, never occur in midst of exertion, precipitated by carbohydrate and salt rich diet. Patient remains alert during attack and initially in between attacks, strength of muscles remain normal. Neurological

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functions are normal, but tingling in the fingers, apathy and paralysis are the common presenting features. During hypokalaemia ECG changes characterized by flattened T-wave and appearance of U-wave in most leads. Periodic paralysis is amenable to treatment and its progression can be reversed. Mode of transmission is autosomal dominant. Exact mechanism is not known but there is a mutation of muscle voltage gated calcium channel (CACLNIA3)[5]. There is some association of periodic paralysis with thyrotoxicosis – mostly seen in Asian young especially Chinese people (TPP) – here decreased activity of calcium pump is claimed[6]. Hypokalaemic periodic paralysis in its acute attack is relieved by potassium therapy but it is prevented by acetazolamide or spironolactone therapy. In clinical practice, some times mistakenly patient is labeled as hysterical conversion reaction; in such cases high level of suspicion, careful clinical examination with electrolyte study guides to correct and conclusive diagnosis.

CASE REPORT

A 55 years old man, hailing from Uttara, married, nonsmoker, worker of a jute mill was admitted to hospital with the history of recurrent attacks of sudden weakness of the limbs for one year. During each episode of attack he was unable to walk. Each attack usually persisted for 2 to 3 hours and he recovered gradually without any medication.

But this time on 10th September 2003, the patient was admitted in this hospital with the history of sudden severe weakness of all four limbs, that developed following intravenous infusion of dextrose in aqua two days back prescribed by a quack due to his decreased appetite and general weakness. There was no history of vomiting or alteration of bowel and bladder habit. The patient did not give any history of convulsion or change of level of consciousness. He was non-diabetic, non-hypertensive and did not give any history suggestive of hyperthyroidism. There was no history of significant medications. On clinical examination he was fully conscious, oriented and mentally alert. His vital signs were normal. On neurological examination his muscle power was diminished to grade 3/5, tone was reduced, reflexes were intact with plantar flexor. Other systems revealed no abnormality. Investigations showed very low serum potassium level (1.49 mmol/l) but serum sodium, serum chloride and serum bicarbonate levels were normal. All other routine investigations including thyroid function test were normal. Immediately after diagnosis, the patient was treated with parenteral potassium (in normal saline) therapy and the patient improved dramatically. Later on he was on oral potassium therapy and spironolactone. Subsequently patient's electrolyte measurement including plasma potassium was reported normal. He was discharged with the advice to take plenty of fruits and vegetables, syrup potassium chloride and

tablet spironolactone and advised to continue these for an indefinite period. He was also advised not to take excess of carbohydrate or salt rich diet or dextrose in aqua infusions.

DISCUSSION

Hypokalaemia is defined as plasma potassium concentration <3.5 mmol/L. It is mainly caused by excessive loss of potassium in the alimentary tract or in the urine. Hypokalaemic periodic paralysis is a rare condition but not uncommon. It is usually undiagnosed or overlooked in clinical practice and sometimes it is labeled as malingering or hysteric conversion reaction. So a careful medical history, clinical examination along with measurement of plasma potassium is necessary to confirm the diagnosis.

Milder attacks of hypokalaemic periodic paralysis usually resolve spontaneously. The acute paralysis improves after the administration of potassium salts. Oral KCl (0.2 to 0.4 mmol/L) should be given in patients with moderate to severe weakness and repeated at 15 to 30-min interval depending on the response of the ECG, serum potassium level and muscle strength. When the patient is unable to swallow or on vomiting or having very low potassium level, intravenous therapy may be necessary. Potassium is administered intravenously preferably in mannitol as the vehicle. The goal of therapy is to eliminate attacks, which also prevents inter attack weakness.

CONCLUSION

Hypokalaemia is a serious condition, which is to be corrected as soon as possible. It may cause fatal cardiac arryhthmias and even death. Diagnosis of hypokalaemic periodic paralysis is established by high index of clinical suspicion, demonstrating a low serum potassium level during a paralytic attack and by excluding secondary causes of hypokalaemia.

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CASE REPORT

MANAGEMENT OF A MAJOR BURN CASE IN A GENERAL SURGICAL WARD

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Z Ummay Humayra[3] MBBS; **S Asmema Shashi**[4] MBBS

ABSTRACT

A 32 years old lady got burn and was admitted in our hospital on the 4th day after accident with a burn wound involving >4o% total body surface area (TBSA) with deep partial thickness and full thickness burn involving bilateral upper limbs, back and majority of upper half of right thigh except the medial surface. On admission the patients was conscious but in a state of shock and the wounds were infected. The case was managed successfully in a general surgical ward and the patient was discharged after 3 months with reasonable physical condition, without any significant disability.

Indexing words : Major burn | General surgical ward | >40% TBSA | Full thickness | Partial thickness burn

[J Med Coll Women Hosp 2004; 2(1): 38-42]

INTRODUCTION

In general terms a major burn is one that involves more than 30% of the body surface area, is a composite of sunburn and inhalation injury, or involves burn in hands, face or genitalia and definitely requires the expertise of a regional burn unit[1]. Globally, in 2000, fire-related burns were ranked among the 15 leading causes of death and burden of disease among children and young adults of 5-29 years. South-East Asia alone accounts for just over one-half of the total number of fire-related deaths worldwide and females in this region have the highest fire-related burn mortality rates globally[2]. Moreover, Study shows in India

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accidental burn in married women results in survival period just more than one day and more than half of them die of Septicaemia[3]. We treated a women who accidentally had a major burn involving >40% TBSA with infected deep and partial thickness wound. The patient is now in good health without any physical disability. The total management of this patient is reported with a review of the literatures.

CASE REPORT

A 32 years old lady accidentally caught by flame to her tightly worn cotton garments, while cooking. Immediately after burn, patient rushed to Dhaka Medical College Hospital but could not get herself admitted due to lack of bed in the hospital. Initially she was managed by local general physician, but when her condition started deteriorating her husband brought her to our Hospital, 4 days after the incidence.

On admission, the patient was in a state of hypovolumic shock and initial assessment of burn area appeared to be >40% TBSA. The most part of the back & both the upper limbs had partial and full thickness burn evidenced by blistering, moist, red colored & edematous skin. Few areas were covered by dark violet colored charred skin, which was due to application of concentrated gentian violet over the whole body. The right lateral surface of waist and upper 1/3rd of thigh had an extensive dark brown, hard, dry, leathery appearance. There was no circumferential burn. The vital signs of the patient were BP 100/70 mm of Hg, Pulse 90/min, temperature 100° F.

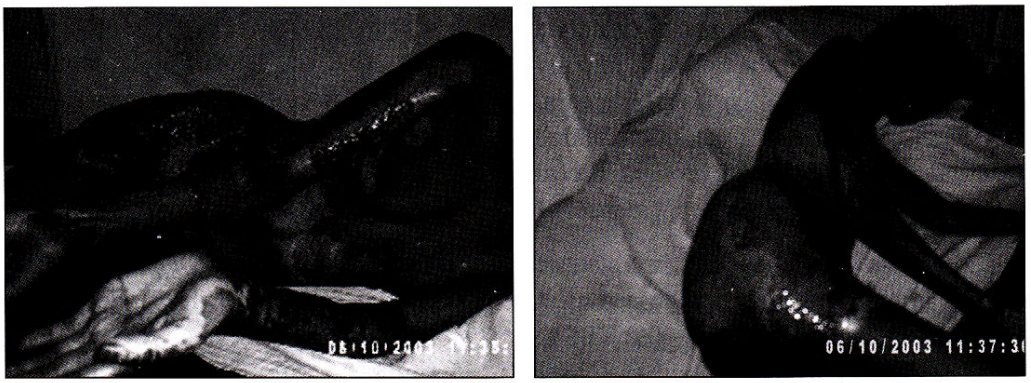
Immediately after admission intravenous channel was established, fluid resuscitation started and a urinary output of >50 ml/hour established. Inj. Tetanus toxoid and TIG were given. one unit of blood was transfused. The surgical excision of the infected dead skin and wound debribement was done. The free pus, collected from underneath the dead skin, was sent for culture sensitivity (C/S). Then regular dressing was continued, initially by 1% gentian violet & later on by Silver sulphadiazine cream; the wound covered with liquid paraffin soaked gauze and roll bandage. Those areas not suitable for closed dressing were kept open. In the first few days (5-7 days) of hospital stay she showed high rise of temperature (102-104° F).

The laboratory values showed Hb% 9.9gm/dl; WBC: Total Count 20,000/cu mm; Differential count: Neutrophil 90%, Lymphocyte 6%, Monocyte2%, Eosinophil 2%; Serum electrolytes: Serum Na^+ 135.1mmol/L, Serum K^+ 3.51mmol/L, Serum Cl^- 101.1mmol/L, Serum HCo_3^- (23.2 mmol/L), blood urea (3.9 mmol/L) and serum creatinine (1.0 mg/dl). Within 3rd day of hospital stay, vital signs were stable, fluid resuscitation was established. The patient's pain was controlled by injection tramadol. Although the C/S report showed multi-drug resistance (MDR), we continued injection Cephradin and Gentamycin as combination that showed a good clinical response as well as control of

high rise of temperature. Time to time kidney function was monitored. Daily dressing by silver sulphadiazine with liquid paraffin soaked and/or betadine soaked gauze was done after a thorough daily wash of whole body by lukewarm water. We followed both open and closed methods of burn dressing and patient was kept in general ward.

To prevent the contracture we kept the elbow and wrist joint of both the upper limb in extended position by using wooden splints, passive and active assisted movements, scar massage & pressure[4].

Around/ Up to 46th days of burn, after daily dressing the wound became gradually shortened and covered with healthy granulation tissue **(photograph)** and a decision of skin grafting was made. The swab from wound, axillary area, throat & nose were sent for C/S showed MDR, only sensitive to cefuroxime and ceftriaxone but there were no evidence of growth of â hemolytic streptococcus. On 48th day autograft was done by partial thickness split skin graft over almost a 12% TBSA. One unit of whole blood was transfused per-operatively and another post-operatively. Post-operative Hb% and S. electrolyte values were assessed and were found within normal limit. Patient showed a good response with injection Ceftriaxone. Dressing in the donor site of right thigh have soaked and were changed on the 3rd POD. On 9th POD recipient sites were opened and donor site was opened on the 15th POD. The grafts were taken well. The patient was discharged on 20th POD with the advice to avoid any job with high exposure to sunlight up to 2 years[5].



Photograph: Front & Lateral view of the patient on 37th day of burn.

DISCUSSION

All burn causes destruction of tissues of the skin. Burns range in severity from minor injuries that require no medical treatment to serious, life-threatening or fatal injuries. Burns are categorized in terms of degrees which are described in Table-1 and Figure-1[5,6,7].

Table 1: Classification of Burn

	Degree	Characteristics	Special note
Superficial Burn *(First Degree)*	• Causes : sunburn, minor scalds • Generally heal in 3-6 days with no scarring	• Minor damage to the skin • Color - pink to red • Painful • Skin is dry without blisters	• Not included in calculations of burn size1
Partial Thickness Burn *(Second Degree)*	• Damages, but does not destroy top two layers of the skin		
• **Superficial Partial Thickness Burn**	• They heal within three weeks with minimal cosmetic defects	• Blisters are present • Under the blister, they are red and moist. • Very painful	• You wouldn't notice the scars with a casual look. • Counted in TBSA
• **Deep Partial Thickness Burn**	• They require longer than three weeks to heal and usually produce severe hypertrophic scarring.	• dry and may appear ivory or pearly white. o Very painful	• A **skin graft** is usually recommended for deep second-degree burns
Full Thickness Burn *(Third Degree)*	• Destroys all layers of the skin • May involve fat, muscle and bone • Will require skin graft for healing	• Skin may be very bright red or dry and leathery, charred, Waxy white, tan or brown • Charred veins may be visible • Area is insensate– the person Is unable to feel touch in areas of full thickness injury	• Best treated with early excision (removal of dead tissue), skin grafting

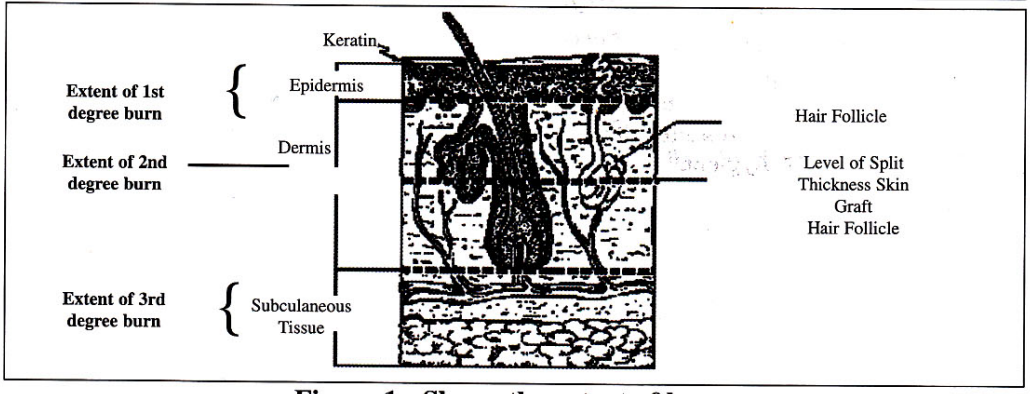


Figure-1 : Shows the extent of burn.

The patient had burn involving the back and both the upper limbs, the right lateral surface of waist and upper 1/3rd of thigh in a combination of partial and full thickness burn with superadded infection. Ideally such a case needs to be treated in Burn care unit, with facilities of intensive monitoring of CVP, Blood gas analysis, proper biochemical review and with all the logistic supports to prevent cross infections and expertise aseptic precaution.

Different studies all over the world have shown major burn cases result in a high mortality rate. American University of Beirut Medical Center, Lebanon, in a study of over 5 year period has shown- "As the % TBSA Burn increases, the hospital stay decreases reflecting the increased mortality for major burns"[8]. Another study of over one year period shows- "the mortality rate has been 100% in burns above 60% TBSA, 69% in 41-60% burns, and 12% in burns of less than 40% TBSA"[9]. Moreover, both morbidity and mortality are directly related to the % of burn and the standard of general care the patient receives on burns ward[10,11]. But as a matter of fact, in the Third world countries the incidence of burn and post-burns infections by MDR are more prevalent due to indiscriminate use of antibiotics, lack of stringent safety regulations and proper hygiene[11]. As the patient had gross socio-economic constraint we continued the conventional approach of burn management in the patient with the highest efforts that could be provided from our side. The patient showed a very positive response within a short period of time.

CONCLUSION

Management of a major burn after secondary infection necessitates the admission and resuscitation in a unit that can provide exact assessment, monitoring of overall burn condition, maintenance of hygiene and above all a keen nursing care. Although there are many evidences of very high mortality (around 70%) of burns involving >40% TBSA in specialized burn centers an honest effort to combat this fatal accidental injury may result in a successful outcome, even when the patient is managed in a general surgical ward.

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ANNOTATION

KAWASAKI DISEASE: EVIDENCE BASED MEDICINE SUPPORTS EARLY RECOGNITION AND PRESUMPTIVE TREATMENT

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ABSTRACT

Kawasaki disease is a systemic febrile vasculitis of unknown etiology. It is now the commonest cause of acquired heart disease in children in developed countries and also a leading cause in many parts of the world. As its cause remains unknown, it presents to doctors with many difficulties in diagnosis. Atypical presentation of Kawasaki disease is also recognized recently. Since there is no specific diagnostic test for the disease many cases are missed leading to fatal out come. As doctors are in dilemma to start the treatment or not, evidence has been sought in recent literature that support early recognition and presumptive treatment. Evidences suggest early recognition is vital to prevent cardiac complications and presumptive treatment should be started in any child with persistent fever and some features of Kawasaki disease, even if symptoms do not meet the full diagnostic criteria and atypical Kawasaki disease, to decrease both the incidence and severity of coronary aneurysm formation.

[J Med Coll Women Hosp 2004; 2(1): 43-47]

Indexing words : **Kawasaki disease** **Atypical Kawasaki disease** **Cardiac complications** **Presumptive treatment**

INTRODUCTION

Kawasaki disease (KD) is an acute systemic febrile vasculitis of unknown etiology predominantly affecting children under five years of age with immediate and long-term cardiovascular sequelae and has been reported from many parts of the world[1,2,3,4,5,6]. In the absence of a specific diagnostic test, clinical criteria have been established to assist the clinician in making the diagnosis[1,2,7]. The term "Atypical Kawasaki Disease (AKD)" has

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been coined to describe patients who have coronary abnormalities of KD, but do not exhibit at least five criteria for the diagnosis of the disease[3]. The most important complication, coronary arteritis leading to formation of aneurysms, occurs in 20-30% of untreated patients. Patients also suffer long term morbidity as a result of scarring of coronary arteries, intimal thickening and accelerated atherosclerosis. Evidences from the recent literature suggest that intravenous immunoglobulin is the most effective treatment of KD given within 10 days of onset (Table-1). This significantly decreases both the incidence and severity of aneurysm formation and provides dramatic resolution of inflammation and relief of symptoms.

DISCUSSION

Dr. Tomisaku Kawasaki described the disease nearly 30 years back[7]. KD is now the commonest cause of acquired heart disease in children in developed countries[8]. Its cause remains unknown and it presents to doctors with many difficulties in diagnosis and management. Kawasaki disease is a systemic febrile vasculitis predominantly affecring children aged under 5 years. The incidence in Britain is 3.4 per 100 000 children aged under 5 years, about a third of the incidence is reported in the United States and a 30th of that in Japan. The most important complication, coronary arteritis leading to formation of aneurysms, occurs in 20-30% of untreated patients. Thrombosis within an aneurysm, myocardial infarction, and dysrhythmias may occur in the acute phase of the illness. The case fatality rate in Britain in 1990 was 3.7%, which compares unfavourably in comparison with the United States and Japan, and in some centres it is as low as 0.1%[9]. Patients also suffer long term morbidity as a result of scarring of coronary arteries, intimal thickening, and accelerated atherosclerosis[10].

There is no specific diagnostic test of the disease, and many cases are missed. Six deaths were recorded in Britain in 1990, but only one case was diagnosed during life[8]. The diagnosis is based on fulfilment of clinical criteria (**Box-1**)[11].

Box-1

Diagnostic criteria for Kawasaki disease: Presence of at least five of six conditions:

- Fever for five days or more.
- Bilateral (non-purulent) conjunctivitis
- Polymorphous rash
- Changes in lips and mouth:
  Reddened, dry, or cracked lips
  Strawberry tongue
  Diffuse redness of oral or pharyngeal mucosa
- Changes in extremities :
  Reddening of palms or soles
  Indurative oedema of hands or feet
  Desquamation of skin of hands, feet, and groin (in convalescence)
- Cervical lymphadenopathy :
  More than 15 mm in diameter, usually unilateral, single, non-purulent, and painful.

However, many common childhood infections have similar clinical features. Furthermore, the diagnostic features of KD may appear sequentially rather than simultaneously. The two features that doctors most often remember are desquamation of the rash and thrombocytosis. Unfortunately, these features are least useful in reaching an early diagnosis because they usually occur later in the disease. Moreover, the clinical diagnostic criteria do not identify every case; and thus "incomplete" or "atypical" cases have come to light as coronary artery aneurysms have been found on echocardiography or at necropsy[12].

Then, how can a doctor distinguish KD from more common causes of fever with a rash **(Box-2).** Remaining alert to the possibility of the diagnosis is critical. Many "Textbook" cases of the disease are missed simply because the diagnosis is not considered. Kawasaki disease can be mistaken for measles, but measles has become less common in Britain since the recent vaccination campaign. Kawasaki disease is a systemic disease and typically affects many systems. Doctors should consider the diagnosis because of less characteristic features such as rhinorrhoea, cough, abdominal pain, vomiting, diarrhoea, pain and swelling of joints, involvement or the central nervous system, abnormal liver function test, and sterile pyuria, which can all occur in KD.

Box-2

Exclusion of diseases with similar presentation:

- Staphylococcal infection (such as scalded skin syndrome, toxic shock syndrome)
- Streptococcal infection (such as scarlet fever, toxic shock-like syndrome). Throat carriage of group A streptococcus does not exclude the possibility of Kawasaki disease
- Measles and other viral exanthems
- Leptospirosis
- Rickettsial disease
- Stevens-Johnson syndrome
- Drug reaction
- Juvenile rheumatoid arthritis

There are some useful clues to the diagnosis. Most of the diseases for which Kawasaki disease is mistaken do not cause fever for more than five days. In addition, the fever in KD is often unresponsive to antipyretics, and the child becomes miserable.

An additional sign sometimes seen in the acute phase is redness and induration at the site of a BCG scar. In KD an inflammatory process and an acute phase response is characteristic and its absence suggests an alternative diagnosis. Presumptive treatment should be started in any child with a persistent fever, with some of the clinical features of KD, and an acute phase response even if symptoms do not meet the full diagnostic criteria.

Intravenous immunoglobulin is the most effective treatment (Table-1). Given within 10 days of onset, this significantly decreases both the incidence and severity of aneurysm formation, as well as providing dramatic resolution of inflammation and relief of symptoms[13,14]. A single high dose of 2g/kg is more effective than the previously recommended regimen of 400 mg/kg for four days[13]. However, despite its proved efficacy, many patients receive delayed or inadequate amounts of immunoglobulin, or even none at all[14]. In Britain in 1990 only 7% of patients were given the recommended optimal treatment, and 39% were not given any immunoglobulin at all.

Table-1: Treatment of Kawasaki disease

Drug	Dose	Notes
Acute phase of disease		
Intravenous immunoglo bulin	Single high dose infusion of 2 g/kg over 10 hours (reduce infusion rate in older children because of risk of fluid overload)	Reactions (shivering, fever, hypotension) are uncommon and usually managed by stopping infusion and restarting at reduced infusion rate Treatment with chlorp-heniramine and hydro-cortisone is occasion-ally required.
Aspirin	30 mg/kg/day in three or four divided doses	Continue for 14 days or until fever subsides, then change to low antiplatelet dose
Convalescent phase of disease		
Aspirin	3-5 mg/kg/day (antiplatelet dose) in single daily dose	If no coronary artery lesions detected on initial echocardiogram then continue until repeat echocardiogram at 6-8 weeks. If coronary artery lesions are detected at any stage then continue long term

There is often uncertainty about the use of immunoglobulin in patients diagnosed more than 10 days after the start of the disease. This has not been tested in a prospective controlled trial. However, many pediatricians recommend the use of immunoglobulin in such patients if there is evidence of ongoing inflammation, as suggested by continuing fever, malaise, and raised acute phase reactants. Re-treatment with immunoglobulin is recommended for persistent or recrudescent disease. Aspirin remains an integral part of treatment, although its use in Kawasaki disease has not been subjected to prospective controlled trials[15].

Many of the difficulties in diagnosing and managing patients with Kawasaki disease would be solved if the cause of the disease was known. The epidemiology strongly suggests an infectious aetiology[16]. There was much interest in the hypothesis that the disease is caused by a bacterial super antigen toxin, similar to those responsible for the staphylococcal and streptococcal toxic shock syndromes[17]. The recent report of a patient who fulfilled the clinical criteria for staphylococcal toxic shock

syndrome but who also had coronary artery lesions typical of KD supports the suggestion that KD and toxic shock syndrome share a common aetiology[18]. However, there is still conflicting evidence for the superantigen theory[19]. Future advances in diagnosis and treatment are likely to depend on the definitive identification of the cause of the disease.

CONCLUSION

As early therapy with high dose intravenous immunoglobulin (IVIG) has been shown to decrease the prevalence of coronary artery aneurisms in patients with Kawasaki Disease, a high index of suspicion for Kawasaki Disease in infants is important and consideration should be given to a cardiac evaluation including an echocardiogram in young infants with prolonged fever who do not fit convincingly into another diagnostic category. A greater awareness about the symptoms and signs which constitute this syndrome would lead to more children with AKD being diagnosed and treated appropriately. It has been reviewed in recent literature and supports that presumptive treatment should be started in any child with a persistent fever, some of the clinical features of KD and an acute phase response even if symptoms do not meet the full diagnostic criteria.

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BRIEF COMMUNICATION

IMMUNOMODULATION OF LDL-CHOLESTEROL METABOLISM

ASM Giasuddin[1] MSc PhD DipImmunol FIBMS MNYAS

ABSTRACT

Cholesterol is a highly decorated small molecule in biology and medicine as thirteen Nobel prizes were awarded for research on it. Several experimental systems have demonstrated that non low – density lipoprotein – receptor (non LDL – R) mediated mechanisms must account for disposal of about one – third of circulating LDL – cholesterol (LDL – Ch). To address this issue, Alving and Wassef proposed an immunological mechanism for immunomodulation of LDL – Ch metabolism through anti – cholesterol antibodies. Recently, many investigators have been trying to exploit the vascular protective effects of immunization with oxidized LDL, HDL and their apoproteins. Inhibition of development of atherosclerosis in mice by activation of immune responses against three ApoB-100 peptide sequences has led to great hopes for the development of a heart attack vaccine for humans. This may help to achieve the ambitious goal set for the elimination of coronary heart disease (CHD) early in the 21st century.

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Indexing words : Cholesterol LDL – Cholesterol Immunomodulation Metabolism

Cholesterol is probably the most highly decorated small molecule in biology, as thirteen Nobel prizes were awarded for research on the structure, conformation and receptor cell biology of it and related molecules [1,2]. The low-density lipoprotein receptor (LDL-R)–mediated pathway for cholesterol homeostasis was discovered by Brown and Goldstein [1]. This has led to the development of drugs and drug strategies aimed at lowering LDL–cholesterol (LDL–Ch), very low–density lipoprotein–cholesterol (VLDL–Ch) and intermediate – density lipoprotein cholesterol (IDL–Ch) levels by inhibiting cholesterol biosynthesis via inhibition of hydroxymethyl glutaryl–CoA (HMG–CoA) reductase and up–regulating LDL-R

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expression [3,4,5]. Based on these drug developments, an ambitious goal has been set for the elimination of coronary heart disease (CHD) early in the 21st century [6]. Despite the splendid array of cholesterol-lowering drugs, the achievement of this goal is still a challenge. LDL-R is characterized by high affinity binding to LDL, with a marked preference for binding LDL rather than high-density lipoprotein (HDL) [7]. However, LDL-R independent uptake of radiolabelled LDL has been readily demonstrated invivo in several experimental systems: (i) genetically deficient patients or rabbits in which LDL receptors are absent; (ii) normal humans or animals in which the radiolabelled LDL that is injected has been chemically modified to block binding to the LDL-R [1,8]. Non LDL-R mediated mechanisms must therefore account for removal of about one-third of circulating LDL-Ch. Cholesterol has been shown to be extremely immunogenic molecule when combined with the proper adjuvant and carrier such as protein-free liposomes, lipid A and silicone [9, 10, 11]. In view of the impressive immunogenecity of cholesterol promoted by lipid A endotoxin, it is perhaps not surprising that naturally occurring antibodies to cholesterol are found in humans and in many animals [9, 12]. Some investigators reported detectable levels of natural antibodies to cholesterol (ranging from very high to very low) in sera of normal human volunteers and in patients with chronic Chaga's disease [13, 14]. Interestingly, naturally occurring antibodies to cholesterol bind specifically to human LDL-Ch, VLDL-Ch and IDL-Ch, but lack specificity for HDL-Ch, and the antibodies bind to cholesterol extracted from HDL, but not to intact HDL [15]. A relatively high protein to cholesterol ratio found at the surface of HDL appears to have an inhibitory effect, perhaps by steric hindrance, charge effects or hydrophobic blocking, on the binding of the antibody molecules to the extremely small 3β-hydroxy group of free cholesterol at the surface of the HDL particle. This implies that antibodies to cholesterol might have easy access to the higher concentration of relatively exposed cholesterol on the surface of LDL, VLDL and IDL particles. These antibodies to cholesterol that differentially bind to LDL may also explain the previous finding that immunoglobulins (IgM & IgA) are the major LDL-binding proteins in normal human plasma[16]. These findings has led to the question of whether antibodies to cholesterol could have the ability to modulate LDL-Ch, VLDL-Ch and IDL-Ch. Could immunological mechanisms play a role in cholesterol metabolism?

To address this issue of immunomodulation of LDL-Ch metabolism by antibodies to cholesterol, Alving and Wassef conducted several experiments immunising rabbits with cholesterol using different immunisation protocols [17]. The results of these investigations showed that antibodies to cholesterol have the capacity to bind and

remove large amounts of LDL-Ch from circulation, thus suggesting that this process could occur under normal conditions. They proposed an immunological mechanism for immunomodulation of LDL-Ch as shown in **Fig-1**. Antibodies to cholesterol bind to LDL (or VLDL or IDL) causing opsonisation by complement and removal of the lipoprotein from the blood by macrophages. The initial binding of the LDL-Ch-antibody-complement complex probably occurs on erythrocytes through the process of immune adherence to complement receptor 1 (CR1), with subsequent shuttling of the immune complex to the macrophages via Fc-recepters. This results in LDL-R-independent uptake, i.e. immunomodulation of cholesterol specific to LDL, VLDL and IDL (lipoproteins containing 'bad' cholesterol) rather than to HDL-Ch (lipoprotein containing 'good' cholesterol). Of the multiple lines of evidence that support the concept of immunomodulation of LDL-Ch metabolism, the major ones include : (i) the ubiquitous existence of antibodies to cholesterol in human serum; (ii) differential binding of the antibodies to LDL-Ch, VLDL-Ch and IDL-Ch, but not to HDL-Ch; (iii) the blocking of diet-induced hypercholesterolemia in rabbits immunized with cholesterol; (iv) the demonstration of immunoglobulins as a major LDL-binding protein in human serum; and (v) the observation that immunosupression (administration of cyclosporine and steroids) is associated with hypercholesterolemia[17, 18, 19].

If regulation of LDL-Ch is at least partially controlled by antibodies to cholesterol, this raises the possibility that increasing titre of the antibodies by direct immunization with a cholesterol vaccine could have a beneficial effect as an unique new tool for lowering serum LDL-Ch level[17]. Many investigators have been trying to exploit the vascular protective effects of immunization with oxidized LDL, HDL and their apolipoproteins[20,21,22]. More recently, synthetic peptides that are replicas of smaller portions of ApoB-100 were designed and tested and evidence was obtained for presence of antibodies against ApoB-100 in human serum. It clearly indicated the possibility of inhibiting development of atherosclerosis by activation of immune responses against three ApoB-100 peptide sequences [22, 23]. Coronary heart disease (CHD) is a burning issue in recent times and the most important risk factor for atherosclerosis leading to CHD is the hyperlipidemia particularly LDL – Ch. These new strategies will therefore have far reaching implications in controlling atherosclerosis. This may help eradication, or at least reduce further the incidence of CHD with the development of a heart attack vaccine in the near future. This would not however be a reason to abandon the proven methods of reducing risk – which are lifestyle modification and medication[3, 4, 5, 24].

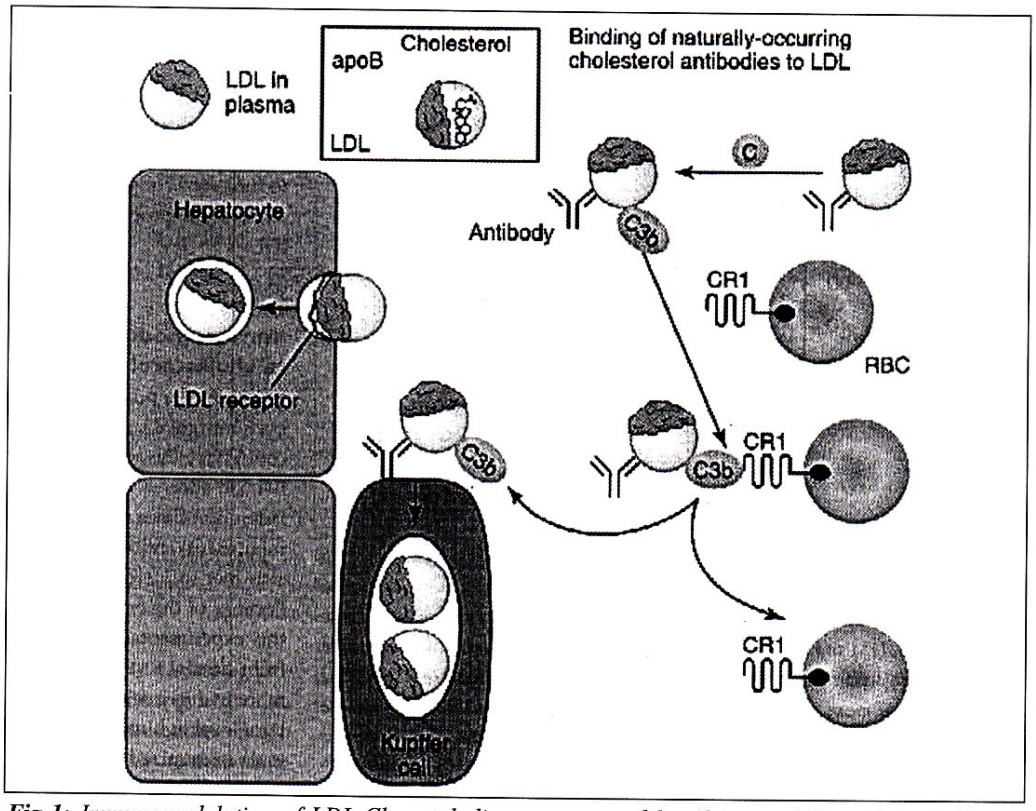

Fig-1: *Immunomodulation of LDL-Ch metabolism as proposed by Alving and Wassef. Antibodies to cholesterol bind to LDL (or VLDL or IDL) causing opsonisation by complement and removal of the lipoprotein from the blood by macrophages. The initial binding of the LDL-antibody-complement complex probably occurs on erythrocytes through the process of immune adherence to CR1, with subsequent shuttling of the immune complex to the macrophages. This results in LDL receptor-independent uptake of lipoprotein. Abbreviations: C, complement; CR1, complement receptor type 1; RBC, Red blood cells; LDL, Low-density lipoprotein; VLDL, Very low-density lipoprotein; IDL, Intermediate- density lipoprotein; apo-B, Apolipoprotein B* ***(taken from ref. no. 17).***

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CLINICAL MEETINGS AT MCW&H

The clinical meetings are held weekly at MCW&H. Some of the presentations made were the following :

DATE : 19.02.2003
TOPIC : Surveillance on 6 Expanded Programme of Immunization (EPI) diseases & Adversed Event Following Immunization (AEFI).
SPEAKER: MCW&H in collaboration with EPI WHO and IOCH; Dr. EGP Haran, Senior Child Health Advisor, IOCH; Dr. Anisur Rahman Siddique. Divisional Co-ordinator, Dhaka division, WHO; Dr. Taslim Uddin, Operation & Surveillance Officer IOCH; Dr. Ulfat Ara Al Jalila, MO, EPI, DGHS; Dr. Tariqul Islam, OSO, Co-ordinator, IOCH-DCC; Dr. Akhter Hamid, NIO, WHO, Dhaka, Bangladesh.
HIGHLIGHTS : This presentation focused on the following : Epidemiology of poliomyelitis, Polio eradication: status & strategies; Acute Flaccid paralysis (AFP) surveillance & role of hospital in AFP surveillance; Neonatal tetanus elimination- epidemiology & surveillance; Measles control epidemiology & surveillance; Diptheria, Pertusis & TB surveillance; Pertusis: Definition & Bangladesh statistics; Tuberculosis epidemiology & control programme; Surveillance management; AEFI surveillance.

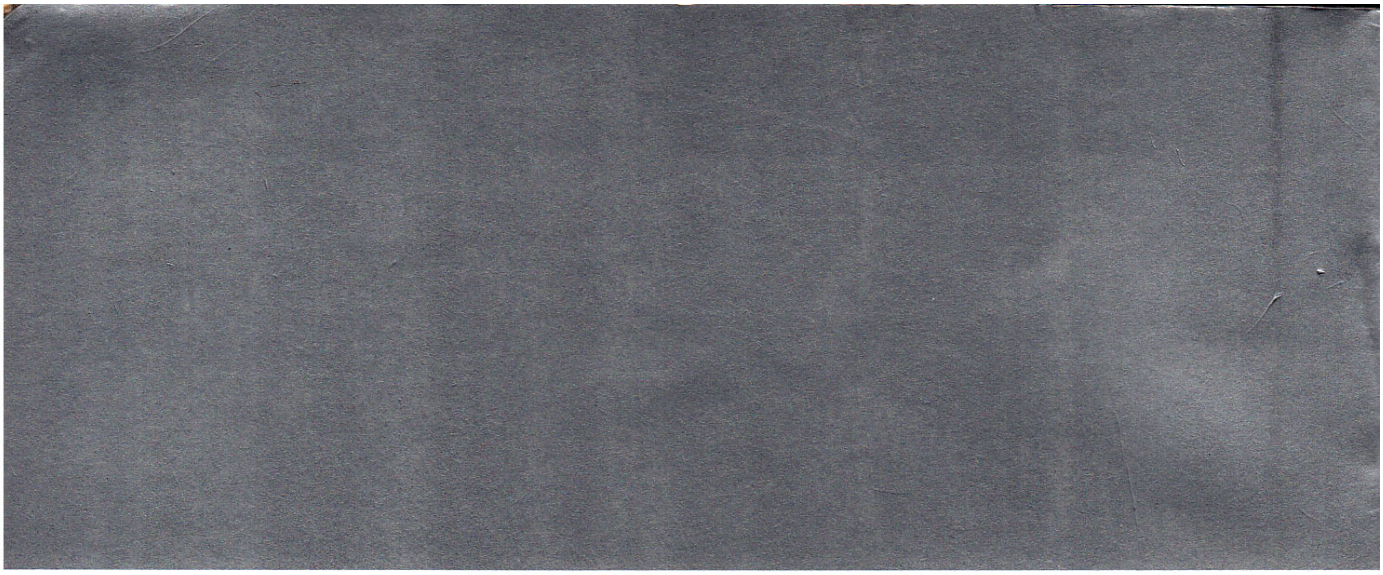
DATE : 16.04.2003
TOPIC : Liver transplant experience in Singapore
SPEAKER : Dr. Zulfikar Rahman Khan, FCPS FRCS FICS
HIGHLIGHTS : Different Aspects of liver transplants discussed were : Indications of liver transplant; Contraindications of liver transplant; Donar selection; Procedure of liver transplant surgery; Prognosis of liver transplant surgery.

DATE : 23.04.03
TOPIC : Maternal nutrition, Diet & Cancer
SPEAKER : Prof. Dr. S.M. Keramat Ullah, Institute of Nutrition & Food Science, Dhaka University, Dhaka, Bangladesh.
HIGHLIGHTS : This presentation focused on : Different aspect of cancer in relation to diet; Impact of Genetic; Environmental & life style risk; Factor on cancer; Different cancer site are associated with different; Dietary risk factor; Preventive measures & early control.

DATE : 23.07.03
TOPIC : Ischaemic Heart Disease
SPEAKER : Maj.Gen (Retd.).Prof. Ziauddin Ahmed, MBBS MCPS FCPS MRCP FRCP(I) FRCP (G), Principal & Head of the Dept. of Medicine, MCW&H, Dhaka, Bangladesh.
HIGHLIGHTS : Different aspects of IHD discussed were as follows : Introduction of IHD; Epidemiology of IHD; Description of anatomical heart; Pathogenesis of IHD;Evolution of an atheromatous plaque; Post mortem findings of heart; Operative specimen; Risk factors of IHD; Clinical presentation of IHD; IHD in women; Management; Recommendation; Barriers to implementation of preventive services; Advice to patient with angina. Conclusion.

DATE : 25.02.04
TOPIC : Kidney & Hypertension
SPEAKER : Maj. Gen (Retd.) Prof. Ziauddin Ahmed, MBBS MCPS FCPS MRCP FRCP(I) FRCP(G); Principal & Head, Dept. of Medicine, MCW&H, Uttara, Dhaka, Bangladesh.
HIGHLIGHTS : Different aspects of kidney & hypertension discussed were : Factors influencing the development of essential hypertension; Etiology; Causes of secondary hypertension; Pathophysiology; Clinical features of hypertension; Long standing hypertension effecting kidney; Acute & chronic renal failure associated with hypertension; Investigation of a hypertensive patient; Recent hypertension level study; General measures in the management of hypertension; Anti hypertensive drug therapy; Strategy for drug therapy in hypertension.

DATE : 31.03.04
TOPIC : Medical ethics & professionalism- A personal feeling.
SPEAKER : Prof. A.K.M. Mahbubur Rahman, MBBS FCPS FRCS(Glasgow), FRCP(Edin).
Dept. of Surgery, MCW&H, Uttara, Dhaka, Bangladesh.
HIGHLIGHTS : This presentation focused on the following : Different definitions of ethics; Ethics in relation to medical science; Moral values; Patient-doctor communication; Doctor's role in ethical point of view; Ethical points in favour and disfavour of general practitioners; Social aspect of ethics; Different personal feelings.



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