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The Evolution of the AIDS Epidemic

Q. AHMAD, FCPS[a], J. YEASMIN, MBBS[b]

Summary :

The first few cases of the disorder that later came to be called AIDS were briefly recorded in the June 5, 1981 issue of the centers for Disease Control's Morbidity and Mortality Weekly Report' and then described in more detail in the New England Journal of Medicine on December10, 1981. This review traces the spread of the virus amongst the different population groups and outlines the efforts to understand the nature of the epidemic. It provides an insight into the evolution of this new epidemic in our midst.

(J. Bangladesh Coll Phys Surg 1992; 10 : 33—38)

Introduction :

A 30 year old homosexual man was referred to UCLA (University of California at Los Angeles) Medical Centre in March, 1981 with a four month history of daily fever and malaise. He had been hospitalized elsewhere in February 1981, at which time generalized lymphadenopathy without hepatosplenomegaly was present. The white cell count was 4200 with 10% lymphocytes. The sedimentation rate was 126 mm in the first hour. Delayed skin tests to five recall antigens were negative. A liver biopsy showed mild focal non-specific hepatitis. Inclusion bodies were absent. The level of antibodies to cytomegalovirus rose from <1:16 to >1:18. Cytomegalovirus was cultured from samples of urine and buffy coat. Antibody evidence of past infection with Epstein Barr and hepatitis B viruses were detected.

On April 21, 1981 the patient was admitted to UCLA Medical Centre, after four days of nonproductive cough, dyspnoea, rising fever, and chills. He reported the recent onset of rectal and oral thrush. He appeared acutely ill, with a temperature of 39° C. The arterial partial pressure of oxygen during breathing of ambient air was 44 mm of Hg, and a chest film showed a diffuse interstitial pattern. Silver staining of bronchial brushings revealed numerous Pneumocystis carinii organisms. A 14 day course of intravenous TMP-SMZ led to resolution of the pneumonia. The white cell count declined to 1900, with 10 percent lymphocytes; the platelet count was 80,000. Bone marrow aspirate and histology were normocellular, with slightly increased numbers of plasma cells. Urine culture grew adenovirus. On June 24, 1981 funduscopic examination revealed multiple discrete white retinal opacities that were consistent with the occurrence of micro infarction of the nerve fiber layer of the retina. There was no history of visual disturbance. The patient was readmitted on August 15, with gradually increasing dyspnoea, cough, and interstitial infiltrates. Bronchial brushings were negative for P carinii; however, numerous pneumocysts were found on open lung biopsy. Cytomegalovirus was cultured from the tissue specimen. The patient's condition deteriorated despite five days of Pentamidine therapy. The fever and pulmonary infiltrates cleared after a four week course of high dose TMP-SMZ, and low dose prophylaxis was instituted after recovery.

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This case report has been taken from the article, "Pneumocystis carinii pneumonia (PCP) and mucosal candidiasis in previously healthy homosexual men: Evidence of a new Acquired Cellular Immunodeficiency", published by Gottlieb et al[1] in the December 10, 1981 issue of the New England Journal of Medicine (NEJM), and describes the first patient of AIDS. In the same issue of the NEJM, two more results were published about patients who had immunological defects indicative of abnormal cell-mediated immunity. In one, Seigal et al[2] described four patients who developed severe chronic ulcerating perianal lesions, and in another, Masur et al[3] described 11 cases of community acquired Pneumocystis carinii pneumonia between 1979 and 1981, in drug abusers or homosexuals or both. PCP had been known to occur in the Pre-AIDS era, but it was almost exclusively limited to patients whose immunity was severely compromised by underlying disease, immuno-suppressive therapy or both. After reviewing requests for Pentamidine in the USA in the period from 1967 to 1970, when it was the only recommended treatment for PCP, Walzer et al [4] and Hughes[5] found only one case of confirmed PCP in a patient without a known underlying condition, which could have caused the immunosupression.

Hymes et al[6] had also reported Kaposi's Sarcoma (KS) in eight homosexual men, who had no cause of immuno-suppression. Though a milder form of KS had been previously reported in elderly people, the agressive form seen in these young patients had only been reported previously in renal transplant patients[7] and those receiving immunosuppressive therapy[8]. Later Friedman et al[9] also reported the incidence of disseminated KS in homosexual men.

Dr. David T. Durack[10] in an editorial titled "Opportunistic infections and Kaposi's Sarcoma in homosexual men" published in the December 10, 1981 issue of NEJM presented the salient features that had emerged about this unexpected outbreak. The patients were typically young homosexual men, most of whom lived in large cities and many of whom used drugs; the infectious agents were low-grade pathogens that often cause opportunistic infections in immunocompromised hosts; and the death rate was fearfully high. The nature and multiplicity of the opportunistic pathogens and the overwhelming severity of many of the illnesses indicated that the normal defences were severely compromised. The most obvious link of this new syndrome was with homosexuality. Cytomegalovirus (CMV) infection was the second common threat amongst the cases recorded so far. But CMV infection was already common in the homosexual community and more than 90 percent of homosexuals had antibodies indicating previous infection[11].

Since June 1981, the Centers for Disease Control (CDC)[5] U. S. A. had learned of an increased occurrence of Kaposi's Sarcoma, Pneumocystis carinii pneumonia, and other serious opportunistic infections concentrated among homosexual men in the United States. The CDC formed a task force to study this new phenomenon. In their report, the task force stated that there were 159 documented cases of KS, PCP and other serious opportunistic infections reported to CDC, between June 1 and November 10, 1981[13]. Of 159 cases with KS, PCP or both, more than 40 percent were reported to have other serious opportunistic infections. These included 24 CMV infections, six progressive or disseminated herpes simplex virus infections, five cryptococcal meningitis infections, twelve invasive gastrointestinal candida infections, seven disseminated mycobacterial infection, out of which five were with atypical mycobacteria, and ten other infections. In each group, many patients were reported to have had a prodromal illness lasting for weeks to months and

characterized by weight loss, lymphadenopathy, fever, and diarrhoea. There was only one woman patient. Ninety two percent of these men were reported to be homosexual or bisexual, where sexual preference was known. The task force was of the view that the current outbreak of KS and PCP among homosexual men of the same age and racial groups who live in the same geographic areas, was suggestive of occurrence of a single epidemic of underlying immunosuppression in these men.

The CDC in early 1982, reported the occurrence of KS and opportunistic infections in persons who were exclusively intravenous drug users and did not indulge in homosexuality[13]. This raised the possibility of drugs in the causation of immunosuppression, especially inhalants containing nitrites.

These are commonly used to intensify orgasm. Users of amyl nitrite are more likely than non-users to have had hundreds of sexual partners and to contract veneral diseases[14]. Some degree of alternation in cellular immune status had already been reported in those indulging in drug abuse[15,16]. But it's effect alone in the causation of immunosuppression was difficult to judge as people using these are also often indulging in all sorts of unusual sexual practices.

Kornfeld et al[17] studied the T lymphocyte sub-populations in homosexual men and reported that altered distribution of T lymphocyte subpopulations were associated with the presence of certain symptoms and with sexual promiscuity but not with the use of inhaled nitrites. There were reduced ratios of T4 (helper cells) to T8 (suppressor cells). Reduced T4/T8 ratios had been previously reported in a variety of diseases including cytomegalovirus induced mononucleosis[18], Epstein Barr virus infection, influenza, acute and chronic hepatitis B infection, and primary biliary cirrhosis[19,20].

Initially the patients suffering from AIDS were mostly confined to homsexuals, bisexuals and drug abusers. But the CDC in 1982 described seven patients in whom classic haemophilia was complicated by PCP[21,22]. All had been treated with Factor VIII, and none of them were homosexual or bisexual and had not indulged in drug abuse. In the six patients tested, the immunological defects were similar to those observed in homosexual men with AIDS:

Late in 1982, came the report of features of AIDS in infants, who had no congenital immunological abnormality[23]. At the same time CDC reported AIDS, possibly transmitted by blood transfusion[24].

The occurrence of AIDS in patients who were neither homosexual nor addicted to drugs suggested that immunity was not conferred or guaranteed by virtue of a traditional life style. The understanding of the disease was misled when it was reported that AIDS was spreading amongst the Haitian immigrant community in the USA[25]. Vieira et al.[26] in January 1983 described AIDS in 10 previously healthy Haitian immigrants who were neither homosexual or bisexual nor had they indulged in any sorts of drug abuse. To identify the characteristics of the acquired immunodeficiency syndrome as it occured in Haiti, Pape et al[27] studied 61 previously healthy Haitians who had diagnoses of either KS, opportunistic infections or both established in Haiti betwen June 1979 and October 1982. The types of opportunistic infections and the clinical course in Haitians with KS and opportunistic infections were similar in most aspects to those in patients with AIDS in the United States.

In March 1983 the Public Health Service of the United States recommended that persons with symptoms suggestive of AIDS and members of groups at increased risk for AIDS should refrain from donating blood or plasma.

Several other organizations associated with blood transfusions had issued similar recommendations and the Food and Drug Administration had issued specific guidelines for blood and plasma collection centers.

With spread of the epidemic, the concept that it may be caused by a transmissible biologic agent was taking firm conviction. Immunodeficiency was reported among female sexual partners of males with AIDS[28]. Harris et al[29] reported in a study of seven female sexual partners of male patients with AIDS. All the seven male partners were drug abusers. Al the women stated that they had maintained exclusively heterosexual relations, only with their Partners. All of them denied the use of intravenous drug or inhalants.

Emeritus Professor Waterson of Royal Postgraduate Medical School, London in March, 1983[30] reviewed the sudden surge of such immune deficiency. Till then 300 patients had been described, of whom 291 came from the United States and only nine from six other countries, but several of those had recently been in the United States. 290 were men, of whom over four fifths were under 45, and most were homosexuals. 135 had PCP, 115 had KS and 28 had both of them. The remaining 22 had various opportunistic infections. More than two-fifths of the earlier patients had already died. The cardinal feature was the occurence of infections which test the host's cellular rather than humoral immunity and which are normally seen only in patients who are naturally or artificially immunocompromised.

The most sinister feature of this acquired immune deficiency at that time was it's communicability, perhaps principally by intimate physical contact. The evidence for this conclusion was the explosive occurrence of the syndrome among those who have ample and exceptional opportunities for the spread of infection from person to person.

Prof. Waterson[30] in searching for a cause of acquired immunodeficiency syndrome pointed to the fact that the two areas at first affected have very high prevalence of homosexuals. Also a statistic of note was that the average number of male sexual partners for life time reported by patients was 1160. The use of drug such as nitrites was widespread amongst this group. The case for the syndrome being linked with an infective agent of some kind was strong, right from the beginning. Viruses such as Hepatitis B virus take good advantage of the homosexual drug abuser ecosystem to make up for the lack of facilities for transfer between humans with less promiscuous sex life. The most likely picture, and it was at that time no more than a conjecture, was that an unrecognized agent, probably a virus, had been enabled by one or more of several circumstances to spread in a way it had previously found impossible. The principal factor was a locus of greatly increased homsexual activity with a background of specifically and overtly homosexual bars, clubs, and so on, combined, perhaps, with the acquisition of new techniques of homosexual activity. This may have been compounded by drug abuse, with the syringe playing its part in boosting parenteral spread from person to person. Certainly this had been found to be a common factor where the disease had appeared in women. As a virus-perhaps present in the blood, intestinal secretion, or semen of some carriers-may not until recently have had the chance to "take off". The agent could 'jump' from the main cycle if, for example, an infected man had heterosexual intercourse. This model could include also a more or less prolonged symptomless phase after first infection, and there was already evidence that subclinical cellular immunodeficiency was far more common among homosexual men than had been realized until recently.

The absence of any effective treatment of these patients underlined the importance of a preventive approach. Ironically, despite all the uncertainities, this disease was even then essentially preventable. The abandonment of promiscuity, homosexuality, and drug abuse could eventually stop the disease in it's tracks-though that was hardly likely to prove an acceptable solution.

By the middle of 1983, AIDS was slowly but inexorably reaching epidemic forms. Though scientists were still at a loss to understand the cause of this disease, much had already been achieved regarding understanding this syndrome. As of June 1, 1983 a total of 1508 cases, of which 575 had been fatal, had been reported to the CDC from 35 states from the United States, and 109 cases had been reported from 17 other countries.

Thus the new disease entity, AIDS had emerged as an epidemic and was ready to leave it's mark on humanity. The epidemic has by now evolved into a pandemic, with 307, 379 AIDS cases being reported to WHO from 158 countries, till December 1990[31]. Down through the ages epidemics have affected nations, politics and even the course of history. The ancient and often irrational responses of the individual and the society to an epidemic has often been a hinderance to it's control. At this fag end of the twentieth century, in the midst of vast technological advances, it is the evolution in human responses to epidemics which may decide the fate of our efforts to solve this world wide problem.

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Natural Course of Viral B Hepatitis In Bangladeshi Population

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Summary :

Hepatitis B virus (HBV) infection is endemic in Bangladesh. This is a follow-up study on 110 patients with HBV hepatitis. The purpose of the study was to know the outcome of this disease in Bangladeshi population. The mean duration of follow-up was 18±3.2 months from January 1987 to June 1990. There were 80 males and 30 females. Their age ranged from 6 to 70 years. In 77(70%) cases there were clinical jaundice and 33(30%) cases presented as anicteric hepatitis. After the initial diagnosis the patients were followed clinically, biochemically and serologically at three months interval. Imaging of hepatobiliary system and liver biopsy were done as and when indicated.

At the end of 18 months, in the anicteric group 13(39.4%) had HBsAg and 10(30.3%) were HBeAg seropositive. Four (12.1%) developed chronic persistent hepatitis, 3(9%) chronic active hepatitis and 12(36.7%) cirrhosis. In the corresponding icteric group 6(7.8%) had HBsAg and 5 (6.5%) were HBeAg seropositive. Three (3.9%) patients developed chronic persistent hepatitis, 6(7.8%) chronic active hepatitis and 8(10.4%) cirrhosis of the liver. Three anicteric and two icteric cases died from hepatic failure.

In conclusion, this study showed that a great majority of the patients with anicteric hepatitis developed chronic liver disease within a short period after HBV infection. In contrast, only a small fraction of patients with icteric hepatitis developed chronic liver disease. Unlike the reports from developed countries the morbidity of patients with HBV infection in Banigadesh appears to be higher. Therefore, integration of HBV vaccine into the Expanded Programme of Immunization by World Health Organisation for this country will save many lives from HBV infection.

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Introduction :

Hepatitis B Virus (HBV) infection is one of the leading causes of morbidity and mortality throughout the world. There are about 300 million HBV carriers in this globe (Maynard et al, 1988). About 80% of them are chinese subjects (lok et al, 1989). Carrier rate of surface antigen range from 8 to 30% in high endemic southeast Asia and sub saharan Africa (Kiire, 1989). Only approximately 25% of the adults following exposure develop jaundice and symptoms of acute hepatitis (Lambert et al, 1980; Stevens et al, 1981; Schulman et al, 1980; Shah et al, 1985). The course is variable in individual patients. Nearly 90% of the patients with acute hepatitis completely recover (Krugman, 1990). Persistence of HBV infection perhaps invariably follows acute neonatal anicteric hepatitis (Schweitzer et al, 1973; Tong et al, 1981). A study from New York population among Chinese origin and white residents demostrated that Chinese ethnics are more prone to become carriers than the white population (Szmuness et al, 1978). There is high prevalence of HBeAg among Chinese than Caucasian carriers (Derso et al, 1978; Szmuness et al, 1981). The natural course of this virus infection probably depends on several factors. These are age of exposure, sex, race, dose of virus innoculum, host immune response, exposure to aflatoxin B, nutrition and alcohol consumption (Lok et al, 1987; Liaw

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et al, 1984; Lambert et al, 1980; Almeida et al, 1969, Madalinski et al, 1979; Szmuness et al, 1978; Edgington et al, 1975; Dienstag et al, 1982; Barker et al, 1972).

This is a common hepatobiliary disease in Bangladesh. It is prevalent in endemic form. About 60% cases of post transfusion hepatitis, 27.2% adult acute hepatitis and 18.2% commercial blood donors in this country possess hepatitis B surface antigen (Islam et al, 1984).

This study is aimed at to know the serological response and clinical outcome of acute icteric and anicteric hepatitis in Bangladesh.

Materials and methods :

The study comprises a total of 110 cases of acute HBV hepatitis. There were 80 males and 30 females. Their age ranged from 6 to 70 years. The patients were collected from in and out patients department of the Institute of Post Graduate Medicine Research and five private hospitals in Dhaka city. This is a clinical and follow-up study on HBV hepatitis. The patients were selected depending on the following criteria.

i) Symptoms suggestive of hepatitits of 1-2 weeks duration.
ii) Rise of AST/ALT more than four fold in the absence of any history of medication.
iii) No evidence of pre-existing liver disease.
iv) Positive HBsAg/HBeAg/IgM antiHBc.

The patients were divided into two groups namely icteric and anicteric. Anicteric patients never developed jaundice during acute illness. The mean duration of follow-up was 18±3.2 months during the period from January 1987 to June 1990. The patients were followed at weekly interval during acute phase of illness for initial four weeks. There after the patients were followed at three months interval except those who showed the features of progressive deterioration in which case they were monitored like acute phase of illness. Besides clinical assessment and routine biochemical Liver function tests including prothrombin time, serological markers of HBV infection such as HBsAg, HBeAg, antiHBc, and antiHBe were determined in every case during their visit. Needle liver biopsy was performed in those who had persistence of HBsAg /HBeAg or showed progressive clinical deterioration after six months. Ultrasonography of hepatobiliary system was done whenever felt necessary. Those who developed fulminant hepatic failure and subacute hepatic failure were excluded from this study. Hepatitis B serology was done by reverse phase passive haemagglutination method. The markers for hepatitis A, C, D and E were not looked in this study.

Results :

Clinical features

Seventyseven patients had prodromal features of acute hepatitis along with clinical jaundice. Thirty three patients presented with sudden lethargy, listlessness and loss of appetite but had no clinical jaundice. Sixtynine (62.7%) patients were in the age group of 20-40 years. None of them was alcoholic. Fifty nine (53.6%) had the habit of smoking.

Alteration of biochemical liver function tests :

Icteric group : At the end of 18 months, 74 (96.1%) had serum bilirubin level less than two times above normal. Whereas in only one case it was more than two times. In 70(90.3%) patients after 18 months serum ALT raised less than two times. Three cases had its range 2-5 times and another two cases exhibited range over five times above normal. Serum albumin was <2gm/dl in 2, it ranged from 2-3.5 gm/dl in 4,6 had range between 3.5-4 gm/dl and it was found to be over 5 gm/dl in 63(18.8%) cases at the end of 18 months follow up. The prothrombin time ranged from 15-17 seconds

in 48(62.3%) and it was between 18-21 seconds in 27(35%) cases. Three patients had prothrombin time range from 22 to over 30 seconds at the end of one year.

Anicteric group : Serum ALT level was raised less than two times in 25(75.7%), 2-5 times in 4 and over 5 times in one case after 18 months. Five had serum albumin level <2gm/dl and 9 (27.2%) between 2-3.5 gm/dl. In another nine patients the range was 3.6-4 gm/dl. Seven cases exhibited serum albumin over 4 gm/dl. Prothrombin time was between 15-17 seconds. In 3 patients prothrombin time was elevated from 20-30 seconds and another 2 patients had its range over 30 seconds.

Course of HBV Serological Markers :

The serological response in icteric and anicteric group of patients during the course of follow up is shown in table 1. About a half of icteric patients had surface antigen and nearly one quarter was e antigen positive at third month. In contrast approximately two third anicteric cases had surface antigen and over a half of them were e antigen positive. Only a few icteric patients in comparison to over one third of anicteric patients were HBs Ag positive after one and a half year. During the same periode antigen positivity rate was much higher in the anicteric group compared to icteric patients.

Clinical and Histopathological outcome :

Three fourth of the icteric and less than one fourth of the anicteric cases passed through an uneventful recovery. In 45 patients liver specimens were collected by trucut needle. These cases had clinical stigmata of chronic liver disease and persistently abnormal liver function tests after six months from onset of hepatitis. The over all out come is shown in Table—2. It appears that vast majority of the anicteric and comparatively minor fraction of the icteric patients progressed to chronic liver disease. In 18 patients the liver tissue contained virus particles. Figure 1,2 and 3 shows ground glass hepatocytes in patients with CPH, CAH and cirrhosis of the liver (cases 19,52,98) respectively.

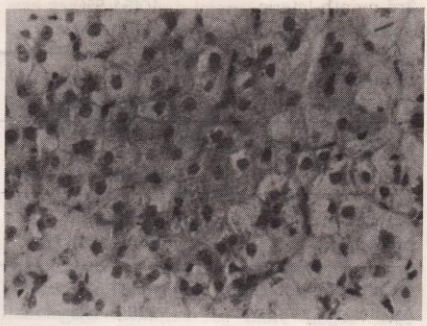

Figure -1: *MS, Age 33 Years, 9 months after acute hepatitis (case 19). Chronic persistent hepatitis. Showing dense mononuclear infiltration with ground glass hepatocytes (440 X).*

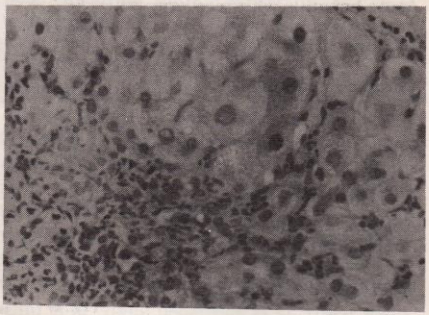

Figure-2 : *ID, Age 20 years, 7 months after acute hepatitis (case 52). Chronic active hepatitis. Showing piece meal necrosis and ground glass cells (440X).*

Table — I

Serological markers in 110 cases of HBV Hepatitis

Follow-up in months	HBsAg	HBeAg	AntiHBc	AntiHBe
3rd month-Icteric	40(51.8%)	20(26%)	54(70.1%)	28(36.3%)
Anicteric	22(66.6%)	19(57.5%)	20(60.6%)	3(9%)
6th month-Icteric	30(38.8%)	18(23.4%)	54(70.1%)	30(39%)
Anicteric	20(60.6%)	19(57.6%)	21(63.6%)	3(9%)
9th month-Icteric	18(23.4%)	10(13%)	55(71.4%)	32(41.55%)
Anicteric	18(54.5%)	16(48.4%)	21(63.6%)	4(12.1%)
12th month-Icteric	9(11.6%)	8(10.3%)	68(88.3%)	45(58.4%)
Anicteric	18(54.5%)	15(45.4%)	28(84.8%)	7(21.2%)
18th month-Icteric	6(7.8%)	5(6.5%)	75(97.4%0	59(76.6%)
Anicteric	13(39.4%)	10(30.3%)	30(90.9%)	11(33.3%)

Table — II

Clinicopathological outcome in 110 cases of HBV Hepatitis

Outcome	No. of Cases (%)
Icteric Group	
Acute hepatitis with recovery	56 (72.7)
Healthy carrier	4 (5.2)
Chronic hepatitis	
CPH	3 (3.9)
CAH	6 (7.8)
Cirrhosis	8 (10.4)
Anicteric Group	
Acute hepatitis with recovery	10 (30.3)
Healthy carrier	4 (12.1)
Chronic hepatitis	
CPH	4 (12.1)
CAH	3 (9)
Cirrhosis	12 (36.4)

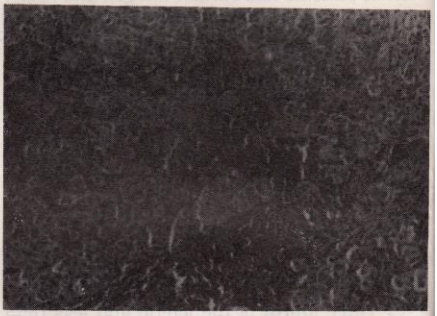

Figure - 3 : *UK, Age years, 10 months after acute hepatitis (case 98), Active cirrhosis. Showing regenerating nodule with ground glass hepatocytes surrounded by dense fibrosis with infiltration of chronic inflammatory cells (110X).*

Discussion :

After the discovery of Australia antigen there have been much advances in our knowledge about the molecular biology, host response in acute and chronic HBV infection

(Tiollais, et al, 1981; Tiollais et al, 1985; Carman et al, 1989; Brunetto et al, 1989). Infection with this virus may lead to a dynamic process. The outcome may be characterized by acute icteric and anicteric hepatitis followed by recovery. Acute icteric and anicteric hepatitis may be followed by chronic hepatitis in the form of chronic persistent hepatitis (CPH), chronic active hepatitis (CAH), cirrhosis and hepatocellular carcinoma (HCC) (AlderShville et al, 1982; Norkrans et al, 1982; Dudley et al, 1972; Smith and Gregory, 1983; Lo et al, 1982; Beasley, 1990).

This is the first systematic follow-up study on HBV hepatitis in Bangladesh. This study reveals the out come of icteric and anicteric acute hepatitis. There was uneventful recovery in 56(72.7%) icteric and 10(30.3%) anicteric hepatitis cases. This is in contrast 90% recovery in developed countries (Krugman et al, 1990).

The range of persistence of HBsAg in self limited infections varies from 1-20 weeks (Lambert et al, 1980). Persistence of surface antigen in serum for a period of six months or longer is defined as carrier state (Seeff and Koff, 1986). Surface antigen was found in 18(23.4%) icteric and18(54.5%)anicteric patients in this study at nine months after acute hepatitis. Nearly half of anicteric and about 13% of icteric patients exhibited antigen at nine months suggesting chronicity. Eighteen (40%) out of 45 liver specimens contained ground glass hepatocytes representing virus particles. Among these cases three had e antigen, four surface antigen and one case was positive for both antigens in the serum. This means that only a minority of the patients had positive serological result while the liver was harbouring the virus particles. This might be due to the use of passive haemagglutination method for detection of viral markers. More number of cases were likely to be detected if sensitive serological tests, immunological staining of liver tissue, recombinant DNA technology and polymerase chain reaction were used (Ray et al, 1976; Omata et al, 1978; Shafritz et al, 1981; saiki et al, 1988). Greater proportion of anicteric patients developed carrier state compared to those whose illness was associated with icterus. This observation confirmed the contention of earlier authors (Shulman, 1971; Ling et al, 1979). Forty out of 110 cases had abnormal liver function tests. No definite correlation has been found between the alteration of biochemical liver function tests, HBV serology, replication of the virus with the degree of liver damage in this study as well as in earlier studies (Periloo et al, 1981; Viola et al, 1981; Tong et al, 1977; Alberti et al, 1983). Some but not all studies found markers of virus replication (HBeAg, HBV DNA, DNA polymerase) predominantly in patients with histological evidence of more active liver disease than those without such markers (Burrell et al, 1984).

The natural course in 100 HBs Ag carriers was studied in Great Britain (Viola et al, 1981). Nineteen of them had the history of acute hepatitis and others were found to be HBsAg positive during routine screening. Histology confirmed 21 CPH, 29 CAH, 8 cirrhosis and 8 HCC. The mean duration of follow-up in their study was 44 months. In about one third patients in this study developed chronic liver disease within 18 months from the onset of hepatitis. This period is undoubtedly very short compared to reports from other countries (Viola et al, 1981). The cause of rapid progression of the disease in our group whether associated with some variants of HBV was not studied. It is possible for some patients with HBV hepatitis to remain viraemic in the presence of anti HBe (Chu et al, 1985; lieberman et al, 1983). They usually develop severe rapidly progressive chronic hepatitis and often go on to develop cirrhosis. None of the patients as yet developed HCC. The estimated relative risk of an HBs Ag

carrier for development of HCC is about 217 fold (Beasley et al, 1981). Therefore, longterm follow-up is necessary.

In conclusion, this study showed that a greater proportion of patients with acute HBV hepatitis in Bangladesh developed chronic liver disease within a short period. HBV infection is preventable by vaccination. Integration of HBV vaccine in the expanded programme of immunization in Bangladesh perhaps will markedly reduce morbidity and mortality caused by this disease.

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Effects of Morphine on the Suppression of Spino-Bulbo-Spinal Reflex Potentials by conditioning Stimuli to the Trigeminal Nucleus in cats

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Summary :

The spino-bulbo-spinal (SBS) reflex potential, following the segmental monosynaptic and polysynaptic reflex potentials was recorded from the lumbar ventral root in cats anesthetized with alpha-chloralose. The intensity of the SBS reflex potential was reduced by conditioning stimuli to the nucleus sensorius superior n. trigemini. Morphine 5 mg/kg intravenously potentiated the SBS reflex itself, and suppressed the influence of conditioning stimuli to the trigeminal nucleus. These effects of morphine were antagonized by naloxone 1 mg/kg intravenously.

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Introduction :

Electrical stimulation of a spinal dorsal root or a peripheral muscle neuron in cats results in the well-known segmental monosynaptic and polysynaptic reflex responses in the spinal ventral root. These responses are followed by an additional delayed reflex response, the spino-bulbo-spinal (SBS) reflex potential[1].

Several investigations have revealed the properties of SBS reflex system, a system which depends upon a relay through the bulbar portion of the brain stem and recurrent projection to spinal motoneurons[2,3,4]. Shimamura et al[4], also reported both inhibitory and facilitative influences associated with the SBS reflex.

Recently, inhibitory effect of electrical stimulation to the trigeminal nucleus on the SBS reflex was revealed by chance[5]. The purpose of the present study was to determine the inhibitory effect of electrical stimulation to trigeminal nucleus on the SBS reflex, and the effect of morphine on it.

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Methods :

Adult cats of either sex weighing 2.5 to 4.0 kg was initially anesthetized with ether, followed by intraperitoneal injection of alpha-chloralose (35mg/kg). The trachea and the femoral vein were cannulated for maintenance of respiration and systemic injection of drugs, respectively. The cats were immobilized by gallamine triethiodide (5mg/kg starting dose) and were on artificial respiration. A dorsal laminectomy was performed over the lower lumbar segments of the spinal cord. The popliteal fossa of left hind limb was dissected to expose peripheral nerves for stimulation. The exposed spinal cord and peripheral nerves were submersed in mineral oil pools and their temperature was maintained at 37±0.2° C by radiant heat.

The left ventral roots of L6, L7, S1 and S2 were severed and cut. Recording from the ventral root was carried out by a pair of Ag-AgCl electrodes placed at the distal cut end of the

ventral root. The tibial nerve of an ipsilateral hindlimb was stimulated by rectangular supramaximal electrical pulse (0.1 msec duration, 0.2Hz). Recordings were taken from the L7 or S1 ventral root and fed through an oscilloscope (VC-11, Nihonkoden, Japan). Four oscillations were averaged by using an averager (QC-110J, Nihon Koden, Japan) and photographs were taken from the oscilloscope with Polaroid camera. Intensity of the SBS reflex was measured by an area under the curve of averaged oscillations. A bipolar coaxial stainless steel electrode was inserted stereotaxically into the nucleus sensorius superior n. trigemini (P4.5, L+5.5, H4.9)[6]. Square wave pulses (250 Hz, 0.1 msec, 4 volleys) were delivered through this electrode prior to the peripheral stimulus which evoked the SBS potentials. The stimulation site in the nucleus was marked by passing a DC current and was confirmed with histological observation after the experiment. All injured or affected parts were treated with 2% procaine. Body temperature was maintained by thermostat equipped heating pad.

Morphine hydrochloride (5mg/kg) and naloxone (1mg/kg) were dissolved in 0.9% sodium chloride solution and administered.

The results were analyzed two-way analysis of variance with subsequent comparisons performed by a paired t-test.

Result :

A representative recording of the segmental and SBS reflex potentials is shown in Fig. 1. The SBS reflex potentials evoked by electrical stimulation of the tibial nerve of the left hindlimb were suppressed following conditioning stimuli of ipsi or contralateral nucleus sensorius superior n. trigemini. Maximum depression was observed at conditioning - testing interval of 40 to 80 msec.

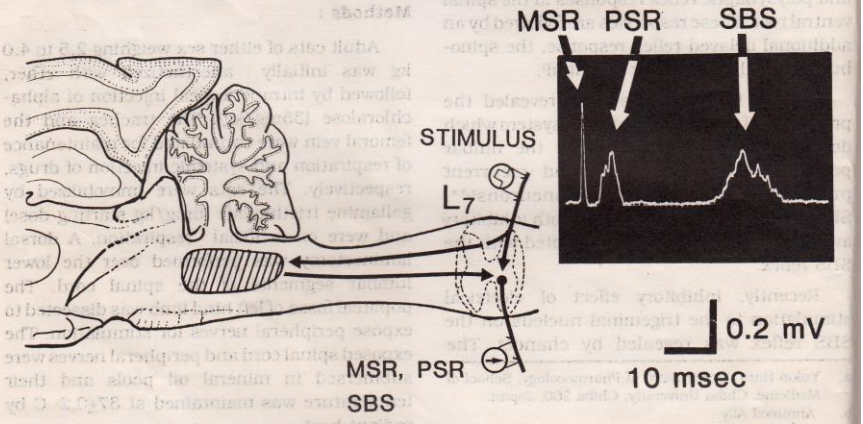

Fig-1 : *Schematic illustration (left) of experimental arrangements and representative Polaroid picture (right) of the reflex potentials. MSR: Monosynaptic reflex potentials, PSR: polysynaptic reflex potentials, SBS: spino-bulbo-spinal reflex potentials. Striped area and arrows in the left illustration were putative center of the SBS reflex and ascending and descending connections mediating segmental and SBS reflexes, respectively.*

The SBS reflex was potentiated by 175% ($p<0.05$) of the preinjection control level, 15 min after morphine 5 mg/kg i.v. (fig. 2-B, upper trace). This increase in intensity of SBS reflex was reversed by naloxone 1mg/kg i.v. by about 97% (Fig. 2-C, upper trace, $p<0.05$, compared with morphine group) of the control level.

Under the conditioning-testing interval of 60 msec, SBS reflex was diminished by

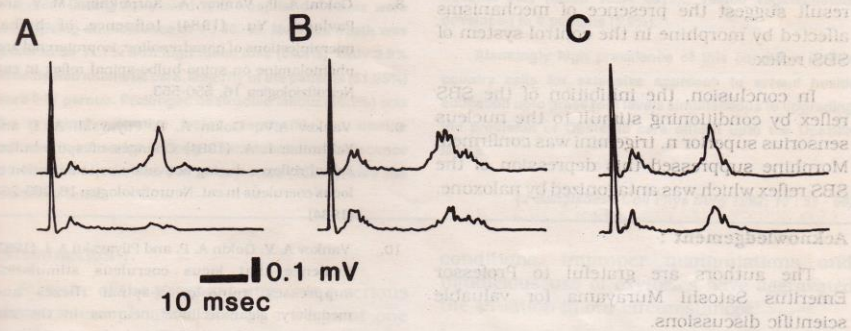

Fig-2 : *Effects of morphine and naloxone on the suppression of the SBS reflex potentials by conditioning stimuli to trigeminal nucleus in alpha-chloralose anesthetized cat. A : Control, B: 15 min after morphine 5 mg/kg i. v., C: 15 min after naloxone 1 mg/Kg i. v. (30 min after morphine) Upper trace : un-conditioned control, lower trace : conditioned. Conditioning stimuli : the nucleus sensorius superior n. trigemini (PA. 5, L5.5, H-4. 9), 250 Hz, 0.1 msec. 4 volleys, Conditioning-Testing interval of 60 msec.*

32.0±4.89% (n=6, Fig. 2-A, lower trace). After administration of morphine 5 mg/kg i.v., the influence of conditioning stimuli was significantly blocked by 62.8+9.16% (Fig. 2-B, lower trace, $p<0.05$) of the unconditioned control. Naloxone 1 mg/kg i. v. antagonized the blocking effect of morphine on conditioning stimuli by 32.0±4.66% (Fig. 2-C, lower trace, $p<0.05$, compared with morphine group).

Discussion :

In the present study the SBS reflex was suppressed by conditioning stimulation of the trigeminal nucleus. This reaction was consistent with previous findings regarding the central influences on SBS reflex[5,7]. Thus, several central influences act on the SBS reflex center and hence control the reflexes. It has been reported that noradrenergic inhibition from the locus coeruleus influenced the SBS reflexes[8,9,10].

The inhibitory effect induced by stimulation of a sensory system, such as the trigeminal nucleus on the SBS reflexes might be regarded as one of the control mechanism at a higher level converging on the SBS reflex center. SBS reflex was more sensitive to the drug than the segmental reflexes.

In the present Study morphine potentiated the SBS reflex and suppressed the inhibitory influence from the trigeminal nucleus to SBS reflex. These effects of morphine were reversed by injection of the morphine antagonist, naloxone. Morphine has a wide range of effect on the bulbo-spinal system. Takagi et al[13]. reported that morphine suppressed the

segmental reflex and the third reflex potentials which correspond to the SBS reflex[13]. They suggested in the same report that morphine activated the ventromedial bulbar inhibitory area to inhibit the spinal segmental reflex in cats. On the other hand, Sinclair[14] reported that morphine antagonized the bulbospinal inhibition of monosynaptic reflex. Morphine acts in the brain to decrease the descending inhibition of spinal nociceptive input[15]. Our result suggest the presence of mechanisms affected by morphine in the control system of SBS reflex.

In conclusion, the inhibition of the SBS reflex by conditioning stimuli to the nucleus sensorius superior n. trigemini was confirmed. Morphine suppressed this depression of the SBS reflex which was antagonized by naloxone.

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The authors are grateful to Professor Emeritus Satoshi Murayama for valuable scientific discussions.

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In conclusion, the inhibition of the SBS reflex by conditioning stimuli to the nucleus sensorius superior n. trigemini was confirmed. Morphine suppressed this depression of the SBS reflex which was antagonized by naloxone.

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Uterine Rupture : An obstetric menace in developing countries

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Summary :

Seventy seven cases of uterine ruptures over one year period in Mymensingh Medical College Hospital were analysed. During the period total hospital deliveries were 3556, giving an incidence of 1 : 46. 18. Maternal death was found to be 11.7% with high foetal loss (84.4%). Only 3.9% patients had antenatal care. Majority of the patients (51.95%) were II-IV parous. Prolonged obstructed labour (56.9%) was the principal causative factor responsible for uterine ruptures in our series. Among other causes scar dehiscence (19.4%) and injudicious use of oxytocics (16.7%) were the important ones.

In majority of patients (56.95%), sub-total hysterectomy was done. Wound infection was the commonest postoperative complication (50%), causing prolongation of hospital stay (mean 16.64 days)., Vesico vaginal-fistula developed in 4 patients (5.9%).

Alarmingly high prevalence of this condition in this country calls for extensive approach to extend health education upto grass root levels simultaneous to upgrading the provision of Obstetric care atleast upto the Upazilla levels.

(J Bangladesh Coll Phys Surg 1992; 10 : 51 - 56)

Introduction :

Rupture of the pregnant uterus is a serious obstetric complication and is a frequent one faced not only by Obstetricians of our country but also by those of other developing countries[1]. Although most cases occur during labour but it can occur during pregnancy also [2]. There has been reports of few cases occurring during the early weeks of pregnancy[3, 4, 5, 6]. UR not only presents as an emergency, but also poses serious threat to the life of both the baby and the mother.

Lack of 'antenatal care' and conduction of labour by unskilled persons are the important factors responsible for such dreadful conditions. Improper manipulations and injudicious use of oxytocics have aggravated the situation in our circumstances.

To ascertain the aetiological factors, clinical condition of the patients and outcome following surgery, this study was undertaken at the Mymensingh Medical College Hospital (MMCH).

Materials and Methods :

From May 1990 to April 1991, any patient presenting to the obstetric unit of MMCH with clinical features conforming to uterine rupture were included in the study. Informations were collected in a data sheet. Clinical findings were correlated with those observed at laparotomy. This was not possible in 8 patients because of their preoperative death. In three of them, clinical diagnosis was in doubt and thus excluded from analysis. Rest 5 were however included although autopsy could not be done for confirmation of clinical diagnosis. Clinical diagnosis were confirmed at laparotomy in 72 cases. This finally made up a total of 77 patients for the present series.

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Details of the clinical settings, presenting clinical features, operative findings and postoperative outcome analysed. Results are expressed as means ± SD wherever applicable.

Results :

Demograpic data and findings related to present labour of all the patients are given in Table-I.

Table- I : *Demographic data & findings related to present labour (n=77).*

1. Age	29.17 ± 5.40 yrs
2. Parity	4.01 ± 2.2 (≤4=52. ≥ 5=25) (Range 2 to 12)
3. Distance of patient's residence from this hospital	33.6 ± 10.1 km.
4. Antenatal care	3 (3.9%)
5. Duration of labour preceding uterine rupture.	50.47 ±28.95 hrs. (Range 10 to 96 hours)
6. Hospital stay (n=68)	16.24 ± 9.43 days.
7. Maternal Mortality	9 (11.7%)
8. Foetal mortality	65 (84.4%)

Majority (51.95%) of our patients belonged to parity between II to IV. Past obstetric history has been tabulated in Table- II

Table-II : *Obstetric history (n= 77)*

Para	Number of patients	(%)
I	12	(15.88%)
II-IV	40	(51.95%)
V-VII	22	(28.57%)
VIII-X or more	03	(3.90%)

On admission 13 patients were in state of shock (mean pressure ≤ 65 mm of Hg) of which cardiovascular variables were not recordable in 11 patients. Result are shown in table III. Even after adequate resuscitative measures to combat shock, nine patients died (5 preoperatively; 1 during operation and 3 in the postoperative period). Four patients survived from their hypovolaemic states.

Table-III.

Cardiovascular variables on admission (n=77)

Pulse	112 ± 21.6 min^{-1}
Systolic BP	108 ± 14.88 mm of Hg.
Diastolic BP	70.23 ± 15.13 mm of Hg.
Hypotensive (mean BP ≤ 65 mm of Hg)	13 (16.88%)

Possible aetiological factors as identified in the 72 operated cases of ruptured uterus are tabulated in Table-IV. Prolonged obstructed labour was the commonest aetiologic factor, occurring in 41 cases (56.9%).

Table-IV : *Aetiological factors.*

Factors	Number of Patients %	Remarks
1. Obstructed labour	41 (56.9%)	
2. Previous caesarean scar	14 (19.4%)	1 in 10 patients & 2 in 4 patients.
3. Use of Oxytocics	12 (16.7%)	im= 10 & iv =2
4. Internal podalic version	4 (5.5%)	
5. Forceps	1 (1.4%)	

On laparotomy, in 46 patients, rupture was found to be of complete variety while it was incomplete in 26 patients. In both the groups

anterior rupture of the lower segment was the commonest ones (of the total 36.11% in complete variety and 33.34% in incomplete variety). Rupture involving both upper and lower segment was found in 15.28% cases. Scar dehiscence was observed at laparotomy in 14 patients (19.4%) with previous Lower Segment Caesarean sections. Details are tabulated in Table-V.

Table-V : *Pattern of uterine rupture (n = 72)*

Site	Complete Number (%)	Incomplete Number (%)
Number	46 (63.88%)	26 (36.12%)
Lower segment		
Anterior	26 (36.11%)	24 (33.34%)
Posterior	4 (5.56%)	2 (2.78%)
Anterior with rupture bladder	2 (2.78%)	——
Upper segment.		
Lateral	3 (4.17%)	——
Combination (upper & lower)	11 (15.28%)	——

Parity and operative findings of the patients at laparotomy dictated the operative procedure. Sub-total hysterectomy was performed in majority (46.95%) of the cases. Uterine repair was performed mainly in patients with incomplete rupture. Surgical procedure adopted in operated patients are tabulated in Table-VI.

Table-VI : *Operative procedure adopted (n= 72)*

Name of operation	Number (%)
1. Subtotal hysterectomy	39 (54.17)
2. Subtotal hysterectomy with bladder repair	2 (2.78)
3. Repair with bilateral tubectomy	24 (33.33)
4. Simple repair	7 (9.72)

Amongst the 68 survivors, wound infection was the commonest postoperative complication (50.00%). In association, other complications were also present, pattern of which is shown in Table-VII.

Table-VII : *Pattern of post operative complications.*

Complications	Number (%)
1. Wound infection	34 (50.00%)
2. Urinary tract infection	17 (25.0%)
3. Vesico vaginal fistula	4 (5.88%)
4. Septicaemia with renal failure	3 (4.41%) (2 died)
5. Paralytic ileus	3 (4.41%)

Discussion :

Uterine rupture is a serious threat both to mother's life as well to that of the foetus. It's overall incidence has been quoted as 1 in 1500 deliveries[7]. The highest incidence so far reported was 1 in 93 deliveries in Uganda[8] while the lowest has been 1 in 11,365 deliveries from Canada[1].

Exact incidence of the condition is difficult to calculate in our country because of lack of birth register and non-attendance of most of the patients at the hospitals. Begum and Tahera[9] reported an incidence of uterine rupture as 1 in 108 deliveries from Dhaka Medical College Hospital. In our hospital, total number of UR was 77 and the number of deliveries during the period reviewed was 3,556, which gives an incidence of 1 in 46.16 deliveries. Our incidence is very high when compared with those of other countries, e. g. 1 in 119 in Conakry / Guniea[10], 1 in 167 deliveries in Ibadan[1]. Our rate is really alarming, pointing to how much we lack in extending antenatal and intranatal care to our mothers in this country. The difference in incidence between Dhaka Medical College

Hospital and that of Mymensingh Medical College Hospital is due probably to the fact that we have a larger catchment area, which covers a population of 123 lacs[11]. Majority of these patients are from remote villages with worst transport system which causes delay in timely transfer of patients from their home to the hospital. Our findings showed the average distance from home to our center was 33.6 km (Table-I).

In our series, only 3.9% patients had antenatal care, i. e. 96.1% were lacking in antenatal care. Rahman in Libya[12] observed that 95% of his cases of ruptured uterus lacked antenatal care.

Begum[13] reported 50 cases of maternal deaths due to uterine ruptures during four year period from 1984-1988 from our hospital, giving an average death rate of 12.6 per year. We lost 9 mothers during one year period of our study. Of them 5 cases died preoperatively, 1 peroperatively and 3 in the postoperative period. Balde et al (10) reported 20.8% maternal mortality in their series from Ibadan.

Foetal mortality was alarmingly high (84.4%) in our series. All the 12 babies those survived in this series were after incomplete ruptures, none survived after complete variety. Our results corresponds very well with those of Balde et al[10]. Foetal mortality in their series was 75.3% and only twenty babies survived after incomplete rupture but none after complete variety.

Mean age of patients in this series was 29.17 ± 5.4 years (range 20-45 years). Majority (53.25%) of uterine ruptures occurred in those over 29 years. Our finding is almost similar to that of Konje et al[1] who reported 57.1% case of UR in the same age group. But this is in contrast to the finding of Balde et al[10] who reported an incidence of 20.85% of UR over the age of 29 years.

Analysis of the data from the present series, reveals the fact that the highest number of UR occurs in II-IV parous women. This is in conformity to other recent reports[9, 10].

As mentioned by Balde et al[10] it is really hard to find out the exact aetiology of uterine rupture. Several factors, like CPD, transverse lie, previous scar, instrumental deliveries and even placenta accreta has been quoted to be responsible for the condition[6,9,14]. With improved obstetric care in developed countries, incidence of UR has become a rarity in western literatures. All the four reports on UR, published in International Journal of Gynecology and Obstetrics in 1990, were from developing countries.

Analysis of our data shows that the preceding labour lasted for prolonged periods (mean 50.47 + 28.95 hours) suggesting obstruction as the cause of uterine rupture. Prolonged obstructed labour (56.9%) was the commonest aetiological factor identified from this study. Transverse lie was responsible for obstructed labour in 16 cases (22.2%), hydrocephalus in one case and impacted breech in one case. Rest of the cases of obstructed labour were most probably due to brow presentation and cephalopelvic disproportion (CPD). The cases of brow presentation and CPD could not certainly be diagnosed as the presenting parts receded after the occurrence of uterine rupture. Bolus doses of oxytocics were given in 12 cases (16.7%) while internal podalic version was responsible for uterine rupture in 4 cases (5.5%).

Dehiscence of scar following previous caesarean section is the common cause of uterine rupture in the developed parts of the world[15]. We observed only 14 cases (19.4%) of scar dehiscence in our series. Begum and Tahera[9] reported uterine rupture after caesarean section in 13.04% cases.

Majority of our patients (83.12%), on admission were stable from cardiovascular point of view. Amongst them rupture was incomplete in 26 cases (36.12%), while in others the uterus found contracted with the extrusion of the foetus into the peritoneal cavity. These factors most probably were responsible for less blood loss in these cases.

The lower segment was the commonest site of rupture in this series (80.56%). This figure was similar to the findings of most other studies[10,12,14]. Rupture of the anterior wall or lower segment occurred in majority of patients (72.22%). This corresponds to the findings presented in other literatures[1,9,10].

We observed complete rupture (63.88%) to be more common than incomplete variety (36.12%) in our series. Konje et al[1] reported a somewhat different incidence (46.8% complete and 53.2% incomplete). The reasons for higher rate of complete variety of UR in our series may be due to delay in diagnosis and / or initiation of treatment, as majority of patients were transferred from remote villages.

Facts and findings that guided us to decide the operative procedure were parity, preoperative clinical condition of the patients and the findings at laparotomy. Majority of our patients had undergone subtotal hysterectomy (56.95%). Begum and Tahera[9] did the same operation in 76.25% cases of their series. Repair of the uterine wound was performed in 43.05% cases where the wound was clean-cut or the rupture was an incomplete one. Bilateral tubectomy was also done in 33.33% of the cases in addition to repair to prevent further pregnancy.

The most common postoperative complication in our cases was wound infection (50%), 25% had urinary tract infection. Septicemia with renal failure occurred in 4.41% cases while vesico-vaginal fistula occurred in 5.88% patients. Many of our patients already had internal examinations without any aseptic precautions before their arrival to hospital and majority of them belonged to poor socio-economic class with poor nutritional status. Possibly all these factors had contributed to high infection rate.

On average the usual stay of patient in our hospital is 7.63 days (Hospital statistics, unpublished). Patients with UR has been found to stay in the hospital for longer periods (mean 16.24 days). which means more cost involvement per patient per day. In a poor country like that of ours with meager and constrained health budget, this finding needs close attention.

In conclusion we would like to say that, uterine rupture is almost a preventable condition. Proper antenatal and intranatal care are the cornerstones to prevent this catastrophe. Ninety six percent of our cases were lacking antenatal care. This emphasises the fact that obstetric care is still inadequate in many parts of rural Bangladesh. Most of our people are still unaware of modern health care facilities and antenatal care. As a result, there is neglect and prolongation of labour, many of which ends with complications such as, uterine rupture, Alarmingly prevalence of the condition in this country calls for extensive approach to extend health education upto grass root levels simultaneous to upgrading the provisions of obstetric care atleast upto the upazilla levels in order to reduce the incidence of such menacing condition.

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Popliteal vascular reconstruction following excision of fibular Osteochondroma in a young patient - A case report.

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Summary :

Multiple injuries were inflicted to the popliteal vessels during excision of huge fibular Osteo-chondroma, in a young patient. Successful reconstruction of damaged popliteal vessels re-established blood circulation and thus saved the limb.

(J. Bangladesh Coll Phys Surg 1992; 10 : 57 - 59)

Introduction :

The osteochondroma is the most common benign neoplasm of bone composed of marked osseous and artilaginous tissue. It is considered by some to be a hamartoma and not a true neoplasm[1]. The excision of such bony tumours may result in damage to the neighbouring vessels and nerves. These injuries are more common in case of huge growths. Following injuries to vessels there may be thrombotic complication, which in turn may pose a serious threat to limb or life. Here we report such a case of multiple injuries to popliteal vessels following excision of fibular osteochondroma. Immediate recognition of those injuries and successful reconstruction saved the limb of a young patient.

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Case Reprot :

A young male of 20 was admitted in the Orthopaedics unit of Rajshahi Medical College Hospital in August, 1989. He complained of constant pain in the right leg, burning sensation in the right sole during walking and a hard mass in the upper 1/3rd of the same limb. The patient gave history of a slow growing nontender mass, of at least 5 years duration. The physical findings consisted of an irregular, hard, fixed palpable mass below the right popliteal fossa. The swelling was not pulsatile. No history of loss of weight or appetite and enlargement of regional lymph nodes was reported. A plain X-Ray of right knee joint revealed an irregular growth of the upper end of right fibula. On the basis of history, physical and radio-logical findings our diagnosis was osteo-chondroma of the upper metaphyseal end of right

fibula (histopathologically it was confirmed later on).

During operation the tumour was found to be huge with irregular bony processes and compressing vessels and nerves in the popliteal fossa. Excision and complete removal of the tumour was done. However in doing so, multiple injuries were inflicted to the popliteal artery and vein. There were multiple lacerations and intimal damage of the popliteal artery over a segment of about 3.5 cm. The popliteal vein was intact through out the length except 2 lateral tears in the neighbourhood of the damaged arterial segment. Those venous tears were repaired using 4/0 atraumatic braided vascular silk (in 8 mm needle) as suture material. The damaged arterial segment was excised out. Simple end to end anastomosis of the artery was tried but not without much narrowing of calibre and tension. Hence grafting with autogenous vein was planned. For this, a 4 cm long segment of autogenous great saphenous vein was dissected out and prepared for grafting. Using reverse-vein-graft technique the venous segment was sutured into the arterial gap in an end to end fashion. Same suture material, as in case of venous repair, was used. Prior completion of the proximal anastomosis, regional heparinization was performed after ensuring distal and proximal clot free vascular tree.

Inspite of postoperative wound infection reconstructed popliteal vessels remained patent, functioned normally and the patient went home on his own feet.

During routine followup at 1, 3, 6 and 12 months interval the condition of operated limb was found to be satisfactory with normal pulsation of the posterior tibial and dorsalis pedis arteries.

Discussion :

Osteochondromas are probably developmental malformations rather than true neoplasms and are thought to originate within the periosteum as small cartilaginous nodules. Occuring with equal frequency in males and females, the osteochondroma is usually discovered during the teenage years as a result of local mechanical symptoms. The metaphysial end of long bones are mostly affected. Any bone may be involved.

In our case upper end of fibula was involved and there were definite features of mechanical compression of nerve and vessels. In such long standing cases malignant transformation usually takes the form of a chondrosarcoma. Histopathologically the excised bony mass was found to be osteochondroma.

There are reports of aneurysm formation of lower extremity vessels (major) as a result of pressure from osteochondroma[2]. The condition of surrounding vessels, in our case, could not be ascertained during enbloc removal of the tumour. On the other hand possibility of popliteal vascular aneurysm cann't be ruled out. Whatever may be the case, the complication in the form of multiple vascular trauma was recognised immediately and appropriate corrective measures were taken. The decision of bridging the popliteal arterial defect with autovenous graft was proper and timely. It, not only helped us to re-establish the continuity of arterial tree but itself remained patent against exogenous infection. Other postoperative complications in the form of thrombo-embolism or graft failure (early and late) were absent.

Such a case, so far known to us, is yet to be reported from Rajshahi Medical College Hospital. The successful out come of this case was mostly due to the team work. In fact, such a complicated major surgery can be carried out successfully if trained personnel and proper instruments are available.

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Carcinoid Tumour of the Larynx - A Case Report

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Summary :

Carcinoid tumour of the larynx was found in a thirty years old farmer. The patient was treated with partial laryngectomy.

(J Bangladesh Coll Phys Surg 1992; 10 : 60 -62)

Introduction :

Carcinoid tumours of the larynx are extremely rare neoplasms characterised by difficulties with tissue diagnosis, a consistently malignant character, and a grave prognosis[1]. Just over twenty cases have been reported in the literature to date. They are unresponsive to radiation and chemotherapy. Ideal treatment is adequate surgical excision. The number of these tumours reported should increase due to the use of electrone microscopy and immunohistologic techniques in making the diagnosis.

Case Report :

A farmer, aged thirty years was admitted into ENT department of Dhaka Medical College Hospital in November 1989 with the chief complaints of dysphagia and hoarseness of voice. Indirect laryngoscopy showed a large, smooth, pink coloured tumour mass arising from the left supraglottic region of the larynx and extending upto the vallecula. Examination of the neck revealed no palpable lymph glands. His liver was not enlarged and ultrasonography of hepatobiliary system did not reveal any evidence of metastasis to the liver. The patient did not complain of flashing of the face, diarrhoea or bronchospasm.

Radiological examination of his chest showed no abnormality but his urinary analysis of 5-Hydroxy indole acetic acid could not be done due to lack of facility. At direct laryngoscopy, a large smooth pink coloured tumour was seen arising from the left ary-epiglottic fold involving pre-epiglottic space. Biopsy of the lesion demonstrated carcinoid tumour which was composed of monotonous cells arranged in small and large alveoli or in sheath with infrequent mitosis. (Fig 1 & 2).

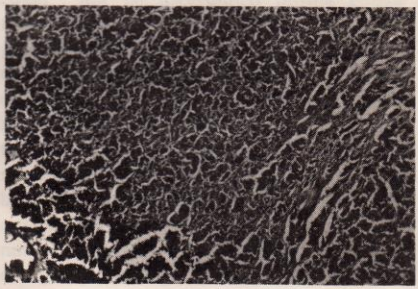

Fig -1 : *Carcinoid tumour of larynx, low power (H & E X 110)*

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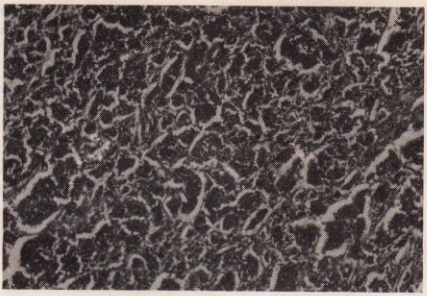

Fig -2 : *Carcinoid tumour of larynx, high power (H & E X 330)*

The tumour was excised by partial laryngectomy. The patient developed wound infection which was controlled by regular dressing and proper antibiotic therapy. He was then discharged and advised to come for follow up every three monthly interval. But after one and a half years, he developed local recurrence. He was then treated with a course of Radiotherapy as he refused further surgery and the tumour was regressed partially. But unfortunately, he was lost from further follow up even after written correspondence.

Discussion :

Carcinoid tumour is a distinctive neoplam arising from a wide variety of locations corresponding to the wide distribution of Kulchitsky (enterochromaffin) cells. The GIT, lung and bronchi are most frequently affected.

By light microscopy, carcinoids are cellular tumours of monotonous blared cells arranged as nests, trabeculae, cords or glands. Newer techniques utilizing immunoperoxidase are useful in the diagnosis. Carcinoid tumours of the larynx are frequently misdiagnosed, most commonly as anaplastic or undifferentiated neoplasms. Unlike undifferentiated carcinoma the carcinoid tumour is universally radioresistant and response to chemotherapy has not been demonstrated. For this reason thorough pathologic evaluation of undifferentiated laryngeal neoplasms is mandatory in order to knowledegeably recommend the proper therapy. Unlike carcinoid tumours in some locations, laryngeal carcinoids do not exhibit secondary systemic symptoms such as the classic carcinoid syndrome secondary to their secretory potential. Therefore, lack of such symptomatology gives no indication of the presence or absence of the laryngeal lesion. The most important point that we wish to emphasize is that although carcinoid tumours of the larynx are universally malignant, aggressive and potentially metastasizing lesions are curable only by adequate surgical extirpation[1,2]. Palliative radiation has been ineffective. New chemotherapeutic drugs or different combinations of existing agents may offer some potential for improvement in both adjuvant and palliative therapy[3].

The absence of clinically recognizable syndromes in patients with laryngeal carcinoid tumours may be related to the production of biologically active products with rapid degradation, intermittent release of biologically active products or the lack of recognition of subtle clinical symptomatology in patients with laryngeal carcinoids[4].

Long term follow up is necessary, since recurrent disease may occur after prolonged disease free interval. Distant metastatic disease occurs frequently and the results of palliative therapy are poor.

The prognosis for patients with laryngeal carcinoid is dismal, with few 5 years survival.

Future therapies must address systemic disease.

Acknowledgement :

We are indebted to prof K. M. Nazrul Islam, Ex-Professor of Pathology, IPGMR, for reviewing the histopathological slide.

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Ectopic Adrenal Tissue within the wall of an Inguinal Hernial SAC : A Case Report and Review of the Literature

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F. TOSHIO ** MD[d] Y. IDEZUKI * MD[e].

Summary :

Ectopic adrenocortical tissue within the wall of a hernial sac was incidentally discovered in a 24 year old man. These are rare and usually symptomless but not without a clinical importance. We present the case here and the literature on ectopic adrenal tissue is reviewed.

(J Bangladesh Coll Phys Surg 1992; 10 : 63 - 65)

Introduction :

Ectopic suprarenal tissue is usually an incidental finding in the human body. Though it rarely gives rise to clinical symptoms, such aberrant adrenal tissue is of considerable interest to surgeons as well as pathologists. In recent years it has become to be considered more common than has previously been reported. The authors present a case of ectopic adrenal cortex within the wall of an inguinal hernial sac and review the literature on the subject.

Case Report :

A 24 years old Indian male presented with a painful irreducible right inguinal hernia. He had a history of reducible swelling at the same place for 5 months. He underwent emergency surgery and a ring like fibrous band was seen in the wall of the hernial sac sorrounding it. Histopathological examination revealed a small nodule of adrenal cortical tissue in the wall of the hernial sac which was not relevant to the fibrous band. The peculiar fibrous band was probably responsible for the irreducibility of the abdominal content from the sac and the presence of the ectopic adrenocortical nodule was incidental. It was a yellowish nodule, oval in shape, hard in consistency and measured 0.2 cm in maximum diameter (Fig. 1).

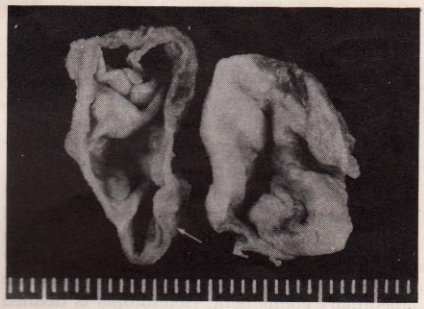

Fig-1 : *The cut section of the hernial sac shows an oval nodule (arrow) in its wall.*

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Histopathology :

Thin sections showed a well-circumscribed oval nodule of adrenocortical tissue embeded in the fibrofatty tissue of the ingunial hernial sac wall. It was composed of fat-laden, compact cells arranged in cords (Fig.2) and encapsulated by delicate connective tissue. No medullary component could be found.

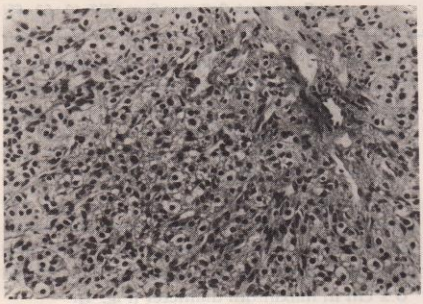

Fig-2 : *Central portion of the nodule shows typical fat-laden and compact cells of adrenal cortex arranged in cords. HE. X450.*

Discussion :

Abnormal location of suprarenal tissue is an interesting anatomical finding within the human body. There have been increasing numbers of reports on aberrant adrenal tissue in diverse locations. These are usually classified into suprarenal heterotopias, accessory suprarenal gland and accessory suprarenal cortical tissue. Accessory adrenal cortical tissue is more common in comparison with suprarenal heterotopia or accessory suprarenal gland, both of which contain the medullary component. Heterotopia is defined as a total displacement of the gland from its normal position.

Accessory suprarenal cortical tissue nodules or adrenal rest may occur almost anywhere in the abdomen, but most commonly are found within the retroperitoneal tissues in proximity to the course of the ovarian and spermatic vessels, alongside and within the spermatic cord[1] and rarely in the wall of the gall bladder[2] and in lungs[3]. These are usually incidental findings and are much more common in infants and neonates than in adults.

In 1740, Morgagni recognized accessory adrenocortical tissue in close proximity to the suprarenals within the peritoneal fat, for the first time in history[1]. These were later described in detail first by Marchand (1883)[1] in children and by Chiari (1884)[2] in adults. McLennan in 1919 first described the presence of adrenal tissue or rests in the wall of hernial sacs[4]. He examined the hernial sacs of 700 children and found nodules of adrenal tissue in 6 of them.

Dhal (1961) examined testes from 100 autopsied male infants and found 15 adrenocortical nodules in 11 cases[5]. In 1980 Abraham reported 12 cases with ectopic adrenal tissue along the spermatic cord among 360 children aged 13 days to 13 years[6].

The less common suprarenal heterotopia can be categorized according to three distinct locations such as adrenal-renal, adrenal - hepatic and cranial.

In contrast with the heterotopic glands, most accessory suprarenal tissue is devoid of a medullary component, however, the structure is complete in a few cases. Medullary tissue could be identified in 50% of the ectopics in Graham's series (1953)[7]. In the Japanese literature, the presence of accessory adrenal tissue was first reported by Kawamura in 1917[8]. Since then more than 80 cases have been reported and among them at least 11 cases contained a medullary component.

Generally, such cases do not produce any symptoms and only a very few can be recognized clinically, when they produce a visible or palpable tumour. For example, Gualtieri and Segal in 1949 removed a 4X3X3 cm accessory adrenal gland from scrotum[9]. Also in 1980 Anderson and McLean reported the removal of a 1 cm in diameter ectopic adrenal gland from the groin[10]. Another important aspect of these ectopic glands is their hyperplastic or neoplastic changes as described by Schechter (1968)[11] and Falls (1955)[12]. In 1978 Gutowski and Gray concluded that an ectopic adrenal cortex is a potential source of neoplasm or of hormone production after adrenalectomy[13]. Steven et al[14] in 1986 reported that a surgically removed myelolipoma was histologically proved to arise from ectopic adrenocortical tissue in the lower abdomen .

In conclusion there is no doubt that accessory suprarenal tissues is of wide occurrence in the human body, despite the paucity of reports. Though it has slight clinical importance, it should be removed whenever diagnosed as it is considered as a source of neoplastic growth.

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Primary Malignant Lymphoma of Bone -A Case Report

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Summary :

This paper reports the case of an adult male who attended the Radiotherapy out patient department, Dhaka Medical College Hospital on 31.10.89 with the history of progressive swelling and pain of the upper part of the left arm for 6 months. He had an operation for biopsy at Rehabilitation Institute & Hospital for the Disabled (RIHD), Dhaka before attending this department. Histopathological examination revealed Non Hodgkin's Lymphoma of left humerus. The patient was treated by Radiotherapy and followed up.

(J Bangladesh Coll Phys Surg 1992; 10 : 66 - 68)

Introduction :

Lymphoma of bone (previously called reticulum cell sarcoma of bone) localized to bone is a rare primary bone tumor (5%) and was first described by Oberling[1] and later by Jackson and Parker[2] in the year 1928 & 1944 respectively. They predominate in males and in patients in the second and third decades of life. It occurs frequently in the long or flat bones but particularly in the femur, clavicle, tibia and humerus. In the long bones, it often starts in the metaphysis and extends to involve the diaphysis[5]. In general, lymphoma presenting in bone is a sign of disseminated (Stage-IV) disease; occasionally it may be true solitary lesion, defined as involvement of single extralymphatic organ or site (Stage IE)[3]. Malignant lymphoma of bone exhibits the same relative radiosensitivity as malignant lymphoma of nodal origin. After irradiation, it regresses rapidly and is controlled with moderate doses. Because of this local radiocurability, radiotherapy can offer the same local control rate as radical surgery; yet the involved extremity or bone segment can be preserved. For this reason radiotherapy is the treatment of choice[4].

A case of primary lymphoma of bone is presented here for rarity, treated only with radiotherapy and the patient is still surviving.

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Case Report :

A 32 year old man had attended the Radiotherapy out patient department of Dhaka Medical College Hospital on 31.10.89 with the complain of progressive swelling & pain in the upper part of the left arm for the last six months.

On local examination the swelling was hard and fixed. Skin over the swelling was mobile but marked with two scars, which was due to surgical interference during biopsy. There was no rise of local temperature and no fever. There was no hepatosplenomegaly and lymphadenopathy. The patient was in good general condition.

X-ray of the left humerus (AP & lateral view) showed osteolytic lesion in the upper part of the left humerus. Whole body bone scan report showed increased concentration of

radionuclide in the upper part of the left humerus. Concentration in other areas of the skeletal system were uniform. Ultrasonogram of the Hepatobiliary system & Brain scan was normal. Liver function tests were also normal. Routine blood examination report was within normal range. No abnormality was found in peripheral blood film.

Histopathological examination of tissue taken from the swollen part of the bone revealed foci of immature lymphoid cells separated by fibro collagenous tissue. Most of the cells were slightly larger than small lymphocytes having a single nucleus and centrally placed single nucleolus. These cells were also seen to have infiltrated into skeletal muscle. The diagnosis was Malignant lymphoma, diffuse, intermediate grade.

The patient was diagnosed clinically as stage I disease and treated with local Radiotherapy (Co60, 4000r in 4 weeks) only. Following Radiotherapy the swelling gradually disappeared. The patient was discharged on 05.12.89 with advice to come for follow-up at 6 weeks interval. The patient came for followup regularly upto 9 months and till last visit in August '90 there was no sign/symptom of local recurrence or metastasis.

Discussion :

Malignant lymphomas primary in bone are rare. They predominate in males and in patients in the second and third decades of life[5], which coincided in this case. This tumor may occur in patients of all ages. but it is rare before the age of 10 years[5]. According to the study of Francis and associates it appears at an average of 34 years of age.[8] This case was presented with the previous history of progressive swelling & pain which are consistent with other findings[6]. A malignant lymphoma is suggested when the tumour is of slow growth and the patient is young and in relatively good condition inspite of advanced local disease[4]. This statement coincided with the present case. Primary malignant lymphoma of bone is most commonly found in the long bones[5], which is also true in this case. Primary malignant lymphoma of bone metastasize to other bones in about one-half of all patients and also to regional lymph modes, liver, and lungs as well as to other sites[5]. Lymph node metastases are not infrequent. McCormack and associates[7] reported regional node metastases in six of their thirty two patients. If untreated, the tumour is moderately aggressive locally and kills with generalized metastases. But in this case, distant and regional metastases were not noted may be due to early presentation of the case. Although, clinical and radiological examinations are suggestive, usually, however the diagnosis is not made until histopathological examination is made[5]. Histopathological examination confirmed the diagnosis in this case. Primary malignant lymphoma of bone is often difficult to differentiate from other lesions like Ewing's sarcoma, Osteosarcoma, metastatic neuroblastoma, multiple myeloma[8], however, diagnosis is not possible until histopathological examination is available[5]. It is important to keep in mind that a primary lymphoma of bone can masquerade as a Ewing's sarcoma[9]. PAS negativity of tumor cells along with other histological parameters are the diagnostic criteria of primary malignant lymphoma of bone[6]. Though clinically and radiologically compatible with other neoplastic lesions, histopathological examination confirmed the diagnosis in this case. Sweet[3] presented a useful algorithm for the evaluation and treatment of bone lymphomas. He emphasized that all patients with a presumed solitary lymphoma of bone should undergo a thorough evaluation for other involvement. Treatment is based on the pathological stage. Stage IE lesions require local radiotherapy and have a reported 90% cure rate[9]. If there is

evidence of more advance disease, chemotherapy is required. The role of surgery is limited to obtaining adequate tissue for diagnosis, treatment of pathologic fracture, and possible resection of an expendable bone[8]. In contrast to the prognosis in patients with generalised malignant lymphoma with secondary bone involvement, patients with primary malignant lymphoma of bone have a relatively good prognosis. Complete cure of more than 50% of patients and local control in a greater number are expected from irradiation alone[4]. In this case, following confirmation of the diagnosis only local radiotherapy was offered as a treatment & gave a very good result.

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COLLEGE NEWS

Continuing Medical Education :

31-10-91 — Dr. Syed Mukarram Ali, Prof. of Pathology BCPS, Dhaka, delivered lecture on Immunoproliferative Small Intestinal Disease.

13-10-92 — Dr. Mr. C. R. Steward, Consultant, Obst. & Gynae. Leicestershire Health Authority Leicester General Hospital, U.K. delivered lecture on Ante-natal Diagnosis of Congenital Malformation.

Workshop on MCQ and Examination System for FCPS-1 Examination :

A workshop on rationalization and Computerization of MCQ and system of examination for FCPS-I Examination was held for 15th days from 2-16th September, 1991 at the College premises. A total of 48 Basic Science and clinical teachers, paper setters and medical experts were present in the workshop as Resource personals, teachers and participants. The workshop was sponsored by the World Health Organization.

In the light of recommendations of the workshop the following decisions have been taken regarding pattern of MCQ and system of examination for FCPS part-I Examination which will be effective from July, 1992 Examination.

1 Group-A (Basic Science) and Group-B (Speciality biased) will not exist in the FCPS Part-I Examination questions which will be combined into one Group only.

2. There shall be separate question paper for each Speciality (i.e. Medicine, Surgery, Obst. & Gynae. and Paediatrics etc. subject-wise).

3. The present system of four format of MCQ will continue. The number of questions for each paper will be- 40 questions from format "A", 40 from format "B", 10 from format "C" and 10 questions from format "D", totaling one hundred (100) questions in each Paper and total marks will be 100.

4. There will be 1.30 hours time for the candidates to answer the questions in the examination.

5. As before, mark will be deducted for wrong answer.

Examination News :

Results of FCPS Part-I, FCPS Part-II and MCPS Examinations held in January, 1992 are given below :

298 candidates appeared in FCPS Part-I Examination held in January, 1992 of which 16 candidates came out successful. Subjectwise results are as follows:

Subject	Number appeared in theory examination.	Number qualified for viva-voce.	Number passed
Medicine	57	19	3
Surgery	70	14	3
Obst. & Gynae.	49	8	2
Paediatrics	35	5	1
Ophthalmology	31	9	2
ENT Diseases	14	0	0
Psychiatry	4	1	1
Anaesthesiology	13	2	2
Radiology	10	1	0
Radiotherapy	1	0	0
Physical Medicine	5	1	1
Haematology	6	1	1
Biochemistry	1	1	0
Histopathology	1	0	0
Microbiology	1	0	0
Total-	298	62	16

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

Roll No.	Name	Graduated from	Speciality
6	Dr. Mansur Habib	Dhaka Medical College	Medicine
13	Dr. Saiyeedur Rahman	Mymensingh Medical College	Medicine
18	Dr. Amar Bahadur Bista	Rajshahi Medical College	Medicine
23	Dr. Fazilatunnesa Malik	Dhaka Medical College	Medicine
24	Dr. Adhikary Kalipada	Sir Salimullah Med. College	Medicine
25	Dr. A. N. M. Farouque-Ur-Rashid	Chittagong Medical College	Medicine
31	Dr. Md. Hayder Rashid	Rajshahi Medical College	Medicine
32	Dr. S. Mohammad. Zahed Chowdhury	Chittagong Medical College	Medicine
34	Dr. Muhammad Rafiqul Alam	Chittagong Medical College	Medicine
41	Dr. Md. Mujibur Rahman	Dhaka Medical College	Medicine
42	Dr. Md. Khalid Hasan	Rajshahi Medical College	Medicine
45	Dr. Khwaja Nazim Uddin	Dhaka Medical College	Medicine

Roll No.	Name	Graduated from	Speciality
48	Dr. A.K.M. Zamanul Islam	Sylhet Medical College	Surgery
52	Dr. Bijoy Kumar Sarker	Rangpur Medical College	Surgery
56	Dr. Syed Mahbubul Alam	Dhaka Medical College	Surgery
59	Dr. Syed Afzal Iqbal	Mymensingh Medical College	Surgery
63	Dr. A. B.M. Golam Robbani	Sir Salimullah Medical College	Surgery
70	Dr. Choudhury Saifuddin Kitchlu	Mymensingh Medical College	Surgery
71	Dr. Md. Abdul Malik	Sylhet Medical College	Paediatrics
72	Dr. Golam Muin Uddin	Chittagong Medical College	Paediatrics
73	Dr. Abu Sayeed Mohammad Iqbal	Dhaka Medical College	Paediatrics
76	Dr. Md. Abid Hossain Mollah	Dhaka Medical College	Paediatrics
77	Dr. Md. Abdul Wahed	Mymensingh Medical College	Paediatrics
79	Dr. Mohammad Nurul Huq	Sher-E-Bangla Barisal.Med.College	Paediatrics
80	Dr. Monnujan Begum	Dhaka Medical College	Obst. & Gynae
82	Dr. Shahanara Chowdhury	Chittagong Medical College	Obst. & Gynae
85	Dr. Ferdousi Begum	Dhaka Medical College	Obst. & Gynae
88	Dr. S. M. K. Alam Mazumder	Chittagong Medical College	ENT Diseases
91	Dr. Md. Monwar Hossain	Mymensingh Medical College	ENT Diseases
92	Dr. Md. Abdul Mannan	Rajshahi Medical College	ENT Diseases
93	Dr. Khabir Uddin Ahmed	Dhaka Medical College	ENT Diseases
96	Dr. Md. Sabbir Quadir	Dhaka Medical College	Ophthalmology
99	Dr. Razia Khanam	Chittagong Medical College	Anaesthesiology
104	Dr. A. K. M. Saiful Islam	Rangpur Medical College	Radiology
105	Dr. Shaikh Golam Mostofa	Sher-E-Bangla Barisal Med. College	Radiotherapy

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

Roll No.	Name	Speciality
2	Dr. Md. Shahidul Islam	Medicine
7	Dr. Md. Shah Ali Miah	Medicine
26	Dr. Md. Shahidul Islam	Surgery
42	Dr. Md. Shahadat Hossain Khan	Paediatrics
44	Dr. Md. Shah Alam	Paediatrics
47	Dr. Syeda Sharifa Merry	Obst. & Gynae.
57	Dr. Hazera Shireen Haque	Obst. & Gynae.

Roll No.	Name	Speciality
58	Dr. Laila Begum	Obst. & Gynae.
61	Dr. Radha Shah	Obst. & Gynae.
63	Dr. Syeda Farida Begum	Obst. & Gynae.
64	Dr. Kohinoor Akhtar	Obst. & Gynae.
67	Dr. Murshed Ahmed Chowdhury	Obst. & Gynae.
71	Dr. S. M. Mahtab-E-Alam	Obst. & Gynae.
72	Dr. Md. Hadi Hossain	Obst. & Gynae.
73	Dr. (Major) Md. Sayedur Rahman	Ophthalmology
89	Dr. Md. Rowshan Isdani	Anaesthesiology
90	Dr. Md. Abdul Bari	Psychiatry
92	Dr. Ashfaque Uzzaman Chowdhury	Psychiatry
93	Dr. Mustafa Najib	Radiology
96	Dr. Shamima Anwar	Radiotherapy
99	Dr. A. B. M. Enamul Huque	Clinical Pathology
103	Dr. Susane Giti	Clinical Pathology
104	Dr. Faruk Ahmed	Clinical Pathology
106	Dr. S. M. Iqbal Shaheed	Dental Surgery
107	Dr. Rafique Ahmed Bhuiyan	Dental Surgery
108	Dr. Anjuman Ara Akhter	Dental Surgery
112	Dr. Zahed Hossain Bhuiyan	Dermatology & Venereology
113	Dr. (Major) Md. Abdul Wahab	Dermatology & Venereology
114	Dr. (Major) Md. Abdul Wahab.	