EDITORIAL

Liver Transplantation—An Update

Since the first orthotopic liver transplantation in 1963 by Thomas E Starzl at Denver, USA[1] now over 4000 liver transplantation are done in a year in USA only[2]. Over 61 centres in USA and many other centres in UK, Australia, Germany, Holland, France, Japan and other countries are performing liver transplantation routinely. In 1983 liver transplantation was accepted as a therapeutic modality for end-stage liver disease[3]. Introduction of Reduced size liver transplantation (RLT) in 1988 and Living related liver transplantation (LRT) in 1989 are milestones for liver transplantation in children[4,5,6]. Thereafter Japan quickly entered into liver transplantation[7]. Orthotopic liver transplantation from brain death donors is not possible in Japan because of local ethical provisions.

Indications of liver transplantation in adults include chronic active hepatitis A, B, C, primary biliary cirrhosis, primary sclerosing cholangitis, Laennec's cirrhosis, acute fulminant hepatitis, neoplasms, metabolic disorders, Budd-Chiari syndrome, irrepairable trauma, and those in children are non correctable or failed congenital biliary atresia, metabolic disorders, fulminant hepatitis, chronic active hepatitis, neoplasms, intrahepatic cholestasis etc.[8].

Until 1980, overall one-year survival was 35% whence introduction of cyclosporine as immunosuppressive agent has tremendously changed the outcome with overall one-year survival of 86%, and three-year survival of about 79% have been achieved[9]. The one-year survival in HBsAg positive recipient was 73% and three-year survival was 51%[10]. Recurrence of HBV infection was found to be about 50%. Use of high doses of HBIg intra - and perioperatively and continuing on long term basis now reduced the incidence of reinfection to 22%[11]. The survival of orthotopic liver transplantation in case of hepatocellular carcinoma is little less. In one report it is stated to be 61% at one-year and 46% at three-year[12]. By RLT an adult liver can be reduced or divided in vitro to use in one or two children[13] and it was possible to gain 94% three-month survival[14].

In LRT where donor is mainly from parents or near relations to donate their left lobe, the acceptibility is more and the one-year survival is 90%[7,15]. Acute fulminant hepatic failure (AFHF) with grade three or four coma with all medical management, the mortality is 80-100%. Now liver transplantation is the single best therapy for such cases with upto 90% survival[16]. The results of liver transplantation in metabolic disorders in children or adult are very good with 90% one-year survival.

The best results of liver transplantation are due to new immunosuppresive drugs, consequent progress in organ preservation, refinement of surgical techniques, adequate medical support and well organisd team spirit. By introduction of University of Wisconsin solution for organ preservation, now hepatic and renal allografts can be safely stored upto 24 and 72 hours respectively[17]. New immunos-ppresive agents are mainstay in improving survival. Initial agents are cyclosporine and methylprednisolone. Rejection episodes are treated with high doses of steroid and OKT3 with or without anti-lymphocyte globulin. A new agent FK506 being used from 1989 is proved more effective in rejection episodes and when primarily used it allows lower doses of steroid and reduces the incidence of rejection[2,8,18].

Wider application of liver transplantation is limited primarily by lack of donors proportional to number of recipients which lead the researchers towards other alternatives. Use of xenografts from baboon is on research and yet to be successful because of specific reactions of naturally occuring antibodies, presence of complement cascade and failure of species specific complement regulatory protein in donor organs to inhibit heterologous complements. These lead to hyperacute rejection in most of the cases. However, some of these grafts survived for a period of months and in future can be used as temporary supports. Temporary hepatic support is a current topic of discussion. Use of isolatd hepatocytes to design a Bioartificial liver (BAL) is now on experiment. Isolated hepatocytes may be less antigenic than endothelial cells, tissue macrophages and other cells present in the liver. In addition, isolated hepatocytes are amenable either to genetic and other manipulations to decrease their antigenicity or to immunoisolation by microencapsulation and other techniques. The design of a BAL is experimentally achieved by cryopreserved isolated matrix anchored normal porcine hepatocytes and plasma perfusion to give extracorporeal liver support[19]. Experimental studies and clinical experience in six patients with BAL suggests that it is safe and possibly beneficial[20]. Various other systems, for extracorporeal liver support to treat both experimental animals and clinical patients with fulminant hepatic failure like cross circulation, whole liver perfusion, plasma exchange, haemadsorption, haemodialysis, total body washouts, use of microsomal enzyme bound carriers etc. are available for short term support[21].

SHAFIQUL HOQUE, FCPS, FACS, FICS.
Associate Professor and Head
Department of Paediatric Surgery
Chittagong Medical College
Chittagong, Bangladesh

(J Bangladesh Coll Phys Surg 1994; 12: 1–3)

References:

1. Starzl TE, Marchiro TL, von Kaulla KN et al. Homotransplantation of the liver in humans. Surg Gynecol Obstet 1963; 117: 656-76.
2. Harland RC. What General surgeons should know about liver and pancreas transplantation. Presented in postgraduate course on diseases of liver, biliary tract and pancreas. 79th Annual Clinical Congress of American College of Surgeons, San Francisco, USA, Oct. 1993.
3. Busuttil RW. Hepatic transplantation 1991. Presented in postgraduate course on diseases of liver, biliary tract and pancreas. 77th Annual Clinical Congress of American College of Surgeons, Chicago, USA, Oct. 1991.
4. Raia S, Nery JR, Mies S. Liver transplantation from live donors. Lancet 1989; ii: 497.
5. Strong RW, Lynch SV, Ong Th et al. Successful liver transplantation from a living donor to her son. N Eng J Med 1990; 322: 1505.
6. Broelsch CE, Whitington PF, Emond JC et al. Liver transplantation in children from living related donors, surgical techniques and results. Ann Surg 1991; 214: 428-439.
7. Tanaka K, Uemoto S, Tokunaga Y et al. Surgical techniques and innovations in living related liver transplantations. Ann Surg 1993; 217:1.
8. National institute of Health Consensus Development Conference Statement. Liver Transplantation. June 20-23, 1983. Hepatology 1984: 107S-110S.
9. Starzl TE, Shapiro R, Simmons RL (eds). Altas of Organ Transplantation 1992. New York. London: Grower Medical Publishing. pp-7.2.
10. Klintmalm GB.Liver transplantation for hepatitis B. Presented in postgraduate course on diseases of liver, biliary tract and pancras. 79th Annual clinical congress of American College of Surgeons, San Francisco, USA, Oct. 1993.
11. Mora NP, Klintmalm GB, Poplawski SS et al. Recurrence of Hepatitis B after liver transplantation. Does Hepatitis B immunoglobulin modify the recurrent disease? Trans Proc 1990; 22: 1549-1550.
12. Chung SW, Toth JL, Rezeig M et al. Liver transplantation for hepatocellular carcinoma. Am J Surg 1994; 167: 317-321.
13. De Hemptinne B, Salizzoni M, Tonk C et al. The technique of liver size reduction in orthotopic liver transplantation. Transplant Proc1988; 20 (Supplement) : 508-511.

14. Frank G M. What's new in paediatric surgery-Transplantation (Liver). Bull Am Coll Surg 1994; 79: 62.

15. Emond JC. Living related liver transplantation (LRT). Presented in postgraduate course on diseases of liver, biliary tract and pancreas. 79th Annual Clinical Congress of American College of Surgeons, San Francisco, USA, Oct. 1993

16. Wood PR. Management of acute fulminant hepatic failure. Presented in postgrauduate course on diseaes of liver, biliary tract and pancreas. 79th Annual Clinical Congress of American College of Surgeons, San Francisco, USA, Oct. 1993.

17. Todo S, Nery J, Yanaga K et al. Extended Preservation of human liver grafts wit UW solution JAMA 1989; 261: 711-4.

18. Bollinger RR. What's new in Transplantation. Bull Am Coll Surg 1994; 79: 75.

19. Rozga J, Williams F, Ro MS et al. Development of a bioartificial liver: properties and function of a hollow fibre module inoculated with liver cells. Hepatology 1993; 17: 258-265.

20. Demetrion AA. Cellular transplantation or support. Presented in postgraduate course on diseases of liver, biliary tract and pancreas. 79th Annual Clinical Congress of American College of Surgeons, San Francisco, USA, Oct. 1993.

21. Brunner G, Mito M (Eds). Artificial liver support; Concepts, methods and results, 1992. Berlin: Springer Verlog.

ORIGINAL ARTICLES

A Study of Testicular Neoplasm at Different Age Groups

MM UDDIN, FCPS[a], PS AKHTAR, FCPS[b]

Summary :

A prospective study of testicular malignant neoplasm covering three and a half years period was done. A total of 168 cases were included in this study. The neoplasm was mostly unilateral (97.62%). Bilateral involvement was found in four cases only.

Histological confirmation was done in 155 cases. Seminoma (65.16%) was the most common variety, followed by teratoma (14.19%), embryonal carcinoma (11.61%) and mixed germ cell tumor (5.16%). Lymphoma, yolk-sac tumor, adenocarcinoma and fibrosarcoma comparised the remaining 3.88%.

Testicular malignant neoplasm has been found in all age groups of patients but was most frequenty at fourth (41.67%) and third decades (25%). Seminoma occured frequently at fourth and fifth decades and teratoma at third and fourth decades. Highest occurence of embroynal carcinoma and mixed-germ cell tumors were observed among patients between 20 and 29 years of age. A case of yolk-sac tumor and another of adenocarcinoma of testis were seen in children below three years of age. Testicular malignancy in subjects above 60 years of age was mostly seminoma.

(J Bangladesh Coll Phys Surg 1994; 12: 39–42)

Introduction :

Testicular malignant neoplasm accounts for 2.85% of all male cancers in Bangladesh[1]. It can occur at any age but most frequently between 20 and 35 years of age[2]. According to Medina et al, age distribution of testicular tumor shows two polar peaks – infancy and 70-80 years old population[3].

Most of the testicular neoplams, aproximately 95%, are of germ cell origin[4]. Among these, seminoma represents the highest fraction followed by embryonal carcinoma, teratoma and choriocarcinoma in that order. Most of these germ cell tumours are highly malignant and capable of rapid and wide dissemination, but optimal combination of surgery, radiotherapy and chemotherapy has improved the treatment outcome of these patients considerably[5]. Tumors, when limited to testis, is curable, even complete remission can be achieved with disseminated disease[5]. Testicular tumors in children are less malignant and require less aggressive therapy[6].

Clinical importance of testicular neoplasm is multiple. First of all, cancer occurs at the age at which a young male adult starts a family and assumes his first work related responsibilities[7]. The most important clinical aspect is the fact that tumors of the testis are curable in high percentage of cases when the three available therapeutic modalities are used appropriately[7].

As there is no dependable statistics regarding testicular malignant neoplasms, the aim of this article is to give an idea about the common histopathology, their probable sites of involvement and age distribution of the patients of testicular tumour.

Materials and method :

This was a prospective study done on all the patients of testicular tumor attending the department of Radiotherapy, Dhaka Medical College Hospital, Dhaka from the beginning

a. Dr. Md. Mokhles Uddin
Radiotherapist, Radiotherapy Department
Dhaka Medical College Hospital
b. Dr. Parveen Shahida Akhtar
Assistant Professor , Radiotherapy,
Dhaka Medical College & Hospital
Correspondence to :
Dr. Md. Mokhles Uddin
Radiotherapist, Radiotherapy Department
Dhaka Medical College Hospital
Received : 16th January, 1994 Accepted: 22nd April, 1994

January, 1990 to the end of June, 1993. One hundred and sixty eight cases of testicular malignant neoplasm who had surgical removal of testis were studied. Short history was taken and thorough physical examination and related investigations were done for each of the subjects.

Results :

In 168 cases, there were patients of all age groups from nine months to 75 years, peak between 30 and 39 years. Mean age was 34.06 years. Tumours were mostly unilateral (97.62%) and seminoma was in the top of the list (65.16%).

Distribution of patients according to age, their sides of involvement and histopathological varieties are shown in the Tables I to III.

Table—I

Agewise distribution of patients of testi cular tumour

Age in years	Numbr of patients	Percentage
Birth - 9	5	2.98
10-19	4	2.38
20-29	42	25.00
30-39	70	41.67
40-49	30	17.86
50-59	10	5.95
60-69	6	3.57
70-79	1	0.59
Total	168	100.00

Table—II

Distribution according to side involved

Sites	Number of patients	Percentage
Unilateral :		
Right testis	90	53.57
Left testis	74	44.05
	164	97.62
Bilateral	4	2.38
Total	168	100.00

Table—III

Histological findings of testicular tumours (N-155)

Histopathology	Total number of patients	Percentage
Seminoma	101	65.16
Teratoma	22	14.19
Embryonal carcinoma	18	11.61
Mixed germ cell tumor	8	5.16
Yolk sac tumor	2	1.29
Malignant lymphoma	2	1.29
Adenocarcinoma	1	0.65
Fibrosarcoma	1	0.65
Total	155	100.00

Table—IV

Agewise distribution histopathological varieties of testicular tumours (N -155)

Age (yrs.)	Seminoma	Teratoma	Embryonal carcinoma	Mixed germ cell tumor	Yolksac tumor	Lym-phoma	Adenoma	Fibros-arcoma
0-9	Nil	Nil	2 (11.11)	2 (25.00%)	1(50%)	Nil	1 (100%)	Nil
10-19	1 (.99%)	Nil	1 (5.56%)	1 (12.50%)	Nil	1 (50%)	Nil	Nil
20-29	15 (14.85%)	9 (40.41%)	8 (44.44%)	4 (50.00)	1 (50%)	Nil	Nil	Nil
30-39	50 (49.50%)	10 (45.45%)	5 (27.78%)	1 (12.50%)	Nil	Nil	Nil	Nil
40-49	24 (23.76%)	2 (9.09%)	2 (11.11%)	Nil	Nil	Nil	Nil	Nil
50-59	7 (6.94%)	1 (4.55%)	Nil	Nil	Nil	1 (50%)	Nil	Nil
60-69	3 (2.97%)	Nil	Nil	Nil	Nil	Nil	Nil	1(100%)
Total :	101	22	18	8	2	2	1	1

Discussion :

Testicular tumours are rare and unique neoplasms[8]. They are the disease of young adult male[9]. SEER (Surveillance, Epidemiology and End Results) programme of the National Cancer Institute[10] shows that malignant tumours of the testis constituted 1% of all cancers in North American men. It accounts for 1% of all male cancers in Western hemisphere too[7]. Sarma et al reported that testicular tumor accounts for 2.85% of all male cancers in Bangladesh[1]. It is more important because it causes loss of significant years of potentially productive life[2].

Testicular tumors are mostly unilateral and in 55-57% cases tumours are present on the right side[7]. They are bilateral in approximately 2-3% cases[7]. In this study, 97.62% of the patients were suffering from unilateral testicular malignant neoplasm. Ninety patients (53.75%) suffered from right sided, 74 (44.05%) from left sided and four (2.38%) from both sided neoplasms (Table - II).

Ninety six percent of all primary testicular tumours are malignant and arise from germinal cells[2]. Seminoma represents the highest fraction (40%)[7]. Embryonal carcinoma represents approximately 20% of testicular tumours and teratoma in adult makes up 10%[5]. Around 40% of testicular tumours are composed of more than one of the pure patterns[5]. Other rare germ cell tumours are yolk sac tumour and choriocarcinoma. In this study, 151 (97.41%) out of 155 histopathologically confirmed cases were germ cell tumours. Seminoma was the most common variety (65.16%), followed by teratoma (14.19%), embroyonal carcinoma (11.61%), mixed germ cell tumours (5.16%) and yolk sac tumour (1.29%). Others were non-germ cell tumours. As the study was done in the department of Radiotherapy, Dhaka Medical College Hospital, the only Radiotherapy centre in Dhaka city and the biggest in the country, there is possibility that more cases of seminoma were concentrated here due to radiotherapy biasness for the treatment of seminoma.

Testicular tumours are the most frequent form of cancer in men between 20 and 34 years of age in United States [11]. We found them at all age groups. They were most frequent between 30 and 39 years (41.67%), followed by in those of age 20 to 29 years (25%). Youngest patient was of age nine months, oldest 75 years and mean age was 34.06 years.

The frequency of seminoma rises to a maximum in men between 35 and 45 years of age[7]. They occur somewhat later than the collective peak[5]. They almost never occur in infants[5]. Embryonal carcinoma occurs mostly in the 20-30 years age group[5]. Teratoma may occur at any age from infancy to adult life[5]. Yolk sac tumour presents more commonly in infants and young children, but also can present in it's histologically distinct form in adults[2]. Malignant lymphoma of testes are seen mostly in elderly men[12]. In this study, seminoma was found at all age groups except in infancy. They were most common in fourth decade (49.50%) and fifth decade of life (23.76%) and none below 18 years of age. Teratoma was present at all ages except infancy. They were found mostly in fourth decade (45.45%) and third decade (40.91%) of life. Embryonal carcinoma was observed among the patients of nine months to 49 years of age. They were most common among 20 to 29 years (44.4%) age group followed by those in 30 to 39 years (27.78%). None was found in patients of age above 50 years. Eight patients were suffering from mixed germ cell tumour and 50% of these were among 20 to 29 years age group . One 2.5 years and another 26 years old patients were suffering from yolk sac tumour. Two patients (one age 55 years and another 19 years) were suffering from primary testicular lymphoma. Adenocarcinoma of testis was found in a two years old boy and fibrosarcoma in a 60 years old man.

There have been some similarities and some dissimilarities between the findings of our study and similar studies in Bangladesh and also in other countries. There are reasons to believe that the findings of this study may not be completely representative but will at least give a general idea about the possible prevalence of different types of testicular neoplasms amongst Bangladesh population.

Acknowledgement :

We are grateful to Dr. Fazle Elahi, Professor of Radiotherapy, Dhaka Medical College, Dhaka for valuable suggestions.

References :

1. Sarma SK, Akhter PS, Alam AMMS, Ansary HR, Uddin MM. Distribution pattern of 3399 New Cancer Patients - A One Year Study. Bangladesh Medical Association Journal (Khulna) 1992; 25 : 15-18.
2. Einhorn LH. Cancer of Testes. In : Devita VT, Hellman S, Rosenberg SF (eds). Cancer-Principle and Practice of Oncology, 2nd Edition 1982. Philadelphia : J.B. Lippincott Company. pp 979-1009.
3. Medina WA, Cadman EA. Testicular Tumors. In : Fischers DS, Marsh JC (eds). Cancer Therapy, 1st edition 1982. Boston: Massachusetts. pp 508-518.
4. Hussey DH . The Testis. In : Text Book of Radiotherapy, 3rd edition 1980, Philadelphia : Lea and Febiger, pp 867-886.
5. Robins SL, Cotran RS, Kumar V. Male Genital System. In : Pathologic Basis of Disease, 4th edition 1989. Philadelphia : WB Saunders Company. pp 1081-1108.
6. Cox JD. Testicle. In: Moss WT, Cox JD (eds). Rdiation Oncology-Rationale, Technique, Results, 6th edition 1989. Baltimore : The C.V. Mosby Company. pp 468-486.
7. Bonadonna G, Cancer of the Male Genital Organs. In: Bonadonna G, Robustelli G (eds). Hand Book of Medical Oncology, 3rd edition 1988. Milano: Masson S.P.A. pp 596-606.
8. Yosida O. Treatment of Testicular Cancer - Current Status and Future Directions. Jpn J Clin Oncol 1990; 20 : 58-66.
9. Frank IN, Keys HM, McCune CS. Urologic and Male Genital Cancer. In : Rubin P, Bakemeier RF, Krackova SK (eds), Clinical Oncology, 6th edition. 1983. American Cancer Society. pp198-21.
10. Young J L, Jr, Percy CL, Asire AJ. Surveillance, Epidemiology and End Results : Incidence and Mortality Data, 1973-77: Natl Cancer Inst Monogr 1981; 57 : 1-1082.
11. Schottenfeld D, Warshauer M E. Testicle. In : Schottenfeld D (editor). Cancer Epidemiology and Prevention 1974. Springfield : Charles C. Thomas Publisher. pp 111.
12. Tepperman BS, Gospodarowicz MK, Bush RS, Brown TC. Non-Hodgkin's Lymphoma of The Tesis. Radiology 1982; 142 : 203-208.

Journal of Bangladesh College of Physicians and Surgeons
Vol. 12. No. 2. May 1994

Comparison of Cardiac Outputs Measured by Transtracheal Doppler and Thermodilution in Anaesthetized Patients

L AZIZ, MBBS[a], S SAEKI MD[b], T NAMBA MD[b], H TOKIOKA MD[c],
K MORITA MD[c], M HIRAKAWA MD[d]

Summary :

Recently, a Doppler ultrasound probe is incorporated into an endotracheal tube (ABCOM°, Applied Biometrics). This allows continuous cardiac output (CO) monitoring in anaesthetized intubated patients. In order to evaluate the accuracy of CO measured by the transtracheal Doppler method (TTD), we compared CO measured by TTD with thermodilution (TD) method. The transtracheal Doppler probe was inserted in eight consenting patients undergoing general abdominal surgery, who required pulmonary artery catheterization. TTD was monitored continuously. TD was measured in triplicate and the average was taken. Comparison of TTD and TD was performed in each patient. Linear regression method was used for statistical analyses. P values less than 0.05 were considered significant. In all cases TTD showed good correlation with TD ($p<0.05$). The transtracheal Doppler was thought to be a useful procedure for monitoring CO under general anaesthesia.

(J Bangladesh Coll Phsy Surg 1994; 12: 43–48)

Introduction :

Noninvasive continuous cardiac output (CO) measurement would be useful for haemodynamic monitoring of patients. Very recently, a technique has been introduced to measure the cardiac output continuously in patients whose tracheas are intubated[1,2,3]. This method uses a 5 mm, 5 MHz pulsed Doppler ultrasound transducer mounted on the tip of a standard endotracheal tube, to obtain the aortic diameter and the Doppler shift of the ultrasound beam reflected by blood in the ascending aorta, proximal to the major aortic arch vessels. From these data, cardiac output is calculated every 12s and displayed. Linear regression statistical analysis has been used in previous studies in dogs[2] and humans [1,3], and a good correlation has been shown between measurements of CO with transtracheal Doppler (TTD) and thermodilution (TD) for a small number of measurements. There have been no studies comparing a relatively large number of simultaneous measurements of CO by TTD and TD. Siegel et al, in heart surgeries, showed that comparison between TTD and TD is not very reliable[4]. Whereas Wong et al showed a reliable correlation in abdominal type of surgeries[5]. This study compares the measurement of CO by TTD with that by TD in anaesthetized patients during major abdominal operations.

a. Lutful Aziz, Research Student
b. Shinsei Saeki,
Taketoshi Namba,
Assistant Professor of Anaesthesiology
c. Hiroaki Tokioka,
Kiyoshi Morita
Associate Professor of Anaesthesiology
d. Masahisa Hirakawa, Professor and Chairman, Dept. of Anaesthesiology
Okayama University Medical School
2-5-1 Shikata Cho, Okayama 700, Japan

Correspondence to :
Lutful Aziz
Dept. of Anaesthesiology & Resuscitology
Okayama University Medical School
2-5-1 Shikata -cho, Okayama 700, Japan
Recieved 21st March, 1994, Accepted : 5th May, 1994

Materials and method :

The transtracheal Doppler probe is described previously in detail[1]. It embodies two important features. The first is the inclusion of a 5 mm diameter ultrasonic transducer at the distal end of the endotracheal tube. The transducer is a lead zirconate titanate piezolectric crystal designed to operate at 5 mHz. The transducer is mounted in a molded, polyvinyl chloride holder to maintain a fixed angle with respect to the longitudinal axis of the endotracheal tube. The

second feature, since ultrasound is highly attenuated in air, is a balloon cuff to assure acoustic contact of the transducer with the anterolateral wall of the trachea. The cuff has been fabricated to be a prolate ellipsoid with its major axis displaced with respect to the longitudinal axis of the endotracheal tube. When inflated, the cuff forces the transducer into contact with the tracheal wall. The electric leads from the transducer are contained within the wall of the endotracheal tube.

The electronics unit used to drive the transducer and process the Doppler ultrasound information was an ABCOM° Applied Biometrics CO computer. The unit operates in a pulsed Doppler mode with a carrier frequency of 5 mHz. When driven by the Applied Biometrics electronics unit, the ultrasound power output was below Food and Drug Administration (FDA) guidelines for cardiac ultrasound use. The continuous CO data were digitized and accumulated for 12s time intervals to average over the variation due to the ventilator cycle.

Doppler determined CO was calculated according to the following equations :

$CO = v \times A$

where;

CO = cardiac output; v = average blood velocity; A = cross–sectional area of the aorta.

The average blood velocity (v) was calculated by :

$$v = \frac{Cxf}{2xf_o x\cos\phi}$$

where :

f = Doppler shift; C = velocity of ultrasound in tissue; f_o = carrier velocity; ϕ = angle of ultrasound relative to flow.

The Doppler shift (f) was determined at the range of peak velocity by time averaging over 12s. The angle of the ultrasound beam relative to the blood flow (ϕ) was determined previously and set at 52.4 degrees. The carrier frequency(f_o) was 5 MHz.

After getting the approval of the Okayama University Ethics Committee on the use of humans in research and also individual informed consents, eight patients older than 20 years of age undergoing elective general abdominal surgery were studied. All the patients required pulmonary arterial catheterization as part of their routine intra-operative monitoring. They did not have any aortic valve diseases or chronic rhythm disturbances. Patients were also excluded if there were any abnormalities or previous surgery involving the aortic arch.

Before the induction of anaesthesia, the pulmonary artery catheter was inserted. Then the patient was anaesthetized and intubated by TTD endotracheal tube. A headset, which transmitted the audio representation of Doppler flow signals, was worn to monitor the Doppler flow signals continuously during intubation. A visual meter was also employed to identify the forward and reverse flow. The TTD probe was positioned by translating the probe along its longitudinal axis and rotating the patients head to maximize both the audio and visual representation of forward flow as determined by Doppler shift. An endotracheal tube holder was used and the cuff was inflated to secure the probe in correct place. The cuff was deflated and the endotracheal tube and patient's head were manipulated as necessary, whenever repositioning of the tube was needed. The balloon cuff was then inflated again. Breath sounds were obtained bilaterally initially and following repositioning.

Measurements of cardiac output were obtained in triplicate with the use of a TD pulmonary artery catheter (93A-131H-7F; American Edwards Laboratory, Santa Ana, CA) and a TD cardiac output monitor computer (series 7010RA; Marquette Electronics, Milwaukee, WI) with a 10 ml saline injectate at 0°C temperature at end-expiration[6]. An average of three measurements was calculated. Any cardiac output significantly aberrant (greater

than±10%) from the other two was excluded, and a repeat measurement was obtained. Concurrent measurements from the transtracheal Doppler and ABCOM° (Applied Biometrics) cardiac output monitor were recorded. Data were not collected during electrocautery. Data were also excluded if the amplitude of the forward flow on the Doppler visual display was inadequate or if the audio representation of the Doppler signal was suboptimal. The adequacy of the Doppler signal was determined without any information about TD measurements. The display of the Doppler cardiac output measurement was updated every 12s. All of the displayed values were recorded during the time interval in which the TD measurements were obtained. The Doppler cardiac output measurements were averaged to obtain the average value for the time interval. Cardiac output was measured at specific moments when possible; it was measured after intubation, after incision, and any time there was a 25% change in pulse or blood pressure or any other clinical indication.

In each patient, correlation of TTD and TD values were assessed by least squares linear regression method. P values less than 0.05 were considered significant.

Results :

Because air does not satisfactorily transmit ultrasound, the ability to obtain velocity profiles from the trachea using Doppler ultrasound verifies acoustic contact with the anterolateral wall of the trachea. Results of fibreoptic bronchoscopy revealed no evidence of tracheal hyperaemia, ulceration, or haemorrhage. Subsequent patients, in whom tracheal suction was performed through the endotracheal tube, again revealed no evidence of bleeding. Four patients complained of a minor sore throat, of whom three had concomitant insertion of a nasogastric tube. Measurement of cardiac output did not interfere with ventilation of the lungs. No airway complications involving positioning of the Transtracheal Doppler probe or measurement of cardiac output were noted. Table-I represents the patients type included in the study and also the type of operation they have undergone. Positioning of the tube and the angle of incidence (ø) are shown in Figure-1. The calculation of the angle of incidence (ø), which was the best least squares fit for the eight patients was $52.4^0 \pm 3.8°$ (±SD).Figure-2 explains the calculation. Figures 3a and 3b shows the scatter plot of Transtracheal Doppler cardiac output (y-axis) versus the TD cardiac output (x-axis) of the eight individual cases. Cases one to

Table—I

Type of surgery done on the subjects

Case Number	Age	Sex	Performed Operation
1	76	M	skin graft for burn
2	82	F	total cystectomy
3	60	M	abdominal aneurysmectomy
4	73	F	adrenal tumor extirpation
5	25	F	aorto-renal bypass
6	70	M	adrenal tumor extirpation
7	77	M	hepatoma enucleation
8	57	F	Miles' operation

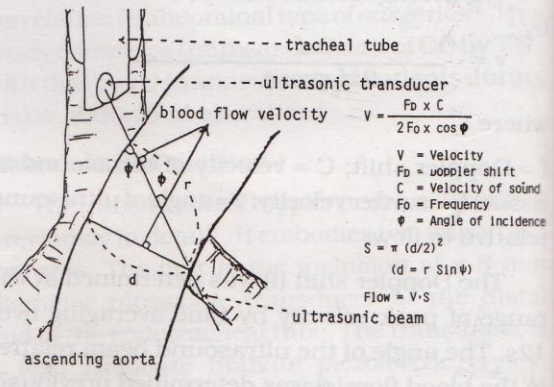

Fig-1: *Positioning of tube and angle of incidence*

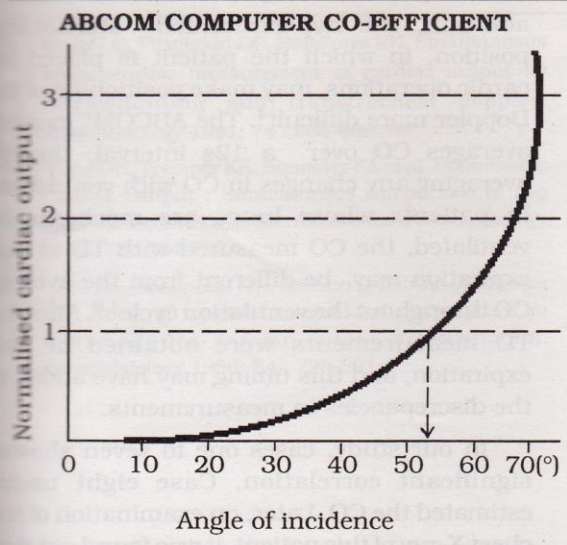

Fig-2: *Explanation of the calculation of angle of incidence*

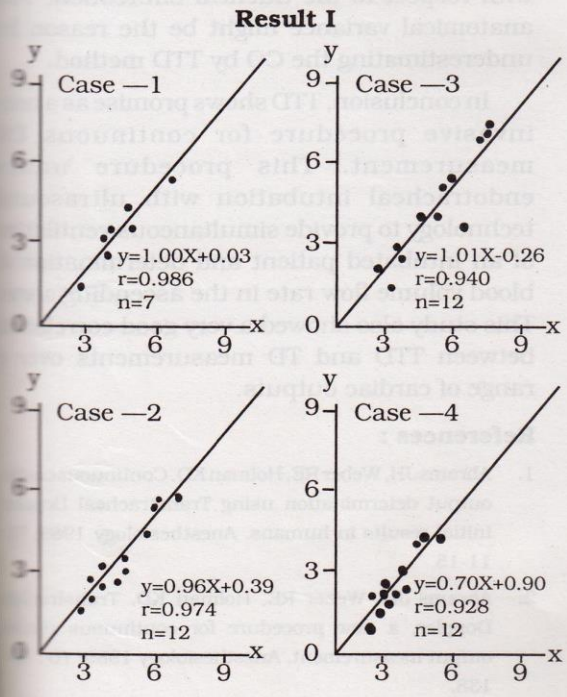

Fig-3a: *Scatter plot of Transtracheal Doppler cardiac output (y-axis) versus Thermodilution cardiac output (x-axis)*

seven shows a significant correlation of the cardiac outputs between the TTD and the TD methods. In case eight, the cardiac output measured by the TTD method underestimated the CO measured by the TD method.

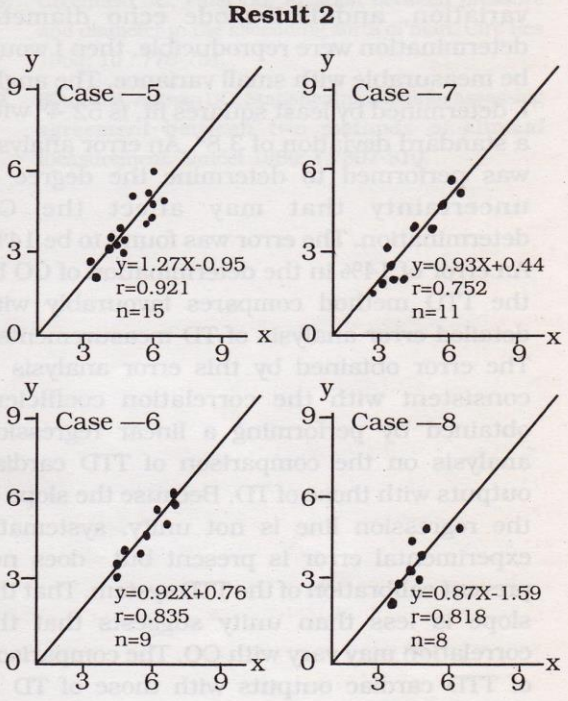

Fig-3b: *Scatter plot of Transtracheal Doppler cardiac output (y-axis) versus Thermodilution cardiac output (x-axis)*

Discussion :

The present study demonstrate that the transtracheal Doppler procedure provides an estimate of cardiac output in humans that compares satisfactorily with thermodilution measurements. Human mediastinal anatomy allows the continuous cardiac output measurements to be performed, using a transtracheal ultrasound window, to measure aortic dimensions and blood velocity. Sources of error in calculating COs include human mediastinal variation, unceratainty in

transducer positioning, uncertainty in B-mode echo ascending aortic diameter determination, and uncertainty in thermodilution CO determination. We reasoned that if transtracheal Doppler probe positioning were reproducible, human mediastinal anatomy had small enough variation, and if B-mode echo diameter determination were reproducible, then f would be measurable with small variance. The angle, f, determined by least squares fit, is 52.4^0 with a standard deviation of 3.8°. An error analysis was performed to determine the degree of uncertainty that may affect the CO determination. The error was found to be 14%. An error of 14% in the determination of CO by the TTD method compares favourably with detailed error analysis of TD measurements[7]. The error obtained by this error analysis is consistent with the correlation coefficient obtained by performing a linear regression analysis on the comparison of TTD cardiac outputs with those of TD. Because the slope of the regression line is not unity, systematic experimental error is present but does not prevent calibration of the TTD system. That the slope is less than unity suggests that the correlation may vary with CO. The comparison of TTD cardiac outputs with those of TD is within the experimental error for both methods. TTD is a relatively new method for monitoring the CO. In a recent publication, it is showed that the comparison between TTD and TD is not very reliable[4]. Most of the operations of that experiment were heart surgeries. But Wong et al showed that the comparison between the TD and TTD method is very reliable if the operations are of abdominal type, and in case of heart surgeries, the results are little different[5]. Many of the patients in Siegel's study had aortic disease that may have affected the Doppler measurement[8]. Surgery requiring manipulation of the aorta might disturb the blood velocity profile in the aorta. Such changes would be expected to have a greater impact on TTD measurements than on TD measurements. In addition, the hyperextended sternotomy position, in which the patient is placed for cardic operations, may make positioning of the Doppler more difficult[4]. The ABCOM° monitor averages CO over a 12s interval, thereby averaging any changes in CO with ventilation. In patients whose lungs are mechanically ventilated, the CO measured with TD at end expiration may be different from the average CO throughout the ventilation cycle[1,9]. All of the TD measurements were obtained at end expiration, and this timing may have added to the discrepancies in measurements.

In our study, cases one to seven showed significant correlation. Case eight underestimated the CO. Later, on examination of the chest X-ray of this patient, it was found out that the aortic arch was lower than the usual position with respect to the tracheal bifurcation. This anatomical variance might be the reason for underestimating the CO by TTD method.

In conclusion, TTD shows promise as a non-invasive procedure for continuous CO measurement. This procedure unites endotracheal intubation with ultrasound technology to provide simultaneous ventilation of an intubated patient and determination of blood volume flow rate in the ascending aorta. This study also showed a very good correlation between TTD and TD measurements over a range of cardiac outputs.

References :

1. Abrams JH, Weber RE, Holman KD. Continuous cardiac output determination using Transtracheal Doppler: Initial results in humans. Anesthesiology 1989; 71 : 11-15.
2. Abrams JH, Weber RE, Holman KD. Transtracheal Doppler, a new procedure for continuous cardiac output measurement. Anesthesiology 1989; 70 : 134-138.
3. Pierpont GL, Weber RE, Kan FK, Ram SK, Abrams JH. Continuous cardiac output monitoring in patients using Transtracheal Doppler ultrasound. Journal of Cardiovascular Technology 1990; 9 : 31-34.

4. Siegel LC, Fitzgerald DC, Engstorm RH. Simultaneous Intraoperative measurement of cardiac output by thermodilution and transtracheal doppler. Anesthesiology 1991; 74 : 664-669.

5. Wong DH, Tremper KK, Stemmer EA et al. Noninvasive Cardiac Output : Simultaneous comparison of two different methods with thermodilution. Anesthesiology 1990; 72 : 784-792.

6. Pearl RG, Rosenthal MH, Nielson L, Ashton JP, Brown BW. Effect of injectate volume and temperature on thermodilution cardiac output determination. Anesthesiology 1986; 64 : 798-801.

7. Fischer AP, Benis AM, Jurado RA, Seely E, Teirstein P, Litwak RS. Analysis of errors in measurements of cardiac outputs by simultaneous dye and thermal dilution in cardiothoracic surgical patients. Cardiovasc Res 1978; 12 : 190.

8. Greenfield JC, Patel DJ. Relation between pressure and diameter in the ascending aorta of man. Circ Res 1962; 10 : 778-781.

9. Bland JM, Altman DG. Statistical methods for assessing agreement between two methods of clinical measurement. Lancet 1986; 1 : 307-310.

Journal of Bangladesh College of Physicians and Surgeons
Vol. 12. No. 2. May 1994

Experience with Intracranial Tumours in Dhaka Medical College Hospital—Report of 72 Cases

M WAHEEDUZZAMAN, FCPS[a], M ZIAUDDIN, MBBS[b], O FARUQUE, MBBS[c]
N M MURSHIDUZZAMAN, DMRD[d], A A KHAN, FRCS[e]

Summary :

Seventy two cases of intracranial tumours were analysed. Mean age of the subjects was 33.84 years. Male to female ratio was about 2 : 1. Twenty five percent cases were blind during initial presentation and 16.66% admitted in unconscious state. In 50 (69.4%) cases diagnoses were confirmed by histopathology. Among those, majority (40%) had glioma. Post-operative mortality was 15.8%. This high mortality was due to late presentation of cases. Early referral and diagnosis of brain tumour will improve results of treatment[1].

(J Bangladesh Coll Phys Surg 1994; 12: 49–53)

Introduction :

Among the intracranial tumours, gliomas are the most common. But the treatment of these tumours is still far from satisfactory. Meningioma constitute about 15 percent of primary brain tumours. These tumours are quite benign and completely resectable. Pituitary tumour represents 10 percent of intracranial tumours. But due to nonavailbility of microsurgical instruments in Bangladesh, treatment of these lesions lead to high mortality and morbidity.

Purpose of this study was to analyse 1) clinical presentation, 2) problems of diagnosis and treatment, 3) location and 4) histopathological variety of intracranial tumours treated over five years period.

Materials and method :

Seventy two consecutive cases of intracranial tumours were analysed. The cases were among those who sought treatment in neurosurgical unit of Dhaka Medical College Hospital from July 1985 to December 1990. All these cases were admitted either from the out-patient department or referred from different hospitals and various units of the same hospital. After admission, the cases were evaluated clinically and available relevent investigations were done. Most (63) of the cases were managed surgically.

Results :

Figure-1 shows the bar chart of incidence of intracranial tumours in different age groups. 65.27% cases were between 20 and 50 years of age. Mean age was 33.84 years.

In the present series the male and female ratio is about 2 : 1. Table-I shows a comparative study of sex incidence of intracranial tumours.

Table—I

Comparative study of sex incidence of intracranial tumours

	Dastur and Lalitha 1968		Present series	
	Male	Female	Male	Female
Total neoplasm	836 (66.35%)	424 (33.65%)	50 (69.40%)	22 (30.60%)
Glioma	448 (70.55%)	187 (29.45%)	14 (70%)	6 (30%)
Meningioma	82 (49.11%)	85 (50.89%)	9 (52.90%)	8 (47.10%)

a. Md. Waheeduzzaman FCPS, Assistant Professor (c.c.)
b. Md. Ziauddin MBBS, Lecturer of Anatomy
c. Omar Faruque MBBS,Assistant Registrar of neurosurgery
d. N.M. Murshiduzzaman DMRD, Professor of Radiology
e. Ata Alahi Khan FRCS, Professor of Neurosurgery
Department of Neurosurgery, Dhaka Medical College, Dhaka.
Correspondence to:
Md. Waheeduzzaman, Assistant Professor (c.c.)
Department of Neurosurgery, Dhaka Medical College, Dhaka.
Received : 15th July, 1992 Accepted : 16th April, 1994

Figure-2 shows duration of symptoms before admission to the hospital.

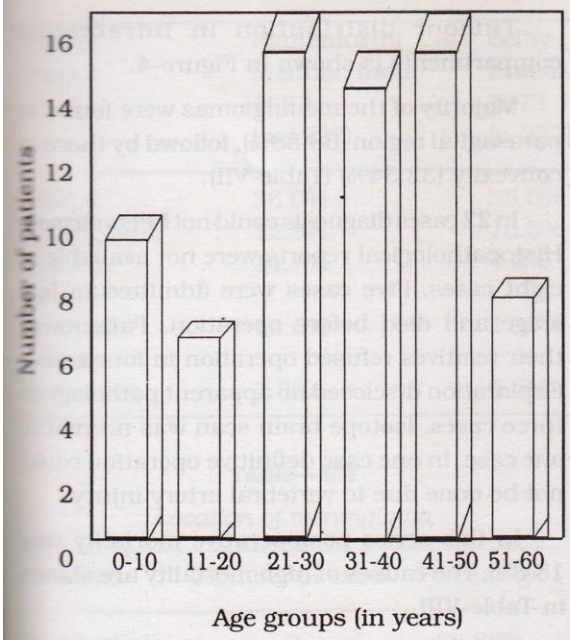

Fig-1: *Age distribution of intracranial tumours*

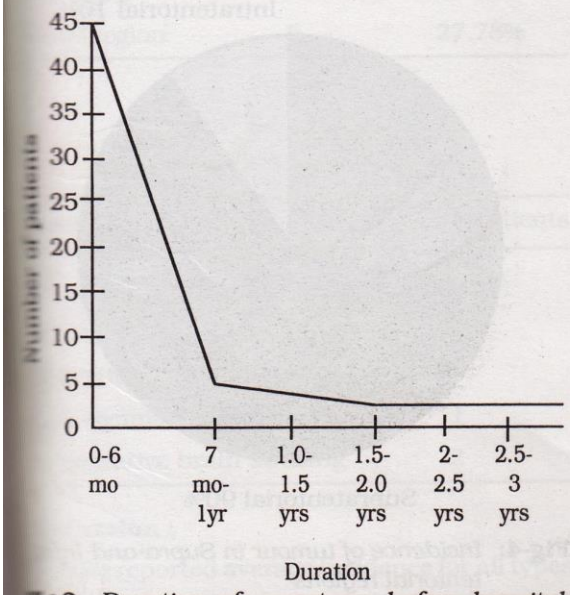

Fig-2: *Duration of symptoms before hospital admission*

Clinical features of intracranial tumours is shown in Table-II. Twenty five percent cases were blind prior to admission in hospital.

Isotope brain scan was done in all cases before the availability of CT scan in Bangladesh. After introduction of CT scan in late 1989, isotope brain scan was done very rarely and only on those who could not afford to do CT scan. The findings are shown in Table-III.

Table- IV shows the percentage of abnormal findings in plain skull X-ray.

Table–II

Clinical features of intracranial tumours in present series

Symptoms	Number of patients	percentage
Headache	52	72.22%
Vomiting	37	51.38%
Blindness	18	25.00%
Seizures	16	22.22%
Unconscious state	12	16.66%
Signs		
Hemiparesis	28	38.88%
Papilloedema	25	34.72%
Cranial nerve palsy	15	20.83%

Table—III

Investigations done for diagnosis and localisation of tumours

Investigation	Number of Patients	Percentage
Plain X-ray of skull	60	83.33%
Isotope brain scan	43	59.72%
CT scan	21	29.16%

Table—IV

Findings of X-ray skull

Number of cases	Normal finding	Abnormal finding	Percentage of abnormal findings
60	40	20	33.33%

Out of 63 cases who underwent surgery, 73.10% had craniotomy. Other neurosurgical procedures were done on the rest (Table-V).

On histopathological examination, 40% lesions were found to be astrocytoma, 36% meningioma and rest other types of tumours (Fig. 3)

Table—V

Type of surgical procedures done in present series

	Name of operation	Number of patients	percentage
1.	Craniotomy	46	73.10%
2.	Suboccipital craniectomy with VP shunt	7	11.10%
3.	Burr hole biopsy	9	14.20%
4.	Transphenoidal hypophysectomy	1	1.50%

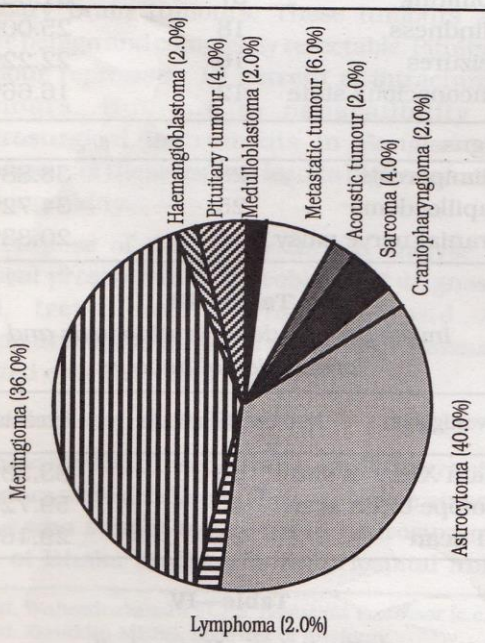

Fig-3: *Types of intracranial tumours (inoldence types)*

Table-VI shows the comparative study of incidence of intracranial tumours[2,3], which indicate a higher incidence of meningioma in present series.

Tumour distribution in intracranial compartments is shown in Figure-4.

Majority of the meningiomas were found in parasagital region (38.88%), followd by those in convexity (32.34%) (Table-VII).

In 22 cases diagnosis could not be confirmed. Histopathological reports were not available in eight cases. Five cases were admitted in late stage and died before operation. Patients or their relatives refused operation in four cases. Exploration disclosed no apparent pathology in three cases. Isotope brain scan was normal in one case. In one case definitive operation could not be done due to vertebral artery injury.

In this series postoperative mortality was 15.8%. The causes of high mortality are shown in Table-VIII.

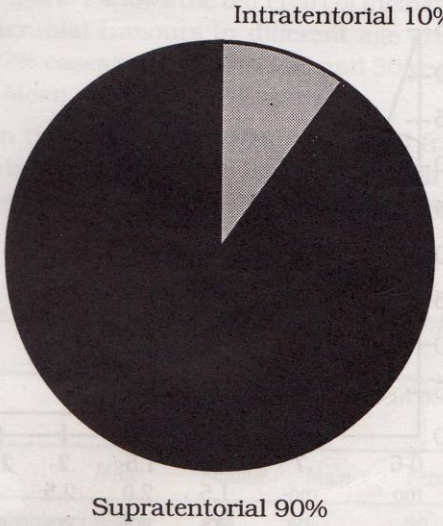

Fig-4: *Incidence of tumour in Supra-and Infra-tentorial regions*

Table—VI

Comparative study of incidence of intracranial tumours.

Type of tumour	Ramamurthi Madras, India 1973 (n-1227)	Selby Kualalumpur 1973 (n-357)	Weinman Colombo, Srilanka 1973 (n-619)	Present series (n-50)
Glioma	36.0%	35.0%	45.7%	40%
Meningioma	12.9%	16.5%	17.9%	36%
Pituitary tumour	14.7%	4.2%	6.8%	4%
Acoustic tumour	12.5%	11.7%	11.4%	2%
Metastatic	7.8%	5.3%	-	6%

Table—VII

Location of meningioma

Location	Number of cases	Percentage
Parasagital	7	38.88%
Convexity	6	33.34%
Basal region	5	27.78%

Table—VIII

Causes of postoperative death

Causes	Number of patients
Late presentation	5
Ventriculitis	1
Aspiration	1
Septicaemia	1
Peroperative brain swelling	2

Discussion :

The reported average incidence for all types of brain tumours in western countries are approximately 10 per 100,000[4]. There is no epidemiological information of such kind available in Bangladesh. Population of Bangladesh is about 110 million and incidence of intracranial tumours would presumably be as high as in other countries.

Age distribution in our cases was the same as other series. Sex distribution of intracranial tumours in the present study is same as that of Dastu's series[5] except for meningiomas. Number of meningiomas in our study was higher in male patients. This is due probably to sociocultural attitude towards females who are usually neglected and priority and necessity of their treatment is not easily perceived.

In this series approximately 50% of cases were presented after six months of onset of symptoms. Evidge et al reported an average duration of symptom for about three years [6] before the patients submit for treatment in cases of astrocytoma. In our series, astrocytomas presented with symptom duration of around two years. On careful analysis we found that, though apparently our cases appear to have come early, their symptoms were quite severe and were in very advanced stage. Further, they had milder symptoms at onset and continued for several years before attending hospital or visiting physicians.

Intracranial tumours are suspected when there is headache, vomiting and papilloedema. This is the classical triad of Harvey Cushing. If the raised intracranial pressure continues without treatment, patient becomes blind due to optic atrophy. There are also secondary effect in other parts of the brain. Later, patient gradually becomes unconscious. In present series 25% cases were blind during initial presentation and 16.66% were in unconscious state.

Initial investigations for intracranial tumour is plain X-ray skull. But it has some limitations. Even in a selected series of 136 patients with proven intracranial tumour reviewed by Stenhouse, the incidence of positive finding was 17.7%[7]. In present series percentage of abnormal finding was 33.3%. The increased positive finding in our series was probably due to late presentation of cases. Occasionally isotope brain scan fails to detect posterior fossa and skull base tumours. In this series isotope scans were negative in three cases. In one of those cerebellopontine angle tumour was detected when CT scan imaging was done.

Meningiomas are the most common neoplasm of nonglial origin arising within the cranial cavity and account for approximately 15 percent of all primary intracranial tumours[8]. Ninety percent of intracranial meningiomas are supratentorial and 10 percent infratentorial. Approximately one third of supratentorial meningiomas arise along the superior sagital sinus or falx, one third over the convexity of the hemispheres and one third from basal region[9]. In the present series incidence of meningioma was about 36% of which 38.8% were in parasagital region. The increased incidence of meningioma in this series was probably due to late presentation of cases. Unlike meningioma, astrocytomas are rapidly growing tumours and some of the patients died before they received proper medical care.

It may be concluded that about half of our cases presented quite late with preoperative complications like coma, mental dullness, hemiplegia, blindness and impaired hearing. Further, they had to be operated with scanty facilities in stages leading to post-operative high morbidity and mortality.

Acknowledgement:

We are very grateful to Dr. A. Sayed and Dr. S.M. Jahangir for considerable help at all stages of preparation of this article.

Reference :

1. Ramamurthi B. Clinical feature of intracranial tumour. In : Ramamurthi B, Tandon PN (eds.). Text Book of Neurosurgery, Vol. II, 1980. India : National Book Trust. pp 800-827.
2. Selby R. Intracranial neoplasm in Malaysia. Internat Surg 1973; 58 : 556.
3. Weinman DF. Incidence and behaviour pattern of intracranial tumours in Ceylon. Internat Surg 1973; 58 : 548.
4. Ramamurthi B. Introduction. In : Ramamurthi B, Tandon PN (eds.). Text book of Neurosurgery, Vol. II, 1980. India : National Book Trust. pp725-733.
5. Dastur HM, Lalitha VS. Pathology. In : Ramamurthi B, Tandon PN (eds.). Text Book of Neurosurgery, Vol. II, 1980. India : National Book Trust. pp733-786.
6. Garfield JS. Malignant intracranial tumours. In : Miller JD (ed.). Northfields Surgery of the Central Nervous System. Chap. 5, 1986. London : Blackwell Scientific Publication. pp178-227.
7. Maravilla KR. Supratentorial glioma : Radiology. In : Wilkins, Rangachary SS (eds.) Neurosurgery. Vol. I. 1985. New York : McGraw-Hill. pp564.
8. Foy PM. The meningioma. In : Miller JD (ed.). Northfield Surgery of Central Nervous System, 1986. London : Blackwell Scientific Publication. pp255-286.
9. Maxwell RE, Chou SN. Preoperative evaluation and management of meningioma. In : Sweet HS (ed.). Operative Neurosurgical Techniques, 1988. Hong Kong: W.B. Saunders company. pp-547-553.

Journal of Bangladesh College of Physicians and Surgeons
Vol. 12. No. 2. May 1994

Patterns of Referral in Psychiatry Outpatients Department

M E KARIM, FCPS[a], M S I MULLICK. FCPS [b], S CHOWDHURY, MCPS, DCAP[c]

Summary :

The study was done to see the pattern of referral and the characteristics of the patients referred for psychiatric consultation in a general hospital psychiatric outpatient department over a period of three months. About 32.5% patients were referred by relatives and followed by 17% and 11.55% referred from out patient department (OPD) of Mitford Hospital and general practitioners respectively. About 93% of referred patients were found to have purely functional disorders and rest had concurrent physical and psychiatric disorders. Schizophrenia was the most common diagnosis (45.5%) followed by depressive disorders (20%), bipolar affective disorders (13.5%) and dissociative or conversion disorders (8%). Most of the patients belonged to age group of 20-40 years. Most of the patients were literate (66%) and 62.5% patients came from the urban background.

(J Bangladesh Coll Phys Surg 1994; 12: 54–58)

Introduction :

Psychiatric consultation services became popular with the establishment of psychiatric units in general hospitals after world war II and enabled psychiatrists to become directly involved in the care of physically ill patients. Epidemiological studies have shown a positive correlation between physical disease and psychiatric disorders. In one of the studies it was found that 30-60% of inpatients and 50-80% of out-patients of general hospitals suffer from psychiatric distress or psychiatric illness of sufficient severity to create a problem for the health professionals[1].

The objective of the study was to see the pattern of referral for psychiatric consultation in a general hospital, frequency of functional disroder among the referred patients, cause of referral, psychiatric diagnosis, organic diagnosis and allied matter regarding the subjects.

a. Md. Enayet Karim, FCPS
Assistant Professor, SSMC Mitford Hospital, Dhaka
b. Md. Sayadul Islam Mullick, FCPS
Assistant Registrar, Institute of Mental Health and Research, Dhaka.
c. Shamim Chowdhury, MCPS, DCAP, Resident Psychiatrist, Central Drug Addiction Treatment Centre, Dhaka.

Correspondence to :
Dr. Md. Enayet Karim
Assistant Professor, Department of Psychiatry
Sir Salimullah Medical College and Mitford Hospital, Dhaka.
Recieved : 20th June, 1993 Accepted: 10th November, 1993

Materials and method :

This study was conducted to determine the characteristics of the patients referred for psychiatric consultation from different referral sources. In this study all those cases were included which were referred for psychiatric consultation at psychiatry OPD of Mitford Hospital, Dhaka from different referral sources from July to September, 1992.

A multipoint questionnaire which included socio-dermographic parameters of the patients, source of referral, reasons for referral etc. was used and information were collected by interviewing the patients and their relatives by the authors. All the referred cases were seen by qualified psychiatrists and the final diagnosis, where it was made, was noted in the questionnaire. The diagnosis was made on the basis of ICD-10.

Results :

Table-I shows the socio-demographic characteristic of the referred patients. Of the patients 55% were males and 45% females. The mean age of the patients was 29.6 years (sd-11.09). Most of the patients were literate (66%) and regarding occupation, 28.5% were houswives and 16.5%,16.5%, 10.5%, 8.5% and 7.5% were students. unemloyed, businessmen, service holders and cultivators respectively. Among the referred cases 62.5% patients were

Table—I

Socio-demographic characteristics of referred patients

Characteristics	Number	Percent
Sex :		
Male	110	55.0
Female	90	45.0
M/F ratio = 1 : 0.82		
Education:		
Illiterate	68	34.0
Primary	41	20.5
Secondary	42	21.0
S.S.C.	19	9.5
H.S.C.	20	10.0
Graduate	9	4.5
Postgraduate	1	0.5
Occupation:		
Student	33	16.5
Housewife	57	28.5
Service	17	8.5
Business	21	10.5
Cultivation	15	7.5
Labour	12	6.0
Self employed	7	3.5
Household	5	2.5
Unemployed	33	16.5
Social background:		
Rural	75	37.5
Urban	125	62.5
Marital status:		
Unmarried	87	43.5
Married	100	50.0
Widow	7	3.5
Divorced	4	2.0
Separated	2	1.0
Economic background:		
Higher	12	6.0
Middle	91	45.5
Lower	97	48.5
Age:		
Upto 20	39	19.5
21-30	83	41.5
31-40	47	23.5
41-50	22	11.0
51-60	6	3.0
Above 60	3	1.5
Mean age = 29.6 (SD=11.09)		

with an urban background, and 56.% were married. Regarding economic condition 48.5%, 45.5% and 6% patients were of lower, middle and higher economic background respectively.

Table-II shows that 32.5% patients were referred by relatives and followed by those from the OPD of Mitford Hospital (17%), by general practitioners (11.5%), by patients treated previously in psychiatry OPD (11.5%), by other medical personnel (9.5%) and from mental hospital (5%) respectively.

Table—II

Source of referral

Source	Number N = 200	Percent
OPD of Mitford Hospital	34	(17.0)
Medicine	20	10.0
ENTD	3	1.5
Paediatrics	3	1.5
Surgery	1	0.5
Orthopaedics	1	0.5
Emergency department of Mitford Hospital	6	3.0
Mental Hospital	10	5.0
Other Hospitals	3	1.5
Specialists	10	5.0
G.P.	23	11.5
Medical students	5	2.5
Other medical personnel	19	9.5
Treated psychiatric pts.	23	11.5
Self	6	3.0
Relatives	65	32.5
Traditional healers	2	1.0

Table—III

Reasons mentioned for requesting consultation

Reason	Number (N=200)	Percent
Diagnosis and treatment	182	91.0
Management	18	9.0

Table-III shows that 91% patients were referred for probable psychiatric diagnosis and management and other 9% were referred for management of already diagnosed cases.

Table-IV shows that 45.5% of the referred patients were suffering from schizophrenia followed by those from depressive disorder (20%), bipolar affective disorder (13.5%), dissociative (or conversion)disorder (8%), substance abuse (4.5%), anxiety disorder (4.5%), phobic disorders (1%), mental retardation (1.5%), dementia (1%) and epilepsy (0.5%).

Table-V shows that 93% of the referred patients were suffering from purely functional disorder and 7% were from functional disorders along with the organic diseases.

Table—IV

Distribution of psychiatric diagnosis

Psychiatric diagnosis	Male (N=110)	%	Female (N=90)	%	Total N. %
Schizophrenia	62	56.36	29	32.22	91 (45.5)
Bipolar affective disorder	15	13.63	12	13.33	27 (13.5)
Depresssive disorder	10	9.06	30	33.33	40 (20)
Anxiety disorder	8	7.27	1	1.11	9 (4.5)
Phobic disorder	1	0.90	1	1.11	2 (1)
Dissociative disorder (or conversion)	2	1.98	14	15.55	16 (8)
Substance abuse	9	8.18	-	-	9 (4.5)
Mental retardation	3	2.72	-		3 (1.5)
Epilepsy	-	-	1	1.11	1 (0.5)
Dementia	-	-	2	2.22	2 (1)

Table—V

Frequency of functional and mixed disorders

	Functional		Total	Mixed		Total
	Male	Female		Male	Female	
Schizophrenia	61	28	89	1	1	2
Bipolar affective disorder	15	12	27	-	-	-
Depressive disorder	6	28	34	4	2	6
Anxiety disorder	8	1	9	-	-	-
Phobic disorder	1	1	9	-	-	-
Dissociative disorder (or conversion)	2	14	16	-	-	-
Substance abuse	9	-	9	-	-	-
Mental retardation	-	-	3	-	3	
Epilepsy	-	-	-	-	1	1
Dementia	-	-	-	-	2	2
			186 (93%)			14 (7%)

Discussion :

The study was done on the basis of information obtained from patients referred from different sources for psychiatric consultation. It was generally found that frequency of functional disorders varied between 14 and 69%[2-7] . In Bangladesh, two surveys of such nature have been done so far and the rate of functional disorder among the patients attending hospital medical outpatients department and general practice were found to be 31% and 29% respectively[2,3].

In our study 32.5%, 17%, 11.5%, 11.5%, 9.5%, 5% and 5% patients were referred by relatives, OPD of Mitford Hospital, general practitioners, previously treated psychiatric patients, other medical personnel, mental hospital and specialists respectively. In one of the studies it was found that 65% of the total referred cases came from medical units for psychiatric consultation[8]. Kligerman and McKegray also reported the same pattern[9]. In our study, majority of the cases referred by relatives and this may be due to increased awareness among the people about treatment of psychiatric disorders and also reflects decrease in social stigmas about mental disorders.

In present study 91% of patients were referred with the request of diagnosis and treatment and 9% referred for management of already diagnosed cases. In one of the studies it was found that 75% and 80% of the reasons for referral were for assistance in diagnosis and advice on patient management or both[10]. Another study reported that 27.7% patients were referred for diagnostic problem only[8].

Of the diagnosed cases, 45.5% were found suffering from schizophrenia. From other studies it was found that 3.1% to 16.49% of the patients were suffering from schizophrenia[8-13]. In our study number of schizophrenic patients were more which might be due to increased attendance of schizophrenic patients in the OPD during the study period. Moreover, minor emotional prblems might have been overlooked or could not be reconginzed at all and patients were only referred when there were overt psychological symptoms or disturbances of social functioning. It was also found that male schizophrenic patients outnumbered female. This variation may be due to the fact that females are less frequently brought for treatment than their male counterpart because of the conservative attitude of our society and because our women play subordinate role in the family as well as in the society, and they are economically dependent.

Of the referred cases, 20% of patients were found to be suffering from depressive disorders and most of them were females. In other studies it was found that about 11 to 57% patients were suffering from depressive disorder[4,8-11,14-16]. It is not surprising, therefore, that depressive disorders account for close to half of psyshicatric diagnoses recorded by liaison psychiatrists[10] and women are more prone to depression than men[6]. About 13.5% and 8% of our patients were found to suffer from biopolar affective disorders and dissociative (or conversion) disorders respectively. A number of studies found that 22.35% patients were of affective disorders[4,8-11] and their findings were more or less consistent with our report.

In our series, 4.5% patients were found to be suffering from anxiety disorders. Similar finding was reported in other studies [4,8,10,11,15]. About 4.5% were referred for substance abuse disorder which is consistent with similar other studies[8,9,15]. This reflects that a burning problem in our society needs to be controlled.

Of the referred cases, 93% were found to be suffering from purely functional disorders having no identifiable physical disease and rest of the patients were having concurrent physical and functional disorders. Similar other studies showed that frequency of functional disorders were between 14 and 69%[2-7] and 20 and 69%

had concurrent physical and psychiatric disorders[6,8-12,17], and majority of their patients were referred from different units of the hospitals. This shows that largest number of cases attending the psychiatric OPD of a general hospital are suffering from purely functional psychiatric disorders and considerable amount of cases have a positive association between physical disease and psychiatric disorder.

Of the referred cases 62.5% came from urban background and this might be due to the location of the hospital where urban people are in more privileged position to avail the treatment. However, a significant proportion, 37.5%, came from rural areas to this city centre. It was found that 66% patients were literate. This figure is much higher than the literacy rate of general population. The urban influence of the sample and educated peoples better health consciousness may explain the higher representation of this group.

In our study, majority of the cases are referred by relatives and this reflects increase awareness among the people about treatment of psychiatric disorders and also gives an idea about gradual decline in social stigmas about mental disorders. Profile of referral from other departments of the hospital was not satisfactory. In one of the studies it was found that 30-60% of inpatients and 50-80% of outpatients of general hospitals suffer from psychiatric distress or psychiatric disorders of sufficient severity. Psychiatric referral system from other departments should therefore be improved so that patients suffering can be minimized and appropriate treatment could be provided at proper time.

References :

1. Lipowski ZJ. Review of Consultation psychiatry and psychosomatic medicine. Psychosom Med 1967; 29 : 201-204.
2. Chowdhury AKMN, Selim M, Sakeb N. Some aspects of psychiatric morbidity in outpatient population of a general hospital. Bangladesh Med Res Counc Bull 1975; 1 : 51-59.
3. Alam MN. Psychiatric morbidity in general practice. Bangladesh Med Res Counc Bull 1978; 4 : 38-42.
4. Murthy RS, Verghese A, Pulimood BM, Kurnivilla K. Psychiatric illness at general hospital medical clinic. J Indian Med Assoc 1976; 66 : 48-51.
5. Goldbergh D, Blackwell B. Psychiatric illness in general practice. Br Med J 1970; 2 : 439-449.
6. Shevitz SA, Silberfarb PM, Liposwki ZJ. Psychiatric consultations in a general hospital. Dis Nerv Syst 1976; 37 : 295-300.
7. Kaufman TD, Bernstein DA. Psychiatric evaluation of the problem patients. JAMA 1957; 163 : 108-110.
8. Taylor G, Doody K. Psychiatric consultation in a Canadian general hospitals. Can J Psychiat 1979; 24: 717-713.
9. Kligerman MJ, Mckegney FP. Pattern of psychiatric consultations in two general hospitals. Psychiat Med 1971; 2 : 126-132.
10. Lipowski ZJ, Wolston EJ. Liaison Psychiatry : Referral patterns and their stability over time. Am J Psychiat 1981; 138 : 1608-1611.
11. Lipowski ZJ. Consultation-liaison psychiatry : An overview. Am J Psychiatry 1974; 131-623-630.
12. Lipowski ZJ, Kirikos RZ. Observation in a neurological hospital. Psychiat Med 1972; 3 : 113-147.
13. Ananda MP. Psychiatric Liaison referrals of elderly inpatients in a teaching hospital. Br J Psychiat 1988; 152 : 45-47.
14. Poe RO, Lowell FM, Fox HM. Depression. JAMA 1966; 195 : 345-350.
15. Karasu TB, Plutchik R, Steinmuller RI, Conte H, Siegel B. Patterns of psychiatric consultations in a general hospital. Hosp Commun Psychiat 1977; 28: 291-294.
16. Wise TN, Mann LS, Puscheck E, Dove H, Kiernan K. Factors affecting anxiety and depression in psychiatric consultations patients. Intl J Psychiat Med 1985-86; 15 : 177-182.
17. Wise MG, Taylor SE. Anxiety and mood disorders in medically ill patients. J Clin Psychiat 1990; 61 : 27-29.

REVIEW ARTICLE

(J Bangladesh Coll Phys Surg 1994; 12: 59–62)

Management of Acute Poisoning with Organophosphorus Insecticide

M A FAIZ[a], M R RAHMAN[b], T AHMED[c]

Organophosphorus compounds are widely used as insecticide in agricultural sector by the farming community in Bangladesh. Studies from some major hospitals indicate that organophosphates account for leading cause of morbidity and mortality due to poisoning in our country [1,2,3,4]. Deliberate self poisoning by ingestion of organophosphates with an attempt of suicide is commoner than accidental poisoning by occupational exposure due to skin contamination and inhalation. Numerous organophosphate and carbamate ester insecticides with diverse structures are available in the market of Bangladesh[5]. However, their toxic effects are identical and associated with inhibition of the nervous tissue acetylcholinesterase (AChE) [6]. AChE is the enzyme responsible for the biological activity of the neurotransmitter acetylcholine (Ach).

The signs of toxicity of organophosphorus insecticides are due to effects of accumulated ACh. ACh causes stimulation of the muscuranic receptors of the parasympathetic autonomic nervous system (increased secretion, bronchoconstriction, miosis, gastrointestinal cramps, diarrhoea, urination, bradycardia); stimulation and subsequent blockage of nicotinic receptors (muscle fasciculation, tremor, muscle weakness, and/or flaccid paralysis); and effects on the central nervous system (restlessness, emotional lability, ataxia, lethargy, mental confusion, loss of memory, generalized weakness, convulsion, cyanosis, coma)[6]. The other manifestations of organophosphate intoxication are Intermediate Syndrome (IS) and Organophosphate Induced Delayed Neurotoxicity (OPIDN). The clinical features of IS include sudden respiratory paresis, several motor cranial nerve paresis, and weakness of proximal limb and neck flexor muscles in 24-96 hours after apparently well-treated acute cholinergic crisis[7]. OPIDN is a predominantly motor distal neuropathy arising 2-3 weeks after exposure to some organophosphorous agents that are capable of inhibiting a distinct esterase enzyme called Neuropathy Target Enzyme (NTE)[8].

The World Health Organization has highlighted the problem of the insecticide poisoning and estimated about 500,000 cases of pesticide poisoning with 5,000 deaths each year in the less developd nations. Despite such a huge health problem, however, authors of widely used textbooks of medicine continued to ignore the topic of insecticide poisoning like the governments of the third world countries ignoring the control of acute pesticide poisoning[10]. In this article the treatment of acute poisoning by organophosphorus compound is emphasised.

The ABC of life should be maintained by removal of oral and respiratory secretions by suction, oxygen administration and main tenance of respiratory rate, cardiac rhythm and blood pressure.

a. Md. Abul Faiz
Associate Professor, Department of Medicine, Chittagong Medical College, Chittagong
b. Md. Ridwanur Rahman
Department of Medicine, Chittagong Medical College, Chittagong
C. Tofayel Ahmed
Professor, Department of Medicine, Sir Salimullah Medical College, Dhaka

Correspondence to:
Md. Abul Faiz
Associate Professor, Department of Medicine, Chittagong Medical College, Chittagong
Recieved: 1st May, 1994 Accepted: 10th May, 1994

Reduction of exposure:

Complete and quick removal of compound which has not yet been absorbed is possible by removal of clothing and washing the skin with an alkaline soap (in case of dermal exposure); and irrigation of eyes with water or saline for 10-15 minutes (in case of eye contamination). When the chemical is ingested or inhaled, the procedure of induction of vomiting in concious patients should be considered. In unconcious patients, gastric lavage may be given with protection of airway. Gastric lavage is most effective if given within half an hour.

Pharmacological treatment :

Atropine : Atropine antagonises the effects of acetylcholine and then reverses the excessive parasympathetic stimulation by competing for identical binding sites at muscuranic receptors. In the patients poisoned by organophosphorus compounds, large doses of atropine are required due to tolerant effect[11]. The amount of atropine to be used depends on the severity of poisoning (Tables-I and II) and must be titrated according to the clinical evidence of atropinization[12]. These are flushing, dry mouth and nose, broncho-

Table—I

Assessment of the severity of poisoning

Grade	Clinical Presentation	Laoratory Finding
Mild poisoning	Dizziness, headche, nausea, vomiting, diarrhoea, salivation, wheezing	RBC AChE >40%
Moderate poisoning	Mild + ataxia, muscle fasciculation, dysarthria, meiosis (pin point)	RBC AchE <40%
Severe poisoning	Above + cyanosis, respiratory failure, coma, convulsion, loss of pupil reflexes	RBC AChE <20%
Intermediate Syndrome	Paralysis of proximal limb, neck flexors, motor cranial and respiratory muscles	

Table—II

Therapeutic approach to acute organophosphorus poisoning

Grade	Treatment					Sequence
	1 Artificial Ventilation	2 Diazepam	3 Decontamination	4 Atropine	5 Pralidoxime	
Mild	-	10 mg SC	+	2 amps I. V. (children 0.01 mg/kg)	-	3,4,2
Moderate	-	10 mg SC	+	6 amps. I. V./ 15 min. (children 0.05mg/kg)	1g over 30 min/12h	4,3,2,5
Severe	+	10 mg SC (repeat if necessary)	+	above or I. V. infusion 100 amps/500cc 5% saline over 4h	1g over 30 min/12h	1,2,4,5,3
Intermediate syndrome	+	-	-	-	-	-

dilatation, disappearance of sweating, dilatation of pupil and increased heart rate. If such signs are not found, the dose of atropine is to be increased. Continuous intravenous infusion of atropine is required in most severe cases[13].

Atropinization should be maintained for at least two days and sometimes for several weeks depending on the severity of poisoning. During the period of withdrawal of atropine, careful monitoring of the patient is necessary to detect relapse of signs of toxicity as organophosphorus esters stored in fat may act as "slow-release" reservoir[14].

Oximes : Atropine has no effect on nicotinic and central nervous system effects, for which oximes are used as specific antidotes for poisoning by organophosphorus compound. Their mechanism of action consists of regeneration of inhibited AChE from acetyl-cholinesterase-organophosphorus complex. These drugs are effective only when phosphorylated AChE has not udergone "ageing" with irreversible loss of function[12]. It potentiates the effect of atropine. Pralidoxime (2-pyridine-aldoxim or 2-PAM; is currently used for reactivation of phosphorylated AChE. Usually it is given in a dose of 1 g intravenous injection to be repeated 2-3 times per day. Oxime treatment is usually effective within 24-96 hours of exposure. Several studies showed that oximes are not very efficient reactivators in reversing the intoxication syndrome[15]. Recently the usefulness of pralidoxime in acute organophosphorus poisoning has been questioned. It is suggested that atropine alone seemed to be as effective as atropine plus PAM in the management of acute organophosphorus intoxication. This lack of efficacy could be that doses of oximes used in those studies were too low and the therapy is stopped too early[16].

Supportive treatment : Diazepam in doses of 10 mg intravenously or subcutaneously have been suggested for its effect in relieving anxiety, antagonising convulsion, and improving morbidity and mortality [17]. Artifical ventilation is required for patients who develop respiratory failure and must be started at the first sign of respiratory failure.

Like many other hospitals in Bangladesh, in Chittagong Medical College Hospital, we usually treat the patients of organophosphorus poisoing with measures to reduce the exposure and with adequate dose of atropine depending on severity of poisoning. Sometime heroic dose of atropine as much as few thousand milligram of the drug is used during the management of severe degree of poisoning. Diazepam is used occasionally. We have limited experience of using pralidoxime with success in selected cases. Provision for artifical respiration is not available in our hospital. A prospective study on different aspects of organophosphorus poisoning including psychological assessment of the patient is going on in Chittagong Medical College Hospital.

In order to make a comparison of patients and assess their management, the cases should be standardized according to severity of poisoning [17]. At present atropine is available in 0.65 mg ampoule in Bangladesh. Availability of considerably large amount of atropine per ampoule (for example, 2 mg/ampoule) may ease the frequent administration that is used in organophosphoruus poisoning. PAM should be available in hospitals or at low cost for general use. Prospective controlled trial is needed before making a firm comment on usefulness or lack of efficacy of PAM in organophosphorus poisoning. Establishment of intensive care unit with facilities for artificial respiration may save many valuable young lives. Prevention of attempted suicide by insecticide should be attempted by legislative measures on storage, sale and usage of insecticides, and as well as by development of public awareness about the potential health hazards of organophosphorus poisoning.

References :

1. Faiz A. Organosphosphorus compound intoxication. Med Dig 1981; 1 : 62-63.

2. Karim S A, Faiz M A, Nabi MN. Pattern of poisoning in Chittagong Medical College Hospital. JCMTA 1993; 4 : 10-14.

3. Khan N I, Sen N, Haque NA. Poisoning in a medical unit of Dhaka Medical COllege Hospital in 1983. Bang Med J 1985; 14 : 9-12.

4. Rahman MM, Mohammad RJ, Khan GK, Amin M, Hossain MZ, Rahman M. Clinical pattern of acute poisoning in Dhaka Medical College Hospital during the year 1993. Abstract of paper read out in the Scientific Seminar of 5th National Convention of Association of Physicians of Bangladesh, Dhaka 1994.

5. Ciba-Geigy Publication 1993. Emergency Medical Treatment for Pesticide Poisoning.

6. Ecobichon D J. Toxic effects of pesticides. In : Amdur M O, Doull J, Kladssen C D (eds) Casarett and Doull's Toxicology. The basic science of poisons. 4th ed. 1991. London : Pergamon Press. pp. 565-618.

7. Senanayake N, Karalliede L. Neurotoxic effects of organophosphorus insecticides. An Intermediate Syndrome. N Engl J Med 1987; 316 : 761-763.

8. Lotti M. The pathogenesis of organophosphate polyneuropathy. Crit Rev Toxicol 1992; 21 : 465-487.

9. Jeyaratnam J. Health problems of pesticide usage in the Third World. Br J Ind Med 1985; 42 : 505-506.

10. Edwards CRW, Bouchier IAD. Acute Poisoning. In: Davidson's Principles and Practice of Medicine, 16th ed. 1991. London : Churchill and Livingstone. pp. 968-983.

11. Golsousidis H, Kokkas V. Use of 19590 mg of atropine during 24 days of treatment after a case of unusaully severe parathion poisoning. Hum Toxicol 1985; 4 : 339-340.

12. Minton NA, Murray VSG. A review of organophosphate poisoning. Med Toxicol 1988; 3 : 350-375.

13. Le Blanc FN, Benson BE, Gilg AD. A severe organophosphate poisoning requiring the use of an atropine drip, J Toxicol Clin Toxicol 1986; 24: 69-76

14. Ecobicon DJ, Ozere RL, Reid E, Crocker J F S. Acute fenitrothion poisoning. Can Med Assoc J 1977; 116 : 377-379.

15. Willems J L. Poisoning by organophosphate insecticides : analysis of 53 human cases with regard to management and drug treatment. Acta Med Milit Belg 1981; 134 : 7-14.

16. Willems J L, Langenbeg J P, Verstraete J L et al. Plasma concentration of pralidoxime methylsulphate in organophosphorus poisoned patients. Arch Toxicol 1992; 66 : 260-266.

17. Lotti M. Treatment of acute organophosphate poisoning. Med J Aust 1991; 154 : 51-55.

CASE REPORTS

Sinus Histiocytosis with Massive Lymphadenopathy - Rosai Dorfman Disease - A Case Report

M M RAHMAN, FCPS[a], P PURKAYASTHA , FCPS, FRCP[b]
K M N ISLAM, M PHIL, FCPS[c], M RAHMAN, MBBS[d]

Summary :

A 38 years old housewife was admitted into the Mitford Hospital with swellings in the neck, axillary and inguinal regions for last eight years. About two years back she developed erythematous rash initially on the face and later throughout the body. On examination, no abnormality was found except the skin lesions and palpable cervical, axillary and inguinal lymphnodes. She had neutrophilic leucocytosis, a very high ESR and nodular opacities in both hilar regions in chest X-ray. Skin biopsy and lymphnode biopsy showed the features of sinus histiocytosis with massive lymphadenopathy-Rosai Dorfman disease. The patient was given only symptomatic treatment. Rosai Dorfman disease is a very rare disorder and this case is presented to acknowledge its presence in Bangladesh and to develop awareness.

(J Bangladesh Coll Phys Surg 1994; 12: 63–67)

Introduction :

Sinus histiocytosis with massive lymphadenopathy (SHML)[1] is a rare benign generally self limited disease that typically appears with cervical lymphadenopathy (often bilateral, painless and massive) usually accompanied by fever. Other lymphnode groups (such as mediastinal, axillary and inguinal) also may be involved[2]. Between a quarter and one third of cases show involvement of extranodal sites including skin[3], orbit and eyelids[4], upper rspiratory tract and salivary glands[5], bones and testis [6] and lung, kidney and peritoneum[7]. The liver and spleen are usually of normal size[8]. Leucocytosis with neutrophilia, an elevatd ESR and polyclonal hypergammaglobulinaemia are common features. The disease occurs in any age but shows a marked predilection for the first and second decades of life. There is no sex predilection[3]. Blacks seem to be more susceptible to this disorder which has a worldwide distribution but is more commonly seen in Africa and West Indies. There is no consistent response to antibiotics, steroids, irradiation or chemotherapy, although local surgery may be of benefit. The course of the disease is almost invariably benign and usually follows a protracted indolent course and leads towards spontaneous resolution, although in some cases lymphadenopathy may last for years.

The cause of SHML remains unknown, it may represent a specific infectious process[8] possibly occuring in patients with an underlyng immunological deficit[9].

The microscopic appearance of the involved lymphnodes is characteristic. There is marked capsular and pericapsular fibrosis with a striking dilatation of the lymphnode sinuses. The later are crowded with inflammatory cells of several types, the predominant cells being sinus histiocytes with large vesicular nuclei and abundant weakly acidophilic cytoplasm. Typically some of the histiocytes contain in

a. Dr. Md. Muhibur Rahman, FCPS, Registrar, Medicine, SSMC Mitford Hospital, Dhaka.
b. Prof. Probhakar Purkayastha, FCPS, FRCP, Prof. & Head, Department of Medicine, SSMC Mitford Hospital, Dhaka
c. Prof. K. M. Nazrul Islam, M. Phil, FCPS, Ex-Professor of Pathology, IPGM&R, Dhaka.
d. Dr. Mizanur Rahman, Asstt. Registrar, Medicine, SSMC Mitford Hospital, Dhaka.

Correspondence to :
Dr. Muhibur Rahman
Registrar, Medicine, SSMC Mitford Hospital, Dhaka
Received: 11th November 1993, Accepted: 5th May, 1994

their cytoplasm a large number of phagocytosed lymphocytes and other inflammatory cells (lymphophagocytic histiocytosis). The sinusoidal histiocytes are predominantly mononuclear with occasional multinucleated cells, some of which contain cytoplasmic Russell bodies. The microscopic appearance of Histiocytosis X primarily affecting lymphnodes may be confused with SHML.

SHML is a rare disorder and as this disease present with lymphadenopathy, it may be confused with other disorders affecting lymphnodes e.g. lymphoma, storage diseases. This case of SHML is presented here to develop awareness about the presence of this rare disease in our country.

Case Report :

A 38 years old housewife was admitted in a surgical unit of Mitford Hospital with the complaints of pain in right upper abdomen for two days which was associated with vomiting. She was diagnosed as a case of acute cholecystitis with cholelithiasis but she was referred to medical unit for some other medical problems.

On admission into medical unit it was revealed that she developed swelling of the neck initially on the right side later on the left side about eight years back. Swelling was painless but gradually increased in size. It was associated with low grade fever, headache, anorexia and malaise. With this complaints she was admitted into an academic hospital where the swelling was found to be due to cervical lymphadenopathy and lymphnode biopsy was done. She was diagnosed to be a case of lymphoma and was treated with radiotherapy, but with no improvement. Gradually she developed swelling in axillary and inguinal regions. About two years back she developed erythematous rash which appeared first on the face later spreaded throughout the body. The rashes had no relation with exposure to sun and did not show any change.

She was again admitted into the same hospital about one year back where a skin biopsy was done and was diagnosed to be a case of post Kala-azar dermal leishmaniasis (PKDL). But she did not give any suggestive history of Kala-azar. Though the patient had anorexia and low grade fever, she did not give any history of marked deterioration of general health.

On examination, the patient was found in good general condition. She was midly anaemic, not icteric or cyanosed and there was no clubbing of fingers or toes. Her pulse rate was 96/minute, regular, BP was 110/60 mm of Hg and body temperature was normal.

Lymphnodes were palpable in cervical, axillary and inguinal region on both sides. But epitrochlear lymphnodes were not palpable. Lymphnodes were of varying sizes from 0.5-2 cm in diameter. Cervical groups were larger than others. Lumphnodes were firm, mostly discrete, nontender and not fixed to the skin or underlying structures. No sinus was visible over the lymphnodes. Rounded and oval erythematous rashes were present on different parts of the body. Rashes on the face were larger varying in size from 2-5 cm with elevated margins from surrounding skin. There was no sensory impairment over the rashes. Rashes on the chest, back, abdomen, upper and lower limbs were smaller than those on the face, little blackish in colour and margins not elevated from surrounding skin.

Examination of cardiovascular system, respiratory system, gastrointestinal and nervous systems did not reveal any abnormality.

Investigations :

Her haemoglobin (Hb) was 62%. Total count of WBC was 12,300/cumm, with 74% neutrophil, 20% lymphocyte, 4% eosinophil and 2% monocyte. ESR was 140 mm in 1st hour (Wester-gren method). Blood sugar (2 hours after 75 gm of glucose) was 6.6 mmol/L. Blood urea-26 mg%. Serum bilirubin, SGOT, SGPT and alkaline phosphatase levels were within normal range. Serum globulin level was estimated at 2.7 gm% (normal 2.6-3.5 gm%). VDRL was non-reactive. Routine examination of urine and stool was normal. X-ray chest P/A view showed nodular opacities in the perihilar region consistent with enlarged lymphnodes. Aldehyde test and CFT for Kala-azar were negative. Ultrasonography of hepatobiliary system and pancreas revealed cholelithiasis. Bone marrow examination showed increased myeloid-erythroid ratio, active marrow with increase in plasma cells and some lymphocytes. No evidence of lymphomatous infiltration or LD bodies was present. Lymphnode biopsy and biopsy from a skin lesion was taken. Skin biopsy showed the dermis densely invaded by large collection of plasma cell and histiocytes. The later cells had abundant foamy or finely granular eosinophilic cytoplasm. Lymphophagocytosis and phagocytosis of red cells and neutrophil were prominent. Lymphnode biopsy showed marked alteration of normal architecture. Like the skin, it revealed collection of palsma cells, lesser number of lymphocytes and a large collection of histiocytes showing lymphophagocytosis. Prominent deposition of hyaline was also present. No malignancy was seen.

Diagnosis was made by histopathological examination which is also suported by other investigations. The patient was given only symptomatic treatment and was briefed adequately about the disease. She was discharged with advice to report after two months.

Discussion :

SHML or Rosai-Dorfman disease is a rare disorder and as it appears with lymphadenoathy, chances of misdiagnosis is quite high. So far there has been no report of this disorder from Bangladesh. Rosai and Dorfman described 34 cases of SHML in 1972 and established the validity of SHML as a definite clinicopathological entity[2]. The first case was described by Azour and Reed in 1966 but the name "Sinus histiocytosis with massive lymphadenopathy" was coined by Rosai and Dorfman in 1969[1].

Of the 34 cases described by Rosai and Dorfman in 1972, 11 cases were from United States, nine from Africa, six from West Indies, three from Europe, two from India and one each from Japan, New Zealand and Mexico. It means that the distribution of SHML is world wide and not limited to single geographical area. So, presence of SHML cases in Bangladesh is also expected. SHML can occur at all ages but shows a predilection for first and second decades of life[2,6,10]. The onset of the illness in our case was at the age of 30. Three cases of SHML with onset of the illness after the age of 20 years have been reported previously[2].

SHML typically presents with massive bilateral painless cervical lymphadenopathy although other lymphnode groups may also be involved. The illness in our case also started with cervical lymphadenopathy which gradually did spread to other lymphnode regions (axillary, inguinal). Similar observations were made by Lampert and Lennert[8] and Rosai and Dorfman[1,2].

The patient complained of low grade fever during initial part of the illness. Fever was also reported in 15 of the 34 cases described by Rosai and Dorfman[2]. An otherwise excellent general condition was found in our case which is corroborative with previous observations [2,10].

Another prominent feature in this case was involvement of skin in the form of rounded and oval erythematous flat papular rashes. Thawerani et al[3] described 14 cases of SHML with cutaneous lesions. The lesions were multiple in seven cases and most lesions were papular or nodular upto 4 cm in diameter and had either a xanthomatous or erythematous appearance. The patient was midly anaemic and had leucocytosis with neutrophilia. Mild anaemia, leucocytosis and neutrophilia had been a frequent finding in previous studies[1,2,6]. ESR was constantly elevated in all previous studies and ESR in our case was 140 mm in first hour. Though hyperglobulinaemia was a frequent finding in previously described cases, hyperglobulinaemia was not found in our case. X-ray chest showed nodular opacities in perihilar region consistent with lymphnode enlargement. Perihilar and mediastinal lymphadenopathy has also been reported in nine out of 34 cases by Rosai and Dorfman[2]. This patient had cholelithiasis and she was admitted to a surgical unit with an attack of acute cholecystitis. Cholelithiasis does not seem to have any relationship with SHML. It appears to be a separate entity and a mere conincidence. Cholelithiasis has not been reported in any case of SHML so far.

Pathologically, the picture of SHML is quite distinct and readily identifiable. The basic abnormality is the proliferation of sinus histiocytes with abundant clear cytoplasm, sometimes resulting in complete effacement of nodal architecture. Another important feature is marked proliferation of plasma cells in medullary cords. The presence of lymphocytes and occasionally other haemopoetic cells within the cytoplasm of histiocytes (lymphophagocytosis) is a constant and a rather striking finding. It has also been seen in other conditions and in lymphnodes involved by salmonellosis, rhinoscleroma and histoplasmosis. Lennert et al[12] considered several possible mechanisms of lymphophagocytosis such as opsonization of the blood cells by immunoglobulins, production of autoantibodies against specific phagocytic activity of the reticuloendothelial system. The later phenomenon has been demonstrated in a variety of infectious diseases such as pneumococcal pneumonia and typhoid fever[13].

There are many possibilities to be considered in regard to the nature of SHML. Lymphoma can be excluded on clinical and pathological grounds. Malignant histiocytosis differs from SHML clinically by virtue of its rapidly fatal course and pathologically by the highly atypical features of proliferating histiocytes. Storage disease can be excluded in view of the absence of any significant amount of abnormal lipid, carbohydrate or mucopolysaccharide in the cytoplasm of the histiocytes as determined by histochemical and biochemical assay. Although from a strictly morphological point of view SHML could be regarded as a diferentiated histiocytosis[14], its clinical and pathological features differ markedly from those of three well recognized members of this group, namely eosinophilic granuloma, Hand-Schuller-Christian disease and Letter-Siwea disease[15].

Two major pathological mechanisms are to be considered as possible causes of SHML; a specific infectious process and an immunodeficiency state. Many of the features of SHML are strongly suggestive of an infectious aetiology. The appearance in a previously healthy individual of an episode of localized lymphadenopathy accompanied by fever, leucocytosis with neutrophilia, elevated ESR and hypergammaglobulinaemia, spontaneously receeding after a variable length of time is certainly consistent with this interpretation, so is the geographical distribution of the disease apparently concentrated in areas of the world associated with lower socio-economic status. On the other hand, attempts to document an infectious aetiology by microscopic examination, culture and other laboratory tests have been

uniformly unsuccessful[12]. There is possibility of salmonellosis in view of the morphology of histiocytes and prominent lymphophagocytosis. The lack of gastrointestinal involvement, presence of leucocytosis instead of leukopaenia and negative serological test go against this interpretation.

The second major possibility is that SHML is the expression of an abnormal immunological response. Clinical and pathological picture does not correspond to any of the recognised variation of the disease resulting from deficiency of humoral or cellular immunity[17] or from disorders of phagocytosis[18,19].

The course of the disease is prolonged and relatively unaffected by various modes of therapy. Eventually the lymphadenopathy and other symptoms regress, although it may take several years for this to occur. Delay in the diagnosis in our case was due to rarity and unfamiliarity with this disorder. Further studies on this disorder is required to ascertain the aetiology and pathogenesis of this clinicopathological syndrome.

References :

1. Rosai J, Dorfman RF. Sinus histiocytosis with massive lymphadenopathy; A newly recognised benign clinico-pathological entity. Arch Pathol 1969; 87 : 63-70.
2. Rosai J, Dorfman RF. Sinus histiocytosis with massive lymphadenopathy; A pseudolymphomatous benign disorder, Analysis of 34 cases. Cancer 1972; 30 : 1174-1188.
3. Thawerani H, Sanchuz RL, Rosai J, Dorfman RF. The cutaneous manifestation of Sinus histiocytosis with massive lymphadenopathy. Arch Dermatol 1978; 114: 191-7.
4. Foucar E, Rosai J, Dorfman RF. The ophthalmologic menifestation of Sinus histiocytoses with massive ly-mphadenopathy. Arch Ophthalmol 1979; 87 : 354-67.
5. Foucar E, Rosai J, Dorfman RF. Sinus histiocytosis with massive lymphadenoathy; ear, nose & throat manifestation. Arch Otolaryngol 1978; 104 : 687-93.
6. Azour FJ, Reed RJ. Histiocytosis, report of an unusual case. New Engl J Med 1966; 274 : 928-30.
7. Wright DH, Richard DB. Sinus histiocytosis with massive lymphadenopathy (Rosai-Dorfman Disease). Report of a case with widespread nodal and extranodal dissemination. Histopathology 1981; 5 : 657-709.
8. Lampert F, Lennert K. Sinus histiocytosis with massive lymphadenopathly : Fifteen new cases. Cancer 1976; 37 : 783-789.
9. Becroft MOD, Dix MR, Gillman JC et al. Benign sinus histiocytosis with massive lymphadnopathy; transient immunologic deficit in a child with mediastinal involvement. J Clinical Path 1973; 26 : 463-9.
10. Vincent TN, Meircort R. Case 9, Eighteenth Seminar of the Penrose Cancer Hospital. Vol. 3, 1967. pp. 246-250.
11. Story P, Hanbury WJ. Morphological changes in Salmonella typhimurium infection. J Pathol Bacteriol 1957; 73 : 443-450.
12. Lernert K, Neidford HR, Bluncke S, Hardmeier Th. Lymphadenitis with massive haemophagocytic sinus histiocytosis. Virchows Arch 1972; 10 : 14-29.
13. Wagner HN, Jr, Iio M, Hornick RB. Studies of reticulo-endothetial system (RES) II. Changes in the phagocytic capacity of the RES in patient with certain infection. J Clinical Invest 1963; 42 : 427-434.
14. Rappaport H. Turnover of the haemopoetic system. In: Atlas of Tumour Pathology. Sect. 3, fasc 8. 1966. Washington DC : Armed Forces Institute of Pathology.
15. Dargeon HWK. Reticuloendotheliosis in childhood; A clinical survey, Sprungfield IU: Charls C Thomas 1966.
16. Barandun S, Riva G, Spengles GA. In : D Bergsma (ed). Imunologic deficiency diagnosis form & current treatment 1966. New York : The National Foundation. pp 40-52.
17. Fundenberg HH et al. Classification of the primary immunodeficiecncy; WHO recommendation. N Engl J Med 1970; 283 : 656-657.
18. Douglas SD. Analytic review, disorder of phagocytic function. Blood 1975; 35 : 851-66.
19. Holmes B, Quie PG, Windhorst DB, Good R A. Fatal granulomatous disease of childhood-An inborn abnormality of phagocytic function. Lancet 1966; 1 : 1225-1231.

Journal of Bangladesh College of Physicians and Surgeons
Vol. 12. No. 2. May 1994

Tuberous Sclerosis with Primary Optic Atrophy—A Case Report

M N ISLAM[a], M A HANNAN[b], A ISLAM[b], M AHMED[b], S M S B TABIB[c]
M M RAHMAN[c], H RAHMAN[c], Z HOSSAIN[c]

Summary:

Tuberous sclerosis, a neurocutaneous syndrome, mainly involves brain, eyes and skin. Other vital organs of the body may often be affected. Ocular pathology of tuberous sclerosis is mostly limited to retina, lens, cornea and conjunctiva. Primary optic atrophy is very unusual. Such an unusual finding in a seven years old male child, alongwith most of the other common features of tuberous sclerosis is presented.

(J Bangladesh Coll Phys Surg 1994; 12: 68–70)

Introduction :

Tuberous sclerosis (TS) is a relatively uncommon disease of dominant autosomal inheritance. It is classified as a neurocutaneous syndrome in which brain, eyes, skin as well as heart, kidneys, lungs and bones may be affected[1]. The diagnosis of TS relies on a high index of suspicion when assessing a child with seizures[2]. Tuberous sclerosis should be suspected in children who have any of the following : mental retardation, seizures, intracranial calcification, skin lesions (especially highly characteristic adenoma sebaceum) or retinal tumours (phakomas). Ocular pathology in TS most commonly involves the retina but may affect the lens, cornea or conjunctiva[3]. but the presence of primary optic atrophy is very unusual. Here a case of TS with primary optic atrophy has been reported with review of diagnostic criterias.

Case Report :

A seven year old male child was admitted in the paediatrics department of the Institute of Postgraduate Medicine and Research, Dhaka with history of recurrent convulsions since six months of age. Seizures were generalised in nature preceded by aura and followed by postictal sleep. Initially the frequency of convulsions was two to three times at about two months interval. He had been taking phenobarbitone for previous three years. No convulsion was noticed for two years from the time of starting phenobarbitone but he developed focal convulsion predominantly affecting the eyes and left side of the face for last one year. The focal convulsion occurred at a interval of one to two months.

He has squint of left eye since birth. The child developed skin rashes over the face, trunk and limbs for the last three years. He had no other systemic problem. He was in grade I and failed to continue his studies. There is history of parental consanguinity. The child has only one sister who is apparently free from any major illness.

Parents are healthy and has no stigmata of disease simulating that of the child. On physical examination the child was found to have multiple brownish and dark brownish papular rash, 1-10 mm in diameter, over the bridge of the nose spreading in a butterfly distribution over the cheeks and also in trunk and limbs which is characteristic of the angiofibromas (adenoma sebaecum) of tuberous sclerosis. There were discrete irregularly thickened slightly elevated brownish patches (Shagren patches) varying from 1-2 cm in diameter and more than five in number distributed over the back of the trunk. There were numerous ovoid or ashleaf shaped

a. M N Islam, Professor
b. M A Hannan, Associate Professor
Afiqul Islam, Associate Professor
Moinuddin Ahmed, Associate Professor
c. S M Shahnawaz Bin Tabib, Assistant Professor
Md. Mizanur Rahman, Astt. Professor
Habibur Rahman, Astt. Professor
Zahid Hossain, Astt. Professor
Deptt. of Paediatrics, Institute of Postgraduate Medicine & Research (IPGMR), Dhaka.
Correspondence to :
Dr. S. M. Shahnawaz Bin Tabib
Asstt. Professor, Paediatrics, IPGMR, Dhaka
Received: 29th Dec., 1993 Accepted: 2nd Feb., 1994

hypopigmented macules varying from 0.2-2 cm in length over the limbs and trunk. A few hyperpigmented areas, 1-3 cm in length, were also present over the limb. Nervous system was thoroughly examined. He had mild mental retardation. On ocular examination, he had left microcornea with left convergent squint with eccentric fixation and poor vision of left eye. On fundoscopy there was primary optic atrophy with chroioretinal degeneration. Right eye was normal.

X-ray skull, X-ray chest, brain scintigraphy and ultrasonography of kidneys revealed no abnormality. EEG was suggestive of seizure disorder. CT scan of brain showed dot like calcifications scattered over both hemisphere of cerebrum and ventricles.No mass effect or midline shift was seen. The child was diagnosed as a case of Tuberous sclerosis with primary optic atrophy. He was given phenobarbitone and diazepam for symptomatic relief of seizures and was discharged with advice to attend neurology follow-up clinic of paediatrics out-patient department of P.G. Hospital, periodically.

Discussion :

Tuberous sclerosis (Bournevilles disease or Epiloia) was first recognised as a specific disease in the 19th century. Rayer gave the first description of the fibrovascular papules and Bourneville reported the case of a mentally retarded girl who also suffered from hemiplegia and epilepsy. Sherlock coined the word epiloia, a telescopic term to indicate the diagnostic triad of epilepsy, low intelligence and adenoma sebaceum[4]. Several recent studies suggest that incidence varies from 1/10,000 to 1/15,000 individuals. The prevalence may actually be higher because patients with one or two findings only may escape diagnosis[5]. Gomez proposed primary and secondary diagnostic criteria to classify this issue[5].

Diagnostic criteria proposed by Gomez[5]

Primary criteria (One required)	Secondary criteria (Two required)
Facial angiofibroma	Infantile spasm
Ungual fibroma	Hypometanotic macules
Cortical tuber	Shagren patch
Subependymal hamartomas	Single retinal hamartoma
Multiple retinal phakomas	Bilateral renal angiomyolipomas Cardiac rhabodomyoma First degree relative with a primary diagnosis of TS

Mental deficiency (60-80%) with seizures (85%) is the most common clinical presentation[6,7,8]. This boy initially presented with generalised seizures and subsequently partial seizures. Adenoma sebaecum of the face is the most readily recognised sign of TS occuring in 80% to 90% of patients [5,9]. Other cutaneous lesions in children include hypometanotic macules (90%), subungual fibroma (20%) and shagren patches (20%). The patient presented here had all the cutaneous lesions mentioned above except subungual fibroma. In this case, a thorough examination revealed other abnormalities of nervous system i.e. mental retardation, left convergent squint with primary atrophy with loss of central vision and intracranial calcification. However, the characteristic diagnostic ocular lesion is the phakoma, a retinal giant cell astrocytoma which was absent in this case. Other less frequent ocular lesions include pedunculated greyish white lesion in conjunctiva, cataract, pigmentary retinopathy, hypopigmented spots in iris and changes associated with pihisisbulbi[3]. But presence of primary optic atrophy in a case of TS is unusual and has not been recorded in literature.

Patient with TS may present purely with visceral lesions : renal (polycystic kidneys), cardiac (rhabdomyoma)[10,11] or pulmonary lesions

(cystic and pulmonary lymphangiomatosis). The correct diagnosis in these cases may be difficult especially in the incomplete form of the disease with no cerebral impairment[10]. This patient did not show any visceral manifestation. As parents of the patient were healthy showing no stigmata of TS, he represents a spontaneous mutation. The incidence of cases resulting from new mutation has been estimated to be as high as 80%[12,13,14]. However, careful diagnostic evaluation of family members in recent studies suggests a figure closer to 50%[5].

In conclusion, it is important to recognize the diagnostic criteria for definitive and presumptive diagnosis of TS and we should emphasize the need for follow-up of the patients regularly to look for appearance of new stigmata of the disease or its complications through baseline studies like urinalysis, renal ultrasound, fundoscopy, X-ray skull, CT scan and wood light skin examination.

References :

1. Hurwitz S, Braveiman IM. White spots in tuberous sclerosis. J Paediatr 1970; 77 : 587-94.
2. Behrman RE, Kliegman M, Nelson W E. Nelson Textbook of Paediatrics, 14th edition. London : W.B. Saunders Company. pp 1510-1511.
3. Warren DG, Robinson D H. Early recognition of tuberous sclerosis by fundoscopic examination. Paediatrics 1969; 75 : 991-95.
4. Champion RH, Burton JL. Tuberous scleroses. In: Textbook of Dermatology, Fifth edition, Vol. I, 1988. London : Blackwell SC Publication. pp 327-330.
5. Joseph C, Alper. Tuberous selerosis. Genetic Disorders of the skin. Mosby Year Book 1990. pp 236-243.
6. Pampiglione G, Pugh E. Infantile spasms and subsequent appearance of tuberous scierosis syndrome. Lancet 1975; 1 : 1046.
7. Akbar MS, Khan Naila Z. Tuberous selerosis : a case report. Bangladesh Journal of Child Health 1986; 10 : 83-86.
8. Monaghan HP, Keafchik B R et al. Tuberous sclerosis complex in children. Am J Dis Child 1981; 135 : 912-917.
9. Sathyamoorthy P. Acute presentation of tuberous selerosis : a case report. Singapore Med J 1993; 34 : 358-360.
10. Rodriguerz VL, Arino I, Liedanna JM, Horndler C, Roncales A R. Bilateral angiomyolipomas of the kidneys: Bournville's tuberous sclerosis. Arch Esp Urol 1989; 42 : 423-31.
11. Smith HC, Watson GH, Patel RG, Supor M. Cardiac rhabdomyomata in tuberous sclerosis. Their course and diagnostic value. Arch Dis Child 1989; 64 : 196-200.
12. Gomez M R. Clinical experience at Mayo Clinic. In : Gomez M R (ed). Tuberous sclerosis. New York : Raven Press, 1979. pp 13-15.
13. Lagos JC, Gomez MR. Tuberous sclerosis, reappraisal of a clinical entity. Proc Mayo Clin 1967; 42 : 26-49.
14. Bundeys, Evans K. Tuberous selerosis; a genetic study. J Neurol Neurosurg Psychiatry 1969 : 32 : 591-603.

COLLEGE NEWS

(J Bangladesh Coll Phys Surg 1994; 12: 71-74)

Continuing Medical Education:

13-01-94 Mr. Peter Hawley
Consultant Ano-Rectal Surgeon
149, Harley Street
London W1N 2DE, U.K.
delivered lecture on
"Ano-Rectal Fistulae".

Examination News :

Results of FCPS Part I, FCPS Part II and MCPS Examinations held in January, 1994 are given below :

512 candidates appeared in FCPS Part I Examination held in January, 1994 of which 52 candidates came out successful. Subjectwise results are as follows :

Subject	Number appeared in theory examination	Number qualified for viva-voce	Number Passed
Medicine	121	19	9
Surgery	120	26	10
Paediatrics	65	17	6
Obst. & Gynae	63	15	4
Ophthalmology	51	15	8
ENT Diseases	16	3	2
Psychiatry	9	3	1
Anaesthesiology	24	2	2
Radiology	8	0	0
Radiotherapy	4	1	0
Physical Medicine	11	6	4
Haematology	10	5	4
Biochemistry	2	1	0
Histopathology	2	2	2
Microbiology	6	3	0
Total	512	118	52

96 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
2	Dr. Shahrukh Ahmed	Dhaka Medical College	Medicine
4	Dr. Fatema Begum	Rangpur Medical College	Medicine
7	Dr. Md. Zakir Hossain	Dhaka Medical College	Medicine
9	Dr. Taimur A.K. Mahmud	Dhaka Medical College	Medicine
10	Dr. Syed Mohammad Arif	Dhaka Medical College	Medicine

Roll No.	Name	Graduated from	Speciality
11	Dr. Musaddiq Husain	Chittagong Medical College	Medicine
13	Dr. Mohammed Abdur Rashid	Chittagong Medical College	Medicine
14	Dr. Paritosh Kumar Roy	Mymensingh Medical College	Medicine
18	Dr. Md. Mohiuddin Ahmad	Dhaka Medical College	Medicine
25	Dr. Dewan Saifuddin Ahmed	Dhaka Medical College	Medicine
29	Dr. Md. Rafiqul Islam	Rajshahi Medical College	Medicine
31	Dr. A.H.M. Rowshon	Rajshahi Medical College	Medicine
38	Dr. Khondker Abul Kalam Azad	Chittagong Medical College	Surgery
39	Dr. Alok Kumar Sinha	Rangpur Medical College	Surgery
41	Dr. Abul Bashar Mohammad Khurshid Alam	Sir Salimullah Medical College	Surgery
46	Dr. S.M. Amjad Hossain	Rajshahi Medical College	Surgery
48	Dr. Md. Yusuf Ali	Rangpur Medical College	Surgery
50	Dr. Kamrun Nahar	Rajshahi Medical College	Surgery
52	Dr. Abdus Salam Muhammad Arif	Dhaka Medical College	Surgery
53	Dr. Md. Mustaque AhmedBarbhuiyan	Sylhet MAG Osmani Medical College	Surgery
54	Dr. Kazi Zaheed Ashraf	Dhaka Medical College	Surgery
55	Dr. Promode Ranjan Singh	Sylhet MAG Osmani Medical College	Surgery
56	Dr. Mohammad Emdadul Haque	Sir Salimullah Med. College	Surgery
65	Dr. Narayan Chandra Saha	Chittagong Medical College	Paediatrics
69	Dr. Suraiy Begum	Mymensingh Medical College	Obst.& Gynae
71	Dr. Hosne Ara Baby	Sir Salimullah Medical College	Obst. & Gynae
72	Dr. Mohammad Abdul Quayyum	Sylhet MAG Osmani Med. College	Obst. & Gyane
73	Dr. Monowara Begum	Rajshahi Medical College	Obst.& Gynae
74	Dr. Farhat Hussain	Dhaka Medical College	Obst. & Gynae
75	Dr. Laila Husna Banu	Rangpur Medical College	Obst. & Gynae
76	Dr. Karuna Rani Karmaker	Sir Salimullah Med. College	Obst. & Gynae
78	Dr. Mosammat Rashida Begum	Sher-e-Bangla Med. College	Obst & Gynae
80	Dr. Salma Rouf	Sher-e-Bangla Medical College	Obst. & Gynae
81	Dr. Md. Abdul Hye	MAG Osmani Medical College	Ophthalmology
82	Dr. Md. Mushahid Thakur	MAG Osmani Medical College	Ophthalmology
85	Dr. Md. Shahinur Rahman	Rajshahi Medical College	Ophthalmology
87	Dr. Mir Hasan Shaheel Mahmood	IPGMR	ENT Diseases
88	Dr. Debasish Banik	Chittagong Med. College	Anaesthesiology
89	Dr. Abdul Khaleque Beg	Sir Salimullah Med. College	Anaesthesiology
91	Dr. Debabrata Banik	Chittagong Med. College	Anaesthesiology
92	Dr. Md. Azharul Islam	Mymensingh Med. College	Anaesthesiology
95	Dr. Nighat Ara	Dhaka Medical College	Psychiatry
97	Dr. A.B.M. Yunus	V.S.S. Medical College, Calcutta	Haematology
98	Dr. Shazadi Nilufar	MAG Osmani Med. College	Biochemistry

161 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. Amirul Khusru	Medicine
5	Dr. Md. Abdur Rob Sarkar	Medicine
8	Dr. Satya Ranjan Sutradhar	Medicine
11	Dr. A.K.M. Fazlur Rahman	Medicine
14	Dr. A.S.M. Towhidul Alam	Medicine
26	Dr. Nirmalendu Roy	Surgery
30	Dr. A.K.M. Abul Hossain	Surgery
36	Dr. Nazneen Eshaque	Paediatrics
37	Dr. Md. Nurul Islam	Paediatrics
39	Dr. Md. Iqbal Hossain	Paediatrics
44	Dr. Sailah Yasmin Talukder	Paediatrics
52	Dr. Hasna Hena Pervin Beauty	Paediatrics
53	Dr. Mahbuba Akhter Banu	Obst. & Gynae
54	Dr. Md. Yusuf Hossain	Obst. & Gynae
55	Dr. Suraiya Begum	Obst. & Gynae
57	Dr. Hosne Ara Begum	Obst. & Gynae
58	Dr. Nasrin Begum	Obst. & Gynae
59	Dr. Akhter Jahan Ummey Yeasmina Begum	Obst. & Gynae
61	Dr. Samar Kumar Ghosh	Obst. & Gynae
62	Dr. Mst. Khaleda Akhtar	Obst. & Gynae
63	Dr. Ali Mahbuba Hasnat	Obst. & Gynae
64	Dr. Abdur Razzaque	Obst. & Gynae
66	Dr. Khandaker Farida Begum	Obst. & Gynae
68	Dr. Delowara Begum	Obst. & Gynae
69	Dr. Md. Shah Alam	Obst. & Gynae
70	Dr. Sk. Md. Mahbubul Haque	Obst. & Gynae
71	Dr. Ferdous Mahal	Obst. & Gynae
72	Dr. Tahira Salwa Jabber	Obst. & Gynae
74	Dr. Parul Jahan	Obst. & Gynae
75	Dr. Shamima Nasreen	Obst. & Gynae
76	Dr. Syeda Husna Akhter	Obst. & Gynae
79	Dr. Ferdousi Begum	Obst. & Gynae
83	Dr. Kazal Rekha Roy	Obst. & Gynae
86	Dr. Md. Nurul Hoque	Obst. & Gynae
89	Dr. Mursheda Akhter	Obst. & Gynae
90	Dr. Moquaddes Akhter Begum	Obst. & Gynae
94	Dr. Hasina Afroz	Obst. & Gynae
102	Dr. Kazi Suraiya Begum	Obst. & Gynae
104	Dr. Lailo Nahar	Obst. & Gynae
105	Dr. Md. Abdur Rashid	Ophthalmology

Roll No.	Name	Speciality
112	Dr. Nazneen Khan	Ophthalmology
120	Dr. Akhter Jahan	Anaesthesiology
124	Dr. Mohammad Farid Uddin	Psychiatry
128	Dr. Enamul Hoque Chowdhury	Radiology
132	Dr. Hazera Khatun	Cl. Pathology
137	Dr. Md. Abu Rayhan Khandakar	Cl. Pathology
141	Dr. A.K.M. Sobhan Moral	Dental Surgery
144	Dr. Q. M. Mahabub Ullah	Dermatology & Venereology
146	Dr. Md. Habibuzzaman Chowdhury	Dermatology & Venereology
148	Dr. Zahedul Karim Ahmad	Forensic Medicine
150	Dr. Md. Abdus Salam	Family Medicine
155	Dr. Md. Shahidur Rahman	Family Medicine
156	Dr. Md. Roushan Ali	Family Medicine
157	Dr. Md. Kashem Ali	Family Medicine
161	Dr. Motahar Ali Chowdhury	Family Medicine