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Acknowledgements

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An official organ of Ad-din Women's Medical College

Editorial

RIRS (Retrograde Intra Renal Surgery)

Urology is one of the rapidly growing subjects in medical science. With the development of anesthesiology as well as bioengineering technologies Urology and Cardiology are now become the most advanced subjects. Technologies for urological procedures developed jointly by medical personnel and engineers are appreciated in all over the world. We, as Urologists would like to acknowledge the contributions of engineers in the development of high-tech urological procedures.

Stone management in urinary system is a very challenging issue in our country like other countries in the globe. PCNL (per cutaneous nephrolithotripsy), ESWL (extra corporeal shock wave lithotripsy), URS+ICPL (uretero renoscopy + intracorporeal pneumatic lithotripsy), laparoscopic urological procedures, are some well known practicing procedures in our country. At present less than 10% kidney related stone problems need open surgical procedures. Ninety per cent of such problems can be managed with high-tech procedures. PCNL is the most practicing procedures at home and abroad for bigger sized kidney stones. In PCNL, puncture of skin and kidney cannot be avoided. The good news is no puncture of skin and kidney is required in newly developed RIRS procedure. Retrograde intrarenal surgery (RIRS) performed using a flexible ureterorenoscope marked the beginning of a new era in urology. The approach attracted a great deal of attention and it was suggested that larger stones could also be treated, albeit over longer operative times which is associated with fewer complications and less morbidity. And whatever the size of kidney stone it could be cleared through per urethral procedure. Technological progress has evolved retrograde intrarenal surgery (RIRS) into a safe and efficacious modality for the treatment of the upper urinary tract and has expanded its potential indications to intrarenal large stones (>25 mm), shock wave lithotripsy (SWL) failure, infundibular stenosis, morbid obesity, renoureteral malformations, musculoskeletal deformities, and bleeding. The development of flexible ureteroscopes and accessory instrumentation like guidewires, ureteral access sheaths, intracorporeal lithotriptors and stone retrieval baskets has facilitated RIRS and has given more safety to the procedure. Safety and efficacy of RIRS has also been confirmed in children. Thus, RIRS potentially may become first-line treatment for intrarenal stones.

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Original article

Serum ammonia level in the patient of cirrhosis with encephalopathy

Md. Ashraf-uz-zaman[1], Nasreen Sultana Lovely[2], Bilquis Ara Begum[3], Md.Masud Hasan[4]

Abstract

Objective : Cirrhosis of the liver is a common medical problem in our country. Hepatic encephalopathy is a frequent complication of cirrhosis which is a medical emergency and increases the rate of mortality.

Method : The present study was designed to detect a laboratory parameter which can early detect the encephalopathy and prompt treatment can be initiated to abort the complication. Ammonia level increases both in cirrhosis with encephalopathy and without encephalopathy. If a cut off value can be established which can differentiate these two clinical entities, the monitoring of the serum ammonia level can early detect the hepatic encephalopathy.

Results : Normal serum ammonia level is 15-33 µmol/L. In this study 30 cirrhotic patient without encephalopathy mean serum ammonia level was 44.13±31.46 and mean ammonia level in patient with encephalopathy was 83.34±34.85.

Conclusion : We can conclude that serum ammonia level is more increased in patient of cirrhosis with encephalopathy than without encephalopathy. The diagnosis of hepatic encephalopathy (HE) is based mostly on clinical criteria. It is important to assess the severity of HE quantitatively for both clinical practice and research: however, this remains a difficult and challenging problem. No single parameter or index has yet been shown to be infallible in assessing the severity of hepatic encephalopathy. Further study and research should be done in this field clinical medicine.

Key Words : Serum ammonia, cirrhosis, encephalopathy.

Introduction

Hepatic encephalopathy is characterized by neuropsychiatric manifestation ranging from slightly altered mental status to coma. This condition of chronic and acute liver disease is a result of failure of the liver to detoxify toxin originating in the intestine. The pathogenesis probably is multifactorial, although the predominant causative agent appears to be ammonia.[1] The major clinical difference from the 20th century is that, hepatic encephalopathy will be seen much less commonly because of the elimination of HBV, HDV and HCV infection. Similarly, the eradication of schistosomiasis will reduce the frequency of portal hypertension, limiting the extent of hepatic encephalopathy associated with it.[2] Continued use of the ethyl alcohol is one of the cause of cirrhosis in the civilized world.

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As the under developed countries become overdeveloped they too; will consume this society accepted hepatotoxin. Eventually, educational programmes akin to those which are now becoming effective for tobacco abuse may gradually become valuable in reducing alcohol abuse.

Ammonia may be a primary diagnostic parameter for portosystemic encephalopathy in the absence of most important diagnostic method (EEG and psychometric test). Moreover ammonia are of great diagnositic importance in patient with coma of unknown origin and can help in prompt management. Ammonia is produced from the breakdown of protein, amino acids, purine and pyrimidine. About half of the ammonia arising from the intestine is synthesized by bacteria, remainder coming from dietary protein and glutamine. This ammonia is detoxified in the liver by synthesis of glutamine in perivenous hepatocyte and through urea cycle in periportal hepatocyte. In cirrhosis, fibrosis follows hepatocellular necrosis. The cell death followed by nodules which disturb the hepatic architecture and full blown cirrhosis develops. As a result hepatocyte cannot perform their metabolic function properly and ammonia is not detoxified fully and serum ammonia level is

increased. When the portal circulation is obstructed, a remarkable collateral circulation and hepatic encephalopathy occurs.

Several studies had been done to establish the correlation of serum ammonia level with hepatic encephalopathy. Ong et al.[3] evaluated correlation between plasma ammonia level and severity of hepatic encephalopathy. Karmar et al.[4] have done a study on relation of ammonia with hepatic encephalopathy. Marsavljivic et al.[5] have showed a prospective study on 25 cirrhotic patients with relation to serum ammonia. Jessy et al.6 have done study in the department of physiology and biophysics university on metabolic change found after the portocaval shunts. Author suggested metabolic alteration occurred due to elevated amonia level.

Methodology

It is a cross sectional study carried out in General Medical Hospital, Elephant Road, Dhaka during the period of January 2014 to December 2015. Sixty cirrhotic patients with ascites was taken. The patients were divided into two groups.

A patient presenting with feature of chronic liver disease was asked for detail history and was examined for evidence of cirrhosis of liver. Presence of ascites was detected clinically on the basis of positive shifting dullness and /or positive fluid thrill examination. After taking informed consent from the patient, 5cc ascitic fluid was drawn for ascetic fluid albumin estimation. At the same time, venous blood sample was drawn to send to department of biochemistry for serum albumin estimation. After obtaining serum albumin level and ascetic fluid albumin level, serum ascitic albumin gradient was determined. Upper gastrointestinal endoscopies of the patients were done following topical anaesthesia in the department of hepatology by video endoscopies. The liver function test (serum bilirubin, SGPT) serum creatinine and chest X-ray were also done. With all aseptic precaution 3 ml venous blood was drawn from anticubital vein by using disposable syringe. Then blood was transfered into previous prepared clear and dry test tube containing, 25 ml EDTA. Plasma was separated within 15 minutes of venipuncture. Then plasma was analyzed immediately. The Vitros AMON Slide is a dry, multilayered, analytical element coated on a polyester support.

A 10µL drop of patient sample is deposited on the slide and is evently distributed by the spreading layer to the underlying layers. Water and nonproteinaceous components travel to the underlying buffered reagent layer, and the ammonium ions are converted to gaseous ammonia. The semipermeable membrane allows only ammonia to pass through and prevents buffer or hydroxyl ions from reaching the indicator layer. After a fixed incubation period, the reflection density of the dye is measured using the white background of the spreading layer as a diffuse reflector. All the data were entered into a personal computer, thoroughly checked for any possible error and then processed and analyzed by SPSS programme. Significance of the test was tested by unpaired 't' test and 'x2' test. 'P' value of <0.05 was taken as statistically significant.

Result

Table 1 : Demographic data of the study group (N=60)

Parameter		Case	Control
Sex	Male	28	25
	Female	2	5
	Total	30	30
Age	Mean	45.30	42.3

Table 2 : Serum ammonia level in study population

Parameter	Case (mean±SD)	Control (mean±SD)	p value
S. ammonia	88.34 ±34.85	44.31 ±13.46	<0.001

Table 3 : Other biochemical parameter of the study group

Parameter	Case (mean)	Control (mean)
S. Bilirubin in mmol/L	103.38	96.04
S.erum Albumin mg/dl	26.95	26.94
S. creatinine mg/dl	0.91	0.91
Prothrombin time in second	18.58	18.73
Serum ascitic albumin	1.84	1.7
gradient		

Table 4 : Serum level of ammonia in various grade of encephalopathy

Serum Ammonia	Mean±SD	P value
G-1	89.25±22.28	0.72
G-2	83.58±40.51	
G-3	97.09±36.64	
G-4	74.00±16.00	

Discussion

Hepatic encephalopathy is a neuropsychiatric syndrome occurring in patients with severe liver disesae and or portosystemic shunting. The exact pathogenesis of hepatic encephalopathy is yet not completely understood but there is a general consensus about the importance of gut derived nitrogenous substance escaping hepatic ditoxification and affecting the central nervous system function. Ammonia is one of these substances, produced both in colon and in the small bowel and extensively metabolized in the liver. In case of impaired liver function and or portal systemic shunt, blood ammonia concentration may increase and exerts its toxic activity on the brain. Although ammonia levels are almost always invariably higher in patient with acute and chronic liver failure, the correlation with severity of hepatic encephalopathy is often variable and inaccurate.

Karmar et al.[4] have done a study to test on serum ammonia level on 56 cirrhotic patient with hepatic encephalopathy. Observation was that clinical grading of hepatic encephalopathy correlated (P <0.001) with both total and partial pressure of total ammonia.

Ong et al.[3] evaluated that correlation between plasma ammonia level and the severity of hepatic encephalopathy. Diagnosis of the hepatic encephalopathty was based on clinical criteria and severity of hepatic encephalopathy was based on West Haven criteria of grading of mental state. Of 121 patients 25% had grade-0 ecenphalopathy, (22%) had grade I cncephalopathy, 19% had grade -II cncephalopathy, 23% had grade III cncephalopathy and 11% had grade IV encephalopathy.

Each of the 4 measures of ammonia increased with severity of hepatic encephalopathy. P value was (<.001). In this study, number of the total cirrhotic patients with encepphalopathy was 30. Of 30 patients, grade I were 4 in number (13%), grade II were 12 in number (42%), grade III were 11 in nuber (30%) and grade IV were 3 numbers (09%). In this study mean(±SD) of serum ammonia level in patient of grade I encephalopathy was 89.25±22.28, in patient of grade II encephalopathy was 83.58±40.51, in patient of grade III encephalopathy was 97.09±36.64 and in patient grade IV encephalopathy was 7.00±16. There was no significant difference in serum ammonia between these 4 grade of encephalopathy.

Nolte et al.[7] have done a study on hepatic encephalopathy and determination of arterial ammonia level was performed in 55 cirrhotic patients treated consecutively by transjugular intraheptic porto systemic shunt. Arterial ammonia increased from a mean of 94±26 µg/dl to 140±28 µg/dl at 3 months after TIPS (P<0.001). In this study mean serum ammonia level without encephalopathy was 44.31±13.46 in cirrhotic patient and mean serum ammonia level in patient with encephalopathy was 88.34±34.85. Serum ammonia level was significantly higher in patient with encephalopathy group (P< 0.001) than patient without encephaloapathy.

Marisavjivic et al.[5] showed one year prospective study on 25 cirrhotic patients with portal systemic encephaloapthy admitted to emergency care centre in Belgrade to investigate the significance of clinical biochemical, electroencephalographic parameter and blood ammonia in the diagnosis, differential diagnosis and prognosis of PSE. Fifteen cirrhotic patients without PSE constituted the control group.

Ammonia levels correlated with the severity of protosystemic encphalopathy (P<0.05) but not with other biochemical parameter (Prothombine time albumin and creatinine). In this study the mean (±SD) level of serum bilirubin. albumin, creatinine, prothombin time and SAAG in the patient with hepatic encephalopathy were 103.38±88.67, 26.29± 8.05, 0.91±0.30, 88.34±34.85 18.58±5.44, 1.84±0.67 respectively. The mean (±SD) of those serum bilirubin, albumin, creatinine, prothombin time and SAAG were 96.04±115.41, 26.94 ±8.26, 0.91±.33, 18.73±4.66 and 1.7±0.55, in cirrhotic patient without encephalopathy respectively. The present study had not showed any, significant difference of serum bilirubin, albumin, prothombin time, SAAG and creatinine in between two groups (P>0.05). But in this study the serum ammonia (mean±SD) were 88.34±34.85 in study group and 44.31±13.46 in control group. The patient with hepatic encephalopathy had higher level of serum ammonia than the patient without encephalopathy. The difference in the means of serum ammonia between study group and control was statistically significant (P<0.001). Nicolao et al.[8] studied to compare the venous, arterial and partial pressure of amonia in 27 consecutive cirrhotic patients with hepatic encephalopathy and 15 cirrhotic patients without encephalopathy. In patients with encephalopathy, ammonia was higher than in patient without encephalopathy. Mean serum ammonia levels was 39.42±44.7 in patient without encephalopathy which was significantly lower than the patient with encephalopathy. In this study, mean serum ammonia level in cirrhotic patient with encephalopathy was 88.34±34.85 and in patient without encephalopathy was

44.31 ± 13.46. Value was higher and statistically significant in patient of cirrhosis with encephalopathy (P<0.001).

Conclusion

We can conclude that serum ammonia level is more increased in patient of cirrhosis with encephalopathy than without encephalopathy. The diagnosis of hepatic encephalopathy (HE) is based mostly on clinical criteria. It is important to assess the severity of HE quantitatively for both clinical practice and research: however, this remains a difficult and challenging problem. No single parameter or index has yet been shown to be infallible in assessing the severity of hepatic encephalopathy. Further study and research should be done in this field clinical medicine.

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Original article

Clinicopathological evaluation of breast lump- a prospective study in tertiary level hospital.

Sadia Armin Khan[1], Muzibar Rahman[2]

Abstract

Objective : To find out the magnitude of breast diseases, its frequency and distribution in different age group among the female patients attending surgical out patients department and admit different units of surgery departments in Dhaka Medical College Hospital and to study the correlation between clinical and histopathological feature in palpable breast lumps .

Methods : This cross sectional descriptive study comprising of 102 cases was done between 01-01-2009 to 31-03-2010 in Dhaka Medical College Hospital. The study was conducted among female patients. Every patient underwent 'a FNAC done on OPD basis, following a thorough clinical check-up. Every patient subjected to FNAC underwent a definitive surgical procedure. All specimens that were obtained sent for histopathology. The results thus obtained from histopathology were matched with clinical feature and a correlation was sought based on statistical tests.

Results : Results of all patients were collected and tabulated. Statistical analysis was performed on the tabulated data. Of all 102 patients selected, 50 cases were duct cell carcinoma (49%),38 cases were firoadenoma (37.3%),9 cases were fibrocystic disease (8.8%) and other benign lesions were 5 cases (4.9%). In this study, carcinoma was common in 3rd, 4th and 5th decade (42.16%), fibroadenoma was common in 2nd and 3rd decade and fibrocystic disease was common in perimenopausal age group. Out of 102 cases, 8 cases were benign on FNAC, but on histopathology these were malignant.

Conclusion : The purpose of this study was to analyze breast lesions causing breast lump and evaluate these cases by histopathology. In this study, most of the cases correlate presenting feature with histopathological reports. In some cases false positive occurs. So all the palpable breast lump must be histopathologically evaluated.

Key words : Breast lump, clinico pathological evaluation

Introduction

A breast lump whether benign or malignant is a cause of anxiety to the patient & her family members. Due to limitation of implementation of early diagnosis of breast cancer by mass screening programme, more than 2/3 of the cancers are already in advanced incurable stage at the time of histopathological diagnosis. This emphasize the requirement of early detection of suspicion of cancer before it is evident clinically by inspection / palpation or by other means[1]. About 5-55%[2] of all women suffer from breast disorders in their life time. Benign disorders of breast is usually seen in the reproductive period of life, is thought to be largely hormone induced and there is a dramatic fall in the incidence, after menopause due to cessation of clinical ovarian stimulation. Benign breast disease is 4-5 times more common than breast cancer[2].

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The concept of ANDI-Aberrations of normal development and Involution is gaining acceptance. Benign proliferations of the breast are often considered as aberrations of normal development and involution. The cyclical changes due to variations in estrogens and progesterone result in increased mitosis around days 22-24 of the menstrual cycle but apoptosis restores the balance across the cycle[3].

ANDI, first proposed by Huges[4] is now universally accepted. So most benign breast diseases are relatively minor aberrations of normal process of development, cyclical hormonal response and involution that interact throughout a woman's life.

In women, the breast Cancer is one of the most common cancer. In developing country, Cancer of cervix is the most common Cancer but the breast Cancer is almost as common & both account for 60% of all Cancer & make the second most common cause of Cancer death of women[1]. It is found that the breast Cancer is on rise in major & metropolitan cities of the world. This appears to be related to late marriage, birth of child in the later age, fewer children and shorter period of breast feeding, which are common practice in Urban Women[1].

Every year in Bangladesh approximately 35,000[5] women develop breast cancer, many of whom never seek treatment. Although the majority of breast lumps ("chakas") are not cancerous and require minimal treatment, some breast lumps require immediate attention.[5]

In this study, we have presented the common types of breast disease, presenting as breast lump in our country. The distribution of different types of breast diseases in different age groups was also studied. Attempts have also been made to correlate the clinical diagnosis with the result of histopathology to evaluate diagnostic accuracy among these cases.

Materials and methods

This cross sectional descriptive study was carried out in different units of the Department of Surgery, and Out patients department of surgery in Dhaka Medical College Hospital.

The study period spanned from January 2009 to March 2010. A total 102 cases who presented with clinically benign or suspicious discrete breast lump in which malignancy couldn't be ruled out and had to undergo FNAC & excision biopsy.

The database form of the study was filled up by interview method and clinical findings and pathological reports were recorded in database form.

Inclusion criteria:

All female patient presenting with palpable breast lump admitted into Dhaka Medical College Hospital and visit SOPD Dhaka Medical College Hospital.

Exclusion criteria: Patients with following criteria were excluded from the study:

1) Patients of pediatric age group (less than 14 years)

2) Patients with breast abscess.

3) Ill-defined or doubtful lump

Detailed history of each patient under study was recorded, with special attention to their age, parity, age of menarche, age of menopause, lactational history, history of breast cancer, history of ovarian cancer. Important and relevant findings on through physical examination of lump which included consistency, temperature, tenderness, fixity of the lump to the skin and underlying structure, draining lymph nodes etc. also be recorded.

In all cases every attempt were made to reach a definitive clinical impression and relevant investigations had been done. Other routine investigations were done and necessary advice were given for follow-up in cases where it was actually needed.

Results

The study includes all female patient presenting with palpable breast lump admitted into different units in Dhaka Medical College Hospital and visited SOPD of Dhaka Medical College Hospital. Every patient underwent a FNAC done on OPD basis following a thorough clinical check-up. Every patient subjected to FNAC underwent a definitive surgical procedure. All specimens that were obtained sent for histopathology.

Common types of breast diseases among presenting breast lumps are duct cell carcinoma 49%, fibroadenoma 37.3%, fibrocystic disease 8.8%, duct ectasia 1.96%, tubercular lesion .98%,phyllodes tumor.98% and medullary carcinoma .98%.surgical procedure (Fig: 1.1). All specimens that were obtained sent for histopathology.

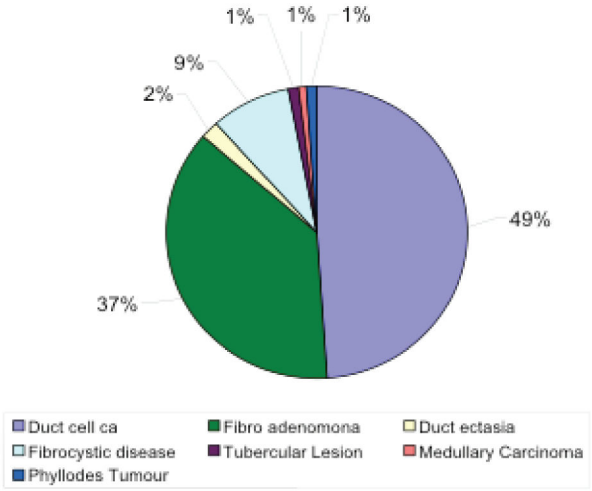

Fig:1.1 : Common types of breast diseases among presenting breast lumps

Age range varied from 15 years to 80 years. In this study, carcinoma was common in 3rd, 4th and 5th decade (43%), fibroadenoma was common in 2nd and 3rd decade, and fibrocystic disease was common in perimenopausal age group. In this study, breast carcinoma was the most common of all lesions (Table 1.1).

Table 1.1 : Age incidence of breast lumps

Breast lumps		Age Group			Total
		15-30 years	31-45 years	>45 years	
Duct cell ca	Count	7 7	24	19	50
	% within Histopathology	14.0%	48.0%	38.0%	100.0%
Fibro adenomona	Count	35	3	0	38
	% within Histopathology	92.1%	7.9%	.0%	100.0%
Duct ectasia	Count	0	2	0	2
	% within Histopathology	.0%	100.0%	.0%	100.0%
Fibrocystic disease	Count	0	5	4	9
	% within Histopathology	0	55.56%	44.44%	100.0%
Tubercular Lesion	Count	0	0	1	1
	% within Histopathology	.0%	.0%	100.0%	100.0%
Medullary Carcinoma	Count	0	0	1	1
	% within Histopathology	.0%	.0%	100.0%	100.0%
Phyllodes Tumour	Count	0	0	1	1
	% within Histopathology	.0%	.0%	100.0%	100.0%
Total					
	Count	48	31	23	102
	% within Histopathology	47.1%	30.4%	22.5%	100.0%

Besides breast lumps, 14.71% patients present with pain, 8.82% patients present with nipple discharge, 2.94% patients present with ulceration of the skin, 1.96% patients present with fever, 24.51% patients present with lymph node involvement.

Out of 102 cases, the onset of menarche of 84 cases (82.4%) were between 12-14 years of age. The onset of menarche of 17 cases(16.7%) were in between 9-11years of age and 1 case (1%) started menstruation after 15 years. 62.7% belongs to average, 27.5% belongs to poor and 9.8% belongs to standard socio-economic group. In this series, 24 cases (23.5%) were obese and rest of them (76.5%) were not obese. 55.88% took different methods of contraceptives. 75.5% patients adequately breastfeed her baby. 6.9% cases had positive family history. 11.8% patients gave history of previous breast disease. considering all lesions of right breast was involved in 58.82% cases and left breast was involved in 35.29%cases. Only 5.8% cases were involved in both breasts. All types of lesions were common in right breast (table 1.2) .

Table 1.2 : Distribution of breast lumps according to the side (right or left) of the breast

Types of lesions	All lesions (n=102)		Right breast (n=60)		Left breast (n=36)		Both breast (n=6)	
	No.	(%)	No.	(%)	No.	(%)	No.	(%)
Fibroadenoma	38	37.25	20	52.62	14	36.84	4	10.52
Fibrodenosis	9	8.8	5	56.55	4	44.44	0	0.00
Other benign breast lump	5	4.7	3	60.00	0	0.00	2	40.00
Breast carcinoma	50	49.00	32	64.00	18	36.00	0	0.00

Only 8.8% patients in this series presented with nipple discharge.

The predominance of duct cell carcinoma (49%). Next common lesion was fibroadenoma (37.3%). Fibrocystic diseasae was in 9 cases (8.8%). Duct ectasia were present in 2 cases (1.96%). There were one case of tubercular lesion, one case of phyllodes tumor and one case of medullary carcinoma (table 1.3).

Table 1.3 : Histologic distribution of breast lumps (n=102)

Histological type	Frequency	Percent
Duct cell ca	50	49.0
Fibro adenomona	38	37.3
Duct ectasia	2	1.96
Fibrocystic disease	9	8.8
Tubercular Lesion	1	.98
Medullary Carcinoma	1	.98
Phyllodes Tumour	1	.98
Total	102	100.0

Table 1.4 shows that 45.1% of cases were clinically diagnosed as fibroadenoma and 41.2% of cases were clinically diagnosed as duct cell carcinoma but according to histopathological findings, most common lesions were duct cell carcinoma (49.01%) and 2nd most common findings were fibroadenoma (37.25%). 8.82%cases were fibrocystic disease, according to histopathlogical study. One case clinically was galactocele, but according to histopath it was duct cell carcinoma. Clinically fibrocystic changes found in eight cases, among them histopathologically, four cases were duct cell carcinoma, duct ectasia found in one case, two cases showed fibrocystic disease and one was tubercular lesion. Five cases (4.9%) were clinically undiagnosed, but according to histopathological report, two cases were duct cell carcinoma, One was fibroadenoma, one was duct ectasia and the rests are fibrocystic disease.

Table 1.4 : Correlation of clinical and histopathological diagnosis of breast lump

Clinical diagnosis		Histopathology							
		Duct cell Ca	Fibro adenoma	Duct ectasia	Fibrocystic disease	Tubercular lesion	Medullary carcinoma	Phyllodes tumour	Total
Duct cell carcinoma	Count	41	0	0	0	0	1	0	42
	% within histopathology	82	0	0	0	0	100	0	41.2
Fibro adenoma	Count	2	37	0	6	0	0	1	46
	% within histopathology	4	97.4	0	66.7	0	0	100	45.1
Galactocele	Count	1	0	0	0	0	0	0	1
	% within histopathology	2	0	0	0	0	0	0	1
Fibrocystic disease	Count	4	0	1	2	1	0	0	8
	% within histopathology	8	0	50	22.2	100	0	0	7.8
Undiagnosed	Count	2	1	1	1	0	0	0	5
	% within histopathology	4	2.6	50	11.1	0	0	0	4.9
Total	Count	50	38	2	9	1	1	1	102
	% within histopathology	100	100	100	100	100	100	100	100

Discussion

A lump in the breast is a common complaint presenting in the surgical out-patient department of all major hospitals, with anxiety regarding a possible malignancy being extremely common. Hence a quick diagnosis of a lump in the breast is essential. Criteria such as cost effectiveness, use of anaesthesia, time between the diagnostic procedure and report, patients hospital stay and most importantly, reliability in deciding subsequent treatment, are all factors to be taken into account in this regard[6]. Although benign breast lumps are about six times more than malignant tumors and the presence of any persistent lump in the breast raises the question of carcinoma, which is the most common malignant tumor of the breast leading cause of death in women[7].

In the present study of 102 patients, 52 (50.98%) lesions were benign. Rest 50 (49.03%) lesions were malignant. Duct cell carcinoma was the most common lesion accounting for 49% (50 lesions) of all and fibroadenoma was the second most common lesion accounting for 37.3% (38 lesions). The third most common lesion was fibrocystic disease comprising 8.8% (9 lesions) and other benign lesions were found in 5 cases (4.9%). The findings in this study correlate with the series of Ahmed Asif (2005, Dissertation, BCPS)[8] where 50% lesions were breast carcinoma, fibroadenoma was 46.55%, fibroadenosis was 3% and other benign lesions were found in 1% cases. A prospective case series of 205 women presenting with breast disease and undergoing treatment at Surgical Unit IV, Jinnah Hospital, Lahore[9], was conducted from January, 1999 to December, 2003 and showed that 30.73% of the patients had benign breast disease whereas 69.26% were diagnosed as having breast cancer. The commonest benign disease was fibroadenoma 60.3%). This result also correlates with this series.

Three most common lumps producing lesions in the breast are fibroadenoma, fibrocystic disease (fibroadenosis) and carcinoma. The relative incidence of these three lesions varies in different studies. Oluwole and Freeman[7] analyzed 282 patients with breast lesions and found fibroadenoma was the most common (34.75%) lesion and second and third most common lesions were carcinoma and fibrocystic disease comprising 28% and 17% respectively. Khan et al.[10] found that among 264 cases of breast diseases, benign breast diseases (BBD) were the commonest lesions of the breast found in this study (93.2%) whereas malignant lesion was infrequent (6.8%). Among benign breast diseases, the commonest lesion was fibroadenoma (32.57%). Thus, findings of this study does not correlate well with those studies as because the hospital admitted patients were also taken in this study as well as surgery outdoor patients. The number of cases taken in this study, was also very small. The percentage of fibrocystic disease in this series was low (8.8%). It is difficult to draw a conclusion because most of the patients with fibrocystic disease do not come to the hospital before appearing a definitive lump in the breast.

Multiple lumps in one or both breasts were found in 6 (5.88%) patients. In 4 (10.52%) cases, fibroadenoma was associated with other types of benign breast lesions. Oluwole and Freeman[7] in their study reported that incidence of multiple lesions was 15% of all benign lesions and fibroadenoma was the most common lesion, occurring with other types breast lesions in the same patient. Nigro and Organ[11] also in their study found that in 10% of cases multiple fibroadenomas were present in one of both breast. All these data unanimous with present study result.

Fibroadenoma was found common in between 15-30 years of age (92.1%). In between 30-45 years frequency was 7.9%. The peak age incidence this series was between 15-30 years of age. Oluwole and Freeman[7] have observed same type of age distribution. They found that peak age incidence was between 16 to 25 years for fibroadenoma. In the study of Khan and Kapoor[10], fibroadenoma was common in second and third decade. All studies showsthat fibroadenoma occurs at earlier age. Present study findings correlates with above studies.

In this study fibrocystic disease was common in 31-45 years age group(55.56%). This observation correlates with the series of Oluwole and Freeman[7]. Peak age incidence for fibrocystic disease was 40-50 years Surgical literature also shows that fibrocystic diseases is the most common between the age of 30 and 50 years. This mild disparity may be due to the small number of patients in this series. In this study breast carcinoma was common in 31-45 years age group (48%), that was not correlate with the series of Khan and Kapoor[10], where it was common in 50-59 years of age(55.5%). In the study of Mushahida et al[9], mean age of the cancer patients was 34.56 years ± 11.5 years, that is more or less similar to this study.

Association of increased risk of breast diseases with early menarche has been reported in many studies. In the present series, out of 102 cases the onset of menarche of 84 cases were between 12-14 years of age which is 82.4% of total cases. The onset of 17 cases (16.7%) were between[9-11] years of age and 1 case started menstruation after 15 years. Age at menarche has been inversely associated with the risk of breast cancer; menarche at a relatively early age is associated with increased risk. Because there is, prolonged exposure to estrogen in early menarche and at higher levels than for those with later menarche[10]. In the study, of Parazinni et al[12], early age at menarche was associated with an increased risk of benign

breast disease, but no definite conclusion can be made on the relationship between the risk of benign breast diseases and the age of menarche. In this study of breast lumps, 55.9% cases used contraceptives. Oluwole and Freeman7 found that high incidence of breast lesions among the users. Present study findings correlates with this findings. Mushahida et al[9] found that risk of carcinoma among oral contraceptive users is 14.08%, that is unanimous to present study.

In this series, 6.9% cases had positive family history of breast disease. 3(6%) patients gave positive family history of breast carcinoma. Oluwole and Freeman[7] also reported similar observation. The risk factor profile for positive family history of breast cancer in first degree relatives in 11.97% of the patients with breast cancer, in Mushahida et al[9] series. Penelop et al[13] found that women with a family history of breast cancer appear to be at increased risk of being diagnosed with BBD, in particular the high-risk types of BBD associated with a greatly increased risk of breast cancer

In this study, 11.8% cases gave previous history of breast disease. 4 cases (8%) of breast carcinoma previously operated as fibrocystic disease. Penelop et al[13] found that women with a history of benign breast disease (BBD) are at increased risk of developing breast cancer.

Lump was the presenting feature of all the cases in this series. Lumps were mild to moderately painful in 14.7% cases. Most of the painful lumps were fibrocystic disease and related with cycle. Nipple discharge was present only in 8.8% (9 cases) of patients, where most of the patient present with blood stained discharge. Occasional fever was complained by 1.96% cases. Which may be due to inflammatory condition of the breast. 2.5% of patients presented with ulceration of skin, all are diagnosed as breast carcinoma according to the histopathological report. 24.5% patients presented with lymph node involvement. Oluwole and Freeman[7] who analyzed breast lesions found that 95% of their patients presented with breast lump, 5% with nipple discharge and 5% with pain. These findings were more or less similar to this study.

Oluwole and Freeman[7] reported that, of all lesions of their series, right breast was involved in 45% of the patients, left breast in 41% and both breast in 14% and multiple lesions were in 15% of cases. Whereas, of the fibroadenoma cases, right breast was involved in 45% of patients, left breast in 42% and both breast in 13% and multiple lesions were present in 10% cases. Findings of this study was not correlate with Oluwole and Freeman[7]. It is found that intramammmary distribution of benign breast lesions are similar to the malignant lesions.

Out of 102 cases, 45.1% of cases were clinically diagnosed as fibroadenoma but according to the histopathological findings, most common lesions were duct cell carcinoma (49%) and second most common findings were fibroadenoma (37.3%). Eight cases (7.8%) were clinically diagnosed as fibrocystic disease but according to the histopathological report, duct cell carcinoma was (8%). 5 cases (4.9%) had no confirm clinical diagnosis, according to the histopathological report, two cases(4%) were duct cell carcinoma, one case(2.6%) was fibroadenoma, one was firocystic changes (11.11%) and the rest was duct ectasia. Clinically one case was galactocele (1.00%), but histopathologically it was duct cell carcinoma (2.00%).

Same picture was noted in series of Ahmed Asif (2005, Dissertation, BCPS)8 where out of 100 cases, 58% of cases were clinically diagnosed as fibroadenoma but according to the histopathological findings, most common lesions were duct cell carcinoma (50%) and the second most common findings were fibroadenoma (46.55%).

From the study, it is concluded that benign lesions of the breast are more common and occur usually during second and third decade of women's life. Malignant lesions of the breast are the second common pattern and occurring during 4th and 5th decade of women' life. Though FNAC is also routinely carried out to diagnose various lesions of breast, histopathological method is finally diagnostic.

In this study, most breast lumps are benign, as in fibroadenoma, a condition that affects mostly women under age 30. Fibrocystic breast disease is present in over 8.8% of all women. The cysts in FBD change in size with the menstrual cycle, whereas a lump from fibroadenoma does not. Fibroadenoma occurs mostly in nulliparous adolescents, whereas fibrocystic disease is found mostly in middle-aged multiparous women.

Breast carcinoma occurs commonly in older age group patients. It is the common malignant breast tumor, leading cause of death in women and its incidence was high in this series. This high incidence reflects the illiteracy, poverty, lack of awareness, lack of medical facilities and screening procedure of our people. Most of the cases of breast lump present with advanced stage when a surgeon is of little help for them. Tubercular lesion, traumatic fat necrosis, phyllodes tumor, duct ectasia are uncommon in this study. From this study and available data from different publications, it is evident that, bilateral involvement, multiple lesions and recurrence in the same patients are not uncommon. It is important that intramammary distribution of benign lesions is similar to that of malignant lesions of the breast.

FNAC of breast is highly accurate and has low false positive and false negative diagnosis. With the result of FNAC, patient can be advised for further treatment. Histological evaluation of the suspicious cases in FNA was

done and was based on excisional biopsy or mastectomy specimen. As the histopathology is 100% sensitive and specific tool available in all the major towns in our country.and as at present early detection and early removal of tumour is only method of curing breast cancer, mass education regarding mass screening by self breast examination, and by other tools i.e., mammography, are the mandatory measures to reduce the morbidity and mortality associated with breast cancer.

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Original article

Sexual dimorphism of fourth digital (4D) length among the adult population of Bangladesh

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Abstract

Objective : Digital lengths of human hand is one of the field of interest in modern science as by virtue of evolutionary changes digital lengths vary from person to person according to age, sex, races, occupation or even environmental influences. It has been found that the digital lengths are not even same in both hands of an individual. In this study, the morphological variations of the ring finger (4D) length has been analyzed and compared among the adult population of Bangladesh.

Materials & Methods : A Cross sectional analytical study was conducted in the department of Anatomy, Dhaka Medical College, Dhaka, from July 2012 to June 2013. The study was performed on 200 MBBS students (≥18 years of age) of Dhaka Medical College, Dhaka. Among them 100 were male and 100 were female. With the help of digital vernier caliper measurements of digital lengths were recorded. Paired and unpaired t tests were done for statistical analysis of the results.

Results : In the present study, the average ring (4D) finger lengths of male were greater than female in both hands (P<0.001). In both sex, the length of right ring finger (R4D) was significantly greater (P<0.001) than left ring finger (L4D).

Conclusion : Digital lengths especially index (2D) and ring (4D) digit lengths are often use to determine sexual dimorphism. Also, study over the variations of digital lengths have great medicolegal importance to determine age, sex and race of an individual.

Keywords : Ring finger length (4D), right ring finger length (R4D), left ring finger length (L4D)

Introduction

Evolutionary history says that any measurements of body parts can change with the alterations in size of the organs involved or general body size and this concept was 1st defined by Levinton[1].Throughout the following decades, one such study has been a marked increase in interest, that is measurements of digital length and its sexual variations. The ring finger located between thumb and middle finger is the second digit (4D) which are usually the most dexterous and sensitive fingers of a human hand[2]. Researchers claimed that the relative lengths of digits are set before birth[3] and interestingly in human hands, the relative lengths of the ring finger differs between male and female[4].

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More recently, the researchers explored the relationship between the index to ring digit ratio (2D: 4D) and a wide variety of human characteristics as 2D:4D ratios appear to correlate with a variety of sex-dependent behavior as stated by Manning J.T. & Fink B.[5] including personality traits like dominance, aggression, reproductive success and sexual performance, sexual orientation, hand preference, verbal skills, physical and mental health and diseases and musical and sporting talents. These associations appear to be often stronger for the right hand[6]. In the study of Manning, it is seen that smaller ring fingers in men have been associated with higher levels of physical aggression throughout their life[7]. This association was only true for physical aggression, not verbal aggression or other types of hostile behaviors[8]. Men with less smaller ring finger are reported as being more masculine and dominant in nature and tend to perform better in a number of physical activities[9]. In human, number of physical and behavioral traits depends on ring finger length (4D) in both sexes which were statistically proven. For example, males with smaller ring finger are more fertile and have high life time reproductive success. Also, they are more aggressive and

assertive in nature and have high musical and sports aptitudes[10] Again, male with excessive smaller ring finger often has some attributes like left-handedness, good visuo-spatial ability[11], fast running speed[12] but they may also experience some severe health related problems like autism, Asperger's syndrome, prostatic carcinoma, Hepatitis-B related hepatocellular carcinoma, urolithiasis and rheumatoid arthritis but male having longer ring finger often has higher risk of early heart disease. On the other hand, females with long ring finger are more fertile, have high reproductive success but also having higher risk of breast cancer and endometrial cancer. Again, female with an excess long ring finger are associated with good verbal fluency but higher risk to have neurodegenerative disorders but, females with smaller ring finger have greater tendency towards homosexuality/ bisexuality, spontaneous abortion, polycystic ovaries and also they are more aggressive and assertive in nature[10].

Materials & Methods

The study was performed on two hundred (200) medical students of Dhaka Medical College, Dhaka age ranging from 20-25 years. Out of them one hundred (100) were male and one hundred (100) were female. With the help of a digital vernier caliper ring finger length (4D) was recorded in millimeters. Length of the ring finger length was measured by measuring the crease-tip (c-t) length where "c" is the midpoint of proximal crease at the base and "t" is extreme end (tip) of the ring finger that is furthest from the hand. The distance between these two points indicates the length of ring finger (4D). Measurements were taken three times independently and the maximum length was taken for analysis. The right ring finger length was termed as R4D and left ring finger length was termed as L4D. Data was expressed as mean ± Standard deviation (±SD) as descriptive statistics in both sexes. Paired and unpaired Student's t-test was done to analyze the differences between lengths of right (R4D) and left (L4D) ring finger and those between male and female respectively. Statistical significance was accepted at ($P < 0.05$).

Result and observations

Right ring finger length (R4D) of male and female was ranged from 6.681-8.683 cm and 6.014-8.074 cm respectively and their mean (± SD) was 7.513± 0.397 cm and 6.828± 0.390 cm respectively. The length of right ring finger (R4D) was significantly larger in male than in female ($P<0.001$). Left ring finger length (L4D) of male and female was ranged from 6.606-8.583 cm and 5.896-8.032 cm respectively and the mean (± SD) of left ring finger length (L4D) was 7.463± 0.419 cm and 6.765± 0.388 cm respectively. Again the length of left ring finger (L4D) was more in male than in female ($P<0.001$). In both sex, right ring finger length (R4D) was higher than left ring finger length (L4D) which was statistically significant ($P<0.001$).

Table 1 : Comparison of ring finger length between male and female

Variable	**Male** (n = 100) mean ± SD	**Female** (n = 100) mean ± SD	***P-value***
R4D	7.513 ± 0.397 (6.681- 8.683)	6.828 ± 0.390 (6.014 - 8.074)	P < 0.001***
L4D	7.463 ± 0.419 (6.606 - 8.583)	6.765 ± 0.388 (5.896 - 8.032)	P < 0.001***

Figures in parentheses indicate range. Comparison of ring finger length between male and female is done by unpaired Student's 't' test, *** = significant at P <0.001

Table 2 : Comparison between right (R4D) and left (L4D) ring finger

Variable	**Groups**	
	Male (n = 100) mean ± SD	Female (n = 100) mean ± SD
R4D	7.513 ± 0.397 (6.681- 8.683)	6.828 ± 0.390 (6.014 - 8.074)
L4D	7.463 ± 0.419 (6.606 - 8.583)	6.765 ± 0.388 (5.896 - 8.032)
P - value	P<0.001***	P< 0.001***

Figures in parentheses indicate range. Comparison between right and left hand in both sex was done by paired Student's 't' test,

*** = significant at P<0.001

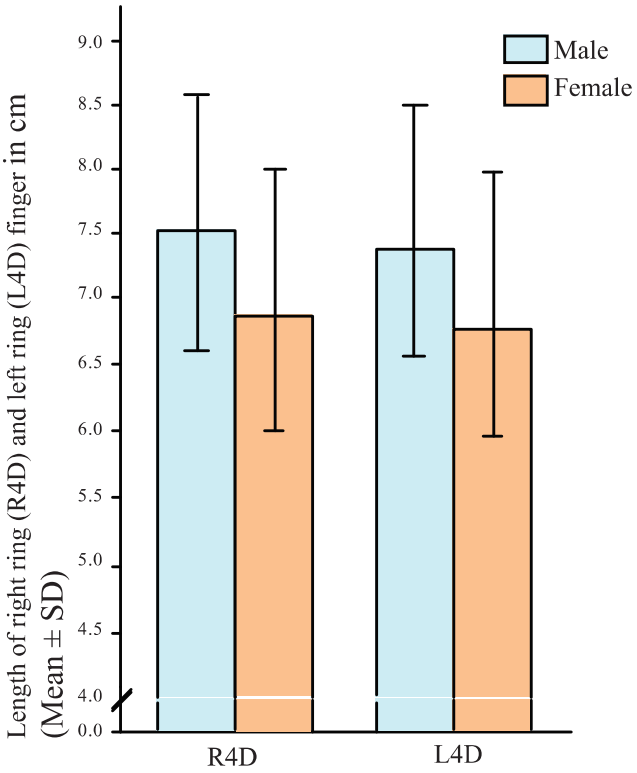

Fig. 1: Comparison of ring finger length between male and female

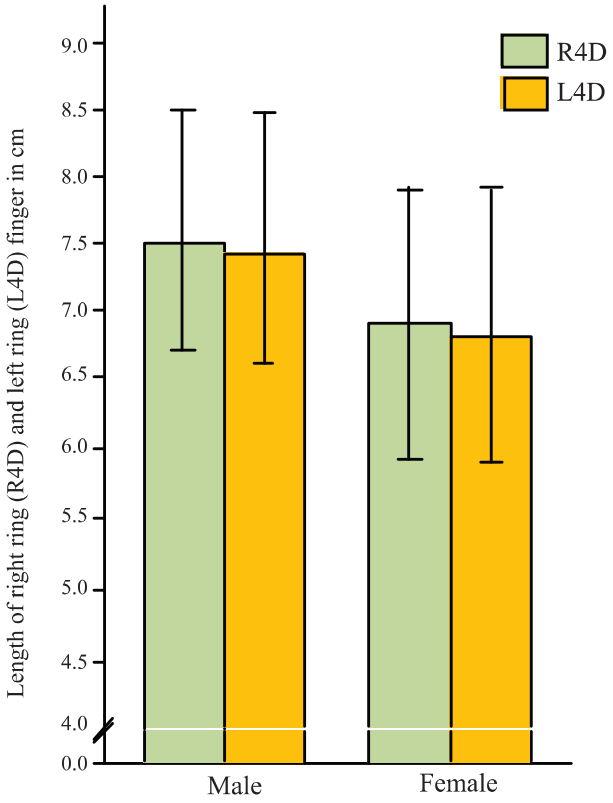

Fig. 2: Comparison between the length of right (R4D) and left (L4D) ring finger

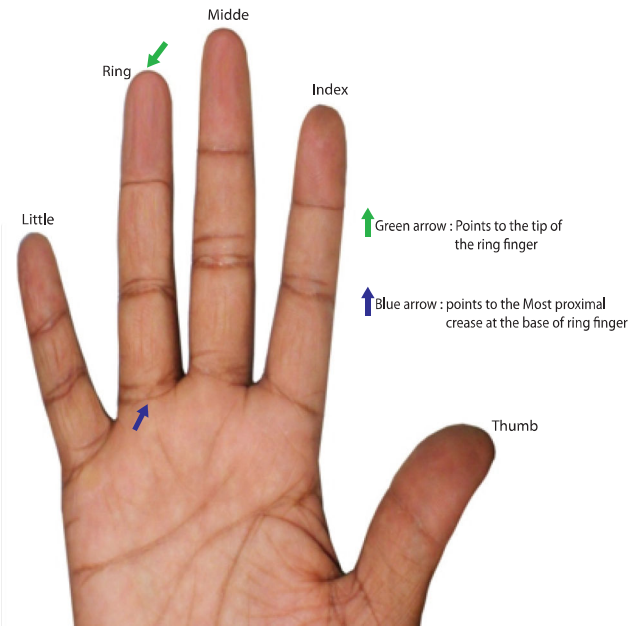

Fig. 3: landmarks for measuring ring finger length

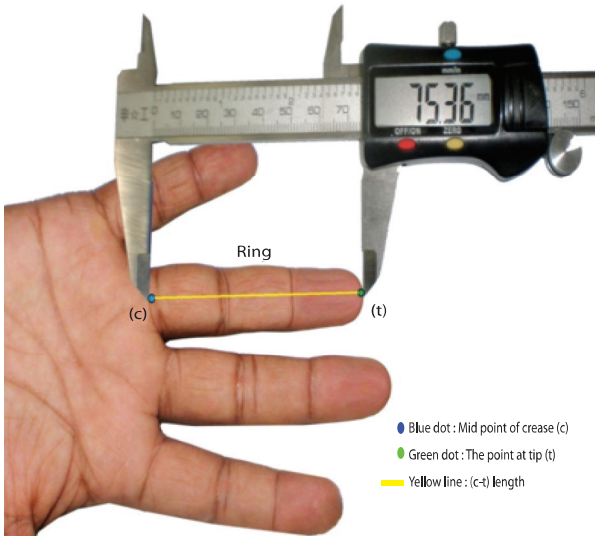

Fig.4 : Measurement of the full breadth of the proximal crease of the ring finger

Discussion

Male have longer ring finger (4D) length than female in both hands which was significant (P<0.001). Also, the length of right ring finger (R4D) was significantly higher than left ring finger (L4D) in both sex (P<0.001). Similar kind of study was conducted by William et al.[13], Lippa, R.A.[14], Rahman Q.[15], Wilson GD.[16], KOSÏF R. and Diramali M. B.[17], Danborno et al.[18] and Ibegbu A.O. et al.[19] where in both sex, the length of right (R2D) and left (L2D) index finger were significantly higher than that of present study (P<0.001) but in the study of Shima M. A. et al.20 the Mean length of both ring fingers (R2D and L2D) of their study were significantly lower than that of present study in both sexes (P<0.001).

Conclusion

Present study showed that Bangladeshi male have larger ring finger length than that of female (P<0.001) in both hands. Again, in both sex, the length of right ring (R4D) finger is larger than left ring (L4D) finger (P<0.001).

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Original article

Serum Thyroid Hormone Levels in Male Autistic Children

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Abstract

Objective : Thyroid hormone plays a key role in the development and physiological functioning of central nervous system. This study was aimed to assess the thyroid status in male children with autism spectrum disorders.

Methods : This analytical type of cross-sectional study was conducted in the Dept. of Physiology, DMC, Dhaka during the period of July 2012 to June 2013.A total number of 60 subjects were selected with age ranging from 3 to 15 years. Among them 30 autistic children were included in the study group. They were selected from Autistic Foundation of Bangladesh, 114/3, Provatibagh, Tilpapara, Khilgaon, Dhaka. Age matched 30 apparently healthy children were included in the control group for comparison. Data were collected in pre-designed structured questionnaire by the researcher herself. For assessment of thyroid function serum FT3 and FT4 levels were estimated by RIA method and serum TSH level was estimated by IRMA method. The statistical analyses were done by unpaired student's 't' test and Pearson's correlation coefficient (r) test.

Results : The mean serum FT3 level was almost similar and within normal range in both the groups. The results showed no statistically significant difference among the groups. The mean serum FT4 level was significantly ($p<0.001$) lower but within normal range in the study group in comparison to that of the control group. Again, the mean serum TSH level was significantly ($p<0.001$) higher in the study group in comparison to that of the healthy group.

Conclusion : It concludes that, subclinical hypothyroidism may be one of the non-genetic risk factors associated with autism spectrum disorders.

Keyword : Thyroid hormones, Hypothyroidism, Autism spectrum disorders

Introduction

Autism spectrum disorders (ASD) are cognitive and neurobehavioral disorders having three core features: Deficits in socialization, deficits in verbal & non-verbal communication and restricted & repetitive patterns of behaviors[1]. Worldwide prevalence of ASD is reported to be 3 to 6 per 1000 children with a male to female ratio of 3:1[2]. In Bangladesh the current estimates of prevalence is nearly 10.5 lakhs. The centre for Child Development and Autism of BSMMU started it's journey in 2001 and only 12 children with autism attended the centre, but the number increased to 105 children in the year 2009[1]. The exact cause of autism is unknown but it is believed to be multifactorial. It usually appears within the first three years of life[3]. Genetics alone do not determine the entire ASD phenotype.

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The process is determined by genetic susceptibility but other non-genetic factors can modify it. So in most cases autism appear to be caused by a combination of autism risk genes and non-genetic factors that influence early brain development[4]. Thyroid hormones are essential for normal growth and development of brain during early years of life. Different patterns of cognitive effects result from prenatal and postnatal thyroid hormone insufficiency. Researchers reviewed the results of structural, neurophysiological and behavioral changes due to alteration of the thyroid axis[5]. Studies indicate that non-genetic factors such as thyroid dysfunction due to endocrine disrupting toxins, teratogens, obstetric complications and prenatal infections such as rubella, cytomegalovirus are responsible for autistic cases[5]. Sensitive populations like persons with marginal dietary iodine deficiency, pregnant women, the new borns and young infants are particularly at risk for TH disruption. It can be induced by environmental contaminants like lead, mercury leading to neurodevelopmental impairments[6]. The diagnosis of autism is based solely on behavioral characteristics, as currently there is no biochemical marker for autism. Research suggests that there are various types of neuroendocrinological abnormalities

present in autistics and possibly TSH could serve as a biochemical parameter of the disease[7]. Several studies were undertaken in other countries to evaluate thyroid hormones as possible biochemical marker for ASD. A recent study showed that children born with very low levels of thyroxine had a higher risk of developing a autism spectrum disorder[8]. Thyroid hormones are essential for normal growth and development of brain during the early years of life[5]. They regulate neuronal proliferation, migration and differentiation in discrete regions of the brain during definitive time periods. Different patterns of cognitive effects results from prenatal and postnatal thyroid hormone insufficiency. It was noticed that nearly three-quarters of children with autism are found to have an underactive thyroid[9]. Insufficiency of thyroid hormone during brain development reduces cell number, synaptogenesis and dendritic arborization; alters cell migration patterns and decreases axonal myelination[8].

At present, researchers are beginning to appreciate the Thyroid-Autism connection and suggesting hypothyroidism as a major contributor to the development of autism[9]. Studies indicate that the general level of TSH was higher in young autistic patients and was most pronounced in those with complete impairment of verbal communication[6]. A different study showed that hypothyroidism is one of the non-genetic factors associated with autism[10]. Autism spectrum disorders are an increasingly important health concern in Bangladesh at present. The heightened awareness has been accompanied by a renewed interest to uncover theunderlying pathophysiologic mechanisms and to find possible causes of the disorder at multiple levels. It has been hypothesized that disturbance in the thyroid hormone availability and metabolism during critical periods of neuronal development may lead to behavioral disturbances as noted in ASD[11]. Deficiency of thyroid hormones may have a significant contribution to the web of causes of ASD but their exact relationship remains debatable.

Hence, the present study was designed to evaluate serum free triiodothyronine (FT3), free thyroxine (FT4) and thyroid-stimulating hormone (TSH) levels in children with autism to explore the role of thyroid hormone deficiency as one of the risk factors associated with autism.

Methods

This analytical type of cross-sectional study was conducted in the Department of Physiology of Dhaka Medical College, Dhaka from July 2012 to June 2013. Ethical permission was taken from Ethical Review Committee of Dhaka Medical College. A total number of 60 children with age range 3-15 years participated in this study. Thirty autistic children diagnosed by psychiatrist according to Childhood Autism Rating Scale were included in the study group from Autistic Foundation of Bangladesh, 114/3, Provatibagh, Tilpapara, Khilgaon, Dhaka and sthirty apparently healthy children selected from personal contact were included in the control group. After selection, proper counseling was done by explaining the aim, objectives, benefits, risks and procedure of the study to the parents of the subjects. They were encouraged for voluntary participation and written informed consent were taken in a prescribed form. Detailed family history and medical history were also taken. Physical and clinical examinations were done and all the informations were recorded in a prefixed questionnaire. Anthropometric measurement including height and weight were taken and BMI was calculated. Then under aseptic precaution, 5ml venous blood was collected from antecubital vein of each subject of both groups for biochemical test. Serum FT3 and FT4 were measured by Radio Immuno Assay (RIA) TSH level was measured by Immuno Radio Metric Assay (IRMA) method using radio isotope I-125 as tracer. These tests were carried out in the Centre for Nuclear Medicine & Ultrasound, Dhaka Medical College, Dhaka. Data were expressed as mean ±SD (Standard deviation). Statistical analysis was done by using SPSS for windows version 17. Unpaired Student's't' test and Pearson's correlation coefficient (r) test were used as the tests of significance and p value <0.05 was accepted as the level of significance.

Results

All the subjects of this study were similar for age & BMI. In this study mean values of serum FT3 in both the groups were almost similar and within normal range. Results showed statistical significant differences.

The mean serum FT4 level was also within normal range but significantly ($p<0.001$) lower in autistic children in comparison to that of normal children.

Again, significantly ($p<0.001$) higher levels of serum TSH were found in the autistic group in comparison to that of the control group (Table I).

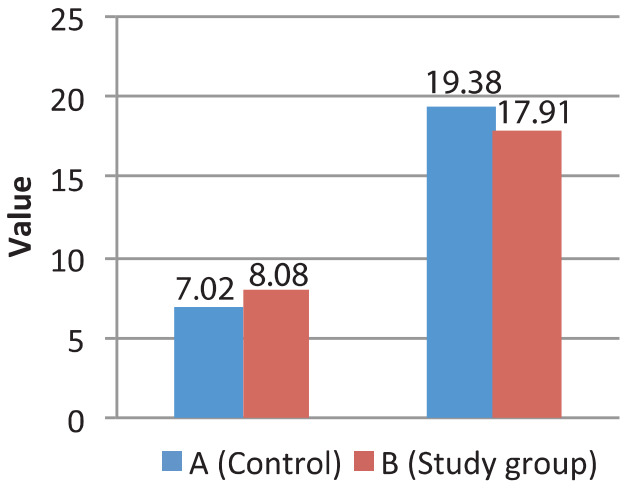

Figure 1 : Mean age (years) and Body Mass Index (BMI) (Kg/m2) of study subjects.

Table I

Serum free triiodothyronine, free thyroxine and thyroid-stimulating hormone levels in both the groups (n=60)

Groups	n	FT3 pmol/L)	FT4 pmol/L)	(TSH (mIU/L)
A-Control	30	4.15±0.87	17.09±2.96	3.15±1.19
B-Study	30	4.24±1.13	13.29±3.55***	10.17±3.72***

Data are expressed as Mean ± SD. Unpaired Student's't' test was performed to compare between groups. The test of significance was calculated and ***p values <0.05 was accepted as level of significance.

Discussion

The present study was undertaken to observe some aspects of thyroid function status in children with autism spectrum disorders by estimating serum FT3, FT4 and TSH levels. All the parameters were also estimated in apparently healthy age and BMI matched children to find out the baseline data and also for comparison. In this study, thyroid hormone levels in the control group were within physiological limit and almost similar to the findings observed by various investigators from different countries[12,13].

Our study showed that, the mean serum FT3 levels in both the groups were within normal range and almost similar and no significant difference was observed among the groups. This finding was in agreement with other researchers of different countries[14,15].

The mean serum FT4 level was also within normal limit but significantly lower in the autistic subjects in comparison to that of the healthy subjects. Similar findings were also made by other investigators[7-9]. Again, elevated serum TSH level was found in the autistic children and these observations are in accordance with other research workers[6,10].

It has been suggested that nervous system growth and differentiation are closely correlated with thyroid hormones in the initial developmental stages. Deficiency of this hormone during the first two years of life may produce morphological brain changes that can have significant deleterious behavioral and cognitive effect[9]. In summary, our findings leads to the suggestion that impairment of mental and cognitive development found in autistic children may result from the subclinical hypothyroidism present in these special children.

Conclusion

From the result of this study, it may be concluded that undiagnosed subclinical hypothyroidism may be one of the non-genetic risk factors associated with autism spectrum disorders.

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Original article

A two years Study of Fatal Burn Injuries in Dhaka Medical College

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Abstract

Objectives : Burn is a global public health problem, occurs since antiquity till the present day. Burn represent an extremely stressful experience for both the burn victims as well as their families. Flame burn and related injuries are major cause of death and disability.

Material and Methods : This study was carried out at the Forensic Medicine department in Dhaka Medical College from the year 2012-2013. This study was based on the post-mortem examination of the dead bodies at the mortuary of Dhaka Medical College. The purpose of this study is to record and evaluate the causes and magnitude of the fatal burn injuries retrospectively.

Results : Among 4898 cases of total autopsy done in 2012-2013, only 295 (6.02%) cases of death due to burn were derived. In this observation, death due to burn was mainly flame burn (97.29%) and superficial and 82% mortality over 40% TBSA (Total Body Surface Area).In age variation 21 to 40 year group were more (66.44%) affected in comparative than other age group. In sex, female (52.88%) were more affected that male (47.12%). In type of burn, flame burns were more (97.29%) than other type. Muslims were more affected (87.46%) than other religion. 82% death occurred over 40%-100% TBSA.

Conclusion : Burn is a common type of traumatic injury causing considerable morbidity and mortality. Detail study regarding flame burn is required to be carried out in this country.

Key words : Flame burn, Acid burn, Neurogenic shock.

Introduction

According to WHO, 2,65000 death occurred due to burn in each year across the world[1]. More than 90% death occur in lower and middle income countries. Even in developed countries more than two million individuals annually are burned seriously and require medical treatment[2]. A burn is an injury, which is caused by application of heat or chemical substance to the external or internal surfaces of the body, which causes destruction of tissues. The minimum temperature for producing a burn is about 44°C for exposure of about 5 to 6 hours. At 65°C, two seconds are sufficient to produce burns and full thickness destruction of skin occurs within seconds above 70°C[3]. In case of superficial burns, there is immediate erythema. If there is formation of blister, then that occurs within 2-3 hours. The erythema around a blister or deep injury passes of by 2nd day. Pus formation occurs by 3rd day. Within the next one or two days there is slough formation, which is shedded off once by the end of the first week. Burn injuries involving the skin and deeper tissues take a minimum of 2 weeks to heal[4].

The majority of deaths occurred within a week (80%) and most of the victims died from neurogenic shock followed by septicemia and Pneumonia. The purpose of this study was to record and evaluate the causes and magnitude of the fatal burn injuries retrospectively.

Materials and Methods

This descriptive cross sectional study of death due to burn was carried out in the Dhaka Medical College from January 2012 to December 2013. The data was collected from inquest report, challan and book of post mortem examination report, kept at the department of Forensic Medicine. This study was based on the post-mortem examination of the dead bodies at the mortuary of Dhaka Medical College. Dead bodies were sent from different Police Station and Dhaka Medical College Hospital. The records reveal various information pertaining to their age,

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sex, type of burn, Total Burn Surface Area (TBSA) and cause of death.

Results

The minimum temperature for producing burn is about 44°C for an exposure of about 5 to 6 hours. At 65°C, two seconds are sufficient to produce burns and full thickness destruction of skin occurs within seconds above 70°C. An analysis of autopsy records revealed that 295 (6.02%) cases of burn death among the total autopsy 4898, done over 2 years period (2012-2013) in the mortuary of Dhaka Medical College(Table-I). Among them 21-40 years age groups (66.45%) were affected more and more than 50% were female (Table-II & III). death mainly occurred by flame burn (97.29%) (Table-IV). Most of the death (35%) occurred due to involvement of 81-100% TBSA (Table-V).

Table-1: Manner of death from 2012 to 2013

Manner of death	Number of death	Percentage of death (%)
Hanging	725	14.80
Homicide	934	19.07
Poisoning	831	16.97
RTA	1149	23.46
GRP	306	6.25
FFH	144	2.94
Burn	295	6.02
Electrocution	88	1.80
Drowning	37	0.75
Natural(in Jail ,under Police)	188	3.84
Stillborn	47	0.96
Others	154	3.14
Total	4898	100

Figure-1 : Shows comparison between frequencies of burn death with other significant number of unnatural deaths.

Manner of death

Table-II : Age distribution of the death victim due to burn from 2012 to 2013.

Age in year	Number of death	Percentage
01-20	66	22.37%
21-40	196	66.45%
41-60	26	8.81%
61 and above	7	2.37%
Total	295	100%

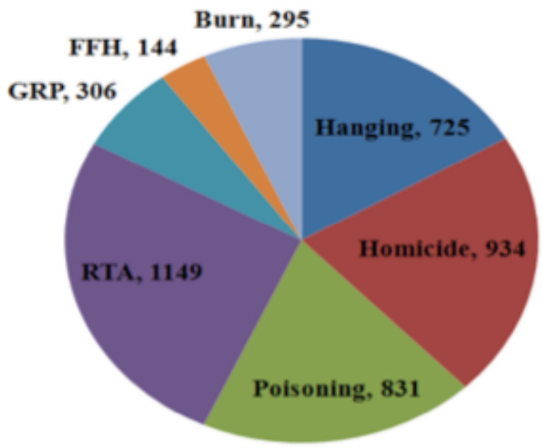

Table-III : Sex distribution of the death victim due to burn from 2012 to 2013

Sex	Number of death	Percentage
Male	139	47.12%
Female	156	52.88%
Total	295	100%

Table-IV : Type of burn causes death from 2012 to 2013

Type of burn	Number of death	Percentage
Flame burn	287	97.29%
Scald	7	2.37%
Acid Burn	1	0.34%
Total	295	100%

Table-V : TBSA (Total Burn Surface Area) of death victim due to burn from 2012 to 2013

Percentage of burn (TBSA)	Number of death	Percentage
Below 40%	56	19%
41-60%	76	25.76%
61-80%	60	20.24%
81-100%	103	35%
Total	295	100%

Discussion

A severe burn injury is the most devastating injury that a person can sustain and yet hope to survive[5]. Like Bangladesh from my study fire related burn or flame burn rate is also high as in lower middle income countries (LMICs)[9]. In our country it is mostly occurred at home, working place like garments or other factory and recently due to some political violence.[6]

In terms of the sex differences, women are usually at higher risk of burn than men. In India 65% burn death victim are women which is nearly same rate to my study[10]. Women are most vulnerable because of cooking, use of loose filling dress, suicide and assault[11]. In this observation 21-40 years age group are more affected than others. In compare with USA, Children under age 5 and older person above 65 years are more affected.[7] It is mostly occurred due to smoking and home fire equipment[12]. But from my study, this middle age group are more exposed in cooking, accident in factories and in some political violence.[8] This study revealed that signs of vitality (Soot in airways and/or digestive tract) were found at autopsy in large majority of victims who died from burn. Sometimes person died during burn but there is no involvement of burn. As there is immediate combustion of oxygen and releasing of carbon particles. These particles are asphyxiant and the person died due to asphyxia[13].

Conclusion

Burns have always been considered as one of the most destructive injuries, causing not only death but also major economic and psychological impacts and long term somatic sequelae as well. Moreover, burns are also among the most expensive traumatic injury, because of long hospitalization and rehabilitation and costly wound and scar treatment. Non fatal burn injuries are a leading cause of morbidity[14].

Now a days burn is a major concern for health practitioners in our country, because of some burn cases occurred due to political violence which lead to death. The result of this study provides the necessary information to develop proper burn prevention programs, thereby reducing the frequency of burns and burn related death. For prevention of burn incidence we should take steps for:

1) Improvement of public awareness by the concern authority.

2) Training of communities in first aid.

3) Modernized health equipments.

4) Proper implementation of law.

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Review article

Fluid and electrolyte management in children

Mahmuda Hassan[1], M. Begum[2], K. Chowdhury[2], T.U. Ahmed[2], A. U. Ahmed[2], H. Rahman[3]

Abstract : Dehydration remains a major cause of morbidity and mortality in infants and young children worldwide. Dehydration is most commonly is due to diarrhea in infant and children specially in the developing countries. Infants are particularly susceptible to development dehydration because of their greater baseline fluid requirements (due to a higher metabolic rate), higher evaporative losses (due to a higher ratio of surface area to volume), and inability to communicate thirst or seek fluid.

Keywords : fluid therapy, electrolytes, pediatrics.

Introduction

The body is comprised of solids and water; the proportion of water changes according to age. Total body water (TBW) is divided between the intracellular fluid (ICF) and the extracellular fluid (ECF), separated by cell membrane. The ECF can be further subdivided into water within the intravascular space (IVS) and the interstitial space (ISS), separated by capillary membrane. At birth, a higher percentage of water is in the ECF, unlike older children and adults where the higher proportion is intracellular. Despite having a similar osmolality (290–320mosmoles), the electrolyte content of the ECF and ICF are very different. The ECF contains a high concentration of sodium, bicarbonate and chloride, with a low concentration of potassium, calcium and magnesium. In contrast, the ICF has a high concentration of potassium and magnesium and a low concentration of sodium and bicarbonate.

Fluid and electrolyte loss from the body due to any reason causes dehydration. Dehydration remains a major cause of morbidity and mortality in infants and young children worldwide. Dehydration is a symptom or sign of another disorder, most commonly diarrhea. Dehydration occurs when there is significant depletion of water and varying degrees of electrolytes from the body.

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Physiologic differences

Total body water content changes drastically from before birth until one year of age. At 24 weeks gestational age, a baby's total body water content is close to 80% of total body weight[1]. This slowly decreases until the child is around one year of age, when total body water content is about 60% of total body weight.2 Most adults' total body water is between 50% and 60% of total body weight.[2]

After birth, infants are expected to lose approximately 5%-15% of their body weight, with more being lost in preterm low birth weight infants.[2,3] In fact, if this weight loss does not occur, there is cause for concern for renal dysfunction and sepsis. In addition to total body water differences, the percent of body weight accounted for by intracellular and extracellular water also changes. In adults, who have about 60% of total body weight as water, about 20% of total body weight is extracellular water and 40% is intracellular water.[2] Newborn babies have more extracellular water—45% of total body weight—compared with only 35% of total body weight that is intracellular water. [2]

Fluid therapy

Physiologic differences also play a role in fluid therapy. The changes that take place as a child grows have a great effect on fluid requirements, making special attention to fluid therapy essential in pediatric pharmacotherapy. There are three classifications of fluid therapy,

1. maintenance, 2. deficit, and 3.replacement, each of which will be discussed separately. When choosing the type of fluid and volume to be administered, careful consideration should be paid to each component of fluid therapy.

Maintenance fluid and the electrolyte requirement:

Physiologic differences of ICF and ECF in children than that of the adult play a role in fluid therapy. The changes that take place as a child grows have a great effect on fluid requirements, making special attention to fluid therapy. There are three classifications of fluid therapy, maintenance, deficit, and replacement. When choosing the type of fluid and volume to be administered, careful consideration should be paid to each component of fluid therapy.

Maintenance fluids are frequently given through an intravenous line, but can also be given orally if the patient is able to tolerate oral therapy. Sensible losses, which include urine output and faecal water, make up the majority of on-going losses, with additional contributions from insensible losses such as respiration and perspiration.[4] The Holliday-Segar in 1957, equation remains the standard method for calculating maintenance fluid requirements in children. They also showed that the water requirement in milliliters was equal to the total energy expended (i.e. 1000 ml of water is required for every 1000kcal expended). Thus, the formula for caloric requirement can also be used to calculate daily water requirements. Thus, while the Holliday-Segar method actually estimates kilocalories lost, it is estimated that a loss of 1 kilocalorie requires 1 ml in replacement, so the kilocalorie estimate is an efficient target for fluid requirements.

Table1: Hourly Maintenance Fluid Requirement (1-hour period).

Examples:
For 10 kg child: 10 kg x 4 ml/kg/hour = 40 ml/hour
For 15 kg child: (10 kg x 4 ml/kg/hour) + (5 kg x 2 ml/kg/hour) = 50 ml/hour
For 25 kg child: (10 kg x 4 ml/kg/hour) + (10 kg x 2 ml/kg/hour) + (5 kg x 1 ml/kg/hour) = 65 ml/hour.

Holliday-Segar Method for Calculating Maintenance Fluid Requirements in Children Maintenance electrolyte requirements must be taken into account, with particular attention paid to sodium requirements, as recent evidence suggests that sodium requirement in hospitalized children are higher than originally thought. Fluid therapy can also have an impact on drug therapy. Hydration status can affect the dose of a drug needed to achieve therapeutic concentrations, and dehydrated patients may be at risk for toxicity even with standard doses of drugs. So monitoring fluid and electrolyte therapy is an important issue in a hospitalized child.

Maintenance fluids are given to compensate the ongoing losses and are required for all patients.[5]

Requirements for children are higher than those for adults for multiple reasons. First, the higher metabolic rate of children requires a greater caloric expenditure, which translates into higher fluid requirements.[5] Secondly, children, especially infants, have a much higher body surface area to weight ratio, and this translates into relatively more water loss from skin compared with adults. In addition, children, especially infants, have higher respiratory rates,[6]

Table 2 : shows Conditions that Modifying Daily Maintenance Fluid Requirements

Requirements Increased	Requirements Decreased
Fever, sweating, vomiting or diarrhea	Meningitis
Diabetes	Congestive heart failure
Burns	Renal failure

Note : with a fever, daily maintenance fluids should be increased by 12% for every degree body temperature above 37.5°C (rectal)

Daily requirements of sodium, potassium and chloride areusually quoted as 30, 20 and 20 mol, respectively, for every1000 kcal expended.

Requirements for sodium are 3 mEq/kg/ day, while the requirements for potassium are 2 mEq/kg/day.[5,7] Chloride needs, which are 5 mEq/kg/day,[7] are usually met by administering sodium and potassium as sodium chloride and potassium chloride salts. 3 mEq/ kg/day of sodium proposed as maintenance by Holliday and Segar is only enough for healthy children, but in sick children electrolyte requirement may varies according to the serum electrolyte value.

Deficit fluid and the electrolyte management in presence of dehydration

Dehydration remains a major cause of morbidity and mortality in infants and young children worldwide. Dehydration is most commonly is due to diarrhea in infant and children specially in the developing countries. Infants are particularly susceptible to development dehydration because of their greater baseline fluid requirements (due to a higher metabolic rate), higher evaporative losses (due to a higher ratio of surface area to volume), and inability to communicate thirst or seek fluid. Dehydration is one of the leading causes of pediatric morbidity and mortality throughout the world. Diarrheal disease and dehydration account for 14% to 30%of

worldwide deaths among infants and toddlers. The American Academy of Pediatrics and the WHO both recommend oral replacement therapy for mild and moderate dehydration. Children with severe dehydration (eg, evidence of circulatory compromise) should receive intravenous fluids. Children who are unable or unwilling to drink or who have repeted vomiting require intravenous fluid replacement. The goal of therapy is to recognize the degree and type of dehydration[8] and to restore any water and electrolyte deficits while meeting maintenance needs and replacing ongoing losses.

Management of fluid in presence of dehydration based on 4 points.

Treatment is best approached by considering separately the 1. Requirements of resuscitation fluid. 2. Current deficit,3. Ongoing losses, and 4. Maintenance requirements. The volume, composition, and rate of replacement differ from person to person. Formulas and estimates used to determine treatment parameters provide a starting place, but treatment requires ongoing monitoring of vital signs, clinical appearance, urine output, weight, and sometimes serum electrolyte levels.

1. Resuscitation fluid in patients with signs of hypoperfusion should receive boluses of isotonic fluid (eg, 0.9% saline or lactated Ringer solution). The goal is to restore adequate circulating volume to restore blood pressure (BP) and perfusion. The resuscitation phase should reduce moderate or severe dehydration to a deficit of about 8% body wt. If dehydration is moderate, 20 mL/kg (2% body wt.) is given IV over 20 to 30 min, reducing a 10% deficit to 8%. If dehydration is severe, sometimes 3 boluses of 20 mL/kg will likely be required. The end point of the fluid resuscitation phase is reached when peripheral perfusion and BP are restored and the heart rate is returned to normal (in an afebrile child).

Fluids lost prior to medical care are termed "deficit fluids." Examples of clinical situations where a patient would present with a deficit fluid include gastrointestinal illness with vomiting and diarrhea, traumatic injuries with significant blood loss, and inadequate intake of fluids over a period of time. One clinical sign of dehydration which can be of use is weight loss. Assess dehydration and Calculate the fluid deficit and correct the fluid deficit accordingly. Deficit fluids, like maintenance fluids, are most easily handled by approaching the needs of the patient in a systematic manner. Clinical signs of dehydration should be taken into consideration first, as they can provide useful understanding of fluid requirement of the patient. One clinical sign of dehydration which can be of use is weight loss by subtracting illness weight from the pre illness weight, but most of the time it is not available. So, assessment with clinical signs include increased thirst, dry mucous membranes, and decreased urine output more convenient[8] Table 3.

Table 3

Feature	Mild Dehydration (< 5%)	Moderate Dehydration (5% to 10%)	Severe Dehydration (> 10%)
Heart rate	Normal	Slightly increased	Rapid, weak
Systolic blood pressure	Normal	Normal to orthostatic, > 10 mm Hg change	Hypotension
Urine output	Decreased	Moderately decreased	Markedly decreased, anuria
Mucous membranes	Slightly dry	Very dry	Parched
Anterior fontanel	Normal	Normal to sunken	Sunken
Tears	Present	Decreased, eyes sunken	Absent, eyes sunken
Skin*	Normal turgor	Decreased turgor	Tenting
Skin perfusion	Normal capillary refill (< 2 seconds)	Capillary refill slowed (2–4 seconds); skin cool to touch	Capillary refill markedly delayed (> 4 seconds); skin cool, mottled, gray
Mental status	Alert	Irritable	Lethargic

*Skin condition is less useful in diagnosis of dehydration in children > 2 years of age[9]

Infants, those less than one year, are mildly dehydrated at a 5% loss, moderateat 10% and severeat 15%.Children, those greater than 1 year, are mildly dehydrated at a 3% loss, moderate at 6% and severe at 9%.

Calculation of the deficit fluid in a 10kg weight child.
Clinical Estimation (note that 1cc = 1gm)
• In case of mild dehydration : 0 - 5% weight loss X body weight (5% X 10kg = 500cc)
• In case of moderate dehydration : 5 - 10% weight loss X body weight (10% X 10kg = 1000cc)
• In case of severe dehydration: 10 - 15% weight loss X body weight (15% X 10 kg = 1500cc)

Calculation of electrolyte requirement will depend according to the type of electrolyte imbalance either isonetramia, hyponatramia or hyernatraemia which is present along with dehydration.

2. Calculation and administration of maintenance fluid

Fluid losses 50% through urine and additional 50% insensible (2/3rd through skin and 1/3 through respiratory system)

Maintenance fluid requirement
4:2:1 rule per hour or 100:50:20 rule per 24hr
4cc/kg/hr. for 0 - 10kg, 2cc/kg/.for next 10 - 20kg, 1cc/kg/hr. from 20kg up to rest of the body weight So, 100cc/kg/24hr for up to 10Kg , 50cc/kg/24hr for 10-20kg and 20cc/kg/hr. for > 20kg to rest of the body weight

Caloric requirement
Same 4:2:1 rule to determine Kcal/kg/hr or 100:50:20 rule for daily caloric requirement
Maintenance Electrolyte requirements
Na+ requirement is 3mEq/kg/24hr
K+ requirement is 2-3mEq/kg/24hr
Kidney produces enough bicarbonate therefore not required until PH is less than 7.0
Glucose: 5g glucose per 100ml maintenance fluid enough to prevent ketosis.
Requirements will increase with fever, ventilation, increase activity.

3. Calculate and administer the ongoing loss if any.
Volume of ongoing losses should be measured directly (eg, NGT, catheter, stool) or estimated (eg. 10 ml/kg per diarrheal stool). Replacement should be milliliter for milliliter in time intervals appropriate for the rapidity and extent of the loss. Ongoing electrolyte losses can be estimated by source or cause (Table 3: Estimated Electrolyte Deficits by Cause).

Table-4 : Estimated Electrolyte Deficits by Cause

Cause	Sodium (mEq/L)	Potassium (mEq/L)
Diarrhea	—	—
Isotonic dehydration	80	80
Hypotonic dehydration	100	80
Hypertonic dehydration	20	10
Pyloric stenosis	80	100
Diabetic ketoacidosis	80	50

Table-5 : Electrolyte status

Definition	Serum[Na+] (mmol/l)
Hyponatraemia	< 135
Normal	135 - 145
Mild Hypernatremia	146 - 149
Moderate Hypernatraemia	150 - 169
Severe Hypernatraemia	≥ 170

1. Intravenous Rehydration for Moderate or Severe Isonatremic Dehydration
Once the degree of dehydration is established, the type of dehydration, defined by serum sodium concentrations, needs to be determined. Most dehydrated patients have an isotonic dehydration.[8] Isonatremic dehydration is defined by sodium of 130 to 150 mEq/L (130 to 150 mmol/L).This reflects an equal proportion of solute and water loss. Isonatremic dehydration typically occurs in patients with secretory diarrhea where the solute concentration of the diarrhea is same as the plasma solute concentration.

Children who are clinically unstable whatever is the type of dehydration, child should receive fluid bolus(es) of 20 ml/kg with 0.9% NS to attain adequate tissue perfusion. Isotonic fluids are used because they provide rapid volume expansion in the plasma and extracellular fluid.

Many recommend initial replacement of 50% of deficit fluids over 8 hours followed by replacement of the remaining 50% deficit over the subsequent 16 hours along with the maintenance fluid. However, this can be clinically impractical and may be associated with an increased risk of too-rapid replacement or difficulty to adjust the rate at the correct time with the junior staffs. If the child received adequate initial resuscitation, replacing the total deficit over 24 hours is generally acceptable.

Maintenance fluid and electrolyte needs should be given and based on the child's normo- volemic weight, either obtained from a prior weight or calculated based on estimated percentage of dehydration.

Based on expert opinion and reasoning, in children with moderate-to-severe isonatremic dehydration, maintenance and deficit fluid and electrolytes should be combined into one solution that is infused over 24 hours. Regardless of the percentage of dehydration, this calculates as 5% dextrose (D5) 1/3 NS + 40 mEq/L (40 mmol/L) of KCl. However, because this is not a standard fluid available in most hospitals, D5 ½ NS + 40 mEq/L (40 mmol/L) KCl can be safely substituted.[10] If there are concerns about renal insufficiency, potassium should not be added to the fluids until the patient is voided. Ongoing fluid and electrolyte losses also should be replaced. Gastric losses should be replaced with 0.45% NS plus 10 to 15 mEq/L (10 to 15 mmol/L) of KCl. For diarrheal losses, bicarbonate substitution for chloride should be considered.

More judicious volumes may be considered in children with congestive heart failure or cerebral edema. Lactated Ringer solution should not be used routinely because it is relatively hypotonic (130 mEq/L [130 mmol/L]) of sodium and could adversely lower the patient's serum sodium. In addition, it contains 4 mEq/L (4 mmol/L) of potassium that could contribute to hyperkalemia. Children with significant emesis may have a contraction alkalosis (increase in blood pH) as a result of fluid losses that could be worsened by the lactate content of the fluid being converted to bicarbonate. Patients with mild to moderate dehydration may be rehydrated with oral therapy, even if diarrhea and vomiting continues.[10,11]

2. Hyponatremic dehydration

Hyponatremic dehydration with a sodium concentration of less than 130 mEq/L (130 mmol/L). Patients with a serum sodium of less than 125 mEq/L are at high risk for serious central nervous system symptoms; lethargy followed by seizures is common.[12] Due to the emergent nature of this condition, boluses with hypertonic saline, usually 3% sodium chloride, are warranted. The volume of 3% sodium chloride is determined by the sodium deficit, which is calculated using the following equation13 (desired serum sodium concentration − current serum sodium concentration) × 0.6 × (weight in kg)

This is generally given over a few hours, with serum sodium checks done 2 to 4 hourly in order to avoid hypernatremia. Serum sodium should not be corrected

faster than 12 mEq/L within 24 hours. Hyponatremic dehydration most typically occurs in older infants and children with gastrointestinal infections. These children are often given fluids with low sodiums content such as water, juice, ginger ale, sodas, or tea. In addition, ADH is often released, further diluting the intravascular solute with the re-absorption of water.[13] As serum osmolality falls, fluid is shifted from the extracellular to the intracellular space, causing more rapid and more severe intravascular compromise. Affected children are most likely to require immediate volume resuscitation. Normal saline should be rapidly infused in 20-mL/kg aliquots to restore intravascular volume. Lactated Ringer solution should be avoided because its lower sodium content may worsen the hyponatremia and the potassium content may contribute to hyperkalemia. Cerebral salt wasting can lead to hyponatremic dehydration is poorly understood, rare condition occurs in patients with central nervous system disorders, most commonly associated with intracranial surgery, meningo encephalitis, and head injury. It typically occurs in the first 10 days of the illness or injury and resolves in 3 to 4 weeks. Cerebral salt wasting is characterized by hyponatremia and intravascular fluid depletion related to inappropriate renal sodium wasting.

Affected children can have very high-volume urine output containing up to 300 mEq/L (300 mmol/L) of sodium and often require replacement with hypertonic saline solutions. Administration of salt tablets and the mineralocorticoid fludrocortisone has been used to help abate sodium and fluid losses.

Using the child's baseline weight, maintenance and deficit fluid and electrolytes are calculated and generally replaced over 24 hours.

Based on expert opinion and reasoning, children with moderate-to-severe hyponatremic dehydration are most likely to need immediate circulatory support. Fluid and electrolyte maintenance and deficit needs usually calculate to be D5 ½ NS + 40 mEq/L (40 mmol/L) KCl. Maintenance plus deficit volumes can be infused over 24 hours, with goal correction of sodium not to exceed 12 to 15 mEq/L (12 to 15 mmol/L) over the 24 hours. Frequent monitoring is recommended, generally every 4 to 6 hours. 10

Administering the deficit fluid faster causes osmotic fluid shifts, which can result in cerebral edema and convulsions.14 Very rarely, precipitous correction of the sodium can result in central pontine myelinolysis. Infants and children can present with seizures related to a rapid drop in the serum sodium concentration. This rapid decrease, generally to less than 120mEq/L (120 mmol/L), overwhelms the cerebral osmo regulatory mechanisms,

resulting in cerebral edema. The seizures can be difficult to abate without partial correction of serum sodium, usually to 120 mEq/L(120 mmol/L). Using the previously cited formula to calculate the sodium replacement, either 0.9% NS or hypertonic 3% saline is given. The choice of solution is generally determined by the volume of saline correction. The correction with 0.9% NS, containing 154 mEq/L (154 mmol/L) of sodium, which generally can be well tolerated in a dehydrated patient. Administration of hypertonic saline, with a sodium content of 513 mEq/L (513 mmol/L) (0.5 mEq/mL[0.5 mmol/mL]), requires approximately one-third of the volume of isotonic saline. However, hypertonic saline may necessitate central access because peripheral administration can be painful and lead to cutaneous tissue necrosis with any extra-vasation.

3. Hypernatremic dehydration

Hypernatremic dehydration is defined as serum sodium greater than 150 mEq/L (150 mmol/L). Despite elevated sodium concentrations, the child actually has total body sodium deficiency, but the water loss exceeds the sodium loss. Hypernatremic dehydration is most commonly seen in young infants receiving inadequate water replacement, typically associated with diarrheal illnesses or poor breast feeding. Because the intravascular contents are hypertonic, fluid shifts from the cells into the intravascular space. Thus, the children may be less hemodynamically compromised, resulting in underestimation of the degree of dehydration. As because intravascular volume is relatively preserved, affected infants often present late for medical care. They are usually somnolent but become hyperirritable when stimulated, often with a high-pitched cry. Their skin feels "doughy" or "velvety". The major concern is cerebral cellular dehydration in the presence of hypertonicity. Resulting brain shrinkage can cause rupture of bridging veins, leading to subdural, subarachnoid, and intra-parenchymal hemorrhage. In addition, thrombosis of the small veins or dural sinuses can occur. Mortality can be high, ranging from 3% to 20%. Up to 40% to 50% of infants can have neurologic sequelae, and in 5% to 10%, the sequelae are severe.

The approach to patients with hypertonic dehydration is quite different, due to the hyper-osmolar state of their circulating blood. The deficit fluid volume should be added to the maintenance fluid volume needed for 48 hours, and the total should be administered over 48 hours. Administering the deficit fluid faster causes osmotic fluid shifts, which can result in cerebral edema and convulsions.[14] For this reason, serum sodium should be corrected by no more than 10 mEq/L/day.[14] Serum sodium should be checked frequently (every 4 to 6 hours) to ensure that rehydration is not occurring so quickly as to cause too rapid decrease in serum sodium.

Based on expert opinion and reasoning, fluid and electrolyte maintenance and deficit needs usually calculate to be D5 ¼ NS + 20 to 40 mEq/L(20 to 40 mmol/L) KCl. Deficit replacement should occur over 48 hours, with goal correction of sodium not to exceed 0.5 mEq/L (0.5 mmol/L) per hour. 10 Infants with presenting serum sodium values of 150 to 160 mEq/L(150 to 160 mmol/L) and with sodium correction of 0.5 mEq/L (0.5 mmol/L) per hour or less over 48 hours is the best. Infants with presenting serum sodium values greater than 160 mEq/L (160 mmol/L) and sodium correction greater than 0.5 mEq/L (0.5 mmol/L) per hour over 48 hours have significantly higher morbidity and mortality.

As with other forms of dehydration, fluid bolus(es) of 20 ml/kg with 0.9% sodium chloride should be administered rapidly if there are any signs of vascular compromise. 4 ml/kg of free water can be administered for every milliequivalent (millimole) of sodium greater than 145 mEq/L (145 mmol/L) or 3 ml/kg of free water administered for every milliequivalent (millimole) if the sodium value is greater than170 mEq/L (170 mmol/L) Potassium should be added once the infant is voiding and is clearly without intrinsic renal disease. Thus, D5 or D10+0.2% NS + 20 to 40 mEq/L (20 to 40 mmol/L) K+ usually appropriate for replacement over 48 hours. Frequent monitoring, generally every 4 to 6 hours, for the change in serum sodium is paramount to a good clinical outcome.

Hyperkalemia

Hyperkalemia is usually defined as a serum potassium of greater than 6 mEq/L, as this is where changes in the electrocardiogram are usually seen.[15] Before deciding to treat hyperkalemia, it is important to discover how the blood was acquired. If the blood came from a heel stick, as is frequently done in infants, cell lysis due to the trauma of the needle can cause intracellular potassium to enter the serum locally, leading to falsely elevated serum potassium.

Hyperkalemia can be treated with a variety of medications. In emergencies, rapid influx of potassium intra-cellularly are useful as they provide an acute decrease in serum levels. These medications include insulin and beta adrenergic agonists such as albuterol.[16] Sodium bicarbonate is sometimes used to treat hyperkalemia, but its mechanism in lowering serum

potassium levels is unknown and generally not immediate.[16] Other medications help by increasing elimination of potassium from the body. Sodium polystyrene sulfonate is an exchange resin which exchanges sodium for potassium in the gut, its use is generally for less emergent situations. Calcium is used in symptomatic patients for cardio-protective effects, as it antagonizes the membrane effects of potassium.[16]

Hypokalaemia

Hypokalemia occurs when a serum potassium concentration is<3.5 mEq/L,and it can become life threatening when the serum potassium concentration falls below 2.5 mEq/L. Hypokalemia can result from intracellular shifts of potassium, increased losses of potassium, or decreased ingestion or administration of potassium . The main cause of hypokalemia in pediatric patients is excessive gastrointestinal losses such as diarrhea or vomiting. Because serum potassium levels do not correlate with intracellular potassium levels, hypokalemia does not reflect total body potassium stores. Dietary potassium is predominantly in the form of potassium phosphate or potassium citrate, which results in the retention of only 40% as much potassium as KCl.[17] Therefore pharmacotherapy of symptomatic hypokalemia should be with KCl. In the presence of cardiac arrhythmias, extreme muscle weakness, or respiratory distress, KCl should be administered intravenously with cardiac monitoring. The intravenous dose of KCl is 0.5 mEq/kg (maximum 20 mEq/dose) administered over 1 to 2 hours based on the severity of the patient's symptoms.[18]

Conclusion

Dehydration is common in infants and children, especially following gastrointestinal illnesses. Oral rehydration can be safely and effectively accomplished in children with mild-to-moderate dehydration with normal serum sodium values. Children with more severe dehydration or with abnormal serum sodium values should be treated with intravenous infusions. It is important to understand how to determine the correct fluid and electrolyte solutions to meet the child's maintenance, deficit, and ongoing losses. In addition, it is also vital to recognize and to monitor the safely of the baby during the correction of dehydration and electrolyte imbalance.

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Case Report

Atypical Presentation of CNS Tuberculosis

Mahmuda Hassan[1]

Abstract : Tuberculosis is an infectious disease which is caused by the pathogenic microorganism Mycobacterium and a highly prevalent disease in developing countries. Approximately at 5-10% of tuberculosis a case central nervous system (CNS) involvement is due to haematogenous spread is not a rare entity. Tuberculoma is one of the manifestations of Central Nervous System (CNS) tuberculosis (TB). A tuberculoma is a tuberculous focus, which enlarges with in brain tissue, firm, avascular, spherical masses, with size varying between 2 cm to 10 cm in diameter and the compressed surrounding tissue shows edema and gliosis. Lesions are most often singular but may be multiple.Our case was a9 years boyhad occasional episodes of severe head ache for 12 months for which he was treated by local doctor. He was admitted at hospital with fever for 1 day with single episode of convulsion. He had also history of undue tiredness during outdoor games with 3 episodes of transient unconsciousness which lasted for 1 to 2 minute and resolved spontaneously. He had no features of signs of raised intracranial pressure or a hemiplegia or cranial nerve palsy and had no history of cough and cold or contact with TB patient.

Key words : Tuberculoma, Computerized Tomography (CT) scan; MRI.

Introduction

Tuberculosis (TB) continues to be an important cause of morbidity and mortality around the world, particularly in the developing as well as in developed countries. Because of the relative ease of travel from endemic areas, the increasing incidence of infections with the human immunodeficiency virus, and the emergence of multiple drug-resistant Mycobacterium strains of tuberculosis[1]are increasing. About 1% of patients with TB develop intracranial tuberculomas, usually as part of miliary TB that arises from extra-cranial spread[2]. In developing countries, the incidence of tuberculoma varies from 5% to 30.5% of all intracranial space occupying lesions.[3,4] Tuberculomas are usually seen in patients with extracranial signs and symptoms tuberculosis. This case is unusual because the patient was young and presented with only with head ache and single episode of convulsion and fever for 1 day before admission to the hospital rather than pulmonary manifestation. The diagnosis was based on clinical and neuroimaging features and response to anti-tuberculous treatment.

Pathophysiology

Most tuberculous infections of the central nervous system are caused by Mycobacterium tuberculosis,as a result of hematogenous spread from a primary location, either the lung or gastrointestinal tract[5]. Initially, small tuberculous lesions (Rich's foci) develop in the CNS, either during the stage of bacteraemia of the primary tuberculous infection or shortly after wards. These initial tuberculous lesions may be inoculated in the meninges; the subpial and subependymal surface of the brain or the spinal cord, and may remain dormant foryears. Later, rupture or growth of one or more of these small tuberculous lesions produces various types of CNS tuberculosis. The type and extent of lesion depend upon the number and virulence of bacilli and the immune response of the host[6]. Pathologically, a tuberculoma is composed of central core of caseous necrosis surrounded by a capsule of collagenous tissues and an outer layer of mononuclear inflammatory cell (including plasma cells & lymphocytes), epitheloid cells and multinucleated Langerhans' giant cells. A tuberculoma harbors few tubercular bacilli within the necrotic center and the capsule. Outside the capsule, there is parenchymal edema and astrocyte proliferation. Unlike caseous tuberculoma, a tubercular abscess has purulent center rich in tubercular bacilli, and lacks epithelioid giant cell granulomatous reaction in its wall.[7]

Clinical presentation

A 9 years boy of a non-consanguineous parent, hailing from a semi-urban area of Dhaka city of low socioeconomic background. He was delivered at term by normal vaginal delivery at a local clinic. Antenatal and natal history was uneventful and was vaccinated against all EPI vaccine. He was admitted at pediatric unit of Ad-din women's medical college hospital with the history fever for 1 day with single episode of convulsion. He had no past history of irregular fever, no history of weight loss or contact with known tuberculous patient. But he had

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history of severe head ache for 12 months for which he was treated by local doctor and also visited to eye specialist. His refraction error was corrected with spectacles and fundoscopy was done for severe head ache which revealed normal. But his headache persisted. He had also history of undue tiredness during outdoor game with 3 episodes of transient unconsciousness which lasted for 1 to 2 minute. On examination the he was conscious, cooperative, well oriented with the surroundings, and normal vital signs. Skin survey revealed BCG mark. Back & spine examination showed no abnormalities. All cranial nerves were intact. On general examination child was mildly pale, not icteric. Admission weight was 21kg, (3th percentiles),height 120cm (5th percentiles). Neurological and all other system including respiratory system reveled normal.

Laboratory investigations

Hb% was 12.8gm/dl, total count were 12,310/cmm and differential count were lymphocyte 10.2%, polymorphs 88.7%, eosinophil 0.1%, monocyte 0.9%, basophil 0%, platelet count was 1,94,000/ cmm. ESR 24 mm in 1st hour.Random blood sugar was 4.4mmol/L, Serum bilirubin was 0.2mg/dl, serum electrolytes and serum calcium were within normal limit. Peripheral blood film showed mature white cells with above distribution. Blood culture showed no growth of bacteria. But the Montous test (MT) was highly positive, induration more than 20 cm.

(Fig. 1 and 2). X ray chest no pulmonary lesion.

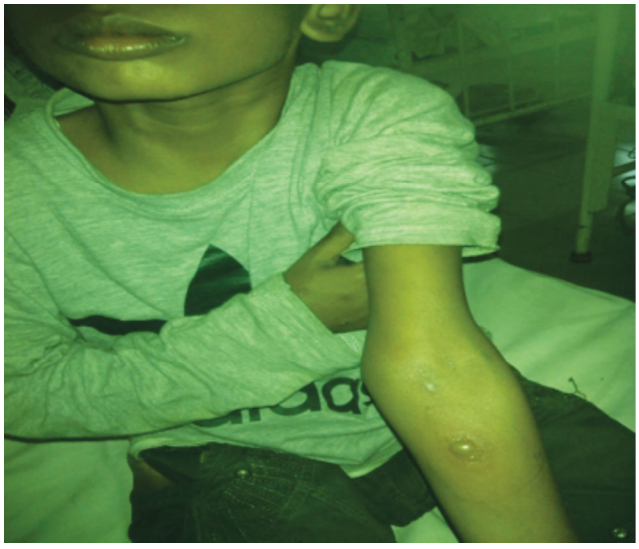

Fig: 1 : Boy with positive MT with blister formation

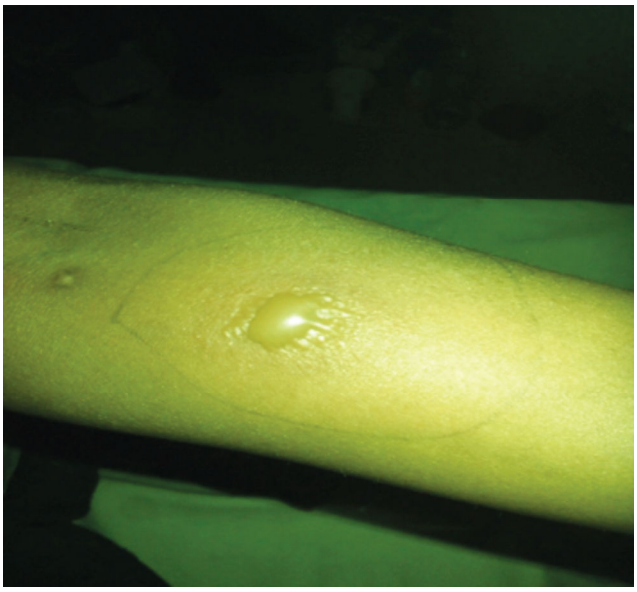

Fig : 2 Large area of indurations with blister formation

CT/MRI scans of the brain

Multiple5 mm axial NECT and CECT scan of the brain was done which shows multiple ring like lesions with rim enhancement, with perilesional hypodense areas, are noted in left frontal, both parieto-occipital region of the brain. Tiny calcification was noted in the hypothalamic area. Frontal horn of the left ventricle was mildly dilated with midline shift to right. Rest of the brain including the scull bone was normal. Report was suggestive of tuberculoma with brain oedema with mild mass effect. MRI shows multi focal T2- FLAIR roundish, conglomerating hypointense mass lesion with huge peri-lesional oedema in left parito-occipital, left frontal and right cerebral hemispheres. After intravenous contrast strong rim as well as nodular enhancement is noted in all the masses. Mass effect is evident by compression on left occipital horn. Evidence of peri-lesional meningeal enhancement is also seen.

Treatment:

On admission child was treated ceftriaxone injection and diazepam for convulsion. Within 48 hours of his admission anti- tubercular drug with RIMCURE 3 FDC (rifampicin 150mg+Isoniazide75 mg+Pyrazinamide 400mg/Tablet) 4 tablets before breakfast, strptomycine with prednisolone injection and pyridoxine. RIMCURE 3 FDC and strptomycine given for 2 months followed by rifampicin and Isoniazide for 10 months. Clinical improvement was observed within48 hours only by remission of headache. Child was vitally stable on regular follow up and there was no complications were observed. After discharge from the hospital progress was assessed regularly in the out-patient clinic and serial CT/MRI examinations were done which showed definite improvement after 12 months treatment and planned to continue for 18 months for complete radiological cure.

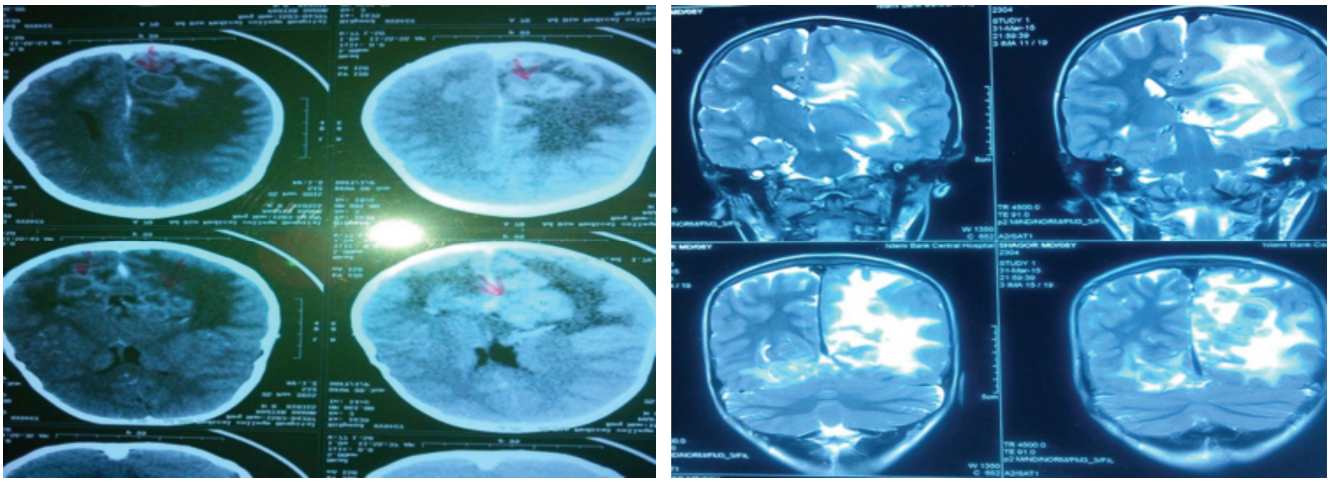

Fig 3 and 4 : CT SCAN Brain -Multiple lesions with peri-lesional hypodense areas with mass effect.

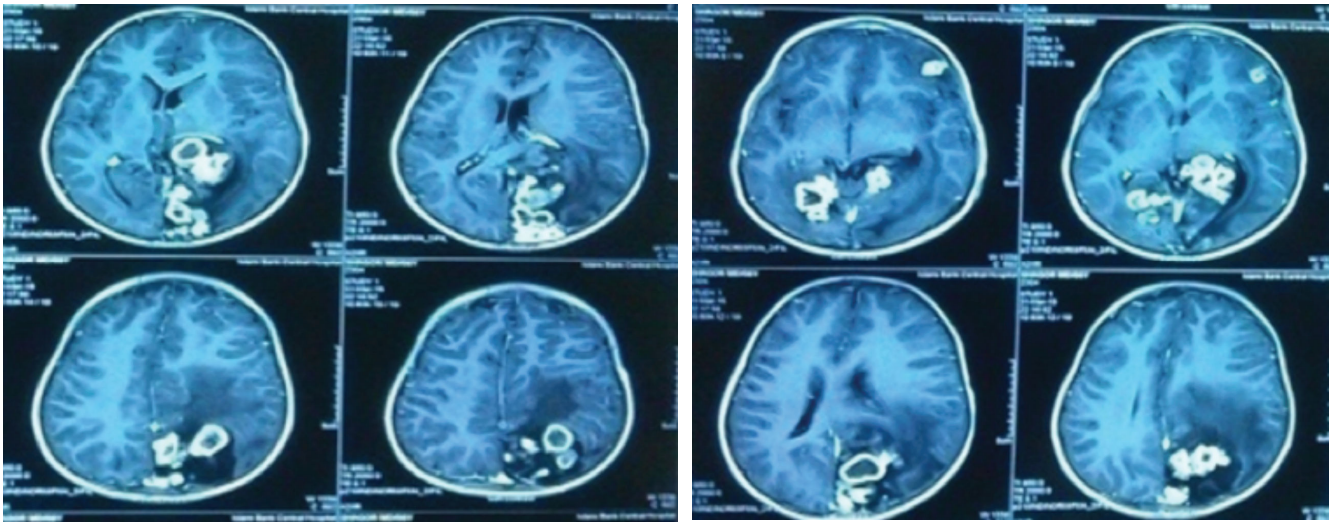

Fig 5: Multiple ring like lesions with rim enhancement after contrast, with perilesional hypodense areas in parieto-occipital region.

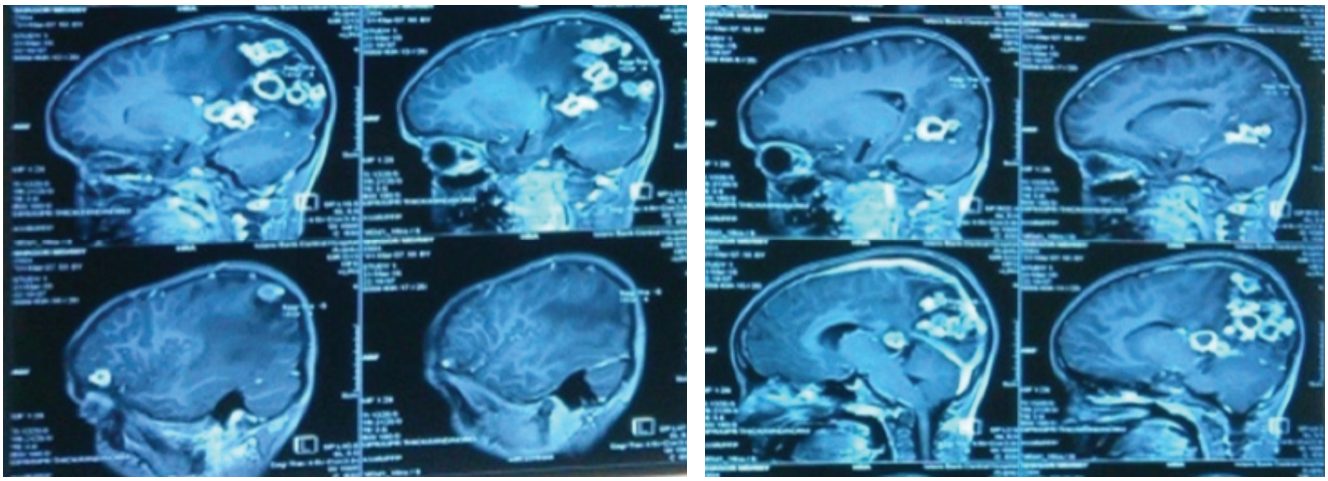

Fig 6: Huge peri-lesional oedema in left parito-occipital, left frontal and right cerebral hemispheres.

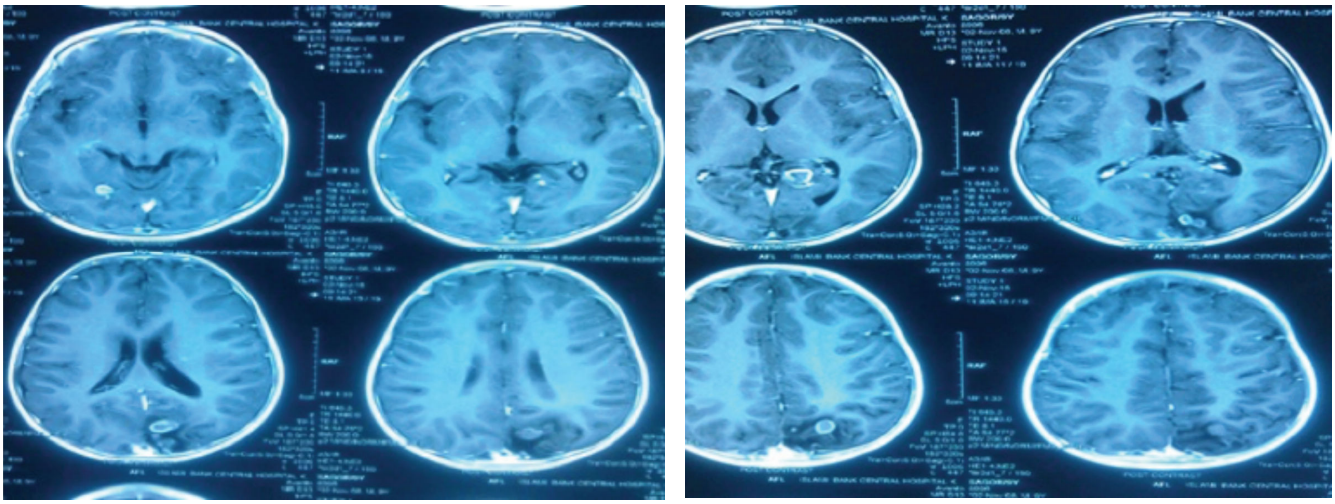

Fig 7: After 9 month treatment with anti TB medications shows improvement on CT SCAN of brain

Discussion

Tuberculomas are firm, avascular, spherical granulomatous masses, measuring about 2–8 cm in diameter. They tend to be located infratentorially in the pediatric population and supra-tentorially in adults. They are well limited from surrounding brain tissuewhich is compressed around the lesion and shows oedema and gliosis. Insideof these masses may contain necrotic areas composed of caseous material, occasionally thick and purulent, in which tubercle bacilli can be demonstrated. Intracranial tuberculomas can occur at any age. In developing countries young adults and children are predominantly affected while in developed countries they are more common in older patients. The symptoms produced by tuberculoma are related to their location. Low-grade fever, headache vomiting, seizures, focal neurological deficit, and papilloedema are characteristic clinical features of supratentorial tuberculomas. Intratentorial tuberculomas are more common in children and may present with brainstem syndromes, cerebellar manifestations and multiple cranial nerve palsies.[8,9,10] Numerous intracranial lesions were found in our patient. However, tuberculomasare usually solitary lesions. Studies

have shown that 15% to 34% of casespresent with multiple lesions.[11] On CT, tuberculomas are characterized as low- or high-density and rounded or lobulated masses and show intense homogenous or ring enhancement aftercontrast administration. They have an irregular wall of varying thickness. Moderateto marked perilesional oedema is frequently present. Tuberculomas may be single or multiple and are more common in frontal and parietal lobes. On CT, the 'target sign', a central calcification or nidus surrounded by a ring that enhances after contrast administration, is considered pathognomonic of tuberculoma.[12] On CT scanning, tuberculoma measure more than 20 mm in diameter, are frequently irregular in outline, and are always associated with marked cerebral oedema with midline shift and progressive focal neurological deficit. The most important factor affecting the prognosis of cerebral TB is early initiation of treatment. In one study 14 patients with tuberculomas of the brain were treated with anti-tuberculous drugs and all of them irrespective of their size, cured by medical treatment.[13] Our case was treated with anti- TB drug only. Surgery is needed with large tuberculoma with space occupy effects even after medical treatment.[14] Another indication of surgery if there is any diagnostic confusion for asrtocytomas. Surgery is also indicated in post tubercular hydrocephalus but surgery can be avoided in 70% cases with conservative management with mannitol, frusemide, acitazolamide and of course dexamethasone.[15]

Conclusion

The diverse clinical manifestations of CNS tuberculosis, a common neurological disorder in developing countries as well as in developed world. Early recognition and timely treatment of CNS tuberculosis is very crucial in order to reduce the mortality and the morbidity of the disease.

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