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EDITORIAL

Micronutrients In Pregnancy- An Often Neglected Issue

Pregnancy is a period of increased metabolic demand with changes in the woman's physiology and the requirements of a growing fetus . During this time, inadequate stores or intake of vitamins or minerals, referred to collectively as micronutrients, can have adverse effects on the mother, such as anemia, hypertension, complications of labor and even death[1]. Furthermore, the fetus can be affected, resulting in stillbirth, pre-term delivery, intrauterine growth retardation, congenital malformations, reduced immunocompetence and abnormal organ development. Recent research suggests that even after the period of infancy, the health of the child and the adult can be influenced by the foetal period. Coronary heart disease, hypertension and type 2 diabetes are thought to originate, in part, from impaired intra-uterine growth and development. These diseases may be a consequence of "programming" whereby a stimulus or insult at a critical, sensitive period early in life has permanent effects on structure, physiology and metabolism [2].

During pregnancy, metabolic changes occur that protect the mother and her pregnancy through an increased metabolic efficiency. The foetus is also relatively protected at the cost of the nutritional status of the mother For micronutrients, similar mechanisms seem to be in place. In a deficiency state of the mother, the foetus will be in part protected with a higher stress on the mother.

Micronutrients :

Iron : Iron is one of the major trace element required during pregnancy from conception, throughout the pregnancy and during lactation. Iron deficiency can cause maternal anaemia , preterm labour and low birth weight[3]**.** Increased iron requirements in pregnancy are not often met by changes in diet or absorption. Iron supplementation during pregnancy has been shown to improve iron stores and reduce anemia**,** which might be expected in turn to reduce the risk of death from complications of pregnancy, such as hemorrhage and also morbidities like sepsis. The recommended daily intake of iron for pregnant women is 27 mg/day.Good dietary sources of iron (more than 2mg/serve) include liver, beef ,fortified cereals, cashew nuts, baked beans etc.Iron supplementation during pregnancy has been recommended by national and international bodies and has become the standard of care in most setting.

Zinc : Zinc deficiency has been associated with complications of pregnancy and delivery, such as pre-eclampsia, premature rupture of membranes, and pre-term delivery, intrapartum haemorrhage, infections and prolonged labour and with fetal growth retardation and congenital abnormalities [4]**.** Trials in developing countries have found that babies whose mothers were given zinc supplements in pregnancy have improved immune function and a reduction in diarrhea and respiratory illnesses in infancy, suggesting effects on immune competence that persist beyond birth [5]**.** Recommended dose in pregnancy is 11mg/day and 12 mg/day during lactation. Firm evidence to warrant a supplement during pregnancy is, however, still lacking.

Iodine : Iodine deficiency during pregnancy is responsible for development defects of the foetus, cretinism and possibly fetal wastage or pre-term delivery, mental retardation, deaf mutism, spastic diplegia, squint, hypothyroidism and dwarfism and the pathologies associated with endemic goitre. Iodine supplementation studies have shown beyond doubt that supplementing iodine during pregnancy can reverse the described abnormalities**.** Maternal health seems not directly affected by iodine deficiency. Salt fortification is now widely practiced throughout the world with an impressive decrease in associated morbidity [6]**.** Recommended dose is >220 micro gram/day. Food sources are sea fish, shellfish, cereals, and grains and fortified salt.

Magnesium : Magnesium is an essential mineral needed in relatively large amounts by humans. In a number of

retrospective studies magnesium levels during pregnancy were found to be associated with the risk of seizures in pre-eclampsia, prematurity, preterm labour and low birth weight [7]. Unfortunately the authors of the Cochrane review concluded that there is at present not enough evidence to show that dietary magnesium supplementation during pregnancy is beneficial.

Selenium : Selenium is thought to have an antioxidant property. Deficiency can result in osteoarticular disorders, cardiac enlargement arrhythmia, heart failure. It may increase risk of cretinism. Recommended dose during pregnancy is 65 microgram/day and to 70 microgram/day during lactation .Food sources are nuts, beef, meat, fish, egg, bread.

Calcium : Deficiency of calcium, may also be associated with abnormal fetal development, pregnancy induced hypertension, and preterm delivery [8]. Adequate calcium supplementation during pregnancy lactation is essential for fetal bone mineralization. A number of observation studies led to the hypothesis that an increase in calcium intake during pregnancy might reduce the incidence of high blood pressure and pre-eclampsia among women with low calcium intake [9]. Recommended dose during pregnancy is 1000-1500 mg/ day. Food sources are yoghurt, cheese, milk and milk product, orange juice, etc

Vitamin A : Vitamin A deficiency during gestation can lead to fetal wastage, growth retardation, preterm birth, low birth weight, placental abruption, although high doses of vitamin A in early pregnancy can be teratogenic as well [10]. In a large vitamin A supplementation trial in Nepal on 44646 women were followed [11] and showed that weekly vitamin A or beta-carotene supplements reduced maternal mortality by 50%. In women at risk of pre-eclampsia a supplement does have a benefit. Vit .A supplementation can increase the hemoglobin concentration by about 4 gm/L in marginally deficient maternal population .In situations where women are deficient it is warranted to correct the deficit to protect the newborn on breast milk. In the first months of life daily intake of 800 microgram is recommended.

Folic Acid : Folate is critically important for foetal development. It is a cofactor essential in the ultimate methylation process of DNA. Interference with DNA synthesis gives rise to abnormal cell division specially rapidly dividing cells, such as those in the haematopoetic system, One of the clinical manifestations of folate deficiency is macrocytic anaemia . There is no doubt that folic acid deficiency is directly linked to neural tube defects. A recent Cochrane review [12] reveals that periconceptual folate supplementation reduced the incidence of neural tube defects by as much as 70% (odds 0.28 C.I. 0.15-0.53). Women with habitual abortions and who have given birth to offspring with neural tube defects had a higher prevalence of hyperhomocysteinemia [13]. Folate supplements reduce significantly the homocysteine concentrations. Limited data suggest that folic acid supplementation of pregnant women may improve fetal growth and reduce the incidence of low birth weight. Improvement in food quality and the use of fortified products seem the only effective strategy. Daily recommended dose is 600 microgram/day .

Vitamin D : Vitamin D helps in absorption of calcium and phosphorus from dietary intake which are required for stimulating bone formation of the fetus. Deficiency of Vit D during pregnancy may result in an infant with rickets or type 1 diabetes mellitus [14]. Recommended daily intake during pregnancy and lactation is 5 microgram/day. Food sources are cod liver oil, milk, oily fish, eggs, cereals.

Vitamin C : Vit.C stimulates better absorption of iron and therefore helps to reduce the risk of maternal anemia .As an antioxidant it guards the body against injurious free radicals. A few studies have shown that vitamin C deficiency plays a role in some pregnancy complications, such as premature rupture of membranes (PRM) and pre-eclampsia). Two such recent trial found that provision of vitamins C and E resulted in a 60 % lowering the rate of preeclampsia [15].The recommended daily intake in pregnancy is 60 mg/day. Dietary sources are various fruits, and vegetables.

Vitamin B group : (1) Vitamin B1 (Thiamine), (2) Riboflavin(B2) , (3) Nicotinic acid (B3), (4) Vitamin B6, (Pyridoxine), (5) Vitamin B12 (Cobalamin)

The group of B vitamins is essential for enhancing the immune system as well as reducing the plasma

concentration of homocysteine which may lead to pre-eclampsia, premature birth, and low birth weight. A recent controlled trial in HIV-infected women using high-dose B vitamins, as well as vitamins C and E, found reductions in intrauterine growth retardation and pre-term births, as well as a reduction in perinatal mortality [16]. Dietary sources are animal products including meat, eggs, fish .and milk. Daily recommended doses are: vit. B1-1-4 mg/day, Vit .B2-1.4mg/day, Vit B3-18mg/day. VitB6-1.9mg/day, Vit.B6-1.9mg/day, Vit B12-2.6microgram/day and folate-600 microgram/day.

There is a large body of evidence supporting the concept that deficiencies in micronutrients adversely affect maternal health and pregnancy outcome. It is important to underline here that not one micronutrient alone is responsible for these adverse effects. . It is therefore very unlikely that the supplementation or correction of one deficiency will yield high effects, as long as other deficiencies remain. For some deficiencies the maximum effect of correction is found when this happens in early pregnancy. For folic acid the supplement should ideally be given before conception and the highest effects of an iron supplement can be expected when taken in the first trimester. In developing countries unfortunately women usually don't consult for a pregnancy until well in the second half of pregnancy.

It seems thus that apart from an iodine fortification program, there is little scope for improving the micronutrient status of pregnant women with supplementation programs alone. Hope of achieving an improvement must lie in upgrading the nutritional status of women of childbearing age in general and providing nutritional advice during pregnancy. A nutrition approach should be integrated in antenatal care programs and they will need an intersectoral approach given the multicausal nature of the problem. In addition, more attention should be focused on dietary approaches, including fortification of foods with micronutrients, which may prove to be more beneficial and sustainable than provision of supplements during pregnancy. Additional research is needed on the possible beneficial and harmful effects of multiple or more selective micronutrient supplements in pregnancy before universal recommendations can be made for developing country populations.

(J Bangladesh Coll Phys Surg 2012; 30:)

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Can Succinylcholine be Used Safely in Severely Burn Patients?

H BEGUM[a], MS ISLAM[b], UHS KHATUN[c]

Summary:

The use of succinylcholine(SC) in burn patients are relatively contraindicated for certain period after lesion aged 4 days – 10 weeks due to chance of hyperkalemia although there are no systemic data to define what period and what level of K^+ is safe. This prospective study was carried out in 60 acute burn patients who were admitted in DMCH Burn Unit and undergone surgery within 3 months of lesion. Most common type of burn was flame burn (33%).Mean age of the patient was 22.60 ± 9.61, TBSA (Total burn surface area) was 22.17 ± 9.57 and duration of burn was 23.36 ± 19.61. Every patient received standard dose of SC (1.5mg/kg) for intubation. The peripheral venous blood samples for serum K^+, Na^+, Cl^- & HCO_3^- were drawn before induction and 3 minutes after injection of SC. On analysis there were no significant change of serum K^+ and HCO_3^- ($p > .05$), on the other hand serum $Na^+$and Cl^- levels were significantly changed ($p<0.05$) due to correction of dehydration. In case of electric burn serum K+ level was raised in every cases but didn't cross the normal high level of serum K+ (5.5mEq/l). Haemodynamic parameters like pulse, NIBP, SPO2 and ECG were analyzed intra operatively and there were no significant change in NIBP and ECG, rather there were significant improvement in pulse and SPO2 ($p< 0.05$). Survival of anaesthetic was 100% and no dysrhythmias or major morbidity were found intra operatively. Therefore, these data taken in the context of a compelling case for rapid intubating condition suggest safety in succinylcholine use in the patients with acute burn.

(J Bangladesh Coll Phys Surg 2012; 30: 5-9)

Introduction:

Since the 1950s, cardiac arrest has been observed in burn patients receiving succinylcholine. A decade later, hyperkalemia was determined to be the cause of cardiac arrest in these patients. Since then, other conditions have been identified that may result in succinylcholine induced hyperkalemia including cerebral vascular accident (CVA), spinal cord injury, Gullain-Barre syndrome and prolonged stays in an intensive care unit (ICU).[1] Further exploration of this phenomenon has been identified upregulation of nicotinic acetylcholine receptors (AchRs) in skeletal muscle as the cause of hyperkalemia.[2] In healthy persons, postsynaptic acetylcholine receptors are restricted to N-M junction. Acetylecholine receptors on postsynaptic membrane are organized in discrete clusters on the shoulders of junctional folds. Each cluster contains a few hundred receptors. Each receptor consists of few subunits, two of which, the alpha (á), are identical. Other three are beta (b), delta (d) and epsilon (e). Receptors are arranged as a cylinder, with a central, normally closed channel-the inophore. Each acetylcholine molecule is involved in opening one ion channel. Acetylcholine receptors are also present on presynaptic area of nerve terminal.[3] but in severe burn, where N-M junctions are affected acetylcholine receptors develop on the adjacent muscle surface. These extrajunctional receptors cause excessive release of k^+ ions from severe burn tissues.[3] Hyper metabolism is also seen with a burn injury of greater than 20% total body surface area. These causes double the cardiac output and metabolic rate over the next 24 to 48 hours. Tissue destruction especially from electrical burn causes hyperkalaemia.[4] Serum potassium may increase 0.3-0.5 mEq/l following administration of paralyzing dose of succinylcholine (SC) in normal patients and last for 3-5 minutes. But in burn patients, massive hyperkalaemia may occur after administration of succinylcholine(SC) for certain period after lesion eg. 4days- 10weeks.[5, 6] in our country, there is no such study, especially on burn patient. We therefore decided to perform such type of study to assess the safety of acute burn patients. This study was carried out to observe morbidity of severe burn patient during anaesthesia

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using succinylcholine and to observe the level of K^+ after using succinylcholine crossing critical level.

Materials and methods:

This prospective clinical study was conducted after obtaining approval from the institutional ethical committee and was carried out in the 50 bedded burn unit, the only dedicated burn facility in Bangladesh with a mean annual admission of 869 patients, under the guidance of professor and head, department. of Anesthesia and ICU of Dhaka Medical College Hospital. Patients were recruited randomly. Purpose of the study was clearly explained to each of the subjects and was recruited only after taking informed written consent. Patient with deep burn >5%- 60% and aged >1 year were included and patients with impaired renal function , failed intubations and the patients were not interested to be included in the study were excluded. All patients were given ideal general anesthesia with same I/V fluid and anesthetic agents eg. Hartman's solution, Inj. Pethidine (1 mg/kg), Inj. thiopental Na (5mg/kg), Inj. Suxamethonium (1.5 mg/kg). 1st blood sample was taken before induction and 2nd blood sample was taken 3 minutes after giving injection succinylcholine. Pulse, noninvasive blood pressure, and electrocardiography (to see any dysrhythmias) were observed in all patients. Readings were recorded according to data collecting sheet, before induction and after giving suxamethonium at 5 min, 10 min, 30 min and 1 hour after suxamethonium injection. Biochemical parameters such as serum potassium level, serum sodium level, serum chloride level, serum bicarbonate level and clinical parameters such as pulse, blood pressure, electrocardiography and SPO2 were analyzed. Data were collected in a specially designed Data collecting sheet. It was analyzed for statistical significance by t-test and ANOVA as appropriate and descriptive statistics were used for demographic profiles. Values was regarded as significant if $p<0.05$.

Results:

A total 60 people were included in this study. Their characteristics including gender, distribution of age, cause of burn, and TBSA burnt and duration of burn are presented in Table - 1. The most common type of burn was flame burn (45%), mean age was (22.6 ± 9.61) and mean duration of burn was (22.36 ± 19.61) days.

The haemodynamic parameters before induction and 5 min, 10 min, 30 min and 60 mins after injection of succinylcholine (SC) were described in Table-II.

Results shows significant improvement of pulse and SPO2 after induction expressed in Table-III.

All patients received a standard dose of succinylcholine (1.5mg/kg.) so called defasciculating doses of nondepolarizing muscle relaxants were not administered before SC. The laboratory parameters were described in Table-IV. The mean baseline value of serum potassium (K+) 4.37 ± .34 and it was not significantly changed 3 minutes after injection of succinylcholine (SC). Serum potassium level increased in all cases of electric burn but didn't cross the normal limit of serum level of potassium (5.5 mEq/l). But serum Sodium (Na+) and Chloride (Cl -) level decreased significantly.

There were no significant changes in laboratory parameters between groups and within groups (ANOVA) shown in Table-V.

Table-I

Characteristics of Patients (n-60)

Age of the Patients			Gender of the patients				Causes of Burn			TBSA % of burn		
Age in yrs	No	%	Male		Female		Type	Number	%	% of burn	No	%
0-10	6	10	No.	%	No.	%	Scald	6	10	<10	6	10
11-20	24	40	33	55	27	45	Flame	27	45	11-20	15	25
21-30	24	40					Electric	21	35	21-30	24	40
31-40	3	5					Gas Explosion	0	0	31-40	9	15
>40	3	5					Chemical	6	10	41-50	1	5
										>50	1	5
Duration of burn			minimum – 4 days				maximum – 78 days			mean – 22.36 ± 19.61		

TBSA = Total Burn Surface Area

Table-II

Haemodynamic changes before and after induction with succinylcholine (SC) (n = 60)

Parameter	Before induction	5 min after induction	10 min after induction	30 min afte r induction	60 min after induction
Pulse	109.0 ± 19.24	108.3 ± 18.30	98.1 ± 23.4	99.5 ± 22.2	95.7 ± 20.2
NIBP systolic	112.7 ± 15.20	109.2 ± 15.00	106.2 ± 12.7	114.2 ± 17.4	111.5 ± 13.1
NIBP diastolic	68.7 ± 16.20	67.2 ± 10.90	67.0 ± 14.0	78.0 ± 12.5	72.7 ± 11.4
SPO2	98.0 ± .88	98.6 ± .58	98.4 ± .82	98.6 ± .67	98.6 ± .48
ECG	1.0 ± .00	1.0 ± .00	1.0 ± .00	1.0 ± .00	1.0 ± .00

Results were expressed as mean ± SD.

Table-III

Haemodynamic changes before and after induction with succinylcholine (SC) (n = 60)

	Parameters	Mean	N	Std. Deviation	Std. Error Mean	TValue	PValue
Pair 1	Pulse before	109.0000	60	19.24633	4.30361	.225	.825
	Pulse_after_5_min	108.3500	60	18.37697	4.10922		
Pair 2	Pulse before	109.0000	60	19.24633	4.30361	2.789	.012*
	Pulse_after_10_min	98.1000	60	23.42266	5.23747		
Pair 3	Pulse before	109.0000	60	19.24633	4.30361	3.316	.004*
	Pulse_after_30_min	99.5000	60	22.22966	4.97070		
Pair 4	Pulse before	109.0000	60	19.24633	4.30361	4.532	.000*
	Pulse_after_60_min	95.7500	60	20.29487	4.53807		
Pair 5	NIBP before	112.7500	60	15.25873	3.41196	1.113	.279
	NIBP_after_5_min	109.2500	60	15.06783	3.36927		
Pair 6	NIBP before	112.7500	60	15.25873	3.41196	2.054	.054
	NIBP_after_10_min	106.2500	60	12.76044	2.85332		
Pair 7	NIBP before	112.7500	60	15.25873	3.41196	-.356	.726
	NIBP_after_30_min	114.2500	60	17.49248	3.91144		
Pair 8	NIBP before	112.7500	60	15.25873	3.41196	.398	.695
	NIBP_after_60_min	111.5000	60	13.18891	2.94913		
Pair 9	NIBP before Dias	103.7500	60	164.21163	36.71884	.992	.334
	NIBP_after_5_min_Dis	67.2500	60	10.93943	2.44613		
Pair 10	NIBP before Dias	103.7500	60	164.21163	36.71884	.956	.351
	NIBP_after_10_min_Dis	67.0000	60	14.08620	3.14977		
Pair 11	NIBP before Dias	103.7500	60	164.21163	36.71884	.822	.421
	NIBP_after_30_min_Dis	73.0000	60	12.50263	2.79567		
Pair 12	NIBP before Dias	103.7500	60	164.21163	36.71884	.829	.417
	NIBP_after_60_min_Dis	72.7500	60	11.41041	2.55144		
Pair 13	SPO2_before	98.0500	60	.88704	.19835	-2.854	.010*
	SPO2_after_5_min	98.6500	60	.58714	.13129		
Pair 14	SPO2_before	98.0500	60	.88704	.19835	-1.506	.148
	SPO2_after_10_min	98.4500	60	.82558	.18460		
Pair 15	SPO2_before	98.0500	60	.88704	.19835	-2.259	.036
	SPO2_after_30_min	98.6500	60	.67082	.15000		
Pair 16	SPO2_before	98.0500	60	.88704	.19835	-2.449	.024
	SPO2_after_60_min	98.6500	60	.48936	.10942		

P < .05 = Significant; * = Significant.

Table-IV

Laboratory parameter changes before induction and 3 minutes after induction with succinylcholine. n = 60

Parameter	Serum level before induction	Serum level 3 minutes after induction	T value	p value
Serum potassium (k+)	4.37 ± .34	4.50 ± .50	- 1.301	.209
Serum Sodium (Na+)	143.54 ± 3.33	142.10 ± 3.33	3.131	.006*
Serum Chloride(Cl -)	104.12 ± 3.67	103.37 ± 3.68	2.76	.012*
Serum bicarbonate(HCO3-)	24.50 ± 3.25	24.37 ± 2.89	.709	.487

Results were expressed as mean ± SD. p < .05 = Significant; * = Significant.

Table-V

Changes in laboratory parameters between groups and within groups (ANOVA); n = 60.

Parameter	Sum of squares between groups df -2	Sum of squares within groups df -58	Sum of squares total df -60	Mean square between groups & within groups	F value	sig
Serum potassium (k+) difference	.467	2.850	3.316	.233.168	1.392	.276
Serum Sodium (Na+) difference	23.589	56.799	80.388	-11.7953.341	3.530	.052
Serum Chloride(Cl -) difference	1.111	26.839	27.950	.5551.579	.352	.708
Serum bicarbonate(HCO3-) difference	2.268	10.514	12.782	1.134.618	1.834	.190

p < .05 = Significant; * = Significant.

Discussion:

It is well known that it is inadvisable to administer succinylcholine to burn patient. there are reports of cardiac arrest during intubation of such patient in the 1960s. These occurred between the 20th and 50th days post injury. [7] The cause was found to be a high plasma potassium level immediately following the administration of the muscle relaxants. [8, 9] There are some case reports and letters have been published reporting hyperkalemia after administration of succinylcholine to patients with burn. In our study we also found mild increase in serum potassium (k^+) level after administration of succinylcholine but we couldn't call it hyperkaleamia (serum potassium (k^+) > 5.5 mEq/l). The level of serum potassium (k^+) in our study was 4.50 ± .50 mEq/l. Dr.Charles D.Deakin reported that succinylcholine contraindicated in burns because of the risk of hyperkalemia. Probably due to entire myocyte cell membrane acting as a receptor. [10] In our study we attempted to evaluate whether any significant morbidity or mortality ensued from the administration of an induction dose of succinylcholine with moderate to severe burned patients within 3 months of injury of different type of burn like flame burn, scald, electric burn, chemical burn etc. And experiences were uneventful although there was transient increase in serum potassium (k+) level. Our findings matched with the study of Adam J. Schow MD et al.[6] who reviewed more than 40,000 general anesthetics administered over 70 months in which succinylcholine was given at the induction. This search yielded 38 patients with a preoperative potassium of 5.6 mEq/l or greater. Survival of the anesthetic was 100% and no dysrhythmias or other major morbidity were documented upon manual review of the intraoperative automated record keeper charts or the patient medical records. Rebecca M. Huggins et al.[2] found administration of succinylcholine to patients without neuromuscular disease results in a small transient increase in a serum potassium (k+) concentration of about .55 mEq/l and serum potassium (k+) increase greater than 5 mEq/l are extremely rare. Maclennan et al.[11] suggest that succinylcholine should not be used beyond 24 hours after a burn injury. However there are no reports in the literature of succinylcholine induced hyperkalemia in human occurring within 1 week after burn injury. Some other investigators found succinylcholine is safe up to 1 week after burn injury, were performed 30 years ago. [9, 12, 13] In these reports

number of patient studied within 1 week after burn injury totaled only three in the three publications, may be the treatment modality at this time was that most burn patients, especially those with big burns did not undergo excision and grafting until the burn escher had separated from the wound, which takes at least 2 weeks for spontaneous separation. Current practice advocates early excision and grafting of wound especially with major burn. [11] Martyn J.A. Jeevendra[14] states that the upregulation of acetylcholine receptors (AChRs) after burns occurs at sites immediately beneath and distant from the burn, a positive correlation between AChR number and the intensity of hyperkalemia after succinylcholine. The upregulation of acetylcholine receptors (AChRs) occurs in muscles beneath the area of burn is as profound as denervation and occur as early as 72 have after burn. Evidence for upregulation of the immature isoform has also been provided by assessment of messenger RNAs for the ã subunit. When depolarized, the immature isoform has a prolonged open channel time, which may aggravate the K^+ efflux that occur with depolarization. Yanez P. et al.[15] reconfirmed that AChRs upregulation with expression of the ã subunit as early as 3 days after a 5% burn over tibialis anterior muscle in rat. In view of Martyn J.A. Jeevendra[14] succinylcholine is probably safe up to 48 hours after burn injury, but it may be wise to avoid it beyond that periods. Geraid A. Gronert, M.D. [16] worked long time since 1959 to 2009 on this topic in different burn unit with the doses of succinylcholine of 0.4, 0.7, and 1.4 mg/kg unlike our result he found succinylcholine was unequivocally contraindicated in the patients with burns. J.A. Jeevendra Martyn [17] also found hyperkalemia with succinylcholine. E. J. Halok et al. [18] found safe (as indicated by an absence of arrhythmias and other morbidity) in patients with preoperative serum concentrations of 5.6 mEq/l or greater. In our study we studied 60 patients with 6 – 50% of mixed to deep burnt patients within 4 – 78 days of injury with succinylcholine without any adverse effect except mild transient increase in serum potassium (K+). We also found that the increase in serum potassium (K+) level was more in case of electric burn relative to other modalities of burn patients like flame burn, scald or chemical burn.

Conclusion:

Succinylcholine can be used safely for major burn patients for early excision and grafting as well as correction of deformity within 3 months of injury. However further study needed to support this inference as there is other work in this field in our country.

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Complications and Immediate Outcome of Pregnant Diabetic Women

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Summary:

Aim: To see complications and immediate outcome among pregnant diabetic women.

Methods: This observational retrospective study included sixty nine pregnant women with diabetes (both pre-gestational and gestational diabetes) those who got admitted and treated at Dhaka Medical College Hospital (DMCH), Dhaka Bangladesh from the 1st August 2007 to the 31st August 2008. Detailed analysis of their obstetric history, ante partum and intra partum complications and mode of delivery were performed.

Results: Majority of the women (76.92%) were admitted through labor emergency. Mean age of the women was 28.9 (18-45) years. Fifty four (54%) percent of women belonged to 21-30 years age group. Sixty two (62.31%) percent of women had gestational diabetes whereas 37.68% had pre-gestational diabetes. All women were followed up both by obstetrician and diabetologist. Twenty five percent(25%) women developed pre-eclampsia and pregnancy induced hypertension, thirteen percent(13%)women developed premature ruptur of fetal membrane(PROM), twenty three percent (23.25%)women had fetal distress, three(2.88%) percent women present with ante-partum hemorrhage(APH) and one percent(1.44%) women develop acute poly-hydromnios. Average gestational age was 36.83(41-28) weeks. Sixty percent (60%) delivered after 37weeks of gestation and forty one (40.58%) delivered before 37weeks of gestation. Ten percent women delivered vaginally and ninety percent (90%) women delivered by caesarean section (CS) because of post CS, repeat CS, breech presentation, preeclampsia, fetal distress and obstructed labor. Maternal mortality is 1.44% and peri-natal is mortality 8.62%.

Conclusion: Ante-partum and intra-partum complications are more common among pregnant diabetic women. Knowledge of the importance of maternal glycemic control, as well as development of surveillance techniques to prevent complications, resulted in a decline in fetal and neonatal mortality.

Key word: ***Pregnancy, diabetes, complications, outcome.***

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Introduction:

Diabetes mellitus is the commonest endocrine disorder during pregnancy and it complicates about 1-2% of all pregnancies [1,2]. Prior to the introduction of insulin, perinatal mortality from this complication was the order of 65% [1,2,3,4]. This has fallen drastically with the introduction of medical obstetric clinic, to a level almost equal to that in non diabetics provided glycemic control is good and tight [1,2,3,4]. In fact many pre-diabetics (IFG and IGT) and potentials diabetics may show chemical evidence of Diabetes mellitus during the course of metabolic stress of pregnancy. In case of Gestational Diabetes Mellitus (GDM), where glucose homeostasis return back to normal after delivery. It increases various risk to the mother and fetus .The duration and severity of maternal diabetes mellitus and quality of its control during pregnancy determine the outcome of pregnancy [5,6]. Pre-eclampsia occurs approximately twice as frequently in diabetic as non-diabetic pregnancies [3]. The risks of preterm labour are three fold higher than that in non diabetics [1,4,7]

Fetal surveillance is important but there is no reliable method for this (ante partum and intra partum). A

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combination of methods must therefore be employed. The value and nature of ante partum fetal monitoring in well controlled GDM remain poorly defined, it is generally accepted that antenatal fetal monitoring may minimize the risk of adverse outcome in complicated pregnant women[2]. There is no universally agreed monitoring method [1,2,3,4].

Risk factors for the occurrence of still births included poorly controlled diabetes, fetal macrosomia, co-existing vasculopathy or pre-eclampsia and hydramnios [1,2,8]. In the past, sudden still birth and unexplained IUD were more common after the 36th week of gestation [1,2]. Hyperinsulinaemia and frequent fluctuations in random glucose level have been described as being responsible for the still Birth[1,4]**.** Vasculopathy induced reduced uterine blood flow and hyperinsuliaemia both causes fetal hypoxia. With better and tighter control of blood glucose level, the incidence of still birth falls dramatically, although it is still higher than in non-diabetic pregnancies[1]. Infant of diabetic mother have 6-9% risk of major congenital anomalies[3], 4% risk of respiratory distress syndrome and 28% risk increased of macrosomia and cardiomegaly(30%) [9]. Macrosomia (birth weight>4000gm) may result in incidence of primary caesarean section***[1]*** or obstetric trauma such as fractured clavicle, Erb's palsy or phrenic nerve palsy due to shoulder dystocia.[2,3]

The timing of delivery in diabetic pregnancies remains controversial. Delivery should be delayed until fetal maturation has taken place, provided that the patient's diabetes is well controlled and antepartum surveillance remains normal.There is little evidence from randomized controlled trials to support either elective delivery or expectant management at term .Many units adopt the policy of elective induction at 38-39 weeks provided the diabetes is well controlled and there is no associated fetal or maternal complication***[1]*** .In presence of complication (either maternal or fetal), delivery should be expedited***[2]*** when the risk of continued intrauterine existence are thought to exceed those of premature delivery particularly respiratory distress syndrome and hyperbilirubinaemia***[3]***. Where glycemic control is good and there is no supervening complication (e.g. abnormal fetal growth) there is no reason to deliver the fetus before 40 weeks of gestation. But some authorities agree that once maturity is achieved, there is little value in delaying delivery ***[1,3]*****.** As pregnancy advances, risk also increases despite good glycaemic control. The major risks are related to operative morbidity, consequent on fetal macrosomia and obstructed labor. The rate of caesarean section in diabetic pregnancies is significantly higher ***[1,2,3,4]*** than that in non-diabetic pregnancies, and may be as high as 50 percent***[1]***. This is partly due to fetal macrosomia with a consequent fear of shoulder dystocia complicating macrosomic vaginal deliveries, fetal distress and also due to failed induction of labors.***[1,3]*** Incidence of fetal distress is high because labor pain stimulates catecholamine***[2]*** release causing glycogenolysis and hyperglycemia. On the other hand uterine contraction causes impaired maternal oxygen release in the utero-placental circulation[1,3].

Aims and Objectives:

The study was designed to find out the complications and immediate outcome of pregnant diabetic women in the third trimester and to compare the results between gestational (**GDM**) and pregestational (**PDM)** diabetic women in labor ward of Dhaka Medical College hospital (**DMCH**), Dhaka.

Methods:

This was a retrospective study done in DMCH from the 1st August 2007 to the 31st August 2008. Data were collected from admission register, patient's file, OT register and report book. During this period total 15283 women were admitted in labor emergency. Among them, 69 had Diabetes Mellitus (PDM and GDM). Irrespective of blood glucose level, all women with pregnancy less than 37 weeks received a course of steroid for fetal lung maturity[3]. Last menstrual period (LMP) and early ultrasound were used for confirmation of duration of gestation. All women with severe pre-eclampsia received a loading dose of magnesium sulphate .Maternal kick count, auscultation of fetal heart sound were used to diagnose fetal distress. We analyzed maternal and fetal complication and outcome depending on type of diabetes and duration of gestation.

Results:

Among 69 pregnant women with diabetes, 43(62.31%) had gestational diabetes (GDM), 26 (37.68%) women had pregestational diabetes (PDM). Fifty-nine percent (59.42%) women progressed to term pregnancy where in 40.58 % pregnant women ended before 37 weeks of gestation. Average pregnancy duration 36.80(28-41)

weeks. Table-l: showed general characteristics of the patients. Table ll: showed age and parity distribution of patients. Mean age of the women was 28.9 (18-45) years. Fifty four percent (53.73%) women belonged to 18-29years age group and 43% percent belonged to 30-39years age group. Twenty four percent women were primi gravida, 21% women were 2nd gravida, 26% women were 3rd gravida and 29% women were 4th gravid and or above. Total 17 (25%) women out of 69 developed hypertensive disorder of pregnancy (PIH and PE). Nineteen percent (19%) women of GDM developed PE in comparison to35% women of PDM, although the difference is not significant statistically (p>0.076) as shown in **table lll.** Sixteen percent (16%) women of GDM developed PROM (preterm rupture of membrane) in comparison to 8% women of PDM. The difference is not significant statistically (p>0.185).One woman of PDM developed severe poly-hydramnios and two women ante-partum hemorrhage (APH). Ten (23.25%) women of GDM developed ante-partum fetal distress in comparison to six (23%) women in PDM, although the result is not significant statistically (p>0.231). Total 62 (90%) women were delivered by cesarean section (CS) as compared to 7(10%) by vaginal delivery. Thirty seven (86%) women of GDM and 25(96%) women of PDM were delivered by cesarean section. The result is not significant statistically (p>0.231). One woman of PDM with PE with IUD died

Table-I

Shows patient's general characteristics

Variables	No of patients(n=69)
Age	28.9(18-45)yrs
Average gravidity	3.03
Average parity	1.38
Gestational age	36.80(28-41)weeks
Term	41(59.42 %)
Preterm	28(40.58 %)
Gestational diabetes(GDM)	43(62.31 %)
Overt diabetes(PDM)	26(37.68 %)

Table-II

Distribution of women according age& Parity (n=69)

Age range	PDM	GDM	Total
18-29yrs	15	21	36(53.73%)
30-39yrs	10	19	29(43.28%)
>40yrs old	1	1	2(2.98%)
Primi gravida	8	8	16(24.24%)
2nd gravid	5	9	14(21.21%)
3rd gravid	6	11	17(25.75%)
4th ≥gravida	7	12	19(28.78%)

Table-III

Pregnancy outcome depending upon type of diabetes. (n=69)

Variables		GDM (n=43)	PDM (n=26)	Total N=69	P-value	Comment
Pre-eclampsia	Yes	8	9	17	0.076	Not significant
	No	35	17	52		
PROM	Yes	7	2	9	0.185	Not significant
	No	36	24	60		
Fetal Distress	Yes	10	6	16	0.231	Not significant
	No	33	20	53		
CS		37	25	62	0.147	Not significant
Vaginal Delivery		6	1	7		
Maternal death	Yes	0	1	1	0.377	Not significant
	No	43	25	68		
Intrauterine death	Yes	3	2	5	0.357	Not significant
	No	40	24	64		

Level of significance 5%

due to septicemia. Regarding peri-natal outcome, Table (lll and lV) showed that live birth 64(91.38%) and IUD 5(8.26%). The occurrence of IUD was higher among GDM as compared to that of PDM (3 and 2 respectively).There were five IUD. Among them four had preeclampsia and in one the cause was unknown. The result is significant statistically (p< 0.01).Average fetal weight 2.89(1.5-3.9) kg. Twenty six percent neonates had weight less than 2.5 (1.5-2.4) kg and remaining 74% had normal birth weight (2.5-3.9kg). There was no case of macrosomia. Nine women had previous bad obstetric history and eight remained uncomplicated in current pregnancy. One woman of GDM had previous history of five IUD and her current pregnancy also ended with IUD.Table V showed indication of caesarean section. Twenty two women had history of previous CS, 5 had breech presentation and 37 women developed ante partum and intra partum fetal distress and delivered by CS. Two women had ante partum hemorrhage. Four percent (4.4%) neonate had severe birth asphyxia and required admission at special care baby unit and 95.55% had normal APGAR score. Table Vl showed pregnancy outcome depending upon complications among PDM and GDM. Among 69 pregnant diabetic women, 49(20+29) women developed complications and 20 (6+14) women remain uncomplicated. Occurrence of PE and fetal distress among complicated PDM and GDM pregnancy are significant (p value are 0.020& 0.011 respectively).Presence of complication also determine duration of gestation and this change is also significant (p value is 0.004)

Table-lV

Shows fetal outcome (n=58)

Variables	Results
Live birth (91.38%)	Average birth weight 2.89kg Normal weight baby(2.5-3.99kg)—73.59% Low birth weight baby(less than 2.5kg) —26.41%
APGAR score	7 and/ or more — (43) 95.55% Less than 6 —(2) 4.44%
IUD (8.62%)	Pre-eclampsia 4(80%) Unknown 1 (20%) 0.01(P value)

Table V

shows Indication of Cesarean section (n=62)

Indication of Cesarean section	No of case
Previous one Cesarean section	17
Repeat Cesarean section	5
Pre-Eclampsia & PIH	16
Intra partum fetal distress	10
Breech presentation	5
Large size baby(weight more than 3.5kg),head high up	6
Ante partum fetal distress(LFM , Oligohydramnios)	6
Bad obstetric history/repeated Pregnancy loss	2
Ante partum hemorrhage	2
Obstructed labor	1
Total	68

*There was overlapping of indications.

Table -Vl

Pregnancy outcome depending on complication.

Variables		Normal (n=20)	Abnormal (n=49)	Total (n-69)	P-value	Comments
Pregestational diabetes Diabetes(PDM)		6	20	26	0.156 significant	Not
Gestational Diabetes(GDM)		14	29	43		
PE and PIH:	Yes	7	9	16	0.020	significant
	Not	38	13	51		
Fetal distress	Yes	2	6	8	0.011	significant
	Not	43	16	59		
PROM:	Yes	6	2	8	0.288	notsignificant
	Not	39	20	59		
Gestational age	≥37 weeks	19	22	41	0.004	significant
	≤37 weeks	4	24	28		

Level of significance 5%

Discussion:

Diabetes is a common medical complication of pregnancy; it is no longer a barrier to conception. The presence of diabetes (gestational and pre gestational diabetes) in pregnancy has been associated with adverse effects on maternal and neonatal outcomes [4]. The incidence of obstetrical and metabolic complications increased, and a continuum has been observed between maternal blood glucose levels and perinatal outcome perinatal mortality, severe congenital malformations, prematurity, and macrosomia [1,8]. Diabetes mellitus is prevalent among 4.8% people of Bangladesh and prevalence of IGT is 8.5%[10]. Among them a significant number are female. Gestational diabetes mellitus (GDM) develops among 6.7% of all pregnancies in our population[11]. In western world 2 to 3% of all pregnancies are currently being diagnosed as GDM [12]. In this study, total number of pregnant diabetic women was 69 and among them 43(62.31%) were GDM and 26(37.68%) were PDM .C B Mahmood [9] in his study of 52 diabetic mother reported that 59.61% had GDM and 40.38% had PDM. The same were found in Begum A [13]and Begum N [14] study. Forty one (59.42%) Women delivered after 37 weeks gestation compared to 28(40.58%) before 37 weeks gestation in this study. Pregnant diabetic women may need to be delivered prematurely due to maternal or fetal problem[2]. C B Mahmood[9] and Ranade *et al* [15] reported 7.6% and 36%of preterm delivery respectively in their study. In this study ,90% women delivered by caesarean section (**CS**) compared to10% by vaginal delivery (VD).[9, 16] S. Mahmuda[17] found 30.23%VD and 69.76% CS, Roksana [18] found 57.45%CS,Metal S reported 38.3%CS, Kasiki OA[19] reported 64.5% VD and 35.5%CS in their study. Regarding perinatal mortality, in this study it was 8.62%. S. Mahmuda [17]found 9.30%, Roksana[18]found 12.77%, Lutale JK et al [20] reported 10% and Huddle K et al[21] reported 6.1% in their study.

The mean birth weight was 2.9(1.5-3.9) kg in the present study. C B Mahmood [9], Ranade *et al*[15], Mohsin F[16] in their study found the mean birth weight of IDM to be 3212±563, 3038±69 and 2970±636gm respectively. In this study there was no case of macrosomia and congenital malformation. Incidence of macrosomia among infant of diabetic mother (IDM) has been reported to be in the range of 20 to32% by C B Mahmood[9], Gabee *et al* [22] and Elliot *et al* [23].Among different pregnancy complications, preeclampsia is commonest and dangerous [1,2,3,4,7]. In this study 17(25%) women had preeclampsia and 4(80%) IUD occurred among pre-eclamptic women. One woman developed left heart failure and admitted in CCU and one woman developed severe PPH, needed caesarean hysterectomy. Occurrence of PE was higher among PDM [2,7] mother as compared to that of GDM mother(35% and 19% respectively) but the difference was not significant

statistically(p>0.076).Incidence of pre-eclampsia among pregnant diabetic women were 9.3%[17], 21.27%[18] ,9.9%[24] and 10.82%[25] found in different studies.

Published data showed incidence of polyhydramnios was 20%[26]. In this study, there was only one case of polyhydramnios. Different study showed the incidence were 6.97%[17], 7.2% [27]and 3.7%[28]. Bivariate analysis (table Vl) revealed strong association between pregnancy outcome, peri-natal loss (p< 0.01), gestational age (p<0.004), and pre-eclampsia (p<0.003).This study revealed that majority of the patient delivered by ceasarean section CS. This might be due to the fact that majority of the patient came with complications such as PIH, pre-eclampsia, IUGR, less fetal movement, bad obstetric history, oligohydramnios and history of previous CS and at the same time previous obstetric history, present pregnancy status, blood glucose level and lack of monitoring facility in the labor ward.

As a result ceasarean section rate increased incidence of birth trauma reduced and rate of premature delivery increased [1,4] .

Conclusion:

Pregnancy in women with diabetes is a high risk one and care must be taken with an aim that both expectant mother and baby must be safe as in a non diabetic person. The abnormal fetal outcome can be changed to a normal acceptable one by pre-pregnancy counseling, optimum antenatal care [29], adequate screening of risk factors followed by proper and timely use of obstetric interventions.

List of abbreviation:

Bad Obstetric history (BOH) Less fetal movement (LFM), Antepartum hemorrhage(APH), Premature rupture of membrane(PROM), Post partum hemorrhage(PPH), Low birth weight baby (LBW), Intrauterine death(IUD), infant of diabetic mother(IDM).

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High Likelihood of Meningitis with Late Onset Septicemia in Newborn

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Summary:

Neonatal meningitis must be recognized and treated quickly to prevent death or disability. Incidence of neonatal meningitis with late onset sepsis is higher in developing countries than those of resource-rich countries. In neonates signs and symptoms of serious infections are often obscured and clinical examination cannot distinguish among septic babies with or without meningitis. Clinicians often differ whether neonates undergo lumber puncture or not to distinguish septic babies with or without meningitis. Abnormal CSF findings are often used to detect neonatal meningitis and determine the type and length of antibiotic therapy with proven sepsis and meningitis cases. This study was conducted to evaluate the bacterial meningitis among the late onset sepsis in newborns and to identify the clinical manifestations that can distinguish septicemia from meningitis in neonates.

Total 1706 admitted patient in NICU of Bangabandhu Sheikh Mujib Medical University from January 2007 to December 2009 were evaluated retrospectively. Among the 133 (27.94%) cases of suspected late onset sepsis 47(35.33%) were proven sepsis, 63(47.37%) were probable sepsis and 23(17.29%) cases were clinical sepsis based on clinical features laboratory reports and blood cultures. Among the proven sepsis 12(42.85%) cases were found to have definitive bacterial meningitis and 16(57.15%) were probable bacterial meningitis. Among the provable sepsis only 1(12.50%) cases were found to have definite bacterial meningitis and 7 (87.50%) cases were probable bacterial meningitis. There were no meningitis have found among the clinical sepsis.

Neonatal meningitis frequently occurred in late onset sepsis. The most frequent presenting clinical features for meningitis cases are more or less similar to those of septicemic cases.

The data of the study suggest that newborns with a positive blood culture are significantly more likely to have meningitis than those with a negative blood culture.

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Introduction:

Bacterial meningitis continues to be a serious problem with high morbidity and mortality.[1-3] Neonatal bacterial meningitis occurs relatively frequently in newborn infant with incidence of 0.2 to 0.5 per 1000 live births based on geographically defined population.[4] Incidence of neonatal meningitis with late onset sepsis is higher in developing countries of South Asian sub-continent than resource- rich countries.[5]

Neonates receiving prolonged intensive care who are prone to nosocomial infection are in high risk group of developing meningitis.[6] Previous studies have reported rates of hospital acquired neonatal infections that are 3 to 20 times higher in resource-poor countries than those of resource-rich countries.[7] There were 49% cases of all sepsis cases occurred between the age 8 to 28 days.[8] About 20% - 30% of neonatal septicemia whether early or late, is complicated by bacterial meningitis.[9-10] Neonates with Early Onset sepsis with obstetric risk factors or respiratory distress alone, lumber puncture may be delayed but lumber puncture should be performed in babies with late onset sepsis because organisms in CSF often affects treatment of choice and duration of the therapy.[11]

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Neonatal meningitis must be recognized and treated quickly to combat preventable death or disability. In Neonates signs and symptoms of serious infections are often obscured and clinical examination cannot distinguish septic babies with or without meningitis. The causative agents of neonatal sepsis vary between geographical areas and with time in any particular locality.[12] In North America and Europe, the prevalent bacterial agents of early onset sepsis are Group B Streptococcus (*GBS), Listeria Monocytogenes* and *E. Coli* and those of late onset sepsis include Coagulase Negative Staphylococcus (*CONS), Kebsiella, E. Coli.*[13-14] In India Shashikala S, Tallur et al. Showed *Klebsiella* was the commonest organism causing neonatal meningitis.[15] On the contrary, blood culture may also be negative in meningitis, as reported in 13-15 percent of patients with CSF culture-proven definite bacterial meningitis among the provable sepsis.[16] CSF studies are recommended for all infants who are found to have positive blood cultures.[17] The importance of Lumber Puncture (LP) as a part of the diagnostic evaluation of the neonate with suspected sepsis has been the subject of debate and clinical practice varies.[11,16,18]

Neonatologists or Pediatricians often ignore the LP when evaluating the newborn infants. Reasons stated include the perceived low risk of meningitis versus the risk of the procedure in the often unstable patient.[11,18-19] We suspected that there would be differences among the centers in the frequency with which an LP was performed and speculated that meningitis may be underdiagnosed because of the failure to perform routinely LP in neonates with suspected late onset sepsis. The aim of this study was to establish the importance of concomitant central nervous system infection in cases of late onset neonatal sepsis. Specifically, we sought to: (i) evaluate the bacterial meningitis among the late onset sepsis in newborns, (ii) identify the clinical manifestations that can distinguish late onset septicemia alone from those complicated by meningitis in neonates.

Materials and Methods:

Medical records of all neonates admitted in the Department of Neonatology, Bangabandhu Sheikh Mujib Medical University (BSMMU) from January - 2007 to December - 2009 were evaluated. Clinical data of the infants and laboratory findings were examined retrospectively.

Late Onset Sepsis (LOS) was defined as infection occurring after 7 days of age up to 28 days of age with two or more clinical features of sepsis like reluctant to feed, lethargy, temperature instability, axillary temperature <36 C or >38 C, feeding intolerance, apnoea, respiratory distress ,capillary filling time >3 seconds on the forehead or sternam, vomiting, diarrhea, abdominal distension, rapid rise of serum bilirubin >15 mg% in the absence of blood group incompatibility, petechiae or bleeding diathesis, mottling, bulged fortanelle and convulsion etc were present.

Laboratory markers of CBC and CRP level were considered abnormal if- total Leukocyte count <5000/ cmm or >25000/cmm, Absolute neutrophil count <1500/ cmm, Immature total neutrophil ratio >0.2 and PBF showed nucleated RBC with presence of toxic granules and band and thrombocytopenia (platelet count <1500000/cmm

CRP had measured quantitatively. A positive test result indicates a CRP level more than 6 mg/L.

A neonate with LOS was diagnosed as a case of meningitis if any significant bacteria was isolated from CSF and/or the fluid contained more than 30 leukocytes/ mm^3, protein e" 200mg/dl and d"40 mg/dl of sugar.

Positive blood and CSF cultures were considered the 'gold standard' against the laboratory values of CBC and CRP level.

Working definition of LOS to be included:

1) Blood culture usually yields growth of organism though in many cases it may be negative along with presence of clinical features and positive other laboratory values of CBC and CRP level and termed as *proven sepsis*.
2) If culture results were negative but other laboratory tests were positive along with two or more clinical features of sepsis were present and termed as *probable sepsis*.
3) If culture results and other laboratory tests were negative but two or more clinical features of sepsis were present and termed as *clinical sepsis*.

Working definition of bacterial meningitis with LOS to be included:

1) *Definite bacterial meningitis* was diagnosed if there were growth of organisms from CSF culture along

with other laboratory values of cytological, Gram staining and biochemical level.

2) *Probable bacterial meningitis* was diagnosed if no pathogens were obtained from CSF culture but other laboratory values of cytological, Gram staining and biochemical level were present.

Criteria to be excluded:

Neonates with gross congenital anomalies and those having birth weight less than 800 gms were excluded from the study.

All patients received standard empirical treatment for neonatal sepsis according to protocol of proven sepsis 14 days, probable sepsis for 7-10 days and clinical sepsis for 7days.All neonates with proven or probable meningitis received treatment for 21 days. Subsequently antibiotics were changed depending on the culture sensitivity report and/or the clinical condition.

Data collection and analysis:

Data were collected from medical records admitted in NICU from January 2007 to December 2009. All the data were analyzed with SPSS software. Clinical features of LOS were compared with the features of LOS alone and those complicated by bacterial meningitis. P values were reached from Chi square test with 95% CI.

Results:

A total of 1706 neonates were admitted in the BSMMU NICU from January 2007 to December 2009.Among them 476 (27.90%) neonates were cases of suspected sepsis (both early and late onset sepsis)

Out of 476 cases 133 (27.94%) were found to have LOS of which 47 (35.34%) cases were *Proven sepsis,* 63 (47.37%) cases were *Probable sepsis* and 22 (16.54%) cases were *Clinical sepsis.*

There were 4 cases from the *proven sepsis* and 12 cases from the *probable sepsis* were excluded from the study as lumbar puncture could not be done due to life threatening condition and/or congenital anomalies

The baseline characteristics of the babies has shown in Table -I

Male: Female ratio was 1.2:1. The mean gestational age (GA) was 34wk ±.2 (range-28-40wks) weeks and mean birth weight was 1700gms ±50 (range-900-4080gms). Among 117 cases of Late Onset Sepsis 65(55.56%) cases were inborn and 68(58.11%) cases were delivered by LUCS.

Clinical features of the cases were analyzed and shown in the Table-II.

The common features were found to have- reluctant to feed, lethargy, feeding intolerance temperature instability, apnoeas, jaundice, respiratory distress etc.

Table-I

The baseline characteristics of the babies (n=117)

Sex	Male-	64 (54.70%)
	Female -	53 (45.29%)
Birth weight	<1500gms-	47(40.17%)
	1500- <2500gms-	38(32.48%)
	2500gm –>2500gms	32(27.35%)
Gestational age (GA)	<30 wks –	37 (31.62%)
	30-34 wks –	35 (29.91%)
	35-<37wks –	24(20.51%)
	37 –>37wks-	21(17.96%)
Mode of delivery	LUCS -	68(58.11%)
	NVD –	49(41.89%)
Place of delivery	Inborn-	65 (55.56%)
	Out born –	52(44.44%)

Table – II

Clinical features of both Septic and Meningitic cases

Symptoms	Total 117		P-Value
	Septic Baby (n=81) Number (Percentage)	Meningitic Baby (n=36) Number (Percentage)	
Reluctant to feed	48 (59.25%)	17 (47.22%)	0.226 NS
Lethargy	45 (55.56%)	18 (50.00%)	0.577 NS
Feeding intolerance	46 (56.79%)	21 (58.33%)	0.876 NS
Temperature Instability	29 (35.80%)	19 (52.78%)	0.084 NS
Apnea	31 (38.27%)	14 (38.89%)	0.949 NS
Abdominal distention	40 (49.38%)	21 (58.33%)	0.371 NS
Vomiting	39 (48.14%)	22 (61.11%)	0.195 NS
Jaundice	17 (20.98%)	8 (22.22%)	0.880 NS
Convulsion	6 (07.40%)	5 (13.89%)	0.267 NS
Respiratory distress	23 (28.39%)	12 (33.33%)	0.590 NS
Bulged fontanel	3 (03.70%)	3 (08.33%)	0.294 NS
High pitch cry	0 (0%)	1 (02.77%)	0.131 NS

P value reached from Chi square test with 95% CI , NS = Not Significant

Features did not differ in the cases with or without meningitis. P values revealed no significant differences in clinical features among the septicemia cases alone or complicated by meningitis.

The most frequent presenting clinical features for meningitis cases were more or less similar to those of septicemic cases; reluctant to feed (59% vs 47%), lethargy (56% vs 50%), feeding intolerance (57% vs 58%), apnoea (38% vs 39 %), abdominal distention (49%vs 58%), vomiting (48% vs 61%) and temperature instability (36% vs 53%).

There were 3 (8.33%) cases found to have bulged fontanels and 5 (13.89%) cases convulsion among meningitic patients. Out of the entire septic baby 6 (7.40%) cases were found to have convulsion and 3 (3.70%) cases were bulged fontanelles.

Among the 43 cases of *Proven sepsis*, 28 (65.11%) cases were found to have meningitis of which 12 (42.86%) cases were *Definite bacterial meningitis* and 16 (57.14%) cases were *Probable bacterial meningitis* based on either by CSF culture positive and/or CSF abnormality.

Among the 51 cases of *Probable sepsis*, 8 (15.68%) cases were found to have meningitis of which 1 (12.5%) case had *Definite bacterial meningitis* as CSF culture was positive. Other 7 (87.5%) cases had *Probable bacterial meningitis* as abnormal CSF laboratory report but CSF culture were negative.

Among the 22 cases of *Clinical sepsis* with strongly clinical suspicion of meningitis, there were no cases found to have *Definite* and/or *Probable bacterial meningitis*.

Finally out of 117 cases of LOS, Bacterial meningitis were found to have in 36(30.76%) cases, shown in Figure I.

Among all the cases of culture positive LOS 18(41.86) cases Klebseilla, 17(39.53%) cases E.coli, 3(6.98%) cases Pseudomonus, 3(6.98%) cases Acinatobacter and 2(4.65%) cases Enterobacter were found.

Out of all the 13 (36.11%) cases of definite bacterial meningitis yielded growth of bacteria of which there were 7(53.85 %) cases of *E.Coli*, 4 (30.77%) cases of *Klebseilla* and 2 (15.38%) cases of *Pseudomonas* which had matched with identification and antibiotic susceptibility profile as their corresponding blood culture ,shown in the Table-III.

Among the 36 cases of bacterial meningitis 12(33.33%) cases were found to have concomitant blood and CSF

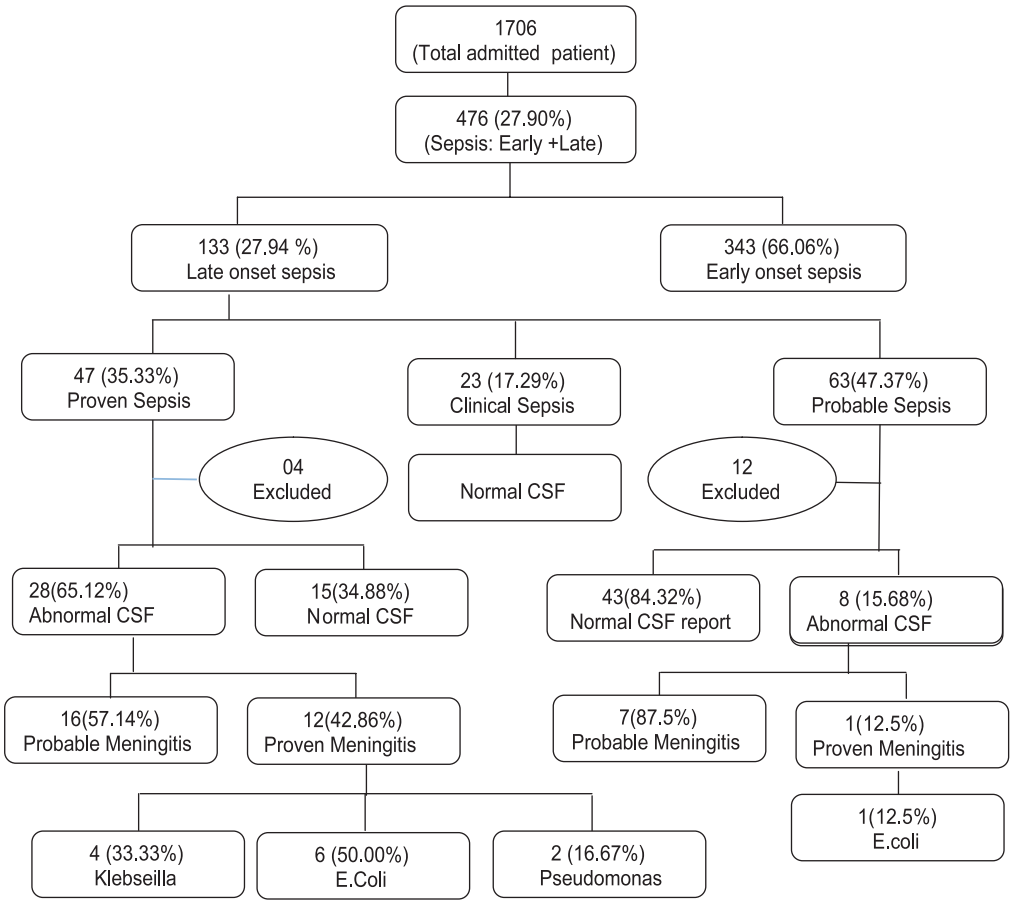

Fig-I: *Flow chart of the Results*

Table–III

Distribution of pathogen in the blood and CSF

Name of Organism	Septic Baby (n=43) Number (Percentage)	Meningitic Baby (n=13) Number (Percentage)
Klebseilla	18 (41.86%)	4 (30.77%)
E .coli	17 (39.53%)	7 (53.85%)
Pseudomonas	3 (6.98%)	2 (15.38)
Acinetobacter	3 (6.98%)	0 (0.00%)
Enterobacter	2(4.65%)	0 (0.00%)

culture positive and 1(2.79%) cases where CSF culture was positive but blood culture was negative.

In neonates with both positive blood and CSF cultures, there was no discordance among the organism isolated.

Discussion:

Neonatal meningitis has a high mortality and significant risk of morbidity and neurological sequelae. But the diagnosis is often difficult or sometimes delayed as clinical features are non specific and LP is not performed routinely.

In some studies it has been shown that 20-30 % cases of neonatal sepsis whether early or late were complicated by neonatal meningitis. In this study we have found 30.76% cases of neonatal meningitis among the suspected cases of LOS.[9 10 20]

The incidence was also very high in the preterm and low birth weight babies which was inversely related to gestational age and birth weight consistent with previous studies.[13, 21]

Commonest organism of neonatal meningitis in the developed countries is Group B Streptococcus (GBS) which is not quite similar in Bangladesh. Studies reported *E. coli, Klebseilla* to be the major causative organism of neonatal meningitis.[20-21] This study has been correlated the previous studies of the country in regard to neonatal sepsis where the organisms were found to have *E-coli, Klebseilla*, and *Pseudomonas* as the important organisms of neonatal meningitis as comparable to other studies of Bangladesh.[20]

Since most cases bacterial pathogens involved in the neonatal sepsis are commonly the same for neonatal meningitis in a given environment. This observation has been corroborated by parallel studies which also identified same organisms as the most frequent pathogens of neonatal sepsis in the same unit. In this study all the culture positive neonatal meningitis cases had the same organisms as in the blood indicating that these were the complication of LOS as in other studies.[13,20]

In this study there were 12(33.33 %) cases where simultaneously blood culture and CSF culture were positive but in other studies there were 50 - 66% cases had concomitant blood culture and CSF culture were positive.[20,22]

As reported by others 13 -38% cases, where blood culture was negative but organisms were isolated in the CSF as culture proven meningitis.[13 22] This study also showed 2.79% cases among the total bacterial meningitis where organisms were not isolated from the blood but isolated from CSF culture.

In neonates with both positive blood and CSF cultures, the organisms isolated were discordant in 3.5% of cases but in this study there were no discordance of isolated organism as found in other study.[20, 22]

Neonates with a positive Blood culture were significantly and more likely to have meningitis than those with a negative blood culture and lumbar puncture should be considered as a part of routine investigation in late onset sepsis .[23-25]

The necessity of performing lumbar puncture in newborn has been debated because of the perceived risks of procedure in very sick infant but other studies have shown that the incidence of meningitis secondary to lumbar puncture is very low and there are no increased risks of death that had a lumbar puncture.[13, 25-26] In this study there were no data of meningitis or death secondary to lumbar puncture during the study period.

Limitation of this study was that the LP was done sometime after receiving antibiotics.

Nonetheless the data confirmed that it is very important to an LP along with blood culture for all suspected cases of LOS.

Conclusion:

Neonatal meningitis is not uncommon in late onset sepsis. The presenting clinical features for meningitis cases are more or less similar to those of septicemic cases. The data of the study suggest that neonates with positive blood culture LOS are more likely to have meningitis than those with negative blood culture.

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Study of EEG Findings in Patients Referred from Psychiatrists

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Summary:

EEG is not so commonly used in patients attending psychiatry department. It is predominantly required to rule out any organic cause behind the behavioral changes. Purpose of this study was to assess the referred cases from psychiatry department and determine the clinical factors associated with an abnormal EEG in patients with psychiatric problem. We retrospectively reviewed and analyzed the data of all the cases referred to EEG lab. of Dept. of Neurology, Dhaka Medical College Hospital from psychiatrist. A total of 50 patients from July 2009 to January 2011 were selected. From the EEG register following information were noted eg. age, sex, socioeconomic status, habitat, rural or urban, clinical features, associated features, probable clinical diagnosis and EEG findings. The results showed that most of the patients belonged to age group 11-20years, comprising 46 % (n-23), 66% (n-33) were female. Though the total rate of abnormal EEG in psychiatry patients are low (n-20). 44% of the patients having seizure as the presenting complaint had the largest number of EEG abnormality (n-16), p value <.001. Patients with primary psychiatric disorders did not have any epileptiform activity (n-22). Where as most of the patients (n-27) who were undiagnosed at the time of referral had the largest number of EEG abnormality (n-19), p value<.002. So any patient presenting to psychiatry department with seizure disorder or any patient with diagnostic confusion should be evaluated with EEG.

Key words: Generalized epilepsy (GE), Localization related epilepsy (LRE), psychogenic nonepileptic seizures (PNES).

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Introduction:

Electroencephalography (EEG) is the recording of electrical activity along the scalp produced by the firing of neurons within the brain. In clinical contexts, EEG refers to the recording of the brain's spontaneous electrical activity over a short period of time, usually 20–40 minutes, as recorded from multiple electrodes placed on the scalp. In neurology, the main diagnostic application of EEG is in the case of epilepsy, as epileptic activity can create clear abnormalities on a standard EEG study. A secondary clinical use of EEG is in the diagnosis of coma, encephalopathy, and brain death.[1]

The electroencephalogram is believed to be of limited value in psychiatry. This is particularly true for patients with neurosis and functional psychosis. With the advent of modern imaging like CT scan or MRI, EEG has got limited value in organic states like tumors, stroke and other focal brain disorders, although former gives a structural and later gives functional assessment of cerebral function. EEG is the investigation of choice in suspected cases of epilepsy and can differentiate between seizure and pseudo seizure. There is well documented relationship between psychosis and epilepsy. In particular temporal lobe abnormalities may present with mental illness.[2] Gruhle, Hill and Pond found psychotic states in epileptic patients that closely resembled schizophrenia. In their review they found

lesions in temporal lobe in most of the patients. Slater studied 69 epileptic patients presenting with a schizophreniform psychosis; 80% had temporal lobe epilepsy (TLE). [3,4,5]

There are doubts and arguments about the role of EEG in psychiatry. Although psychiatrists send patients for EEG where there is doubt about seizure, pseudo seizure and psychosis. With this background the aim of this study is to correlate various clinical presentations of psychiatric patients to EEG findings and to guide clinicians in cases where EEG is indicated to differentiate between a primary psychiatric disorder and epilepsy.

Materials & methods:

This is a retrospective observational study carried out in EEG lab of Department of Neurology of DMCH from July 2009 to January 2011. All the patients referred from Department of Psychiatry or psychiatrists for EEG were enrolled for the study. Information of non co-operative patients or EEG with marked artifact were discarded Data of first 50 patients were taken from EEG register. From the EEG register following information were noted eg. age, sex, socioeconomic status, habitat, rural or urban, clinical features, associated features, probable clinical diagnosis, EEG findings and diagnosis. EEG was done in international 10/20 system using surface electrodes. After proper recording EEG were analyzed and reported by two Neurologist of Department of Neurology of DMCH. EEG showing focal epileptiform activity was labeled as LRE (Localization Related Epilepsy) and generalized epileptic activity was labeled as GE (Generalized Epilepsy). Data were recorded and analyzed using SPSS system.

Results:

The number of cases referred from the department of Psychiatry to Neurology for EEG evaluation is sparse. We just had 50 cases from the psychiatry department. We carried out a retrospective observation of patients from hospital records. The results of observation are quite interesting. Most of the patients belong to age group 11-20years, comprising 44 % (n-22) and 62% of patients was below 20years of age. 66% of the patients were female (Table-I). 34% patients experienced sudden onset of symptoms whereas 66% had gradual onset of illness. Symptom analysis revealed that seizure (true or pseudo seizure) was present in 22 patients (44%) (Table-II). At the time of referral 54% of the patients did not have any specific diagnosis on underlying psychiatric disorder (Table-III). Analysis showed a strongly positive association between seizure event at presentation and likelihood of getting an abnormal EEG diagnosis, p value 0.0001 (Table-IV). During referral 46% patient had specific diagnosis of underlying psychiatric disorder. But only 1 patient had a LRE diagnosed. Rest of the patients (n-27) who were undiagnosed had the highest number of EEG positive cases (Table-V).

Table I

Socio demographic profile of the patients

Parameter		n	%
Age	<10 yr	9	18
	11-20 yr	22	44
	21-30 yr	9	18
	31-40 yr	7	14
	41-60 yr	2	4
	>60 yrs	1	2
Sex	Male	17	34
	Female	33	66
Habitat	Rural	32	64
	Urban	18	36
Socioeconomic status	Lower class	22	44
	Middle class	25	50
	Higher class	3	6

Table I: Most of the patients belong to age group 11-20years, comprising 44 % (n-22) and 62% of patients was below 20years of age. 66% of the patients were female. Majority of them were from rural areas (64%) and belonged to middle class (50%).

Table II

Clinical symptoms and associated features

Parameter		n	%
Onset	Sudden	17	34
	Gradual	33	66
Duration	Years	21	42
	Months	18	36
	Weeks	6	12
	Days	2	4
Symptoms	Seizure	22	44
	Unconsciousness	22	44
	Altered behavior	21	42
	Headache	13	26
	Burning head	7	14
	Psychosis	7	14
	Others	8	16
Associated features			
	Family History	5	10
	Birth injury	3	6
	Cerebral palsy	5	10
	H/O encephalitis	3	6
	Substance abuse	4	8
	Family dispute	5	10
	Mental retardation	4	8
	Decreased school performance	7	14
Drug History			
	Antidepressant	12	24
	Anti epileptic	10	20
	Antipsychotic	7	14
	No drugs	21	42

Table II: 34% patients experienced sudden onset of symptoms whereas 66% had gradual onset of illness. Symptom analysis revealed that seizure (true or pseudo seizure) was present in 22 patients (44%), unconsciousness in 22 patients and altered behavior in 21 patients. history of cerebral palsy was found in 10% and Birth asphyxia in 6%. Many of them received antidepressant (24%) and antiepileptic (20%) drugs.

Table III

Underlying psychiatric disorder and EEG

Parameter		n	%
Underlying psychiatric disorder			
	Conversion disorder	6	12
	Somatoform disorder	3	6
	Depression	6	12
	Schizophrenia	7	14
	Others	1	2
	Undiagnosed	27	54
EEG Diagnosis	Normal	30	60
	LRE	14	28
	GE	6	12
EEG Findings	Normal	30	60
	Spike and wave	14	28
	Sharp and wave	4	8
	Focal slow wave	2	4
Site of epileptiform activities			
	Temporal lobe	8	16
	Frontal lobe	2	4
	Paracentral	3	6
	Hemispheric	1	2
	Generalized	6	12

Table III: At the time of referral 54% of the patients did not have any specific diagnosis on underlying psychiatric disorder. Conversion disorder was present in 6 cases, Somatoform disorder in 3, depression in 6 and schizophrenia in 7 cases. EEG was normal in 60% cases, LRE in 28%, GE in 12%. Most common EEG abnormality was in the form of Spike and wave in 28%, most common location was Temporal lobe (16%).

Table IV

Seizure and EEG diagnosis

Seizure	Normal	LRE	GE	Total	
Present	Count	5	12	5	22
	% within seizure	22	54	22	100
	% within EEG Diagnosis	16	85	83	44
	% of total	10	24	10	44
Absent	Count	25	2	1	28
	% within seizure	89	7	2	100
	% within EEG Diagnosis	83	14	16	56
	% of total	50	4	2	56
Total	Count	30	14	6	50
	% within seizure	60	28	12	100
	% within EEG Diagnosis	100	100	100	100
	% of total	60	28	12	100
Chi-Square test		df			*p*
	Pearsons Chi-Square	2	0.0001		
	Likelihood ratio	2	0.0001		

Table V

Underlying psychiatric disorder and EEG diagnosis

Underlying Psychiatric disease		N	LRE	GE	Total
Conversion Disorder	Count	6	0	0	6
	% within EEG Diagnosis	20	0	0	12
	% of total	12	0	0	12
Somatoform Disorder	Count	3	0	0	3
	% within EEG Diagnosis	10	0	0	6
	% of total	6	0	0	6
Depression	Count	6	0	0	6
	% within EEG Diagnosis	20	0	0	12
	% of total	12	0	0	12
Schizophrenia	Count	7	0	0	7
	% within EEG Diagnosis	23	0	0	14
	% of total	14	0	0	14
Others	Count	0	1	0	1
	% within EEG Diagnosis	0	7	0	2
	% of total	2	0	0	2
Undiagnosed	Count	8	13	6	27
	% within EEG Diagnosis	27	93	100	54
	% of total	16	26	12	54
Total	Count	30	14	6	50
	% of total	60	28	12	100
Chi-Square test		df			*p*
	Pearsons Chi-Square	10		.002	
	Likelihood ratio	10		.000	

Table IV: Analysis showed a strongly positive association between seizure event at presentation and likelihood of getting an abnormal EEG diagnosis, p value 0.0001

Table V: During referral 46% patient had specific diagnosis of underlying psychiatric disorder. But only 1 patient had a LRE diagnosed. Rest of the patients (n-27) who were undiagnosed had the highest number of EEG positive cases. The association of underlying psychiatric disorder and positive EEG diagnosis is highly significant, p value <.002.

Discussion:

The value of EEG in psychiatry is an issue of debate since the advent of these electrophysiological studies in the 1930. Interestingly there are few credible studies in this area in home and abroad, and much of the earlier studies had poor research design and hence unwarranted conclusions.

Among the cases in our study, 66% (n-33) were female and 34% (n-17) were male. Here female outnumbers male. Most of the patients belong to age group 11-20years, comprising 44 % (n-22) and 62% of patients was below 20years of age. Next was the age range from 0-10, having 9 patients. Only one patient was above 60 years. In this study 64% (n-32) of the patients were from urban areas and 36% (n-18) from rural area. 44% (n-22) of patients belonged to lower social class and only 6% belongs to higher class. Onset of illness was gradual in 66% of the patients. 42% patients suffered for years together while 12% waited for weeks before getting their EEG done. Here it is evident that patients with combined psychiatric neurologic symptoms suffer for long time before getting consultation from specialists. Analysis of symptoms revealed that seizure (true or pseudosizure) was present in 44% (n- 22) patients, in 44% (n-22) patients there was history of unconsciousness, 21 patients had history of altered behavior, headache in 13 patients and psychotic features were present in 4 patients. Psychogenic nonepileptic seizure (PNES) or pseudoseizures are common at epilepsy centers, where they are seen in 20-30% of patients referred for refractory seizures. PNES are probably also common in the general population, with an estimated prevalence of 2-33 cases per 100,000 populations. Misdiagnosis of epilepsy is common. Misdiagnosis occurs in approximately 25% of patients with a previous diagnosis of epilepsy that does not respond to drugs. Most cases of misdiagnosed epilepsy are eventually shown to be psychogenic nonepileptic seizures (PNES) or, more rarely, syncope.[6,7,8] 44% (n-17) of the patients with abnormal EEG findings had seizure at onset of illness. Analysis revealed a strong association between seizure and EEG abnormality, p value .0001 (Table-IV). So any patient with seizure that visits psychiatry department should be evaluated with EEG for further management. Out of 50 patients 10% (n-5) patients had cerebral palsy, 8% (n- 4) patients had mental retardation, there was presence of birth injury in 3 patients, and 5 patients had history of family dispute. Bruck showed the overall prevalence of epilepsy was 62% in cerebral palsy. Incidence of epilepsy was predominant in patients with hemiplegics and tetraplegic palsies: 70.6% and 66.1%, respectively.[9,10,11]. The cumulative risk of seizures and epilepsy was investigated in a prospectively identified cohort of 221 children with mental retardation (MR) born between 1951 and 1955 in Aberdeen, Scotland. By age 22 years, 33 (15%) had epilepsy.[9] At the time of referral for 24% (n-12) were on antidepressant, 20% on antiepileptic, 14% were on antipsychotic drugs where as 42% were without these drugs. Probable psychiatric diagnosis was Dissociative disorder in 6 patients, Somatoform disorder in 3 patients, Depression in 6 patients, Schizophrenia in 7 patients and diagnosis was not mentioned in 27 patients. 1 patient thought to be of psychiatric disorder actually had LRE (Table- III) Analysis shows that 50% of the conversion disorder patients had seizure at onset, while 63% of the patients who did not have a definite diagnosis of psychiatric illness had seizure at presentation. Co morbidities of epilepsy and psychiatric disorder is common, yet the most common are depression, nervousness, anxiety, PNES, and less common being Schizophrenia and psychosis.[10-14]

EEG revealed that 60% (n-30) had normal findings, 28% (n-14) had Localization related epilepsy (LRE), and 12% (n-6) had Generalized epilepsy (GE). 54% of the patients who were undiagnosed at the time of referral, had the most number of EEG abnormality. Analysis showed a strong association between underlying psychiatric disorder and EEG negativity, p value .002 (table-V). This signifies the role of EEG in any patient of psychiatry with diagnostic confusion. Out of 16 patients of LRE 8 originated from Temporal lobe and rest from Frontal, Parietal lobes and Central and Para central areas. All the patients suspected of Dissociative disorder had normal

findings in EEG. Interestingly enough, most of the patients among the undiagnosed subgroup at the time of referral had positive EEG findings 54% (Table-V). In 14 patients, abnormal EEG was in the form of spike & wave, and in 4 patients sharp & wave.

Conclusion:

EEG is not routinely advised in patients suspected of primary psychiatric disorder. Whereas it has important role in classification and management of seizure disorders. Patients with epilepsy often have got psychiatric symptoms and co morbidities specially in temporal lobe epilepsy eg. complex partial seizure. On the other hand in patients with PNES symptoms, EEG might be helpful in differentiating from true seizure. So judicious use of EEG might be helpful in management of patients with psychiatric and neurological symptoms.

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Pre-Anaesthetic Fitness for Surgery

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Introduction:

Pre-anaesthetic fitness for surgery requires pre-anaesthetic evaluation of the patient which is one of the essential parts of perioperative care. Several large scales epidemiological confidential study into peri-operative death showed that inadequate preparation of the patients were the major contributory factors for perioperative mortality [1]; the mortality rate is 0.01% [2]. The factors include recognition of existing problems of the patient at the earliest opportunity; the process must be designed to allow the problematic patients to be seen by an experienced anaesthesiologist well in advance of proposed surgery [3]. This can be achieved if proper anaesthetic check up is done as soon as the patient is scheduled for operative procedure. The main purpose is to keep the patients in their best possible physiological condition to undergo anaesthesia and to ensure that the benefit of surgery must not be outweighed by perioperative risks. There should not be any lapse in the routine procedure of pre-anaesthetic formalities. Many a time a small little gap in this practice could bring severe catastrophe to the patient and to the operative team.

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Procedure of Pre-Anaesthetic Assessment

The preoperative visit enables the anaesthesiologist to meet the patient and to make a rapport so that the patient is relieved of imaginary fear and anxiety. The anaesthesiologist should explain about the anaesthetic planning with assurance of adequate perioperative care. Then the anaesthesiologist will proceed to obtain history, perform physical examination and order special investigations as dictated by the findings. The informed consent should be taken and the anaesthetic record sheet should be properly documented.

A. History

A series of questions to be asked. These includes

i) Presenting condition and concurrent medical history: The preoperative history should clearly establish the patient's present problem which will help to plan the peri-operative anaesthetic management. The indication for surgery may influence anaesthetic management quite dramatically. The systemic effects of disease process must be quantified. There are many diseases which may have a significant impact on anaesthetic management and its outcome, particularly disease of the cardiovascular or respiratory systems. Their presence or absence are usually ascertained by direct questioning and should be recorded carefully.

ii) Family history: There are number of inherited conditions that have a significant influence on different aspects of planned anaesthetic management, such as malignant hyperthermia, cholinesterase abnormalities, porphyria, certain heamoglobinopathies and dystrophia myotonica. If such a condition is suspected, a full investigation of relevant family member is beneficial.

iii) Drug history: Many patients requiring surgical procedure might suffer from unrelated disease and about 42% of them receive regular drug therapy [4, 5]. These drugs may have interaction with anesthetic agents or may cause problems related to their sudden withdrawal during preoperative period. There are other substances (alcohol, tobacco. opioids and cocaine, etc) taken habitually by some patients that can also have a significant influence on the process of anesthesia.

iv) Anaesthetic history: Obtaining record of previous admission and previous anaesthesia is very important. This will help to avoid repetitions of complications and planning of anaesthesia. So, details of administration and outcome of any previous anaesthetic exposure are important and mandatory. History of any pre-operative fear, nausea, vomiting, sore throat or headache should be taken into account and the anaesthetist must attempt to clarify their clinical significance. History of any difficult intubation should be evaluated by physical examination.

i) History of allergy /hypersensitivity: Although severe anaphylactic reactions to anaesthetic drugs are rare they do represent an important cause of serious morbidity or mortality. Ratio of anaphylactic reaction to population was 1:25000 [6]. Common drugs causing anaphylactic reactions are antibiotics especially co-trimoxazole, penicillin and aspirin. Non-steroidal anti-inflammatory drugs (NSAIDS) are used commonly in the preoperative period may have a risk of cross-sensitivity to the patient. Patients having history of atopy may demonstrate greater sensitivity to release histamine or other vasoactive chemicals with increased reactivity of the cardiovascular or respiratory system on exposure to noxious stimuli. A small proportion of patients may complain of an allergic reaction to previous anaesthetic. The exact nature of symptoms and signs must be asked for, as the term allergy is not always understood properly by the patient.

ii) Personal History

(a) Smoking: Cigarette smoking is one of the factors involve with adverse peri-operative outcomes. There are several potential mechanisms by which smoking exerts short term and long term adverse effects on various organs. Short term effects are due to inhalation of cigarette smoke which contains nicotine and carbon monoxide. Nicotine acts on the sympathetic nervous system producing tachycardia, hypertension and increased coronary vascular resistance; all these cause an increase in myocardial oxygen demand. Carbon monoxide binds with haemoglobin to form carboxyhaemoglobin. In a heavy smoker this may results in a significant decrease (as much as 25%) in oxygen delivery to the tissues[7]. The half–life of carboxyhaemoglobin is short and therefore stopping of smoking for about 12 hours lead to an increase in arterial O_2 content[7]. This is very important for the patient suffering from ischaemic heart disease. There is evidence that cessation of smoking improves the symptoms of angina.

Long term problems of smoking include depression of immune function, impaired clearance of secretion from the tracheobronchial tree and chronic airway diseases[7]. It is suggested that stopping of smoking for 6-8 weeks results in reduced bronchoconstriction and mucous secretion in the tracheobronchial tree[7].

(b) Alcohol: Patient may present with acute intoxication or sequence of chronic alcohol consumption. Once the diagnosis is established, it must be decided whether to continue alcohol consumption during admission or to run a course of withdrawal during pre-operative period, which has its own risk of morbidity and mortality.

The authors prefer continuation of alcohol rather than withdrawal.

(c) Vomiting tendency: This may modify the choice of anaesthetics which would reduce the likelihood of post operative nausea and vomiting.

(d) Menstrual history: Elective surgery should be postponed in early pregnancy, where possible until second trimester[8] to avoid the potential hazards for the fetus, which include exposure

to teratogenic effect of anaesthetic agents, intraoperative hypoxaemia or asphyxia, risk of spontaneous abortion or preterm delivery.

B. Pre-operative patient questionnaires

1. Do you suffer or have you suffered from any of the following:

Stroke	Yes /No
Heart disease ·	
• Palpitations·	
• High Blood pressure·	
• Chest pains·	
• Swelling of ankles·	
• Shortness of breath during walking up a single flight of stairs	
Asthma	
Bronchitis	
Diabetes	
Epilepsy	
Ulcer trouble or hiatus hernia	
Jaundice	
Liver disease	
Kidney disease	
Anaemia	
Arthritis	

2. Are you taking any tablets, pills, inhalers or medicines? If yes, please list:
3. Have you any allergies? If yes, please list.
4. Do you smoke? If yes, how many a day?
5. Do you drink more than a moderate amount of alcohol? (more than 8 pints beer/week or 10 glasses wine/week)
6. Do you bruise easily or bleed excessively?
7. Have you had any operations or general anesthesia before? If yes, please list, including approximate dates:
8. Were there any complications?

   If yes, please give details.
9. Have any members of your family had any problems with anesthesia?
10. Is there anything about yourself or your family's medical history you think we should know?

    If yes, please details.

C. Physical examination

Physical examination is a simple, safe and cheap method of providing important pre-operative information. A full clinical examination should be performed on every patient and the findings must be documented. Beside history and routine clinical examination, there are areas where special preferences are to be given during examination such as airway for difficult intubations.

D. Investigations

Laboratory tests are essential tools for appropriate diagnosis and to quantify a disease process. The relevance of investigation for anesthesia can be extended to provide a pre-operative baseline data with which peri-operative change can be compared. In general, results of some investigations can be predicted if a detailed history or examinations is available. Before ordering extensive investigations, the anesthesiologist should confirm that the investigations will alter the management of the patient. Instead of doing a series of investigations as a matter of routine procedure, a guideline can be followed which will give relevant information.(Table II)

RISK ASSESSMENT

An attempt has been made to classified or score patients preoperatively in order to identify those at greater risk of adverse outcome.

1. ASA (American society of anesthesiologist) grading

In ASA grading the patients are classified according to disability related to patient's general health, which correlates to some extend with risks of preoperative complications[9]. It predicts poorly when used alone as it does not embrace all aspects of anaesthetic risk such as age, severity of the presenting disease or the proposed surgery and it does not identify factors which can be altered pre-operatively to improve outcome. Nevertheless it is useful in average prediction of the risk and perioperative mortality rate. (Table III, IV)

Table-I

Clinical examination before anaesthesia

Systems	Points to examine
General	General well-being, nutritional state, build, colour of skin, temperature and hydration state.
Cardiovascular	Pulse rate, rhythm, volume, jugular venous pressure and pulsations, cardiac impulse, blood pressure, auscultatory heart sounds, carotid pulsation, sacral oedema.
Respiratory	Auscultation of lung fields, observation for dyspnoea.
Central nervous System	Function of special senses and cranial nerves, peripheral motor and sensory function.
Airway	Mouth opening, neck movement, dental records.

Table 1I

Guideline for preoperative Investigation

Investigations	Indication
Urine analysis	This should be performed on every patient. There may be undiagnosed DM or urinary tract infection.
Urea, creatinine	• All patients over 65 years of age or with a positive result from electrolytes and urine analysis. • All patients with cardio-pulmonary disease or taking vaso-cardiovascular active drugs, diuretics or steroids. · All patients with a history of liver or renal disease, diabetes or an abnormal nutritional state. • Any patient with a history of diarrhea, vomiting or metabolic illness. Patients who have been on i.v. fluid therapy for more than 24 h.
Blood glucose	All patients with history of DM, vascular disease and the patient receiving cortico-steroid.
Liver Function Test	Any history of liver disease, alcoholism, previous hepatitis or unexplained fever, an abnormal nutritional state.
Full blood count	• All female patients regardless of general health or reason for admission. • All male patient > 50 years of age. • History of blood loss. • History of previous anaemia. • History of heamatopoitic disease, cardio-respiratory disease or possibility of significant blood loss during surgery.
Coagulation screen	• Any patient with a history of coagulation disorder. • Significant alcohol consumption, • Drug abuse • On anticoagulant medication • All patient of ethnic group carry risk of sickle gene
Electrocardiogram	• Smoker > 45 years old • Diastolic pressure > 95 mmHg during admission • Hypertension or Heart disease • Patient on diuretics or cardiovascular active drugs. • Patient with symptomatic chronic or acute-on-chronic pulmonary disease.
Chest X-ray	• Cardiovascular and/or respiratory disease, • History suggestive of possible abnormalities e.g trauma. • A previously abnormal chest film. • Any patient with thyroid enlargement(with a thoracic inlet view)
Pulmonary Function Tests	Patient with severe dyspnoea on mild to moderate exertion should go for Peak expiratory flow rate, FVC and FEV_1.
Arterial blood gas analysis	All patients with dyspnoea; patient scheduled for elective thoracotomy.

Table-III

The ASA Physical Status Scale

Class	Definition
P1 (Class I)	A normal healthy individual
P2 (Class II)	A patient with mild systemic disease
P3 (Class III)	A patient with severe systemic disease that is not incapacitating
P4 (Class IV)	A patient with incapacitating systemic disease that is a constant threat to life.
P5 (Class V)	A moribund patient who is not expected to survive 24h with or without operation.
P6 (Class VI)	A brain-dead patient whose organs are being removed for donor purposes.
Class E	Added as a suffix for emergency operation.

The ASA Classification as amended on October 1984.

Table-IV

Mortality rate of anesthesia and surgery for each ASA physical status

ASA rating	Mortality rate %
I	0.1
II	0.2
III	1.8
IV	7.8
V	9.4

2. PAFS (Pre-operative assessment of fitness score)

This classification is based on physiological information, demographic feature and basic laboratory test for the assessment of peri-operative survival [11]. The specificity is 80%. It includes various scoring (Table-V) for the assessment of post-operative complication such as pneumonia, sepsis, non-infective organ failure within 30 days of surgery. Prospective identification of independent predictors of severe peri-

Table –V

Pre-operative Assessment of Fitness Score (PAFS)

Score	Preoperative factor
Score I for each	Cardiac symptoms controlled by treatment.
	Dyspnea on climbing stairs.
	Morning cough.
	Stroke or myocardial infarction > 6 month age.
	Hemoglobin <10g.dl-1.
	Serum albumin 30-35 g.litre-1
	Plasma urea 10-19mmol.litre-1
	Steroid treatment
	Controlled diabetes
Score 2 for each	Age 70-79 years.
	Cardiac symptoms poorly controlled by treatment.
	Dyspnoea on walking.
	Persistent cough with sputum.
Score 3 for each	Clinical Jaundice.
	Serum albumin <9g. litre- 1
	Loss of 10% body weight in 01 month.
	Plasma urea>20mmol.
	Dyspnoea at rest.
	Myocardial infarction >6months back.
	Confusion.
	Cytotoxic treatment.
Score 4 each	Age> 80 years.
	Palliative operation for surgery.
	Intestinal obstruction.
	Perforation, pancreatitis and intraperitoneal abscess. Hemorrhage or anaemia.

operative adverse outcome is of utmost importance. Forrest and co-workers have undertaken a large scale study, analyzing independent predictors of severe peri-operative adverse outcome over 17000 patients [10]. A history of some cardiovascular disease, the needing abdominal or cardio thoracic surgery, specific demographic factors were found to be the most important predictors of severe cardiovascular or respiratory events.

Evaluation of PAFS score according to phyforth et al[12]:

*A total score of less than 6 indicates low risk (10%)

*A score of 6-10: high risk of postoperative death or major complication within 30 days of surgery.

*The major complications are defined as pneumonitis, sepsis or non infective organ failure

PREDICTION OF SPECIFIC ADVERSE EVENTS

These are

(a) The difficult airway; (b) Adverse cardiac events; (c) Respiratory complications

(a) Prediction of difficult airway

Physical features related with difficult intubations includes

1. General appearance of the neck, face, maxilla and mandible.
2. Jaw movement, mouth opening
3. Head extension and neck movement
4. The teeth and oropharynx
5. The soft tissues of neck
6. Recent chest and cervical spine X-ray
7. Previous anesthetic records.

Unfortunately, difficult intubations still unexpectedly occur, causing more anaesthetic morbidity and mortality. Mallampati and colleagues devised a classification based on visible pharyngeal structures when the patient opens the mouth maximally and protrudes the tongue[13]. This was subsequently modified by Samson & Young (Table VI[14]. This is a simple bedside test but sometimes related with a high incidence of false positive to improve upon the observers variability, Wilson and colleagues described a five point features which includes weight; movement of head, neck and jaw; presence of mandibular recession or absence of buckteeth. These also produce a significant number of false positive[15, 16]. When Mallapati test is combined with thyromental distance (TD) the false positive is reduced. Now it is suggested that any patient with thyromental distance of less than 7cm and Mallampati grade III or IV may present with intubation problem[17]. Cormack and Lehane described a standard method of grading depending on laryngoscopic view (Table VII) [18].

Table-VI

Mallampatis modified classification

Grade	Description
I	Pharyngeal pillars soft palate and uvula visible
II	Only soft palate and uvula visible
III	Only the soft palate visible
IV	Soft palate not visible

Table-VII

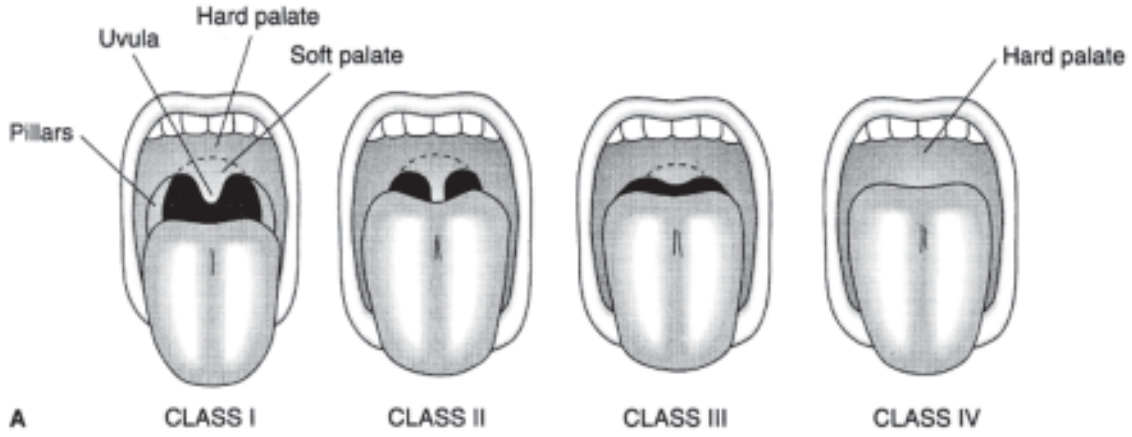
Cormack & Lehane's grading on laryngoscope view:

Grade	*Structures visible*
I	Vocal cord visible.
II	Arytenoid cartilages and posterior part of the vocal cords visible.
III	Epiglottis visible.
IV	No exposure of the glottis, or of the corniculate cartilages.

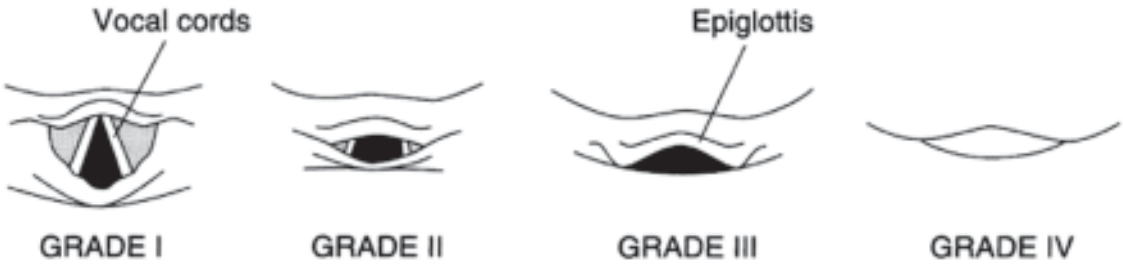
Evaluation of Cormack & Lahane grading

Grade I	No difficulty
Grade II	Slight difficulty
Grade III	Severe difficulty
Grade IV	Intubations impossible without special method

Mallampati classification of oral opening



Cormack and Lehane's grading on laryngoscope view:



(b) Adverse cardiac events

Opinions are conflicting regarding prediction of serious perioperative cardiac events. Goldman and colleagues are renowned for their retrospective study on cardiac event in patient undergoing non cardiac surgery"[19, 20]. Their risk indices (Table-VIII and IX) give a guide to major cardiac complications.

Similar risk indices have described more recently (Table-X), although controversy persists about the most accurate predictors of serious pre operative cardiac events[12]. One of the most sensitive factors is the presence of pre-operative hypertension. Gross hypertensive responses, with ECG evidence of ischaemia on some occasions are likely to occur due to noxious stimuli during anesthesia in hypertensive patients. Whether treated or not, if the pre-operative diastolic pressure exceeds 110 mm Hg, there is a chance of ST changes with an increased incidence of postoperative myocardial infarction. So the patients should be prepared for surgery in such a way that these changes are less likely to occur. Thus patients, who are presented with a diastolic arterial pressure more than 110 mmHg, should receive antihypertensive treatment. Several days or weeks may be required to stabilize the cardiovascular system. Controlled or uncontrolled hypertension is usually associated with increased cardiac peri-operative morbidity [21, 22]. On the other hand over aggressive treatment of hypertension, that is diastolic pressure less than 85 mmHg may itself increase morbidity or mortality in those with ischaemic heart disease, perhaps due to inadequate coronary artery perfusion pressure[23]. Hypertensive patients with left ventricular hypertrophy are associated with an increased risk of peri-operative myocardial ischaemic due to imbalance of myocardial oxygen supply and demand, even in the absence of coronary artery disease[24].

Table-VIII

Goldman's multifactorial Cardiac Risk Index (CRI)

A patient's score is totaled and used to calculate the risk of major complications associated with surgery

Risk factor		Points
1.	History	
	Age> 70 years	5
	Myocardial infarction in preceeding 6 months	10
2.	Physical examination	
	Third heart sound or gallop rhythm	11
	Aortic stenosis	3
3.	ECG·	
	Rhythm other than sinus or atrial ectopic beats on ECG	7
·	More than 5 ventricular ectopics per minutes	7
4.	General status·	
	PO_2 <8kPa (<60mmHg) or PCO_2 > 6.7kPa (>50 mmHg)	3 each
·	K+ <3mmol/L or bicarbonate < 20 mmol/L	
·	Blood urea nitrogen (BUN) > 8.3mmol?l or creatinine >270 mmol/L	
·	Abnormal liver enzyme or signs of chronic liver disease	
·	Patient bedridden from non-cardiac causes	
5.	Operation	
	Intraperitoneal, intrathoracic, or aortic operation	3
	Emergency operation	4
	Total possible score	53

Table-IX

Computation of cardiac risk (Goldman et al.[18]

Risk of major complications associated with surgery

Class (number of points)	Cardiac death	Life-threatening complications
I (0 – 5)	0.2%	0.7%
II (6 – 12)	2%	5%
III (13 – 25)	2%	11%
IV (> 26)	56%	22%

Table-X

Incidence of perioperative re-infarction in relation to interval between first MI and Surgery

Interval since last MI	Re infarction risk
Under 3 months	up to 30%
3-6 months	up to 15%
Over 6 months	up to 6%

Table-XI

Incidence of perioperative MI : Retrospective studies

- - 0.4 % MI, in previous healthy patient
- 3.2 - 7.7 % MI, in patient with previous MI
- 50% are Silent
- Occurrence - majority after 3rd day of surgery
- Mortality - 40% - 60% in preoperative MI

Table-XII

Incidence of Perioperative Re-infarction in relation to duration of operation and site of operation.

Duration of operation (h)	Upper abdominal intrathoracic operation %	Other operative site%
<3	5.9	3.6
>3	15.9	3.8

*P <0.05 compared with other site

(C) Respiratory complication

Although the post-operative pulmonary complications are very frequent, pre-operative respiratory functional tests are not necessarily helpful in their prediction. One retrospective study by Nunn and colleagues examined patients undergoing elective surgery who had a severely limited forced expiratory volume (FEV_1< 1) on pre-operative assessment[25]. They found the only useful predictors of the need for postoperative ventilation to be the combination of a pre-operative arterial oxygen tension of less than 9kpa and the presence of dyspnoea at rest.

Preoperative therapy guideline

Disease	Therapy
Respiratory disease	• Chest physiotherapy, • sputum for bacteriological test and culture, • Appropriate antibiotic therapy • Bronchodilators – where applicable • Avoidance of drug which releases histamine and SRS
Cardiovascular disease·	Antihypertensive drug. Diastolic pressure
• Hypertension	should be < 110 mmHg
• MI	Interval between the attack of MI and surgery should be > 06 months
• Valvular Heart Disease	Antibiotic therapy against bacterial endocarditits
• Arrhythmias	Drug therapy
• Conduction Defect	Insertion of pace-maker before operation, if necessary.
Renal disease	Up-to-date blood urea, electrolytes, serum creatinine estimation.Correction of uremia and potassium imbalances
Liver disease·	Obstructive jaundice · Mannitol at or just before induction.·
• Hepato-renal syndrome	• IV fluid should be started night before surgery.
• Bleeding problems	• Inj. Vit K 10mg iv for 3 days before surgery.
Smoking habit	Stop smoking 12 hrs for reversal of short term effect and 6 – 8 weeks for reversal of long term effects
Alcohol consumption	Better allow than to have withdrawal syndrome
Endocrine disease	Control by direct suppression of endocrine over activity or its effect on target organs.
Diabetes mellitus	Close control of blood glucose concentration.
Steroid	Additional steroid cover is required before induction and start of operation.
Contraceptive pill	
• For-progesterone containing pill	Medication need not be stopped.
• For oestrogen containing pill:	• Stop 4 weeks pre-operatively and recommence at the start of first menstrual cycle post operative.
If early mobilizationpost operatively	• Heparin prophylaxis is not required.
If pill not stopped &/or-early mobilization isvnot possible postoperatively.	• Low-dose S/C heparin is indicated.
Hormone replacement therapy(HRT)	• No special precaution is required.
Dental condition	•
• Loose teeth·	• May be removed before anaesthesia to prevent dislodgement and aspiration.
• Poor hygiene	• Referral to an oral surgeon before operation.

Preoperative fasting

American Society of Anaesthesiology Fasting Guidline.

Ingested material	Minimum fasting [a]
Clear liquid [b]	2 hours
Breast milk	4 hours
Infant formula	6 hours
Non-human milk	6 hours
Light meal [c]	6 hours

a. Fasting times apply to all ages.

b. Example: water, fruit juice without pulp, carbonated beverages, clear tea, black coffee.

c. Example: dry toast and clear liquid. Fried or fatty foods may prolong gastric emptying time. Both amount and type of food must be considered.

The guidelines recommend no routine use of gastrointestinal stimulants, gastric acid secretion blockers or oral antacids.

Providing information to the patient and obtaining consent:

- All patients should be told of common complications.
- All patients should be told what they may experience.
- All patients should be given the opportunity to ask questions.

Consent

- The patient must have the capacity to consent to the treatment offered.
- The patient must have sufficient information to enable him/her to make a balanced decision to consent.
- The consent must be voluntary.
- If in doubt - Consent should be sought from a person with parental responsibility. Capacity may also be invalidated by a patient's confusion, pain, shock, or fatigue, and administration of some drugs such as opioid, analgesics or benzodiazepine premedication.
- Appropriate advice should be sought if there is any concern.

Conclusion:

It is strongly recommended that the anaesthesiologist should be careful enough in preanaesthetic evaluation to recognize the risk factors. Necessary treatment of the co-existing diseases should be arranged to bring the patient in his/her best possible physiological state prior to operation. This can be done in the anesthetic clinic, where the patient prior to surgery be referred. Discussion between anesthesiologist and surgeon is essential for optimum prediction of risk. This will reduce the perioperative morbidity and mortality. Prevention is always better than treating the catastrophe, which may fall due to the lack of proper pre-anaesthetic evaluation.

Moreover, informed consent of the patient and proper documentation of anaesthetic record sheet are emphasized. These will allow the anaesthesiologist involved to stand on a solid foundation for patient's perioperative care and medico-legal question, if any.

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Moyamoya Disease: A Rare Entity Report of One Case

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Summary:

Moyamoya disease is a disease of the cerebral vessels at the base of the cranium and is most prevalent in Japan. It is relatively rare in other parts of the world. Though few cases have been reported from different Asian and some European countries, to the best of our knowledge, no case of Moyamoya disease has been reported in our country so far. Here we report a case of Moyamoya disease in a 9 years old girl diagnosed in the department of neurosurgery of Bangabandhu Sheikh Mujib Medical University. We believe this is the first detected case in our country.

key words: Moyamoya, Puff of smoke, MRI, MR angiogram.

(J Bangladesh Coll Phys Surg 2012; 30: 41-43)

Introduction:

Moyamoya disease is a clinical entity with bilateral stenosis or occlusion of the large intracranial vessels at the base of the brain, with proliferation of fine collaterals.[1] The term "moyamoya" was introduced by Suzuki and Takaku in 1969 to characterize the angiographic appearance of the condition, which shows a collateral network of blood vessels at the base of the brain. As is described in the text, the Japanese word 'moyamoya' means "something hazy just like a puff of cigarette smoke drifting in the air."[2-4]

Case Report:

A 9 years old girl came to us with the complaints of occasional seizures especially following crying or eating hot spicy food. Clinical examination revealed that she is a quite intelligent and playful girl without any neurological deficit. She did not give any history of head injury or family history of epilepsy. Her CBC and other relevant biochemical parameters including blood glucose, renal functions, liver functions and serum electrolytes were within normal limits. So, to find out the cause of the seizure an MRI of brain was done and in T2WI (Fig-1) and FLAIR (Fig-2) it showed multiple flow voids in the area of circle of Willis and in the basal ganglia. MR angiogram revealed characteristic Moyamoya vessels with typical "puff of smoke" appearance (Fig-3).

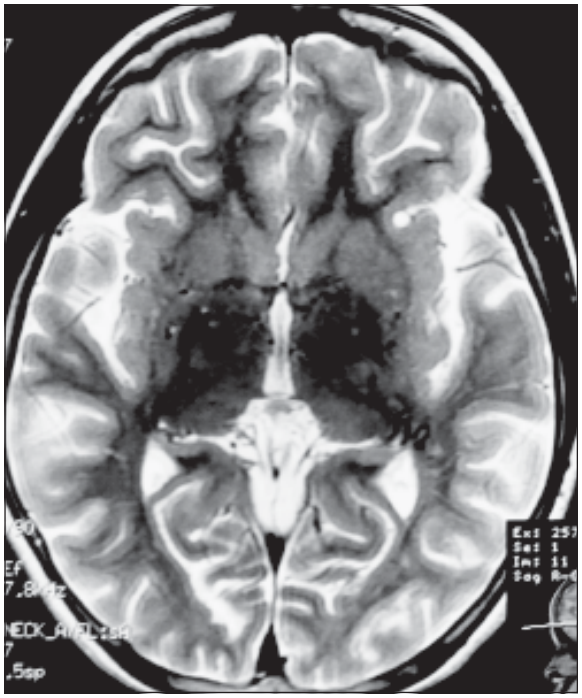

Fig.-1: *MRI in T2WI (Axial view) shows multiple flow voids in the area of circle of Willis and in the basal ganglia.*

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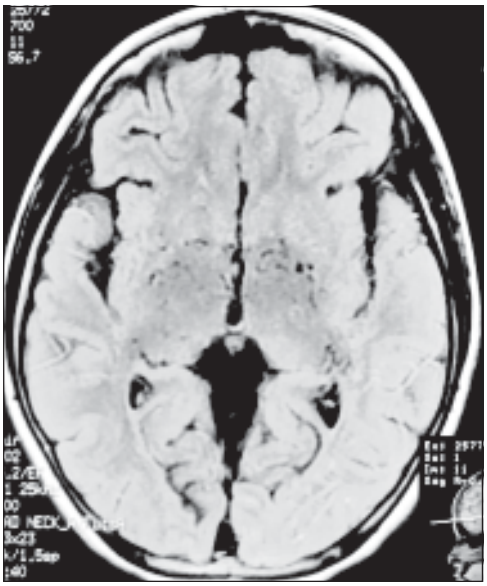

Fig.-2: *FLAIR image of MRI (Axial view) showing multiple flow voids in the area of circle of Willis and in the basal ganglia.*

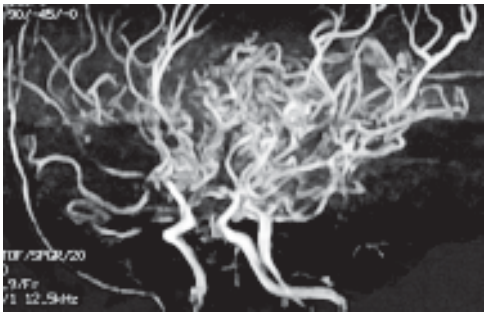

Fig.-3: *MR Angiogram (Right oblique view) showing characteristic Moyamoya vessels with typical "puff of smoke" appearance.*

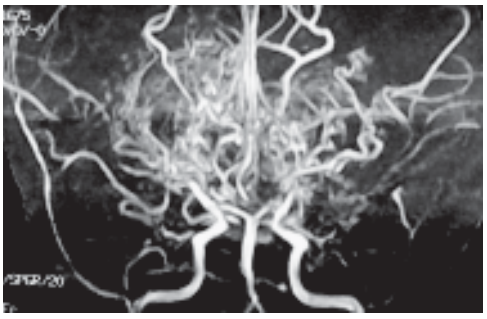

Fig.-4: *MR Angiogram (A P view) shows stenosis of bilateral internal carotid arteries.*

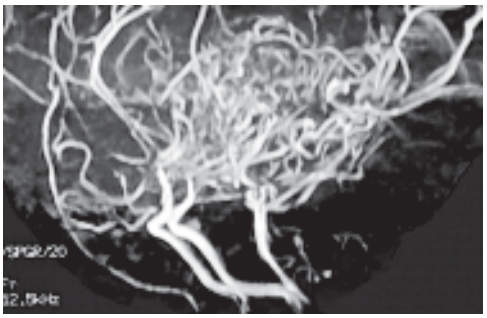

Fig.-5: *MR Angiogram (Lateral view) showing collateral Moyamoya vessels with "puff of smoke" appearance.*

Discussion:

Moyamoya disease is a rare idiopathic vaso-occlusive disease characterized by progressive irreversible occlusion of main blood vessels to the brain as they enter into the skull. The blockage tends to cause strokes or seizures.[4] Many clinical features specific to moyamoya disease have been reported and cited in textbooks. The types of clinical findings can be divided into four subgroups: (a) ischaemia, including infarction and transient ischaemic attacks; (b) haemorrhage; (c) no symptoms and (d) other symptoms, including headaches, seizures and involuntary movements.[5] Cerebral infarction and disturbed cerebral haemodynamics were detected in 20% and 40% of the involved hemispheres, respectively. [06] Our patient presented only with having seizures while under stress and intake of spicy food, most likely because of failure to meet the required metabolic demands from disturbed cerebral haemodynamics. She was otherwise asymptomatic probably because she presented at a very early stage before developing any clinical manifestation other than seizure. A Japanese study also revealed that patients with asymptomatic moyamoya disease are not always rare and the percentage of asymptomatic cases was 17.8% in Japan. Higher figures than previous reports probably reflect the availability of appropriate diagnostic tools and the brain check-up system that has been extensively developed in Japan. [05] She presented in her first decade and one of the well-known specific features of moyamoya disease is its two-peak pattern of age distribution and its higher incidence in childhood. The first peak being in the first decade and the second being in the fourth decade and the female to male ratio is 2.2 : 1.[5] Though conventional angiography or DSA

are the investigations of choice, MR angiography has become a reliable diagnostic modality for moyamoya disease. [07] MRI not only reveals areas of infarctions but also allows direct visualization of collateral vessels as multiple small flow voids. MR angiography is used to confirm the diagnosis and to see the anatomy of the vessels involved. It typically reveals the narrowing and occlusion of proximal cerebral vessels and extensive collateral flow through the perforating vessels demonstrating the classic 'puff of smoke' appearance.[4] The T2WI of MRI of our patient showed multiple flow voids at and around the base of the brain. Her MR angiogram revealed stenosis of the bilateral carotid systems (Fig-4) as well as the typical "puff of smoke" appearance (Fig-5) of collateral vessels. Several surgical procedures, which can be classified as direct and indirect bypass methods, have been proposed for the treatment of this disease. The direct bypass techniques that have been proposed are vein grafts and EC-IC arterial anastomoses (STA-MCA and OA-MCA anastomoses).The indirect techniques are as follows: 1) Encephaloduroarteriosynangiosis; 2) Encephalo-myosynangiosis; 3) Encephalomyoarteriosynangiosis; 4) the use of multiple cranial bur holes; and 5) transplantation of omentum. Other options such as cervical carotid sympathectomy and superior cervical perivascular ganglionectomy have also been proposed. Medical management had not been proven capable of controlling or improving the disease.[2] We explained and counseled the parents about the young girl in details specially regarding the prognosis and outcome of the disease with and/or without surgery and offered for an EC-IC bypass. The parents informed us that they would let us know their decision later. So, for the time being the girl has been given prophylactic anticonvulsant and is kept under observation at the moment.

Conclusion:

The patient fulfilled the clinical and angiographic criteria for definitive diagnosis of moyamoya disease, which is a rare entity in our country. And to our knowledge it is the first diagnosed case in Bangladesh.

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Jejunogastric Intussusception: A Case Report

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Summary:

Jejunogastric intussusception is an established complication following any type of gastroenterostomy. In its acute form it presents with abdominal pain and lump suggestive of obstruction. It is also a rare cause of haematemesis. Chronic and intermittent presentation has also been described. It is a surgical emergency in its acute form. Early diagnosis and prompt treatment is required to avoid mortality. We report here a case that had a history suggestive of recurrent symptoms and ultimately presented as an acute emergency in the emergency department of Dhaka Medical College Hospital, Dhaka, Bangladesh.

(J Bangladesh Coll Phys Surg 2012; 30: 44-47)

Introduction:

Jejunogastric intussusception (JGI) is a rare but well recognized postoperative complication of any type of gastroenterostomy. It may occur any time and has been reported after 2 days up to 35 years[1]. In its acute form, it is a surgical emergency. Early diagnosis and prompt surgical intervention is crucial in such cases. Chronic and intermittent form has also been described. Here we present such a case that presented to us with abdominal pain, lump and haematemesis.

Case Report:

A 40 year old male patient was admitted into a medicine unit of our hospital, and later transferred to the surgery department, with the complaints of severe upper abdominal pain, haematemesis and a lump over mid-abdomen for 6 hours. Pain was localized to upper abdomen, severe colicky at first and then became excruciating in nature. It was associated with several episodes of spontaneous, projectile vomiting and haematemesis. Patient underwent elective gastrojejunostomy bypass operation in 1994 in DMCH for pyloric stenosis due to chronic duodenal ulcer. Over the period of last 10 years, patient had been experiencing moderate to severe abdominal pain associated with nausea and vomiting on and off. During this period he was hospitalized several times and managed conservatively.

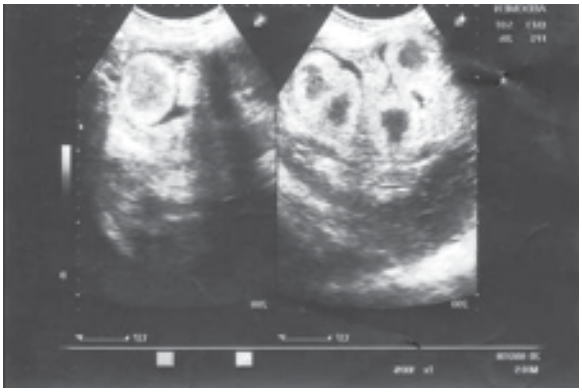

Fig 1: *Preoperative USG showed a large central abdominal mass containing encysted collection with thick walled bowel loops.*

On examination, he was moderately anemic and dehydrated. Pulse was 96 /min, BP was 105/60 mm of Hg. There was a hemispherical lump, about 6x7cm in size, firm, slightly tender, mobile, free from anterior abdominal wall occupying umbilical and left lumbar region. There was no ascites. Bowel sound was absent. Per rectal examination was normal.

The routine biochemical profiles were within normal limit including serum amylase. Plain X-ray abdomen in erect posture was normal. Abdominal ultrasound revealed a fairly large mass containing encysted

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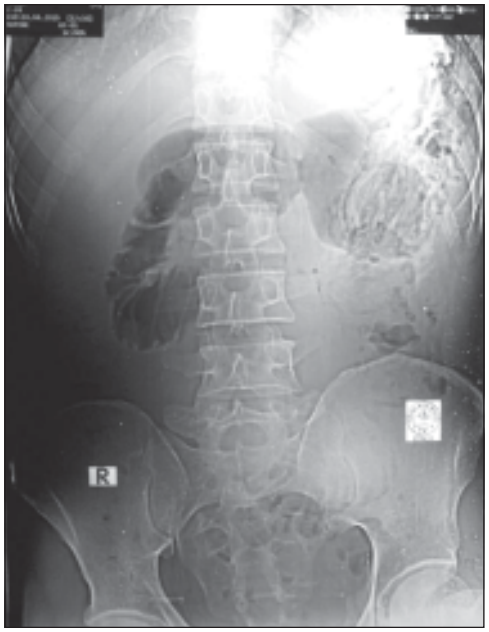

Fig 2: *Water soluble contrast x-ray – filling defect within the stomach. The contrast medium failed to pass beyond the stoma.*

collection with thick walled bowel loop in the upper part of central abdomen and in the left side. Water soluble contrast x-ray of stomach and duodenum showed filling defect inside the stomach and absence of contrast medium beyond the stoma. Upper GI endoscopy showed intussusception of jejunum into the stomach through gastrojejunostomy stoma. Mucosa of the protruded intestinal loop (intussusceptum) was black due to ischaemic necrosis. The diagnosis of JGI was established and an emergency laparotomy was performed.

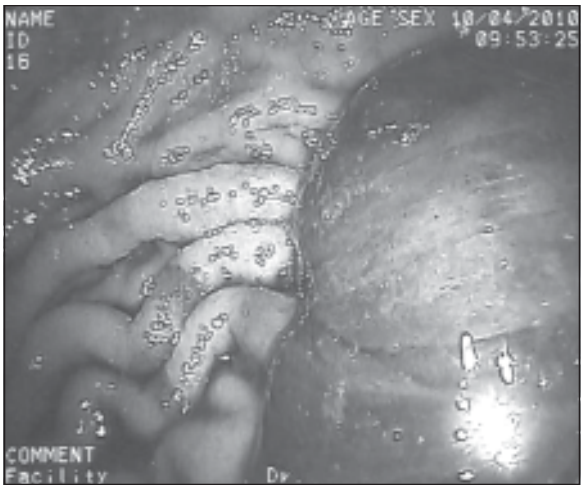

Fig 3: *Preoperative endoscopy revealed gangrenous jejunal loop (intussusceptum) within the stomach.*

At laparotomy, the efferent loop was found intussuscepted in a retrograde way into the gastric lumen. Reduction of the intussusception was performed. The gangrenous segment was resected followed by jejunojejunal anastomosis. A feeding jejunostomy tube was placed distal to anastomosis. Postoperative recovery was uneventful.

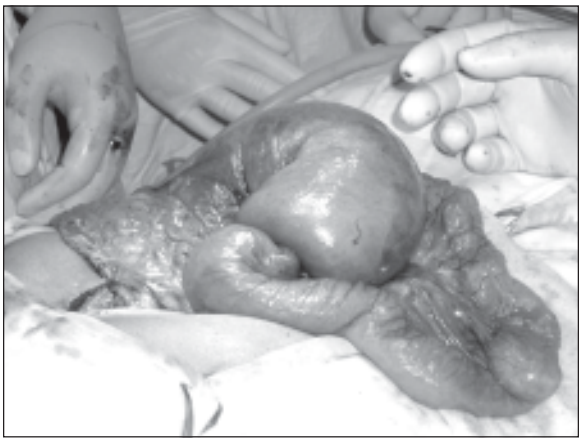

Fig 4: *Intraoperative photograph demonstrating the JGI*

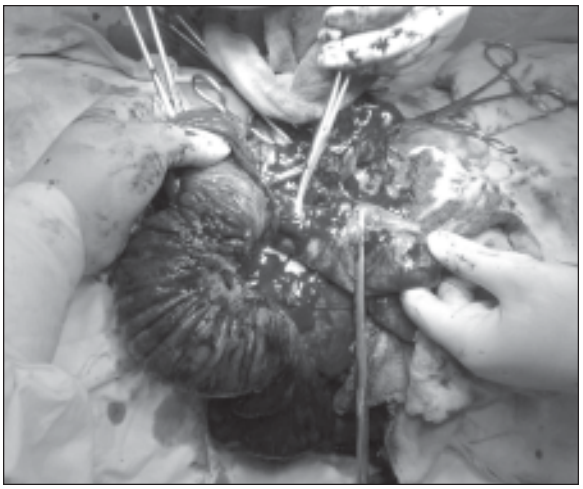

Fig 5: *Intra operative photograph showing gangrenous jejunal segment after dismantling*

Discussion:

Retrograde JGI was first described in a case of gastrojejunostomy by Bozzi in 1914[1]. Later it was also reported after partial gastrectomy and other operations.

It is a rare entity and just over 200 cases have been reported till 2008[2].

Commonly held anatomical classification proposed by Shackman distinguishes three categories of JGI:

Type I – where afferent loop is the intussusceptum constituting 15% cases;

Type II – where efferent loop enters the stomach in a retrograde way. This is the commonest group (75-80%);

Type III – represents a combined form in about 10% cases

Recently a fourth type where intussusception occurs through enterostomy (Braun anastomosis) stoma has also been described[3].

The exact cause or mechanism of JGI is poorly understood, but various factors have been implicated. These include: (1) long afferent loop, (2) jejunal spasm with abnormal motility, (3) increased motility of efferent loop, (4) adhesions leading to intussusception of a more mobile segment into fixed segment, (5) widening of upper jejunum, (6) hyperacidity, (7) increased intraabdominal pressure[2,3]. Retrograde peristalsis, seen in people prior to gastric surgery, seems to be accepted as a cause of retrograde JGI[5,6].

Clinically two forms of JGI have been widely accepted: an acute and a chronic form. Again, there are two clinical types in an acute variety[5,7]. In the first type, the patient is suddenly seized with an acute attack of epigastric pain followed by a sensation of severe constriction of abdomen. A mass may be palpable in the central abdomen. Here, early operation has proved to be life saving in 90% of the cases. The second variety may be confused with a bleeding stomal ulcer, afferent loop syndrome or obstruction due to adhesions. Vomiting is frequent, being at first bloodstained and then frankly haemorrhagic. Since the medical line of treatment is usually tried first, a delay in surgery occurs causing more morbidity and mortality. In both types, however, spontaneous reduction is rare. It should be kept in mind that sudden onset of severe epigastric pain, vomiting and subsequent haematemesis and a palpable abdominal mass in a patient having a history of previous gastric surgery are thought as the classic triad of JGI[8].

The chronic variety is characterized by recurrent bouts of epigastric distress, nausea and colicky abdominal pain. Intermittent and sometimes severe vomiting may also occur. This may be confused with nonspecific abdominal pain and postoperative adhesions. Our case was probably a chronic form for last 10 years, which this time presented as acute variety.

Early diagnosis of the acute form is of paramount importance. The clinical picture is almost diagnostic. It can be confirmed by an upper GI endoscopy which will reveal the jejunal segment migrating into the stomach through the gastrojejunostomy stoma. A water-soluble upper GI contrast study may reveal a "coiled-spring" appearance within the stomach[4]. CT scan is also helpful and findings are characteristic and similar to enteroenteric intussusceptions. The chronic form may pose a diagnostic dilemma simply because the upper GI endoscopy or contrast x-ray is avoided in the symptomatic period. However it has been suggested that in the asymptomatic period, the provocation of JGI during endoscopy by the use of a jet of water directed towards the anastomotic stoma may be diagnostic of the chronic form[9].

The treatment of acute JGI is surgical intervention as soon as possible. Most authors report a mortality of 10% if operation is performed within 48 hours after the onset of severe symptoms and as high as 50%, if operation is delayed[10]. Surgical options vary according to peroperative findings. If the involved segment of jejunum is viable, simple reduction will suffice. Future recurrence of JGI can be prevented by anchoring the involved jejunal segment to either the neighboring jejunal limb of small intestine or to the transverse mesocolon. Gangrenous JGI requires resection of the gangrenous segment of jejunum along with revision of anastomosis. The treatment of the chronic recurrent variety of JGI is symptomatic. If symptoms persist, revisional surgery is performed. The surgical options include reduction, resection, revision of the anastomosis and taking down the anastomosis[11].

Conclusion:

Jejunogastric intussusception, though rare, is a surgical emergency and requires prompt diagnosis. Endoscopy remains the investigation of choice. Early surgical intervention significantly reduces morbidity and mortality. Therefore clinicians should be aware of this possibility in similar cases. For those with chronic intermittent form, endoscopy or imaging at the time of attack or recurrent symptoms may establish diagnosis.

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Dentatorubral Pallidoluysian Atrophy (DRPLA)-A Rare Neurological Disorder

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Abstract:

Dentatorubral pallidoluysian atrophy (DRPLA) is a rare type of an autosomal dominant progressive late onset cerebellar ataxia. It is caused by a defect in a gene [CAG repeat in atrophin-1 (ATN1) on chromosome 12] and results in damage to the cerebellum, and to its connections within the central nervous system. It is also known as Haw River Syndrome and Naito-Oyanagi disease. This disorder seems to be very rare except in Japan. Patients with DRPLA can exhibit a variety of psychiatric symptoms in addition to extrapyramidal and cerebellar symptoms. We report a case who was 28 years old unmarried man presented at BSMMU, Dhaka in 2010. The present work highlights the variable mode of presentation of DRPLA and the difficulty of an early diagnosis without facility of genetic analysis. The aim of our report was to describe typical clinical presentations of the disease without positive family history. Our patient presented with unsteadiness during walking, irrelevant behavior and talking, incontinence of urine and stool with occasional myoclonic seizure and dementia.

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Introduction:

Dentatorubral-pallidoluysian atrophy (DRPLA) is a neurodegenerative disorder characterized by an unstable CAG trinucleotide repeat in the DRPLA gene on chromosome 12q12, leading to pathological changes in the brain.[1,2] The human genome contains two atrophin genes; DRPLA has been correlated to the expansion of the polyglutamine region of the atrophin-1 gene on chromosome 12p13.3[3]. A normal number of CAG repeats in the atrophin-1 gene is 7-34. Affected individuals display 49-93 repeats[4]. Usually, patients with large expansions have an early onset and a rapid disease progression[1]. DRPLA is a rare condition with an estimated prevalence of 0.2–0.7/100,000[5] population in Japan. There is significant reduction in CNS tissue throughout the brain and spinal cord, with brain weights of DRPLA patients often becoming less than 1000g[6].

In regions lacking obvious neuronal depletion, atrophy of the neuropil is noted. The globus pallidus and subthalamic nucleus demonstrate consistent neuronal loss and astrocytic gliosis. In general, the pallidoluysian degeneration is more severe than the dentatorubral degeneration in juvenile-onset and the reverse is true for the late adult-onset[7]. Patients with DRPLA manifest various combinations of symptoms such as epileptic seizures, myoclonus, ataxia, dementia[8,9] and psychiatric symptoms[10-12]. There are two forms of DRPLA- the juvenile and adult forms[7], In juvenile onset patients, particularly under the age of 30years, it tends to present with a progressive myoclonic epilepsy-picture with more marked dementia than is seen in patients with Huntington's Disease. In patients with adult onset typically above the age of 30years, DRPLA typically presents with a milder phenotype with ataxia, mild chorea and milder cognitive impairment[13]. Diagnosis of DRPLA rests on positive family history, clinical findings, and genetic testing. Family history can be difficult to obtain if a relative was misdiagnosed, died young, or experiences late onset of symptoms. Here we report the patient with DRPLA of juvenile onset which could not confirmed by genetic identification.

Case history

A 28 years-old ex-smoker, right handed, nondiabetic, nonhypertensive, unmarried male from Comilla, Bangladesh, reported us with the complaints of unsteadiness during walking for 8 years, irrelevant behavior and talking for 2years, incontinence of urine

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and stool for the same duration. According to the statement of mother of the patient he was reasonably well 8years back. Since then he developed progressive unsteadiness during walking. At the same time she also noticed sudden, involuntary, jerky movement of head followed by fall without loss of consciousness. For this reason they consulted several consultants and got some sort of relief. But from November 2007, he experienced many episodes of seizure. The seizure was generalized with loss of conciousness, lasted for several minutes, associated with incontinence of urine and stool. He also developed difficulty in swallowing which was more for liquid, poverty of speech, forgetfulness and abnormal posture of limbs. His speech became irrelevant. He could not take food himself without caregiver's support for last 3 years. All his symptoms have been gradually progressing and reaching to such an extent that he became bed bound. There was no significant or relevant disease before the development of this disorder. His birth history was uneventful. The patient comes from a lower middle class family. He took alcohol occasionally but his mother could not mention the duration of his alcohol intake, used to take illicit drug whose name and duration of intake could not mentioned by his attendants. The alcoholism and substance abuse was preceded by the ailment. He completed higher secondary level of education and could not complete his graduation due to this illness. His father and mother are alive with good health. There was no parental consanguinity of marriage. He has one brother and one sister; both are younger to him and are in good health.

Fig.1: *Wheelchair bound patient of DRPLA.*

On general examination he was found apathic, with good body built, well nourished, poorly dressed and awkwardly postured. His blood pressure was 110/90 mm of Hg, pulse-70/min, respiration rate 16/min. There was no organomegaly. No other abnormality was found on general examination. K.F. ring was absent. Examination of higher cerebral function revealed incoherent, irrelevant and incomprehensible speech. MMSE score was 0. All the cranial nerves including fundoscopic examination were normal. Regarding motor system examination; bulk of the muscles was normal, no involuntary movement or tremor was noticed, tone was increased in the form of rigidity, muscle power was MRC grade 4 in four limbs and all the deep tendon reflexes were exaggerated, Hoffman sign was positive with bilateral extensor plantar response. Palmomental reflex and glabellar tap were present but other primitive reflexes were absent. Patient could not stand or walk without support. Co-ordination and sensory examination could not be done as he was unable to follow command properly. Other systems examination revealed no abnormality.

We diagnose the case as DRPLA on clinical basis by excluding some other differentials through clinical ground and available investigations. In the neurology department of BSMMU the patient was treated with Clonazepam 2mg daily and vitamin E capsule 400mg daily for six months. Six month latter (on July 2010) we admitted the patient again in BSMMU, Dhaka for follow up. This time patient was euphoric, not responding to command verbally but can speak or sing a song at his will. He was seizure free, relatively mute and other features were as before. We added memantine 10mg 12 hourly, Co-dopa 110mg 12 hourly to continue for indefinite period. This time psychiatric evaluation was done. The psychiatrist was also agreed with the diagnosis and advised to continue the ongoing treatment as the patient was not aggressive. We advised the attendant follow up visits every 6 (six) months.

The laboratory investigation reports are summarized in the table below:

Sl. No	Name of investigation	Date	Result/comment
1	Urine R/M/E	6.01.10	Protein-trace.
2	TC of WBC	7.01.10	11000/cmm
3	DC of WBC	7.01.10	Neutrophil-66%, Lymphocyte-24%, Monocyte-04%, Eosinophil-06%.
4	ESR	7.01.10	10 mm in 1st hour.
5	Haemoglobin	7.01.10	14 g/dl
	PBF	7.01.10	Unremarkable.
6	Chest X-ray	7.01.10	Unremarkable.
7	Random blood sugar	7.01.10	5.4 mmol/L
8	S. creatinine	7.01.10	0.7 mg/dl
9	S. bilirubin	7.01.10	0.8 mg/dl
10	S. ALT	7.01.10	26 U/L
11	S. calcium	7.01.10	8.8 mg/dl
12	S. electrolyte	7.01.10	Na-137 mmol/L, K-4.4 mmol/L,Cl-99 mmol/L,TCO_2-21 mmol/L
13	VDRL	7.01.10	Non reactive.
14	TSH	7.01.10	0.29 mIU/L.
15	Vitamin B_{12} Assay	7.01.10	430 pgm/ml.
16	Anti-HIV (1and 2)	9.01.10	Negative
17	ECG	9.01.10	Normal
18	EEG	11.11.09	Normal.
19	CT scan of brain	10.11.09	Generalized cerebral cortical atrophy of brain with ventriculomegaly.
20	MRI of brain	10.01.10	Marked degenerative atrophy of the whole brain with compensatory ventriculomegaly.
21	USG of abdomen	25.01.10	Normal study
22	CSF	2.2.10	Cytology-TC-2/cmm. 100% lymphocyte.Protein-40mg/dl. Sugar-73.26mg/dl.Gram stain and AFB stain- negative.Culture and sensitivity-no growth.VDRL-non reactive.

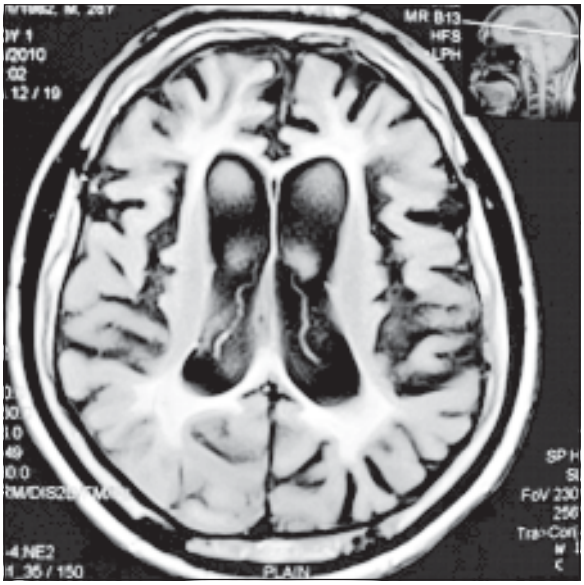

Fig.2: *FLAIR axial image of Brain MRI showing gross cerebral atrophy with compensatory lateral ventricular dilatations and periventricular white matter changes.*

Discussion:

Our patient developed a picture of severe ataxia, spastic quadriparesis, dystonia in the form of abnormal posture, myoclonic epilepsy, rigidity, dementia, and psychosis. The MRIs showed a marked degenerative atrophy of the brain with evidence of demyelination. Previous MRI studies in DRPLA showed atrophy of the cerebral cortex, cerebellum, brainstem and in some instances, white matter signal alterations in T2 and fluid-attenuated inversion recovery sequences[14-16]. White matter involvement was not uncommon in a number of autopsy specimens of Japanese and non-Japanese patients with DRPLA[16-19]. Our case highlights the fact that structures other than brainstem and cerebellum may play an important role in disease progression in some patients with DRPLA.

No test for excluding hereditary metabolic diseases of nervous system could be done due to non-availability of tests in our country and patient's financial constraints. So we have to exclude some differential diagnosis on the basis of clinical features and available investigations. We considered presenile dementia of Alzheimer type,

Huntingtons disease (HD), Creutzfeldt-Jakob disease (CJD), Frontotemporal dementia (FTD), lewy body dementia (LBD), alcoholic dementia, postencephalitic syndrome, metachromatic leucodystrophy (MLD), Niemann-Pick disease, Sialodosis, â-mannosidosis, juvenile and adult onset Sandhoff disease and Tay-Sachs disease as our differential diagnoses. Our patient had seizures, gait disturbance and incoordination at the onset or very early in the course of the illness. According to NINCDS-ADRDA criteria for the diagnosis of alzheimer's disease (AD) these features made the diagnosis of AD unlikely.[20] Symptoms of HD commonly become noticeable between the ages of 35 and 44 years. The most characteristic initial physical symptoms are chorea. In HD suicidal thoughts and suicide attempts are more common than in the general population.[21] Muscle atrophy, cardiac failure, impaired glucose tolerance, weight loss, osteoporosis and testicular atrophy usually found in HD.[22] These findings were inconsistent with our case. The constellation of dementia, myoclonus, and periodic electrical bursts in EEG, an afebrile middle aged patient generally indicates CJD. There should be a history of accidental transmission of CJD. Death usually occurs within 5 years of onset.[23] EEG was normal in our patient. In FTD significant impairment of frontal lobe tests in the absence of severe amnesia, aphasia, or perceptuospatial disorder; predominant frontal and/or anterior temporal abnormality is frequently present.[24] But our patient had severe generalized brain atrophy. In LBD there is usually fluctuating cognition with pronounced variations in attention and alertness, recurrent visual hallucinations which are typically well-formed and detailed, repeated falls, syncope, transient loss of consciousness, systematized delusions and hallucinations in other modalities[25] which are lacking in our patient. Alcoholic dementia is a form of dementia caused by long-term or excessive drinking. The front part of the brain is mostly affected, resulting in poor judgment, difficulty in making decisions, and lack of insight. Long-time alcohol abuse can often lead to poor nutritional problems causing parts of the brain to be damaged by vitamin deficiencies. These problems could also cause personality changes in some people.[26] Because these warning signs are so similar, alcohol dementia can be difficult to diagnose. Our patient was an occasional drunker and he had generalized atrophy of brain. He had some additional features like seizure, psychosis and parkinsonian features. In his third decade of life with all other features, it was unlikely to be an alcoholic dementia. In postencephalitic dementia there should be a history of encephalitis followed by dementia. Symptoms of the adult form of MLD are- onset after 16 years, mental deterioration, impaired concentration, depression, psychiatric disturbances, ataxia, tremor, dementia, reduced muscle tone, quadriplegia, leukodystrophy, megacolon, muscle wasting, muscle weakness, developmental delay and progressive vision loss. But our patient had neither visual loss, nor tremor, or muscle wasting. In addition our case had no megacolon, or developmental delay. He had epilepsy. So it may not be a case of metachromatic leukodystrophy. Dystonia, splenomegaly, hepatomegaly, dementia, seizures, slurred and irregular speech, cataplexy, tremors, vertical supranuclear gaze palsy and ataxia are the usual features of Niemann-Pick disease [27]. Our case had no organomegaly, cataplexy, tremors, vertical supranuclear gaze palsy. Characteristic features of juvenile onset Sialodosis, are: dysostosis multiplex, cherry-red spots on macula, decreased visual activity, myoclonus and angiokeratomas[28,29]. Most of the features were lacking in our patient. Severe psychomotor retardation, bone deformities, gargoylism, recurrent skin and respiratory infections indicate â-mannosidosis[30]. Sandhoff disease symptoms are clinically indeterminable from Tay-Sachs Disease. Main features of these diseases are cognitive impairment, incoordination of muscle and the characteristic red spots in the retina. The adult form of the disease, however, is sometimes milder and may only lead to muscle weakness that impairs walking or the ability to get out of bed[31]. These features are not consistent with our case.

Treatment:

No information is available about the treatment of DRPLA, the clinician should be guided by the nature and severity of symptoms[32]. Treatment is only symptomatic.

Limitations:

In Bangladesh we have no available facilities for the genetic analysis of this kind of disease. The patient also had no family history of such disease. So we are not confirmed about the diagnosis of the case. For confirmation further evaluation can be done.

Conclusion:

Sporadically mutated cases of autosomal dominant diseases are difficult to diagnose in the absence of facilities for genetic analysis. DRPLA may be found in non Japanese asian people if carefully searched. It is a progressive disease with no curative treatment available at present. Treatment should be symptomatic. Proper

nutrition, hygiene, regular physiotherapy, nursing and counseling are essential part of management of this type of patient.

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Addison's Disease, Idiopathic Intracranial Hypertension and Empty Sella - In a 40-Year-Old Woman

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Summary:

A 40-year-old woman having normal menstrual history got admitted into Bangabandhu Sheikh Mujib Medical University (BSMMU) because of vomiting for previous two months, with a background history of irregular steroid intake for her bodyache for about six years. She was gaining weight and gradually losing vision in her right eye and noticed increased body pigmentation. Physical examination revealed BMI 29 kg/m^2, generalized pigmentation more marked in palmar crease and oral mucosa, secondary optic atrophy in right eye and papilloedema in left eye. After biochemical and radiological investigation she was diagnosed as a case of Addison's disease with idiopathic intracranial hypertension (IIH) with empty sella.

(J Bangladesh Coll Phys Surg 2012; 30: 53-55)

Introduction:

Idiopathic intracranial hypertension (IIH) can rarely be associated with an underlying endocrine disorder such as Addison's disease[1], Cushing's syndrome, hyperthyroidism, hypoparathyroidism or with administration of thyroxin or growth hormone. Though cases of IIH associated with Addison's disease have been in reported in children, there are very few documented case reports of this association in adults[2, 3]. We describe a case of an adrenal insufficiency associated with IIH leading to empty sella in a 40-year-old female.

Case Presentation

A 40-year-old woman with a normal menstrual history was admitted to BSMMU because of vomiting for previous two months. She was taking tablet predisolone 5mg irregularly about 6 years for her bodyache. She was gaining weight and at the same time noticed increased body pigmentation. She was also gradually losing vision in right eye. She had no history of postpartum hemorrhage. On examination she was conscious, obese (BMI 29 kg/m^2), afebrile and her blood pressure was 100/70 mmHg with no postural drop. She had generalized pigmentation including pigmentation of her palmer crease (Fig 1) and oral mucosa. Opthalmoscopy revealed secondary optic atrophy in right eye and papillodema in left eye. No focal sign was found on neurological examination. All her secondary sex characteristics were normal. Initial biochemical analysis revealed serum sodium 130mmol/L, serum potassium 4.1mmol/L, serum creatinine 1.2 mg/dl, plasma random glucose 5.9 mmol/L, normal complete blood count with normal PBF, ESR 40mm in first hour. MRI of brain demonstrated empty sella (Fig 2). Lumbar puncture performed in lateral decubitus position revealed CSF pressure of 200mm of water. CSF analysis showed WBCs 4/mm^3, RBC nil, organism nil, protein 0.3g/L and glucose 3.5mmol/L. MT was 5mm, x-ray chest normal, and abdominal ultrasound including adrenal glands revealed no abnormality. Her baseline endocrine test is seen in Table 1.

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Table-I

Baseline endocrine test reports

Test	Result	Comment
ACTH	207.0 pg/ml	high
Serum Cortisol	25.5 nmol/L	low
LH	39.02 IU/L	normal
FSH	19.43 IU/L	normal
Prolactin	14.90 ng/ml	normal
TSH	1.49 mIU/L	normal
FT4	11.20 pmol/L	normal

Initially she was put on oral hydrocortisone 30mg, ondensetrone 24mg and acetazolmide 500mg daily in divided doses. At the same time CSF drainage was done. As vomiting continued, with consultation of endocrinologist and neurologist fundal photgraph (Fig 3) and perimetry were done and she was switched to intravenous dexamethasone 15mg, ondensetrone 24mg daily in divided doses. In addition lumbar drainage was done. Vomiting subsided and vision also improved significantly as evidenced by follow up fundal photograph (Fig 4) and perimetry after three weeks. She was then switched to oral dexamethasone followed by oral prednisolone. Her follow-up electrolyte showed persistent hyponatraemia which was corrected by addition of fludrocortisone 0.1mg with prednisolone 7.5mg per day.

Discussion:

According to her background history of steroid intake, obesity and opthalmoscopic findings, initially we labeled her as a case of iatrogenic Cushing syndrome with raised intracranial pressure (ICP). Her vomiting can be attributable to raised ICP. However, presence of pigmentation was the clinical clue that led us to think an alternative possibility. After biochemical analysis she was found to be a case of Addison's disease and her raised ICP could be attributed to Addison's disease. As very low serum cortisol and very high ACTH were strongly suggestive of diagnosis of Addison's disease, so we did not go for synecthine test. Her long history of irregular low dose steroid intake was responsible for chronicity of illness but was not sufficient for replacement. There are a few reports about the development of raised ICP in Addison's disease[2,3]. Increased serum and CSF arginine vasopressin peptide (AVP) in a glucocorticoid deficient state is the likely cause of raised ICP in Addison's disease[4]. The mechanism of empty sella in this case is probably due to raised ICP or an autoimmunity which is associated with autoimmune adrenal failure[5]. Our main concern was cortisol replacement and to halt the progression of visual loss. During management with the physiological replacement dose of corticosteroid her hyponatraemia was persistent, which was corrected by addition of fludrocortisone. As her vision was improved by acetazolamide and lumbar drainage we did not go for our initial plan of lumbo peritoneal shunt.

IIH is defined as the clinical syndrome of raised intracranial pressure in the absence of the space occupying lesion or vascular lesion, without the enlargement of cerebral ventricles for which no causative factor can be identified[6]. Historically IIH was referred to as pseudotumor cerebri as it mimics an intracranial tumor. More recently it has been referred to as benign intracranial hypertension (BIH). This term has also been abandoned because a small but significant number of patients develop visual impairment or visual loss, as in our case. However, even the current term IIH is inaccurate because this condition frequently associated with obesity or with the use of medication including various antibiotics (tetracyclines, nitrofurantoin and nalidexic acid), amiodarone, cyclosporin, both systemic steroids use and withdrawal[1] and oral contraceptive pill. Moreover various endocrine disorders have also rarely been reported in association with IIH, including Cushing syndrome[7] hyperthyroidism[8] as well as administration of thyroxin or growth hormone[9]. There are very few reported cases of IIH associated with Addison's disease in adult[1, 2]. Our case is an addition to the world literature of such a rare association.

Although the pathophysiology of IIH is uncertain the mechanisms that have been proposed for its development include increased production of CSF, reduced CSF absorption or increase cerebral venous pressure causing a secondary increase in CSF pressure. Analysis of the CSF arginine vasopresssin peptide (AVP) in the patient with IIH demonstrate it to be elevated compared to healthy controls[4] This reveals that patient with glucocorticoid deficiency have increased

plasma level of AVP and a sustained hypersecretion of AVP despite plasma dilution[10]. Thus it is possible that increased serum and possibly CSF AVP may mediate IIH in Addison's disease.

It is to be mentioned here that we were unable to measure serum or CSF AVP to provide the mechanistic link amongst Addison's disease, ICP and AVP. We also failed to rule out the role of autoimmunity in Addison's disease and empty sella. Finally, we could not establish raised ICP purely due to Addison's disease or steroid or combination of both.

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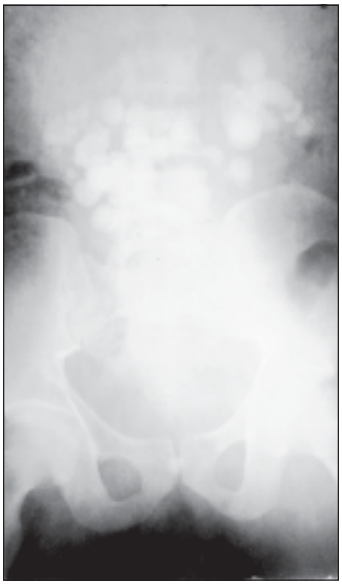
Plain X-ray can be a Good Tool for the Diagnosis of Horse Shoe Kidney

MM RAHMAN

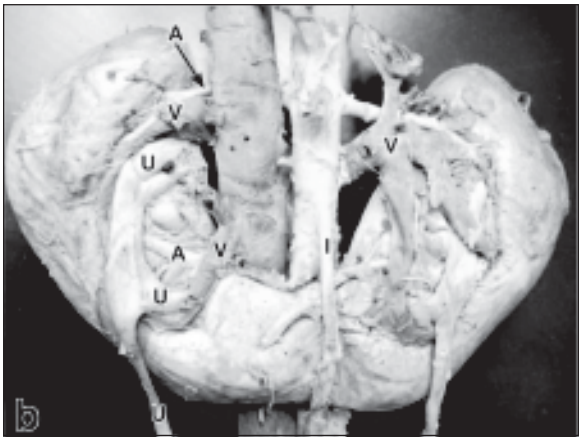
(J Bangladesh Coll Phys Surg 2012; 30: 56-57)

A 32 year-old male presented with non specific abdominal pain for last five years, diffuse swelling lower abdomen, and recurrent attacks of burning micturitation and passage of stone per urethra once. Plain Xray KUB showed multiple round to oval radio opaque shadows suggestive of urinary stones along the upper pelvis and lower abdomen. Ultrasonography and related investigations(IVU) settled it as Horse shoe kidney. The plain X-ray beautifully demonstrated as the stones maintained the line of a horse shoe kidney.

Most of the time, a horseshoe kidney is an incidental finding on an examination for some other condition that the patient is having. But once it is discovered, there are many options for imaging the anomaly. The kidneys can be seen on plain radiographs, but the definition is not as clear as in some of the other modalities. They will be discovered on plain radiographs because of their lower location and the location of the lower poles being more medially rotated than would be expected, ureters as well, to see if there are any abnormalities lower in the system.



A. Plain X ray KUB shows palisades of radioopaque shadows of different sizes suggestive of stones maintaining the shape of horse shoe kidney.



B. Horseshoe kidney shown with descending aorta and inferior vena cava.

Note. From "A horseshoe kidney with partial duplex systems," by K. Ongeti, J. Ongeng, and H. Saidi 2011, *International Journal of Anatomical Variations, 4,* p 56.

Horseshoe kidney is the most common fusion anomaly in the kidney and occurs in about 1 in 400 people, or about 25%. It is also twice as likely to occur in males as in females [1]. Although it is not highly common, it isn't uncommon either. Normally one-third of the patients that have horseshoe kidney are asymptomatic and the condition is noticed incidentally on radiologic examination [2]. Although most patients are

Prof. Md. Mizanur Rahman, Professor, Dept. of Surgical Oncology, National Institute of Cancer Research and Hospital, Mohakhali, Dhaka.

asymptomatic, there are certain conditions that go with horseshoe kidney quite frequently.

Anatomy of the Horseshoe Kidney

Horseshoed kidney is the most common type of fusion anomaly. For the most part, the horseshoe kidney functions as a normal kidney. Many times, kidney malformations are accompanied by lower urinary tract anomalies as well. This is understandable because the kidney and the ureter arise from the same single embryonic structure[3]. With horseshoe kidney, the kidneys can be located anywhere along the normal embryologic ascent of the kidneys. Normally they are located lower in the pelvic region of the body. In 90% of cases, the fusion of the kidneys occurs in the lower poles. In this condition, both kidneys are malrotated and their lower poles are joined. In this condition, both kidneys are malrotated and their lower poles are joined. With the fusion, there is an isthmus that crosses the midline of the body to connect the two kidneys.

Symptoms and Complications:

It is also twice as likely to occur in males than in females[4], Normally one-third of the patients that have horseshoe kidney are asymptomatic, and the condition is noticed incidentally on radiologic examination [2]. Although most patients are asymptomatic, there are certain conditions that go with horseshoe kidney quite frequently.

When symptoms are present, they are usually because of obstruction, stones or infection with urinary tract infection being the most common presenting symptom in children" [1]. Then next most common occurrence is presence of kidney stones. Kidney stones will develop in anywhere from 20%-60% of patients. Kidney stones go hand-in-hand with obstructions; they have a tendency to cause one another.

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LETTER TO THE EDITOR

(J Bangladesh Coll Phys Surg 2012; 30: 58-59)

To

Editor in Chief

Journal of Bangladesh College of Physicians and Surgeons

Sir,

I had gone through the case report of your valuable journal (Vol . 29,No. 4,October 2011) title with " Chronic Disseminated Histoplasmosis in an Immunocompetent Man Presented as Bilateral Adrenal Masses with Partial Adrenocortical Insufficiency- A Rare Condition" by ABM Sarwar-e-Alam et al with keen interest and have few observations.

a. The case report was well written and the contents and illustrations were nice.

b. Disseminated Histoplasmosis involving adrenal gland is a rare disease and under diagnosed and underreported but it is not the first case in Bangladesh as there was another report published in Journal of Medicine 2011;12:81-85. Adrenal involvement is seen in disseminated disease but sometimes it may be the only site of demonstrable disease.[1]

c. Pure form of dissemination of a disease need involvement of two or more than two sites, but in this case only one site is mentioned.

d. Serological diagnosis (antigen and antibody based)[2] is important for diagnosing chronic disseminated histoplasmosis. In this paper no such diagnostic tool was used.

e. The recommended treatment is amphotericin B for critically ill hospitalized patients[3]. Less severe cases can be treated with oral Itraconazole which is the first line drug. In this case Amphotericin was given as first line drug though it was not severe form of disease. Among patient without AIDS, amphotericin B is effective in 68-72% of the cases, itraconazole is effective in 100% of the cases, ketoconazole is effective in 56-70% of the cases and fluconazole is effective in 86% of the cases.[4]

The authors have rightly pointed out that high degree of clinical suspicion for this rare disease should be developed.

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Dr Rukhsana Parvin
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Author's Reply

To

Editor-in-Chief

Journal of Bangladesh College of Physicians and Surgeons.

Dear Sir,

I have got the following reply of the comments mentioned by Dr Rukhsana Parvin:

a. Thanks for her kind remarks,

b. We submitted our case report to your office on 06/10/2010 prior to the publication mentioned by her which was published in Journal of Medicine 2011; 12:81-85.

c. The literal meaning of "Disseminated histoplasmosis" is progression of histoplasmosis beyond the initial focus in the lung (usually) or skin (rarely) to other organs. In other word we can say, disseminated histoplasmosis is a progressive extra-pulmonary infection. So there was no doubt about disseminated histoplasmosis in our case. Involvement of two sites or more sounds the diagnosis anatomically and clinically as

disseminated form. In our case, two adrenals were involved.

d. We agree that the specific diagnostic laboratory tests are –detection of antigen of *H. Capsulatum* in urine and serum, histopathology, antibody to *H. Capsulatum* in serum, culture of blood or tissue samples. There is no doubt, culture or biopsy is the definite tool for diagnosis of disseminated histoplasmosis. Both the tests were done in our case. Tests for antigen and antibody may be falsely +ve or –ve in good number of cases. Moreover In non-AIDS patients with disseminated histoplasmosis, serological test is of limited value.

e. Our patient was an elderly man (75 years) with fever and symptomatic postural hypotension (lying 100/62→standing 70/55 mmHg) which was due to adrenal involvement. So we chose step-down therapy i.e., initially Injection amphotericin-B followed by oral itraconazole because itraconazole does not eradicate fungemia as rapidly as amphotericin-B (we followed the clinical practice guidelines for the management of patients with histoplasmosis updated in 2007 by the infectious diseases society of America).

Dr. A B M Sarwar-e-Alam ,
Professor Zahidul Hasan,
Professor Md. Aminul Islam Khan,
Square Hospitals Limited, Dhaka

FROM THE DESK OF EDITOR in CHIEF

(J Bangladesh Coll Phys Surg 2012; 30: 60)

Let me start by thanking all of you who have contributed to the journal over the years. It is you who have helped us to bring out the issues without hindrance. Still a lot is left to ask for. In-spite of all your effort we are getting very few quality original articles. This is due to the lack of interest in performing original research among the young physicians. It is the younger generation who are going to lead the future of scientific commons of this country and they should come up with new and innovative ideas for the advancement of medical science in Bangladesh. I therefore urge the new generation of specialists to dedicate more time in research work and this will ensure publication of papers of international stature.

HAM Nazmul Ahasan
Editor-In-Chief

Obituary

(J Bangladesh Coll Phys Surg 2012; 30: 61)

The following Fellows who died between October to December 2011

Professor Probhakar Purkayestha

Professor Probhakar Purkayestha died on 22nd October, 2011. He passed fellowship in Medicine in July, 1975 from Bangladesh College of Physicians and Surgeons (BCPS).

Professor Md. Aftabuzzaman

Professor Md. Aftabuzzaman died on 19th November , 2011. He passed fellowship in Medicine in July, 1972 from Bangladesh College of Physicians and Surgeons (BOPS).

Professor Syed Kamaludddin Ahmed

Professor Syed Kamaludddin Ahmed died on 21st December, 2011. He passed fellowship in Psychiatry in July, 1982 from Bangladesh College of Physicians and Surgeons (BCPS). He was Editor-in-Chief, Journal of Bangladesh College of Physician & Surgeons from 1997 to 2001.