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CONTENTS

ACUTE RESPIRATORY FAILURE (ARF)—A REVIEW

Momtaz Ahmed

Key words :
Acute respiratory failure, ARDS, PEEP.

Introduction :

Acute respiratory failure (ARF) constitutes the 5th leading cause of death in the United States—next to deaths caused by highway fatalities. Over 70% of deaths due to pneumonia are attributed to ARF. It is conceivable that the problem of ARF in Bangladesh is in no way small.

Definition :

Grossly speaking respiratory failure exists where the respiratory apparatus fails to maintain normal ABG at sea level under normal BTPS. ARF, however, the subject matter of our discussion, may be defined as a life threatening condition with profound derangement of ABG under a varieagated precipitating conditions and manifested by a wide spectrum of clinical severity *either in a normal healthy lung or in a previously diseased lung* (*Summer et al, 1984*).

ARF may be divided into two broad main categories :

A) That manifested predominantly or in its entirety by hypoxemia together with normal or reduced CO_2 tension i.e. Hypoxic R.F. or Type I.

B) That manifested predominantly by Hypercapnia and associated with Hypoxemia i. e. Hypercapnic-Hypoxic R.F. or Type II.

Momtaz Ahmed. M D. Modern Clinics of Surgery and Midwifery Rd, 131. Gulshan, Dhaka, Bangladesh

Acute Hypoxic R.F. is again defined as any condition reflecting a severe arterial Hypoxemia i.e. paO_2 of 50 mm Hg or less *and which can not be corrected by increasing the inspired O_2 concentration to 50% or more i. e. FIO_2 of 0.5 or greater.* This is often associated with normal or low $PaCO_2$. In this connection both PaO_2 of 50 mm Hg and FIO_2 of 0.5 are arbitrary figures representing a critical physiologic milestone, since at PaO_2 of 50 mm Hg the Hb is approximately 80% saturated. Further reduction of arterial O_2 tension would result in profound hypoxemia and give rise to severe constitutional symptoms *in an otherwise normal healthy lung*. Similary, an FIO_2 of 0.5 is used here since it can be readily achieved at patients' airway by closed system i.e. without recoursing to intubation or non-rebreathing mask. In addition, an FIO_2 of 0.5 usually corrects a vast majority of hypoxic conditions in which gas exchange is not a dominant clinical problem. If hypoxemia could be corrected by this means the management of ARF would have been immensely simplified.

Hypercapnic-Hypoxic R.F i.e. Type II may be defined as an acute emergent condition where *CO_2 elimination by the lung* is inadequate. As CO_2 accumulates in lung fractional concentration of 0_2 also decreases in the alveolus which, therefore, reflects in hypoxemia.

Interestingly, ARF has often been defined in terms of absolute blood gas values which is often erroneous as severity of clinical condition depends largely on :

1. Various precipitating conditions under which it develops.

2. On the state of pulmonary health of the individual at that time. For example, in a previously healthy lung a $PaCO_2$ of 45 mm of Hg constitutes ARF e. g. acute bronchial asthma or acute onset of myasthenia gravis whereas a $PaCO_2$ of 55 mm Hg or greater usually spells ARF in a patient of chronic lung condition having normal $PaCO_2$ previously.

In this context a few words about ARDS is extremely appropriate. This relatively new entity has great significance in the management and epidemiology under which it develops (Clowes, 1974). Various hypothetical theories as to its etiology have been put forward and many excellent reviews have been published to date discussions o which are beyond the scope of this article but while discussing ARF a brief outline of ARDS is mandatory.

What factors actually contribute to the precipitation of this interesting condition are still speculative and hypothetical. However, it has been postulated that *lung reacts peculiarly to various obnoxious stimuli, the common denominator of response appears to be injury at the alveolo-capillary membrane level.*

Clinical Presentation :

It can present itself, under a variety of clinical conditions and express itself in a wide spectrum of clinical severity varying from *mild pulmonary insufficiency to progressive and eventually pulmonary fatal failure.* For clinical purposes it has been conveniently divided into four arbitrary clinical stages :

Stage I : *Respiratory Alkalosis*

1. Hyperventilation and beginning hypocapnia (*normocapnia in early stages*)

2. Diminished pulmonary compliance.

3. Mixed metabolic acidosis and respiratory alkalosis.

4. Normal chest X-ray.

5. Very little or no *auscultatory* findings.

Stage II : *Circulatory stability and beginning respiratory failure*: This stage may be as long as several hours to several days.

1. Persistent hyperventilation and progressive hypocapnia.

2. Increased cardiac output due to correction of hypovolemia and other resuscitative measures used in non-thoracic injury.

3. Progressive decrease in effective pulmonary compliance.

4. Progresssive decrease in FRC i. e. resting lung volume.

5. Falling oxygenation.

6. Progressive worsening of V/Q abnormality. In other words, increased intrapulmonary shunt. The last four are the hallmark of this clinical syndrome.

Stage III : *Progressive pulmonary failure.*

Stage IV : *Terminal hypoxia and asystols.*

With the progress of these clinical stages there are striking changes in the chest X-ray varying from early interstitialoedema followed by diffuse, sometimes, patchy homogenous and perhaps symmetrical infiltrates which may ultimately pro-

gress to widespread areas of consolidation. Finally, in the terminal stage the lung may appear "wet" the so-called wet-lung syndrome.

There are various causes of hypoxemia and ARDS is one of them.

1. Hypoventilation.

2. Diffusion defects i.e. alveolar-capillary block.

3. V/Q abnormalities i. e. ventilation/perfusion inequalities.

4. Intrapulmonary shunting.

o Majority cases of hypoxemia are due to hypoventilation and there are numerous causes of hypoventilation which is not necessary to discuss here as one can take reference of any text book of medicine.

o Diffusion defects can theoretically result from interstitial edema and alveolar thickening and they should, however, respond to 100% oxygen administration by face mask. This is not the case in "Shock lung" or ARDS. So diffusion defect alone appears to be an unlikely cause of this syndrome.

o V/Q abnormalities is a very important and serious problem in the realm of hypoxic conditions, particularly in its management which will be dealt with later on. When a group of alveoli becomes non-ventilated or underventilated there is a reflex compensatory mechanism that brings about a commensurate decrease in pulmonary circulation and *this appears to be lost in certain pathologic conditions.* Now, when this imbalance is reflected in normal ventilation and low or decreased perfusion in a group of alveoli it is known as high V/Q predominance or dead space ventilation. Whereas when the opposite phenomenon occurs in a group of alveoli where there is low or absent ventilation but normal perfusion, the condition is then called low V/Q predominance or the existence of so-called shunt. This latter condition is a very serious clinical state (see fig. 1).

The exact mechanism by which lung reacts to critical injury and a host of obnoxious agents, is not clearly known. However, the basic underlying pathology is one of capillary injury and pulmonary leakage resulting in massive alveolar collapse (Rinoldo & Roger, 1982). The latter is responsible for intrapulm-shunts which is lethal unless prompt and vigorous mechanical devices are employed to improve oxygenation. Several factors have been incriminated either singly or in combination as to the pathogenesis of this syndrome (Rosen, 1975):

1. Microscopic emboli as seen in DIC following sepsis and other serious injuries. These emboli release a variety of vasoactive and bronchoconstrictive substances.

2. Direct injury to the endothelial cell which is primarily *due to sepsis lining the single most important cause.*

3. Diminished production and activity of pulmonary surfectant.

4. Activation of alternate pathway of complement cascade thereby releasing lysozymes and superoxide radicals that injure endothelial cell lining of the capillaries.

5. O_2 toxicity.

Diagnostic Criteria (Udwadia, 1979)

1. High index of suspicion and anticipation of a post-injury, post-anesthesia and other common precipitating conditions are important prerequisites as for example,

sepsis, massive injuries and massive blood transfusions particularly of stored blood. In fact, ARDS may be the first manifestation of sepsis in severe burns, cerebral injuries and pulmonary contusions.

2. *Falling oxygenation and progressive hypocapnia not responding to 50-100% O_2.*

3. (A-a) DO_2 of 200 mm or increasing—the hallmark of intrapulmonary shunt.

4. Other parameters of defective oxygenation and impaired ventilation :

i) PaO_2/FIO_2 ratio : 300 or less

ii) $PaCO_2$ of 30 mm or decreasing.

iii) Minute Volume of 12 L/min and increasing.

5. Lung-thorax mechanics (Comroe et al, 1962)

i) Respiratory rate of 25/min and increasing.

ii) Effective pulmonary compliance which is decreasing i. e. stiff lung syndrome (Ceff : V_T divided by peak airway pressure).

iii) Decreasing FRC.

These are typical accompaniment of ARDS.

Management (Udwadia, 1979) :

Reversing the pathogenetic mechanism that is operative at the time.

1. *Manipulating pulmonary blood flow*: Increasing the perfusion of well ventilated units and decreasing the perfusion of poorly ventilated units.

2. *Directly reducing the capillary leak*: by reversing the membrane injury i. e. directly attacking at the precipitating conditions.

3. Indirectly by reducing the interstitial edema i.e. hypothetical use of diuretics, steroids etc.

4. Finally, improving ventilation of poorly ventilated segments and preventing further alveolar filling and collapse. This parameter of treatment is probably the most rational approach in salvaging such patients. This involves using ventilatory support designed to support and increase alveolar volume—the sheet anchor of treatment of such critically ill patients (Moyer, 1974).

Definitive Treatment :

1. To identify high risk patient—already mentioned in diagnostic criteria.

2. Prompt and keen preparedness to meet all contingencies of emergency measures which require a high skilled team of personnel in a well equipped respiratory intensive care unit.

3. A reliable blood gas laboratory and respiratory therapists the services of which should be available 24 hours of the day.

4. Close monitoring of pulmonary functioning at bedside as well as monitoring all the parameters of adequate oxygenation, ventilation and lung-thorax mechanics.

It is better to leave the E-T tube for 4-6 hours in post-operative ward if one anticipates such complication while closely monitoring the patients progress. If several days of intubation is contemplated a N-T tube may be substituted in the operating room.

A pre-requisite for optimal lung functions are :

1. Normal cardio-vascular status.

2. Adequate concentration of Oxy-hemoglobin and optimum condition for its carriage by hemoglobin (Duhm et al,

1971 & Shapell & Lenfant, 1971) an elaborate discussion of which is beyond the scope of this article. Another attempt to review this important aspect in future might prove helpful.

Hemodynamic Monitoring (Boysen, 1981) :

Includes routine vital signs, bedside electrocardiograph and monitoring of central venous pressure or better pulmonary artery wedge pressure, if facilities of Swan-Ganz catheter is available. Serial body weight, intake and output balance, bacteriologic studies of daily sputum, coagulation profile and chest X-rays, etc. should be carefully maintained.

As a general principle, isolated determinations of these data as opposed to serial measurements & a careful maintenance of flow sheets is an essential part of treatment in the total management of such cases. Partial pressure of arterial 0_2 is the hallmark of adequacy of oxygenation which will, therefore, dictate the appropriate measures to be taken to achieve that end.

The adequacy of ventilation is similarly reflected in the partial pressure of arterial CO_2. An increase indicates hypoventilation and/or dead space ventilation whereas a decrease means hyperventilation that is typically observed in the problem in question in this article. An improvement likewise of $PaCO_2$ approaching normalcy is an important indicator of adequate response to the therapeutic modality used.

Tidal volume (TV) can readily be measured with a modestly priced respirometer. TV multiplied by respiration rate gives the minute ventilation. The effective compliance is a valuable parameter in the assessment of such patients. This is derived by dividing TV with peak air way pressure. Decreased Ceff indicates the stiffness of the lung, which means increased extravascular lung water, increased airway resistance and increased chest wall resistance. Although highly desirable, measurement of work of breathing is difficult to achieve in a critically ill patient as it rsquires intraesophageal balloon for its measurements.

Means of Support of Ventilation :

This is the mainstay and perhaps the treatment of ARDS. As already mentioned this condition is refractory to the usual mundane method of delivering O_2 by closed technique which fails to achieve adequate oxygenations. Assisted ventilation with a volume respirator like MA-1 or MA-2 or other types of ventilators are life saving. Pressure cycled ventilator may be used, if not available, but they are so unpredictable since volume delivered is dependent on pulmonary resistance and peak flow is not accurately adjustabte. (Moyer, 1974 & Kirby et al, 1977)

Indication :

There are no universal guidelines for the institution of ventilatory therapy. The most common indicator is high index of suspicion and that *the hypoxemia is refractory to ordinary O_2 therapy*. As already mentioned, while defining ARF, there is no absolute numerical values that can determine or dictate the ultimate need of instituting ventilator. Perhaps, PaO_2 of 50 mm Hg or less in an otherwise normal healthy lung may constitute enough grave prognostic sign to introduce such modality.

Purpose :

1. Ventilating marginally ventilated alveoli and recrulting collapsed or partially

collapsed alveolar segments thereby increasing the FRC which was falling.

2. Thus correcting the V/Q abnormality in other words, the intrapulmonary shunts and thereby improving and maintaining adequate oxygenation which is so vital to the survival of an organism.

Operation (Kirby et al, 1977).

The technique of operation is so variable that any set rules can not be advocated, rather it should be tailored according to individual requirements. Generally speaking, the settings could be as follows :

1. Volume to be delivered initially may be set at 10-15 ml/kg
2. Rate : 12-14 breaths/min
3. FIO_2 : 0.4
4. I & E ratio : 1 : 2
5. Sigh: 12-17 ml/kg. This is sometimes used in different centers to prevent atelectasis. No uniform results to its effectiveness have been conclusively shown.
6. Humidification is essential for the insipred air with a heated nebuliser.

Effectiveness of treatment is gauged by serial measurements of ABG and Ceff. If Ceff is increasing with increased V_T benefit from increasing V_T may be expected whereas decreased Ceff usually denotes too much volume is being delivered, *since the compliance curve follows the starling curve of cardiac function.* In such case lower tidal volume should be employed to avoid complications and would need the applications of PEEP eventually. In some centers IMV has been used claiming beneficial results in shortening the weaning period. (Feely & Hedley-White, 1975) Howevre, our personal experiences do not corroborate to such reports.

AIM :

A. To achieve acceptable level of PaO_2 i.e. 65-80 mm of Hg :

1. Manipulating the FIO_2.

2. *Support of lung volume* by the addition of PEEP or CPAP.

B. To control of $PaCO_2$: around 35 mm of Hg :

1. Increasing FIO_2 :

Since pulmonary shunts are due to continued perfusion of nonventilated areas simply increasing FIO_2 will not improve oxygenation rather it will wash out the N_2 of already ventilated alveolar segments and, therefore, contribute to more atelectasis. Besides, high FIO_2 for prolonged period will lead to O_2 toxicity that will promote and/or aggravate an already existing ARDS.

2. Addition of PEEP or CPAP is the best alternative choice in recruiting the collapsed or nearly collapsed alveoli.

Technique of PEEP (Powers, 1974) :

This is achieved by applying continuous positive end-expiratory pressure to the airway. A simple device is by inserting an "air-flow resistance" at the expiratory valve.

Result :

1. Increased FRC and increase of effective compliance.
2. Increased PaO_2
3. Conversion at low V/Q ratio areas to high V/Q ratio
4. Decreased intrapulmonary shunts.

Initially it is to be started with 5 cm of H_2O with gradual increment of 3-5

cm of HO_2 not to exceed 20 cm of HO_2. ABG should be monitored frequently. One should wait for ½-1 hour or longer to see its efficacy. Serial bedside measurement of Ceff would be ideal, for proper guide to therpy.

Complications of PEEP :

1. Cardio-vascular :

(i) Decreased cardiac output.

(ii) Decreased venous return and poor right-ventricular filling.

This is particularly significant when intravascular volume is low.

Therefore, adequate hydration must be ensured prior to initiation of therapy.

Similarly, monitoring PAWP and Cardiac output is essential to avoid serious complications.

2. Pulmonory :

Excessive pressure applied to terminal bronchioles may overdistend and rupture normal aloveoli leading to total or partial pneumothorax. PEEP is mostly used when there is decreased FRC. In patients with increased FRC e. g. COPD, any level of PEEP is undesirable and most dangerous.

B. Control of $PaCO_2$:

Hyperventilation is very common in this setting. Hypocapnia is extremely deleterious to both cerebral and pulmonary circulation causing vasoconstriction. With high tidal volume ventilation hypocarbia is an expected phenomenon. Attempts have been made to counteract this by increasing the inspired concentration of CO_2 or adding couple of feet of dead space in the ventilator with equivocal and inconsistent results. Most effective controls require heavy sedation or the use of muscle relaxants e. g. curare to control respiration and thereby decreasing the work of breathing. Decrease of $PaCO_2$ to 30 mm Hg is frequently associated with fatal arrythmia, especially in patients on digitalis and steroids.

Anciliary Therapy :

Success of mechanical ventilation depends largely on how carefully the attendant deficiencies have been taken care of:

i) Oxygen carrying capacity of blood : Depends on adequate concentration of Hb and the storage age of blood used. These must be ensured for successful treatment of hypoxemia (Cherniack & Cherniack, G, 1961 & Duhm et al, 1971).

ii) Adequate hydration : is a must before ventilator therapy is started since hypovolemia and decreased cardiac coutput is highly inimical, especially when PEEP is instituted. Again, overhydration can aggravate the water logging condition of ARDS.

iii) Diuretics, steroids and Heparin : may be used when specifically indicated as in the case of fluid overload, septic shock, aspiration of gastric fluids and DIC respectively.

iv) Antibiotics : indiscriminate use of antibiotics is extremely dangerous as it promotes emergence of resistant strains of organism difficult to treat. Specific antibiotics are used to treat documented infection and their choice is dictated by serial cultures of sputum, blood and other materials as the case may be.

v) Finally acidosis and alkalosis (Shapell & Lenfant, 1971) Either metabolic or respiratory has profound therapeutic implications in the successful management of ARF. It is beyond the scope of this article to explain in detail the role it plays. As it is already known

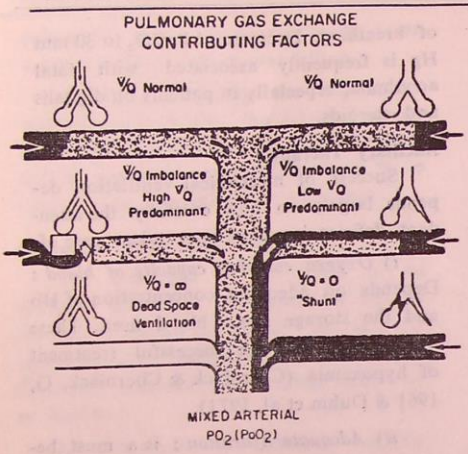

Fig 1: *Diagramatic Representation of Ventilation Perfusion Ratio (V/Q) Abnormalities.*

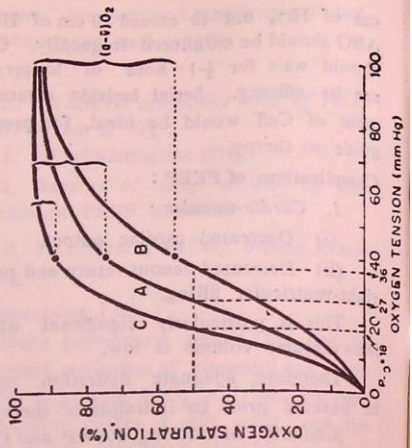

Fig 2: *Oxygen hemoglobin Dissociation curves. If Arterial and Venous Ozygen Tensions Remain Constant, Arteriovenous Oxygen Difference Decreases as the Curve Moves Towards the Left.*

that acidosis shifts the p-50 i. e. Oxy-Hb-dissociation curve to the right thereby facilitating the unloading of the O_2 to the tissues while alkalosis shifts the curve to the left which increases the affinity of O_2 to Hb and therefore, difficult to release O_2 in the tissues at the time when tissue is extremely hypoxic due to dangerously low level of PaO_2 approaching "critical PO_2". Therefore, alkaline pH is highly undesirable especially in hypoxic patients with low fixed cardiac utput (Fig. 2.)

Addendum :

This article has been read in part at the Ninth Annual Conference of Bangladesh Heart and Lung Association.

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MANAGEMENT OF PULMONARY HYPERTENSION AFTER SURGICAL CORRECTION OF CONGENITAL HEART DISEASES

G N Himmelfarb[1], T Z Karimova[2], N M Khawaja[3], N M Gerasimov[4]

Key Words :

Pulmonary Hypertension, infusion of Phosphobion.

Summary :

40 patients who had pulmonary arterial pressure of 41.3±2.8 mm Hg were given Phosphobion infusion. Phosphobion infusion (0.05 mg/kg/min) into pulmonary artery caused a fall in pulmonary arterial pressure to 20.4±2.2 mm Hg. Phosphobion therapy is effective in patients of pulmonary hypertension with congenital heart diseases.

Introduction :

Ligation of Patent ductus arteriosus (PDA), closure of septal defects of heart (HSD) in patients with pulmonary hypertension often does not lead to normalisation of pulmonary circulation during post-operative period.

Functional factors like development of vasospastic reactions (Doty & Lamberth WC, 1985; Pass H, Crawford & Sade, 1984) degree of which is determined by alterations in pulmonary vasomotility ; concentration of humoral regulators (histamine, serotonin, lactic acid) (Corrin, 1982; Bakhle & Chellian, 1983; Heinemann & Fishman, 1969) plays important role in pathogenesis of pulmonary hypertension.

Pathogenesis of pulmonary hypertension in patients with congenital heart diseases has been studied inadequately, and its therapy is not worked out. Taking into account certain shortcomings described in literature about such vasodilators as aminophylline, sodium nitroprusside, nitroglycerine, calcium antagonists (Estanove, 1980; Oliveira, 1981), we paid our attention to reports on ATP vasodilatation effects (Newberg, Millo & Michenfeder, 1985; Sodeyama, Murata & Mizatori, 1983).

Aim of the study :

To study some aspects of pathogenesis of pulmonary hypertension in patients during surgical correction of PDA and HSD

1. G N Himmelfarb, Dr sc med, professor, Head of the Department of Anaesthesiology Tashkent branch of All-Union Research Centre of Surgery of Academy of Medical Science.
2. T Z Karimova, Ph.D, Scientific worker, anaesthesiology, Tashkent branch of All-Union Research Centre of Surgery of Academy of Medical Science.
3. N M Khawaja, Ph.D, Scientific worker, Cardiovascular surgery, Tashkent branch of All-Union Research Centre of Surgery of USSR Academy of Medical Science.
4. N M Gerasimov, Ph.D, Scientific worker, Tashkent branch of All-Union Research Centre of Surgery of USSR Academy of Medical Science.

and during awakening after operation; to evaluate therapeutic efficacy of ATP infusion into the pulmonary artery in relieving pulmonary hypertension during this period.

Material and Methods :

40 patients during ligation of PDA and 34 patients during surgical correction of HSD were investigated in the age group of eight to 36 years. 30 patients were subjected to double ligation and oversewing of patent ductus arteriosus, 10 patients were subjected to ductal division with its arterial and truncal end suturing, 15 patients have undergone plasty of HSD under extracorporeal circulation (EC), and 19 patients suturing of HSD under EC.

All patients have undergone catheterization of redial artery and internal jugular vein by Seldinger technique. Right heart and wedged pulmonary arterial pressure were measured with Swan-Genz catheter (N6-7) of "Elecam" model. Values of arterial pressure, cardiac contraction rates (CCR), pulmonary arterial pressure (PAP) were fixed on polygraph "Salute". Cardiocomputer "Cardiomax" of farm Columbus Instrument (USA) was used for determination of cardiac output (CO) indices. Total peripheral resistance (TPR), total pulmonary vascular resistance (TPVR), pulmonary arteriolar resistance (PAR) and cardiac index (CI) values were calculated with generally accepted formulas. Acid-base and oxygen balance in arterial and venous blood (micromethod of Astrup) were studied.

Concentrations of some biologically active substances in mixed venous blood such as serotonin, histamine, adrenaline and noradrenaline (NA) were studied with spectrofluorometric method on fluorescent spectrophotometer and also cyclic nucleotides (cAMP and cGMP) and prostaglandines of two groups (PGF 2α and PG E+A) were studied with radioimmunologic method to elucidate pathogenesis of residual pulmonary hypertension in these groups of patients in the nearest post-operative period.

The investigations were performed during following steps : I—initial state aftee premedication on the operative table ; II—at the end of operation during awakening of patients and restoration of spontaneous respiration with 100% oxygen through intubation tube ; III—within 15 minutes ; IV—within 30 minutes ; V—within 1 hour from the begining of 1% phosphobion solution infusion.

Phosphobion infusions (1% adenosintriphosphoric acid bisodium salt solution) into pulmonary artery in drops in the dose of 0.05 mg/kg/min (total dose of 50 mg) were used for treating residual pulmonary hypertension in the nearest post-operative period. Obtained results were processed with variation statistic method using Student's significance criteria and with determination of correlative dependence by Spirman technique.

Results :

Our studies (table I & II) indicated that in the second step of the study following ligation of PDA, mean PAP reduced by 18% ($p < 0.05$) from the initial level, however, it remained above normal by 99,4% with PaO_2—152.5±9.1 mm Hg, PvO_2– 70.5±3.6 mm Hg, $HbaO_2$—98.8±0 08%, $HbvO_2$—85.0±0.8%. Following surgical correction of HSD mean PAP remained at the initial level at this step, and TPR in-

Table—I

Values of systemic and pulmonary circulation hemodynamics, gas exchange in infusion during postoperative period of 1% phosphobion solution

Values	stages :	I	II	III	IV	V
mean AP	PDA	95.2±2.8	126.2±3.0	125.2±3.0	125·0±3.2	121.7±3.7
mm Hg	HSD	112.7±2.8	112.6±3.0	110.3±3.0	110.5±3.5	100.5±2.6**
mean PAP	PDA	41.3±2.8	33.9±2.3*	31.5±2.2	26.13±2.1**	20.4±2.2**
mm Hg	HSD	32.2±1.6	32.8±2.6	31.5±2.0	28.6±2.1	24.4±3.1**
HR	PDA	103.0±1.6	112.8±3.2	110.5±3.3	110.3±3.5	96.8±4.7**
beat/min	HSD	34.1±2.1	101.7±2.6	100.0±2.0	100.0±2.0	95.4±3.5
MTR	PDA	6.3±0.3	6.6±0.4	6.6±0.4	7.0±0.4	7.9±0.5
L/min	HSD	4.9±0.2	5.0±0.3	5.0±0.3	5.2±0.3	6.0±0.3**
TPR	PDA	217.0±59.4	310.8±23.5	286.3±20.5	237.5±30.1	156.9±42.3**
din. s/cm^5	HSD	293.4±45.6	410.1±46.1*	393.7±52.1	357.5±35.6	244.0±42.5**
PAR	PDA	145.3±28.5	210.2±18.4	190.0±15.6	167.2±14.3	86.1±11.5**
din. s/cm^5	HSD		281.4±40.4	260.0±40.6	237.5±45.0	166.0±50.0
HI	PDA	3.9±0.2	5.1±0.3			
L/min/m^2	HSD	4.0±0.2	4.1±0.2	4.1±0.2	4.3±0.2	5.0±0.2**
TPR	PDA	1180.3±56.8	1478.6±96.7	1450.5±85.5	1448.3±96.9	1217.2±196.3
din. s/cm^5	HSD	1243.9±47.0	1780.4±45.4*	1744.1±30.9	1747.2±36.6	1589.1±36.0**
PaO_2	PDA	154.2±7.0	152.5±9.1			144.9±15.1
mm Hg	HSD	154.9±15.3	163.3±7.3			172.8±15.2

Table—II

Values of some biologically active substances in mixed venous blood in 1% phosphobion solution infusion in postoperative period

Stages	I		II		V	
Substrate	PDA	HSD	PDA	HSD	PDA	HSD
NA mmol/l	5.05±0.6		6.1±0.8	7.3±0.7	4.1±0.5**	2.9±0.7**
PG (F+A) mkg/l	3.1±0.03	3.82±0.2	3.84±0.6	4.6±0.5	4.7±0.9	4.08±0.7
$PGF_{2\alpha}$ mkg/l	0.56±0.09	0.52—0.06	0.83±0.1*	0.62—0.1	0.66—0.06**	0.78±0.05
cAMP hmol/l	31.1±3.5	44.2—4.9	42.5±4.3*	45.3—5.5	45.6—3.0	34.2±8.9
cGMP hmol/l	8.5±0.7	12.9—1.1	7.8±0.7	14.8±1.1	7.9±0.9	10.7±1.8**

* $p < 0.5$ in comparison with the I stage

** „ „ „ „ „ II stage.

creased by 39.7% ($p<0.08$), with following gas exchange indices : PaO_2—163.3±7.3 mm Hg; PvO_2—61.7±2.2 mm Hg; $HbaO_2$—98.4±0.4%; $HbvO_2$—88.8±0.6%.

Alterations in concentration of biologically active substances in mixed venous blood were the following.

In patients after ligation of PDA at the second step of the investigation, concentration of vasopressor $PGF_2\alpha$ in mixed venous blood increased significantly by 41.6% ($p<0.5$) in comparison with the initial level cGMP by 26.9% ($p<0.05$). Positive correlation coefficient between $PGF_2\alpha$ and mean PAP values made up 0.47, and between cGMP and mean PAP—0.54 that permitted to conclude on the essential role of these mediators in the functional component of pulmonary hypertension in these patients during post-operative period.

In patients after surgical correction of HSD (step II), the concentration of vasopressor mediators in pulmonary vessels, NA, $PGF_2\alpha$ and cGMP remained at the initial high level. Thus, after open heart surgery for HSD under EC, initial high level of pulmonary hypertension was probably maintained with high concentrations of NA, $PGF_2\alpha$ and cGMP elevated in response to such intraoperative influence of EC.

In both groups of patients pulmonary vascular vasoconstriction in the immediate post-operative period was not of hypoxic etiology, but was due to elevated concentrations of mediators like $PGF_2\alpha$, NA and cGMP. Residual pulmonary hypertension during this period was accompanied by disturbances of ca diac rhythm : wite sinus tachycardia in 20 patients, appearence of nodal rhythm in 10 patients and alterations of intraventricular conduction in two patients. Considerably high pulmonary hypertension & appearence of cardiac rhythm disturbance in this period induced us to conduct its therapy.

Results of our investigation indicated that significant changes in hemodynamics of pulmonary circulation were noted within one hour from the begining of phosphobion infusion when its total dose made up 50 mg.

In patients after ligation of PDA, mean PAP reduced by 49.7% ($p<0.05$), TPR by 49.6% ($p<0.05$), PAR by 59.1% ($p<0.05$). and MOC elevated by 16.5% ($p<0.05$) with following gas exchange indices : PaO_2 144.9±15.1 mm Hg ; PvO_2—66.1±4.3 mm Hg ; $HbaO_2$—87.6±0.09% ; $HbvO_2$—92.3± 0.8%.

In patients after surgical correction of PDA, mean PAP reduced by 10.8% ($p<0.05$), TPR by 10.8% ($p<0.05$), mean PAP by 27.7%($p<0.05$) and mean TPR by 40.6% ($p<0.05$). Cardiac activity indices indicated MTR elevation by 16.7% ($p<0.05$) and CO by 18% ($p<0.05$). Disturbance of cardiac rhythm disappeared, and only in 2 patients intraventricular conduction disturbance remained. Gas exchange indices were as follows : PaO_2—172.8±15.2 mm Hg, PvO_2—71.6±6.6 mm Hg, $HbaO_2$—98.6±0.4% and $HbvO_2$—92.3±0.7%.

Discussion :

Phosphobion infusion into pulmonary artery in patients with PDA and HSD after their surgical correction permitted to reduce pulmonary vascular resistance and pulmonary arterial pressure down to optimal level. With such method we did not observe significant reduction in peripheral resistance & mean arterial pressure. Such

differences in pulmonary and peripheral vessel reactions can be explained with that, in phosphobion infusion into pulmonary artery in mentioned doses, relaxation of pulmonary vessels occurs first of all. Considerable amount of phosphobion (ATP) is included into the metabolism of numerous number of endothelial cells of pulmonary vessels (Fukunaga, Flako & Bloor, 1982) and thus gets utilised, and small amount of it reaches systemic circulation. Here we see the advantage of ATP infusion in treating pulmonary hypertension in comparison with other vasodilators that are not metabolised in the lungs.

According to the data in literature, the mechanism of vasodilating effect of ATP lies in inhibition of NA synthesis, elevation of intracellular cAMP concentration and blockade of calcium channels (Bakhle & Vane, 1974, Newberg, Millo & Michenfeder, 1985). According to our findings, within one hour of begining of phosphobion infusion into pulmonary artery after ligation of PDA, reduction of concentration of NA was observed by 32.8% ($p < 0.05$) and $PGF_{2\alpha}$ by 20.5% ($p < 0.05$). And in patients after surgical correction of HSD, we have observed significant reduction of only NA concentration by 60.3% ($p < 0.05$) in venous blood.

Thus, our investigations confirm the data indicated in literature (Sodeyama, Murata & Mizatori, 1984) that ATP inhibits NA synthesis in organs and tissues. It has additionally revealed that ATP is also an inhibitor of $PGF_{2\alpha}$ synthesis in organs and tissues in patients after PDA ligation.

Thus, utilisation ATP infusion after ligation of PDA and surgical correction of HSD in patients with pulmonary hypertension permits to improve systemic and pulmonary circulation indices, and to increase cardiac index preserving stable indices of gas exchange, and to decrease the incidence of arrhythmias.

Conclusion :

In pathogenesis of pulmonary hypertension in patients after ligation of patent ductus arteriosus, elevated concentration of vasopressor $PGF_{2\alpha}$ and in patients after surgical correction of septal defects of heart elevated concentrations of catecholamines, $PGF_{2\alpha}$ and cGMP play an important role. Differences are due to character of intraoperative procedures, in particular, to extracorporeal circulation.

Phosphobion infusions (0.05 mg/kg/min) into pulmonary artery are the method of treatment of pulmonary hypertension in immediate post-operative period in patients after ligation of PDA and surgical correction of HSD, as it reduces vasopressor concentrations of NA, prostaglandines of $F_{2\alpha}$ group in pulmonary vessels.

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CAMPYLOBACTER DIARRHOEA IN RIYADH, SAUDI ARABIA

M A Samad Talukder[1], Suraya P Talukder[2], Wafa Y Jauhargy[3],

Key words :

Campylobacter jejuni, diarrhoea, management, rehydration.

Summary :

The aim of the study was to correlate laboratory isolation of Campylobacter jejuni with clinical manifestation. The organisms were isolated and identified by culturing the faeces on blood agar media containing antibiotics and incubating in presence of carbon dioxide and hydrogen at 42°C. Clinical findings were recorded from previously written case notes. Patients' main symptoms were painful diarrhoea but a few cases were asymptomatic. There were 58 patients, 39 males and 19 females aged from less than one year to 69 years. The conventional culture yeilded other organisms. Associated organisms were five Salmonella species, one Enteropathogenic Escherichia coli 078 and microscopic examination showed four Giardia lamblia. Patients recovered either spontaneously or after five days of treatment with erythromycin. Patients with Giardia lamblia and C. jejuni were treated with metronidazole to which the C. jejuni were sensitive.

1. M A Samad Talukder, Formerly Consultant Microbiologist, Head of Microbiology and Chairman, Control of Infection Committee (Present address and for correspondene : 95, D O H S, Banani, Dhaka Cantonment, Dhaka 1206, Bangladesh).
2. Suraya P Talukder, Formerly Primary Care Physician.
3. Wafa Y Jauhargy Primary Care Physician, Riyadh Armed Forces Hospital, P O Box 7897, Riyadh—11159, Kingdom of Saudi Arabia.

Introduction :

The main cause of death in children is diarrhoeal disease, respiratory infection and maluntrition. One of the commonest cause of diarrhoea in the world today is *Campylobacter jejuui* (Pai et al, 1979 ; Skirrow, 1977 ; Blaser et al and the study group, 1983). A new blood agar media containing antibiotics has been devised by Skirrow (1977) for the isolation of *Campylobacter jejuni* (*C. jejuni*) which is increasingly reported in the United Kingdom (Anonymous, 1981) and the number of isolates has gone up from 1349 in 1977 to 9506 in 1980. The rates of isolation of *C. jejuni* from diarrhoeal stool have been reported as follows: Canada 4.3% (Pai et al, 1979), Riyadh 4.5% (Robertson et al, 1981), 0.9% (Chowdhury and Mahgoub, 1981), Australia 5.8% (Steel and McDermott 1978), Belgium 5.1% (Butzler et al, 1973), England 7.1% (Skirrow, 1977), Sweden 10.99% (Svedhem and Kaijser, 1980), U S A 4.6% (Blaser et al and the study group, 1983) and Bangladesh 14% (Glass et al, 1983).

The purpose of this study is to report symptomatology of Campylobacter diarrhoea and to discuss about management.

Materials and Methods :

Faeces were routinely examined for ova, parasites and cultured for other organisms including *Campylobacter jejuni*. The period of study was six months in

1982 and 1983. Faeces were cultured promptly on blood agar media containing vancomycin, trimethoprim and polymixin and incubated in an anaerobic jar without catalyst in presence of BBL gas pack with 10 ml water as a source of hydrogen, carbon dioxide and reduced oxygen for 24 to 48 hours at 42°C. The organisms were identified by their characteristic morphology i. e. Gram negative curved rod, motile, oxidase positive and do not grow in ordinary media at 25°C and 37°C but grew at 42°C. Microscopic examination of faeces showed protozoa such as *Giardia lamblia* and by conventional cultural technique *Salmonella sp.* and Enteropathogenic *Escherichia coli* 078 were isolated. Sensitivity testing were performed in anaerobic jar on DST agar plate with 5% lysed blood using erythromycin 15 μg and metronidazole 4 μgm discs.

Results :

By the method described above 58 patients (39 males and 19 females) were found to have *C. jejuni* and their age ranged from under one year to 69 years, majority being children. The main presenting features were diarrhoea, abdominal pain, vomiting, nausea, anorexia, fever, and blood and/or mucus in stool. Six patients were asymptomatic. In 10 patients second organism was present such as *Salmonella sp.* in five patients, *Giardia lamblia* in four and Enteropathogenic *Escherichia coli* 078 in one patient. All *Campylobacter jejuni* were sensitive to metronidazole and erythromycin except two *C. jejuni* which were resistant to erythromycin.

Discussion :

Campylobacter jejuni is well known to cause watery or bloody diarrhoea with abdominal pain. In almost all cases symptoms were present as previously described by other authors (Pai et al, 1979 ; Chowdhury and Mahgoub, 1981 ; Steel and McDermott, 1978) but in this study six were asymptomatic i.e. had no diarrhoea which is different from other studies as they have not found any *C. jejuni* in control groups except one positive culture for *C. jejuni* is mentioned by Pai et al (1979) from asymptomatic contact from Canada. It is reported by Glass et al (1983) that they have isolated *C. jejuni* from patients with watery diarrhoea and some asymptomatic cases from Bangladesh. Epidemiological studies in the International Centre for Diarrhoeal Disease Research, Bangladesh showed that painful watery diarrhoea occurs in Bangladesh (Glass et al,1983) whereas in USA, Canada and Europe *C. jejuni* causes dysentry like symptoms (Blaser et al and the study group, 1983 ; Karmali and Fleming, 1979 ; Pai et al, 1979 ; Skirrow, 1977).

Males suffer more from *C. jejuni* diarrhoea as found in this study is in agreement with other workers (Pai et al, 1979 ; Chowdhury and Mahgoub, 1981 ; Palasundaram, 1982). In 50% of patents prodrome of headache, malaise, dizziness, myalgia, fever and cramping abdominal pain, followed by mild to severe diarrhoea & bloody stools for two to three days may be present (Gopalakrishna, 1986). This is a leading cause of diarrhoea in the United States of America (Gopalakrishna, 1986 ; Blaser et al and the study group, 1983). Blaser and the study group (1983) recommended stool culture for the diagnosis of *C. jejuni* diarrhoea and that is the practice in their laboratories otherwise the diagnosis of this important pathogen will

be missed. Other authors are also of the same opinion (Pai et al, 1979 ; Gopala krishna, 1986). This is the practice in our laboratory and we recommend to culture stool when indicated, to include culture for *C. jejuni* on appropriate medium containing antibiotics at right temperature and atmosphere.

The recognition of this organism requires prompt despatch of stool specimens to the laboratory and awareness by the clinical staff of the presence of this organism as a cause of diarrhoea, will ensure more rapid detection and proper managment. Patients recovered spontaneously or after five days of treatment with erythromycin. Patients with *Giardia lamblia* and *C. jejuni* responded well to treatment with metronidazole. Often, there is no need to treat Campylobacter diarrhoea as the patient usually recovers spontaneously unless disease is severe when erythromycin 500 mg q.i.d. orally for five days, is the treatment of choice (Gopalakrishna, 1986). in this study all *C. jejuni* were sensitive to erythromycin except two but all were sensitive to metronidazole so in erythromycin resistant cases metronidazole will be useful. One of asymptomatic patients who was to be operated on for abdominal condition was treated with erythromycin prior to surgery.

Campylobacter jejuni may cause bacteraemia or septicaemia in debilitated patients either with or without diarrhoea (Talukder et al, 1986). *C. jejuni* may even cause meningitis in neonates (Thomas et al, 1980). Prevention of these conditions are desirable.

A paper is published by Omer (1983) from Sudan on bacterial diarrhoea without mentioning anything about *C. jejuni* and the reason may be that the study was done from January to December, 1978 and did not have the facilities for isolating campylobacter. Jegathesan (1984) mentioned about *C. jejuni* and quoted five to 14% of diarrhoeal cases and in Malayasia it can be three to four precnt of children with diarrhoea. The author did not categorically mention that they have done the study but only hypothesised. It is recently reported from children under three years from Saudi Arabia that two (0.5%) of 361 patients had *C. jejunni* but of the 100 known positives only two had this organism (Talukder et al, 1987). This is a selected age group. Whatever may be the reason of diarrhoea, fluid therapy is vital in all diarrhoea to replace fluid loss and then comes specific treatment. Severely dehydrated patients should be hospitalised if facilities are available. The oral rehydration in watery diarrhoea remains the mainstay of therapy regardless of the organism (Gopalakrishna, 1986 ; Anonymous, 1983). The oral rehydration solution containing both glucose and glycine (as the actively transported substrate to promote water and electrolyte absorption) was significantly better than a solution containing glucose and electrolytes (Nalin et al, 1979). This reduces stool volume and duration of watery diarrhoea. Less expensive, can easily be prepared at home and readily acceptable by patients is a rice salt solution. It can be prepared by putting 50g or two handful of dry rice, soaked in water, ground as powder and added to 1 litre (1 seer) of water, boiled for five to seven minutes stirred continaously till smooth solution is formed and then add three finger pinch of salt twice and this rice salt oral rehydration solution is ready for oral use (Molla and

Molla, 1986). This is not only for *C. jejuni* diarrhoea but also for any watery diarrhoea causing dehydration. Improperly cooked chicken, unpasteurised or raw cow's milk, infected water are potential source of *C. jejuni* (Chowdhury and Mahgoub, 1981 ; Gopalakrishna, 1986). Person to person transmission from diarrhoea patient due to *C. jejuni* is possible. Domestic animals such as cats or dogs could be source of this organism.

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STUDY OF BONE MARROW IN 30 CASES OF LYMPHOMAS

A H K Chowdhury[1], M J Rahman[2], T A Nasir[3], M Tahir[4], Q Huda[5]

Key Words :
Lymphoma, Bone marrow.

Summary :

30 cases of lymphomas (Non-Hodgkin's lymphomas 21, Hodgkin's lymphomas 9) diagnosed by lymph node biopsy, were included in this study. Bone marrow biopsy was done in all of them which showed marrow involvement in 8 (37.1%) cases of N H L and in 2 (22.5%) cases of H L. The proliferative pattern was focal nodular and paratrabecular. Reactive changes were found in 12 cases in the form of hypocellularity, eosinophilic infiltration and erythroid hyperplsia. 10 patients were upstaged (stage iv) by marrow biopsy.

Introduction :

Bone marrow biopsy has been considered to be an integral part of investigation of patients with lymphoproliferative disorders (Rosenberg, 1979). Involvement of the bone marrow is one of the criteria for systemic dissemination of malignant lymphomas. (Bartl et al, 1982). Bone marrow involvement is also important for clinical staging of lymphomas. Webb et al (1970) reported bone marrow involvement in 22% of Hodgkin's lymphoma (H L) while Bartl et al (1982) and Stein et al (1976) found it in 16-75% cases of non Hodgkin's lymphoma (N H L). The reasons for this wide variations in N H L are : a) use of marrow aspirations which have a high rate of false negatives ; b) the unequal proportions of patients with early and advanced disease in diflerent studies & c) the varying biopsy sizes, sites and technical preparations. Adequate bone marrow biopsy provides information regarding extent, pattern and the state of non-involved marrow (Dorren, 1987).

In view of the above infomations, this paper reports experiences with marrow aspirations as well as bone marrow biopsies regarding their usefulness in cases of malignant lymphomas.

Materials and Methods :

Thirty cases of lymphomas (diagnosed histologically) who were admitted in IPGMR and in different private clinics during October 1984 to November 1987, were included in this study. A thorough clinical assessment and laboratory investigations were done in all cases. Bone marrow aspiration was performed from sternum or iliac crest and trephine biopsy was done from posterior iliac crest.

A positive bene marrow was defined in this study as unequivocal evidence of lymphoma in the marrow as was cosidered in

1. Abul Hussain Khan Chowdhury, Resident Madical Officer
2. Md. Jalilur Rahman, Asstt. Professor of Haematology
3. Tarek Al Nasir, Asstt. Professor of Pathology
4. Md. Tahir, Professor of Medicine
5. Quamarul Huda, Professor of Medicine.
Institute of Postgraduate Medicine & Research.

a previous study (Maitreyan et al 1987). Patients were treated with combination chemotherapy using 'COPP and CVP'.

Results:

Of the 30 cases, 21 were of N H L and nine H L. The age of the patients varied from 13 years to 72 years. Among 21 patients of N H L, 15 were males and six females; where as in cases of H L, six were males and three females. The presenting features of these 30 cases are shown in Table I. Results of laboratory investigations were not significant except moderate anaemia in 25 cases and eosinophilia in two cases of Hodgkin's disease.

Table—I

Signs and symptom in 30 cases of malignant lymphomas.

Signs and symptoms	N H L	H L
Symptoms :		
Fever	10	5
Jaundice	—	2
Aches and pains	5	1
Signs :		
Lymph node enlargement:		
Cervical	14	9
Axillary	10	4
Inguinal	12	3
Hepatomegaly	8	3
Splenomegaly	7	2
Pallor	19	6
Pleural effuslon	2	—

Presentation was classical in majority of patients except the presence of jaundice and pleural effusion.

Bone marrow biopsy showed tumour deposits in two cases of HL and eight cases of NHL. (Table-II). The pattern of involvement was focal nodular in eight cases and paratrabecular in one case each of HL and NHL. In a case of NHL about 90% of marrow was necrosed and a few viable lymphocytes and occasional lymphoma cells were seen.

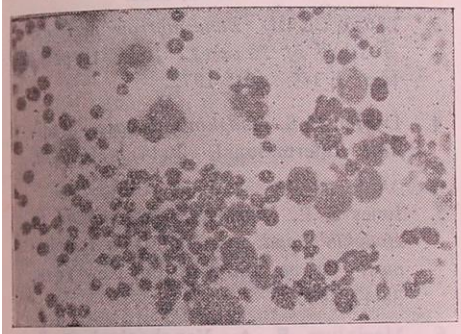
Table-II

Bone marrow histopatholosy in 30 cases of malignant lymphomas

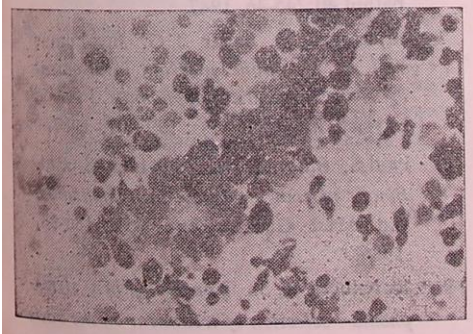
	N.H.L	H.L
Cellularity :		
Normocellular	12	7
Hypocellular	9	0
Myeloid series :		
Normal	12	7
Hypoplasia	9	2
Eosinophilia	0	2
Lymphocytosis	2	0
Erythroid series :		
Normal	3	7
Hypoplasia	8	0
Hyperplasia	0	1
Aplasia	1	0
Megakaryocytes :		
Normal	3	9
Decreased	8	0
Absent	1	0
Tumour deposits	8	2
Fibrous tissue	1	0
Necrosis	1	0

Reactive changes were found in 12 cases. Erythroid hyperplasia was found in one and eosinophilia in two cases of HL. Hypocellular marrow was found in nine cases of NHL. Myelofibrosis was observed in one case of NHL.

Bone marrow infiltration was seen in a case of HL which was earlier categorised as clinical stage II (Ann Arobor staging).



Fig—1 *Marrow Showing Lymphoma cells (H & EX 495).*



Fig—2 *Marrow Showing Lymphoma cells (H & EX 640).*

Positive bone marrow was also found in one case of HL, and three cases of NHL who were in clinical stage III & five cases of NHL who were in clinical stage IV ; so all of them were finally put in stage IV.

Discussion :

In the present study 37.1 percent and 22.5 percent positive marrow biopsies were observed for NHL and HL respectively. These finding are in conformity with observations made by Maitreyan et al (1987) for NHL and Webb et al (1970) for H.L. Pattern of involvement was focal nodular in eight cases (seven NHL and one HL) and paratrabecular in one case each of NHL and HL. It has been observed that patients with nodular marrow involvement had significantly longer survival time than those with other patterns (Bart et al. 1982). Besides the involvement of marrow by disesase itself, adequate marrow biopsy also provides information regarding uninvolved marrow and tumour myelopathy (Dorren, 1987).

In nine cases of NHL, there were reactive changes in the form of hypocellular bone marrow in this study. Similar observation was made in a previous study. (Paul, 1986). Out of these, in one case focal myelofibrosis was also there.

In one case of HL, there was erythroid hyperplasia and in another two cases the bone marrow was hypercellular with diffuse infiltration by eosinophils. Studies by Bart et al (1982) showed that patients with normal bone marrow had favourable prognosis while those having leukemoid reaction, low content of lymphocytes and hypocellular marrow were

associated with shorter life expectancy. In one of the retrospective studies (Macintyre et al 1981) is has been found that bone marrow eosinophilia had no effect on life expectancy rather it is a non-specific reaction to Hodgkin's disease. It has no prognostic importance unless it is very selective (Vaughanhudson, 1986). We also can not comment on prognostic significance of findings due to failure of follow up of our patients.

In this study, 90% necrosis of bone marrow was found in a case of NHL. In one of the studies chemotherapy, sepsis, disseminated intravascular coagulation and radiation have been considered to be responsible (Brown, 1972). Others (Jones, 1989) suggested that in uncomplicated & untreated patients, rapidly progressive disease process may spontaneously cause marrow necrosis. In this study, rapidly progressive disease may be the aetiological factor for marrow necrosis as other causes were not applicable to the particular case.

One case of H L included in stage IV due to positive bone marrow biopsy was considered in stage II by prior clinical assessment. Several workers (Bartl et al, 1983) in past also made similar observations. So, bone marrow biopsy should be considered as useful diagnostic tool for staging the patients of malignant lymphomas and to know about uninvolved marrow.

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ENDOSCOPIC FINDINGS OF 180 DYSPEPTIC SUBJECTS IN NARAYANGANJ HOSPITAL

M Rahman[1], S K Shaha[2]

Key Words :

Peptic ulcer, Fibreoptic endoscopy, Upper gastrointestinal tract.

Summary :

Endoscopic examination carried out on 180 dyspeptic subjects in the Narayanganj Disrtict Hospital over a period of six months. These cases were referred from various departments of Narayanganj Hospital. The clinical diagnosis was peptic ulcer in 97.7% carcinoma of stomach in 1.7% and cirrhosis of liver in 0.6% cases. At endoscopy, however, normal upper gastrointestinal tract was found in 30.5%, chronic duodenal ulcer including uppet gastrointestinal bleeding in 45.2%, chronic duodenitis in 14.4%, pyloric stenosis in 7.7%, carcinoma of stomach in 2.2%, oesophagitis in 0.6% and ascariasis in 1.2% cases. The most frequent cause of the upper abdominal symptoms appeared to be chronic duodenal ulcer. This is a common condition in Bangladesh. The findings of this study are consistent with those of earlier studies from this country. The endoscopic examination was well tolerated and well accepted by the patients in this district hospital.

1. Dr. M Rahman, Consultant, Department of Medicine.
2. Dr. S K Shaha, Ph.D, Registrar, Department of Medicine,
Narayanganj Hospital.

Introduction :

Narayanganj District Hospital was started in 1985 with 200 in patient beds. Upper gastrointestinal endoscopic examination was started in 1986 in this hospital. In this communication we are presenting the endoscopic findings of the first 180 subjects referred to our department with various clinical diagnosis. The two previous but similar studies (Azad Khan, A K et al 1982 and Roy, P K et al, 1986) from Bangladesh were carried out in teaching hospitals but none was done in a district hospital.

Materials and Methods :

In all 180 patients were studied—110 males with their age ranging from 14 to 70 years (average 39.8 years) and 70 females with age ranging from 10 to 65 years (average 36.8 years) (Table I). These sujects were referred from both indoor and outdoor departments of Medicine, Surgery and Paediatrics of Narayanganj Hospital with history of upper abdominal pain and/or dyspepsia. Those who had jaundice or suspected liver diseases were advised to have HBsAg test. Those who were positive were excluded.

Endoscopy was done with a Machida forward viewing panendoscope. Endoscopy was carried out in the morning in empty stomach about 12-14 hrs. after the last feed. The procedure was explained to the patient. The examination was done in the left lateral position. The endoscopist was assisted by a person whose job was to hold the mouth guard in position. The tube was passed to the oesophagus, without using any anaesthetic spray, by asking the patient to swallow and, while in the oesophagus it was manouvered through the oesophagus, stomach and passed upto the second part of the duodenum.

Results :

The clinical diagnosis of the patients are shown in Table II. Chronic duodenal ulcer was clinically diagnosed in 153 cases. Next majority group of cases were diagnosed as chronic duodenal ulcer with haematemesis & melaena (14). The other clinically diagnosed cases were chronic duodenal ulcer with pyloric stenosis-8, carcinoma of stomach-3 and cirrhosis of liver-1.

Table—I

Showing the age & sex wise distribution of the patients investigated

Age (yrs)	Sex	
	Male	Female
11-20	4	6
21-30	27	25
31-40	32	17
41-50	30	17
51-60	13	4
61-70	4	1

Table—II

Showing the clinical diagnosis of the patients investigated

Diagnosis	Number	Percentage (%)
1. Chronic duodenal ulcer	153	85
2. Chr. D. U. with Haematemesis & melaena	14	7.8
3. Chr. D. U. with pyloric stenosis	8	4.4
4. Stomal ulcer	1	0.6
5. Carcinoma of stomach	3	1.7
6. Cirrhosis of liver	1	0.6

At endoscopy the clinical diagnosis was corroborated and the resuls are shown in Table-III. Most common diseases diagnosed endoscopically were chronic duodenal ulcer which was found in 64 patients. Normal upper gastrointestinal tract was found in 55 cases. Other endoscopically diagnosed cases were as follows : chronic duodenitis-26, oesophagitis-1, ascariasis-2, chronic duodenal ulcer with haemorrhage-14 and carcinoma of stomach-5.

It is obvious from the presented tables II & III that the patients suffering from chronic duodenal ulcers outweigh other diseases of the upper gastrointestinal tract. Most common disease in the series was found, both clinically and endoscopically, to be chronic duodenal ulcer. However, gross discrepency was observed between the clinical and endoscopic diag-

Table—III

Showing the endoscopic diagnoses of the patients investigated

Diagnosis	Number		Percentage (%)	
	Male	Female	Male	Female
1. Normal GI tract	19	36	17.3	51.4
2. Chr. Duodenitis	11	15	10.0	21.4
3. Chr. D. U.	56	8	50.9	11.4
4. Pyloric stenosis	12	2	10.9	2.9
5. Chr. D. U. with haemorrhage	9	5	8.2	7.1
6. Carcinoma of stomach	4	1	3.6	1.4
7. Oesophagitis		1		1 4
8. Ascariasis		2		2 9

Table—IV

Showing the causes of haematemesis & melaena

Diagnosis	Number	Percentage (%)
1. Chr. duodenal ulcer	11	78.6
2. Cirrhosis of liver	1	7.1
3. Prepyloric erosion	2	14.3

noses. Clinically 85% of these cases were diagnosed as chronic duodenal ulcers, whereas endoscopically only 35.6% cases were proved to be so. A large number of clinically diagnosed peptic ulcers (30.6%) cases were proved to have no lesson in the upper gastrointestinal tract. These cases were named nonulcer dyspepsia having symptoms like heart burn, pain in the abdomen but no ulcer or any other organic lesion in the stomach or duodenum (Roy, PK et al, 1986).

History of upper gastrointestinal haemorrhage in the form of haematemesis & melaena or both was present in 14 (7.8%) cases. At endoscopy chronic duodenal ulcer was found to be the main offender in 10 cases (78.5%), prepyloric erosion in two cases (14.2%) and oesophageal varices in one case (7.1%).

Interestingly we could detect the presence of Ascaris Lumbricoides in the duodenum of two female patients (2.9%).

Endoscopic examination was well tolerated and there was no complication in any patient.

Discussion :

The disease pattern revealed by the endoscopic study reflects the frequency of different gastrointestinal diseases. Our study indicates that chronic duodenal ulcer is a common disease in Bagnladesh. Clinically it is more frequently diagnosed. These finding are consistent with the studies carried out in academic institutions of Bangladesh earlier (Azad et al, 1982, Hasan et al, 1985 and Tovey, 1979). No case of gastric ulcer was recorded in this series. This is probably due to the fact that gastric ulcer is less frequently met in our country (Hasan et al, 1985).

Most frequent and sometimes dramatic complication of peptic ulcer is haemorrhage which explains unpassable interests of the investigators to the study of disease (Hunt, 1984, Beloysov et al, 1976, Fisher et al, 1985 and Pinsky et al, 1985). Loca-

lizing and revealing the cause of upper gastrointestinal bleeding is difficult. Radiology is not of sufficient help in diagnosing acute ulcers or erosions. Sometimes more than one source is seen in as many as 33% cases (Poul et al, 1972). In this study we could successfully detect the causes of haematemesis & melaena: duodenal ulcer (78.5%), ruptured oesophageal varices (7.1%) & prepyloric erosion (14.2%).

Complications during gastroduodenoscopy are mainly limited to the mechanical trauma of the mucous membrane, perforation of the oesophagus, stomach, duodenum and haemorrhage. According to different authors perforation occured in 0.033% cases, haemorrhage in 0.03% cases, cardiorespiratory complication in 0.071% cases and mortality in 0.0047% cases (Cotton, 1973). None of our patients had mortality or major complications. It may be stressed here that on correct observation of all technical formalities complications only very rarely develop. It is needless to stress that endoscopy is a well tolerated and safe method of investigation (Cotton, 1973). This study is a proof to the fact that endoscopy is a useful diagnostic tool in upper gastrointestinal diseases and can be carried out in a district hospital if trained personnels and equipments are available.

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PSEUDO-MEIG'S SYNDROME IN BANGLADESH

A K M Shahabuddin[1]

Key Words :
Pseudo-Meig's Syndrome, Meig's Syndrome, Ovarian Tumour.

Summary :

Pseudo-Meig's syndrome is a very rare condition. Three cases of Pseudo-Meig's syndrome is reported and their prognosis after treatment discussed.

Introduction :

Meig's syndrome and Pseudo-Meig's syndrome are both rare conditions. But Meig's is even rarer. Surprisingly, more cases of Meig's syndrome have been reported in the world literature than the Pseudo-Meig's syndrome (Ong et al, 1979 & Palaniappan et al, 1980). It appears that, Pseudo-Meig's syndrome are either not identified or not often reported. But proper identification of such cases usually improves prognosis (Hartstein et al, 1980 & Morell et al, 1980).

At Sher-E-Bangla Medical College, Barisal, we came accross three cases of Pseudo-Meig's syndrome within a period of six months, where surgical removal of the ovarian tumour cured two subjects and improved longivity of the remaining one.

Materials and Methods :

Case No—1

A forty years old female presented with amenorrhoea, rapidly increasing lower abdominal mass and ascites. At the time of examination her haemoglobin was 44 percent and temperature 37.8°C. She had moderate degree of ascites and bilateral small pleural effusion. There was an irregular mass (6" x 6") fixed in left lower abdomen.

Before coming to us the patient underwent paracentesis several times, but every time, ascites reappared quickly.

After admission, a course of amoxycillin was given and the temperature subsided within two days. Laparotomy was performed.

At laparotomy, 1500cc of clear ascitic fluid was drained. A tumour of about 5" x 5", arising out of the left ovary was found adherent to all surrounding structures. This was removed. All the other viscerae were found normal. Histology of the tumour showed benign mucinous cystadenoma.

The patient made uneventful recovery and was discharged on the 20th day after the operation, when she was completely free of ascites and pleural effusion. The patient was followed for two and a half years and was found completely nnomal.

Case No—2

A sixteen years old, unmarried girl presented with ascites and general weak-

Dr. A K M Shahabuddin, FCPS, Professor of Obstetrics and Gynaecology.
Rangpur Medical College, Rangpur.

ness for six weeks. At admission, in addition to tense ascites, she had a right lower abdominal mass (8" x 8") slightly irregular, firm and with some restricted mobility. She had moderate right pleural effusion, temperature of 37.2°C and haemoglobin of 40 percent.

At laparotomy, 2.5 litres of clear ascitic fluid was drained. A left ovarian tumour of 8" x 8", slightly adherent to omentum was removed. Patient made uneventful recovery. X-ray chest at two weeks showed no effusion, and there was no ascites.

As the facilities for histopathology at our hospital was not good, the resected specimen was sent to Dhaka for examination, but unfortunately, was lost during transport.

Patient remained well for about two and a half months, but was readmitted after three months and laparotomy showed malignant tumour in the remaining right ovary with extensive infiltration in the peritoneal cavity. The ovarian mass was removed and the patient was treated by chemotherapy and radiotherapy. She improved and remained well for about a year after which she deteriorated again and died after about nine months.

Case No—3

A forty five years old female, presented with swelling of abdomen and tiredness for about five months. At admission, she had moderate ascites and a mass of about 4"X4" could be felt through fornices per vaginal examination. It was firm, round and freely mobile. She had moderate right pleural effusion.

At laparotomy, 1500 cc of haemorrhagic fluid was drained. A tumour of 4"X4" size was found arising from the right ovary; uterus and the other ovary were found to be infiltrated. Total hysterectomy with bilateral salpingo-oophorectomy was done. Histopathology revealed mucinous cystadenocarcinoma.

Postoperative course was uneventful. X-ray chest on the 10th postoperative day showed complete disappearance of the pleural effusions, and there was no reccurrence of ascites by that time. Patient was given chemotherapy and radiotherapy. She was discharged three weeks after surgery but could not be traced for follow up since then.

Discussion :

Pseudo-Meig's syndrome is usually defined as ascites and pleural effusion (usually of the right side), due to benign or malignant tumour of the ovary other then a fibroma (Jeffcoat 1975 & Meig, 1975).

All the above cases presented with ascites, pleural effusion and ovarian tumour. In two cases, history excluded Meig's syndrome (fibroma). In the remaining one case, although histology could not be done, but reccurrence and metastasis, proved malignant nature of the disease and thus excluded Meig's syndrome.

Importance of recognising Pseudo-Meig's syndrome lies in improved prognosis after surgical removal of the tumour. To our knowledge, no case of Pseudo-Meig's syndrome has been reported from Bangladesh. The presence of a firm to hard abdominal mass, ascites and pleural effusion may suggest disseminated malignancy and lead us to abandone laparotomy (Feroze, 1981). But all the three cases presented in this report shows that, surgical removal of the tumour greatly improves prognosis

in cases of Pseudo-Meig's syndrome, even if the primary condition is malignant.

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MULTIPLE RECURRENT SCHWANNOMA OF THE CHEST WALL WITHOUT MUTIPLE NEUROFIBROMATOSIS

Ahmed Mokhtar[1], T A A Abioye[2], Hassan Raffa[3]

Key Words :
Schwannoma, Neurofibromatosis.

Summary :

A case of malignant recurrent schwannoma of the chest wall unassociated with multiple neurofibromatosis is presented. This is the first case to be reported from the King Abdulaziz University Hospital. The clinical, macroscopic and microscopic features of this unique malignant and recurrent chest wall tumour in a young man are discussed.

Introduction :

Solitary malignant Schwannomas are rare but they occur more frequently within the context of neurofibromatosis. They do not occur in common sites of benign schwannomas and it is highly doubtful whether a benign schwannoma ever undergoes malignant change. The high rate of local recurrence after inadequate excision has been previously emphasised. The diagnosis of malignant schwannoma in a chest wall tumour in a young patient without clinical features of Von Rechlinghausen's disease is, therefore, of sufficient importance and interest to warrant documentation.

The purpose of this paper, therefore, is to report the first case of recurrent malignant schwannoma in a young Yemeni patient presenting at the King Abdulaziz University Teaching Hospital, Jeddah.

Case Report :

The patient was a male 29 years old Yemeni who, when first seen in October 1982, gave a history of left sided chest wall mass of 8 months' duration. He gave a past history of similar mass in 1966, a mass which was removed some 12 years later in 1978. It was histologically diagnosed then as neurofibroma.

On admission, physical examination revealed an irregularly shaped mass measuring 25 X 20cm, on the left side of the anterior chest wall away from the nipple.

1. Ahmed Mokhtar, M D, Associate Professor of Surgery, King Addulaziz University Hospital, KSA
2. T A A Abioye, M D, Professor of Pathology, King Abdulaziz University Hospital, KSA.
3. Hassan Raffa, M D, FRCS, D Sc (Hon), Assistant Professor of Cardiothoracic Surgery, King Abdulaziz University Hospital, KSA.

Address for correspondence : Dr. A Mokhtar, Associate Professor of Surgery, King Abdulaziz University Hospital, P. O. Box 6614, Jeddah 21452, Saudi Arabia.

The mass was mobile, well defined, firm in consistency and attached to the overlying skin only in its central part. Also in the centre of the mass there was a foul smelling ulcer with raised and everted edges. Examination of all the other systems showed no abnormality.

The mass was later on excised and sent for histological examination. About two months later, the patient presented again with a mass in the area of previous resection.

Pathology :

Microscopic appearance—The specimen received consisted of a very firm nodular mass weighing one kilogram (1 kg.) and covered by an ellipse of skin. The specimen measured 19 X 10 X 13 cm in dimensions. There were few pigmented nodules on the surface. Cut surface showed a multi-lobulated greyish-white fibro-fatty tissue with marked congestion.

Histological examination : revealed sections consisting of a fibroblastic tumour showing much nuclear pallisading. It was very vascular with large cells arranged in whorls alongside the large blood vessels. Mitoses were somewhat frequent and a partially intact nerve bundle from which the tumour appeared to have originated could be discerned in the background. The overall appearance was that of a well-differentiated malignant schwannoma.

Discussion :

The terminology of peripheral nerve tumours composed essentially of a spindle cell growth pattern with cells arranged in tight, wavy or interlacing bundles with plump ovoid and vesicular nuclei is rather confusing. In this paper, the term malignant schwannoma as used by Gupta & Brasfield (1970), is adopted for a primary tumour of similar appearance to previously reported case of malignancy in peripheral nerves. There are few well documented cases of the neoplasm which may arise independently or in association with Von Rechlinghausen's disease.

For many years, Pathologist and Clinicians have been greatly interested in malignant tumours of the peripheral nerves because of their controversial histogenesis and rarity. These tumours, whether benign or malignant, arise from the schwann cells, the lining cells of the nerve sheath. In 1935, Stout first interpreted this malignant growth as fibrosarcoma, though he revised his opinion in 1949 after observing the outgrowth of Schwann cells in-vitro from such tumour (Murray & Stout, 1940 & 1942 : Stewart & Copeland, 1931,: Vieta & Pack, 1951) and Das Gupta & Brasfield have contributed to an increased understanding of this tumour (Stewart & Copeland, 1931 and Vieta & Pack, 1951). The histogenesis of this tumour is still controversial. Presently it is considered to originate either from Schwann or perineural cells. Stout (1970) regarded the Schwann cells as the proliferating element in a schwannoma. From a review of the literature, opinion concerning the natural history of this tumour vary from "a high propensity for local recurrence and a low incidence of metastases to a highly malignant and a frequently lethal tumour". Because of this controversy, the aggressiveness of primary treatment has varied from simple excision to amputation. Malignant schwannoma tend to recur after removal, becoming more anaplastic with successive recurrence. The present case is in this category, particularly in its biological behaviour. Although

it is stated that malignant schwannomas probably account for the majority of malignant tumours in neurofibromatosis, our patient is free from neurofibromatosis. However, its association with previously diagnosed neurofibroma removed from the same site some two years back coupled with histological identification of a possible origin from a nerve bundle, strengthened our diagnosis. This is further enhanced by the fact that cases that arise unequivocally from peripheral nerves are included among the acceptable examples. In addition to the foregoing, is the fact that the well-differentiated form of the tumour tends to retain the pattern or interlacing bundles of spindle cells with their characteristic reticulin pattern which are prominent histological features in our present case.

From a clinical standpoint the presentation of maligant schwannoma is clearly indistinguishable from that of any mesenchymal tumour of the chest wall and, in any case, since operative treatment is apt to be the same, this point may appear insignificant. However, the question of whether a neurogenic tumour is benign or malignant and its attendent prognosis deserves consideration. No doubt, in this case, one is dealing with a rapidly growing malignant tumour.

Satisfactory treatment of malignant schwannoma of the chest wall is bedevilled as in the case of neurogenic sarcoma in other sites, by the problem of recurrence following surgical excision. The high local recurrence rate of moderate and high grade malignant schwannoma indicates that a reappraisal of local disease therapy is warranted. Recurrence rates are known to be quite high depending on whether local or radical excision has been practised. With extensive radical excision of adjacent soft parts, Clark (1957) sound that local recurrence rates fell from 61% to 10%.

We presume that with the radical excision of the recurrent tumour, our patient may be completely controlled and escape the appearance of pulmonary metastases which are known to be frequent in this disease.

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HYPOGLYCAEMIA AS A COMPLICATION OF TREATMENT OF CEREBRAL MALARIA WITH PARENTERAL QUININE— A CASE REPORT

Md. Abul Faiz[1], Md. Nurun Nabi[2], A K M Monjur Murshed[3]

Key Words :

Cerebral Malaria, Quinine, Hypoglycaemia.

Introduction :

Cerebral Malaria with neurological features are the commonest complications of falciparum malaria (Faiz M A, et al, 1985). Hypoglycaemia has only recently been recognised as a potential complication of quinine therapy in falciparum malaria (White N J, et al 1982 & Okitolonda W et al, 1987). No such case was reported in Bangladesh. Here, we report a case of severe hypoglycaemia encountered during treatment of cerebral malaria with parenteral quinine.

Case Report :

A 55 years old male labourer of Comilla District working at Bandarban town was admitted at Medical Unit-I of Chittagong Medical Collage Hospital on 5-3-1988 in an unconscious State. Prior to his unconsciousness he had five days' history of fever with chills and rigor, headache and vomiting.

He was normotensive, febrile (100°F) and anaemic. His grade of consciousness in Glasgow coma scale on admission was eyes open-none, best verbal response-none, best motor response-extension to pain (Fig-1). He had signs of neningeal irritation, brisk tendon jerks, normal size & equal pupil reacting to light normally, bilateral extensor plantar response and herpes simplex in lips. His liver and spleen was not palpable He had no icterus. Fundoscopy revealed no abnormality.

Laboratory investigations revealed plasmodium falciparum parasitamia (34,200/cmm of blood) (figure 2).

Total and differential count of WBC was normal. Hb%-9.5gm% & ESR-25mm in 1st hour. CSF study showed raised pressure, protein-75 mg%, sugar—70 mg%, few lymphocytes, bacteriologically sterile and had no malarial parasite on staining. Random blood sugar was 180 mg%. Urine showed no abnormality, skull and chest skiagrams were normal.

The patient was treated with injection quinine dihydrochloride 600 mg diluted in 250 cc of 5% Dextrose in saline I. V. drip eight hourly for 72 hours followed by oral

1. Md. Abul Faiz, FCPS (Bang.), Consultant,
2. Md. Nuruu Nabi, FRCP, Professor and Head
3. A K M Monjur Murshed,
Registrar, Department of Medicine
Chittagong Medical College Hospital,
Chittagong.

NAME OF THE PATIENT : M.Y. AGE : 55 YEARS SEX : MALE

DIAGNOSIS = CEREBRAL MALARIA

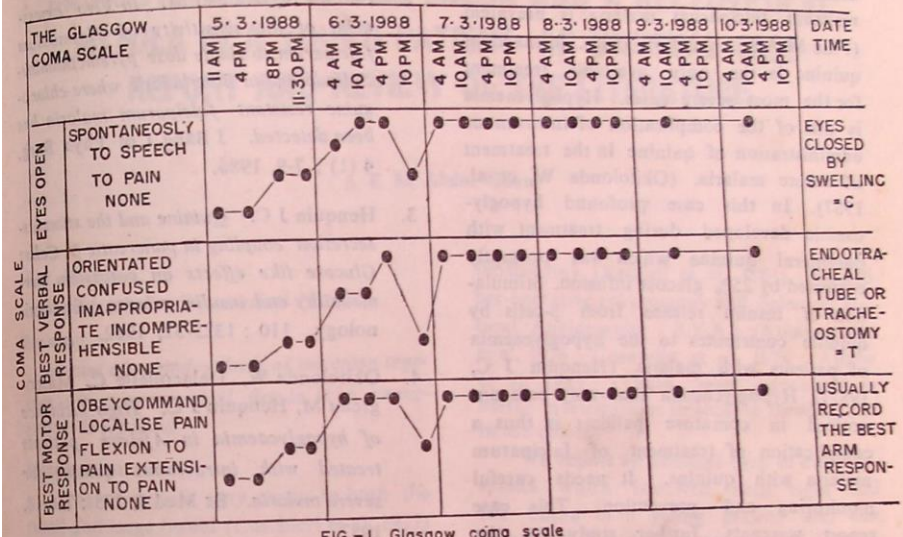

FIG.–1. Glasgow coma scale

Figure—2. *Blood film Showing Trophozoite Stage of P Falciparum.*

quinine sulfate 600 mg. eight hourly for subsequent four days.

While recovering from unconsciousness suddenly his condition deteriorated on the second day with deepening coma and drenching sweats. His pulse rate was 96/minute and B.P. 160/100 mm Hg.His tendon jerks were brisk. Blood sugar level was 40 mg%. 200 cc of 25% Glucose was administered following which his consciousness improved.

During unconsciousness, his nutrition was maintained by nasogastric feeding, fluid intake and output was balanced and an indwelling catheter was introduced. The patient recovered without any neurological deficit.

In this patient EEG, brain scan, plasma insulin and quinine level were not assessed.

Discussion :

Malaria is endemic in eastern border areas of Bangladesh where chloroquine resistant falciparum malaria is prevalent (Faiz M A & Ahmed M, 1988). Intravenous quinine is the only available treatment for the most severe cases. Hypoglycaemia is one of the complication of intravenous administration of quinine in the treatment of severe malaria. (Okitolonda W, et al, 1987). In this case profound hypoglycaemia developed during treatment with parenteral quinine which was promptly corrected by 25% glucose infusion. Stimulation of insulin release from β-cells by quinine contributes to the hypoglycaemia of patients with malaria (Henquin J C, 1982). Hypoglycaemia that may pass unnoticed in comatose patients is thus a complication of treatment of falciparum malaria with quinine. It needs careful monitoring and prevention. This case report warrants further study on the detection and treatment of hypoglycaemia in patients treated with parenteral quinine.

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ANGIODYSPLASIA OF THE COLON AND A RECTOVESICAL FISTULA IN A YOUNG PATIENT—AN UNUSUAL CASE REPORT AND REVIEW OF THE LITERATURE

A K M Abdul Ghani

Key Words :
Angiodysplasia, Rectovesical Fistula.

Summary :
A case of angiodysplasia of the colon together with a rectovesical fistula in a young patient is reported.

Introduction :

Angiodysplasia may occur in both the small and large bowel (Cuschieri et al, 1972) the latter being more commonly involved than the former. According to Boley et al (1977) colonic angiodysplasia is an important cause of bleeding per rectum in the elderly and so is diverticular disease of the colon. This vascular anomaly involves the right side of the colon more than the left side (Cuschieri et al, 1972) and should be suspected when the conventional investigations have failed to localise the source of gastro-intestinal bleeding. The cause may be congenital or acquired, the latter perhaps being the result of chronic partial intermittent obstruction of the submucous veins. It is highly desirable to identify the bleeding site before surgery as there is a high failure rate of localisation at blind laparotomy (Retzleff et al, 1961). Pre and per-operative colonoscopy and Selective Visceral Angiography (S.V.A) (Athanasoulis et al. 1976 ; Casarella et al, 1974 ; Athow et al, 1985 ;) have been advocated as useful investigations to localise bleeding site in the colon.

We report an unusual case of angiodysplasia which involved both right and left colon in a young patient and was associated with a large recto-vesical fistula.

Case History :

A 30 year old Saudi male ambulance driver presented at casualty department with bleeding per rectum, frank haematuria and dysurea of 24 hours' duration. There was no past history of any colonic or peptic ulcer disease. However, he suffered from recurrent urinary tract infections in the past two years. Physical examination including his pulse and blood pressure recorded on admission were normal. Sigmoidoscopic examination revealed dark red blood in the rectum coming from above and a large recto-vesical fistula with chronic inflammatory changes around it. The patient was catheterised for continous drainage and irrigation of the bladder. Urine was frankly blood stained which was presumably due to blood leaking from the rectum through the fistula.

Dr. A K M Abdul Ghani FRCS Ed,
Consultant General Surgeon, Hera General Hospital, Makkah, Saudi Arabia.

Haemoglobin on admission was 12.4 gms/dl and the clotting studies were within normal limit. Upper gastro-intestinal endoscopy did not reveal any source of bleeding in the oesophagus, stomach or duodenum. An urgent intravenous urography showed normal upper urinary tract and leakage of the dye into the rectum. On the following day the patient had further episodes of profuse rectal bleeding. His blood pressure dropped to 85/50 mm Hg and haemoglobin to 6.45 gms/dl. The patient was resuscitated for hypovolemic shock and blood was transfused. After general condition had improved an emergency Baenema was done. However, the x-ray failed to show any significant colonic lesion to account for bleeding.

An exploratory laparotomy was performed for uncontrolled bleeding. Blood was seen in the whole colon without any visible or palpable source. The small bowel did not contain any blood. The right and left colon were inspected by a rigid sigmoidoscope through colotomies after a thorough colonic lavage. The examination revealed areas of diffuse small mucosal abrasions covered with fine clots in the caecum, ascending and descending colon. After washing off the clots fresh blood was seen collecting in these areas. It was concluded that these abraded areas were the sources of bleeding and accordingly sub-total colectomy had to be done. The urinary bladder was opened. Its interior appeared normal apart from the fistulous opening in the basal part with chronic inflammatory changes. The fistula was closed with interrupted stitches using 'O' chromic catgut. The bladder was drained by a suprapubic catheter. The terminal ileum was taken out as an ileostomy in the right iliac fossa and the rectum as a mucous fistula through the lower part of the main wound. The clinical diagnosis of angiodysplasia was confirmed on angiographic and histological examination of the specimen.

The patient made an uneventful recovery. There was no further bleeding and the ileostomy worked satisfactorily. The rectovesical fistula was confirmed on proctoscopy and cystoscopy. The urinary catheter was removed and the patient was able to pass urine without any problem.

Six weeks later an ileo-rectal anastomosis was done. However, the patient had several post-operative complications but eventually he recovered and could be discharged. At follow-up in the outpatient clinic he had no further bleeding and the bowel action varied from two to three times per day. A recent sigmoidoscopy revealed normal looking rectal musosa with no sign of recurrence of the fistula.

Discussions :

Massive gastro-intestinal bleeding may originate any where between the oesophagus and the anal canal. Upper gastrointestinal bleeding can readily be diagnosed in 80-95% of cases by an oesophago-gastro-duodenoscopy (Cotton et al, 1973). However, bleeding from the lower intestinal tract may pose a diagnostic challenge. Any ano-rectal lesion such as haemorrhoid, neoplasm or ulcer can be diagnosed on proctoscopy or sigmoidoscopy. If the conventional investigations fail to identify any bleeding in the upper gastro-intestinal tract or ano-rectal region it is highly possible that the source lies in the large bowel diagnosis of which may be really difficult. A Barium-enema can diagnose any gross lesion such

as diverticulosis, neoplasm which, however, may not be the site of bleeding. Vascular anomaly, for example angiodysplasia cannot be diagnosed by Barium-enema as it happened in this case. Although a pre-operative colonoscopy has been advocated to diagnose the cause of colonic bleeding this is unlikely to be helpful in a patient with massive haemorrhage. One distinct advantage of colonoscopy is that an attempt may also be made to diathermise minute bleeding points. Our patient did not have any colonoscopy as there was none available at that time. Selective Visceral Angiography which was introduced in 1965 by Baum et al (Baum et al, 1965) is now widely used to investigate unexplained intestinal bleeding. Extravasation of blood must be 0.5 ml/minute or more for bleeding to appear on angiography (Cuschieri et al, 1972; Nusbaum & Baum, 1963). However this is not always free from complications. Preoperative angiography was not done for our case because of lack of facility and the patient could not be sent to another place as his life was threatended by profuse bleeding. Scintiography using radio-active technicium may detect bleeding (Markisz et al, 1983) but does not localise it accurately as does angiography. In vascular anomaly angiograph reveals a tortous feeding artery and dilated draining veins with an intervening clustre of fine vessels together with leakage of dye into the lumen if there is active bleeding. During angiography attempts may be made to embolize the source of bleeding. The main risk is infarction.

Pre-operative colonoscopy after orthograde colonic wash through a caecostomy (Scott et al, 1986) has been claimed successful to locate the sites of bleeding. In our case we had no choice but to perform blind laparotomy as the patient's life was threatened. Because of non-availability of a colonoscope we used a rigid sigmoidoscope to inspect the right and left colon through colotomies after the bowel had been thoroughly washed with saline by a Foley's Catheter. We succeeded to identify the bleeding sites in the caecum, ascending and descending colon. Primary anastomosis was not attempted because of the rectovesical fistula pathogenesis of which could not be explained properly. However, this was inflammatory, probably non-specific in nature and concidental. Although angiodysplasia is known to be associated with aortic stenosis (Cuschieri et al, 1972, Galloway et al, 1974) and or Rendu-Osler-Weber disease (Cuschieri et al, 1972) there was no evidence of either in our patient. We advocate from our experience in this unusual case that the colon should be inspected thoroughly by appropriate techniques even if this necessitates the use of a rigid sigmoidoscope, to idnetify the bleeding sites when facilities for colonoscopy or Selective Visceral Angiography are not available before subjecting the patient to any blind limited resection which may not always be successful to control the bleeding as the lesion may also be in the remaining portion of the colon.

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COLLEGE NEWS

Separate Syllabus for FCPS I Examination in Psychiatry :

The separate Syllabus has been prepared and approved by the Council of the College for FCPS Part I Examination in Psychiatry. This new Syllabus will be effective from January, 1989 Examination.

Continuing Medical Education Programme :

March 31, 1988 — Dr. M. Nabi Alam Khan
Professor of Cardiac Surgery, ICVD, Dhaka
delivered a lecture on "Study on Close Mitral Commissurotomy in 139 cases of severe Mitral Stenosis of Juvenile age group between 8-15 years in Bangladesh".

April 28, 1988 — Dr. M. Q. K. Talukder
Professor of Paediatric Nutrition and Gastroenterology, IPGMR, Dhaka, delivered a lecture on "Nutrition, Malnutrition, Growth and Development of Children in Bangladesh".

August 25, 1988 — Dr. M. A. Salam
Senior Consulant, General Hospital, Comilla delivered a lecture on "A review on Modern Medical Education System".

Examination News :

Results of FCPS Part I, FCPS Part II and MCPS Examination held in July, 1988 are as follows :

343 candidates appeared in FCPS Part I Examination in different subjects and only 27 candidates came out successful. Subjectwise result are as follows :

Subject	Number appeared in Theory exam.	Number qualified	Number Passed
Medicine	72	32	15
Surgery	83	22	4
Obst. & Gynae	47	12	2
Paediatrics	44	5	1
Ophthalmology	43	6	1
ENT Diseases	16	7	2
Psychiatry	6	1	1
Radiology	8	1	0
Anaesthesiology	14	1	1
Radiotherapy	2	0	0
Clinical Pathology	8	4	0
	343	98	27

Candidates securing 12 grade marks in theory examination in more than two subjects or below 12 grade mark in any subject were not called for Viva-voce Examination.

74 candidates appeared in FCPS Part II Examination in different subjects. List of candidates who satisfied the board of examiners are as follows :

Roll No	Name	Name of Medical College from where graduated.	Subject
1	Dr. Syed Atiqul Haq	Chittagong Med. College	Medicine
5	Dr. Firoz Ahmed Quraishi	Rajshahi Med. College	Medicine
6	Dr. Md. Abdul Hannan	Sylhet Med. College	Medicine
15	Dr. Manabendra Nath Nag	Mymensingh Med. College	Medicine
16	Dr. Hasan Askari Md. Nazmul Ahasan	Dhaka Med. College	Medicine
17	Dr. Sk. Md. Bahar Hussain	Chittagong Med. College	Medicine
24	Dr. Md. Shahab Uddin	Dhaka Med. College	Medicine
29	Dr. Harunur Rashid	Dhaka Med. College	Surgery
35	Dr. M.I.Md. Nasim Sobhani Khondker	Dhaka Med. College	Surgery
36	Dr. Najib Mohammad	Sir Salimullah Med. College	Surgery
37	Dr. Md. Shahidur Rahman	Rajshahi Medical College	Surgery
41	Dr. Israil Biswas	Sir Salimullah Med. College	Surgery
42	Dr. Mahmud Hasan	Mymensingh Med. College	Surgery
44	Dr. Mrinal Kanti Roy	Mymensingh Med. College	Surgery
45	Dr. Mohammad Mohsin	Sir Salimullah Med. College	Surgery
53	Dr. Panna Lal Saha	Rajshahi Medical College	Surgery
54	Dr. Saleha Begum Chowdhury	Chittagong Medical College	Obst. & Gynae
55	Dr. Kamrun Nahar	Dhaka Medical College	Obst. & Gynae
60	Dr. Choudhury Habibur Rasul	Dhaka Medical College	Paediatrics
62	Dr. Sayed Zahid Hossain	Sher-e-Bangla Med. College	Paediatrics
63	Dr. Soofia Khatoon	Dhaka Medical College	Paediatrics
64	Dr. Abdul Hannan	Sylhet Medical College	Paediatrics
66	Dr. Devabrata Roy	Sir Salimullah Med. College	Paediatrics
67	Dr. A. A. Md. Rashed Nizam	Sylhet Medical College	Ophthalmology
69	Dr. Md. Abdullah	Sylhet Medical College	E. N. T. D.
70	Dr. Ranjit Kumar Nath	Chittagong Medical College	E. N. T. D.
71	Dr. A. M. M. Shariful Alam	Mymensingh Med. College	Radiotherapy
72	Dr. Md. Enayet Karim	Rangpur Med. College	Psychiatry
73	Dr. Md. Golam Rabbani	Rajshahi Med. College	Psychiatry
74	Dr. Abul Hasnat Mohammad Firoz	Dhaka Medical College	Psychiatry

65 candidates appeared in MCPS examination in different subjects. List of candidates who satisfied the board of examiners are as follows :

Roll No.	Name	Subject
5	Dr. Abu Naser Ziauddin Ahmed	Medicine
7	Dr. Mohammed Mahtabuddin Hasan	Medicine
19	Dr. Rehana	Obst. & Gynae
24	Dr. Shufia Rashid	Obst. & Gynae
28	Dr. A. K. M. Majibur Rahman Khan	Obst. & Gynae
31	Dr. Moksuda Khanom	Obst. & Gynae
35	Dr. Muhammad Anisur Rahman	Paediatrics
36	Dr. Syed Misbahul Hoque	Paediatrics
45	Dr. Md. Azizul Haque	Paediatrics
47	Dr. A. S. M. Sajjad Hosain	Paediatrics
52	Dr. Md. Shafiul Alam	ENTD
56	Dr. Md. Shamsul Alam	Anaesthesiology
59	Dr. Muhammad Sayeedur Rahman	Cl. Pathology
61	Dr. Md. Shahidullah	Dermatology &V.D.
62	Dr. S. M. Akhtar Kamal Chowdhury	Dental Surgery
63	Dr. Muhammad Sadeque	Forensic Medicine

Recognition of Training :

The Council of Bangladesh College of Physicians and Surgeons has decided to recognise the training of doctors in the department of Medicine, Surgery, Obst. & Gynae in the Dhaka National Medical Institute Hospital provisionally for a period of 2 years w. e. from 21.4.88 on condition that only 1 year will be accepted as approved training as prerequisites for appearing in Fellowship Examination of the College.

The Council has also decided to recognise the training of doctors in the department of Anaesthesiology in the Holy Family Red Crescent Hospital, Dhaka for a period of 3 years w. e. from 9.6 88 on same condition that only 1 year will be accepted as approved training as prerequisites for appearing in FCPS Part II Examination in Anaesthesiology.