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PERFORATION OF TUBERCULOUS ENTERITIS
Report of a Case

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WITH THE general fall in the incidence of tuberculosis, one is apt to forget the profound aftermaths of the infection. With the decline in the number of pulmonary tuberculosis the incidence of intestinal affections have become notably uncommon and rarer still, the free perforation of tuberculous ulcer of the bowel (Mitchell & Bristol, 1954). Indeed, the recorded morbidity and mortality arising from these are high but with early diagnosis and an aggressive management regime the final outcome of these cases with otherwise poor prognosis, can be favourably modified, (Sweetman & Wise, 1959; Bhausali *et al.*, 1968).

This paper reports an uncommon case of perforated tuberculous enteritis which was diagnosed in the late stage of the disease. It is the object of this paper to draw fresh attention to the severe implications of this condition and to plead for its inclusion into the differential diagnosis of gastro-intestinal disorders, more so in areas where tuberculosis is prevalent.

CASE REPORT

The patient was a 26 year old female clerk with a history of malaise, anorexia, weight loss, intermittent low grade fever, central abdominal pain and diarrhoea for six months duration. She was first admitted to a district hospital where on physical examination no significant clinical abnormality could be detected.

The following investigations were carried out. Blood study revealed haemoglobin of 6.0 gm%, total white cell count of 9,000/c.mm, platelet count of 400,000/c.mm, and a sedimentation rate of 20 mm/hour. Serum protein was in the order of 4.4 gm% (Albumin 1.4 gm%, globulin 3.0 gm%), alkaline phosphatase 14 KA units and the total serum bilirabin was 0.6 mg%. Urine analysis was normal and stool for occult blood was positive. The Mantoux test was negative. Radiological examination of the chest revealed no abnormality.

During her stay at the district hospital her condition deteriorated with loss of weight and development of dependent oedema and generalized ecchymosis. Empirically, she was started on a course of ampicillin pending a definite diagnosis. She developed abdominal distension and four days later was transferred to the medical unit, General Hospital, Kuala Lumpur.

On review of the history, her husband was found to be a known case of pulmonary tuberculosis who had defaulted treatment. On physical examination her general condition was noted to be poor, associated with severe pallor, mild clubbing, ankle oedema and generalized ecchymosis. The pulse rate was 80/min. and the systemic blood pressure was recorded at 120/70 mm Hg. On auscultation of the lungs basal crepitations were heard. The abdomen was distended, resonant on persussion with decreased bowel sounds. A working diagnosis of pyrexia of unknown origin was made and several differential diagnosis considered.

Blood investigation revealed haemoglobin of 10 gm%, total white cell count of 12,000/c.mm, differential count showing a neutrophil concentration of 95% and lymphocyte of 6% with sedimentation

rate of 4 mm/hr. Biochemical estimation of electrolytes showed sodium 115 meq/litre, potassium 2.4 meq/litre, and chloride 101 meq/litre. The blood urea concentration was 14 mg%. Sputum examination for acid bacilli was positive.

For the treatment of her diarrhoea she was commenced on lomotil and intravenous fluid therapy was instituted to combat the electrolyte imbalance. Daily injection of 1 gm streptomycin and 400 mg of INH orally were added to this therapy.

After four days, she was referred to the surgical unit as a case of sub-acute intestinal obstruction. On physical examination, she was now found to be cachexic with poor general condition. The pulse rate was 100/min. and the blood pressure was recorded at 90/60 mm Hg with a sub-normal temperature of 96.8F°. The abdomen was markedly distended with tense and shiny skin. No mass was palpable and on percussion the liver dullness was obliterated. Bowel sound was absent and per rectal examination revealed no abnormality. A diagnosis of tuberculous enteritis with perforation of gut was made. Scout film of the abdomen confirmed the pseumoperitoneum.

Immediate resuscitation measures were commenced with intravenous injection of hydrocortisone 500 mg and blood transfusion. As the patient was already on ampicillin 500 mg six hourly, intramascular injection of clindamycin 150 mg was added in eight hourly doses.

An emergency exploratory lapatotomy was performed revealing gross pneumoperitoneum and peritonitis yielding 1000 mls. of purolent aspirate from the pelvic cavity. Examination of the bowel showed multiple small whitish follicle on terminal ileum and caecum. The ileum was observed to be oedematous with multiple small perforations and the caecum was thickened with enlarged mesenteric lymph nodes. The liver, gall bladder, stomach and colon were found to be normal.

A right hemicolectomy with end to end ileocolic anatomosis was performed and two drains were inserted – one in the pelvic cavity and the other in the right para-colic gutter. Post-operatively the intramascular injection of streptomycin was reduced to 0.75 gm, INH 400 mg, Rifampicin 600 mg and pyridoxin 10 mg daily were added to the drug therapy. To combat her poor general condition intravenous hyperalimentation was started.

Following surgery slight post-operative improvement was noted as recorded by a rise in the blood pressure to 100/60 mm Hg, the return of normal pulse rate and a normal body temperature. Slight improvement was noted in her general condition but the urine output remained poor.

Blood investigation revealed haemoglobin of 14.8 gm%, total white blood cell of 14,000/c.mm with differential count showing neutrophil of 86% and lymphocytes of 14%. Blood urea was 34 mm%. Serum electrolytes were as follows:- sodium 136 meq/litre, potassium 5.2 meq/litre and chloride 101 meq/litre.

On the fourth post-operative day, the patient developed high fever together with hypotension, tachypnoea but no cyanosis. Clinical examination of chest revealed bilateral basal crepitations and the abdomen was soft with audible bowel sounds. Solumedrol 1.5 gm was instituted, oxygen therapy through mask commenced and one litre of plasma was given. She remained refractory to the resuscitative measures and succumbed the same day.

Histolopathological examination of tissues taken at laparotomy confirmed the diagnosis of tuberculous enteritis with ulceration and perforation.

DISCUSSION

It is stated that the rate of intestinal tuberculosis is directly proportional to the incident of pulmonary tuberculosis (Mitchell & Bristol, 1954). Prior to the advent of anti-tuberculous drug 70% of patients with advanced pulmonary tuberculosis developed concommitant tuberculous enteritis commonly at the ileo-caecal region (Weisburgh & Luongo, 1961). The reason advanced for this was the presence of abundant lymphoid tissue anatomically located in these areas (Bombart *et al.*, 1961).

Tuberculosis enteritis commonly manifests in either of the three gross patholigical forms, namely – ulcerative, hypertrophic or mixed ulcerative hypertrophic type. Prior to the advent of antituberculous chemotherapy perforations of these lesions were rare but since its introduction have become more prevalent (Jordan & De Bakey, 1954). This is alleged to result from the reduction of local tissue inflammatory reaction to the infection thereby reducing fibrous tissue formation. When the lesion affects the Payer's patches the perforations are longitudinal but when the lesion takes the form of a diffuse involvement these are usually pinhead in nature. Haemorrhage and fistulae formation have also been known to occur in the severe cases.

The clinical presentation of tuberculous enteritis is determined by the type of pathological reaction it provokes. Hypertrophic tuberculosis results in

stenosis with manifestations of obstruction. The more common ulcerative form causes diarrhoea or constipation and only occasionally is it known to cause progressive inaniation. General symptoms of fever, night sweats, anorexia, malaise and weight loss may occur in all these variety of symptoms but these symptoms are often falaciously ascribed to any co-existent pulmonary lesion. A helpful physical sign is the presence of a mass, often tender, in the right ilac fossa. Barium study (barium meal with follow through or enema) may be helpful in confirming the diagnosis.

The treatment recommended for tuberculous enteritis *per se* is non-operative consisting of anti-tuberculous drug therapy with general supportive measures. When associated with complications they warrant surgical intervention. With perforations, suture of the perforation or limited bowel resection is the recommended treatment. With the less aggressive simple sutures the mortality is quoted to be in the region of 50%. In view of this, he had recommended resection of the lesion with primary anastomosis (Sweetman & Wise, 1959).

In this case the lesion was of the ulcero-hypertrophic variety. Procrastination in the diagnosis of tuberculous enteritis led to perforation which even with concentrated resuscitative measures and surgical resection failed to control the peritonitis that led to fatal septicemia.

SUMMARY

Following pertinent features emanate from the study of this case:-

(a) That the duration of ill health and the general symptomatology were suggestive of tuberculosis.

(b) Despite this the diagnosis of tuberculous enteritis was never entertained; hence no specific therapy was instituted.

(c) And, only when the pathology was advanced and complications had set in was tuberculosis considered.

(d) The failure to arrive at a provision diagnosis early in the course of the disease and the empirical use of ampicillin precluded the timely use of antituberculous drugs with disastrous consequences.

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