Pseudomembranous Colitis

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Antibiotic associated diarrhea is a well described common side effect of the use of antibiotics. The diarrhea is usually self limited and often stops after the antibiotic is discontinued. Infrequently the diarrhea becomes a more serious problem than the initial disease for which the antibiotic was prescribed, and progresses to dehydration, electrolyte imbalance, shock and even death(1). This severe form of antibiotic associated diarrhea is called pseudomembranous colitis, due to the formation of a pseudomembrane in the colon visualized on sigmoidoscopy. Rarely, in severe cases the pseudomembranes may coalesce and be shed(2). *Clostridium difficile,* an anaerobic organism, has been incriminated as the etiologic agent(3). This condition in children was reported in the Indian literature for the first time in 1985(4).

Case Report

A 4½-year-old girl was on out-patient treatment with amoxicillin for a respiratory infection. On the fifth day of treatment, she had a recurrence of fever with sudden onset of severe diarrhea with copious foul smelling watery green stools. On examination she looked ill, had a temperature of 103°F and was drowsy; pulse rate was 192/min, thready and BP was 68/40 mmHg. She had oliguria. There was mild abdominal distension and bowel sounds were weak. Investigations revealed Hb 11.5g/dl, TLC 3600/cu mm with 86% polymorphs which included 66% band forms. Majority of the polymorphs showed toxic granules. Serum electrolytes, CSF studies, liver function tests, blood urea and creatinine were normal. Malarial parasite was not detected on peripheral blood smear examination. Blood and urine cultures were sterile.

She was managed with fluids, dopamine drip and broad spectrum antibiotics. A day later she passed a three feet long structure per rectum resembling a cast of intestine (*Fig.*). With a possibility of pseudomembranous colitis in mind, her antibiotics were stopped and oral metronidazole 200 mg every 8 hours started.

Histopathological examination of the specimen revealed acute inflammatory tissue with necrotic cells, polymorphs, fibrin, macrophages and lymphocytes. There was no epithelial tissue. Stool culture for *Clostridium difficile* was negative. Cytotoxin assay in stool could not be done.

She made a good recovery thereafter and on discharge had normal clinical and laboratory parameters.

Discussion

Antibiotic associated colitis is reported following administration of a variety of antibiotics and drugs, commonest being clindamycin, ampicillin and amoxicillin. The usual presentation in children is acute onset of profuse watery diarrhea during the

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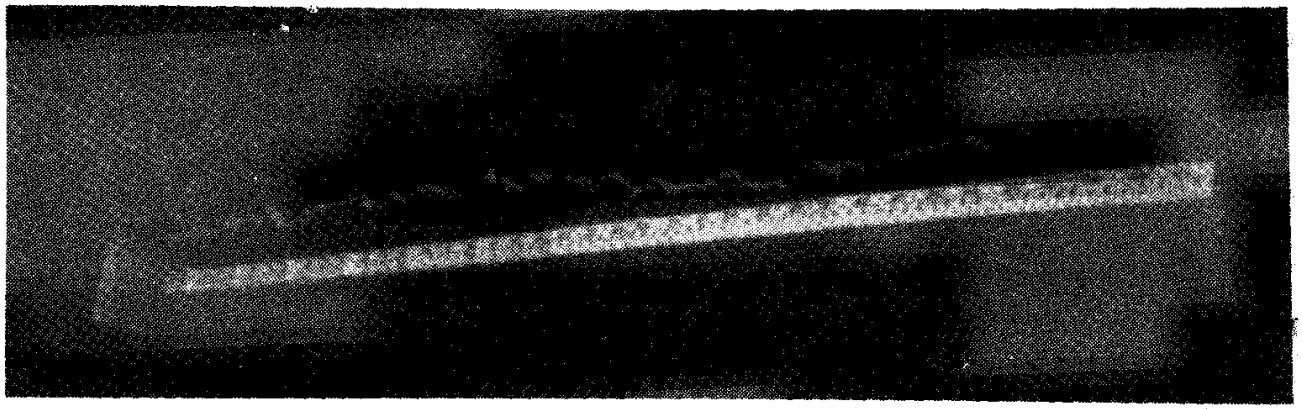

Fig.. Coalesced pseudomembrane 36" long.

first week of therapy. Diarrhea may be bloody. In uncomplicated cases it resolves shortly after stopping the antibiotic. However, in severe cases, there may be toxic megacolon, peritonitis, septicemia and hypoalbuminemia. Occasionally, the disease may occur 2-3 weeks after completion of antibiotic therapy. The commonest site involved is the descending colon. However, sometimes the entire colon or the small intestine may be involved. Sigmoidoscopy reveals a red, edematous friable mucosa with multiple, raised yellow white plaques. These plaques may be small, patchy or confluent in severe cases(1). However, a long confluent pseudomembrane of the size seen in our case is very rare. Histopathological examination shows a pseudomembrane consisting of mucin, fibrin, necrotic cells and polymorphs.

There is overgrowth of the anaerobe *Clostridium difficile* due to antibiotic suppression of normal gut flora, and a cytotoxin produced by this organism has been incriminated in the causation of this disease(3). Toxin assay could not be done in our patient due to nonavailability of this facility, which would otherwise have confirmed the diagnosis.

Both vancomycin and metronidazole are effective in treatment, but as vancomycin, the drug of choice is extremely expensive, metronidazole is good alternative(5). Overall mortality reported is about 28%(6) and is usually associated with severe disease.

A case of pseudomembranous colitis following amoxicillin therapy with severe manifestations is presented; the unusual feature being the passage of a long, confluent pseudomembrane 3 feet in length.

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Serum Magnesium, Calcium, Zinc in Infantile Tremor Syndrome

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Infantile tremor syndrome (ITS) is a condition peculiar to this country, characterized by tremors, anemia, skin pigmentation and nutritional deficiency in a plump child. Many theories on the etiopathogenesis have been postulated but none seems convincing(1,2). Many workers have tried to correlate the symptoms with certain trace element deficiency states without satisfactory results. In order to find out the relation of levels of serum magnesium, calcium and zinc to etiopathogenesis of ITS, this study was undertaken.

Material and Methods

The present study was carried out on 32 patients with clinical diagnosis of infantile tremor syndrome admitted to Umaid Hospital for Women and Children, Dr. S.N. Medical College, Jodhpur. An equal number of age and sex matched normal children served as controls. The patients of ITS were further classified in two weight groups: weighing more than 60% and less than 60%(3). The extent of tremors, as to the part of body involved, was carefully noted. The levels of serum magnesium, calcium and zinc were detected by atomic absorption spectrophotometer (Model No. SP 90; Pye Unichem)(4).

Results

Age and sex distribution of patients and controls is shown in *Table I.* All the patients were less than 2 years of age and 53.13% patients belonged to the age group 6-12 months.

The mean levels of serum magnesium, calcium and zinc in ITS and controls are shown in *Table II.* The mean serum magnesium level was markedly reduced while mean serum calcium and zinc were marginally reduced. Mean serum levels of magnesium, caclium and zinc were significantly reduced in cases of ITS with less than 60% weight (Grades III and IV malnutrition). The relation of mean serum magnesium levels and extent of tremors is shown in the *figure.*

Discussion

Mean serum magnesium level was markedly reduced in patients of ITS as compared to controls, the difference being highly significant ($p<0.001$). Chapparwal studied magnesium levels in cerebrospinal fluid (CSF) and serum in 25 patients of ITS; 80% had markedly low levels of magnesium in CSF and 36% had frank hypomagnesemia(5). Gerald reported that

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