The common symptoms of capillariasis are diarrhea, recurrent vague abdominal pain, weight loss, malaise, and anorexia. However, there has been no report of capillariasis presenting with chronic intestinal pseudo-obstruction.

A 35-year-old Thai man came to the hospital with history of chronic voluminous, watery diarrhea with 8 to 10 stools daily for 4 months, loss of appetite, and loss of 6 kg body weight. The patient was a farmer working in Prachinburi Province in the northeast of Thailand. The patient suffered from recurrent vague abdominal discomfort with progression of the symptoms prior to admission. Physical examination revealed body weight of 43 kg, muscle wasting, pale conjunctiva, and abdominal distention with prominent borborygmi. Minimal ascites was detected. Laboratory investigation showed Hb 8 g/dl, WBC 6,500/mm³, neutrophils 41%, lymphocytes 48%, eosinophils 7%, monocytes 3%, basophils 1%, and adequate platelet count. Blood chemistry showed Na 130 mEq/l, K 2.5 mEq/l, total Ca 7.7 mg/dl, fasting plasma glucose 70 mg/dl, creatinine 0.95 mg/dl. Liver function test showed total bilirubin 0.74 mg/dl, SGOT 21 U/l, SGPT 22 U/l, albumin 1.8 mg/dl, globulin 2.3 mg/dl, prothrombin time 15.4 seconds (control 12.4 seconds). Anti-HIV was negative. Ascites fluid showed transudative profile. Stool examination revealed Capillaria philippinensis ova. Plain abdomen film demonstrated multiple air-fluid levels in small intestine.

**Fig 1**—An upright abdominal film on admission showed air-fluid levels in small intestine.

**Fig 2**—Barium study on prone position showed malabsorption pattern of small intestine.
intestine which indicated intestinal obstruction (Fig 1). After hypokalemia and hypocalcemia were corrected, the abdominal film still was not changed. Further investigations to confirm intestinal obstruction were performed. Barium enema of the colon followed by small bowel follow-through demonstrated a malabsorption pattern of small intestine (Fig 2) without mechanical intestinal obstruction in the small and large bowel. Endoscopy with upper jejunal biopsy showed nonspecific inflammation with eosinophil, lymphocyte, and mononuclear infiltration. After excluding mechanical obstruction of the intestine, investigations for secondary causes of intestinal pseudo-obstruction were carried out: thyroid function was normal, carcino-embryonic antigen was negative, antinuclear antibody was negative, and chest film was normal. However, diagnostic laparotomy with full-thickness resection of the small intestine was not attempted.

The patient was treated with albendazole 200 mg twice daily for 10 days. After 2-week treatment the frequency of daily stools decreased to once daily with formed stool. Stool examination was normal and no parasite was found. Plain abdominal film taken 3 weeks later showed the same finding as the previous film on admission. The patient was discharged from the hospital. Five months later, the patient came to hospital for follow-up with a normal stool habit. His weight increased to 52 kg with good appetite. Laboratory investigations including complete blood count, liver function tests, serum Na and Ca, and stool examination were normal. Plain abdomen film showed improvement of intestinal pseudo-obstruction pattern with fewer air-fluid levels in the small intestine than the previous films.

On admission, the plain abdominal film of this patient showed intestinal pseudo-obstruction which might occur in any patient with hypokalemia or hypocalcemia. However after correction of electrolyte imbalance, the abdominal film still showed the same pattern. Thus, electrolyte imbalance were not attributed to intestinal pseudo-obstruction in this patient. Barium studies were performed to exclude mechanical obstruction of intestine. Investigations to find the possible secondary causes of pseudo-obstruction were carried out but they showed negative results. The patient improved after anti-helminthic treatment with increased weight and appetite and normal stool. The abdominal film taken 5 months later was improved. Explored laparotomy was not attempted because of the patient’s marked clinical improvement without abnormal gastrointestinal symptoms.

Chronic intestinal pseudo-obstruction is a rare clinical syndrome caused by ineffective intestinal propulsion and characterized by symptoms and signs of intestinal obstruction in the absence of an occluding lesion of the intestinal lumen (Faulk et al, 1978). It is caused by a large number of disorders of intrinsic smooth muscle, myenteric plexus, or extraintestinal nervous system. Radiologically, there is pronounced distension of the bowel (especially the small intestine like this patient) mimicking intestinal obstruction.

Capillariasis may present with chronic watery diarrhea due to small intestinal malabsorption. Malnutrition contributed to weight loss, muscle wasting, anemic, hypoalbuminemia, and prolonged prothrombin time in this patient. Endoscopic examination of the upper jejunum revealed only nonspecific inflammation. Since most of the worms are found in the jejunum, endoscopic diagnosis is not easily made (Lee et al, 1993). The parasites are known to induce intestinal inflammation with mucosal damage and atrophy (Freshet et al, 1972). However, no information is available on the mechanism of chronic intestinal pseudo-obstruction in capillariasis. The possible mechanism of chronic intestinal pseudo-obstruction in this patient may be due to atrophy or hypotrophy of small intestinal muscle with poor contraction from malnutrition (resulting from chronic diarrhea with malabsorption). Chronic intestinal pseudo-obstruction was believed to be associated with capillariasis in this patient, because the x-ray pattern of pseudo-obstruction markedly improved in 5 months after treatment. Although this patient was properly treated, intestinal smooth muscle may take many months to become normal with good function. However, this patient should be further followed-up to see when pseudo-intestinal obstruction disappeared.

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