

in Crohn's disease. Furthermore, pancreatitis and exocrine pancreatic insufficiency have rarely been reported as initial manifestations of Crohn's disease in children (10).

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### INTESTINAL PSEUDOObSTRUCTION IN ACUTE LYME DISEASE: A CASE REPORT

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We report here a case of acute Lyme disease in a 61-yr-old man who developed a facial nerve paralysis and a relentless intestinal pseudoobstruction 2 wk after the initial prodrome. Both the facial nerve paralysis and pseudoobstruction persisted for a month until the patient sought medical attention. Both lesions resolved only after treatment for Lyme disease was initiated. The temporal association of the pseudoobstruction with the somatic cranial neuropathy and the response of both to specific therapy for Lyme disease suggest that the former was likely the result of a reversible autonomic neuropathy or dysfunction. (*Am J Gastroenterol* 1998;93:1179–1180. © 1998 by Am. Coll. of Gastroenterology)

#### INTRODUCTION

Intestinal pseudoobstruction has not been previously described in Lyme disease. The temporal association of intestinal pseudoobstruction with Bell's palsy occurring in a patient with Lyme disease forms the basis of this case report.

#### CASE REPORT

Six weeks before presenting to our hospital, a 61-yr-old man in robust health, who enjoyed the outdoors and who took no medications, developed lethargy and aches in his joints and muscles. As these symptoms abated over a 2-wk period, he noticed drooping of the right side of his face, an inability to close his right eye, gradually progressive nausea, abdominal distention, and severe constipation, having to strain to pass a stool once in several days. Before this time he used to have a well formed bowel movement every day. He saw a physician at another facility only 4 wk later, a few days before being seen by us, and was prescribed dicyclomine hydrochloride 10 mg, *t.i.d.* He took the tablets only for 1 day. Other than a right sided, lower motor neuron seventh nerve paralysis and generalized abdominal distention with tympany and normal bowel sounds, he had no other abnormalities detected on physical examination. The patient's blood counts, serum electrolytes, calcium, phosphorous, magnesium, blood urea nitrogen, creatinine, liver function tests, amylase, and lipase were in the normal range. Stool examination for ova and parasites and stool culture were negative. The Lyme titer was positive (Lyme IFA 1:1024). Supine, erect, and decubitus films of the abdomen revealed diffusely dilated air-filled loops of small and large bowel together with fecal matter in the colon. As air was present in the rectum also, the appearance was consistent with pseudoobstruction rather than mechanical obstruction (Fig. 1).

The patient was treated for acute Lyme infection with a tapering dose of prednisone and a 3-wk course of doxycycline. Follow-up plain x-rays of the abdomen done 4 days later showed improvement, although the intestines were still dilated. In 1 month, the patient's neurologic deficit completely resolved and his bowel

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FIG. 1. Abdominal x-ray on presentation, showing dilated loops of small and large bowel.

habits gradually returned to normal. A colonoscopy done at that time was normal.

#### DISCUSSION

During the course of systemic infections, mild evanescent gastrointestinal symptoms are not uncommon. In fact, anorexia, nausea, vomiting, and right upper quadrant tenderness may be seen early on in the course of Lyme disease in a small percentage of patients (1). Such symptoms however, are not the result of pseudoobstruction. Our patient's symptoms were progressive and relentless over several weeks. He developed constipation culminating in obstipation, with diffuse small and large bowel dilation, in the absence of mechanical obstruction. These are the hallmarks of intestinal pseudoobstruction (2). Intestinal pseudoobstruction can be either myogenic or neurogenic in origin. Although it is impossible to be certain of the underlying mechanism in our patient, it is unlikely for this episode to have been caused by an intestinal myopathy because most myopathic causes, such as advanced scleroderma (3), are not reversible. In contrast, autonomic visceral neuropathies may be reversible and are known occasionally to accompany somatic neuropathies such as the Guillain-Barré syndrome (4). Such neuropathies may be caused by structural neuronal lesions or may result from autonomic dysfunction arising as epiphenomena from systemic stress, as frequently seen in Ogilvie's syndrome. It is therefore tempting to speculate that our patient's intestinal pseudoobstruction was the result of autonomic dysfunction that accompanied the somatic neuropathy of the seventh cranial nerve. Symptoms suggestive of intestinal pseudoobstruction have been reported with infections such as herpes zoster (5), infectious mononucleosis (6), and rubella (7).

To the best of our knowledge, neither autonomic dysfunction nor pseudoobstruction has previously been reported in association with Lyme disease. Although one cannot rule out a chance association of intestinal pseudoobstruction with Lyme disease in this case, this seems unlikely. Our patient had no previous history of similar symptoms, nor did he have an underlying disorder that might result in a reversible form of intestinal pseudoobstruction,

such as peritoneal irritation, chronic hypocalcemia, hypokalemia, hypomagnesemia, or hypothyroidism. The patient had been prescribed dicyclomine, but this in fact was for symptoms of pseudoobstruction that had already been present for >3 wk. Besides, he took only two to three 10-mg tablets and had discontinued the medication 2 days before seeing us, as he felt it exacerbated his problem. That the symptoms completely resolved, and have not recurred over the past 9 months, effectively rules out primary pseudoobstruction or causes of permanent secondary pseudoobstruction.

Recovery from sensory neuropathies associated with Lyme disease generally takes several weeks after specific therapy is initiated, as did the facial nerve palsy in our patient. Although the onset of recovery of his abdominal symptoms began 4 days after the initiation of treatment, their complete resolution took considerably longer.

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