Laminated intestinal calculi – A rare complication of Crohn’s disease

HUGH J FREEMAN MD, ANDREW SEAL MD, DAVID LI MD

INTTESTINAL FOREIGN BODIES MAY be either exogenous or endogenous (1). Endogenous foreign bodies may be primary and formed within the intestinal tract (eg, enteroliths), or secondary and enter the gastrointestinal tract from adjacent structures (eg, gallstones). Enteroliths may be further subdivided into a false type formed from clumping of insoluble ingested material or by inspissation of bowel contents (eg, phytobezoars, trichobezoars, fecoliths), or a true type formed from precipitation of substances present in the lumen (eg, bile or mineral salts, such as calcium) (1).

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Laminated intestinal calculi detected during the course of clinical evaluation may be associated with congenital (eg, Meckel’s diverticulum, ileal duplication cyst) or acquired conditions (eg, anastomotic or postinflammatory strictures, such as Crohn’s disease of the small intestine). In both, local stasis of the intestinal contents is critical to stone development (1).

Although small intestinal strictures are extremely common in Crohn’s disease, enterolithiasis is very rare, being recorded to date in only 14 patients...
Detection is important, however, since intestinal calculi may be associated with obstruction, bleeding, refractory anemia and, in rare instances, perforation.

**CASE PRESENTATION**

A 51-year-old male was initially referred for assessment of Crohn’s disease in November 1984. A diagnosis of Crohn’s disease involving the small intestine was first established in 1956 in the United States followed by repeated admissions to a teaching hospital in Montreal for abdominal pain and bloody diarrhea over a two-year period. A 20 cm small bowel resection with an ileocolic anastomosis was done in 1959 followed by a second resection for recurrent ileal disease in 1969. Pathological review revealed characteristic features of Crohn’s disease with granulomatous inflammation. In 1970, barium radiographic studies demonstrated recurrent small bowel disease with stricture formation. The patient was treated with sulfasalazine. Although he remained virtually asymptomatic, barium studies of the upper and lower gastrointestinal tract in 1974 revealed a jejunal stricture with small bowel dilation proximal to this narrowed segment.

In 1984, abdominal pain recurred. Laboratory studies, including a hemoglobin, white blood cell and platelet counts, liver chemistry tests, sedimentation rate, serum iron, folic acid and vitamin B12, were normal. Urinalysis was normal. Colonoscopy and biopsies were normal. Barium studies revealed similar changes to 1974; in addition, thickening and nodularity of the mucosa in the descending duodenum were now evident. Sulfasalazine treatment was continued and the patient’s symptoms appeared to resolve with a lactose-free diet. Clinical evaluations and blood tests done semi-annually over the next eight years were normal. In 1990, abdominal pain recurred temporarily; barium studies now showed a normal duodenum as well as previously defined strictures but no new radiological findings and, following this study, his symptoms spontaneously resolved.

In May 1992, the patient was re-evaluated because of recurrent and severe abdominal pain. Laboratory studies revealed (normal range in brackets): hemoglobin, 126 g/L (140 to 180), mean cell volume, 76 fl (82 to 98),
white blood cell count, 7.3×10^9/L (4.0 to 11.0), serum ferritin, 16 µg/L (18 to 250), serum iron, 6 µmol/L (7 to 23) and serum iron saturation 0.09 (0.20 to 0.55). A barium study revealed a markedly dilated loop of small bowel proximal to a stricture; in the dilated loop, a filling defect was seen (Figure 1). In delayed films done 24 h later, residual barium was seen in the dilated loop; two radiolucent and mobile filling defects were clearly seen (Figure 2). Following treatment with oral 5-aminosalicylic acid his pain subsided.

In September 1992, pain recurred in the patient’s right upper quadrant with development of erythema and swelling over the right rectus sheath. Within 24 h a small amount of purulent drainage was seen from an apparent fistula. Except for a hemoglobin level of 112 g/L, other laboratory studies, including a white blood cell count, were normal. An abdominal film revealed two structures with faint peripheral calcification (Figure 3). An abdominal computed tomography scan showed a thickened right rectus muscle with an irregular 3 cm fluid collection typical of an abscess. A dilated segment of small bowel was present with two peripherally calcified structures (Figure 4). After initiation of parenteral nutrition and intravenous antibiotics, a fistulogram demonstrated communication with the small intestine; again, the two filling defects were seen (Figure 5). After failure of the fistula to heal with conservative management, a small intestinal resection was done including the enterocutaneous fistula. The resected segment of small bowel revealed characteristic features of Crohn’s disease with two enteroliths (Figure 6).

DISCUSSION

Primary enterolithiasis or intestinal calculi are rare but may be a clue to occult intestinal disease and must be differentiated from secondary calculi that originate elsewhere and subsequently enter or erode into the gastrointestinal tract, such as gallstones. Formation of intestinal calculi requires the proper chemical milieu within the lumen of the intestine (1,8,10) as well as conditions that lead to local stasis, as may be observed with congenital abnormalities such as a Meckel’s diverticulum or a duplication cyst of the distal ileum. Occasionally, acquired causes of intestinal stasis may be responsible, including postoperative or postinflammatory strictures such as occurs in Crohn’s disease. Although our patient had long-standing Crohn’s disease, the initial detection of intestinal calculi (eg, with abdominal films) in a new patient should lead to further studies including barium radiographs to exclude unrecognized intestinal disease, including Crohn’s disease. Enteroliths are always clinically significant because they may represent the first clue to an occult small bowel diverticulum or a stenosing lesion. Misinterpretation as biliary or urinary tract calculi or as innocuous concretions in the peritoneal or extraperitoneal spaces could result in inappropriate treatment (1).

To date, there have been 14 patients reported in the literature with Crohn’s disease and a clinical course that has been complicated with intestinal calculi (1-10). Usually, but not exclusively, these are detected in the small intestine. Single or multiple calculi with smooth or faceted borders have been described, usually, as in our patient, in a chronically dilated small bowel loop proximal to a stricture. In some patients, intestinal calculi may have been primarily responsible for the obstructing symptoms along with chronic or recurrent blood loss and iron deficiency anemia that has been refractory or difficult to manage (10). In very rare patients with Crohn’s disease and intestinal calculi, the presentation has been dramatic with perforation (5); thus, while rare, the potentially serious outcome of intestinal stones deserves emphasis. In all patients described with Crohn’s disease and intestinal calculi, the average duration between the
clinical onset of Crohn’s disease and the detection of enterolithiasis was prolonged, usually over a decade, range five to 33 years (10). In our patient, who had calculi detected in 1992, the Crohn’s disease was first established elsewhere 36 years earlier in 1956.

Although stasis appears to be a critical factor in the pathogenesis of these stones, their chemical composition is believed to depend on the site of formation along the length of the small intestine as well as the pH of the intestinal chyme at the location of the stones. The relatively lower pH of the duodenum and proximal jejunum apparently favours deposition of bile acids that tend to be radiolucent on plain abdominal radiographs. In contrast, ileal stones usually contain mineral salts that tend to precipitate in the more alkaline environment of the distal small intestine. These calculi tend to be laminated and more radiopaque; a variety of minerals such as calcium carbonate, calcium oxalate and, rarely, magnesium or even barium sulphate may be present (1,8,10). The possible role that dietary factors, medications such as antacids or investigative barium studies may play in stone development is not known.

REFERENCES