# Meckel's enterolith : a rare cause of mechanical small bowel subobstruction

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# Abstract

Meckel's diverticulum is the most common congenital gastrointestinal malformation and may present with bleeding, obstruction and diverticulitis. Symptomatic Meckel's diverticulum is associated with age < 50 years, male gender, diverticular length > 2 cm and ectopic mucosa. Formation of enteroliths is a rare complication of Meckel's diverticulum and the majority of stones will remain in the diverticulum. Factors promoting enterolith formation through precipitation of calcium in the small intestinal alkaline environment include stasis as well as diverticular anatomy and histology. Mechanical obstruction due to liberation of enteroliths is even more rare and other mechanisms include intussusception, adhesions, volvulus and neoplasms. Visualization of enteroliths on plain abdominal films is challenging because not all stones are radiopaque. Surgical diverticulectomy or segmental bowel resection with anastomosis is preferred in case of complications. We present a case of mechanical small bowel subobstruction resulting from an expelled Meckel's enterolith. (Acta gastroenterol. belg., 2018, 81, 534-537).

Key words : Meckel's diverticulum ; enterolith ; small bowel obstruction.

**Abbreviations** : CRP, C-reactive protein ; CI, confidence interval ; CT, computed tomography ; GI, gastrointestinal ; OR, odds ratio ; WBC, white blood cell.

#### Introduction

Mechanical small bowel obstruction due to liberation of a Meckel's enterolith is a rare complication of Meckel's diverticulum and other mechanisms of obstruction complicating Meckel's diverticulum include intussusception, adhesions, volvulus and neoplasm. Visualization of enteroliths on plain abdominal films is challenging because not all stones are radiopaque. Differential diagnoses include appendicolith, biliary and urinary calculi, vascular or omental calcifications, leiomyoma and teratoma. We present a case of mechanical small bowel subobstruction resulting from an expelled Meckel's enterolith.

# **Case report**

A 42-year-old Caucasian man presented to the emergency department with colicky abdominal pain, which he had been experiencing intermittently for the past 10 days. He also reported nausea with bilious vomiting and denied previous biliary colic or (abdominal) surgery. His medical history was unremarkable except for Salmonella enteritis with normal ileocolonoscopy but

hemorrhagic colitis with crypt abscesses on pathology. His last bowel movements the previous day were normal and he was recently admitted for similar abdominal complaints with an episode of non-bloody diarrhea occurring 48h after a barbecue. He was treated with intravenous fluids, analgesia and peroral Azithromycin 500mg O.D. for suspected Campylobacter enteritis. However, stool cultures, Shiga toxin and parasite examination with stool antigen tests were negative and he was discharged home on the second day. His wife also suffered from gastrointestinal (GI) symptoms and both returned from holidays in Mallorca 3 weeks ago. Upon current presentation, vital signs, cardiac and pulmonary examinations were normal. There was mild abdominal distention and tenderness with no evidence of peritoneal signs, hernias or masses. Laboratory testing revealed an increased C-reactive protein (CRP) level at 119 mg/L (normal range: 0-5 mg/L) with normal total white blood cell (WBC) count. Creatinine, urea and electrolytes and liver function tests were within normal limits. Plain abdominal X-rays showed dilated small bowel loops with air fluid levels. Abdominal computed tomography (CT)-scan with intravenous contrast confirmed small bowel sub-obstruction due to a well-defined intraluminal nodular structure (35 mm in diameter) with a central high-density component located proximal to the caliber change in the ileum, suggestive of an impacted food bolus (Figure 1A). There was no evidence of pneumobilia, appendicitis or terminal ileitis. Following surgical consult, conservative treatment was proposed with intravenous broad-spectrum antibiotics (Ciproxine 400 mg B.I.D. and Metronidazole 500 mg T.I.D.), gastric decompression and nil by mouth. Serial radiographic abdominal examination was performed with persistence of air-fluid levels and progressive clinical deterioration with development of localized peritonitis. Laparoscopy was performed, revealing a Meckel's diverticulum located 50 cm proximally from the ileocecal valve, which was dissected from the mesentery and transsected at the base with the use of a gastrointestinal anastomosis (GIA<sup>TM</sup>) stapling device (Figure 2A). Histology confirmed a

CASE REPORT

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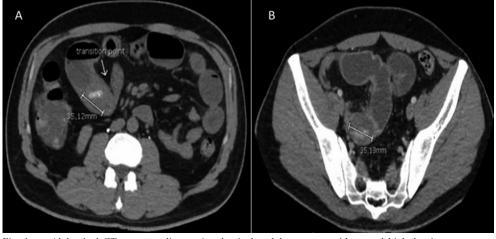


Fig. 1. — Abdominal CT-scan revealing an intraluminal nodular process with central high-density component proximal to the transition point in the ileum (A), displaced more distally after resection of a Meckel's diverticulum when using the stapling line as reference (B).

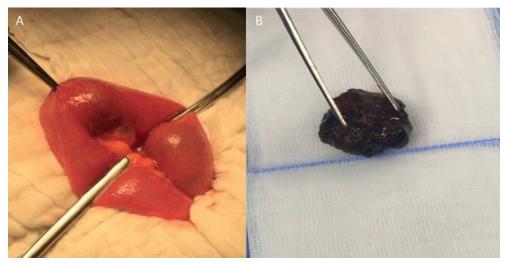


Fig. 2. — Surgically removed Meckel's diverticulum (A) and enterolith (B).

Meckel's diverticulum measuring  $6.0 \ge 4.5 \ge 1.5$  cm with intestinal-type mucosa and no evidence of malignancy.

The initial postoperative course was uneventful. However, on the second post-operative day, he experienced recurrent abdominal pain and vomiting, with clinical suspicion and radiological confirmation of small bowel obstruction. Control abdominal CT-scan revealed the round structure as previously described that was displaced more distally and proximal to the transition point. On laparotomy, multiple loops of distended ileum surrounded by non-purulent fluid were seen and an impacted enterolith was removed by longitudinal enterotomy with primary anastomosis (Figure 2B). Biochemical analysis showed a composition of 60% cholesterol, 30% bilirubin and some calcite. A diagnosis of small bowel obstruction secondary to an expelled Meckel's enterolith was made. The further postoperative course was uneventful and the patient was discharged home on the third post-operative day with no further episodes of abdominal pain.

#### Discussion

Meckel's diverticulum is the most common congenital GI malformation with a reported prevalence of 2-4% (1). It is a remnant of the vitelline or omphalomesenteric duct and is located on the antimesenteric border of the ileum, located 60 cm from the ileocecal valve (2). The 'rule of 2's' (prevalence of 2% of the general population, male-to-female ratio of 2:1, generally found before the age of 2, containing two types of ectopic tissue (i.e. gastric and pancreatic), located 2 feet (60 cm) from the ileocecal valve and measuring 2 inches (5 cm) in length) is commonly quoted despite inaccurate data (2). The lifetime complication risk was estimated at 6.4% (3), whereas Park et al. reported 16% of operatively found Meckel's diverticula were symptomatic with bleeding, obstruction and diverticulitis as the most common presentations (4). Mechanisms of obstruction related to Meckel's diverticulum include intussusception, adhesions, volvulus, neoplasm and the formation of

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enteroliths. Variables associated with a symptomatic Meckel's diverticulum in the Mayo cohort were age < 50 years (OR 3.5 (95% CI 2.6-4.8), P< .001), male sex (OR 1.8 (95% CI 1.3-2.4), P< .001), diverticular length > 2 cm (OR 2.2 (95% CI 1.1-4.4), P= .02) and histologic evidence of ectopic tissue (OR 13.9 (95% CI, 9.9-19.6), P < .001) (4). Our patient had several risk factors for symptomatic Meckel's diverticulum (< 50 years old, male and a diverticular length of > 2 cm) and presented with obstruction secondary to an expelled Meckel's enterolith. Ectopic tissue is mostly associated with complications of bleeding, diverticulitis and perforation (1,2). However, heterotopic gastric mucosa and the resulting acidic environment prevent enterolith formation, usually resulting from precipitation of calcium in the small intestinal alkaline environment in combination with stasis through strictures (in case of inflammatory bowel disease), chronic infection, scarring or blind pouches after surgery (5). The wide diverticular neck and peristaltic activity of a true diverticulum, containing all layers of the small intestine, also prevent enterolith formation in the majority of Meckel's diverticula (6). The presence of intestinal tissue, the small diverticular neck and possible scarring from previous enteritis in our patient were the most important factors in enterolith formation in the absence of inflammatory bowel disease or abdominal surgery.

Small bowel obstruction secondary to an expelled Meckel's enterolith is even more rare as most of the formed enteroliths will remain in the diverticulum (5,7). A prevalence of 6% in symptomatic and 0.7% in asymptomatic surgical patients was found (4), although it may be as high as 10% (6). In addition, Mirizzi-type compression and small bowel obstruction due to localized scarring of Meckel's enteroliths have also been reported (8,9). Radiographical demonstration is challenging because only one-third of enteroliths is radiopaque, although visualization on plain abdominal films was possible in 88% in one study (6) A mobile mass while repositioning the patient and peripheral calcifications with a radiolucent center in the context of small bowel obstruction are suggestive or enteroliths (5,7). CTappearance of an obstructing enterolith is described as a nodular structure with air-fluid levels (10). Differential diagnosis include appendicolith, biliary and urinary calculi, vascular or omental calcifications, calcified lymph nodes or fibroids, leiomyoma and teratoma (11). The visualization of a central high-density component in the present case contrasts with the peripheral calcification and radiolucent centers described in the literature, which may explain the initial suspicion of an impacted food bolus. However, laminated stones are also possible. Manipulation of the Meckel's diverticulum during initial laparoscopy may have resulted in further displacement of the enterolith with subsequent small bowel obstruction. This was also evident on the control CT-scan after Meckel's diverticulum resection, when using the stapling line as reference. Because the stone contained some calcite, it was formed in the distal small intestine as proximal stones are entirely calcium free due to the acidic environment preventing precipitation of calcium (11).

Treatment options differ depending on the cause of obstruction and include milking the enterolith distally into the cecum without incising the bowel (but with risk of perforation) or proximally into less edematous small bowel with removal through enterotomy. Resection of the Meckel's diverticulum is indicated to prevent complications and segmental bowel resection with primary anastomosis is often performed in cases of inflammation, perforation and/or necrotic bowel (5,7,13). Prophylactic excision of incidentally found asymptomatic Meckel's diverticulum has also been proposed with the presence of risk factors for symptomatic diverticula (4), which was also performed in our patient at the time of laparoscopy. Because of displacement of the enterolith causing small bowel obstruction, laparotomy was finally performed with removal of the enterolith by longitudinal enterotomy with primary anastomosis.

### Conclusion

In conclusion, mechanical small bowel obstruction due to an expelled enterolith is a very rare complication of Meckel's diverticulum. Symptomatic Meckel's diverticulum is more common with age < 50 years, male gender, diverticular length > 2 cm and ectopic mucosa. In the absence of ectopic mucosa, complications of bleeding and inflammation are less common than obstruction. Preoperative diagnosis is challenging when calculi are not radio-opaque and a characteristic CT-appearance has been described. Differential diagnosis includes appendicolith, biliary and urinary calculi, vascular or omental calcifications, leiomyoma and teratoma. Surgical resection is the preferred by diverticulectomy or segmental bowel resection with primary anastomosis in case of complications.

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