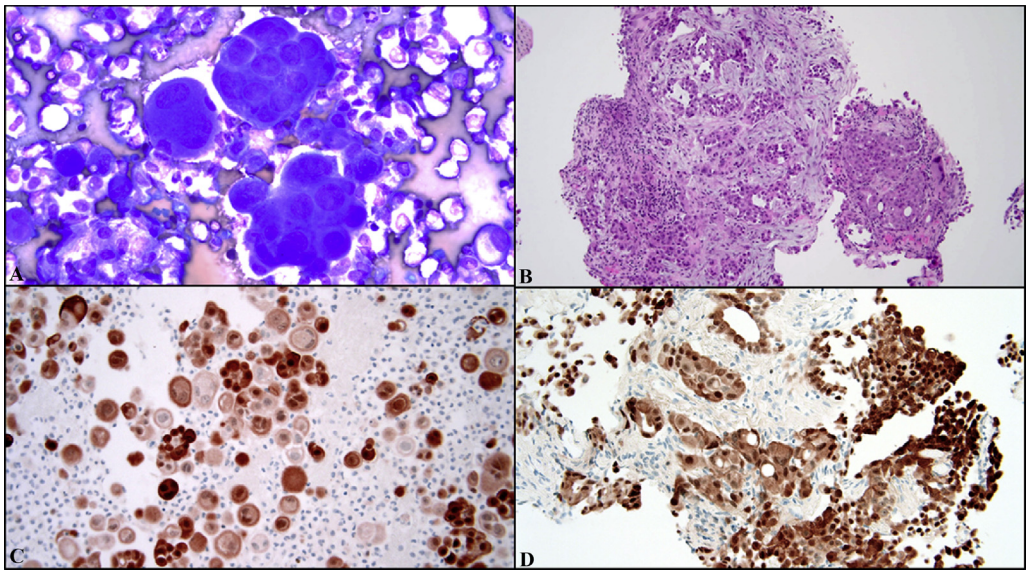


**Fig. 2** Ascitic fluid contained malignant cells forming large morulae (A). Peritoneal biopsy showed a 'gland-forming' large cell malignancy (B). Positive labelling of tumour cells with calretinin was seen in both (C) ascitic fluid and (D) peritoneal biopsy.

site. Whenever there is any doubt, a panel of immunohistochemical markers should be utilised, with at least two mesothelial markers and two carcinoma markers.[8] In addition, in female patients with radiological findings of ascites, omental and peritoneal thickenings, primary peritoneal serous carcinoma should also be included in the differential diagnosis.[7]

In some instances, peritoneal mesothelioma is a result of transdiaphragmatic spread of primary pleural malignant mesothelioma,[9,10] thus the diagnosis of primary peritoneal malignant mesothelioma should be established with a multidisciplinary approach involving the clinical, radiological and pathological input.

To the best of our knowledge, this is the first reported case of such a presentation with tissue diagnosis from both primary and metastatic sites. Only one case of metastatic diffuse peritoneal mesothelioma detected on endoscopic biopsy of a localised gastric antral mass is documented in the English literature, but in this case there was no tissue diagnosis of the primary site (peritoneum) and the diagnosis of peritoneal mesothelioma was established on a CT scan of the abdomen following the initial gastric biopsy result.[9]

The treatment of peritoneal mesothelioma differs depending on the disease extent. Chemotherapy alone is considered as a palliative treatment for patients not eligible for radical surgery. Cytoreductive surgery and perioperative intraperitoneal chemotherapy has been introduced as a curative treatment option over the last decade with an overall 5-year survival rate of 29–63%.[11]

**Conflicts of interest and sources of funding:** The authors state that there are no conflicts of interest to disclose.

**Yanling Yang**
**Dimuth Gunawardane**
**David Moffat**
**Christopher Carter**

*SA Pathology, Adelaide, SA, Australia*

Contact Dr Yanling Yang.
E-mail: cindy.yl.yang@gmail.com

1. Rodríguez D, Cheung MC, Housri N, *et al*. Malignant abdominal mesothelioma: defining the role of surgery. *J Surg Oncol* 2009; 99: 51–7.
2. Frontaria SC, Loveitt A, Goldenberg-Sandau A, *et al*. Primary peritoneal mesothelioma resulting in small bowel obstruction: a case report and review of literature. *Am J Case Rep* 2015; 16: 496–500.
3. Taylor S, Carpentieri D, Williams J, *et al*. Malignant peritoneal mesothelioma in an adolescent male with BAP1 deletion. *J Pediatr Hematol Oncol* 2015; 37: e323–7.
4. Sethna K, Sugarbaker PH. Localized visceral invasion of peritoneal mesothelioma causing intestinal obstruction: a new clinical presentation. *Hepatogastroenterology* 2005; 52: 1087–9.
5. Kindler HL. Peritoneal mesothelioma: the site of origin matters. *Am Soc Clin Oncol Educ Book* 2013: 182–8.
6. Kurimoto R, Kishimoto T, Nagai Y, *et al*. Malignant peritoneal mesothelioma: quantitative analysis of asbestos burden. *Pathol Int* 2009; 59: 823–7.
7. Pickhardt PJ, Bhalla S. Primary neoplasms of peritoneal and subperitoneal origin: CT findings. *Radiographics* 2005; 25: 983–95.
8. Hyuan TS, Barnes M, Tabatabai AL. The diagnostic utility of D2-40, calretinin, CK5/6, desmin and MOC-31 in the differentiation of mesothelioma from adenocarcinoma in pleural effusion cytology. *Acta Cytol* 2012; 56: 527–32.
9. Daniele L, Righi A, Ferraris A, *et al*. Metastasis of malignant peritoneal epithelioid mesothelioma in endoscopic gastric biopsy: a diagnostic pitfall. *Int J Surg Pathol* 2011; 19: 217–9.
10. Gutzeit A, Reischauer C, Hergan K, *et al*. Secondary malignant peritoneal mesothelioma of the greater omentum after therapy for primary pleural mesothelioma. *Case Rep Oncol* 2013; 6: 236–41.
11. Mirarabshahii P, Pillai K, Chua TC, *et al*. Diffuse malignant peritoneal mesothelioma—an update on treatment. *Cancer Treat Rev* 2012; 38: 605–12.

DOI: http://dx.doi.org/10.1016/j.pathol.2016.11.012

## Enteroliths and multiple neuroendocrine tumours in a Meckel's diverticulum

Sir,
Meckel's diverticulum is a true diverticulum, which arises from the anti-mesenteric border of the ileum. It occurs when

the proximal part of the omphalomesenteric (vitelline) duct fails to completely obliterate by weeks 7−10 of gestation.[1,2] The diverticulum may retain its connection to the umbilicus via a fibrous cord, or may be associated with other anomalies of the duct including fistulas, sinuses and cysts.[1] It is the most common congenital abnormality of the gastrointestinal tract and was identified by Fabricus Hildanus in 1598. The German anatomist Johann Friedrich Meckel first published a detailed description in 1809.[3]

The anomaly is most often found incidentally during laparotomies or autopsies. Autopsy series have found an incidence between 0.14% and 4.5%. The average length is 3 cm and 90% of cases are 1−10 cm in length. The longest recorded is 100 cm.[2] Distance from the ileocaecal valve varies with age, ranging from an average of 34 cm in infants to 67 cm in adults.[4] It is often, but not always, discovered in childhood and the male to female ratio is 1:1.[2] Therefore, the often quoted 'rule of 2s' (incidence 2%, length 2 inches, distance from ileocaecal valve 2 feet, age at diagnosis usually <2 years of age, male to female ratio 2:1) does not accurately represent the evidence.

Meckel's diverticula are lined by ileal mucosa. Heterotopic tissue is found in approximately 50% of cases, usually consisting of gastric (60−85%) or pancreatic mucosa (5−16%).[5] Rarely, colonic, duodenal, or hepatobiliary mucosa are found.[5] Heterotopic tissues which produce strong acids (gastric mucosa) or alkalis (pancreatic mucosa) may cause ulceration and bleeding.[5]

Complications occur in approximately 4−6% of cases, with a male to female ratio of 1.8−3:1.[5] There is still dispute as to whether the rate of complications decreases with age.[5] For all ages combined, complications in order of frequency are obstruction, diverticulitis, haemorrhage, perforation, neoplasm and fistula.[3,4]

Meckel's diverticula have been perforated by a range of objects, including fish bones, gallstones, marbles and toothpicks.[2]

Symptomatic cases present with unexplained abdominal pain, nausea and vomiting, or intestinal bleeding. The broad differential diagnosis includes appendicitis, cholecystitis, peptic ulcer disease, Crohn's disease, acute mesenteric lymphadenitis, angiodysplasia, gastroenteritis, food allergy or intolerance, and colonic or ileal diverticulitis.[1,5] The non-specific presentation and multiple common causes of the clinical features often results in a Meckel's diverticulum going unsuspected.

An 82-year-old man presented to his local emergency department with abdominal pain, severe vomiting and confusion. His medical history included early dementia, hypertension, viral myocarditis complicated by post-viral cardiomyopathy, and insertion of a permanent pacemaker for atrial flutter-associated bradycardia. There were no gastrointestinal diagnoses.

Examination findings included signs of dehydration, mild abdominal distension and generalised abdominal tenderness. Bowel obstruction was suspected.

Venous blood showed lactate 3.9 (0.5−2.0 mmol/L), urea 11.6 (3.1−8.1 mmol/L), creatinine 123 (60−110 μmol/L), protein 81 (60−80 g/L), albumin 47 (32−46 g/L), CRP 27 (<5 mg/L), WCC 14.9 (4−11×$10^9$/L), neutrophils 13.2 (2.0−8.0×$10^9$/L). The patient was not known to have chronic renal failure and the findings were consistent with dehydration and acute inflammation.

A supine abdominal X-ray showed multiple calcifications in the right upper quadrant, reported as 'most likely gallstones'. The bowel gas pattern was non-specific and there was no free intraperitoneal gas.

Computed tomography (CT) scan of the abdomen and pelvis showed a small bowel obstruction, with a transition point in the right iliac fossa. The favoured aetiology was adhesions. There was also calcification associated with what appeared to be a blind ending loop of small bowel, most likely a Meckel's diverticulum, which did not appear to be the cause of the obstruction. There was intraperitoneal free fluid adjacent to the liver and in the right iliac fossa. There was no free gas. The appendix, gallbladder and pancreas appeared normal.

Surgery revealed straw-coloured ascites and dilated small bowel to the distal ileum, with haemorrhagic patches on the serosa but no overt ischaemia changes. The transition point appeared to be the Meckel's diverticulum. The tip of the diverticulum felt hard and the surgeons suspected a tumour. An end-to-end anastomosis was performed.

Macroscopic examination revealed a broad-based Meckel's diverticulum. A fibrous band tethered the segments of ileum on either side of the diverticulum, forming a horseshoe shape. The tip of the diverticulum contained hard, mobile masses. Opening the diverticulum revealed a lumen with a pinpoint diameter at the neck and dilatation at the tip. Within the dilated tip there were three enteroliths and sludge. Two of the enteroliths were triangular and flat, the third was ovoid. The external surfaces of all enteroliths were hard and dark green with brown areas (Fig. 1). After decalcification, cutting demonstrated a hard, thin shell around a softer, friable brown core with an odour of faeces. On opposing sides of the tip, on the serosa, there were two firm, pale areas. The cut surfaces of the wall at these sites revealed two separate tumours, both well-circumscribed, solid and tan-coloured (Fig. 2A).

Histology of one enterolith showed a core of vegetable matter and debris surrounded by a thin shell of calcified material. Fouchet's stain proved that both the shell and contents contained bile (Fig. 3). Chemical analysis of a second enterolith found calcium oxalate. Histology of the tumours revealed two separate low grade neuroendocrine tumours (carcinoids), both arising in heterotopic non-oxyntic gastric mucosa (Fig. 2B).

The patient had post-operative complications unrelated to the pathology in the diverticulum or the anastomosis, before full recovery and discharge home. There was no clinical or radiological evidence of metastatic disease.

Symptomatic Meckel's diverticula are rare in people >80 years. From 1950−2002 the Mayo Clinic recorded only 13 cases in this age group and the oldest person who required an operation for a symptomatic Meckel's diverticulum was 91 years.[3]

Asymptomatic Meckel's diverticula discovered incidentally by imaging are not resected. In asymptomatic paediatric patients and young adults, Meckel's diverticula found incidentally during surgery are usually resected. In the case of adults, resection of Meckel's diverticula found incidentally during surgery remains a matter of debate. Older age and comorbidities increase the risks of surgery and must be