Congenital Peritoneal Encapsulation in A Pregnant Woman: A Case Report

Abstract

Congenital peritoneal encapsulation (CPE) is a very rare congenital malformation of the gastro-intestinal tract which is characterised by the presence of an accessory peritoneal membrane in which the small bowel is contained and communicates with the rest of the peritoneal cavity by means of a small opening. We report a 26-year-old primigravida who presented with an acute onset abdominal pain and was found at laparotomy to have complications resulting from CPE. The embryological basis, clinical and pathological features of the disease are also considered. Knowledge of this condition will help guide the surgeon in making prompt decision when confronted with it.

Keywords: *Accessory, congenital peritoneal encapsulation, peritoneal membrane*

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Introduction

Congenital peritoneal encapsulation (CPE) is a very rare congenital malformation of the gastro-intestinal tract.[1] It has been described in less than 50 case reports since its first description by Cleland.[1,2] This condition is characterised by the presence of an accessory peritoneal membrane, in which the small bowel is contained. It is bounded by the omentum and meso-colon, and communicates with the rest of the peritoneal cavity by means of a small opening.[1]

We report on a 26-year-old primigravida without prior abdominal symptoms who presented with an acute onset abdominal pain at a public referral hospital. During laparotomy, gangrenous small bowel loops were identified extruding from an opening in a peritoneal sac consistent with CPE.

Case Report

Our patient is a 26-year-old primigravida at estimated gestational age of 28 weeks who was referred to us from a peripheral centre on account of intraoperative findings of gangrenous bowel.

She was well until 2 days prior to presentation when she developed sudden onset of sharp supra-umbilical pain. She had associated rapidly progressive abdominal distension, constipation and 2 episodes of nonbilious vomiting.

She neither had haematemesis nor melaena.

She also has no fever, discharge from the vagina, nor urinary symptoms.

She did not have any abdominal surgery in the past; neither does she have any anterior abdominal wall or groin protrusion, prior change in bowel habit nor any history of abdominal trauma.

Her pregnancy was booked at the referring centre where she had been regular with her antenatal care. Pregnancy had not been adversely eventful.

Following her symptoms, she presented at the referring centre where she had surgery done, but was referred to us based on the intraoperative finding of gangrenous bowel and unavailability of expertise at the centre.

On examination, she was acutely ill-looking, pale, anicteric, afebrile, not dehydrated, with no pedal oedema.

Her pulse was 160 beats/min, blood pressure was 97/61 mmHg, and respiratory rate was 24 cycles/min. Her oxygen saturation was 95% on room air.

Her chest was clinically clear with vesicular breath sounds.

Her abdomen was distended. She had a mid-line laparotomy wound tagged with nylon sutures with no significant discharge. There was generalised tenderness which was

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worse in the left hypochondrial, left lumbar and umbilical regions. Bowel sounds were absent.

Gynaecological/obstetric examination revealed normal vulva and vagina, posterior cervix which was firm, 3 cm long with closed internal os.

Foetal heart was not heard using sonicaid.

Digital rectal examination revealed normal findings.

We made the following diagnoses:

1. Acute abdomen secondary to? Gangrenous bowel, and
2. Intra-uterine foetal death.

She was commenced on resuscitation: nasogastric tube was passed which drained bilious effluent, urethral catheter was passed for monitoring of urine output. She was placed on nil by mouth (nothing to be ingested), intravenous fluids, antibiotics and analgesics.

The initial investigations done include:

Full blood count—packed cell volume 29%, haemoglobin concentration 8.7 g/dL, white blood cells 44.1×10^9/L (neutrophils 74%, lymphocyte 20%, monocytes 6%), platelet 286×10^9/L. Blood film showed nucleated red blood cells and leucocytosis with left shift.

Electrolytes, urea and creatinine—Na—139 mmol/L, K—5.2 mmol/L, urea—4.1 mmol/L, creatinine—216 µmol/L.

Obstetric ultrasound scan confirmed intra-uterine foetal death.

Following 36 h of resuscitation (including transfusion of 2 units of whole blood), her parameters were as follows:

Pulse—130 beats/min, blood pressure—121/74 mmHg, respiratory rate—28 cycles/min, oxygen saturation—94% (on room air), 98% (on oxygen by facemask).

Repeat full blood count showed: packed cell volume 30%, haemoglobin concentration—9.9 g/dL, white blood cells—19.3×10^9/L (neutrophils 89%, lymphocyte 3%, monocytes 8%), platelet 142×10^9/L. Blood film showed leucocytosis and neutrophilia with left shift.

Serum electrolytes urea and creatinine—Na—138 mmol/L, Cl—105 mmol/L, K—5.3 mmol/L, urea—12.9 mmol/L, creatinine—319 µmol/L.

She was thereafter booked for exploratory laparotomy. However, at this time, she had commenced the process of labour, and she had a normal vaginal delivery of a female stillbirth. The third stage of labour was, however, complicated by retained placenta. She was subsequently wheeled into the operating room.

She had an exploratory laparotomy with findings of:

A peritoneal sheet attached to the mesenteric border of the ascending colon down to the sigmoid with a defect at the region of the terminal ileum, caecum and proximal ascending colon. The peritoneal sheet projected inferiorly to the retro uterine region.

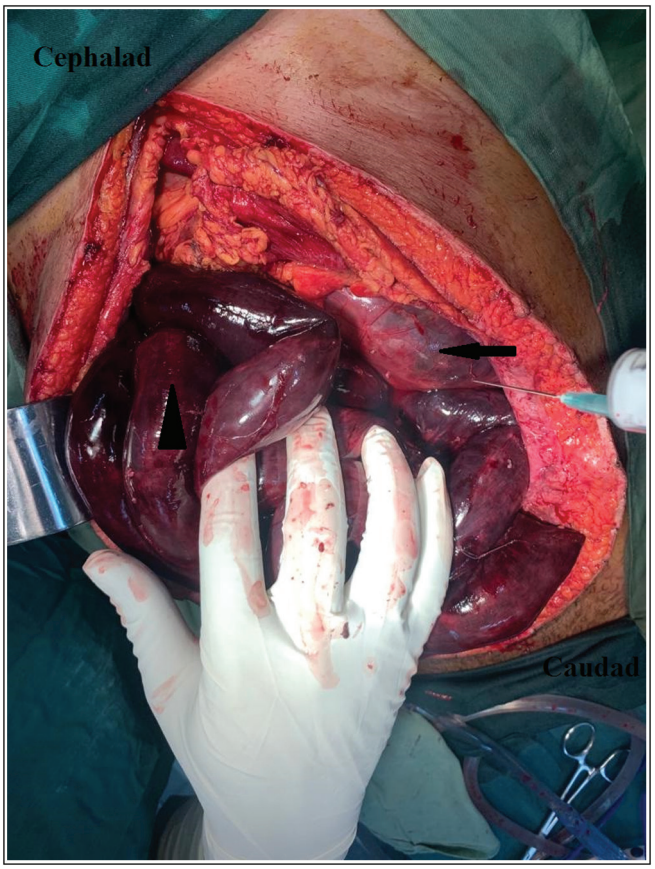

Figure 1: Arrow head-gangrenous bowel; arrow-aberrant peritoneal layer covering the rest of the bowel

There is herniation of a long segment of gangrenous small bowel through the peritoneal defect and also a 270º anticlockwise torsion of the herniating bowel on its mesentery [Figures 1 and 2]. Part of the non-herniating loops were also gangrenous due to the torsion.

The uterus was bulky and intra-abdominal [Figure 2]. Other intra-abdominal structures appeared normal.

The peritoneal sheet was opened widely, and she had the entire gangrenous segment resected with the remaining viable portion being the proximal 90 cm of the jejunum and distal 10 cm of the ileum. She had an end-to-end jejuno-ileal anastomosis done.

Attempt at manual evacuation of retained placenta under anaesthesia proved abortive.

Her wounds were closed over a passive peritoneal drain.

She was monitored in the intensive care unit till 5th post-operative day after which she was transferred to the open ward.

Post-operative challenges included frequent watery bilious stools which was controlled with loperamide and small frequent

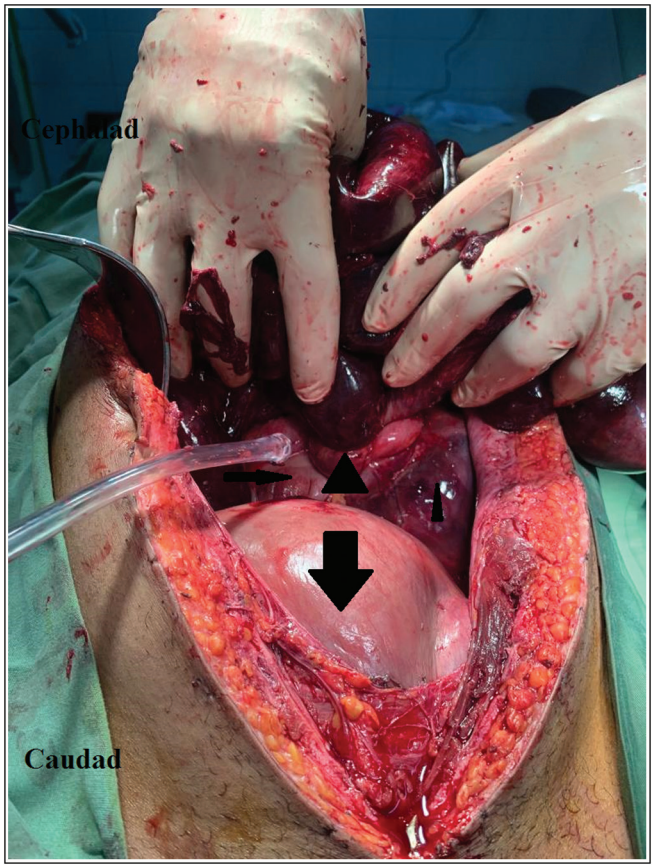

Figure 2: Small arrow-caecum; large arrow-uterus; small arrowhead-aberrant peritoneum covering the rest of the bowel; large arrowhead-tight opening through which small bowel herniated and got strangulated

meals. Her placenta separated on the 20th post-operative day, and she was discharged to be followed up in clinic.

Discussion

During foetal development, there is physiologic herniation of the mid-gut around the sixth week and reduction of the herniation at about the ninth week. It was postulated by Papez[3] that CPE develops as a result of adhesions occurring between the proximal portion of the herniated bowel and the edge of the hernial sac. On reduction of the herniated bowel back into the abdominal cavity, it pulls along with it the sac which is peeled off the umbilicus. This then forms the accessory peritoneal sac characteristic of CPE. The membrane is morphologically and histologically identical to peritoneum and it is lined by mesothelium.[4]

CPE is a very rare disease making its incidence and prevalence difficult to quantify. It has been reported in less than fifty times in the literature since the first case was reported in 1868.[1] Presentation occurs in all age groups ranging from 11 to 82 years.[5,6] According to Dave *et al*, females are diagnosed earlier, with a majority presenting prior to 30 years as is the case with our index patient. There has, however, not been any report of CPE in pregnancy as in this our patient. There has not been any report of CPE in pregnancy as in our patient. There is a male preponderance with male:female of 5:3.[1]

CPE is typically asymptomatic and incidentally detected during laparotomy performed for other indications or at autopsy.[1,7] Others, however, present with complications resulting from this rare condition, commonly features of strangulation or intestinal obstruction.[1,7] Most of the cases reported presented as a result of complications from this condition; hence, it is likely that this condition is more common than reported.

Another encapsulating bowel condition which is a close differential diagnosis of CPE is sclerosing encapsulating peritonitis (SEP) which can be idiopathic (abdominal cocoon) or secondary.[1,4] Secondary causes include local factors (e.g., peritoneal dialysis, abdominal trauma, abdominal surgery, peritoneal shunts, peritoneal tuberculosis, peritoneal foreign body and intra-peritoneal chemotherapy) and systemic factors (e.g., beta blocking agents, methotrexate, cirrhosis, systemic lupus erythematosus, malignancy and sarcoidosis).[1,4]

Morphologically, SEP appears as a thick, firm fibrotic membrane.[1] It is separate from the peritoneum, but may have significant adhesions to the peritoneum and other surrounding structures.[1] At surgery, a major difference between CPE and SEP is that, in CPE, the membranous layer can be separated easily from the underlying loops of bowel which appear normal and are not adherent to each other.[6] Histologically, SEP is characterised by dense connective tissue proliferation, chronic inflammatory cell infiltration and dilated lymphatics.[1,4]

Surgical management of CPE involves adhesiolysis and peritonectomy.[1,8] Adhesions at the neck of the sac must be carefully resected to ensure complete release of the bowel and prevent bowel obstruction and ischaemia post-operatively.[1] In our experience, we resected the whole extent of the gangrenous bowel along with the neck of the sac. We also opened the residual sac widely without completely excising it.

Prognosis following prompt surgical management of CPE is excellent.[1] Morbidity and mortality are however expectedly increased in cases presenting with extensive bowel gangrene and/or bowel perforation. In our patient, there were features of short bowel syndrome in the early post-operative period likely due to the extent of bowel resection. These, however, significantly resolved with medical therapy.

In conclusion, it is important for the abdominal surgeon to know about this very rare congenital condition as it is not routinely reported in conventional anatomical texts. This will help guide prompt decision making when confronted with this condition in the elective or emergency setting either with the open or minimal access approach.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and

other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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