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Case Report

Congenital Peritoneal Encapsulation with Complications: a Rare Case Report

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Abstract:

Congenital peritoneal encapsulation is an exceptionally rare congenital anomaly characterized by the existence of an accessory peritoneal membrane that encapsulates a variable portion of the small intestine. The pathogenesis remains largely elusive; however, it is hypothesized to result from abnormal adhesions within the peritoneal lining during the physiological hernia phase of fetal mid-gut development. The phenomenon was first documented in 1868, and since then, only 45 cases have been reported in the medical literature. The condition usually remains asymptomatic and is an incidental diagnosis during surgery or autopsy. We report a case of congenital peritoneal encapsulation in a 30-years-old male who was diagnosed per operatively during laparotomy for perforation of caecum. Clinical examination revealed features of perforation, plain X-ray abdomen didn't show free gas shadow under right dome of diaphragm and ultrasonography of the whole abdomen revealed mild free fluid in the peritoneal cavity with matted gut loops. On exploration, a large sac-like structure was found which encased the distal part of the jejunum, whole ileum, perforated caecum and part of the ascending colon. Affected portion of caecum was resected and an end ileostomy was done.

Key words: Congenital peritoneal encapsulation, Perforation.

Introduction:

Congenital peritoneal encapsulation (CPE) is an exceedingly rare condition and was first reported by Cleland in 1868 as a "congenital anomaly that occurs secondary to an abnormal return of abdominal wall contents during the 12th week of gestation"[1]. We reported a case demonstrating a thin membrane encasing the small bowel forming an accessory peritoneal sac. These cases usually present with small bowel obstruction or can be an incidental finding during laparotomy. Our case is diagnosed during laparotomy for caecal perforation.

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Case Report:

A 30-years-old male presented to emergency department of Faridpur Medical College Hospital (FMCH) with severe pain in the right lower abdomen with vomiting, fever and abdominal distension for 1 day. Pain was of gradual in onset, moderate to severe, colicky in nature, aggravated by coughing, and was slightly relieved by lying still. For the last 1 day, the patient developed abdominal distension.

Physical examination revealed muscle guarding all over the abdomen with board like rigidity, mild anaemia with raised temperature.

Plain X-ray abdomen in erect posture revealed no free intraperitoneal gas. Ultrasonography of the whole abdomen revealed mild free fluid in the peritoneal cavity with matted gut loops along the right iliac fossa. With the clinical diagnosis of perforation of gas-containing hollow viscus he underwent emergency exploratory laparotomy under general anesthesia after adequate resuscitation. After opening the abdominal cavity, a large sac-like structure was found that occupied almost entire abdominal cavity.

Figure-1: Exteriorized peritoneal sac.

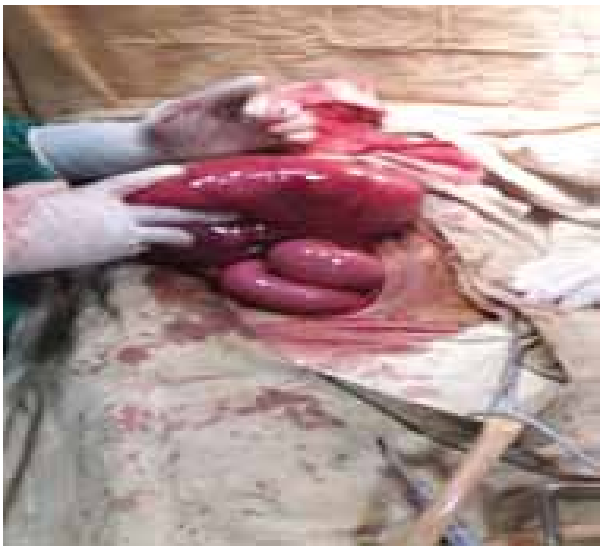

Figure-2: The entry point of guts into the sac.

Figure-3: Perforated and gangrenous caecum.

It had the appearance of a supplementary peritoneal capsule through which the 'trapped' small bowel loops were visible. After extending the incision upwards, the whole sac was exteriorized, and an entry point or opening was found on the undersurface of the sac. Through that point, the gut entered the sac. The whole ileum and distal part of the jejunum with the caecum and part of the ascending colon were gently pulled out of the sac through the opening.

The sac was then identified clearly, which aroused from the side of the root of the mesentery and encroaching over the D-J junction above, on the mesentery, and over the ascending colon below. Appendix was healthy, Caecum was gangrenous and perforated. Ileocaecal artery was found thrombosed and this may be the cause of caecal perforation secondary to ischemic necrosis. There was no lymphadenopathy. Unhealthy portion of caecum was resected. The peritoneal sac was excised. The ileum was resected near the terminal ileum, and end ileostomy was done.

Discussion:

Congenital peritoneal encapsulation was first described in 1868 by Cleland1. There are 45 case reports described in the literature[2]. Sclerosing encapsulated peritonitis (SEP) and abdominal cocoon (AC) are other rare entities causing small bowel encapsulation[3]. These are all separate pathologies: CPE is an embryological malformation, while AC is idiopathic, and SEP is predominantly associated with peritoneal dialysis, local irritation, peritoneal shunts, peritoneal tuberculosis, intraperitoneal chemotherapy, etc[4]. Recently, Dave et al.[2] have proposed a classification system for various encapsulated bowel diseases based on their etiology. Notably, CPE should be considered in the differential diagnosis alongside fibrotic peritoneal encapsulation (FPE), which encompasses idiopathic cocoon syndrome and SEP attributed to well-defined inflammatory triggers. The designation of FPE has been introduced to delineate CPE from acquired forms of encapsulated bowel disease. Additionally, FPE is characterized by a thick fibro-collagenous layer encasing the small and large bowel, and thus the associated membrane differs for morphology and histology from the peritoneal sheet encountered in the CPE. In fact, the membrane present in CPE is typically thin, semi-transparent, and vascularized, differing consistently from the thick, white, and fibrotic membrane typical of FPE.

CPE is a developmental abnormality encasing part or the entire small bowel in an accessory sac derived from the yolk sac. The accessory peritoneal membrane is derived from the peritoneum of the yolk sac at 12 weeks of gestational age, which withdraws into the abdominal cavity with the small bowel instead of remaining at the base of the umbilical cord. This is attached to the ascending and descending colon laterally, the transverse mesocolon superiorly and merges with posterior parietal peritoneum inferiorly[5]. The membrane has two openings, one around the duodeno-jejunal flexure and the other at the Ileocaecal junction[3].

CPE seems to be predominant in males and the most frequent age range was between 10 and 29 years[2]. It is not always associated with abnormal intestinal rotation[6].

The two most common clinical presentations are acute small bowel obstruction or incidental diagnosis during laparotomy for another condition; however, many cases are incidental findings at autopsy. Some patients may have episodes of intermittent colicky abdominal pain or episodes of sub-acute small bowel obstruction, before a definitive diagnosis[7]. Only 3% cases of small bowel obstruction are thought to be caused by congenital bands formed by abnormal adhesions of the peritoneal folds during embryogenesis[8].

Rarely CPE can lead to idiopathic sclerosing peritonitis[9]. It can occur with incomplete situs inversus and abnormal artery (both are congenital anomalies) or as an incidental finding at the time of gastric surgery or during colon cancer surgery[10].

Concerning the physical examination, a patient with intestinal obstruction caused by peritoneal encapsulation presents some clinical signs: asymmetrical and fixed abdominal distension, peristalsis without variation, and differences in consistency on abdominal palpation[11].

Because of the diagnostic difficulty based on clinical findings, imaging studies often play an important role in the diagnosis. Radiographic studies are usually normal or show nonspecific features of intestinal obstruction. Plain abdominal X-rays are suggestive of mechanical bowel obstruction. Ultrasonographic findings will guide towards the complications that arise from encapsulations, such as perforation in our case. On computed tomography (CT) scan, the findings are very nonspecific. The small bowel is seen enveloped in a thin membrane and may be suggestive of peritoneal encapsulation[12].

The treatment of peritoneal encapsulation typically necessitates the complete excision of the aberrant membrane and the release of both openings. In asymptomatic cases, surgical intervention is generally not indicated. However, bowel resection may become essential if the membrane cannot be removed atraumatically or if there is evident gangrene of the affected bowel segment. There is no reoperation reported in the literature after dividing the encasing membrane in peritoneal encapsulation[12].

Conflict of Interest:

Authors declare no conflict of interest.

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