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# Adhesive mesenteric lymphadenitis due to pancreatic necrosis a cause of Intestinal obstruction

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**Abstract**
Pancreatic necrosis is the presence of nonviable pancreatic parenchyma or peripancreatic fat, it can be manifested as focal area or diffuse involvement of gland. Up to 20% of patients with acute pancreatitis develop pancreatic necrosis. It is important to identify and proper treatment because most patients who develop multiorgan failure have necrotizing pancreatitis. Necrotizing pancreatitis associated with bacterial translocation usually involving enteric Flora such as gram negative rods e.g. *E. coli*, Klebsiella and pseudomonas and enterococcus. While an uncommon occurrence, it is possible for patients diagnosed with necrotizing pancreatitis to develop colonic ileus, obstruction or perforation. The definite treatment of infected pancreatic necrosis is surgical debridement with necrosectomy, closed continuous irrigation or open packaging.

**Keywords:** Pancreatic necrosis, peripancreatic fat, mesenteric lymphadenitis

## Introduction
Tuberculosis (TB) is a chronic granulomatous disease caused by Mycobacterium tuberculosis. The usual site of infection is the lung, but other organs may be involved.
Abdominal tuberculosis represents the sixth most frequent form of extra-pulmonary tuberculosis after lymphatic, genitourinary, bone and joint, miliary, and meningeal tuberculosis. Tuberculous bacteria reach the gastrointestinal tract via hematogenous spread, ingestion of infected sputum, or contiguous spread from adjacent organs. Almost all cases of abdominal TB are caused by Mycobacterium tuberculosis. The predilection of the bacillus for the ileocecum, is attributed mainly to three factors: Relative physiological stasis of the area, the high rate of absorption, with more complete digestion (permitting free contact of the organism with the mucosal lining), and the abundance of lymphoid tissue at this site. There are three gross morphological forms of tuberculous enteritis:
Ulcerative, hypertrophic, and ulcerohypertrophic. The ulcerative type, which commonly affects the ileum and jejunum, is characterized by a single or multiple transverse ulcers, the healing of which leads to stricture formation, and may perforate, bleed, or form fistulas. The hypertrophic and ulcerohypertrophic types commonly affect the ileocecum and cause obstruction or present as a mass. Grossly, peritoneal tubercles and enlarged, matted, caseous mesenteric lymph nodes may be seen.
Perforation is a serious complication of abdominal TB, associated with high morbidity and mortality. The low incidence of tuberculous perforation is due to reactive fibrosis of the peritoneum. However, in recent years, intestinal perforation, which was relatively rare in the past, has been reported more frequently. The cause of this remains unknown.
Vasculitis is a well-established feature of tuberculosis of the central nervous system (CNS), lungs, and kidneys, but scarcely described in intestinal tuberculosis. Few studies have evaluated the role of mesenteric vasculitis in the natural history of intestinal tuberculosis. Ischemic changes have been said to underlie perforation and stricture formation. However, an in-depth analysis into the cause of intestinal changes in abdominal tuberculosis and the association between macroscopic and microscopic features (i.e., perforation, ulceration, and stricture formation) with changes in mesenteric vasculature has not been done previously. This study attempts to elucidate the changes in mesenteric vessels and their association with perforation, and also document various gross morphological types of intestinal TB and their correlation with the microscopic features.

## Case report
A 30 yr. Old male patient presented with abdominal discomfort and abdominal distension for last 4 days. It was associated with complain of nausea and stool not passed since last 4 days.

Also history of vomiting before 3 days. There was no history of fever, jaundice, Melena, hemoptysis, hematemesis, dysuria, hematuria, hemoptysis, seizures or worm infestation.

On clinical examination vitals parameters were found within normal limit with no pallor, icterus, pedal edema, lymphadenopathy. Per abdomen examination revealed generalized tenderness and tense & distended abdomen. Per rectal examination revealed mild ballooning.

Laboratory tests shows hemoglobin 14.0, WBC count of 17,400. High RBS 383.His liver function tests- total proteins, total bilirubin in normal limit. Amylase 79.4 and S. lipase 32.80.

A chest radiograph showed no infiltrates in lungs. Ultrasound abdomen revealed an intestinal obstruction. X ray abdomen standing shows multiple air fluid level (4-5 in number) suggestive of intestinal obstruction.

Rules tube insertion and catheterization was done. Ryle's tube aspiration shows approx.800 cc gastric material in uro bag.

Therefore, patient was prepared for exploratory laparotomy. Patient underwent exploratory laparotomy, which revealed necrosed pancreas along with enlarged lymph node in mesentery and adhesion between loops of small bowel.

**Surgical technique**

The patient was treated surgically under general anesthesia and was placed in supine position. A vertical incision was kept in midline which revealed approx. 100-150 cc purulent peritoneal fluid grossly in the abdomen. The mesenteric border of jejunum had a single perforation of ~ 0.5*0.5 cm2 and a complete stricture was found distal to perforation with dilated proximal jejunal segment.

Peritoneal fluid was sent for cytology and resected specimen was sent for Histopathological Examination.

The post-operative period was uneventful. The histopathological report of the resected specimen that were sent intra operatively revealed acute supportive inflammation at the junction of mesentery (PANICULITIS) and the fluid cytology revealed Klebsiella. On receiving this report, the patient was started on anti-tubercular treatment a remained well on follow up. Efforts were made to detect a primary focus of tuberculosis, but were unsuccessful.

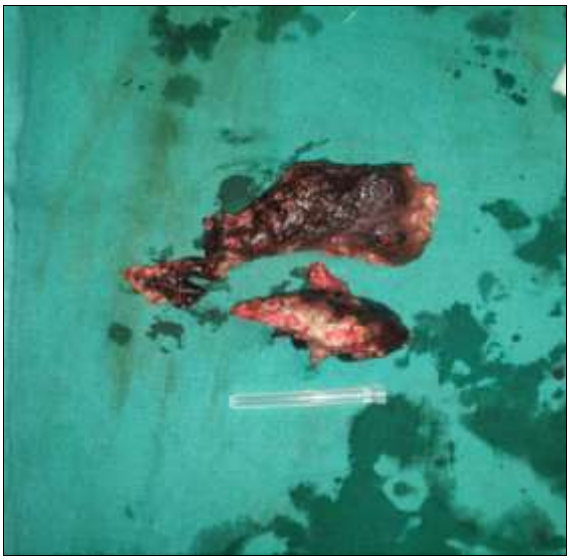

**Fig 1:** Shows in the patient was treated surgically under general anesthesia and was placed in a supine position.

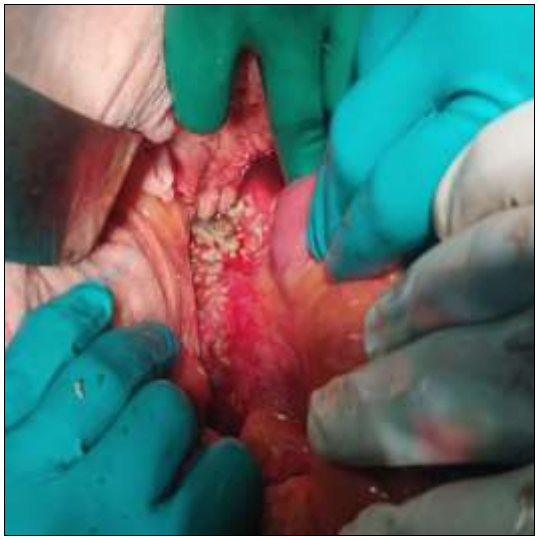

**Fig 2:** Acute adjuvant inflammation was detected at the junction of the mesentery (panniculitis) and fluid cytology revealed Klebsiella.

**Discussion**

Bowel complications of necrotizing pancreatitis such as paralytic ileus, ischemic necrosis, perforation and mechanical obstruction are relatively infrequent.

Mechanical bowel obstruction as a result of necrotizing pancreatitis has been described in the literature and is more likely to occur in the splenic flexure and transverse colon. This is believed due to:

(1) Severe inflammation of the body and tail of pancreas causing extrinsic compression.
(2) Retroperitoneal extravasation of pancreatic enzymes causing pericolitis.
(3) Thrombosis of mesenteric arteries (associated with hypercoagubility)
(4) Infarction/Ischemic necrosis of watershed area secondary to systemic hypotension.

Retroperitoneal inflammation may also lead to the involvement of other segments of bowel including small bowel as was the case with our patient.

Other obstructive manifestations of necrotizing pancreatitis include colonic stenosis which may present as a "pseudo carcinoma" with classic "apple-core" appearance on imaging a few months following an episode of necrotizing pancreatitis. This complication has been most frequently described at the splenic flexure and, like mechanical obstruction, may be the result of the intimate anatomic relationship shared between the pancreatic tail and splenic flexure. The pancreatic tail lies in the phrenolienal and phrenocolonic ligament, which is contiguous with the splenic flexure of the colon. This shared communication facilitates direct extension of inflammatory enzyme-rich material to the splenic flexure with progressive stricturing of the colon segment. Additionally, the splenic flexure is a watershed area often supplied by a poorly developed marginal artery making this area more vulnerable to ischemic insult.

Similar anatomic relationship exists between the anterior surface of the pancreas and transverse colon where the two layers of the transverse mesocolon cover the head and body of the pancreas. Thus, enzyme-rich inflammatory extravasation can easily access the transverse colon leading to local complications (including mechanical obstruction).

Furthermore, the peritoneal reflection from the anterior surface of the pancreas facilitates communication to the small bowel mesentery making the small bowel vulnerable to inflammatory complications.
Finally, colonic paralytic ileus is a relatively more common and less severe complication of necrotizing pancreatitis than true mechanical obstruction. The etiology of ileus is not entirely understood but may arise from a viscerally mediated reflex within the superior mesenteric plexus secondary to retroperitoneal inflammation and/or transient colonic ischemia.
Colonic obstruction secondary to necrotizing pancreatitis needs surgical intervention.

## Conclusion

Clinicians must be aware of mechanical small bowel obstruction as a complication of necrotizing pancreatitis which is rare, are potentially deadly and therefore should be managed aggressively.

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**Ethical approval:** The study was approved by the institutional Ethics Committee.

**Conflicts of interest:** None declared

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