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**Spontaneous perforation of the common bile duct in pregnancy**

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Spontaneous perforation of the bile duct is rare. Bile duct perforation occurring during pregnancy is even rarer. We report a 21-year-old primigravida with sealed bile duct perforation at 28 weeks of amenorrhoea. (Indian J Gastroenterol 2001;20:198-199)

**Key words:** Billary peritonitis

Spontaneous rupture of the common bile duct (CBD) is rare, only 40 cases having been reported earlier. It may result from increased intraductal pressure due to associated calculi, and necrosis of the bile duct wall secondary to thrombosis. Early diagnosis and aggressive therapy are mandatory to alleviate sepsis and achieve decompression of the bile duct.

A 21-year-old primigravida with 28 weeks' amenorrhoea presented with continuous upper abdomen pain since one day associated with 7-8 episodes of vomiting. The patient had no leaking or bleeding per vaginum and felt normal fetal movements. On examination her pulse rate was 124/min and blood pressure was 90/70 mmHg. Tenderness and guarding was noted in the upper abdomen. The uterus was 28 weeks' size by palpation. Fetal heart sounds were normal. Bowel sounds were normal on auscultation. Per rectum examination was normal.

Abdominal sonography showed mild hepatomegaly. Gall bladder was distended and normal; the CBD diameter was 5 mm. There was no free fluid in the abdomen. Uterus was normal with a live 28-week fetus. Hemoglobin was 11 g/dL, leucocyte count 8400/mm³; liver and renal profile and serum amylase were normal. Abdominal paracentesis in the right hypochondrium showed the presence of clear yellow bile-like fluid.

At laparotomy, there was 200 mL of clear yellow bile in the supracolic compartment along with bile staining of the CBD just below the opening of the cystic duct. The gall bladder was tense and distended. A large impacted lower-end CBD stone was palpated, which could not be removed at surgery. The rest of the abdomen was normal. A diagnosis of spontaneous sealed perforation of the CBD was made. In view of the general condition of the patient and risk of preterm labor, a decision to temporarily drain the CBD with a T-tube was taken. Postoperative T-tube cholangiogram showed complete cut-off of the lower-end CBD with a stone there. Two weeks later, ERCP was attempted for removal of the impacted CBD stone. However, it was unsuccessful since the stone was 3 cm in size. The patient was discharged with the T-tube drain in situ.

Two months later, at 36 weeks, the patient had a normal vaginal delivery. Three weeks post delivery she underwent cholecystectomy with CBD exploration and T-tube drainage. The T-tube was removed on the 14th day after T-tube had shown no residual CBD stones.

A wide variety of causative factors are reported to be responsible for spontaneous bile duct perforations. These include erosion of calculi through the CBD; thrombosis of a mural vessel, which may lead to ischemia in the duct wall; intraductal infection leading to weakness in the bile duct wall; infective diverticulum or mucous cyst; reflux of active pancreatic secretions. There is very little evidence to support any of these theories.

In pregnancy, the presence of gallstones at the weakest point of the cystic duct and hemodynamic changes associated with higher pressure in the vena cava could be the probable cause of bile duct perforation.

The clinical presentation may be insidious or acute. The former is more common and is characterized by progressive jaundice, painless abdominal distention, ascites, and clay-colored stools. The acute form presents with abdominal distension, fever, vomiting and signs of fulminant bile peritonitis.

Recommended treatment includes cholecystectomy and CBD exploration with T-tube drainage of the CBD for several weeks in cases with no ductal abnormality or Roux-en-Y ductal anastomosis if ductal disruption is severe. In high-risk patients simple external bile duct drainage helps to tide over the initial crisis, as was done successfully in our case. Endoscopic sphincterotomy can be done safely in pregnancy with fluoroscopic control (lead apron protecting the fetus) or without fluoroscopic control.

**References**

1. Piotrowski JJ, Van Stegmann G, Liechty RD. Spontaneous
Colonic wall necrosis due to tuberculosis in HIV-seropositive patient

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We report a 40-year-old man with sloughing off of the colonic due to tuberculous involvement with HIV infection. He presented with lump in the abdomen, distension and vomiting suggestive of intestinal obstruction. Proximal loop ileostomy with closure of colonic perforation was performed, with good recovery. This was followed by antitubercular chemotherapy.

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Key words: Intestinal obstruction

Colonic tuberculosis is an uncommon condition. We report an HIV-seropositive patient with colonic tuberculosis with sloughed-off wall of the ascending and transverse colon.

A 40-year-old man presented with distension of the central abdomen, colicky abdominal pain and vomiting for two months, which had increased 4-5 days prior to admission. He had been previously diagnosed to have pulmonary tuberculosis and was prescribed four drug anti-tuberculotics therapy, which he had taken irregularly for two months. Examination revealed pallor and tachycardia. Abdominal examination revealed distention of the central abdomen with palpable bowel loops and tenderness on deep palpation but no guarding or rigidity. X-ray revealed a few air-fluid levels in the abdomen but no free gas. X-ray chest showed right upper zone infiltration due to pulmonary tuberculosis. He was explored after correction of dehydration and electrolyte imbalance since obstruction was not relieved with conservative treatment.

At laparotomy, the anterior wall of the ascending and transverse colon was completely sloughed off, with thick adhesions between the posterior wall of the ascending colon and the posterior abdominal wall. Inseparable adhesions were also present between the transverse mesocolon and stomach. The colonic mucosa was edematous and grossly inflamed, extending from the ascending colon just beyond the cecum up to the splenic flexure. The small intestine and ileocolic junction were normal. There was no mesenteric lymphadenopathy. Proximal loop ileostomy and suturing of colonic wall was performed in view of gross fecal peritonitis and inseparable adhesions. The patient recovered well postoperatively. Anti-HIV antibody test done after surgery was positive. Histology of the colon wall showed mixed inflammatory exudates containing lymphoid aggregates with occasional multinucleate giant cells. No epithelioid cells or caseation were seen (Fig). Anti-tuberculosis treatment was continued. Two months later the patient is better and is awaiting a definitive procedure and/or ileostomy closure.

Although extrapulmonary tuberculosis is common in patients with AIDS, colonic involvement is rare.1 Diffuse pancolitis due to tuberculosis has been reported in one case.2 Obstruction and perforation of the gastrointestinal tract are rare in AIDS.3 Perforation of tuberculous intestinal lesions may account for 1%-10% of cases with peritonitis among the HIV-positive.4

Our patient had completely sloughed off anterior wall of the ascending and transverse colon. Thrombosis of small vessels on the antimesenteric border may have caused ischemic necrosis of the colonic wall. The treatment options available were complete resection of the involved colon with terminal ileostomy and descending colostomy. Another option was proximal diverting loop ileostomy with suturing of the colonic wall followed by colonic resection and anastomosis when the inflammation subsides. The latter option was selected in this case due to local factors.

References