cutaneous tissue. Aspiration of pus revealed acid-fast bacilli. She responded to 9 months of antitubercular treatment. [Indian J Gastroenterol2003;22:190-191]

Key words: Hepatic tuberculosis

Abdominal wall cold abscesses are commonly a result of tracking of a spinal or paraspinal tubercular lesion. We report a patient with abdominal wall cold abscess secondary to subcutaneous tracking of subcapsular tubercular liver abscess.

A 22-year-old woman presented with right lower chest pain, breathlessness, low-grade fever and anorexia of 3 months' duration. Chest X-ray showed right-sided pleural effusion. On pleural fluid analysis, she was diagnosed to have tubercular pleural effusion and was started on 4-drug therapy.

After 15 days she started experiencing localized pain in the right upper quadrant of the abdomen, which was intermittent and throbbing. She continued to have fever and anorexia, but chest symptoms had decreased. General examination revealed mild pallor. Abdominal examination revealed a firm, nontender, globular mass, 3 cm x 3 cm, in the abdominal wall in the right hypochondrium. Liver was palpable 2 cm below the right costal margin. There was no splenomegaly or free fluid. Chest examination revealed right-sided pleural effusion.

Investigations: hemoglobin 10.9 g/dL, white cell count 9400 cells/mm³ (P 70%, L 30%), ESR 75 mm in 1st hour, normal liver profile, serum electrolytes and blood sugar; HBsAg and HIV negative. Ultrasonography revealed an inhomogenous hypodense lesion in the oblique muscles, suggestive of abdominal wall cold abscess. CT scan revealed mild hepatomegaly with a septated subcapsular abscess, 5.5 cm x 2 cm x 6 cm, anterior to the right lobe of the liver, and a similar 2 cm x 2 cm lesion anterior to the left lobe. The right lobe abscess was extending into subcutaneous tissues (Fig). There was no ascites or retroperitoneal lymphadenopathy. Ultrasound-guided aspiration of pus demonstrated acid-fast bacilli.

The patient was continued on the 4-drug regimen. After 3 months, since ultrasonography showed no decrease in the size of the abscess, percutaneous guided needle aspiration was done. On completion of nine months of treatment, the patient improved symptomatically and gained 3 Kg weight, the abdominal wall mass disappeared. Ultrasonography showed complete resolution of the abscess, with normal liver.

Focal liver involvement such as abscess secondary to tuberculosis is rare probably because of the relatively low tissue oxygen tension in the liver. Subcapsular liver abscess is even more rare and to the best of our knowledge this is only the second report in indexed literature. Development of tubercular abscess has been reported in patients on antitubercular drugs.

Complications described secondary to tubercular liver abscess include duodenal fistula and bronchobiliary and gastrobiliary fistulae. Our patient had abdominal wall abscess, which has been described secondary to paraspinal/spinal tuberculosis but not with tuberculous liver abscess. Antitubercular drugs remain the mainstay of therapy. Some authors recommend transcatheter infusion of these drugs into the abscess cavity. Percutaneous transcatheter or surgical drainage may be required in large abscesses.

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Micro pneumatosis coexistent with Helicobacter pylori and its improvement

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Micro pneumatosis intestinalis is the occurrence of gas-filled circular voids with diameter of 20-200 microns, not lined with epithelium. We report a 39-year-old man with superficial gastritis and Helicobacter pylori infection who also had gastric, duodenal and colonic micro pneumatosis. Endoscopic biopsy after treatment for H. pylori gastritis showed no micro pneumatosis in gastric, duodenal or colonic mucosal sections. We suggest that H. pylori may be one of the causative factors for micro pneumatosis. [Indian J Gastroenterol2003;22:191-192]

Key words: Pseudolipomatosis
Microvesicular pneumatisis intestinalis, also called "pseudolipomatosis," for resembling fatty infiltration, is characterized by the presence of small gas voids in the gastrointestinal wall, especially in the mucosa. These voids are not lined by epithelial cells. Although the pathogenesis and etiology of this rare condition remain unresolved, there are some associations and possible causes reported. Commercially available endoscope disinfecting solutions of hydrogen peroxide have been incriminated in some cases. It has also been reported as an air pressure-related complication of colonoscopy.

We present a patient with duodenal and colonic pseudolipomatosis who also had *Helicobacter pylori* infection in the antral mucosa, and tubular adenoma in the transverse colon. The pseudolipomatosis resolved after treatment for *H. pylori*.

A 39-year-old man presented with abdominal pain, heartburn, belching and dyspepsia for 13 years. He had no history of smoking and no recent usage of any medication. Cholecystectomy was done 14 years ago. His father was operated on for peptic ulcer and brother suffered from peritoneal carcinomatosis. Examination revealed abdominal distension.

Stool examination showed no parasite or ova; laboratory findings were all normal. Gastroduodenoscopy showed grade III esophagitis, salt-and-pepper appearance in the antrum, and elevations over the mucosa in the second part of the duodenum. Biopsy specimens from these lesions showed microcysts that looked like lipocytes, in the lamina propria and muscularis mucosa of the duodenum (Fig). Superficial gastritis and *H. pylori* were observed in the antral specimen.

Since duodenal microcystosis was diagnosed, biopsies were also taken from the colonic mucosa; microcystosis was seen here as well. A 0.6 cm diameter sessile polyp was removed from the transverse colon; histology showed tubular adenoma without dysplasia. Gastrroduodenoscopy was repeated and biopsy specimens were taken from the same spots as before. Histology showed microcystosis in the gastric submucosa and duodenal mucosa.

The patient was given a course of *H. pylori* eradication therapy, namely, Lansoprazole 30 mg daily for 15 days, and clarithromycin 500 mg bid and amoxicillin 1000 mg bid for 10 days. One month after treatment the patient was free of symptoms. Microcystosis intestinalis was not seen in gastric, duodenal or colonic mucosa at repeat endoscopic biopsies.

Mechanical, bacterial and biochemical theories exist to explain the pathogenesis of pneumatosi. We are not aware of any publication on the role of *H. pylori*.

Pneumatosi cystoides intestinalis has been reported to be present together with other conditions such as pseudomembranous enterocolitis, necrotizing enterocolitis, bowel infarction, chronic obstructive pulmonary disease, intestinal obstruction, collagen vascular diseases and systemic amyloidosis, late-stage AIDS with cryptosporidial diarrhea, and Crohn's disease. Microcystosis is observed in most cases of pneumatosi cystoides intestinalis. It is also reported to be caused by disinfectant hydrogen peroxide solution. It may also be a complication of colonoscopy and disappears three weeks after the procedure.

In our patient microcystosis was diagnosed on initial endoscopy specimens. After treatment for *H. pylori* gastritis, microcystosis foci were not observed. These findings suggest that *H. pylori* may be one of the causative factors for microcystosis.

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Thrombocytopenic purpura as initial presentation of acute hepatitis A

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Extrahepatic immune manifestations are rare in hepatitis A virus infection. We report a 4½-year-old girl who presented with thrombocytopenic purpura as initial manifestation of hepatitis A virus infection. She re-

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Fig: Sections of duodenum reveal lipocyte-like multiple microcysts in lamina propria (H & E, 200X)

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