Microvesicular pneumatosis intestinalis, also called pseudolipomatosis, 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 implicated 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. Gastroendoscopy 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 mucosae of the duodenum (Fig). Superficial gastritis and H. pylori were observed in the antral specimen.

Since duodenal microcystomatosis was diagnosed, biopsies were also taken from the colonic mucosa; microcystomatosis was seen here as well. A 0.5 cm diameter sessile polyp was removed from the transverse colon; histology showed tubular adenoma without dysplasia. Gastroendoscopy was repeated and biopsy specimens were taken from the same sites as before. Histology showed microcystomatosis 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. Microcystomatosis 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 pneumatosis. We are not aware of any publication on the role of H. pylori.

Pneumatosis 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. Microcystomatosis is observed in most cases of pneumatosis 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 microcystomatosis was diagnosed on initial endoscopy specimens. After treatment for H. pylori gastritis, microcystomatosis foci were not observed. These findings suggest that H. pylori may be one of the causative factors for microcystomatosis.

References

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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-
sponded to steroid therapy. [Indian J Gastroenterol 2003;22:192-193]

**Key words:** Thrombocytopenia, viral hepatitis

**Hematologic manifestations following hepatitis B and C virus infections are commonly reported in literature. But the association of hepatitis A virus and thrombocytopenia has been described rarely.** Most of the cases have been reported in adults and older children. Immune thrombocytopenic purpura, to our knowledge, has not been reported in children less than 5 years.

A 4½-year-old girl was brought with hematuria, hematemesis and skin bleeds. She did not have fever or any other illness prior to the onset of these bleeding manifestations. On examination she was pale, mildly icteric, and had purpuric rash all over the body and subconjunctival hemorrhage. There was no lymphadenopathy. Abdominal examination revealed mild hepatomegaly; spleen was not palpable and there was no ascites. Rest of the systemic examination including fundoscopy was normal.

**Investigations:** hemoglobin 10 g/dL, white cell count 6,500/mm$^3$, (N 62%, L 38%), and platelet count 5,000/mm$^3$. Serum bilirubin 4.0 mg/dL (direct 2.6), total protein 7.0 g/dL (albumin 3.7), AST 2070 U/L, ALT 2130 U/L, alkaline phosphatase 299 U/L. Prothrombin time, partial thromboplastin time and serum creatinine were normal. Bone marrow aspiration showed increased number of megakaryocytes, consistent with peripheral platelet destruction. Serologic studies were positive for anti-HAV IgM antibodies and negative for viral hepatitis B, C, E and HIV. dsDNA and complement levels were normal.

Serial monitoring of platelet count revealed falling platelet counts and hemoglobin. Since she was symptomatic she received pulse doses of dexamethasone 0.8 mg/Kg/day for 4 days, after which she was discharged on oral prednisolone 1 mg/Kg/day in divided doses. Her platelet count after 4 days of dexamethasone was 70,000/mm$^3$. She was followed up after 4 weeks and after 3 months. Her platelet counts were normal and she did not have any bleeding or jaundice clinically.

**Extrahepatic immune manifestations are rare with hepatitis A virus infection although it is commonly associated with hepatitis B virus infection.** Hematological manifestations in hepatitis A can be severe, independent of the severity of liver disease.** Thrombocytopenia can follow 6 weeks after an attack of jaundice** or rarely as initial presentation.

Several mechanisms have been postulated for the development of thrombocytopenia, which include bone marrow depression, hypersplenism secondary to splenomegaly, disseminated intravascular coagulation, and a liver regulating factor that depresses platelet release. Some patients undergo spontaneous resolution; if not, corticosteroid therapy can be given.

**References**


**Association of ulcerative colitis with pulmonary sarcoidosis, subcutaneous lipomatosis and appendiceal adenocarcinoma**

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We report a 52-year-old man with left-sided ulcerative colitis for 5 years and pulmonary sarcoidosis diagnosed 3 years back. He presented with subcutaneous lipomatosis and a right iliac fossa mass, which was diagnosed histologically as appendiceal adenocarcinoma. He was treated with right hemicolectomy, followed by chemotherapy. [Indian J Gastroenterol 2003;22:193-194]

**Key words:** Appendix, idiopathic ulcerative colitis

**Primary appendiceal adenocarcinoma developing in a patient with left-sided idiopathic ulcerative colitis (IUC) is rare. Its association with pulmonary sarcoidosis and subcutaneous lipomatosis has not been reported in English literature.**

A 53-year-old man presented with low to moderate fever associated with chills for two months, weight loss of 8 Kg, and a palpable mass in the right iliac fossa for three weeks. He had been diagnosed to have IUC for the last 5 years, and was on mesalamine. He had one or two relapses every year, and these were controlled with short courses of prednisolone. Two years later, he developed cough and breathlessness on exertion. Chest X-ray revealed nodular-reticular shadows in both lung fields, with hilar and mediastinal lymphadenopathy. Transbronchial lung biopsy showed non-caseating epithelioid cell granuloma with Langhan's type of giant cells, which was negative for acid-fast bacilli. A diagnosis of pulmonary sarcoidosis was made, and he was treated with 40 mg of prednisolone per day. He became asymptomatic by the fourth week. Prednisolone dose was subsequently tapered off. There has been no recurrence of