trating segment IV of the liver. No significant lymphadenopathy was present. An extended cholecystectomy with 2-cm wedge resection of the liver beyond the infiltration and hepatoduodenal lymphadenectomy was performed.

The resected specimen had an irregular surface, with a firm grey-white nodule in the gall bladder fossa. The nodule was around 4 cm x 3.5 cm in diameter, near the gall bladder wall but not involving it. On histologic examination, the tumor was composed of fascicles of plump spindle cells intermixed with numerous plasma cells and variable number of lymphocytes and histiocytes (Fig). The spindle cells were arranged in fascicles with a moderate amount of intercellular collagen demonstrable with Masson trichrome stain. There were no abnormal mitoses. The veins entrapped within the lesion showed features of endophlebitis. The adjacent liver parenchyma showed moderate portal fibrosis with mild chronic inflammation in the portal tract. The bile ducts showed concentric fibrosis. Immunohistochemistry revealed strong positivity for smooth muscle antigen (SMA) in the spindle cells, indicating their myofibroblastic origin. CD68 immunostaining revealed occasional presence of histiocytic cells. Immunostaining for Epstein-Barr virus was negative. The postoperative period was uneventful and the patient is doing well 6 months later.

Inflammatory pseudotumors of the liver are relatively rare, but more than 200 cases have been reported in the literature. Because of the many reports of spontaneous regression of hepatic pseudotumors, most patients are treated with simple observation or conservative therapy. In contrast to peripheral hepatic pseudotumors, those involving the porta hepatis require treatment for obstructive jaundice. Fourteen cases have been reported at the hepatic hilum till 2001, apart from a recently reported case. None of the cases occurred at the gall bladder fossa.

The exact etiology of this lesion is unknown, but it is generally regarded as a benign reactive inflammatory condition. Evidence of Epstein-Barr virus infection has recently been documented. The presence of obliterating phlebitis has been reported, as in the present case. It was hypothesized that micro-organisms gaining access to the hepatic parenchyma through the portal vein subsequently elicit an inflammatory reaction that results in obliterating phlebitis. Portal hypertension has been reported to be an associated feature.

The prognosis of inflammatory pseudotumor of the liver is generally considered good. Most of the patients recover after resection.

References

Severe esophagitis in a child with Henoch-Schönlein purpura presenting as protein-losing enteropathy

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A 4-year-old boy was seen for vomiting, diarrhea and peripheral edema. He had no evidence of nephrosis or liver dysfunction. Upper gastrointestinal endoscopy was performed for investigating the etiology of protein-losing enteropathy. It showed severe esophagitis and multiple ulcers in the descending duodenum. The symptoms and endoscopic mucosal abnormalities subsided after three weeks of hospitalization without specific therapy. Ten days after being discharged he was seen again with characteristic rash of Henoch-Schönlein purpura and arthritis without gastrointestinal symptoms. Biopsy of the skin rash revealed leukocytoclastic vasculitis. [Indian J Gastroenterol...]

Fig: Microphotograph shows fascicles of variable sized spindle cells entrapping a portal tract (H&E, 280X)
Henoch-Schönlein purpura (HSP) is characterized by purpuric rash, renal manifestation, arthritis, and gastrointestinal involvement. Although abdominal pain and bleeding are well known gastrointestinal manifestations, intussusception, perforation and protein-losing enteropathy are less common. Esophageal involvement has not been described in children before.

A 4-year-old boy was admitted with complaints of vomiting and diarrhea since seven days. Swelling of his eyelids, lower extremities and abdomen along with diminished urine output within the last two days were reported by his parents. Physical examination revealed slight distension of the abdomen; the liver and spleen were not palpable. There was marked edema of the eyelids and lower extremities. He had petechial rash on his earlobes and around the lateral malleoli.

**Investigations:** Hemoglobin and platelet counts were within normal levels. Leukocytosis (WBC 42,000/mm³) and toxic granulation in peripheral blood smear were noted. Serum biochemistry was normal except for low serum protein (3.6 g/dL), albumin (1.8 g/dL) and calcium (7.2 mg/dL). Leukocytes and erythrocytes were seen on microscopic analysis of stool. Bacteriologic and virologic studies of stool gave negative results. Urine analysis was normal; there was no proteinuria by spot and 24-h urine analysis. The immunologic profile was normal except for low immunoglobulin G and C3. Antinuclear antibodies, anti dsDNA, rheumatoid factor, and nuclear cytoplasmic antigens were negative. ESR was normal. Abdominal ultrasonography showed dilated bowel segments and minimal ascites.

Endoscopy of the upper gastrointestinal tract showed severe esophagitis (Fig); gastric mucosa and duodenal bulb were normal. There were multiple ulcers with surrounding normal mucosa in the descending duodenum. Biopsy of the esophagus showed chronic esophagitis with ulceration. Cytomegalovirus and Candida were absent in tissue specimen. Antral biopsy showed chronic gastritis with ulceration. Biopsy of the descending duodenum showed severe inflammation characterized by focal ulceration. There was no evidence of reflux on gastric scintiscan. Immunoperoxidase studies revealed normal lambda and kappa chains. Colonoscopy and colonoscopic biopsies were normal.

The patient was treated with third-generation cephalosporine and sucralfate. The day after hospitalization the petechial rash resolved. Diarrhea, vomiting and edema disappeared within three weeks. At discharge, stool analysis, and serum albumin, calcium, complement and immunoglobulin levels were within normal limits. Endoscopic examination revealed normal esophagus and healing duodenal mucosa. After ten days, he was rehospitalized because of purpuric rash of the lower extremity and right knee arthritis. He did not have any gastrointestinal complaints. Stool analysis and albumin levels were within normal limits. Cutaneous biopsy showed leukoclastic vasculitis. Arthritis and rash resolved after three days without residual deformity.

Hypoproteinemia and edema are commonly related to renal involvement in HSP. Protein-losing enteropathy has been reported in only a few children with HSP. Mucosal erosions, ulcers and increased vascular permeability could be related to enteric protein loss in HSP. Gastointestinal tract involvement is due to vasculitis with ulceration. In 1973, Akdamar et al second reported the endoscopic appearance of HSP in a child. Multiple submucosal hemorrhagic lesions and superficial aphthoid ulcers in the stomach, duodenum, terminal ileum and colon have been reported in HSP but findings in the esophagus have not been described previously.

The gastrointestinal symptoms of HSP respond to corticosteroids. Because of the atypical presentation of disease during the first hospitalization, we did not give any steroid therapy to our patient. Normalization of the gastrointestinal findings without specific therapy could be spontaneous remission of the disease.

**References**


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