**Reply from the authors**

We have a different viewpoint from Dr Upasani’s about whether conservative treatment is less dangerous if the surgeon is less experienced and does not have access to adequate equipment. Generally, appendectomy is regarded as a Resident’s surgery, supervised by another Resident doctor. This is especially true in a public hospital set-up, where we work. But in the more than 20 years that one of us (SK) has worked in such a hospital, there has not been a single instance when we regretted an appendectomy done by a Resident doctor. Of course, the appendectomy in all these situations was for acute appendicitis. But whenever we followed the rule of doing appendectomy for appendicular mass (in our set-up or in a private set-up), complications invariably followed. That prompted us to undertake this study. So, appendectomy is a basic surgery and risks are negligible unless it is done in the presence of appendicular mass. And that is one of the messages we wanted to convey.

The conservative treatment we followed has been well described in our methods. It is essentially the same as the Ochsner-Sherren regimen.

Despite the results of our randomized trial, one may need to modify our recommendations to suit special situations.

Sunil Kumar, Sundeep Jain

**Henoch–Schönlein purpura probably due to montelukast presenting as subacute intestinal obstruction**

Henoch–Schönlein purpura (HSP) is a small-vessel vasculitis that involves the skin, joints, GI tract, and kidneys. GI symptoms occur in up to 85% of the patients. Montelukast is a leukotrine inhibitor used in the management of bronchial asthma. We report a man with HSP who was on montelukast for one month before presenting initially as subacute intestinal obstruction.

A 20-year-old man presented with colicky abdominal pain since one day. The pain was periumbilical in location, associated with recurrent bilious vomiting and obstipation. There was no history of fever. He had history suggestive of allergic rhinitis for which he was taking montelukast for one month. On examination, the patient was pale and in agony; pulse was 110/minute, blood pressure 140/100 mmHg. Per abdominal examination revealed diffuse tenderness, mild guarding but no rigidity. There was no organomegaly and bowel sounds were exaggerated. A diagnosis of subacute intestinal obstruction was made.

**Investigations:** hemoglobin 10 g/dL, ESR 80 mm in first hour, leukocyte count 26,000/mm³ with 10 percent eosinophils. Liver and kidney function tests, serum electrolytes, calcium and magnesium, and urine examination did not show any abnormality. X-ray abdomen revealed multiple air-fluid levels. Chest X-ray was normal. Montelukast was stopped.

The next day, the patient started passing flatus but continued to have colicky abdominal pain of less severity. Repeat leukocyte count was 24,000/mm³ with eosinophil count of 9000/mm³. On the third day he developed arthralgias and palpable purpura that was distributed over the buttocks and the posterior aspect of arms and legs. Repeat urine examination showed 7-10 RBC/hpf. Stool for occult blood was positive. Serum IgE level was 0.9 mg/L (normal <0.0005). Antineutrophilic cytoplasmic antibodies (ANCA) were positive. Endoscopy revealed diffuse mucosal redness and hemorrhagic erosions in the stomach and multiple ulcers in the first and second parts of the duodenum. Biopsies from the ulcers showed prominent infiltration by eosinophils and red cell extravasation in the mucosa and submucosa. Barium meal follow-through showed thickened jejunal folds, small barium flecks and shallow ulcers in the jejunum. Skin biopsy showed infiltration of small vessels by polymorphonuclear leukocytes, suggestive of leukocytoclastic vasculitis. A diagnosis of HSP was confirmed and the patient started on tablet prednisolone 60 mg a day. The patient improved clinically and investigation reports normalized.

Montelukast has been reported to cause systemic eosinophilia and small-vessel vasculitis, namely, Chürg-Strauss syndrome. An objective causality assessment revealed that montelukast leading to HSP as adverse drug reaction in this case was probable. We could not find any reported case of montelukast-associated HSP.

Sudeep Khanna, Bhupesh Uniyal, Dhirender Kumar, Jagdish Vij

Department of Gastroenterology, Pushpawati Singhania Research Institute for Liver, Renal and Digestive Diseases, Sheikh Sarai-Phase II, New Delhi 110 019

**References**


**Lymphoplasmacytic sclerosing pancreatitis mimicking pancreatic cancer**

Ramachandran *et al.* recently reported two cases of autoimmune pancreatitis treated with steroids. This condition often mimics pancreatic cancer in its clinical presentation though the CT findings of a diffusely enlarged pancreas without a discreet mass may suggest the diagnosis of lymphoplasmacytic sclerosing pancreatitis...
A 37-year-old man presented with complaints of pain in the right hypochondrium associated with progressive jaundice of three weeks’ duration. There was no fever. The patient was a non-alcoholic and a non-smoker. There was a history of skin allergy in the past, for which he was put on short-term steroids. On examination, he had marked icterus. Investigations: bilirubin 16.2 mg/dL (direct 12.2), alkaline phosphatase was raised (342 IU/L). Ultrasonography showed dilated common bile duct (CBD) with no evidence of stone disease and a hypoechoic mass in the head of the pancreas. CT scan showed markedly dilated CBD up to the duodenum with dilatation of intrahepatic biliary radicles, with diffuse enlargement of the entire pancreas (Fig). Endoscopic ultrasonography showed dilatation of the CBD with narrowing in the intrapancreatic region, with a hypoechoic mass in the head of the pancreas, suggestive of malignancy. ERCP with stenting of the CBD was done. Tumor markers (CEA, CA 19-9) were within normal limits.

At surgery, the pancreatic head was hard and grossly enlarged. The rest of the gland was uniformly enlarged and firm to hard in consistency. The peridochal nodes were enlarged with dense adhesions in the peripancreatic region. Frozen sections from the pancreas showed evidence of fibrosis with atrophic lobules, suggestive of chronic pancreatitis. A pylorus-preserving pancreateoduodenectomy was nevertheless done. The post-operative phase was uneventful except for wound infection.

On microscopy, the pancreas revealed areas of fibrosis and a diffuse inflammatory infiltrate of lymphocytes and plasma cells. The lobules showed acinar atrophy with fibrosis. In places the inflammation was predominantly periductular. There was no evidence of malignancy. The histological picture was consistent with LPSP – intermediate type. Further tests showed elevated ANA levels (1.3 IU/L). The patient has been followed up for 6 months and is asymptomatic.

Both the cases reported by Ramachandran et al\textsuperscript{1} presented with recurrent episodes of pain suggestive of recurrent pancreatitis. Our patient was 37 years old, whereas most patients reported earlier were in the 5th-6th decade, and had no clinical evidence suggestive of recurrent episodes of pancreatitis or chronicity.

In conclusion, though many patients with LPSP (a rare entity in itself) may undergo pancreateoduodenectomy because of inability to exclude the possibility of pancreatic neoplasia, the possibility of LPSP needs to be considered in patients with suspected but unproven pancreatic cancer.

Prasad K Wagle, Guruprasad S Shetty, C Tampi*
Departments of GI Services and *Pathology, Lilavati Hospital and Research Center, Mumbai

References

**Safe liver biopsy in a patient with chronic hepatitis C under continuous ambulatory peritoneal dialysis treatment**

Besides the bleeding tendency generally occurring in patients with end-stage renal disease (ESRD), patients on continuous ambulatory peritoneal dialysis (CAPD) are also prone to peritonitis. We describe a safe and reproducible approach for liver biopsy in patients with ESRD and hepatitis C virus (HCV) infection on CAPD.

A 48-year-old woman with ESRD due to unknown etiology for a year was treated with CAPD. Investigation showed anti-HCV antibody positivity; serum transaminases level and ultrasonography of liver were normal. HCV genotype was 1b and viral load was 32,860 copies/mL.

Prior to liver biopsy, desmopressin was given via intravenous infusion over 30 min at 0.3 mg/Kg body weight diluted in 50 mL of saline.\textsuperscript{1} Thirty minutes after infusion, ultrasound-guided liver biopsy was performed. Eight hours later, the peritoneal cavity and liver were screened with ultrasonography. There was no bleeding or any other complication. Since blood urea nitrogen, creatinine, electrolytes and arterial blood gases were normal 24 hours after biopsy, the CAPD-off period was prolonged. First peritoneal exchange was performed 48 hours after the biopsy and the solution was pinkish. There was no abnormality macroscopically or microscopically in the peritoneal solutions on subsequent exchanges. Histological examination revealed chronic hepatitis with Knodell score 5. She is under interferon therapy now.

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\textbf{Fig: CT scan showing diffuse enlargement of pancreas and dilated common bile duct.}