Autoimmune pancreatitis

The article by Ramachandran et al\textsuperscript{1} needs several clarifications. The authors state that hyperglobulinemia is a feature of this condition, but their patients had either normal or only marginally raised globulin level. Amylase levels were normal in both patients, although the authors mention high values of enzymes accompany the diagnosis of pancreatitis. ANA/dsDNA was positive in the first patient, but was negative in the other patient. No tests were performed to rule out other causes of acute recurrent pancreatitis, e.g., microlithiasis, sphincter of Oddi dysfunction.

Both their patients had cholestasis (alkaline phosphatase elevated >3 times normal) but the first patient also had ALT >10 times normal, which does not favor extrahepatic cholestasis but rather hepatitis associated with cholestasis. Both patients had pain, although the authors mention that patients usually have mild pain. The second patient had features of sclerosing cholangitis but had negative ANA and imaging findings were non-specific.

Resolution of pancreatitis after two months may be spontaneous (unrelated to steroid). Total resolution of pancreatic abnormality (stricture / dilatations) after steroid course in the first patient, and remaining normal for a year without maintenance dose of steroid (when pancreatic parenchyma may be edematous) is surprising. I wonder whether the duct abnormalities observed were due to early ERCP in acute pancreatitis with resolution by the time of the second ERCP.

Finally, the title states “chronic pancreatitis”. In the patients reported, the structural changes appear to have happily resolved. Though this article describes a rare condition, we need more stringent criteria to make such a diagnosis.

Reference


**References**


**Sporadic, multiple adenomatous polyposis coli treated with ileo-endorectal pull-through**

Adenomatous polyps are extremely rare in children less than 12 years of age.1

A six-year-old boy presented with bleeding per rectum for one year. The child was anemic (hemoglobin 5 g/dL) and hypoproteineminc (serum protein 3.5 g/dL). Digital rectal and proctoscopic examination revealed a bunch of polyps; one of these was sent for histological examination. Barium enema and colonoscopy showed that the whole colon was studdied with polyps. Histology showed adenomatous polyp. Upper gastrointestinal endoscopy did not show any polyps. There was no peri-oral or axillary fold hyperpigmentation. Ophthalmoscopic examination did not show any retinal lesions. Pedigree charting of two generations did not show any such case. Ophthalmoscopic examination of family members did not show any retinal lesion.

Due to the poor general condition of the child, ileal pouch reconstruction was deferred and total colectomy with ileo-endorectal pull-through with ileo-anal anastomosis at the anal columns was done. Examination of the specimen showed that the rectum and entire colon was studded with polyps. Two years later, the child is passing well-formed stools 3-4 times a day and there has been no recurrence of polyps.

Adenomatous polyps are extremely rare in children. Kottmeier and Clatworthy1 reported only three cases in a group of 50 patients with intestinal polyps. We have seen about 500 cases of various types of intestinal polyps in the past 10 years (unpublished data); this is the only one case of its type.

Fig: Bisected specimen showing rectum, colon and cecum studded with polyps.

**Familial adenomatous polyposis**

Familial adenomatous polyposis are now recognized to be Mendelian dominant and the gene responsible (APC gene) has been located on the short arm of chromosome 5.2 Pigmented spots in the retina and DNA tests for FAP gene are important screening methods.2 There was no symptomatic, genetically related individual in two generations of our case. None had pigmented spots in the retina. DNA tests for FAP gene could not be performed. In all probability our case had sporadic disease.

Total colectomy with removal of the rectal mucosa and ileo-endorectal pull-through is a simple and dependable procedure. With passage of time the neorectum, i.e., terminal ileum undergoes dilatation and there is spontaneous correction of the problem of stool frequency. If this does not occur even after one year a reservoir can be constructed when the patient is relatively healthy and can withstand a bigger procedure.

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**References**


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**Dysphagia due to olanzepine, an antipsychotic medication**

Olanzapine is currently one of the most commonly used anti-psychotic medications. The side-effect profile of the drug is considered favorable compared to older medications.

A 24-year-old man, a known patient of bipolar affective disorder, was admitted with a diagnosis of mania. He was started on olanzapine 20 mg/day and sodium valproate 1000 mg/day. He complained of increased salivation and difficulty in swallowing his saliva 5 days after starting his medication. Over the next few days he had difficulty in taking food orally or drinking water; a nasogastric tube was inserted and his nutrition and hydration were maintained. ENT examination ruled out local pathology. The patient did not have fever, rigidity, involuntary movements, other weakness, focal neurological deficit or signs of parkinsonism. The olanzapine dose was reduced to 10 mg/day and was stopped over the next 5 days. The dysphagia resolved over the next week, and the patient was continued on sodium valproate 1000 mg/day and clonazepam 4 mg/day.

Dysphagia is a rare side effect of olanzapine therapy. Reports of dysphagia due to risperidone1 and