immunity in children (68%) among 15 year olds, and seropositive children had higher mean number of household members compared to the seronegative ones. The rate in Tehran among school children was 22.3%, and 93.2% in a report from Delhi.4

In our country, therefore, there is no need for routine vaccination against HAV virus in chronic liver disease except for younger patients and probably those who live in less populated families.

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Hepatitis A in pediatric acute liver failure in southern India

Analysis of 1612 subjects in different parts of India demonstrated that almost 50% of children under 5 years of age are at risk for hepatitis A.1 A recent report showed that the relative contribution of hepatitis A to acute viral hepatitis in children has increased to over 80% in 1994-1997 as compared to 51% in 1978-81.2 However, a number of studies in school children in northern and southern India have reported evidence of prior infection in up to 98% of 10-year-old children.3,4

We retrospectively analyzed data of children between 0 and 15 years, admitted in our pediatric intensive care unit (ICU) between January 2001 and July 2004, with acute liver failure-related diagnoses. The total number of children admitted with such diagnoses was 55 (mean age 5.5 years). Of these, 22 children (40%) were admitted with hepatic encephalopathy. Other diagnoses included acute-on-chronic liver disease (n=12; 22%), acute liver failure (11; 20%), fulminant hepatitis (4; 7%) and neonatal hepatitis (4; 7%). One patient each had Reye’s syndrome and drug-induced hepatitis. Pre-existing liver disease was present in 17 of 55 patients.

IgM anti-HAV serology was positive in 13/27 (48%) patients tested; the mean age of patients who tested positive was 7.1 years. Two of the 13 (15%) had pre-existing liver disease. HBsAg was positive in only 1/33 (3%), anti-HCV in 1/10, and anti-HEV in 2/4 tested. Eleven patients died and two were discharged with poor prognosis. Two patients who died had evidence of acute hepatitis A.

This retrospective review showed a high incidence of acute hepatitis A infections of sufficient severity to require ICU admission in children with no pre-existing liver disease.

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References

Technical modification for difficult intubation during endoscopic variceal ligation

Endoscopic variceal ligation (EVL) has supplanted esophageal sclerotherapy because of greater efficacy and fewer associated complications.1,2,3 The present procedure for EVL includes diagnostic endoscopy followed by removal of endoscope, assembly of the multi-band ligating unit, followed by reintroduction of the endoscope. Occasionally it is difficult to negotiate the endoscope across the upper esophageal sphincter (UES) following the assembly of the ligating unit. We describe simple techniques to overcome this difficulty.

After assembly of the ligating unit, if it is not
possible to negotiate the scope across the UES despite reasonable attempt, the endoscope is positioned proximal to the UES. A guide wire (GW) is then passed through the accessory channel of the endoscope, and the endoscope is negotiated into the esophagus over the GW. Because of the addition of the ligating unit, the GW has to be passed from the irrigation port or the diaphragm through which the trip wire passes.

1. In ligating units where the irrigation port is on the top (Fig 1) (V-Gripp; IMI, Kolkata), the irrigation cap is removed and the GW is passed through it and down the accessory channel and guided under vision into the esophagus. The endoscope is then guided gently over the GW into the esophagus under vision. The GW is removed, the irrigation cap is replaced and EVL is done. Use of the 0.035 inch GW does not disturb the release mechanism of the ligation unit.

2. In ligating units where the irrigation port is on the side (Fig 2) (Superseven; Microvasive Boston Scientific, USA), the port cannot be used to pass the GW. Also in the Saeed 4/6 Shooter system (Wilson Cook, USA) there is no irrigation port. In such a situation the GW is passed through the diaphragm through which the trip wire passes.

3. In patients in whom it is known previously that it is not possible to intubate with the band assembly, the GW is passed at the time of initial endoscopy and the endoscope is removed. After assembly of the ligating unit, an ERCP catheter is passed down the accessory channel as above and as it comes out of the scope, the distal end of the GW is threaded through it retrograde and brought out. The ERCP catheter is removed and the endoscope is guided over the GW. The hole is sealed with a tape so that good suction is generated for suction of varix into the cylinder.

We used this procedure successfully in 3 patients (all men, median age 50 years) on 5 occasions (V-Gripp in 3 and Saeed 6 Shooter in 2). One patient underwent EVL on 3 occasions, as it was not possible to intubate the esophagus conventionally on all the 3 times with this technique. Initially we used a Savary Gilliard GW (Wilson Cook, USA), but this interfered with the release of the band due to stiffness of the GW. Now we use a 0.035 inch GW, which does not disturb the release mechanism.

EVL is increasingly used to treat bleeding esophageal varices. The ligating band cylinder increases the effective diameter of the endoscope and makes it stiff and difficult to negotiate across the UES. If bleeding ensues with repeated attempts it further hampers the already compromised vision due to the cylinder. The exact incidence of this problem is not known, but when it occurs, EVL has to be abandoned or replaced by sclerotherapy.

This wire-guided intubation technique that we have described for intubation is simple. Previously GW or washing pipes have been used for difficult intubation. Hurwitz et al described a similar technique in which they passed the 0.035 inch GW blindly or under the vision for Saeed 6 Shooter. We have further modified this to suit different models of ligating units available, and for patients in whom difficulty in intubation is anticipated.

In summary, we describe a technique to overcome the difficulty in intubating the esophagus after assembly of the ligating unit.

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References


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Autoimmune pancreatitis

The article by Ramachandran et al1 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.


Reply from the authors

Dr Joshi has raised questions challenging the diagnosis of AIP in the two patients we described.

Detailed etiological work-up, including bile for microlithiasis, serum calcium, lipid profile and ANA, was performed in both patients. There was no history of abdominal trauma, drug intake or family history of pancreatitis. For the sake of brevity negative tests were not mentioned in the report. Sphincter of Oddi dysfunction does not present with the constellation of structural abnormalities described in our case report.

The first patient was diagnosed to have AIP based on positive ANA and ds DNA, characteristic imaging features, and return of all abnormalities to normal after a course of steroids.

The second patient was diagnosed based on hyperglobulinemia and characteristic imaging studies as in the first patient, lymphoid aggregates seen on FNAC of the pancreas, and return of biochemical and imaging abnormalities and elevated blood sugar to normal after a course of steroids.

The Japanese Pancreas Society has laid down three criteria for the diagnosis of AIP, where criterion 1 must be present together with criterion 2 or 3:1 1) pancreatic imaging studies showing diffuse narrowing of the main duct with irregular wall and diffuse enlargement of the pancreas; 2) elevated serum gamma globulins / IgG or the presence of autoantibodies; 3) pathological examination of pancreas showing lymphocytes and plasma cell infiltration. American investigators consider response to steroid therapy an important diagnostic criterion.

We are unaware of any disease of the pancreas other than AIP where strictures of the pancreatic duct and diabetes resolve after a course of steroids. Furthermore, the patient remaining asymptomatic for one year without therapy also fits in with a diagnosis. Raised liver enzymes in the absence of hepatitis, normalizing with steroid therapy, has also been described in AIP.2

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Case Snippets
