Editorial

Portal hypertensive intestinal vasculopathy:
the jejunal story

Variceal bleeding, whether esophageal or gastric, is a known life-threatening cause of upper gastrointestinal (GI) bleeding in patients with portal hypertension. During the last two decades, another cause of upper GI bleeding has been recognized in these patients— that occurring from the gastric mucosa. These mucosal lesions have been labeled portal hypertensive gastropathy (PHG). Lesions classified as severe are responsible for most of the bleeding episodes. Rather than being a source of massive bleeding, PHG leads to chronic blood loss and anemia.

Other parts of the GI mucosa such as the duodenum and jejunum11-15 and the colon (portal hypertensive colopathy)16-29 have also been noted to be affected because of portal hypertension. There are reports of bleeding from mucosal lesions in the colon and anorectal and colonic varices.15,19,20,22,25,28 Thus, it appears that the entire GI tract that is drained by the portal venous system is affected in patients with portal hypertension—portal hypertensive intestinal vasculopathy.

The small intestine is relatively inaccessible, resulting in only a limited number of studies in this region. The study in this issue of the Journal by Desai et al30 is therefore welcome. The authors studied 40 patients with portal hypertension and 43 control subjects (patients with non-ulcer dyspepsia with normal upper GI endoscopy) using a pediatric colonoscope as an enteroscope. They evaluated the stomach, the duodenum, and the proximal part of the jejunum endoscopically and histologically. Endoscopic features of jejunalopathy were seen in six patients and none of the control subjects. All six patients had endoscopic evidence of PHG and five of them also had duodenopathy. These changes were not seen in the control group. Histological examination of jejunal pinch biopsies showed vascular changes to be similar in patients and control subjects.

Some questions are unanswered in the article. We presume that the endoscopies were performed by more than one experienced endoscopist. Was there interobserver variation and, if so, how was it settled?

The selection of cases is also problematic. Thirty-three cirrhotics and seven non-cirrhotics (extrahepatic portal vein obstruction 6, non-cirrhotic portal hypertension 1) were enrolled and they have been clubbed as one group. The common factor is portal hypertension, but had the seven non-cirrhotics not been included, it would have been a more homogenous group and the findings would have been easier to interpret.

It would have helped if the cause of cirrhosis and the grades of esophageal varices, gastric varices and portal hypertensive gastropathy were mentioned by the authors. The association between these parameters and endoscopic and histologic PHG, portal hypertensive duodenopathy and jejunalopathy would have been interesting to study. Further, the influence of endoscopic sclerotherapy should have been mentioned.

Another pitfall was the selection of the control group. The authors selected patients with non-ulcer dyspepsia with a rider that upper GI endoscopy had to be normal. This effectively meant that no comparison could be made between the endoscopic changes present in patients with portal hypertension and the control group. The significance of diffuse erythema in the jejunum is not clear and also how cherry-red spot could be differentiated from angiodysplasia.

The histological findings in this study are contrary to those reported earlier from the same city by Nagral et al. While Nagral et al observed abnormally dilated capillaries in 84% of their patients, which was significantly more than in the control group, Desai et al30 noted dilated vessels in 42% of the patients and 66% of the control subjects (p=ns). The difference may be due to the techniques of obtaining jejunal biopsies. While Nagral et al14 used a Watson biopsy capsule, Desai et al10 obtained pinch biopsies during jejunoscopy. Capillary dilatation may be non-specific and has been attributed to portal hypertension, other transient factors, and even as an artifact due to the pinch-avulsion technique of most commonly used biopsy forceps. Dilated and congested mucosal blood vessels may therefore be a poor marker of portal hypertension.5,11,12

In an earlier study, using pinch biopsies, we had observed dilated capillaries more frequently in the jejunum of patients with portal hypertension.15 We also noted thickened jejunal capillary walls in patients with portal hypertension. Capillary wall thickening has been observed in the stomach7 as well as in the colon.22 Desai et al30 did not measure the thickness of the capillaries, though they had facilities for doing it. This finding could have been important in either proving or dis-proving the histological changes observed earlier.7,13,22

The lack of correlation between endoscopic and histologic finding observed by Desai et al30 is not surprising. Similar findings have been reported earlier for PHG. Moreover, the endoscopic changes were noted in only six patients with portal hypertension; a type II error would be likely in such a small sample size.

The clinical importance of the findings by Desai et al30 is not clear. They started by stating that the small
bowel mucosa is a potential source of bleeding but do not mention whether they found bleeding from the jejunum in any of their patients. Nonetheless, their study is important in our understanding of the spectrum of GI changes associated with portal hypertension. It further confirms the view that the whole gut drained by the portal venous system is involved in portal hypertension. The authors have opened up areas for further research on portal hypertensive intestinal vasculopathy.

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References

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