bile ascites may not require surgical intervention, but the progress of the patient needs monitoring.

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References

Ultrasonic Demonstration of Diminished Respiratory Caliber Variation in Portal Vasculature: A Sure Sign of Portal Hypertension

Sir,

Previous workers1-3 considered absence of respiratory caliber variation in portal vessels to be a specific sign of portal hypertension (PH). However, they did not define a lower limit of the respiratory variation below which the diagnosis of PH could be predicted with certainty. Kurrol et al4 observed quantitative respiratory variations in normal subjects, and mean respiratory variations of 40%, 150%, and 80% respectively in the portal (PV), splenic (SV), and superior mesenteric (SMV) vein diameters, but they did not correlate the value in patients with PH.

This prospective study involved 100 normal healthy subjects (78 men, 22 women; mean age 32 years, range 17-50) and 50 patients with PH (39 men, 11 women; mean age 36 years, range 13-63). The diagnosis of PH was based on the clinical features and the documentation of esophageal varices by esophagogastroscopy. The normal subjects and the patients were examined with real-time linear-array ultrasound scanner (EUB-24F) using a 5.0 MHz transducer. To avoid the effects of meal and posture,5 the subjects were studied after an overnight fast, and all measurements were taken in the supine position. The portal vessels were displayed in longitudinal section and the diameters were measured at their broadest at the same point during deep inspiration (I) and deep expiration (E). The respiratory caliber variation in percentage was calculated as (I-E)/E × 100%; the mean ± SD values were compared using Student's t test.

Sonography could visualize the PV, SV, and SMV in 100%, 98% and 96% of healthy volunteers, and in 100%, 92% and 82% of the patients. The mean ± SD values and the range of percentage increase in caliber with inspiration in the PV, SV and SMV were 55 ± 10.2 (22.2 - 58.3), 67.8 ± 12.0 (36.7 - 100.0) and 62.7 ± 12.4 (40.0 - 100.0) in normal subjects; while these values in patients were 0.5 ± 3.4 (0.0 - 7.1), 0.7 ± 2.8 (0.0 - 8.4) and 0.7 ± 2.3 (0.1 - 7.7). The respiratory variation was statistically significant (p < 0.001) in normal subjects, but insignificant (p > 0.1) in patients.

Hemodynamic and morphologic changes in the normal portal vasculature during respiration (an increase in the diameters on inspiration and decrease on expiration) have been shown on pathophysiological as well as ultrasonic studies.1-6 Bolondi et al4 observed a significant respiratory caliber variation in normal SV and SMV, but not in PV. The present study shows a significant caliber variation in all the three veins, and thus confirms and extends the findings of Kurrol et al.4 On quantitative estimation, we did not encounter in any case a respiratory variation less than 22%, 36% and 40% in the PV, SV and SMV; the respective values in an earlier study were 12%, 32% and 29%. Previous workers1-3 attached importance to the absence of caliber variation as being specific of PH; this was seen in 78-89% of their patients. In our study, the respiratory difference in the diameters of the PV, SV and SMV was absent in 98%, 94% and 90% of the patients respectively; in the remaining cases too, the difference was insignificant. Furthermore, in no case did the respiratory difference exceed the lower limit of caliber variation seen in normal subjects.

Hence, a diminished (rather than absent) respiratory caliber variation in the PV, SV and SMV of less than 22%, 36% and 40% respectively could be considered a specific, sensitive and accurate ultrasound diagnostic index for patients with suspected portal hypertension. Furthermore, in contrast to previous studies,1-3 we attach greater significance to the portal vein caliber variation because this vein could be traced out easily in all cases.

References
Fatal Strongyloidiasis in India

SIR,

Dr Sudha Sane reported two fatal cases with disseminated strongyloidiasis to highlight their abnormal clinical presentations, i.e., hematemesis in one and anuria in the other.1 We would like to point out some deceptive statements in this paper. Firstly, the incidence rate mentioned is not true. No reference was provided to support the statement that the incidence of strongyloidiasis in populations living in unhygienic conditions is 35% to 85%. The highest prevalence rate, not the incidence rate, of this infection even in a very vulnerable population, i.e., the Burma-Thai prison's prisoners of war, is only 37%.2-3 In India its prevalence rate is less than 1%.4 Moreover, the author did not quote a single reference of Indian studies. Dr Sane states that this parasite is limited to tropical countries. However, there are various reports from developed and temperate countries like the USA, UK, Italy and USSR.5-6

Dissemination of Strongyloides stercoralis especially in compromised hosts, as were these patients, may occur in any organ and severe disseminated disease manifests in various ways according to the site of involvement and extent of the damage to the organ.7 Chronic gastritis and gastric ulceration along with massive edema of the gastric outlet or duodenum may lead to delayed emptying of the stomach and vomiting which may be blood tinged. Though rare, massive hematemesis is a documented phenomenon,8 contrary to the author's statement. Finally, the author did not specify which aspect of the present cases has not been reported from India. If it is fatal strongyloidiasis, then this statement too is not true.9

Recently, we treated 10 cases with strongyloidiasis, including one fatal case, of 2100 stool samples processed during one year 1988-89. A 40 year old male labourer presented with severe epigastric pain, constipation and vomiting for one month. The vomiting was aggravated and the vomitus became blood stained after administration of oral H2 receptor antagonists. A barium meal series revealed complete obstruction of the gastric outlet. An upper GI endoscopy 15 days later showed numerous hemorrhagic ulcers in the gastric mucosa; the endoscope could not pass beyond the gastric antrum due to gross edema of the duodenal mucosa. Gastric biopsy showed non-specific gastritis with numerous hemorrhagic ulcers. His stools, examined then by formal-ether concentration method, showed numerous rhabditiform larvae of Strongyloides stercoralis and hookworm ova. Unfortunately, before the stool report was communicated, the patient became comatose and died at home two days after endoscopy.

It is possible that H2 receptor antagonists aggravated the condition by virtue of their anti-secretory activity with consequent rise in gastric pH. Whether dissemination of the parasite occurred following endoscopic trauma is not known.

S SINGH
J C SAMANTARAY
M P SHARMA

References

Reply from the Author

SIR,

Since my report was not meant to be a review article, only pointedly relevant references were quoted.

My first reference states, "In certain areas among low socioeconomic groups and in warm humid climates the rate of infection may be as high as 85%". Unfortunately, the reference number 1 was missing at the end of the sentence in the text.