India Elsewhere


Reports on the role of *H. pylori* in the pathogenesis of portal hypertensive gastropathy (PHG) are conflicting. This prospective study analyzed the prevalence of *H. pylori* in patients with PHG and its relationship to the severity of PHG.

37 cirrhotic patients (24 men, mean age 39.2 [14.9] years; alcohol-related 9, post-hepatic 18, cryptogenic 10; Child class A 18, B 15, C 5) with PHG (fundus 14, body 2, antrum 16, diffuse 5; mild 27, moderate 9, severe 1) were evaluated. 43% of patients with PHG were positive for *H. pylori* by histology and urease test. A linear trend with age was seen in *H. pylori*-infected patients. There was no difference in the prevalence rate with respect to sex, alcohol consumption, or site of lesion. *H. pylori* status declined from 52% in patients with mild PHG, 22% with moderate PHG, to nil with severe PHG.

The authors conclude that PHG does not provide a favorable environment for the colonization of *H. pylori*. This bacterium is unlikely to contribute to the pathogenesis of PHG.

Bhattachar BNS, Sharma CLN, Gupta SN, Mathur MM, Reddy DCS (Departments of Surgery, Anatomy and Preventive and Social Medicine, Institute of Medical Sciences, Varanasi). Study on the anatomical dimensions of the human sigmoid colon. *Clinical Anatomy* 2004;17:236-43

This study provides data on the anatomical measurements of the sigmoid colon in 70 subjects (51 alive; 27 men, mean age 36.2 [12.6] years; undergoing laparotomy for non-colonic disease) and 19 cadavers (17 men, age unknown; no sigmoid colon disease).

The sigmoid colon was completely emptied by finger stripping before taking measurements. There was a wide variation in the measurements. 80% of subjects had a sigmoid colon length of 34-60 cm (median 47), the width at the apex was 3-5 cm (3.5), and the width at the distal end was 3-5 cm (4). In the live subjects the sigmoid colon was wider at the apex and distal end than at the proximal end; but the widths at the apex and the distal end were not different from each other.

In 80% of individuals the sigmoid mesocolon vertical length was 10-16 cm (median 13), the maximum width 7-10 cm (8), and the width at the root was 4-7 cm (6). Seven shapes of the sigmoid loop were seen. The commonest was dolichomesocolic (mesocolon vertically longer than wide, the sigmoid loop having its maximum convexity located a little proximal to the apex), but brachymesocolic pattern (width of mesocolon greater than vertical length) was also seen. The mesocolon in females was brachymesocolic whereas in males it was dolichomesocolic. The measurements did not change with gender as age advances from 16-60 years. All parameters were similar in cadavers and live subjects, except the width of the mesocolon which was lower in cadavers (probably due to shrinkage).

This study demonstrates that the mesocolon has several variations in shape, which could account for the difficulty encountered occasionally in negotiation during colonooscopy. Males are more prone to develop sigmoid volvulus as their mesocolon is dolichomesocolic.


Mutations in the cationic trypsinogen (protease, serine, I trypsin 1; PRSS1) and serine protease inhibitor, Kazal type 1 (SPINK1) have been reported to be associated with hereditary and non-hereditary chronic pancreatitis (CP). This study evaluated the role of PRSS1 and SPINK1 mutations in Indian patients with CP.

Genomic DNA was isolated from leukocytes and coding regions of PRSS1 and SPINK1 genes were sequenced in 198 patients (156 males; 120 idiopathic CP (ICP); 41 alcoholic CP (ACP); 37 hereditary pancreatitis (HP)), and also in 24 unaffected relatives from HP families and 290 healthy volunteers. Mutation in the PRSS1 gene was not detected in any patient or in the controls. 77 patients (38.9%) with CP had at least one SPINK1 mutation, with a majority carrying the N34S allele (n=75; homozygote 15, heterozygote 60) and 2 carrying the P555 in the heterozygous state. The N34S mutation in the SPINK1 gene was seen in 73% of HP (homozygote 7), 26.8% of ACP (homozygote 1), and 32.5% of ICP (homozygote 7) patients. Only 2.8% of the control population and 25% of unaffected relatives of HP patients had the N34S mutation. The P555 mutation was observed in one ICP, one ACP and 3 controls. Genotype-phenotype correlations did not suggest any difference in the age of onset, severity of disease, or pancreatic endocrine insufficiency in patients with or without mutations in SPINK1 gene and irrespective of the allelic status of N34S SPINK1.

The authors conclude that established mutations in PRSS1 are not a common cause of CP in India. N34S SPINK1 mutations were strongly associated with all types of CP, although the penetrance is very low.

Compiled by Sundeep Shah