surgery is as distasteful as those following laparoscopic cholecystectomy and that the operation will be performed by surgeons with an interest in both oesophageal disease and endoscopic surgery.


Serum amylase levels and acute pancreatitis

EDITOR.—We fully agree with the conclusion reached by Winslet et al (Gut 1992; 33: 982–6) that serum amylase is not a sensitive test for acute pancreatitis especially when the cause is related to alcohol. In a recent study we found that a serum amylase (>3 times normal) had a sensitivity of only 55% in detecting acute alcoholic pancreatitis. This is much lower than the 86% noted by Winslet et al. In our study, we included only patients with image proved pancreatitis (ultrasound or computed tomography scanning) as opposed to the clinical criteria adopted by Winslet et al and this could explain the difference.

In their study Winslet et al correctly speculate that serum lipase may be a better test in these circumstances. And we have proved this in our study. We found that serum lipase (>3 times normal) had a sensitivity and specificity of 100% in detecting acute pancreatitis of alcoholic cause.

In the past, the unpopularity of serum lipase was attributed to defects inherent to its assay. With the advent of new techniques and the information obtained from our study, we feel that serum lipase should replace serum amylase as the initial test for acute pancreatitis.

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Reply

EDITOR.—We are grateful for the comments by Dr V Gumaste regarding our recent publication. The paper referred to does not give the time from when symptoms first occurred to the serum tests being performed. The finding of a sensitivity of 55% for serum amylase in patients with acute pancreatitis because of alcohol suggests that this was undertaken on average 2–3 days after the start of the attack.

Although not all our patients had confirmed evidence of the attack, it is worth pointing out that the conclusions in those that did (298 patients) differed little from those that did not (119 patients). In fact, all but four of the patients had at least one ultrasound examination in our study but in most failure to visualise the pancreas because of gas present implied an accurate diagnosis. Gumaste et al found ultrasound accurately diagnosed acute pancreatitis in 25 of 32 (78%) patients. It would be important to know how many patients were in fact patients with chronic pancreatitis presenting with an exacerbation. This type of patient was excluded from our study and may fundamentally affect the interpretation of results.

Nevertheless, the finding of a highly sensitive and specific diagnostic rate using the Kodak Ektachem slide lipase test in this difficult group of patients appears to be an important advance. Comparative assessment in a British group of patients is warranted.

J P NEOPTOLEMOS

Colonic tuberculosis

EDITOR.—I have read with interest the report by Shah et al on colonic tuberculosis (Gut 1992; 33: 347–51). In the opening statement of the discussion, the authors state that the caecum is most affected in colonic tuberculosis. But, it is the ileocolic region that is not affected, that is involved in tuberculosis. In colonic tuberculosis, involvement of the ileocaecal region is a distinct entity and should be considered separate from small bowel (ileal and jejunal) and large bowel (colonic) tuberculosis. Even in their own results, the authors state that 35 of 50 patients had involvement of the ileocaecal region. Another recent report commented the same semantical error and described 60 patients of “large bowel” tuberculosis to be included as many as 49 with ileocolic involvement.

The histological hallmark of tuberculosis, central caseation in a granuloma, was seen in only nine patients. The diagnosis in at least some of the 24 patients with non-caseating granulomas, seven with agglomeration of epithelial cells and five with chronic inflammation could as well be non-specific enteritis or Crohn’s disease, which though uncommon is not unknown in India.

The authors state that in most colonic (including ileocaecal) lesions, the differential diagnosis included Crohn’s disease then justified in advocating anti-tubercular chemotheraphy even when colonicoscopic biopsy examination is negative for tuberculosis? It would have been interesting to know how many patients who were clinically suspected to have tuberculosis, but had negative colonicoscopic biopsy for tuberculosis, finally turned out to have other diseases like neoplasia, lymphoma, Crohn’s disease, etc. Also coexisting tuberculosis and carcinoma is not unknown. Moreover, empirical anti-tubercular chemotherapy, in the absence of definite histological diagnosis of tuberculosis, may create problems of diagnosis later, as it changes the histological picture so that differentiation from Crohn’s disease becomes difficult.

Patients with ileo-caecal and colonic tuberculosis usually present with lump or features of severe acute intestinal obstruction. In patients with colonic tuberculosis, a third presented with subacute intestinal obstruction and a quarter had lump. Anti-tubercular chemotherapy causes healing which is accompanied by varying amounts of fibrosis and may result in further narrowing of the already compromised lumen and increase the risk of aggravating the obstruction. The authors state that all patients responded to anti-
Serum amylase levels and acute pancreatitis.

V Gumaste

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