Adult coeliac disease, dermatitis herpetiformis and smoking

EDITOR.—Snook and colleagues report that cigarette smoking seems to exert a protective effect against the development of adult coeliac disease (Gut 1996; 39: 60–2). Dermatitis herpetiformis is an uncommon blistering skin disease of unknown aetiology. Importantly, gluten sensitive enteropathy is present in almost 100% of patients after gluten loading, giving rise to the hypothesis that coeliac disease and dermatitis herpetiformis may share a common pathogenesis. Several studies have suggested that a possible link between dermatitis herpetiformis and cigarette smoking exists. Since then, examination of coeliac disease and dermatitis herpetiformis in adults has also been shown (Gut 1990; 31: 821–4).

Risk factors for pancreatitis

EDITOR.—Dr De Beaux, Carter, and Palmer in their thoughtful provocative editorial have examined a number of possible risk factors for pancreatitis occurring after ERCP (Gut 1996; 38: 799–800).

However, several studies have failed to show that factors commonly thought to be at fault do actually present a risk, for example, the type of contrast used in the examination of the pancreas, bacteraemia, 3,4 Sphincter of Oddi manometry does seem to be a risk factor5 as does sphincterotomy for stones, although this does not seem to be related to duct clearance. It is clear therefore that particularly for diagnostic ERCP, the cause of post-ERCP pancreatitis is unknown. We would like to propose the possibility that glutaraldehyde residues remaining after endoscopy cleaning, could be at fault.

This hypothesis is based on our own experience following an outbreak of pancreatitis related to changes in our endoscopic cleaning methods. Our usual rate of diagnostic pancreatitis of approximately 1% increased to 15% with a change from manual cleaning and use of the Keymed autoclave (Keymed, Stock Road, Southend on Sea, Essex) to an automatic closed circuit washing machine (Cus- toms Ultrascopes Automatic System 83-2, Specialist Endoscopy Equipment, Ormskirk, Lancashire). After a worrying six week period and based on the knowledge that even small spaces of gluing agent toxic to mucous membranes cancolonic epithelium, we very carefully examined our change in cleaning practice. It became apparent that rinsing of the elevator wire channel (forceps rete) was possibly less than adequate, after automatic cleaning with glutaraldehyde. Our practice now is to rinse the elevator wire channel manually with at least six rinses of sterile water. Since then, examination of our pancreatitis rate for the past year has shown that of 309 ERCPs, there was one case of pancreatitis in 174 diagnostic ERCPs (including 123 pancreateograms). An incidence of 0-6%. The one case was in a 15 year old girl with recurrent pancreatitis. 1% of 74 patients undergoing sentin ent placement, with or without access sphincterotomy, there was one case of pancreatitis (1-3%). There were four cases of pancreatitis among 61 sphincterotomies for stones (an incidence of 6-5%). This appeared unrelated to success or otherwise of duct clearance. Four cases were mild with discharge of the patient from hospital within five days. One case where duct clearance was inadequate was however, severe.

We believe that there is circumstantial evidence to suggest that glutaraldehyde might be implicated in the aetiology of post-diagnostic ERCP pancreatitis. Proof of this hypothesis will require an animal model, but in the meantime we recommend that units carrying out ERCP, carefully evaluate their rinsing procedures and consider the possibility of changing them if they are found to be less than satisfactory.

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LETTERS TO THE EDITOR

Gastric ulcers

EDITOR.—Interest in the effects of corticosteroids on the healing of gastric ulcers goes back a long time. The paper by M Carpani deKaski and colleagues is a valuable contribution to this field which demonstrates that corticosteroids do reduce the regenerative repair of the epithelium in experimental cryoprobe-induced ulcerations in the rat stomach (Gut 1995; 37: 613–6). The earliest work they cite on the healing of experimental ulcer with prednisone is that of Kuwayama and Eastwood, published in 1988.1 But their memories do not go back far enough. My colleagues and I, with Franklin Hollander, and I presented what we believed was the only extant report at that time (1957) on the effects of cortisone and corticotropin on the healing of gastric ulcers in an experimental study in the dog. Using explants of the entire gastric wall of this species transplanted to the anterior abdominal wall, and protected by a mechanical metal guard, we induced circular ulcers whose diameters ranged from 5 to 25 mm and included the muscularis mucosae. Doses of cortisone of 10 or 20 mg/kg, or ACTH of 5 or10 mg/kg, significantly delayed the healing but did not completely inhibit it. So now, infinitely improved, we may report on some of the mechanisms that may be operative in the healing of human gastric ulcers is indeed a welcome addition to our knowledge.

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