LETTERS TO THE EDITOR

Oral submucous fibrosis—a chronic disseminated intravascular coagulation syndrome with local coagulopathy

EDITOR,—We have read the leading article in Gut with great interest. (Gut 1992; 33: 4-6).

We were, however, disappointed as the work on oral submucous fibrosis published in Indian journals1 and the American Journal of Clinical Pathology2 has not been covered by the authors. After working for about 15 years on oral submucous fibrosis, we cannot agree with the views expressed.

According to Jayanthi et al, oral submucous fibrosis has been attributed to local irritation caused by tobacco and chillies used in cooking and that the progression of the disease can be halved by stopping tobacco, pan, etc but it does not seem to be that simple or straightforward.

We have seen young female patients who were not exposed to such iritants and they still developed the disease.

We have seen that oral submucous fibrosis is a chronic disseminated intravascular coagulation syndrome but it is well compensated for in most patients. We have shown that there is a thrombin like substance identified as fibrin producing factor in the saliva of patients suffering from oral submucous fibrosis.3 This is in contrast with the findings in normal saliva.

In normal saliva fibrinolitic substances have been shown, however, we have found considerable amounts of proactivator and plaminogen in mixed, parotid, and submaxillary sublingual saliva. Several centres in India have confirmed the presence of fibrin producing factor in oral submucous fibrosis (personal communication).

In 1984, using a haemagglutination inhibition technique (Wellcome Kit HA:14) we showed that fibrinogen/fibrin degradation products—which we prefer to call molecules immunologically similar to fibrinogen (MISFI) were detected both in the plasma and sera of the patients with oral submucous fibrosis.4 These MISFI were like fibrin monomers, because paracoagulation tests were positive and erythrocyte aggregations were present.

In addition to the discovery of MISFI we have done global or first line clotting time tests in oral submucous fibrosis (unpublished data). These screening tests—activated partial thromboplastin time, prothrombin time, and thrombin time—yield interesting information. The clotting times of oral submucous fibrosis are either prolonged or normal or even shortened.

When working on oral submucous fibrosis plasma, it is important to appreciate that there is a strong tendency for the formation of cryofibrinogen so that tests should be performed on fresh plasma and sera. When the plasma is stored at −20°C, cryofibrinogen can develop in a matter of two to six hours.

We find cryofibrinogen in almost all patients with oral submucous fibrosis. The presence of cryofibrinogen suggests that a small quantity of thrombin like material is being added to the circulating plasma.5 Our data suggest that this thrombin like procoagulant is present in the saliva of patients with oral submucous fibrosis.

Furthermore, we are tempted to suggest that the varying clotting times, the presence of MISFI (or fibrinogen/fibrin degradation products) and cryofibrinogen should point to hypercoagulation. According to Wintrobe6 hypercoagulation and chronic disseminated intravascular coagulation are superficially different but basically the same phenomena and disseminated intravascular coagulation is virtually always associated with fibrinolysis.

Oral submucous fibrosis seems to have a genetic basis. It is suggested that in oral submucous fibrosis the local fibrinolytic mechanism in the oral cavity is defective and cannot match or matches imperfectly with the hypercoagulation produced by the fibrin producing factor.

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Concentrations of 5-ASA and AC-5-ASA in human ileocolonic biopsy homogenates

EDITOR,—I read with interest the paper by (Gut 1992; 33: 1338-42) Vos et al. This is a new approach to the study 5-ASA compounds in human subjects and is necessary to correlate these concentrations with clinical benefit in inflammatory bowel diseases.

Firstly, I would like to comment about the experimental protocol. In their study, Vos et al state that their patients a laxative to colonic ileocolonic biopsy and to a lesser extent of Salazopyrin and the release modified drugs.

We did not study the influence of the washout on the mucosal concentrations of 5-ASA after Asacol and Claversal 500 because we did not expect

Reply

EDITOR,—We thank Dr Phatak for his interest shown in our paper on oral submucous fibrosis. The article was based on the work done in the Indian consortium on factors that might be responsible for oral submucous fibrosis and placed in perspective the impact that 'betel nut' and 'betel leaf' (pan) consumption would have among south Asians in the United Kingdom.

Oral submucous fibrosis is a multifactorial disease, some factors are initiators, others cofactors, and some promoters. Dr Phatak's suggestion of a defect in the fibrinolytic mechanism in genetically predisposed subjects is well taken, but their hypothesis does not suggest the group which needs screening for fibrin producing factor. Also the ill effects of 'betel nut' chewing cannot be readily dismissed and legislations to ban their import in the United Kingdom and those in Indian subcontinent cannot be over emphasised.

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Reply

EDITOR,—We agree with Chausssade and Sogni that the influence of the washout on our results remains a difficult problem. We emphasise that we used the washout only to facilitate the endoscopy and not to mimic a condition similar to ulcerative colitis.

We agree that because of the mechanical removal of the intraluminal contents, an impairment in the splitting of the azo bond can be expected for Dipentum and Salazopyrin. The comparison of mucosal concentrations of 5-ASA and AC-5-ASA is not sufficient to assess the influence of this drug in inflammatory bowel disease.


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A G Phatak

Gut 1993 34: 713
doi: 10.1136/gut.34.5.713

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