Correspondence

Essential fatty acids and peptic ulcer disease

Sir,—We read with interest the reply by Hollander and Tarnawski on the role of dietary essential fatty acids in peptic ulcer disease (Gut 1987; 27: 914–6). To support our contention that essential fatty acids themselves could be protective without conversion to prostaglandins, we would like to add the following:

In a recent study Huang et al.1 have shown that aspirin induced gastric haemorrhage can be prevented by gamma linolenic acid (GLA) enriched diet. They have also shown that the concentrations of linoleic acid (LA) were significantly increased, whereas those of arachidonic acid (AA) were decreased in plasma and liver phospholipids of aspirin treated animals fed linoleic acid. This according to Huang et al. suggest diminished desaturation and elongation of LA to AA. In addition, they have also shown that treatment with GLA prevented these changes. As they used aspirin in their studies, the question of conversion of GLA to prostaglandins cannot be the mechanism for the protective effect of GLA on aspirin induced gastric haemorrhage as aspirin is a powerful cyclo-oxygenase inhibitor. The results of these experiments also indicate that AA concentrations are low in animals with gastric haemorrhage. Further, it is known that aspirin can block the activity of the enzyme delta-6-desaturase which is necessary for the conversion of LA to GLA. This implies that a deficiency of AA and GLA as a result of the decreased activity of delta-6-desaturase may play a crucial role in gastric ulceration and haemorrhage. This does not mean that prostaglandins do not have a role in peptic ulcer disease. We feel that in addition to prostaglandins, the role of essential fatty acids especially that of GLA, AA and EPA in peptic ulcer disease should not be pushed to the background. Further research is needed to prove or disprove our point.

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References


Neurofibromatosis and small bowel adenocarcinoma—an unrecognised association

Sir,—Further to the case report by T L Jones and T L Marshall (Gut 1987; 28: 1173–6) I would report a further case of a patient with peripheral neurofibromatosis and a periampullary tumour.

The patient, a woman now aged 67, presented with obstructive jaundice 10 years ago. At that time she had established neurofibromatosis, which also had affected her father. I carried out a local excision after which she remained well until 1984 when a further local resection was made of the ampulla. She remains alive and well when seen at follow up one month ago. Neurofibromatosis was present in the duodenum at both operations.

Unlike the reported cases, this patient has survived 10 years. Drs Jones and Marshall believe the association with small bowel cancer is unlikely to be caused by chance. Three of the five cases had carcinoma of the periampullary region which is also unlikely to be caused by chance.

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Books


Sir Christopher Booth needs no introduction as a spokesman for medical science, but, in these essays, he reveals other and less familiar facets of his ability. He is well known to the cognoscenti of medical history; this book should bring wider recognition of his skills both as scholar and writer. Most of the essays are devoted to 18th century medical scientists; his choice of personalities betrays his origins as a son of the Yorkshire Dales, his leaning towards non-conformism, and his commitment to the experimental method in medicine. His hero is Hermann Boerhaave of Leyden, lector in medicine at Leyden between 1701 and 1735, an exponent of the scientific method who directly or indirectly influenced many English physicians of the time. But Booth has not confined his attention to notables such as Boerhaave.
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