LETTERS TO THE EDITOR

Pigbel-like syndrome in a vegetarian in Oxford

EDITOR,—We were interested to read the case report by Farrant et al (Gut 1996; 39: 336–7) as we too had a similar recent report of unexplained ischaemic or necrotising enterocolitis. In our patient also the terminal ileum and caecum were primarily affected, and Gram positive organisms were identified on Gram staining of bowel wall. It seems, despite the background of tropical exposure we do not agree with Farrant et al’s interpretation of the findings as being consistent with Pigbel syndrome. The pathology of this condition is well described by Cooke and seems to affect the jejunum in a patchy fashion along the antimesenteric border and rarely involves the ileum, but never the colon. It is classically described as occurring rapidly following eating a rich meal in the setting of protein energy malnutrition, and the requirement for dietary trypsin inhibitors seems to be at the time of toxin ingestion rather than three months previously. The finding of Gram positive organisms in the bowel wall is not conclusive as clostralid species are ubiquitous in faecal flora, and indeed we have demonstrated their presence in the tissues of colectomy specimens with infection due to vascular thrombosis. The fact that Pigbel syndrome has been well controlled by the introduction of vaccination to the Clostridium perfringens type C β-toxin suggests that the presence of this organism or its toxin is necessary to make this diagnosis. The mere presence of Gram positive organisms in this case without toxicological proof, given the unusual circumstances and the unlikely distribution of the lesion, makes the presumptive diagnosis of Pigbel syndrome somewhat tenuous.

We would suggest instead that the clinical picture is more that of a “non-occlusive mesenteric ischaemia”-like syndrome which is known to have a predilection for the terminal ileum and caecum as in this case and has been associated with diarrhoea, ileus, vasoconstrictors such as cocaine, hypovolaemia, haematological malignancies, and even Marathan running. Although such cases are rare and the aetiology obscure, the presence of splanchic vasoconstriction and diminished circulating volume would seem to be critical and could have occurred in this case due to the combination of infective diarrhoea and physical training.

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Bone disease after liver transplantation should not be underestimated

EDITOR,—In their leading article on bone disease after liver transplantation (Gut 1996; 39: 505–7), Losowsky and Hussaini rightly emphasise the pathogenetic role of pre-existing osteopenia and osteoporosis. However, the suggestion that post-transplantation bone disease is “becoming less of a problem” does not accord with our own experience or that of others, who have reported marked incidence loss between 20 and 30% in the first year after transplantation.1,2

The clinical significance of post-transplantation bone disease lies in the morbidity associated with fragility fractures, with emphasis on bone mineral density as an indicator of disease may be misleading in this group of patients. During the early phase of bone loss, there is an increase both in bone turnover and the depth of osteopenia,2 which changes which will result in trabecular thinning and penning in cancellous bone, reducing its mechanical strength and increasing fracture risk. Although bone mineral density may be permanently compromised. In addition, the skeletal distribution of bone loss may be heterogeneous and measurements performed in the lumbar spine and proximal femur may not reflect loss in other parts of the skeleton—for example, the thoracic spine. These considerations emphasise the importance of using fracture, not bone density, as the main outcome of post-transplantation bone disease; the majority of fractures occur in the spine and, since as many as two thirds of vertebral fractures may be asymptomatic, vertebral radiographs both before and after transplantation are required to establish fracture incidence accurately. Losowsky and Hussaini state that in the study of Hawkins et al, no patient showed radiological or clinical evidence of vertebral collapse or hip fractures after transplantation, but spine radiographs were performed postoperatively in only 50 (61%) patients and fracture incidence may thus have been underestimated. The recent report from Lees’ contains no data on fracture incidence and the clinical significance of these changes in bone mineral density is thus unclear.

In common with the Lees group, we have found that the reduction in bone mineral density after liver transplantation is less than that reported in earlier studies,3,4 possibly as result of the smaller doses of glucocorticoids now used for immunosuppression. Nevertheless, the incidence of fractures during the first postoperative year remains high, resulting in significant long term morbidity and bone development of effective prophylactic strategies in these patients should be regarded as an important research priority.

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Gut 1997 40: 695
doi: 10.1136/gut.40.5.695

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