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 have had a similar recent case 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 in the bowel wall. However, 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 Cooke¹ 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 a protein 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 clostridial species are ubiquitous in faecal flora, and indeed we have demonstrated their presence in the tissues of colectomy specimens with infarction 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 -toxin² 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 caecum³ as in this case and has been associated with diarrhoea,⁴ ileus,⁵ vasoconstrictors such as cocaine,⁷ hypovolaemia, haematological malignancies,⁸ and even Marathon running.⁹ Although such cases are rare and the aetiology obscure, the presence of splanchnic 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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Reply

EDITOR,—Drs Woodward and Sanders state incorrectly that Pigbel syndrome never affects the colon. On the contrary, any part of the small or large intestine may be involved.^{1,2} The time scale of the changes in diet has been misunderstood. We stated in our report that shortly before becoming ill, our patient had consumed a large amount of fish, fermented soybean and peanut butter sauce. In the discussion we covered the fact that fermented soybean and peanut butter sauce contain antitrypsins. We agree that serological proof would have been ideal in confirming the diagnosis. However, 50% of cases of Pigbel syndrome are serologically negative for antibody to the β -toxin of *Clostridium perfringens* type C. We therefore felt justified in labelling the case "Pigbel-like syndrome". We do not agree with the suggestion that our case was one of "non-occlusive mesenteric ischaemia". The patient had been well until two weeks before admission. There was no preceding illness causing hypovolaemia. There was no history of use of cocaine. Her strenuous exercise predates her illness. At the time she became unwell she had been resting and not taking strenuous exercise.

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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 a fracture incidence between 20 and 30% in the first year after transplantation.^{1–5}

The clinical significance of post-transplantation bone disease lies in the morbidity associated with fragility fractures, and undue 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 cavities eroded by osteoclasts,⁶ changes which will result in trabecular thinning and penetration in cancellous bone, reducing its mechanical strength and increasing fracture risk. Although bone mineral density shows a tendency to return towards baseline values six months to one year after transplantation,⁷ the loss of connectivity resulting from these early changes in bone remodelling is unlikely to be reversible and bone strength 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 bone 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*,⁸ no patient showed radiological or clinical evidence of vertebral collapse or hip fractures after transplantation, but spinal radiographs were performed post-operatively in only 50 (61%) patients and fracture incidence may thus have been underestimated. The recent report from Leeds⁹ contains no data on fracture incidence and the clinical significance of the observed changes in bone mineral density is thus unclear.

In common with the Leeds group, we have found that the reduction in bone mineral density after liver transplantation is less than that reported in earlier studies,⁵ possibly as a 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 the development of effective prophylactic strategies in these patients should be regarded as an important research priority.

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