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 had a similar recent case of unexplained ischaemic or necroting enterocolitis. In our patient also the terminal ileum and caecum were primarily affected, and Gram positive organisms were identified on Gram staining of 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 Cooke and seems to affect the jejunal 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 the parent 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 coloectomy 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 Marathorn 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.

J M WOODWARD
D S A SANDERS
Queen Elizabeth Hospital,
Edgbaston,
Birmingham B15 2TH


Reply

EDITOR,—Dr 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

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 is unusual but does contain anti-trypsins. 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 justifiably 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 predisposes her illness. At the time she became ill she had been resting and not taking strenuous exercise.

J M FARRANT
Gastroenterology Unit, Royal United Hospital, Bath BA1 1NG

Z TRAIL
Department of Radiology, John Radcliffe Hospital, Oxford

C COLON
Nuffield Department of Medicine, John Radcliffe Hospital, Oxford

B WARNEN
Department of Histopathology, John Radcliffe Hospital, Oxford

N MORTENSEN
John Radcliffe Hospital, Oxford

F V GLEESON
Department of Radiology, John Radcliffe Hospital, Oxford

D P JEWELL
Gastro-intestinal Research Unit, Radcliffe Infirmary, Oxford

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 no net increase loss between 20 and 30% in the first year after transplantation.1–3

The clinical significance of post-transplantation bone disease lies in the morbidity associated with fragility fractures, and the 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 bone osteoclasts,4 changes which will result in trabecular thinning and penetration in cancellous bone, reducing its mechanical strength and increasing fracture risk. Although bone loss may be permanent, it is unlikely that 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 changes 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,5 no patient showed radiological or clinical evidence of vertebral collapse or hip fractures after transplantation, but spine radiographs were performed post-operatively in only 50 (61%) patients and fracture incidence may thus have been underestimated. The recent report from Lees’6 contains no data on fracture incidence and the clinical significance of these changes in bone mineral density is 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,7 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 loss of effective prophylactic strategies in these patients should be regarded as an important research priority.

J E COMPSTON
G A ALEXANDER
Department of Medicine,
University of Cambridge Clinical School of Medicine,
Addenbrooke’s Hospital,
Cambridge CB2 2QG

Reply

Editor,—The message from our leading arti-
cle in this issue and the recent article in the Journal of Bone and Joint Surgery (Carr J 2000;82-B:715) on bone transplantation are quite timely. We do not deny that bone disease after transplantation is a problem. However, we wish to emphasise that with better selection of patients, for liver transplantation, shorter hospital stays and the use of steroids in lower dosages, with steroid withdrawal in some pro-
grammes, there is a lower morbidity asso-
ciated with post-transplantation bone disease compared with a decade ago.

We agree that bone fracture is an important endpoint with regard to post-transplantation bone disease. However, we suggest that the symp-
tomatic bone fracture is the most important endpoint for the morbidity related to post-
transplantation bone disease. Thus, the paper by Hawkins and co-workers, although possibly underestimating asymptomatic spinal frac-
tures, does accurately reflect the rate of symptomatic fractures after transplantation. In an own series (unpublished data) the rate of symptomatic bone fractures was low, with only eight (14%) of 54 patients experiencing frac-
tures. One patient experienced a fracture of the lumbar spine; the remaining patients had femoral neck fractures. We agree that all patients did not undergo systematic axial and spinal screening radiology, we may have under-
estimated the rate of asymptomatic fractures. None the less, a fracture rate of 14% is substan-
tially lower, compared with 65% of patients sustaining at least one fracture in the first three months after transplantation.1 Thus, the com-
bination of a low symptomatic fracture rate combined with a reduction in the loss of bone mineral density, compared with earlier studies, leads to the suggestion that post-transplantation bone disease is “less of a problem”.

We concur that bone disease and fracture after liver transplantation are still important contributors to post-transplant morbidity and thus important for further research. However, we emphasise further the need to re-assess the magnitude of the problem, in the light of current clinical practice.

S H HUSSAINI
Lecturer in Medicine
M LOSOWSKY
Emeritus Professor of Medicine and Head, Academic Division of Medicine, Level 7, Clinical Science Building, St James’s University Hospital, Leeds LS9 7TF

1. Eastell R, Dickson ER, Hodgson SF, Wiener RH, Porysky MK, Wahner HW, et al. Rates of vertebral bone loss before and after liver transplantation in women with pri-

BOOK REVIEWS


My first reaction on receiving this book was “Not another book on bile duct stones”. I find it remarkable and remarked to myself on the large numbers of black and white radiographs of the biliary tree taken by all kinds of techniques: laparoscopy and ERCP in particular. There were also quite a few colour photographs. However, I have taken dozens of laparoscopic biliary tree photographs which could have been of better quality. I scanned one chapter and noted three things that irritated me: the author consistently confused the common bile duct with the com-
mon hepatic duct and C with F; B, most irksome was to refer to radiological contrast as a “dye” (visions of my teenage children and what they do to their hair) I put the book to one side.

Later, I went through the list of the 24 multidisciplinary contributors drawn princi-

cally from North America but also Europe, Australia and South Africa – it was a distin-
guished list. I then felt that this book might be useful after all. After digesting several chapters I (rarely read a book in sequence), I realised that my initial unfavourable impres-
sions were misplaced.

Gastroenterologists frequently give the view that the therapeutic revolution of bile duct stones with the widespread introduction of ERCP was final. But then at one point (only five years ago) ESWL was believed to be the answer to gallstone problems. However, such an expectation is underestimation.

Thus, now there emerged laparoscopic bile duct exploration. Many surgeons viewed this as a gimmick. Moreover, gastroenterologists often castigated the innovators, who spent two to three hours undertaking a laparoscopic chol-
edocholithotomy when this could be done by ERCP in “five minutes”. Any such negative views are effectively destroyed by this book.

For the “theoretical” gastroenterologist, this book defines multiple areas in which ERCP should be used and brings together powerful arguments for the primary use of laparoscopic bile duct exploration (notwithstanding the established roles of ERCP in acute cholangitis and in acute pancreatitis, in elderly unfit patients and in patients with a repaired or recurrent bile duct stone). For the surgeon, it is an outstanding technical manual.

For the practising gastroenterologist, it is extremely valuable in helping to understand the kind of problems that may be created by laparoscopic surgery and the difficulties in contributing to possibly diagnostic or therapeutic procedures as well as defining the roles of other imaging modalities such as computed tomog-
raphy scanning.

The references are remarkably up to date and highlight the enormous advances and accelerating worldwide experience in laparoscopic bile duct surgery. This book needs to be read by every trainee and every surgeon undertaking surgery for gallstones. Several chapters alone


Magnetic resonance cholangiopancreatography (MRCP) is a relatively new diagnostic investigation that is becoming quite rapidly used in clinical practice. This book aims to convey details of the techniques, results and clinical indications. This is quite a challenging task for the author but the book is very comprehensive in terms of clinical and technical evaluation of MRCP, and the information presented is a distillation of current published work combined with a good measure of the personal experience and views of the authors.

And large, their aim is achieved in a series of short chapters that discuss the details of the various MRCP techniques in current use and the place of MRCP in most clinical situations. Only MR radiologists are likely to benefit from the physics and techniques chap-

ter, but the other chapters are more widely accessible. The evaluation of jaundice is the area most studied using MRCP and the potential to replace diagnostic ERCP in many situations is illustrated. This is reinforced in the chapter on choledocholithiasis which puts MRCP in the context of other diagnostic techniques, although I was surprised to see no discussion of CT contrast cholangiographic methods. Benign and malignant biliary steno-

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Bone disease after liver transplantation should not be underestimated.

J E Compston and G A Alexander

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