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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Reply

EDITOR,—The message from our leading article was not to underestimate bone disease after liver transplantation, but to put the problem in perspective with regard to the current practice of liver transplantation. We do not deny that bone disease after transplantation remains a problem. None the less, we suggest 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 programmes, there is a lower morbidity associated 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 symptomatic 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 fractures, does accurately reflect the rate of symptomatic fractures after transplantation. In our own series (unpublished data) the rate of symptomatic bone fractures was low, with only eight (14%) of 54 patients experiencing fractures. One patient experienced a fracture of the lumbar spine; the remaining patients had femoral neck fractures. We agree that as all patients did not undergo systematic axial and spinal screening radiology, we may have underestimated the rate of asymptomatic fractures. None the less, a fracture rate of 14% is substantially lower, compared with 65% of patients sustaining atraumatic fractures in the first three months after transplantation.¹ Thus, the combination 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 warrants further research. However, we emphasise further the need to re-assess the magnitude of the problem, in the light of current clinical practice.

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

mary biliary cirrhosis. *Hepatology* 1991; 14: 296-300.

BOOK REVIEWS

Bile ducts and bile duct stones. Berci G, Cuschieri A. (Pp 179; illustrated; £80.00.) Philadelphia: WB Saunders, 1996. ISBN 0-7216-1488-4.

My first reaction on receiving this book was "Not another book on bile duct stones". I flicked through the pages 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 principally taken during laparoscopy 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 common hepatic duct and "C with 'F'; the 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 principally from North America but also Europe, Australia and South Africa – it was a distinguished list. I began to feel this book might be useful after all. After digesting several chapters (I rarely read a book in sequence), I realised that my initial unfavourable impressions 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 gallstones before laparoscopic cholecystectomy was developed. Then, slowly there emerged laparoscopic bile duct exploration. Many surgeons viewed this as a gimmick. Moreover, gastroenterologists often castigated the innovative surgeon for spending two to three hours undertaking a laparoscopic choledocholithotomy 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 when called upon to contribute diagnostically or therapeutically by ERCP (as well as defining the roles of other imaging modalities such as computed tomography 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

("The role of preoperative ERCP and sphincterotomy in the laparoscopic era", "The unsuspected bile duct stone", "Preoperative endoscopic sphincterotomy", "Management of retained stones", "Early diagnosis of bile leakage", "Endoscopic management of postoperative bile leaks", and "A cost-effective approach to the treatment of common bile duct stones with surgical versus endoscopic techniques") also make this book a must for practising gastroenterologists.

J P NEOPTOLEMOS

MR Cholangiopancreatography Techniques. Results and Clinical Indications. Edited by P Pavone, R Passariello. (Pp 150; illustrated; DM 148.00.) Berlin: Springer Verlag, 1997. ISBN 3-540-61349-8.

Magnetic resonance cholangiopancreatography (MRCP) is 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 challenge given the rapid progress that is occurring both in techniques and clinical 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.

By and large, their aims are 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 chapter, 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 tests, although I was surprised to find no discussion of CT contrast cholangiographic methods. Benign and malignant biliary stenoses are well covered and the particular diagnostic advantages of MRCP in "mapping" the biliary tree well illustrated. Pancreatography receives less attention, reflecting the problems of spatial resolution and of correlating anatomical changes with clinical features of pancreatitis, although pseudocyst demonstration by MRCP may well be of value in clinical practice. The value of MRCP in relation to laparoscopic cholecystectomy is appended somewhat as an afterthought and this is an area still lacking in large trials, preventing proper evaluation.

Strong points are the copious good quality illustrations and the numerous up to date references. A debatable point is the emphasis on the particular technique that the authors have used although this does not detract from their overall conclusions. The not infrequent grammatical, translation and spelling errors are irritating and can make the text somewhat challenging to read. An additional chapter on the potential limitations of the technique would have been helpful for the clinician and some feel for the likely future technical developments such as 3D breath-hold and dynamic functional studies would have been useful. Overall, this is a timely snapshot overview of a new diagnostic technique that will be of value to those involved in the investigation and management of pancreatic and biliary disease.

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