Letters to the Editor

Metastatic hypernephroma from a native kidney after cadaver renal transplantation

Sir,
Cystic degeneration of kidneys in patients with end-stage renal failure and undergoing haemodialysis is well recognized. The incidence and extent of the cystic changes is related to the duration of the haemodialysis (Dunnill *et al.*, 1977). Forty five percent of patients with these changes can expect to develop a hypernephroma, but there is only one recorded case of these tumours metastasizing. (Ratcliffe *et al.*, 1983). A search through the literature and communication in the Transplant Tumour Registry has failed to reveal any previous reports of metastases from tumours in the native kidneys in patients who have undergone successful cadaver renal transplantation.

A 67 year old male developed end-stage renal failure due to a combination of diabetes mellitus, gout and hypertension. He commenced haemodialysis in January 1980, and received a cadaver renal transplant six months later. He remained well for three and a half years, when he presented with recurrent urinary infections.

Examination revealed an obese man with signs of peripheral vascular disease. There was a hard, fixed lymph node in the right supraclavicular fossa. Biochemical investigations showed good graft function. Serum creatinine was 95 nmol/l. Chest radiograph showed multiple lesions throughout both lung fields. Abdominal CT scan showed a mass at the inferior pole of his right native kidney. No abnormality was seen in the transplant kidney. A diagnosis of metastatic hypernephroma was made.

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References

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Gallstone perforation of the ileum without obstruction

Sir,

The case report (Shankar *et al.*, 1984) of gallstone perforation of the ileum without obstruction was described as a sequel to asymptomatic gallstone impaction. Although the distal migration of a gallstone within the small bowel lumen is often asymptomatic, gallstone impaction must result in obstruction. The commonest site for this to occur is in the terminal ileum and it is the delay in presentation of such a distal small bowel obstruction which gives rise to the poor clinical condition of patients and high operative mortality (Van Landingham & Broders, 1983). At laparotomy, it is not uncommon to find pressure necrosis at the site of impaction and with increasing tension within the ileal wall it is not surprising that perforation may occur either spontaneously or with surgical manipulation. Indeed, studies have shown that with an increase in faecal organisms due to obstruction, mucosal necrosis occurs early (Ellis, 1982). Perforation will allow the bowel to decompress with loss of the radiographic features of obstruction and the clinical picture changes to that of peritonitis. Clinical deterioration is rapid due to release of large volumes of small bowel content with a high concentration of faecal organisms consequent upon the obstruction (Ellis, 1982). This sudden severe peritonitis with septicaemia may be the initial clinical presentation of 'gall-stone ileus'. Recent experience of such a case is presented.

A 74 year old woman presented with a 6 d history of lower abdominal pain of sudden onset. Vomiting had been an early feature without abdominal distension. There was no past history of biliary tract disease. On examination she was dehydrated and toxic with signs of generalized peritonitis and a blood pressure of 70/50 mm Hg. Abdominal X-rays showed signs of a generalized ileus with gas and fluid in non-dilated small bowel and gas was present throughout the colon. After resuscitation, laparotomy was undertaken. A longstanding peritonitis was evident and a perforation was identified in the terminal ileum adjacent to a large gallstone. A cholecystoduodenal fistula was present containing a palpable gallstone and two further gallstones were present within the gallbladder. The gallstone was removed from the terminal ileum and the perforation closed transversely with sutures. The presence of further gallstones dictated cholecystectomy with excision of the fistulous tract and closure of the duodenum. Despite aggressive resuscitative measures including tetracycline, peritoneal lavage and systemic broad spectrum antibiotics (cefuroxime and