Page kidney: successful radiological management of acute renal failure

Sir,

Page kidney is the external compression of a kidney. The condition is usually caused by a subcapsular haematoma, associated with high blood pressure and occasional renal failure, as described by Irwin Page in an animal model with cellophane papers wrapped round the kidney in 1939 and, subsequently, clinically in 1955 [1,2]. The present case illustrates, in a patient with a single functioning kidney, the successful management of a spontaneous subcapsular haematoma that was causing both acute renal failure and hypertension.

A 36-year-old female presented with a left-sided loin pain. At the age of 2 years she had undergone right nephrectomy for a Wilms tumour and left-sided lower lobectomy for pulmonary metastatic disease, followed by chemotherapy and radiotherapy. Six months before presentation, following her first pregnancy, she was diagnosed with dilated cardiomyopathy, for which she was given candesartan, carvedilol and warfarin.

She was in pain, with tenderness over the left costo-vertebral angle. Her blood pressure was 94/57 mmHg. Investigation showed INR 2.7, C-reactive protein 20 mg/l and serum creatinine 136 μmol/l.

An ultrasound scan revealed a mixed reflective collection, adjacent to the postero-lateral aspect of her left kidney. Spectral Doppler analysis of the segmental arteries within the kidney showed a high-resistance pattern with complete loss of normal diastolic flow; but the renal vein was patent. A non-contrast computed tomography (CT) scan confirmed the finding of a subcapsular haematoma (Figure 1).

In the following 48 h she became hypertensive, oliguric and her serum creatinine rose to 508 μmol/l. Drainage was arranged.

After reversal of anticoagulation, an 8F pigtail catheter was inserted percutaneously into the haematoma under ultrasound guidance and 100 ml sero-sanguineous fluid was drained. Ultrasound after 24 h showed a reduction in size of the haematoma and an improvement in diastolic blood flow within the kidney. Over the subsequent week, the creatinine fell to 102 μmol/l, with further improvement in diastolic flow. The patient’s blood pressure, however, remained high (180/100 mmHg), requiring more antihypertensive medications. A renal angiogram, performed subsequently, was normal.

Her acute renal failure was presumably due to a decreased perfusion of the single kidney as a result of pressure exerted by the subcapsular haematoma. Pre-existing warfarin therapy may have been a contributory factor [3]. Relief of the pressure and restoration of blood flow by percutaneous drainage led to the recovery of renal function.

Previously, the treatment of Page kidney has been exclusively surgical, but recently, laparoscopy- and radiology-assisted drainage has been used successfully [4,5]. However, radiology-assisted drainage of a subcapsular haematoma in a single functioning kidney has not been reported previously.

Management of hypertension with Page kidney has often involved nephrectomy [6], which we were able to avoid, in this patient with a single kidney, by using more antihypertensives and careful fluid management.

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1Department of Renal Medicine
St George’s University of London
London, UK
Email: Debasish.Banerjee@stgeorges.nhs.uk

2Department of Radiology
St George’s University of London
London, UK
Email: Rebecca.Suckling@stgeorges.nhs.uk

3Department of Cardiology
St George’s University of London
London, UK
Email: James.Pilcher@stgeorges.nhs.uk

4Department of Surgery
St George’s University of London
London, UK
Email: John.B.Eastwood@stgeorges.nhs.uk

5Department of Oncology
St George’s University of London
London, UK
Email: Iain.A.MacPhee@stgeorges.nhs.uk

6Department of Endocrinology
St George’s University of London
London, UK
Email: Fhorkan.Uddin@stgeorges.nhs.uk

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Long-term lamivudine therapy is not reasonable for HBV-associated nephropathy

Sir,

With the article by Izzedine et al. [1] recently published in Nephrology Dialysis Transplantation, we feel the continued treatment with lamivudine for as long as possible after initial remission is not reasonable, because the proportion of patients with a documented lamivudine-resistant mutation increases from 23% in year 1 to 65% in year 5 [2,3]. Patients with lamivudine-resistant mutations experienced significantly
more flare-ups of hepatitis than patients without \((P < 0.005)\) [2]. Although Tang et al. [4] described one patient who had a relapse of nephropathia after 2 years of complete remission when lamivudine was withdrawn, there no patient whose nephropathy was in complete or partial remission and who was being treated by an angiotensin-converting enzyme inhibitor at 12 months ended up with end-stage renal disease by 3 years of follow-up. It is supposed that the renal function is conserved when patient’s proteinuria declines into remission levels. Therefore, long-term lamivudine therapy for the patients with HBV-associated nephropathia should be considered unnecessary, perhaps even unreasonable. Although the optimal duration of treatment and the criteria for stopping it have not been established, maintaining lamivudine therapy for 4–6 months following chemotherapy was suggested [5].

We would like to reinforce the point that long-term lamivudine therapy should be used only for patients who do not experience remission under supportive treatment, or for those with relapse of nephropathia after lamivudine withdrawal, so as to prevent lamivudine-resistant mutations and hepatitis flare-ups.

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A renal transplant patient with a solitary plasmacytoma in the oral cavity

Sir,
Post-transplant lymphoproliferative disorder (PTLD) is a serious complication of transplantation because it can occur early after transplantation and carries a high morbidity and mortality. Not more than 4% of the malignant tumors detected in organ recipients are plasmacytomas [1,2]. Although the upper respiratory tract and oral regions are favourable sites for the extramedullary or solitary plasmacytoma, solitary plasmacytoma after a renal transplantation is extremely rare [3,4]. We report a renal transplant patient with a solitary plasmacytoma of the mandible that developed 12 years after a living related renal transplantation. A 26-year-old male patient presented with a mild gingival enlargement in the left mandibular molar area. His medications were cyclosporine A (CsA) 200 mg/day, azathioprine 50 mg/day and methylprednisolone 6 mg/day. Physical examination revealed no abnormalities except a buccogingival mass measuring 1×1 cm between the lower left first and second molar teeth. Laboratory investigation revealed: an erythrocyte sedimentation rate 98 mm/h, leukocytes 6910/μl, haemoglobin 10.7 g/dl, serum creatinine concentration 2.1 mg/dl and proteinuria 0.5 g/day. An excisional biopsy was performed and microscopic examination revealed diffuse infiltration of plasma cells with kappa light chain monotype. A pathologic diagnosis of a plasmacytoma was made. Bone marrow biopsy and examination for multiple myeloma revealed no evidence of systemic involvement. Azathioprine was stopped. CsA was switched to rapamycin and surgical excision was performed. After six months of outpatient clinical follow-up, a new buccogingival mass measuring 1×1 cm between the lower right first and second molar teeth was detected. An excisional biopsy was performed again and microscopic examination revealed diffuse infiltration of plasma cells with kappa light chain monotype. Immunosuppressed organ allograft recipients are at risk of developing lymphoproliferative disorders as a consequence of immunosuppressive therapy and long-term antigenic stimulation from both the graft and possible viral infections. In this rare case, although the PTLD regressed after changing the immunosuppressive treatment with concurrent excisional surgery, solitary plasmacytoma recurred in a different site.

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Yasar Caliskan
Department of Internal Medicine
Istanbul School of Medicine
Istanbul University
Istanbul, Turkey
Email: ykcaliskan@yahoo.com

Mehmet Sukru Sever
Istanbul University
Istanbul School of Medicine
Department of Internal Medicine
Istanbul, Turkey
Email: ykcaliskan@yahoo.com

Yasemin Sahin
Department of Internal Medicine
Istanbul University
Istanbul, Turkey
Email: ykcaliskan@yahoo.com

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