Case Report

The reappearing kidney: an unusual complication of renal biopsy

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Introduction

In 1939 Page described impaired renal function due to compression of renal parenchyma accompanied by renal ischaemia and high renin hypertension [1]. In humans this is usually seen as a consequence of perinephric or subcapsular haematoma, often related to blunt trauma [2]. We present a case of 'Page' kidney following renal biopsy. This case was particularly unusual because of a reduction in proteinuria observed after temporary loss of function in the biopsied kidney.

Case

An asymptomatic, diet-controlled, diabetic 65-year-old smoker was referred for investigation of persistent proteinuria, 10 months after an uncomplicated anteroseptal myocardial infarct. Examination revealed a blood pressure of 160/90 mmHg with grade I hypertensive changes on fundoscopy. The patient was being treated with atenolol. No bruits were heard over major vessels and there were no missing peripheral pulses. Sacral oedema was noted.

A 24-h urine collection demonstrated a creatinine clearance of 76.5 ml/min and proteinuria of 9.6 g/24 h. Urea, electrolytes, creatinine, liver function tests, albumin, full blood count and clotting screen were all normal. Ultrasound examination showed two kidneys with bipolar diameters of 9 and 10 cm for the right and left kidneys, respectively. The left kidney was biopsied without immediate complications and the patient was discharged after 24 h. At no time was there any clinical evidence of bleeding or hypovolaemia. Following the procedure blood pressure was transiently elevated to 215/115 mmHg but had returned to baseline within 24 h without additional treatment.

On examination of the biopsy 6/12 glomeruli were normal, 2/12 were globally sclerosed and 4/12 demonstrated focal hyalinosis and mesangial sclerosis. Light-microscopy also revealed mild tubular atrophy and hyaline thickening of small arteries and arterioles. Electron-microscopy showed capillary basement membrane thickening without significant electron-dense deposits and patchy foot process fusion. Immunofluorescence was unremarkable and the appearances were most in keeping with focal segmental glomerulosclerosis (FSGS) with additional hypertensive or ischaemic changes.

In view of his smoking history, coronary artery disease and mild hypertension, the diagnosis of a renal artery stenosis (RAS) needed to be excluded. Serendipitously, a dynamic renogram [Tc-99m diethylamine penta-acetic acid (DTPA)] was performed 5 days after the biopsy. This demonstrated a non-functioning left kidney (Figure 1). A large perirenal haematoma was shown on ultrasound scan and Doppler studies suggested reduced left renal arterial flow. However, selective renal angiography showed no evidence of renal artery stenosis, and similarly, venography showed a patent vein. The serum creatinine remained unchanged and no further intervention was undertaken. The addition of a loop diuretic controlled the oedema without a significant reduction in blood pressure. A repeat DTPA scan at 2 weeks was unchanged but dynamic renography 8 weeks later revealed that the left kidney had recovered and contributed 30% of the total function. Throughout this recovery period the plasma creatinine did not alter appreciably but proteinuria fell to less than 1 g/24 h.

The patient has now been followed up for 4 years following the biopsy. Renal function has remained stable with a serum creatinine of 123 μmol/l and creatinine clearance of 62 ml/min. The most recent dynamic renogram (Tc-99m MAG3) shows that the left kidney is still responsible for 30% of the total renal function (Figure 2). However, the amount...
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whose research interests included hypertension and arteriosclerosis. In 1939 he reported the observation of renal ischaemia and high renin hypertension when dog kidneys were wrapped tightly in cellophane [1]. The clinical correlate of this experimental technique may occur in humans and is usually due to perinephric or subcapsular collections [2]. McCune et al. [3] reviewed 80 cases in the literature, 53 of which were clearly related to trauma, approximately half of these occurred as a result of sporting injuries. Cases following percutaneous biopsy of native [2–5] and transplant kidneys [6,7] are recorded in the literature.

The DTPA renogram performed almost immediately after the renal biopsy in our case was planned to investigate the possibility of renovascular disease. Without this investigation the complication is likely to have gone unrecognized.

Although hypertension is a cardinal feature of the experimental Page kidney, the blood pressure in our patient, treated with low-dose betablockade at presentation, was raised for less than 24 h following the biopsy. Hypertension is a feature of all the previous reports of Page kidney following native renal biopsy. However, the duration of hypertension and the ease with which it can be controlled are highly variable. Spontaneous resolution at 5 days [5] is recorded, but McCune and his colleagues report hypertension persisting 9 months after a biopsy and requiring three antihypertensive agents [3]. Although the magnitude and position of post-biopsy bleeds are likely to be important it is not clear which patients will develop this complication. It is interesting that in a large series where selective angiography was performed following renal biopsy only one of seven large perirenal haematomas was associated with hypertension [8]. The Page phenomenon has been described as a consequence of percutaneous biopsy of renal allografts [6,7]. In both cases allograft dysfunction was accompanied by significant hypertension and surgical decompression was required to restore graft function.

Proteinuria may accompany both hypertensive and renovascular disease [9]. Renovascular disease is not typically associated with heavy proteinuria, although it is of interest that Thadhani et al. [10] demonstrated nephrotic-range proteinuria and FSGS in association with renovascular disease. However, we did not find significant renal artery stenosis at selective renal angiography in our patient.

Perhaps the most intriguing and unexplained feature of this case is the reduction in proteinuria after this episode of transient left renal nonfunction. Successful treatment of the patient’s oedema with a loop diuretic did not significantly lower systolic or diastolic blood pressure. Since we would expect both kidneys to be affected equally by the process of FSGS, the almost complete abolition of proteinuria cannot be explained simply by loss of left kidney function, unless this kidney was responsible for most of the renal function.

Discussion

We have described a case of Page kidney occurring after a native renal biopsy with a clearly reversible deterioration in function caused by a perirenal haematoma. The phenomenon was described by and named after I. H. Page, a US physician and director of the Lilly laboratory of proteinuria has gradually increased and is now 4.8 g/24 h. The patient remains well on a small dose of loop diuretic.
Fig. 2. Three years later a Tc-99m MAG3 renogram now clearly demonstrates that both kidneys are functioning. The left kidney now contributes around 30% of the overall renal function.

References


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