Interesting Case

Dead on biopsy is not always dead

Nitin Kolhe¹, Mark Downes², Patrick O'Donnell³ and Paul Carmichael¹

¹Department of Nephrology and ²Department of Radiology, Canterbury Hospital, Canterbury UK and
³Department of Histopathology, St Thomas's Hospital, London, UK

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Introduction

Renal infarction usually occurs in a setting of atrial fibrillation or severe renal artery stenosis. Patients with renal infarction usually present with loin pain and may mimic symptoms of acute pyelonephritis. Anuria is usually uncommon unless renal infarction occurs in the setting of a solitary kidney or severe bilateral disease. We report a case of renal artery stenosis in a solitary kidney, presenting with renal failure and oligo-anuria. Subsequent renal biopsy demonstrated renal infarction. Partial recovery of renal function occurred for reasons stated below.

Case

An 80-year-old lady presented with a 3-day history of back pain, vomiting, and dysuria with decreasing urine output. She was treated with cephelexin by her GP. Past history included a right nephrectomy for renal cell carcinoma in 1988 and hypertension since 1975. Medication included lisinopril 20 mg/day, amlodipine 10 mg/day, and co-proxamol (two tablets on as required basis). She had smoked 15 cigarettes a day for the past 40 years. On examination her blood pressure was 140/72 mmHg, pulse 72/min and regular, peripheral pulses were present and there were neither femoral nor carotid artery bruits. Her temperature was 37.8°C, jugular venous pressure was normal, and there was neither pedal oedema nor skin rash. Systemic examination was essentially unremarkable. Investigations were as follows: haemoglobin 13.1 g/dl, white cell count 20.2×10⁹/l (neutrophilia), plasma sodium 131 mmol/l, potassium 6.7 mmol/l, urea 21.3 mmol/l, creatinine 609 µmol/l, alkaline phosphatase 97 IU/l, albumin 33 g/l, serum calcium 2.77 mmol/l, phosphorous 1.91 mmol/l, bicarbonate 18 mmol/l, and C-reactive protein 380 mg/l. Urinalysis revealed protein 1 g/l, sugar negative, leucocytes 15/ml, red blood cells 200/ml. X-rays of the chest and abdomen were normal and ECG revealed a sinus rhythm. Ultrasound scan revealed a solitary left kidney of 12 cm with good cortical thickness and no evidence of hydronephrosis. The provisional diagnosis was acute renal failure secondary to acute pyelonephritis in a single kidney. She was treated with i.v. antibiotics and haemodialysis. After three sessions of haemodialysis, the patient's urine output improved to 1 l a day and she became dialysis independent after 6 days. Serum protein electrophoresis, autoantibody screen including ANCA, and blood and urine cultures were all negative. Renal function failed to improve (creatinine 600 µmol/l) and a renal biopsy was performed. This demonstrated infarcted cortical tissue with up to an 80% loss of tubular elements (Figure 1).

The patient then underwent renal angiography, which demonstrated a 99% stenosis of a single left renal artery. A DMSA scan demonstrated infarction affecting the lower pole of the kidney (Figure 2). The patient, therefore, underwent renal angioplasty with stenting (Figure 3A and B).

Her present renal function is stable at a creatinine level of 290 µmol/l and antihypertensive requirement has decreased from three to one drug.

Discussion

Renal infarction is frequently misdiagnosed because of non-specificity of symptoms. One of the most frequent misdiagnosis is renal colic and acute pyelonephritis. Renal infarction has been reported widely in the literature secondary to thrombo-embolic events [1–3], NSAID ingestion [4], and also occasionally in patients with systemic lupus erythematosus and antiphospholipid syndrome [5]. It requires heightened suspicion and appropriate radiological investigations. In a small study by Lumerman et al. [6], CT scanning was found

Correspondence and offprint requests to: Dr Nitin Kolhe, Specialist Registrar, Department of Renal Medicine, Kent and Canterbury Hospital, Ethelbert Road, Canterbury CT1 3NQ, UK. Email: nitinkolhe@hotmail.com

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to be useful in the early diagnosis of segmental infarction. Radionuclide imaging has also been found useful in diagnosing acute segmental infarction [7]. Chabova et al. [8] found that deterioration of renal function and mortality risk were greatest in patients with bilateral stenosis or stenosis to a solitary functioning kidney. The long-term preservation of renal function and improvement in hypertension make solitary renal revascularization worthwhile [9].

This case illustrates the need to correlate renal histology to the clinical scenario. In our case, acute pyelonephritis was thought initially to be the cause of back pain and oligo-anuria, which was excluded after appropriate investigations. However, the true cause became apparent on further investigations. The patient, apart from age and smoking history, did not fulfill the typical criteria for renovascular disease with no peripheral vascular disease, ischaemic heart disease, demonstrable bruits, or difficult to control hypertension. Such a case demonstrates the need to continually review one’s diagnosis. We believe that segmental renal infarction in our patient may have been due to differential intra-renal blood flow with the lower pole having a more impaired flow secondary to the critical stenosis. An alternative explanation may be that the intra-renal vascular disease was more severe in the vessels supplying the lower pole. In this setting angioplasty and stenting had a favourable outcome as the entire kidney would undoubtedly have infarcted in the near future.

References


Fig. 1. Infarction of cortical tubules. Methanamine silver;H&E × 100.

Fig. 2. The DMSA scan showing irregular lower pole of the left kidney consisting with scarring secondary to infarction.

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Fig. 3. (A) The figure demonstrates more than 90% stenosis of the renal artery. (B) The figure demonstrates successful angioplasty and stenting of the renal artery.