Case Report

Running repairs: renal artery dissection following extreme exertion

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Case

A 43-year-old champion New Zealand athlete presented acutely with the onset of right flank pain. He gave a previous history of 'exercise-induced' arrhythmia and vertebral degeneration, which were attributed to a lifetime of sporting activity. Only the day before, he had competed in a gruelling triathlon race consisting of a 11.5 km swim, a 25 km cycle and a 10 km run in summer temperatures reaching at least 32°C (94°F).

On examination, he was normotensive but had moderate renal angle tenderness. Urinalysis showed white cells $>10^7-8$ cells/ml but no red cells or casts, and an abdominal ultrasound was unremarkable. He was discharged with simple analgesia but represented 7 days later with increasing abdominal pain and malignant hypertension. A renal isotope scan demonstrated the absence of perfusion of the right kidney. Subsequent angiography confirmed the presence of dissection and dilatation of the right renal artery with a segmental accessory artery perfusing the lower pole, as well as the kidney as a whole, via co-lateralization (Figure 1).

At surgery, the origin of the right renal artery appeared to be above the diaphragmatic crus, with a normal proximal centimetre of right renal artery angling downwards. The artery became angulated more horizontally adjacent to the muscular crus of the diaphragm that contained a fibrous band. The dilatation of the artery medial to the cava was immediately anterolateral to the crus and the dissection appeared to be angulating distally from this point. There was thrombus in the vessel extending back to the bifurcation of the renal artery at the hilum. The upper two-thirds of the kidney was dusky in colour but normalized following aorto-renal saphenous vein grafting with an end-to-end anastomosis to the renal artery stump. Intra-operative Doppler confirmed flow in the graft.

He was discharged 10 days later, normotensive and with normal renal function, on felodipine 20 mg and metoprolol 95 mg daily. He represented 2 weeks later with accelerated hypertension. Isotope scanning again confirmed the absence of right renal perfusion, and Doppler flow studies with contrast failed to show flow in the aorto-renal graft. He was commenced on single agent captopril 25 mg tds with immediate blood pressure control. Subsequently he has undergone a nephrectomy. Gross pathology demonstrated an infarcted kidney with preservation of the lower pole. There was no evidence of dysplasia of the renal artery. Adjacent to areas of frank renal infarction there was striking juxtaglomerular expansion in areas of preserved glomeruli, consistent with his hyper-reninaemia (Figure 2).
Renal artery dissection is an uncommon condition usually associated with atherosclerotic disease, fibromuscular dysplasia or after angioplasty [1]. Sporting activities including aerobics and American football have been associated with dissection, where it is thought that acceleration/deceleration angulation of the artery leads to intimal tearing [1,2]. It is possible that, as in this case, significant shearing forces were generated by the diaphragmatic crura on abnormally high renal vessels, particularly as the result of extreme and repetitive exertion. Spontaneous dissection in otherwise normal vessels has been recorded in the literature, invariably affecting young men and more frequently involving the left kidney [1]. Interestingly, an unusually high position of the kidney or artery, including upper polar vessels, supernumerary kidneys and thoracic kidneys are also more common in men and also preferentially involve the left side [3]. Although a recent report has detailed ischaemic renal artery following stretching injury to the renal artery of a displaced solitary kidney [4], this is believed to be the first recorded association of a high renal artery origin and dissection.

An alternative hypothesis for the pathological angulation of the renal artery seen in this case is the abnormally low right diaphragmatic crus, possibly due to exertional hypertrophy of the diaphragm or due to the fibrous band seen at operation. Men characteristically have larger vital capacity, especially with exertion. Larger, more muscular crura may create greater proximal angulation of the renal artery and, consequently, a risk for arterial trauma with jarring exertion, particularly if the vessel is adjacent. Diaphragmatic hypertrophy has not, however, previously been associated with arterial injury.

In this patient, the cardiac, vascular and skeletal injuries resulted from exertional wear and (ultimately) an exertional tear. In this modern age of athletes who push themselves ‘to the edge’, it seems likely that the limits of human endurance may increasingly become exposed. This case draws attention to the necessity for an early consideration of arterial damage and the requirement for angiography to confirm the diagnosis. Surgical repair or replacement of the damaged vessel is effective in preventing renal loss and controlling hypertension [1]. However, a late diagnosis, despite adequate flow being restored, can result in poor outcome and the consequent need for nephrectomy.

References


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