

*Teaching Point*

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## The patient with low back pain and acute oliguric renal failure

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### Case report

A 53-year-old male patient was referred from another hospital with a diagnosis of acute renal failure. Thirty-six hours prior to admission, he had sustained severe sudden-onset low-back pain while he was gardening at home. He had also noted paraesthesias in the right leg but had still been able to walk without any difficulty. The patient presented to another hospital where a vascular event of the right leg was suspected on the basis of absent pulses and duplex studies. Serum creatinine was 320  $\mu\text{mol/l}$  and urea was 18  $\text{mmol/l}$ ; both had been normal 2 months prior to admission. The patient now reported anuria of 12 h duration. A vascular surgeon deferred further evaluation pending a workup of acute renal failure (ARF) and the patient was transferred to our unit.

The patient was a retired sports teacher who had never smoked. He had been in good health until 1993 when aortic valve replacement had been performed for aortic regurgitation and a-v block had necessitated fitting of a pacemaker. He was on warfarin ever since. The patient's brother had recently undergone valve replacement, probably of the aortic valve, for an unknown disorder. The remainder of the medical history was unremarkable and there was no evidence of previous renal disease.

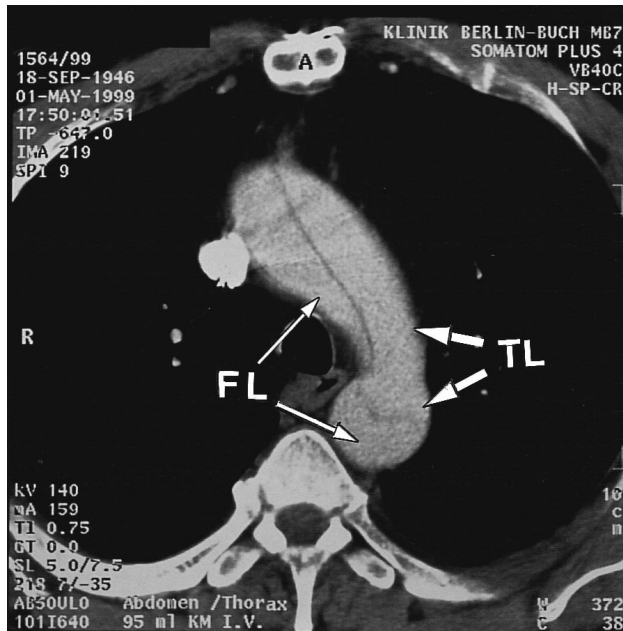
On examination, the patient appeared in pain but not acutely ill. Blood pressure was 150/100 mmHg, heart rate 96/min and temperature 36.4°C. The chest was clear and examination of the heart showed a

normal prosthetic click without significant murmur. The abdomen was soft but both flanks were tender to palpation. The right leg was warm but pale; femoral and distal pulses were absent with delayed capillary refill. The remainder of the physical examination was unremarkable. Laboratory studies showed serum creatinine of 350  $\mu\text{mol/l}$ , urea 25  $\text{mmol/l}$ . Dipstick analysis of small amounts of dark, turbid urine was ++ for protein, + for blood and negative for leucocytes and bacteria. Examination of the sediment showed many hyaline and waxy casts and epithelial cells presumed to be of tubular origin. On ultrasound, the kidneys were normal in size and morphology without any evidence of pericaliceal or ureteric distention. A computed tomography (CT) scan showed an intimal flap extending from the aortic root into the iliac arteries in keeping with Stanford type A aortic dissection (Figures 1 and 2). Little, if any, perfusion of the renal vessels was observed (Figure 3). The patient underwent emergency supracoronary replacement of the ascending aorta and intimal tear; a 26-mm Hemashield-Gold graft was used. Histology of the specimen showed severe degeneration of the aortic media. The patient made an uneventful recovery and was discharged in good health on the 28th post-operative day with a normal serum creatinine.

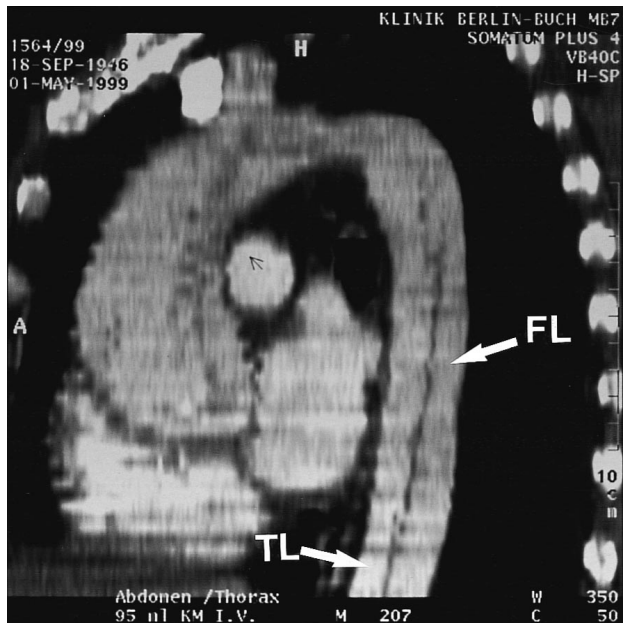
### Discussion

Timely diagnosis and management of acute aortic dissection have remained challenging ever since DeBakey and colleagues first reported repair of the descending aorta in 1955 [1]. Since then, mortality rates have declined substantially with the advent of new imaging modalities, improvements in surgical technique and better post-operative care. Still, the diagnosis is not infrequently made at autopsy and there is reason to believe that physicians are not sufficiently aware of the disorder and its presentation. In the case under

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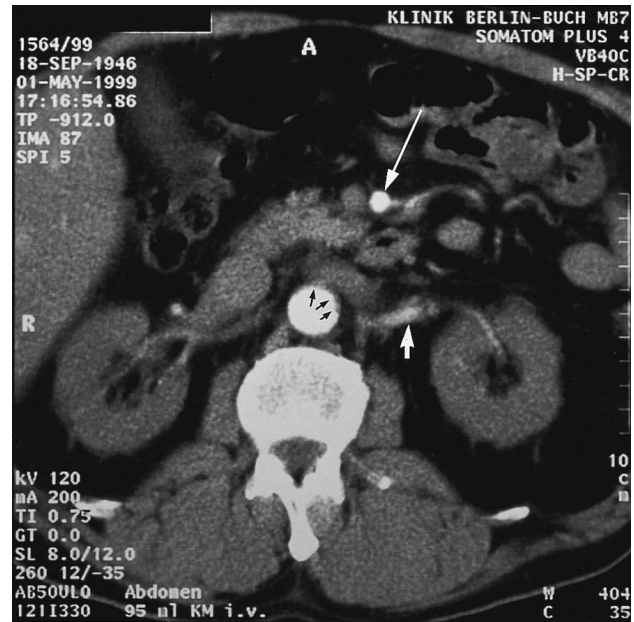


**Fig. 1.** CT scan of the ascending aorta and aortic arch just above the prosthetic valve obtained after the injection of contrast material: true lumen (TL) and false lumen (FL) can be easily identified.



**Fig. 2.** Sagittal reconstruction of the thoracic aorta derived from images obtained by CT scan after administration of intravenous contrast material: true lumen, false lumen and intimal flap are clearly visualized (the anterior chest wall is depicted on the left).

discussion, a fair number of doctors, among them a vascular surgeon, missed the diagnosis. In retrospect, a history of aortic valve replacement in conjunction with ARF and peripheral vascular compromise should have raised the suspicion of aortic dissection immediately; this suspicion was reinforced by a family history of aortic disease which, in the absence of Marfan's or von Ehlers Danlos syndrome, seemed to point at some



**Fig. 3.** CT scan image at the level of the renal arteries; the true lumen is the small irregular structure (small black arrows) which is somewhat displaced to the left. Accordingly, there is still some perfusion of the left renal artery (short white arrow) whereas the right renal artery is almost not perfused. In contrast, the superior mesenteric artery (long white arrow) appears to be well-perfused.

*forme fruste* of collagen disorder or cystic disease of the aortic media. Stanford type A aortic dissection was then easily diagnosed by CT scan thus allowing for timely surgical treatment with favourable outcome.

Only a few cases of aortic dissection presenting with ARF have been reported in the literature. However, transient compromise of one or both renal arteries by an intimal flap may be far more common than frank ARF and may occasionally go unnoticed [2]. Moreover, it is difficult to ascribe any impairment of renal function during the course of aortic dissection to the disease itself since other pre-renal factors such as exposure to contrast material, shock, and surgery may equally be held responsible. Fann and colleagues, reporting single-centre experience of 272 consecutive patients, encountered preoperative renal dysfunction in 8% of patients [3]. In their 1993 series of 325 patients, Cambria and co-workers reported proven renal vascular compromise in 17 patients with six of them being completely asymptomatic as to renal function and blood pressure [4]. Aldridge and colleagues were the first to report ARF and fatal outcome in a 71-year-old man with a remote history of nephrectomy who was then diagnosed with aortic dissection [5]. At presentation, signs and symptoms such as flank pain commonly prompt urologic evaluation, accounting for months of delay until appropriate treatment is initiated [6,7]. Management of aortic dissection is still controversial. However, excision and grafting are mandatory in Stanford type A dissection and were successfully performed in this patient. Notably, repair of the ascending aorta and aortic arch can reverse peripheral

vascular compromise provided that the intimal tear is properly repaired. However, despite considerable progress in this field, acute mortality still exceeds 20% [1]. Recently, evidence has emerged suggesting that acute aortic dissection can be managed by endovascular stent-graft sealing of the intimal tear averting the need for extensive high-risk surgery [8].

### Teaching point

Aortic dissection, a vascular catastrophe associated with high morbidity and mortality, is often missed and appropriate treatment therefore delayed, due to misleading signs and symptoms at presentation. Occasionally, oliguria may be a leading symptom. In patients presenting with acute renal failure, nephrologists should employ a high degree of suspicion to diagnose this rare but treatable cause of renal vascular disease.

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