Sir,

Anatomical anomalies of the upper extremity arterial system caused by aberrant embryological development are not rare, and most of them are reported in cadaver studies [1–3]. In recent years, angiography has provided an alternative tool for uncovering these embryological variants. It is important for surgeons and nephrologists to be aware of the possible arterial variations, because using anomalous arteries to establish arteriovenous access could be considered a viable alternative in some instances. To the best of our knowledge, this is the first case report of a patient undergoing maintenance haemodialysis via arteriovenous graft (AVG) with an anomalous brachial artery as the feeding artery.

Case. A 68-year-old man was an end-stage renal disease patient undergoing haemodialysis for the last 15 years. The present vascular access was a polytetrafluoroethylene graft placed in his right arm 3 years ago. After placement, no numbness or paraesthesia was noted and access function was optimal. However, recently decreased blood flow and increased venous pressure were found at the access site. Surgical thrombectomy was performed smoothly and palpable thrill was restored. He underwent an immediate postoperative angiography to evaluate the residual stenosis. Retrograde brachial arteriography was performed at the cubital fossa level and initially failed to show an AVG. Another set of angiography studies performed via the AVG clarified the course of the feeding artery (Figure 1) and showed multiple stenotic lesions in the distal part of the AVG. Retrograde brachial arteriography at the axillary level demonstrated that the feeding artery originated from the axillary artery and was measured as 7 mm in diameter. He underwent successful percutaneous transluminal angioplasty with a 5*20 SMASH balloon (Boston Scientific).

At 2 months follow-up, the dialysis course was rather smooth and the thrill was prominent.

Comment. Anomalies of the upper extremity arterial vasculature occur in 9–20% of the general population in cadaver studies [4,5]. Although the number of reported cases is small, the prevalence of anomalous arteries is high enough to warrant their use in clinical practice. Anomalous arteries are not as easily identified as a brachial artery (as we discovered in our case), but the superficial brachial artery usually has the same caliber as that of the main one and hence is suitable as a feeding artery for arteriovenous access. Easy accessibility, normal vessel caliber, and adequate blood flow qualify its use in arteriovenous shunt. Although aberrant vasculature caused ischaemic complications in patients undergoing radial or ulnar artery flap [6], our patient experienced neither ischaemia nor paraesthesia with adequate arterial inflow. In conclusion, if the brachial artery is neither optimal nor available, the anomalous vessel may be a viable alternative for establishing arteriovenous access.

Conflict of interest statement. None declared.

6. doi:10.1093/ndt/gfi047

Advance Access publication 16 August 2005

Using an anomalous brachial artery as an alternative choice of arteriovenous shunt feeding artery

Fig. 1. Graft angiography. With hand compression of the proximal upper arm, the course of the feeding artery (black arrow) and AVG (white arrow) were identified. The prime brachial artery was also shown (asterisk).
Vascular calcification and increased mortality in dialysis patients: is the baroreflex sensitivity the answer?

Sir,

In their interesting paper, Chesterton et al. [1] gave evidence for a possible link between vascular calcification, increased arterial stiffness (determined by time to shoulder, TTS) and impaired autonomic function (reduction in baroreflex sensitivity, BRS). This study adds new insights in the currently ongoing discussion dealing with vascular calcification and increased mortality in dialysis patients [2]. Impaired autonomic control of blood pressure due to vascular calcification could not only be a possible explanation for dialysis-induced hypotension but also a significant risk factor for the excessive cardiovascular mortality found in the dialysis population. Although Chesterton et al. [1] showed a significant association between vascular calcification and BRS (5.67 ± 0.76 vs. 3.43 ± 0.38 ms/mmHg in the group with and without calcification) as well as a significant but low correlation between TTS and BRS (r = 0.41), they failed to show a significant association between vascular calcification and increased arterial stiffness. This raises the question whether other variables not included in their analysis but obviously present in their study population are involved in reduced BRS, elevated arterial stiffness and vascular calcification. These variables include diabetes mellitus and smoking, which have been shown to impair BRS, enhance vascular calcification and lead to arterial stiffening [3,4]. Furthermore, the use of different antihypertensive drugs, such as α- and β-blockers or angiotensin-converting enzyme inhibitors, can significantly influence autonomic control and therefore alter BRS independently of arterial function and structure [5]. For that reason, information on the antihypertensive drugs used in the study population should be given by the authors. The link between alterations in arterial structure, arterial function and autonomic regulation might offer new pathophysiological insights, but larger studies controlling for the above mentioned factors have to be done to prove this relationship.

Conflict of interest statement. None declared.

Clinical Division of Nephrology
Emanuel Zitt
Laboratory of Experimental Alexander R. Rosenkranz
Nephrology
Innsbruck Medical University
Anichstrasse 35
6020 Innsbruck
Austria
Email: emanuel.zitt@uibk.ac.at

Sir,

Can rhabdomyolysis be the only cause of acute renal failure in leptospirosis?

Sir,

Acute renal failure (ARF) is a well-known complication of leptospirosis in its severe form (Weil’s syndrome). Generally, ARF is accompanied by jaundice and thrombocytopenia, with only sporadic case descriptions of milder forms of ARF in anicteric patients (rarely requiring dialysis). Thrombocytopenia is also closely correlated with ARF occurrence [1] and is described in all anicteric cases with ARF. The pathophysiology of ARF in leptospirosis evolves hypovolaemia, direct tubular toxicity and rhabdomyolysis. This case describes a patient with leptospirosis, severe rhabdomyolysis and ARF, with no jaundice or thrombocytopenia. Clinical and laboratory findings point to rhabdomyolysis as the major factor responsible for kidney injury.

A 38-year-old man, with no previous disease, had a history of myalgia 2 days previously and noticed reddish urine. Physical examination was unremarkable, except for extremely

Conflict of interest statement. None declared.

Sir,

In their interesting paper, Chesterton et al. [1] gave evidence for a possible link between vascular calcification, increased arterial stiffness (determined by time to shoulder, TTS) and impaired autonomic function (reduction in baroreflex sensitivity, BRS). This study adds new insights in the currently ongoing discussion dealing with vascular calcification and increased mortality in dialysis patients [2]. Impaired autonomic control of blood pressure due to vascular calcification could not only be a possible explanation for dialysis-induced hypotension but also a significant risk factor for the excessive cardiovascular mortality found in the dialysis population. Although Chesterton et al. [1] showed a significant association between vascular calcification and BRS (5.67 ± 0.76 vs. 3.43 ± 0.38 ms/mmHg in the group with and without calcification) as well as a significant but low correlation between TTS and BRS (r = 0.41), they failed to show a significant association between vascular calcification and increased arterial stiffness. This raises the question whether other variables not included in their analysis but obviously present in their study population are involved in reduced BRS, elevated arterial stiffness and vascular calcification. These variables include diabetes mellitus and smoking, which have been shown to impair BRS, enhance vascular calcification and lead to arterial stiffening [3,4]. Furthermore, the use of different antihypertensive drugs, such as α- and β-blockers or angiotensin-converting enzyme inhibitors, can significantly influence autonomic control and therefore alter BRS independently of arterial function and structure [5]. For that reason, information on the antihypertensive drugs used in the study population should be given by the authors. The link between alterations in arterial structure, arterial function and autonomic regulation might offer new pathophysiological insights, but larger studies controlling for the above mentioned factors have to be done to prove this relationship.

Conflict of interest statement. None declared.

Clinical Division of Nephrology
Emanuel Zitt
Laboratory of Experimental Alexander R. Rosenkranz
Nephrology
Innsbruck Medical University
Anichstrasse 35
6020 Innsbruck
Austria
Email: emanuel.zitt@uibk.ac.at

Sir,

Can rhabdomyolysis be the only cause of acute renal failure in leptospirosis?

Sir,

Acute renal failure (ARF) is a well-known complication of leptospirosis in its severe form (Weil’s syndrome). Generally, ARF is accompanied by jaundice and thrombocytopenia, with only sporadic case descriptions of milder forms of ARF in anicteric patients (rarely requiring dialysis). Thrombocytopenia is also closely correlated with ARF occurrence [1] and is described in all anicteric cases with ARF. The pathophysiology of ARF in leptospirosis evolves hypovolaemia, direct tubular toxicity and rhabdomyolysis. This case describes a patient with leptospirosis, severe rhabdomyolysis and ARF, with no jaundice or thrombocytopenia. Clinical and laboratory findings point to rhabdomyolysis as the major factor responsible for kidney injury.

A 38-year-old man, with no previous disease, had a history of myalgia 2 days previously and noticed reddish urine. Physical examination was unremarkable, except for extremely

Conflict of interest statement. None declared.