reached the level of statistical significance (Wilcoxon’s $P=0.055$). No reciprocal relations between the above HGF values, their per-dialytic change, enoxaparin dose or HD session duration were found.

In conclusion, we saw that salivary HGF concentration normalized during HD sessions. Our working hypothesis turned out to be apparently incorrect. The specific and important quest for the cause behind periodontal disease epidemics in maintenance HD patients has just begun and should be pursued.

Conflict of interest statement. None declared.

Conflict of Interest statement. None declared.

1Department of Nephrology Kenan Keven 1
2Biostatistic Ankara Başol Canbakan 1
University School of Medicine Atilla H. Elhan 2
Ankara Turkey
Email: keven@medicine.ankara.edu.tr

doi:10.1093/ndt/gfi134

Advance Access publication 14 September 2005

To haemodialysis and back: saving a kidney graft by treatment of an arteriovenous fistula

Sir,

With an incidence of around 16%, arteriovenous fistula (AVF) is a frequent complication of percutaneous renal biopsy (PRB) after kidney transplantation [1]. In only 0–5% of cases, however, an AVF causes clinical signs, such as systolic–diastolic murmur in the area of the graft, haematuria or hypertension [1–3]. A more serious consequence of AVF is a decline in renal function caused by shunting of part of the blood flow directly through the AVF into the venous system, bypassing the glomerular vessels. This leads to a shortage in circulation at the glomerular level in the flow area behind the AVF. The perfusion outside the flow area of the AVF will also be reduced since a substantial part of the blood flow directly through the AVF into the venous system, bypassing the glomerular vessels. This leads to a shortage in circulation at the glomerular level in the flow area behind the AVF. The perfusion outside the flow area of the AVF will also be reduced since a substantial part of the blood flow in this region will be shunted towards the affected flow area. Due to this diminished blood flow the affected flow area will activate the renin–angiotensin system causing hypertension and sodium-retention [1,4,5].

Recently a 36-year-old male with end-stage renal failure due to type 1 diabetes received a simultaneous pancreas–kidney transplant in our clinic. Due to limited improvement of renal function (Figure 1) in total three PRBs were performed during follow-up, all taken from the upper pole. The first two biopsies showed signs compatible with cyclosporine and tacrolimus toxicity, respectively, but cessation of these drugs did not improve renal function. After the second PRB a systolic–diastolic murmur was heard above the graft and presence of an AVF was suspected. Colour-coded Doppler sonography (CCDS), however, did not support this diagnosis. Forty-five days after transplantation the patient had to return to haemodialysis and a third PRB was performed, showing signs of focal tubular necrosis.
Clinically there was now hypertension and mild urinary sodium retention, both suspected to have a renovascular cause, which strengthened earlier suspicions of the existence of an AVF despite the negative CCDS. Subsequent angiography revealed two large AVFs with rapid venous outflow in the upper pole of the graft (Figure 2). Both AVFs were coiled using superselective transcatheter embolization, which resulted in disappearance of the murmur, improvement of renal function, normalization of blood pressure and urinary sodium-excretion. Currently, more than 1.5 years after transplantation, the patient has an excellent renal function with a creatinine clearance of 54 ml/min. This is to our knowledge the first report of successful treatment of a patient with ‘features of end stage renal failure’ after PRB-induced AVF. This case clearly shows that sometimes an AVF can cause serious problems and therefore requires treatment. However, due to the low incidence of symptomatic AVFs after PRB there is little attention to this phenomenon in clinical practice. With this case we want to stress the importance of considering AVF as a complication of a PRB in the differential diagnosis of poorly understood renal dysfunction after kidney transplantation.

Acknowledgements. No funding was received for the writing of this article.

Conflict of interest statement. None declared.