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¹Department of Nephrology and Dialysis, HELIOS Kliniken Schwerin, Schwerin, Germany

²Department of Urology and Transplantation, University of Rostock, Rostock, Germany

³Department of Tropical Medicine and Nephrology, University of Rostock, Rostock, Germany
Email: norbert.richard.braun@t-online.de

Norbert Braun¹
Ronald Walther¹
Oliver W. Hakenberg²
Emil C. Reisinger³

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Renal resistance index—think of more than just the kidney

Sir,

In a recent Nephroquiz, Mitsides *et al.* describe Doppler ultrasound of the segmental renal arteries of a renal allograft obtained from a patient with bigeminus [1]. They and others point out that extrarenal factors can affect the intrarenal resistance index (RI) [2]. To extend the list of these factors, we want to report the case of a 60-year-old woman, who had preemptively received a living donor kidney allograft from her husband.

The cause of her renal disease was vascular nephropathy. Immunosuppressive therapy consisted of tacrolimus, mycophenolate and prednisolone, and she also received several antihypertensive drugs (metoprolol, indapamide, doxazosin, felodipine). The post-operative course was uneventful, and serum creatinine at discharge was 90 µmol/L. Despite normal renal function, a Doppler ultrasound of the allograft showed a complete absence of diastolic flow in the interlobar and segmental arteries, giving an RI of 1 (Figure 1). A transplant biopsy taken 6 months later showed a mild polyoma virus nephropathy. Acute rejec-

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Fig. 1. Doppler signal of the distal segmental artery.

tion was excluded, and arteries and arterioles appeared normal. Therefore, the high RI could not be explained by intrarenal abnormalities, pointing to an extrarenal cause. One such cause could be stiffness of the pre-renal arterial vessels. Our patient had suffered from coronary heart disease, insufficiency of the aortic valve and an aneurysm of the ascending aorta. Therefore, 7 years before transplantation, she had undergone to aortocoronary bypass grafting and implantation of a prosthetic aortic valve, and a vascular graft (Hemashield Vantage®) of the ascending aorta. Dacron grafts are extremely stiff compared to the healthy aorta [3]. Therefore, they cannot expand during systole, and contraction during diastole, the main determinant of diastolic aortic flow, is absent. We suggest that this phenomenon explains the missing diastolic perfusion in the patient's renal allograft. In addition, Doppler ultrasound of the abdominal aorta and the superior mesenteric artery also showed a complete absence of diastolic perfusion.

In conclusion, this case demonstrates that an increased RI in a renal allograft may not always be a consequence of intrarenal pathology, but may also be caused by impairment of the function of pre-renal arterial vessels. Whether the absence of diastolic blood flow in the transplanted kidney will have a negative impact on long-term graft function is, at present, unknown. Eighteen months after transplantation, the patient's allograft function is excellent with an actual serum creatinine of 120 µmol/L.

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Department of Nephrology
and Dialysis, Academic
Teaching Hospital Feldkirch,
Feldkirch, Austria
Email: karl.lhotta@lkhf.at

Philipp Rein
Erich Wöss
Karl Lhotta

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Hepatitis C virus core antigen test in virological monitoring of patients on long-term dialysis

Sir,

Despite the control of hepatitis C virus (HCV) transmission, hepatitis C is still being observed among patients un-

dergoing dialysis [1]. In this population, HCV-positive subjects have an increased mortality risk compared with HCV-negative subjects. The diagnosis of HCV infection is currently based on detection of HCVAb, but enzyme immunoassays cannot distinguish between active and cleared infection, and aminotransferase activities lack diagnostic usefulness in dialysis patients. The direct detection of HCV depends on nucleic acid amplification technology (NAT) techniques with several problems: frequent unavailability, considerable skill requirement, limited reproducibility and overall important costs. Few studies exist about the efficacy of HCV core antigen test in the dialysis population [2,3]. From September 2009 to February 2010, we screened 168 long-term dialysis patients: 93 underwent haemodialysis and 75 peritoneal dialysis. We evaluated HCV antigen by chemiluminescent assay (Architect Abbott) and HCVRNA by PCR (TaqMan Roche). HCVRNA testing was performed on 90 subjects. We detected 142 HCVAb-negative and 26 HCV-positive patients. All HCVAb-negative subjects were HCVAg negative. HCVRNA testing was performed in 66 of the 142 HCVAb-/HCVAg-negative patients. The result was always negative. Among the 26 HCVAb-positive patients, we detected 18 who were HCVAg positive. All these HCVAb-/HCVAg-positive patients were HCVRNA positive; eight HCVAb-positive patients were HCVAg negative; six of these HCVAb-positive/HCVAg-negative patients were HCVRNA negative; we could not perform an HCVRNA test in two HCVAb-positive/HCVAg-negative subjects. Therefore, in 90 patients, the HCVAg test did not show any discrepancy towards the HCVRNA test. We think serological detection of HCVAg may be an alternative to NAT techniques; it can improve virological monitoring and integrate the diagnosis of acute hepatitis C in the dialysis population. The minimal cost and its easiness make this assay useful for routine long-term dialysis treatment patients.

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Nephrology and Dialysis
Division, Civic and Di
Cristina Hospital, Palermo
Italy
Email:
gioacchinolicavoli@libero.it

Gioacchino Li Cavoli
Carmela Zagarrigo
Onofrio Schillaci
Angelo Tralongo
Ugo Rotolo

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