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Cardiorenal syndrome or irreversible kidney damage: dilemmas in transplantation medicine

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Introduction: Renal dysfunction in advanced heart failure may reflect reversible cardiorenal physiology, intrinsic kidney disease, or both. Distinguishing reversible from permanent dysfunction guides selection for isolated heart transplantation (HT) vs. (simultaneous) heart-kidney transplantation (HKT).[1] We present a patient initially designated for HKT who, following haemodynamic optimisation, required isolated heart transplantation.

Case report: 58-year-old man with advanced heart failure due to ischemic heart disease (LVEF 24%, NYHA class III), presenting with appropriate implantable defibrillator shocks despite ventricular tachycardia ablation, with long-standing nephrolithiasis, was evaluated for HT. His renal work-up revealed a hypotrophic left kidney with cysts, extensive calculi and an encrusted JJ stent implanted for pelviureteric junction stenosis, while the right kidney was morphologically normal (**Figure 1**). Corrected creatinine clearance was 29 ml/min, eGFR 25mL/min/1.73 m^2, and proteinuria up to 823 mg/24 h. Right heart catheterisation revealed elevated left and right filling pressures, severe combined pre- and post-capillary pulmonary hypertension, reduced cardiac index (CI 1.4 L/min/m^2) and reversibly elevated PVR. Multidisciplinary team indicated HKT, with possible left nephrectomy later in the course



FIGURE 1. Computed tomography scan of the abdomen.

of treatment. Due to symptoms of low cardiac output, levosimendan was administered, but the CI remained low (1.8 L/min/1.73 m^2). Following 3 levosimendan cycles, full decongestion and a modification of RAAS inhibitor therapy, eGFR improved to 40–51 ml/min/1.73 m^2 and corrected clearance to 55 ml/min. Five weeks after the initial decision, the HKT indication was withdrawn, yet keeping the patient on the HT list. Two months later he underwent HT with a preoperative eGFR of 36 ml/min/1.73 m^2. Postoperative heart graft function was normal (LVEF 53%, CI 3.44 L/min/m^2), with normal filling pressures and PVR of 1.6 WU. After a transient further reduction of kidney function (nadir of eGFR 31 ml/min/1.73 m^2), it nearly normalized at discharge on postoperative day 20 with eGFR of 74 ml/min/1.73 m^2, an improvement to any pre-transplant value. At two months, while on tacrolimus and other potentially nephrotoxic therapies, only a slight worsening of eGFR was noted (52 ml/min/1.73 m^2) (**Figure 2**).

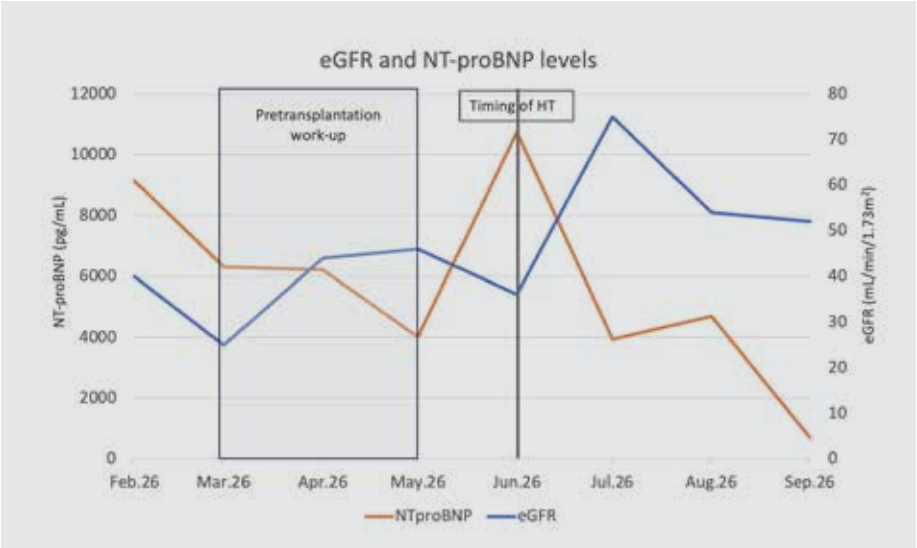


FIGURE 2. Estimated glomerular filtration rate (eGFR) and N-terminal pro-brain natriuretic peptide (NT-proBNP) trajectories.

Conclusion: Renal function can be affected by hemodynamic conditions such as congestion and low cardiac-output, but special caution should be exercised in patients with serious structural renal abnormalities, such as in our patient. Thorough haemodynamic optimisation is needed prior to definitive decision on modality of transplantation treatment.

LITERATURE

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