ACUTE KIDNEY INJURY SECONDARY TO CARDIOGENIC SHOCK? BEYOND KIDNEY REPLACEMENT THERAPY IN NECROTIZING FULMINANT MYOCARDITIS

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INTRODUCTION

Necrotizing fulminant myocarditis is a particularly severe form of myocardial inflammation that presents with cardiogenic shock and multiorgan failure. With goal-directed therapies and extracorporeal life support, like ventricular assisted devices, its prognosis has improved over the last decade. Even so, its mortality rate is still as high as 50%. Autoimmune mechanisms play a central role in this disease even though circulating cardiac auto antibodies cannot always be detected. In this setting, critical care nephrologists can not only provide extracorporeal support but also techniques directed against its pathophysiology.

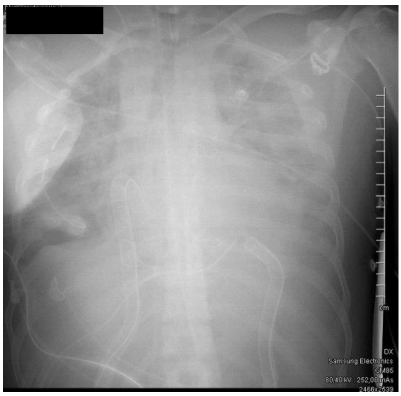
CASE REPORT

A male in his mid-twenties with a one-week history of cough, fever and progressive shortness of breath was admitted due to severe cardiac function impairment, requiring intubation, inotropic support and circulatory support with an intra-aortic balloon pump and extracorporeal membrane oxygenation (ECMO). On admission he had severe biventricular disfunction with an estimated ejection fraction (LVEF) of 20%. Routine viral serologies were negative except for Epstein Bar virus and parvovirus B19 IgG. A left ventricular endomyocardial biopsy showed a mixed inflammatory infiltrate with detectable Parvovirus B19 genome, although negative in plasma. Immunological profile, including cardiac auto antibodies, was negative. Suspecting a viral trigger immune-mediated cardiac injury he was started on glucocorticoids pulses (Methylprednisolone 1 gr daily for 3 days).

At that point antimyocardial antibody involvement was suspected and the patient had worsening hemodynamic instability with progressive fluid overload, so Critical Nephrology Department was contacted. Blood work up showed a creatinine of 2.8 mg/dL with eGFR of 36.39 mL/min/1.73 m2 and a K of 5.7 mmol/L with lactic acidosis. Daily urine volume was 1500 mL with intravenous furosemide. Kidney ultrasound showed normal sized kidneys with no hydronephrosis and signs of systemic congestion. A diagnosis of KDIGO's stage 3 acute kidney injury secondary to cardiocirculatory failure due to suspected Immune-mediated fulminant myocarditis was made and one session of plasma exchange (PEX) was performed in-line with the VA ECMO circuit with albumin reposition followed by immunoglobulin infusion (IVIG). Subsequently Continuous Venovenous Hemodiafiltration (CVVHF) was started (Prismaflex, oXiris filter) with a 35 cc/kg/h dialysis dose.

Unfortunately, the patient developed a profound refractory cardiogenic shock requiring a third-generation biventricular assistdevice (Centrimag Levitronix) (figure 1). He underwent another endomyocardial biopsy showing an eosinophilic infiltrate with extensive vasculitis with necrosis. Urinary sediment, although he was catheterized, showed microhematuria (++) and a 2000 mg/g protein/creatinine ratio.

Figure 1. Chest X ray following Centrimag implantation



As the patient presented with fulminant myocarditis-associated necrotizing coronary vasculitis secondary to Parvovirus B19 infection further methylprednisolone pulses were given (500 mg daily for 3 days) followed by IVIG. He slowly improved his hemodynamic status so that both ventricular support and kidney replacement therapy could be removed. He underwent another endomyocardial biopsy showing resolution of inflammation and vasculitis, being discharged from the ICU with an eGFR of 36 ml/min/1.73 m2 on oral furosemide and tapering steroid regime with oral prednisolone (1 mg/kg) with a global LVEF of 43%.

DISCUSSION

Although kidney failure was initially attributed to hemodynamic instability due to severe refractory cardiogenic shock, the possibility of renal vasculitis rose when the second myocardial biopsy proved vasculitis signs and urinary sediment was consistent with glomerular involvement. Even though the immune profile was within normal range and renal histological confirmation could not be obtained empirical PEX, IVIG and steroid treatment were started alongside with vital support, with progressive renal and ventricular function improvement. To our knowledge, there are no case reports published to this date on kidney involvement in patients with fulminant myocarditis with necrotizing vasculitis.

Knowledge of its pathophysiology can provide goal-directed therapies to prevent further hemodynamic deterioration. Viral infections are one of the most common etiologies. Molecular mimicry between viral and cardiac antigens is suspected to be a key mechanism of myocardial injury. As a result, myocardial cytotoxic T cells are activated and anti-cardiac antibodies develop, although they are not always detected in plasma. In this setting, nephrologists can offer not only renal replacement therapy support but also expertise in immunosuppressive agents and apheresis techniques, providing therapies directed to the intricate basis of this disease.

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