The fourth “T”

Dr Joanne Skinner¹, Dr Louis Peters¹, Dr Clare Macewen¹
¹Oxford University Hospitals NHS Foundation Trust, Oxford, United Kingdom

Introduction

Uraemic pericarditis is well described in patients with acute renal failure, but is not often considered in the differential of haemodynamic instability during haemodialysis. Although uncommon, progression of uraemic pericarditis to tamponade must be promptly recognised to ensure survival. We report a case of cardiac tamponade during acute haemodialysis, which we believe has a number of important learning outcomes for the renal community.

Case Summary

A 65-year-old Caucasian male with rapidly progressive diabetic nephropathy was transferred to our tertiary renal unit with fluid overload and refractory hyperkalaemia. He had presented to the referring hospital the previous week with an infected diabetic foot ulcer, complicated by acute on chronic renal failure. He required a session of haemofiltration to manage hyperkalaemia on the local intensive care unit. Echocardiography revealed moderate impairment of LV function, but no pericardial effusion. He had been stepped down to the ward for 3 days but had not shown any renal recovery.

On examination, the patient was drowsy, disoriented, and grossly fluid overloaded, with evidence of pulmonary oedema and anasarca. He was haemodynamically stable and saturating at 94% on 4L/min nasal cannulae. There was no pericardial rub and serum urea was 24 mmol/L. A temporary femoral line was inserted to facilitate acute haemodialysis. On bleed out into the circuit (roughly 150mls), he became hypotensive, bradycardic and hypoxic. This progressed to pulseless electrical activity despite delivery of atropine, and return of the circuit. Intubation and cardiopulmonary resuscitation proceeded, including delivery of a 250ml fluid bolus. Return of spontaneous circulation was achieved after 5 minutes. Bedside ultrasound revealed a 3-4cm deep pericardial effusion with fibrin stranding, causing clear tamponade with right ventricular diastolic collapse. Whilst awaiting transfer to cardiac theatres, the patient became periarrest and we proceeded to urgent ultrasound-guided bedside pericardiocentesis on the renal ward using a standard central line kit. 170mls of haemorrhagic serous fluid was drained, with restoration of blood pressure and resolution of echocardiographic features of tamponade. The central line was left in the pericardial space for 24 hours on the intensive care unit, until it stopped draining. The patient was discharged home 2 weeks later with ongoing outpatient haemodialysis three times weekly and remains well.

Discussion

This case highlights the need to consider uraemic pericarditis with pericardial effusion as a cause of haemodynamic compromise on haemodialysis. Important learning points include the rapidity of effusion development; the minimal change in intravascular volume required to precipitate tamponade; the relatively low serum urea (which is only a marker of “uraemia”); and the lack of clinical clues. A rub can only be heard when there is minimal fluid between pericardial layers, and autonomic neuropathy may prevent compensatory tachycardia. Bedside ultrasonography is available on most renal units for vascular access and
renal biopsy. We suggest that renal physicians consider training in limited bedside echocardiography, eg subcostal view with an abdominal probe, to facilitate diagnosis of this condition.