Myocardial Stunning During Acute Renal Replacement Therapy

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Introduction

The circulatory stress of chronic haemodialysis (HD) results in repetitive subclinical myocardial ischaemia (myocardial stunning), which contributes to adverse patient outcomes. Currently it is unknown whether this process occurs during renal replacement therapy (RRT) for acute kidney injury (AKI). Acute RRT differs in both its delivery and because patients do not display the circulatory changes of chronic uraemia that increase ischaemic susceptibility. We therefore aimed to determine whether acute RRT is capable of inducing myocardial stunning.

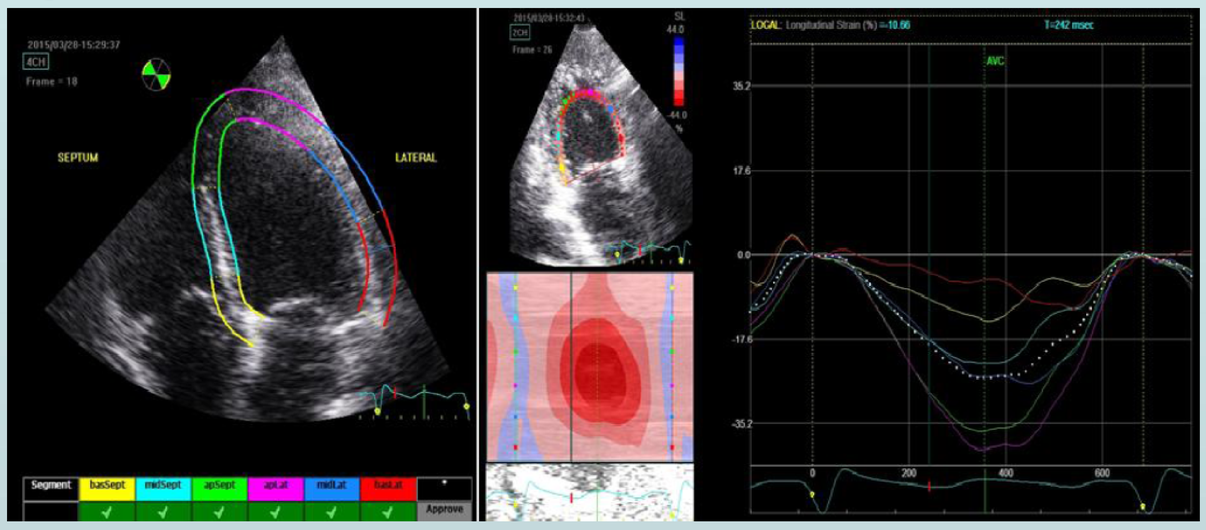
Methods

Patients admitted between October 2014 and September 2015 to our renal high dependency unit with acute kidney injury (AKI) requiring dialysis were considered for inclusion.

12 patients were recruited (2 patients were subsequently excluded due to poor image quality). All patients underwent a 4 hour haemodialysis session using an AN69 ST dialyser (Nephral, SA 1.3m^2), dialysate flow rate of 500 ml/minute and a blood flow rate between 190-250mls/minutes. The dialysis prescription and ultrafiltration volumes were determined by the clinical team.

Serial echocardiography (apical 4 & 2 chamber views, figure 1) was performed before, during and after a single RRT session and images analysed off-line using 2D speckle-tracking software. Myocardial stunning was defined as ≥2 new left ventricular (LV) regional wall motion abnormalities (RWMAs) during dialysis. LV systolic longitudinal strain was used to measure global and segmental LV function, with RWMAs defined as ≥20% decline in strain from pre-dialysis values. Ejection fraction, segmental LV strain and global longitudinal strain (GLS) were recorded. Blood pressure (BP) and systemic haemodynamics were measured continuously using thoracic bioreactance. Intra-dialytic hypotension (IDH) was defined as >20 mmHg fall in systolic blood pressure (SBP) or SBP <90 mmHg. Biochemistry data was collected pre and post dialysis.

Figure 1,

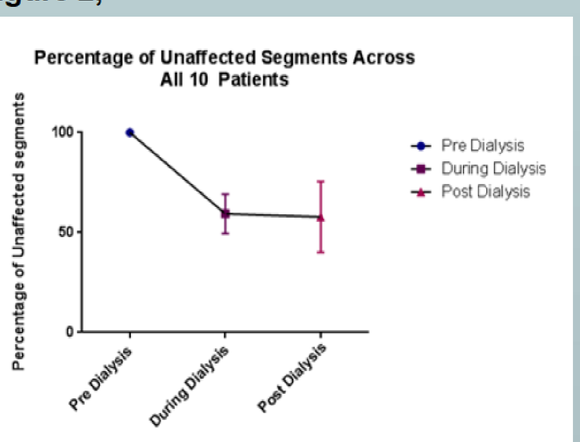


Results

Mean age of the patients was 60±15 years, 8 were male and 5 were diabetic. Baseline eGFR was 79±28 ml/min, 4 patients had pre-existing CKD stage 3. All 10 patients demonstrated dialysis-induced stunning during dialysis (≥2 new RWMAs). The majority of these segments remained dysfunctional at 30 minutes post-dialysis (figure 2). The median number of affected LV segments during dialysis was 4.5 (IQR 3-6) versus 5.5 (IQR 3-6) post dialysis (p=0.47)

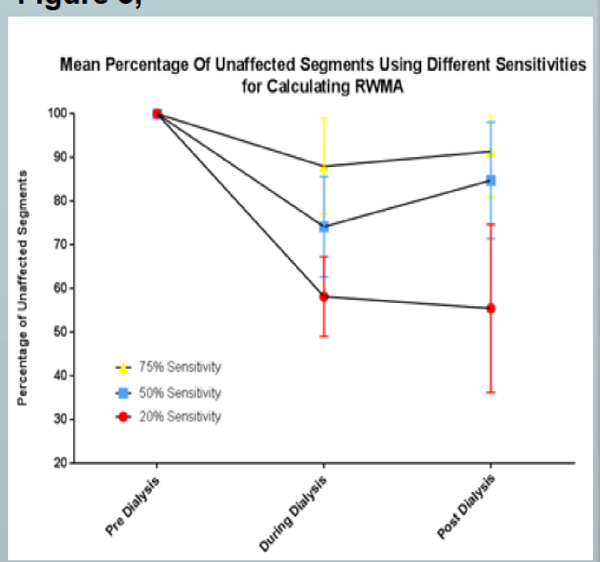
In a sensitivity analysis (defining segmental dysfunction as ≥50% reduction in strain from baseline) myocardial stunning was still observed in 9 patients (figure 3).

Figure 2,



Percentage of Unaffected Segments During Dialysis 59.4% ±3.11 to 57.8% ±5.75 post dialysis

Figure 3,



GLS significantly declined from a pre-dialysis level of -17.1±4% (within normal range) to below the normal range during dialysis (-14.3±3%, p=0.035), remaining low at 30min post-dialysis (-14.45%±0.9), figure 4.

Baseline mean ejection fraction (EF) was 71±8%, and fell significantly during dialysis to 59±13% (p=0.03 versus baseline) and only partially recovered post dialysis (62±8%, p= 0.04 versus baseline), figure 5.

Figure 4,

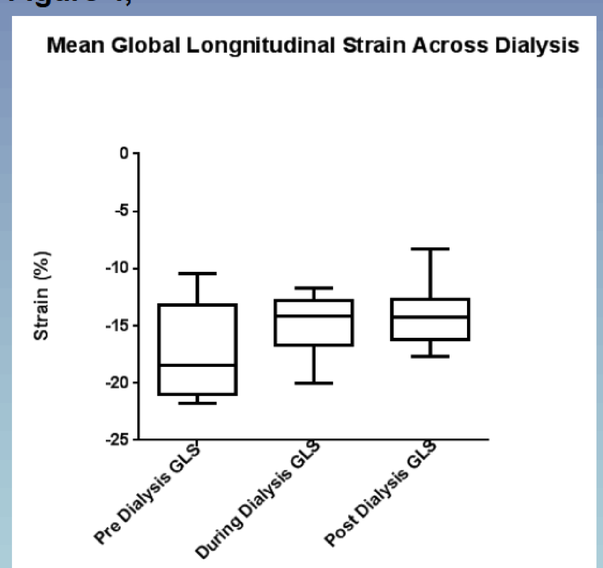
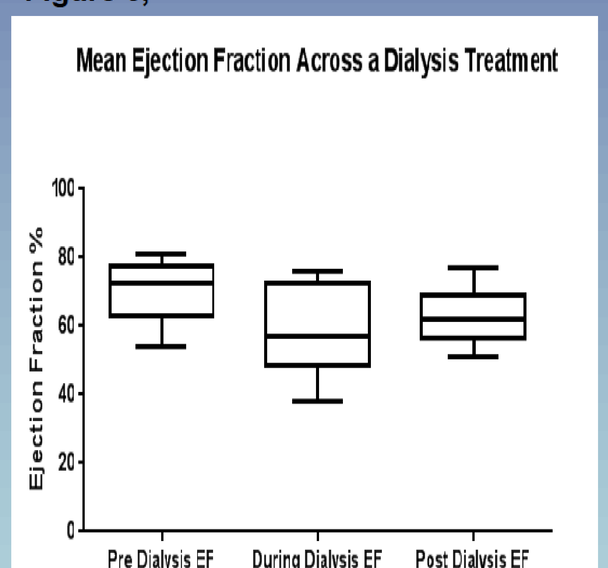


Figure 5,



The number of stunned LV segments was associated with ultrafiltration volume (r=0.65, p=0.04) and the presence of intradialytic hypotensive episodes (r=-0.36, p=0.35)

In the 6 patients who experienced intradialytic hypotension, GLS was significantly lower than those with stable BP (-13.0±2% vs. -16.2±2%, p=0.04).

NT pro-BNP increased post dialysis as compared to predialysis levels 12977±3948 ng/L versus 10438±468 ng/L, p=0.005, (figure 6), but Troponin T measurements did not differ significantly (figure 7).

All patients survived to discharge, and mean length of stay (LOS) was 25.6 +/- 15 days. 3 patients remained haemodialysis dependant, 2 of these patients died after hospital discharge.

Figure 6,

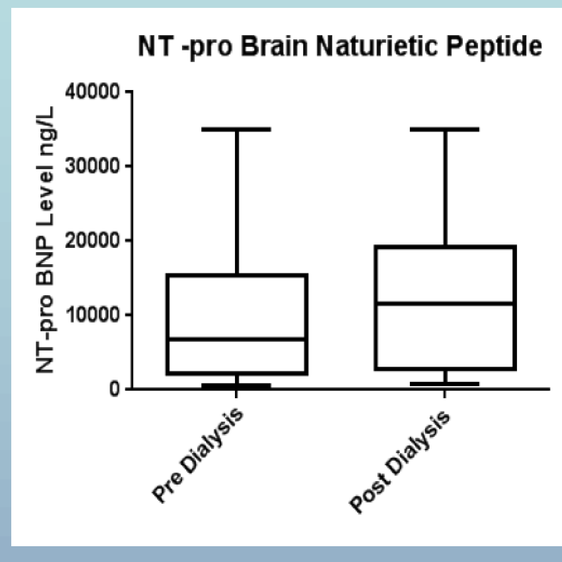
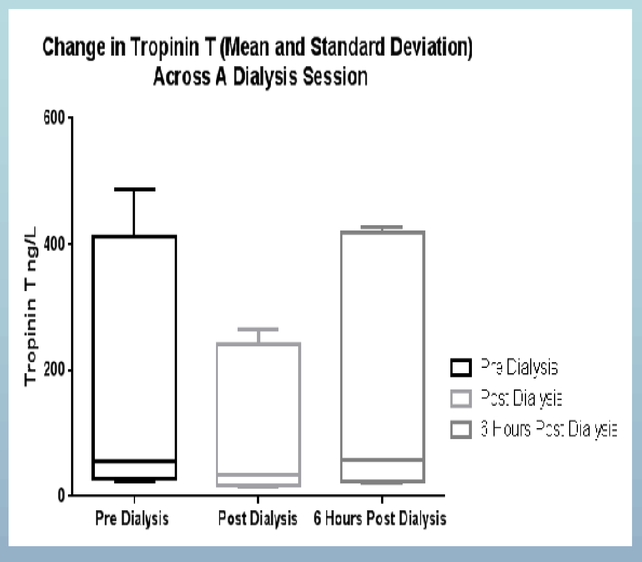


Figure 7,



Conclusion

This study demonstrates for the first time that dialysis induced myocardial stunning occurs during acute RRT and is associated with a global reduction in LV contractility and EF that persist after the dialysis is completed. Myocardial dysfunction was most notable in patients with higher ultrafiltration volumes and intradialytic hypotension. This suggests that cardiac injury during RRT is driven by dialysis-related factors (as opposed to being dependent on the cardiovascular changes of chronic uraemia). Further study is required to determine whether dialysis induced stunning contributes to the very poor outcomes that are well recognised in this patient group and whether therapeutic dialysis-based interventions in the acute setting reduce the rate and severity of myocardial dysfunction.

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