ISSN: 0975-833X

*Available online at http://www.journalcra.com*

*International Journal of Current Research*
*Vol. 10, Issue, 04, pp.68314-68316, April, 2018*

**INTERNATIONAL JOURNAL OF CURRENT RESEARCH**

## CASE REPORT

# UREMIC CARDIOMYOPATHY WITH MYOCARDITIS A RARE COMPLICATION IN END STAGE RENAL DISEASE, A CASE REPORT AND REVIEW OF LITERATURE

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**ARTICLE INFO**

***Article History:***
Received 17th January, 2018
Received in revised form
20th February, 2018
Accepted 29th March, 2018
Published online 30th April, 2018

***Key words:***
Uremic cardiomyopathy, ESRD, CKD.

**ABSTRACT**

Uremic cardiomyopathy is responsible for high morbidity and mortality rates among patients with chronic kidney disease (CKD) or end stage renal disease (ESRD) even though the patients on dialysis. Among dialysis patients with left ventricular hypertrophy (LVH) are more than two thirds die from heart failure or sudden cardiac death. Present case is about a case of CKD on dialysis who died due to myocarditis and cardiomyopathy.



**Citation: Patne, SS., Gadgil, PA., Pandit, GA. and Mahamuni, NM. 2018.** "Uremic cardiomyopathy with myocarditis a rare complication in end stage renal disease, a case report and review of literature", *International Journal of Current Research*, 10, (04), 68314-68316.

## INTRODUCTION

Cardiac diseases are leading cause of death among chronic kidney disease patients on dialysis accounting for 49% of all mortality (Rigatto and Parfrey, 2001). Uremic cardiomyopathy is defined as cardiac disease in chronic kidney disease patients because of collective disordes of perfusion (ischemic disease) or disease of structure and function. Even though coronary artery disease is major cause of death in patients with ESRD, some authors have suggested that more deaths are caused by left ventricular hypertrophy (LVH) and congestive heart failure (CHF) than by CAD (Eyad Alhaj *et al*., 2013). Uremic cardiomyopathy is a result of uremia , pressure overload and volume overload that leads to LVH. There are specific and nonspecific factor affecting uremic cardiomyopathy, Specific factors are uremic toxins and parathyroid hormone (PTH). (Kristian Kuze *et al*., 1998) Nonspecific and potentially reversible factors which affects uremic cardiomyopathy are anemia, hypertension and overhydration (Kristian Kuze *et al*., 1998).

### Case report

Sudden death of a 35years male a known case of CKD on dialysis since 4years. At autopsy all organs were in their normal anatomical position.

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Lungs were spongy with thickened pleura. Heart weighed 400gms, left ventricular wall of thickness 2cm. White patches on left ventricular anterior, posterior wall and inter ventricular Septum (Fig. 1). Aorta showed atherosclerotic patch. Both kidneys were reduced in size (Fig. 1) with asymmetric involvement. External surface showed scars, granularity, capsule adherent. Corticomedullary differentiation was not possible in both kidneys with cystic change (Fig. 2).

### Microscopy

Sections from lung show thickened pleura with inflammatory cell infiltrate. Sections from left ventricular wall showed hypertrophy and attenuation of cardiomyocytes with increased interstitial connective tissue with focal areas of mononuclear inflammatory cell infiltrate with myocytolysis (Fig. 3). Coronaries and aorta showed atherosclerosis. Section from Kidney showed (Fig. 4) diffuse glomerular sclerosis, tubules showed thyroidisation and the interstitium showed fibrosis and diffuse mononuclear inflammatory cell infiltrate. Marked arterial sclerosis was noted. Cyst were lined by flat to cuboidal epithelium.

## DISCUSSION

Cardiovascular disease account >50% of mortality in uremic patients in CKD treated with dialysis (Nolan, 2005). Compared to general population the relative importance of atherosclerotic coronary artery disease is diminished in CKD and that of left ventricular hypertrophy (LVH), heart failure and sudden

cardiac death are increased (David sample *et al*., 2011). Uremic cardiomyopathy is heterogenous (systolic and diastolic dysfunction) and multi factorial disease (Kristian Kuze *et al*., 1998).

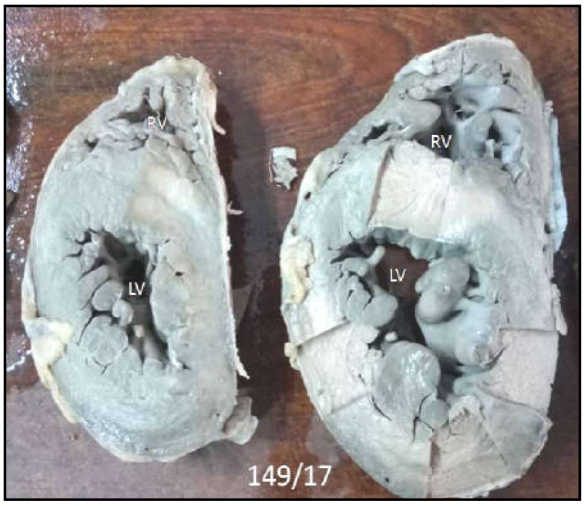


**Fig. 1. Transverse section of heart- LVH with White patches**

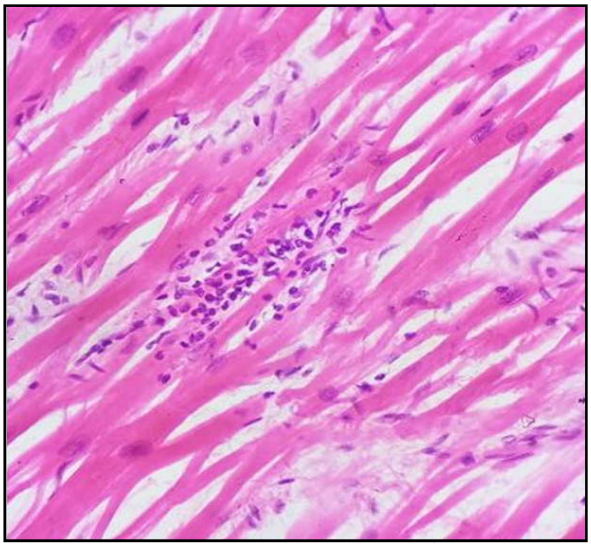

**Fig. 2. Cardiomyocytes- hypertrophy, myocytolysis and inflammatory cell infiltration**

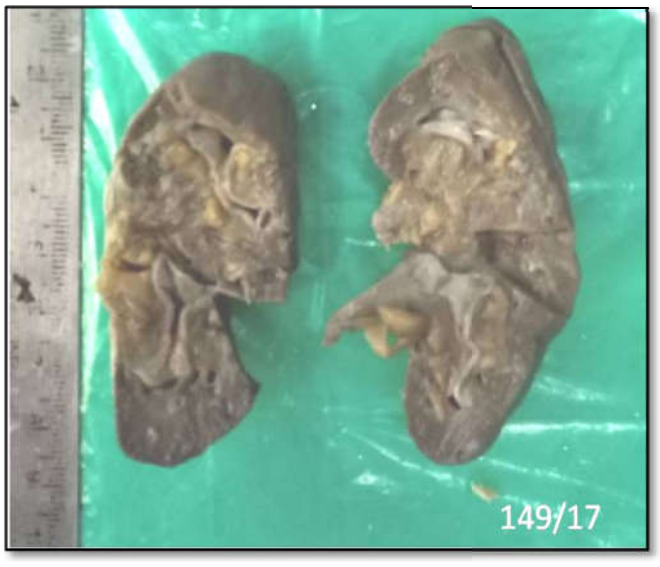


**Fig. 3. Kidneys- Reduced in size with loss corticomedullary differentiation**

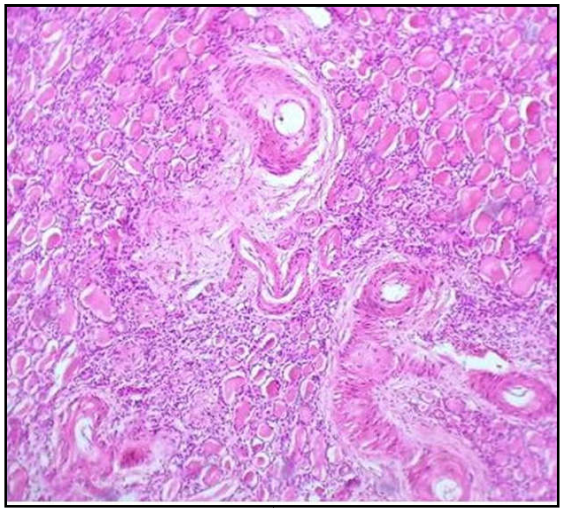

**Fig. 4. Microscopy kidney – CKD 3rd stage**

Uremic cardiomyopathy is as a result of impaired renal function influencing on the myocardial function causing left ventricular hypertrophy (Eyad Alhaj *et al*., 2013). Current treatment strategy offer only improvement in extending life (David sample *et al*., 2011). There are few cardiovascular abnormalities commonly encountered in CKD or ESRD patients are LVH, left ventricular (LV) dilation and left ventricular (systolic and diastolic) dysfunction (Kristian Kuze *et al*., 1998). There are potential risk factors (RF) for cardiomypathy in uremic individuals and are traditional and uremic factors. Traditional RF are the smoking, hypertension, dyslipidaemia diabetes and left ventricular hypertrophy. Uremic haemodynamic factors are anemia, AV (arterio-venous) fistula, volume overload and uremic metabolic factors are hypoalbunemia, hyperhomocysteineamia, oxidative stress, chronic inflammation and secondary hyperparathyroidism. Parathyroid hormone is potentially cardiotoxic (Bostom and Lathrop, 1997; Amann *et al*., 1994; London *et al*., 1987). Cardinal features of uremic cardiomyopathy are LVH, reduced capillary density, fibrosis and ventricular remoldeling. (Eyad Alhaj *et al*., 2013) LVH increases in prevalent from 26% in patients with stage 3 CKD to 75% in patients undergoing hemodialysis (David sample *et al*., 2011). LVH is an attempt to maintain wall stress and is a beneficial and adaptive response to continuous LV pressure and volume overload. It leads to maladaptive changes in cardiomyocytes and death of cardiomyocytes.

It further exacerbated by diminished perfusion, malanutrition uremia and hyperthyroidism. Loss of cardiomyocytes predisposes to LV dilation and LV (diastolic and systolic) dysfunction. (Eyad Alhaj *et al*., 2013) Cardiac fibrosis in uremia in CKD patients noted on post mortem is reactive type of consequence of endothelial to mesenchymal transition followed by activation and proliferation of interstitial fibroblasts which causes ventricular stiffness and diastolic dysfunction, arrythemias and also affects molecular exchange between cardiomyocytes and capillary bed (David sample *et al*., 2011). Molecular pathophysiology of pressure and volume overloads one thought to stimulate LVH through integrins. It initiate intracellular signalling in response to a stretch of extracellular matrix which promote release of local ligands (angiotensin II, and endothelin I) and these bind to receptors on cardiomyocytes then stimulating intracellular signalling pathway.

Accumulation of hypertrophic substances of uremic state like endothelin I, PTH, TNFα, leptin, ILIα and IL 6 causes LVH in CKD which is independent of volume and pressure overload (Yutao *et al*., 2006; Yamazaki *et al*., 1995; Yamazaki *et al*., 1996; Winchester, 2006). Dialysis reduces LV mass. Few studies have evaluated that anti hypertensive drugs causes regress in LVH. Cannella *et al* observed that triple association of beta blockers, calcium channel antagonists and ACE inhibitors markedly reduces LV mass and arterial blood pressure (Cannella *et al*., 1993). Kidney transplant has been shown to reverse uremic cardiomyopathy and to confer a significant survival advantage over hemodialysis. High perioperative mortality noted at the time of renal transplant because of LV diastolic dysfunction associated with pulmonary oedema (Eyad Alhaj *et al*., 2013). We reported this case for its rare complication of cardiomyopathy with myocarditis and atherosclerosis leading to death in a known case of CKD.

**Conclusion**

Uremic cardiomyopahty is heterogenous (systolic and diastolic dysfunction) and multi factorial complication responsible for high morbidity and mortality in CKD or ESRD patients. Cardinal features of uremic cardiac disease are LVH, reduced capillary density, fibrosis and ventricular remoldieing. LVH is a strong predictor of CHF and sudden death in CKD patients. Targetting future therapies at the underlying cellular mechanism may reduce the burden of uremic cardiomyopathy in CKD and ESRD population.

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