Reversal of Severe Left Ventricular Dysfunction Following Kidney Transplantation: A Case Report

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Uremic cardiomyopathy (U-CMP), also known as chronic kidney disease cardiomyopathy (CKD-CMP), is a phenotype of non-ischemic cardiomyopathy frequently seen among patients with chronic kidney disease. Left ventricular (LV) systolic dysfunction is seen in approximately 13% of patients, and LV ejection fraction (LVEF) below 40% has been reported in 5.8% of patients [1]. Severe LV dysfunction may be considered a relative contraindication to renal transplantation. We present a case of complete recovery of ventricular function following renal transplantation in a patient with severe U-CMP.

PATIENT DESCRIPTION

A 52-year-old patient was admitted to the intensive cardiac care unit due to an acute non-ST-segment elevation myocardial infarction (NSTEMI). His past medical history included non-ischemic cardiomyopathy with a dilated, hypertrophic LV, and severe LV systolic dysfunction, which was first documented in 2012. His estimated LVEF was 20–30% on serial transthoracic echocardiograms between 2012 and 2019. Other chronic medical conditions include hypertension and dyslipidemia as well as a history of smoking. The patient had end-stage kidney disease, secondary to IgA nephropathy, requiring hemodialysis. The patient received a kidney transplant from a cadaver in 2020 and his arteriovenous fistula was ligated. Regular medications included prednisone, tacrolimus, mycophenolate mofetil, esomeprazole, atorvastatin, bisoprolol, and ramipril. There was no history of alcohol or recreational drug use.

On admission an echocardiogram demonstrated a non-dilated, hyperdynamic LV with an estimated LVEF of 75% and reduction of wall thickness compared to the patient's previous echocardiographic studies. The improvement in LVEF and the rest of echocardiographic findings were adjudicated by two expert echocardiographers. Cardiac catheterization demonstrated triple vessel coronary artery disease. The patient underwent percutaneous coronary intervention and insertion of drug eluting stent to the right coronary artery.

Cardiovascular diseases are by far the leading cause of mortality of patients with chronic kidney disease (CKD) [1]. CKD and ischemic heart disease (IHD) share some risk factors, such as diabetes mellitus and older age. In addition, some IHD risk factors are aggravated by the presence of CKD. One such example is hypertension, which is often difficult to control in patients with CKD. Last, the metabolic state associated with CKD has an adverse effect on the heart. Therefore, U-CMP often coexists with other phenotypes of cardiomyopathy, such as ischemic or hypertensive cardiomyopathy, making the diagnosis challenging.

Severe LV dysfunction among dialysis patients may be considered as a relative contraindication to renal transplantation, as operative risks are higher and heart failure may be aggravated by steroid treatment. However, if LV dysfunction is potentially reversible by transplantation this may actually present a better reason to proceed to transplantation.

COMMENT

U-CMP is particularly common in patients with end-stage kidney disease but can be seen in patients with any degree of CKD. Numerous mechanisms are thought to contribute to U-CMP. One such mechanism is the accumulation of uremic toxins [2].

Urea, often used to guide the need for dialysis and its effect, is a minor contributor to cardiomyopathy. Con-

versely, other molecules, not routinely measured in laboratory tests, play a more pronounced role in the disease mechanism. Some molecules, such as trimethylamine-N-oxydase, are small, potentially dialyzable molecules. However, dialysis membranes are often not optimized for their removal. Moreover, some of those toxins accumulate in serum more rapidly than urea, leading to intermittently high toxin levels in patients undergoing intermittent hemodialysis. Other uremic toxins, such as indoxyl sulphate, are protein-bound, and therefore not dialyzable [3].

A second proposed mechanism involved in U-CMP is accumulation of cardiotonic steroids. Substances such as oubain or marinobufagenin can alter the function of Na^+-K^+ pump, leading to diminished myocardial contractility.

A third proposed mechanism in U-CMP is mineral and bone disorder (CKD-MBD) [4]. Previous studies linked secondary hyperparathyroidism with vascular calcification, LV hypertrophy and systolic dysfunction. Hyperphosphatemia is associated with increased cardiomyocyte apoptosis. Increased fibroblast growth factor 23 (FGF-23) and reduced serum levels of its cofactor, alpha-klotho are associated with LVH and sudden cardiac death as well. Other postulated mechanisms include anemia, chronic volume overload, and chronic inflammation.

One more mechanism linking CKD and cardiomyopathy is accelerated atherosclerosis, also known as cardiorenal syndrome type 4. As glomerular function deteriorates, calcium deposition in coronary artery increases [4]. Both autopsy and imaging studies link advanced CKD with diffuse fibro-calcific coronary artery plaques, with both intimal and medial calcification. The interplay between coronary artery calcification, endothelial dysfunction, and bone-mineral metabolism abnormalities leads to diffuse, severe atherosclerotic disease in CKD patients compared to matched, non-CKD patients.

Some patients experience improvement of their clinical condition and their imaging-evidence of U-CMP following renal replacement treatment [5]. However, some uremic toxins are not readily removed by dialysis. Moreover, hemodialysis on its own might aggravate cardiomyopathy: the high-output state associated with arteriovenous fistula leads to renin-angiotensin-aldosterone activation, increased myocardial oxygen demand and subendocardial ischemia, particularly in the presence of concurrent coronary artery disease. Those caveats can be avoided with peritoneal dialysis. Kidney transplantation is considered the ultimate solution for U-CMP, allowing reversal of the metabolic changes without the potential harmful effects of dialysis.

CONCLUSIONS

U-CMP is a distinct phenotype of cardiomyopathy often seen in CKD patients on hemodialysis, which is potentially completely reversible with renal transplantation. Despite the lack of systematic randomized studies to assess the potential benefit of renal transplantation in this population, it might contribute to recovery from cardiomyopathy. We suggest that in the absence of documented other causes for severe LV dysfunction, renal transplantation should not be denied and there is hope for significant resolution of LV dysfunction following transplantation.

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References

1. Li Q, Qiu L, Long M, et al. Prevalence and prognostic impact of abnormal left ventricular ejection fraction in hemodialysis patients with end-stage renal disease. *BMC Nephrol* 2025; 26 (1): 384.
2. Nguyen TD, Schulze PC. Cardiac metabolism in heart failure and implications for uremic cardiomyopathy. *Circ Res* 2023; 132 (8): 1034-49.
3. Ahmadmehrabi S, Tang WHW. Hemodialysis-induced cardiovascular disease. *Semin Dial* 2018; 31 (3): 258-67.
4. Sarnak MJ, Amann K, Bangalore S, et al; Conference Participants. Chronic Kidney Disease and Coronary Artery Disease: JACC State-of-the-Art Review. *J Am Coll Cardiol* 2019; 74 (14): 1823-38.
5. Hiraiwa H, Kasugai D, Okumura T, Murohara T. Implications of uremic cardiomyopathy for the practicing clinician: an educational review. *Heart Fail Rev* 2023; 28 (5): 1129-39.

Life is an adventure in forgiveness.
Norman Cousins (1915–1990), author, editor, journalist, and professor

I would rather try to persuade a man to go along, because once I have persuaded him he will stick. If I scare him, he will stay just as long as he is scared, and then he is gone.
Dwight D. Eisenhower (1890–1969), U.S. general and 34th president