SILENT CHALLENGE. EARLY KIDNEY TRANSPLANT FAILURE AND CARDIOMYOPATHY.

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Key clinical Message

Cardiac remodeling occurs frequently in hypertensive patients with chronic kidney disease. However, other diseases with myocardial involvement should be considered. This clinical image shows the importance of the endomyocardial biopsy.

Abstract

Differentiation of hypertrophic cardiomyopathy phenotypes is challenging but crucial for appropriate management. We report a case of myocardial oxalate deposition as an infrequent cause of infiltrative cardiomyopathy.

We present a 57-year-old woman with history of essential arterial hypertension since 2008 adequately controlled, and chronic kidney disease of unknown etiology undergoing dialysis since 2014. Normal cardiac echocardiographic findings until 2018, when progressive development of left ventricular hypertrophy was observed in repeated echocardiographic examinations (initially attributed to hypertensive cardiomyopathy). She received a cadaveric kidney graft in 2019 and presented early renal failure after transplantation. A kidney biopsy revealed tubulointerstitial nephropathy due to oxalate crystals and genetic study confirmed Primary Hyperoxaluria type 1.

Echocardiogram showed left ventricular dysfunction and severe wall thickness increase with granular texture **(figure 1A)**, right ventricular wall thickening, biatrial enlargement and a restrictive mitral Doppler signal. Endomyocardial biopsy (EMB) demonstrated rosette-like calcium oxalate crystals in myocardial fibers **(figure 1B, 1C)** and interstitial fibrosis. Myocardial T1 mapping in cardiac magnetic resonance (CMR) showed diffuse fibrosis, dilated left ventricle, pleural and pericardial effusion **(figure 1D)**. Right heart catheterization revealed postcapillary pulmonary hypertension (mean pulmonary artery pressure 44 mmHg, pulmonary capillary wedge pressure 30 mmHg, pulmonary vascular resistance 2,4 UW).

End-stage oxalosis cardiomyopathy was made after kidney transplant failure and a multidisciplinary approach was embraced. EMB was essential to confirm etiology of myocardial thickening and patients' prognosis.

Authors contributions

RS, FV, MG and JQ treated the patient and wrote the manuscript. All authors contributed to manuscript revisions. All Authors approved the final version of the manuscript and agree to be held accountable for the content therein.

Conflict of interest

The authors have no conflict of interest to disclose

Ethical approval

Written consent for publication was obtained from the patient and is available upon request.

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Reference section

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Figure legends

A: Two-dimensional 4 chamber view during diastole. Symmetrical left and right ventricular wall thickness with echo-dense speckled reflection.
B: Endomyocardial biopsy showing numerous oxalate crystals (circles). Hematoxylin and eosin (H-E) x 40.
C: Endomyocardial biopsy showing oxalate crystal viewed under polarized light (H-E x 200).
D: Non-contrast T1 mapping (CMR), short axis orientation. Increase myocardial native T1 value. Note presence of pericardial and pleural effusion.