Spontaneous chordae rupture of tricuspid valve in patient with chronic renal failure†

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Spontaneous chordae rupture of the tricuspid valve is relatively rare, unlike the mitral valve. We present a 27-year-old male with chronic renal failure on hemodialysis therapy for treatment of parathyroid adenoma. The echocardiography showed the severe tricuspid regurgitation with chordae rupture which was not noted 1 year ago. In addition, the papillary muscle of both mitral and tricuspid valve was shortened and calcified extensively. However, in his clinical history, the specific causes for chordae rupture, such as chest trauma or endocarditis, were not disclosed. It was presumed that dilated right ventricle with volume or pressure overloading and secondary hyperparathyroidism are probably responsible for the chordae rupture of tricuspid valve.

**KEYWORDS**
Tricuspid valve; Chordae rupture; Hyperparathyroidism; Echocardiography

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**Case report**

A 27-year-old male, diagnosed as chronic renal failure with IgA nephropathy, presented to our hospital for operation of parathyroid adenoma. He was being treated with hemodialysis and also, hypertension was accompanied with hemodialysis. One year ago, the echocardiography showed that papillary muscles of both mitral and tricuspid valve were severely calcified with shortened and calcified chordae of tricuspid valve (Figure 1), with decreased systolic function and dilated chambers. On this admission, the follow-up studies showed isolated severe tricuspid regurgitation with chordae rupture, calcified papillary muscle and limited motion of both leaflets (Figure 2). We excluded the infective endocarditis through the transesophageal echocardiography (TEE) and more detailed clinical history for trauma or physical injury. In his serologic test, the values of parathyroid hormone (1882 pg/mL), calcium (11.0 mg/dL) and phosphate (8.6 mg/dL) were all increased, respectively, and the product of calcium by phosphate was more over 70.

**Discussion**

The most common cause of tricuspid regurgitation is secondary form due to annular dilation and malcoaptation of the leaflet from underlying disorders such as pulmonary hypertension, primary left heart disease, or disease of the right ventricular myocardium. In addition, the etiology of isolated tricuspid valve regurgitation, especially rupture of chordae tendineae, was congenital, chest trauma or infective endocarditis. However, spontaneous chordae rupture of the tricuspid valve has been rare and the effects of chronic renal failure with long-term hemodialysis on the cardiac valve are not clear. In 1982, the report by Abrahams et al. which was based on examination of the heart at autopsy in patients with secondary hyperparathyroidism and long-term hemodialysis, showed the fibrosis, thickening and extensive calcification in the mitral valve apparatus. For these valvular changes, other potential explanations would be the increased cardiac output and mechanical stress on the valve apparatus.

Besides valvular calcification, Kunhali et al. reported the case of tricuspid papillary muscle rupture, which was associated with primary pulmonary hypertension. In that study, one explanation for papillary muscle rupture would be the increased wall tension in a dilated ventricle leading to the increased tension with predisposing to rupture.

In the present case, it is tempting to speculate that this is a consequence of long-standing progression of hyperparathyroidism and increased wall tension resulting from the...
hemodialysis, which would be the etiology of isolated spontaneous rupture of tricuspid valve apparatus in selected patients.

References

