

Tricuspid valve prolapse secondary to excessive long chordae evaluated by transthoracic echocardiography

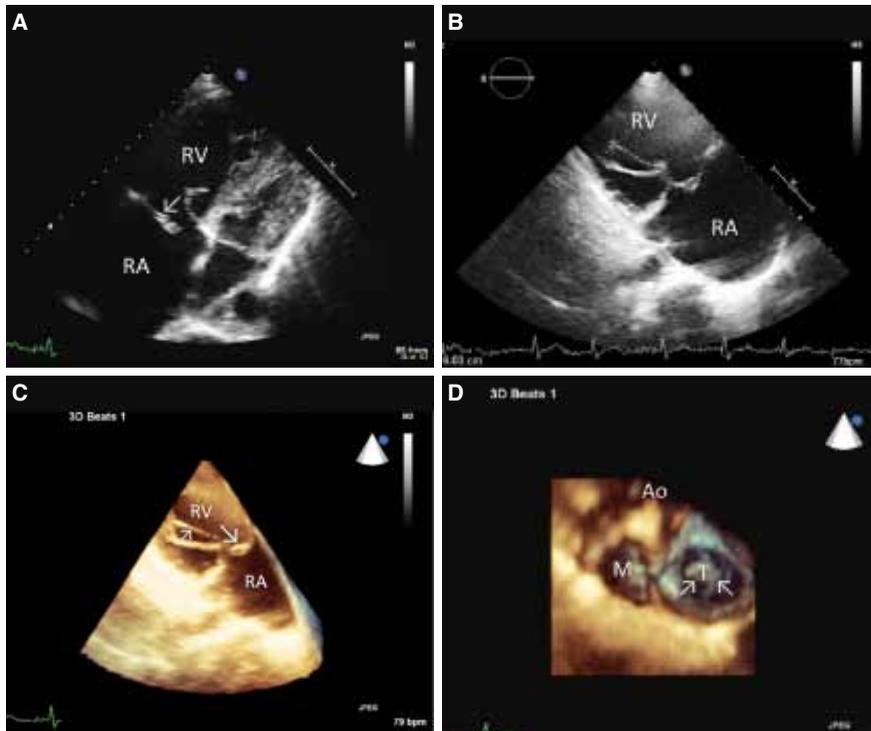
Uzun kordaya sekonder gelişen trikuspid kapak prolapsusunun ekokardiografik olarak değerlendirilmesi

Hayati Eren
Macit Kalçık
Gökhan Kahveci
Mehmet Özkan

Department of Cardiology,
Kartal Kosuyolu Training and
Research Hospital, Istanbul

A 48-year old man was referred to our outpatient clinic for pretibial edema and New York Heart Association Class 2 dyspnea. His chest radiogram showed mild pulmonary congestion with a normal cardiothoracic index, and electrocardiogram revealed normal sinus rhythm with right bundle branch block. Laboratory findings were not remarkable except for moderately elevated alanine transaminase, aspartate transaminase and brain natriuretic peptide levels. Two-dimensional transthoracic echocardiography (TTE) showed normal left ventricular systolic functions. The Apical four chamber view demonstrated moderately enlarged right atrial and ventricular cavities and tricuspid valve prolapsus (TVP) (Figure A) due to excessive long chordae (Video 1*). The chordae of the anterior leaflet was measured as 6 cm in length (Figure B). Severe tricuspid and mild mitral

regurgitant jets were obtained by color Doppler imaging. Estimated pulmonary arterial systolic pressure from tricuspid regurgitant flow was 60 mm Hg. Subsequently, a real-time three-dimensional (RT-3D) TTE was performed and the presence of anterior leaflet prolapsus due to elongated chordae was confirmed (Figure C and D, Video 2 and 3*). A two-dimensional TTE was unable to evaluate all the leaflets to rule out other possible etiological factors so, an RT-3D-TTE was performed to clarify the tricuspid valve which did not have any other primary pathologies except for the long chordae. Finally, valve repair surgery was recommended to the patient, which he refused. The most common cause of tricuspid regurgitation is not primary tricuspid valve disease, but rather an impaired valve coaptation caused by dilatation of the right ventricle and/or the tricuspid annulus. In the present case, the patient had no secondary reason for TVP and the long chordae were the only primary pathology. Here, excessive long chordae is highlighted as a rare cause of TVP to be reported in the current literature.



Figures– Two dimensional transthoracic echocardiography apical four chamber view demonstrated the displacement of the anterior leaflet of the tricuspid valve towards the left atrium during systole (A) due to excessive long chordae (B). (RA: Right Atrium, RV: Right Ventricle, Arrow shows prolapsed anterior leaflet of tricuspid valve). Three dimensional transthoracic echocardiography confirmed the presence of excessive long chordae (C) causing anterior leaflet prolapsus (D). (Ao: Aorta, M: Mitral Valve, RA: Right Atrium, RV: Right Ventricle, T: Tricuspid Valve, Arrow shows prolapsed anterior leaflet of tricuspid valve and long chordae). *Supplementary video files associated with this presentation can be found in the online version of the journal.