INTRODUCTION

Tricuspid regurgitation (TR) is characterized by classic physical examination and echocardiographic findings. The following case highlights some of the echocardiographic features of this very common and sometimes overlooked valvular disease.

CASE PRESENTATION

We present the case of a 50-year-old man with end-stage renal disease secondary to focal segmental glomerulosclerosis on home dialysis with a medical history significant for tricuspid valve (TV) endocarditis that developed approximately 7 years previously because of an indwelling catheter–related infection. The endocarditis resolved with an extended course of antibiotics, and the patient responded well to medical therapy, ultimately undergoing successful cadaveric renal transplantation 2 years later. According to the patient, he was doing well and had resumed a high level of physical activity, walking 2 to 3 miles a day at a brisk pace at least three times a week. Unfortunately, the patient experienced shortness of breath and decreased exercise tolerance despite adherence to standard therapy, and he needed renal replacement therapy 3 months before presentation.

Shortly thereafter, the patient began noticing increasing shortness of breath and decreased exercise tolerance despite adherence to medical therapy and dialysis. During a recent consultation for potential second kidney transplantation, he was found to be in congestive heart failure with right ventricular (RV) volume overload. On physical examination, there was a grade 4/6 murmur at the right sternal border that increased with inspiration, along with a pulsatile liver, but no RV heave. Transthoracic echocardiography was performed for further assessment.

Transthoracic echocardiography demonstrated severe right atrial dilation (area 37.3 cm²), mild RV dilation (TV annular diameter 5.3 cm), and low-normal RV systolic function (tricuspid annular plane systolic excursion 2.3 cm, fractional area change 38%). The anterior and septal leaflets of the TV were slightly restricted, as demonstrated on the apical four-chamber view focused on the right ventricle (Video 1). In a zoomed parasternal short-axis view, the anterior and septal leaflets of the TV appeared shortened, with a large central area of noncoaptation (Video 2). In addition, there was a freely mobile linear echodensity on the septal leaflet prolapsing into the right atrium in systole (Video 2).

Color Doppler flow revealed severe TR with vena contracta width of 3.2 cm (Figure 1, Videos 3 and 5). The RV inflow view also demonstrated a shortened and restricted posterior leaflet with the aforementioned large central area of noncoaptation (Video 4). Continuous-wave (CW) Doppler through the TV demonstrated a triangular jet envelope with a peak velocity of 2.56 m/sec, resulting in a gradient of 26 mm Hg between the right atrium and right ventricle on the basis of the modified Bernoulli equation (Figure 2).

Subcostal images demonstrated a dilated inferior vena cava (IVC) (maximum diameter 3.2 cm, minimum diameter 1.9 cm) with rhythmic, cyclical dilation and collapse secondary to severe TR (Figure 3, Video 6). With the IVC diameter >2.1 cm and <50% collapse, right atrial pressure was estimated to be 15 mm Hg. Combined with the previously mentioned TR velocity, calculated pulmonary artery systolic pressure was 41 mm Hg using the modified Bernoulli equation. Pulsed-wave Doppler of the hepatic veins also depicted systolic flow reversal (Figure 4). Of note, the left ventricle was remarkable for concentric hypertrophy (relative wall thickness 0.51, left ventricular mass index 119 g/m²) with mild global left ventricular systolic dysfunction (left ventricular ejection fraction 47%). In addition, there was septal flattening in both systole and diastole, consistent with right-sided pressure and volume overload (Video 7). There were no other significant valvular abnormalities.

DISCUSSION

Normal patients demonstrate variations in IVC diameter during respiration. The phasic pattern demonstrated above is most likely related to transmitted RA pressures. Pulsatile IVC flow and systolic portal vein flow reversal are commonly seen with severe TR. In severe TR, distal vascular impedance in the portal circulation is increased, highest in early ventricular systole. This impedance may hinder antegrade flow in the portal vein, leading to congestion. Chronic hepatic vein congestion can lead to cirrhotic changes in the liver, splenomegaly, and ascites.

This patient had a dilated right ventricle with distorted or flail leaflets and a dense triangular CW jet with an early systolic peak. The CW jet profile signifies rapid equalization of pressure between the right atrium and right ventricle, suggestive of severe TR. In this scenario, the peak velocity and pulmonary artery systolic pressure calculation...
would underestimate the degree of TR because the right ventricle does not need to generate a lot of pressure in the presence of two systolic outlets, the pulmonary artery and TV. Interventricular septal flattening throughout the cardiac cycle (RV volume and pressure overload) and reduced RV systolic function are also hallmarks of late-phase TR.

From the anatomic views in Videos 1 and 2, all three TV leaflets appeared shortened, retracted, and with freely mobile echodense material, likely representing fibrous tissue. Furthermore, there was leaflet tethering, likely related to RV dilation. Therefore, the most logical explanation for severe TR etiology in this case is a multifactorial process involving endocarditis-related valve or valvular apparatus damage along with RV remodeling. Chronic fibrosis of the TV leaflets after treatment likely resulted in RV volume overload and progressive RV dilation.

Primary disorders of the TV that can lead to significant degrees of TR include rheumatic heart disease, prolapse, congenital disease (i.e., Ebstein’s anomaly), infective endocarditis, prior radiation therapy, carcinoid heart disease, blunt chest wall trauma, RV endomyocardial biopsy–related trauma, and pacemaker or defibrillator leads. Approximately 80% of TR cases are functional (or secondary) in origin and related to tricuspid annular dilation and leaflet tethering in the setting of RV remodeling due to pressure and/or volume overload.

CONCLUSION

Transthoracic echocardiography is indicated to evaluate TR severity and etiology, RV systolic function, pulmonary artery systolic pressure, and IVC size in patients presenting with right-sided heart failure. We present a patient with a markedly pulsatile IVC due to severe TR. Typical echocardiographic findings of severe TR are a large, broad-based pansystolic jet profile on CW Doppler and a dilated IVC. Systolic flow reversal should also be present in the IVC and hepatic veins. During rapid equalization of right atrial and RV pressures, the maximum TR velocity may not be significantly elevated, but the CW Doppler jet profile will change from parabolic to triangular (Figure 2). Surgical intervention should be considered only in symptomatic patients who have indications for left-sided valve surgery or in whom medical therapy is unsuccessful.
SUPPLEMENTARY DATA

Supplementary data related to this article can be found at http://dx.doi.org/10.1016/j.case.2017.04.001.

REFERENCES

1. Bathla G, Singh S, Khandelwal G, Maller V. Pulsatile portal vein sign—an indication of underlying tricuspid regurgitation with congestive failure. Internet J Radiol 2008;9. Available at: http://ispub.com/IJRA/9/2/8978.

2. Abu-Yousef MM, Milam SC, Farner RM. Pulsatile portal vein flow: a sign of tricuspid regurgitation on duplex Doppler sonography. AJR Am J Roentgenol 1980;155:785-8.

3. Nishimura RA, Otto CM, Bonow RO, Carabello BA, Erwin JP III, Guyton RA, et al. 2014 AHA/ACC guideline for the management of patients with valvular heart disease: executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. Circulation 2014;129:2440-92.