Utility of 320-Slice Multi-Detector Computed Tomography for the Diagnosis and Evaluation of Cardiac Structures in a Patient With a Double-Chambered Right Ventricle

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Figure 1. (A) 320-slice multidetector computed tomography (MDCT): coronal section through the right ventricular outflow tract (RVOT) indicating hypertrophy of the right ventricle (RV) wall, an anomalous muscle band dividing the RV, and a post-stenotic dilated RV outflow trunk. (B) 320-slice MDCT: sagittal section through the RVOT. Hypertrophy of the infundibulum and RV free wall and anomalous muscle band resulted in the division of the RV into 2 chambers. Note that severe stenosis was found within the RV in the end-systole images (arrows). All examinations were performed using an Aquilion ONE CT scanner (Toshiba, Otawara, Japan). Contrast medium (iopamiron, Nihon Shering, Osaka, Japan; 65ml) was given i.v. at the rate of 4.0ml/s. A bolus tracking program was used to time the start of the scanning after the contrast material injection. The trigger threshold level was set at an increase of 100HU over the baseline CT number of the ascending aorta. The parameters were as follows: 120kV; 350mA; prospective ECG gating; 0–100% phase window; 16-cm craniocaudal coverage; detector configuration, 320×0.5mm; gantry rotation time, 350ms). (C) Right ventriculograms in the anterior–posterior projection showing the stenosis (arrows) in the RV where the stenosis was found on MDCT. (D) Measurement of intra-cardiac pressure. PA, pulmonary artery; RA, right atrium.
A 69-year-old woman was admitted to hospital with exertional dyspnea (New York Heart Association [NYHA] functional class III). This symptom had first appeared at the age of 59, and her symptoms had been getting worse. Physical examination demonstrated jugular venous distension and a grade 4/6 systolic murmur on the left sternal border at the second intercostal space. Electrocardiogram (ECG) demonstrated atrial fibrillation (AF) with a QRS rate of 46/min and left ventricular (LV) hypertrophy. Non-sustained ventricular tachycardia was also found on ECG monitoring. Chest X-ray demonstrated cardiomegaly (cardiothoracic ratio 68%). Echocardiography indicated an enlarged right atrium with severe tricuspid regurgitation and thickened apical portion of the ventricles. Intraventricular narrowing was also suspected in the right ventricle (RV), but a precise diagnosis was difficult because of the poor images obtained. The LV end-diastolic diameter and ejection fraction were 41 mm and 45%, respectively.

In spite of the data acquisition during chronic AF, 320-detector-row computed tomography (MDCT) clearly demonstrated RV hypertrophy with marked trabeculations and an anomalous muscle bundle dividing the RV into an inflow chamber and outflow chamber (arrows; Figures 1A, B; Movies S1, S2). These bands reached inward from the interventricular septum and the anterior free wall, and severe stenosis was noted within the RV on the end-systole images. No marked thickening of the ventricular septum was found. All those findings were suggestive of a double chambered RV (DCRV).\(^1\)

The patient refused myocardial biopsy and cardiac surgery, and cardiac catheterization was performed for a definitive diagnosis and assessment of the therapeutic effectiveness of apical pacing and/or cibenzoline. Right ventriculography produced the same findings and stenosis at the identical RV site as was confirmed on MDCT (arrows; Figure 1C). The pulmonary artery and RV outflow tract had a normal pressure, but an abnormally high pressure was recorded (90/5 mmHg) in the RV inflow tract, indicating a significant stenosis with a pressure gradient of 60 mmHg at this portion (Figure 1D). Right ventricular apical pacing (VVI pacing; 70 ppm) or a single i.v. dose of cibenzoline (70 mg) were each effective in decreasing the pressure gradient (Figure 2). Furthermore, a prominently synergic effect of the simultaneous treatment with RV pacing and cibenzoline was observed, and the pressure gradient decreased to 13 mmHg (Figure 2). The patient received an implantable cardiac defibrillator with VVI pacing (70 ppm) and oral cibenzoline (200 mg/day). One month after receiving the hybrid therapy with RV apical pacing and cibenzoline, the patient’s status improved to NYHA class II, and Doppler confirmed an improvement in the systolic pressure gradient of 10 mmHg. The septal-to-posterior wall motion delay increased from 77 to 280 ms,\(^2\) and the time difference of the first peak between the earliest and latest segments on the time-radial strain curve of 149 ms to 339 ms,\(^3\) indicated the appearance of LV dyssynchrony. LV function was preserved, however, with an ejection fraction of 45%. The patient has done well with no deterioration of her heart failure during 10-month follow-up.

Sixty-four-slice MDCT has been widely used for cardiac imaging in the clinical setting,\(^4\) but it requires approximately

Figure 2. Effect of right ventricular apical pacing and/or cibenzoline on the intracardiac pressure gradient in the right ventricle. RV (OT), right ventricular (outflow tract).
5–8 heartbeats to capture the entire heart, and banding artifacts and stair-step artifacts inherent in 64-slice technology cannot be completely avoided.4,7 Recently advanced wide-area coverage MDCT, such as 256-slice and 320-detector row MDCT, however, has great advantages over 64-slice MDCT in terms of quality, reduction in the radiation and contrast dose, and imaging in the setting of arrhythmias or irregular heart rates.6,7 All the scan data for the 320-slice MDCT can be acquired in the same R–R interval (1 heartbeat), making it possible to scan patients with arrhythmias or heart rate irregularities without stair-step artifacts.6,7 In the present case, although CT was performed during AF, detailed and precise anatomical and functional information on the DCRV were provided by the clear-cut rest and animated images with no artifacts.

A DCRV is a rare congenital heart disorder in which the RV is divided into a high-pressure inflow chamber and lower pressure outflow chamber by anomalous muscle bundles.1 Although it is difficult to differentiate between a DCRV with no other anomaly and an intraventricular RV obstruction in hypertrophic cardiomyopathy (HCM), we think that the absence of marked thickening of the ventricular septum in this case is supportive of the diagnosis of DCRV.8 The only established therapy for DCRV is the surgical removal of the anomalous muscle band, and no medical therapy has been established to reduce the intraventricular pressure gradient.1 In the present case, we demonstrated for the first time the synergetic effect of RV apical pacing and cibenzoline in reducing the RV pressure gradient in DCRV. All or some of the reported proposed mechanisms of the effectiveness for intraventricular RV obstructions in HCM,8–11 such as suppressing the myocardial contractility and improving the diastolic dysfunction due to a marked reduction in the intracellular Ca2+ concentration by cibenzoline and modifying the contractile behavior, improving the myocardial perfusion, and eliminating drug-induced bradycardia by pacing, might act to attenuate the dynamic RV obstruction contributing to the gradient.1 This therapy may be promising and become an alternative in patients who refuse cardiac surgery.

Disclosure

No author has a real or perceived conflict of interest.

References


Supplemental Files

Supplemental File 1

Movie S1. 320-slice multidetector computed tomography (coronal section) demonstrating hypertrophy of the wall of the right ventricle (RV), an anomalous muscle band dividing the RV, and a post-stenotic dilated right ventricular outflow trunk.

Supplemental File 2

Movie S2. 320-slice multidetector computed tomography (sagittal section) demonstrating hypertrophy of the infundibulum and right ventricular free wall, and the anomalous muscle band resulting in a division of the right ventricle into 2 chambers.

Please find supplemental file(s);