International Journal of Cardiology 220 (2016) 828–832

Contents lists available at ScienceDirect

International Journal of Cardiology

journal homepage: www.elsevier.com/locate/ijcard

Editorial

Apical aneurysm, apical thrombus, ventricular tachycardia and cerebral hemorrhagic infarction in a patient of mid-ventricular non-obstructive hypertrophic cardiomyopathy: A case report

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ARTICLE INFO

Article history:
Received 26 April 2016
Accepted 27 June 2016
Available online 29 June 2016

Keywords:
Dabigatran
Mid-ventricular hypertrophic cardiomyopathy
Aneurysm
Thrombus
Cerebral hemorrhagic infarction

1. Background

Hypertrophic cardiomyopathy (HCM) is a genetic cardiomyopathy with segmental myocardium hypertrophy, in terms of clinical course and phenotypic expression [1,2]. As a rare and often missed subgroup of HCM, mid-ventricular hypertrophic cardiomyopathy presenting mid-ventricular muscular constriction, often accompany with high risk to presenting apical aneurysm, apical thrombus, embolic events and ventricular tachycardia (VT), especially in patients with obstruction [3–6]. However, these complications can similarly happen in patients without obstruction. In this report we present a rare non-obstructive mid-ventricular hypertrophic cardiomyopathy case combined with cerebral hemorrhagic infarction, apical aneurysm, apical thrombus and sustained VT.

2. Case presentation

A 42-year-old man was admitted to the local cardiology department because of sustained ventricular tachycardia (Fig. 1A). He frequently felt chest distress and palpitation at activity for past five years. Several past echocardiogram demonstrated mid-ventricular hypertrophic cardiomyopathy with dubiously apical aneurysm. His family history was suspicious positive. His parents died of sudden death without determined etiology at sixty years old. The echocardiogram of his two brothers and a daughter was normal. His electrocardiogram at sinus rhythm demonstrated ST-segment elevation (≥1 mm), T-wave inversion and R wave high voltage in V4 through V6 (Fig. 1B). Unfortunately no drugs were prescribed by his doctor. He suddenly felt palpitation after gentle activity and went to the nearest hospital after one hour. Then his electrocardiogram showed wide QRS wave tachycardia at 206 bpm. Electric cardioversion was prescribed after the failure of recovering to sinus rhythm through intravenous injection of amiodarone. Sinus rhythm restored quickly after electric cardioversion but could not be maintained. His left limb's activity disorder appeared after seven times of electric cardioversion. Then this patient was transferred to our hospital. The magnetic resonance imaging (MRI) examination of his head showed ischemic stroke in large area affecting frontal lobe, parietal lobe, temporal lobe and occipital lobe (Fig. 3A). Cerebral hemorrhage in basal ganglia was found one week after his ischemic stroke (Fig. 3B). The brain magnetic resonance angiography (MRA) presented right middle cerebral artery without occlusion (Fig. 3C). The echocardiogram in our hospital displayed hypertrophy of left mid-ventricle (the most septal thickness was 22 mm) and a large thin-walled apical aneurysm with a large apical thrombus at a size of 27 mm × 20 mm (Fig. 2A). The constricted mid-ventricle separated left ventricle to two sections, apex and base respectively. His left ventricular end-diastolic dimension (LVEDD) was 49 mm, with ejection fraction was 67.6%. Continuous Doppler tracing revealed the intraventricular peak flow velocity of the diastolic paradoxic jet flow and systolic flow (Fig. 2B, C and D). But no intracavitary gradient between apex and base of left ventricle was identified, implying that the accurate diagnosis of our patient was non-obstructive mid-ventricular hypertrophic cardiomyopathy. Cardiac MRI also showed mid-ventricular hypertrophy, apical aneurysm and a big apical thrombus (Fig. 2E and F). Coronary angiography exhibited normal vessels. The left ventricular angiography in the right anterior oblique projection at end systole exhibited a mid-ventricular muscular constriction resulting a dumbbell shape and separating distinct proximal and distal chambers (Fig. 2G). An implantable cardiac defibrillator (ICD) was implanted with the defibrillation lead placing at the right ventricular apex after one month of his stroke (Fig. 1D). Dabigatran was administered after three days of his implantation at

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http://dx.doi.org/10.1016/j.ijcard.2016.06.282
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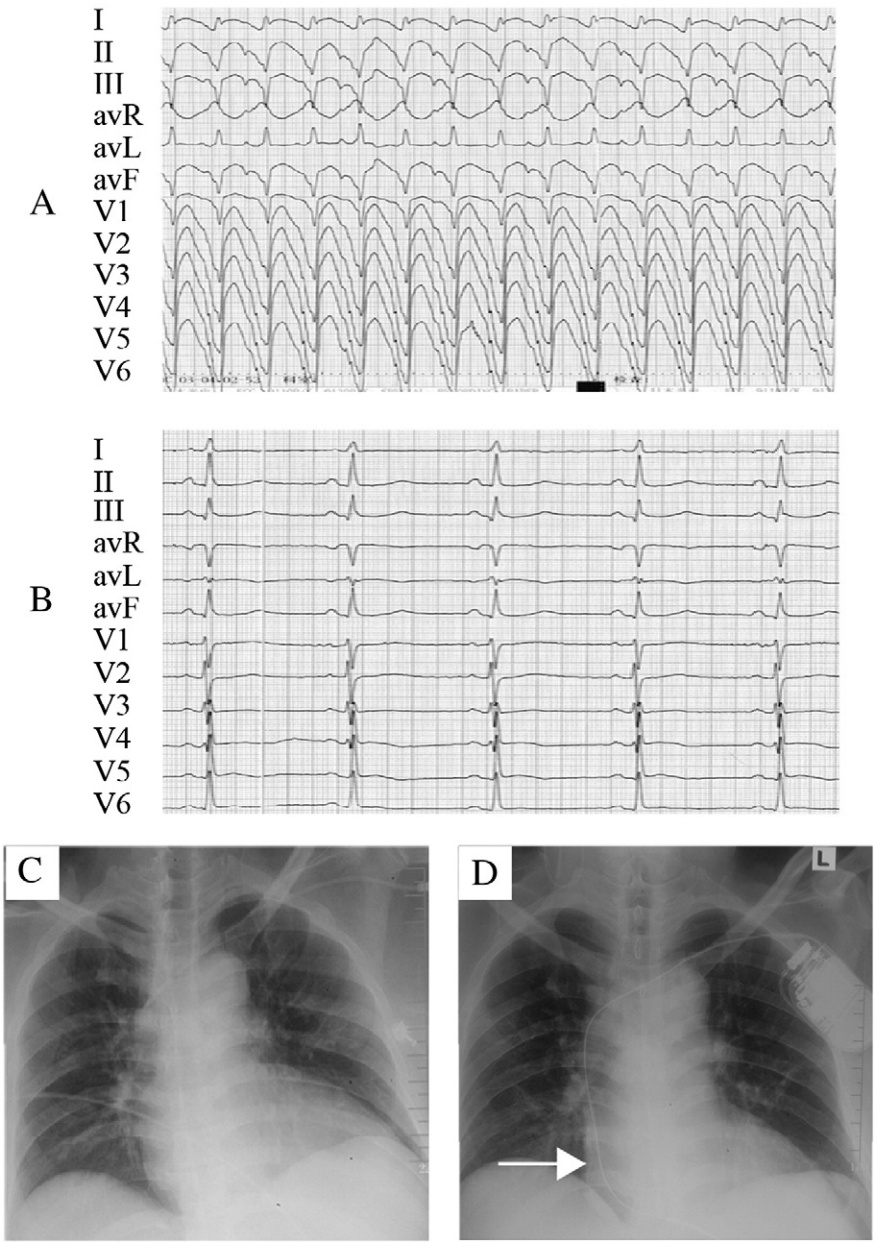

Fig. 1. A, the cardiogram of this patient exhibiting ventricular tachycardia, B, the cardiogram at sinus rhythm demonstrating ST-segment elevation (≥1 mm), T-wave inversion and R wave high voltage in V4 through V6. C, the chest X-ray at beside showing enlarge of heart. D, the chest X-ray at beside after ICD implantation indicating defibrillation lead locating in right ventricular apex (arrow).

110 mg per day for his cerebral hemorrhage was stable. Then the big apical thrombus in the heart became smaller significantly, with the size reducing from 27 mm × 20 mm to 13 mm × 10 mm after one week, and to 9 mm × 10 mm after two week of the administration of dabigatran (Fig. 3D, E and F). Then he discharged and prescribed with dabigatran (110 mg, BID), metoprolol (47.5 mg, QD), amiodarone (200 mg, TID). VT never occurred at one-month follow-up, with the apical thrombus disappeared.

3. Discussion

HCM is the most common type of cardiomyopathy, with a reported prevalence of 0.2% in the common population, characterized by marked difference in morphological expression and clinical history [1–2]. Mid-ventricular hypertrophic cardiomyopathy is a rare form of hypertrophic cardiomyopathy characterized by mid-ventricular muscular constriction which leads to a dumbbell shape of left ventricle [4]. Echocardiogram and cardiac magnetic resonance imaging (MRI) are two main means in confirming the diagnosis of mid-ventricular hypertrophic cardiomyopathy with special presentations. Mid-ventricular obliteration because of marked septal hypertrophy of left mid-ventricle is the main manifestation. If it presents with the peak instantaneous gradient between apex and base of left ventricle exceeding 30 mm Hg, mid-ventricular obstructive hypertrophic cardiomyopathy (MVO or MOHC) was diagnosed. The whole prevalence of MOHC varies from 8% to 12.9% in the entire HCM patients [4,5,7]. These reports also realized that MVO patients tended to be more symptomatic and had more outcome events. Therefore, MVO was a distinct phenotype of HCM patients accompany with unfavorable prognosis, sudden death and lethal arrhythmia [4]. However, those patients without the left ventricular gradient exceeding 30 mm Hg, called non-obstructive mid-ventricular hypertrophic cardiomyopathy (MNOHC), could induce same serious events as same as MOHC [8,9], like the occurrence of apical aneurysm formation. In our patient, the mid-ventricular gradient was

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