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Case Report

Myocardial bridging in adult with hypertrophic cardiomyopathy: Imaging findings with coronary computed tomography angiography ☆,☆☆

Vefa Celenk, MD[a], *Cetin Celenk, MD*[b,*]

[a] *Department of Cardiology, Institute of Cardiology, İstanbul University- Cerrahpasha, İstanbul, Turkey*
[b] *Department of Radiology, Faculty of Medicine, Ondokuzmayıs University*

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ABSTRACT

Myocardial bridgin in an adult with hypertrophic cardiomyopathy is a rare congenital coronary artery anomaly. It is often detected incidentally, and its true incidence in the general population is not known. Myocardial bridging may cause compression of a coronary artery, and it has been suggested that myocardial ischemia may result. Symptoms of myocardial bridging in the adult with hypertrophic cardiomyopathy are syncope, palpitations, dyspnea, and chest pain. Also, arrhythmia and myocardial infarction can be seen; these can cause sudden death, especially in athletes and young people. We present a case of a 48-year-old male with hypertrophic cardiomyopathy and myocardial bridging detected by coronary computed tomography angiography who complained of chest pain.

Introduction

Myocardial bridging (MB) is a congenital coronary artery anomaly in which a band of myocardium overlies a segment of the normal epicardial coronary artery.

While the average prevalence of MBs in adults is 25%, it is seen that this rate is 41% in adults with hypertrophic cardiomyopathy (HCM). This high rate demonstrates the importance of examining coronary CT scans of patients with HCM for MB [1].

Some reports have suggested an association between MB and increased severe symptoms, such as ventricular arrhythmias and sudden death in HCM [2,3].

Therefore, it is necessary to understand the diagnosis of MB in patients with HCM. Different modalities can be used for diagnosis. Coronary computed tomography angiography (CCTA) is a noninvasive imaging method preferred for patients with low to intermediate risk of coronary artery disease due to its high negative predictive value.

We present a case of a 48-year-old male patient who was diagnosed with HCM and MB on coronary CT angiography.

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* Corresponding author.
E-mail address: ccelenk@hotmail.com (C. Celenk).
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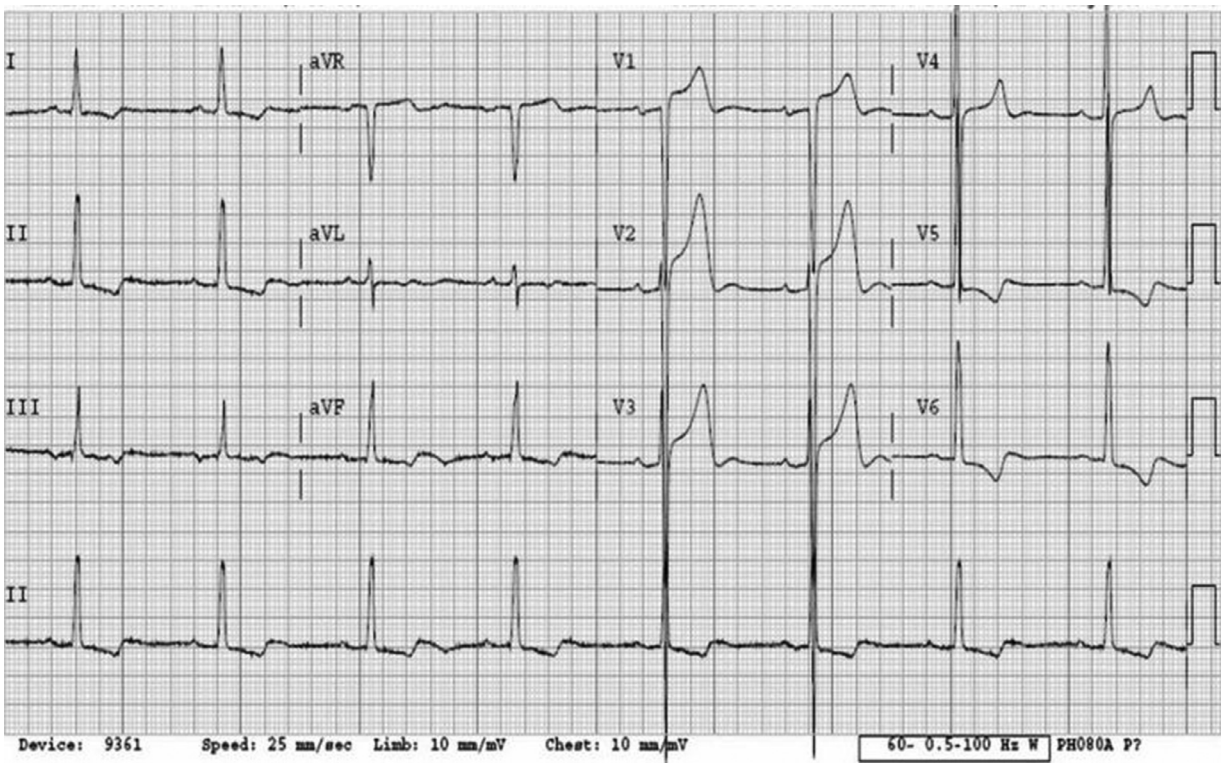

Fig. 1 – In the 12-lead ECG taken, sinus rhythm, heart rate 65/min, left ventricular hypertrophy and strain pattern in the precordial leads were observed.

We also present a literature review of this rare congenital anomaly.

Case report

A 48-year-old male patient with known hypertension, followed up with perindopril 2.5 mg tablet, applied for routine cardiology examination. No significant change was observed in the physical examination and laboratory findings of the patient. Blood pressure was 130/65 mm-Hg.

In the 12-lead ECG taken, sinus rhythm, heart rate 65/min, left ventricular hypertrophy and strain pattern in the precordial leads were observed (Fig. 1).

In the transthoracic echocardiography, both ventricular systolic and diastolic functions were within normal limits, and the left ventricular ejection fraction was 67% (measured by the Simpson method). A thickening reaching 17 mm was observed in the left ventricular basal segment, no gradient increase was observed in the left ventricular outflow tract. Valve functions and pericardium were normal (Fig. 2).

CCTA was planned because of the patient's history of chest pain symptoms consistent with CCS Class 1 Angina that had persisted for the last few years.

Written informed consent was obtained from the patient. The patient underwent CCTA to exclude coronary artery disease. The size, shape, and position of the heart were normal in CCTA. The right coronary system was dominant The right coronary artery (RCA), left main coronary artery (LMCA), and left anterior descending (LAD) had a normal origin, course,

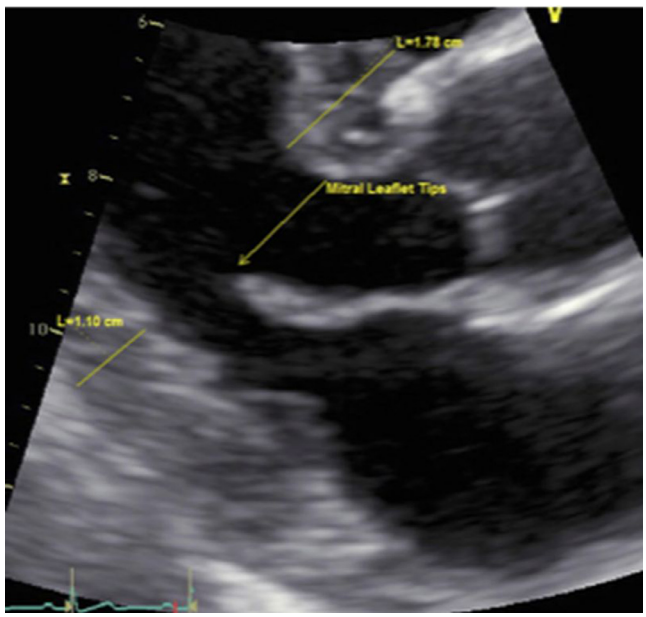

Fig. 2 – The parasternal long axis (PLAX) view showing the asymmetrical hypertrophy of the interventricular septum.

length, and luminal diameter. There was no pericardial effusion. The right coronary artery and the left main coronary artery arose from the right and left sinus Valsalva, respectively. There is a myocardial bridge localized on the distal tract of the

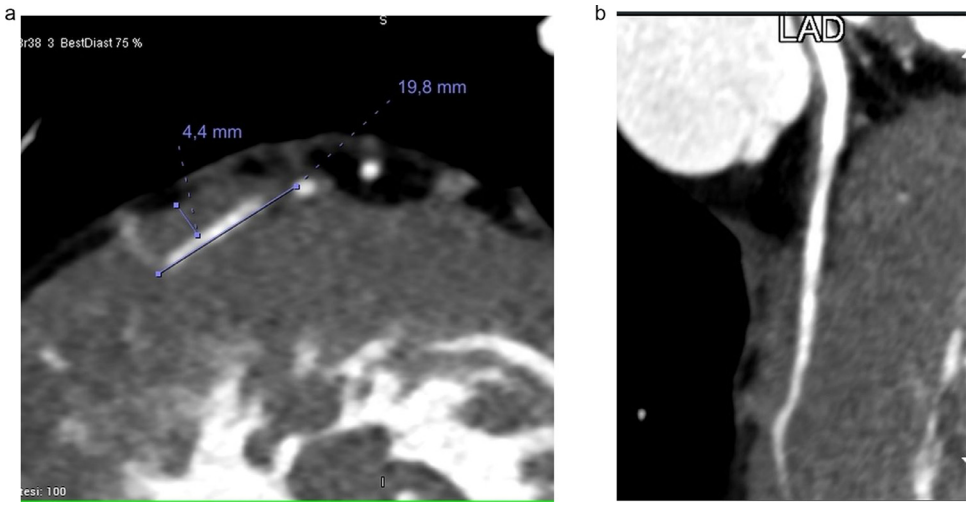

Fig. 3 – (A) Axial and (B) sagittal left ventricular cross-section views showing myocardial bridging in LAD central portion 5 mm deep and 20 mm in length.

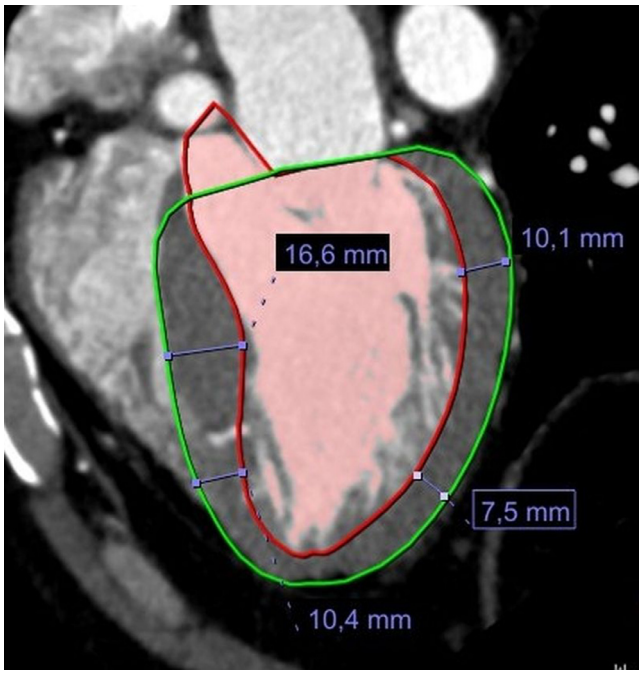

Fig. 4 – Left ventricular sagittal cross-section CT image showing asymmetric septal wall hypertrophy in diastole.

LAD, approximately 4 mm deep and with a 20 mm tunneled segment (Figs. 3A, B).

A CCTA revealed that; septal wall thickness in the anterior and central of 10 mm and 17 mm and free wall thickness in the anterior and posterior of 8 mm and 10 mm, respectively (Fig. 4). Septal wall thickness is suitable with HCM. Also, systolic narrowing was seen in the LAD central portion (from 22 to 1.9 mm) (milking effect) (Figs. 5A, B). No plaque or stenosis was detected in these arteries.

After CTA, metoprolol 50 mg was added to the current treatment of the patient.

Discussion

HCM is characterized by left ventricular hypertrophy (wall thickness >12-15 mm; normal wall thickness is 12 mm or less, measured during diastole) without obvious etiology.

HCM is a common inherited heart disease that is characterized by cardiac hypertrophy, unexplained by abnormal loading conditions [4].

Baxi et al. defined HCM in 7 different types morphologically [5]. In our patient, focal basal septum, HCM (C) is seen.

The asymmetric septal form is the most common morphologic variant of HCM and accounts for up to 60%-70% of cases [6–11]. In asymmetric HCM, the ventricular septum is disproportionately enlarged, with the anteroseptal myocardium most commonly involved. The septal hypertrophy can be limited to the subaortic, midventricular, or apical regions.

MB occurs when a portion of an epicardial coronary artery takes an intramuscular course and is tunneled under an overlying muscular bridge [12].

Anatomically, MB varies in length, degree of contractility, depth/thickness, and location, and is more common in the LAD coronary artery than other branches [13,14].

MB frequency has been reported to be 0.8%-4.9% in catheter angiography studies [15,16].

The prevalence ranges anywhere from 0.5% to 85%, noted mostly on coronary angiography and autopsy [17].

However, autopsy studies showed wide variance, ranging from 5% to 86%.

In CCTA studies, however, MB prevalence was shown much higher (3.5%-30.5%) than catheter angiography [14,18].

Compression of the LAD coronary artery is found in 30%-50% of adults with HCM [19]. It is not clear why, on average, patients who had bridging were older than patients without bridging when their HCM was diagnosed. Longitudinal angiographic studies would need to be done to determine whether the MB was congenital or acquired and what factors are linked to the development of bridging [20].

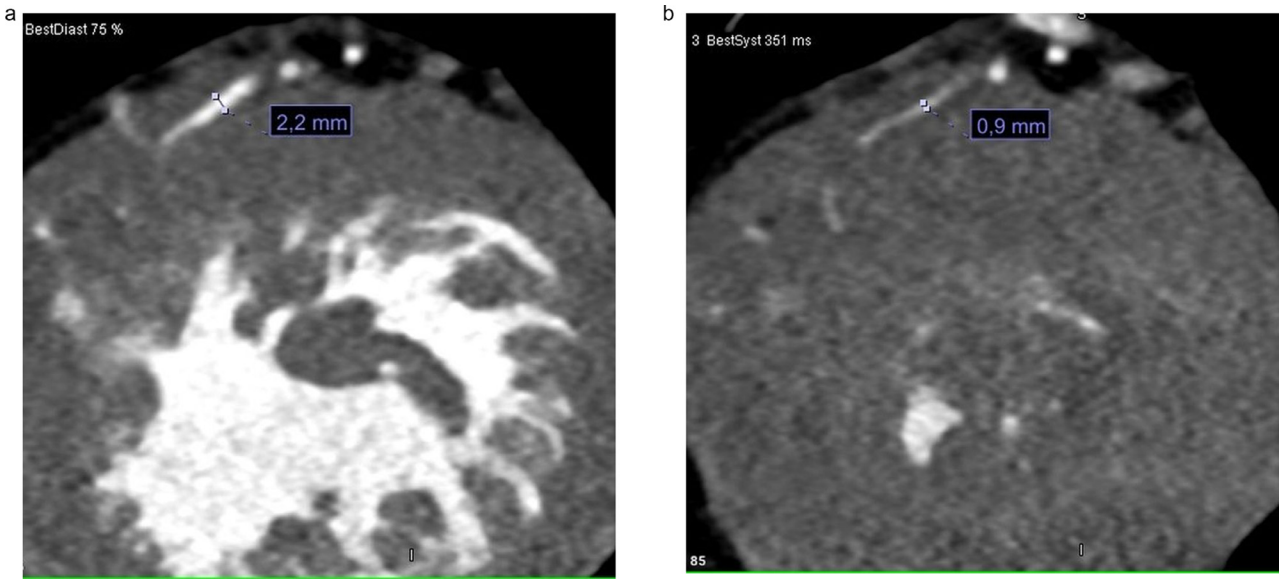

Fig. 5 – Left ventricular sagittal oblique cross-section view showing: (A) in LAD in bridging segment lumen diameter is 2.2 mm in diastole. (B) In LAD in bridging segment lumen diameter is 0.9 mm in systole.

Syncope, palpitation, dyspnea, chest pain, arrhythmia, and myocardial infarction can be seen in patients with hypertrophic cardiomyopathy disease (HCD). It is often revealed by sudden death, especially in young adults and athletes [21,22].

In general, narrowing at systole and widening at diastole in the coronary artery segment during the catheter coronary angiography (it is called the milking effect) has been accepted as diagnostic for MB [23,24]. The milking effect phenomenon is a pathognomonic angiographic finding in the MB of coronary arteries. Systolic compression of coronary vessels with partial or complete decompression during diastole may describe as a milking effect in CCTA.

Conventional coronary angiography and CCTA can be used in the diagnosis of MB with HCM. CCTA is preferred in the diagnosis of MB in the adult with HCM because it is a noninvasive diagnostic imaging method.

CCTA offers excellent spatial resolution and constitutes an alternative modality in patients. Noninvasive studies such as CCTA and MRI can also be used to diagnose MB. Also, invasive studies such as intravascular ultrasound and intracoronary Doppler sonography can be used for diagnosis [25].

CCTA is a currently evolving method as an alternative to diagnosing coronary anomalies. It provides information regarding the lumen and walls of the coronary arteries and is effective in evaluating coronary anomalies, as well as atherosclerosis, stent patency, bypass grafts, and myocardial irregularities [5].

Conclusion

MB in the adult with HCM is an uncommon abnormality rarely diagnosed in living patients. CCTA is the good imaging method to evaluate the origin, course, length, and luminal diameter of the coronary arteries. Activity may be restricted in children and young people because sudden death may occur. Here we present a case of MB in an adult with HCM as diagnosed by coronary CT angiography. Treatment was given to treat symptoms and a follow-up strategy was determined.

Ethics approval

Yes

Patient consent

Written informed consent was obtained from the patient for the publication of this case report, including accompanying images.

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