Available online at www.elixirpublishers.com (Elixir International Journal)

Cardiology

Elixir Cardiology 114 (2018) 49452-49454

ISSN: 2229-712X

Unknown Hypertrophic Cardiomyopathy Mimmicking Myocardial Infarction

M. Bennani, S. Ratbi, A. Daouda, J. Kheyi, I. Asfalou, M. Sabry and Z. Lakhal
Cardiology department of Mohamed V military hospital.

ARTICLE INFO

Article history:
Received: 9 November 2017;
Received in revised form:
24 December 2017;
Accepted: 5 January 2018;

Keywords
Hypertrophic Cardiomyopathy,
Cardiac MRI,
Myocardial Infarction.

ABSTRACT
Hypertrophic cardiomyopathy is a vast pathology, and may mimic coronary disease. Clinical data, electrical manifestations and laboratory parameters may drift toward the diagnosis of myocardial infarction. We report the case of a patient of 39 years, who presented a rare case of hypertrophic cardiomyopathy that manifested by acute coronary syndrome with ST segment elevation in anterior septum. Troponine was moderately high. Coronary angiography by cardiac catheterization was normal. Echocardiography and cardiac MRI allowed to pin down the diagnosis.

Introduction

Hypertrophic cardiomyopathy may be responsible for different clinical pictures. Among these manifestations, there is an acute painful form, with symptoms that may mimic coronary heart disease including myocardial infarction, and the presence of disorders of repolarization on electrocardiogram contributes to this confusion. Echocardiography is the first-line examination for the diagnosis of hypertrophic cardiomyopathy. In contrast, cardiac magnetic resonance imaging allows a comprehensive study of all segments of the left ventricle, the detection of areas of fibrosis in the myocardium, and evaluation of the prognosis of this disease. We report a patient who manifested with this problem of differential diagnosis between the existence of an unknown hypertrophic cardiomyopathy and myocardial infarction in an emergency setting.

Patient observation

Mrs. AM 39 years old, having as cardiovascular risk factors; android obesity, without a family history of sudden death, was admitted to the emergency room with suspected 5 hour acute chest pain of myocardial infarction.

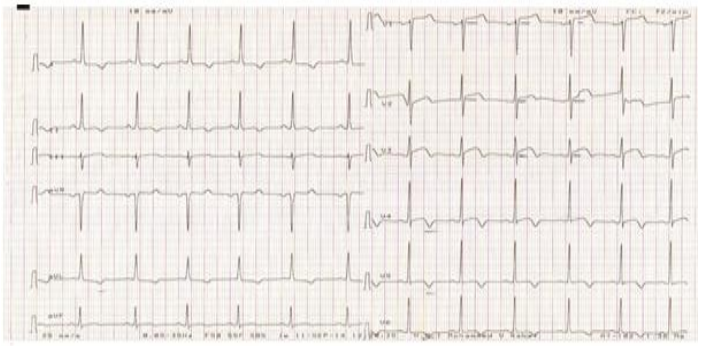

Figure 1. 12-lead ECG recording an additional ST segment shift in antero-septal, left ventricular hypertrophy and secondary repolarization disorders.

On clinical examination in the emergency she was conscious, in agony, eupneic at rest, able to lie supine. Blood pressure was 120 / 70mmHg with a heart rate of 70 beats / min and O2 saturation of 100% on room air. The rest of the examination was entirely normal.

The 12 lead electrocardiogram (ECG) showed regular sinus rhythm at 72 beats / minute, an extra ST segment encompassing the T wave in the anterior-septum, biphasic T waves in septo-apical and left ventricular hypertrophy type systole (Figure 1). The chest radiograph was normal. Troponin and cardiac enzymes were moderately elevated.

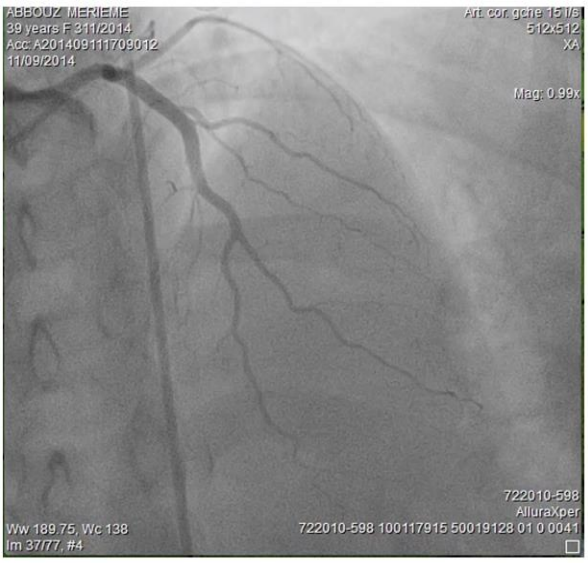

Figure 2. RAO 10 ° CRAN 40 incidence coronary angiography showing the entire non-atheromatous left coronary artery.

The patient had had coronary angiography that showed healthy arteries (Figure 2).Transthoracic echocardiography revealed a left ventricular hypertrophy of the septal wall (14 to 15 mm), a non-dilated left ventricle (46mm / 26mm), with a good global and segmental contractility. There was no left intraventricular obstruction.

Tele:
E-mail address: dr.s.ratbi@gmail.com

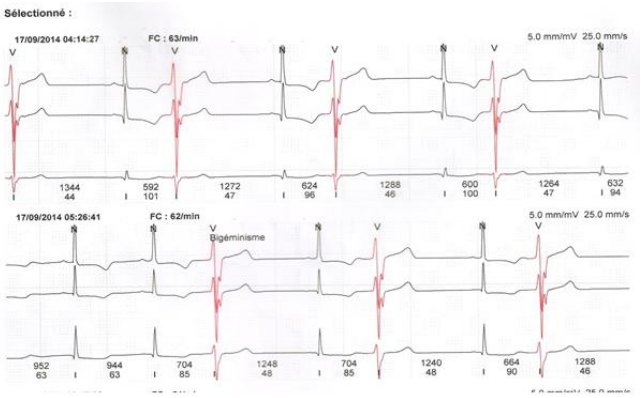

Figure 3. Holter ECG 24H: Objectively detecting the presence of isolated bimorphic ventricular extrasystoles, organized into bighetinism.

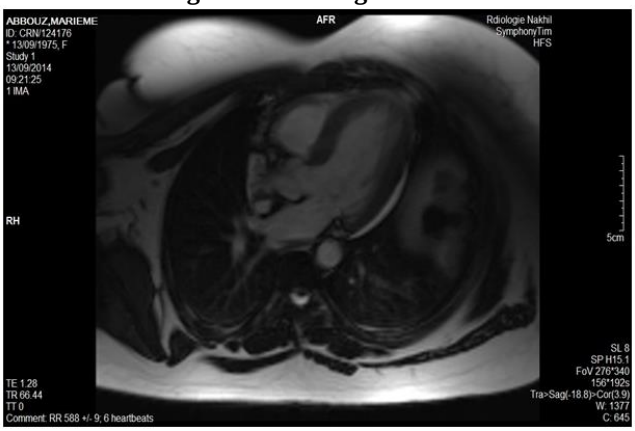

Figure 4. Cardiac MRI: Long-axis section showing asymmetric hypertrophic cardiomyopathy (CMH) of the mediospinal wall (15 mm).

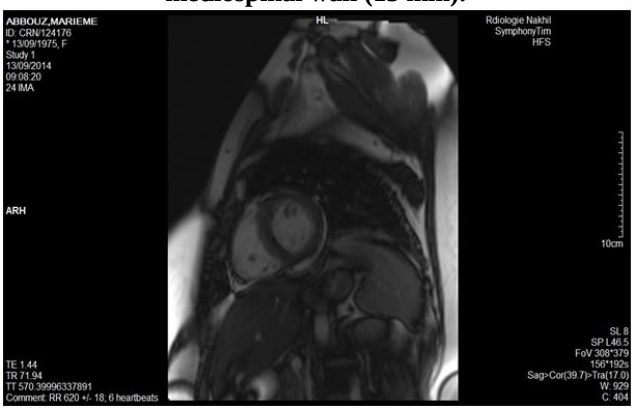

Figure 5. Cardiac MRI: Small axial cut showing an asymmetric CMH of the mediospinal wall (15mm).

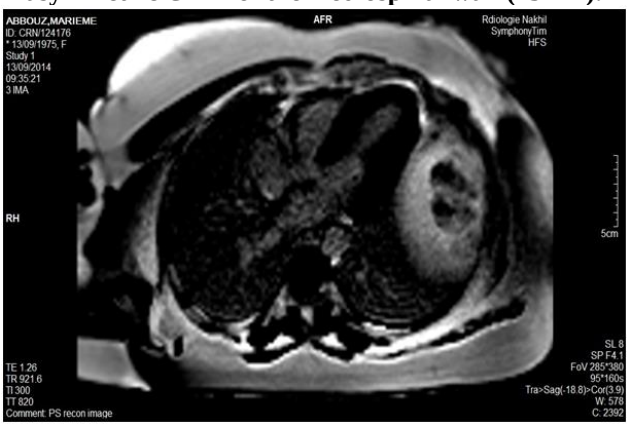

Figure 6. Cardiac MRI: Study of late enhancement showing no enhancement after 10 min.

Cardiac MRI revealed septal hypertrophic cardiomyopathy (base: 14mm, Medial: 15mm, Apex 9mm). Volumes and ventricular ejection fraction (73%) were normal. There were no signs of myocardial inflammation or fibrosis foci (Figure 4,5,6). The stress test was inconclusive. Holter ECG recorded bimorphic isolated premature ventricular contractions, bigeminy and trigeminy in organization (Figure 3), Stage II LOWN. There was no ventricular tachycardia, nor conduction disorders. Genetic evaluation and family screening is underway in this patient.

Discussion

Hypertrophic cardiomyopathy is a genetic disease with autosomal dominant pattern of inheritance characterized by a localized or diffuse hypertrophy of the left ventricle. It represents the first cause of sudden death in young adults and sports men. [1] Its prevalence is 0.2% of the general population.

Hypertrophic cardiomyopathy can be manifested by different clinical and electrical panels sometimes wrongly orienting towards the diagnosis of coronary heart disease. Chest pain at effort or rest is part of the clinical picture in 40 to 50% of cases. [2] This pain in not soothed in fact can be worsen by taking nitrates derivatives.

The mechanism appears multifactorial: a disease of the small intra-myocardial vessels[3], compression of perforating septal arteries in systole [4], spasm of small coronary arteries [4], a limitation on the possibility of extracting oxygen on increasing the demand and finally, inadequate capillary density [5].

The electrocardiogram is abnormal in 92% of cases. The three types of the most common defects are: cavitary hypertrophy, conduction disorders, and primary disorders of repolarization which are more or less a type of sub-epicardial ischemia. We also see subendocardial ischemia, rarely sub-endocardial lesions or sub-epicardial as is the case in our patient. Q waves, often fine and deep, called pseudo necrosis Q wave are present in 36 to 56% of cases.

Doppler echocardiography is the tool of choice for the diagnosis of this disease and its monitoring. Left ventricular hypertrophy associated with normal left chamber or of reduced size makes its diagnosis: one retains a diastolic threshold in adults 13 mm in familial forms and 15 mm in sporadic forms. Hypertrophy is generally asymmetric septal with a septum / posterior wall ratio > 1.3, that can affect the anterior portion of the septum (10%), the entire septum (20%) and this is the case of our patient, the septum and anterolateral wall (52%) or affecting other regions instead of the septum (18%).

MRI is useful in case of diagnostic doubt. It allows, thanks to its spatial resolution, the exploration of all segments of the left ventricle and particularly the antero-lateral and apical regions [6,7] which are sometimes difficult to visualize in echocardiography. It also allows to precisely measure the mass of the left ventricle ejection fraction. MRI also helps to stratify patients by studying late enhancement to identify areas of fibrosis and thus distinguish patients at high risk of sudden death [8], who may benefit from an implantable defibrillator for primary prevention.

Conclusion

Hypertrophic cardiomyopathy poses real diagnostic problems. Myocardial infarction or angina can be the first on differential diagnosis however the diagnosis is reached after carrying out specific examinations.

For this familial disease, the diagnosis should, after being discovered in a subject, be sought in all first-degree relatives by repeated clinical, ECG and ultrasound scan examinations.

References

1.Maron B.J., Roberts W.C., Epstein S.E. Sudden death in hypertrophic cardiomyopathy: a profile of 78 patients *Circulation* 1982 ; 65 : 1388-1394

2.Marcaggi X, Barraud P, Schaurdin C, Rozan JY, Lusson JR, Cassagnes J. Les myocardiopathies hypertrophiques primitives. Diagnostic et traitement. Rev Prat 1990;40: 2219-26

3.Maron BJ, Wolfson JK, Epstein SE, Roberts WC. Intramu-ral ("small vessel") Coronary artery disease in hypertro- phic cardiomyopathy. J Am Co// Cardioll986;8 :545-57

4.Pasternac A, Noble J, Streulen Y, Elier R, Henschke C, Bourassa MG. Pathophysiology of chest pain in patients with cardiomyopathy and normal coronary arteries. Circu- lation 1982;65 :778-8

5.Nishimura K, Nosaka H, Saito T, Nobuyoshi M. Another possible mechanisme of angina in hypertrophic cardiomyo-pathy [Abstract]. Circulation 1983;68 (suppl lll):lll-162

6.Rickers C, Wilke NM, Jerosch-Herold M, et al. Utility of cardiac magnetic resonance imaging in the diagnosis of hypertrophic cardiomyopathy. Circulation 2005 ; 112 : 855-61.

7.Moon JC, Fisher NG, McKenna WJ, Pennell DJ. Detection of apical hypertrophic cardiomyopathy by cardiovascular magnetic resonance in patients with non-diagnostic echocardiography. Heart 2004 ; 90 : 645-9.

8.Kim RJ, Judd RM. Gadolinium-enhanced magnetic resonance imaging in hypertrophic cardiomyopathy: in vivo imaging of the pathologic substrate for premature cardiac death? J Am Coll Cardiol 2003 ; 41 : 1568-72.