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RESEARCH ARTICLE

HYPERTROPHIC CARDIOMYOPATHY SIMULATING AN ACUTE CORONARY SYNDROME

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Abstract

Hypertrophic cardiomyopathy corresponds to myocardial hypertrophy >15mm or >13mm in case of familial hypertrophic cardiomyopathy (HCM).We report the case of a 31-year-old patient with no cardiovascular risk factors and no notable history who was admitted to the emergency room for the management of prolonged resting chest pain associated with electrical changes in the electrocardiogram (EKG)given the clinical symptomatology and the overshift with the mirror image. A coronary angiography was performed and came back without any particularity. The diagnosis of non-obstructive septal HCM was made based on echocardiography and cardiac magnetic resonance imaging (MRI) findings.The patient stayed in the cardiac intensive care unit with a good clinical course, he was declared to be discharged under symptomatic treatment given the non-obstructive nature and the low risk of sudden death.

Introduction:-

Hypertrophic cardiomyopathy (HCM) is defined as increased parietal thicknesses not explained by abnormal loading conditions [1]. This definition applies to both adult and pediatric populations, with no preconceived notions of etiology or underlying myocardial pathology.

There are two different types of HCM: sarcomeric HCM and non-sarcomeric HCM. The first is the most common form HCM and results from different genetic mutations that affect a beta myosin heavy chain (MYH7) or one of several other proteins than make up the sarcomere. How these genetic changes cause a thickened heart muscles is not completely understood.Non-sarcomeric HCM includes disorders formerly called "idiopathic" or "disorder of unknown cause." The most common mutation associated with non-sarcomeric causes is MYBPC3.

HCM is associated with various clinical presentations, and it is a challenging diagnosis because of its wide range of clinical, electrocardiographic, echocardiographic and imaging manifestations. In addition, HCM has been known to mimic other cardiac diseases such as acute coronary syndrome (ACS). The present report illustrates this particular point with a 31-year-old patient without cardiovascular risk factors and without medical or surgical history. The recognition of these clinical forms is crucial to limit more and more the invasive attitude in the diagnosis and the management.

Presentation of the case

A 31-year-old patient was admitted to the emergency room for acute chest pain associated with blockpnea. This pain initially occurred on exertion and then worsened 3 days before admission, when the patient presented with

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prolonged chest pain at rest. Thequestionningrevealed a patient without cardiovascular risk factors and without medical or surgical history. Moreover, there was no similar case in the patient's family. On examination, the patient was conscious, overweight with a BMI of 27kg/m^2. His heart rate was 58 B/M, his blood pressure was borderline at 91/46 with an average blood pressure of 57mmhg. He was eupneic with a respiratory rate of 12 breaths/min and an O2 saturation of 98% on room air.There were no signs of right or left heart failure. The complementary physical examination was normal. TheEKG showed a regular sinus rhythm with a ventricular rate (VR**)**of 56 beats per minute, a constant PR of 200ms, a 3mm ST-segment elevation in the septal and AVR, as well as diffuse negative T waves. The chest X-ray was unremarkable, and the transthoracic echocardiography (TTE) confirmed the presence of an asymmetric septal hypertrophy with a diameter of 30mm with no signs of obstruction. The left intra-ventricular gradient was 10mmhg (**Fig. 3**). It was decided to perform a cardiac MRI based on the radiological findings and given the clinical context. The results showed an aspect of hypertrophic cardiomyopathy involving the septum, the lateral wall without obstacle to the ejection of the left ventricle with the presence of foci of diffuse intramyocardial fibrosis involving the septum and the anterior wall of the left ventricle.

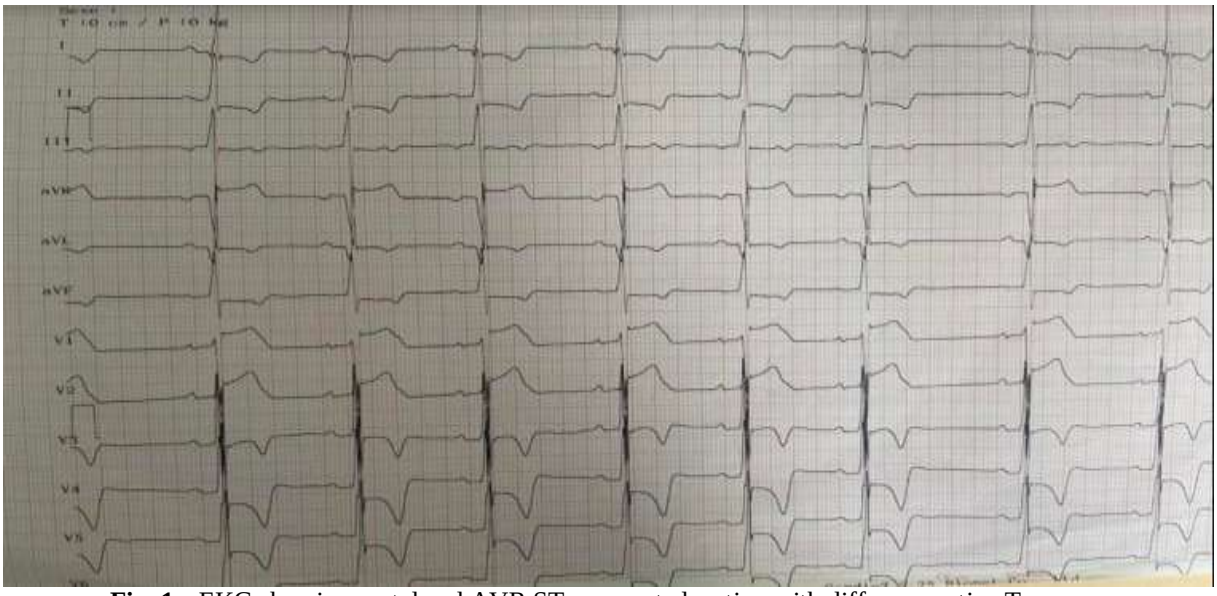

Fig. 1:- EKG showing septal and AVR ST-segment elevation with diffuse negative T waves.

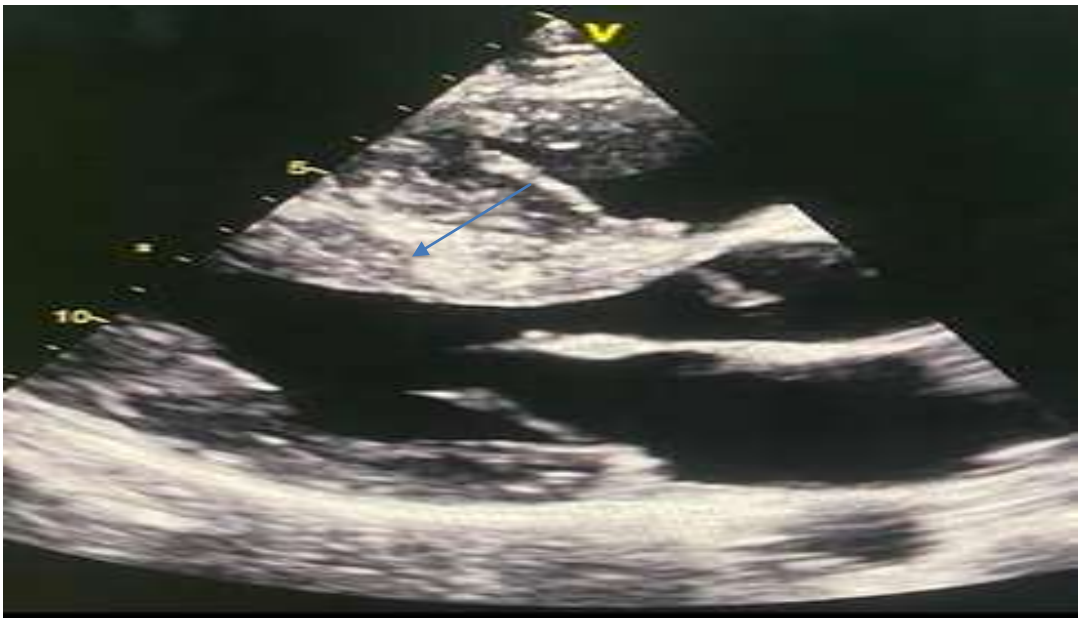

Fig. 2: Parasternal long axis section showing septal hypertrophy

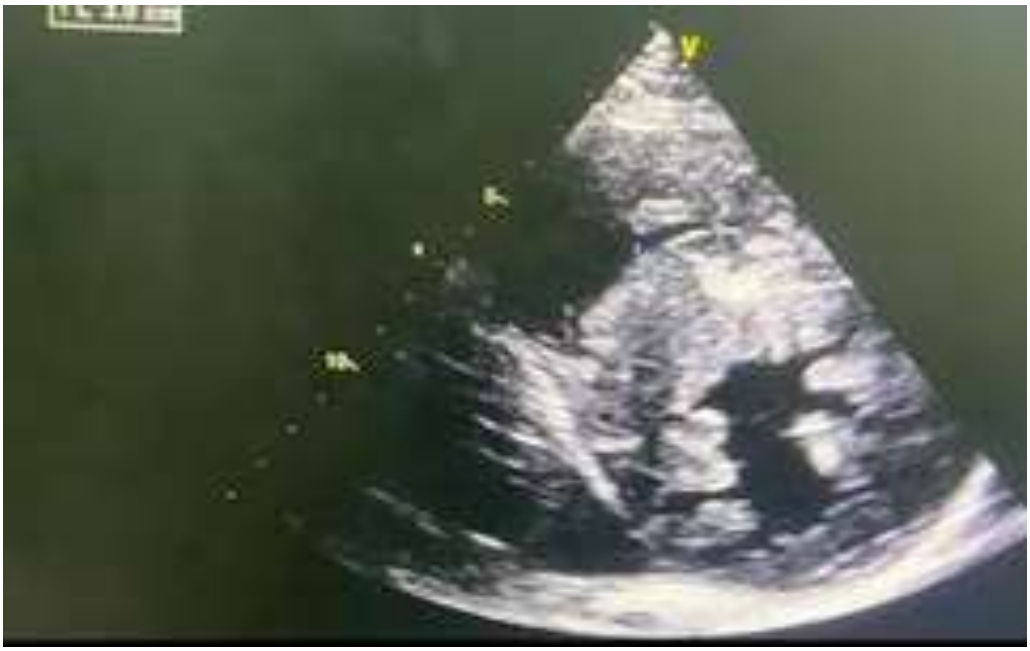

Fig. 3:- Parasternal small-axis section showing 30mm septal hypertrophy.

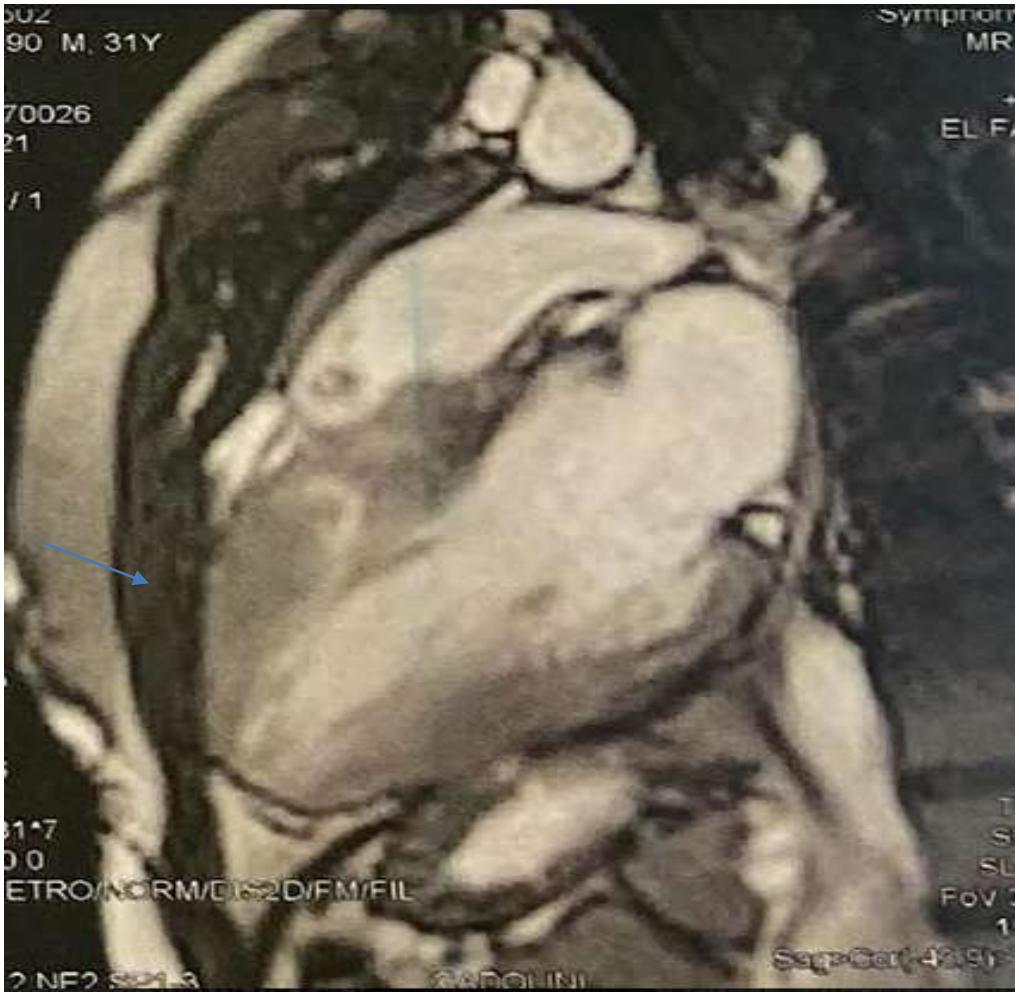

Fig. 4:- Cardiac MRI showing hypertrophic cardiomyopathy involving the septum and lateral wall.

Biologically, his troponin on admission was negative on 2 occasions, the C reactive protein (CRP) was negative 1.6mg; hepatic assessment and renal function were unremarkable.

Differential diagnoses included acute coronary syndrome, myocarditis, and other causes of acute chest pain.

The patient was admitted to the cardiology intensive care unit where he was conditioned and scoped. The diagnosis of hypertrophic cardiomyopathy was made on the basis of echocardiographic, biological, electrical and clinical evidence.

The patient did not receive any antiarrhythmic treatment because of the non-obstructive nature of his cardiomyopathy and his low risk of sudden death, and he was put on oral analgesic treatment on request.

It should be noted that the patient remained stable throughout his hospitalization and his chest pain disappeared on the first day of his hospitalization.He stayed 3 days in cardiological intensive care where iterativeEKGs were performed and which did not objectify any changes compared to the baseline ECG. He then was transferred to clinical cardiology; thereafter, he was declared discharged with a control appointment in a month.

Discussion:-

- Hypertrophic cardiomyopathy (HCM) is the most common genetic disease associated with more than 1000 mutations in 11 genes [7]. HCM is caused by an autosomal dominant mutation in genes that encode sarcomere proteins or sarcomere-associated proteins [8]. Evidence shows that 8 genes are known to definitively cause HCM: beta-myosin heavy chain, myosin-binding protein C, troponin T, troponin I, alpha tropomyosin, actin, regulatory light chain, and essential light chain [9,10].

- In the literature, several clinical situations have been described that may simulate STEMI on EKG ,It has been estimated that up to 2.3% of patients undergoing catheterization with suspected STEMI may be due toEKG mimicry. [2].

-Chest pain in HCM is multifactorial, it is due both to cardiac muscle hypertrophy with increased oxygen requirements especially during exercise, and on the other hand to compression of the coronary arteries secondary to cardiac hypertrophy. It should also be noted that microvascular dysfunction is a recognized feature of HCM, and its degree is a predictor of clinical deterioration and death [11]. This dysfunction may precede clinical deterioration by several years.

-The dynamic changes of the ST segment systematized in a territory and the borderline value of the ultrasensitive troponin or even slightly elevated values can aggravate this diagnostic confusion with ACS [3].Many cases have been described of apical HCM which is a rare form that predominates in Asian patients (25% of HCM in Japan vs. 2 to 9% in the West). Initially described by the Japanese (Sakamoto 1976), it is generally accompanied by precordial negative T waves and does not cause dynamic obstruction to LV ejection (Sato 2007 [4], Sakamoto 2001 [5]).Dynamic ST segment variations of ischemic origin are generally associated with mirror images, which is not usual in HCM [6) .

-In our case, the patient had presented, next to the thoracic pain, an elevation of the ST segment in V1-V2 and in AVR with a diffuse sub-shift. He was sent to the catheterization room in emergency where he had benefited from a coronary angiography, which came back without any particularity. Considering this context, a cardiac MRI was indicated coming back in favor of a septal CMH, which was at the origin of the electrical modifications simulating a STEMI.

-Cardiac imaging by magnetic resonance is gaining more and more importance in the etiological assessment of various cardiopathies; in recent years, it has made it possible to avoid numerous unnecessary coronary procedures.

Conclusion:-

-HCM is a pathology with a variable prognosis depending on the etiology.

-The calculation of the risk of sudden death is systematic in the presence of any HCM in order to discuss the implantation of an ICD if the risk is high.

-The treatment of HCM is initially based on antiarrhythmic drugs such as beta blockers.

-Interest of family screening in sarcomal HCM.

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