RESEARCH ARTICLE

CAN WE SURVIVE AFTER A LEFT VENTRICULAR FREE WALL RUPTURE WITHOUT SURGERY?: WHEN PATIENT’S WILL MATTERS!

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Abstract

Left Ventricular Free Wall Rupture (LVFWR) is the second most lethal complication of acute myocardial infarction, after cardiogenic shock. It occurs within a few days of an extensive myocardial infarction. Two distinct patterns are described; the first and most famous is the complete rupture leading to sudden death from cardiac tamponade. The second form, more rare, is rather less dangerous, appearing as an incomplete rupture or pseudo aneurysm formed by recurrent bleeding in the pericardium, which might also lead to fatal outcomes. Imaging tools especially echocardiography and cardiac magnetic resonance, which are performed depending on the clinical and hemodynamic conditions of the patient, confirm the diagnosis. Surgery is the first-line treatment of this condition. Herein we describe a case of a 53-year-old patient who presented to our department two weeks after myocardial infarction with an advanced right heart failure. However, his hemodynamic condition remained stable during hospitalization. Transthoracic echocardiogram showed pericardial effusion and signs of cardiac pre-tamponade. MRI showed the rupture site protected by a thrombus inside an apical pseudo-aneurysm. The patient was immediately subjected to cardiac surgery with the diagnosis of cardiac rupture. Unexpectedly he refused surgery and asked for immediate discharge. Three month later, we noticed a good clinical and echocardiogram outcome. We reviewed the literature of this lethal complication of acute myocardial infarction. This case report highlights the fact that in a world full of invasive therapy, non-invasive management could sometimes matter.

Introduction:-

Nowadays, Left Ventricular Free Wall Rupture (LVFWR) occurs in less than 1% of myocardial infarctions, thanks to the advent of reperfusion therapy (1). It is among the worst mechanical complications of myocardial infarction, which was first reported in 1647 by William Harvey (2).

Commonly, the acute form leads to sudden death from immediate cardiac tamponade. However, there is a less disastrous form designated as “sub-acute” or “contained” rupture, or left ventricular pseudo aneurysm, (3). We illustrate the latter with a clinical case to showcase its unusual favorable evolution without surgery, through a review of literature.
Case report:
A 53-year-old man with long history of smoking was referred to our emergency room with a right heart failure clinical presentation that had begun 2 weeks earlier, concomitantly to a chest pain neglected by the patient.

At admission, he was hemodynamically stable with peripheral edema and NYHA functional class III-IV. Vital signs included a BP of 110/60 mmHg, HR at 75 beats/min, temperature of 37°C, and oxygen saturation of 92% on room air. Heart auscultation revealed a discreet systolic murmur.

ECG showed a low QRS voltage at the peripheral leads, with Q waves of necrosis in the anterior leads and diffuse isoelectric T waves (Figure 1).

2D Echocardiography (Vivid 9, Cardiology B Department, Rabat, Morocco) performed immediately, showed a pericardial effusion of moderate to severe abundance, without diastolic collapse of heart chambers (Figure 2), the blood flowed from the LV to the pericardial space and diastolic re-flow was from the pericardial space to the LV with suspicion of LV apical image. Was also found an ischemic heart disease with mild systolic dysfunction.

Cardiac MRI, performed after stabilization of the patient, revealed a huge apical thrombus filling a pseudo-aneurysm of the LV contained by pericardial adhesions, clogging the wall tear (Figure 3).

Coronary angiography, performed to assess coronary anatomy, showed an occlusion of the proximal part of the LAD and along tight proximal and middle circumflex arterstenosis.

Thus, an intensive medical therapy was administered and an immediate surgical repair was recommended. However, the patient disapproved of the invasive management considering the high per and post-operative mortality, and was discharged against medical advice.

Unexpected improvement was noted three months later when the patient came back for checkup. TTE showed a regression of the effusion.

Figure 1:- ECG showing a low QRS voltage at the peripheral leads, with Q waves of necrosis in the anterior leads and diffuse isoelectric T waves.
Figure 2: Two-dimensional (2D) Transthoracic Echocardiogram (PSSA), showing severe pericardial effusion.

Figure 3: Four-chamber view on Cine Cardiac MRI(SSFP) showing a left ventricular pseudoaneurysm (red arrows) clogged by thrombus, and pericardial effusion.
Discussion:-
Sub-acute free wall rupture is a gradual or incomplete rupture of the infarcted area with slow or recurrent bleeding into the pericardial sac, contained by epicardial adhesions, causing progressive or recurrent cardiac tamponade. (4)(5)

This rupture of myocardium is contained by epicardial adhesions. It is a more rare complication compared to the complete rupture or true postinfarction aneurysms. Its true incidence is unknown since death can occur before hospital admission and autopsy reports are poor and variable. However, it is estimated that the incidence of LVFWR has significantly decreased in the era of reperfusion therapy, before which it could easily reach 6%, nowadays it is less than 2% (6) (7)

Actually, the starting point of a free wall rupture is an endocardial tear. A Japanese team evaluated 50 sudden out-of-hospital death cases (post-mortem) and concluded that endocardial tears concentrate on two sites: at or near the base of the papillary muscles, and in the area where the septum meets the cardiac free wall. (8) They result from the stretching of the adynamic, infarcted myocardium – becoming thin and soft due to proteolytic enzymes of necrosis - by left ventricular contraction as well as systolic pull of the mitral valve apparatus. (8) Some authors also blamed “reperfusion injury” due to thrombotic therapy and genetic predisposition leading to an inter-individual susceptibility (2).

Commonly, clinical presentation might be congestive heart failure, recurrent or persistent chest pain, ventricular arrhythmias and even embolization. Some patients remain asymptomatic and subacute ruptures are discovered several years later. (3) (10)

Diagnosis can be confirmed by several imaging techniques especially TTE and CMR. Routine echocardiography may detect a pseudo-aneurysm in an asymptomatic patient who is recovering from an acute myocardial infarction as well as a tamponade patient. Ultrasound has the advantage of being a rapid, innocuous, and readily available technique at the patient’s bedside.

CMR established the diagnosis in our case; in fact, it allowed distinction of pseudo aneurysm from a true aneurysm.

Surgical treatment is always recommended as the risk of outright rupture occurs in nearly half the patients, associated with a good intensive medical therapy as well. Pericardiocentesis is also an option to be used to stabilize the patient before definitive therapy. (3)(9)(10)

What happened in the case of our patient?
The contained rupture was attributed to pericardial adhesions, which might have been present at the time of infarction or may have developed de novo during rupture as the tract is usually small and narrow. An intrapericardial collection of clotted blood was formed, which might have elicited an inflammatory and fibrous reaction.

Thus, our patient survived by virtue of adherent thrombi or pericardial adhesion. Takuda also reported that the histological age of the thrombi found in situ was independent of those of the myocardial infarction (8).

Conclusion:-
Left Ventricular Free Wall Rupture is a fatal complication of acute myocardial infarction. Pseudo-aneurysm occurs when this rupture is incomplete as it is contained by adherent pericardium. Diagnosis is difficult and challenging with the absence of a specific clinical presentation. Hence, TTE remains a judicious first step that should be followed by other imaging techniques with higher sensitivity, such as CMR.

Surgery can save these patients. Untreated pseudoaneurysms have an approximately 30% to 45% risk of rupture. (10)

References:-
1. Toshimasa Okabe, MD Howard M. Julien, MD, MPH Antony G. Kaliyadan, MD Henry Siu, MD Gregory D. Marhefka, MD, FACC. Prompt Recognition of Left Ventricular Free-Wall Rupture Aided by the Use of Contrast Echocardiography. Volume 42, Number 5, 2015
2. Pradeep Vaideeswar, Jayashri P Chaudhari, Jagdish Butany. Mechanical complications of myocardial infarction.
3. Eren E, Bozbuga N, Toker ME, et al. Surgical treatment of post-infarction left ventricular pseudoaneurysm. Tex Heart Inst J 2007; 34: 47e51.
4. Purcaro A, Costantini C, Ciampani N, Mazzanti M, Silenzi C, Gili A, Belardinelli R, Astolfi D. Diagnostic criteria and management of subacute ventricular free wall rupture complicating acute myocardial infarction. Am J Cardiol. 1997;80:397–405.
5. Satoshi Honda, MD; Yasuhide Asaumi, MD, PhD; Takaumi Yamane, MD; Toshiyuki Nagai, MD, PhD; Tadayoshi Miyagi, MD; Teruo Noguchi, MD, PhD; Toshihisa Anzai, MD, PhD; Yoichi.
6. Trends in the Clinical and Pathological Characteristics of Cardiac Rupture in Patients With Acute Myocardial Infarction Over 35 Years. J Am Heart Assoc. 2014;3:e000984doi: 10.1161/JAHA.114.000984.
7. Becker RC, Gore JM, Lambrew C, Weaver WD, Rubison RM, French WJ, Tiefenbrunn AJ, Bowlby LJ, Rogers WJ. A composite view of cardiac rupture in the United States National Registry of Myocardial Infarction. J Am Coll Cardiol. 1996;27:1321–1326.
8. Moreyra AE, Huang MS, Wilson AC, Deng Y, Cosgrove NM, Kostis JB. Trends in incidence and mortality rates of ventricular septal rupture during acute myocardial infarction. Am J Cardiol. 2010;106:1095–1100.
9. Aya Takada, Kazuyuki Saito, Tatsuya Murai, Akihiko Hamamatsu. Endocardial tears and rupture tracts of left ventricular ruptures during acute myocardial infarction: An autopsy study of 50 out-of-hospital sudden death cases Received 7 July 2006; accepted 18 August 2006.
10. Yeo TC, Malouf JF, Oh JK, Seward JB. Clinical profile and outcome in 52 patients with cardiac pseudoaneurysm. Ann Intern Med 1998; 128: 299e305.
11. Frances C, Romero A, Grady D. Left ventricular pseudoaneurysm. J Am Coll Cardiol 1998;32:557-61.