CHEST

A 46-Year-Old Man Presenting With Orthopnea, Hypotension, and Abdominal Pain

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VIDEO

CHEST 2017; 151(3):e53-e56

A 46-year-old man was brought to the ED with severe acute dyspnea and signs of systemic hypoperfusion that had evolved for the previous 2 hours. He also manifested abdominal and chest pain. His medical history revealed active smoking; family medical history was unremarkable.

On presentation, his vital signs were a regular heart rate at 150 beats per minute, blood pressure at 80/40 mm Hg, respiratory rate of 46 breaths per minute, and temperature at 36°C. Pulse oximetry saturation was 70% at room air. The physical examination revealed a severely distressed, orthopneic, and poorly cooperative patient. He was extremely pale, diaphoretic, and had generalized skin mottling. He could scarcely state that the pain was located in the epigastrium but also radiated to the precordium and had evolved over the preceding 3 days. The pain was moderate in intensity, did not reproduce with palpation, and was accompanied by nausea. Cardiac auscultation revealed a rapid and regular heart rate; no murmurs were noted. Central and peripheral pulses were symmetrically filiform; jugular ingurgitation was absent. Respiratory examination showed an effortful tachypnea and wheezing; no rales were evident. ECG showed a regular sinus rhythm at 150 beats per minute and Q waves and a 1-mm ST-segment elevation in inferior leads. A specular ST-segment depression was also observed in high lateral leads. Notable laboratory data showed troponin I and creatine kinase MB isoenzyme elevated 5 and 10 times above the upper reference limit, respectively. A medical ICU consult was called. After initial history, physical examination, ECG, and laboratory review, bedside ultrasound [transthoracic echocardiogram (TTE) and lung ultrasound (US)] was performed to evaluate the etiology of dyspnea, chest and abdominal pain, and shock (Videos 1, 2).

Question: Based on Videos 1 and 2, the ECG, the patient's clinical presentation, and the laboratory data, what is the most likely diagnosis?

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DOI: http://dx.doi.org/10.1016/j.chest.2016.07.047

Answer: Rupture of the posteromedial papillary muscle, complicating an acute inferior myocardial infarction

Discussion

The patient presented with a cold and wet shock and abdominal and chest pain of a myocardial ischemic origin by means of clinical and ECG findings (Video 3). Biomarkers of myocardial infarction were elevated. The diagnosis of a mechanical complication [eg, acute mitral regurgitation (MR)] of an acute inferior myocardial infarction (AMI) was first considered as well as a proximal aortic dissection complicated with a severe acute aortic regurgitation and compromise of a sinus of Valsalva and thus coronary involvement. The latter is particularly relevant because approximately 20% of patients with a proximal dissection have ECG evidence of acute ischemia or AMI and/or wall motion abnormalities detected by TTE (particularly left ventricular inferior hypokinesia).[1] Another diagnosis also taken into account was an aortic or mitral valve endocarditis with acute severe regurgitation and septic coronary embolism causing AMI. The absence of fever and other indicators of infection decrease (although does not completely exclude) the likelihood of this condition. Other diagnoses, such as ruptured abdominal aorta, right ventricular infarction, acute pulmonary embolism, or hypovolemia, were not clinically considered at first glance because of the patient's presentation. Bedside US was considered of extreme importance to confirm one of our presumptive diagnoses, and therefore delineate the correct treatment, which in this case was considered a surgical emergency.

As shown in Video 3, lung US (Video 1, Clip 1) showed a diffuse bilateral and severe B-line pattern, corresponding to pulmonary edema (PE). This evaluation was obtained in seconds and performed with the patient seated because he was not able to tolerate supine decubitus. The presence of normal lung sliding, absence of subpleural consolidations, and a nonpatchy distribution of B lines and orthopnea strongly suggest the diagnosis of a PE of cardiogenic origin.[2] Parasternal long-axis view (Video 1, Clip 2) showed a hyperdinamic left ventricular systolic function, a normal aortic root and aortic valve, left ventricular posterior wall hypokinesia and a flail posterior mitral valve leaflet, which looked too long because it had attached to a thin structure corresponding to a chordae tendineae, and also a thick region compatible with a ruptured papillary muscle (PM) head. The presence of normal left ventricular size and function in the context of cardiogenic PE (drastic change in the pressure-volume relationship) should immediately raise the possibility of acute valvular regurgitation.[3] Color Doppler US showed an MR jet that was directed anteriorly. As a general rule, when there is a flail or prolapsed leaflet, the regurgitation jet follows an opposite direction. Because of the rapid pressure equilibration between the left ventricle (LV) and the left atrium (LA) in the context of severe acute MR, the MR jet is not seen as massive, and this explains why clinically an MR murmur is subtle or is not evident on cardiac auscultation.[3] Furthermore, because of its eccentric direction and tachycardia, the real magnitude of the MR can also be underestimated. Parasternal short-axis views at basal and midventricular levels (Video 1, Clip 3) showed a hyperdinamic left ventricular systolic function and clear inferior and posterior hypokinesia. When looking in detail, a mobile isoechoic structure was observed in the inferoposterior region, which was compatible with the flail mitral subvalvular apparatus. Right ventricle geometry looked normal. An apical four-chamber view (Video 2, Clip 4) showed hyperdinamic left ventricular systolic function and a flail posterior mitral valve leaflet, a flail chordae tendineae, and a ruptured PM head (Fig 1). An eccentric and anteriorly directed MR jet was evident on color

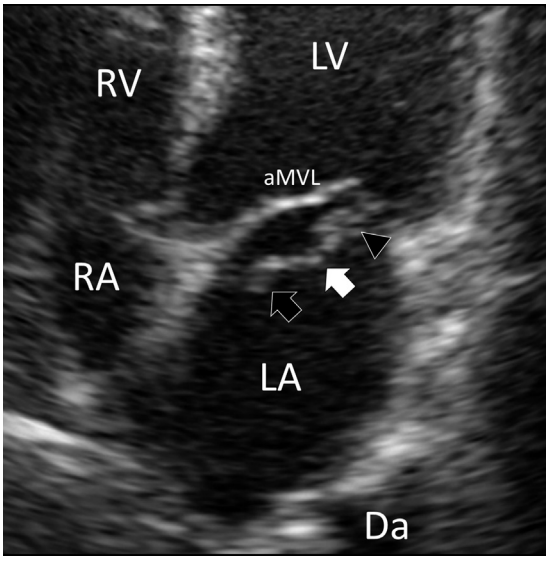

Figure 1 – *Transthoracic echocardiogram, apical four-chamber view. A flail posterior mitral valve leaflet is observed (arrowhead), with a thin structure in continuity with the leaflet corresponding to a flail chordae tendineae (white arrow) and a thick segment (black arrow) corresponding to a ruptured papillary muscle head. aMVL = anterior mitral valve leaflet; Da = descending aorta; LA = left atrium; LV = left ventricle; RA = right atrium; RV = right ventricle.*

Doppler US. Right ventricular size and function were normal. At an apical two-chamber view (Video 2, Clip 5), a hyperdinamic LV and inferior hypokinesia were also observed. In this view, a thin structure entering into the LA and attached to the posterior mitral valve leaflet was observed; this is compatible with a flail chordae tendineae (Fig 2). In a subcostal longitudinal view (Video 2, Clip 6), the inferior vena cava looked depleted and with full collapsibility; abdominal aorta was normal in size with no flap seen inside (not shown). The inferior vena cava, as observed in this case, is a real example teaching that this parameter cannot be used in an isolated manner to rule in or rule out cardiogenic PE. This is presumably explained from the large negative pleural pressure swings occurring with the patient's breathing pattern. No pleural or pericardial effusion was noted.

Acute mitral regurgitation (AMR) is one of the main mechanical complications of AMI, along with cardiac rupture and ventricular septal defect.[4] AMR complicating AMI can be categorized as functional (PM dysfunction) or mechanical in origin; the latter is because of partial (occurring at either one of the muscle heads) or complete PM rupture or chordae tendineae rupture. The posteromedial PM is more susceptible to ischemia because it generally has a single blood supply from the right coronary or circumflex artery; in contrast, the anterolateral PM is less vulnerable to ischemia because it typically has a dual blood supply from the anterior descending and circumflex arteries.[3,4] This difference in blood supply explains why rupture of the posteromedial PM is three to 12 times more likely than anterolateral PM rupture.

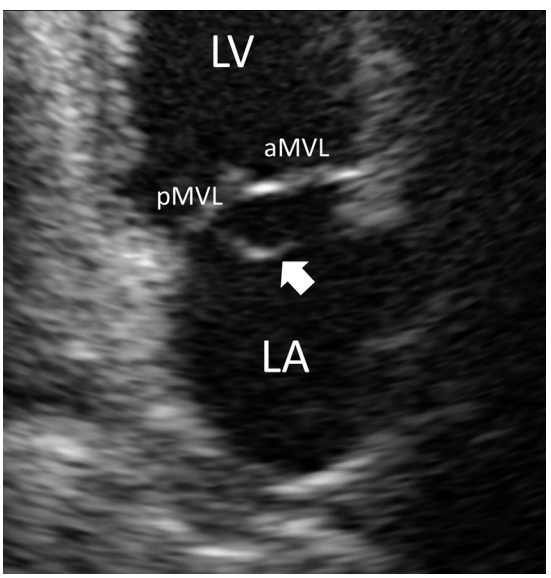

Figure 2 – *Transthoracic echocardiogram, apical two-chamber view. A flail posterior mitral valve leaflet is observed, with a thin and mobile structure in continuity with the leaflet (arrow) corresponding to a flail chordae tendineae. pMVL = posterior mitral valve leaflet. See Figure 1 legend for expansion of abbreviations.*

Globally, PM rupture occurs in 0.25% of patients after AMI,[4] usually coexisting with an inferior or inferoposterior AMI; this is explained by the shared coronary circulation of the aforementioned LV walls and the posteromedial PM and represents up to 7% of patients in cardiogenic shock after AMI. PM rupture is clinically evident between 2 and 7 days after AMI; however, the median time to PM rupture is approximately 13 hours.[4] Most commonly seen in transmural infarctions, PM rupture is increasingly observed in non-ST-elevation AMI, where both diagnosis and revascularization can be delayed.[5] Risk factors for a PM rupture are delayed hospitalization (> 24 hours) and first AMI (absent collaterals), as seen in the patient presented here, advanced age, and female sex. It is postulated that the preserved contractility exerts increased stress on an already compromised PM, eventually leading to rupture.[6] Mortality rate is high, with an in-hospital mortality of 80% to 90% with medical treatment, and an operative mortality of 26.9% with a 15-year survival of 39%.[4]

Clinically, the typical patient with PM rupture and severe AMR presents with cardiogenic shock and acute PE because of the massive regurgitant volume that leads to a low cardiac output and an extreme rising in LA pressure, respectively. In patients with acute chronic MR, a previously dilated LA can accommodate further regurgitant volume without elevating considerably their pressure; therefore, it could be best tolerated thanks to the implementation of compensatory short- and long-term mechanisms.[3]

Although chest radiograph can detect PE, using this method for this purpose is best avoided because of the excellent and superior diagnostic capabilities of lung US. Moreover, chest radiograph does not aid in defining neither the origin nor the mechanism of cardiogenic PE. In this way, bedside US can be considered as a first-line tool to diagnose a patient suffering from acute PE and/or shock of any origin, but especially to rule in or rule out a mechanical complication of AMI and a proximal aortic dissection. TTE diagnostic criteria of PM rupture include the presence of a flail leaflet, chord tendineae and PM head, and an MR jet, commonly eccentric and in opposite direction to the flail subvalvular apparatus.

The reported sensitivity of TTE for PM rupture is 65% to 85%.[5] Although TTE is diagnostic in most of the cases, a transesophagic echocardiogram is mandatory when TTE is of poor quality and to best delineate the valvular anatomy and planning the surgical procedure, enabling decision-making regarding the replacement or repair of the mitral valve.[7]

The patient was intubated and mechanically ventilated, with improvement in perfusion and oxygenation because of the beneficious effect of lowering LV afterload. Then, he was emergently transferred to another center for further care. A counterpulsation balloon pump was placed along with sodium nitroprusside to lower LV afterload and MR leakage and to improve coronary and systemic circulation. Transesophagic echocardiogram confirmed the partial rupture of the posteromedial PM. A coronary angiogram unexpectedly showed a codominant coronary circulation and an isolated occlusion of the obtuse marginal artery (a circumflex artery branch), which could not be revascularizated because of the evolutionary time of the coronary lesion. The rest of the coronary arteries were normal. After initial stabilization, he successfully underwent a mitral valve replacement (mechanical valve). The next day, after a profound and irreversible vasoplegic syndrome, he died.

This case demonstrates the great value of US for study patients with acute PE and shock in the context of AMI, aiding in delineating highly fatal complications such as PM rupture.

Reverberations

1. *Bedside TTE is a helpful method to diagnose AMI-related mechanical complications.*
2. *Acute severe MR caused by a ruptured PM must be thought of and carefully evaluated by TTE in a patient presenting with acute PE and/or shock and LV inferior/posterior ischemia on ECG and/or LV inferior/posterior hypokinesia.*
3. *Typical findings of a ruptured PM are a flail leaflet with attached free chordae tendineae and a thick structure corresponding to a PM head. Usually, MR is severe and LV systolic function is hyperdinamic and cardiac chambers are normal in size. The PM usually involved is the posteromedial one because of its one blood vessel circulation.*

Acknowledgments

Financial/nonfinancial disclosures: None declared.

Other contributions: *CHEST* worked with the authors to ensure that the Journal policies on patient consent to report information were met.

Additional information: To analyze this case with the videos, see the online article.

References

1. Erbel R, Alfonso F, Boileau C, et al. Diagnosis and management of aortic dissection. Recommendations of the Task Force on Aortic Dissection, European Society of Cardiology. *Eur Heart J*. 2001;22(18):1642-1681.
2. Blanco PA, Cianciulli TF. Pulmonary edema assessed by ultrasound: impact in cardiology and intensive care practice. *Echocardiography*. 2016;33(5):778-787.
3. Stout KK, Verrier ED. Acute valvular regurgitation. *Circulation*. 2009;119(25):3232-3241.
4. Kutty RS, Jones N, Moorjani N. Mechanical complications of acute myocardial infarction. *Cardiol Clin*. 2013;31(4):519-531.
5. Murad K, Missov E. Papillary muscle rupture following non-ST-elevation myocardial infarction: a case report. *Echocardiography*. 2016;33(6):923-925.
6. Fradley MG, Picard MH. Rupture of the posteromedial papillary muscle leading to partial flail of the anterior mitral leaflet. *Circulation*. 2011;123(9):1044-1045.
7. Lee SK, Heo W. A new surgical repair technique for ischemic total papillary muscle rupture. *Ann Thorac Surg*. 2015;100:1891-1893.