Resonancia magnética precoz en infarto transmural y pericarditis epistenocárdica con coronariografía normal

Early MRI on transmural infarction and epistenocardic pericarditis with normal coronariography

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ABSTRACT

A 53-year-old male presented in the emergency room with two hours of intense squeezing retrosternal pain, irradiating to both arms, hyperhidrosis and nausea while resting. On admission, myocardial necrosis markers were in the normal range, EKG demonstrated ST elevation on inferior leads and transthoracic echocardiography showed hypokinesis of mid- and apical segments of inferior wall and inferior septum. The patient was referred to coronary angiography in which it did not demonstrate significant lesions. Therefore, we considered possible revascularized acute infarction after treatment with unfractionated heparin versus coronary vasospasm.

Still, a few hours later, pain relapsed, first without but afterwards with pleuritic characteristics associated with PR depression and maintained ST elevation on EKG, and Troponin T peak value at 36 hours after initial event. He began colchicine and non-steroidal anti-inflammatory drugs for pain management after suspicion of perimyocarditis.

Cardiac MRI was performed demonstrating recent transmural infarction on distal right coronary artery/posterior descending artery territories including a central area of microvascular obstruction and signs of post-infarction pericarditis.

Hence, the patient probably developed a rapidly established transmural infarction that was revascularized by administration of heparin with the progressing post-infarction pericarditis.

This case shows the predicament of administration of reperfusion agents by allowing the rapid revascularization which is beneficial for the patient but depriving the definite diagnosis from the clinicians. It also demonstrates the importance of cardiac MRI to elucidate and confirm our suspicions.

Keywords: Myocardial infarction, pericarditis, coronary angiography, ventriculography.

RESUMEN

Un varón de 53 años acudió a urgencias con dos horas de intenso dolor retroesternal opresivo, irradiado a ambos brazos, hiperhidrosis y náuseas en reposo. En el momento del ingreso los marcadores de necrosis miocárdica estaban en rango normal, el electrocardiograma demostró elevación del ST en derivaciones inferiores y la ecocardiografía transtorácica mostró hipoquinesia de segmentos medios y apicales de pared inferior y septo inferior. El paciente fue remitido a coronariografía en la que no se demostraron lesiones significativas. Por tanto, se consideró posible infarto agudo revascularizado tras tratamiento con heparina no fraccionada frente a vasoespasmo coronario.

Aún así, pocas horas después, el dolor recidivó, desarrollando características pleuríticas asociadas a depresión del PR y elevación del ST mantenida en el EKG, y valor pico de Troponina T a las 36 horas del evento inicial. Inició colchicina y antiinflamatorios no esteroideos para el tratamiento del dolor tras sospechar perimiocarditis.

Se realizó una resonancia magnética cardiaca que demostró un infarto transmural reciente en los territorios distales de la arteria coronaria derecha/arteria descendente posterior, incluida una zona central de obstrucción microvascular y signos de pericarditis postinfarto.

Por lo tanto, el paciente probablemente desarrolló un infarto transmural de instauración rápida que se revascularizó mediante la administración de heparina con la pericarditis postinfarto progresiva.

Este caso muestra el predicamento de la administración de agentes de reperfusión al permitir la revascularización rápida que es beneficiosa para el paciente pero privando del diagnóstico definitivo a los clínicos.También demuestra la importancia de la RM cardiaca para dilucidar y confirmar nuestras sospechas.

Palabras clave: Infarto de miocardio, pericarditis, angiografía coronaria, ventriculografía.

CLINICAL CASE

A 53-year-old male presented in the emergency room with two hours of intense squeezing retrosternal pain, irradiating to both arms, hyperhidrosis and nausea while resting. As for vascular risk factors, he was an active smoker.

Initial EKG demonstrated ST elevation on inferior leads and immediately was given 250 mg of acetylsalicylic acid, 5.000 U of unfractionated heparin, morphine, and nitrates.

On admission, myocardial necrosis markers were in the normal range and transthoracic echocardiography showed hypokinesis of mid- and apical segments of inferior wall and inferior septum. Upon this presentation, patient was referred for urgent percutaneous coronary intervention.

However, coronary angiography did not demonstrate significant lesions. Ventriculography was performed and confirmed akinesis of

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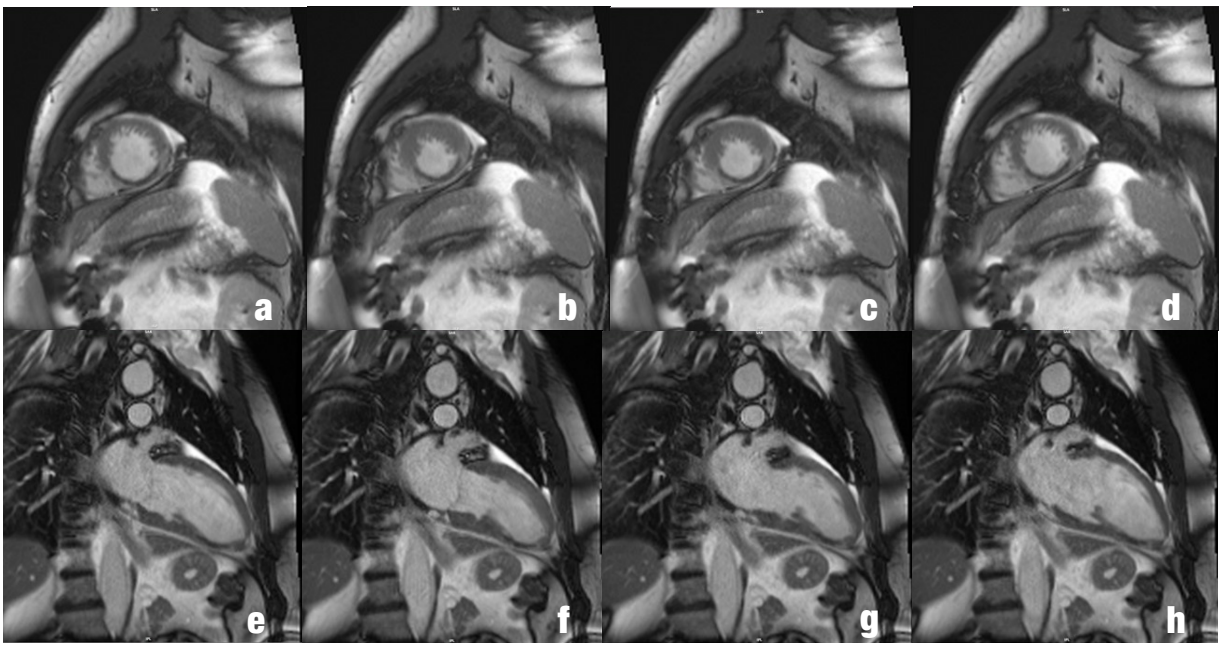

Figure 1. Sequence of short-axis (a-d) and two-chamber long-axis (e-h) cine images demonstrating inferoseptal akinesis and apical inferior wall dyskinesis. A central and subendocardial hyposignal region in the apical and septal segments of the inferior wall, indicating microvascular obstruction.

apical segment of inferior wall with preserved left ventricular systolic function. Aortography excluded aortic dissection. Therefore, we considered possible revascularized acute infarction after treatment with unfractionated heparin versus coronary vasospasm.

A few hours after coronary angiography, pain relapsed, at first without alteration during breathing cycle nor position and sustained ST elevation. We suspected a perimyocarditis instead of MI and administered anti-inflammatory drugs but without total resolution of the complaints.

Less than 24 hours after the initial event, worsened pain recurred with pleuritic characteristics associated with PR depression and maintained ST elevation on EKG, and continuous elevation of cardiac markers with peak values of Myoglobin of 539 ng/mL and Troponin T of 3270 ng/L, 36 hours after clinical onset. He began colchicine and non-steroidal anti-inflammatory drugs for pain management after suspicion of post-infarction pericarditis. Pain slowly reduced.

Cardiac MRI was performed 24hours after the initial event demonstrating recent transmural infarction on distal right coronary artery/posterior descending artery territories including a central area of MVO and signs of pericarditis (figures 1-3).

DISCUSSION

STEMI patients account for 25 to 35% of Acute Coronary Syndrome (ACS) and demand rapid reperfusion therapy[1,2]. It is usually caused by acute occlusion of a coronary artery or its main branch by thrombosis of preexisting coronary atherosclerosis and plaque rupture[3]. Very often they progress to transmural infarction[1]. Despite reinstatement of epicardial arteries, flow may not be adequate, specially within the microcirculation, visualized by cardiac MRI as microvascular obstruction (MVO)[3,4,5]. MVO causes persistent or recurrent chest pain with associated ST elevation even after revascularization.[1]

Normal coronary angiography does not exclude acute myocardial infarction as they may have angiographically invisible plaques in the culprit vessels that can be seen in coronary computed tomography and ischemic changes seen on cardiac MRI.[2,6,7]

The absence of a culprit vessel identification can be explained by recanalization, travelling embolus or vasospasm.[7]

Hence, in cases where angiography is not clarifying, cardiac MRI allows characterization, detection of complications and risk stratification for future major cardiac events.[1,5,8]

According to these findings, we believe that the administration of heparin dissolved the occlusion within the culprit vessel, although the lesion had already progressed and evolved to an established transmural infarction with central core of MVO. Along with this rapidly progressed event, it was associated with an epistenocardic pericarditis complication.

This case shows that prompt administration of anticoagulation before cardiac angiography allows rapid revascularization which is beneficial for the patient but denies the definite diagnosis for the clinicians. It also demonstrates the importance of early cardiac MRI to elucidate and confirm our suspicions.

Hence, in cases where angiography is not clarifying, cardiac MRI allows establishment of final diagnosis, helps detection of complications and further risk stratification for future major cardiac events.[1,5,8]

Thus, this case shows the importance of performing cardiac MRI in an early phase and, consequently the correct treatment orientation.

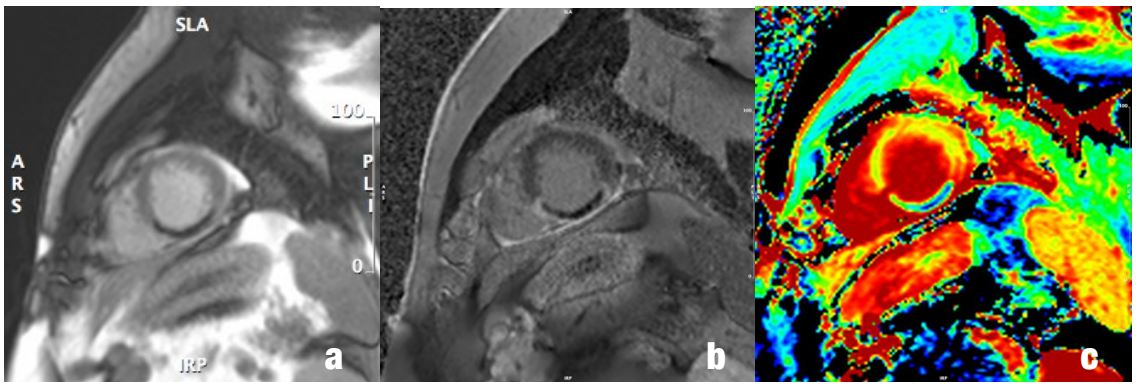

Figure 2. Early (a) and late (b) gadolinium enhancement in short-axis images showing acute transmural infarction in distal inferior and inferoseptal segments with a central and subendocardial core region of low signal intensity, indicating microvascular obstruction. Also, there is evidence of pericardial enhancement adjacent to the infarcted segments, suggestive of pericarditis. T1 mapping images after contrast (c) showing an area of absent contrast uptake in blue (arrow) corroborating the presence of microvascular obstruction.

Many studies have shown the benefit of early cardiac MRI, within the first week after the cardiac event[1,5,8]. A few parameters detected in cardiac MRI, such as MVO, are associated with worst prognosis and require strict follow-up and rigorous risk factor control.

Therefore, besides individual experience in recognizing certain pathophysiologic events, the right early imaging support orientate us to the right path and allows us to prevent further complications.

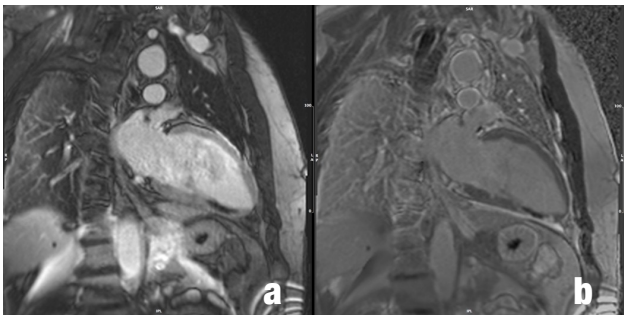

Figure 3. Early (a) and late (b) gadolinium enhancement in two-chamber long-axis images showing acute transmural infarction in the apical inferior segment with an area of microvascular obstruction. On late gadolinium enhancement (b), the hypersignal of pericardial inferior and lateral walls of the left ventricle are compatible for pericarditis.

CONFLICT OF INTEREST
The authors declare that they have no conflict of interests.

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ETHICAL ASPECTS
All participants submitted a consent form to be included in this study.

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