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An unusual cause of myocardial infarction

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Background: In order to direct the treatment it is well established that is fundamental to clarify the aetiology of heart failure and the cause of myocardial infarction (MI) with non obstructive coronary artery disease (MINOCA), with CMR being one of the methods of choice in both clinical situations.

Case report: A 70 years-old male patient was admitted in our emergency department with complaints of irregular palpitations, progressive dyspnoea and fatigue on exertion, with two weeks of evolution; these symptoms were associated to retrosternal chest pain in the last twelve hours. He had a previous medical history of dyslipidemia, no other cardiovascular risk factors were known. On admission, his heart rate was 130bpm, with an irregularly irregular pulse and the pulmonary auscultation revealed bibasal crackles. The remaining physical examination was unremarkable. The ECG showed an atrial fibrillation rhythm, with a mild ST elevation and T wave inversion in inferior leads. The echocardiogram revealed a diffuse hypokinesia of left ventricle with an ejection fraction of 35-40%. The lab tests documented an elevation of troponin (hs-TnT 210ng/L) and NTproBNP (1945pg/ml). The coronary angiogram showed no lesions.

It was admitted acute heart failure with reduced ejection fraction. To further clarify the aetiological diagnosis a CMR was performed revealing diffuse hypokinesia, which was more severe at the apical segments of the inferior and septal walls, where subendocardial LGE was observed. The diagnosis of myocardial infarction (MI) of embolic aetiology in association to probable tachycardiomyopathy due to uncontrolled atrial fibrillation was assumed and the patient was medicated accordingly. During follow-up, there was a reversal of the LV function, only remaining the wall motion abnormalities due to the MI.

Discussion/Conclusion: This patient was referred for an acute coronary syndrome and acute heart failure of unknown aetiology. After the exclusion of coronary atherosclerotic disease it is important to investigate other less common causes.

In our case CMR revealed wall motions abnormalities and a pattern of LGE compatible with ischemic necrosis probably due to an embolic MI in a patient with "de novo" atrial fibrillation and not anticoagulated. In this unusual case, a tachycardiomyopathy was associated due to the rapid ventricular response. CMR was essential to establish the final diagnosis.

Abstract P568 Figure. CMR - Pattern of LGE

