CASE PRESENTATION

On the Precipice of Disaster – Fulminant Acute Myocarditis Mimicking ST-Elevation Myocardial Infarction in a Young Patient

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ABSTRACT

Although acute myocarditis and coronary vasospasm are common differential diagnoses in the case of young patients with persistent ST elevation, the association of coronary vasospasm and acute fulminant myocarditis is a rare situation[1,2,3]. We present the case of a 21 year-old male who presented with chest pain, ECG changes and biomarker levels initially interpreted as ST elevation myocardial infarction (STEMI), in which severe coronary vasospasm was identified. Shortly after, he developed cardiogenic shock and fulminant acute myocarditis was suspected.

Keywords: acute myocarditis, coronary vasospasm, STEMI, cardiogenic shock.

REZUMAT

Deși miocardita acută și vasospasmul coronarian sunt diagnostice diferențiale comune în algoritmul pacienților tineri care se prezintă cu supradenivelare persistentă de segment ST, asocierea vasospasmului coronarian cu miocardita acută fulminantă este o situație mai rară[1,2,3]. Prezentăm cazul unui tânăr de 21 de ani care se prezintă cu durere toracică anterioară, modificări ECG și dinamică enzimatică, ce au fost interpretate inițial ca STEMI, la care coronarografia a evidențiat vasospasm sever. La scurt timp după prezentare, pacientul dezvoltă șoc cardiogen, ridicându-se astfel suspiciunea unei miocardite acute fulminante.

Cuvinte cheie: miocardita acută, vasospasm coronarian, STEMI, șoc cardiogen.

CASE PRESENTATION

We present the case of a 21 years-old male patient who presented to the emergency department of his local hospital with severe ongoing chest pain, which began at rest two hours prior to admission. His detailed medical history was fairly unremarkable: except for being a regular smoker the patient did not have any personal or family history of heart disease, he denied recreational drug use, any recent history of respiratory or gastrointestinal symptoms. His clinical exam at presentation was normal. The ECG strip recorded in the ER revealed sinus tachycardia, PR segment depression, important concave ST elevation in the inferior and lateral leads with reciprocal ST depression in aVR and V1 (Figure 1). Blood analyses revealed high troponin levels (Hs cTnI- 11831ng/l), leukocytosis, neutrophilia and inflammatory syndrome. Echocardiography showed LVEF 45%, inferior and lateral wall hypokinesis, no significant valvulopathies, no pericardial effusion.

At this moment he was interpreted as Killip I infero-lateral ST elevation myocardial infarction (STEMI). As timely PCI in the recommended 2 hours timeframe was not available at the local hospital, he was administered fibrinolytic therapy and thereafter transferred to our institution for further care and evaluation. Patient underwent urgent coronary angiography revealing important vasospasm of ostial and proximal segment of right coronary artery which subsided after intracoronary Nitroglycerin bolus. No atherosclerotic

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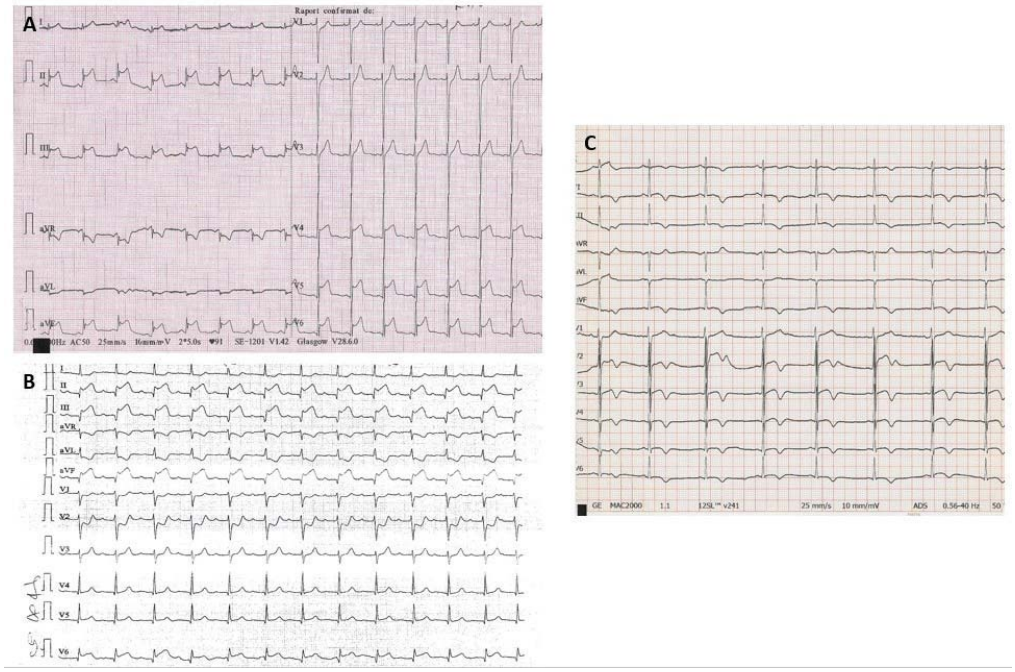

Figure 1. ECG: strip A – at admission to local hospital; strip B – at admission to our hospital; strip C – day 6 after admission to our hospital.

burden was identified by angiography (Figure 2). Repeated blood analyses showed higher troponin levels (Hs cTnI- 36779ng/l), increased heart failure markers (NTproBNP-2521pg/dl) and confirmed elevated leucocyte count with neutrophilia, elevated ESR, as well as high C-reactive protein and fibrinogen, while procalcitonin, influenza test and urinary toxicology screen were negative. Shortly after coronary angiography the patient's clinical condition rapidly deteriorated with the development of overt cardiogenic shock. Bedside echocardiography revealed mild left ventricular (LV) dilatation, global severe hypokinesia with LV ejection fraction of 10%, and severe mitral regurgitation, without pericardial effusion. He was admitted to the

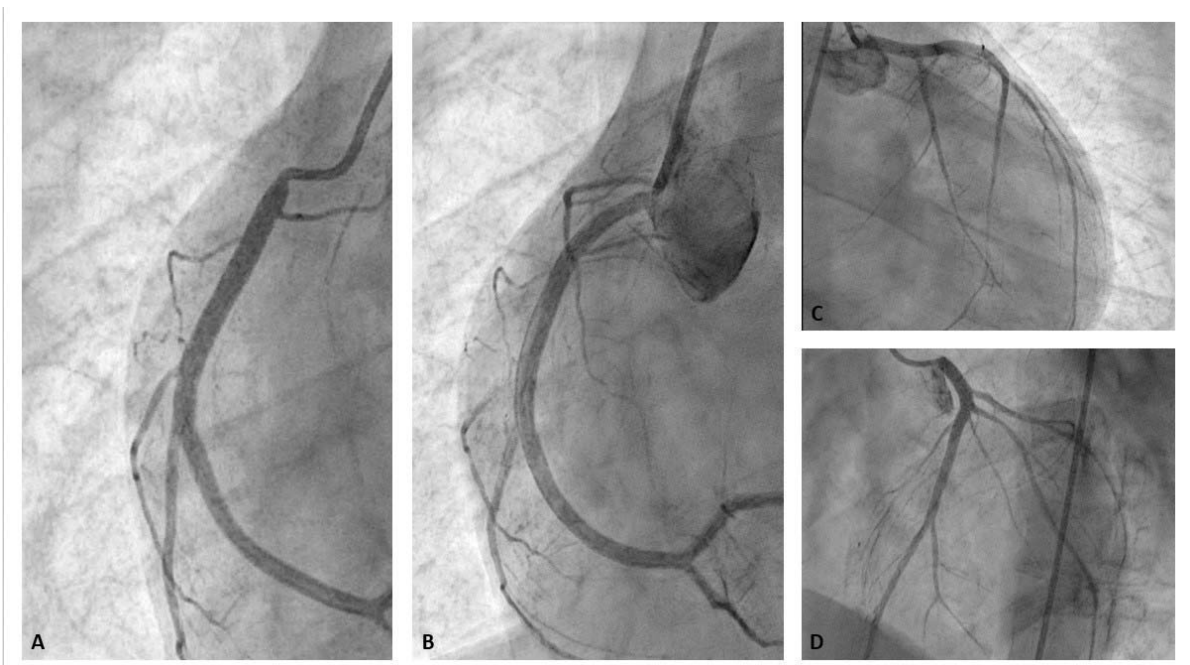

Figure 2. Coronary angiography showing (A) important spasm of right coronary artery which subsided after intracoronary Nitroglycerin administration (B); normal left coronary system (C and D).

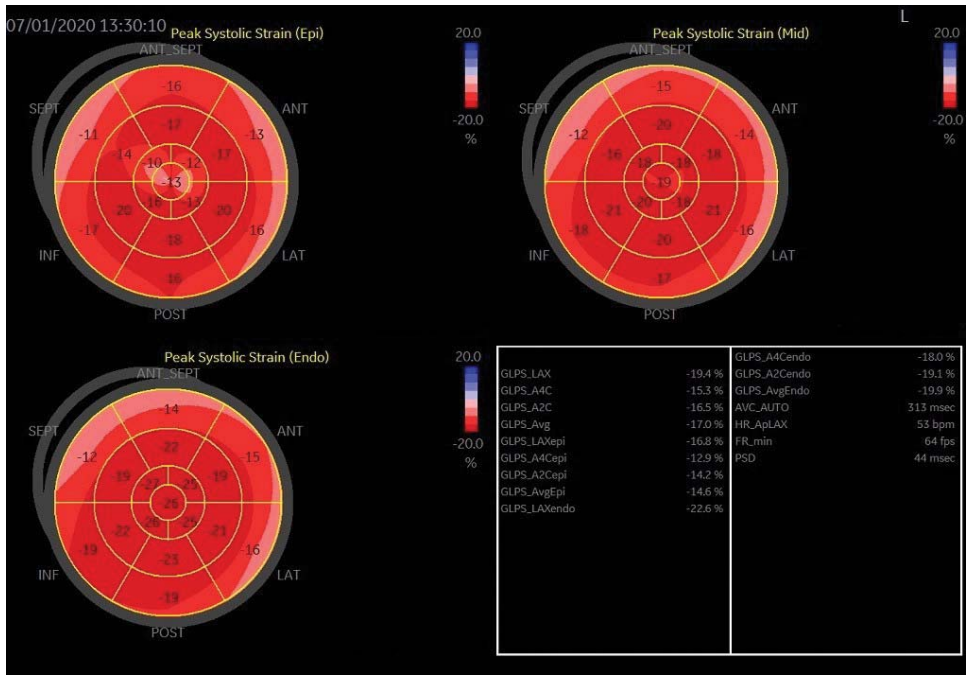

Figure 3. Speckle tracking echocardiography performed 8 days after admission showing decreased global longitudinal strain, especially in the epicardial layer, with a patchy and diffused distribution of reduced strains across all segments.

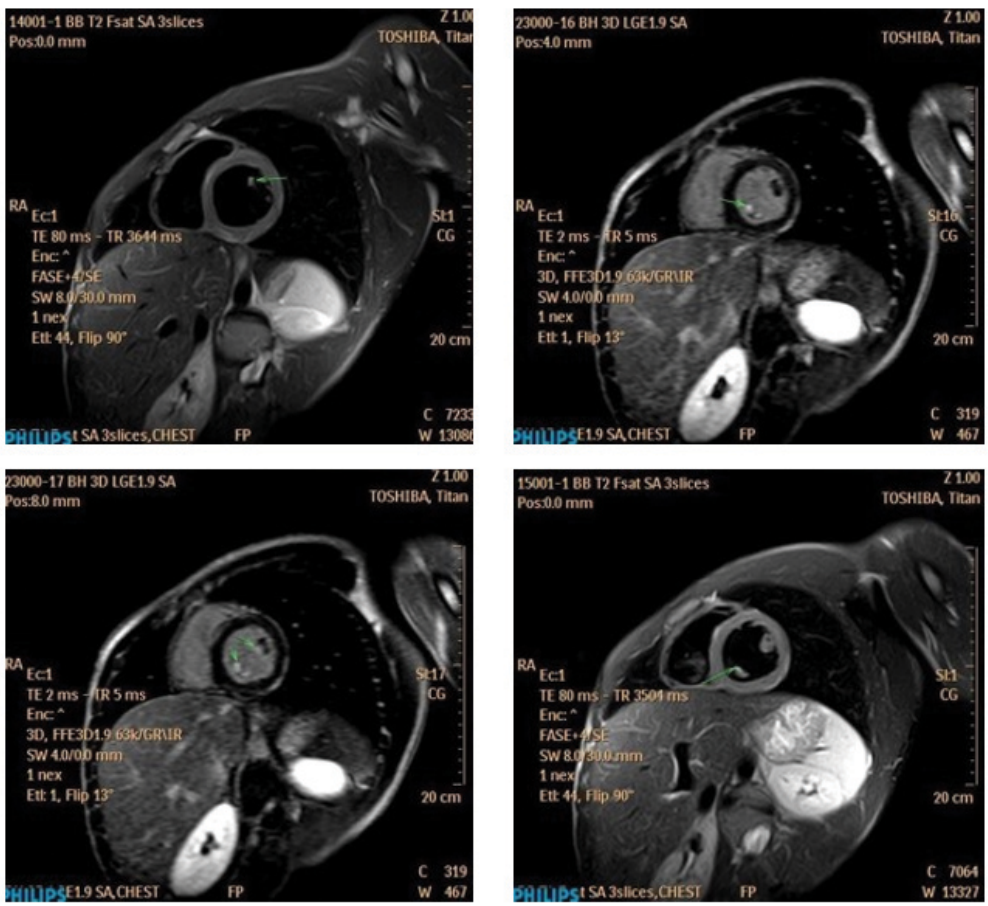

Figure 4. CMR scan done at 14 days. LGE and T2-weighted sequences showing edema at the distal portion of the left ventricular papillary muscles (arrow).

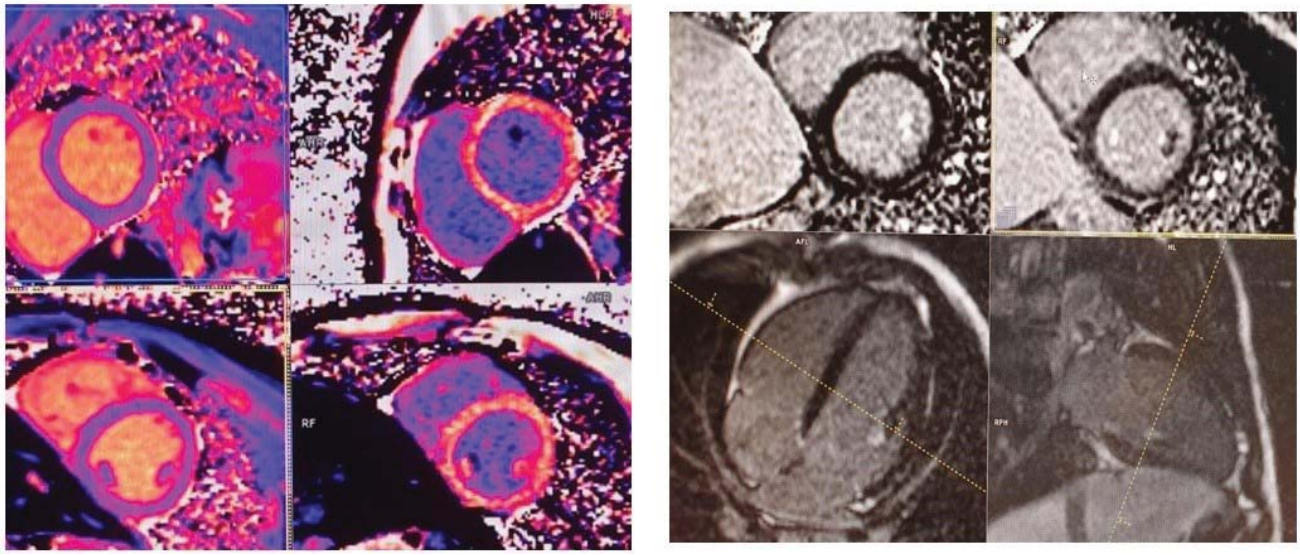

Figure 5. CMR scan repeated at 30 days. T1 pre- and post- contrast mapping and LGE acquisitions: fibrosis in the distal portion of papillary muscles (arrow).

Intensive Care Unit for circulatory support receiving inotropic and vasopressor therapy. The patient's status progressively improved allowing for weaning of supportive therapy 4 days after admission in the ICU. Conventional echocardiography performed after 6 days from admission showed significant improvement of LV function (40% in the sixth day) and mild mitral regurgitation. Speckle tracking echocardiography performed after 8 days from admission showed decreased global longitudinal strain (GLS), especially in the epicardial layer, with a patchy and diffused distribution of reduced strains across all segments (Figure 3). Taking into account the clinical presentation with rapid onset of cardiogenic shock in the absence of recurrent chest pain and ECG changes (Figure 1), serum biomarkers, coronary angiography and echocardiography findings, as well as significant improvement over a short period of time, we considered the diagnosis of acute fulminant myocarditis (FM), complicated with coronary vasospasm and cardiogenic shock. Cardiac magnetic resonance (CMR) was done 14 days after symptoms onset (Figure 4) and showed normal left ventricular volumes with mildly reduced LV ejection fraction (55.95%). T2-weighted sequences and Late Gadolinium Enhancement (LGE) imaging described features suggestive of myocardial oedema at the distal portion of the left ventricular papillary muscles. Thus, Lake Louis updated criteria[4] for acute myocarditis were not met. The patient's subsequent course was favourable and he was discharged 16 days after admission, on beta-blocker, ACE inhibitor and nitrate. Given the peculiar aspect revealed by CMR a repeat study was done 30 days after the initial presentation (Figure 5). The assessment did not demonstrate deterioration of left ventricular systolic function, or cardiac chamber enlargement. Morphologically, no areas of inflammatory and fibrotic left ventricular myocardial insults were recorded. However, T1 pre and post contrast mapping and LGE acquisitions depicted evidence of fibrosis in the cranial segment of left ventricular papillary muscles.

DISCUSSIONS

Cardiovascular disease in young patients can evolve with dramatic presentations in the acute setting. Early diagnosis and timely therapies are the cornerstones that give the patient a maximized chance of survival and recovery. Sometimes the clinical presentation and paraclinical investigations can give rise to sensitive problems regarding the possible differential diagnoses and implicitly the therapeutic approach. Particularly in this case, the initial patient presentation was considered acute Killip I infero-lateral STEMI. Following investigations depicted FM, complicated by coronary vasospasm and cardiogenic shock. Peculiar to this case is the presence of vasospastic angina, rarely described as a mechanism of chest pain in acute myocarditis[1,2,3]. Possible pathogenic mechanisms of coronary spasm associated with FM are: acute viral myocarditis resulted in an early virus-platelet interaction leading to an intracoronary aggregation of platelets, release of vasoactive substances, and consequent coronary artery va-

sospasm[5]; altered neurohumoral tone seen in patients with acute heart failure[6]; immunologically mediated coronary vasculitis[7].

In acute myocarditis is more likely to detect diagnostically important evidence of myocardial damage if the scan is performed within 2 weeks of presentation and the addition of tissue mapping, which was not available at first CMR evaluation, yield significant improvement in diagnostic accuracy over traditional Lake Louise criteria.[8] Even if CMR offers unique insights into tissue-level pathologies, unfortunately, in the case of our patient, was possible to be performed only after 14 days from symptoms' onset and did not fulfil the revised Lake Louise criteria.

Papillary muscle fibrosis without rupture is common, as they are the last portion of the heart to be perfused, and even if coronary ischaemia is the most common cause, this condition can also be caused by cardiogenic shock, induced by FM. The aspect seen on CMR, on LGE acquisitions is focal enhancement, confined to the apical portions of the papillary muscles[9]. Consequently, we consider that the papillary muscle fibrosis is the hallmark of cardiogenic shock.

Endomyocardial biopsy is the gold standard diagnostic test, but taking into consideration the fibrinolytic therapy and subsequent haemorrhagic risk, in addition to rapid and spectacular improvement of hemodynamic status, we did not consider opportune to perform it.

In conclusion, although current guidelines and consensus papers are comprehensive in leading the physician to a prompt diagnosis and appropriate treatment for the vast majority of patients, in particular cases a more individualised approach is needed. Moreover, despite the advancement in diagnostic procedures, and especially the progress of imaging modalities, a sound clinical judgement remains fundamental for a correct approach of patients.

Compliance with ethics requirements:

The authors declare no conflict of interest regarding this article. The authors declare that all the procedures and experiments of this study respect the ethical standards in the Helsinki Declaration of 1975, as revised in 2008(5), as well as the national law. Informed consent was obtained from all the patients included in the study.

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