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CASE REPORT / ПРИКАЗ БОЛЕСНИКА

Focal myocarditis, an unusual imitator – case report and short review

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SUMMARY

Introduction Acute myocarditis is a serious inflammatory condition of the myocardium. Clinically, symptoms may differ from case to case, and as such can pose a significant diagnostic dilemma. Here we present a case of acute focal myocarditis with markedly elevated troponins, in which diagnosis was finally made using cardiac magnetic resonance (CMR).

Case outline A male patient, 26-year-old, without cardiovascular risk factors presented with severe chest pain, diaphoresis, pallor, and dyspnea. Blood pressure was 160/110 mmHg, and electrocardiogram (ECG) showed ST-segment elevation in inferior leads. In laboratory there was an extreme elevation of Troponin. Inferior-posterior-lateral STEMI was suspected, and initial treatment was given according to that suspicion. The patient was then sent to catheterization laboratory for further evaluation, which showed absence of coronary artery disease. A working diagnosis of myocardial infarction with non-obstructive coronary arteries (MINOCA) was established. To distinguish MINOCA from other causes of myocardial injury with elevated troponins, a CMR was done, and its finding was consistent with focal myocarditis of inferolateral localization. Further treatment consisted of beta blockers, angiotensin-converting-enzyme inhibitors and avoidance of strenuous activity for the next six months. The patient fully recovered and had no further complications with ECG only showing flat T-wave in D3 lead.

Conclusion Focal myocarditis is an unusual manifestation of myocardial disease and can confuse physicians, especially if it occurs along with elevated cardiac markers and ST-elevation, but in a young patient, without any known comorbidity, this diagnosis must be considered. Here, a CMR may be a useful tool.

Keywords: myocardial infarction with non-obstructive coronary arteries; cardiac magnetic resonance; troponin

INTRODUCTION

Acute myocarditis is a serious inflammatory condition of the myocardium, the middle layer of the heart that contains cardiac muscle cells. It affects people of all ages and has a broad etiology (Table 1). Clinically, symptoms may differ from case to case, and as such can pose a significant diagnostic dilemma [1, 2]. Dramatic and acute electrocardiogram (ECG) ST-elevation can be wrongly interpreted as acute coronary syndrome, thus misleading physicians. Invasive coronary angiography is necessary for evaluation of myocardial injury. Absence of coronary artery stenosis (50% or greater) leads to the working diagnosis of myocardial infarction with non-obstructive coronary arteries (MINOCA). There are different algorithms in evaluating this diagnosis of myocardial injury. For Figure 1 illustration, we used modified algorithm mentioned by Occhipinti et al. [3]. In this case, careful history, clinical exam, and eventual cardiac magnetic resonance (CMR), could provide a solution for the dilemma [4]. Here we present a case of acute focal myocarditis with markedly elevated troponins, in which diagnosis was finally made using CMR.

CASE REPORT

A male patient, 26-year-old, without cardiovascular risk factors presented with severe chest pain, diaphoresis, pallor, and dyspnea. He had no fever. Blood pressure was 160/110 mmHg. ECG is shown in Figure 2. In laboratory findings, there was an extreme elevation of Troponin (I day – 6.374 ng/ml, II day – 16.947 ng/ml, III day – 10.302 ng/ml, V day – 0.207 ng/ml). Other laboratory parameters were normal. As there was an increase in troponin levels along with inferior leads ischemic ST elevation, initial diagnosis of acute coronary syndrome-Inferior-posterior-lateral STEMI was suspected. Initial treatment was given according to that suspicion and consisted of acetylsalicylic acid, clopidogrel, pantoprazole, tramadol, ramipril, bisoprolol, diazepam. Echocardiography showed normal ejection fraction (52%), heart size and ventricle wall thickness, with hypokinesia of medial basal segment of inferior wall of left ventricle. The patient was then sent to catheterization laboratory for further evaluation, which showed absence of coronary artery disease (Figure 3). A working diagnosis of MINOCA was established. Viral serology was negative for acute infection with cytomegalovirus, Epstein–Barr virus, parvovirus B19, adenovirus and coxsackievirus, with only

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Table 1. Causes of myocarditis-illustrative

Viruses	Bacteria and rickettsia	Fungi and protozoa	Parasites	Immune and systemic	Drug reactions and toxins
Coxsackie B Echovirus Epstein–Barr virus Cytomegalovirus Adenovirus Influenza virus Mumps Measles Rubella HIV Hepatitis B and C Varicella-zoster virus Respiratory-Syncytial virus Sars-CoV-2 Parvovirus B19 Herpesvirus Arbovirus Polio virus	*Corynebacterium Diphtheriae* *Staphylococcus* *Streptococcus* *Tuberculosis* *Clostridium tetani* *Mycoplasma pneumoniae* *Brucella* *Neisseria gonorrhoeae* *Hemophilus influenzae A* *Actinomyces* *Salmonella* *Rickettsia* *Borrelia burgdorferi* *Leptospira* *Tropheryma whipplei* *Francisella tularensis* *Vibrio cholerae*	*Aspergillus* *Candida* *Cryptococcus* *Coccidioides* *Histoplasma* *Trypanosoma cruzi* *Malaria spp.* *Leishmania spp.* *Toxoplasma gondii*	*Ascaris lumbricoides* *Trichinella spiralis* *Echinococcus granulosus* *Taenia solium* *Paragonimus westermani* *Schistosoma* *Visceral larva migrans* *Toxocara canis*	Kawasaki disease Scleroderma Multisystem inflammatory syndrome in children and adults Systemic lupus erythematosus Vasculitis Toxic shock syndrome Hypereosinophilia Wegener granulomatosis Sarcoidosis Inflammatory bowel disease Phaeochromocytoma	Chemotherapy agents Antipsychotics Antibiotics [Penicillin Cephalosporins Tetracyclines Sulfonamides] Tricyclic antidepressants Lithium Diuretics Alcohol Arsenic Snake venom Tetanus toxoid Carbon monoxide Heavy metals [Iron, Copper] Insect bites Cocaine Methyldopa

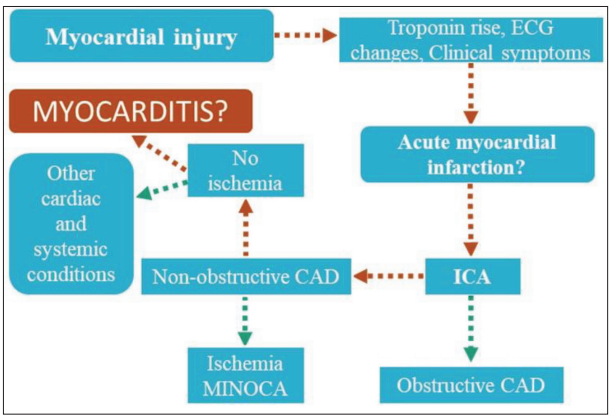

Figure 1. Example of focal myocarditis diagnostic pathway

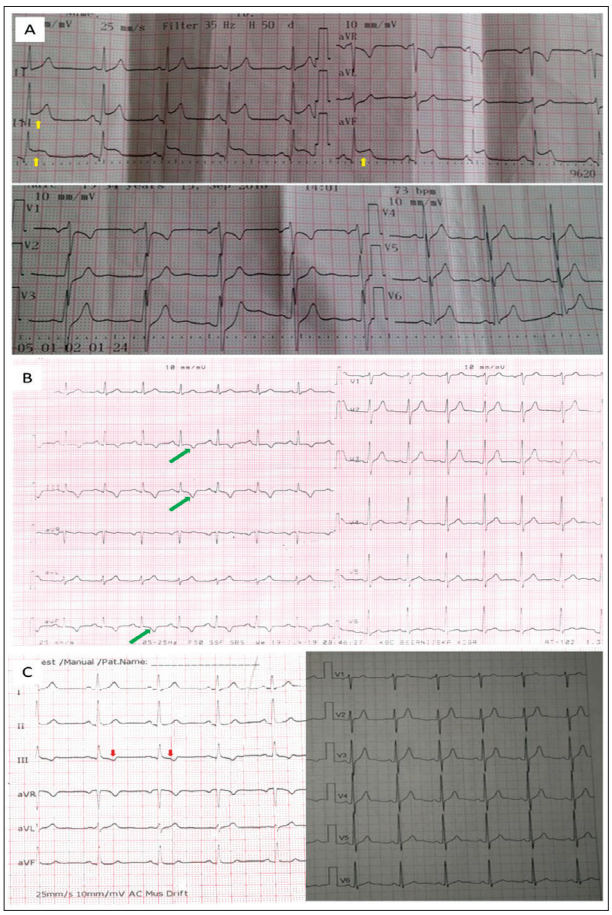

Figure 2. A – Echocardiogram (ECG) on admission: sinus rhythm, heart rate = 110/min, ST- elevation (yellow arrows) in D2, D3, aVF up to 4 mm; B – ECG one month later: negative T-wave (green arrows) in D2, D3, aVF up to 4 mm; C – last ECG, year after the event: sinus rhythm, heart rate = 75/min, negative T-wave in D3 (red arrows)

IgG antibodies present for cytomegalovirus, Epstein–Barr virus, and parvovirus B19. During next days of hospital stay, patient complained of chest discomfort, but there were no further ECG changes, and troponin levels normalized. To distinguish MINOCA from other causes of myocardial injury with elevated troponins, a CMR was done (example of CMR myocarditis is shown in Figure 3). CMR finding showed inferolateral subepicardial edema on T2 and STIR sequences with late gadolinium enhancement (LGE) in the same region. The finding was consistent with focal myocarditis of inferolateral localization. Further treatment consisted of beta blockers, angiotensin-converting-enzyme inhibitors and avoidance of strenuous activity for the next six months. The patient fully recovered and had no further complications with ECG only showing flat T-wave in D3 lead. This case report was approved by the institutional ethics committee, and written consent was obtained from the patient for the publication of this case report and any accompanying images.

DISCUSSION

Myocarditis is an inflammatory disease of myocardium that is likely underdiagnosed. The prevalence of myocarditis has been reported from 10.2 to 105.6 per 100,000 worldwide and is more common in men with about 1:2–4 female–male ratio, with women reported to have better survival of dilated cardiomyopathy [5]. It is a significant public health issue, especially for young adults.

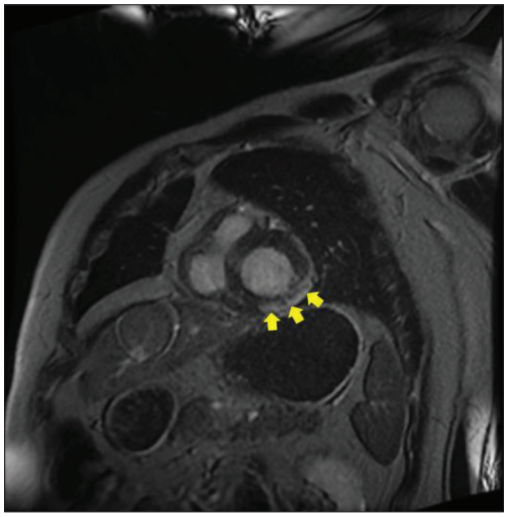

Figure 3. Short axis late gadolinium enhancement pathologic intramyocardial and pericardial late gadolinium enhancement (10–20 min) in lateral and inferior wall of the left ventricle; case courtesy of Dr. Igor Yamola (radiopaedia.org, rID: 98955)

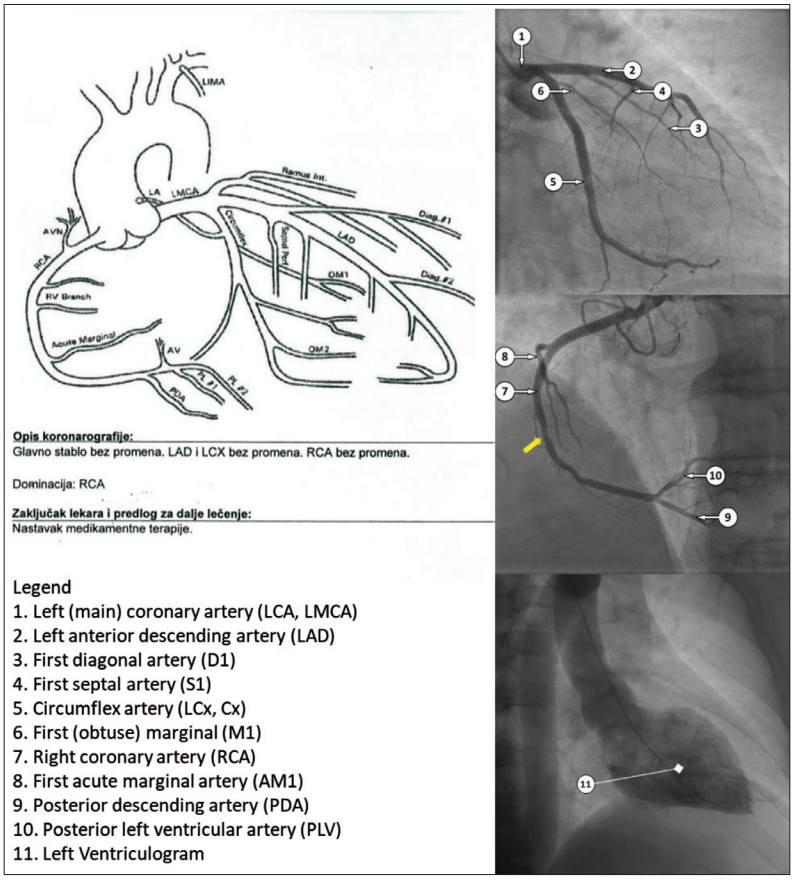

Figure 4. Coronary angiography report of our patient along with near normal coronary angiography (only minor mid right coronary artery stenosis-yellow arrow, in a 45-year-old male); case courtesy of Dr. Craig Hacking (radiopaedia.org, rID: 63081)

Myocarditis has a generally good prognosis, but in up to 20% of cases, it may progress to a dilated cardiomyopathy [6, 7]. Acute myocarditis can be defined as a period of < 1 month between symptom onset and diagnosis. Most common symptoms noted are chest pain (in 85–95% of cases), fever (in about 65%) and dyspnea (19–49% of cases) [8]. Another entity to be aware of in differential diagnosis besides acute coronary syndrome is left dominant arrhythmogenic cardiomyopathy, which is underrecognized, but can also present with chest pain and elevated cardiac enzymes [9]. Here we presented a case of a young patient with acute focal myocarditis and extremely elevated troponins, that was initially considered acute myocardial infarction, then evaluated as MINOCA, and finally diagnosed using a CMR. The absence of fever in our patient was an aggravating feature for making the diagnosis of focal myocarditis.

Focal myocarditis has already been described as a myocardial infarction simulator. In cases, were endomyocardial biopsy was ordered, etiology was mostly viral (ADV, cytomegalovirus, Epstein–Barr virus, PVB19) [10, 11]. Young patients that present as STEMI must be evaluated in direction of focal myocarditis, especially if there is a positive history of recent viral illness.

Endomyocardial biopsy is the gold standard for diagnosis of myocarditis. After using a transfemoral, or radial approach, a specimen is histologically and immunologically evaluated for presence of infectious agents, and tissue architectonic is considered for other (e.g., autoimmune) forms of inflammation [12]. In our case, due to focal nature of inflammation, endomyocardial biopsy could not be done. Patchy and focal inflammation of myocardium can sometimes work against this diagnostic approach.

ECG with ST-segment elevation may occur in pericarditis, myocarditis, myocardial infarction (STEMI), LV aneurism, and in some rare or uncommon conditions (e.g., Brugada syndrome, Takotsubo cardiomyopathy), and benign conditions (e.g., benign early repolarization) [13].

Elevated cardiac markers must be inclusively considered along with clinical and other diagnostic findings, as they do not define type of myocardial injury. The treatment should therefore be aimed at the cause of myocardial injury. Troponins may be an indicator of a disease severity and may rise in myocarditis, but they do not carry an adverse prognosis as in acute coronary syndrome [14, 15].

CMR may be crucial in diagnosis of acute focal myocarditis and is best to be done early after symptoms onset and can also be used to track disease progression/resolution. Time window for optimal sensitivity for diagnostic imaging is few weeks from its presentation. In our case, CMR has been proven diagnostically two weeks after disease onset. As edema is a universal marker of inflammation that also occurs in focal myocarditis, a T2-weighted imaging and high STIR signal may be indicative of myocardial edema [16, 17]. Lake Louise criteria for CMR in myocardial inflammation were established in 2009. For diagnosis of acute myocarditis, two out of three of the following features are needed: edema (with T2-weighted sequences), hyperemia (with early Gadolinium Enhancement) and necrosis or fibrosis (with LGE) [18]. Updated Lake Louise criteria were established in 2018 and include main criteria ("2 out of 2"): T2 based imaging – Regional high (10 pixels) T2 signal intensity or

global T2 signal intensity ratio ≥ 2.0 in T2-weighted images or regional or global increase of myocardial T2 relaxation times; T1 based imaging – Regional or global increase of native myocardial T1 relaxation times or extracellular volume (highly sensitive to detecting both acute and chronic forms of increased free water content within the myocardium). Supportive criteria are pericardial inflammation and left ventricular disfunction. All of mentioned criteria may as well be an indicator of focal myocarditis [19, 20].

Focal myocarditis is an unusual manifestation of myocardial disease and can confuse physicians, especially if it occurs along with elevated cardiac markers and ST-elevation, but in a young patient, without any known comorbidity, this diagnosis must be considered. Here, a CMR may be a useful tool.

Conflict of interest: None declared

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Фокални миокардитис, необични имитатор – приказ болесника и кратак преглед литературе

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САЖЕТАК

Увод Акутни миокардитис је озбиљно инфламаторно стање које погађа миокард. Клинички, симптоми се разликују од случаја до случаја, представљајући дијагностичку дилему. Приказаћемо случај акутног фокалног миокардитиса са изразито високим тропонинима, код којег се до дијагнозе дошло употребом магнетне резонанце срца.

Приказ болесника Мушкарац старости 26 година, кардиваскуларних фактора ризика, јавља се због изразитог бола у грудима, дијафорезе, бледила и диспнеје. Притисак на пријему био је 160/110 *mmHg*, а ЕКГ је показивао елевацију *ST*-сегмента у доњим одводима. Лабораторијски је евидентирана висока вредност тропонина и постављена је сумња на инферо-постеро-латерални *STEMI*. Иницирана је терапија под том сумњом. Пацијент је потом послат на коронарографију, која је показала одсуство коронарне болести срца. Постављена је радна дијагноза инфаркта миокарда без опструкције коронарних артерија. Како би се утврдило да ли је заиста у питању ова дијагноза, затражена је магнетна резонанца срца, чији је налаз био конзистентан са фокалним миокардитисом инферолатералне локализације. Даља терапија садржала је бета-блокаторе, *ACE* инхибиторе и избегавање захтевне физичке активности наредних шест месеци. Пацијент се потпуно опоравио и није имао даље компликације, осим што је на ЕКГ-у перзистирао налаз аплатираних Т-таласа у Д3 одводу.

Закључак Фокални миокардитис је необична манифестација болести миокарда и може збунити лекаре, нарочито ако се дешава у склопу повишених срчаних ензима и *ST*-елевације на ЕКГ-у, али код младих особа, без познатих коморбидитета, ова дијагноза се мора узети у обзир. У овом случају, кардиоваскуларна магнетна резонанца може бити користан алат.

Кључне речи: инфаркт миокарда без опструкције коронарних артерија; магнетна резонанца срца; тропонин