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Case of Myocardial Infarction with Nonobstructive Coronary Arteries Caused by Thebesian Veins Thrombosis

Caso de Infarto de Miocardio con Arterias Coronarias No Obstructivas por Trombosis de las Venas de Tebesio

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SUMMARY

Introduction: *The causes of myocardial infarction with nonobstructive coronary arteries are heterogeneous and include different reasons. Clinical cases of myocardial infarction without obstruction represent a difficult diagnostic task where cardiac magnetic resonance is increasingly used.* **Objective:** *This research aimed to demonstrate the evaluation protocol of myocardial infarction with nonobstructive arteries.* **Method:** *The object of this study was a 49-year-old male patient who presented at the emergency department with atypical chest pain, notably elevated troponin levels. To establish the diagnosis were performed electrocardiogram, echocardiography, stress-echocardiography, coronary angiography, and cardiac magnetic resonance.* **Results:** *Echocardiography didn't demonstrate significant abnormalities of the heart's wall movement but due to elevated troponin level and were performed coronary angiography and the myocardial infarction with nonobstructive coronary arteries were established. Further cardiac magnetic resonance confirmed the diagnosis and established that the cause of myocardial infarction was Thebesian vein thrombosis. A cardiac magnetic resonance imaging (MRI) was performed, which showed myocardial edema in the inferior wall on T2-weighted visualization. Late gadolinium enhancement showed focal areas of subendocardial infarction. The patient received appropriate medical treatment and experienced good clinical outcomes. This case study demonstrates how the role of cardiac MRI in the diagnosis and management of myocardial infarction with nonobstructive coronary arteries (MINOCA) patients has become increasingly crucial, as the understanding of troponin elevation and its various mechanisms continues to evolve.*

Keywords: *Cardiovascular magnetic resonance; coronary angiography; MINOCA; stress-echocardiography.*

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RESUMEN

Introducción: *Las causas del infarto de miocardio con arterias coronarias no obstructivas son heterogéneas e incluyen diferentes motivos. Los casos clínicos de infarto de miocardio sin obstrucción representan una tarea diagnóstica difícil donde se utiliza cada vez más la resonancia magnética cardíaca.* **Objetivo:** *Esta investigación tuvo como objetivo demostrar el protocolo de evaluación del infarto de miocardio con arterias no obstructivas.* **Método:** *El objeto de este estudio fue un paciente varón de 49 años que acudió al servicio de urgencias por dolor torácico atípico, destacando niveles elevados de troponina. Para establecer el diagnóstico se realizaron electrocardiograma, ecocardiografía, ecocardiografía de estrés, angiografía coronaria y resonancia magnética cardíaca.* **Resultados:** *La ecocardiografía no demostró anomalías significativas del movimiento de la pared del corazón, pero debido al nivel elevado de troponina se realizó una angiografía coronaria y se estableció el infarto de miocardio con arterias coronarias no obstructivas. Una resonancia magnética cardíaca adicional confirmó el diagnóstico y estableció que la causa del infarto de miocardio fue la trombosis de la vena de Tebas. Se realizó una resonancia magnética (MRI) cardíaca, que mostró edema miocárdico en la pared inferior en la visualización potenciada en T2. El realce tardío con gadolinio mostró áreas focales de infarto subendocárdico. El paciente recibió tratamiento médico adecuado y experimentó buenos resultados clínicos. Este estudio de caso demuestra cómo el papel de la resonancia magnética cardíaca en el diagnóstico y tratamiento de pacientes con infarto de miocardio con arterias coronarias no obstructivas (MINOCA) se ha vuelto cada vez más crucial, a medida que la comprensión de la elevación de troponina y sus diversos mecanismos continúa evolucionando.*

Palabras clave: *Resonancia magnética cardiovascular, angiografía coronaria, MINOCA, ecocardiografía de estrés.*

INTRODUCTION

The traditional approach to acute chest pain is considered to be closely related to myocardial ischemic conditions, which in most cases are expected to be the result of coronary atherosclerosis. This conception conditioned diagnostic, therapeutic management, and prognostic evaluation of myocardial ischemic conditions. Guidelines of 4th Universal Definitions of Myocardial Infarction, which were published by (1), presented new conceptions in understanding myocardial infarction. Since then, there have been clear demarcations between myocardial infarction and myocardial injury. Today myocardial injury means non-obstructive coronary artery myocardial infarction (MINOCA) and Takotsubo syndrome. The consequences of this new conception consider large changes in future protocols of evaluation and management of patients with acute ischemic myocardial pain (2). Currently, in most clinical cases of patients with typical symptoms of myocardial infarction but no atherosclerotic obstruction, cardiologists often face skepticism, and the diagnosis is frequently denied (3). Despite the numerous reviews from Ukrainian and European societies, that specialize in the evaluation and management of patients with acute coronary syndromes, a lot of clinicians still deny the possibility of myocardial infarction in cases with non-obstructive coronary arteries.

The first stage when cardiologists are faced with a case of non-obstructive myocardial infarction is to determine which instrumental diagnostic methods can confirm this diagnosis. After changes in terminology, it was established that cardiac magnetic resonance (CMR) plays a crucial role in the differential diagnosis of non-ischemic causes of myocardial infarction. Lintingre et al. (4) and Pathik et al. (5) consider that the efficacy of CMR diagnostic of conditions with suspected MINOCA is between 60 % and 87 %. The issues regarding patient selection criteria for CMR diagnosis, the timing of CMR diagnosis (early or late), and the choice of CMR protocol for visualization still need to be addressed. After establishing a diagnosis, cardiologists are faced with the challenge of determining the treatment protocol. MINOCA encompasses various underlying causes, including coronary artery spasms, microvascular dysfunction, myocarditis, etc. Each etiology demands a distinct therapeutic approach, making it challenging to adopt a uniform treatment protocol. Besides, unlike the well-established guidelines for myocardial infarction with obstructive coronary arteries, there is a lack of standardized protocols for managing MINOCA. The absence of clear guidelines can lead to uncertainty among cardiologists regarding the most appropriate course of action. It remains unclear if the treatment protocols for patients

with acute myocardial infarction with obstructed coronary arteries are suitable for MINOCA cases.

Matta et al. (6) divided all pathological mechanisms of MINOCA into two groups: ischemic reasons and non-ischemic reasons. Ischemic reasons include spontaneous coronary artery dissection (SCAD), disruption of plaques, coronary spasm, dysfunction of the microvascular net, embolism or thrombus in coronary arteries, and mismatch in supply-demand mechanisms. The non-ischemic reasons include myocarditis, cardiomyopathy of Takotsubo, hypertensive disease of the heart, tachyarrhythmias, chemotherapeutic agents, cardiomyopathies, and intoxications with cardiotoxins. Fedorov et al. (7) analyzed the current position regarding MINOCA and consider that management of MINOCA remains challenging. Pharmacological treatment, indeed, remains relevant but uncertain due to the unclear etiology. Khanyukov et al. (8) consider that results from randomized trials in multicenter studies are eagerly awaited to find new pathobiological treatments.

This research aims to represent the typical case of MINOCA and describe the standard protocol of evaluation and treatment based on etiology.

Literature Review

Physicians can often misdiagnose acute myocardial infarction in cases of absence obstruction process in coronary arteries. As a result, the patients without correct diagnosis do not receive appropriate medical therapy and are wrongly reassured and discharged. Previous studies by Tamis-Holland et al. (9) and Talebi et al. (10) have examined the etiologies, diagnosis, and treatment of MINOCA. Specifically, Tamis-Holland et al. summarized the different possible causes of MINOCA and outlined appropriate diagnostic and therapeutic approaches in their conclusions. Meanwhile, Talebi et al.'s review highlighted the value of using diverse imaging modalities to determine the underlying etiology in all MINOCA cases. These two studies underscore the need for a thorough diagnostic workup using multiple imaging techniques to identify the specific causes of MINOCA and guide appropriate management.

Reynolds et al. (11) highlighted the use of intracoronary imaging and advanced methods of imaging modalities to determine the etiology of MINOCA. His study showed that nearly 40 % of patients with MINOCA cases have had signs of plaque disruptions in the past. These plaque disruptions include ruptured plaque, erosion of vessels, and calcified areas of the vessels. The performing of intracoronary imaging approved these signs. MINOCA cases with plaque disruption, approved by intracoronary imaging, on CMR imaging showed large areas of edema, which testified about the temporary ischemic condition in large vessels. Sometimes, a necrotic area was observed within the edema area (12). The only way to distinguish what reasons could lead to such appearances is the intracoronary provocative test or CMR imaging in a smaller, well-defined area of gadolinium enhancement in the late period (LGE) which can indicate that the most likely reason for myocardial necrosis was atherothrombotic debris embolization from the area of disruption (13). The CMR imaging is preferable to the intracoronary provocative test.

Collet et al. (2) in current guidelines for the management of MINOCA cases with ruptured plaque recommended medical treatment similar to plaque rupture in cases of obstructive coronary artery disease. Patients diagnosed with both plaque disruption and MINOCA are recommended to undergo dual antiplatelet therapy for one year, followed by single antiplatelet therapy for the remainder of their life. Patients with non-obstructive coronary artery disease, even if the atherosclerosis is at an early stage, are prescribed statin therapy as a treatment option. Lee et al. (14) described the case of MINOCA in a patient with sickle cell disease. In this research, a 49-year-old man with sickle cell disease in the past medical history with complaints of chest pain was hospitalized. Diagnosis of MINOCA used as secondary to microvascular obstruction. The patient received antiplatelet therapy and was discharged.

Despite the growing recognition of MINOCA as a distinct entity within the spectrum of acute myocardial conditions, several research gaps remain that merit further exploration. The absence of universally accepted treatment guidelines for MINOCA patients poses a

significant challenge. While guidelines for myocardial infarction with obstructive coronary arteries are well-established, MINOCA patients present a diverse array of underlying causes. The development of standardized protocols according to different etiologies is essential for optimizing patient outcomes. The optimal choice and timing of imaging modalities for diagnosing MINOCA remain uncertain. Comparative studies assessing the effectiveness of different modalities, such as CMR, echocardiography (ECG), and intracoronary imaging, could guide clinical decision-making. The mechanisms underlying subendocardial ischemia, as observed in some MINOCA cases, warrant further investigation. Advancing an understanding of potential biomarkers or imaging features that can predict treatment outcomes would facilitate more precise targeting of therapeutic strategies. Addressing these knowledge gaps through further investigation is key to unraveling the intricacies of MINOCA, optimizing diagnostic and management protocols, and ultimately improving prognosis and quality of life for patients affected by this complex syndrome.

MATERIALS AND METHODS

The object of this research is the clinical case of a 49-year-old male patient, who has visited the cardiologists with complaints of mild pain, "pressure" and "heaviness" behind the sternum radiating to his left arm. After significant physical exertion, shortness of breath and diaphoresis were observed. A case of this patient included previous cardiac ischemia. During a previous visit to the cardiologist, echocardiography was performed and mitral regurgitation of the I stage was established. Blood chemistry established hyperlipidemia, but the patient did not take prescribed statin therapy for the last 2 years. Holter monitoring registered supraventricular tachycardia. At the time of hospitalization, his blood pressure was 114/56 mm Hg. The heart rate was 57 bpm. The temperature was 36.4°C (97.6°F). The respiratory rate was 21 per minute. Blood saturation in room air was 98 %. The 12-lead ECG didn't show specific changes in the ST segment (Figure 1), but troponin increased to 11.5 ng/mL. ECG also did not show the disturbed function of the left ventricle. Coronary angiography didn't reveal any coronary segments with more than 50 % obstruction. A CMR was performed, which showed myocardial edema in the inferior wall on T2-weighted visualization. Late gadolinium enhancement showed focal areas of subendocardial infarction.

Table 1 shows laboratory results, which were sampled at the moment of hospitalization.

A transthoracic echocardiogram (TTE) was performed according to the guidelines of the British Society of Echocardiography (15). TTE showed no significant wall motion disorders. The ejection fraction was 59 %, and the heart volumes and wall thickness were normal.

Figure 1. ECG of the patient

Table 1

Shows laboratory results, which were sampled at the moment of hospitalization

Table 1. Laboratory tests

Parameter	Result	Reference range
Complete blood count		
Leukocytes, x10^9/L	16.4	3.5-10.6
Erythrocytes, x10^{12}/L	4.63	4.0-5.2
Haemoglobin, g/L	129	110-160
Haematocrit, %	24	35-47
Mean corpuscular volume, fl	95.7	75-98
Mean corpuscular haemoglobin, pg	31.7	27-34
Mean corpuscular haemoglobin concentration, g/dL	34.4	31.5-36
Platelets, x10^9/L	469	150-390
Erythrocytes distribution width (RDW-SD)	47.1	37-54
Erythrocytes distribution width (RDW-CV)	14.2	11-16
Platelets distribution width, fl	15.3	10-18
Mean platelets volume, fl	9.2	6.5-11
Erythrocyte sedimentation rate, mm/h	24	<15
Blood differential test		
Neutrophils, x10^9/L	4.57	1.7-7
Lymphocytes, x10^9/L	2.87	1-4.8
Monocytes, x10^9/L	0.38	0-0.8
Eosinophils, x10^9/L	0.08	0-0.45
Basophils, x10^9/L	0.01	0-0.02
Coagulogram		
Prothrombin time, s	10.2	9.8-12.5
Prothrombin index, %	96.3	70-130
International normalization ratio	1	
Activated partially thromboplastin time, s	30.5	22-32
D-fibrinogen, g/L	3.15	2-4
Thrombin time, s	18.4	14-21
Lipidogram		
Cholesterol, mmol/L	6.2*	<5.2
Triglycerides, mmol/L	2.12	<2.26
High-density lipoproteins, mmol/L	1.51*	>1.68 – no risk
Low-density lipoproteins, mmol/L	3.8*	<2.59 – the optimal level
Biochemistry		
Serum glucose, mmol/L	4.92	4.11-5.89
Creatine phosphokinase, U/L	1234	39-308
C-reactive protein, mg/dL	1.34	5

Evidence of pericardial effusion was absent. Aortic dimensions were within the normal range. In the apical projection, increased trabeculation of the apex of the left ventricle was noticed. Pulmonary regurgitation of the I stage and mitral regurgitation of the I stage was noted. Complaints of discomfort behind the sternum and shortness of breath after physical exertion, dyslipidemia, and the age of the patient become the prescription to undergo stress echocardiography (stress EchoCG). Stress EchoCG was carried out on a horizontal bicycle ergometer Shiller erg 911L on a Toshiba Aplio 50 scanner and a Mortara scribe electrocardiograph and performed according to the ABCDE Stress-Echo protocol (16). The results showed that the patient was indicated to pass over coronary angiography.

This study aligned with the ethical principles of research, including anonymity, confidentiality, and beneficence. Ethical approval of the study was obtained from the Health Research Ethics Commission of the Bogomolets National Medical University with No. MO-178.

RESULTS

After the diagnosis of MINOCA was established, the goal of the treatment strategy was to identify the underlying etiology and prescribe appropriate pathoetiological medical treatment. To achieve this, a follow-up TTE was ordered after the coronary angiography. Protocol of TTE on MINOCA cases was performed on expert class devices. In this case, TTE was performed on the GE Vivid E9 scanner. All measurement calculations were performed during three cardiac cycles, except continuous wave (CW) Doppler and pulse wave (PW) Doppler. The ejection fraction was recalculated to Simpson's rule and became 58 %. Classification of regional movements of cardiac walls made by visual interpretation of the wall's width. Segments, which become thicker more than 30 %, are regarded as normokinetic. Less than 10 % are regarded as akinetic. Segments can be regarded as dyskinetic if there is systolic thinning or eccentric excursion. In this patient, pathological dismovement was not observed.

2D loops from the projection of three apical cameras were analyzed with the use of the Q-analysis module in longitudinal 2D strain in EchoPAC BT11.2. When the area of interest is marked with the borders, this module generates strain curves and peak systolic strains in semi-automatic mode. The software utilized in this study automatically generated strain curves and peak systolic strains by marking the myocardial borders as the region of interest (ROI). Additionally, it calculated the post-systolic index, which measures the proportion of shortening after aortic valve closure and was found to be less than 0.2. Temporal derivation of strain rate was also performed. Both Global longitudinal strain (GLS) and segmental values for all variables are recorded. The Tissue Doppler Imaging (TDI) Q-analysis module utilized in this study analyzed loops from color-coded tissue Doppler obtained from the same views in apical projection. The software module placed ROIs in the two basal segments in each imaging view to evaluate the maximal systolic and diastolic velocities in the early stages, the timing of the cardiac cycle events, and the myocardial performance index for both ventricles. The analysis revealed a myocardial performance index of 0.3.

Mitral inflow velocities, which include E-speed (850 mm/s) and A-speed (550 mm/s) were calculated. E/E' was 6, and pulmonary venous flow was 450 mm/s. In this investigation, a custom interface designed in LabVIEW was utilized to examine the E waves during free breathing and after the Valsalva maneuver. To study the E waves, the equation for a simple harmonic oscillator was applied, and the constants k, c, and x were calculated by fitting the curve to the maximum velocity envelope of each E wave. The values of these constants represent chamber stiffness, viscoelastic energy loss, and load, respectively. Moreover, the time constant of isovolumic chamber relaxation (IVRT) was assessed and was found to be 84 ms in the patient under observation. The stiffness and relaxation components of the deceleration time (DT) of the E wave were measured to be approximately 180 ms. These outcomes facilitated the computation of a load-independent index of diastolic filling and stiffness.

Considering complaints of discomfort behind the sternum and shortness of breath during physical exertion, dyslipidemia (increased levels of total cholesterol and low-density lipoproteins, decreased level of high-density lipoproteins), as well as age, the patient was indicated to undergo stress echocardiography (stress EchoCG) (Table 2).

On the initial ECG, a sinus regular rhythm and a slightly negative T wave in lead III were recorded. On the initial echogram, there was a zone of hypo-/akinesis in the region of the apical lateral segment in the 4-chamber view and the local effect of spontaneous contrasting of the rounded shape with a diameter of about 1.2 cm, which was suspicious of a hypoechoic intracardiac thrombus located in the region of the trabeculae of the apex-lateral walls of the left ventricle. At a load of 50 W, in the apex-lateral segment of the left ventricle, normokinesis

Table 2

Stress-Echo protocol

Stage	Workload, W	Time, min	Heart Rate, bpm	BP, mmHg	ECG	EchoCG	Complaints
Initial (0)	0	3:00	67	120/75	"-" T wave in lead III	Hypo-/akinesis of the apical lateral segment in the 4-chamber view, the local effect of spontaneous contrasting in the region of the apex-lateral wall of LV	none
1	50	3:00	111	130/80	-//-	normokinesis	none
2	100	1:03	130	130/80	Horizontal depression 1.2 mm in V5-6, deepening of "-" T in lead III.	Normo-/hypokinesis of the apical lateral segment in the 4-chamber view, a small rupture of the lateral wall of the LV, the release of the part of the thrombus beyond the pericardium of LV lateral wall	cyanosis dyspnoea
Restitution	0	3:00	105	125/75	"-" T in lead III	normokinesis	none
Rest	0	5:00	67	120/70	As initial	Hypo/akinesis of the apical lateral segment of LV	none

Note: LV – left ventricle.

is registered in contrast to hypo-/akinesis as previously at the initial stage. It was decided to continue the study, as there were no complaints or negative ECG trends. At a workload of 100 W after 1 min. 3 s., the appearance of shortness of breath, and mild cyanosis of the lips were noticed, as well as horizontal depression of the ST segment with a maximum amplitude of 1.2 mm in leads V5-6, deepening of the negative T wave in lead III were registered on ECG. On the echogram, attention is drawn to a small rupture of the lateral wall, the lumen of which was filled with a thrombus with the release of a part of the thrombus beyond the pericardium of the lateral wall. At the same time, the size of the cardiac thrombus within the left ventricle was decreased. In addition, there was mild hypokinesis in the apical segment of the intraventricular septum in the 4-chamber view (Figure 2).

The restitution period was normal. At 5 minutes of rest, the condition of the patient, ECG, and contractility of the left ventricle on EchoCG returned to baseline. Given the fact that the patient was suspected of myocardial infarction, coronary angiography was performed. Based on the findings from the coronary angiography, it was concluded that the patient did not have any significant obstructive lesions in the left coronary artery. Nevertheless, a distinct contrast blush was observed at the endocardial surface, generating a ventriculogram through the extensive presence of multiple micro fistulae originating from the diagonal branch of the left anterior descending artery and emptying into the left ventricular cavity (Figure 3).

A peculiar contrast blush was observed in both the left (A and B) and right (C and D) coronary arteries. This unusual flow drained into the left ventricular chamber, and it was traced to the Thebesian venous network by following the arrows. Interestingly, the visualization of the endocardial border was facilitated by this contrast blush, which was particularly evident in images B and D (Figure 3). The intermittent visualization of these fistulae may indicate thrombosis in the Thebesian veins, which could

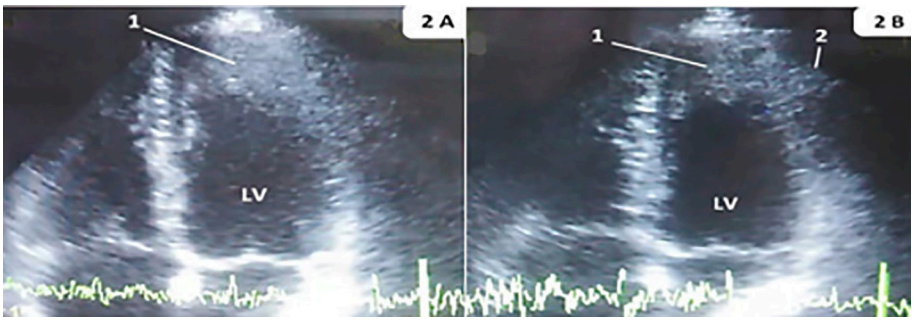

Figure 2. Apical 4-chamber view during stress-EchoCG test: 2A – before the test; 2B – after stage 2 (1 – the local effect of spontaneous contrasting; 2 – the part of the thrombus beyond the pericardium).

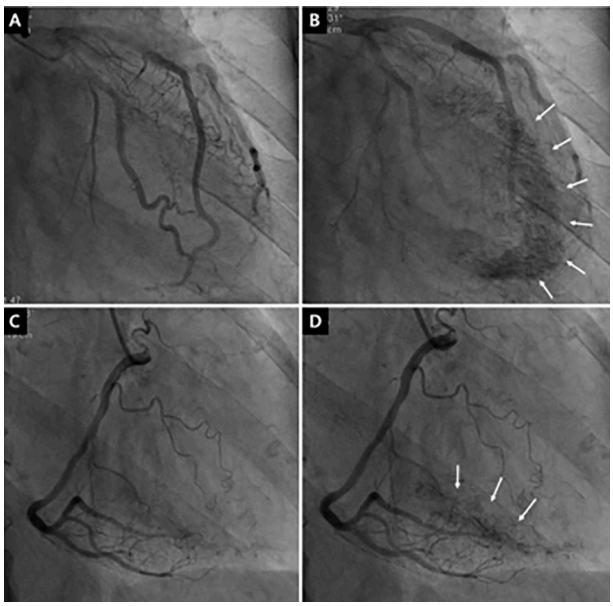

Figure 3. Coronary angiography showing the absence of significant stenosis.

be the underlying cause of the symptoms. Based on a retrospective analysis of this clinical case, it was determined that the most effective laboratory investigations should encompass routine clinical chemistry. In addition, it is recommended to perform a comprehensive array of tests to evaluate different domains, such as haematology, cystatin C, glucose, hsTroponinT, lipids, NT-proBNP, thyroid function, D-dimer, electrolytes, metoxycatecholamines, and the albumin to creatinine ratio in the urine. In every case, an oral glucose tolerance test (OGTT) must be performed along with measurements of HbA1C and cortisol levels in saliva (morning and evening). Additionally, all patients, which are suspected of MINOCA should undergo haematological testing after 6 months to rule out the risk of thromboembolism and systemic lupus erythematosus.

Moreover, during the right coronary angiography, a comparable capillary blush was noticed, which drained into the left ventricular cavity. This capillary blush was identified as originating from the branch of the posterolateral artery and posterior descending artery. The observed results suggested the existence of an arterioluminal form of the Thebesian venous network that collects blood from the coronary arteries and empties it directly into the ventricle. Further testing with CMR revealed a definite subendocardial perfusion defect at the level of the base to the middle part of the left ventricle during the rest perfusion sequence. The discovery implies that noticeable Thebesian veins may instigate subendocardial ischemia, even when at rest, and present as an acute coronary syndrome. Late enhancement of gadolinium, signifying myocardial infarction in the subendocardial layer, was confirmed at a later stage. To treat the patient's condition, bisoprolol, valsartan, and a small dose of anticoagulants to decrease cardiac afterload were administered alongside antiplatelet therapy. The patient's symptoms ameliorated, and he was subsequently released from medical care. Follow-up at the outpatient

clinic revealed that the patient remained stable for three months.

It is crucial to conduct an outcomes-based clinical trial to assess the efficacy of MINOCA patient management treatment guided by CMR. A thorough CMR evaluation including functional imaging and advanced tissue characterization techniques like mapping and late gadolinium enhancement can help diagnose and predict outcomes in MINOCA patients, enabling personalized treatment. It should be included as a primary diagnostic test in clinical guidelines. This protocol should be performed within one week of acute presentation with complete coverage of the left ventricle to provide a thorough evaluation. In cases where patients exhibit a clear non-ischemic cause, the typical protocol for acute coronary syndrome may not be beneficial and could even be harmful. Additional research is needed to determine whether the standard heart failure treatments, including beta-blockers and ACE inhibitors, can reduce the risk of progressing dilated cardiomyopathy and severe arrhythmias for patients with acute myocarditis, especially those who have impaired left ventricular ejection fraction, regional wall motion abnormalities, or extensive late gadolinium enhancement imaging. The benefits of utilizing CMR to guide treatment strategy in MINOCA patients extend beyond a precise diagnosis of myocardial infarction and distinguishing it from non-ischemic causes. It also provides valuable information about the extent and functional impact of acute myocardial injury, allowing for a more individualized treatment approach for each patient. This can include identifying the presence of myocardial inflammation, fibrosis, or scarring, which may require specific therapies or interventions. Additionally, CMR can help monitor the response to treatment over time, allowing for adjustments as necessary to optimize patient outcomes.

An optimal approach for managing MINOCA patients would involve a thorough and non-invasive assessment of both the upstream coronary arteries and downstream myocardium, which would enable the development of a personalized treatment plan. The use of a comprehensive evaluation, such as multiparametric CMR imaging, would be highly beneficial for diagnosing and predicting outcomes in MINOCA patients and should be considered a primary diagnostic tool in clinical guidelines. It is promising that CMR has demonstrated additional clinical benefits in diagnosing and forecasting outcomes in patients with MINOCA, and it should be included in clinical guidelines as one of the primary diagnostic tests to allow for further classification of these patients. This integration will provide a more comprehensive understanding of the extent and functional consequences of acute myocardial injury, as well as inform personalized treatment plans.

DISCUSSION

The results of this study provide valuable insights into the diagnostic and treatment challenges associated with MINOCA, particularly when caused by intracardiac thrombosis involving Thebesian veins. The case demonstrates that MINOCA can present with mild or atypical symptoms, minimal changes on ECG, and even absent pain syndrome, highlighting the importance of considering alternative diagnostic methods beyond traditional markers of acute coronary syndromes. The utility of cardiac magnetic resonance (CMR) as a comprehensive diagnostic tool is underscored, as it enables the identification of subtle myocardial abnormalities and the differentiation between ischemic and non-ischemic causes.

Thygesen et al. (1) defined 5 types of myocardial infarction. It was highlighted that myocardial injury taxonomy needs improvement, as it is still rather challenging to differentiate its subtypes and choose appropriate therapeutic approaches depending on the cause.

MINOCA is not associated with stenosing atherothrombosis of the coronary arteries and occurs in patients with intact coronary arteries or initial coronary atherosclerosis when atherosclerotic plaque is less than 50 %. At the same time, atherosclerotic lesion of the coronary arteria with the presence of "young" plaques with a thin fibrous cap and a large lipid core, which can be damaged under various conditions and lead to partial or complete thrombosis followed by spontaneous thrombolysis, is considered as one of the causes of MINOCA. As a result, Sucato et al. (17) defined that interrupted myocardial

infarction can occur when the area of necrosis is noticeably smaller or even in very rare cases is not registered. This clinical case represents a similar situation when the area of necrosis was so small, that even ECG didn't show significant abnormalities. In such situations, Kovacs I noticed the phenomenon of spontaneous thrombolysis with reflow. In addition, Meah and Williams (18) noted that an atherosclerotic plaque can be completely or partially located eccentrically (so-called "positive" remodeling) when there is no narrowing of the coronary arteries lumen, but the risk of injury and myocardial infarction still exists. The experience of this study confirms that MINOCA cases require investigation of every abnormality that was noted.

The heart veins in their number and size significantly exceed the arteries. The coronary venous system is divided into two groups of veins. While coronary sinus and tributary veins, as well as veins draining the right ventricle and the atria, belong to the greater cardiac venous system, the lesser cardiac venous system is represented by Thebesian veins, particularly small vascular channels, and venous sinusoids. Thebesian veins with a diameter of 50 to 200 microns are localized within the myocardium. Sirjuddin et al. (19) studied that in the atrium, their structure is similar to veins, and in the ventricles, Thebesian veins look like sinusoids – narrow vascular fissures with the endothelium in the myocardium. In this research, knowledge of variant coronary venous anatomy in CMR played a role in the diagnosis of Thebesian vein thrombosis. Four types of Thebesian veins are described. The first one has a short tree-like trunk, which is the basis of a network of vessels extending into the deep layers of the thickest sections of the myocardium. The second type of vessels occur in areas of the myocardium of small thickness, practically do not have a trunk, and immediately diverge in a star-like manner in the form of thin branches anastomosing with each other. The third type of vessel – long channels, almost without branches and passing through the myocardium. Finally, the fourth type of vessel is small veins with a small number of branches passing through the deep layers of the heart muscle.

Heart veins are mentioned mostly regarding resynchronization therapy, left ventricle pacing and arrhythmia ablation, drug-targeted therapy, and delivery of stem cells to the infarcted myocardium. In this case, the patient's medical history is not burdened by anything like this and it remains unclear what led to thrombosis of the Thebesian veins. Ansari (20) considered the significance of Thebesian veins and defined that they supply blood to the myocardium in coronary arterial occlusion, acting as a natural nutrient channel. Consequently, Thebesian veins are of great compensatory importance in conditions of insufficient coronary blood supply. Moreover, Boonysirinant et al. (21) established that they can regress and even disappear after myocardial infarction. It is logical to assume the theory that in this case, Thebesian veins could have appeared as a compensatory mechanism for previously occurred myocardial ischemia and will regress over time, but it is not possible to verify this theory. The connection of sinusoids with the branches of the coronary system of the heart is noted. Cernica et al. (22) assumed that it may be a compensatory mechanism of myocardial blood supply, especially in ischemic patients. In the current patient, thrombosis of Thebesian veins opening on the endocardium of the left ventricle was noted in the region of myocardial infarction.

Padfield (23) described similar to this research clinical case where Thebesian veins were localized in the region of the trabeculation of the left ventricle and were sclerosed during coronary atherosclerosis. In addition, turbulent blood flows develop in the area of trabeculae and chords, which eventually damage the endothelium of these vessels (24). It is in these places that a venular intraventricular thrombus can form. It is formed, as a rule, due to increased blood turbulence and, possibly, due to aseptic inflammation and sclerosis of Thebesian veins (25, 26). In this case, a thrombus can cause microembolization and thrombosis of the microvasculature of the myocardium, followed by focal necrosis and gradual thinning of the myocardium which, perhaps, leads to myocardial microrupture in the left ventricle of the current patient.

At the same time, these processes can occur gradually and strictly locally, which significantly changes the clinical manifestations of MINOCA. The pain syndrome can be mild or atypical or absent with insufficient or even no changes on ECG, as it happened in the current patient (27). Regarding EchoCG, in the area of myocardial

damage in the apical lateral segment of the left ventricle, hypo-/akinesis developed as a protective reaction to injury (stunning of the myocardium) (28-30). At stage, I, the area of the viable myocardium had to be differentiated from the hibernating myocardium. Consequently, it was decided to increase the load. At stage II, normo-/hyperkinesis was registered, which made it possible to exclude the hibernating myocardium due to severe coronary atherosclerosis. However, it was under this load that a micro-rupture of the lateral wall of the left ventricle was registered, which was the main cause of myocardial stunning.

As for the tactics of the management of this patient regarding a cardiac thrombus, most likely it is necessary to highlight two tactical points. First, if a fresh intracardiac thrombus was located in the area of trabeculae without lateral wall hypokinesis, it is possible, after examining the patient for thrombophilia, to start using antiplatelet agents with small doses of new oral anticoagulants (31, 32). In the presence of hypokinesis of the left ventricle lateral wall, it is necessary to remember the possible damage not only to the fibrous capsule of the CA atherosclerotic plaque but also to the endomyocardium. Transesophageal echocardiography may be useful in these patients to evaluate endocardial damage. In such patients, anticoagulants should be used with caution, as the presence of an intracardiac clot may be beneficial for the patient's survival (33, 34). In this present case, the clot was located inside the heart and may have covered the microruptures in the left ventricle.

While this study provides important insights, some limitations should be noted when interpreting the findings. The research presented results from a single patient case, which may not fully represent the broader MINOCA population. The individual patient characteristics and clinical presentations could limit the applicability of the obtained results to other cases. The small sample size of one patient limits the statistical power and generalizability of the findings. A larger patient number is needed to validate the trends observed in this study. The short follow-up period post-treatment does not allow us to assess the long-term effectiveness and durability of the treatment strategy employed. Longer follow-up periods are necessary to evaluate the sustained benefits.

CONCLUSIONS

The results of this research showed that clarifying MINOCA diagnosis requires significant diagnostic capabilities. In this case, EchoCG, stress-EchoCG, coronary angiography, and CMR were required to discover the reason for MINOCA. As previously discussed, MINOCA should be established after coronary angiography. In this case, during stress-EchoCG, was observed the phenomenon of a partial release of the thrombus through small ruptures in the lateral wall of the left ventricle into the pericardial cavity. Although this event may not be the cause of the symptoms, it could be a step toward explaining their etiology. Coronary angiography showed a vast capillary blush of Thebesian veins, draining into the cavity of the left ventricle. Despite the low distinctiveness of such a small branch, obstruction is observed in some of them. Further, CMR with late gadolinium enhancement showed subendocardial myocardial infarction at the delayed phase. In this way, "suspicious" results of coronary angiography and diagnosed myocardial infarction on CMR make it possible to establish the etiology of this MINOCA case – thrombosis of Thebesian veins. The patient was prescribed bisoprolol, valsartan, and antiplatelet therapy with small doses of anticoagulants. The benefit of using CMR in the case of MINOCA is obtaining the accurate reason for myocardial infarction to differentiate it from non-ischemic reasons. This can help to make the treatment of each patient more effective and individualized. Based on the research findings, the following recommendations for future clinical practice can be made. The promotion of multi-center research involving a large number of patients who potentially have a diagnosis of MINOCA can improve the guidance of therapy and enhance the prognosis of such patients. These studies can refine diagnostic criteria, treatment approaches, and risk factor assessment. Long-term outcome assessments will shed light on the effectiveness of interventions guided by CMR findings. It is necessary to develop standardized protocols for CMR and stress-EchoCG, ensuring consistent diagnostic procedures across diverse clinical settings. This will facilitate meaningful comparisons of results and outcomes. Specialized training for healthcare

professionals, along with patient education initiatives, can elevate the accurate diagnosis and management of MINOCA cases and is crucial to enhancing awareness and education. Eventually, a collaborative, multidisciplinary approach involving cardiologists, radiologists, and other specialists is crucial to refining MINOCA diagnosis and management.

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