Folia Cardiologica 2022
vol. 17, no. 4, pages 251–256
DOI: 10.5603/FC.2022.0046

ISSN 2353–7752
e-ISSN 2353–7760

Comorbidity of cholecystitis and myocardial infarction with non-obstructive coronary arteries in 65 years old female patient

Współwystępowanie zapalenia pęcherzyka żółciowego i zawału serca bez istotnych zwężeń w tętnicach wieńcowych u 65-letniej chorej

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Abstract

Myocardial infarction with nonobstructive coronary arteries (MINOCA), is increasingly recognized as an important and separate cause of myocardial infarction among patients with the acute coronary syndrome (ACS). The dominant pathophysiological mechanisms of MINOCA include coronary pathologies (epicardial spasm, coronary microcirculation disorder, spontaneous coronary dissection, coronary thrombosis/embolism) with the absence of disease with significant angiographic artery stenosis. This study presents a clinical course of myocardial infarction (MI) without significant stenosis in the coronary arteries and cholecystitis, in 65 years old woman, with some risk factors of atherosclerosis (smoking, obesity, atherogenic dyslipidemia, high levels of C-reactive protein, old age and physical inactivity). In the first days, myocardial infarction was complicated by an atrioventricular block. Coronarography revealed no significant stenosis confirming the occurrence of MINOCA.

Key words: MINOCA, myocardial infarction, non-obstructive coronary artery disease, microvascular disease, cholecystitis, inflammation

Folia Cardiologica 2022; 17, 4: 251–256

Introduction

The first reports of myocardial infarction without significant stenosis in the coronary arteries date back to the 1980s, and recently such a course of the disease was defined as MINOCA with an occurrence of 5–10% of all coronary syndromes. The aetiology of MINOCA is heterogeneous and can be divided into coronary and noncoronary (cardiac and non-cardiac) causes [1]. MINOCA is more common in women and younger patients and is less often associated with classical risk factors (e.g. diabetes, hypertension, dyslipidemia). The techniques used to make the initial diagnosis that is the basis for determining the cause of MINOCA are clinical history, myocardial enzymes, electrocardiography (ECG), echocardiography, coronary artery angiography, and left ventricular angiography [1, 2].

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Case report

A case is presented of a 65-year-old female patient, untreated for chronic diseases, admitted to the cardiology clinic due to pain in the epigastric region, mild diarrhoea and weakness. Electrocardiogram showed signs of acute coronary syndrome without ST-T elevation: ST-segment depression in I, II, V4–V6 leads to T-wave inversion in I, II, III, aVL, aVF, V2–V6 and with escape rhythm 50 beats per minute as well third-degree atrioventricular block and right bundle branch block (Figure 1). The levels of cardiac troponin T (cTNT) determined with the highly sensitive method were 2253 ng/L (0.0–14.1) and 2191 ng/L after one hour. Creatine kinase was elevated (1215 U/L [20–180]), together with N-terminal fragment of B-type natriuretic propeptide (6803.00 pg/mL [0.00–125.00]) and alkaline phosphatase (201 U/L [35–104]). Echocardiography revealed general hypokinesis with an ejection fraction of 40%, (Figure 2). The patient was treated with dual antiplatelet therapy (acetylsalicylic acid, clopidogrel), and received also angiotensin-converting enzyme inhibitors (ACEI). No beta blocker was added because of atrioventricular block and no statins — of the presence of symptoms of acute biliary disease. Abdominal pain has moved downward (with the localization on palpation typical for cholecystitis, laboratory tests results were consistent with the clinical picture: elevated bilirubin levels 2.24 mg/dL [0.10–1.20], increased C-reactive protein [CRP] 48.30 mg/L [0.60–5.00]), leukocytosis 16,56 K/uL [3.98–10.04] and anaemia (RBC 3.88 M/uL [3.90–5.10]) (Table 1). She was consulted surgically with ultrasound examination of the abdominal cavity (gallbladder of normal size with a thickened wall and calcified 9 mm stone, unexpanded bile ducts) and qualified to the pharmacologic, conservative treatment: antibiotic

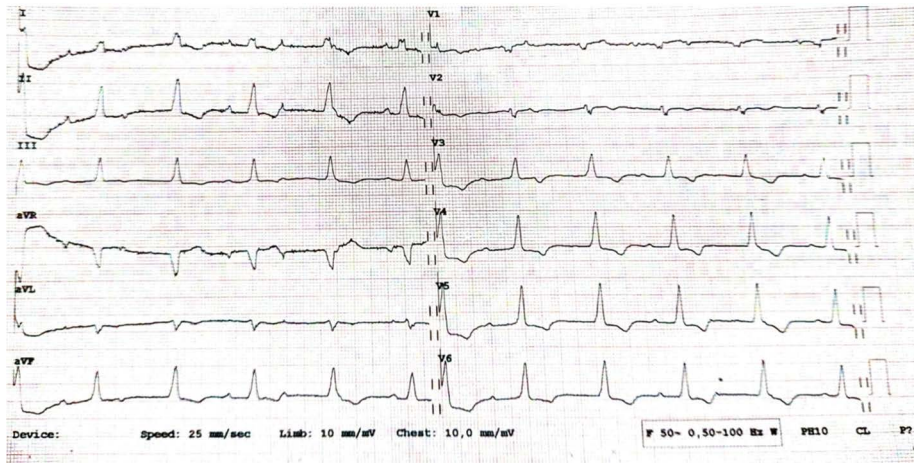

Figure 1. Electrocardiogram showing signs of acute coronary syndrome without ST-T elevation: ST-segment depression in I, II, aVF, –V6 leads with T-wave inversion in I, II, III, aVL, aVF, V2–V6 and with escape rhythm 50 beats per minute, third degree atrioventricular block and right bundle branch block

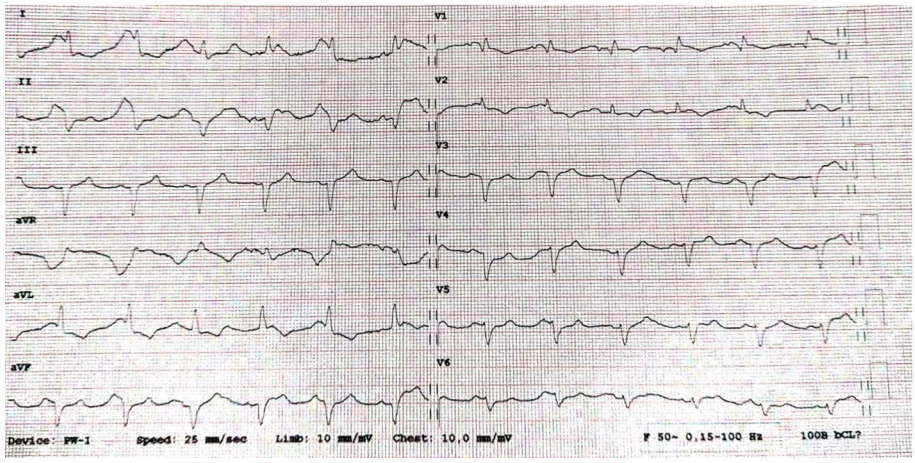

Figure 2. Electrocardiogram records showing ST segment depression occurred only in aVL lead, QS segment in lowering of R wave in III, aVR, aVF, V3, V4, V5, V6 leads

Table 1. Laboratory results of blood tests

Blood parameters	Value	The norm of value
cTNT	253 ng/L	0.0–14.1
cTNT after one hour	2191 ng/L	0.0-14.1
CK	1215 U/L	20–180
NT-proBNP	6803.00 pg/mL	0.00–125.00
ALP	201 U/L	35–104
Bilirubin level	2.24 mg/dL	0.10–1.20
CRP	48.30 mg/L	0.60–5.00
WBC	16,56 K/uL	3.98–10.04
RBC	3,88 M/uL	3.90–5.10
LDL	104 mg/dL	
TG	155 mg/dL	35–135 mg/dL
HDL	18 mg/dL	> 46 mg/dL
Hyperuricemia (uric acid)	12 mg/dL	3–7 mg/dL

ALP — alkaline phosphatase; CK — cretine kinase; CRP — C-reactive protein; cTNT — cardiac troponin T; HDL — high-density lipoprotein; LDL — low-density lipoprotein; NT-proBNP — N-terminal fragment of B-type natriuretic propeptide; RBC — red blood cells; TG — triglycerides; WBC — white blood cells

therapy (Ceftriaxone), antispasmodics and intravenous fluids therapy. Some typical risk factors of atherosclerosis were found: smoking, obesity (body mass index = 37.5 kg/m^2) atherogenic dyslipidemia (low-density lipoprotein 104 mg/dL, triglycerides 155 mg/dL, high-density lipoprotein 18 mg/dL), hyperuricemia (uric acid 12 mg/dL), high levels of CRP 48.30 mg/L (Table 1), old age and physical inactivity [3]. During hospitalization glucose parameters and blood pressure levels were normal. Due to all signs of the clinical picture, lowering TNT, and symptom relief after comprehensive treatment on the first day of hospitalization, a non-ST segment elevation myocardial infarction was diagnosed. During the first day of hospitalization, her general condition improved and the pain disappeared. Furthermore, the concentration of CRP decreased, so she was qualified for planned surgical treatment of the gallbladder.

Due to all signs of the clinical picture, lowering TNT, and resolution of symptoms after comprehensive treatment on the first day of hospitalization, non-ST segment elevation myocardial infarction in the evolution period was diagnosed.

On the second day of hospitalization ECG records showed ST segment depression only in aVL lead, QS syndrome in II, III, aVR, aVF, V3–V6 lead and in lowering of R wave in III, aVR, aVF, V3–V6 lead. Left ventricular ejection fraction assessed by echocardiography had increased by 46%, found regional disturbances were limited to hypokinesis of 4 segments: basal — inferoseptal, anteroseptal, of inferior wall, and medial of interior wall, with the improvement of longitudinal strain function (Figure 3). In the fifth day

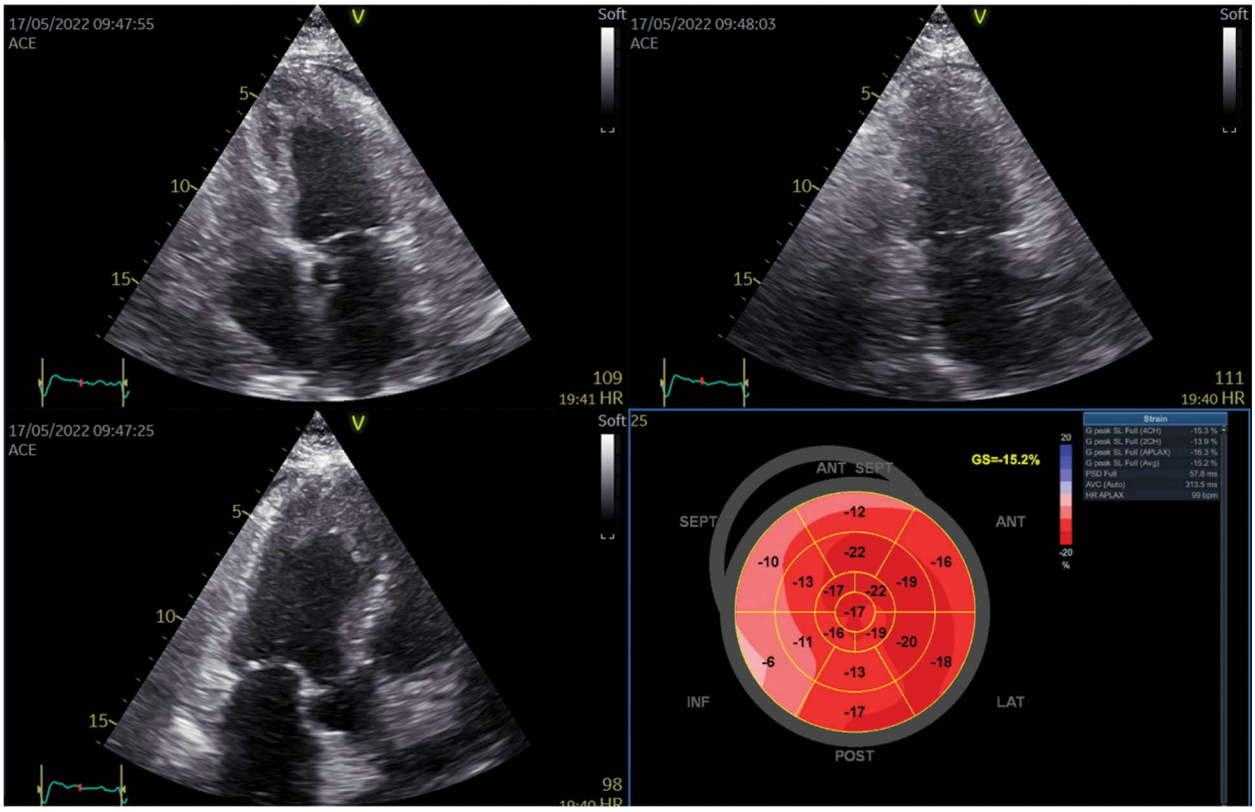

Figure 3. Transthoracic echocardiography: hypokinesis of 4 segments: basal — inferoseptal, anteroseptal, of inferior wall, and medial of interior wall, with mildly reduced global function of left ventricle, with reduction of global longitudinal strain

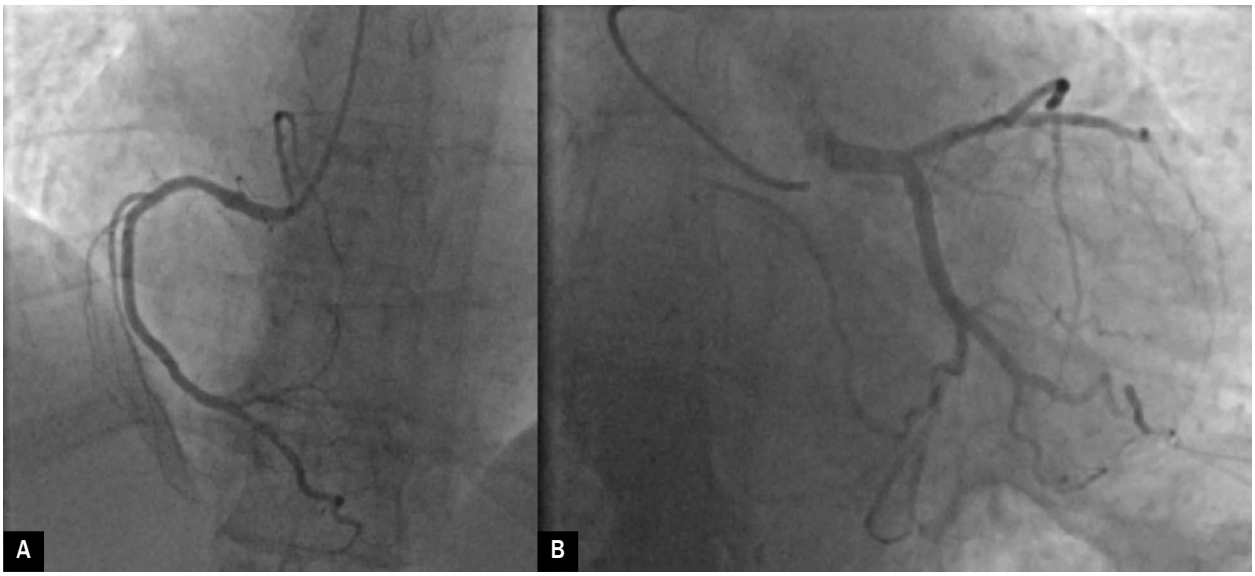

Figure 4. Normal picture of coronary angiography: right (**A**) and left (**B**) coronary arteries

of hospitalization, after lowering of TNT to 847.1 ng/L, coronarography was performed, but no lesions significantly narrowing the lumen of the coronary vessels were found, however, there were found para mural changes in RPD (posterior descending branch) and left ventricular branch of right coronary artery (Figure 4).

Then, 24-hour Holter ECG monitoring was performed, and sinus rhythm was found, with normal frequency (minimum 52 beats per minute, maximum 102 beats per minute) with 1 episode of nonsustained ventricular tachycardia — 6 beats. The invasive electrophysiological examination revealed: a third-degree paroxysmal atrioventricular block, with correct conduction time from the proximal His bundle to the ventricular myocardium 56 ms. The patient was qualified for pharmacologic treatment, without pacemaker implantation.

The ECG performed on the last day of hospitalization showed a regular sinus rhythm of 80 beats per minute, left anterior hemi block positive, and negative T-waves in V2–V6. The patient was discharged home after more than a dozen days of observation and treatment, with a recommendation of non-pharmacologic treatment (lipid-lowering diet, regular physical activity, cardiac rehabilitation in outpatient clinic) and pharmacotherapy: dual antiplatelet drugs, ACEI, proton pump inhibitor, symbiotic, drotaverine, and follow-up ambulatory cardiology and surgical and laboratory blood check-ups (morphology, ionogram, uric acid, urea, creatinine, alanine aminotransferase levels, lipid panel, blood glucose level).

Discussion

MINOCA is increasingly recognized as an important and separate cause of myocardial infarction among patients suffering from ACS. The dominant pathophysiological mechanisms of MINOCA include coronary pathologies (epicardial spasm, coronary microcirculation disorder, spontaneous coronary dissection, coronary thrombosis/ /embolism) with the absence of disease with significant angiographic artery stenosis (≥ 50% lumen narrowing) in any major epicardial vessel. The definition of the syndrome includes diagnosis of myocardial infarction by the guidelines, lack of ≥ 50% stenosis of coronary arteries, and no other alternative clinical cause of the disease. Includes patients with: normal coronary arteries (without angiographic stenosis), mildly expressed irregularity in the outline of the vessel lumen (angiographic strictures < 30%), and indirect atherosclerotic lesions of the coronary arteries (stenosis > 30% but < 50%) [4]. It can exclude myocarditis, Takotsubo syndrome, and cardiomyopathies, as well as provide imaging confirmation of ACS [5, 6].

In patients with MINOCA, the plaque in the coronary arteries tends to develop eccentric so the majority of coronary angiography cases show normal to mild stenosis [7]. In the case of the patient, no additional diagnostic tools were needed, because the symptoms and performed tests confirmed the diagnosis. If the diagnosis is uncertain, cardiac magnetic resonance imaging should be considered. Computed tomography, intravascular ultrasound,

transoesophageal and/or contrast echocardiography are the basic diagnostic tools in that situation [12].

While there is no obvious coronary stenosis in MINOCA patients, most of them have varying degrees of heart damage and are still at high risk of adverse cardiovascular events, survival rate is higher compared to people with significant changes in the coronary arteries, although lower than in healthy people matched for sex and age. Treatment and prognosis are strongly equated with pathogenesis, so it is especially important to successfully discover the disease causes. Using statins and an ACEI/angiotensin-receptor blockers to improve the long-term prognosis of MINOCA patients has been shown to have significant benefits [6, 7]. The pharmacotherapy recommendations focus on slowing the progression of coronary artery dysfunction, i.e. the appropriate treatment of risk factors and comorbidities [6].

Smoking, obesity, and atherogenic dyslipidemia can have a prothrombotic impact, and be the next factor contributing to flow disturbances in the coronary artery.

The typical risk factors of cholecystitis that the patient presents are old age, female gender and obesity. They may have contributed to the onset of this condition. Furthermore, gallstone disease has been related to cardiovascular risk factors, independently of traditional causes [10].

Atrioventricular block during myocardial infarction may be a temporary condition if it occurs within its first 7 days [11]. The indications for pacing in patients with his bundle branch block (BBB) are: 1) BBB with unexplained fainting and abnormal EPS result; 2) Variable BBB; 3) BBB with unexplained fainting, inconclusive diagnostics; 4) Asymptomatic BBB, however pacing is inadvisable in asymptomatic patients with BBB [11]. Cholecystitis, cholelithiasis and manipulation within the area of the gallbladder and other visceral organs during laparoscopic surgery may initiate coronary vasoconstriction and give rise to symptoms of myocardial infarction. The mechanism of ECG changes includes reduced coronary blood flow due to a distended bile duct, tachycardia, increased blood pressure and retinol levels mediated by the vagal reflex. Contraction of coronary vessels is a consequence of the above mechanism [8, 9].

Inflammation initiates clotting, reduces the activity of the natural anticoagulant mechanisms and impairs the fibrinolytic system. Inflammatory cytokines are the main mediators involved in the activation of clotting. Natural anticoagulants keep the blood levels of cytokines low. Inflammation affects the haemostatic balance, disrupting it and contributing to increasing the level of fibrinogen in the blood. In the present patient, the cholecystitis could have been a trigger for intravascular coagulation and caused myocardial infarction [13].

The presented case is an example of the presence of myocardial infarction without significant stenosis in the coronary arteries, but with some para mural changes, in a patient with risk factors of atherosclerosis influencing coagulative state, confirming the occurrence of MINOCA which was finally directly triggered by cholecystitis.

Conflict of interest

None declared.

Funding

None.

Streszczenie

Zawał mięśnia sercowego bez istotnych zwężeń w tętnicach wieńcowych (MINOCA) jest coraz częściej uznawany za ważną i oddzielną przyczynę zawału serca wśród pacjentów z ostrym zespołem wieńcowym (OZW). Dominujące mechanizmy patofizjologiczne MINOCA obejmują patologie wieńcowe (skurcz nasierdziowych tętnic wieńcowych, zaburzenia mikrokrążenia wieńcowego, samoistne rozwarstwienie tętnicy wieńcowej, zakrzepica/zatorowość tętnicy wieńcowej) przy braku istotnego zwężenia tętnicy wieńcowej towarzyszącego chorobie w badaniu angiograficznym. W niniejszym opisie przypadku klinicznego przedstawiono zawał mięśnia sercowego bez istotnego zwężenia tętnic wieńcowych i zapalenie pęcherzyka żółciowego u 65-letniej kobiety z czynnikami ryzyka miażdżycy (palenie tytoniu, otyłość, dyslipidemia aterogenna, wysoki poziom białka C-reaktywnego, podeszły wiek i brak aktywności fizycznej). W pierwszych dniach zawał mięśnia sercowego był powikłany blokiem przedsionkowo-komorowym. Badanie koronarograficzne nie wykazało istotnych zwężeń, potwierdzając tym wystąpienie zawału typu MINOCA.

Słowa kluczowe: MINOCA, zawał mięśnia sercowego, nieobturacyjna choroba wieńcowa, choroba mikronaczyniowa, zapalenie pęcherzyka żółciowego, zapalenie

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