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CASE STUDY/PRACA KAZUISTYCZNA

The 39-year-old patient with myocardial infarction with nonobstructive coronary arteries (MINOCA) in the course of acute cholecystitis

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Abstract

As defined, the use of the term MINOCA (Myocardial infarction with nonobstructive coronary arteries) is considered initially at angiography, as a working diagnosis until further assessment leads to the exclusion of other possible causes of troponin increase. As a MINOCA is a group of heterogeneous diseases with different mechanisms of pathology, an individual approach and adjustment to the patient's current condition are essential. This case report presents a 39-year-old patient with a myocardial infarction in the setting of a rare cause that may be acute cholecystitis.

Key words: myocardial infarction, AMI, MINOCA, cholecystitis, troponin

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Introduction

As defined, the use of the term MINOCA (Myocardial infarction with nonobstructive coronary arteries) is considered initially at angiography, as a working diagnosis until further assessment leads to the exclusion of other possible causes of troponin increase. The term encompasses a heterogeneous group of factors underlying cardiac injury, including both coronary and non-coronary pathogenic factors, involving cardiac and non-cardiac disorders [1].

Patients with a diagnosis of MINOCA presented a lower cardiovascular risk compared to patients with obstructive-CAD (Coronary Artery Disease), they were younger, and without

diabetes, hypertension, or hyperlipidemia. That suggests the predominant importance of non-atherosclerotic etiology and atypical risk factors such as psychosocial aspects, insulin resistance and inflammation [2]. Although, compared to patients with significant coronary atherostenosis, patients with MINOCA had similar outcomes, including mortality and psychosocial and functional status [3]. This case report presents a myocardial infarction (MI) in the setting of a rare cause that may be acute cholecystitis.

Case report

A 39-year-old woman with no history of chronic disease was admitted to the Department of Cardiology with suspected acute coronary syndrome (ACS). Before the admission, the woman was at a social event during which she felt palpitations and chest pain. The pain did not subside and after six hours, when the woman returned home, she briefly lost consciousness, after which she felt severely weak. The patient called the emergency medical team and was transported to the local ED.

In the ED, high levels of high sensitivity troponin were found, echocardiography showed reduced left ventricular ejection fraction (LVEF) and ECG showed sinus rhythm and intermittent right bundle branch block (RBBB). Abdominal ultrasonography examination showed a not enlarged gallbladder with a significantly thickened, stratified wall (10–15 mm wide), surrounded by fluid which is suggestive of inflammation (Figure 1).

On the clinical admission, the patient was hemodynamically stable, she denied any chest pain. Physical examination showed heart rhythm 74/min, blood pressure 104/70 mmHg, and normal vesicular breath sound without pulmonary congestion, no abdominal pain, and no signs of peritonitis.

Laboratory tests revealed an elevated level of N-terminal pro-B-type natriuretic peptide 5030.0 pg/mL, increasing concentration of high-sensitive troponin typical of the ACS (maximum concentration — 4586 ng/dL), elevated hepatic enzymes, leukocytosis and slightly reduced level of total proteins.

In electrocardiogram, there was regular sinus rhythm, without features of MI.

Another echocardiography was performed which showed a mildly decreased ejection fraction of the left ventricle (EF 43%) and generalized wall hypokinesis (Figure 2).

The patient received pharmacological treatment for heart failure in the form of beta-blocker, ACEI and torasemide. The next day, the patient reported abdominal pain occurring after meals, which was the patient's only complaint. Due to elevated troponin levels in laboratory

tests, the patient underwent coronary angiography which revealed no signs of CAD (Figure 3) and pulmonary arteries angiography that excluded pulmonary embolism. In addition, gastrointestinal consultation was performed at which it was decided to start empirical antibiotic therapy (ciprofloxacin and metronidazole) due to cholecystitis. In the following days, the patient's condition remained stable. In control abdominal ultrasound examination, the gallbladder was not enlarged, with a hyperechoic wall up to 5.5 mm wide (Figure 4). In the control of high-sensitive troponin was 162 ng/L. On the 5th day of hospitalization, the patient developed an allergic reaction after administration of ciprofloxacin at the injection site, which was resolved by changing the antibiotic to ceftriaxone. A follow-up ECHO scan was performed where an increase in ejection fraction from the last scan to 49% was observed. A follow-up laboratory test showed a decrease in NTproBNP levels (to 745 pg/mL) and a normalization of liver enzymes. The next day torasemide was discontinued and replaced with 25 mg eplerenone. The patient was consulted by a gastroenterologist and was advised to undergo gastroscopy and serological tests to diagnose coeliac disease and Helicobacter pylori infection. A diagnosis of gastropathy, duodenopathy and gastric polyp was made. The urease test was negative.

Considering the whole clinical picture, a diagnosis of myocardial infarction without coronary lesions (MINOCA) in the course of cholecystitis was made. The patient was discharged from the hospital in stable condition without stenocardial complaints. The recommended home treatment was 25 mg eplerenone, 5 mg bisoprolol and 2.5 mg ramipril per day. Echocardiographic follow-up after one month reveals no evidence of contractility disturbances (Figure 5). The LVEF (calculated using the modified Simpson's biplane method) is 57.8%, showing improvement of left ventricle function.

Conclusions

This case report illustrates therapeutic challenges in managing patients with MINOCA indicated by cholecystitis. As a MINOCA is a group of heterogeneous diseases with different mechanisms of pathology, it makes therapeutic management more difficult. An individual approach and adjustment to the patient's current condition are essential. Managing such a patient with a presumptive diagnosis of MINOCA should be treated and monitored according to the guidelines for the underlying disease, in this case with antibiotics.

Discussion

According to the ESC statement in order to diagnose MINOCA, the criteria for AMI, as

defined in the Third Universal Definition of Myocardial Infarction, must be met. The coronary arteries must be free of significant stenosis (I.e. no greater than 50% stenosis in the large epicardial artery), and there must be no other specific cause that could be an alternative of the clinical picture [4].

Management should include an initial assessment of LV wall contractility. Episodic wall motion abnormalities may indicate an epicardial cause of MINOCA or other specific causes, the finding of which will lead to the exclusion of this diagnosis.

Pulmonary embolism should also be considered as an alternative diagnosis and possible cause of myocardial damage and can be ruled out by additionally determining D-dimer levels, BNP or performing CT pulmonary angiography if indicated.

In addition, other circumstances associated with an imbalance between oxygen supply and oxygen demand or associated with an increase in cardiac troponin levels, including hypertensive crisis, tachyarrhythmias, sepsis and anemia, should also be considered as potential causes of myocardial damage.

Patients with a presumptive diagnosis of MINOCA and an underlying cause of cardiac injury identified at diagnosis should be treated and monitored according to the guidelines for specific diagnosis. Patients with a final diagnosis of NSTEACS (ACS without ST-segment elevation) or MINOCA of unknown cause should be monitored as post-ACS patients with significant coronary artery stenosis.

As part of the treatment of MINOCA, acetylsalicylic acid, statins, angiotensin-converting enzyme (ACE)/angiotensin receptor blockers (ARBs) and beta-blockers may be suggested. In a long-term observational study in the SWEDEHEART registry, these drugs showed significant long-term beneficial effects on overall mortality, cardiovascular mortality, the incidence of acute myocardial infarction, stroke and major adverse cardiac events [5].

Additional information

Author contribution

Equal division of responsibilities during writing the paper

Conflict of interest

The authors declare no conflict of interest

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Streszczenie

Wśród pacjentów z MINOCA (Ostrym zespołem wieńcowym bez istotnych zwężeń w tętnicach wieńcowych), przyczyna zawału serca nie zawsze jest oczywista. Chorzy z rozpoznaniem MINOCA manifestują niższe ryzyko sercowo-naczyniowe w porównaniu z pacjentami z chorobą wieńcową. Rzadziej współistnieją u nich cukrzyca, nadciśnienie czy hiperlipidemia, co sugeruje dominujące znaczenie innych nietypowych czynników ryzyka. Ponieważ postępowanie różni się w zależności od etiologii, niezbędne jest indywidualne podejście do diagnozy oraz terapii każdego przypadku MINOCA. W niniejszym opisie przedstawiono przypadek 39-letniej pacjentki, u której wystąpił zawał serca w przebiegu ostrego zapalenia pęcherzyka żółciowego.

Słowa kluczowe: zawał serca, ostry zespół wieńcowy, MINOCA, zapalenie pęcherzyka żółciowego

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Figure 1. First abdominal Ultrasonography: Cholecystitis — significantly thickened, delaminated, surrounded by fluid gallbladder wall 10–15 mm wide.

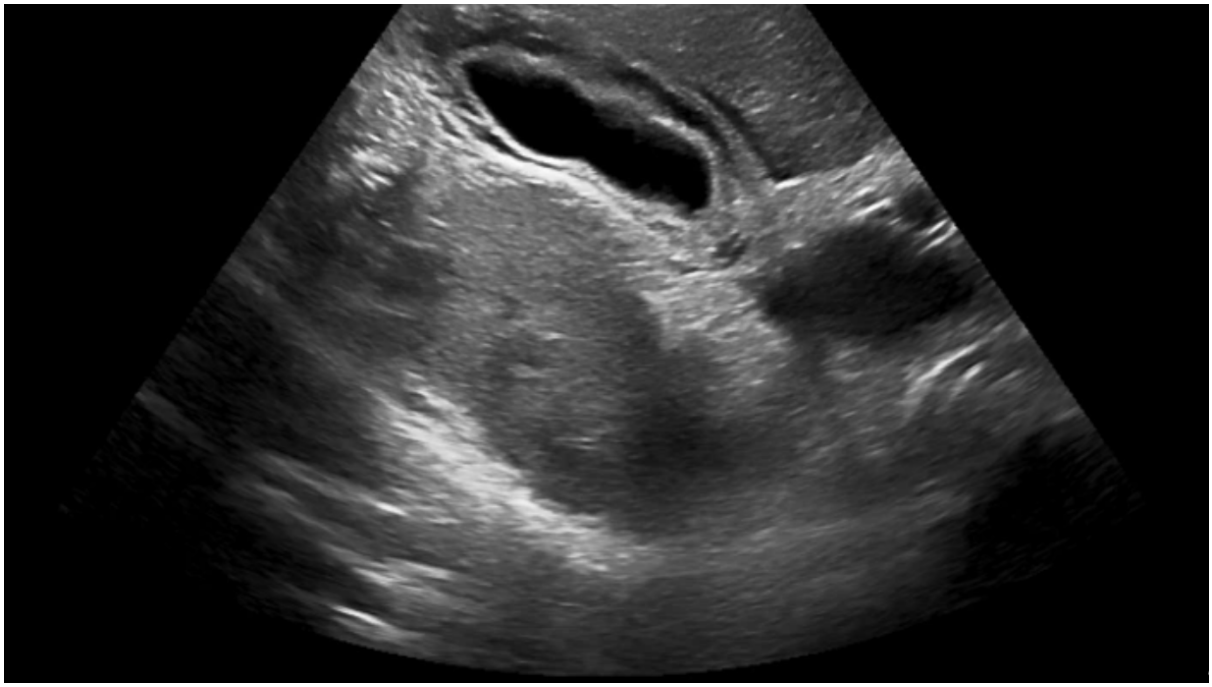
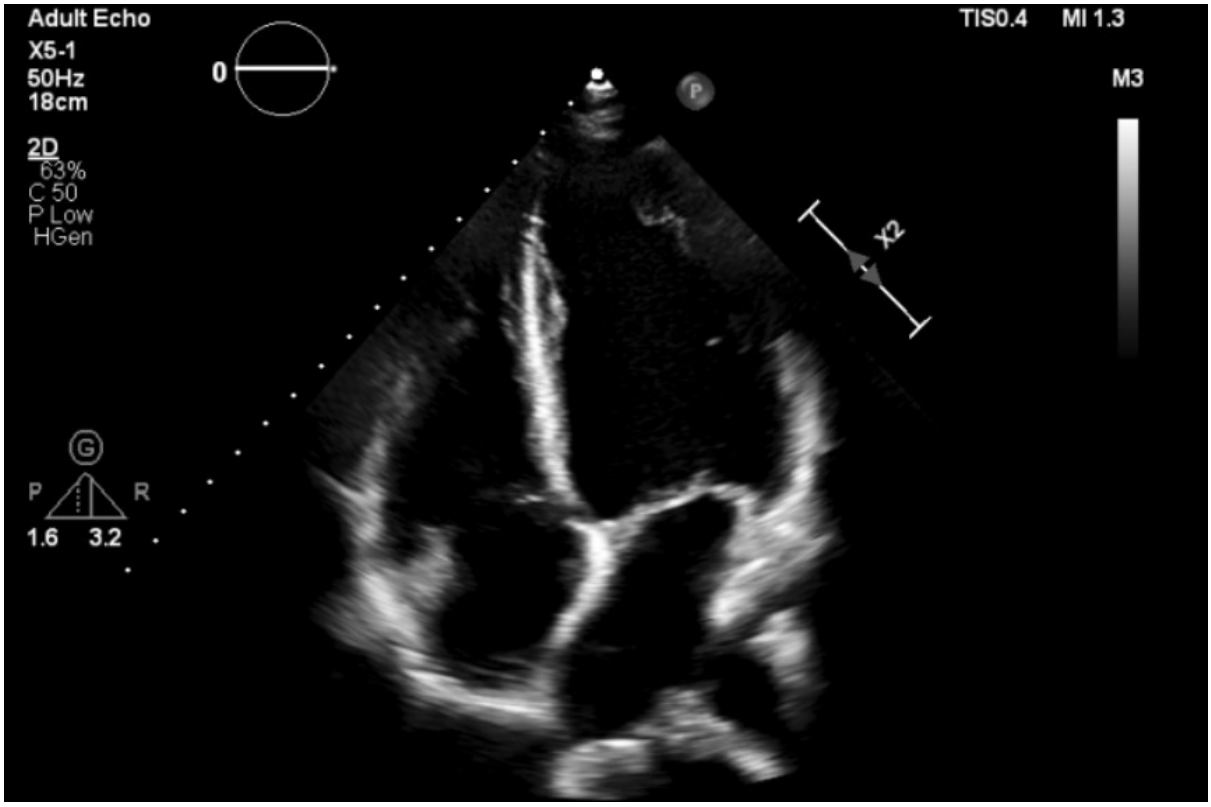


Figure 2. Echocardiography: Apical four-chamber view in end-diastolic (Panel A) and end-systolic phases (Panel B).

Panel A:



Panel B:

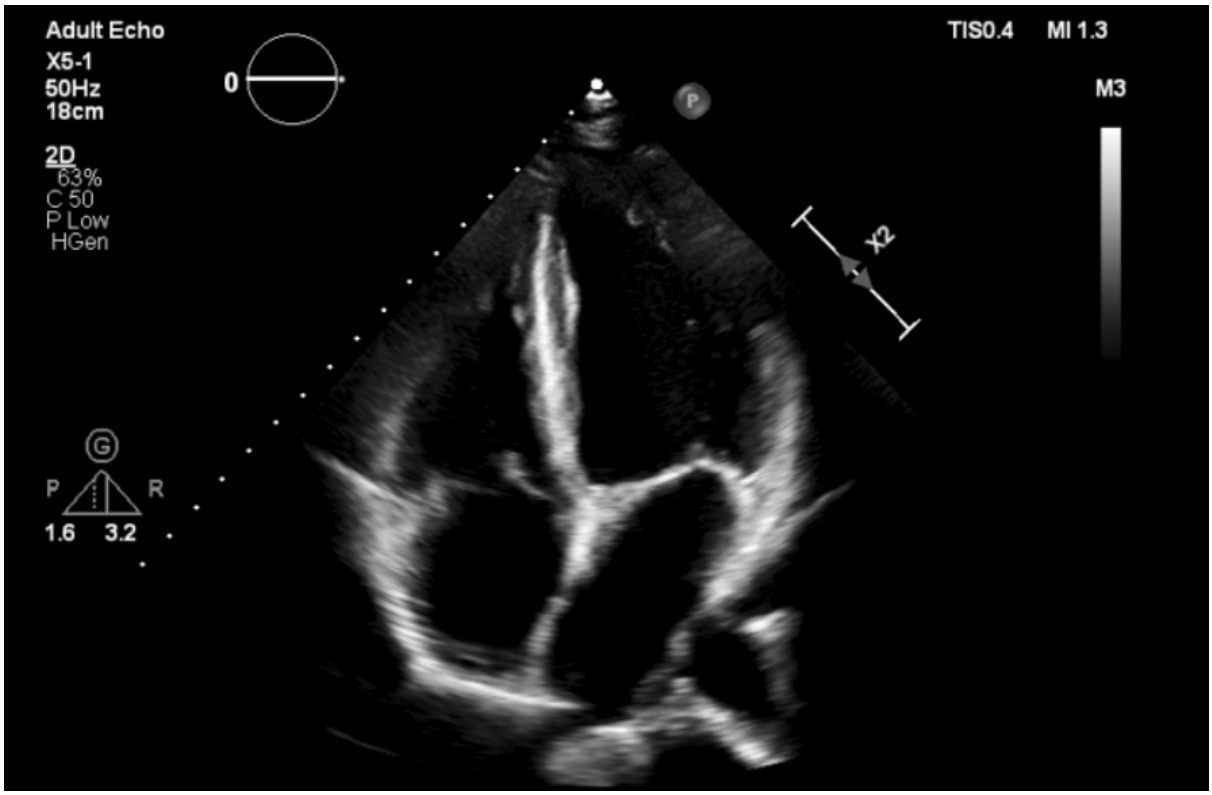
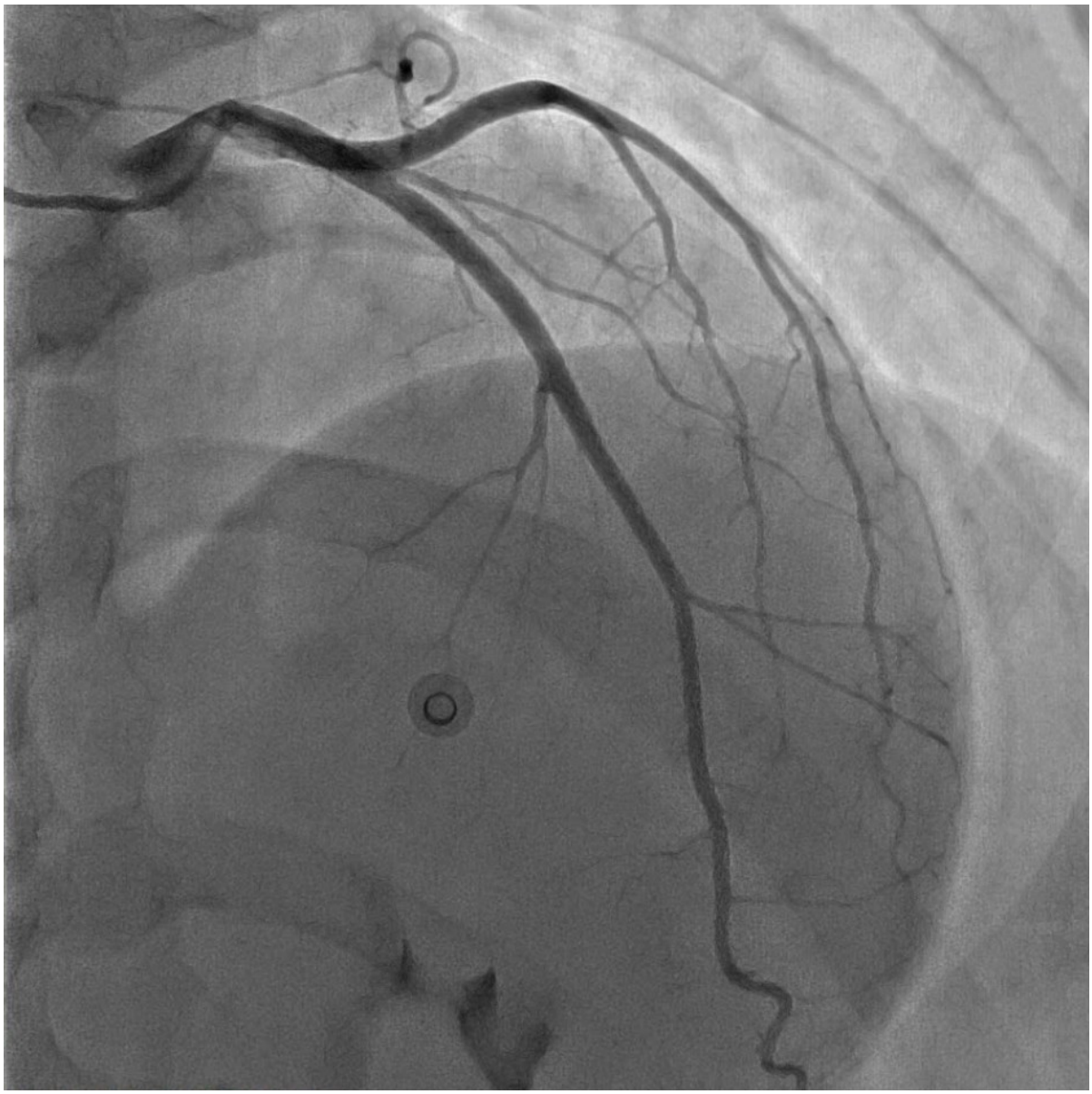


Figure 3. Coronarography (Panel A — Left coronary artery, Panel B — Right coronary artery) without significant stenosis.

Panel A:



Panel B:

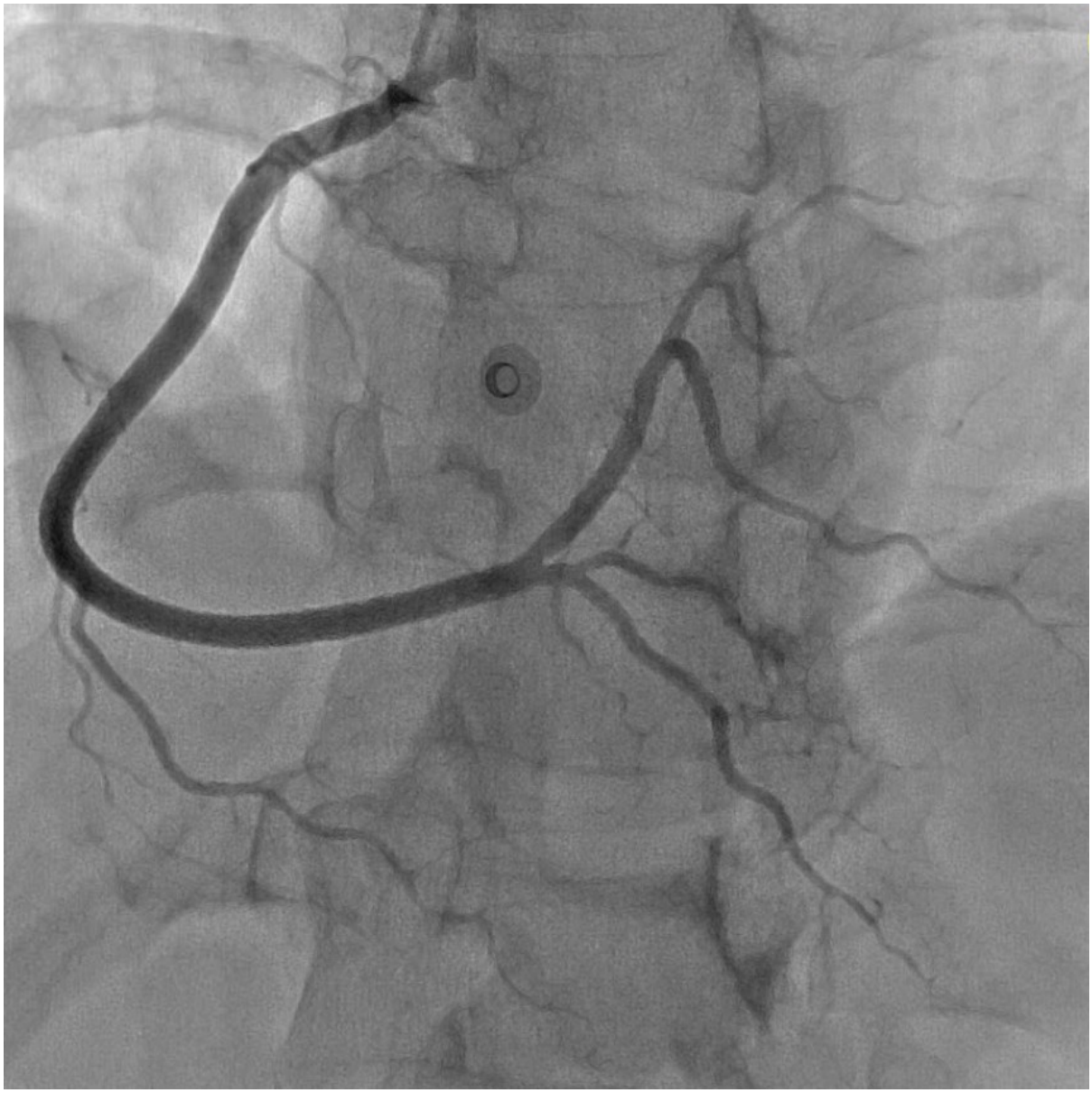


Figure 4. Follow-up abdominal Ultrasonography after 3 days: non-enlarged gallbladder with hyperechoic wall up to 5.5 mm wide, no deposits.

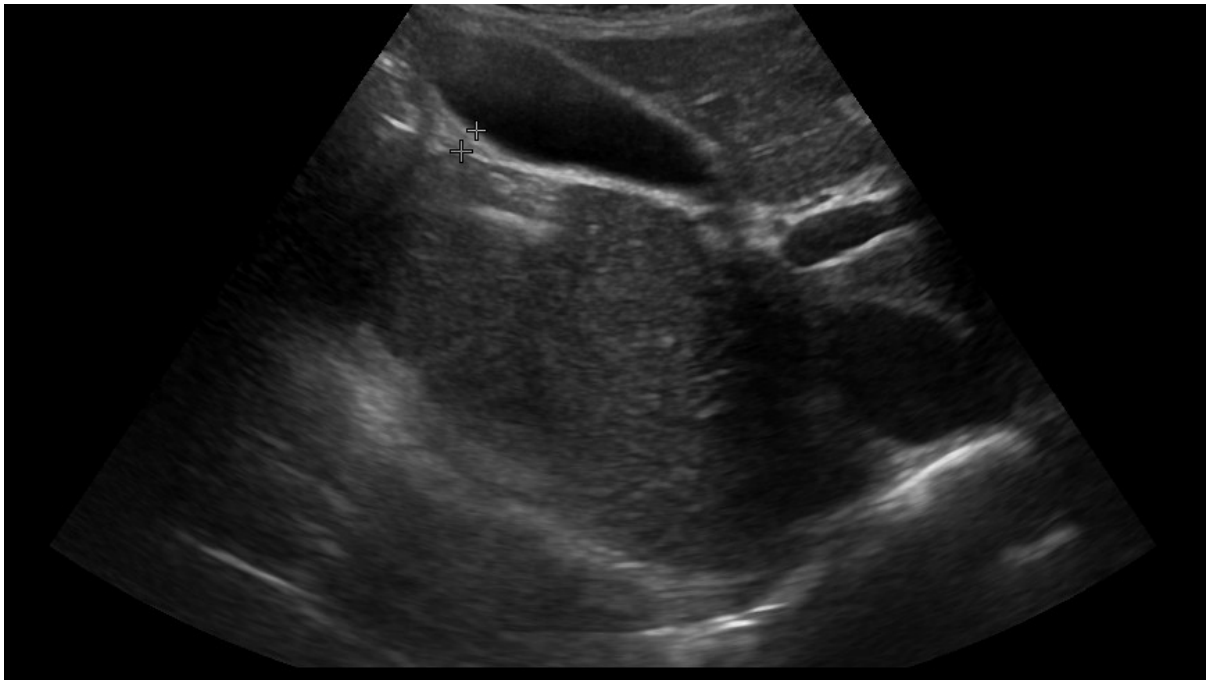
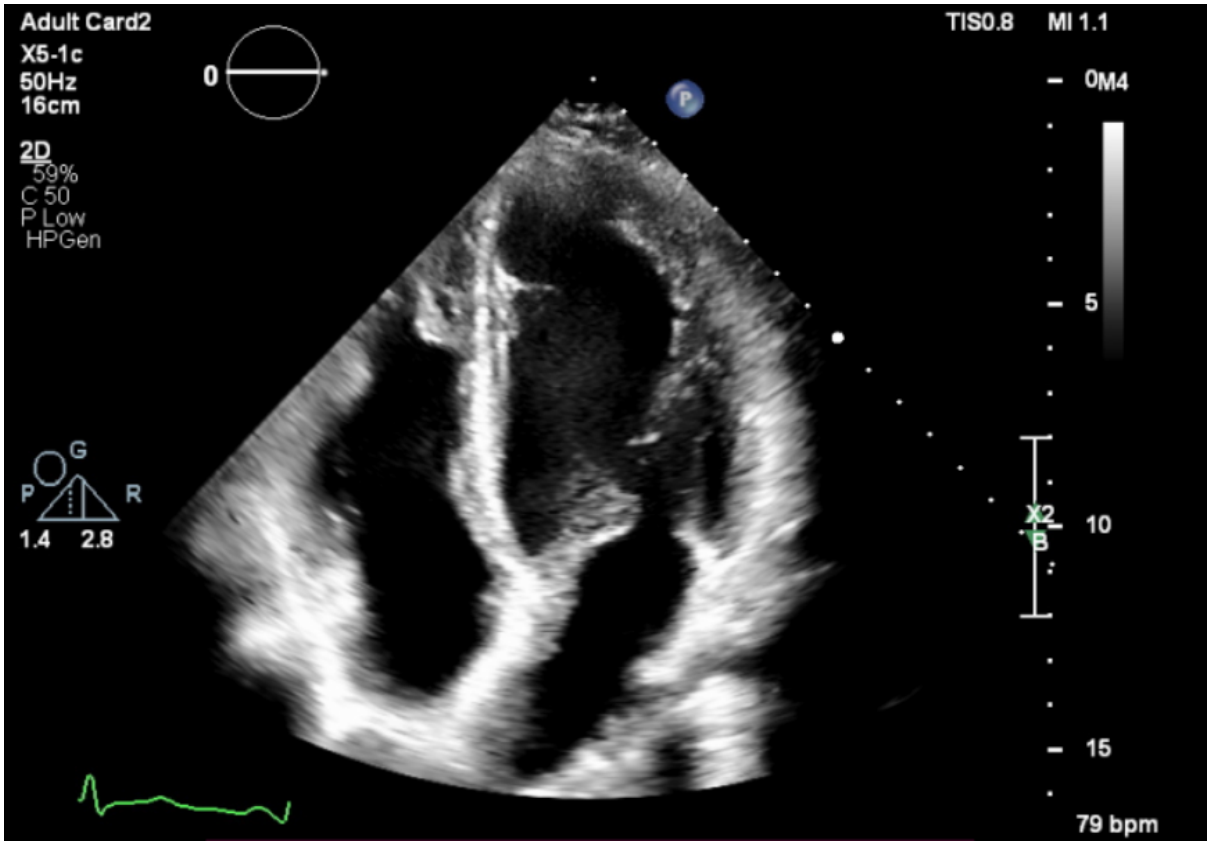


Figure 5. Follow-up echocardiography: Apical four-chamber view in end-diastolic (Panel A) and end-systolic phases (Panel B).

Panel A: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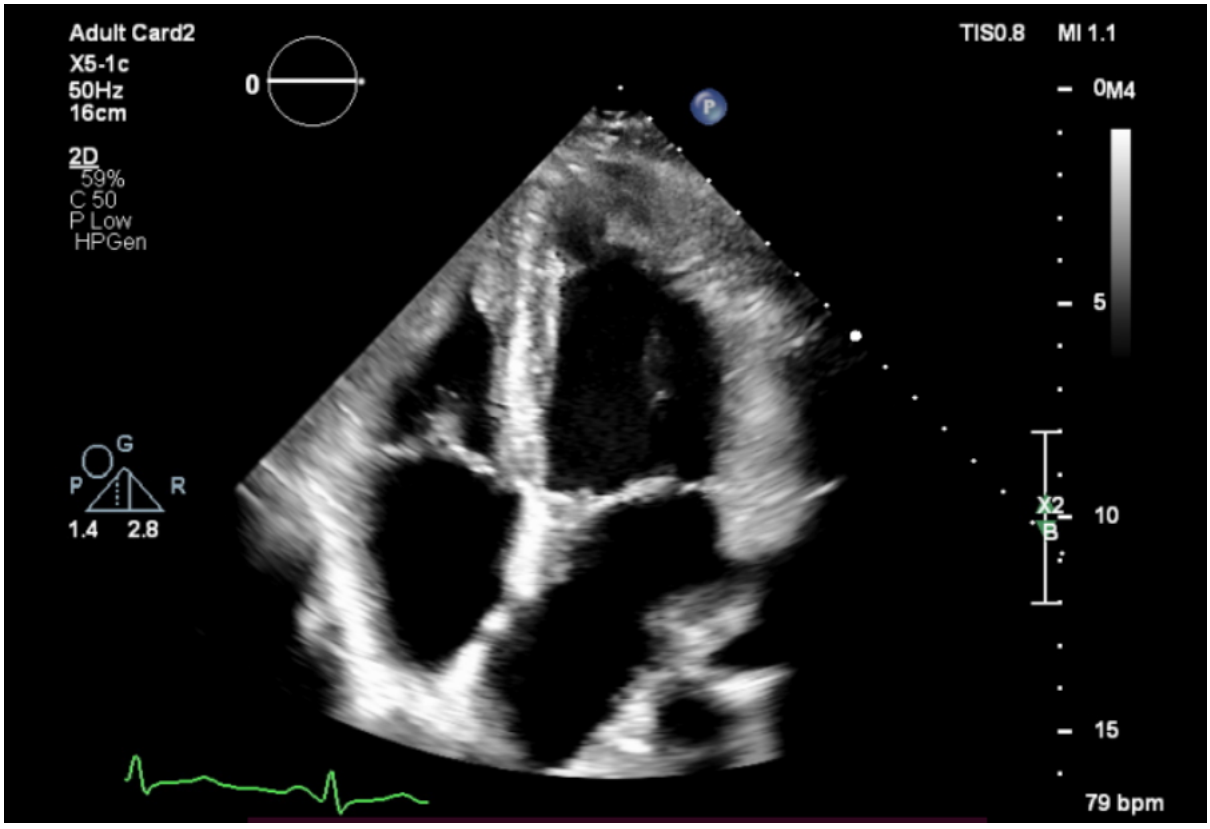
50Hz
16cm
2D
59%
C 50
P Low
HPGen
TIS0.8
MI 1.1
0M4
5
10
15
79 bpm
G
P
R
1.4
2.8
X2

Panel B: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X5-1c
50Hz
16cm
2D
59%
C 50
P Low
HPGen
TIS0.8
MI 1.1
0M4
5
10
15
79 bpm
G
P
R
1.4
2.8
X2