A Case of Ischemia with Non-Obstructive Coronary Arteries (INOCA) in a non-smoker 50-Year-Old Female: Diagnostic Challenges and Management Strategies

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Key clinical message

Myocardial infarction/Ischemia with non-obstructive coronary arteries (MINOCA/INOCA) is a form of acute myocardial infarction (MI) without significant coronary artery blockage. Accounting in up to 15% of all MI cases, INOCA disproportionately affects women.

INOCA requires thorough diagnostic evaluation, including advanced multimodality imaging, to identify underlying mechanisms and guide treatment.

It is important to highlight that current expert recommendations for INOCA management are not fully aligned, with conflicting opinions and differing interpretations.

1 | INTRODUCTION

For many years, myocardial infarction (MI) has been observed in patients without significant obstructive coronary artery disease (CAD) or an identifiable culprit artery, until 2013 when this condition was formally termed "myocardial infarction/ischemia with non-obstructive coronary arteries – MINOCA/INOCA" [1,2], followed by the paper of European Society of Cardiology (ESC) in 2017 when was published a paper on MINOCA introducing diagnostic criteria based on the third universal definition of MI as follows:

- the presence of positive cardiac biomarker with clinical evidence of infarction,
- absence of stenosis ([?]50%) in any epicardial coronary arteries on coronary angiography, and
- lack of any alternative diagnosis for the index presentation [3].

However, in recent times there has been a tendency to use the term INOCA, which means ischemia but without the presence of myocardial infarction, therefore in the following text we will stick to this term even though the scientific papers that will be cited have the same MINOCA etiology and pathology.

MINOCA is recognized as a heterogeneous working diagnosis, with an estimated prevalence ranging from 3% to 15% (6% to 8% according to ESC) among all acute myocardial infarction (AMI) patients [4,5]. This variability is partly due to differences in the conditions classified as MINOCA and the definitions applied. A pooled analysis of 23 studies found that the prevalence of MINOCA was 8.1% among 806,851 consecutive AMI patients [6]. National registries from countries such as the US, Japan, Poland, and Sweden report MINOCA incidences between 2.9% and 10.2% [5, 7-9]. Compared to myocardial infarction with obstructive coronary artery disease (CAD), MINOCA patients tend to be younger, with a median age of around 61 years, and are more frequently identified in Black and Hispanic populations [5, 10, 11]. Additionally, MINOCA patients are less likely to exhibit traditional cardiovascular risk factors, including hypertension, dyslipidemia, diabetes, and current smoking history [6].

MINOCA disproportionately affects women, with recent meta-analyses indicating that females account for up to 50% of MINOCA patients [12]. In the VIRGO study (Variation in Recovery: Role of Gender on Outcomes of Young AMI Patients), young women with acute myocardial infarction (AMI) had approximately five times the odds of experiencing MINOCA compared to men (14.9% vs. 3.5%, OR: 4.84, 95% CI: 3.29–7.13) [11]. Among the 269 women with MINOCA in the study, the majority (75%) had an undefined cause, while 4% were attributed to spasm, 21% to dissection, and 1% to embolization [11]. An analysis of MINOCA patients from the ACTION Registry-GWTG (Acute Coronary Treatment and Intervention Outcomes Network Registry-Get with the Guidelines) revealed a higher incidence of in-hospital major adverse cardiovascular events (MACE)—a composite of death, reinfarction, cardiogenic shock, or heart failure in females compared to males (5.4% vs. 4.1%; $p < 0.0001$) [13].

At the time of angiography, INOCA is regarded as a provisional diagnosis, pending the exclusion of other causes of myocardial injury and identification of the underlying ischemic mechanisms [14].

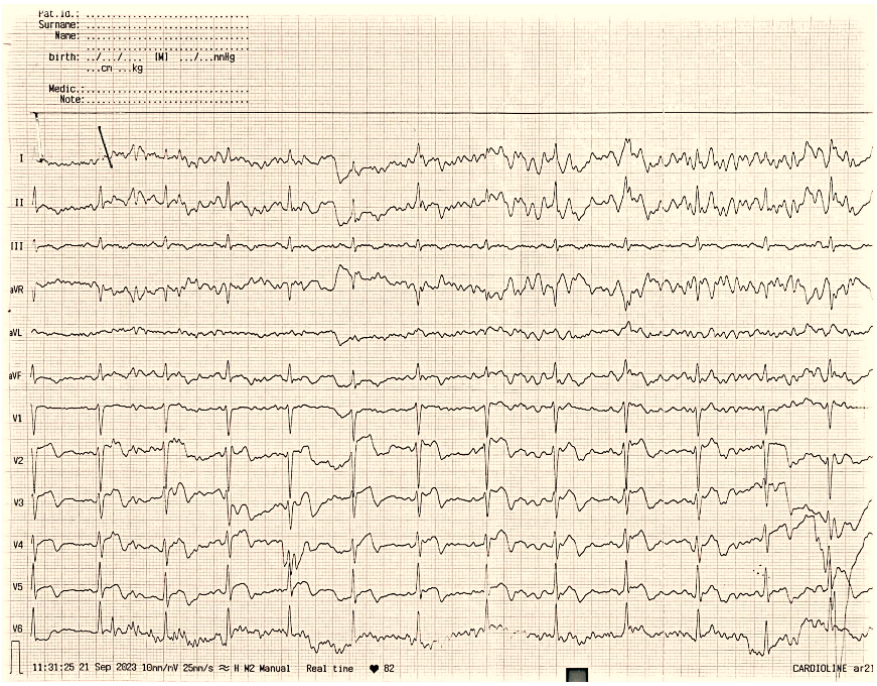
The initial diagnostic investigations for identifying the causes of INOCA include clinical history, electrocardiogram (ECG), cardiac enzymes, echocardiography, coronary angiography, and left ventricular (LV) angiography [2]. Notably, regional wall motion abnormalities observed during LV angiography can indicate an **"epicardial pattern"** when limited to a single epicardial coronary artery territory which includes: plaque rupture or erosion, coronary dissection and coronary artery spasm; or a **"microvascular pattern"** when they extend beyond this territory including: Takotsubo cardiomyopathy, myocarditis, coronary thromboembolism, other forms of type 2 MI, and INOCA of uncertain etiology [15].

In 2018, the universal definition of myocardial infarction (MI) was revised to encompass only ischemic mechanisms linked to myocardial injury, thereby excluding non-ischemic conditions such as Takotsubo cardiomyopathy, myocarditis, and non-ischemic cardiomyopathy [14]. Consequently, the American Heart Association (AHA) scientific statement in 2019 refined the definition of MINOCA by excluding these non-ischemic mechanisms, categorizing them as MINOCA mimickers [16].

INOCA patients present a clinical challenge due to the wide range of possible etiologies and pathogenic mechanisms linked to the condition. Therefore, selecting the appropriate treatment often necessitate further diagnostic evaluation.

2 | CASE PRESENTATION

2.1 | Case history and examination



A 50-y/o female patient, a non-smoker, presented to the primary care health institution with chest pain described as a tightening sensation and pressure lasting 30 minutes. The ECG revealed ST segment elevations in leads V2-V5 (presented in figure no.1), and her troponin level was significantly elevated at 5400 ng/L initially. She received treatment in accordance with myocardial infarction protocols and was subsequently referred to the Cardiology Clinic for further investigation and treatment. Her past medical history includes hypertension, diagnosed 2 years prior, and a right leg fracture 2 months ago. Family history is non-contributory, and she is currently taking Enalapril 5 mg twice daily, with no regular measurements and follow-up by the family doctor.

2.2 | Diagnosis, investigation and treatment

From the laboratory findings: Troponin: 14375 ng/L, CRP: 2.8, Na^+: 137 mmol/l, K^+: 4.4 mmol/l, Urea: 5.9 mmol/l, Creatinine: 65 µmol/l, Fasting Plasma Glucose (FPG): 5.6 mmol/l, Hgb: 131 g/l, RBC: 4.2 x10ˆ12 cells/l, WBC: 10.5 x10ˆ9 cells/l, PLT: 223 x10ˆ9 cells/l, D-dimer: 326 ng/ml. These results indicate significant elevation in troponin levels, consistent with myocardial injury, with a normal range of the blood count, electrolyte status, degradation products, inflammation marker, fasting plasma glucose and D-dimer, which in this case could indicate one of the theories of the pathophysiological causes of INOCA in the scope of microvascular pattern, respectively coronary thromboembolism, which in this case is also reasonable to suspect due to the fracture that the patient had two months before this event.

Because of the complexity of the patient and situation, in the beginning Top of Form

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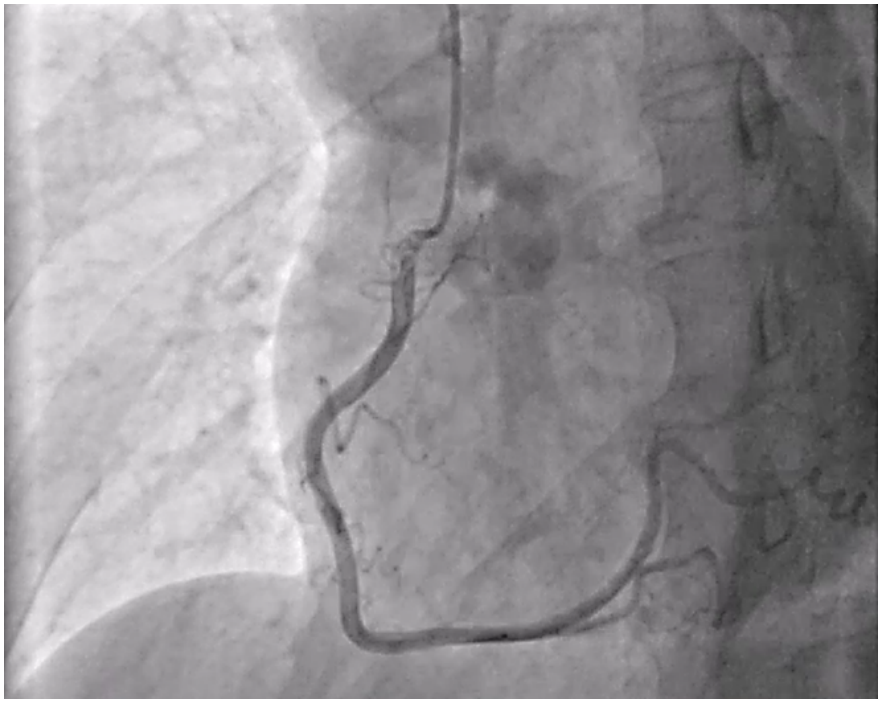
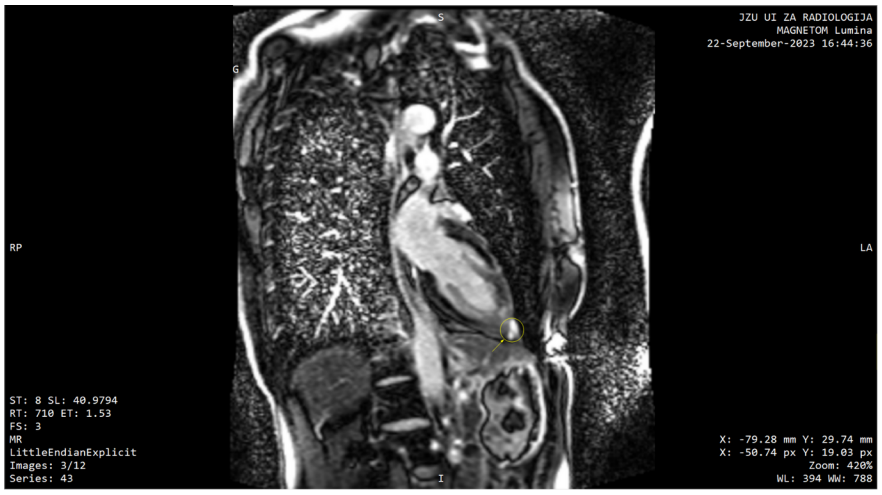
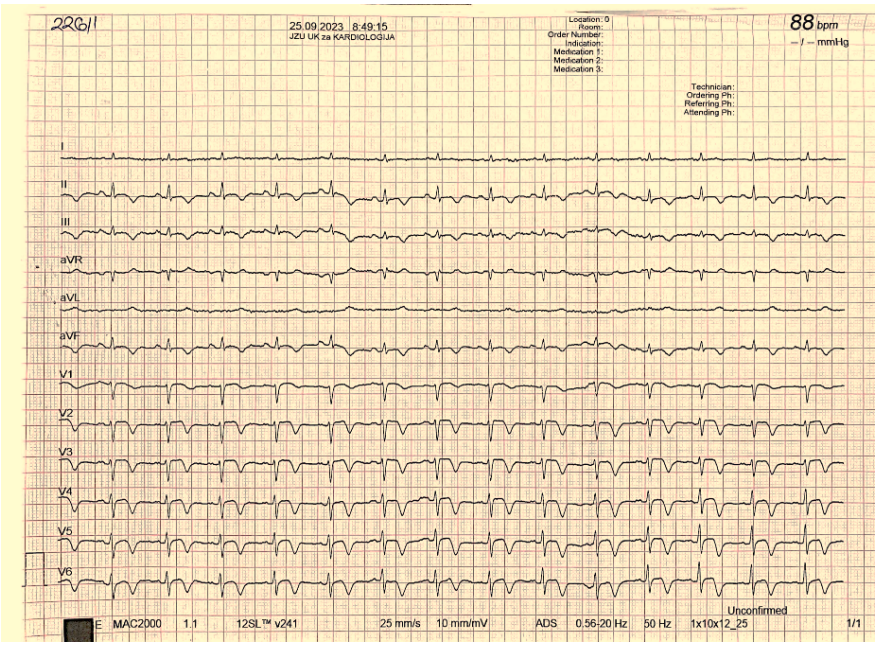


coronary angiography was performed, revealing no significant stenosis and normal findings were reported (figure no.2), and after that the next day an echocardiogram was indicated showing an impaired global longitudinal deformation with a GLS -15% (ref values >-20%) of the left ventricle involving the entire myocardium, except the basal segments of the anterior and inferior walls of the left ventricle with an impression of dyskinetic motion at the apex of the left ventricle, necessitating further investigation, with a normal systolic function (EF 63%).



According to ESC guidelines for diagnosis of INOCA a cardiac MRI was performed with early post-contrast series shows transmural enhancement of signal in the apical segment, indicating a necrotic ischemic zone. A delayed post-contrast series reveals clear enhancement along the apical inferior wall and towards the mid septal anteroseptal and inferoseptal walls, as well as significant enhancement of the aneurysmal dilated apex,

predominantly due to fibrotic changes, changes which correspond to ischemic lesions, excluding myocarditis and Takotsubo syndrome and confirming the INOCA (figure no.3).



In cases of diagnostic uncertainty like in our case, advanced intracoronary imaging modalities such as optical coherence tomography (OCT), intravascular ultrasound (IVUS) and fractional flow reserve (FFR) may be considered but these techniques were not available at the time to perform. However, these should be used cautiously due to the potential risk of propagating coronary dissection.

Regarding our case, it was decided to discharge the patient with the following therapy due to the extensive areas of ischemia guided by the findings in MRI and ECG changes before admission and discharge (figure no.4): **Tbl.Acetyl-salicylic acid a 100mg 1x1, Tbl.Clopidogrel a 75mg 1x1, Tbl.Rosuvastatin a 40mg 1x1, Tbl.Zofenopril 7,5mg 2x1, Tbl.Pantoprazole a 40mg 1x1.**

2.3 | Outcome and follow-up

Following the administration of the therapy recommended by the expert opinion and studies conducted in this domain, patient showed significant improvement clinically, but not only, the high sensitive troponin which was 14375 ng/l in the admission, four days after we noticed a significant decrease before discharge from the hospital, 2720.7 ng/L, and all other laboratory findings in the normal range. The patient was discharged hemodynamically and rhythmically stable with a recommendation of hygiene-dietetic regimen and follow-up in our clinic after one month. In the first consultation after a month patient showed no complication or subjective complaints. After six months tbl. Clopidogrel was discontinued and the patient has a control echocardiography and magnetic resonance of the heart in November.

3 | DISCUSSION

Patients with an identified underlying cause of INOCA benefit from targeted, cause-specific treatment. Additionally, secondary atherothrombotic prevention measures should be considered based on the specific etiology [14]. No randomized clinical trials have been published for the treatment of INOCA, so management should be individualized and tailored to the underlying cause of each case, but there are few randomized controlled trials and observational studies have reported lower mortality in MINOCA patients treated with ACE inhibitors/ARBs and statins. One such study, involving 9,138 patients from the SWEDHEART registry,

demonstrated improved outcomes with these treatments, analyzes the positive effect of treatment with beta-blockers and the neutral effect of dual antiplatelet therapy [17, 18].

MINOCA-BAT is a study involving 3500 patients investigating the effects of beta-blockers and ACE/ARB compared to placebo. It is being conducted to assess mortality, recurrent myocardial infarction, stroke, or heart failure, with the study expected to conclude in 2025 [19]. The PROMISE trial will recruit 180 patients diagnosed with MINOCA and randomly assign them into two groups. One group will undergo a precision medicine approach that includes coronary optical coherence tomography (OCT), cardiac magnetic resonance imaging (CMRI), and coronary spasm testing to guide tailored medical therapy. The other group will follow the standard approach to managing acute coronary syndrome. This study aims to evaluate the effectiveness of personalized diagnostics and treatment strategies compared to conventional care in INOCA patients.

It is important to highlight that current expert recommendations for INOCA management are not fully aligned, with conflicting opinions and differing interpretations. For example, the European Society of Cardiology (ESC) guidelines for managing non-ST segment elevation acute coronary syndromes recommend using conventional secondary prevention medications for INOCA, similar to myocardial infarction with obstructive coronary artery disease (MI-CAD), when the underlying cause is unclear [20].

The ESC also advises routine use of aspirin, statins, and calcium channel blockers (CCBs) for vasospasm [16]. In contrast, the American Heart Association (AHA) suggests that statins and antiplatelet therapy should only be used in INOCA cases involving plaque disruption and avoided in type 2 MI, where they may be contraindicated [14].

Table 1. Therapeutic target based on the etiology of INOCA (adopted from Samaras et al. [21])

MINOCA/INOCA	ETIOLOGY	TREATMENT
	Plaque Disruption	ASA, Clopidogrel or Ticagrelor, Statins, RAASi,
	Epicardial Coronary Vasospasm	Isosorbid dinitrate, CCBs (low-dose ASA, statins
	Coronary Microvascular Dysfunction	β-blockers, CCBs, (RAASi, statins, nitrates)
	Spontaneous Coronary Artery Dissection	Conservative therapy (β-blockers, statins, RAASi
	Coronary Artery Embolism	PFO closure, OAC

ASA—acetylsalicylic acid; RAASi—renin–angiotensin adrenergic antagonists; CCBs—calcium channel blockers; OAC—oral anticoagulation; PFO—patent foramen ovale

Currently, targeted therapies for INOCA based on specific pathophysiological mechanisms have not been sufficiently studied and in the table below we will list the optimal therapeutic treatment based on the etiology of INOCA.

It remains uncertain whether conventional secondary preventive therapies are effective in reducing post-infarct angina in INOCA patients. An individualized approach should be adopted based on the underlying cause (etiology) of its development.

4 | CONCLUSION

INOCA is a syndrome that predominantly affects women and presents as a heterogeneous working diagnosis, often understudied, underdiagnosed, and undertreated. Despite having a prognosis similar to myocardial infarction with obstructive coronary artery disease, INOCA requires thorough diagnostic evaluation, including advanced multimodality imaging, to identify underlying mechanisms and guide treatment. While randomized clinical trials on secondary prevention are ongoing, further research is necessary to establish targeted treatments based on specific pathophysiological mechanisms.

FUNDING INFORMATION

None.

CONFLICT OF INTEREST STATEMENT

None.

DATA AVAILABILITY STATEMENT

The data are not publicly available due to privacy or ethical restrictions

CONSENT

Written informed consent was obtained from patient to publish this report in accordance with the journal's patient consent policy.

AUTHOR CONTRIBUTIONS:

Antonio Georgiev: Conceptualization, Formal analysis, Investigation, Methodology, Supervision, Visualization, Writing - original draft, Writing - review & editing.

Alsada Abazi: Investigation

Vegim Zhaku: Conceptualization, Formal analysis, Methodology, Supervision, Visualization, Writing - original draft.

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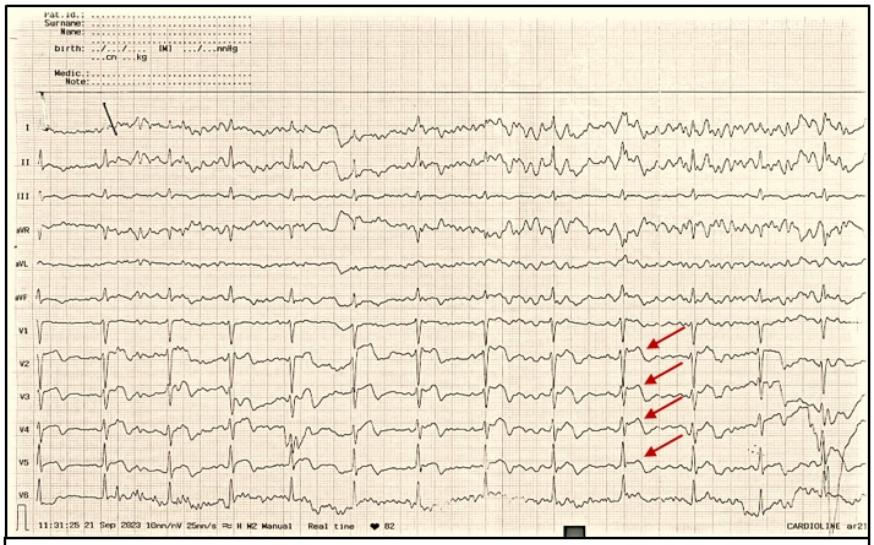

Figure no.1 ECG at admission. ST segment elevation V2-V5.

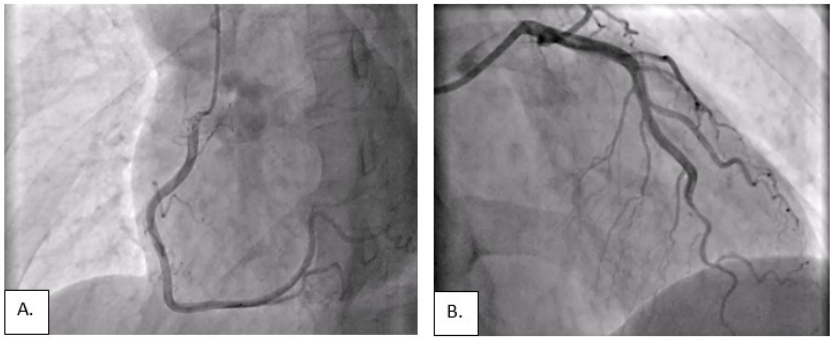

Figure no.2. Normal findings in coronary angiography. **A:** RCA (right coronary artery); **B:** LM (left main), LAD (left anterior descendent), Cx (circumflex) and Dg (diagonal)

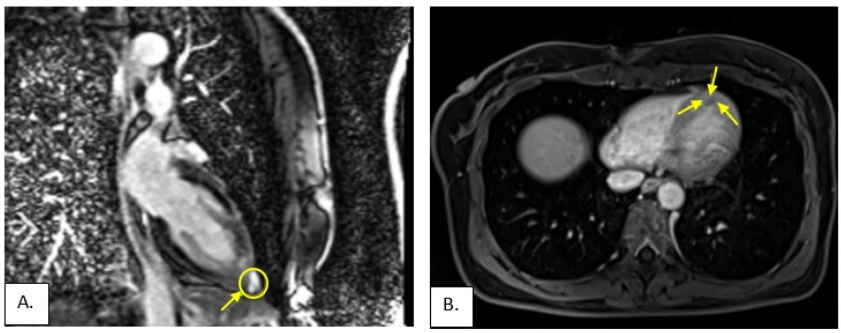

Figure no.3. Cardiac MRI showing **(A)** transmural enhancement of signal in the apical segment, indicating a necrotic ischemic zone and **(B)** an aneurysmal formation in the apex.

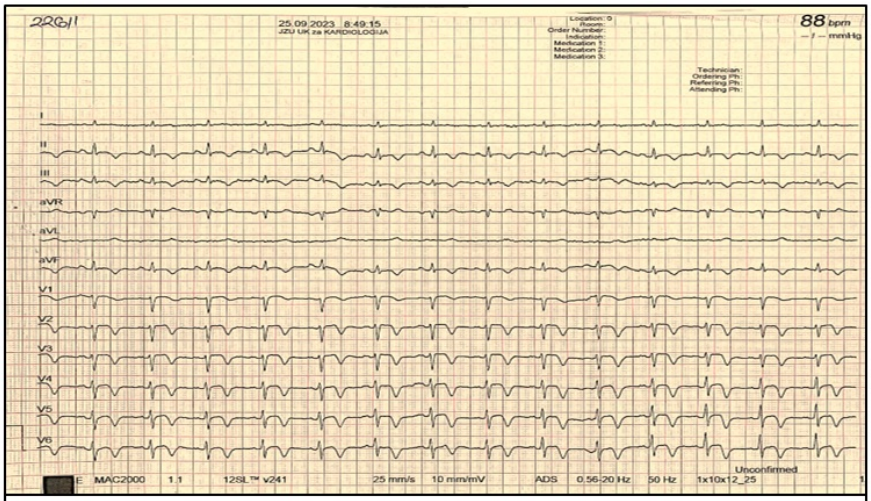

Figure no.4 ECG before discharge. Persistent concave ST segment elevation in the precordial leads, inversion of T waves in inferior and precordial leads.

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Table.1 (in editable version).docx available at https://authorea.com/users/840726/articles/1230902-a-case-of-ischemia-with-non-obstructive-coronary-arteries-inoca-in-a-non-smoker-50-year-old-female-diagnostic-challenges-and-management-strategies