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Myocardial infarction with non-obstructive coronary arteries (MINOCA) in septic patient. Case report

[1]Guliko Kiliptari, [2]Murman Kantaria, [3]Lali patsia

1.Head of critical care department of university clinis after Kipshidze, Professor of state medical university
2. Head of cardiology department of university clinis after Kipshidze, MD, PHD
3.Cardiologist, MD, PHD

Abstract: We report a case of myocardial infarction with non –obstructive coronary arteries (MINOCA), in a critically ill patient with cardiac ventricular dysfunction, hemodynamic instability and multiorgan involvement. Despite nonobstructive coronary arteries, the patient exhibited marked troponin elevation, regional wall motion abnormalities and delayed coronary flow. The patient's condition progressed rapidly, peading to refractory shock and cardiac arrest . this case highlights the diagnostic complexity and high mortality risk of MINOCA in severe settings

Conclusion: Sepsis induced cardiac, cardiomyocyte disfunction is generated from inflammatory involvement of the heart as a part of systemic infectious process. Different pathogenic pathway, including mitochondrial injury, microvascular thrombosis, complement system activation, endothelial dysfunction, occurrence of an arrhythmia, linking sepsis to myocardial disfunction and cardiogenic shock.

MINOCA is a complex and heterogeneous syndrome requiring careful diagnostic evaluation, mechanism-based management, close monitoring. This case highlights the importance of advanced imaging, vigilant ICU care and comprehensive therapy in critically ill patient with non-obstructive MI

1. INTRODUCTION

Myocardial infarction with non-obstructive corinary arteries(MINOCA) is a heterogeneous and clinically significant entity characterized by evidence of myocardial infarction without obstructive coronary artery disease. Despite the absence of significant stenosis, MINOCA can result in substantial morbidity and mortality, particularly in patient with hemodynamic instability, sepsis and multiorgan involvement.

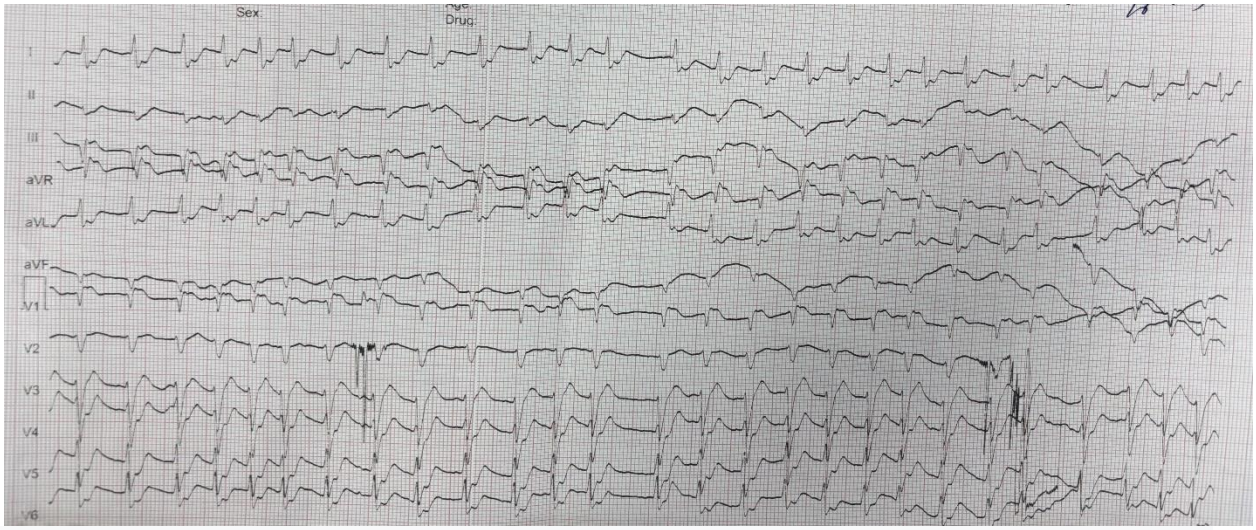
MINOCA encompasses multiple underlying mechanisms, including epicardial coronary injury, oxygen supply-demand mismatch, and non-coronary cardiac causes. The sepsis induced myocardial dysfunction pathway is mitochondrial injury and cardiomyocyte dysfunction, with inflammatory mediator production and microvascular thrombosis Recognition and mechanism -based management are critical, especially in patients with hemodynamic instability and multiorgan involvement

Case presentation

We present one case of myocardial infarction without coronary arteries occlusion. Patient. male, 70 years old, was admitted in hospital by ambulance., was admitted with severe chest pain, sincope, chills, dyspnea. past medical history -- hypertension, paroxysmal atrial fibrillation. Hyprglycemia on admission, arterial pressure 50/20 mm.hg, SaO2 80 % , RR -38 , patient was intubated and started mechanical ventilation . Hemodynamic instability required escalating norepinephrine and epinephrine infusions.

Stabilization was Initiated with pressors infusion (Sol. Noradrenalin k200 0.5mcg/kg/min, Sol. Adrenaline k100 0.3mcg/kg/min)

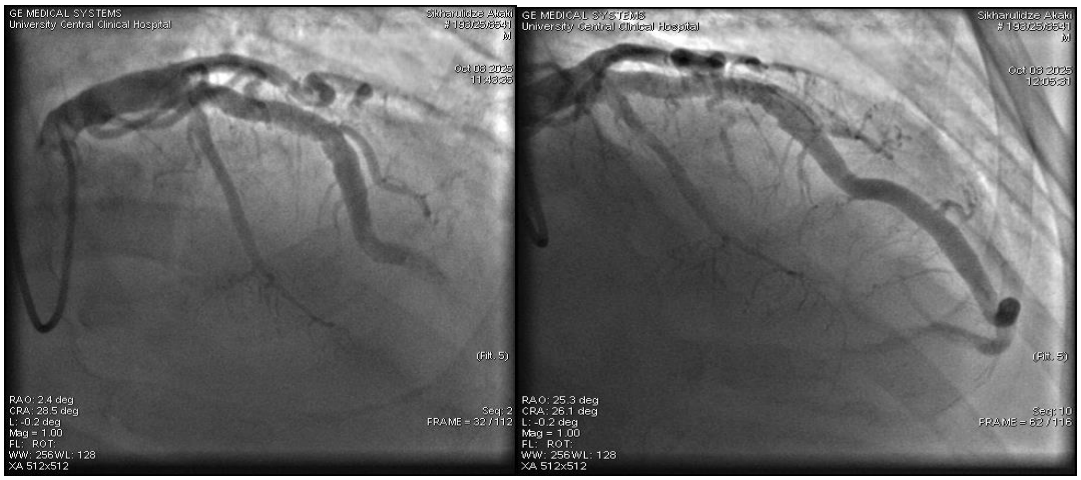
ECG demonstrated atrial fibrillation, ST segment elevation in inferior leads II, III, aVF, aVR, with reciprocal depressions in precordal leads, initially suggesting an inferior STEMI.

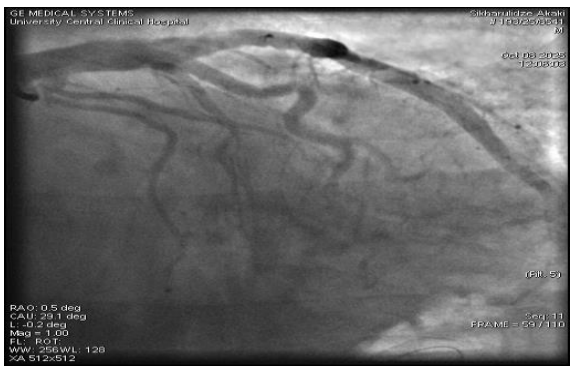


1. Echocardioskopy findings -- EF 50%, Comparative hypokinesia of the left ventricular inferior wall, Right ventricular free wall akinesia,LA 42 mm, LVDD 50 mm, RA 50 mm, RV 50 mm, AO.asc 32 mm, IVS 14 mm, PW 12 mm Mild mitral regurgitation, Moderate tricuspid regurgitation

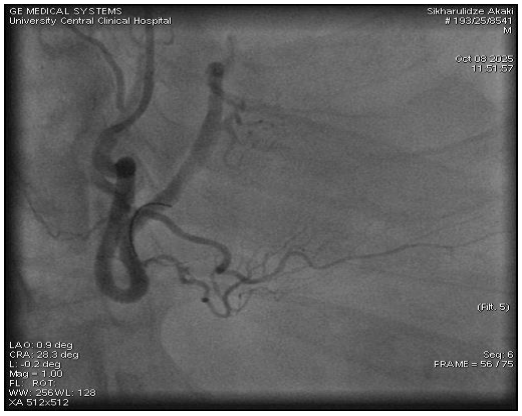
Gas analisis --Lactat acidosis, pH-7.1 BE (B) – (-)10.5 mmol/l, BE (ecf)- (-) 10.2 mmo/l, HCO3-18.40 mmol/l,Lac – 8.24 mmol/l ,Glu -202 mg/dl. Troponin I level -- - <0.100 ng/ml

Coronary angography detected: The left coronary artery—the main artery and anterior descending artery—is dilated, large-caliber, and fills with a slow, turbulent flow, with complete penetration of the contrast agent. However, the distal segment of the anterior descending artery completely fills after 8–9 cardiac cycles, which, under stress conditions, will contribute to the development of secondary ischemia. The distal segment of the right coronary artery fills with a slow flow. The descending artery is dilated (up to 10 mm in the proximal segment), extending beyond the apex and posteriorly to the interventricular groove, without significant narrowing. The circumflex and intermediate arteries also show no significant narrowing.



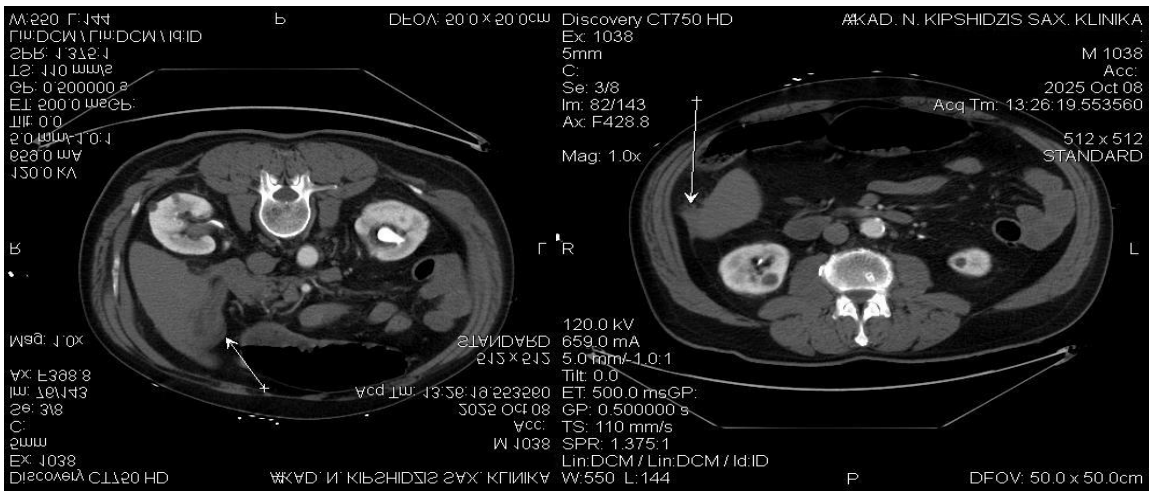


The distal segment of the right coronary artery fills with a slow flow. Right coronary artery is dilated, no obstructive lesions

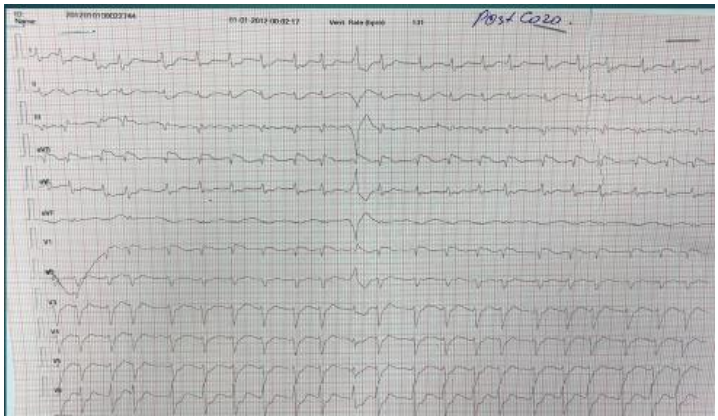


Considered possibilities included: Pulmonary embolism, myocarditis,takotsubo cardiomyopathy.
CT scan pulmonary angiography reveled infiltrative foci with consolidation in dorsal segments of both lower lobes, bilateral pleural effusion, no free fluid or air. Pulmonary trunk, main, lobar, segmental, subsegmental arteries fully opacified, no thromotic masses. Thoracic aorta normal in calibre.

Abdominal and pelvic CTA, Gallbladder enlarged with wall thickening, infiltrative changes with chronic cholecystitis, diverticula in the sigmoid colon wall, no other acute intraabdominal or pelvic pathology. The abdominal aorta and its branches are filled with a contrast agent; thrombotic formations in the lumen are not visible. The pancreas is visualized along its entire length; its structure is lobulated, with no dilation of the duct. Both kidneys are of normal shape, size, and anatomical location. The renal parenchyma is saturated with contrast. Several cystic inclusions are visualized on both sides, Bilateral dilation of the renal pelvis is not detected



Brain CT- Bilateral subcortical and frontal lobe ischemic foci of varying age at the level of lateral ventricles and semioval centers, reduced white matter density, periventricular encephalopathy present suggestive of chronic cerebrovascular disease.

ECG—Without any changes. 

Echocardioskopy findings: LA a.42 (N<38) mm, m.l. (N<38), mm, s.i. mm; LVIDD **50** (N<56) mm, LVISDb (N<40)mm;RA m.l.50 (N<38) mm, s.i. 57 (N<46)mm; RV **50** (N<36)mm; AO **32** (N 20–37)mm, As
AO (N 20–35)mm;IVS —14 (N 6–11) mm, LVPW12 mm; EF **22** % (N - 55%);
MV. Reg.+1 , MaxPG. (N <7), mmHg; MeanPG. (N<5); AV. Reg.+ MaxPG (N<10) mmHg, MeanPG mmHg, TV. Reg. +3 , IVC 32 (N12–23)mm;PASP mmHg; kolab. 50 (N>50) %;

Marked dilation of the right ventricle, sclerosis of the aortic and carotid artery walls. Hypertrophy of the interventricular septum, diffuse critically impaired global contractile function of the left ventricle, ejection fraction of 22%, complete akinesia of the right ventricular free wall, moderate mitral and severe tricuspid regurgitation, in dynamics, echo data have deteriorated sharply, shock has deepened, pressor infusion rate has increased. Laboratory findings -- WbC-19.71 10^3 / µL ALT-114 U/L AST-153U/L GGT-88U/L ,PCT-9.38 ng/ml ,Troponin I – 9.92 ng/ml .

The treatment aimed to stabilize hemodynamics, support organ perfusion, antimicrobial ------ and address potential causes of MINOCA. Initial troponin I was <0.100ng/ml, lactic acidosis was present, consistent with tissue hypoperfuson, follow-up troponin I-9.92 ng/ml, indicating significant myocardial injury. Furthermore, procalcitonin level increased sharply, reaching >9 ng/ml, along with leukocytosis and a left shift in the white blood cell count. Bilateral pneumonia and refractory shock (history of viral infection) represent the epitome of sepsis and septic shock.

The case illustrates MINOCA in a critically ill patient with sepsis, ventricular disfuncton, delayed coronary flow and multiorgan involvment. The presence of hemodynamic instability, high troponin and echocardiographic wall motion abnormalities underscores the severity.

Coronary ectasia with turbulent and delyed distal flow likely contributed to myocardial ischemia in the absence of obstructive lesions. Differential diagnosis including pulmonary embolism, myocarditis and Takotsubo cardiomyopathy were systematically excluded.

2. CONCLUSION

Sepsis induced cardiac, cardiomyocyte disfunction is generated from inflammatory involvement of the heart as a part of systemic infectious process. Different pathogenic pathway, including mitochondrial injury,microvascular thrombosis ,complement system activation , endothelial dysfunction ,occurrence of a arrhythmia, linking sepsis to myocardial disfunction and cardiogenic shock .

MINOCA is a complex and heterogeneous syndrome requiring careful diagnostic evaluation, mechanism-based management, close monitoring. This case highlights the importance of advanced imaging,vigilant ICU care and comprehensive therapy in critically ill patient with non-obstructive MI.

REFERENCES

1. Current challenges in understanding, diagnosing and managing sepsis-induced cardiac dysfunction. lCatalina Paraschiv [a b], Mihaela Roxana Popescu Moraru [a b], Livia Florentina Paduraru [a b], Claudia Andreea Palcau [a b], Andreea Catarina Popescu [a b], Serban Mihai Balanescu [a b] **J Crit Care** 2026 Feb:91:155250. doi: 10.1016/j.jcrc.2025.155250. **Epub 2025 Sep 13.**
2. Cardiac dysfunction in severe sepsis and septic shock.

3. Zanotti-Cavazzoni SL, Hollenberg SM.Curr Opin Crit Care. 2009 Oct;15(5):392-7. doi: 10.1097/MCC.0b013e3283307a4e.PMID: 19633546Review Cardiac dysfunction and circulating cardiac markers during sepsis.Latini R, Caironi P, Masson S.Minerva Anestesiol. 2016 Jun;82(6):697-710. Epub 2016 Jan 21. PMID: 26795362
4. Laboratory and Microbiological Considerations in Sepsis-Induced Cardiac Dysfunction. Paraschiv C, Nicolaescu DO, Popescu MR, Vasile CC, Moisa E, Negoita SI, Balanescu SM.Medicina (Kaunas). 2025 Sep 30;61(10):1765. doi: 10.3390/medicina61101765.
5. Martin, L.; Derwall, M.; Al Zoubi, S.; Zechendorf, E.; Reuter, D.A.; Thiemermann, C.; Schuerholz, T. The Septic Heart: Current. Understanding of Molecular Mechanisms and Clinical Implications. Chest **2019**, 155, 427–437.
6. Troponin elevation in severe sepsis and septic shock: the role of left ventricular diastolic dysfunction and right ventricular dilatation*,Giora Landesberg [1], Allan S Jaffe, Dan Gilon, Phillip D Levin, **Crit Care Med ,2014** Apr;42(4):790-80
7. Myocardial Infarction With Nonobstructive Coronary Arteries (MINOCA): The Past, Present, and Future Management, Sivabaskari Pasupathy, BSc(Hons) ,PhD, Rosanna Tavella, BSc(Hons), PhD, and John F. Beltrame, BSc, BMBS, PhDAUTHOR INFO & AFFILIATIONS, Circulation, Volume 135, Number 16,2017
8. Systematic review of patients presenting with suspected myocardial infarction and nonobstructive coronary arteries. *Circulation*. 2015; *131:861*–870. doi: 10.1161/**CIRCULATIO**N, AHA.114.011201. Pasupathy S, Air T, Dreyer RP, Tavella R, Beltrame JF,*Circulation.* 2015;*131*:861–870. doi:
9. Medical therapy for secondary prevention and long-term outcome in patients with myocardial infarction with non-obstructive coronary artery disease. Lindahl B, Baron T, Erlinge D, Hadziosmanovic N, ***Circulation.*** **2017**; *135:1481*–1489. doi:,
10. Pathophysiology, diagnosis and management of MINOCA: an update, G. Scalone , G. Niccoli , F. Crea, European Heart Journal - **Acute CardioVascular Care** , Volume 8 , Issue 1 , Pages 54 - 62