

MedPeer Publisher

Abbreviated Key Title: MedPeer

ISSN : 3066-2737

homepage: <https://www.medpeerpublishers.com>

MINOCA REVEALED BY RENAL INFARCTION ASSOCIATED WITH LEFT VENTRICULAR THROMBUS IN A YOUNG PATIENT: A CASE REPORT AND LITERATURE REVIEW.

DOI: 10.70780/medpeer.000QGWO

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ABSTRACT

Myocardial Infarction with Non-Obstructive Coronary Arteries (MINOCA) is a heterogeneous clinical entity characterized by evidence of myocardial infarction in the absence of significant obstructive coronary artery disease. Its diagnosis remains challenging, particularly in young patients presenting with atypical extracardiac manifestations. We report the case of a 33-year-old man with chronic tobacco use and occasional cannabis consumption who was initially admitted for right flank pain in a pseudo-infectious setting. Abdominal computed tomography revealed a hypodense lesion of the lower pole of the right kidney suggestive of either pyelonephritis or renal infarction. Subsequent cardiac investigations demonstrated severe dilated cardiomyopathy with major left ventricular systolic dysfunction. Cardiac magnetic resonance imaging showed extensive transmural late gadolinium enhancement in the left anterior descending artery territory associated with a 5-mm left ventricular thrombus. Coronary angiography revealed normal coronary arteries, leading to the diagnosis of MINOCA complicated by probable systemic renal embolization. This case highlights the major diagnostic value of cardiac magnetic resonance imaging in MINOCA and underlines the possibility of extracardiac manifestations revealing silent myocardial infarction.

KEYWORDS

MINOCA, renal infarction, left ventricular thrombus, cardiac magnetic resonance imaging, systemic embolism, silent myocardial infarction

MAIN ARTICLE

INTRODUCTION

Myocardial Infarction with Non-Obstructive Coronary Arteries (MINOCA) is defined by the presence of myocardial infarction according to universal diagnostic criteria in the absence of significant obstructive coronary artery disease, generally defined as coronary stenosis below 50% on angiography [1]. MINOCA accounts for approximately 5% to 15% of all myocardial infarctions in contemporary series [2]. Previously considered a relatively benign condition, MINOCA is now recognized as a clinically significant entity associated with substantial cardiovascular morbidity and mortality [2].

MINOCA does not represent a single disease but rather a syndrome encompassing multiple pathophysiological mechanisms including plaque disruption, coronary embolism, coronary vasospasm, microvascular dysfunction, and spontaneous coronary artery dissection [1,3]. Identification of the underlying mechanism is essential because it directly influences therapeutic management and prognosis.

Cardiac imaging, particularly cardiac magnetic resonance imaging (CMR), currently plays a pivotal role in the diagnostic evaluation of MINOCA [4]. CMR allows differentiation between ischemic and non-ischemic myocardial injury such as myocarditis or Takotsubo syndrome [4,5].

We report the case of MINOCA revealed by probable embolic renal infarction in a young patient presenting with severe dilated cardiomyopathy.

CASE PRESENTATION:

A 33-year-old man with no significant past medical or surgical history presented to the emergency department with right flank abdominal pain associated with nausea and several episodes of diarrhea in a clinical setting initially suggestive of acute gastroenteritis.

Cardiovascular risk factors included active tobacco smoking estimated at eight pack-years, occasional alcohol consumption, and occasional cannabis use.

The patient denied chest pain, dyspnea, palpitations, or syncope. On admission, he was hemodynamically stable with a blood pressure of 114/59 mmHg and a heart rate of 62 beats per minute. Physical examination revealed right costovertebral angle tenderness without

additional abnormalities. Cardiac auscultation was unremarkable, and there were no clinical signs of heart failure.

Initial laboratory investigations demonstrated marked inflammatory syndrome with leukocytosis of 17.86 G/L predominantly involving neutrophils and elevated C-reactive protein at 109 mg/L. High-sensitivity troponin was mildly elevated at 8 ng/L, while NT-proBNP was elevated at 345 ng/L. Renal function remained preserved.

Contrast-enhanced abdominal computed tomography demonstrated a hypodense lesion in the lower pole of the right kidney suggestive of either pyelonephritis or renal infarction.

Specialized radiologic review additionally identified morphological abnormalities of the left ventricle, prompting further cardiac evaluation.

Electrocardiography revealed sinus rhythm with QS waves in the anteroseptal-apical and high lateral leads suggestive of previous anterior myocardial necrosis.

Transthoracic echocardiography demonstrated a dilated non-hypertrophied left ventricle with severe global hypokinesia predominantly involving anterior segments. Anteroseptal akinesia associated with myocardial wall thinning was observed. Left ventricular ejection fraction was severely reduced at 28%. Left atrial dilation was also noted. No intracavitary thrombus was initially identified.

Cardiac magnetic resonance imaging revealed a dilated left ventricle with severely impaired systolic function and a left ventricular ejection fraction of 21%. Late gadolinium enhancement imaging demonstrated extensive transmural enhancement greater than 75% involving the anteroseptal, anterior, and apical segments corresponding to the left anterior descending artery territory. These findings were consistent with extensive non-viable ischemic myocardial scar. CMR also identified a 5-mm left ventricular thrombus attached to the basal anterolateral wall.

Twenty-four-hour Holter electrocardiographic monitoring demonstrated sinus rhythm without atrial fibrillation or significant conduction abnormalities. Subsequent coronary angiography revealed no significant obstructive coronary artery disease.

Initial thrombophilia workup, including antiphospholipid antibody testing, was negative.

Based on the overall clinical, imaging, and angiographic findings, the diagnosis of MINOCA with extensive anterior myocardial infarction sequelae complicated by left ventricular thrombus and probable systemic renal embolization was established. Anticoagulation with rivaroxaban was initiated in association with guideline-directed medical therapy for heart failure with reduced ejection fraction.

DISCUSSION

MINOCA currently represents a complex and heterogeneous clinical entity constituting a major diagnostic challenge in modern cardiology [1,3]. Patients presenting with MINOCA are generally younger than those with obstructive myocardial infarction and less frequently exhibit traditional cardiovascular risk factors [2]. A large systematic review involving more than 55,000 patients reported a MINOCA prevalence of approximately 6% among all myocardial infarctions [2]. The same analysis demonstrated that atypical clinical presentations were more frequent in these patients [2].

Our case perfectly illustrates this unusual presentation since the patient exhibited no suggestive cardiac symptoms and initially presented with a pseudo-infectious abdominal syndrome. This atypical clinical presentation initially oriented the diagnosis toward acute pyelonephritis, especially given the marked inflammatory biological findings including leukocytosis and significant elevation of C-reactive protein.

Renal infarction is a rare and probably underdiagnosed condition [6]. Clinical presentation is frequently misleading and may mimic pyelonephritis or renal colic [6]. A retrospective study investigating renal infarction reported that the main etiologies included cardioembolic disease, thrombophilia, and vascular disorders [6]. In our patient, the subsequent identification of a left ventricular thrombus strongly supports the hypothesis of systemic renal embolization.

One of the major aspects of this observation lies in the decisive diagnostic contribution of cardiac magnetic resonance imaging. International recommendations currently emphasize the central role of CMR in the evaluation of MINOCA [1,4]. Through tissue characterization and late gadolinium enhancement analysis, CMR allows differentiation between ischemic and non-ischemic myocardial injury such as myocarditis or Takotsubo syndrome [4,5].

In ischemic myocardial injury, late gadolinium enhancement is typically subendocardial or transmural and follows a coronary distribution [4]. In contrast, myocarditis usually presents with mid-wall or epicardial enhancement patterns [4,5]. Several studies have demonstrated that CMR can identify a precise etiology in nearly 70% to 80% of MINOCA cases [4,5].

In our observation, the extensive transmural enhancement pattern, coronary territorial distribution, and associated myocardial wall thinning made myocarditis or primary non-

ischemic cardiomyopathy highly unlikely. CMR therefore allowed reclassification of an initially suspected primary dilated cardiomyopathy into ischemic cardiomyopathy secondary to MINOCA.

The exact pathophysiological mechanism underlying MINOCA in our patient nevertheless remains speculative. Several mechanisms have been described in the literature [1,3]. One possible explanation is transient coronary thrombosis with spontaneous lysis before coronary angiography. Several studies suggest that coronary thrombi may rapidly recanalize, leading to angiographically normal coronary arteries despite established myocardial infarction [1,3]. Another possible mechanism is prolonged coronary vasospasm. Severe vasospasm may induce transmural myocardial infarction even in the absence of fixed coronary stenosis [1]. Occasional cannabis use in our patient is noteworthy because several reports have described associations between cannabis consumption, coronary vasospasm, and acute coronary syndromes in young individuals [3].

Coronary embolism also remains a potential mechanism, particularly in younger patients [1]. However, no emboligenic arrhythmia was identified on Holter monitoring. Transesophageal echocardiography could further complement the etiological workup by investigating potential intracardiac embolic sources not identified on transthoracic echocardiography.

The left ventricular thrombus identified on CMR represents a classical complication of extensive anterior myocardial infarction associated with severe left ventricular systolic dysfunction [7]. A recent meta-analysis demonstrated that the risk of left ventricular thrombus remains significant despite the era of primary percutaneous coronary intervention, particularly in patients with anterior myocardial infarction, apical akinesia, and severely reduced ejection fraction [7]. In our case, all these factors were present. The ventricular thrombus most likely constituted the source of systemic renal embolization.

Contrary to previous assumptions, MINOCA is not a benign condition. Several contemporary studies have demonstrated significant long-term cardiovascular mortality [2]. Therapeutic management should therefore be individualized according to the suspected underlying mechanism [1]. In our patient, anticoagulation was justified by the presence of left ventricular thrombus, while conventional guideline-directed therapy for heart failure with reduced ejection fraction was initiated.

CONCLUSION

MINOCA is a complex clinical entity requiring a rigorous multimodal diagnostic approach. This case illustrates an atypical presentation revealed by probable embolic renal infarction in a young patient without suggestive cardiac symptoms.

Cardiac magnetic resonance imaging played a decisive role in identifying the ischemic origin of myocardial injury as well as detecting the associated left ventricular thrombus. This observation also emphasizes that the absence of obstructive coronary artery disease does not exclude myocardial infarction and highlights the importance of comprehensive evaluation in young patients presenting with severe dilated cardiomyopathy.

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FIGURES

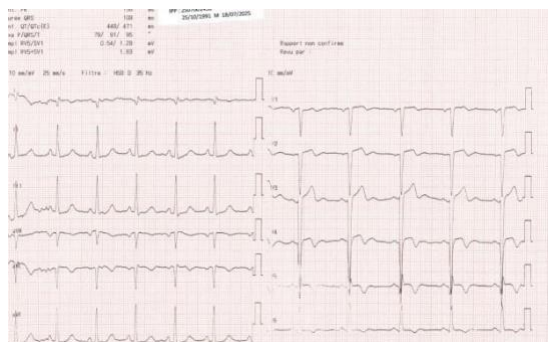


Figure 1: EKG: QS waves in the anteroapical and high lateral leads, and repolarization abnormalities at the same territory.

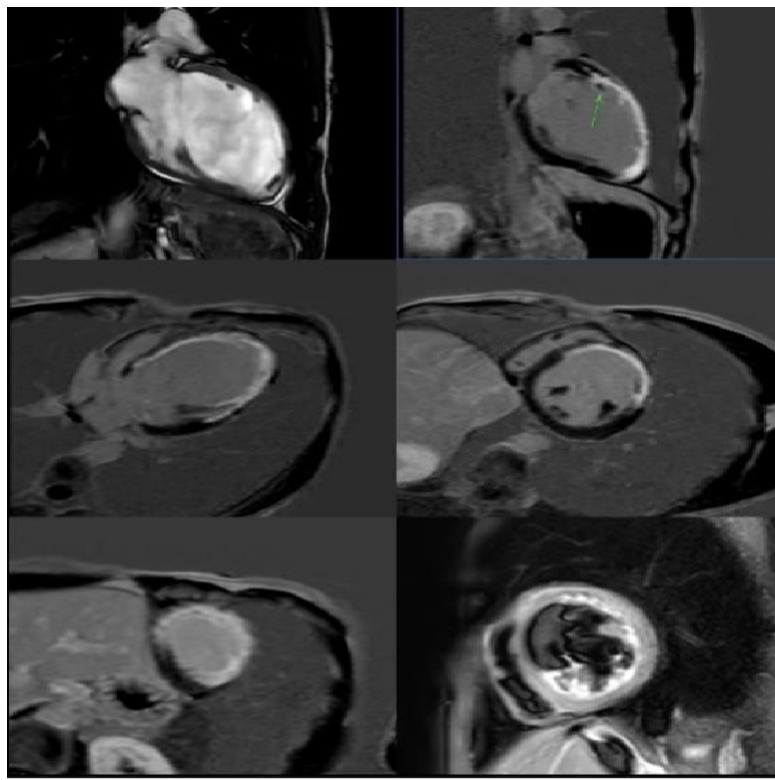


Figure 2: CRI: Extensive transmural enhancement involving the anteroseptal, anterior, and apical segments, thrombus attached to the basal anterolateral wall.

ACKNOWLEDGEMENTS

The authors have no acknowledgements to declare and report no conflicts of interest.

REFERENCES

1. Tamis-Holland JE, Jneid H, Reynolds HR, Agewall S, Brilakis ES, Brown TM, et al. "Contemporary Diagnosis and Management of Patients With MINOCA." *Circulation*. 2019;139:e891-e908. doi:10.1161/CIR.0000000000000670
<https://doi.org/10.1161/CIR.0000000000000670>

<https://pubmed.ncbi.nlm.nih.gov/30913893/>

2. Pasupathy S, Air T, Dreyer RP, Tavella R, Beltrame JF. "Systematic review of patients presenting with suspected myocardial infarction and nonobstructive coronary arteries." *Circulation*. 2015;131(10):861-870. doi:10.1161/CIRCULATIONAHA.114.011201
<https://doi.org/10.1161/CIRCULATIONAHA.114.011201>

<https://pubmed.ncbi.nlm.nih.gov/25587100/>

3. Niccoli G, Scalone G, Crea F, Camici PG. "Acute myocardial infarction with no obstructive coronary atherosclerosis: mechanisms and management." *Eur Heart J*. 2015;36(8):475-481. doi:10.1093/eurheartj/ehu469
<https://doi.org/10.1093/eurheartj/ehu469>

<https://pubmed.ncbi.nlm.nih.gov/25387757/>

4. Liang K, Alviar CL, Manandhar P, et al. "The Role of Cardiac Magnetic Resonance in Myocardial Infarction With Nonobstructive Coronary Arteries." *Front Cardiovasc Med*. 2022;8:821067. doi:10.3389/fcvm.2021.821067
<https://doi.org/10.3389/fcvm.2021.821067>

<https://pubmed.ncbi.nlm.nih.gov/35071722/>

5. Bhandari A, Srinivas P, Murthy A. "Cardiac MRI in myocardial infarction without obstructive coronary artery disease helps arrive at correct diagnosis: A case report and discussion." *J Cardiol Cases*. 2022;26(5):290-293. doi:10.1016/j.jccase.2022.06.008
<https://doi.org/10.1016/j.jccase.2022.06.008>

<https://pubmed.ncbi.nlm.nih.gov/36176961/>

6. Oh YK, Yang CW, Kim YL, Kang SW, Park CW, Kim YS, et al. "Clinical Characteristics and Outcomes of Renal Infarction." *Am J Kidney Dis*. 2016;67(2):243-250. doi:10.1053/j.ajkd.2015.09.019
<https://doi.org/10.1053/j.ajkd.2015.09.019>

<https://pubmed.ncbi.nlm.nih.gov/26545635/>

7. Robinson AA, Jain A, Gentry M, McNamara RL. "Left ventricular thrombi after STEMI in the primary PCI era: A systematic review and meta-analysis." *Int J Cardiol*. 2016;221:554-559. doi:10.1016/j.ijcard.2016.06.236
<https://doi.org/10.1016/j.ijcard.2016.06.236>

<https://pubmed.ncbi.nlm.nih.gov/27424314/>