International Research Journal of Medical Sciences and Health Care (IRJMSHC)
eISSN: 3087-405X
Volume03 Issue10 | Page No: - 06-13

Received: 25 August 2026, Revised: 10 September 2026, Accepted: 29 September 2026, Published: 14 October 2026

DOI: https://doi.org/10.55640/irjmshc-v03i10-02

Coronary Microvascular Pathomorphology In Myocardial Infarction with Non-Obstructive Coronary Arteries: Current Evidence and Limitations of Morphological Verification

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ABSTRACT: Background. Myocardial infarction with non-obstructive coronary arteries (MINOCA) is a working clinical category rather than a distinct morphological entity. In the absence of ≥50% stenosis of the major epicardial coronary arteries, infarction may be caused by plaque rupture or erosion, vasospasm, spontaneous coronary artery dissection, thromboembolism, or coronary microvascular dysfunction. Aim. To analyze which structural and functional abnormalities of the coronary microcirculation may contribute to MINOCA and which of them are supported by direct morphological evidence. Materials and methods. This narrative review is based on a targeted search of PubMed/MEDLINE, open-access full-text sources, documents of the European Society of Cardiology and the American Heart Association, Russian clinical guidelines, and Russian cardiology journals. Results. Direct pathological studies of the microvasculature in patients with clinically established MINOCA are scarce. The strongest morphological evidence concerns microvascular obstruction and microembolization, endothelial injury, arteriolar remodeling, perivascular fibrosis, and capillary rarefaction; however, much of it was obtained in other forms of ischemic heart disease, in reperfusion injury, or in conditions that mimic MINOCA. Myocarditis and takotsubo syndrome have distinct morphological features and explain troponin elevation in the absence of epicardial obstruction. Microvascular spasm is mainly a functional phenomenon and usually leaves no specific histological signature. Conclusion. Established morphological signs of infarction and microvascular injury should be distinguished from presumed functional spasm, and morphological findings should be interpreted together with clinical and imaging data.

KEYWORDS: MINOCA; myocardial infarction; coronary microcirculation; microvascular dysfunction; microthrombosis; microembolization; endothelial injury; pathology; myocarditis; takotsubo syndrome.

1. Introduction

In modern cardiology, the absence of a hemodynamically significant stenosis on coronary angiography (CAG) is no longer considered sufficient grounds for excluding acute myocardial infarction. According to the Fourth Universal Definition of Myocardial Infarction and subsequent documents of the European Society of Cardiology (ESC) and the American Heart Association (AHA), myocardial infarction with non-obstructive coronary arteries (MINOCA) is diagnosed when the criteria for acute myocardial infarction are met and there is no obstructive stenosis in the major epicardial arteries. This diagnosis is a working one and requires identification of the specific mechanism of injury [1], [2], [3], [4]. The 2024 Russian clinical guidelines likewise use the term MINOCA and emphasize the need for subsequent clarification of the cause [5].

From the standpoint of pathomorphology, the problem of MINOCA lies in the mismatch between the clinical definition of the syndrome and the possibilities for its morphological verification. CAG mainly assesses the lumen of the large epicardial arteries and practically does not visualize intramural arterioles, precapillary vessels, or the capillary network [6]. A normal or near-normal angiogram may therefore coexist with microcirculatory impairment that has no direct angiographic correlate.

In recent years, interest in the coronary microcirculation has grown markedly. Studies of coronary microvascular dysfunction (CMD) distinguish two interrelated components: a structural one (arteriolar

remodeling, capillary rarefaction, perivascular fibrosis) and a functional one (impaired endothelium-dependent vasodilation, microvascular spasm) [6], [7], [8], [9], [10]. However, extrapolating these data to MINOCA requires caution: a considerable proportion of histological studies were performed in patients with chronic coronary artery disease (CAD), arterial hypertension, or other forms of vascular pathology rather than in specifically selected MINOCA cohorts.

For the pathologist, it is important to answer not only the question of which microcirculatory changes are possible, but also a stricter one: which of them have been morphologically proven in MINOCA specifically, which are merely pathogenetically plausible, and which belong to alternative diagnoses that mimic MINOCA. The present review is devoted to drawing this distinction.

2. Aim of the review

To critically analyze the current literature on the pathomorphology of the coronary microcirculation in MINOCA, to identify the best-supported structural changes of the microvasculature, to characterize their relationship to the mechanisms of ischemic myocardial injury, and to define the limits of morphological verification of this syndrome.

3. Methods

The work was carried out as a narrative literature review with a targeted source search. The search and verification of publications were performed in October 2026 in the PubMed/MEDLINE and PubMed Central databases, as well as in official ESC and AHA materials, Russian clinical guidelines, and publications in specialized medical journals.

Preference was given to peer-reviewed original studies, systematic reviews and meta-analyses, consensus documents, and clinical guidelines. To describe microvascular morphology, earlier fundamental pathological studies were also used if they directly described microthrombi, microemboli, microvascular obstruction, or histological features of conditions clinically similar to MINOCA. The results of such studies were not regarded as direct evidence of MINOCA morphology.

An important methodological limitation: this work is not a systematic review conducted according to the PRISMA protocol (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) and does not involve quantitative pooling of morphological results. Because of the insufficient number of direct pathomorphological studies of MINOCA, a qualitative synthesis was performed, with mandatory separation of direct data, indirect morphological analogies, and functional clinical observations.

4. Results

4.1. MINOCA as a clinical rather than a morphological category

Current guidelines regard MINOCA as a working diagnosis. The 2023 ESC guidelines emphasize that it unites a heterogeneous group of causes and requires clarification in the course of further diagnostic work-up [2], [4]. The Russian guidelines of 2024 take the same position: MINOCA is established when the criteria for infarction are met and there is no stenosis ≥50% in the major coronary arteries, after which the specific cause of myocardial injury must be determined [5]. Consequently, the diagnosis of MINOCA by itself does not allow a single histological picture to be predicted.

This is essential for the pathologist. In some cases, the morphological picture corresponds to true infarction caused by transient spasm, microembolization, or destabilization of an atherosclerotic plaque not detected on angiography; in others, the clinical diagnosis of MINOCA is revised in favor of myocarditis or takotsubo syndrome after cardiac magnetic resonance imaging (CMR) [2], [11], [12], [13]. The pathomorphological conclusion should therefore describe the changes found rather than turn the MINOCA syndrome into a distinct morphological nosological entity.

4.2. The structural substrate of coronary microvascular dysfunction

The coronary microcirculation includes precapillary vessels, arterioles, the capillary network, and the venular segment. In the current literature, the structural variant of CMD is associated with remodeling of the wall of small vessels, capillary rarefaction, and perivascular fibrosis [6], [7], [8], [9], [10]. Morphologically, these processes may manifest as thickening of the vascular wall, an altered lumen-to-wall area ratio, accumulation of perivascular collagen, and a reduced number of functionally competent capillaries.

The most convincing data come from the autopsy study by Campbell et al. (2024), which histologically examined the free wall of the left ventricle in individuals who died suddenly of non-cardiac causes and had pronounced coronary atherosclerosis (stenosis ≥75% by area in at least one coronary artery), and in sex- and age-matched individuals with no CAD or minimal CAD; cases of myocardial infarction and other cardiac pathology were excluded. In the presence of CAD, the authors found lower arteriolar density and shorter capillary length per unit volume of myocardium, as well as more pronounced myocardial fibrosis [8]. These results are important for understanding the morphological basis of CMD, but they were not obtained

in patients with MINOCA; they should therefore be regarded as pathophysiological context rather than a specific morphological criterion of MINOCA.

A similar approach is seen in reviews on remodeling of the coronary microcirculation: structural changes may limit perfusion reserve and increase the vulnerability of the myocardium to brief disturbances of coronary flow [7], [8], [10]. At the same time, it cannot be reliably established morphologically whether the remodeling found was the cause of the acute event or a pre-existing background on which the infarction developed.

4.3. Microvascular spasm: a functional mechanism with limited morphological verification

Microvascular spasm is among the mechanisms discussed in MINOCA. In clinical practice it is detected with functional tests, in particular the acetylcholine provocation test and assessment of the coronary response [4], [9], [10]. Unlike thrombus or dissection, transient spasm does not necessarily leave specific morphological changes.

The notion that microvascular spasm can be seen on a histological specimen is therefore incorrect. The pathologist may find the consequences of ischemia — cardiomyocyte injury and necrosis, interstitial edema, hemorrhage, and later granulation tissue and fibrosis — but is not always able to prove retrospectively that spasm was the triggering factor. This is particularly important in late autopsy, when the functional state of the vascular wall can no longer be assessed.

4.4. Microthrombosis, microembolization, and microvascular obstruction

The most evident morphological mechanism of microcirculatory injury involves intravascular material capable of mechanically occluding the lumen of microarteries and arterioles. In a classic pathomorphological study of sudden coronary death, Schwartz et al. identified microemboli and occlusion of intramural vessels containing fibrin and platelets in their lumen; these were most often found in the territory of the left anterior descending artery, and emboli in vessels less than 200 μm in diameter were more often associated with plaque erosion than with plaque rupture [14]. The study was carried out not in a MINOCA cohort but in acute coronary thrombosis; nevertheless, it directly demonstrates that the pathological process can extend from the epicardial artery to the level of the microcirculation.

In MINOCA, there may be situations in which embolic material or a microthrombus is no longer preserved by the time of examination or lies in vessels inaccessible to standard macroscopic inspection (Fig. 1). The absence of a large coronary thrombus at autopsy therefore does not exclude a microcirculatory mechanism. At the same time, the finding of a microthrombus cannot automatically be taken as proof of a causal relationship: the clinical context, the timing of the event, the possible source of embolism, and the localization of the lesion must be taken into account.

Morphologically, microvascular obstruction may be accompanied by zonal ischemic injury, interstitial edema, small hemorrhages, and cardiomyocyte necrosis. With reperfusion, endothelial injury, vascular wall edema, and intravascular cellular debris are added. These changes are well known in infarction and reperfusion injury and are not specific to MINOCA [4], [14].

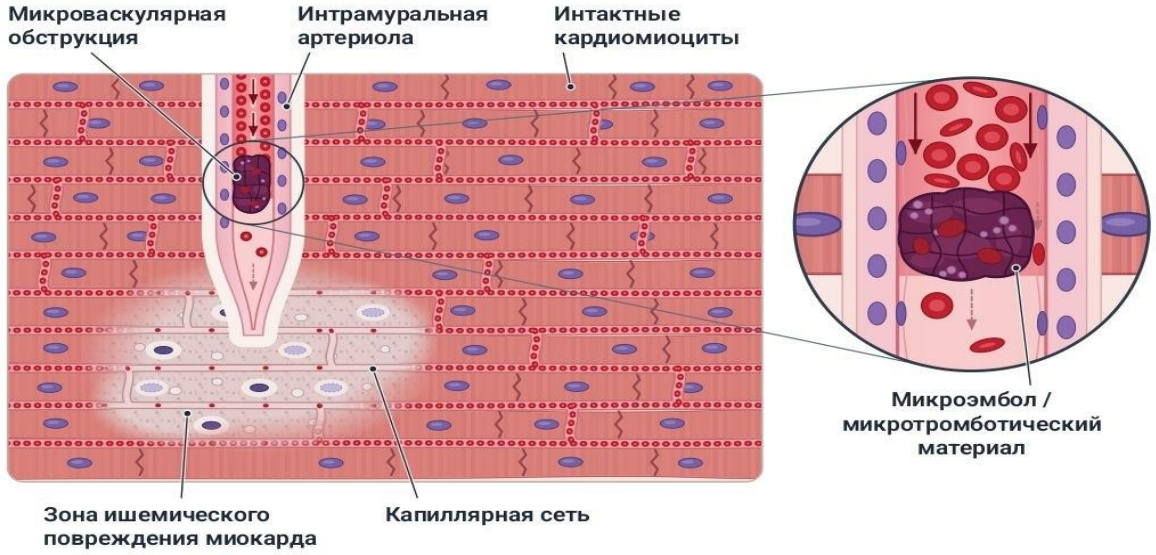


Figure 1. Schematic reconstruction of microvascular obstruction in MINOCA. Microembolic/microthrombotic occlusion of an intramural microvessel leads to impaired distal blood flow, reduced capillary perfusion, and the formation of focal ischemic myocardial injury (the illustration is the author's schematic based on published pathomorphological and pathophysiological data and is not an original photomicrograph).

4.5. Endothelial injury and inflammatory lesions of the microcirculation

Endothelial injury may be not only functional but also morphologically evident. Among inflammatory heart diseases, myocarditis is of particular importance; it is not infrequently found in patients with an initial working diagnosis of MINOCA. According to a meta-analysis and a prospective CMR study, a substantial proportion of patients referred with presumed MINOCA show signs of myocarditis after further diagnostic work-up [11], [12].

A reported case of fatal parvovirus B19-associated myocarditis is instructive for the pathologist. The viral genome was detected by in situ hybridization predominantly in the endothelial cells of venules and, to a lesser extent, in small arteries and arterioles; endothelial activation and perivascular T-cell inflammation were noted at the same time. The authors linked the endothelial infection to impaired coronary microcirculation and secondary cardiomyocyte injury [15]. This case should not be considered a morphological variant of MINOCA, but it illustrates that microvascular endothelial injury can produce a clinical picture of ischemia without obstruction of the large coronary arteries.

When examining autopsy or biopsy material, attention should be paid to perivascular and interstitial inflammatory infiltration, stromal edema, endothelial injury, and signs of vasculitis. At the same time, overdiagnosis must be avoided: minimal perivascular cell clusters without the appropriate context do not in themselves prove a causal relationship with acute infarction.

4.6. Takotsubo syndrome as the most important differential diagnosis

Takotsubo syndrome may clinically mimic acute infarction with non-obstructive coronary arteries, but in the modern diagnostic concept it is identified as a separate diagnosis after the cause of myocardial injury has been clarified [4], [16], [17]. For the pathologist it is important primarily because it has characteristic, although non-specific, histological features.

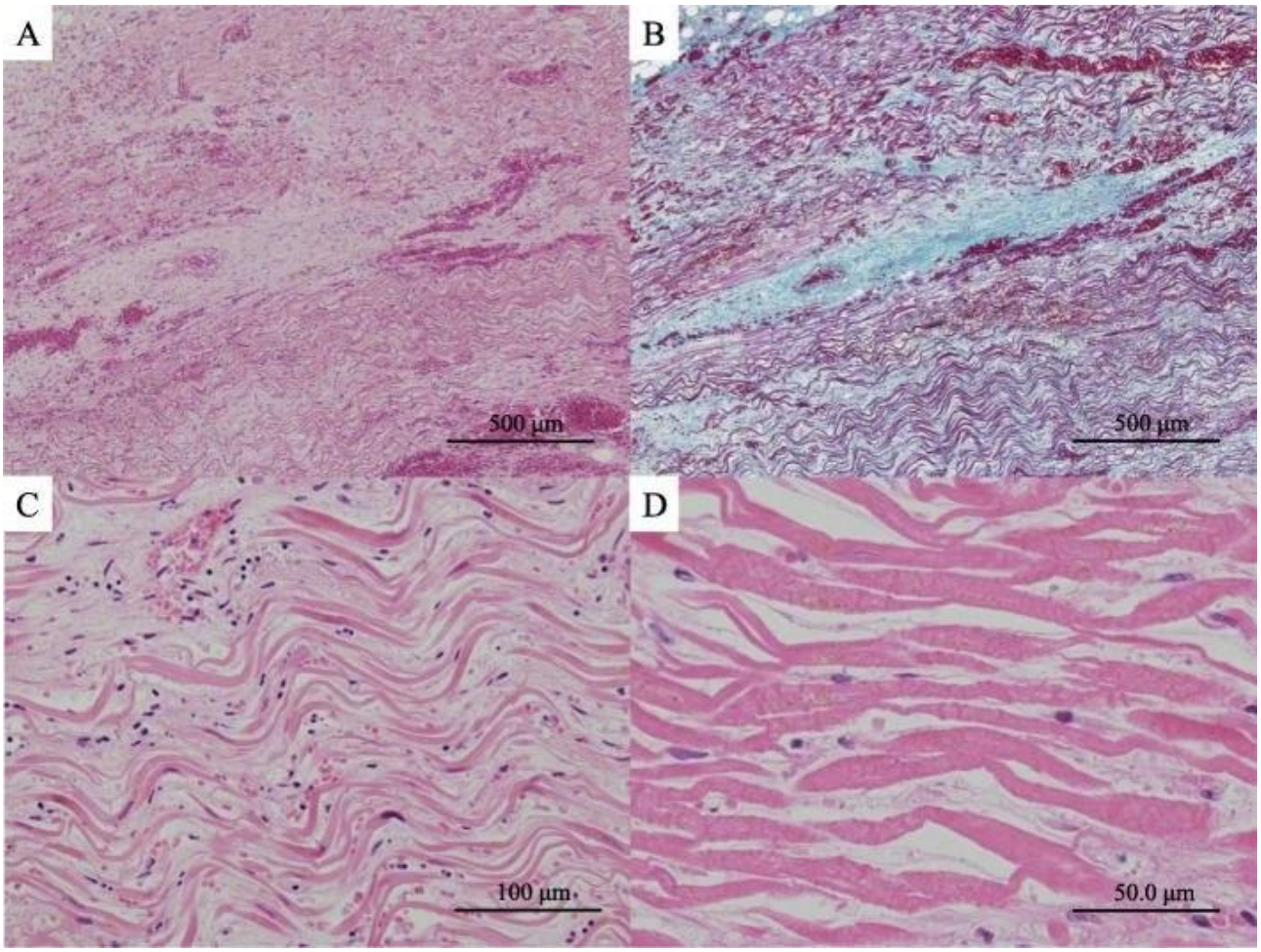


Figure 2. Pathomorphological changes of the myocardium in takotsubo cardiomyopathy. Histological examination shows degenerative changes of cardiomyocytes, foci of injury and necrosis, interstitial fibrosis, and hemorrhage. A characteristic morphological feature is contraction band necrosis of cardiomyocytes, reflecting the marked damaging effect of catecholamines on the myocardium. The changes presented illustrate the structural substrate of severe myocardial injury in takotsubo cardiomyopathy.

Published biopsy and autopsy observations describe focal cardiomyocyte injury, interstitial edema, minimal inflammatory infiltration and, above all, necrosis with contraction bands (contraction band necrosis) [18], [19] (Fig. 2). Histologically, contraction bands are dense eosinophilic transverse bands resulting from hypercontraction of sarcomeres and are associated with catecholamine-induced injury. However, this feature is not pathognomonic: contraction bands are also seen in agonal states, in reperfusion, and in other forms of catecholamine load [18], [19], [20].

Autopsy observations in takotsubo syndrome also show that the lesion may be multifocal and may not correspond to the territory of a single epicardial artery [19]. In individual cases, hemorrhages and microthrombi in small coronary vessels have been described, but these findings are inconsistent and do not allow takotsubo syndrome to be reduced to microvascular occlusion alone [17].

4.7. Pathomorphological characteristics of ischemic necrosis in the absence of obstruction of the large epicardial arteries

If a decedent with clinically established MINOCA truly has a myocardial infarction, the microscopic signs of necrosis should correspond to the stage of ischemic injury. In the acute period, coagulative necrosis of cardiomyocytes, loss of cross-striations, cytoplasmic eosinophilia, and nuclear changes are expected; these are followed by neutrophilic infiltration, macrophage resorption, granulation tissue, and scar. With a microcirculatory mechanism, the size and distribution of the foci may be small, patchy, and mosaic.

The distribution of necrosis must be correlated with coronary artery anatomy. A focus that does not correspond to the territory of a single epicardial artery, or multiple small foci, suggest an embolic, spastic, or other non-classical mechanism, but do not in themselves prove it. Intravascular findings — microthrombi, fibrin, platelet aggregates, vascular debris, hemorrhages — increase the likelihood of microcirculatory involvement, but their interpretation depends on the age of the process.

4.8. Morphological methods and their diagnostic capabilities

Light microscopy with hematoxylin and eosin staining remains the main method for assessing the morphology of infarction and inflammatory myocardial lesions. Connective tissue stains, including picrosirius red and trichrome methods, allow quantitative or semiquantitative assessment of interstitial and perivascular fibrosis [8]. Immunohistochemical endothelial markers, for example CD31 (cluster of differentiation 31; synonym — PECAM-1, platelet endothelial cell adhesion molecule), are used to visualize capillaries and for their morphometry; this approach was used in an autopsy study of the coronary microcirculation [8].

Electron microscopy makes it possible to assess interendothelial junctions, the basement membrane, mitochondria, and ultrastructural signs of cardiomyocyte injury; however, in MINOCA its use in clinical practice is limited, and it rarely determines the final etiology. Intravascular methods — optical coherence tomography (OCT) and intravascular ultrasound (IVUS) — are not pathomorphological in the strict sense but are of great importance for detecting mural thrombi, erosion, dissection, and other changes that may remain inaccessible on routine CAG [3], [11], [21].

For the pathologist, a multimodal approach is the most productive: macroscopic examination of the heart and coronary arteries, serial examination of the myocardium, routine histology, immunohistochemistry and special stains when necessary, and then correlation with CAG, OCT/IVUS, and CMR data. No single method can by itself fully reveal the mechanism of MINOCA. The main morphological findings and the strength of their evidence in MINOCA are summarized in Table 1.

Table 1. Morphological findings and the strength of their evidence in MINOCA

Mechanism / finding	What may be found morphologically	Interpretation in MINOCA
Structural CMD	Arteriolar remodeling, perivascular fibrosis, capillary rarefaction	Morphologically proven in related conditions; specificity for MINOCA not established [7], [8].
Microvascular spasm	Usually no specific histological trace; consequences of ischemia are assessed	A functional diagnosis, confirmed mainly by provocation tests [4], [9], [10].
Microthrombosis / microembolization	Fibrin, platelets, and embolic material in intramural vessels; microhemorrhages	Has a direct morphological basis, but most data were obtained outside specifically selected MINOCA cohorts [14].
Endothelial inflammatory lesion	Endotheliitis and vasculitis-like changes, perivascular infiltration, edema	May point to myocarditis or vasculitis as an alternative cause [15].
Takotsubo syndrome	Contraction bands, focal cardiomyocyte injury, edema, sometimes inflammation	An important differential diagnosis; the findings are not pathognomonic [17], [18], [19], [20].

Mechanism / finding	What may be found morphologically	Interpretation in MINOCA
Myocardial infarction	Coagulative necrosis, neutrophils, macrophages, granulation tissue, fibrosis	Morphologically confirms the fact of infarction but does not always allow its mechanism to be established.

5. Discussion

The analysis shows that the main difficulty of the topic lies not in a shortage of pathophysiological hypotheses but in the limited direct morphological evidence base. Clinical studies demonstrate that functional abnormalities of the coronary microcirculation are found in some patients with MINOCA, and current guidelines recognize microvascular dysfunction as one of the possible mechanisms [4], [9], [10]. However, confirming this mechanism morphologically in the human heart in reliably established MINOCA is considerably more difficult: spasm is transient, microthrombi may lyse or migrate, and structural remodeling may exist long before the acute event.

Therefore, the results of CMD studies in chronic CAD, arterial hypertension, diabetes mellitus, or myocardial ischemia with non-obstructive coronary arteries cannot be mechanically transferred to MINOCA. Thus, the autopsy study by Campbell et al. showed reduced arteriolar and capillary density in severe CAD [8], but the hearts examined had no infarction and did not reflect the MINOCA population. Similarly, the data of Schwartz et al. on microemboli in vessels less than 200 μm in diameter confirm that microcirculatory occlusion is real, but the group studied consisted of individuals with sudden coronary death and acute coronary thrombosis [14].

It is precisely here that the pathologist's approach must be especially rigorous: morphological examination must not be replaced by pathogenetic reconstruction based solely on clinical context. If characteristic signs of myocarditis are identified (primarily inflammatory infiltration with cardiomyocyte injury), confirmed when necessary by additional studies, it is more correct to regard myocarditis as the cause of the initial clinical picture than to retain a formal diagnosis of MINOCA. The same applies to takotsubo syndrome, which has its own diagnostic context and a combination of histological features characteristic of catecholamine injury [17], [18], [19], [20].

Cardiac MRI occupies a special place in the diagnostic chain. According to a meta-analysis and prospective studies, in a substantial proportion of patients with an initial working diagnosis of MINOCA, CMR reveals an alternative or more specific explanation of myocardial injury, primarily myocarditis or takotsubo syndrome [11], [12]. From the pathological standpoint, this means that at subsequent autopsy one must assess not only the coronary arteries but also the nature of myocardial injury, its distribution, and the presence of inflammation, fibrosis, microhemorrhages, and vascular material.

The term "microvascular dysfunction" deserves separate discussion. It combines structural and functional states and is therefore not in itself a morphological diagnosis. In the work of Collet et al. [7], structural remodeling is considered one dimension of CMD, whereas endothelial dysfunction and spasm belong to its functional components; a similar division is used in other reviews [9], [10]. This division should be preserved in pathomorphological analysis as well: the presence of a structural substrate does not prove acute spasm, and functional spasm does not necessarily leave structural changes.

Thus, the best-supported pathomorphological model of MINOCA is not a single picture but several overlapping scenarios. The first is true infarction with small-focal ischemic necrosis due to a transient or microvascular mechanism. The second is infarction due to microembolization or microthrombosis, in which visualization of intravascular material is possible. The third is inflammatory or catecholamine-dependent myocardial injury initially mistaken for infarction. The fourth is a combination of several factors: a structurally altered microcirculation lowers the ischemic threshold, and the triggering event is a brief spasm or embolization.

Limitations of the review and of the evidence base

The main limitation is the absence of large standardized autopsy series in which the diagnosis of MINOCA was made during life according to current criteria and the microvasculature was systematically examined after death with morphometry of arterioles and capillaries. Most morphological studies concern either infarction in obstructive coronary disease or individual conditions that mimic MINOCA. The question therefore remains open as to what proportion of MINOCA cases is directly caused by primary structural pathology of the microcirculation.

Additional limitations relate to postmortem changes in vascular tone and the possible loss of transient intravascular structures. Moreover, the microscopic picture depends on the time elapsed since the onset of ischemia, the fixation methods, and the site from which sections were taken. The absence of a particular

morphological sign therefore does not always mean the absence of the corresponding mechanism in the acute period. Finally, a limitation of the review itself is its narrative format: the search was not systematic and the selection of publications was not governed by a formalized protocol, which does not exclude incomplete coverage of the literature and subjectivity of selection.

6. Conclusion

MINOCA should be regarded as a working clinical category that unites different mechanisms of acute myocardial injury in the absence of obstructive lesions of the large epicardial coronary arteries. The pathomorphological picture is accordingly not uniform. Of greatest importance are ischemic cardiomyocyte necrosis, microthrombosis and microembolization, structural remodeling of the microcirculation, endothelial inflammatory injury, and the features of conditions that mimic infarction — above all myocarditis and takotsubo syndrome.

At the same time, direct morphological evidence of a primary microvascular cause specifically in MINOCA remains limited. Structural signs of CMD have been convincingly shown in related forms of vascular pathology, and microemboli and microvascular obstruction are well described in acute coronary thrombosis, but transferring these data to MINOCA requires caution [8], [14]. Microvascular spasm is predominantly a functional phenomenon and has no reliable specific histological marker.

In the pathologist's practical work, a comprehensive approach is the most important: morphological verification of infarction and/or alternative myocardial injury must be supplemented by examination of the coronary arteries, a search for microvascular thrombosis and embolism, and assessment of the endothelium and perivascular space, and the results must be interpreted together with angiographic, intravascular imaging, and CMR data. Such an approach avoids both underestimation of microcirculatory injury and the erroneous transformation of MINOCA into a single "morphological disease."

Practical significance

When MINOCA is suspected, it is advisable in pathological practice to:

1) carefully examine all the major epicardial coronary arteries, looking for minimal atherosclerotic changes, thrombi, and signs of dissection and embolization;

2) perform serial examination of the myocardium from zones of macroscopic and suspected microscopic injury;

3) assess small intramural vessels for thrombotic material, endothelial injury, perivascular inflammation, and hemorrhagic changes;

4) when necessary, use immunohistochemical detection of CD31 and special stains to assess the vascular network and fibrosis;

5) consider myocarditis and takotsubo syndrome as mandatory alternative explanations for elevated troponin in non-obstructive coronary arteries.

Such an algorithm, naturally, does not replace the clinical diagnostic work-up, but it increases the diagnostic value of morphological examination.

References

1. Thygesen K, Alpert JS, Jaffe AS, et al. Fourth Universal Definition of Myocardial Infarction (2018). Eur Heart J. 2019;40(3):237–269. doi:10.1093/eurheartj/ehy462. PMID:30165617.
2. Agewall S, Beltrame JF, Reynolds HR, et al. ESC working group position paper on myocardial infarction with non-obstructive coronary arteries. Eur Heart J. 2017;38(3):143–153. doi:10.1093/eurheartj/ehw149. PMID:28158518.
3. Tamis-Holland JE, Jneid H, Reynolds HR, et al. Contemporary Diagnosis and Management of Patients With Myocardial Infarction in the Absence of Obstructive Coronary Artery Disease: A Scientific Statement From the American Heart Association. Circulation. 2019;139(18):e891–e908. doi:10.1161/CIR.0000000000000670. PMID:30913893.
4. Byrne RA, Rossello X, Coughlan JJ, et al. 2023 ESC Guidelines for the management of acute coronary syndromes. Eur Heart J Acute Cardiovasc Care. 2024;13(1):55–161. doi:10.1093/ehjacc/zuad107. PMID:37740496.
5. Averkov OV, Arutyunyan GK, Duplyakov DV, et al. Acute coronary syndrome without ST-segment elevation on the electrocardiogram. Clinical guidelines 2024. Russian Journal of Cardiology. 2025;30(5):6319. doi:10.15829/1560-4071-2025-6319. EDN: CXJUIB. (In Russ.)
6. Shmatova EN, Grinshtein YuI. Microvascular angina: pathogenesis, clinical presentation, diagnosis, and principles of therapy. RMJ. Medical Review. 2020;4(7):425–430. doi:10.32364/2587-6821-2020-4-7-425-430. (In Russ.)
7. Collet C, Sakai K, Mizukami T, et al. Vascular Remodeling in Coronary Microvascular Dysfunction. JACC Cardiovasc Imaging. 2024;17(12):1463–1476. doi:10.1016/j.jcmg.2024.07.018. PMID:39269414.

8. Campbell DJ, Francis VCM, Young GR, Woodford NW. Association of Coronary Microvascular Rarefaction and Myocardial Fibrosis With Coronary Artery Disease. J Am Heart Assoc. 2024;13(21):e037332. doi:10.1161/JAHA.124.037332. PMID:39424420.

9. Shome JS, Perera D, Plein S, Chiribiri A. Current perspectives in coronary microvascular dysfunction. Microcirculation. 2017;24(1):e12340. doi:10.1111/micc.12340. PMID:27976459.

10. Severino P, D'Amato A, Prosperi S, et al. Coronary microcirculation in myocardial ischemia: A genetic perspective. J Mol Cell Cardiol. 2025;203:67–75. doi:10.1016/j.yjmcc.2025.04.002. PMID:40220989.

11. Reynolds HR, Maehara A, Kwong RY, et al. Coronary Optical Coherence Tomography and Cardiac Magnetic Resonance Imaging to Determine Underlying Causes of Myocardial Infarction With Nonobstructive Coronary Arteries in Women. Circulation. 2021;143(7):624–640. doi:10.1161/CIRCULATIONAHA.120.052008.

12. Balakrishna AM, Ismayl M, Thandra A, et al. Diagnostic Value of Cardiac Magnetic Resonance Imaging and Intracoronary Optical Coherence Tomography in Patients With a Working Diagnosis of Myocardial Infarction With Non-obstructive Coronary Arteries: A Systematic Review and Meta-analysis. Curr Probl Cardiol. 2023;48(6):101126. doi:10.1016/j.cpcardiol.2022.101126. PMID:35120967.

13. Niccoli G, Camici PG. Myocardial infarction with non-obstructive coronary arteries: what is the prognosis? Eur Heart J Suppl. 2020;22(Suppl E):E40–E45. doi:10.1093/eurheartj/suaa057. PMID:32523437.

14. Schwartz RS, Burke A, Farb A, et al. Microemboli and Microvascular Obstruction in Acute Coronary Thrombosis and Sudden Coronary Death: Relation to Epicardial Plaque Histopathology. J Am Coll Cardiol. 2009;54(23):2167–2173. doi:10.1016/j.jacc.2009.07.042. PMID:19942088.

15. Bültmann BD, Klingel K, Sotlar K, et al. Fatal parvovirus B19-associated myocarditis clinically mimicking ischemic heart disease: an endothelial cell-mediated disease. Hum Pathol. 2003;34(1):92–95. doi:10.1053/hupa.2003.48. PMID:12605372.

16. Sucato V, Comparato F, Ortello A, Galassi AR. Myocardial Infarction with Non-Obstructive Coronary Arteries (MINOCA): pathogenesis, diagnosis and treatment. Curr Probl Cardiol. 2024;49(7):102583. doi:10.1016/j.cpcardiol.2024.102583. PMID:38679151.

17. Khattab E, Karelas D, Pallas T, et al. MINOCA: A Pathophysiological Approach of Diagnosis and Treatment—A Narrative Review. Biomedicines. 2024;12(11):2457. doi:10.3390/biomedicines12112457. PMID:39595023.

18. Nef HM, Möllmann H, Elsässer A. Tako-tsubo cardiomyopathy (apical ballooning). Heart. 2007;93(10):1309–1315. doi:10.1136/hrt.2006.101675. PMID:17890711.

19. Kinbara T, Hayano T, Otani N, et al. An autopsy case of tako-tsubo cardiomyopathy presenting ventricular tachycardia after pacemaker implantation. J Cardiol Cases. 2013;8(4):134–137. doi:10.1016/j.jccase.2013.06.007. PMID:30546765.

20. Mizutani K, Shioya A, Hirose Y, et al. Serious takotsubo cardiomyopathy: an autopsy case presenting severe irreversible myocardial damage after frequent episodes of recurrence. Diagn Pathol. 2020;15:90. doi:10.1186/s13000-020-01006-x. PMID:32693809.

21. Vorobyevsky DA, Kozlov KL, Shcherbak SG, Lazakovich DN. The role of intravascular imaging in determining treatment strategy for a patient with NSTEMI and no obstructive coronary artery disease. Kardiologicheskii Vestnik. 2024;19(2):67–71. doi:10.17116/Cardiobulletin20241902167. (In Russ.).