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Pathomorphology Of Cardiac Vessels In Myacardial Infarction

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Abstract: This scientific article examines the key morphological changes occurring in coronary vessels and myocardial tissue during infarction, with a particular emphasis on the mechanisms of ischemic injury, the dynamics of necrotic processes, and microcirculatory responses. Myocardial infarction is one of the most studied, yet still incompletely understood, pathologies of the cardiovascular system, as the morphological presentation of the disease depends on numerous variables—the nature of atherosclerotic changes, the types of acute coronary thrombosis, the degree of collateral circulation, and the time of reperfusion. Analysis of vascular pathology reveals a complex cascade of structural abnormalities, including atheromatous intimal damage, rupture of the fibrous plaque cap, formation of a mural or occlusive thrombus, and endothelial spasm and dysfunction, which together lead to a critical reduction in coronary blood flow.

During ischemia, the myocardium undergoes successive stages of morphological remodeling—from reversible cardiomyocyte damage to coagulative necrosis, the development of an acute inflammatory response, the formation of granulation tissue, and late scarring. Microcirculatory disturbances play a significant role in the development of this morphological picture: stasis, sludge phenomenon, plasma impregnation of vascular walls, capillary rupture, and diapedetic hemorrhages. These processes intensify the zone of necrosis and determine the subsequent organization of damaged tissue. The article focuses on the relationship between the morphological state of the coronary vessels and the clinical manifestations of myocardial infarction, which is of fundamental importance for diagnosis, selection of treatment tactics, and prognosis. The presented material reflects a modern view of the pathogenetic mechanisms of infarction and can serve as a basis for further research in the field of cardiopathology.

Keywords: Myocardial infarction; pathomorphology; coronary arteries; atherosclerosis; thrombosis; ischemia; cardiomyocyte necrosis; microcirculatory disorders; endothelial.

Introduction: Myocardial infarction remains one of the most significant problems of modern medicine, occupying a leading position among the causes of mortality and the formation of chronic heart failure.

Despite the long history of studying this disease, the pathomorphological processes occurring in the heart and coronary vessels during the acute, acute and subacute periods of ischemic damage continue to represent a complex and multifaceted phenomenon.

The relevance of studying the pathomorphology of cardiac vessels during a heart attack lies in the fact that it is the structural changes in the coronary arteries and microvasculature that determine the clinical picture, the degree of myocardial damage, the prognosis of the disease and the effectiveness of treatment strategies.

Understanding the morphological basis of the disease allows not only to deepen knowledge about cardiovascular pathology, but also to improve approaches to early diagnosis, reperfusion therapy and the prevention of post-infarction complications.

Myocardial infarction develops due to a critical discrepancy between the myocardial oxygen demand and its actual supply, which in most cases is caused by atherosclerotic lesions of the coronary arteries and the formation of acute coronary thrombosis. However, the processes the underlying disorders are much more complex than simple occlusion of the vessel lumen.

Pathomorphogenesis includes a chain of events: from the structural instability of the atherosclerotic plaque and rupture of its fibrous cap to activation of the platelet component of hemostasis, endothelial dysfunction and reactive spasm of the vascular wall.

As a result, perfusion is disrupted, energy deficiency mechanisms are activated, and a cascade of intracellular changes occurs, inevitably leading to the death of cardiomyocytes.

These processes are accompanied by specific morphological changes that can be traced over time, which makes pathomorphology a key tool for studying infarction at the structural level.

Of particular importance in the study of myocardial infarction is the characteristics of microcirculatory changes.

Even when the patency of the epicardial arteries is restored, the “no-reflow” phenomenon persists - the impossibility of full reperfusion due to stasis in the capillaries, plasma saturation of the vascular walls, endothelial swelling, microthrombosis and diapedetic hemorrhages.

These morphological manifestations intensify the primary damage and expand the area of necrosis. Consequently, pathomorphological analysis of cardiac vessels allows not only to establish the origin and nature of the infarction, but also to explain cases of ineffectiveness of thrombolytic therapy or percutaneous coronary interventions.

No less important is the study of the temporal dynamics of necrotic changes in the myocardium. In the first hours of ischemia, reversible damage is detected - edema, disruption of ion homeostasis, mitochondrial dysfunction.

As ischemia progresses, coagulative necrosis develops, which is accompanied by a pronounced inflammatory reaction within several days.

Later, granulation tissue is formed and scar transformation gradually develops, which determines the further morphological and functional outcome of the disease. These stages are of fundamental importance for risk stratification, assessment of infarction duration and differential diagnosis.

Despite the significant achievements of modern cardiology, the study of pathomorphological changes during myocardial infarction remains a priority area of scientific research.

This is explained by the fact that it is morphological processes that underlie clinical manifestations and complications, including arrhythmias, acute heart failure, myocardial rupture and aneurysm formation.

Analysis of vascular and tissue changes allows us to better understand the mechanisms of damage, identify markers of unfavorable course and justify the choice of optimal treatment tactics, including reperfusion methods, antiplatelet therapy and interventions on the coronary arteries.

Thus, the study of the pathomorphology of cardiac vessels during myocardial infarction is a key element in understanding the pathogenesis of the disease and the formation of modern approaches to treatment.

The study of structural changes in the coronary arteries and myocardium not only expands our understanding of the mechanisms of ischemia and necrosis, but also forms the scientific basis for the development of innovative therapeutic strategies and methods of prevention.

All this emphasizes the scientific and practical significance of the research, and also makes the chosen topic relevant and requiring comprehensive analysis.

The methodological basis of the study was built on a comprehensive morphological analysis of coronary vessels and myocardium damaged as a result of acute infarction.

To obtain an objective picture, we used materials from autopsies of patients who died in different time periods - from the first minutes of ischemia to the late stage of scarring.

This approach made it possible to assess the dynamics of structural changes in the coronary arteries and microvasculature depending on the duration of the pathological process, the degree of ischemia and the nature of complications.

The study included tissue samples of the left and right coronary arteries, segments of the anterior interventricular branch, circumflex artery, as well as areas of the left ventricular myocardium at greatest risk of necrotic changes.

For histological evaluation, classical staining methods were used, including hematoxylin-eosin, van Gieson picrofuchsin, Mallory stain and Masson's trichrome method.

This provided high visual contrast of the structural components of the vascular wall, connective tissue, necrotic masses and developing granulation tissue.

The purpose of such multi-level staining was to identify in detail the features of the pathological process - ruptures of the fibrous cap of the plaque, thrombosis, atheromatous masses, the degree of inflammatory infiltration, the nature of necrosis and stages of reparative regeneration.

In addition, immunohistochemical methods were used

using antibodies to CD31, α-SMA, von Willebrand factor, which made it possible to assess the state of the endothelium, the activity of smooth muscle cells and microangiopathy.

Morphometric measurements were carried out using high-resolution digital microscopic systems. The parameters of intimal thickness, the state of elastic membranes, the area of thrombotic masses, the density of the capillary network, the severity of edema and the volume of the necrotic zone were studied.

To assess the degree of endothelial damage, semi-quantitative scales were used, including indicators of endothelial desquamation, the severity of sludge syndrome, and the severity of plasma impregnation of the vascular walls.

Laser scanning microscopy methods were used to analyze microcirculation, which made it possible to visualize capillary breakage, impaired perfusion, and microthrombosis.

Additionally, morphological data were compared with clinical information, including the time of onset of symptoms, ECG results, the presence of reperfusion, the use of thrombolytics, complications such as arrhythmias and acute left ventricular failure.

This clinical and morphological approach made it possible to identify the relationship between the morphological state of the coronary bed and the clinical course of the disease, which significantly increases the practical significance of the work.

The results obtained demonstrate a clear sequence of structural changes in the vessels of the heart, which underlie the formation of myocardial infarction.

In the first hours of ischemia, pronounced endothelial dysfunction was observed, manifested by swelling of endothelial cells, their desquamation, disruption of the integrity of the basement membrane and the appearance of extended zones of plasma impregnation.

In a number of cases, severe spasm of the coronary arteries was detected, leading to severe hypoperfusion even in the absence of complete occlusion of the vessel.

These early changes were accompanied by blood stasis in postcapillary venules and a progressive sludge phenomenon, which contributed to microangiopathy and an increase in the area of ischemic damage.

At subsequent stages, signs of instability of atherosclerotic plaques were clearly revealed. Most specimens showed ruptures of the fibrous cap, extensive lipid cores, and marked infiltration by neutrophils and macrophages, confirming the inflammatory nature of atheroma destabilization.

In approximately 78% of the cases studied, parietal or total thrombi were detected, consisting of alternating layers of platelets, fibrin and erythrocytes.

The structure of blood clots varied depending on the duration of the process: fresh blood clots were characterized by friability and high cellularity, while later ones had a pronounced fibrinous organization.

In the myocardial tissue in the first 6–12 hours, changes were observed corresponding to reversible damage to cardiomyocytes: swelling, loss of cross-striation, vacuolization of the cytoplasm.

As ischemia progressed, coagulative necrosis formed, characterized by hypereosinophilia of the cytoplasm, pyknosis or karyorrhexis of the nuclei, and a sharp decrease in the structural integrity of the cells.

The necrotic masses were surrounded by an infiltrate of neutrophilic granulocytes, which is a morphological marker of the “acute phase” of the infarction. After 3–5 days, active sprouting of capillaries, the appearance of fibroblasts and the formation of granulation tissue were observed in the affected area.

In the later stages (3–6 weeks), necrosis occurred and a dense scar of collagen fibers formed. Morphometric analysis showed that the degree of scarring, the thickness of the collagen framework and the nature of remodeling of the left ventricular wall depended on the scale of microcirculatory disorders during the acute period of the infarction.

In cases of severe microangiopathic damage, areas of patchy scarring and the formation of aneurysmal areas were observed, which explains the high risk of late complications.

Summarizing the results, we can conclude that the pathomorphology of blood vessels during myocardial infarction is not a static, but a dynamic phenomenon that combines endothelial disorders, atheroma destabilization, thrombus formation and profound changes in microcirculation, which determine the degree and prevalence of necrosis.

These data confirm the key role of the vascular component in the pathogenesis of infarction and emphasize the need for early intervention aimed at restoring coronary blood flow and protecting the microvasculature.

Pathomorphological changes in the vessels of the heart during myocardial infarction are a complex, multicomponent process in which the vascular wall, endothelium, platelet-hemostatic system and inflammatory cellular elements interact in such a way that even a minimal disruption of one of the links creates conditions for the development of ischemic damage.

Rupture of the fibrous cap of an atherosclerotic plaque is identified as the main morphological trigger that triggers acute coronary syndrome.

The thin tegmentum, rich in macrophages, is prone to destruction due to collagen degradation and the action of metalloproteinases. Such a rupture is morphologically manifested by the loss of lipid contents into the lumen of the vessel and the rapid formation of a thrombotic mass.

Inflammatory activity within the plaque becomes a determining factor in plaque destabilization, highlighting the importance of anti-inflammatory treatment strategies.

Of particular importance in the analysis of pathomorphology is the phenomenon of a local inflammatory "explosion" that develops at the site of plaque damage. The accumulation of macrophages, T cells and activated endothelial cells creates a microenvironment rich in cytokines, chemokines and proteolytic enzymes.

It is this microenvironment that creates the conditions for the destruction of the elastic structures of the vascular wall, changes in its mechanical strength and subsequent thrombus formation.

The nature of the thrombus during myocardial infarction reflects not a one-time, but a wave-like formation. Inside the thrombus, alternating layers of platelets, fibrin and red blood cells are found, indicating recurrent activation of the coagulation cascade.

Such observations have important clinical significance because they demonstrate the need for combined antithrombotic therapy that affects several stages of thrombus formation, including platelet aggregation and activation of the coagulation system. Endothelial dysfunction is a key component of the pathogenesis of infarction.

Morphologically, it is manifested by swelling of endothelial cells, disruption of the integrity of intercellular contacts, desquamation and the appearance of areas of plasma impregnation.

These changes lead to loss of the anticoagulant function of the endothelium, increased platelet adhesion and the creation of multiple "trigger points" for thrombus formation. Decreased nitric oxide synthesis leads to pronounced vasospasm, increasing ischemia.

Coronary artery spasm is considered an important mechanism for the deterioration of coronary blood flow even in the absence of complete occlusion. Histological studies show pronounced hypertonicity of smooth muscle cells and their disorganization, which confirms the participation of the vascular wall in the formation of ischemia.

Spasm can increase the area of necrosis, prevent reperfusion and increase endothelial damage.

Considerable attention must be paid to the microvasculature. The studied samples indicate a pronounced sludge phenomenon, blood stasis in the venules, breakage of capillaries and local areas of microthrombosis.

These processes form the "no-reflow" phenomenon, in which restoration of patency of a large artery is not accompanied by adequate restoration of microcirculation. It is microangiopathy that explains cases of ineffectiveness of reperfusion therapy, even performed at the optimal time.

Ischemic myocardial damage is characterized by a strict temporal sequence of morphological changes. In the early stages, there is a reversible disruption of ion exchange, edema, disappearance of cross-striations and swelling of mitochondria.

As it progresses, coagulative necrosis develops, characterized by hypereosinophilia of the cytoplasm, fragmentation of nuclei and destruction of sarcomeres. These changes are accompanied by massive neutrophil infiltration.

The acute inflammatory response developing in the necrotic zone plays a dual role: on the one hand, it promotes the removal of damaged tissue, on the other, it enhances the local destruction of myocardial structures. Large numbers of neutrophils promote the formation of free radicals and proteases, which worsen the damage.

Subsequently, neutrophils are replaced by macrophages, which is the beginning of the reparative phase.

The formation of granulation tissue is a critical step in healing. Active capillary sprouting, fibroblast proliferation and collagen deposition create the morphological basis for scar formation.

It is the quality of granulation tissue that determines the strength of the future scar and the risk of late complications, such as aneurysm or myocardial rupture. Myocardial scarring is a long and complex process. In the later stages, a dense collagen framework forms in the affected area, which gradually replaces necrotic tissue.

However, the severity of remodeling depends on the amount of initial damage and the degree of microcirculatory disorders. The discovered differences in the density of collagen fibers and the heterogeneity of the scar structure explain the functional heterogeneity of the post-infarction myocardium

A comparison of morphological changes in different types of infarction showed that transmural forms are accompanied by more pronounced disturbances of the vascular wall and more massive thrombus formation.

In contrast, subendocardial infarctions are characterized by relatively less damage to the epicardial arteries, but more pronounced stasis in the microcirculation. This difference highlights the importance of accurate morphological classification.

Analysis of the state of atherosclerotic plaques demonstrates that stable and unstable plaques have fundamentally different morphologies. Unstable plaques contain a thin fibrous cap, a large lipid core, and significant inflammation. Stable plaques, on the other hand, have a thicker cap and less inflammatory component.

These differences determine the risk of their rupture. Of particular importance is the assessment of the elastic structures of the vascular wall. Their destruction under the influence of proteases leads to loss of elasticity of the artery, which increases the possibility of plaque rupture.

Pathology of elastic fibers was observed in almost all samples with severe thrombosis, which confirms their important role in pathogenesis.

The findings demonstrate that vascular damage during infarction is not limited to the epicardial arteries but also involves profound disruption of the intramuscular microcirculation.

This explains situations where the size of the infarction does not correlate with the level of arterial stenosis, but is determined by the condition of the capillaries and arterioles.

Of interest are cases where a combination of thrombosis and severe vasospasm was detected. This combination creates the most unfavorable conditions for the myocardium, since it reduces the reserve capacity of collateral circulation.

Data confirm that the vasospastic component of infarction is underestimated in clinical practice. Pathology shows that reperfusion injury is an important factor aggravating the course of infarction.

Re-entry of blood into the ischemic area causes free radical stress, endothelial damage and an increase in the area of necrosis. Morphologically, this is expressed by hemorrhagic impregnation and fragmentation of capillaries.

The results of the study highlight the need for a comprehensive analysis, including macroscopic, histological and immunohistochemical evaluation. Only a combination of these methods makes it possible to accurately determine the mechanism of vascular damage and the severity of necrotic changes.

The data obtained also confirm that individual characteristics of the vascular wall structures and the level of systemic inflammation play a key role in the prognosis of infarction. Patients with significant inflammatory activity and unstable plaques are more likely to develop large transmural lesions.

. Summarizing all of the above, it can be argued that the pathomorphology of the heart vessels during myocardial infarction is a dynamic, complex process that determines the clinical picture of the disease, complications and outcome. Understanding the structural mechanisms of damage opens the way to improving preventive and therapeutic technologies.

The study of pathomorphological changes in the vessels of the heart during myocardial infarction allows us to conclude that this pathology is not only a localized lesion myocardial tissue, but also a complex multi-level process, including a whole range of disorders in the coronary arteries and the microcirculation system. The data obtained convincingly show that it is the condition of the vascular wall that largely determines the onset, severity and outcome of a heart attack.

The basis of an acute coronary event is the mechanisms of inflammatory destabilization of the atherosclerotic plaque, impaired endothelial function, activation of the platelet-vascular link and severe microangiopathy, which form a critical decrease in coronary blood flow and trigger the ischemic cascade.

Of particular importance are the identified vascular changes, demonstrating that myocardial infarction is a dynamic process, where different stages - from plaque rupture to scar formation - are closely interrelated.

The structural instability of atherosclerotic lesions and the presence of a large number of activated immune cells and proteolytic enzymes create conditions for disruption of the integrity of the vascular wall and subsequent thrombus formation.

Along with this, the identified microcirculatory disorders confirm that even complete restoration of the patency of the epicardial arteries does not guarantee adequate myocardial reperfusion due to the pronounced sludge phenomenon, blood stasis and microthrombosis.

Morphological analysis demonstrated a clear dependence of the nature of necrosis, the degree of inflammatory response and the rate of scarring on the severity of vascular disorders in the acute period of the disease.

The presence of a massive thrombotic component and severe endothelial damage predetermines the formation of transmural lesions and increases the risk

of fatal complications, including myocardial rupture, aneurysm formation and severe arrhythmias.

On the contrary, less pronounced microcirculatory disorders in subendocardial infarctions are accompanied by a smaller depth of damage, but are characterized by a high probability of recurrent ischemic episodes.

The results obtained emphasize the need for early and comprehensive diagnosis of vascular changes, since timely assessment of the morphological instability of plaques, the degree of endothelial dysfunction and the severity of microthrombosis is crucial for choosing the optimal treatment tactics.

Modern reperfusion methods, antiplatelet and anti-inflammatory therapy must take into account the morphological features of the vascular bed, since the effectiveness of restoring blood flow and preventing recurrent coronary events depends on their correct combination.

Thus, the pathomorphology of cardiac vessels during myocardial infarction reflects deep structural changes that underlie the clinical picture of the disease. The study of these changes is not only of fundamental, but also of applied importance, since it forms scientific the basis for further improvement of methods of prevention, diagnosis and treatment. Understanding the mechanisms of atheroma destabilization, thrombus formation and microangiopathy allows us to predict the course of the disease, reduce the risk of complications and maintain vital myocardial function.

All this makes pathomorphological analysis a key tool in modern cardiology and pathology, determining the accuracy of clinical decisions and the quality of medical care.

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