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Myocardial Infarction Pathogenesis

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Abstract: Myocardial infarction remains a leading cause of morbidity and mortality worldwide, yet gaps persist in understanding its multifactorial pathogenesis and optimal management strategies. While extensive research has explored the role of atherosclerosis and ischemic imbalance, the interplay of thrombotic mechanisms and metabolic disturbances requires further investigation. This study employs a qualitative review methodology, synthesizing existing literature on myocardial infarction classification, pathophysiology, and treatment approaches. The findings confirm that atherosclerosis-induced thrombosis is the predominant cause, with ischemic imbalance exacerbating myocardial damage through metabolic acidosis and impaired cardiac electrophysiology. Results emphasize the need for early risk assessment, advanced thrombolytic therapies, and personalized treatment strategies to mitigate infarct progression. These insights have significant clinical implications, advocating for enhanced diagnostic biomarkers, targeted pharmacological interventions, and preventive measures to reduce cardiovascular disease burden. Future research should focus on molecular mechanisms and precision medicine approaches to improve early detection, treatment efficacy, and long-term patient outcomes.

Keywords: Myocardial infarction, ischemic imbalance, "white" thrombus, cleroderma, rheumatismberlipril, prendopril, enalapril.

Introduction

Etiology: Myocardial infarction is divided into the following types based on international agreements.
Type 1. Spontaneous myocardial infarction. Spontaneous myocardial infarction caused by ischemia due to primary coronary injury (eg, sudden rupture, erosion or rupture of atherosclerotic capillaries; coronary dissection)

Type 2. Myocardial infarction observed as a result of ischemic imbalance. Ischemia due to increased oxygen demand (eg, hypertension) or decreased oxygen delivery (eg, coronary artery spasm or embolism, arrhythmia, hypotension).
Type 3. Fatal myocardial infarction in cases where it is not possible to identify biomarkers. Sudden cardiac death is defined as sudden cardiac death occurring when myocardial ischemia is suspected or new bundle branch block is detected on ECG.

Type 4a. Myocardial infarction associated with percutaneous coronary
intervention in the heart and blood vessels. Diagnosed when a previously normal troponin value exceeds the 5 × 99th percentile or greater than 20% of normal after percutaneous interventions.
Type 4b. Myocardial infarction associated with thrombus of stent in coronary vessels.

Associated with an implanted stent or clinical myocardial ischemia, confirmed stent thrombosis, and changes in the dynamics of cardiospecific enzymes

Type 5. Myocardial infarction associated with aortic-coronary shunting. It is based on a high cardiac troponin index (10 × 99) or an increase of 20% or more of the existing index of this biomarker.

Materials and Methods

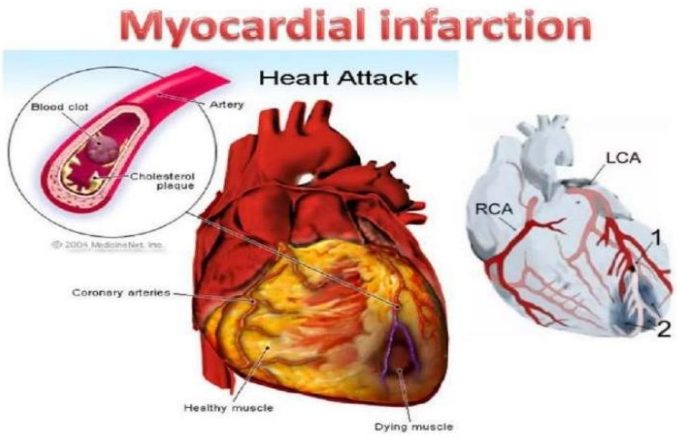
The methodology for this study on myocardial infarction pathogenesis employs a qualitative research approach, focusing on a comprehensive review of existing literature and clinical studies. The research process involved an in-depth analysis of academic sources, including textbooks, peer-reviewed journal articles, and medical guidelines, to synthesize current knowledge on the etiology, classification, and pathogenesis of myocardial infarction. The study critically examines myocardial infarction types based on international classifications, highlighting spontaneous cases, ischemic imbalance-related infarctions, fatal infarctions without biomarker identification, and infarctions associated with coronary interventions and bypass surgeries. The data collection process incorporated studies on thrombus formation, coronary artery occlusion mechanisms, and the metabolic disturbances resulting from ischemia, with particular attention given to the role of atherosclerosis as the predominant cause. Clinical observations and experimental findings were analyzed to understand the progression of myocardial infarction from the prodromal stage to post-infarction recovery. The methodology integrates pathophysiological insights by examining myocardial ischemia's impact on cardiac electrophysiology, metabolic disturbances, and necrosis expansion. Pharmacological treatment strategies, including thrombolytics, anticoagulants, and antiarrhythmic agents, were reviewed to assess their effectiveness in preventing infarct progression. Preventive measures, such as lifestyle modifications and risk factor control, were also evaluated based on existing medical recommendations. By synthesizing multidisciplinary perspectives from cardiology, internal medicine, and pathology, this study aims to provide a structured understanding of myocardial infarction mechanisms and therapeutic approaches. The findings contribute to improving early diagnosis, intervention strategies, and patient management to mitigate the burden of myocardial infarction.

Results and Discussion

Features of the course of the disease:

- is primary
- again
- relapsing

Location of myocardial infarction: Anterior septum, anterior lateral wall, anterior basal (front upper), anterior diffuse (anterior septal area, apex and anterior lateral wall); Backdiaphragmatic, back-basal, back-side wall, back-spread (back-diaphragm and back-side wall), side wall, right ventricle;



Periods of illness:

- prodromal - pre-infarction period 0-18 days (up to 1 month);
- extremely acute period - up to 2 hours from the onset of myocardial infarction;
- acute period - up to 10 days from the onset of myocardial infarction;
- subacute period - from 10 days to the end of 4-8 weeks;
- period after infarction - in most cases from 4-8 weeks to 6 months.

Pathogenesis: Myocardial infarction is based on atherosclerosis of coronary arteries (in most cases), spasm and bleeding in atherosclerotic wrists. Its main cause is atherosclerosis in 95% of cases. The remaining 5% are caused by coronary embolism (infective endocarditis, intraventricular thrombus) and congenital and acquired other types of damage (systemic lupus erythematosus, scleroderma, rheumatism, and coronary arteritis in rheumatoid arteritis), and in this case, myocardial infarction is a separate clinical form of MI. rather than as a complication of the main disease. In most cases, a sudden stop or limitation of blood circulation in the coronary vessels occurs due to thrombosis formed in the area of "complicated" atherosclerotic plaque in them. It activates tissue thromboplastin and collagen and blood platelet and plasma coagulation factors. First, a walled thrombocytic "white" thrombus is formed. At the same time, a number of biologically active substances with a strong vasoconstrictor effect (endothelin, serotonin, thrombin, antithrombin A2) are released in this area. In addition, small platelet aggregates lead to embolism of small coronary vessels and further deteriorate the coronary circulation. If the natural activation of the fibrinolytic system and spontaneous dissolution of the thrombus as a result of thrombolytic treatment is not observed in the patient, it gradually enlarges and completely closes the vessel opening and Q-shaped (transmural) MI develops. If, for some reason, the coronary artery is not completely closed or if the thrombus suddenly dissolves on its own (even in cases where the large coronary vessels are completely closed and the collaterals are well developed), subendocardial or intramural (without Q wave) MI develops.
In some cases (75%), the process of thrombus formation, which completely closes the coronary artery, lasts from 2 days to 2-3 weeks. This period corresponds to the passing of unstable angina from a clinical point of view (pre-infarction syndrome). In very rare cases (in ¼ of patients), the process of formation of a thrombus, which completely blocks the vessel, takes place at lightning speed. Such patients do not have a pre-infarction (prodromal) period.

The development of acute necrosis in the heart muscle can be caused by the following conditions:

- Obvious spasm of coronary vessels;
- Collateral vascular system is not well developed;
- Sudden increase in oxygen demand of the myocardium due to physical and emotional stress, sudden increase in heart rate and other reasons. These 3 factors cause the center of necrosis to grow and increase in size. On the contrary, MI may not develop in some cases even as a result of complete occlusion of the coronary artery, which occurs slowly when collateral blood circulation is well developed.

Cardiac muscle ischemia causes serious metabolic disturbances in cardiomyocytes and cells of the cardiac conduction system. Hypoxia develops when blood circulation in the coronary vessels decreases or stops altogether, and this change has a negative effect on the synthesis of ATF in mitochondria. In this case, the contractility of cardiomyocytes decreases rapidly. The arrival of fatty acids and glucose, which are energy substrates of cells, in very small quantities through coronary blood flow activates the process of anaerobic glycolysis. Glycogen metabolism is broken down only to lactate, which is not further oxidized in oxygen deficiency. An increase in the concentration of lactate and other non-oxidized products in the blood of the heart muscle and coronary sinus leads to acidosis, a shift of pH in the myocardium towards acidity, which further

reduces its electrical activity and contractility. These changes are different in the areas of the myocardium affected by ischemia, creating conditions for the electrophysiological properties of cardiomyocytes to be different, which causes heart rhythm disturbances.

Treatment. All patients with myocardial infarction are hospitalized in special intensive care units of the hospital. Treatment measures should be aimed at the complete elimination of pain syndrome, prevention of heart rhythm and conduction disturbances, and limitation of the focus of necrosis. To achieve this goal, patients are given thrombolytics (streptokinase, streptodecasa), anticoagulants (heparin, fraxiparin), antiaggregants (aspirin, cardiomagnyl, stasis (clopidogrel), nitrates (nitrosorbide, nitrong, monosan, olicard), P-blockers (atenolol, egilok, nebilet). , AAFI (berlipril, prendopril, enalapril), antiarrhythmic (kordarone, P-blockers, allapinin) drugs are prescribed in individual doses depending on the patient's condition. When recommending them, it is necessary to take into account the presence of indications and contraindications.

Prevention. Elimination of risk factors that lead to ischemic heart disease: smoking cessation, weight control, consumption of a diet low in animal fat, concomitant treatment of hyperglycemia and diabetes, control of blood uric acid and hypercholesterolemia. modification.

Conclusion

The study on myocardial infarction pathogenesis underscores the critical role of atherosclerosis as the primary cause, with thrombosis and ischemic imbalance serving as key contributors to disease progression. The findings highlight the multifactorial nature of myocardial infarction, encompassing spontaneous coronary events, procedural complications, and ischemia-induced infarctions, all of which necessitate timely diagnosis and intervention. The metabolic disturbances induced by ischemia, particularly the shift towards anaerobic glycolysis and subsequent acidosis, further exacerbate myocardial damage and increase the risk of adverse cardiac events. These insights have significant clinical implications, emphasizing the necessity for early risk assessment, improved thrombolytic strategies, and personalized treatment regimens that address the underlying pathophysiology. Furthermore, preventive measures, including lifestyle modifications and pharmacological interventions, are essential in mitigating disease incidence and severity. Given the complexity of myocardial infarction, further research is warranted to explore novel biomarkers for early detection, optimize pharmacological therapies, and develop targeted interventions that enhance myocardial recovery and prevent recurrent infarctions. Advancements in molecular cardiology and precision medicine may offer new avenues for improving patient outcomes and reducing the global burden of cardiovascular diseases.

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