JEDIC
Journal of Ethics and Diversity in International Communication
| e-ISSN: 2792-4017 | www.openaccessjournals.eu | Volume: 3 Issue: 2

# Late Complication of Myocardial Infarction—Dressler's Syndrome, (Patient Complaints, Modern Methods of Treatment)

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**Abstract**. In this article, the late complication of Myocardial infarction—the clinical presentation of Dressler's syndrome (patient complaints, modern methods of treatment) is described in detail.

**Key words:** myocardial infarction, Dressler's syndrome, modern treatment methods, etc.

Myocardial infarction is based on atherosclerosis of coronary arteries (in most cases), spasm and bleeding into atherosclerotic plaques. Its main cause is atherosclerosis in 95% of cases. In the remaining 5%, coronary embolism (infective endocarditis, intraventricular thrombus) and congenital and acquired other types of damage (systemic lupus erythematosus, scleroderma, rheumatism and coronary arthritis in rheumatoid arthritis) lie, and myocardial infarction is not a separate clinical form of YUIK, but considered as its complication. 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. It activates tissue thromboplastin and collagen and blood platelet and plasma clotting factors. First, a premural 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 worsen coronary circulation. If the patient's natural activation of the fibrinolytic system or spontaneous dissolution (lysis) of the thrombus as a result of thrombolytic treatment is not observed, it gradually enlarges and completely closes the vessel opening, and a Q-shaped (transmural) MI develops. Subendocardial or intramural (without Q-wave) MI is formed if the coronary artery is not completely closed for some reason or if the thrombus suddenly dissolves on its own (even when the large coronary vessels are completely closed and the collaterals are well developed).

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 (a quarter of patients), the process of formation of a thrombus, which completely closes 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 three conditions:

- Marked spasm of coronary vessels;
- Collateral vascular system is not well developed;
- Due to physical and emotional stress, a sharp rise in blood pressure and other reasons, a sharp increase in myocardial oxygen demand. These three factors cause the center of necrosis to grow and increase in size. On the other hand, MI may not develop in some cases even as a result of the complete occlusion of the coronary artery, which occurs gradually when the collateral blood circulation is well developed.

Palpation and percussion of the heart: palpation reveals local pain in the left precardial area, if the patient has no history of diseases that can cause left ventricular enlargement (AG, atherosclerotic or post-infarction cardiosclerosis, etc.), during the acute period of MI, the heart expansion of the borders is not observed. Auscultation of the heart: due to the decrease in contractility of the left ventricular myocardium affected by ischemia, there is a weakening of the I tone at the peak of the heart. Second tone decreases due to slowing of early diastolic relaxation of the left ventricle or a decrease in pressure in the aorta. Due to the increase in pressure in the pulmonary artery as a result of venous damping within the small blood circulation, an accent of the II tone is determined in its projection. In some patients, an accent of tone II can be heard in the aorta due to obvious atherosclerosis of the aorta. When acute vascular insufficiency develops, BP decreases mainly due to systolic pressure. At the same time, the fullness and height of the pulse decreases and accelerates.

Dressler syndrome is a type of pericarditis, an inflammation of the sac that surrounds the heart (pericardium). Inflammation is a sign of protecting the heart from damage caused by a heart attack, surgery, or trauma. Treatment includes pain management and inflammation reduction. Recommended treatment includes medication or even surgery if complications develop. People who have had a recent heart attack, recent heart surgery, or heart procedures are more prone to Dressler syndrome. Other terms used for Dressler syndrome are post-injury syndrome, post-myocardial infarction syndrome, or post-pericardiotomy syndrome. Dressler syndrome can occur 2-6 weeks after a heart attack or heart surgery. However, signs and symptoms may take several months to appear. Various tests are used to diagnose Dressler syndrome, such as EKG, complete blood count, chest X-ray, cardiac MRI and CT, echocardiogram, etc. Treatment for Dressler's syndrome involves the use of non-steroidal anti-inflammatory drugs (NSAIDs). Anti-inflammatory medications such as aspirin, ibuprofen, naproxen, and others can help reduce pain and inflammation. Aspirin is the most common NSAID used to treat Dressler syndrome. Depending on the patient's condition, other NSAIDs may be preferred.

The acute period is the time from the onset of the first clinical and/or instrumental (ECG) signs of acute myocardial ischemia to the formation of the necrosis center (from 30 minutes to 2 hours). During this period, it is possible to reverse the morphological changes in the myocardium and prevent the formation of a necrosis center by timely use of thrombolytic drugs that allow the restoration of blood flow in the coronary vessels. The first clinical sign of MI is associated with the completion of the process of thrombotic occlusion of the coronary artery or its sudden occurrence (in the absence of symptoms of the prodromal period in the patient). There are several clinical types of its onset: painful (status anginosus); asthmatic (status asthmaticus); abdominal (status abdominalis); arrhythmic; cerebrovascular; less symptomatic (symptomatic) type. In 80-95% of patients, MI begins with angina - painful type. At its beginning, it is common to have an attack of severe pain in the heart. Strong, pressing, pressing, burning, "dagger" pains are mainly located behind the sternum, are transmitted to the left arm, under the shoulder blade, to the jaw, are

accompanied by a feeling of fear of death, and in most cases It lasts 40-60 minutes, and sometimes hours (status anginosus). Multiple doses of nitroglycerin, long-acting nitrates, and non-narcotic analgesics are ineffective. Pain usually decreases or disappears after narcotic analgesics. They sometimes pass in waves, that is, after a severe pain lasting 20-30 minutes, there is a break of 10-15 minutes, and a strong attack begins again. In some cases, it can go away on its own even if no help is provided.

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