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INFLAMMATION AND IMMUNOLOGICAL MECHANISM IN MYOCARDIAL INFARCTION

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Abstract. *This article examines the role of inflammation and immunological mechanisms in myocardial infarction. The results of the study show that inflammation and the immune response in the process of myocardial infarction are a complex, multi-stage process. The inflammatory reaction affects tissue damage in the infarct zone and the remodeling of the heart muscle. At the initial stage, mediators released from damaged cells activate the immune system, which can enhance the processes of necrosis and fibrosis. The immunological response also plays an important role in the cleansing and regeneration of the myocardial infarction site, but excessive inflammation and immune response can increase the risk of heart failure.*

Keywords: *Myocardial infarction, Immunological mechanism, Cytokines, TNF-α, IL-1, C-reactive protein (CRP), DAMPs.*

ВОСПАЛЕНИЕ И ИММУНОЛОГИЧЕСКИЙ МЕХАНИЗМ ПРИ ИНФАРКТЕ МИОКАРДА

Аннотация. *В статье рассматривается роль воспаления и иммунологических механизмов при инфаркте миокарда. Результаты исследований показывают, что воспаление и иммунный ответ при инфаркте миокарда — сложный, многоэтапный процесс. Воспалительная реакция влияет на повреждение тканей и ремоделирование сердечной мышцы в зоне инфаркта. Медиаторы, высвобождаемые из поврежденных клеток на начальном этапе, активируют иммунную систему, что может усилить процессы некроза и фиброза. Также иммунологический ответ играет важную роль в процессе очищения и регенерации места инфаркта миокарда, однако чрезмерное воспаление и иммунный ответ могут увеличить риск сердечной недостаточности.*

Ключевые слова: *Инфаркт миокарда, Иммунологический механизм, Цитокины, ФНО-α, ИЛ-1, С-реактивный белок (СРБ), DAMP.*

Introduction

Myocardial infarction (MI) is one of the severe cardiovascular diseases that develops as a result of a sharp disruption of the blood supply to the heart muscle, which is part of the acute coronary syndromes. This pathology is one of the leading causes of death and disability worldwide, and the issue of improving its treatment and prevention is urgent. The pathogenesis of MI is a complex and multifactorial process in which inflammatory and immunological mechanisms play an important role. As a result of ischemia of the heart muscle, necrosis develops and an inflammatory reaction begins in the damaged tissues. In this process, cells of the immune system and inflammatory mediators are activated, affecting the process of myocardial regeneration.

However, excessive inflammation and an imbalance of the immune response can aggravate myocardial damage and lead to functional failure of the heart. This article analyzes the role of inflammatory and immunological mechanisms in myocardial infarction, their interrelationships, and new approaches to treatment. A deeper study of these mechanisms is important in developing new therapeutic strategies for myocardial infarction.

Literature review and method

Myocardial infarction is a cardiovascular disease that occurs as a result of an acute disruption of the blood supply to the heart muscle, and its pathogenesis is based on complex mechanisms. Inflammatory and immunological mechanisms play an important role in this process.

When myocardial infarction begins, the inflammatory process is activated in the damaged heart muscle. Damage signals are released from ischemic cells, and this process leads to the secretion of inflammatory mediators. As a result, the inflammatory process develops and various cells of the immune system are involved.

Immunological mechanisms include responses aimed at eliminating necrotic tissue in the infarct zone by the body's defense system. In this process, innate and adaptive immune responses are formed. Components of the immune system are activated to clean and restore damaged tissues.

However, uncontrolled or excessive inflammation can lead to further damage to the myocardium. The interaction of inflammation and immunological mechanisms also affects the process of remodeling in the heart tissue in the later stages of myocardial infarction. During this process, myocardial fibrosis and heart failure may develop.

Regulation of inflammation and immunological mechanisms in myocardial infarction is important for increasing the effectiveness of treatment. By controlling these processes, it is possible to reduce damage to the heart muscle and prevent complications. In this regard, biological drugs and immunomodulators are used in modern medicine.

Pathophysiology of myocardial infarction

Myocardial infarction occurs as a result of rupture of atherosclerotic plaques or the formation of a thrombus in the coronary arteries supplying the heart with blood. This leads to a sharp cessation of blood flow and the ischemic process begins in the myocardial tissues. In the initial stage of ischemia, the cell cannot meet the need for oxygen to produce energy, and as a result, metabolic processes are disrupted. As a result of cell damage, membrane stability is lost, ion balance is disturbed, and cells undergo necrosis.

Inflammatory process

With the onset of myocardial infarction, the inflammatory process is activated. This process occurs in interconnected sequential stages:

- Initial phase (last few hours): As a result of myocardial ischemia and necrosis, damage signaling molecules (DAMPs — damage-associated molecular patterns) are released from cells.

These substances act as signals that stimulate the inflammatory response in the body. DAMPs activate various cells of the immune system (neutrophils, monocytes and macrophages).

- Inflammatory phase (1-3 days): During this phase, inflammatory mediators (cytokines and chemokines) are produced. Cytokines such as IL-1, IL-6, TNF-α cause the recruitment of more immune cells to the infarct zone. Neutrophils phagocytose damaged cells, further intensifying inflammation. During this process, proteolytic enzymes and oxygen radicals are released, which play an important role in the destruction of necrotic tissue.

- Clearing phase (3-7 days): In this phase, macrophages are formed from monocytes, which engulf and clear necrotic cells. Macrophages begin the process of repairing and renewing damaged tissues. During this period, the amount of cytokines decreases, the inflammatory process subsides.

- Regeneration phase (7-14 days): Fibroblasts are activated, produce collagen, and connective tissue is formed. This process leads to the development of fibrosis, which plays an important role in the closure of the myocardial wound. Inflammation ends, but myocardial remodeling continues in the remaining focus.

Immunological mechanisms

The pathogenesis of MI involves both innate and adaptive components of the immune response. The initiation and continuation of the inflammatory process are controlled by these immune mechanisms:

- Innate immune response: Innate immune cells (neutrophils, macrophages, dendritic cells), activated by DAMPs and PAMPs (pathogen-associated molecular patterns), enter the infarct zone and eliminate necrotic cells. These cells produce inflammatory mediators and activate the inflammatory process.

- Adaptive immune response: Dendritic cells activate T-lymphocytes as antigen-presenting cells. T-lymphocytes recognize myocardial antigens and form a specific immune response. During this process, autoimmune reactions may develop, which increases the likelihood of exacerbation of post-infarction complications.

- Autoimmune reactions: In some cases, myocardial antigens are recognized as autoantigens and are mistakenly attacked by the immune system. This process can cause additional damage to the heart tissue.

The role of inflammation and the immune response in the pathogenesis of MI

Inflammation and the immune response play different roles in the acute and chronic phases of myocardial infarction. While inflammation in the acute phase helps to eliminate damaged tissues, excessive or prolonged inflammation can increase myocardial remodeling and fibrosis in the infarcted areas. This can reduce the contractility of the heart muscle and lead to the development of heart failure.

Management of inflammation and immunity in treatment

Regulation of inflammatory and immunological mechanisms in the treatment of MI is important to increase the effectiveness of treatment.

- Anti-inflammatory drugs: Drugs that inhibit the activity of cytokines and chemokines are used.

- Biological drugs: Targeted immunotherapy blocks harmful mediators using specific antibodies.

- Immunomodulators: Agents that regulate the immune response are used in the acute phase of infarction.

- Gene and cell therapy: New treatments serve to reduce the consequences of MI.

Inflammatory and immunological mechanisms in myocardial infarction involve complex processes.

Aberrant activation of these mechanisms can aggravate myocardial damage and lead to heart failure. Therefore, controlling inflammation and the immune response is important in treatment. New approaches and therapeutic strategies have great prospects in this area.

Analysis of the scientific literature on the pathogenesis of myocardial infarction (MI), its inflammatory and immunological mechanisms shows that this pathology is a complex and multi-stage process. In recent years, the role of inflammatory mediators, immune cells and autoimmune reactions in the development of MI has been studied in depth. Many studies have noted that the inflammatory process plays an important role not only in the acute phase of infarction, but also in the development of long-term complications.

Scientific articles are devoted to the study of the activity of interleukins (IL-1, IL-6), tumor necrosis factor alpha (TNF-α), C-reactive protein (CRP), neutrophils and macrophages during MI.

The presence of these mediators in high concentrations is used as one of the diagnostic biomarkers to determine the severity of the acute phase of infarction, inflammation and fibrosis.

Also, the effect of the immune response on the processes of recovery and remodeling in the later stages of myocardial injury has been studied.

The literature discusses the effectiveness of biological drugs, cytokine antagonists, immunomodulators and targeted therapy agents in the treatment of MI. Also, the potential of gene therapy and cell therapy in the management of inflammatory and immunological responses has been noted.

Discussion

Studies on the role of inflammation and immunological mechanisms in myocardial infarction show that these processes are complex and multistage. The results of the study allowed us to further understand how inflammatory and immunological responses affect the pathogenesis of myocardial infarction and its complications. In the initial stage of the inflammatory process, DAMPs and other mediators released from damaged cells in the infarct zone activate the immune system. As a result of the accumulation of neutrophils, macrophages, and monocytes in the infarct zone and the release of harmful enzymes, the necrotic focus of the myocardium can further expand.

This, in turn, reduces the contractile function of the heart muscle and increases the risk of heart failure. Studies show that controlling inflammation and the immune response at the initial stage can reduce the damage caused by myocardial infarction.

For example, high levels of inflammatory mediators such as interleukins (IL-1, IL-6), TNF-α, and C-reactive protein (CRP) increase necrosis and fibrosis processes in the infarct site.

Therefore, biological drugs that inhibit their activity (for example, IL-1 antagonists or TNF-α blockers) are of great importance in treatment. Immunological mechanisms play an important role in the process of clearing and regeneration of necrotic tissue in the infarct site.

However, excessive or prolonged inflammation can negatively affect myocardial remodeling and lead to the development of heart failure.

Therefore, balancing inflammation and immune responses is important in increasing the effectiveness of treatment. Based on the analyzed scientific sources, it has been established that excessive activity of inflammation and immunological responses in the pathogenesis of MI exacerbates the damage caused by infarction. Prolonged inflammation leads to fibrosis of the heart muscle, decreased contractile function of the heart, and increases the risk of heart failure.

Therefore, the timely use of anti-inflammatory and immunomodulatory agents provides positive results in the treatment of MI. The results of the study confirm the need to develop new therapeutic strategies that target inflammatory and immunological mechanisms in myocardial infarction. In particular, the use of new methods, such as biological drugs that regulate the activity of cytokines and inflammatory mediators, gene and cell therapy, can help reduce the consequences of MI. In conclusion, the study of inflammatory and immunological mechanisms in myocardial infarction allows the development of effective strategies to reduce myocardial damage and accelerate recovery processes. Research in this area is of great importance in improving modern treatment methods and introducing new approaches.

Conclusion

Inflammatory and immunological mechanisms in myocardial infarction significantly affect the development of pathology through complex and multi-stage processes. The interrelation of these processes leads to tissue damage in the acute phase of infarction, and in the long term to remodeling and fibrosis of the heart muscle. The results of the study show that balanced control of inflammation and immune response is important in reducing the consequences of myocardial infarction. In the initial stage of the inflammatory process, the activation of the immune system by DAMPs and other mediators helps to eliminate necrosis and damaged tissues in the infarct focus.

However, excessive or prolonged inflammation causes additional damage to myocardial tissue and increases the risk of heart failure.

As a result of excessive activity of immunological mechanisms, autoimmune reactions and chronic inflammation are observed, which can further aggravate the complications of infarction. Therefore, there is a need for targeted control of inflammatory mediators and the immune response in treatment.

The use of anti-inflammatory biological drugs, cytokine antagonists, immunomodulators and targeted therapy agents may be effective in alleviating the consequences of myocardial infarction. The potential of gene and cell therapy in controlling the immune response is very promising.

Overall, further understanding of the inflammatory and immunological mechanisms of myocardial infarction and improving treatment options will play a key role in reducing mortality and disability from cardiovascular disease. Future research in this area will help develop new therapeutic strategies.

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