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THE IMPORTANCE OF IMMUNE INFLAMMATION IN THE DEVELOPMENT OF SENILE AORTIC STENOSIS

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Abstract

Senile aortic stenosis (SAS) is a heart disease associated with narrowing of the aortic valve, which is usually age-related and causes serious cardiovascular problems. The role of immune inflammation in the development of SAS has been confirmed by scientific studies. Inflammatory processes can cause fibrosis and calcification of the valve tissue, leading to narrowing of the valve. Many studies show that patients with SAS have altered immune system activity, inflammatory foci, and cell migration. These inflammatory processes lead to a change in the structure of the valve and a decrease in its function. Thus, immune inflammation plays an important role in the development of SAS, and scientific research and new approaches are needed to improve or prevent this condition.

Keywords: SAS- Senile aortic stenosis, inflammation, immunotherapy.

SENIL AORTAL STENOZINING RIVOJLANISHIDA IMMUN YALLIG'LANISHNING AHAMIYATI

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Annotatsiya

Senil aorta stenozi (SAS) aorta qopqog'ining torayishi bilan bog'liq bo'lgan yurak kasalligi bo'lib, odatda yoshga bog'liq va jiddiy yurak-qon tomir muammolarini keltirib chiqaradi. SAS rivojlanishida immun yallig'lanishning roli ilmiy tadqiqotlar bilan tasdiqlangan. Yallig'lanish jarayonlari fibroz va qopqoq to'qimalarining kalsifikatsiyasiga olib kelishi mumkin, bu esa qopqoqning torayishiga olib keladi. Ko'pgina tadqiqotlar shuni ko'rsatadiki, SAS bilan og'rigan bemorlarda immunitet tizimi faolligi, yallig'lanish o'choqlari va hujayralar migratsiyasi o'zgargan. Ushbu yallig'lanish jarayonlari vana tuzilishining o'zgarishiga va uning funktsiyasining pasayishiga olib keladi. Shunday qilib, immun yallig'lanish SAS rivojlanishida muhim rol o'ynaydi va bu holatni yaxshilash yoki oldini olish uchun ilmiy tadqiqotlar va yangi yondashuvlar zarur.

Kalit so'zlar : SAS- Senil aorta stenozi, yallig'lanish, immunoterapiya.

ЗНАЧЕНИЕ ИММУННОГО ВОСПАЛЕНИЯ В РАЗВИТИИ СТАРЧЕСКОГО СТЕНОЗА АОРТЫ

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Аннотация

Старческий аортальный стеноз (САС) – заболевание сердца, связанное с сужением аортального клапана, которое обычно является возрастным и вызывает серьезные сердечно-сосудистые проблемы. Роль иммунного воспаления в развитии САС подтверждена научными исследованиями. Воспалительные процессы могут привести к фиброзу и кальцификации ткани клапана, что приводит к его сужению. Многие исследования показывают, что у пациентов с САС изменена активность иммунной системы, воспаление и миграция клеток. Эти воспалительные процессы приводят к изменению структуры клапана и снижению его функции. Таким образом, иммунное воспаление играет важную роль в развитии САС, и необходимы научные исследования и новые подходы для улучшения или предотвращения этого состояния.

Ключевые слова: САС-сенильный аортальный стеноз, воспаление, иммунотерапия.

Introduction. In recent years, many studies have been conducted on the role of immune inflammation in the development of SAS. This article reviews the role of the immune system in the development of SAS and analyzes the influence of inflammatory mediators, cellular responses, and autoimmune processes on the pathogenesis of SAS[1]. Also SAS will provide information on immune-inflammation control methods, new therapeutic targets, and prospects for immunotherapy. These data may be important for the development of new strategies for the treatment of SAS. Senile aortic stenosis is a heart valve disease common among elderly people, which is characterized by degenerative and calcific changes in the aortic valve [2].

Main body. Although the development process of SAS was initially considered as a degenerative process without the involvement of inflammation, recent studies have shown that immune-inflammatory processes play an important role in the pathogenesis of this disease [3]. Inflammatory mediators and cell reactions, along with calcium deposits in aortic valve tissue, are one of the main factors shaping the pathogenesis of SAS. This article examines the role of immune-inflammatory processes in SAS. Understanding how the immune system and inflammatory processes are related to the pathophysiological changes in the aortic valve will provide new opportunities for the development of effective treatment strategies for SAS [4,5,6]. At the same time, the prospect of targeting immune-inflammatory processes as new targets in the treatment of SAS is of great importance. Recent

studies show that this process is not limited to mechanical degeneration but inflammatory and immune processes are also prominent as the main pathogenetic factors. The calcification process begins with endothelial cell dysfunction, lipoprotein accumulation, and activation of inflammatory markers. The importance of immune-inflammatory processes in the pathogenesis of SAS has been confirmed in many studies [7]. The process of calcification in the tissues of the aortic valve is not only mechanical, but also controlled by inflammatory mediators. Inflammatory markers such as C-reactive protein (CRP), interleukins (IL-1, IL-6), TNF-alpha play an important role in the development of SAS. These markers enhance aortic valve calcification changes associated with SAS and trigger immune-inflammatory processes [8,9,10,11]. Also, some of the inflammation in SAS is related to autoimmune processes, which cause the body to produce antibodies against its own tissues. This process accelerates the development of SAS and enhances the calcification process. Autoimmune reactions are important in the early stages of the development of SAS, and their analysis may provide a basis for new therapeutic strategies. Inflammatory mediators play an important role in the development of SAS. The inflammatory process in the aortic valve begins with the infiltration of monocytes and macrophages, these cells trigger the calcification process [12]. At the same time, inflammatory mediators, including interleukins and chemokines, activate fibroblasts and myofibroblasts in the valve tissue, accelerating their transformation into calcification. The role of these mediators in the pathogenesis of SAS is particularly evident in the calcified aortic valve. Inflammatory markers such as interleukins (IL-1, IL-6) and TNF-alpha stimulate the transformation of fibroblasts into osteoblasts, which leads to the accumulation of calcium and hardening of the valve [13,14]. Therefore, controlling immune inflammation in SAS may be important in slowing disease progression. Targeting immune-inflammatory processes in the treatment of SAS opens up new therapeutic opportunities. A number of anti-inflammatory drugs (eg, statins, omega-3 fatty acids) have been found to reduce the inflammatory processes associated with SAS. But in recent years, anti-inflammatory biological therapy, such as interleukin antagonists and TNF-alpha blockers, has been proposed as a new strategy in the treatment of SAS. In addition, the possibility of treating SAS through immunotherapy is being investigated. Immunomodulatory therapies, such as antibody-based therapies, may be promising in the future for patients with SAS. These approaches are aimed at slowing the progression of SAS by controlling inflammatory processes [15,16,17,18].

Conclusion. Senile aortic stenosis (SAS) is a progressive disease primarily driven by calcification and degenerative changes, but growing evidence points to immune-mediated inflammation as a key player in its pathogenesis. The activation of inflammatory pathways, the presence of immune cells within the aortic valve, and the

role of inflammatory mediators suggest that SAS shares mechanisms with other chronic inflammatory conditions, such as atherosclerosis. Understanding the significance of immune inflammation in the progression of SAS not only sheds light on the underlying biology of the disease but also opens new therapeutic avenues [19,20]. Targeting the immune-inflammatory response may offer novel strategies to slow disease progression and improve patient outcomes. Anti-inflammatory therapies, including biologics and immunomodulatory drugs, hold potential in addressing the chronic inflammatory state that underlies valve calcification. Further research is necessary to fully elucidate the complex interactions between immune cells, inflammatory mediators, and calcific processes in SAS. A deeper exploration of these mechanisms may lead to more effective, targeted treatments that could delay or prevent the need for surgical intervention in patients with senile aortic stenosis [21,22,23].

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