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- 14.00.28 - Neyroxirurgiya;
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- 14.00.34 - Yurak-qon tomir xirurgiyasi

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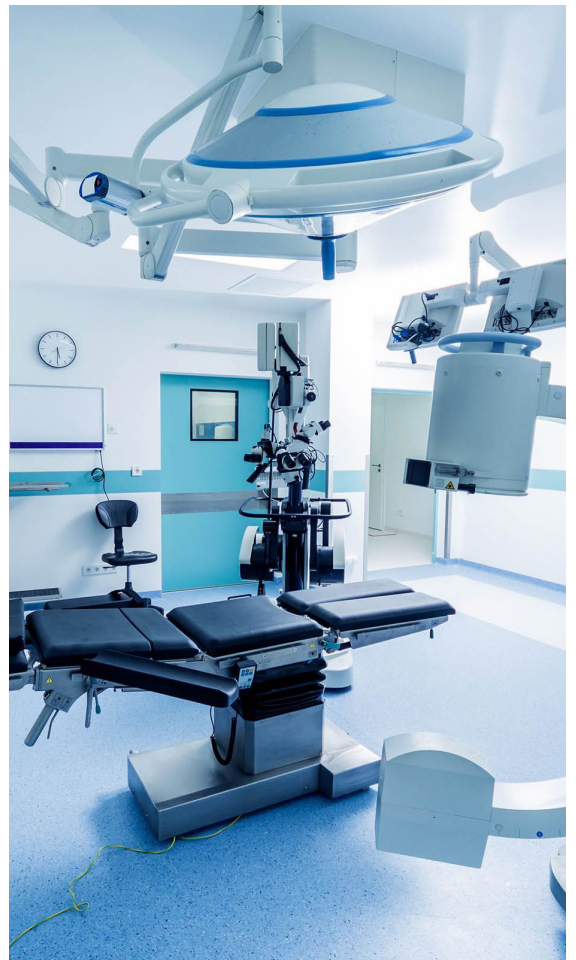
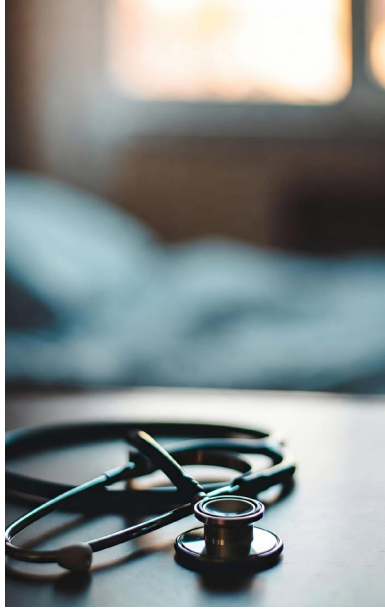


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KARDIORENAL SAMARADORLIK NUQTAI NAZARIDAN FINERENON VA EPLERENONNING CHAP QORINCHA OTISH FRAKSIYASI SAQLANGAN YURAK YETISHMOVCHILIGI BEMORLARIDAGI QIYOSIY BAHOLANISHI.....	21
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EFFECTIVENESS OF TREATMENTS FOR CORONARY ARTERY DISEASE

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Abstract: This review is to evaluate the current evidence on the effects of biomaterials on different cell types involved in cardiac repair and regeneration following myocardial infarction. Particular attention is given to the interactions of biomaterials with cardiomyocytes, fibroblasts, endothelial cells, macrophages, cardiac progenitor cells, and mesenchymal stromal cells, as well as their effects on inflammation, angiogenesis, fibrosis, extracellular matrix remodeling, and cellular survival. A structured literature review was conducted to identify and evaluate studies investigating the effects of biomaterials on cellular processes involved in cardiac repair and regeneration following myocardial infarction. The review was designed in accordance with the principles of the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA 2020) guidelines. The literature demonstrates a substantial expansion of biomaterial-based approaches for myocardial repair following myocardial infarction (MI). The biological effects of biomaterials were found to depend strongly on their composition, mechanical properties, degradation characteristics, architecture, surface chemistry, and the presence of incorporated biological signals. Biomaterials can support cardiomyocyte survival, promote endothelial cell-mediated angiogenesis, regulate macrophage-driven inflammation, modulate fibroblast activation and extracellular matrix remodeling, and improve the retention and regenerative activity of stem and mesenchymal stromal cells. These coordinated cellular effects may contribute to reduced adverse ventricular remodeling and improved structural and functional recovery of the injured myocardium.

Key words: Myocardial infarction, biomaterials, inflammatory cardiovascular diseases, hyaluronic acid.

Annotatsiya: Ushbu sharhda miokard infarktidan keyin yurak to'qimasini tiklash va regeneratsiya qilishda ishtirok etadigan turli hujayra turlariga biomateriallarning ta'siri bo'yicha mavjud ma'lumotlar baholanadi. Biomateriallarning kardiomiotsitlar, fibroblastlar, endotelial hujayralar, makrofaglar, yurak progenitor hujayralari va mezenximal stromal hujayralar bilan o'zaro ta'siriga, shuningdek, ularning yallig'lanish, angiogenez, fibroz, hujayradan tashqari matritsani qayta qurish va hujayralarning omon qolishiga ta'siriga alohida e'tibor qaratiladi. Miokard infarktidan keyin yurak to'qimasini tiklash va regeneratsiya qilishda ishtirok etadigan hujayra jarayonlariga biomateriallarning ta'sirini o'rganuvchi tadqiqotlarni aniqlash va baholash uchun tuzilgan adabiyotlar sharhi o'tkazildi. Sharh PRISMA 2020 (Tizimli sharhlar va meta-tahlillar uchun afzal ko'rilgan hisobot elementlari) ko'rsatmalariga muvofiq ishlab chiqilgan. Taqdim etilgan adabiyotlarda miokard infarktidan (MI) keyin miokardni tiklashga biomaterial yondashuvlarini qo'llashda sezilarli



kengayish kuzatilgan. Biomateriallarning biologik ta'siri ularning tarkibi, mexanik xususiyatlari, parchalanish xususiyatlari, arxitekturasi, sirt kimyosi va kiritilgan biologik signallarning mavjudligiga sezilarli darajada bog'liqligi aniqlangan. Biomateriallar kardiomiotsitlarning omon qolishini qo'llab-quvvatlashi, endotelial hujayralar vositasida angiogenezni rag'batlantirishi, makrofaglar keltirib chiqaradigan yallig'lanishni tartibga solishi, fibroblastlarning faollashishi va hujayradan tashqari matritsani qayta tuzishni modulyatsiya qilishi hamda ildiz va mezenximal stromal hujayralarning saqlanishi va regenerativ faolligini yaxshilashi mumkin. Ushbu muvofiqlashtirilgan hujayrali ta'sirlar qorinchalarning salbiy qayta tuzilishini kamaytirishga va shikastlangan miokardning strukturaviy va funksional tiklanishini yaxshilashga yordam berishi mumkin.

Kalit so'zlar: Miokard infarkti, biomateriallar, yallig'lanishli yurak-qon tomir kasalliklari, gialuron kislotasi.

Аннотация: Данный обзор посвящен оценке имеющихся данных о влиянии биоматериалов на различные типы клеток, участвующих в восстановлении и регенерации сердечной ткани после инфаркта миокарда. Особое внимание уделяется взаимодействию биоматериалов с кардиомиоцитами, фибробластами, эндотелиальными клетками, макрофагами, клетками-предшественниками сердца и мезенхимальными стромальными клетками, а также их влиянию на воспаление, ангиогенез, фиброз, ремоделирование внеклеточного матрикса и выживаемость клеток. Был проведен структурированный обзор литературы для выявления и оценки исследований, изучающих влияние биоматериалов на клеточные процессы, участвующие в восстановлении и регенерации сердечной ткани после инфаркта миокарда. Обзор был разработан в соответствии с принципами руководства PRISMA 2020 (Preferred Reporting Items for Systematic Reviews and Meta-Analyses). Представленная литература демонстрирует существенное расширение применения биоматериальных подходов к восстановлению миокарда после инфаркта миокарда (ИМ). Было установлено, что биологические эффекты биоматериалов в значительной степени зависят от их состава, механических свойств, характеристик деградации, архитектуры, химии поверхности и наличия включенных биологических сигналов. Биоматериалы могут поддерживать выживаемость кардиомиоцитов, способствовать ангиогенезу, опосредованному эндотелиальными клетками, регулировать воспаление, вызванное макрофагами, модулировать активацию фибробластов и ремоделирование внеклеточного матрикса, а также улучшать сохранение и регенеративную активность стволовых и мезенхимальных стромальных клеток. Эти скоординированные клеточные эффекты могут способствовать снижению неблагоприятного ремоделирования желудочка и улучшению структурного и функционального восстановления поврежденного миокарда.

Ключевые слова: Инфаркт миокарда, биоматериалы, воспалительные заболевания сердечно-сосудистой системы, гиалуроновая кислота.

INTRODUCTION

Long-term CVD frequently results in myocardial infarction (MI), which is caused by blockage of one or more coronary arteries, depriving the myocardium of oxygen and nutrients. The ischemic event induces a switch from aerobic to anaerobic respiration, resulting in acidosis due to lactic acid accumulation, the formation of reactive oxygen species (ROS), and a considerable loss of cardiac myocytes. The ischemic event triggers a switch from aerobic to anaerobic respiration, leading to acidosis due to lactic acid accumulation, the generation of reactive oxygen species (ROS), and a significant loss of cardiac myocytes. In the inflammatory phase, apoptotic and necrotic cardiomyocytes release cytokines that signal the recruitment of neutrophils and monocytes. After the removal of cellular debris, macrophages release growth factors and cytokines, leading to the maturation of granulation tissue through angiogenesis and the promotion of fibroblast proliferation, marking the beginning of the transition from the inflammatory phase to the proliferation phase. Biomaterials can take many forms, from injectable hydrogels to solid patches, and can serve a variety of purposes. A left ventricular restraint is a device that is surgically implanted on the outer wall of the heart in order to provide increased mechanical support to the weakened ventricle of the infarcted heart [1]. Cardiac patches, which can be created in the laboratory and sutured or applied to the surface of the tissue at the site of myocardial injury, have also been employed. These integrate with the host tissue and can be used to deliver therapeutics such as drugs, growth factors, or small molecules [2]. Recently, there has been an increasing amount of research into injectable hydrogels that can be delivered directly into the cardiac tissue. Although much research has been performed on the development of biomaterials to enhance cell transplantation therapies, it is increasingly evident that biomaterials can also be effective on their own without cells, which is the focus of this review.

Due to the high metabolic requirements of cardiomyocytes, oxygen is one of the most important factors in heart function. During infarction, the oxygen supply is blocked or reduced to a point where the oxygen demand exceeds the supply, causing excessive cardiomyocyte death. This cell death leads to a thinning of the ventricular wall in the affected area, making it susceptible to rupture. In order to prevent ventricular rupture, the dead muscle is replaced with a rigid fibrous scar that does little more than provide mechanical support. Several

studies have investigated hydrogels for the release of growth factors that can improve cardiomyocyte survival. For example, an injectable poly(lactide-co-glycolide) (PLGA) nanoparticle was used to bind and deliver insulin-like growth factor-1 (IGF-1), a growth factor that can promote cardiomyocyte proliferation and survival [3]. IGF-1-loaded PLGA nanoparticles were injected immediately post-MI, resulting in increased phosphorylation of the Akt (protein kinase B) and a reduction in the number of TUNEL+ apoptotic cardiomyocytes at 24 h post-injection, compared with injection of IGF-1 alone. After 3 weeks, hearts that received injections of IGF-1 nanoparticles had reduced scar size and superior LVEF compared with controls. It appears that the biomaterial treatment in conjunction with sustained IGF-1 release provided early cardioprotective effects, leading to reduced remodeling and enhanced function of the infarcted heart. Another study utilized a biomaterial derived from porcine ECM in combination with an engineered hepatocyte growth factor (HGF) fragment. Hyaluronic acid (HA)-based biomaterials have also shown promise in preserving ventricular morphology and cardiac function post-MI. For example, MI pig hearts treated with a degradable HA hydrogel (delivered 30 min after induction of MI) had decreased LV end-systolic and end-diastolic volumes at 1 week post-treatment compared with saline controls, indicating initial preservation of function [4].

Furthermore, there was a trend toward increased function (LVEF) in HA-treated animals at all time points, which reached significance at 8 weeks post-MI. This increased function may be due, in part, to the preservation of ventricular thickness and decreased LV volumes conferred by the biomaterial at 4, 8, and 12 weeks post-MI compared with saline. The same group has also reported on the use of a HEMA-HA hydrogel containing PLGA microspheres as a bulking agent for treating the MI heart. It was found that injection of a fast-degrading HEMA-HA hydrogel containing microspheres led to increased thickness of the apical infarct area compared with HEMA-HA alone and infarct control [5]. This thickening was accompanied by greater cell infiltration surrounding the PLGA microspheres and an increase in blood vessel density.

LITERATURE REVIEW

Myocardial infarction (MI) remains one of the major causes of cardiovascular morbidity and mortality worldwide. It is primarily caused by acute obstruction of coronary blood flow, resulting in an imbalance between myocardial oxygen demand and oxygen supply. Ischemia rapidly disrupts cardiomyocyte metabolism, leading to a shift from aerobic oxidative phosphorylation toward anaerobic glycolysis, accumulation of lactate, intracellular acidosis, depletion of ATP, ionic imbalance, mitochondrial dysfunction, and excessive production of reactive oxygen species (ROS). These pathological changes contribute to cardiomyocyte apoptosis and necrosis and initiate a complex process of myocardial remodeling [10–13]. The response to myocardial injury is characterized by several overlapping phases, including inflammation, proliferation, and scar maturation. During the inflammatory phase, necrotic and apoptotic cardiomyocytes release damage-associated molecular patterns and inflammatory mediators that promote the recruitment of neutrophils and monocytes to the infarcted region. Subsequently, macrophages participate in the removal of cellular debris and regulate the transition from inflammation to tissue repair. Activated macrophages release cytokines and growth factors that stimulate angiogenesis, fibroblast proliferation, and extracellular matrix (ECM) remodeling [10,12]. Conventional treatment strategies for MI, including reperfusion therapy, antiplatelet treatment, lipid-lowering therapy, and neurohormonal blockade, primarily aim to restore coronary perfusion, prevent recurrent ischemic events, and limit adverse ventricular remodeling. However, these approaches cannot directly replace lost cardiomyocytes or reconstruct the damaged myocardial extracellular matrix. Consequently, tissue engineering and regenerative medicine have attracted increasing attention as potential strategies for myocardial repair [13–15].

Biomaterials can be used in several forms, including injectable hydrogels, cardiac patches, nanoparticles, scaffolds, and extracellular-matrix-derived matrices. Depending on their composition and design, these materials may provide mechanical support, create a favorable regenerative microenvironment, modulate inflammation and fibrosis, promote angiogenesis, and facilitate the localized and controlled delivery of therapeutic agents [13–16]. Natural materials such as collagen, fibrin, gelatin, alginate, hyaluronic acid (HA), and decellularized extracellular matrix have been extensively investigated for cardiac patch fabrication. Synthetic polymers, including poly(lactic-co-glycolic acid) (PLGA) and polyethylene glycol (PEG)-based materials, provide greater control over mechanical properties, degradation kinetics, and drug-release characteristics [14–16]. Experimental studies have demonstrated that degradable HA-based hydrogels can preserve ventricular morphology following MI. Treatment with HA hydrogels has been associated with reduced left ventricular end-systolic and end-diastolic volumes and improved ventricular function compared with control treatment [15,16]. The beneficial effects of HA-based materials may extend beyond mechanical support. HA can influence cell migration, inflammatory responses, extracellular matrix remodeling, and tissue organization. Consequently, HA-based hydrogels may provide both structural and biological support during post-infarction repair [15,16].



RESEARCH METHODOLOGY

The review also aims to identify the principal mechanisms underlying biomaterial-mediated cardiac repair, compare the regenerative potential of different biomaterial platforms, and highlight current limitations and future directions for their translation into clinical practice. A comprehensive literature search was performed in the PubMed/MEDLINE, Scopus, Web of Science, and Google Scholar databases. The search covered publications from January 2015 to September 2026, with earlier studies included when they represented landmark research directly relevant to the development of biomaterial-based cardiac regeneration. The search strategy combined terms related to myocardial infarction, biomaterials, cardiac regeneration, and cellular responses. The main search terms included: “myocardial infarction,” “cardiac regeneration,” “cardiac repair,” “biomaterials,” “biomaterial scaffolds,” “hydrogels.”

ANALYSIS AND RESULTS

The reviewed literature demonstrates a substantial expansion of biomaterial-based approaches for myocardial repair following myocardial infarction (MI). During the past decade, biomaterials have evolved from relatively passive structural scaffolds to multifunctional platforms capable of modulating the post-infarction microenvironment and interacting with multiple cardiac and immune cell populations. Cardiomyocyte loss represents one of the central pathological consequences of MI. Because adult cardiomyocytes have limited proliferative capacity, extensive myocardial injury is generally followed by scar formation rather than restoration of functional myocardium. The reviewed evidence indicates that biomaterials can improve the post-infarction environment and support the survival and function of remaining cardiomyocytes. Injectable hydrogels and cardiac patches may provide mechanical reinforcement to the ventricular wall, reduce local mechanical stress, and create a three-dimensional environment that supports cellular survival. Biomaterials can also be used to deliver growth factors, nucleic acids, extracellular vesicles, and other cardioprotective signals directly to the injured myocardium. Importantly, current strategies increasingly attempt to reproduce structural and functional characteristics of native myocardium. Conductive and anisotropic biomaterials have been investigated to improve electrical integration and organization of cardiomyocytes. Experimental studies suggest that biomaterials with appropriate mechanical and electrical properties may facilitate more physiological cellular organization and contribute to improved cardiac function.

Several studies have investigated hydrogels for the release of growth factors that can improve cardiomyocyte survival. For example, an injectable poly(lactide-co-glycolide) (PLGA) nanoparticle was used to bind and deliver insulin-like growth factor-1 (IGF-1), a growth factor that can promote cardiomyocyte proliferation and survival [6]. IGF-1-loaded PLGA nanoparticles were injected immediately post-MI, resulting in increased phosphorylation of the Akt (protein kinase B) and a reduction in the number of TUNEL+ apoptotic cardiomyocytes at 24 h post-injection, compared with injection of IGF-1 alone. After 3 weeks, hearts that received injections of IGF-1 nanoparticles had reduced scar size and superior LVEF compared with controls. It appears that the biomaterial treatment in conjunction with sustained IGF-1 release provided early cardioprotective effects, leading to reduced remodeling and enhanced function of the infarcted heart.

Another study utilized a biomaterial derived from porcine ECM in combination with an engineered hepatocyte growth factor (HGF) fragment [7]. Biomaterials for treating MI have also been developed from a variety of synthetic polymers. For example, poly(ϵ -caprolactone)-2-HEMA and poly(*N*-isopropylacrylamide) (PNIPAAm) were added to a biodegradable dextran chain to yield a Dex-PCL-HEMA/PNIPAAm hydrogel [8]. Upon injection into the MI heart, this material formed a gel rapidly and reduced scar size while increasing ventricular wall thickness and preventing LV dilation compared with PBS-injected controls. Hydrogels based on PEG, a commonly used synthetic polymer, have also been shown to have therapeutic potential for the treatment of MI. One study evaluated the effect of timing of delivery on its therapeutic efficacy [9]. It was shown that injection of the hydrogel at 1 week post-MI led to greater ventricular wall thickness compared with animals that were treated with the PEG hydrogel immediately after induction of MI (day 0). This observation was attributed to more rapid degradation of the material in the acute inflammatory environment when injected at the earlier time point, thus reducing its effectiveness as a bulking agent.

CONCLUSION

A biomaterial can affect cells directly or indirectly via downstream signaling, and understanding these signaling processes will enable enhanced regulation of the healing process. It may also be that multiple biomaterial strategies will need to be applied in combination to promote appropriate repair responses from the different cell types. Overall, the available evidence supports the concept that biomaterials can influence myocardial repair through coordinated interactions with multiple cellular populations. The therapeutic effect should not be viewed as a direct replacement of damaged myocardium by an artificial scaffold.

Rather, biomaterials can act as regulators of the post-infarction microenvironment, providing structural support while modifying cellular communication, inflammation, angiogenesis, fibrosis, and tissue remodeling. The most promising strategies appear to be those that combine several biological functions within a single platform. For example, a biomaterial may simultaneously provide mechanical support, promote vascularization, modulate macrophage activity, reduce fibrosis, and deliver regenerative signals to surviving cardiomyocytes or transplanted cells.

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Jurnalga istalgan payt quyidagi rekvizitlar orqali obuna bo'lishingiz mumkin. Obuna bo'lgach, @iqtisodiyot_77 telegram sahifamizga to'lov haqidagi ma'lumotni skrinshot yoki foto shaklida jo'natishingizni so'raymiz. Shu asosda har oygi jurnal yangi sonini manzilingizga jo'natamiz.

Mazkur jurnalda maqolalar chop etish uchun quyidagi havolalarga maqola, reklama, hikoya va boshqa ijodiy materiallar yuborishingiz mumkin.

Materiallar va reklamalar pullik asosda chop etiladi.

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