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CLINICAL AND PATHOGENETIC ASPECTS OF HEART DAMAGE IN SYSTEMIC CONNECTIVE TISSUE DISEASES

Sadikova Raykhon Rakhimbekovna,
Assistant Professor, Department of Propaedeutics of Childhood Diseases No. 2, TSMU, Tashkent State Medical University

Abstract

Systemic connective tissue diseases (SCTDs) represent a heterogeneous group of chronic, often progressive, autoimmune disorders characterized by the involvement of collagen and elastin-containing tissues. Among the most common SCTDs are systemic lupus erythematosus, systemic sclerosis, polymyositis, dermatomyositis, Sjögren's syndrome, and rheumatoid arthritis. These conditions can affect almost any organ system, but one of their most significant and life-threatening complications is cardiac involvement. Understanding the clinical and pathogenetic aspects of heart damage in the context of systemic connective tissue diseases is essential for improving prognosis, management strategies, and patient survival.

Keywords: Systemic connective tissue diseases, cardiac involvement, autoimmune inflammation, myocarditis, pericarditis, accelerated atherosclerosis, immunosuppressive therapy.

Introduction

Аннотация:

Системные заболевания соединительной ткани (СЗСТ) представляют собой гетерогенную группу хронических, зачастую прогрессирующих, аутоиммунных расстройств, характеризующихся поражением тканей, содержащих коллаген и эластин. К наиболее распространённым СЗСТ относятся системная красная волчанка, системная склеродермия, полимиозит, дерматомиозит, синдром Шегрена и ревматоидный артрит. Эти заболевания могут поражать практически любую органную систему, однако одним из самых значимых и угрожающих жизни осложнений является поражение сердца. Понимание клинических и патогенетических аспектов

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сердечного поражения при системных заболеваниях соединительной ткани крайне важно для улучшения прогноза, тактики лечения и выживаемости пациентов.

Ключевые слова: Системные заболевания соединительной ткани, поражение сердца, аутоиммунное воспаление, миокардит, перикардит, ускоренный атеросклероз, иммуносупрессивная терапия.

Annotatsiya:
Sistemali biriktiruvchi to‘qima kasalliklari (SBTK) – kollagen va elastin tutgan to‘qimalarni zararlovchi, surunkali va ko‘pincha progressiv kechuvchi avtoimmun xastaliklar guruhini tashkil etadi. SBTKlar orasida eng ko‘p uchraydiganlari – sistemali qizil volchanka, sistemali skleroz, polimiozit, dermatomiozit, Sjögren sindromi va revmatoid artritdir. Bu kasalliklarning deyarli barcha a’zolarga zarari mumkin, ammo eng xavfli va hayot uchun tahdidli asoratlardan biri yurak zararlanishi hisoblanadi. Sistemali biriktiruvchi to‘qima kasalliklari kontekstida yurak zararlanishining klinik va patogenetik jihatlarini o‘rganish prognozni yaxshilash hamda davolash strategiyalarini tanlash va bemor omonligini oshirish uchun muhim ahamiyatga ega.

Kalit so‘zlar: Sistemali biriktiruvchi to‘qima kasalliklari, yurak zararlanishi, avtoimmun yallig‘lanish, miokardit, perikardit, tezlashgan ateroskleroz, immunosupressiv terapiya.

INTRODUCTION

Cardiac manifestations in SCTDs are often multifaceted and may range from silent, subclinical changes detected only by advanced imaging modalities to overt, life-threatening conditions such as heart failure, arrhythmia, and sudden cardiac death. These manifestations may involve any part of the heart, including the myocardium, pericardium, endocardium, coronary arteries, heart valves, and conduction system. The frequency, type, and severity of cardiac involvement largely depend on the underlying disease, its duration, activity, and the presence of other comorbidities. Pathogenesis of cardiac involvement in SCTDs is complex and multifactorial. Autoimmune inflammation plays a pivotal role, promoting cellular and humoral immune responses with the production of

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autoantibodies, activation of T lymphocytes, and the release of various cytokines and chemokines that ultimately affect cardiac tissues. Additionally, immune complexes deposit into cardiac structures, leading to complement activation and subsequent inflammation and tissue injury. Vascular endothelial injury, microvascular dysfunction, and chronic inflammation drive accelerated atherosclerosis, which increases the risk of ischemic heart disease in these patients even in the absence of traditional cardiovascular risk factors.

MATERIALS AND METHODS

In systemic lupus erythematosus (SLE), cardiac involvement is reported in a substantial proportion of patients and may include pericarditis, myocarditis, endocarditis (notably Libman-Sacks endocarditis), and increased propensity for accelerated coronary artery disease. Pericarditis is the most common cardiac complication of SLE and frequently manifests with chest pain and characteristic electrocardiographic changes. Immune complex-mediated inflammation of the pericardium results in exudative effusions and can sometimes progress to constrictive pericarditis if not appropriately managed. Myocarditis, although relatively rare, can manifest as unexplained heart failure, arrhythmias, or conduction abnormalities. Libman-Sacks endocarditis is characterized by the formation of sterile vegetations, most commonly on the mitral and aortic valves, which may be complicated by embolic phenomena [1].

Systemic sclerosis (SSc) is another SCTD frequently associated with cardiac involvement, which includes myocardial fibrosis, pericardial disease, arrhythmias, conduction disturbances, and pulmonary arterial hypertension with resultant right heart dysfunction. Myocardial fibrosis is especially characteristic of SSc and results from chronic microvascular ischemia, reperfusion injury, and activation of fibroblasts which synthesize excess extracellular matrix proteins. Advanced imaging modalities such as cardiac MRI and echocardiography reveal patchy myocardial involvement that often precedes clinical symptoms. Cardiac involvement may progress insidiously, leading to diastolic and systolic dysfunction, restrictive cardiomyopathy, and heart failure.Patients with polymyositis and dermatomyositis also demonstrate a spectrum of cardiac abnormalities including myocarditis, conduction defects, arrhythmias, and, less commonly, pericarditis. Inflammatory infiltration of the myocardium by lymphocytes, degeneration of myocardial fibers, and small vessel vasculitis

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contribute to pathogenesis. This group is at particular risk for sudden cardiac death due to conduction system defects and ventricular arrhythmias. Electrophysiological studies and imaging help identify patients at risk. Myocarditis may be asymptomatic or present with overt heart failure [2].

RESULTS AND DISCUSSIONS

Rheumatoid arthritis (RA) is associated with increased cardiovascular morbidity and mortality, a risk not explained solely by traditional risk factors. Chronic systemic inflammation is thought to be the key driver, with increased atherogenesis, endothelial dysfunction, and upregulation of pro-inflammatory cytokines. RA-associated pericarditis is common but is often clinically silent. Long-term inflammation may lead to fibrous thickening of the pericardium and rarely, constrictive pericarditis. Myocardial involvement is less common but can manifest as granulomatous or nonspecific myocarditis. Valvular involvement, especially thickening and regurgitation of the aortic and mitral valves, can also occur. Cardiac involvement in Sjögren's syndrome remains less common and less well described, but increased risk of arrhythmia and conduction disturbances, as well as possible myocarditis and pericardial effusion, have been documented. The pathogenesis of cardiac damage in SCTDs involves both direct immune-mediated injury and indirect mechanisms. Autoimmune reactions directly injure cardiac myocytes, the conduction system, and vascular endothelium. Chronic inflammation, oxidative stress, and immune complex deposition may cause tissue remodeling, fibrosis, and atherosclerotic plaque formation. Moreover, drug therapies commonly utilized in SCTDs, such as corticosteroids and certain immunosuppressive agents, can also contribute to cardiovascular morbidity through their effects on blood pressure, lipid profiles, insulin sensitivity, and vascular health [3].

Clinically, cardiac manifestations in SCTDs are often asymptomatic in the early stages. Subtle changes may be detectable only by advanced imaging techniques or biomarkers prior to the development of symptoms. As the cardiac involvement progresses, patients may develop exertional dyspnea, chest pain, palpitations, presyncope or syncope, peripheral edema, and even sudden death. Physical examination findings are often nonspecific, highlighting the need for a high index of suspicion in patients with known SCTDs. Diagnostic approaches to heart involvement in SCTDs have evolved considerably. Echocardiography remains a

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first-line tool for the detection of pericardial effusions, chamber size and function, valve abnormalities, and pulmonary pressures. Cardiac MRI excels at identifying myocardial inflammation, fibrosis, and perfusion abnormalities. Laboratory studies, including cardiac biomarkers such as troponin and NT-proBNP, may assist in the detection of myocardial damage. Electrocardiography and ambulatory Holter monitoring are valuable for detecting conduction disturbances and arrhythmias. In selected cases, coronary artery imaging or invasive hemodynamic monitoring may be indicated to clarify the extent of disease.Management of cardiac involvement in SCTDs requires a multidisciplinary approach. Therapy must be individualized based on the underlying disease, the type and severity of cardiac injury, and the overall health status of the patient. Immunosuppressive therapies such as corticosteroids, cytotoxic agents, and biologics are commonly used to control the underlying autoimmune process and mitigate further cardiac damage. For pericarditis, NSAIDs and corticosteroids form the mainstay of treatment, although colchicine and immunosuppressive agents may be needed for recurrent or refractory cases. In cases of myocarditis, aggressive immunosuppression may be required alongside symptomatic management of heart failure [4].

Management of heart failure follows standard cardiology guidelines with particular attention to the underlying autoimmune process. Patients with significant arrhythmias or conduction disturbances may require device implantation such as a pacemaker or implantable cardioverter-defibrillator (ICD). Close monitoring for adverse drug effects and potential drug interactions is crucial, as many SCTD patients are receiving multiple medications. Cardiovascular risk reduction by controlling traditional factors like hypertension, diabetes, obesity, hyperlipidemia, and smoking cessation is essential since these factors can compound the risk created by chronic inflammation. Prevention and early detection of cardiac involvement are also important. Regular cardiac assessment, including imaging and biomarkers, should be an integral part of disease monitoring in patients with systemic connective tissue diseases, even in the absence of symptoms. Screening for subclinical cardiac involvement can help initiate timely treatment and potentially improve long-term outcomes. Genetic, environmental, and hormonal influences further complicate the pathogenesis of heart damage in SCTDs. Recent discoveries highlight the importance of genetic predisposition to both autoimmunity and accelerated atherosclerosis, epigenetic

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factors, and the complex interplay of sex hormones — particularly in diseases with a pronounced female predilection such as SLE and SSc.

Promising research is ongoing to identify novel biomarkers for early detection, to clarify the molecular pathways driving cardiac involvement, and to develop targeted therapies that can modulate inappropriate immune activation without causing undue immunosuppression. Biologic agents targeting specific cytokines, immune checkpoints, or cell types are being evaluated in clinical trials, with hopes of offering more effective and less toxic alternatives for patients at risk of severe cardiac complications. The prognosis of patients with SCTDs and cardiac involvement is closely linked to the underlying disease severity, the degree of cardiac dysfunction, and the responsiveness to intervention. Early detection, judicious use of immunosuppressive therapies, and aggressive management of both traditional and disease-specific cardiovascular risks offer the best prospects for improving survival and quality of life [5].

In summary, heart damage in systemic connective tissue diseases is the result of multifactorial pathogenetic processes, including autoimmune inflammation, vascular dysfunction, and accelerated atherogenesis. Cardiac involvement may involve any cardiac structure, presenting with a wide variety of clinical manifestations ranging from subclinical to life-threatening. Because cardiac damage often progresses insidiously, early recognition, thorough assessment, and interdisciplinary collaboration are pivotal to optimize patient outcomes. Ongoing research will hopefully provide further insights into the molecular and cellular mechanisms at play and inform the development of more precise therapies to mitigate the burden of these devastating diseases.

Conclusion

Cardiac involvement is a major determinant of morbidity and mortality in patients with systemic connective tissue diseases. Its pathogenesis is complex and is driven by autoimmune processes, inflammation, microvascular injury, and subsequent tissue remodeling, which together contribute to a broad spectrum of clinical presentations from silent disease to overt cardiac dysfunction and sudden death. Improved understanding of these pathogenetic mechanisms has led to better diagnostic strategies and evolving approaches to therapy that integrate immunosuppression with optimal cardiovascular care. Early detection, regular monitoring, and tailored management are critical in preventing advanced cardiac

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damage and improving outcomes for patients with SCTDs. Future research focused on biomarker discovery and targeted therapies offers hope for even greater improvements in the care of these complex patients.

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