THE USA JOURNALS
THE AMERICAN JOURNAL OF MEDICAL SCIENCES AND PHARMACEUTICAL RESEARCH
(ISSN – 2689-1026)
VOLUME 06 ISSUE09

PUBLISHED DATE: - 30-09-2024
DOI: - https://doi.org/10.37547/TAJMSPR/Volume06Issue09-07 **PAGE NO.: - 41-44**

RESEARCH ARTICLE **Open Access**

FEATURES OF CENTRAL HEMODYNAMICS IN PATIENTS WITH BRONCHIAL ASTHMA

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Abstract
Bronchial asthma (BA) is a serious medical problem. The prevalence of asthma is increasing in most countries. Deterioration of pulmonary function is as strong a predictor of cardiovascular mortality as major cardiovascular risk factors. In this review, we tried to reflect the current current understanding of the adverse effects of asthma on cardiovascular remodeling.

Keywords Bronchial asthma, pulmonary hypertension, cor pulmonale.

INTRODUCTION

In the last decade, chronic nonspecific lung diseases have taken third place in terms of prevalence, morbidity and mortality among other types of pathology. According to 2020–2023 World Health Organization (WHO) estimates, nearly 262 million people worldwide suffered from bronchial asthma, resulting in 455,000 deaths [1].

Bronchial asthma (BA) is a heterogeneous disease characterized by chronic inflammation of the airways, the presence of respiratory symptoms such as wheezing, shortness of breath, chest congestion and cough, which vary in time and intensity, and occur together with variable airway obstruction [1,2]. Based on the pathogenesis of asthma, a number of cytokines and growth factors relevant to the chronicity of airway inflammation are produced by normal resident cells of the bronchial tree (fibroblasts, myofibroblasts, epithelial cells and smooth muscle cells). Fibroblasts play a key role in airway remodeling and inflammation. They produce collagen, reticular and elastic fibers, proteoglycans and glycoproteins. Myofibroblasts promote tissue remodeling by releasing interstitial collagen, fibronectin and laminin, and producing growth factors for blood vessels, nerves and smooth muscle [3]. In bronchial asthma, the number of myofibroblasts increases and their number correlates with the thickness of the reticular basement membrane. Thus, as a result of the pathological process in bronchial asthma, the reversible component of bronchial obstruction predominates, which consists of contraction of smooth muscles, edema of the mucous membrane, and obstruction of the bronchial lumen by mucus. However, with long-term inflammation in the bronchi, wall remodeling and proliferation of connective tissue (fibrosis) occurs, which is an irreversible part of bronchial obstruction. This in

turn will lead to multiple complications from the cardiovascular system [3,4].

Frequent exacerbations of attacks in asthma are most clearly demonstrated by the close anatomical and functional connection between the heart and lungs. As a result, the problem goes from pulmonary to cardiopulmonary. In patients with asthma, changes in the cardiovascular system are often recorded with the development of pulmonary hypertension, damage predominantly to the right side with the formation of chronic pulmonary heart disease, the development of myocardial ischemia and heart rhythm disturbances. The issues of the combined course of cardiovascular diseases (CVD) and asthma are actively discussed in the literature. [5]. Cardiac remodeling, which occurs in response to damage, leading to a change in its geometry and impaired contractility, ultimately determines the prognosis of life for patients with chronic obstructive pulmonary pathology. Despite the fact that the main function of the circulatory system is transport, its participation in physiological and pathological processes in the body is very diverse. It should be noted that the clinical, functional and morphological changes that occur in the cardiovascular system during inflammation, as a rule, represent a complex chain of cause-and-effect and closely related manifestations of pathology [6,7]. Therefore, pathological changes that can be objectively assessed are usually the result of the simultaneous influence of multiple factors such as hypoxemia, hypercapnia, bronchial obstruction and associated ventilation disorders, intoxication with products of altered tissue metabolism, pathological effects of biologically active substances, and disturbances in the rheological properties of blood [8]. The severity of changes in the cardiovascular system and, accordingly, its clinical manifestations depend on the prevalence of bronchial lesions, as well as the phase of the process. It is known that changes in the pulmonary circulation in patients with bronchial asthma can be associated with both disturbances of central hemodynamics and disorders of pulmonary microcirculation.

According to the observations of many authors, in patients with mild impairments in the function of external respiration, normal levels of oxygen tension in the arterial blood, pressure in the pulmonary artery and minute blood volume do not exceed normal figures [3,9]. Against the background of repeated exacerbations, with the progression of respiratory dysfunction, changes in hemodynamics also become more pronounced. Increasing disturbances in bronchial obstruction, diffusion capacity and lung volume parameters, leading to chronic hypoxemia and hypercapnia, can cause the development of moderate pulmonary hypertension at rest and an increase in right ventricular filling pressure [10]. Systolic and diastolic function of the right ventricle in patients with asthma. Previous studies have revealed that the state of diastolic function of the right ventricle in asthma depends on the severity of the underlying disease, the formation of the level of afterload, the values of pulmonary hypertension and the severity of enlargement and hypertrophy of the right ventricle. In various groups of patients with asthma, pathological load against the background of the lack of pharmacotherapeutic control of asthma (increased resistance in the pulmonary circulation, myocardial hypoxia) leads to structural and functional changes in the pancreas [11]. An increase in the severity of bronchial asthma naturally leads to even more pronounced remodeling of the right ventricle and changes in regional contraction indices at the inflow level. The dependence of progressive changes in the diastolic function of the right

ventricle on the severity of asthma has been established. Pulmonary hypertension in patients with moderate and severe asthma is associated with the development of diastolic dysfunction in the right ventricle and changes in the morphometric parameters of the right ventricle [4,12].

It should be noted that the appearance of hypertrophy, dilatation and subsequent failure of the right ventricle can be recorded at relatively low pressure levels in the pulmonary artery, not exceeding 35 mmHg. This may indicate that pulmonary hypertension is not the only cause of the formation of chronic cor pulmonale [13]. In the diagnosis of asthma, correlation analysis data in the group of asthmatics with moderate disease showed an inverse relationship between the degree of right ventricular hypertrophy (RVH) and the FEV1/FVC value. Outside of exacerbation of the disease, a negative relationship was revealed between RVH and FEV1. In a cohort of patients with severe asthma during an exacerbation, a negative relationship between FEV1/FVC and the diameter of the pulmonary trunk was also recorded [7]. Correlation analysis of echocardiographic data with body plethysmography data in patients with moderate asthma during exacerbations established a negative relationship between residual lung volume and maximum late filling rate. In patients with severe asthma, a significant positive relationship was found between the thickness of the anterior wall of the right ventricle and the residual lung volume (RLV), the RLV/RLV ratio, as well as between the RLV and end-diastolic pressure in the pulmonary artery. During the period of remission of the disease in the group of patients with moderate asthma, a positive relationship was established between expiratory air resistance and the value of MPAP. A combination of changes in the shape of the right ventricular cavity (an increase in the ratio of transverse to longitudinal dimensions) with the development of diastolic dysfunction of the right ventricle was also identified. The identified disorders are a reflection of the general process associated with the structural and functional remodeling of the right heart in patients with bronchial asthma. Changes in the right ventricular myocardium are closely associated with persistent bronchial obstruction, impaired lung volumes, and chronic inflammation present in the airways. It has been established that disturbances in the diastolic function of the right ventricle progress in parallel with an increase in the severity of bronchial asthma [13,14].

Thus, the risk of cardiovascular diseases is closely related to asthma; disturbances in intracardiac blood flow in patients with asthma are associated with the appearance of an obstructive syndrome, which periodically resolves in the form of changes in the blood flow of the lungs and pulmonary circulation. The study of circulatory disorders in asthma can play an important role in their treatment.

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