The Inextricable Silent Connection: Unraveling the Link Between Atherosclerosis and Asthma

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Abstract

Despite previous studies suggesting a possible association between atherosclerosis and asthma, the available data remains confined and isolated. This article delves into the correlation between these two diseases. By scrutinizing the existing literature highlighting shared risk factors and biomarkers between atherosclerosis and asthma, it becomes evident that the inflammatory cells common to both ailments may play a role in the pathogenesis of both disease processes. Some research sheds light on the protective effect of some asthma medications and the harmful aspect of others. An increasing body of evidence looks at the age asthma onset and its association atherosclerosis. Future research should focus on the early detection of atherosclerosis and addressing the tacit connection between atherosclerosis and asthma to improve the morbidity and mortality of those with asthma.

Keywords

Asthma, atherosclerosis, arterial plaque, cardiovascular disease, inflammation, inflammatory cells.

Introduction

Inflammation plays a significant role in the pathogenesis of both asthma and atherosclerosis. In asthma, chronic airway inflammation leads to reversible airway hyper-responsiveness and remodeling. The contribution of cytokines released from mast cells, lymphocytes, eosinophils, macrophages, endothelial dysfunction evidenced by endothelial micro-articles (EMPs), and other inflammatory mediators to arterial atherosclerotic plaque formation in asthmatics has been established. This article examines the link between asthma and atherosclerosis while controlling for other -

of cardiovascular disease in patients using asthma medications, and the effect was more pronounced in women [13].

Understanding how the inflammatory cells can be targeted to control the disease progression in asthma and atherosclerosis is needed to improve the morbidity and mortality related to cardiovascular events in asthmatics.

Limitations

This article's findings are limited by the need for studies analyzed, highlighting the need for additional reports to be included. Furthermore, more randomized-controlled trials are needed to validate the conclusions drawn from the present study.

Conclusion

Inflammation is a shared antecedent between asthma and atherosclerosis, with asthmatics exhibiting more inflammatory cells and an increased risk of cardiovascular disease due to atherosclerosis. There is much room for research in this area that delves into establishing a link between the pathological and pharmacological aspects of asthma and the possible association with cardiovascular and cerebrovascular events while controlling for other variables and excluding the potential confounders. To improve survival outcomes for individuals with asthma, it is essential to explore targeted therapeutic approaches to reduce the inflammatory cell burden, which forms the underlying basis of both conditions.

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Discussion

Atherosclerosis, a scourge upon the vasculature, is a malady characterized by the formation of focal lesions in the innermost wall of blood vessels. These lesions, comprising a tender nucleus rich in cholesterol and an outer sheath of fibrin, appear as early as childhood in the form of fatty streaks. But in adulthood, they progress to calcified plagues that may obstruct most vessel lumen. Inflammation is inextricably intertwined with the pathogenesis of atherosclerosis, and its hallmarks include the infiltration of inflammatory cells throughout the disease's various stages. Notably, atheromatous plaque cells secrete C-reactive protein (CRP), a biomarker of atherosclerosis. These lesions are intimately associated with macrophages, lymphocytes, and mast cells [1].

Asthma, an enduring ailment of the airways, is defined by recurring reversible episodes of wheezing, breathlessness, cough, and chest constriction. It is marked by the narrowing of the bronchioles that stems from an over-reactivity of the airways to various non-pathogenic stimuli. It is also characterized by persistent inflammation, sustained by a host of immune cells, such as lymphocytes, eosinophils, and mast cells. As the disease progresses, mucus production and the structural transformation of the airways increase. This engenders fibrotic alterations below the mucosal layer, accompanied by heightened glandular dimensions and vascularity, and amplified proliferation of smooth muscle cells, leading to augmented sensitivity to allergens [2].

Asthma as a disease entity has deleterious effects on the blood vessels, but the medications, specifically oral corticosteroids used to treat asthma, also wreak havoc on the vasculature. Increasing data have suggested increased IgE, fibrinogen, lipoprotein A, CRP, and total leukocyte number in asthma. A differential count shows an increasing trend in the number of eosinophils and thrombocytes while a declining trend in the number of lymphocytes. Elevated levels of fibrinogen, CRP, and lipoprotein A have been correlated with an increased risk of developing cardiovascular diseases. It was also observed that the more severe the magnitude of the illness in asthmatics, the more profound the pathological lesions and burden of atherosclerosis [3]. This may manifest as increased intima-media thickness in carotid and femoral vessels in asthmatics compared to the controls [4].

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Invariably, asthma has been linked with more cardiovascular complications, a risk factor for stroke and ischemic heart disease [3]. A study with a mean follow-up time of about 9.1 years showed an association of persistent asthma with heart disease. In addition to increased CRP and fibrinogen, the study also noted that patients with asthma had higher levels of markers of inflammation, such as interleukin six (IL-6) and D-dimer, compared to the controls [5]. On the contrary, a comprehensive study that followed asthmatic patients for about fourteen years concluded that asthma is not linked to an increased risk of heart disease but is positively linked with stroke development [6].

Although asthma is caused by an alteration in the balance between the Th-1 and the Th-2 immunity, while atherosclerosis is linked to Th-1 immunity, these two diseases seem intertwined based on common inflammatory system components, including macrophage and lymphocytes found in the airways and the blood vessel mucosa. Activated mast cells by crosslinking of IgE degranulate to release histamine and cytokines, which mediate an asthma attack, are also found in the atherosclerotic vasculature and triggered by various stimuli; they lead to plaque vulnerability. This might provide the basis for improved lipid profile in the long-term users of mast-cell stabilizers, but this observation needs validation by more research [7]. It has also been researched that inhaled corticosteroids (ICS), in contrast to oral corticosteroids, may be protective against atherosclerosis which was evident in reduced thickness of intima-media of the carotid vessels in the ICS-treated asthmatics compared to the nonasthmatics hence showing the negative correlation [8,9].

Other cell types that form a common link between asthma and atherosclerosis are macrophages, T-lymphocytes, and eosinophils [7]. Evidence also suggests a positive correlation between asthma and peripheral arterial disease, but this association's exact cause remains unclear [10].

Some evidence points to the association between adultonset asthma in women and increased carotid artery atherosclerosis. However, it could be partly attributable to these subjects' significant smoking history [11]. However, the atherosclerotic changes seen in childhood-onset asthma were not significantly different from the non-asthmatic controls, as seen by a casecontrol study [12]. This observation was supported by a prospective study that reported an increased prevalence 195(1), pp. 129–137. doi:10.1016/j.atherosclerosis.2006.09.004.

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