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CASE REPORT

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Eosinophilic myocarditis and coronary arteritis in a fatal case of asthma

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✓ Abstract

Mortality is very unusual in the case of asthma. We recently came across a fatal case of asthma which showed a rare combination of unusual complications like eosinophilic myocarditis, coronary arteritis, biventricular cardiac hypertrophy, eosinophilic pneumonitis and pulmonary hypertension.

Keywords: Asthma, biventricular hypertrophy, coronary arteritis, eosinophilic pneumonitis, eosinophilic myocarditis, pulmonary hypertension

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✓ Introduction

With recent advances, diagnosis of asthma or chronic obstructive pulmonary disorders is never a problem, and with good drugs available now, most of the patients lead a comfortable life. However, complications do come up now and then but are rarely fatal. One such rare complication is eosinophilic myocarditis. ^{[1],[2],[3]} We are hereby presenting a fatal case of asthma which showed unusual complications.

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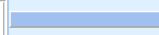
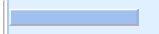
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Case Report

A 30-year-old male, a known asthmatic, was admitted to the casualty in gasping state. The past history revealed that the patient had intermittent attacks of breathlessness along with cough with expectoration. The patient was also a chronic alcoholic and smoker. He expired within half an hour of admission and a complete autopsy was performed.

At autopsy, the heart weighed 700 g with marked bilateral ventricular hypertrophy and congestion of myocardium [Figure 1]. Pericardium and myocardium were unremarkable. Both the lungs showed emphysematous changes, numerous sub-pleural bullae and dilated airways, few plugged with inspissated mucous. Pleural membranes were moderately thickened. Other organs were unremarkable. On microscopy, the heart showed severe myocardial hypertrophy in the numerous sections studied, including the section from the septum. There was mild interstitial fibrosis with moderate inflammatory infiltrate comprising predominantly eosinophils focally destroying the myocytes [Figure 2]. There was no evidence of any parasite. Sections from the left anterior descending and right coronary artery showed marked intimal fibrosis with damage to the internal elastic lamina and the media was infiltrated with mixed inflammation including eosinophils [Figure 3]. Sections from lungs showed thickened pleura and widened alveolar septae with marked congestion and eosinophilic infiltrate. The bronchi showed marked thickening and destruction of wall by a predominant eosinophilic infiltrate and few were plugged with mucous secretions [Figure 4]. Pulmonary arteries were markedly thickened with few showing recanalized thrombi, suggesting coexistent pulmonary hypertension. These changes were attributed to his asthmatic condition and final cause of death was given as eosinophilic myocarditis and pneumonitis leading to cardiorespiratory failure.



Figure 1 : Transverse section of the heart showing marked biventricular hypertrophy and areas of congestion

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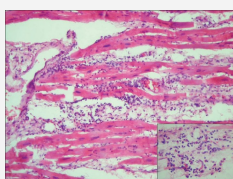


Figure 2 : Photomicrograph of heart showing hypertrophy of myocardial fibers and moderate inflammation in the interstitium, predominantly comprising the eosinophils (inset) and focally destroying the myocytes (H and E, ×100)

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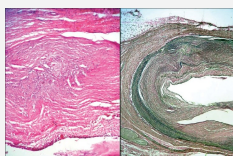


Figure 3 : Left: Section from the right coronary artery showing intimal fibrosis and with underlying media. medial thickening with moderate inflammatory infiltrate (H and E, ×100). Right: Focal destruction of the internal elastic lamina (Von-Geison 's stain, ×100)

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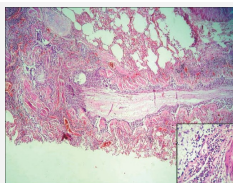


Figure 4 : Photomicrograph of the lungs showing inspissated mucus in the large bronchus with destruction of the wall with dense inflammatory infiltrate comprising predominantly eosinophils (inset) (H and E, ×100)

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Discussion

The overall prevalence of asthma in the general population is 2-3% and up to 5% in the children, and mortality rate due to asthma is 0.1% to 3.3 % in patients with repeated attacks of acute severe asthma. [4] However, the exact incidence of fatal changes in lung and heart is not documented.

Myocarditis is non-ischemic myocardial infarction of known and unknown causes like infectious agents, hypersensitivity or immune related. It is noted in autopsy series in 1-5% of cases. [3] Depending on the nature of the infiltrate, the etiologic agent can be determined. Eosinophilic myocarditis is associated with hypersensitivity, parasitic infections and hypereosinophilic syndromes. Other causes are asthmatic bronchitis, mycoplasma pneumoniae or idiopathic. In our case, asthma was the only known culprit which could have lead to myocarditis along with coronary arteritis which is very unusual and only rarely reported. [1],[2] Hypereosinophilic syndrome can present with perennial rhinitis and asthma, and can rarely be fatal with

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histopathology showing extensive myocarditis and eosinophilic pneumonia as seen in our case.

[5] Eosinophilic myocarditis can be seen even in the absence of pronounced eosinophilia in the peripheral blood. [6] However, absolute eosinophil counts were not available in our case. Right ventricular hypertrophy is another feature commonly noted in asthmatics. [4] However, in our case, there was significant biventricular hypertrophy which has not been described with asthma.

Eosinophilic myocarditis can present as acute myocardial infarction with chest pain, ST elevation on an electrocardiogram and increase in cardiac enzymes, and thus should be considered in the differential diagnosis of myocardial infarction in the appropriate clinical setting. [6] It can also occur in asthmatic patients who have begun treatment with antileukotriene drugs following withdrawal of steroids. [7] However, our patient did not give any history of using any of these drugs. Endomyocardial biopsy is the gold standard for the diagnosis of myocarditis. [3]

Pulmonary changes in asthma are largely overdistended lungs, atelectasis, airways plugged with thick tenacious mucus plugs with microscopy showing severe eosinophilic infiltrate. [4] These changes were seen in our case correlating with the diagnosis of asthma. Pulmonary hypertension is rarely described with asthma and is being attributed to nitric oxide produced in moderate asthmatics. [8]

The other differential diagnosis that we considered was Churg-Struass syndrome (CSS). CSS is a form of systemic vasculitis, and multiple organ involvement in the form of asthma, allergic rhinitis, pulmonary infiltrates, cardiac disease, gastrointestinal tract, renal, skin and musculoskeletal system is reported. [9]

However, in our case, the eosinophilic infiltrate was limited to the lungs and the heart. Vasculitis was noted only in the coronary vessels.

To conclude, this was a case of asthma showing a rare combination of unusual complications like eosinophilic myocarditis, coronary arteritis, biventricular cardiac hypertrophy, eosinophilic pneumonitis and pulmonary hypertension in a single case.

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Figures

[Figure 1], [Figure 2], [Figure 3], [Figure 4]

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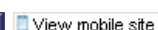
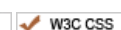
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