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RESEARCH ARTICLE

POLYMYOSITIS COMPLICATED WITH COMPLETE ATRIOVENTRICULAR BLOCK: A CASE REPORT AND LITERATURE REVIEW

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Abstract

Background: Cardiac involvement in polymyositis is often asymptomatic. Electrocardiogram (ECG) remains a cornerstone for the screening of conduction disturbances and other electrocardiographic abnormalities.

Case presentation: We report the case of a 35 year-old-woman with polymyositis complicated with a complete atrioventricular block 10 years after the beginning of her symptoms. She was admitted to the hospital for syncope and benefited from a pacemaker insertion without incident.

Conclusion: Cardiac effects of polymyositis are associated with poor prognosis and might appear in patients in remission of the disease.

Introduction:-

Polymyositis is a chronic inflammatory muscle disease clinically characterized by muscle weakness and fatigue, and histopathologically by inflammatory cell infiltrates in skeletal muscle.

The frequency of heart involvement in patients with myositis varies between 9 and 72% [1, 2]. Progression to complete atrioventricular block might not be clinically relevant, thus the importance of reporting such case, unfortunately diagnosed at the syncope stage, occurring in a patient ten years after the onset of muscle pain and weakness, in order to highlight the value of a systematic electrocardiographic screening for a better management.

Case History:

We report the case of a 35-year-old woman presenting with syncope. From her medical history we learned that she was admitted in 2009 for pain and stiffness in both thighs, difficulties standing erect and rising from the supine position. There was no history of rheumatic fever, systemic disease, exposure to toxic chemicals nor family history of muscular disease.

Physical examination on admission revealed a hemodynamically stable, apyretic patient, with a normal cardiovascular examination. We noticed a pronounced muscle atrophy of both thighs.

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Laboratory investigations at the time of diagnosis yielded the following results: HGB: 11g/dl, MCV: 78 μ^3, WBC: 12000 / mm^3, PLT: 230000 /mm^3, Urea: 0.4 g/l, Creatinine: 11 mg/l, C-Reactive Protein: 20 mg/l. Creatine kinase (CK) levels were elevated (1200 U/L). Muscle biopsy and electroneuromyography showed an aspect suggestive of polymyositis. The patient was treated with corticosteroids with good clinical outcome.

In April 2019, she returned to the emergency department for dizziness along with three episodes of syncope. Clinical examination on admission revealed bradycardia with a heart rate of 30 per minute, no audible murmurs on auscultation. An electrocardiogram showed a complete atrioventricular block, with a ventricular rate of 32 per minute and a QRS duration of 0.18 seconds. Chest X-ray showed no cardiac enlargement nor lung parenchymal abnormalities. Transthoracic echocardiography and routine laboratory tests were normal. A permanent pacemaker was inserted without incident.

Discussion:-

Cardiac involvement is a common cause of death in polymyositis with a frequency increasing with age. Reported manifestations include essentially: left ventricular diastolic dysfunction, hyperkinetic state, myocarditis, pericarditis, mitral valve prolapse, coronary heart disease and vasospastic angina.

ECG abnormalities observed in idiopathic inflammatory myopathies occur in 33–72% [1,3,4,5] and are represented mainly by: atrial and ventricular arrhythmias, bundle branch blocks, A-V blocks, high-grade heart blocks, ventricular premature beats, left atrial abnormalities, abnormal Q-waves as well as non-specific ST-T wave changes.

Infiltration, degeneration, regeneration and fibrous replacement of the electrical conduction system of the heart may account for the variety of cardiac conduction abnormalities observed [2,6]. In detailed necropsy studies, histological changes were found in the conduction system, with the presence of lymphocyte infiltration and sinoatrial node fibrosis [7,8] which may explain the presence of conduction disorders in patients with idiopathic inflammatory myopathies. However, as has been illustrated in the two largest series [1,9], the histological correlation of electrocardiographic abnormalities may be missing.

In a longitudinal study, abnormal ECG was independent of signs of active disease in skeletal muscle activity. Progression of ECG abnormalities can occur under treatment, in the course of disease flare, or even during clinical remission [8,10]. In our case, the period between the beginning of the symptoms and occurrence of complete AV block was about 10 years and the exact beginning of this conductive disorder is unknown. Hence the importance of integrating systematically the ECG in examinations and follow-up of these patients, given that evolution is often asymptomatic.

Key Messages:

Cardiac manifestations may be apparent at the time of myositis diagnosis but may also develop and become manifest after initiating the treatment.

The possibility of a silent cardiac involvement warrants systematic explorations: ECG, Holter and Echocardiography even in patients in remission of the disease.

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