

## Anti-Mi2 Antibody Positive Dermatomyositis with Cardiac involvement in Young African Woman: A Case Report

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### Abstract

### Case Report

Dermatomyositis (DM) is a type of idiopathic inflammatory myopathy characterised by the presence of DM-specific antibodies. Cardiac involvement in DM is uncommon. A 35-year-old Senegalese woman presented with muscle weakness, dysphagia, arthralgia and palpitations. Creatine Kinase was elevated at 1291 UI/L, and Anti-Mi2 was positive. Echocardiogram demonstrated tachycardia with subepicardial ischemia. Echocardiography showed slight enlargement of the right ventricle and a moderate reduction in left ventricular ejection fraction at 43 %. Computed Tomography pulmonary angiography was normal. The diagnosis of Anti-Mi2 positive DM with cardiac involvement was made. She was started on Prednisone, Hydroxychloroquine Methotrexate, and cardiac protection drugs (Ramipril and Bisoprolol) which led to an improvement of symptoms and biochemical markers. Cardiac involvement may occur in cases of anti-Mi-2-positive DM, hence the importance of systematic cardiac evaluation to avoid diagnostic delays.

**Keywords:** Dermatomyositis, Anti-Mi2 antibodies, Cardiac involvement, Idiopathic inflammatory myopathy, Senegal, Heart failure.

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## BACKGROUND

Dermatomyositis (DM) is a type of idiopathic inflammatory myopathy mainly characterised by muscular (proximal muscle weakness), skin involvement (Gottron papules, heliotrope rash) and histological patterns (perifascicular atrophy, perimysial and perivascular inflammatory infiltrates) [1, 2].

It can be systemic, affecting the lungs, heart, blood vessels, joints, and digestive system [3]. DM is an autoimmune disease distinguished by the presence of DM specific autoantibodies (anti-Mi2, anti-NXP2, anti-MDA5, anti-TIF1 $\gamma$ , anti-SAE) [4]. Cardiac involvement is an important cause of morbidity and mortality in DM [5].

In Africa, DM with cardiac involvement is rarely described, especially with the presence of DM-specific antibodies. Herein, we describe a case of Anti-Mi2-positive DM with cardiac involvement in a young African woman.

## CASE PRESENTATION

A 35-year-old Senegalese woman with a two-year history of myalgia and polyarthralgia presented with muscle weakness, dysphagia, arthralgia, and palpitations with exertional dyspnoea.

On admission, vital signs, including body temperature, were normal. Physical examination revealed proximal upper and lower muscle weakness (muscle testing: grade 3/5) with peripheral polyarthralgia (without deformities or ankyloses). She also presented dysphagia without other digestive symptoms. She had a heliotrope rash without other symptoms, including Gottron's papules, Raynaud's phenomenon, oral ulcers, Sicca symptoms, parotidomegaly, hepatomegaly, lymphadenopathy, or night sweats.

Laboratory findings showed normochromic normocytic anemia at 10.2 g/dl without other abnormalities on the blood count test, elevated serum C-reactive protein at 24 mg/l (normal < 6), and elevated Creatine Kinase at 1291 IU/L (normal < 170). Anti-Mi2a and Anti-Mi2b antibodies were positive. Other,

Myositis-specific antibodies were negative. Otherwise, Anti-CCP, Rheumatoid Factor, anti-DNA were negative. Other Anti-ECT antibodies were negative. ASAT was elevated at 260 UI/L (5-35) and ALAT at 107 UI/L (5-40). Serology of HIV, Hepatitis B, and Hepatitis C was negative. Other laboratory results, including TSH, creatinine, glomerular filtration rates, calcaemia, and 24-hour urinary protein were within the normal limits.

The Electrocardiogram showed tachycardia with subepicardial ischemia. Echocardiography demonstrated slight enlargement of the right ventricle and a moderate reduction in left ventricular ejection fraction at 43 %. Otherwise, Computed Tomography pulmonary and angiogram were normal. NT-proBNP and cardiac troponin were unavailable.

The diagnosis of Anti-Mi2 positive DM with cardiac involvement was made. We started therapy with Prednisone 1 mg/kg/day, Methotrexate (20 mg per week), Hydroxychloroquine (400 mg/day), Omeprazol (20 mg/day) and cardiac protection drugs (Bisoprolol (5mg/day) and Ramipril (5mg/day).

After three months of treatment, the patient had an overall improvement in her symptomatology, and CK decreased at 525 UI/L (Normal < 170), CKmb at 3 ng/ml (normal: 0-6), ASAT at 99 UI/L and ALAT at 42 UI/L.

## DISCUSSION

Cardiac involvement in DM was first reported in 1899 by Oppenheim [6]. The frequency of cardiac involvement in patients with DM varies between 9% and 72% [7]. The incidence of myocarditis in DM patients was 2.1 per 1000 patient-year in a recent large DM cohort [8].

The pathophysiology of cardiac involvement in DM is linked to inflammation of the myocardium (myocarditis). DM is a microangiopathy with capillary lesions and secondary ischaemic changes in muscle fibres [9]. Muscle damage (including myocardium) in DM is believed to be mediated by humoral factors (antibodies and complement system) directed against endomysial capillary endothelial cells. As a result of T cells being present in the muscle biopsy of DM and the HLA-DR association, a role for both adaptive and innate immunity in the development of muscle inflammation may be hypothesised [10]. This autoimmune inflammatory process manifests in the heart as mononuclear inflammatory infiltrates located in the endomysium and perivascular areas, accompanied by degeneration of cardiac myocytes. Histological samples of cardiac tissue revealed the presence of myocarditis, fibrosis (affecting the sinoatrial node and conduction system), with lymphocytic infiltration and band necrosis. Vascular alterations in the coronary arteries have also been reported [11, 12].

Cardiac involvement in DM mainly manifests as palpitations, exertional dyspnea, orthopnea, chest pain, peripheral edema and syncope [5]. Blood tests may reveal elevated levels of cardiac troponin (T and I), CPK-*mb*, and N-terminal pro B-type natriuretic peptide (NT-pro BNP) [13]. The electrocardiogram shows non-specific changes. Among these, ST-T wave changes, premature ventricular contractions, chamber hypertrophy, left anterior bundle branch block, and right bundle branch block are the most common [13]. Regarding Echocardiography, the most common finding is diastolic dysfunction. The deterioration of diastolic dysfunction may develop during the disease course due to the ongoing fibrotic transformation of the myocardium, finally leading to clinically symptomatic heart failure. Two other common Echo abnormalities are hyperdynamic left ventricular contraction and prolapse of the mitral valve [14]. Cardiac magnetic resonance is useful for preclinical diagnosis of cardiac involvement in DM patients [14].

Regarding the treatment of dermatomyositis with cardiac involvement, there are no specific recommendations; however, treatment includes glucocorticoids, conventional synthetic Disease-Modifying Anti-Rheumatic Drugs (Methotrexate, Azathioprine, Mycophenolate mofetil), Intravenous immunoglobulin (IVIG), Rituximab and cardiac protection drugs (Beta-blockers, Angiotensin-Converting Enzyme inhibitors).

## CONCLUSION

Cardiac involvement may occur in cases of anti-Mi-2-positive DM, hence the importance of systematic cardiac evaluation to avoid diagnostic delays. The combination of prednisone, methotrexate, hydroxychloroquine, and cardiac protection drugs appeared to be effective in our patient.

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