



## A Case Report and Literature Review on Dermatomyositis Following Heart Transplantation

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### Abstract

**Background and objects:** Cardiac involvement has been well honored in cases with dermatomyositis (DM) and polymyositis (PM) with a variable frequency between 9 and 72. Still, clinically significant heart involvement in DM/ PM is fairly occasional and there have been rare reports of cardiac transplantation in DM. Our points were to describe a case of severe cardiac involvement in DM taking heart transplantation and review the literature of cardiac complaint in DM and PM.

**Method:** A case with dermatomyositis who was admitted to our institution with severe heart failure is described. Pathology of the case's cadaverous and cardiac muscle is reviewed. A MEDLINE database hunt of reports of cardiac involvement in DM and PM was also conducted.

**Results:** A 36 time-old man with DM presented with severe heart failure to our institution for evaluation of heart transplantation. After a three month hospitalization he passed successful cardiac transplantation. Pathological examination of his explant heart revealed a pattern of inflammation and damage analogous to DM in cadaverous muscle. The case is presently doing well, 20 months post-transplant, and is maintained on tacrolimus, collect, rituximab, and low cure prednisone. To our knowledge, this is the first case report of heart transplantation in dermatomyositis in which the muscle pathology is analogous in both heart and cadaverous muscle.

**Conclusion:** Severe cardiac involvement taking transplantation is rare in dermatomyositis but does do and appears to be related to a analogous seditious process as noted in the cadaverous muscle.

**Keywords:** Dermatomyositis; Seditious myopathy; Cardiomyopathy; Cardiac transplantation; Orthotopic heart transplant

### Introduction

Dermatomyositis (DM) and polymyositis (PM) are both idiopathic seditious myopathies (IIM) characterized by proximal muscle weakness and seditious cell infiltrates within the cadaverous muscle. Cardiac involvement similar as conduction abnormalities, arrhythmias, congestive heart failure, valvular/ pericardial/ coronary roadway complaint and left ventricular dysfunction has been reported as a common cause of death. Severe cardiac involvement in IIM is rare and only two cases of cardiac transplant in IIM have been reported, one in a case with PM and the other in which the cardiac muscle pathology showed giant cell myocarditis. In this report, we describe a case with severe cardiac involvement in DM taking heart transplant and review the literature of cardiac complaint in DM and PM [1].

### Materials and Method

A 36 time old African American joker preliminarily in good health presented to an outside installation with verbose muscle pain and proximal muscle weakness. He described difficulty raising his arms above his head and climbing stairs. He'd a pruritic, popular rash on his upper reverse and anterior casket and complained of itching and swelling around his eyes, coarse voice, and swelling and stiffness of his hands. Labs were significant for a creatine phosphokinase (CPK) of 12,006 and MRI of bilateral femurs showed verbose muscle edema. He was started on prednisone at 80 mg daily for possible myositis. He latterly developed dysphagia, and a muscle vivisection of his left ham showed severe seditious myopathy with perivascular inflammation and zones of visage- and perifascicular atrophy harmonious with dermatomyositis or variant. Two months after starting prednisone, the case began methotrexate at 15 mg daily and the prednisone was phased. Due to patient muscle weakness and CPK elevation after 6 weeks on methotrexate, rituximab was added. Within 6 months of

donation, the case developed severe fatigue and briefness of breath. He was set up to have cardiomyopathy with an ejection bit of 10 – 15 and normal coronary highways [2]. Over the posterior 4 months he'd multiple sanitarium admissions at an outside installation with heart failure complicated by atrial fibrillation, ventricular tachycardia, gastrointestinal bleeding with haemoptysis, and a lower extremity deep venous thrombosis. The case was transferred to our installation for evaluation of orthotopic heart transplantation (OHT). Upon admission to our installation, the case had residual lower extremity proximal muscle weakness and a mild hyperpigmented rash on his upper casket and back. He was entering prednisone 10 mg daily, MTX 25 mg SQ daily and rituxan was cured 7 months previous to admission. CPK was 126 IU/L. Serologic testing showed the presence of ananti-Ku antibody. The case had a complicated sanitarium course including cardiogenic shock taking placement of anintra-aortic balloon pump followed bybi-ventricular help bias (VADs). Immunosuppressive specifics weren't increased due to concern regarding biVAD infections by the Cardiology Transplant service which would avert OHT [3]. A month following his original admission, the case had bleeding and purulent discharge from his VAD spots and the methotrexate was held and prednisone was dropped to 7 mg daily. The case's lower extremity weakness worsened and hoarseness of his voice returned. His methotrexate wasre-initiated at

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10 mg weekly, still after several weeks, his CK position rose to 1400 IU/L and moderate cure prednisone at 40 mg daily and IVIG were initiated. Within 2 weeks of this DM flare, the case passed a successful orthotopic heart transplant. On examination of the explanted heart valvular circumferences were within the normal high limits. The tricuspid (13.2 cm in circumference), mitral (11.7 cm in circumference) and aortic cusps (6.8 cm in circumference) appeared mildly thickened. Mitral chorda were attached to both papillary muscles. All three pulmonary cusps were normal and 6.8 cm in circumference. Pathology of the case's cadaverous and cardiac muscle is shown in detail. Towel samples attained from explanted heart included left and right ventricular wall and papillary muscle. Histologic examination showed a multifocal habitual and severe fibrosing myocarditis in all areas examined. Active myocardiocyte injury was estimated using non-specific esterase (NSE), an enzyme response with propensity to describe lysosomal activation and active myodegeneration [4]. This comprised of foci of exertion and degeneration also multifocal and of variable inflexibility ranging from single fiber necrosis to large areas of perifascicular injury. Perifascicular active myofiber injury and microvascular immunoreactivity with antibodies to membrane-attack-complex (c5b9) were more prominently noted in samples from right ventricular wall. "Active" mononuclear inflammation was present and moderate in viscosity. This comprised substantially of a T-lymphocytes linked in the area incontinently continuous to the fascicles (perimysium) and adjoining connective tissue. Both CD4-aided and CD8- cytotoxic T cells were present with no apparent ascendance [5].

## Discussion

Cardiac involvement in DM and PM has been well described in the literature with a reported prevalence between 9 and 72 depending on patient selection and styles of discovery. Still, clinically significant cardiac involvement is much less common [6]. A methodical review reported EKG and Holter cover abnormalities similar as conduction blights, ST-T changes, and frequent atrial or ventricular unseasonable beats in over to 85 of IIM cases. Other modalities for cardiac evaluation included echocardiogram showing wall stress, valvular and pericardial abnormalities in over to 62 of cases Technetium-99m-pyrophosphate (99mTc-PYP) scintigraphy, and cardiac MRI showing abnormal improvement in a study of 4 cases which bettered after corticosteroid treatment. The most constantly reported clinical symptom of cardiac involvement is congestive heart failure although coronary roadway complaint including acute myocardial infarction is also increased in cases with seditious myopathies. Cases with DM/PM have increased mortality compared to the general population with cardiopulmonary complaint as the leading cause of death.

Two case reports have preliminarily been published regarding cardiac transplantation in myositis. The most recent report described a case of fulminant mammoth-cell myocarditis in a case with possible DM diagnosed two weeks previous to donation [7]. The case was treated with high boluses of methylprednisolone and 2 boluses of IVIG but progressed to cardiogenic shock fleetly and eventually passed a successful orthotopic heart transplant. Histological examination of the explanted heart revealed foci of mixed seditious cells admixed with multinucleated giant cells/ sinking myocardial filaments, harmonious with fulminant mammoth-cell myocarditis. The alternate case report described a case with polymyositis who developed severe heart failure over 7 times following opinion, and eventually passed successful heart transplantation. The pathology of the explant heart wasn't considerably described in this report. Fresh necropsy studies assessing the histopathology of myocarditis in PM cases have described a verbose

interstitial and perivascular mononuclear cell insinuate analogous to the seditious changes seen in the affected cadaverous muscle still further detailed descriptions, particularly in DM, are lacking [8].

In the current work, we detail the cardiac pathology and also report parallels observed between the myocardium and the cadaverous muscle particularly those pertaining to characteristic pattern of DM injury. The most notable resemblance was the perifascicular distribution of atrophy and damage in the cadaverous muscle which was matched by zones of supplemental fiber damage in the cardiac muscle. Violent perifascicular alkaline phosphatase reactivity, specific of DM was also noted in both cardiac and cadaverous muscle. The multifocal and variable nature of the complaint from region to region was analogous to the pattern of injury generally seen in dermatomyositis affecting cadaverous muscle. Eventually, membrane attack complex (MAC) overexpression in the vasculature, a hallmark sign of DM pathology, was noted in cornucopia in both cadaverous and cardiac muscle microvasculature in the current case. Similar histologic parallels suggest that dermatomyositis is indeed a systemic complaint that may beget muscular inflammation and damage beyond the cadaverous muscle. Myocardial findings were indeed limited in comparison to the features available on cadaverous muscle vivisection. Still, the ultimate dated a time previous and thus not representative of the supplemental cadaverous muscle complaint at the time of the OHT [10].

## Conclusion

Although utmost cases of cardiac involvement in DM are subclinical, it's important to realize that severe cases of cardiac involvement taking transplantation do, in which case transplant may be lifesaving. The histopathologic study suggests a pattern of analogous seditious damage in the heart as noted in the cadaverous muscle.

## Conflicts of Interest

None

## Acknowledgment

None

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