Journal of Cardiovascular Disease Research

ISSN: 0975-3583, 0976-2833 VOL 16, ISSUE 1, 2025

A Rare Case Report

WEAK MUSCLES, STRONG CLUES: DIAGNOSING DERMATOMYOSITIS- A RARE CASE REPORT

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Received: 15-11-2024 / Revised: 18-12-2024 / Accepted: 08-01-2025

Abstract

Dermatomyositis is a rare idiopathic inflammatory myopathy characterised by proximal muscle weakness & distinct dermatological features. In this article we report the case of Dermatomyositis without coexisting malignancy.

INTRODUCTION

Dermatomyositis is an idiopathic inflammatory myopathy characterised by progressive symmetrical muscle weakness, elevated muscle enzymes [1]. Muscle involvement usually manifests as proximal muscle weakness initially with or without myalgia or tenderness. Other important clinical features of DM include the presence of ILD[2]. The prevalence of DM is 1 in 100000 with women being more affected than men[3].

Case Description:

A 44 yr female patient presenting to Afaque Hospital, Vijayapura with complaints of weakness & myalgia in both her shoulders & upper limbs since 2 months, the weakness was bilateral, gradual in onset, progressive in nature to cause difficulty in lifting hands above shoulders & performing day to day activities, she has also experienced noticeable swelling of face particularly around the eyes with violet to purplish discolouration along with puffiness & tightness of skin. Additionally she mentions shortness of breath, especially after exertion or moderate physical activity. There was no history of fever, dysphagia or joint pain.

On GPE, her vitals were normal, rashes were seen on her face with distinctive purplish discoloration on the malar prominence (heliotropic rash) and over neck, shoulders and upper back region(shawl sign). The neurological, respiratory, cardiovascular and abdominal systems were normal.

Laboratory findings:

Laboratory investigation revealed ESR, done by westergren tube method to be 56 mm/hr, Hb 11.5 gm% and WBC to be 9440 cells/cumm, complete hemogram was measured by automated Erba H 360 & is presented in Table 1. Biochemical panel was measured by semi automated Erba Chem 5X and revealed CK to be 3586.6 and ANA, done with immunofluorescence was Mild Positive, LFT showed slight rise in AST 179 and ALT 103 (Table 2).

ISSN: 0975-3583, 0976-2833 VOL 16, ISSUE 1, 2025

Table 1		
Parameter	Result	Normal Range
Hb	**11.5 gm%**	12-16
RBC	4.66 mil/cumm	3.5-5.5
PCV	36 %	36-46
MCV	77.25 fl	80-100
MCH	**24.68 pg**	25.4-34.6
MCHC	31.94 %	31-36
WBC	9440 cells/cumm	4000-11000
Neutrophils	**77 %**	40-70
Lymphocytes	**18 %**	20-45
Eosinophils	05 %	2-8
Monocytes	0 %	1-6
Basophils	0 %	0-1
PLT	3.34 L/cumm	1.5-4.5
ESR	**56 mm/hr**	0-20

Note: Abnormal values are indicated in bold.

CXR shows left sided homogeneous opacities , 2D Echo revealed structurally normal heart with EF ~60%, Based on the above findings a diagnosis of Dermatomyositis was made.

Table 2		
Parameter	Result	Normal Range
S. creat	0.8 mg%	0.6-1.4
S. uric acid	6.0 mg%	3.2-6.4
Rheumatoid factor	17.6 IU/mL	0-30
S. calcium	9.9 mg%	8.4-10.5
S. electrolytes		
sodium	135 mmol/l	135-150
potassium	3.8 mmol/l	3.5-5.5
chloride	109 mmol/l	94-110
ionic calcium	1.24 mmol/l	1.10-1.35
LFT		
total bilirubin	0.3 mg%	0.2-1.3
direct bilirubin	0.2 mg%	0.1-0.4
indirect bilirubin	**0.1** mg%	0.2-0.6
Total protein	5.5 mg%	6.3-8.2
Albumin	**3.0** mg%	3.5-5.0
Globulin	2.5 mg%	1.5-3.0
A:G	1.2	1.1-2.3
AST	**179 IU/L**	14-36
ALT	**103 IU/L**	9-52
ALP	73 IU/L	38-126
CPK	**3586.6 IU/L**	25-192
ANA	**Mild Positive**	

Note: Abnormal values are indicated in bold.

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She was treated with 40mg Prednisone with improvement in muscle strength, At present she is put on Prednisone 10mg OD, orally, Methotrexate 7.5mg weekly once PO and Folic acid 5mg OD PO for maintenance therapy.

Discussion:

Dermatomyositis is an autoimmune condition characterised by skin rashes & progressive muscle weakness, classified under Idiopathic Inflammatory Myopathies (IIM), Often coexisting with extramuscular manifestations, such as ILD, Arthritis and Malignancies.[4]
The pathogenesis of DM is multifactorial, complex and incompletely understood. MSA are antibodies that are exclusively associated with a diagnosis of an IIM, DM specific antibodies include anti-Mi2, anti-melanoma differentiation-associated protein 5 (MDA5), anti-NXP2, anti-TIF1, and anti-small ubiquitin-like modifier activating enzyme (SAE)[5]

The hallmark cutaneous manifestations include Heliotropic rash, a macular reddish purple rash on eyelids and Violaceous/Erythematous macular rash on anterior of chest & neck (V-sign) or back (shawl sign) or lateral hips (holster sign) and over bony prominences of knuckles, knees, & Elbows. Gottron rash/papules are raised, smooth, indurated, reddish violet lesions on knuckles, elbows and knees. [6,7,8]

Management: The treatment of choice is high dose of oral prednisone which must be initiated early to improve muscle weakness .In situations where Prednisone cannot be used, second-line agents such as methotrexate and azathioprine will be appropriate. Rituximab, intravenous immunoglobulin (IVIG), and other biologics are useful in patients who developed resistance to therapy.Antipruritics, topical steroids may be employed to treat superficial skin disease.[9]
The systemic options include Antimalarials (HCQ's), Immunosuppressants (Methotrexate or Mycophenolate mofetil), IVIG, possessing immunomodulatory effects, Janus kinase inhibitors (Tofacitinib), Ruxolitinib impairs signalling through multiple signalling receptors.[10]

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