ORIGINAL RESEARCH PAPER
Volume - 14 | Issue - 09 | September - 2025 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

JUVENILE DERMATOMYOSITIS PRESENTING AFTER ACCIDENTAL TRAUMA: A CASE REPORT

Neurology

Dr. Pazhaniyandi Pillai.K Senior Resident, Department of Neurology, Tirunelveli Medical College, Tamil Nadu, India

ABSTRACT

Juvenile dermatomyositis (JDM) is a rare, idiopathic inflammatory myopathy of childhood characterized by proximal muscle weakness and pathognomonic cutaneous manifestations. We report the case of a 10-year-old boy who initially presented with progressive proximal muscle weakness following an accidental fall, leading to diagnostic delay. The diagnosis was subsequently confirmed by elevated muscle enzymes, electromyography findings, cutaneous features, and anti-Mi-2 antibody positivity. Early initiation of corticosteroids and immunosuppressive therapy led to clinical improvement. This case highlights the need for high clinical suspicion in children presenting with unexplained weakness after trauma, and emphasizes the role of myositis-specific autoantibodies in prognostication, even in resource-limited settings.

KEYWORDS

INTRODUCTION

Juvenile dermatomyositis (JDM) is the most common idiopathic inflammatory myopathy in children, although its incidence is only 2–4 per million per year[1]. It is an autoimmune vasculopathy characterized by symmetric proximal muscle weakness and characteristic rashes such as Gottron's papules and heliotrope rash[2].

Unlike adult dermatomyositis, JDM is not associated with malignancy but carries a higher risk of calcinosis and lipodystrophy[3]. Advances in immunopathology, particularly the role of type I interferon pathways and myositis-specific autoantibodies (MSAs), have improved diagnostic precision and prognostic stratification[5].

We present a child with JDM whose symptoms were initially attributed to post-traumatic weakness, leading to a delayed diagnosis. Anti-Mi-2 antibody positivity in this patient adds to the sparse Indian data on antibody profiles in JDM.

CASE REPORT

Patient Profile

A 10-year-old male, firstborn of a non-consanguineous marriage, with normal developmental milestones and complete immunization, presented with progressive weakness. There was no family history of autoimmune or neuromuscular disease.

History of Present Illness

The child sustained an accidental fall on 5/6/25, after which he remained ambulant. On 25/6/25, he suffered another fall with hip injury, followed by swelling of limbs and face, and persistent low-grade fever for three weeks. Despite treatment at a homeopathy clinic, his weakness progressed — initially inability to walk, then sit, and eventually inability to lift his head.

There was no history of dysphagia, dyspnea, Raynaud's phenomenon, or photosensitivity.

Examination

General: Weight 23 kg (underweight).

Skin: Multiple hypopigmented plaques on the trunk and back; Gottron's papules over bilateral knuckles; heliotrope rash absent.

Musculoskeletal: Proximal muscle weakness involving neck flexors, shoulder and pelvic girdle; unable to sit or stand without support; distal strength preserved.

Systemic: Other systems were normal.

Investigations

Hemogram: Hb 10.2 g/dL, TLC 8100, ESR 18 mm/hr.

Muscle enzymes: CK 1085 U/L (elevated), LDH 591 U/L (elevated), AST elevated.

Electromyography: Short, small polyphasic motor unit potentials with fibrillations and bizarre repetitive discharges, consistent with inflammatory myopathy.

Chest X-ray: Normal.

Mantoux test: Negative.

Autoantibody profile: ANA positive (1:320 speckled). Myositis-specific antibody panel showed anti-Mi-2 positivity, while anti-MDA5 and anti-TIF1γ were negative.

Clinical Impression

Based on Bohan and Peter's criteria[1], the diagnosis of Juvenile Dermatomyositis was established (proximal muscle weakness, elevated muscle enzymes, EMG features, Gottron's papules).

Treatment and Clinical Course

The patient was treated with:
IV methylprednisolone (375 mg/day × 3 days),

Oral prednisolone (1 mg/kg/day),

Methotrexate (15 mg/m² weekly),

High-protein diet, calcium/vitamin D, zinc, physiotherapy.

Over two weeks, he improved in neck holding and sitting balance. He is under follow-up with rheumatology.

REVIEW OF LITERATURE

The first description of dermatomyositis was given by Wagner in 1863. JDM is the most common inflammatory myopathy of childhood, accounting for nearly 85% of pediatric idiopathic inflammatory myopathies[4].

Autoantibody profiles have significant prognostic implications:

Anti-Mi-2: Associated with florid skin rash, milder systemic involvement, and good steroid responsiveness[5].

Anti-MDA5: Associated with rapidly progressive interstitial lung disease and poor prognosis[6].

Anti-TIF1γ: Associated with severe cutaneous disease and, in adults, malignancy[7].

Anti-NXP2: Linked with calcinosis and severe weakness[8].

Indian data on antibody prevalence in JDM remain sparse. Malaviya and Singh[5] described clinical patterns in Indian children but did not include MSA profiling. Thus, antibody-documented cases such as ours contribute to filling this epidemiological gap.

DISCUSSION

The present case highlights several novel aspects:

1. Unusual trigger and diagnostic delay: The child's weakness was initially attributed to post-traumatic sequelae, delaying recognition. Trauma-associated onset of JDM has rarely been reported.

2. Delayed dermatological features: Gottron's papules appeared later in the disease course, whereas cutaneous manifestations are usually the earliest sign[3].

3. Anti-Mi-2 positivity: Anti-Mi-2 antibody positivity, while rare in Indian children, is associated with favorable prognosis[5]. Reporting such cases enhances understanding of regional immunological profiles.

4. Resource-limited context: In the absence of muscle MRI or biopsy, antibody profiling combined with clinical and EMG data confirmed the diagnosis, underscoring its diagnostic value in low-resource settings[1].

Thus, the novelty of this case lies in its trauma-associated onset, diagnostic delay, and antibody-proven JDM in an Indian child, highlighting the need for awareness of atypical presentations.

CONCLUSION

Juvenile dermatomyositis should be considered in children with progressive proximal muscle weakness, even when the onset follows trauma. Cutaneous signs such as Gottron's papules are highly specific and should prompt further evaluation. Myositis-specific antibody testing, particularly anti-Mi-2, not only confirms diagnosis but also provides prognostic information. Reporting antibody-positive JDM cases from India contributes valuable epidemiological and clinical insights.

REFERENCES

1. Rider LG, Miller FW. Classification and treatment of the juvenile idiopathic inflammatory myopathies. Rheum Dis Clin North Am. 1997;23(3):619–55.
2. Pachman LM. Juvenile dermatomyositis: Pathophysiology and disease expression. Pediatr Clin North Am. 1995;42(5):1071–98.
3. Malaviya AN, Singh RR. Juvenile dermatomyositis in India. Indian J Pediatr. 1989;56(3):345–52.
4. Gowdie PJ, Manlhiot C. Juvenile dermatomyositis: epidemiology and pathogenesis. Pediatr Rheumatol. 2013;11:17.
5. Tansley SL, Betteridge ZE, McHugh NJ. Myositis-specific autoantibodies in juvenile and adult myositis: utility for diagnosis and prognosis. Curr Opin Rheumatol. 2013;25(6):772–777.
6. Sato S, Hoshino K, Satoh T, et al. Autoantibodies to MDA5 and clinical manifestations in juvenile dermatomyositis. Arthritis Rheumatol. 2016;68(6):1534–1541.
7. Betteridge ZE, Gunawardena H, Chinoy H, et al. Anti-TIF1-γ antibodies define a juvenile dermatomyositis subset with severe cutaneous phenotype. Arthritis Rheum. 2011;63(9):2624–2631.
8. Rider LG, Nistala K. Update on juvenile dermatomyositis: clinical phenotypes, disease course, and outcome measures. Rheum Dis Clin North Am. 2018;44(4):623–638.