


CASE REPORT **OPEN ACCESS**

Juvenile Dermatomyositis Triggered by Influenza B: A Case Report on Viral-Induced Autoimmunity

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ABSTRACT

A previously healthy 13-year-old boy developed juvenile dermatomyositis (JDM) shortly after a confirmed influenza B infection, presenting with progressive proximal muscle weakness and classic cutaneous findings. Laboratory tests revealed elevated muscle enzymes and myositis-specific autoantibodies, supporting the diagnosis. The temporal association suggests a potential post-viral autoimmune trigger, highlighting influenza B as a possible environmental factor in JDM pathogenesis. This case reinforces the need for heightened clinical awareness and further research into virus-associated autoimmune mechanisms in pediatric myopathies.

1 | Introduction

Idiopathic inflammatory myopathies (IIMs) [1] in children are rare autoimmune disorders characterized by proximal muscle weakness and characteristic cutaneous findings, including heliotrope rash and Gottron's papules. Juvenile dermatomyositis (JDM) [2] is the most common form.

Compared to adult-onset dermatomyositis (DM), JDM typically presents more acutely and is associated with more prominent vasculopathy [3], a higher risk of ulcerative skin lesions, and a near-universal pattern of muscle involvement [4]. Calcinosis is also significantly more frequent in JDM than in adults [5]. In contrast, adult DM is more often associated with malignancy and interstitial lung disease, both of which negatively affect prognosis. Despite treatment advances, JDM can lead to chronic disability, especially if diagnosis or therapy is delayed [6].

The exact pathophysiology of this group of diseases remains incompletely understood. Previous studies have demonstrated a

crucial role of the innate immune system in JDM pathogenesis, highlighting dendritic cells and macrophages as key players in the development of cutaneous lesions and inflammatory muscle damage [7]. Several theories have been proposed regarding potential triggers. Among the most studied are ultraviolet (UV) radiation exposure and viral infections [8, 9], both of which may play crucial roles in initiating an immune response in genetically susceptible individuals.

Viral infections have been suggested as potential triggers for JDM because of their ability to induce aberrant immune activation. Various pathogens, including Epstein-Barr virus, enteroviruses, parvovirus B19, *Mycoplasma pneumoniae*, and *Toxoplasma gondii*, have been implicated in the onset of the disease in some patients [8]. Notably, up to 70% of patients with JDM report a preceding infection as a possible or probable trigger [10].

However, the role of influenza virus (A or B) as an immune trigger remains less clearly established, despite sharing similar

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pathophysiological mechanisms with other implicated pathogens. Influenza infections, especially influenza B, may serve as environmental triggers for autoimmunity in genetically predisposed individuals [11]. While cases of benign, self-limited myositis have been reported [12], the capacity of influenza B to initiate sustained autoimmune responses resulting in the muscle and skin inflammation typical of JDM remains insufficiently studied.

In this case report, we present a 13-year-old boy who developed JDM following an influenza B infection, highlighting the potential association between viral infections and the onset of autoimmune diseases.

2 | Case Report

A previously healthy 13-year-old male presented with newly developed cutaneous lesions, accompanied by weakness and myalgia, which significantly impacted his daily life, including his regular participation in sports. Approximately 1 month prior, he experienced flu-like symptoms, which were confirmed as an influenza B infection via a nasal swab. One week later, the patient developed severe muscle soreness, accompanied by characteristic cutaneous lesions. These included erythematous to violaceous facial lesions predominantly involving the malar and periorbital regions, extending to the nasolabial folds (features compatible with heliotrope rash), and erythematous papular lesions on the dorsal surfaces of the metacarpophalangeal (MCP) and proximal interphalangeal (PIP) joints, which were clinically compatible with Gottron papules (Figures 1 and 2).

On physical examination, the patient exhibited mild proximal muscular weakness, notably affecting the cervical musculature,



FIGURE 1 | Facial erythema in a malar distribution extending to the nasolabial folds, with associated periorbital edema (not shown), characteristic of JDM.

deltoids (especially left side), and quadriceps muscles, which were more pronounced on the right side. The Childhood Myositis Assessment Scale (CMAS) [13] revealed mild weakness, with a score of 45 out of 52. Additionally, periungual erythema and capillary abnormalities were noted on nailfold inspection. No Raynaud's phenomenon, photosensitivity, arthralgia, fever, or systemic symptoms such as weight loss or gastrointestinal manifestations were reported. There was no known family history of autoimmune or rheumatic diseases.

Laboratory tests revealed leukopenia (3850 cells/ μ L) and significantly elevated muscle enzymes (CK 3997 U/L (reference range 46–171), AST 171 U/L (5–34), ALT 80 U/L (10–49), LDH 510 U/L (120–246)). Antinuclear antibodies (ANAs) were positive at a titer of 1:320, showing a fine speckled nuclear pattern (AC-4). Anti-transcription intermediary factor 1- γ (Anti-TIF1- γ) autoantibodies were strongly positive. Anti-Jo1, anti-Scl70, anti-Ro52, anti-MDA5, and other myositis-specific antibodies were negative. Serological tests were negative for Epstein–Barr virus, parvovirus B19, HIV, CMV, *Toxoplasma*, and *Coxiella*. Initial *Mycoplasma* IgM positivity, detected in the first analysis, was not confirmed when the same sample was retested, suggesting a false positive result. *Mycoplasma* IgG was negative. PCR results for influenza B (as well as influenza A, respiratory syncytial virus, and SARS-CoV-2) were negative at admission, but influenza B had been previously confirmed prior to symptom onset.

Magnetic resonance imaging demonstrated diffuse muscle edema in both shoulder and pelvic girdles, which was consistent with inflammatory myopathy (Figure 3). Prior to admission, a skin biopsy revealed nonspecific findings of interface dermatitis without mucin deposition or immune deposits on immunofluorescence, consistent with the clinical suspicion of DM.

The patient was diagnosed with JDM, supported by clinical, biochemical, immunological (positive anti-TIF1- γ antibodies), imaging, and histological evidence. Initial management included intravenous methylprednisolone boluses (500 mg/day for three consecutive days), followed by oral prednisone (1 mg/kg daily)



FIGURE 2 | Dorsal view of both hands revealing erythematous, scaly papules over the metacarpophalangeal and interphalangeal joints, characteristic of Gottron's papules. Periungual erythema was also noted.

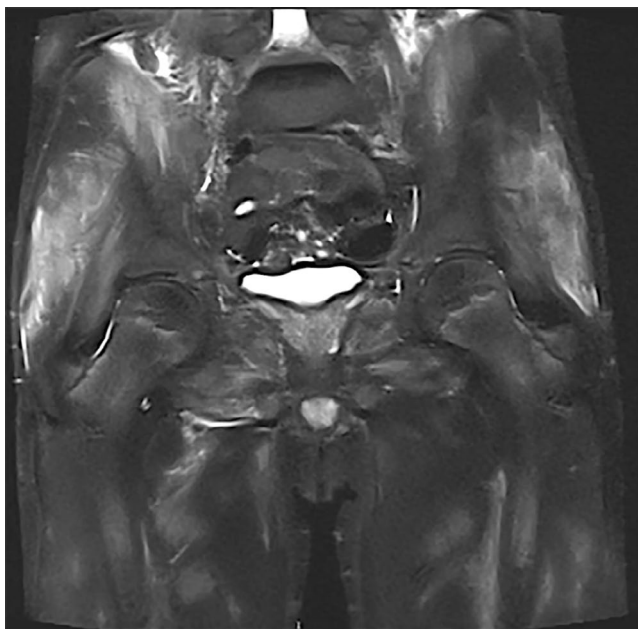


FIGURE 3 | Magnetic resonance imaging (MRI) STIR (Short Tau Inversion Recovery) sequence illustrating coronal pelvis view with hyperintensity in iliopsoas muscles.

and subcutaneous methotrexate. Clinical improvement was observed rapidly after corticosteroid initiation, with partial resolution of muscle weakness and significant improvement in skin lesions. While there is limited published evidence quantifying the expected time to response in JDM, our clinical experience suggests that patients with a high inflammatory burden often show prompt improvement following corticosteroid therapy. Follow-up laboratory tests after glucocorticoid boluses revealed significant biochemical improvement, with reductions in creatine kinase (1613 U/L), as well as improvements in other muscle-related parameters.

The patient remains under multidisciplinary follow-up with pediatric rheumatology, dermatology, and rehabilitation services.

3 | Discussion

Influenza viruses, members of the Orthomyxoviridae family [14], include four types (A–D), with types A and B responsible for most seasonal epidemics in humans. Influenza A is divided into H and N subtypes and has pandemic potential due to antigenic shifts. In contrast, influenza B circulates only in humans and causes recurrent seasonal outbreaks through antigenic drift [15]. Though less likely to cause pandemics, influenza B can lead to severe illness, particularly in children and immunocompromised individuals. Its ability to replicate beyond the respiratory tract may contribute to systemic immune dysregulation [16].

Type I interferon (IFN-I) is a key antiviral response, inducing interferon-stimulated genes (ISGs) [17] in infected tissues, including muscle and blood—a signature also seen in JDM. Among these, interferon-induced transmembrane (IFITM) proteins, especially IFITM3, restrict influenza replication and spread.

Although IFN-I signaling is essential for viral defense, its persistent activation can lead to excessive inflammation and contribute to autoimmune pathogenesis, as observed in JDM. In affected JDM patients, CD14+ monocytes produce oxidized mitochondrial DNA (oxmtDNA) [18], which acts as a powerful stimulator of IFN-I expression, further amplifying immune activation and sustaining muscle inflammation.

Additionally, chronic IFN-I activation alters CD8+ T-cell responses [19], promoting their infiltration into skeletal muscle, where they contribute to tissue damage and autoantigen presentation. This finding supports the hypothesis that acute viral infections such as influenza B can act as triggers or exacerbating factors in JDM for genetically predisposed individuals. Yet, why such infections lead to diseases like JDM in only a few (despite widespread exposure in the population) remains an unanswered question. Could it be explained by subtle genetic variants [20], epigenetic factors, or a specific immunological threshold? [9] The mechanisms underlying this selective vulnerability remain to be fully elucidated.

Recent transcriptomic studies [21] of JDM muscle biopsies have demonstrated the upregulation of nearly all 2',5'-oligoadenylate synthetase (OAS) system genes, suggesting a strong link between OAS activation and disease pathogenesis. Notably, 39.9% of modulated genes in JDM overlap with those activated by polyinosinic-polycytidylic acid (poly(I:C)), a synthetic analogue of double-stranded RNA (dsRNA), further supporting the hypothesis that viral mimicry triggers autoimmune activation. The overexpression of OAS genes in JDM muscle tissue, coupled with their known ability to detect viral RNA and activate RNase L-mediated degradation, indicates that influenza B infection could initiate a cascade leading to prolonged immune activation and muscle damage. These findings highlight the need for further research into the role of the OAS pathway in post-viral autoimmunity and its potential as a therapeutic target in JDM.

Beyond IFN-I and OAS-mediated responses, toll-like receptors (TLRs) further contribute to the innate immune response [22]. Influenza virus engages TLRs, particularly TLR3 and TLR7, whose expression has been found to be elevated in monocytes and dendritic cells from influenza-infected patients compared to healthy controls. While this mechanism helps control viral replication, it may also contribute to excessive immune activation, potentially worsening autoimmune conditions such as JDM.

Taken together, these findings suggest that influenza infection, particularly in genetically predisposed individuals, may act as a potent environmental trigger in JDM through multiple immune-mediated pathways. A better understanding of these mechanisms is essential for developing targeted therapeutic approaches that can mitigate virus-induced immune dysregulation in JDM and other inflammatory myopathies.

4 | Conclusion

This case reinforces the hypothesis that influenza B may act as a trigger for JDM by inducing a strong type I interferon response, which can amplify immune dysregulation in predisposed individuals. Recognizing this link is essential for early diagnosis

and intervention, potentially reducing disease complications. Increased awareness of post-viral autoimmune conditions may improve patient outcomes and guide further research into the mechanisms of virus-induced autoimmunity.

Author Contributions

Santiago Dans-Caballero: investigation and writing – original draft. **Miguel Juan-Cencerrado** and **Carmen Mochón-Jiménez:** writing – review and editing and resources. **Rosa Roldán-Molina:** writing – review and editing and conceptualization. **Verónica C. Pérez-Guijo:** writing – review and editing, conceptualization, resources and supervision.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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