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Viral Myositis and Dermatomyositis: Key Diagnostic Differences

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Abstract

Myalgias causing an inability to ambulate after a viral illness is a concerning presentation for children, their parents and healthcare providers. Benign acute childhood myositis (BACM) and post-viral myositis (PVM) are typically distinguishable from juvenile dermatomyositis (JDM), but in exceedingly rare cases, they may present similarly, complicating the diagnosis. We report a unique case of a 4-year-old boy with severe myalgias and inability to ambulate following a norovirus infection. He also exhibited periorbital edema resembling a heliotrope rash, accompanied by significantly elevated levels of creatine kinase (CK), alanine transaminase (ALT), and aspartate transaminase (AST), raising concern for juvenile dermatomyositis, especially as he had a poor response to conservative medical management and his symptoms had persisted for two months. This case underscores the diagnostic challenges in distinguishing BACM/PVM from JDM, particularly when an uncommon viral pathogen like norovirus presents with a heliotrope rash—an eruption often considered pathognomonic for dermatomyositis.

Keywords

Benign Acute Childhood Myositis, Post-Viral Myositis, Juvenile Dermatomyositis, Norovirus

1. Case Report

A previously healthy 4-year-old Caucasian male developed a gastrointestinal infection caused by norovirus, presenting with low-grade fever, decreased oral intake, and fatigue. These symptoms resolved within 48 hours. However, three days later, he exhibited new symptoms, including periorbital edema with an erythematous lace-like macular rash across his eyelids resembling a heliotrope rash, wors-

ening fatigue, and severe myalgias affecting both proximal and distal extremities, resulting in significant difficulty with ambulation (Figure 1). His symptoms persisted for one month with lab findings, showing a markedly elevated CK (5500 U/L), ALT (122 U/L), and AST (220 U/L) consistent with myositis (Table 1, Table 2).

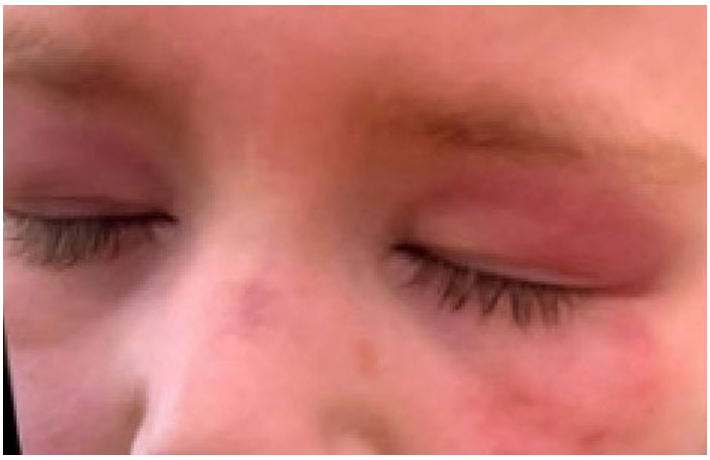
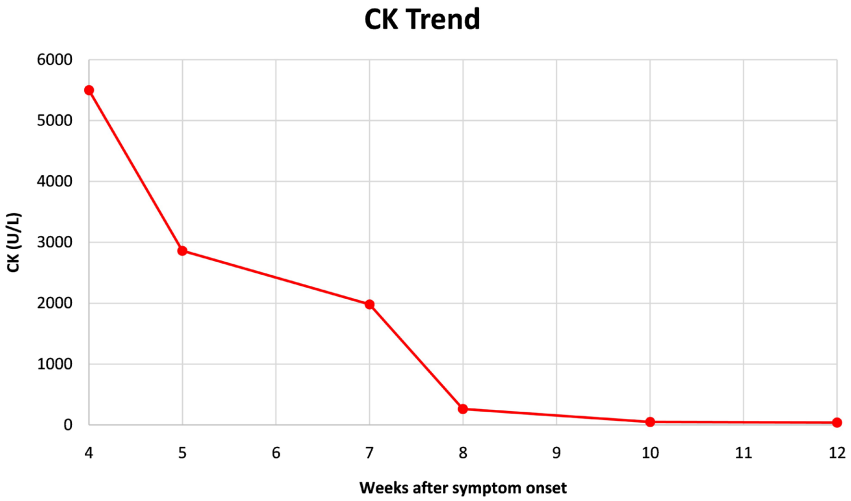


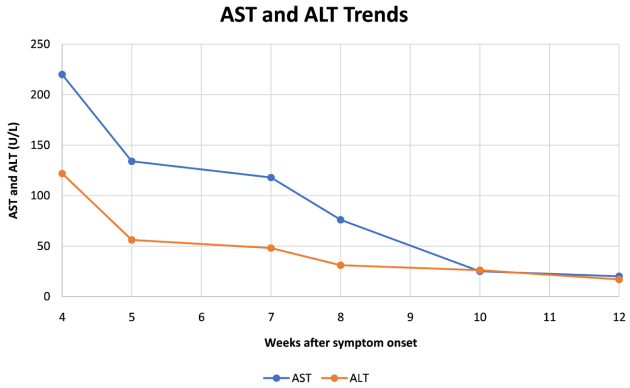
Figure 1. A bilateral macular faint violaceous and erythematous rash with periorbital edema consistent with a heliotrope rash.

Table 1. Creatine phosphokinase trend.



Weeks after symptom onset	CK (U/L)
4	5500
5	2864
7	1983
8	262
10	49
12	38

Table 2. Liver enzymes trend through illness.



Weeks after symptom onset	AST (U/L)	ALT (U/L)
4	220	122
5	134	56
7	118	48
8	76	31
10	25	26
12	20	17

Eight weeks after symptom onset, his creatinine kinase and liver enzymes were improving but remained elevated, while his inflammatory markers with erythrocyte sedimentary rate (ESR) and C-reactive protein (CRP), were normal. His examination revealed intermittent waxing and waning dry papular rashes on the elbows, knees, and dorsal aspect of the feet, while muscle strength and tone were normal in all extremities. However, he was still unable to ambulate independently and continued to report of persistent pain in his thighs and calves. Further investigation with comprehensive lab testing ruled out other metabolic, endocrine, nutritional, infectious, autoimmune, and genetic etiologies for myositis. MRI imaging of the brain and total spine showed no abnormalities. Given concerns for juvenile dermatomyositis, an MRI with myositis protocol was performed of the proximal lower extremities, revealing diffuse myositis in a symmetric pattern (Figure 2).

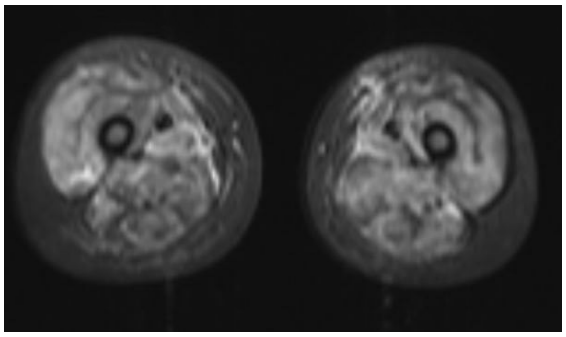


Figure 2. MRI w/myositis protocol with T2 imaging highlighting diffuse edema in a symmetric pattern of the Quadriceps and Hamstring muscles.

A biopsy of the skin rash revealed subtle vacuolar interface dermatitis with increased dermal mucin deposition and perivascular lymphocytes, once again raising concern for juvenile dermatomyositis (JDM). Ultimately, a muscle biopsy was performed for definitive diagnosis, which demonstrated acute and subacute myopathic changes with mild inflammatory infiltrates, occasional necrotic and regenerating fibers, and no evidence of vasculitis findings consistent with post-viral myositis (Figure 3). By week 10, his laboratory values continued to improve despite the absence of any medical intervention. At that time, he did receive IVIG and transitioned to a brief taper of prednisolone and by week 12, he was able to ambulate independently with all of his labs returning to normal (Figure 4).

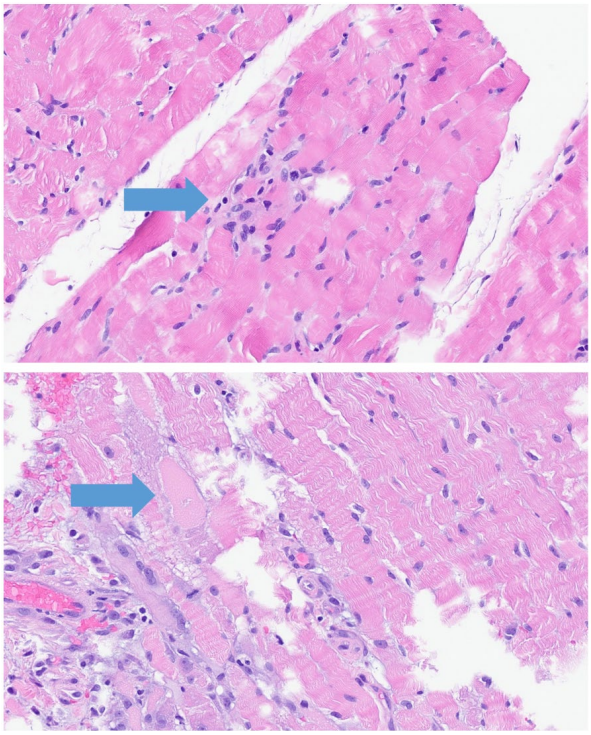


Figure 3. **Top panel:** A high-magnification photomicrograph of a formalin-fixed, paraffin-embedded tissue section stained with hematoxylin and eosin shows mild variation in muscle fiber size. There is a mixed lyphohistiocytic infiltrate (arrow) in a muscle fascicle. **Bottom panel:** A high-magnification photomicrograph of another area of the muscle biopsy shows necrotic (arrow highlights one example) and regenerating muscle fibers with basophilic cytoplasm.

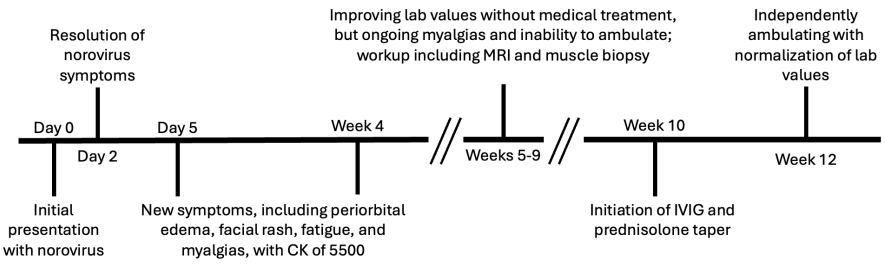


Figure 4. Time line of disease symptoms.

2. Introduction

Viral-induced myositis typically presents with abrupt-onset muscle pain and weakness following a viral illness. Most cases are self-limiting, resolving within days to weeks with conservative management. However, a subset of refractory cases may exhibit atypical features, including rashes, persistent myalgias, profound weakness, and significantly elevated creatinine kinase levels. These cases can mimic juvenile dermatomyositis—an autoimmune inflammatory myositis characterized by proximal muscle weakness and distinctive skin rashes, creating a significant diagnostic challenge.

Laboratory workup for autoimmune myositis includes myositis-specific antibodies/myositis-associated antibodies (MSA/MAA), which are highly specific (95%) but have poor sensitivity (20%) [1] [2]. Consequently, a negative antibody test does not rule out juvenile dermatomyositis (JDM) as a diagnosis. Our review of current literature found no data quantifying how often viral myositis may be misdiagnosed as JDM; therefore, integrating clinical presentation, laboratory findings, imaging, and histopathology is essential for achieving an accurate diagnosis. Our paper further examines the distinctions between benign acute childhood myositis, post-viral myositis, and juvenile dermatomyositis, emphasizing their similarities and differences to facilitate accurate differentiation. Additionally, we document a previously unreported clinical presentation of viral myositis featuring a heliotrope rash.

3. Discussion

3.1. Differential Diagnosis

1) Benign Acute Childhood Myositis (BACM)

The association between viral infections and severe myositis has been recognized for decades. In 1955, Dr. Lundberg observed cases of children in Stockholm, Sweden, who experienced viral prodromal syndromes that subsided within four days, but then soon developed severe myalgias in the calves, rendering them unable to walk [3] [4]. The myalgias typically resolved within three days, leaving the children asymptomatic. Dr. Lundberg later termed this condition as "myalgia cruris epidemica," now known as benign acute childhood myositis (BACM).

BACM is a rare condition that presents with severe, symmetrical myalgias, predominantly in the calves, leading to difficulty in ambulation following a viral illness. Clinically, affected children typically exhibit a wide-based gait with stiff legs or tiptoe walking [3] [5] [6]. The condition is most commonly associated with influenza A (13%) and influenza B (19%) but has also been linked to coxsackievirus (6%), adenovirus (4%), and echovirus (2%) [3] [5]-[7]. Cases tend to surge during the winter months in the Northern Hemisphere and exhibit a male predominance, with a male-to-female ratio of 2 - 3:1 [3] [5] [7] [8].

Laboratory findings in BACM are marked by significant elevations in CK levels, often exceeding 20 times the upper limit of normal [3] [5]. Although an intensive diagnostic workup is generally unnecessary, muscle biopsies in select cases have

demonstrated variable histopathological findings, ranging from normal morphology to segmental rhabdomyolysis or myositis with moderate necrosis and interstitial inflammation [7]-[9]. BACM has an excellent prognosis, with most children fully recovering with conservative management within a few days to weeks [5].

2) Post-Viral Myositis (PVM)

Acute viral or post-viral myositis can occur with nearly any virus, although influenza A and B are the most common causes in the United States. Other implicated viruses include enterovirus, herpesvirus, hepatitis, and parvovirus [10] [11]. Clinically, PVM presents with diffuse myalgias, particularly in the limb girdle and paravertebral muscles with minimal muscle weakness [8]. The exact mechanism remains unclear but is hypothesized to involve either direct viral invasion of muscle tissue or molecular mimicry, leading to immune cross-reactivity between viral and muscle proteins.

Norovirus infection typically manifests as acute gastroenteritis. While it is not commonly linked to myositis or myalgias, rare documented cases have been reported. For example, Yamamoto *et al.* reported BACM secondary to combined rotavirus and norovirus infection in a 2.5-year-old girl [12]. Nishio *et al.* described a case of rhabdomyolysis linked to norovirus in a 2-year-old Japanese boy [13].

3) Juvenile Dermatomyositis (JDM)

JDM is a rare autoimmune disease characterized by symmetrical proximal muscle weakness and distinctive skin rashes, often accompanied by systemic involvement [14]-[16]. The annual incidence of JDM is between 1.6 to 4 cases per million children, with a prevalence of 2.5 cases per 100,000 [14]-[16]. It primarily affects children aged 5 to 14 years, with a female predominance (2.3:1) [14]-[17]. The pathophysiology of JDM involves a combination of polygenetic risk factors and environmental triggers leading to immune dysregulation.

The strongest genetic association in both adult and pediatric myositis is within the ancestral haplotype 8.1 region of the human genome [15] [18] [19]. This haplotype includes HLA-A1, HLA-B8, HLA-DR3, and HLA-DQ2, which are prevalent in Caucasians and encompass a significant portion of the major histocompatibility complex on chromosome 6. This region is associated with several autoimmune conditions, including inflammatory myositis, autoimmune liver disease, Sjogren's syndrome, and myasthenia gravis [19] [20].

Multiple environmental exposures may trigger immune dysregulation in inflammatory myositis, including ultraviolet exposure, medications, and bacterial and viral infections such as Parvovirus, Coxsackie B virus, Epstein-Barr virus (EBV), polyomavirus and even COVID-19 [15] [21]-[24]. Additionally, inhaled exposures to pollutants, carbon monoxide, smoking, gasoline vapor, and chalk/dust during pregnancy have been implicated in the onset of JDM [15] [22]-[26].

3.2. Pathophysiology

Greco *et al.*, in 1976, documented a case of post-viral myositis in a 19-year-old

female who presented with severe myositis and CPK levels exceeding 2400 U/L, three weeks after experiencing viral symptoms [27]. A muscle biopsy revealed patchy necrosis, while electron microscopy identified numerous virus-like particles within degenerative fibers [27]. These spheroidal structures with spiky surface projections were consistent with myxoviruses, and were absent in normal muscle fibers [27] [28] Although viral cultures from the tissue could not be replicated, these findings suggest that direct viral invasion may result in myositis. Culturing viruses from muscle biopsies remains challenging, largely due to the resistance of mature skeletal muscle to infection. This may explain why children with immature skeletal muscle tissues often present with severe forms of myositis [7] [28]-[31]. However, there are isolated reports of successful viral culture. For instance, Kessler *et al.* (1980) successfully isolated the influenza A virus from muscle tissue [28] [30] [31].

The pathophysiology of dermatomyositis is multifaceted, with one of the crucial pathways involving interferon signaling [4] [26] [32] [33]. Interferons are released in response to pathogen-associated molecular patterns (PAMPs) that recognize viral proteins or genetic material. PAMPs bind to toll-like receptors (TLR), initiating downstream signaling cascades that result in increased production of type 1 interferons [4] [26] [32]-[34]. Additionally, TLR-independent pathways, including RIG-1, MDA-5, and STING, promote interferon production via mitochondrial antiviral signaling proteins (MAVS) [4] [26] [32] [33]. In JDM, plasmacytoid dendritic cells (pDCs) in perifascicular and perivascular regions produce large quantities of interferons. These interferons bind to interferon receptors (IFNAR), leading to the upregulation of interferon-stimulated genes (ISGs) [4] [32] [33].

ISGs subsequently release multiple cytokines and chemokines, including CXCL 9, CXCL 10, and CXCL 11 [4] [16] [32]-[34]. These chemokines bind to the CXCR 3 receptor, facilitating the migration of T cells, NK cells, and macrophages [32] [33].

Finally, the adaptive immune system also plays a significant role in dermatomyositis, as demonstrated by the presence of T and B cells in muscle biopsies. T-cells amplify innate immune responses by recruiting neutrophils and macrophages, whereas B-cells contribute to the development of high-affinity myositis-specific antibodies [33].

3.3. Histopathology

The histopathological features of muscle biopsies in viral myositis differ significantly from those observed in inflammatory myositis. Bove *et al.* analyzed muscle biopsies from 12 children and reported patchy necrosis with minimal inflammatory infiltration [35]. Similarly, Agyeman *et al.* identified muscle degeneration and necrosis with minimal inflammation in 28 out of 35 cases [9]. While large-scale studies are lacking, these findings suggest that muscle necrosis, rather than inflammation, is the predominant histopathological feature in viral myositis. This contrasts sharply with dermatomyositis, where tissue biopsies consistently demonstrate

pronounced inflammation with $CD4^+$ and B cells within the perivascular and perimysial regions, leading to perimysial atrophy [15] [16].

Histopathological changes in skin biopsies, such as vacuolated interface dermatitis, are highly associated with dermatomyositis, though they are not exclusive diagnostic findings. Wolstencroft *et al.* examined 228 skin biopsies from dermatomyositis patients and observed a strong association with several histopathological changes, including perivascular inflammation (85%), mucin deposition (81%), basal vacuolization (75%), and dyskeratotic keratinocytes (74%) [36] [37]. Over 90% of the biopsies exhibited at least one of these findings [37]. However, interface dermatitis is not unique to dermatomyositis; as it can also occur in systemic lupus erythematosus, viral exanthems, drug reactions, erythema multiforme, and graft-versus-host disease [38] [39]. Therefore, the pathological findings of interface dermatitis must always be interpreted in the appropriate clinical context to ensure accurate diagnosis.

3.4. Imaging Findings

MRI plays a pivotal role in the diagnostic evaluation of myositis, aiding in distinguishing patterns associated with various etiologies. It is particularly useful for identifying areas most likely to yield diagnostic results on muscle biopsy. In viral myositis, MRI often reveals diffuse muscle involvement characterized by focal or patchy edema, but without muscle atrophy or fatty infiltration [40]. In JDM, MRI findings vary with the disease phase. During the acute phase, symmetric muscle edema is typically observed in the proximal extremities, which may also involve fascial and subcutaneous soft tissues [40]. In the chronic phase, MRI commonly demonstrates muscle atrophy with fatty replacement and calcinosis [40].

3.5. Diagnosis

Virus-induced myositis is a clinical diagnosis typically characterized by the abrupt onset of symptoms following a viral infection. In contrast, the diagnosis of dermatomyositis can be more challenging and is often guided by established classification criteria. The initial criteria for inflammatory myositis were proposed by Peter and Bohan in 1975, encompassing symmetric proximal muscle weakness, laboratory evidence of muscle damage, histopathological evidence of myositis, myopathic changes on EMG, and the presence of a typical rash consistent with dermatomyositis [15] [16]. Since then, advances in the identification of muscle-specific antibodies have significantly refined the diagnostic process. The 2017 EULAR/ACR classification criteria incorporate these antibodies and introduce a scoring system with a sensitivity of 93% and specificity of 88% for diagnoses supported by muscle biopsy. When a biopsy is not feasible, the sensitivity is 87%, and the specificity is 82% [41]. A total score of 5.5 without a biopsy or 6.7 with a biopsy indicates probable myositis, while scores of 7.5 without biopsy or 8.7 with biopsy indicate definitive myositis [41].

Characteristic skin manifestations in dermatomyositis include heliotrope rash,

Gottron's papules, and Gottron's sign [42]-[44]. These cutaneous findings can often precede the onset of myositis by several months [45]. While the 2017 EULAR criteria include major cutaneous manifestations, minor criteria such as the V-sign, shawl sign, and holster sign are also frequently observed [36]. Recognizing both major and minor skin changes is crucial for timely diagnosis. Amyopathic dermatomyositis, which lacks significant muscle involvement, poses diagnostic challenges and can be easily confused with lupus erythematosus. Da Silvia *et al.* reported that 37% of patients with dermatomyositis were initially misdiagnosed with cutaneous or systemic lupus erythematosus [43] [46].

4. Conclusions

This case presented a diagnostic dilemma, as the patient exhibited symptoms consistent with post-viral myositis but failed to respond promptly to conservative medical treatment, raising suspicion of other causes of myositis, particularly juvenile dermatomyositis. His physical examination revealed a heliotrope-like rash, which is often pathognomonic for dermatomyositis; however, his clinical presentation was atypical of the diagnosis, as he exhibited both proximal and distal myalgias but not weakness. Viral myositis can closely mimic dermatomyositis, as both conditions may present with overlapping clinical features and laboratory findings, as noted by Narayanappa *et al.* [11]. In this case, the occurrence of a heliotrope-like rash associated with viral myositis is very unusual and, to our knowledge, has not been previously documented.

Due to this clinical ambiguity, a biopsy of his dry eczematous rashes on the elbows and knees was pursued, revealing subtle features of interface dermatitis, raising suspicion for an atypical presentation of juvenile dermatomyositis. Further evaluation with MRI imaging highlighted symmetric myositis in the proximal lower extremities, yet again prompting suspicion of dermatomyositis. Ultimately, a muscle biopsy was performed to clarify the diagnosis, revealing significant necrosis and regenerating muscle fibers with minimal inflammatory infiltrate, findings consistent with post-viral myositis.

A diagnosis for dermatomyositis can be achieved with a classic presentation of symmetric proximal muscle weakness, elevated levels of creatinine kinase and a characteristic skin rash associated with dermatomyositis. Muscle-specific antibodies and biopsy are not always required to establish a diagnosis; however, they can be particularly useful in differentiating virus-induced myositis from dermatomyositis, as patients with a virus-induced myositis will not have antibodies and will recover with conservative management [5] [41]. While myositis-specific antibodies have clinical utility, they are not particularly useful in acute settings, as results can take weeks to return. Therefore, myositis-specific antibodies should not replace a clinician's astute judgment, which may include histopathological evaluation when necessary. In our case, the delayed results of these antibody tests were negative, serving only to confirm our clinical and pathological diagnosis of post-viral myositis through muscle biopsy.

Alternative diagnoses were considered but ultimately excluded through comprehensive laboratory testing and clinical decision-making. Routine labs with complete metabolic panel and thyroid assessments ruled out common metabolic and endocrine causes of myositis. Infectious etiologies, including tick-borne diseases and viral pathogens such as SARS-CoV-2, influenza, parainfluenza, adenovirus, respiratory syncytial virus, and toxoplasmosis, all tested negative. Finally, extensive genetic testing excluded muscular dystrophies (Duchenne, Becker, facioscapulohumeral dystrophy, limb-girdle dystrophy) and myotonic disorders (McArdle's, Pompe's, and Tauri's disease).

Severe cases of post-viral myositis, including those rarely associated with myositis, such as norovirus, can lead to profound generalized weakness, significant elevation of creatine kinase levels, and a heliotrope-like rash. These features closely mimic dermatomyositis, presenting significant diagnostic challenges. This case highlights the importance of thorough clinical evaluation and diagnostic workup in distinguishing between these conditions. It also underscores the need for clinicians to be vigilant in recognizing atypical presentations of viral myositis to avoid diagnostic errors.

Learning Points

1) Norovirus, albeit rarely, can present with severe myositis.

2) A heliotrope rash is pathognomonic for dermatomyositis, but we present a case where it is associated with viral myositis.

3) Viral myositis can closely mimic inflammatory myositis; therefore, meticulous attention to detail is paramount to avoid diagnostic pitfalls.

Declaration

Informed written consent was obtained from the patient's parent for this case report.

Authors' Contributions

All authors contribute to planning, literature review and conduct of the review article. All authors have reviewed and agreed on the final manuscript.

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Conflicts of Interest

The authors declare no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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