When Inflammation Follows Infection A Case of Post Viral Inflammatory Myositis in a Child

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ABSTRACT

Background: Muscle pain and weakness following a febrile illness in children may indicate a myopathic process. Although benign acute childhood myositis typically follows a brief, self-limiting course, persistent or progressive weakness warrants evaluation for other inflammatory muscle disorders and neurological conditions, including Guillain–Barré syndrome. This report describes an 11-year-old girl with progressive symmetrical limb weakness following a febrile illness, markedly elevated creatine kinase, and electrophysiological findings supporting inflammatory myositis of suspected post-viral origin.

Case Presentation: This case report describes an **11-year-old female patient with post-viral inflammatory myositis**. Post-viral inflammatory myositis is an uncommon but increasingly recognized complication of acute viral infections in children. It can present a diagnostic challenge due to its overlapping features with **Guillain-Barré syndrome (GBS)** and other inflammatory myopathies. A markedly elevated **creatine kinase (CK)** level and electrophysiological findings consistent with myositis, in the setting of a preceding febrile illness, can help distinguish this condition from neuropathic disorders.

Conclusion

This case highlights the diagnostic challenges of progressive muscle weakness following a febrile illness in children. Markedly elevated creatine kinase and a myopathic electromyographic pattern supported muscle involvement over a primary neuropathic disorder. Although the preceding illness suggested a post-viral trigger, the absence of virological confirmation and further myositis-specific investigations limits diagnostic certainty. Continued assessment of muscle strength, enzyme levels, and functional recovery is important to monitor progression and identify persistent inflammatory myopathy.

Key Words: *Post-viral myositis, Paediatric myositis, Creatine kinase, Electromyography, Guillain-Barré syndrome, IVIG, Inflammatory myopathy.*

Background

In the pediatric population, viral illnesses may result in acute myositis; however, this is an infrequent event. The presentation can range from benign acute childhood myositis (BACM), which is self-limited and characterized by bilateral calf pain lasting only for a few days, to more severe or persistent inflammatory myositis requiring treatment with immunosuppressant medications [1,2] The majority of cases of BACM are seen in school-aged children and have been associated with influenza B virus; however, influenza A or other infectious agents may also be responsible for the condition[3]. Clinical findings typically include bilateral lower extremity pain, an altered walking pattern (known as gait disturbance), and an elevated serum CK level (significantly higher than normal). Supportive care alone usually leads to full recovery in the majority of patients within a few days; some patients who are unable to walk normally or have significantly elevated CK levels at the initial visit may represent a chronic autoimmune process requiring treatment with immunosuppressive therapy.[4] The diagnostic challenge in post-viral myositis lies in distinguishing it from Guillain-Barré syndrome (GBS), which also presents with post-infectious ascending weakness in children.

Patient Demographics and History

Yashfa is an 11-year-old female (10 years and 10 months) from Gujranwala, Pakistan, who was admitted to the Paediatric Intensive Care Unit (PICU) at Dr. Akbar Niazi Teaching Hospital (ANTH), Islamabad, on 7 April 2026. Her presenting complaints were limb weakness for one-month, generalized body swelling for two weeks, and intermittent fever for two weeks. According to her mother, Yashfa had been healthy approximately one month before admission when she developed a high-grade fever of up to 102°F, which was intermittent in nature. The fever was not associated with rigors, chills, diarrhea, vomiting, cough, rash, headache, or body aches. She was given Panadol and an unknown injection at a local clinic, following which the fever subsided within one day.

Soon after the fever resolved, she developed progressive weakness and pain in both lower limbs. The weakness gradually increased, was symmetrical, and followed an ascending pattern, subsequently involving the upper limbs and back. There was no difficulty breathing; however, during the last few days before admission, she developed pain on swallowing, along with tingling sensations in both the upper and lower limbs.

Approximately two weeks after the onset of weakness, generalized swelling developed, initially involving the lower limbs and gradually progressing to the upper limbs.

Physical Examination on Admission

On physical examination, the patient appeared ill and had a limping gait. Her vital signs were: heart rate 110/min, respiratory rate 32/min, temperature afebrile, and SpO2 98% on room air. No lymphadenopathy was noted. A BCG scar was present, and the tongue and oropharynx appeared normal.

Respiratory examination: Bilateral equal air entry was present, with no adventitious sounds.

Abdominal examination: The abdomen was soft and non-tender, with no visceromegaly. Bowel sounds were audible.

Cardiovascular examination: S1 and S2 were audible, with no added sounds.

Central nervous system examination: The patient had a **GCS of 15/15** and was oriented to person, place, and time. A limping gait was noted, with no muscle wasting. The calves were mildly tender, with no local edema, erythema, or warmth. Muscle tone was slightly increased bilaterally in all four extremities. Muscle power was **3/5 in both lower limbs and 5/5 in both upper limbs**. Reflexes were normal in all extremities. Plantar responses were equivocal, with an extensor response noted. Sensory examination was intact in all dermatomes, and all accessible cranial nerves were intact. Mildly slurred speech was noted. A darkly pigmented lesion was observed over the right distal lower limb.

The patient passed **dark-yellow, non-frothy urine without haematuria**. Stool was yellow, with normal consistency and frequency. There was no evidence of jaundice.

Haematological Investigations

The differential leukocyte count showed neutrophilia and lymphopenia. Neutrophils were elevated at 85% (reference range: 35–65%), while lymphocytes were reduced to 12% (reference range: 23–53%).The absolute neutrophil count was 9,250/mm³ (reference range: 2,800–8,400/mm³), while the absolute lymphocyte count was 1,290/mm³ (reference range: 2,000–6,000/mm³).

Inflammatory Markers

The sample taken on 8 April 2026 showed a high-sensitivity C-reactive protein (CRP) level of 4.44 µg/mL, which was below the threshold of 6.0 µg/mL, indicating low to no evidence of an acute inflammatory response. This correlated with the ESR of 15 mm/h on 8 April 2026, which was within or slightly above the upper limit of the normal range.

Muscle Enzyme Profile

The patient's creatine phosphokinase (CK) was markedly elevated on 7 April 2026, with a value of 3,462 U/L, compared with the normal range for females of up to 170 U/L. This represented approximately a 20-fold increase above the upper limit of normal. On repeat testing on 15 April 2026, CK had decreased to 1,724 U/L, demonstrating a decline but remaining markedly elevated. On 9 April 2026, CK-MB was 202 U/L (normal range: 0–25 U/L), representing an approximately eight-fold increase above the upper limit of normal. However, troponin-I was 0.10 ng/mL (negative <0.3 ng/mL), which was within the negative range and did not indicate significant myocardial injury.

Liver Functioning and Autoimmune Serology

Liver function tests performed on 7 April 2026 showed a total bilirubin of 0.30 mg/dL (normal), albumin of 3.6 g/dL (low-normal), ALT of 104 U/L (elevated; reference range: 10–40 U/L), ALP

of 156 U/L (within the reference range: 30–645 U/L), and AST of 221 U/L (markedly elevated; reference range: 9–40 U/L). Total protein was 6.1 g/dL, within the reference range of 6–8 g/dL.ANA/ANF testing performed on 9 April 2026 was negative (ANA 0.1; negative <0.9, borderline 0.9–1.1, positive >1.1). TSH was 1.80 µIU/mL, within the normal reference range of 0.35–4.94 µIU/mL, indicating normal thyroid function and making thyroid dysfunction an unlikely cause of the myopathy. The 25-hydroxy vitamin D level was 36.04 ng/mL, indicating sufficient vitamin D levels (>30 ng/mL).

Urine Analysis

Serial urine analysis performed on 7 April 2026 revealed pale-yellow urine, with a pH between 6.0 and 7.5 and a specific gravity between 1.005 and 1.015. No protein, blood, glucose, nitrites, or leukocyte esterase were detected. There were only 1–3 pus cells, with no red blood cells or epithelial cells observed per high-power field. There was no evidence of myoglobinuria, making significant rhabdomyolysis less likely. Rhabdomyolysis is a serious complication of severe muscle injury characterized by the release of myoglobin into the bloodstream, which can result in renal tubular injury.

Electrophysiology: NCS and EMG

Nerve conduction studies (NCS) showed decreased compound muscle action potential (CMAP) amplitudes in the bilateral common peroneal nerves, while distal motor latencies and motor nerve conduction velocities were preserved. The bilateral tibial nerves, left median nerve, and left ulnar nerve showed normal CMAPs, distal motor latencies, and motor nerve conduction velocities. F-wave responses were normal in the bilateral tibial nerves, left median nerve, and left ulnar nerve. Similarly, the bilateral sural nerves and the left median and ulnar nerves demonstrated normal sensory nerve action potentials (SNAPs), sensory peak latencies, and sensory nerve conduction velocities (SNCVs). EMG revealed fibrillations (Fibs) and positive sharp waves (PSWs), along with small motor unit action potentials (MUAPs) and an early recruitment pattern in the bilateral tibialis anterior muscles. The left brachioradialis showed no Fibs or PSWs but demonstrated small MUAPs and an early recruitment pattern. The left quadriceps showed a normal electromyographic pattern.

Overall, the electrophysiological findings demonstrated abnormal parameters consistent with a myopathic process, supporting the diagnosis of myositis.

Differential Diagnosis

Guillain-Barre syndrome (GBS): The development of ascending, bilateral weakness in the days following a febrile illness led to the initial suspicion of GBS. However, normal sensory conduction on NCS and preserved F-waves, along with a myopathic EMG pattern, ruled this out. Furthermore, the CK level (20 times normal) was very high and therefore indicated a muscle disorder rather than GBS since CK elevations are usually mild in GBS.[6]

Juvenile Dermatomyositis (JDM):

JDM, the most common paediatric inflammatory myopathy, typically presents with proximal muscle weakness, skin rash, and elevated muscle enzymes[7]. Although this patient had weakness and raised CK, there were no cutaneous features and ANA was negative. JDM without rash remains possible, but the temporal association with the preceding febrile illness supported post-viral myositis[8].

Immune-Mediated Necrotising Myopathy (IMNM):

IMNM is a rare form of inflammatory myopathy characterized by severe weakness in proximal muscles and very high CK levels.[9] Viral infections may trigger IMNM. In this case, IMNM was considered a possibility due to the progressive weakness and high CK level; however, the associated antibody tests to confirm IMNM were not done.

Benign Acute Childhood Myositis (BACM):

BACM is a self-limiting condition that is typically associated with an influenza B infection and is characterized by acute bilateral calf pain, inability to walk, and moderate elevations of CK (mean 2483 U/L reported). It usually resolves between 1-5 days after the onset of symptoms. While the CK level of this patient (3462 U/L) fits the range for BACM, the ongoing and progressive nature of the symptoms, in addition to the involvement of both upper extremities (arms), slurred speech, and need for admission into the Pediatric Intensive Care Unit (PICU), is not typical of any case of BACM and indicates a more significant inflammatory process taking place.

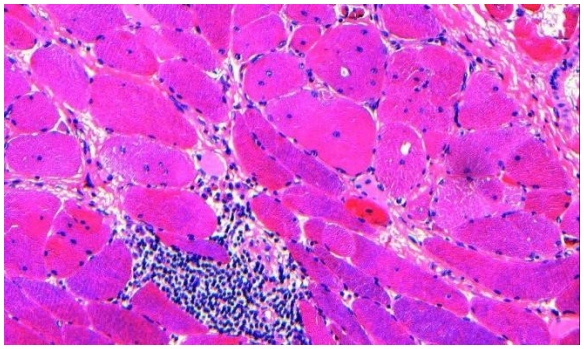


Figure 1. *Histological section showing inflammatory cell infiltration between muscle fibers.*

Discussion

This case illustrates several important teaching points regarding post-viral myositis in children:

The Post-Viral Immune Cascade: The inflammatory muscle disease known as myositis can be triggered by a viral infection through several mechanisms; either direct invasion of muscle cells by the virus, molecular mimicry between viral protein or components that resemble those of muscle proteins, or nonspecific activation of the immune system of muscle cells due to infections elsewhere in the body. In this case, there was a strong temporal association between the development of weakness and febrile illness, suggesting that the mechanism is immunemedicated and post-infectious and not caused by viral invasion of the muscle cells. Therefore, the febrile illness was probably caused by either influenza virus or another type of viral infection; however, the viral serology, PCR or any other testing cannot be determined for this patient.

The Significance of Markedly Elevated CK: Skeletal muscle damage is evaluated using a serum creatine kinase (CK) assay. CK has been extensively used for diagnosing and tracking progression of myositis. In this case, the patient's initial admission CK level was approximately 20 times above the upper limit of the normal range, indicating that she was suffering from severe myositis. The CK level decreased from 3462 U/L to 1724 U/L over an 8-day treatment period in response to successful treatment (plateau). It has been well documented that CK normalization corresponds to a clinically meaningful decrease in the severity of inflammatory myopathy.[9] Thus, CK should be periodically monitored after initiation of therapy in all patients with myositis to determine their response to treatment.

CK-MB Elevation Without Cardiac Involvement: The high CK-MB level in this patient (202 units/liter) was a concerning finding for this patient; but the fact that his troponin-I level was normal (0.1 ng/ml) and his echocardiogram was completely normal helped rule out the diagnosis of myocarditis. CK-MB is released from regenerating skeletal muscle cells, and will frequently be elevated in inflammatory myositis cases without associated cardiac involvement, as CK-MB is also produced in that tissue. The ratio of CK-MB to total CK was approximately 5.8%, which falls into the range of what you would expect to see if the source of CKMB was skeletal muscle as opposed to being from cardiac tissue; in the case of cardiac tissue, the ratio of CK-MB to total CK would be greater than 6%, raising the concern for cardiac injury.

EMG/NCS as the Gold Standard for Differentiation: The outcome of the electromyography and the nerve conduction studies established a definitive diagnosis. The abnormal myopathic pattern of fibrillations, positive sharp waves, as well as the small size of the motor unit action potential with early recruitment along with normal sensory conduction velocities and F waves were conclusive that Guillain-Barre syndrome could be ruled out and inflammatory myopathy confirmed. Studies show that EMG is less sensitive in children (10% to 86%) depending on technique used and physician experience, as a result of less insertions being performed and their ability to cooperate with the study.[10] In this case the findings are without question. The selective involvement of the bilateral anterior tibialis with relative sparing of the quadriceps indicates a patchy pattern of muscle inflammation which is characteristic of inflammatory myositis versus a generalized involvement as occurs with myopathy**.**

Treatment and Management

1. First-line corticosteroids: For inflammatory myositis, high-dose oral glucocorticoids are usually the initial treatment. Depending on the severity of the illness and the extent of organ involvement, steroid-sparing medications are often added early.[11]

2. Steroid-sparing immunosuppressive agents: Methotrexate, azathioprine, and mycophenolate mofetil are examples of steroid-sparing immunosuppressants used when steroids alone are insufficient, cause unacceptable adverse effects, or when the disease relapses during steroid tapering.[12]

3. Intravenous immunoglobulin (IVIG): Following a double-blind, placebo-controlled trial, IVIG was approved by the FDA for the treatment of adult dermatomyositis. It may be used as an adjunctive treatment or as an

alternative when conventional immunosuppressive therapy is ineffective, poorly tolerated, or contraindicated. [13]

4. Physiotherapy and Exercise: A 2021 controlled trial demonstrated that a 24-week training program involving strength, endurance, and activities of daily living significantly improved muscle function without increasing inflammation levels. A 2025 multicentre randomized trial also showed that high-intensity interval training was more effective than moderate exercise in improving aerobic capacity in patients with recent-onset myositis. [14,15]

Conclusion

This case highlights several important learning points: **(1)** markedly elevated creatine kinase (CK) is a key feature of inflammatory myositis and should prompt further evaluation with electromyography (EMG) and nerve conduction studies (NCS); **(2)** elevated CK-MB in the absence of troponin elevation does not necessarily indicate cardiac disease in the setting of myositis; **(3)** elevated transaminases, particularly AST, may reflect skeletal muscle injury rather than primary liver disease; **(4)** myopathic motor unit action potentials (MUAPs) with fibrillatory potentials and preserved sensory nerve conduction on EMG/NCS support a myopathic process and argue against Guillain–Barré syndrome (GBS); and **(5)** management of significant inflammatory myositis may include corticosteroids with intravenous immunoglobulin (IVIG), depending on disease severity and clinical response.

Declarations

Ethics Approval: Ethical approval for this case report was obtained from the Institutional Review and Ethics Board (IREB), Islamabad Medical and Dental College (IMDC), under approval number **303/IMDC/IREB-2026.**

Consent, and Confidentiality: Informed consent was obtained from the patient for publication of the case report and any accompanying clinical information or images, as applicable. Patient confidentiality and privacy were maintained throughout the preparation of this report.

Conflict of Interest: The authors declare no conflict of interest.

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