

Case Report

Benign acute childhood myositis: a self-limiting condition with observed bone marrow suppression and vitamin D deficiency

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Received: 09 July 2026

Revised: 11 August 2026

Accepted: 29 September 2026

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ABSTRACT

Benign acute childhood myositis (BACM) is a rare, self-limiting condition seen primarily in school aged boys following viral infections, especially influenza. It presents with sudden onset bilateral calf pain, gait disturbances, and elevated creatine kinase (CK) levels. We report an 8 year old male with BACM following a febrile illness, severe calf pain, difficulty walking, leucopenia, thrombocytopenia, and hypocalcemia. Supportive care with non-steroidal anti-inflammatory drugs (NSAIDs), Intravenous fluids, and antivirals, led to full recovery in 10 days. This case highlights the importance of recognizing BACM to prevent unnecessary interventions and explores its association with transient bone marrow suppression and vitamin D deficiency.

Keywords: Benign acute childhood myositis, Transient bone marrow suppression, Vitamin D

INTRODUCTION

Benign acute childhood myositis (BACM) is considered rare, with estimates placing it at around 2.6 cases per 100,000 children under 18 years old during epidemic periods and significantly lower (0.23 cases per 100,000) during non-epidemic times; primarily affecting school-aged boys.¹ The incidence of BACM in India is not well-documented, and large scale population based studies specifically addressing BACM are scarce. It usually occurs at the early convalescent phase of the viral illness, particularly influenza B infection.² Characterized by a sudden reluctance to walk due to severe bilateral calf pain and increased creatine kinase (CK) level. Histopathologic examination of muscle reveals degeneration and necrosis, with overall little inflammatory infiltrates. The diagnosis is made by a febrile prodrome, otherwise normal systemic examination, positive CK level and viral studies.^{3,4} Notably, about 19% of affected patients completely avoid bearing weight on their legs, and many exhibit a "Frankenstein gait"-a wide-based, stiff-legged

walking pattern despite having a normal neurological exam, a critical clue in differentiating BACM from other neuromuscular disorders and presenting with acute onset of gait abnormalities.⁵ The low awareness about BACM often lead to delayed diagnosis and unneeded ancillary investigations. Influenza B is the virus most commonly associated with BACM, followed by Influenza A; a novel association with Sars-CoV-2 has been also detected.⁶

CASE REPORT

An eight year old boy presented to the Emergency Department with 4 days of fever, 2 days of cough and severe bilateral calf pain with inability to walk for 1 day. The calf pain was so intense that the child could only walk on his heels, imitating a spastic walking pattern. There was no relevant family history of neuromuscular conditions. No reports of recent travel, injuries, strenuous physical activity or previous occurrences of limb pain before the symptoms began. The child had received all required vaccinations and their medical history did not indicate any significant illnesses.

On examination child appeared well nourished but had fever and was in distress due severe bilateral calf muscle tenderness. Respiratory system examination revealed bilateral crepitations with no signs of respiratory distress. Other systemic examinations, including the CNS appears within normal limits.

Laboratory parameters shows increased creatine phosphokinase (CK) levels, leucopenia and thrombocytopenia with decreased calcium (7.62 mg/dL) and vitamin D3 levels (3 ng/ml). The serum ferritin was 2000 ng/ml (Table 1).

During the first few days of hospitalization, there was a notable increase in the CK-MB and CK-Total levels, which are indicative of muscle injury. The Total leucocyte counts and platelet counts showed a gradual decrease, which raised concern for an ongoing inflammatory or infectious process causing bone marrow suppression. An X-ray chest revealed bilateral infiltrates, which could indicate a viral respiratory infection.

Respiratory viral panel samples were found to be positive for influenza B virus as tested by RT-PCR. BACM was kept as the main working diagnosis.

Table 1: Laboratory findings, from disease onset to follow-up.

Days	CK-total (U/L)	CK-MB (U/L)	AST (IU/L)	ALT (IU/L)	Hb (g/dL)	TLC (10 ³ /μL)	Platelet count (10 ³ /μL)	Calcium mg/dL	Vitamin D3 ng/ml	Serum ferritin ng/ml
Day 1	6299.6	81.37	126	33	11.3	1.98	119	7.7	3	
Day 2	7894.8	99.08			11.9	2.15	110			2000
Day 4	5671	82.37	448.7	247.1	11.2	1.88	90	7.6		
Day 5	1257.9	39.50	289	224	10.8	1.52	107			
Day 7					10.5	2.59	150			
Day 10	151.5	28.13	37	122	11.5	5.03	324	8.8		
Day 17 (Follow up in OPD)	81	19.17	27.5	49	11.2	7.73	346	9.76		

*AST: Aspartate Aminotransferase, ALT: Alanine Aminotransferase, CK: Creatine phosphokinase, TLC: Total leucocyte count.

The child was managed with paracetamol (NSAID) for fever and muscle pain, intravenous fluids (IV) to maintain hydration. Syrup Oseltamivir was initiated due to the suspicion of a viral etiology, particularly given the bilateral infiltrates on the chest X-ray and the cough.

Gradually the calf muscle tenderness subsided and the child was able to walk without significant pain. His fever resolves, and his inflammatory markers began to normalize. The child was discharged after a 10-days of hospital stay. At the time of discharge, the child was ambulatory with minimal discomfort and follow-up instructions were given for outpatient visits, including repeat laboratory tests and clinical evaluation to ensure full recovery.



Figure 1: Chest X-ray picture.

DISCUSSION

BACM is a self-limiting condition that primarily affects school-aged children, often following a viral illness, particularly influenza B.³ First described by Lundberg in 1957 as myalgia cruris epidemica, BACM is characterized by acute onset bilateral calf pain, difficulty walking, and elevated serum CK levels.^{7,8} While the condition resolves spontaneously, its presentation often leads to unnecessary investigations and concern for more serious neuromuscular disorders.

In our case, the patient presented with classic BACM symptoms, including a viral prodrome followed by acute onset bilateral calf pain and gait disturbances. His clinical course was notable for transient bone marrow suppression, including leucopenia and thrombocytopenia, which, although uncommon, has been reported in some cases of BACM.⁴ These hematologic findings are likely a result of the viral infection rather than BACM itself. The presence of respiratory symptoms and bilateral infiltrates on chest X-ray further supported a viral etiology.⁵

The exact pathophysiology of BACM remains uncertain, with hypotheses including direct viral invasion of muscle tissue, immune-mediated myotoxicity, and cytokine-induced muscle damage. While biopsy studies have shown muscle degeneration and necrosis with minimal inflammatory infiltrates, the absence of persistent weakness and spontaneous recovery supports the notion of a transient, non-inflammatory myositis.⁹

A distinguishing factor in this case was the concurrent presence of vitamin D deficiency and hypocalcemia. Chronic vitamin D deficiency is associated with muscle weakness and myopathy, which could have contributed to the severity and duration of symptoms. The delayed normalization of serum calcium and vitamin D levels may have played a role in the prolonged recovery phase observed in this patient.

Vitamin D exerts its biological effects through the vitamin D receptor (VDR), which is expressed in hematopoietic progenitor cells as early as embryonic development. Developmental vitamin D deficiency or absence of VDR reduces hematopoietic progenitor cell frequency and alters immune cell development, with long-term consequences on host immunity, supporting a mechanistic link between vitamin D status and hematopoietic regulation.¹⁰

Further, vitamin D regulates hematopoietic differentiation by promoting monocyte-macrophage lineage commitment and inhibiting granulocyte colony formation. It also activates intracellular signalling pathways such as the phosphatidylinositol-3-kinase (PI3K-AKT) and mitogen activated kinase (MAPK), which influence marrow cell fate decisions. Vitamin D influences haematopoiesis and immune modulation through these molecular pathways, further supporting its relevance in conditions involving transient marrow suppression.¹¹

These findings provide biological plausibility for transient bone marrow suppression observed in our case. While speculative, vitamin D deficiency may have contributed to observed hematologic abnormalities, though further studies are needed to confirm any causal link.

A key lesson from this case is the importance of recognizing BACM to prevent unnecessary interventions such as extensive neuromuscular workups or invasive procedures like muscle biopsy or bone marrow aspiration. Awareness of BACM among clinicians, particularly its association with transient hematologic abnormalities, can aid in timely diagnosis and reassurance for both patients and caregivers. Management remains conservative, focusing on symptomatic relief with analgesics, hydration, and close monitoring until spontaneous resolution occurs, typically within a week.

This case also underscores the potential role of vitamin D status in pediatric myopathies. While the direct impact of vitamin D deficiency on BACM remains unclear, screening for and addressing nutritional deficiencies in pediatric patients presenting with myositis may be beneficial.¹²

CONCLUSION

This case underscores the importance of recognizing BACM as a self-limiting condition to prevent unnecessary interventions. Our findings generate the hypothesis that transient hematologic abnormalities and metabolic deficiencies, particularly vitamin D deficiency may influence its clinical course. This case highlights the need for further investigation into the potential role of nutritional and metabolic factors, such as vitamin D, in pediatric viral myopathies. Future research should explore the interplay between vitamin D metabolism, bone marrow suppression, and viral-induced myositis to clarify pathogenesis and inform supportive management strategies.

Funding: No funding sources

Conflict of interest: None declared

Ethical approval: Not required

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Cite this article as: Ansari MA, Kaiser A, Yadav S. Benign acute childhood myositis: a self-limiting condition with observed bone marrow suppression and vitamin D deficiency. *Int J Contemp Pediatr* 2026;13:2355-8.