

# A crawling case of benign acute childhood myositis

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## Abstract

Benign acute childhood myositis is a relatively uncommon complication of viral illness, particularly influenza A and B infections. In this abstract, we present a case report of benign acute childhood myositis secondary to influenza. On presentation, he was ill-appearing, febrile and had significant calf pain that limited mobility. Examination revealed mildly erythematous throat and bilateral calf tenderness with pain on dorsiflexion bilaterally. A respiratory virus panel was positive for influenza A subtype H3 and he was found to have an elevated creatine phosphokinase. He was diagnosed with benign acute childhood myositis secondary to influenza type A infection and symptoms gradually resolved following supportive management. Management involves oral hydration, pain management, and follow-up to ensure resolution. Extensive laboratory evaluation and hospitalization are often unnecessary. Benign acute childhood myositis is self-limiting with an excellent prognosis and should be included in the differential for a child who develops difficulty walking, particularly when presentation follows a respiratory infection.

## Keywords

Influenza, benign acute childhood myositis, BACM

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## Introduction

Benign acute childhood myositis (BACM) was first described by Ake Lundberg in 1957 and was initially called myalgia cruris epidemica.<sup>1</sup> BACM is now recognized as a sequela of viral illnesses, most commonly influenza A and B,<sup>2</sup> characterized by lower extremity myalgia, weakness, leg pain, and refusal to walk after a viral prodrome.<sup>3</sup> The overall incidence of BACM is relatively low with 2.6 cases per 100,000 children occurring during epidemics,<sup>4</sup> however, actual prevalence is unknown. BACM may be overlooked when a child presents with acute onset of weakness in pursuit of other diagnoses such as Guillain-Barre syndrome (GBS).<sup>5</sup> Given the self-limited natural history of BACM and its relatively simple diagnostic workup, it is important to include in the differential diagnosis of acute weakness and calf pain. We present a case report of BACM and include a limited differential diagnosis for the pediatric patient.

## Case report

A 9-year-old male presented with a 3-day history of fever (up to 40.3°C), dry cough, anorexia, and severe right calf pain. Fevers were accompanied by delirium and chills, which

were resolved with scheduled use of antipyretics. Right calf pain radiated to his ipsilateral ankle and thigh, without joint pain, swelling, or erythema. Leg pain was so severe that he was crawling at home and brought into the clinic in an infant stroller. Further review of systems was unremarkable. His past medical history included Kawasaki disease, angioedema, innocent heart murmur, allergic rhinitis, and chronic urticaria. The patient had an allergy to amoxicillin with accompanying erythema multiforme. He was up to date on vaccinations, except had not received his annual influenza vaccination.

On presentation, he was alert but ill-appearing and febrile. He had difficulty bearing weight and required help to get out of the infant stroller and onto the exam table. Examination revealed mild throat erythema and bilateral calf tenderness to palpation with increased pain on

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**Table 1.** Differential diagnosis of acute weakness in a pediatric patient.

Diagnosis	Symptoms/signs	Supporting laboratory results	Timing	Type of muscle weakness
BACM <sup>5</sup>	Fever, constitutional symptoms, rhinorrhea, cough, nausea and vomiting, myalgia, weakness	Elevated CPK, LDH, transaminases Normal ESR and CRP	Acute onset 1–3 days following fever or influenza like illness	Typically in the calf muscles
Guillain-Barre Syndrome <sup>12</sup>	Fatigue, paresthesia and pain, ataxia; can progress to respiratory muscle weakness	Elevated CSF protein with normal CSF WBC	Onset of muscle weakness 2–4 weeks after prodrome of gastrointestinal illness	Ascending, progressive, symmetrical muscle weakness that typically begins in the lower limbs
Hypokalemia <sup>13</sup>	Cardiac arrhythmias, polyuria, gastrointestinal symptoms, weakness	Serum potassium < 3.5 mEq/L	Onset of muscle weakness when serum potassium < 2.5 mEq/L	Begins in proximal muscles of limbs and ascends toward the trunk
Rhabdomyolysis <sup>14</sup>	Myalgia, weakness, and tea-colored urine. Infectious symptoms may be present depending on cause.	Elevated CPK (1500–100,000 IU/L) Myoglobinuria	Acute onset within hours following muscle injury, seizure, or strenuous exercise. Can occur after viral illness, medication exposure. See full review for additional details <sup>14</sup>	Variable depending on the muscle group involved
Poliomyelitis <sup>15</sup>	Viral prodrome with fever, fatigue, headache, vomiting, stiff neck, pain in limbs, weakness	CSF studies with elevated lymphocytes and protein Virus isolation in stool or cerebrospinal fluid sample	Acute onset weakness, progressing over the course of two to ten days	Flaccid weakness May have bulbar involvement

BACM, benign acute childhood myositis; CPK: creatine phosphokinase; LDH: lactate dehydrogenase; ESR: erythrocyte sedimentation rate; CRP: c-reactive protein; CSF: cerebrospinal fluid; WBC: white blood cells; IUL: international units/L.

dorsiflexion. He had full range of motion of all joints and no joint swelling, warmth, or erythema. The remaining portions of his cardiac, respiratory, and abdominal exams were unremarkable. Given the season of his presentation and clinical course, initial evaluation included a respiratory virus panel that was positive for influenza A subtype H3. Further evaluation revealed elevated CPK of 2,102 m/L (normal 49–379), with leukopenia of 2.60 K/mL (normal 5.4–9.9 K/mL), thrombocytopenia of 166 K/mL (normal 219–350 K/mL), and normal complete metabolic panel (CMP), c-reactive protein (CRP), erythrocyte sedimentation rate (ESR), and urinalysis.

He was diagnosed with benign acute childhood myositis (BACM) caused by influenza type A infection. The patient returned home to continue supportive care, including antipyretics and oral hydration. Antiviral therapy was not indicated due to presentation 5 days after symptom onset. Parents reported he returned to baseline functioning within the week. Repeat laboratory evaluation was recommended to be repeated within several weeks of presentation. Four months after initial presentation, CPK and platelet levels normalized, while mild leukopenia of 4.10 K/mL (normal 5.4–9.9 K/mL) persisted.

## Discussion

Benign acute childhood myositis (BACM) is an uncommon complication of viral illness. In the United States, it is most commonly associated with influenza A and B infections; however, BACM can also occur after other viral infections

such as coxsackie, parainfluenza, adenovirus, enterovirus, human T-cell leukemia-lymphoma virus or hepatitis B and C and even the SARS-coronavirus.<sup>3,5–8</sup> In a retrospective study of influenza infection in children, BACM was reported more frequently in cases of influenza B than influenza A, at 33.9% vs 5.5% respectively.<sup>9</sup> The overall incidence of myositis remains low at 0.23 cases per 100,000 children under 18 years old at baseline and up to 2.6 cases per 100,000 during an epidemic.<sup>4</sup> School aged males are more commonly affected.<sup>1,5,10</sup> Although the exact pathogenesis remains unknown, several theories have been proposed to explain this complication. Some suggest that BACM is due to direct invasion of muscle cells by the viral agent, release of myotoxic cytokines in response to the viral infection, or a virus-induced immunologic process leading to muscle damage.<sup>5,7,11</sup>

BACM presents with muscle pain and tenderness 1 to 3 days after symptoms of viral illness. Pain tends to be localized in the muscles of the lower extremities such as the calf or quadriceps and children may present with difficulty or refusal to walk secondary to pain or weakness. Children may present with a classic gait of either “toe-walking or a wide-based stiff-legged gait.”<sup>5,8</sup> The neurologic and strength exams should remain normal. Although most cases of BACM can be diagnosed clinically, laboratory investigations may reveal elevations in CPK, aspartate aminotransferase (AST), and lactate dehydrogenase (LDH) while the ESR and CRP remain normal.<sup>5,8</sup> Leukopenia is also reported.<sup>8</sup> As BACM is self-limiting, management involves

a triad of oral hydration, pain management and close follow-up. CPK levels typically return to normal within 2 weeks, preceded by the resolution of clinical symptoms, usually within three days of onset.<sup>11</sup> Antivirals are unlikely to be beneficial due to the presentation late in the course of illness.<sup>8,11</sup> BACM should resolve with little to no intervention and hospitalization is rarely required.<sup>5</sup> Rarely, cases can evolve into rhabdomyolysis and patients will report tea-colored urine.<sup>5,11</sup>

Due to the relatively low incidence of BACM, it may be overlooked in the differential diagnosis of acute weakness presenting in a child.<sup>12</sup> Clinicians may focus on more menacing causes of muscle weakness or pain such as Guillain-Barre syndrome, hypokalemia, or rhabdomyolysis. A comparison of the clinical manifestations and laboratory findings of these diagnoses are listed in Table 1. A detailed history and thorough physical exam should be performed to distinguish the accompanying features of BACM from other neuromuscular etiologies. Therefore, it is important for clinicians to consider this diagnosis when a child presents with acute weakness following a viral illness in the absence of other alarming symptoms.

## Conclusion

The onset of influenza-associated BACM is rare and presentation can be alarming, leading to potentially unnecessary invasive testing and hospitalization. Recognition of BACM can help to reduce unnecessary interventions.

## Author contributions

AH, MR, AS, NH contributed to the conception, analysis, and interpretation. AH and MR drafted the manuscript. AH, MR, AS, NH contributed to and critically revised the manuscript. All authors gave final approval.

## Declaration of conflicting interests

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## Informed consent

Written informed consent was obtained from minor patient's parent for their anonymized information to be published in this article. The parent was the legally authorized guardian of the minor subject.

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