Polyarticular Gout Flare Masquerading as Sepsis

To the Editor:

In patients with poorly controlled gout, polyarticular gout attacks occur more frequently than in those with less advanced disease. Such flares can induce a systemic inflammatory response syndrome that may be misdiagnosed as sepsis. Distinguishing these two entities, which often present with similar findings, is challenging. We present a case of an acute polyarticular gout attack masquerading as sepsis and the clinical pitfalls associated with this diagnosis.

CASE SUMMARY

A 64-year-old African-American man with a history of chronic tophaceous gouty arthropathy presented with four days of severe pain in his left foot and ankle associated with fevers, fatigue, and weakness. He denied a history of trauma and had recently completed two courses of corticosteroids for acute gout flare. He had recently completed two courses of corticosteroids for acute gout flare and had recently completed two courses of corticosteroids for acute gout flare.

On physical examination, he was uncomfortable and diaphoretic. His vital signs showed a temperature of 38°C, pulse rate of 126 beats/min, and blood pressure level of 141/86 mm Hg. Purulent-appearing material drained from open wounds on the first and fourth toes of the left foot. The whitish exudate and soft tissue swelling obscured a probe-to-bone test. Bilateral olecranon-bursa nodules were observed. Laboratory findings revealed a white blood cell count of 18,700/μL (16.5 K/mcL bands), C-reactive protein level of 42.5 mg/L, and erythrocyte sedimentation rate of 120 mm/h. Plain film radiography of the left foot was concerning for osteomyelitis of the first toe.

Initially, the patient was managed for presumed sepsis secondary to osteomyelitis. Despite broad-spectrum antibiotics and intravenous fluid resuscitation, the patient remained febrile and tachycardic with a persistent leukocytosis. General surgery was consulted for potential operative intervention given the presumed diagnosis of osteomyelitis of the left great toe. Further evaluation with magnetic resonance imaging of his left foot showed nonspecific inflammatory changes without clear evidence of osteomyelitis. In addition, gram stain and culture of the exudate from his open wounds were negative for microorganisms. Examination of the purulent-appearing material under polarized light microscopy revealed copious negatively birefringent urate crystals. Oral prednisone was initiated, and antibiotic therapy was discontinued. The patient’s fever, tachycardia, and pain improved rapidly. Blood and wound cultures remained sterile.

DISCUSSION

This case illustrates an acute polyarticular gout attack with systemic inflammatory response syndrome masquerading as sepsis. Distinguishing these two entities, which often present with similar findings, is challenging for several reasons. First, our patient’s previous flares were not associated with abnormal vital signs, drainage of purulent-appearing material, or radiography suggestive of osteomyelitis. Polyarticular gout flares, though, may present as pseudo-sepsis, characterized as a systemic inflammatory response due to a non-infectious etiology. In mouse models, it has been demonstrated that monosodium urate crystals, serving as danger signals, can activate the intracellular cryopyrin-inflammasome complex. This triggers release of cytokines, which may lead to a sterile systemic inflammatory response. A similar mechanism has been postulated for pseudogout-induced systemic inflammatory response syndrome.

In addition, on physical examination, liquefied monosodium urate crystals may be mistaken for purulent exudate as seen in this case. Finally, chronic tophaceous gout leads to nonspecific inflammatory changes in bones and joint spaces that may appear similar to osteomyelitis on plain film radiography and mimic a source of infection.

CONCLUSIONS

In patients with chronic tophaceous gout, it is important to recognize that polyarticular gout attacks can induce a systemic inflammatory response syndrome without an associated infection. As exam and imaging findings may be unreliable, microscopic examination of exudates or synovial fluid with both polarized light microscopy and gram stain is necessary to distinguish this entity from sepsis due to osteomyelitis or other infectious sources. Early recognition may prompt a trial of corticosteroids, thereby avoiding unnecessary antibiotic therapy or surgical intervention.

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References


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