



Bifocal pyomyositis in a 3-year-old child with eczema: a case report

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Pyomyositis is an infection of the skeletal muscles, resulting in a pus-filled abscess. Immunodeficiency of the patient is considered to play an important role in pathogenesis. We report a case of a 3-year-old child, who presented with multifocal pyomyositis in the gastrocnemius muscle, extending to the posterior muscles of the thigh. Even though there was no evidence of immunodeficiency, the presence of atopic eczema in the big toe of the affected limb could interfere with the immune system response, and therefore, could be associated with pyomyositis. The increasing incidence of pyomyositis in non-tropical areas and its severe complications requires an acute clinical awareness.

Key words: Pyomyositis; child; eczema; immune system; immunodeficiency; multifocal; non-tropical.

Pyomyositis is a primary acute pyogenic infection of skeletal muscles, usually accompanied by an abscess formation. This form of infection was first described in 1885 by Scriba, as an endemic disease in the tropics.^[1] Pyomyositis is far less common in temperate climates, where it is responsible for 1 per 3000 pediatric admissions.^[2,3] It is believed that pyomyositis is associated with conditions that impair the immunodeficiency level in these regions.^[4] Pyomyositis may cause diagnostic problems for physicians working in temperate climates, due to its rarity and the obscure, non-specific, or misleading signs and symptoms. We present a case of bifocal pyomyositis located in posterior thigh and calf muscles in a 3-year-old child.

Case report

A three-year-old male was brought by his parents to the emergency department, with a ten day history of fever (approximately 37.5 degrees Celsius), and associated fatigue. On the fifth day of the fever, the

mother had consulted the child's pediatrician, who suspected a viral infection and prescribed paracetamol for the symptomatic treatment. Despite the treatment, the symptoms persisted. On the last two days his fever increased to 39.5 degrees Celsius and he was unable to walk. There were no other reported symptoms. His medical and surgical history was clear. There were no similar symptoms in the past. There was no family history of diabetes, malignancy or other factor for potential immunodeficiency.

Upon his physical examination, the patient exhibited a diffused tenderness to the gastrocnemius muscle, causing a slight limp. There was edema to the posterior surface of the left leg, with associated local erythema and increased local temperature. Tenderness was extending to the posterior surface of the thigh. Bilateral inguinal lymph nodes were palpable and not tender. Physical examination revealed eczema on the medial side of the first left toe (Fig. 1). According to the mother, the eczema was first noticed



Fig. 1. Presence of eczema on the medial side of the first left toe. [Color figure can be viewed in the online issue, which is available at www.aott.org.tr]



Fig. 2. Normal foot (a) and leg (b) radiographs.

approximately one month before the initial symptoms. Clinical examination of the cardiovascular, pulmonary and other systems was unremarkable.

Blood tests showed an increase in the white blood cell count ($14.6 \times 10^3/\mu\text{l}$) with 48% neutrophils. Erythrocyte sedimentation rate and C-reactive protein were significantly increased, 75 and 142 respectively. All biochemistry tests, including creatinine kinase, were normal. All tests for the immune system function were also normal.

The clinical signs and the laboratory test results suggested pyomyositis. After blood cultures were obtained, the patient was given penicillin combined with cefuroxime and dicloxacillin intravenously. Radiographs of the lower extremity did not reveal signs of osteomyelitis in the tibia, fibula or the femur (Fig. 2). Radiographs of the first metatarsal and pha-

langeal bones of the first toe were also normal. Magnetic resonance imaging (MRI) of the affected limb showed diffuse enlargement of the left gastrocnemius and other posterior muscles. On the affected areas, there was an intermediate intensity signal on T1-weighted images and hyperintense signal on T2-weighted images (Fig. 3). A small amount of fluid was detected underneath the fascia, with a high intensity signal in T2-weighted images. Post-gadolinium scans demonstrated enhancement of the affected muscles with hyperintense signal in T1-weighted images (Figs. 4a-d). These findings suggested an initial stage of pyomyositis with associated fasciitis. There was no abscess formation.

The blood cultures revealed *Streptococcus haemolyticus* type A. Based on the results of the antibiotic sensitivity tests, the treatment was

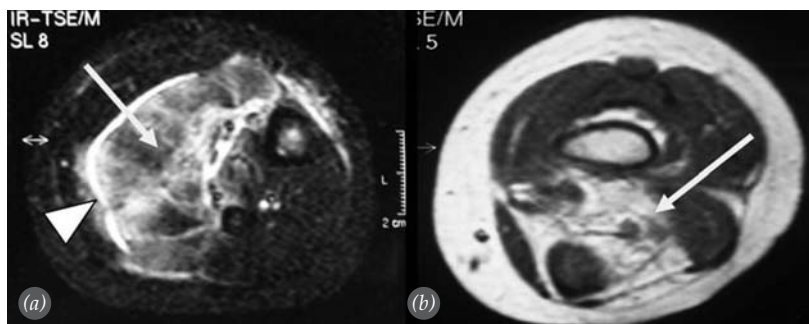


Fig. 3. Magnetic resonance imaging revealed enlargement of gastrocnemius and hyperintense signal of muscle (arrow) and deep fascia (arrowhead) in axial T2 STIR sequences (a), while axial post gadolinium T1-weighted images showed enhancement in biceps femoris muscle (arrow) (b).

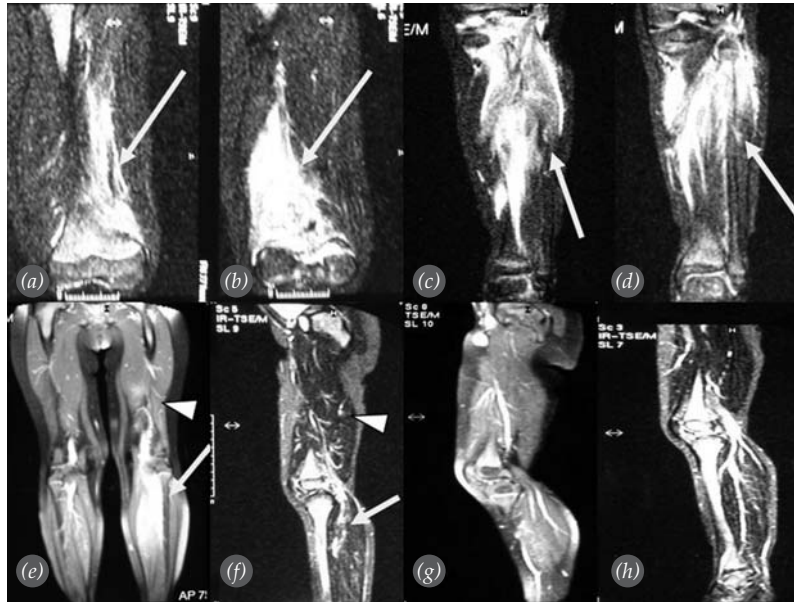


Fig. 4. Magnetic resonance imaging revealed enlargement and hyperintense signal of gastrocnemius and biceps femoris muscles (arrows) in coronal T2 STIR sequences (a-d). After 15 days, remission of symptoms was reported. A control MRI showed small high signal area in the gastrocnemius muscle (arrows both in e and f) in coronal post gadolinium T1-weighted images (e) and in sagittal STIR T2-weighted images (f), whereas biceps femoris had normal signal (arrowheads both in e and f). Last MRI, 2 months after the initial diagnosis, illustrated no signs of infection in sagittal post gadolinium T1-weighted images (g) and in sagittal STIR T2-weighted images (h).

changed to penicillin and vancomycin, given intravenously for 15 days.

The fever resolved on the 4th day of treatment. The control MRI, taken 15 days after the intravenous antibiotic treatment, showed complete remission of inflammatory changes in the thigh and mild changes in the gastrocnemius muscle (Figs. 4e and f). The patient was discharged on the 20th day. He was asymptomatic and his white blood cell count was $11 \times 10^3/\mu\text{l}$. The erythrocyte sedimentation rate and C-reactive protein level returned to normal, being 40 and 1 respectively. Oral penicillin was given for another 20 days. There was no sign of infection on the last MRI taken 2 months after the initial diagnosis (Figs. 4g and h). Follow-ups at six and 12 months after the discharge were normal and there was no recurrence.

Discussion

Pyomyositis represents an infection in the muscle tissue. Typically, it causes an intramuscular abscess formation. It usually affects young adults. Initially, it

has been reported as “pyomyositis tropicans”, due to the high incidence rate in populations in tropical regions.^[1]

Patients in tropical climates tend to have higher incidence rate in two age groups; one between 2 and 5 years old and the other between 35 and 40. The mean age is 28.^[2] On the contrary, it is believed that there is no pattern or higher incidence in specific age groups in non-tropical cases (with a mean age at 34 years).^[4] The male gender has an increased incidence rate, compared to the female, with a male-to-female ratio being approximately 3/1. This ratio is present in non-immunocompromised patients, but it is even higher in patients with the HIV infection.^[4]

Different theories were proposed in the past, in an effort to correlate the tropical climate with the disease. In some patients, vitamin C and vitamin B deficiency could have a predisposing role for the disease.^[5,6] Recently, it was proposed that pyomyositis in temperate climates is directly related to conditions that impair the immunity.^[4] Conditions, such as HIV infection, malignancy, diabetes mellitus, and

intravenous drug use are considered as high-risk factors for the development of pyomyositis.^[7]

In our case, there was no evidence of immune system deficiency. The presence of eczema on his left toe must be taken into consideration, because there are reports associating atopic eczema with immune system deficiency.^[8,9] The skin lesion may also be directly responsible for the muscle infection, as it is the entry site for the microorganism.

The overall pathogenetic mechanism remains unclear. Although bacteraemia rarely leads to muscular infection, transient bacteraemia may complicate local trauma, intensive exercise, or any other case of muscle injury.^[10] This hypothesis is consistent with the findings of Miyake^[11] who proposed that local infection and subsequent abscess formation may be seen after a muscle injury. Male predominance and the higher incidence of musculoskeletal trauma in men support this pathogenetic mechanism.^[2] Most authors report a definite history of muscular trauma or muscle exertion being present in certain patients, especially in those, with no risk factor.^[2-4,12]

Multifocal involvement is reported in approximately 13% of the cases. This rate is even higher in tropical climates and in patients with risk factors.^[2,13] The multifocal nature of the present case suggests the haematological spread of the disease to the affected muscles as the possible pathogenetic mechanism.

The organism mostly identified in both abscess and blood specimens is staphylococcus aureus.^[4] This organism has been held responsible for more than 90% of the cases in tropical regions and only 70% of the cases in non-tropical.^[2,4] *Streptococcus pyogenes* is the causative microorganism in approximately 9% of the non-HIV cases in patients without an underlying medical condition.^[4] The presence of *Streptococcus haemolyticus* type A in blood cultures support our hypothesis that transient bacteraemia could be the potential pathogenetic mechanism.

Blood tests usually reveal elevation of the white blood cell count, with dominance of the neutrophils. Eosinophilia is found in 10% of the patients with pyomyositis tropicans, which suggests a potential co-existing parasitic infection.^[2] This finding suggests a poor immunodeficiency level of the patient, being prone to parasitic infections, rather than an etiological contribution of the parasites.

In conclusion, we report a case of an immunodeficient child, who developed multifocal pyomyositis from *Streptococcus haemolyticus* in a temperate climate. The presence of atopic eczema could be an early sign of immunodeficiency and could be associated with the pathogenesis of the infection. Primary pyomyositis is an infrequently reported condition that is becoming more commonly recognized. A high level of suspicion is essential for the diagnosis of pyomyositis, especially in patients with potential immunodeficiency. Early diagnosis and treatment will minimize the morbidity of the disease by preventing the complications and subsequent surgical procedures.

Conflicts of Interest: No conflicts declared.

References

1. Scriba J. Beitrag zur Aetiologie der Myositis acuta. Dtsch Z Chir 1885;22:497-502.
2. Chiedozi LC. Pyomyositis. Review of 205 cases in 112 patients. Am J Surg 1979;137:255-9.
3. Gibson RK, Rosenthal SJ, Lukert BP. Pyomyositis. Increasing recognition in temperate climates. Am J Med 1984;77:768-72.
4. Crum N. Bacterial pyomyositis in the United States. Am J Med 2004;117:420-8.
5. Idoko JA, Jimoh O, Onyewotu II. Has lack of vitamin C any role in the aetiology of tropical pyomyositis? J Infect 1990;21:7-9.
6. Engel D. Tropical pyomyositis, a thiamine-deficiency disease. Med Hypotheses 1981;7:345-52.
7. Christin L, Sarosi GA. Pyomyositis in North America: case reports and review. Clin Infect Dis 1992;15:668-77.
8. Byrom NA, Timlin DM. Immune status in atopic eczema: a survey. Br J Dermatol 1979;100:491-8.
9. Howell MD, Boguniewicz M, Pastore S, Novak N, Bieber T, Girolomoni G, Leung DY. Mechanism of HBD-3 deficiency in atopic dermatitis. Clin Immunol 2006;121:332-8.
10. Smith IM, Vickers AB. Natural history of 338 treated and untreated patients with staphylococcal septicaemia (1936-1955). Lancet 1960;275:1318-22.
11. Miyake H. Beiträge zur Kenntnis der sogenannten Myositis infectiosa. Mitt Grenzgeb Med Chir 1904;13: 155-98.
12. Spiegel DA, Meyer JS, Dormans JP, Flynn JM, Drummond DS. Pyomyositis in children and adolescents: report of 12 cases and review of the literature. J Pediatr Orthop 1999;19:143-50.
13. Gubbay A, Isaacs D. Pyomyositis in children. Pediatr Infect Dis J 2000;19:1009-13.