A Rare Case of Tuberculous Pyomyositis Masquerading as Dermatomyositis

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Intro: Tuberculosis (TB) can manifest as two diseases: latent tuberculosis (LTI) or active tuberculosis. TB frequently involves the lung but can be extrapulmonary. Primary tuberculous myositis accounts for <1% of skeletal TBI.[1] Here we discuss a rare case of primary tuberculous pyomyositis. The patient was treated for what was thought to be dermatomyositis (DM) given his presentation with bilateral upper extremity swelling and positive markers on the myositis panel. Immunosuppressive therapy was administered for approximately 9 months. After many hospitalizations, including ICU admissions, he was eventually diagnosed and treated for tuberculous pyomyositis.

Case: A healthy 44 year-old-male with no past medical history was admitted to the hospital for bilateral hand swelling, R periorbital edema, dyspnea and fever. On admission he was found to be hypoxic to 91%. Chest CT that showed ground glass opacities, nodules with one 5 mm nodule in the right upper lobe. His COVID PCR and Quantiferon Gold were negative. AST/ALT were elevated 485/301. Autoimmune hepatitis workup revealed +anti-smooth muscle antibody (1:160) and +ANA (1:80) with negative biopsy. He was diagnosed and treated for community acquired pneumonia and discharged. One month later he presented with the exact same symptoms. Myositis panel was positive for Anti-MDA-5 and Anti-SSA 52. He was discharged with a prednisone taper and was started on mycophenolate a few days later in the outpatient setting. Approximately four months later he was re-admitted for severe left upper extremity swelling. Pathology of the left forearm biopsy showed mild myositis with four necrotizing granulomas with negative AFB and fungal cultures. There was low suspicion for an acute infectious process at the time. Methotrexate (MTX) was added to his regimen of methylprednisolone and mycophenolate in the outpatient setting due to worsening swelling. Approximately 2 weeks after starting MTX he was readmitted for worsening symptoms. He was found to have lactic acidosis, ESR 57, CRP 66.9, and CPK 244. AFB sputum cultures were negative. Karius testing was positive for TB. RIPE therapy was started and immunosuppressive therapy was discontinued. The patient was unable to tolerate RIPE therapy at home and was readmitted 1 month later for septic shock. He started amikacin, moxi and ethambutol therapy. Of note the nodules found in his prior admission now appeared to be cavitating. During this hospitalization he was found to have a new L thigh abscess with +PCR and a repeat muscle biopsy of the L forearm showed necrotic granulomas and + AFB stain. He was discharged with rifampin, moxifloxacin, and ethambutol. After one year of this therapy, all of his symptoms resolved.

Discussion: This case report signifies the importance of considering tuberculosis pyomyositis as a potential diagnosis for a patient that fails to improve with immunosuppressive therapy and has risk factors for TB. It also emphasizes the downfall of relying on screening exams to diagnose and rule out conditions. Due to its rarity and variability in presentation the diagnosis and treatment of tuberculous pyomyositis is frequently delayed. In addition to this although poorly understood there is documented evidence of autoimmune processes resulting in autoimmune antibodies in TB. Providers should consider tuberculous pyomyositis for patients who are at risk for TBI and are unresponsive to immunosuppressive therapy.

Conclusion: The case report highlights the importance of tuberculosis pyomyositis in the differential diagnosis of patients who are at risk. As well as the rarity, variable in presentation presentation and frequently delayed diagnosis of tuberculous myositis. There is also a poorly understood but well documented presence of the development of autoantibodies in TB infections.

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