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Disseminated Gonococcal Arthritis Associated with Systemic Lupus Erythematosus

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Abstract

Signs and symptoms of gonococcal infection in patients with systemic lupus erythematosus (SLE) are overlapping and sometimes difficult to distinguish from the manifestations of SLE itself. We are reporting on a patient with a recent SLE flare who developed disseminated gonococcal infection (DGI) as a result of the nature of SLE and the management of a flare.

Keywords: Disseminated gonococcal arthritis; Systemic lupus erythematosus; CRP; Suppurative arthritis syndrome; Tenosynovitis-dermatitis syndrome

Introduction

SLE is a chronic autoimmune disease characterized by multisystem inflammation and the presence of circulating autoantibodies directed against self-antigens according to Nelson Textbook of Pediatrics (841). In SLE, any organ system can be involved, with the most common complaints of fever, arthralgia, and arthritis to be the presenting symptoms which can be difficult to differentiate from a Gonococcal infection. A simple Gonococcal infection, more likely to be falsely denied in the adolescent population, can more rapidly disseminate during an SLE flare due to the nature of the disease as well as the treatment of the flare. The unique relationship between an SLE flare and DGI, their musculoskeletal manifestations, clues to differentiating the diagnoses, and DGI treatment are discussed.

Case

A 17-year-old African-American female with Systemic Lupus Erythematosus (SLE) that was treated for a polyarthritis due to Lupus flare. Her symptoms were controlled with 20 mg oral prednisone twice daily and hydroxychloroquine 300 mg daily. Laboratory tests at that time showed positive antinuclear (ANA), and double-stranded DNA antibodies (ds-DNA). Also the RNP,

SSA and SSB antibodies were positive. Her WBC was 8000/mm³ with normal differential, and C-reactive protein (CRP) was slightly elevated at 3 mg/dl (normal 0-0.5 mg/dL). The hemoglobin and hematocrit were 10.6 g/dl and 31.9%, respectively (normal 1214g/dL and 3641%, respectively). The complement components C3 and C4 levels were decreased at 35.4 and 5.0 mg/dL (normal 83-177 and 15-45 mg/dL, respectively), and IgG level was elevated at 2230 mg/dl (normal 595-1275 mg/dL).

Two weeks later the patient presented to the emergency room with complaints of pain and swelling originating in her right ankle that recently progressed bilaterally to her knees, ankles, wrists and fingers. She noted this pain as "more painful than usual," claiming it was unresponsive to her usual treatment of NSAIDs and opiates. Also noted was a history of fevers with spikes around 102 °F and a raised papular rash spreading to her face. She denied any previous sexual activity. A physical examination found that the patient appeared to be in severe pain, with swelling of her knees, ankles, elbows and wrists, which displayed redness and tenderness to palpation. In addition there was an evidence of tenosynovitis in both hands, and non-blanching pink patches and plaques ranging from 1-3 mm in diameter on her palms, wrists, arms and face.

Laboratory testing showed an elevated white blood cell count of 14290/mm³ (normal 4500-13000/mm³) with 89% segmented neutrophils, a significantly elevated C-reactive protein (CRP) of 17.5 mg/dL (normal <2 mg/dL), and normal platelets of 182,000/mm³. The patient's hemoglobin and hematocrit were reduced to 10.3 g/dL and 24.9%, respectively. No growth was noted from the blood culture. Urinalysis showed 1+ protein, 25-35 white blood cells, 15-25 red blood cells with trace leukocyte esterase and negative bacteria. Complete metabolic panel was within normal limits. Her complement components C3 and C4 were both slightly decreased at 78 mg/dL and 16.1 mg/dL, respectively. Quantitative immunoglobulin IgG was elevated at 1890 mg/dL. The IgA and IgM were both within normal limits.

Due to the high fever, rash, tenosynovitis and uncharacteristic and unresponsive severe pain, a diagnosis of gonococcal

infection concurrent to the patient's underlying diagnosis of SLE was considered. The patient's right knee was aspirated, and the culture showed moderate growth of *Neisseria gonorrhoeae*. Based on these findings she was diagnosed with DGI and treated with ceftriaxone (1 g intravenous) as well as her usual SLE flare treatment of intravenous Methylprednisolone, oral hydroxychloroquine, cyclobenzaprime and meloxicam. After being pressed further, the patient admitted to being sexually active. Upon discharge she received a seven day course of doxycycline (100 mg daily) and cefixime (400 mg twice a day). There was no evidence of arthritis, morning stiffness, muscle pain, fevers, or rash at follow-up three weeks later.

Discussion

Patients with SLE carry an increased risk for severe gonorrheal infection due to the nature and management of the disease. Reasons include an iatrogenically induced immunosuppressive state from being on immunosuppressive agents; inherited or acquired complement deficiencies [1] that occur secondary to increased C3 and C4 consumption during an SLE flare [2]; delayed recognition; and the impairment of the disposal of immune complexes and bacteriolysis by the mononuclear phagocytic system [3]. Of note, CRP is a poor marker for a SLE flare, and any elevated CRP warrants a search for coincident inflammation or infection [4]. In our case, the patient's CRP was only mildly elevated during the presentation of her recent SLE flare, yet became extremely elevated during the DGI.

SLE flare and disseminated gonococcal infection (DGI) can be difficult to differentiate due to overlapping rash and arthritis symptoms. The classic suppurative arthritis syndrome presentation of DGI with a single hot, swollen joint is only present in 20% to 30% of patients, while the tenosynovitis-dermatitis syndrome involving polyarthritis/arthritis and skin lesions resembling an SLE flare are present in approximately half of initial manifestations [5]. It is not uncommon to have clinical manifestations of the both DGI variants, as is evident in our patient.

The severe monoarticular pain that was unresponsive to her usual SLE treatment of NSAID's and opiates, as well as the character and increased intensity of our patient's tenosynovitis, hinted that her symptoms were not solely the result of an SLE flare. *Neisseria gonorrhoeae* is a highly transmissible, sexually transmitted infection that causes an estimated 820,000 new infections per year in the United States [6], less than half of which are detected and reported. DGI occurs in 1% to 3% of all gonococcal infections [7]. The majority of infections occur in sexually active teens and young adults aged 15 through 24, with the rate of infection higher in females than males, and 17 times more common in black females than white females [8]. The infection can also spread vertically from mother to neonate during childbirth. Potential manifestations range from mild to severe symptoms affecting the pharynx, urethra, anus, endocervix and epididymis. Early infections can be asymptomatic in all populations, allowing the infection in 10% to 20% of untreated women to spread into the uterus and fallopian tubes causing silent to life-threatening manifestations of pelvic inflammatory disease (PID); nearly 20% of these untreated

women experience subsequent uterine tubal scarring, which increases the risk of ectopic pregnancy, tubo-ovarian abscess and involuntary infertility. As the infection disseminates it can cause petechial or pustular dermatitis, asymmetrical arthralgia, osteomyelitis, meningitis, septic arthritis, tenosynovitis, perihepatitis known as Fitz-Hugh-Curtis, and septic shock [7].

Specific testing for diagnosis includes culture, PCR by nucleic acid hybridization test, and nucleic acid amplification tests (NAATs) on urine and other body fluid samples. In suppurative arthritis syndrome, synovial fluid cultures are positive in 45% to 55%, while blood cultures are generally negative. In tenosynovitis-dermatitis syndrome, 30% to 40%, blood cultures are positive, while synovial cultures are generally negative [7]. Recommended therapy for DGI is ceftriaxone or cefotaxime for a 7 day total course. Chlamydia trachomatis should be presumptively treated for if not ruled out [9].

Conclusion

A recent SLE flare carries an increased risk for DGI due to an iatrogenically immunosuppressed state, acquired complement deficiencies, delayed recognition, and impairment of the mononuclear phagocytic system, yet can be difficult to differentiate due to the overlapping rash and arthritis symptoms. A mildly elevated CRP with a baseline character and intensity of arthritic symptoms would likely suggest a SLE flare, while a pronounced CRP and atypical character and intensity of the arthritic symptoms would more likely suggest a DGI. Confirm Gonococcal infection with urine or blood PCR. Blood cultures and synovial fluid cultures can help differentiate between both DGI types.

Author Contributions

EL is a pediatric resident that managed the patient with us and wrote the short communication. AB and AG are the rheumatology attending in charge of the management. ED assisted with technical editing.

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