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Polyarthrititis in a 19-Year-Old Female With Systemic Lupus Erythematosus

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Case Presentation

A 19-year-old female with a history of systemic lupus erythematosus (SLE) and class V lupus nephritis presented to the emergency department with complaints of left wrist pain and swelling as well as a several-day history of bilateral ankle and foot pain. Acetaminophen initially provided symptomatic relief but by the day of presentation, pain and swelling diminished use of her left hand and her feet and ankles were swollen. She denied any concurrent fevers, upper respiratory symptoms, vomiting, diarrhea, urinary symptom, or rashes. She had been seen by her rheumatologist approximately 2 weeks prior at which time she had hypertension, elevated inflammatory markers, as well as anemia, hypoalbuminemia, and proteinuria, thought to be due to a flare of the lupus involving the kidney.

On examination in the emergency department, the patient had limited range of motion accompanied by tenderness to palpation of the left wrist, left second proximal interphalangeal joint, left third distal interphalangeal joint and bilateral ankles but without erythema, warmth, or swelling. She also had tenosynovitis of the left hand extensor tendons and right foot extensor tendons. Except for hypertension (154/99 mm Hg) and a left buccal mucosal sore, the remainder of her physical examination was within normal limits. Laboratory evaluation again demonstrated the elevated inflammatory markers (erythrocyte sedimentation rate 66 mm/h [range = 0–20] and C reactive protein 5.46 mg/dL [normal <1 mg/dL]) along with hematuria (15–19 cells/high-power field), proteinuria (109.4 mg/dL protein [normal <12 mg/dL]), mild anemia (hemoglobin 11.4 g/dL) and hypoalbuminemia (2.8 g/dL). The combination of physical and laboratory findings resulted in the rheumatologist diagnosing the patient as having a flare of her lupus. She received 100 mg of methylprednisolone intravenously (IV) in the emergency department and was discharged to home with a prescription to take 20 mg per day of oral prednisone.

The persistence of symptoms despite steroid treatment led the patient to return to the clinic for evaluation by her rheumatology provider. She continued to have joint swelling and pain

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of the left wrist and right ankle and the pain in her right ankle became so severe she was using crutches to assist her in ambulation, which was concerning since it is unusual for such severe arthritis pain to be associated with lupus. The patient denied fevers or rash. Her physical exam was remarkable for progressive right ankle warmth, redness, and swelling. Additional history discovered the patient reported recent sexual activity but with a single partner. A radiograph of her right ankle was obtained as well as a nucleic acid amplification test (NAAT) of the urine for *Neisseria gonorrhea* and *Chlamydia trachomatis*. The radiograph revealed an ankle effusion, synovial thickening, and swelling of the right lateral malleolus without any evidence of fracture. The NAAT was positive for *N gonorrhea*.

Final Diagnosis

Disseminated gonococcal infection.

Hospital Course

After the diagnosis of *N gonorrhea* was made, the patient was admitted to the hospital for further testing, including arthrocentesis of the right ankle. Four milliliters of bloody synovial fluid was obtained but a cell count was unable to be performed because of immediate clotting of the fluid. A gram stain of the fluid demonstrated many white blood cells but no organisms and the culture subsequently was sterile. A blood culture obtained the same day as the arthrocentesis was also sterile. Results of HIV, syphilis, and trichomonas testing were negative. Further history revealed that her last menstrual period had started within 7 days of the onset of her initial symptoms. She was administered an initial 2000 mg dose of IV ceftriaxone followed by 1000 mg IV daily for 7 days. She also was administered a 7-day course of oral doxycycline at a dose of 100 mg twice a day. Within 24 hours of initiation of antimicrobial therapy, her left wrist pain and swelling had resolved. Two days later, her right ankle pain had improved, and by day 6 of therapy, mild right ankle edema was her only persistent symptom.

Discussion

Neisseria gonorrhea remains the second most common sexually transmitted disease in the United States, with an estimated 700 000 new cases occurring each year.¹ Disseminated gonococcal infection (DGI) is well described, once estimated to occur in 0.5% to 3% of patients with gonorrhea.² However, the frequency of DGI appears to be decreasing so when it does occur, the diagnosis may be delayed significantly as occurred in the current case.

Joint involvement from DGI has 2 reported forms: arthritis–dermatitis syndrome and septic arthritis. Arthritis–dermatitis syndrome occurs during the bacteremic phase of illness and often involves fevers, chills, and other systemic symptoms. In addition, polyarthralgias, often involving the knees, elbows, wrists, ankles, and other distal joints, are common and may occur in conjunction with tenosynovitis.^{3, 4} Up to 50% of patients will have a positive blood culture in this stage.⁴ Up to 75% of patients may experience skin lesions, which are often painless and include papules, pustules, vesicles, or bullae that may have a hemorrhagic component.^{2, 4} Untreated patients may progress to septic arthritis of one or more of the

previously inflamed joints.^{2, 3, 5} However, the initial presentation of DGI may be septic arthritis either because the arthritis–dermatitis syndrome was unrecognized or did not occur.⁴ Other less common clinical syndromes that can occur with DGI include endocarditis, myocarditis, pericarditis, hepatitis, and meningitis.³

Disseminated gonococcal infection is thought to be the result of dissemination of *N gonorrhea* in the bloodstream to other locations, but other indirect immunologic methods may also be contributing.^{2–4} Specifically, the frequency of positive blood cultures in the early stages of DGI appears to point to a more direct mechanism of infection, and in fact *N gonorrhea* possesses a potent endotoxin on the surface of viable or even nonviable bacteria.^{3, 4} However, gonococcal antigen–antibody immune complex deposition similar to other syndromes such as serum sickness has been postulated, although not yet proven.^{3, 4}

Features of *N gonorrhea* strains leading to DGI include the ability to resist the bacteriocidal action of human serum and cause minimal genitourinary inflammation. They often belong to the porin 1A serotype, which can bind to complement downregulatory molecules and decrease the local inflammatory response.^{2, 4} The critical role of complement and humoral immunity in preventing DGI is further illustrated by the fact that up to 13% of patients with DGI will have complement deficiencies.^{2, 4}

Female gender is another risk factor for DGI with more than 80% of cases reported in women, likely related to women often having asymptomatic primary infections.^{3–5} Timing of menstruation is also a risk factor, with DGI occurring most commonly during the first 7 days of the menstrual cycle.^{2–5}

Although blood cultures may be positive in up to 50% of patients with DGI, bacteremia often is intermittent so a minimum of 3 blood cultures should be obtained when DGI is suspected.^{2, 4} Synovial fluid leukocyte counts in septic arthritis typically range between 40 000 and 60 000 cells/mm³ with greater than 80% polymononuclear cells.⁴ In approximately 30% of the cases, *N gonorrhea* may be isolated from cultures of synovial fluid.^{2, 3} Rarely, organisms may be recovered from cultures of pustular skin lesions.^{2, 3} The most sensitive test for diagnosis of DGI is culture or NAATs of urine or genital secretions, which would be expected to be positive in up to 80% of patients even if they are not experiencing any urogenital symptoms.^{2, 4} Any patient with DGI should also be evaluated for *C trachomatis*.

Treatment of DGI typically involves hospitalization for initial therapy with the administration of ceftriaxone 1000 mg IV or intramuscularly (IM) every 24 hours. Alternate regimens include cefotaxime or ceftizoxime 1000 mg IV every 8 hours. IV or IM therapy should be continued for 24 to 48 hours after clinical improvement, at which time therapy can be changed to oral cefixime 400 mg twice daily to complete 7 days.¹ With treatment, joint symptoms typically resolve within a few days, and no treatment failures have been reported with the recommend regimens.^{1, 5} Patients treated for DGI should always be treated presumptively for *C trachomatis* even if testing is negative.¹

Conclusion

Adolescents with SLE are in a unique risk group for undiagnosed DGI. Sexually active adolescents are at higher risk for sexually transmitted infections than the general population, and adolescent attitudes about disclosure of sexual activity to health care providers vary. In addition, SLE is associated with hypocomplementemia and renal disease, which may impede a person's ability to fight gonococcal disease. Furthermore, the presentation of DGI often includes rash and arthritis, which can be mistaken for a lupus flare. This case underscores the importance of considering DGI in a sexually active adolescent with SLE who presents with polyarthritis.

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