Brief Communication

Bacterial Monarthritis Due to *Neisseria meningitidis* in Systemic Lupus Erythematosus

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Abstract. **Two patients with systemic lupus erythematosus developed monarticular infectious arthritis, in which *Neisseria meningitidis* was recovered from knee synovial fluid. In one instance, the sole manifestation of meningococcal disease was a chronic, indolent, erosive monarthritis. In the second, a febrile, bacteremic illness presented with an acute, septic arthritis.** *(J Rheumatol 8:* **145-148, 1981)**

Key Indexing Terms:
INFECTIOUS ARTHRITIS MENINGOCOCCAL ARTHRITIS
SYSTEMIC LUPUS ERYTHEMATOSUS INFECTION IN SLE

Meningococcal infection may present as meningitis, endocarditis, urethritis, or as a fulminant, subacute, or chronic bacteremic illness. Fam, *et al*[1] and others[2-5] have described and categorized several forms of meningococcal articular disease: a) Type I – early onset acute polyarthritis with systemic meningococcemia; b) Type II – late onset post-infectious mono- or oligoarthritis; c) Type III – isolated acute infectious arthritis without meningococcemia; d) Type IV – arthritis of chronic meningococcemia. Two patients with systemic lupus erythematosus (SLE) and monoarticular meningococcal septic arthritis are reported, an association not described previously. The sole feature of meningococcal disease in the 1st case was a chronic, indolent, erosive monarthritis (Type III). Meningococcal infection presented as an acute septic arthritis (Type I) in the 2nd patient.

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CASE REPORTS

Case 1. A 49 yr old woman with SLE of 9 months' duration was referred because of left knee pain and swelling. Fever, pleurisy, alopecia, oral mucosal erosions, acute glomerulonephritis with nephrotic syndrome and renal insufficiency, transient left ventricular failure, and arthritis were recent problems treated with tapering doses of prednisone, digoxin, and diuretics. A several year history of episodic painful swelling of the left knee without

preceding trauma, locking, or buckling was elicited. Four months before, knee pain and swelling progressed, without constitutional manifestations. Roentgenograms 1 month later showed a 0.5 cm erosion of the medial, proximal tibia.

Abnormal findings included alopecia, and a swollen, warm, tender left knee with limited motion (20-120°). The hemoglobin (Hb) was 9.8 g/dl, white blood cell (WBC) count was 4,000/mm^3 (67% PMN), and trace proteinuria was present. Roentgenograms showed a 1.0 cm erosion of the left tibia (Figure 1a). Arthrocentesis yielded 10 ml of purulent fluid with a poor mucin clot, WBC count of 18,000/mm^3 (84% PMN), protein of 6.0 g/dl, and glucose of 18 mg/dl (with normal blood glucose). Synovial fluid culture grew *Neisseria meningitidis*, with weak reaction to type W-135 antiserum.

Treatment consisted of intravenous aqueous penicillin (10 million units daily for 1 wk, then 20 million units daily for 3 wk, in divided doses), bed rest, range of motion and muscle strengthening exercises, and 2 further joint aspirations. The tibial erosion diminished and was surrounded by a rim of bony sclerosis (Figure 1b). At discharge the knee was asymptomatic with full motion, but a small effusion, thought to represent reactive arthritis, persisted.

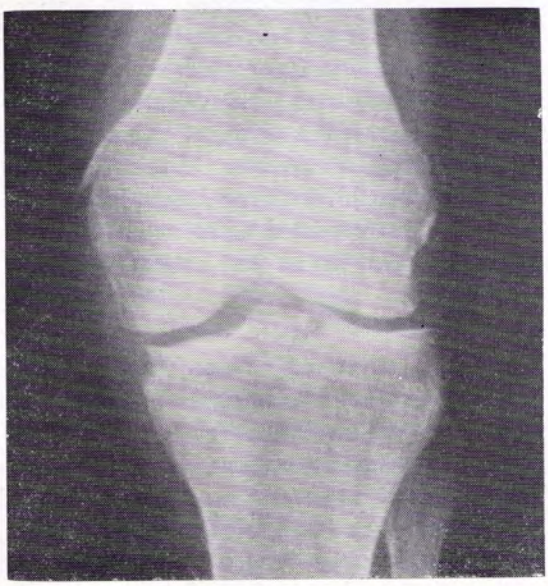

Fig. 1a. (Case 1) Left knee roentgenogram taken August 23, 1979, prior to antibiotic treatment, showed a subchondral erosion on the medial tibial plateau.

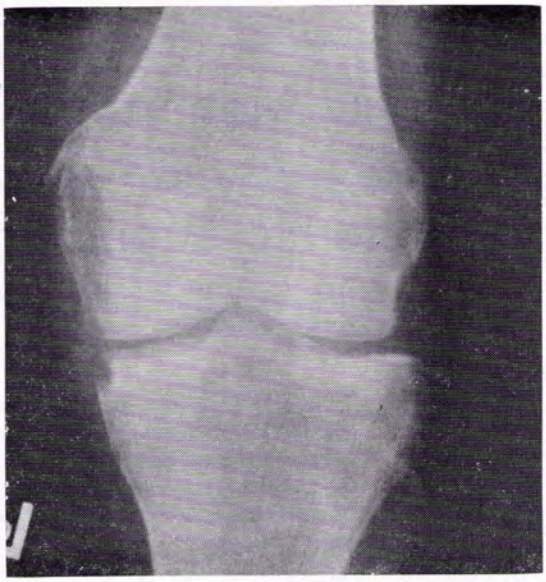

Fig 1b. Roentgenogram of the same joint taken October 1, 1979, after 4 wk of iv penicillin therapy, showed a reduction in erosion size and surrounding sclerotic rim.

Case 2. A 21 yr old woman was hospitalized because of right knee pain, swelling, and limitation of motion of 24 h duration. Two recent episodes of undocumented fever and chills were reported. Four yr previously SLE was diagnosed, based on fever, arthralgias, lymphadenopathy, hemolytic anemia, antinuclear antibody (ANA) titer of 1:8,000, and positive LE preps. Prednisone (60 mg/d) and physical therapy were prescribed for severe proximal myopathy, nephrotic syndrome, and cerebritis when arthritis occurred.

Physical examination revealed a Cushingoid woman with fever (37.7°C), inappropriate affect, paranoid ideation, and memory lapses. A butterfly rash, facial acneform eruption, alopecia, and abdominal striae were observed. The right knee was warm, erythematous,

tender, and swollen; flexion was limited to 25°. The proximal lower extremity muscles were profoundly weak and atrophic.

The Hb and hematocrit were 11.3 g/dl and 36.5% respectively; WBC count – 18,000/mm^3 with a left shift; uric acid 15.3 and 5.7 mg/dl; creatinine – 1.6 mg/dl; BUN – 35 mg/dl; glucose – 300 and 168 mg/dl, lactic dehydrogenase 379 units, Westergren erythrocyte sedimentation rate – 58 mm/h, and fluorescent ANA-positive with a homogenous pattern. Knee roentgenograms showed soft tissue swelling. Numerous other tests were normal or negative. Twenty cc of serosanguinous fluid was aspirated from the right knee. Synovianalysis revealed 2,300 WBC/mm^3, 2,400 RBC/mm^3, glucose-6 mg/dl, and gram negative cocci in pairs. Blood and synovial fluid cultures grew *N. meningitidis* (serologic group not reported) on the 3rd day of hospitalization. Cerebrospinal fluid and nasopharyngeal cultures, obtained after penicillin therapy was initiated, were negative.

Penicillin G therapy (2 million units q 4 h iv) and hydrocortisone (250 mg q 6 h iv) were administered, and arthrocentesis was repeated twice. All articular symptoms and findings resolved within 7 d.

DISCUSSION

Septic arthritis is one of the less frequent infectious complications of SLE[6,7]. Joint trauma, intraarticular injections, lupus synovitis, and corticosteroid treatment appear to be predisposing factors[8]. In contrast to rheumatoid arthritis wherein *Staphylococcus aureus* is the most common cause of joint sepsis, *N gonorrheae* and gram negative bacilli are equally responsible pathogens in SLE[8].

Chronic meningococcal monarthritis was unassociated with fever, rash, meningitis, or constitutional symptoms and signs in the 1st patient (Type III infection). High dose corticosteroid therapy for lupus nephritis may have masked signs of joint infection initially, but fever, leukocytosis, and other evidence of infection did not develop while on low-dose, alternate-day steroids. The onset of infection could not be determined, in part because timely arthrocentesis was not accomplished, although roentgenographic changes were evident more than 2 months prior to diagnosis. Destructive changes from meningococcal arthritis are observed rarely in the antibiotic era, and presumably resulted from delayed diagnosis and treatment[5,9].

In the 2nd case, meningococcal bacteremia was associated with acute septic knee arthritis which resolved rapidly after parenteral penicillin therapy (probably Type I infection, despite absence of rash and meningitis). Altered host defense mechanisms may have predisposed to meningococcal sepsis with metastatic localization to the knee.

Infectious arthritis should be considered in any lupus patient who develops a joint effusion, even if other features of articular inflammation or sepsis are subtle or lacking. Diagnosis requires immediate arthrocentesis; elevation of synovial fluid WBC count and depression of glucose level strongly suggest bacterial infection. Initial antibiotic choice should be determined by gram stain findings. However, an unrevealing gram stain should not deter prompt antibiotic administration. In such cases, antimicrobial therapy generally is determined by the clinical setting, pending culture results.

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