Prognosis

Risk for septic arthritis was increased in adults with incident diagnosis of gout

Clinical impact ratings: ★★★★★☆ ★★★★★☆ ★★★★★☆ ★★★★★☆ ★★★★★☆

Question
In adults with an incident diagnosis of gout, what is the risk for septic arthritis?

Methods

Design: Inception cohort followed for a mean of 5 years.

Setting: The Health Improvement Network (THIN) database, representing 580 general practices in the UK.

Patients: 72,073 adults ≥ 40 years of age (mean age 65 y, 71% men) who had an incident diagnosis of gout. The control group comprised 358,342 adults without gout, matched for age, sex, date of study entry, and enrollment year. Exclusion criteria were history of septic arthritis, gout, osteoarthritis, rheumatoid arthritis, or other inflammatory rheumatic condition (seronegative spondyloarthopathies, psoriatic arthritis, connective tissue disease, vasculitides, or crystal arthropathy).

Prognostic factors: Lifestyle factors (smoking and alcohol use), body mass index, socioeconomic deprivation index (Townsend Deprivation Index score), number of physician visits, glucocorticosteroid use, and Charlson comorbidity index.

Outcomes: Incident septic arthritis diagnosis associated with hospital admission or prescription for an antibiotic.

Main results

90 cases of septic arthritis occurred in the gout group and 166 cases in the control group. At 5-year follow-up, patients with incident gout had increased risk for septic arthritis compared with those without gout (Table).

Conclusion

Adults with an incident diagnosis of gout had increased risk for septic arthritis.

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Commentary

We know that patients with rheumatoid arthritis have an increased risk for acute septic arthritis, and case reports have confirmed that septic arthritis can also be associated with acute gouty arthritis. Lim and colleagues, in a novel cohort analysis of a large UK database, show that a first bout of gout was associated with a 2.6-fold increased risk for septic arthritis compared with patients without gout. However, due to numerous clinical exclusions (including pre-existing gout), they may have underestimated the true increased risk for septic arthritis in patients with gout.

Acute inflammatory monoarticular and oligoarticular arthritis are common conditions with an extensive differential diagnosis, most commonly acute crystal arthritis (gout or, less frequently, calcium pyrophosphate deposition disease or pseudogout), rheumatoid arthritis, or septic arthritis. Diagnostic workup mandates synovial fluid aspiration to examine for the presence of crystals and to obtain a Gram stain, nucleated cell count and differential, and bacterial culture (1).

Although a high leukocyte count in the synovial fluid (> 50,000/ mm³ with > 90% neutrophils) is highly suggestive of septic arthritis (2, 3), acute gout and pseudogout are also characterized by high leukocyte counts, typically 25,000 to 50,000/mm³ with neutrophilic predominance (3). Crystals can be identified in the synovial fluid of most patients with acute crystal arthritis (4), while about 75% of patients with septic arthritis will have a positive culture, with up to 50% having a positive Gram stain (2, 3). What is challenging is the 5% of patients with acute monoarticular arthritis or oligoarthritis with crystals who also have septic arthritis (5).

The study by Lim and colleagues reaffirms that synovial fluid obtained from patients with acute monoarticular or oligoarticular arthritis must routinely be analyzed for infection even when urate crystals are seen. Because the results of synovial fluid culture will be negative in up to 25% of patients with septic arthritis (2, 5)—especially if the patient has received empirical antimicrobial therapy—we should retain an aliquot of synovial fluid from patients with acute crystal arthritis for later analysis by 16S ribosomal RNA polymerase chain reaction. This assay can rapidly and accurately identify the bacterial pathogen in culture-negative septic arthritis in up to 50% of cases (6). If the patient has a high fever or signs of systemic infection at the outset or does not respond promptly to targeted antinfiammatory therapy, but Gram stain and bacterial cultures are negative, the specimen can be tested for evidence of infection by 16S ribosomal RNA polymerase chain reaction.

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References


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