Coll. Antropol. **29** (2005) 2: 743–746
UDC 616.72-002:616.379-008.64
Case report

Septic Acromioclavicular Arthritis in a Patient with Diabetes Mellitus

Nadica Laktašić-Žerjavić[1], Đurđica Babić-Naglić[1], Božidar Ćurković[1], Kristina Potočki[2] and Dragica Soldo-Jureša[3]

[1] University Department of Rheumatology and Rehabilitation, University Hospital Center »Zagreb«, Zagreb, Croatia
[2] University Department of Radiology, University Hospital Center »Zagreb«, Zagreb, Croatia
[3] Department of Immunology, University Department of Medicine, University Hospital »Dubrava«, Zagreb, Croatia

A B S T R A C T

A 44-year-old diabetic man with isolated septic arthritis of the left acromioclavicular joint (A–C) caused by Staphylococcus aureus is described. He was admitted to the Department of Rheumatology with clinical symptoms of left shoulder arthritis and fever. Laboratory findings showed leukocytosis, elevated levels of erythrocyte sedimentation rate and C-reactive protein, all indicating septic arthritis. Blood culture was positive for Staphylococcus aureus*. Left A–C joint x-ray and ultrasonography, and whole body scintigraphy with 99 mTc radiolabeled autologous leukocytes pointed to septic arthritis of the A–C joint. The patient was treated for six weeks with antibiotics successfully. Infection of the A–C joint is uncommon, even in conditions such as immunodeficiency, renal dialysis and intravenous drug abuse which are associated with unusual joint infections, and can be differentiated from shoulder joint infection, by maximal tenderness over the A–C joint on examination, and findings of A–C joint widening, effusion, and bony erosions on imaging studies.*

Key words: *acromioclavicular joint, septic arthritis, diabetes mellitus*

Introduction

Infectious arthritis represents invasion of the joint space by a variety of microorganisms. Although any infective agent can cause arthritis, bacterial pathogens lead to most rapid joint destruction. Despite advances in antimicrobial therapy, septic arthritis is often responsible for residual functional impairment (an irreversible loss of joint function in 25–50% of survivors)[1–3], leading to significant rates of morbidity and even mortality (mortality rate, 10–15%)[1–4].

Risk factors for septic arthritis are divided into local joint abnormalities, systemic factors, or both. They include older age, pre-existing joint disease (e.g., osteoarthritis, arthropathy), vulnerability of the host to infection due to disease or medication (e.g., rheumatoid arthritis, diabetes mellitus, HIV infection, injecting drug use, use of systemic corticosteroid or cytotoxic medications), joint trauma, and surgery or prosthesis[4–7].

The knee is the most commonly involved joint, followed by the hip and shoulder[4,7]. Infection of the acromioclavicular (A–C) joint is uncommon, and rarely seen even in immunocompromised patients, renal dialysis patients and intravenous drug users which tend to have unusual localization of joint infections[8–10].

Staphylococcus aureus accounts for up to two-thirds of the organisms identified in blood or joint cultures of patients with septic arthritis[4,7,11].

This case report describes septic arthritis of the A–C joint caused by *Staphylococcus aureus* in a diabetic patient.

Case Report

A 44-year-old male patient with the symptoms of left shoulder arthritis was admitted to the Department of Rheumatology. Careful interview revealed the pain in the left shoulder had started six days before and had been unsuccessfully treated with various analgesics including the conventional non-steroidal anti-inflammatory drug indomethacin in anti-inflammatory dosage.

Received for publication June 11, 2004

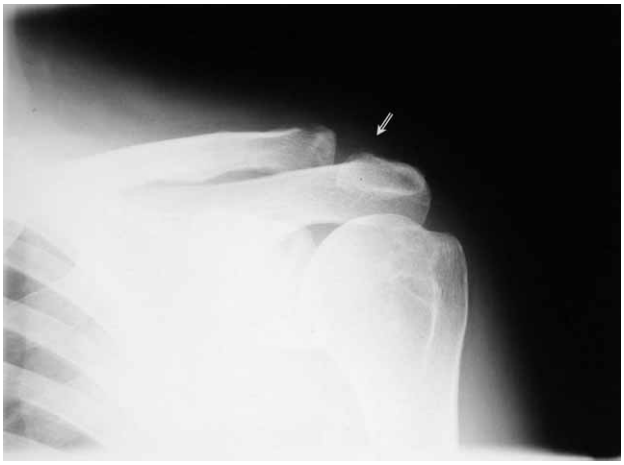

Fig. 1. Acromioclavicular (A–C) joint x-ray. Note subchondral osteopenia of the joint facets, especially of the upper part of the acromion (arrow) with slight widening of the joint space. No bony erosions are present.

After three days the patient became febrile with axillary temperature of 38.9°C. On the sixth day the shoulder pain became almost unbearable. The patient had been treated for high blood pressure and elevated serum glucose for 13 years, being on insulin therapy for the last seven years. The patient had no predisposing conditions for septic arthritis other than diabetes mellitus.

On admission, clinical examination revealed a slightly swollen left shoulder with warm and reddish skin discoloration over the upper aspect of the shoulder. There was no skin laceration or joint penetration. The range of the left shoulder movement was considerably reduced. The point of maximal tenderness and redness was detected over the left A–C joint. All other physical findings and vital signs short of temperature of 38°C were normal. Urgent laboratory tests were performed yielding the following findings: elevated erythrocyte sedimentation rate (ESR) 70 mm/h), C-reactive protein (CRP) 125 mg/L, peripheral blood leukocytes 12.3×10^9/L with left shift in white blood cell count; serum level of uric acid was normal. Plain radiograph of the left shoulder showed no marked pathology. Chest x-ray and electrocardiogram (ECG) were normal.

Based on the clinical and laboratory findings, and the history of diabetes mellitus, septic arthritis of the shoulder was suspected. Peripheral blood, urine, stool, and oral and nasal mucosa samples were obtained, and systemic antibiotic therapy was immediately administered. *Staphylococcus aureus* was considered as a possible pathogen, so the patient received cloxacillin (2 g every 6 h) and gentamicin (160 mg every 12 h) intravenously. Three peripheral blood cultures obtained simultaneously before the administration of systemic antibiotic therapy were positive for *Staphylococcus aureus* and sensitive to previously administered antibiotic therapy.

Normal plain radiograph of the left shoulder showed discrepancy with the clinical and laboratory findings that suggested septic arthritis of the shoulder. Additional imaging was done to reach an accurate diagnosis. The A–C joint x-ray demonstrated subchondral osteopenia of the joint facets with slight widening of the joint space. No bony erosions were noticed (Figure 1). Ultrasonography of the left shoulder showed normal glenohumeral joint. Careful ultrasound review of the A–C joint revealed marked focal tenderness over the A–C joint and irregular A–C joint facets with capsular distension (Figure 2). Whole body scintigraphy was done with ^{99m}Tc-HMPAO radiolabeled autologous leukocytes to visualize the suspected infectious foci. It showed clear focal accumulation of radiolabeled leukocytes in the left A–C joint (Figure 3). Based on these diagnostic findings supported by clinical findings, the diagnosis of acute septic arthritis of A–C joint caused by *Staphylococcus aureus* was made.

The patient received intravenous antibiotic therapy for three weeks, followed by three weeks of peroral anti-

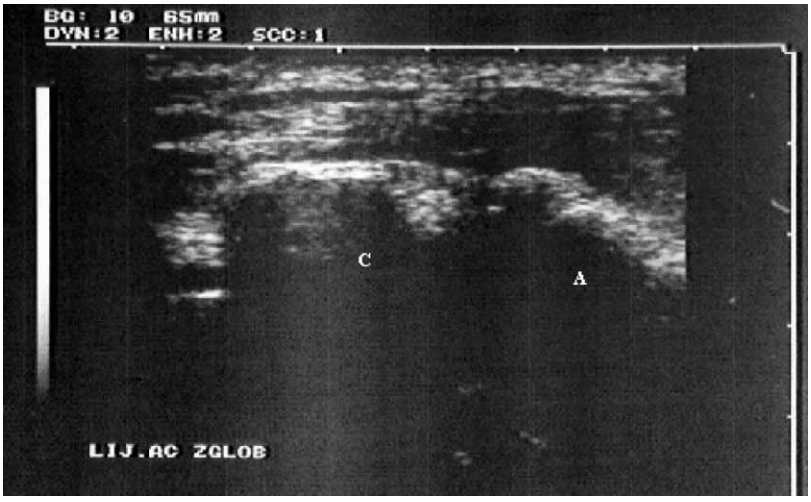

Fig. 2. Ultrasound of the left acromioclavicular (A–C) joint. Note irregular A–C joint facets, especially on the acromial side.

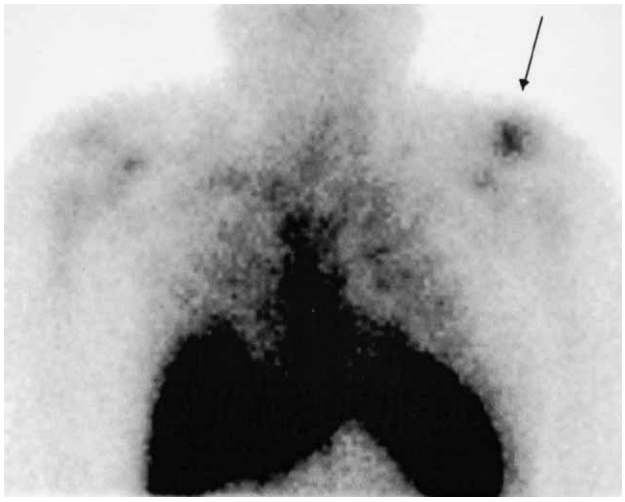

Fig. 3. Scintigraphy of the whole body with radiolabeled autologous leukocytes. Note clear focal accumulation of radiolabeled leukocytes in the left acromioclavicular (A–C) joint (arrow).

biotic therapy. The consulting orthopedic surgeon did not perform needle aspiration or drainage of the infected A–C joint because ultrasonography showed no major effusion of the joint space. Also the patient's condition improved and the accurate etiologic diagnosis was made on the basis of positive blood culture.

The patient responded favourably to antibiotic therapy. On the second day of therapy, the patient became afebrile, and on third day his white blood cell count was normal. On day 21, CRP level was normal. After six weeks of antibiotic therapy the patient was free from complaints and restriction in shoulder movements.

Discussion

Infections of the A–C joint caused by pyogenic microorganisms are very rare. Bacterial infections are usually monoarticular and involve the knee in 50% of cases. The hips and shoulders are the second most commonly involved joints[4,7]. Computer literature search identified only few publications in the last 20 years describing patients with septic A–C arthritis[8–10]. The A–C joint is rarely involved in septic processes, even in conditions such as intravenous drug abuse and immunodeficiency[7,9].

In this case of septic A–C arthritis there was nothing unusual either in the causative bacterial pathogen or in the underlying disease. *Staphylococcus aureus* is the most common bacterial pathogen causing septic arthritis, accounting for up to two-thirds of the organisms identified in blood or joint cultures of patients with septic arthritis. Also, diabetes mellitus is one of the most common concurrent medical diseases, being present in up to one-fourth of patients with septic arthritis[4–7].

History and physical examination are sometimes sufficient for the diagnosis of septic arthritis. The clinical features of A–C joint arthritis are very similar to those observed in shoulder joint arthritis, thus it is a challenge to recognize it and differentiate it from shoulder arthritis. Clinical examination reveals localized swelling and the point of maximal tenderness over the anterior aspect of the A–C joint. The range of movement in the shoulder joint is limited due to referred pain.

When the presence of an infective focus is suspected, it can be visualized accurately with radiolabeled autologous leukocytes. Scintigraphy of the whole body with radiolabeled autologous leukocytes is the gold standard of nuclear medicine techniques to image acute or chronic infection or inflammation, with a sensitivity on visualizing infectious or inflammatory foci exceeding 95%[12].

On detecting soft tissue lesions in arthritic A–C joints, magnetic resonance imaging (MRI) is superior to ultrasonography, however, the latter can also detect A–C joint changes reliably. Ultrasound is able to rule out joint inflammation when the ultrasonographic distance of the joint capsule from the bone is less than 3 mm [10,13]. In a setting of possible shoulder sepsis, a normal glenohumeral joint on ultrasound examination should prompt careful review of the A–C joint, and if capsular distension is present joint aspiration is advised.

Computed tomography (CT) is the best method to reveal bony surface changes, whereas plain radiography is least sensitive but quite specific[13].

In this case, the accurate diagnosis was obtained by clinical examination and positive blood culture, combined with the findings of diagnostic ultrasonography, scintigraphy and plain radiography of the A–C joint.

When septic arthritis is suspected antimicrobial therapy should be started immediately. Although the optimal length of therapy is not established, most authorities recommend antibiotic treatment for a minimum of 4 to 6 weeks. If effusion of the joint is present joint aspiration is advised. Surgery is indicated to drain abscesses or to debride infected bone and cartilage[14]. In this case the patient was treated for 6 weeks with antibiotics successfully. After 6 weeks of antibiotic therapy the patient was free from complaints and shoulder movement was normal.

In conclusion, septic arthritis of the A–C joint occurs seldom and can be differentiated from shoulder joint infection, by virtue of point tenderness over the A–C joint and findings on imaging studies, CT, MRI or ultrasound.

REFERENCES

1. KAANDORP, C. J., P. KRIJNEN, H. J. MOENS, J. D. HABBEMA, D. VAN SCHAARDENBURG, Arthritis Rheum., 40 (1997) 884. — 2. VINCENT, G. M., J. D. AMIRAULT, Clin. Orthop., 251 (1990) 241. — 3. YU, L. P., J. D. BRADLEY, S. T. HUGENBERG, K. D. BRANDT, Scand. J. Rheumatol., 21 (1992) 142. — 4. DUBOST, J. J., M. SOUBRIER, B. SAUVEZIE, Joint Bone Spine, 67 (2000) 11. — 5. HO, G. JR., Curr. Opin. Rheumatol., 4 (1992) 509. — 6. KAANDORP, C. J. E., D. VAN SCHAARDENBURG, P. KRIJNEN, J. D. F. HABBEMA, M. A. F. J. VAN DE LAAR, Arthritis Rheum., 38 (1995) 1819. — 7. STIMMLER, M. M., Postgrad. Med., 99 (1996) 127, 135. — 8. BLANKSTEIN, A., J. L. AMSALLEM, E. RUBINSTEIN, H. HOROSZOWSKI, I. FARIN, Arch. Orthop. Trauma Surg., 103 (1985) 417. — 9. ZIMMERMAN, B. III, A. D. ERICKSON, D. J. MIKOLICH, Arthritis Rheum., 32 (1989) 1175. — 10. WIDMAN, D. S., J. G. CRAIG, M. T. VAN HOLSBEECK, Skeletal Radiol., 30 (2001) 388. — 11. DUBOST, J. J., M. SOUBRIER, C. DE CHAMPS, J. M. RISTORI, J. L. BUSSIERE, B. SAUVEZIE, Ann. Rheum. Dis., 61 (2002) 267. — 12. BOERMAN, O. C., H. RENNEN, W. J. G. OYEN, F. H. M. CORSTENS, Semin. Nucl. Med., 31 (2001) 286. — 13. ALASAARELA, E., O. TERVONEN, R. TAKALO, S. LAHDE, I. SURAMO, J. Rheumatol., 24 (1997) 1959. — 14. BARZAGA, R. A., P. A. NOWAK, B. A. CUNHA, Heart Lung, 20 (1991) 692.

N. Laktašić-Žerjavić

University Department of Rheumatology and Rehabilitation, University Hospital Center »Zagreb«, Kišpatićeva 12, 10000 Zagreb, Croatia

PRIKAZ BOLESNIKA SA SEPTIČKIM ARTRITISOM AKROMIOKLAVIKULARNOG ZGLOBA I ŠEĆERNOM BOLESTI

SAŽETAK

U radu je prikazan 44-godišnji dijabetičar s utvrđenim septičkim artritisom lijevog akromioklavikularnog zgloba (A–C), uzrokovanog zlatnim stafilokokom. Bolesnik je zaprimljen u reumatološku Kliniku sa simptomima artritisa lijevog ramena i vrućicom. Laboratorijski utvrđena leukocitoza, izrazito ubrzana sedimentacija eritrocita i visoke vrijednosti C-reaktivnog proteina ukazale su na septički artritis. U kulturi krvi je izoliran zlatni stafilokok. Ultrazvučni pregled lijevog A–C zgloba i scintigrafija cijelog tijela vlastitim, 99 mTc obilježenim leukocitima potvrdile su dijagnozu septičkog artritisa lijevog A–C zgloba. Bolesnik je uspješno liječen šest tjedana sistemskom primjenom antibiotka. Infekcija A–C zgloba je neuobičajena. A–C zglob je rijetko zahvaćen septičkim procesom čak i u imunodeficijentnih i dijaliziranih bolesnika, kao i u intravenskih narkomana u kojih su češće prisutna neuobičajena sijela artritisa. Razlikuje se od artritisa ramenog zgloba kliničkim pregledom s točkom najjače boli u području A–C zgloba, kao i prikazom proširenja zglobne pukotine, izljeva u zglobu i erozija zglobnih tijela metodama oslikavanja.