CASE REPORT

Diagnostic challenges in acromioclavicular septic arthritis

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Accepted 19 May 2016

SUMMARY

A 69-year-old man with Klinefelter's syndrome presented with a painful shoulder and staphylococcal sepsis. He received intravenous antibiotics while investigations were performed to locate the source of infection. MRI demonstrated infection in the acromioclavicular joint (ACJ). The patient clinically improved and a further 5 weeks of oral antibiotics were given. He remained asymptomatic at 2-year follow-up. Although ACJ septic arthritis is rare, independent of immune-competent status, a high index of suspicion is essential for prompt diagnosis. The condition presents additional diagnostic challenges due to unfamiliarity, the challenges of interpreting imaging, desire for radiological guided arthrocentesis and low volume aspirates. Overcoming these pitfalls is essential to avoid significant morbidity and mortality.

BACKGROUND

Prompt diagnosis of small joint septic arthritis requires a high index of clinical suspicion, to avoid significant morbidity and mortality. In particular, acromioclavicular joint (ACJ) septic arthritis is a rare entity that can potentially be mistaken for glenohumeral joint septic arthritis given its similar presentation in the early stage, higher prevalence and anatomical proximity. Both diagnoses can present with signs and symptoms common to septic arthritis of any joint, such as joint heat, swelling, pain and overlying skin erythema. A careful examination may differentiate the affected joint by detecting maximal tenderness and erythema overlying the ACJ. A case of ACJ septic arthritis is presented with discussion of the diagnostic challenges in the case, as well as a wider examination of the diagnostic difficulties in small joint septic arthritis.

Of the 20 cases of ACJ septic arthritis reported in the literature, most occurred in patients with impaired or compromised immune systems, such as from: diabetes mellitus,^{1–3} multiple myeloma,² intravenous drug use,⁴ prior joint disease,⁴ a recent history of intra-articular injections,⁴ a remote history of surgery,⁴ AIDS⁵ and chronic steroid use.⁶ The patient presented did not have any of these issues. A literature search found a single case in a patient with no immunodeficiency.² The patient presented consented to a case report with exclusion of any personal identifiable information.

CASE PRESENTATION

A 69-year-old right-handed man presented to accident and emergency department, with acutely worsening severe left shoulder pain. The patient

reported progressive swelling, redness and a reduced range of movement, with associated fevers and rigors for 4 days. Two days before admission, the patient had reported to his general practitioner (GP) of shoulder pain noticed after lifting furniture. The GP diagnosed a muscular-skeletal injury and advised rest and expectant management. The patient described no concurrent infection, and no recent hospitalisations/procedures. Systemic enquiry provided no additional information.

The medical history was significant for metabolic syndrome (hypertension, hypercholesterolaemia, obesity and impaired glucose tolerance), Klinefelter's syndrome and mild renal insufficiency. The patient denied alcohol use, smoking or drug abuse.

On admission, the patient was feverish (38.0°C), tachycardic (125 bpm) and normotensive. Physical examination demonstrated a hot, erythematous and diffusely tender shoulder. Active and passive range of movement was limited due to pain, most notably in forward flexion and abduction. There were superficial scratches on the patient's arms, caused by a pet puppy.

INVESTIGATIONS

A working diagnosis of glenohumeral septic arthritis prompted an ultrasound guided arthrocentesis by the emergency physicians, but no fluid was obtained. Left shoulder radiographs revealed no abnormality (figure 1). The initial white cell count was $7.3 \times 10^9/L$ ($4.0\text{--}11.0 \times 10^9/L$), C reactive protein (CRP) 285 nmol/L (<5 nmol/L), bilirubin 31 $\mu\text{mol/L}$ (<26 $\mu\text{mol/L}$) and haemoglobin was 143 g/L (140–170 g/L).

Figure 1 Anteroposterior radiograph of the left shoulder, demonstrating no abnormalities. Plain radiographs, although not useful for septic arthritis diagnosis, are routinely performed to exclude underlying osteomyelitis.

To cite: Williams M. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2016-216034

DIFFERENTIAL DIAGNOSIS

At presentation, the differential diagnosis included a crystalopathy, synovitis, cellulitis, acute osteomyelitis and acute rheumatic fever. Glenohumeral septic arthritis was the primary diagnosis requiring exclusion in the first instance, given its potential for long-term morbidity.

TREATMENT

The patient was consented for and underwent an arthroscopic washout of the left shoulder within 9 hours of admission. A single posterior porthole, with the patient in the lateral position, allowed wash out with 3 L. Visualisation of the subacromial space demonstrated mild inflammation, an intact cuff and neither pus nor abscess collection.

Joint fluid microscopy demonstrated neither crystals nor bacterial growth. Postwashout, intravenous flucloxacillin (2 g, four times a day) was started empirically. The patient's vital signs normalised within 12 hours of starting the antibiotic. Shoulder ultrasound demonstrated no soft tissue collection, no glenohumeral effusion and no significant ACJ effusion. Mildly deranged admission liver function tests prompted an abdominal ultrasound scan, which was unremarkable. In addition, a transthoracic echocardiogram obtained suboptimal images (technically difficult due to obesity) but demonstrated no obvious evidence of bacterial endocarditis. Blood cultures taken on admission isolated a *Staphylococcus*, later confirmed to be *Staphylococcus aureus*, which was methicillin sensitive. The microbiology consultant recommended the addition of intravenous clindamycin (900 mg, four times a day), which the patient received for 6 days.

Clinically, the shoulder symptoms improved, reflected by improving inflammatory markers. Re-examination of the shoulder elicited tenderness localising to the ACJ and pain on cross-body adduction. This prompted an MRI scan 7 days after admission, which demonstrated increased signal centred around the ACJ, with some evidence of oedema extending to the acromion (figure 2A, B).

OUTCOME AND FOLLOW-UP

Given the patient's clinical improvement, further surgery for irrigation and debridement of the ACJ was not required. The patient was discharged after 8 days in hospital, with 5 weeks of oral flucloxacillin (1 g, four times a day) and weekly follow-up. One week after discharge, there were no restrictions of shoulder movements and the CRP was 8 nmol/L. Follow-up at 2 years found that the patient's shoulder had remained asymptomatic.

DISCUSSION

Septic arthritis most commonly affects the knees, hips and shoulders, although, as this case demonstrates, nearly any synovial joint can become infected. The ACJ is a gliding synovial joint and is therefore vulnerable to septic arthritis. In the absence of trauma, most cases of septic arthritis arise from haematological seeding to joints with a rich blood supply.⁷ This explains the higher incidence of seeding to the hip, knee and glenohumeral joints. Conversely, given the comparatively poorer blood supply to the ACJ, it is not surprising that it is rarely implicated. The causative organisms in ACJ septic arthritis, as is true with the more frequently affected joints, include *S. aureus* and *Streptococcus*.^{1–3} In addition, there are individual reports of both *Cryptococcus neoformans* and *Haemophilus parainfluenzae* causing ACJ septic arthritis.^{6,8} In this regard, failure to diagnose and identify the causative organism would lead to rapid

Figure 2 (A) Transverse T2-weighted MRI scan demonstrating oedema extending into the acromion. (B) Axial T2-weighted MRI scan demonstrating the acromial joint. Findings of synovial enhancement, perisynovial oedema and joint effusions support a diagnosis of anteroposterior septic arthritis.⁷

cartilage destruction, with long-term morbidity and even the potential for mortality.⁷

Small joint septic arthritis reported in the literature typically occurs in patients with impaired immune systems.^{3–6,9} Although a case has been reported in a patient with no immunodeficiency, this is not the norm. Klinefelter's syndrome is a sex chromosomal disorder in males, resulting in infertility. This state of androgen deficiency is considered to alter the immune system and have various effects on the body pathology. Indeed, the higher incidence of autoimmune disease in Klinefelter's has been the subject of research and debate.¹⁰ The association of Klinefelter's with metabolic syndrome, and therefore impaired glucose tolerance, is well documented in the literature.¹¹ However, independent of its association with metabolic syndrome, it is uncertain if Klinefelter's syndrome diagnosis confers reduced immunity.

A relatively straightforward diagnostic pathway for suspected large joint septic arthritis exists, with emphasis on timely arthrocentesis. Costantino *et al*¹² described the use of ultrasound for arthrocentesis of glenohumeral joints by emergency physicians to establish the diagnosis more readily. On the other hand, time to a small joint septic arthritis diagnosis can be delayed. In ACJ, septic arthritis radiographs are rarely useful while ultrasonography may detect an ACJ effusion. Widman *et al*⁹ further concluded that in patients with a suspected septic shoulder and a

normal glenohumeral joint ultrasound, careful review of the ACJ is needed. One limitation to using ultrasound in diagnosis is lack of familiarity of findings, given the disease rarity. MRI can be used when seeking a diagnosis of ACJ septic arthritis with findings of synovial enhancement, perisynovial oedema and joint effusions supporting a clinical diagnosis.¹³ However, for any suspected septic joint, no radiological technique is sufficiently sensitive or specific to be diagnostically definitive. Furthermore, significant delays can arise if specialist image guidance is employed to obtain small joint aspirations, as compared with simple large joint arthrocentesis employing landmark techniques.

As alluded to, prompt arthrocentesis and therefore microscopic analysis and culture of synovial fluid are fundamental in the diagnosis of septic arthritis. Microscopic analysis can confirm the presence of micro-organisms or crystals, however, Gram staining is positive in only 50% of cases.¹⁴ Indeed, the sensitivity of detecting causative organisms is often misunderstood, leading to false reassurance and delayed diagnosis. Furthermore, small joint aspirates will obtain low volumes,

likely further affecting microscopic Gram staining sensitivity. This difficulty is unlikely to be overcome by using a PCR assay, as this does not offer any advantage over bacterial culture in the microbiological diagnosis of staphylococcal or streptococcal joint infection.¹⁵ Once a diagnosis of small or large joint septic arthritis is confirmed, treatment principles are the same, namely, antibiotic treatment and, usually, joint drainage. Given the diagnostic uncertainties associated with imaging and microscopic synovial analysis, a clinical decision to treat a small joint septic arthritis may be based exclusively on the clinical examination and biochemical tests.

Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

- Hammel JM, Kwon N. Septic arthritis of the acromioclavicular joint. *J Emerg Med* 2005;29:425–7.
- Chirag AS, Ropiak CR, Bosco Ili JA, et al. Septic arthritis of the acromioclavicular joint—a report of four cases. *Bull NYU Hosp Jt Dis* 2007;65:308–11.
- Laktasić-Zerjavić N, Babić-Naglić D, Curković B, et al. Septic acromioclavicular arthritis in a patient with diabetes mellitus. *Coll Antropol* 2005;29:743–6.
- Bossert M, Prati C, Bertolini E, et al. Septic arthritis of the acromioclavicular joint. *Joint Bone Spine* 2010;77:466–9.
- Zimmerman B III, Erickson AD, Mikolich DJ. Septic acromioclavicular and osteomyelitis in a patient with acquired immune deficiency syndrome. *Arthritis Rheum* 1989;32:1175–8.
- Adams R, McDonald M. Cryptococcal arthritis of the acromio-clavicular joint. *N C Med J* 1984;45:23–4.
- Carpenter CR, Schuur JD, Everett WW, et al. Evidence-based diagnostics: adult septic arthritis. *Acad Emerg Med* 2011;18:781–96.
- Carey TW, Jackson K, Roue R, et al. Acromioclavicular septic arthritis: a case report of a novel pathogen. *Am J Orthop* 2010;39:134–6.
- Widman DS, Craig JG, van Holsbeeck MT. Sonographic detection, evaluation and aspiration of infected acromioclavicular joints. *Skeletal Radiol* 2001;30:388–92.
- Koçar IH, Yesilova Z, Ozata M, et al. The effect of testosterone replacement treatment on immunological features of patients with Klinefelter's syndrome. *Clin Exp Immunol* 2000;121:448–52.
- Groth KA, Skakkebaek A, Host C, et al. Clinical review: Klinefelter syndrome—a clinical update. *J Clin Endocrinol Metab* 2013;98:20–30.
- Costantino TG, Roemer B, Leber EH. Septic arthritis and bursitis: emergency ultrasound can facilitate diagnosis. *J Emerg Med* 2007;32:295–7.
- Karchevsky M, Schweitzer ME, Morrison WB, et al. MRI findings of septic arthritis and associated osteomyelitis in adults. *AJR Am J Roentgenol* 2004;182:119–22.
- Weston VC, Jones AC, Bradbury N, et al. Clinical features and outcome of septic arthritis in a single UK health district 1982–1991. *Ann Rheum Dis* 1999;58:214–19.
- Jalava J, Skurnik M, Toivanen A, et al. Bacterial PCR in the diagnosis of joint infection. *Ann Rheum Dis* 2001;60:287–9.

Learning points

- ▶ The case outlined presents the rare clinical entity of acromioclavicular joint (ACJ) septic arthritis, found in a higher frequency but not exclusively in patients with immunodeficiency.
- ▶ Prompt recognition and identification of the causative organism is essential for effective treatment.
- ▶ An ACJ septic arthritis diagnosis, as with other small joint septic arthritis, can be challenging and delayed for several reasons, such as: their low incidence meaning septic arthritis fails to be a differential diagnosis for a presenting arthropathy; radiological imaging is unlikely to be diagnostic and is often difficult to interpret; the desirability for radiological guided arthrocentesis leading to time delays; and low volume joint aspirates affecting Gram stain and culture yields.
- ▶ All physicians must remain vigilant to the potential of septic arthritis in any synovial joint.

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