Glenohumeral Joint Sepsis after Joint Injection through a New Tattoo

Richard Becker[1*], Sean Haight[2]

1. Department of Radiology Naval Hospital Pensacola, FL, USA

2. Aviation Medicine Clinic Training Wing 5, Milton, FL, USA

* **Correspondence:** Richard Becker, Department of Radiology Naval Hospital Pensacola, 6000 West Hwy 98, Pensacola, FL, 32507 USA (*richard.l.becker.mil@mail.mil*)

Radiology Case. 2017 Nov; 11(11):28-35 :: DOI: 10.3941/jrcr.v11i11.3197

ABSTRACT

We report a case of Propionibacterium acnes septic arthritis of a shoulder following two joint access procedures. Methodical investigation of these two procedures was performed using the Joint Commission root cause analysis matrix, which involved examination of protocols, interviews with technologists and physicians, and inspection of the procedure rooms and equipment. No procedural error or other causal factor for infection was found other than the injection through a five day-old tattoo during the first procedure. We could find no other reported cases of Propionibacterium acnes seeding a joint via an arthrogram needle. This may be an under-reported phenomenon or could be a unique case. In either instance, the occurrence bears notice by anyone performing joint access procedures.

CASE REPORT

CASE REPORT

A 24 year-old male right handed former collegiate throwing athlete presented to his primary care physician with a two year history of right shoulder pain ranging in severity from 1/10 to 5/10. His physician suspected a labral tear and an MRI arthrogram was ordered. Five days prior to his procedure the patient had a tattoo placed that covered his entire upper chest and the anterior aspect of both shoulders. He neglected to mention the recent placement of this tattoo to the radiologist performing the procedure and there were no visible indicators of inflammation. He denied any post procedure pain and had a normal MRI arthrogram (see Figure 1). He followed up with orthopedics five days later and reported 4/10 pain without constitutional symptoms and an exam remarkable for a positive Hawkins sign. He was treated with a subacromial steroid injection with relief of symptoms and released to physical therapy.

Over the following month of physical therapy the patient continued to report 3/10 pain and decreasing range of motion (ROM). At his next orthopedic follow up he reported 3-4/10 pain that increased with anterior and lateral movement and decreased ability to raise his right arm secondary to pain. Vitals signs were normal and he denied constitutional symptoms. Radiographs were obtained that showed only mild inferior subluxation of the humeral head (Figure 2). The differential diagnosis was expanded to include bicipital tendonitis, Parsonage-Turner syndrome, adhesive capsulitis, or aseptic inflammation from the previous arthrogram. Bicipital groove and glenohumeral joint steroid/anesthetic injections were performed with minimal relief. He was released back to physical therapy for another month.

At his next orthopedic follow up, he reported continued pain and decreased shoulder function. Muscle atrophy of the shoulder girdle was noted. A subacromial steroid injection was performed with improvement in pain and symptoms. An MRI C-spine and repeat shoulder MRI were ordered. The MRI C-spine was normal but the shoulder MRI revealed joint effusion, superior rotator cuff tear, articular cartilage loss and possible labral tear, intra-articular biceps tendon rupture and extensive edema in the supraspinatus and infraspinatus muscles as well as focal subcortical edema in the superior postero-lateral humeral head (See Figure 3). The radiologist initially postulated the findings could represent post-traumatic

injuries from anterior-inferior dislocation/relocation. Joint infection was suspected after consultation between the radiologist and orthopedic surgeon revealed no known history of trauma and on noting the additional finding of significant articular cartilage loss.

Labs showed erythrocyte sedimentation rate of 12 mm/hr (normal 0-22), C-reactive protein of 0.55 mg/dL (normal < 3.0), and white blood cell count 6,500 (normal 4,000-11,000). Right shoulder radiographs revealed irregularity of the subchondral bone of the humeral head and severe joint space narrowing, new from the prior (See Figure 4). Right shoulder joint aspiration was performed without immediate return of fluid, requiring normal saline infusion and aspiration to acquire a serosanguinous sample. The gram stain was negative and patient was taken to the operating room the same day for washout. Operative findings included severe chondrolysis, rotator cuff fraying and biceps tendon rupture. No pus was encountered and bone biopsy and culture were obtained. The patient was admitted and an infectious disease consultation was requested. The patient was started on empiric intravenous (IV) vancomycin and ciprofloxacin and the next day the antibiotics were switched to IV daptomycin and oral ciprofloxacin and he was discharged with outpatient IV antibiotics therapy and continued physical therapy. He was evaluated by neurology with electromyography performed and diagnosed with brachial plexopathy with differential consideration of multiple peripheral neuropathies.

Joint aspirate culture was initially negative, and tissue cultures were positive for light *Staphylococcus aureus*. Given the slowly progressive clinical course which is atypical for *Staphylococcus aureus* joint sepsis, the orthopedic surgeon requested that specimens be kept longer to culture for *Propionibacterium acnes* (*P. acnes*) which he knew could cause an indolent infection. The final aspirate and tissue cultures were positive for *P. acnes* with confirmation from Infectious Disease as the most likely etiological agent, and Staphylococcus aureus most likely a contaminant. Final histopathology of the bone and soft tissue biopsies from the Joint Pathology Center yielded lymphoplasmacytic infiltrate without necrosis indicating chronic inflammation of non-specific etiology. Electron microscopy of the samples was negative for the presence of gadolinium. After completing antibiotic therapy, the patient's post-infectious osteoarthritis progressed, but with improved muscular function and unfortunately had to undergo hemi-arthroplasty within a year.

DISCUSSION

Etiology & Demographics:

Glenohumeral joint sepsis is an acknowledged, uncommon, complication of intra-articular injections [1,2]. In our case a young male suffered a right shoulder joint infection that caused severe early onset degenerative joint disease. Septic arthritis predominantly affects the weight bearing joints of the knees and hips (approximately 60 percent) [2] with only about 3 percent of cases affecting the shoulder joints [3]. It is very uncommon for septic arthritis of the shoulder to occur after an MRI with gadolinium arthrogram [4]. *P. acnes* infections are most commonly associated with acne in teenagers and young adults, in surgical wound infections and in invasive deep-seated infections, usually in the setting of an implantable device. It is an uncommon cause of joint infections outside of surgical procedures and shows a predilection for the shoulder joint over other joints [5]. *P. acnes* inhabit sebaceous glands and follicles in skin where antiseptic solutions do not penetrate well [6,7]. *P. acnes* is a member of the Actinobacteria phylum of microorganisms, and is a gram-positive anaerobic bacillus which is nonmotile, slow-growing and of low virulence. This bacterium is capable of adherence and biofilm formation which explains its association with prosthesis infections. Given the low virulence of this bacterium, the infections they cause are usually indolent and clinical manifestations may be delayed for months or even years [8] and are not characterized by classic infection or inflammatory symptoms. These infections are more common in men than in women [9], possibly due to men having more sebaceous glands and hair follicles. Neither perioperative topical antisepsis nor intravenous cefazolin is effective in eliminating *P. acnes* colonization [10]. These organisms can be difficult to identify because of their slow growth, especially in samples that contain other common, readily recovered organisms and they can easily be missed if cultures are discarded after the typical three to five days of incubation.

Clinical & Imaging findings:

Synovial joints are a hospitable environment for bacteria and can be seeded either by direct inoculation/contiguous spread or by hematogenous route. Risk factors such as recent invasive procedures, indwelling hardware, infection of another organ system, chronic arthritis, or intravenous drug use may be noted. Early infections may present with local and systemic inflammatory signs and symptoms and progress to joint erythema, warmth, pain and stiffness. Decreased ROM and pain with passive ROM are often present. Usually a clinical diagnosis is made by history, exam and arthrocentesis. Imaging studies may be important in unusual cases where clinical indicators are uncertain or if synovial fluid cannot be obtained. Radiographs may be normal at the early stages of the disease. Joint effusion may be evident, manifesting as joint space widening or subluxation of the humeral head if involving the shoulder. As the disease progresses, there will be periarticular osteopenia, narrowing of the joint space secondary to cartilage destruction, loss of subchondral bone on both sides of the joint and, if left untreated, reactive juxta-articular sclerosis and ankylosis. Though not used in our case, ultrasound can be a useful imaging modality for evaluating joint effusions, particularly in children. Typically it will demonstrate a joint effusion with echogenic debris. Color Doppler may show increased peri-synovial blood flow. Computed tomography (CT) was not used in our case, but is more sensitive than plain films for the detection of early bone destruction and effusion and there may be a fat-fluid level in the absence of trauma. MRI is most sensitive for early cartilage damage, showing thinning and/or pitting of and hyperintense T2 signal within the articular cartilage. Loss of T1 weighted signal in the bone marrow and marrow enhancement indicate concomitant osteomyelitis. Perisynovial edema and joint effusion will be most apparent on T2 images. Synovial enhancement and the presence of a joint effusion

have been reported to have the highest correlation with the clinical diagnosis of a septic joint [11].

Treatment & Prognosis:

Left untreated, classic septic arthritis can result in permanent joint damage within 48 hours. However, *P. acnes* can cause a slow, indolent infection and cases of hardware infection have been reported more than two years after surgery. Because this bacterium can form a biofilm the usual treatment is removal of any hardware [12, 13] and surgical debridement of the joint. Antibiotic therapy is then guided by antimicrobial susceptibility and tailored to the specific organism. *P. acnes* is usually very susceptible to penicillins, clindamycin, cepalosporins, and carbapenems, and most laboratories do not routinely perform susceptibility testing for this pathogen [14]. Infectious disease consultation is highly recommended if *P. acnes* is suspected or isolated in culture.

Differential Diagnosis:

The shoulder radiographic findings of inferior subluxation of the humeral head could be due to a joint effusion, large labral tear, acute rotator cuff tear or rotator cuff muscle/nerve paralysis.

The second shoulder MRI revealed joint effusion, superior rotator cuff tear, cartilage loss and possible labral tear, intra-articular biceps tendon rupture and extensive edema in the supraspinatus and infraspinatus muscles. The differential diagnosis includes:

Trauma

Normally a history of severe trauma including a dislocation event would be provided, which would help to explain this degree of injury. In the absence of any history of trauma this diagnosis was excluded.

Parsonage-Turner

Also known as brachial neuritis or neuralgic amyotrophy, Parsonage-Turner Syndrome is a peripheral nerve disorder characterized by rapid onset of pain in the shoulder and arm, thought to be the result of an abnormal immune-mediated response to insult such as trauma or infection, but can be idiopathic. Routine chest and spine films may show altered posture secondary to muscular weakness or reveal secondary bone injury. MRI may be normal initially, but will eventually show increased T2 signal in the associated muscles due to denervation, followed by streaks of high T1 signal and reduced muscle mass from fatty atrophy. This was likely a co-existing disease process in our patient.

Charcot Arthropathy

Charcot arthropathy is an uncommon neuro-arthropathy seen among diabetics of uncertain pathogenesis that is probably due to a combination of mechanical and vascular factors resulting from peripheral neuropathy and metabolic bone abnormalities. This commonly presents with sudden onset of unilateral warmth, redness, and edema over the joint, and in the shoulder should raise concern for syringomyelia [15]. Early plain films may be nonspecific, showing only soft tissue edema, loss of joint space, or osteopenia. In later stages, bone resorption may predominate with possible fragmentation or total resorption of the humeral head. MRI will show decreased T1 signal in the periarticular bone marrow and increased T2 signal due to edema, as well as patchy hyperenhancement and hypoenhancement in the periarticular soft tissues due to neurogenic edema and necrosis. Diagnosis is made based on history, physical and radiographic findings and infection should be excluded.

Rheumatoid Arthritis

Rheumatoid arthritis is an autoimmune inflammatory polyarthropathy that can have a waxing and waning or progressive course. It causes synovial hyperplasia that leads to painful joint swelling and erodes the subchondral bone at the joint margin. The aberrant inflammatory process also causes diffuse articular cartilage loss. Patients have warm, swollen and tender joints, usually affecting the small joints in the distal extremities, but eventually more proximal larger joints. A variety of extra-articular manifestations of the disease may be present. Early radiographs are usually without osseous abnormality, but with progression, periarticular demineralization and marginal erosions with axial joint space narrowing are evident. MRI will show synovial thickening and T2 hyperintensity which may mimic a joint effusion, but enhancing post-contrast in contra-distinction to the peripheral synovial enhancement of a septic joint with joint effusion. Articular cartilage loss will be evident. Subchondral marrow signal changes may also be present.

Septic Joint

Clinical and imaging findings of joint sepsis were detailed previously.

Summary

This young patient has suffered a severe deterioration of the right shoulder joint due to an infection that was likely the result of an inoculation from one of the two joint access procedures that occurred at the time of the first normal MRI arthrogram and before the markedly abnormal second MRI without contrast. Methodical investigation of these two procedures using the Joint Commission root cause analysis matrix, which involved examination of protocols, interviews with technologists and physicians, and inspection of the procedure rooms and equipment revealed no procedural error or other causal factor for infection other than the injection through a five-day old tattoo during the first procedure. The patient's clinical worsening during the period between the two procedures also implicates the first procedure. There was no illness or any other skin injury or procedure to suggest a hematogenous route of infection. Of note, a stylet needle was used for the procedure which should minimize the volume of tissue translocation into the joint. Because a thorough review of the literature did not reveal a previously reported case of a tattoo-related joint infection, and no procedural error was discovered, the infection could only be theoretically linked to the tattoo. This patient's clinical course was confounded by the slow progression and concomitant development of a brachial plexopathy (likely Parsonage-Turner Syndrome), which delayed the diagnosis of septic joint.

Arthrography is considered a safe procedure with an established incidence of infection around 0.003% [16];

however, infection can cause severe permanent arthritis, osteomyelitis or even secondary necrotizing fasciitis [17] any of which can be devastating. A literature review from 1966 to 2006 showed that infection was the most common complication of musculoskeletal injections [18]. In a series of 19 patients treated arthroscopically for shoulder sepsis, 15 had the joint accessed with a needle prior to the infection [2]. *Staphylococcus aureus* is the leading cause of septic arthritis, accounting for more than 60% of cases [19], while *P. acnes* is an uncommon causal organism and it has been associated with shoulder surgery rather than joint injections and is commonly cultured from skin [20]. Given that the infection occurred in the absence of surgery, and in consideration of the patient's clinical timeline, it is believed that the infection was a result of the arthrogram shoulder injection and the presence of skin and subcutaneous inflammation related to the recent tattoo placement probably increased the risk of transmission of skin flora into the joint.

TEACHING POINT

An arthrogram through a new tattoo may increase the risk of joint sepsis, which could have an indolent course and not be suspected clinically. The diagnosis should be considered on follow-up imaging where a joint effusion, cartilage loss and subchondral marrow signal changes are present, and Parsonage-Turner Syndrome though uncommon is a possible co-morbidity.

REFERENCES

1. Tjoumakaris F, Anakwenze O, Kancherla V, Pulos N. Neuralgia Amyotophy (Parsonage-Turner Syndrome). J Am Acad Orthop Surg. 2012;20:443-449. PMID: 22751163

2. Mook WR, Klement MR, Green CL, Hazen KC, Garrigues GE. The Incidence of Propionibacterium acnes in Open Shoulder Surgery: A Controlled Diagnostic Study. J Bone Joint Surg Am. 2015 Jun; 97(12):957-63. PMID: 86085528

3. Mathews CJ, Kingsley G, Field M, et al. Management of septic arthritis: a systematic review. Ann Rheum Dis. 2007;66(4):440-445. PMID: 18508984

4. Klinger HM, Baums MK, Frechie S, Nusselt T, Spahn G, Steckel H. Septic arthritis of the shoulder joint: an analysis of management and outcome. Acta Orthop Belg. 2010 Oct;76(5):598-603. PMID: 21138213

5. Busfield BT. Glenohumeral Joint Sepsis After Magnetic Resonance Imaging Arthrogram. Am J Orthop. 2012 Jun;41(6): 277-278. PMID: 22837992

6. Mook WR, Klement MR, Green CL, Hazen KC, Garrigues GE. The Incidence of Propionibacterium acnes in Open Shoulder Surgery: A Controlled Diagnostic Study. J Bone Joint Surg Am. 2015 Jun; 97(12):957-63. PMID: 86085528

7. Carricajo A, Nuti C, Aubert E, et.al. Propionibacterium acnes contamination in lumbar disc surgery. J Hosp Infect. 2007;66(3):275. PMID: 17573158

8. McLorinan GC, Glenn JV, McMullan MG, Patrick S. Propionibacterium acnes wound contamination at the time of spinal surgery. Clin Orthop Relat Res. 2005 Aug; (437):67-73. PMID: 16056028

9. Kanafani ZA, Sexton DJ, Pien BC, Varkey J, Basmania C, Kaye KS. Postoperative joint infections due to Propionibacterium species: a case-control study. Clin Infect Dis. 2009;49(7):1083. PMID: 19725786

10. Levy PY, Fenollar F, Stein A, et. al. Propionibacterium acnes postoperative shoulder arthritis: an emerging clinical entity. Clin Infect Dis. 2008;46(12):1884. PMID: 18462110

11. Koh CK, Marsh JP, Drinkovi? D, Walker CG, Poon PC. Propionibacterium acnes in primary shoulder arthroplasty: rates of colonization, patient risk factors, and efficacy of perioperative prophylaxis. J Shoulder Elbow Surg. 2016 May;25(5):846-52. PMID: 26704361

12. Lutz MF, Berthelot P, Fresard A, Cazorla C, Carricajo A, Vautrin AC, Fessy MH, Lucht F. Arthorplastic and osteosynthetic infections due to Propionibacterium acnes: a retrospective study of 52 cases, 1995-2002. Eur J Clin Microbiol Infec Dis. 2005;24(11):739. PMID: 16328558

13. El Rafei A, Desimone DC, Sohail MR, Desimone CV, Steckelberg JM, Wilson WR, Baddour LM. Cardiovascular Implantable Electronic Device Infections due to Propionibacterium Species. Pacing Clin Electrophysiol. 2016;39(6):522. Epub 2016 Apr 22. PMID: 26970081

14. Roberts SA, Shore KP, Paviour SD, Holland D, Morris AJ. Antimicrobial susceptibility of anaerobic bacteria in New Zealand: 1999-2003. J Antimicrob Chemother. 2006;57(5):992. PMID: 16507560

15. Cullen AB, Ofluoglu O, Donthineni R. Neuropathic Arthropathy of the Shoulder (Charcot Shoulder). MedGenMed. 2005;7(1):29. PMID: 16369334

16. Newberg AH, Munn CS, Robbins AH. Complications of arthrography. Radiology. 1985;155:605-606. PMID: 4001360

17. Birkinshaw R, O'Donnell J, Sammy I. Necrotising fasciitis as a complication of steroid injection. J Accid Emerg Med. 1997; 14(1):52-54. PMID: 9023629

18. Cheng J, Abdi S. Complications of joint, tendon, and muscle injections. Tech Reg Anesth Pain Manag. 2007;11(3):141-147. PMID: 18591992

19. Dubost JJ, Soubrier M, De Champs C, et al. No changes in the distribution of organisms responsible for septic arthritis over a 20 year period. Ann Rheum Dis. 2002;61(3):267-269. PMID: 11830437

20. Lutz MF, Berthelot P, Fresard A, et al. Arthoplastic and osteosynthetic infections due to Propioribacterium acnes: a retrospective study of 52 cases, 1995-2002. Eur J Clin Microbiol Infect Dis. 2005 Nov; 24(11):739-44. PMID: 16238558.

FIGURES

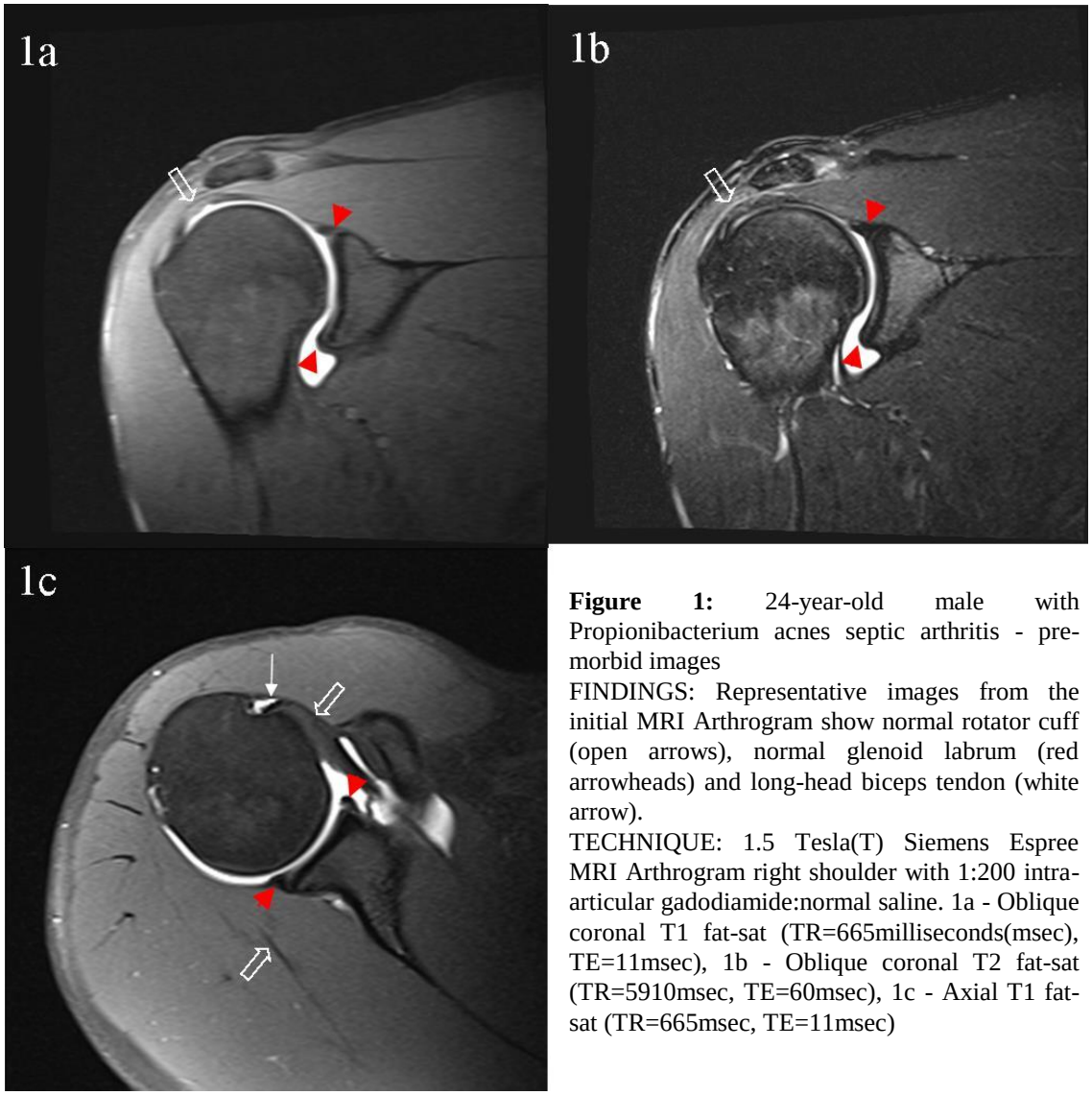

Figure 1: 24-year-old male with Propionibacterium acnes septic arthritis - pre-morbid images
FINDINGS: Representative images from the initial MRI Arthrogram show normal rotator cuff (open arrows), normal glenoid labrum (red arrowheads) and long-head biceps tendon (white arrow).
TECHNIQUE: 1.5 Tesla(T) Siemens Espree MRI Arthrogram right shoulder with 1:200 intra-articular gadodiamide:normal saline. 1a - Oblique coronal T1 fat-sat (TR=665milliseconds(msec), TE=11msec), 1b - Oblique coronal T2 fat-sat (TR=5910msec, TE=60msec), 1c - Axial T1 fat-sat (TR=665msec, TE=11msec)

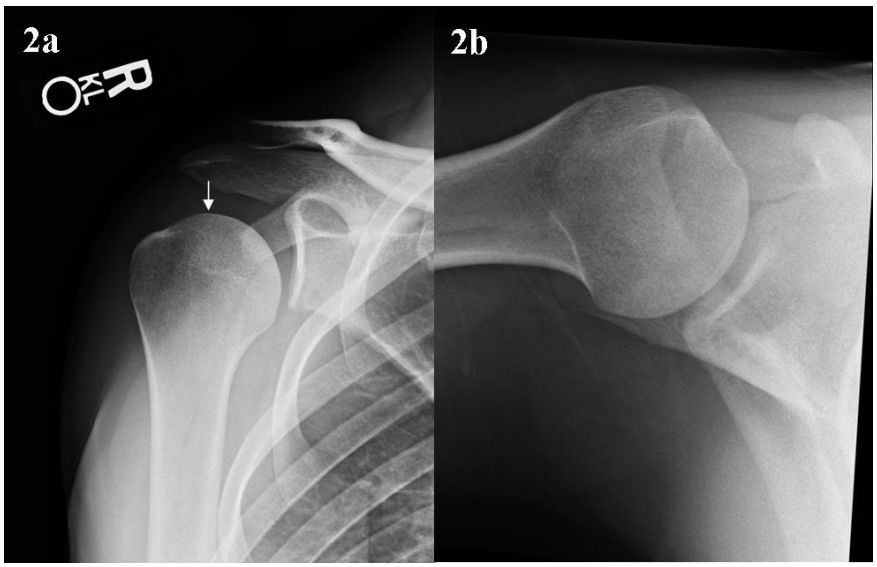

Figure 2: 24-year-old male with Propionibacterium acnes septic arthritis - One month after arthrogram.
FINDINGS: Inferior subluxation of the humeral head (2a - arrow) without dislocation (2b). No abnormality of the subchondral bone.
TECHNIQUE: Radiographs right shoulder with Grashey view (2a) and axillary view (2b).

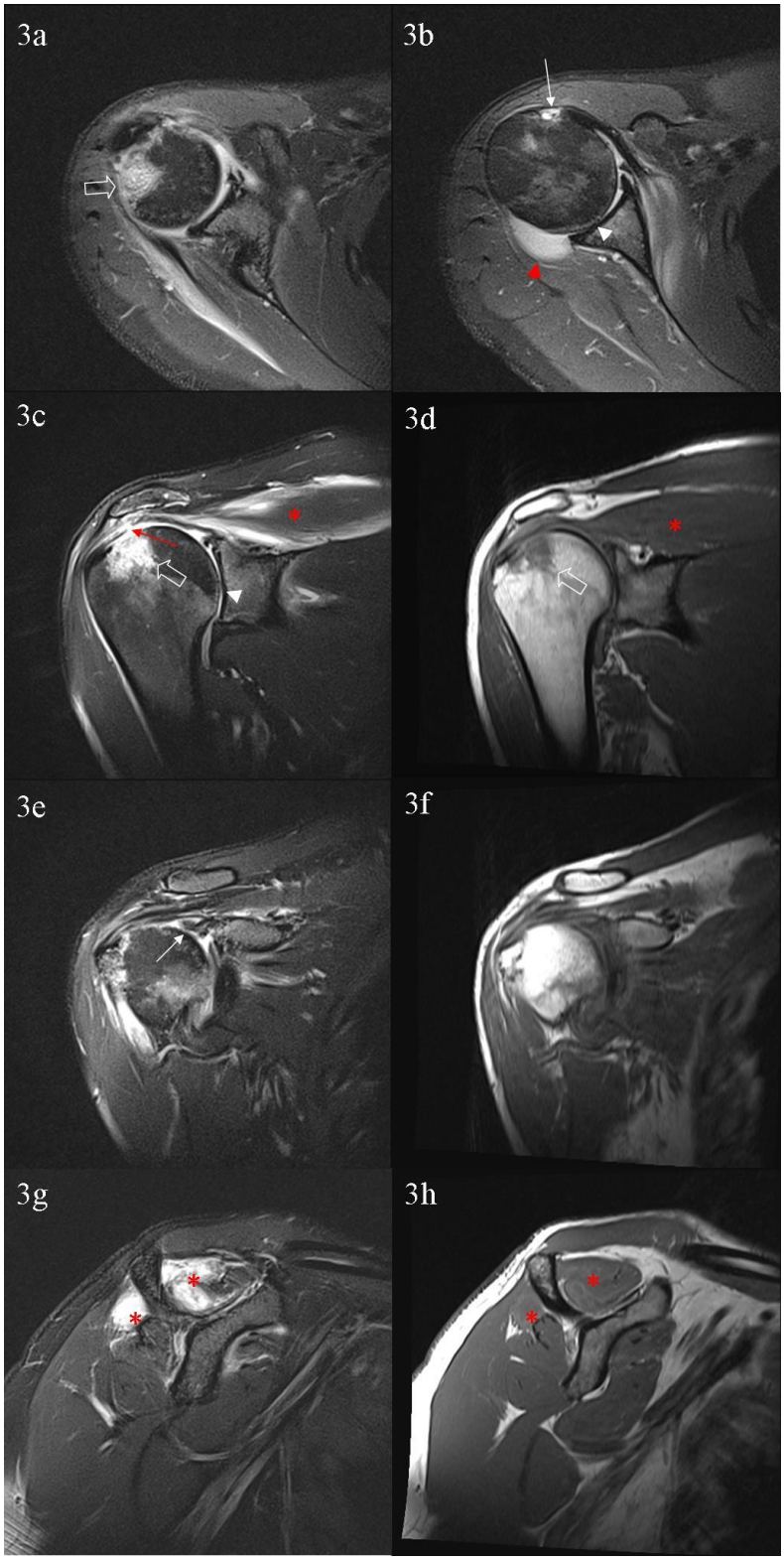

Figure 3: 24-year-old male with Propionibacterium acnes septic arthritis - two months after arthrogram
FINDINGS: There is a joint effusion (3b - red arrowhead), focal area of T2 signal hyperintensity and T1 hypointensity in the subchondral marrow of the superior posterolateral humeral head (3a, 3c, 3d - open arrows), extensive T2 hyperintensity and T1 hypointensity in the supraspinatus and infraspinatus muscles (3c, 3d, 3g, 3h - red asterisks) with probable full-thickness supraspinatus tendon tear (3c - red arrow) and long head biceps tendon rupture (3b, 3e - white arrows). Constellation of findings could be post-traumatic, possibly due to transient dislocation, but absence of inferior glenoid contusion and presence of articular cartilage loss (3b, 3c - white arrowheads) indicates a different etiology.
TECHNIQUE: 1.5T Siemens Espree MRI right shoulder without contrast. 3a/3b - Axial proton density fat-sat (TR=2300msec, TE=35msec), 3c/3e - Oblique coronal T2 fat-sat (TR=6700msec, TE=69msec), 3d/3f - Oblique coronal T1 (TR=667msec, TE=15msec), 3g - Oblique sagittal T2 fat-sat (TR=6030msec, TE=69msec), 3h - Oblique sagittal T1 (TR=1210msec, TE=87msec).

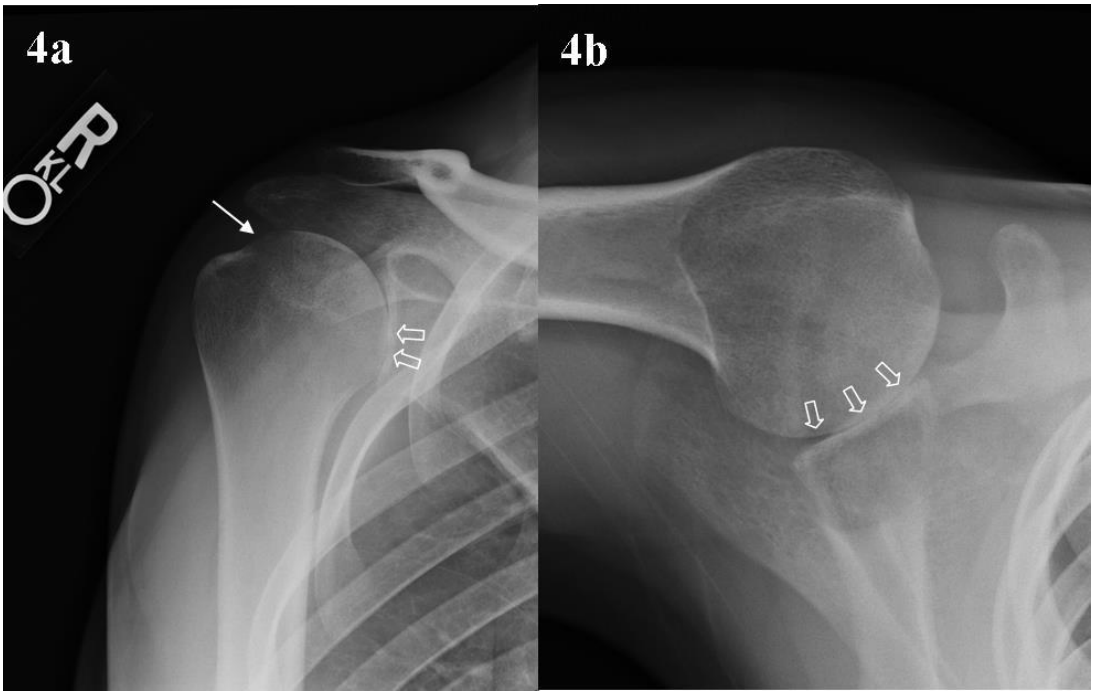

Figure 4: 24-year-old male with Propionibacterium acnes septic arthritis - two months after arthrogram
FINDINGS: Subchondral lucency at the superolateral humeral head (4a - arrow), subtle irregularity of the subchondral bone plate and marked joint space narrowing (4a, 4b - open arrows).
TECHNIQUE: Radiographs right shoulder with Grashey view (4a) and axillary view (4b).

Etiology	*Propionibacterium acnes* is a relatively common cause of shoulder infection following orthopedic surgery, but this is the only case we can find of it being caused by a radiologic procedure.
Incidence	Overall joint sepsis following arthrogram is on the order of 0.03%, with only about 3% of these involving the shoulder. Incidence of joint sepsis related to a tattoo is unknown.
Gender ratio	More common in men due to men likely due to more sebaceous glands and hair follicles around the shoulder.
Age predilection	No known predilection
Risk factors	Shoulder surgery
Treatment	Surgical debridement and appropriate antibiotic therapy.
Prognosis	Loss of joint function depending on degree of necessary debridement, virulence of infection, and length of infection.

Table 1: Summary table for Septic Arthritis secondary to Propionibacterium acne infection.

	History and Physical Exam	Radiograph	Magnetic Resonance Imaging (MRI)
Parsonage-Turner	• Acute onset severe shoulder or arm pain followed by weakness and numbness • Progresses from shoulder to arm • Affected muscles become weak and atrophied and potentially paralyzed • May have paralysis without pain, and sometimes just pain without paralysis	• May reveal an inciting or secondary osseous injury	• Intramuscular edema early • Intramuscular fatty infiltration and atrophy • Involves one or more muscles of the shoulder • Often supraspinatus and infraspinatus
Charcot arthropathy	• Acute onset monoarticular warmth, redness and edema • Painless • When not complicated by infection the inflammatory markers are often negative	• Early stage- mild soft tissue swelling, loss of joint space or osteopenia • Later stage- Bone resorption, Loss of joint space, Scattered chunks of bone, Fracture, Worsening soft tissue edema	• Periarticular bone marrow edema • Patchy periarticular soft tissue hyperenhancement and hypoenhancement • In shoulder, should evaluate for syringomyelia
Septic Joint	• Acute onset monoarticular warmth, redness and edema. • Restricted painful passive ROM • Majority febrile • Risk factors of recent invasive procedure, indwelling hardware, chronic arthritis, infection or intravenous drug use	• Usually normal early • Signs of joint effusion • Peri-articular osteopenia • Narrowing of the joint spaces due to cartilage loss • Destruction of the subchondral bone on both sides of the joint	• Joint effusion • Synovial edema and enhancement • Subchondral marrow edema • Articular cartilage loss with high T2 signal • Loss of T1 signal within subchondral bone and bone marrow enhancement
Trauma	• History of dislocation/ relocation • Poor active ROM with pain • Rotator cuff weakness/pain • Apprehension	• Any fracture or dislocation • Posterior superior humeral head notch (Hill Sachs) • Inferior glenoid insufficiency (Osseous Bankart)	• Rotator cuff tendon or biceps tear without tendinosis • Labral tear • Bone contusions or impaction fractures • Osteochondral injury • Joint effusion
Rheumatoid Arthritis	• Polyarticular pain/swelling • Warm, tender joints • Tenosynovitis • Extra-articular manifestations • Waxing/waning or progressive	• Early- normal • Later- Periarticular osteopenia • Marginal erosions	• Synovial thickening, edema and enhancement • Mimics joint effusion • Articular cartilage thinning • Marginal erosions

Table 2: Differential diagnosis table for Septic Arthritis (secondary to Propionibacterium acne infection).

ABBREVIATIONS

CT = Computed Tomography
Fat-sat = Fat saturation
IV = Intravenous
MRI = Magnetic resonance imaging
msec = millisecond
P. acnes = Propionibacterium acnes
ROM = Range of motion
T = Tesla

KEYWORDS

septic arthritis; arthrogram; arthrography; joint injection; tattoo; shoulder septic arthritis; Propionibacterium acnes; Parsonage-Turner; brachial neuritis; brachial plexopathy; neuralgic amyotrophy

ACKNOWLEDGEMENTS

The authors would like to acknowledge the leadership of Naval Hospital Pensacola who encouraged this case to be published.