

A Patient Having Calcific Tendinitis with Intraosseal Penetration, Differential Diagnosis with Osteomyelitis-Malignancy of the Proximal Humerus

¹ Mete ÖZER^a, ¹ Mahmut Kürşat ÖZŞAHİN^a, ¹ Mahmut Görkem GÜRÇİNAR^a, ¹ Arın CELAYİR^a,
¹ Nuri AYDIN^a

^aDepartment of Orthopedics and Traumatology, İstanbul University-Cerrahpaşa, Cerrahpaşa Faculty of Medicine, İstanbul, Türkiye

ABSTRACT Calcific tendinitis is a tendinopathy characterized by the formation of calcium deposits within or around the rotator cuff tendons in the shoulder, leading to severe pain. It most commonly occurs in the supraspinatus and infraspinatus tendons due to the accumulation of calcium hydroxyapatite crystals. Diagnostic tools such as radiography, magnetic resonance image, and ultrasound are useful for establishing the diagnosis. Intraosseous penetration is a rare condition. Cases of intraosseous penetration of calcific tendinitis have been reported in the insertions of the pectoralis major and gluteus maximus in the humerus and femur. The region most commonly affected by intraosseous penetration is the greater tuberosity of the humerus. In cases of intraosseous penetration, differential diagnosis should consider malignancies and osteomyelitis.

Keywords: Calcific tendinitis; intraosseous penetration; osteomyelitis; malignancy

Calcific tendinitis is a tendinopathy characterized by the formation of calcium deposits within or around the rotator cuff tendons in the shoulder, leading to severe pain. It most commonly occurs in the supraspinatus and infraspinatus tendons due to the accumulation of calcium hydroxyapatite crystals.¹

The etiology is not fully understood, but it is considered a risk factor for diabetes and gout. Patients with calcific tendinitis have been reported to have other endocrinological diseases. Adhesive capsulitis and concomitant rotator cuff tears can accompany the disease. It affects individuals between the ages of 40 and 65, with a higher prevalence in women. Conservative treatment can be chosen as the initial approach, and surgical treatment is indicated for cases unresponsive to conservative treatment. Acute cases generally respond better to conservative treatment, while chronic cases often require surgical intervention.²

Patients typically present with shoulder pain, restricted range of motion, and decreased joint mobility. The most significant limitation in motion is usually seen in abduction and flexion because the supraspinatus tendon is the most commonly affected. In some cases, night pain may also be present.^{1,2}

Tools such as radiography, magnetic resonance image (MRI), and ultrasound are useful for establishing the diagnosis. Idiopathic skeletal hyperostosis or degenerative tendinopathies are included in the differential diagnosis. The region around the greater tuberosity of the humerus is the most frequently affected area.³⁻⁵

Intraosseous penetration is a rare condition. Cases of intraosseous penetration of calcific tendinitis have been reported in the insertions of the pectoralis major and gluteus maximus, as well as in the humerus and femur. The region most commonly af-

Correspondence: Arın CELAYİR

Department of Orthopedics and Traumatology, İstanbul University-Cerrahpaşa, Cerrahpaşa Faculty of Medicine, İstanbul, Türkiye

E-mail: arin.celayir@iuc.edu.tr



Peer review under responsibility of Türkiye Klinikleri Journal of Case Reports.

Received: 10 Sep 2023

Accepted: 18 Dec 2023

Available online: 20 Dec 2023

2147-9291 / Copyright © 2023 by Türkiye Klinikleri. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

ected by intraosseous penetration is the greater tuberosity of the humerus.^{5,6}

Radiological findings that may be encountered in calcific tendinitis with bone penetration include medullary involvement, periosteal reaction, and more commonly, cortical erosion. In addition to these findings, MRI may show a focal rim that mimics widespread bone marrow edema or infection, which can resemble malignancy or infection. History, physical examination findings, imaging techniques, and histopathological examinations play an important role in the differential diagnosis of malignancy or infection.

Conservative treatment includes local injection, ultrasound-guided barbotage or extracorporeal shock-wave therapy, and range of motion exercises. There is no consensus on surgical treatment, but options include removal of calcific deposits, acromioplasty, and repair of tendon defects.⁷

CASE REPORT

A 28-year-old male patient complained of left shoulder pain, particularly at night, for the past few months. He did not have any underlying medical conditions or regular medication use. He had no history of surgical intervention. Patient consent was taken before any treatment procedure.

Physical examination revealed that both shoulder levels were the same and symmetric. There was no swelling or redness in the shoulder area. Joint range of motion was nearly full in both shoulders, but abduction and flexion of the left shoulder were slightly painful. The Hawkins and Neer tests for the left shoulder were positive, while the Jobe test was negative. Palpation revealed slight tenderness in the superolateral aspect of the left shoulder. The acromioclavicular joint was painless.

The patient’s blood tests were normal, with no signs of infection. C-reactive protein, sedimentation rate, and white blood cell count were within normal ranges. Laboratory investigations showed no pathological findings.

Radiological examinations revealed a bone lesion in the proximal left humerus at the level of the

supraspinatus insertion, with bone marrow edema around the greater tuberosity, cortical erosion, and soft tissue extension (Figure 1). The lesion was reported to potentially indicate osteomyelitis or malignancy (Figure 2, Figure 3). The available imaging was evaluated, and an intralesional biopsy procedure was planned.

An intralesional biopsy was performed, and the biopsy specimens were sent for histopathological ex-

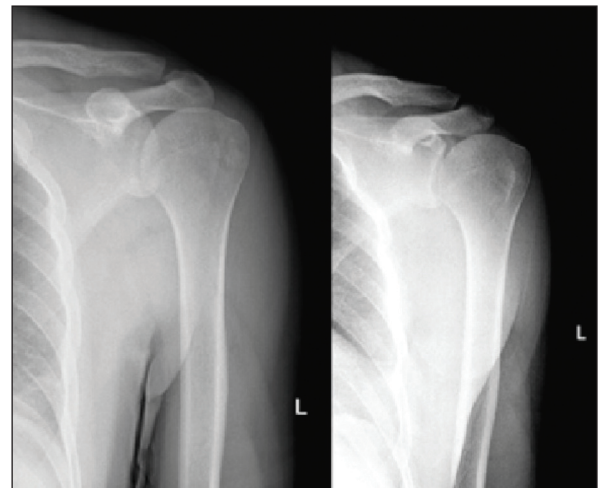


FIGURE 1: Preoperative shoulder radiographs.

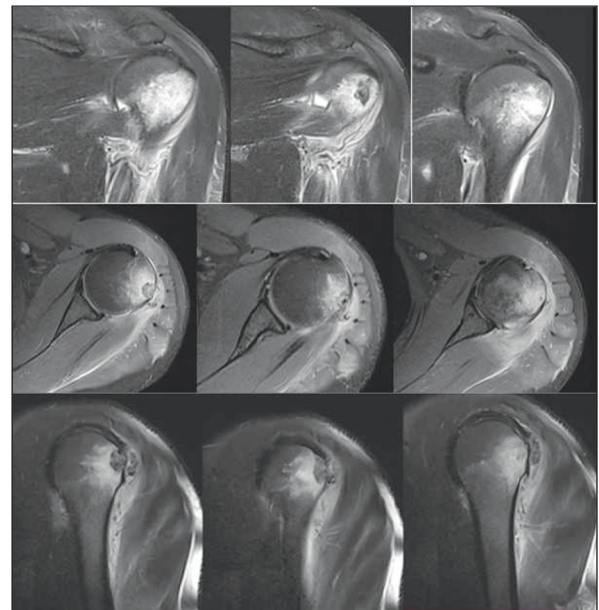


FIGURE 2: Preoperative magnetic resonance imaging. A lesion with intraosseous penetration is observed in the superolateral aspect of the humerus. The dimensions were measured approximately 13×11 mm. Perilesional bone marrow edema is observed.

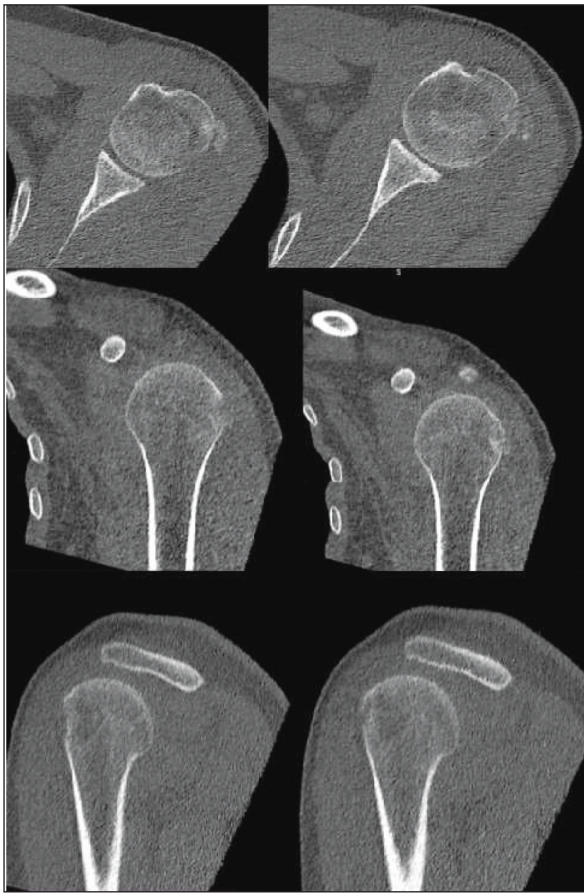


FIGURE 3: Preoperative computed tomography images, showing cortical erosion at the level of the greater tuberosity.

amination (Figure 4). Passive elbow exercises and pendulum exercises were initiated on the evening of the surgery, and the patient was discharged one day later.

During follow-up, the patient did not experience any complications related to the surgical site. There were no complaints of pain or restricted movement.

Despite not performing calcific deposit extraction, the patient’s pain complaints resolved in the postoperative period.

The histopathology report confirmed calcific tendinitis with intraosseous penetration, and no evidence of malignancy or osteomyelitis was found. MRI performed at the 3-month postoperative follow-up confirmed the lesion as calcific tendinitis originating from the attachment site of the supraspinatus muscle.

DISCUSSION

It has been reported that conservative treatment can yield successful results in up to 80% of patients with acute calcific tendinitis who are younger than 40 years old and do not have endocrine disorders. In a similar case with our patient but at an older age, successful outcomes were achieved with conservative treatment in a patient diagnosed with intraosseous calcific tendinitis following evaluation with radiography, MRI, and whole-body bone scintigraphy. The improvement of the patient’s symptoms, despite the absence of surgical interventions such as calcific deposit extraction, bursectomy, or acromioplasty, was likely due to conservative treatment involving analgesic use and range of motion exercises.

The course of calcific tendinitis is described as a three-stage process: pre-calcific, calcific, and resorptive phases. The pre-calcific phase involves fibrocartilaginous metaplasia, followed by the accumulation of calcium deposits in the calcific phase, and it progresses with neoangiogenesis and phagocytosis in the resorptive phase.⁸

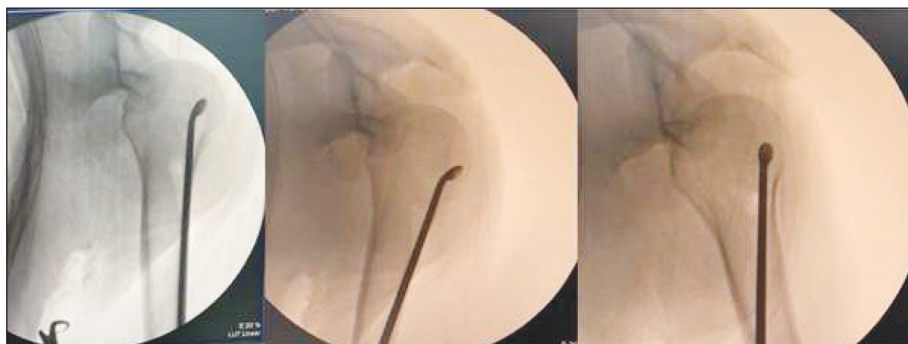


FIGURE 4: Intraoperative fluoroscopy images.

During the investigation of a sclerotic lesion observed at the proximal aspect of the greater tuberosity in a case presentation, calcific tendinitis was noted in the supraspinatus tendon in a radiograph taken 6 months prior.⁹ Since it is likely in the resorptive phase, the disappearance of the calcific deposit in the supraspinatus tendon could be considered a normal occurrence. This highlights the importance of understanding the phases and radiological findings of the disease.

There is no consensus on the choice of surgical treatment. Complete removal of the calcific deposit and the absence of tuberosity osteolysis have been reported to be associated with better functional outcomes.¹⁰ Even in the presence or absence of bone penetration in calcific tendinitis, the removal of the calcific deposit has been reported to be associated with good clinical outcomes, and repair is recommended in the presence of tendon defects.^{11,12}

Some authors argue that the accurate evaluation of radiological findings of the disease can be effective in the choice of treatment. The combination of findings such as erosion of the greater tuberosity in MRI, presence of edema around the lesion, “snowstorm” appearance in computed tomography, and “comet tail” appearance in radiography have been reported

to support the consideration of intraosseous penetration for correct diagnosis and to prevent aggressive surgical interventions.^{13,14}

Source of Finance

During this study, no financial or spiritual support was received neither from any pharmaceutical company that has a direct connection with the research subject, nor from a company that provides or produces medical instruments and materials which may negatively affect the evaluation process of this study.

Conflict of Interest

No conflicts of interest between the authors and / or family members of the scientific and medical committee members or members of the potential conflicts of interest, counseling, expertise, working conditions, share holding and similar situations in any firm.

Authorship Contributions

Idea/Concept: Nuri Aydın, Mahmut Kürşat Özşahin; **Design:** Nuri Aydın, Mahmut Kürşat Özşahin; **Control/Supervision:** Nuri Aydın; **Data Collection and/or Processing:** Arın Celayir, Mete Özer, Mahmut Görkem Gürçınar; **Analysis and/or Interpretation:** Mete Özer, Mahmut Görkem Gürçınar, Arın Celayir; **Literature Review:** Mete Özer, Arın Celayir; **Writing the Article:** Arın Celayir, Mete Özer; **Critical Review:** Nuri Aydın, Mahmut Kürşat Özşahin; **References and Findings:** Nuri Aydın, Mahmut Kürşat Özşahin.

REFERENCES

1. DE Carli A, Pulcinelli F, Rose GD, Pitino D, Ferretti A. Calcific tendinitis of the shoulder. *Joints*. 2014;2(3):130-6. [[Crossref](#)] [[PubMed](#)] [[PMC](#)]
2. Kim MS, Kim IW, Lee S, Shin SJ. Diagnosis and treatment of calcific tendinitis of the shoulder. *Clin Shoulder Elb*. 2020;23(4):210-6. [[Crossref](#)] [[PubMed](#)] [[PMC](#)]
3. Albano D, Coppola A, Gitto S, Rapisarda S, Messina C, Sconfienza LM. Imaging of calcific tendinopathy around the shoulder: usual and unusual presentations and common pitfalls. *Radiol Med*. 2021;126(4):608-19. [[Crossref](#)] [[PubMed](#)] [[PMC](#)]
4. Draghi F, Cocco G, Lomoro P, Bortolotto C, Schiavone C. Non-rotator cuff calcific tendinopathy: ultrasonographic diagnosis and treatment. *J Ultrasound*. 2020;23(3):301-15. [[Crossref](#)] [[PubMed](#)] [[PMC](#)]
5. Malghem J, Omoumi P, Lecouvet F, Vande Berg B. Intraosseous migration of tendinous calcifications: cortical erosions, subcortical migration and extensive intramedullary diffusion, a SIMS series. *Skeletal Radiol*. 2015;44(10):1403-12. [[Crossref](#)] [[PubMed](#)]
6. Flemming DJ, Murphey MD, Shekitka KM, Temple HT, Jelinek JJ, Kransdorf MJ. Osseous involvement in calcific tendinitis: a retrospective review of 50 cases. *AJR Am J Roentgenol*. 2003;181(4):965-72. [[Crossref](#)] [[PubMed](#)]
7. Verstraelen FU, Fievez E, Janssen L, Morrenhof W. Surgery for calcifying tendinitis of the shoulder: A systematic review. *World J Orthop*. 2017;8(5):424-30. [[Crossref](#)] [[PubMed](#)] [[PMC](#)]
8. Uthoff HK, Loehr JW. Calcific tendinopathy of the rotator cuff: pathogenesis, diagnosis, and management. *J Am Acad Orthop Surg*. 1997;5(4):183-91. [[Crossref](#)] [[PubMed](#)]
9. Martin S, Rapariz JM. Intraosseous calcium migration in calcifying tendinitis: a rare cause of single sclerotic injury in the humeral head (2010: 2b). *Eur Radiol*. 2010;20(5):1284-6. [[Crossref](#)] [[PubMed](#)]
10. Porcellini G, Paladini P, Campi F, Pegreffo F. Osteolytic lesion of greater tuberosity in calcific tendinitis of the shoulder. *J Shoulder Elbow Surg*. 2009;18(2):210-5. [[Crossref](#)] [[PubMed](#)]

11. Caliskan E, Eren I, Aslan L, Koyuncu O, Seyahi A, Demirhan M. Intraosseous calcific tendinitis of the rotator cuff yields similar outcomes to those of intratendinous lesions despite worse preoperative scores. *Knee Surg Sports Traumatol Arthrosc.* 2022;30(7):2485-91. [[Crossref](#)] [[PubMed](#)]
12. Seyahi A, Demirhan M. Arthroscopic removal of intraosseous and intratendinous deposits in calcifying tendinitis of the rotator cuff. *Arthroscopy.* 2009;25(6):590-6. [[Crossref](#)] [[PubMed](#)]
13. Pereira BP, Chang EY, Resnick DL, Pathria MN. Intramuscular migration of calcium hydroxyapatite crystal deposits involving the rotator cuff tendons of the shoulder: report of 11 patients. *Skeletal Radiol.* 2016;45(1):97-103. [[Crossref](#)] [[PubMed](#)]
14. Marinetti A, Sessa M, Falzone A, Della Sala SW. Intraosseous migration of tendinous calcifications: two case reports. *Skeletal Radiol.* 2018;47(1):131-6. [[Crossref](#)] [[PubMed](#)]