



TGCT of the shoulder – a case series and literature review

Josefa Stadelmeier¹ · Filip Bijeljac¹ · Alexander Klein¹ · Felix Winden¹ · Paul Reidler² · Hans Roland Dürr¹

Received: 10 October 2025 / Accepted: 11 February 2026
© The Author(s) 2026

Abstract

Introduction Tenosynovial giant cell tumor (TGCT), also known as pigmented villonodular synovitis (PVNS), involving the shoulder is extremely rare and can present with a challenging clinical course. Due to the complex anatomy of the shoulder, both diagnosis and treatment are demanding. This study aims to evaluate the diagnostic and therapeutic management of shoulder TGCT based on a case series and a review of the literature.

Materials and methods Between 2005 and 2021, four patients (2 females, 2 males) with shoulder TGCT (1 localized, 3 diffuse) underwent surgical treatment at our institution. The minimum follow-up was 39 months (range: 39–233 months). Functional outcomes were assessed using the Disabilities of the Arm, Shoulder and Hand Questionnaire (DASH), the Oxford Shoulder Score (OSS), and the Short Form 36 Health Survey (SF-36). Additionally, a literature review of 65 studies comprising 108 patients was performed.

Results Treatments consisted of arthroscopic and open resections, tendon repair, adjuvant radiosynoviorthesis, and radiotherapy, as appropriate. All patients were recurrence-free at the last follow-up, except for one with stable residual disease after radiotherapy. The mean interval from symptom onset to diagnosis was 23.6 ± 28.1 months. In the literature cohort, the mean patient age was 50.3 ± 19.7 years, with a nearly equal gender distribution. Diffuse TGCT was more common (66.1%) than nodular TGCT (33.8%). Treatment was primarily surgery, arthroscopic (53.2%) or open (42.9%), with adjuvant therapies applied in 9.3% of cases. After a mean follow-up of 37.6 ± 36.2 months in 67 cases, diffuse in 44.8%, nodular in 19.4% (no data regarding TGCT-type in 35.8%) the reported recurrence rate was 10.6% and 4.5% remained with residual disease.

Conclusions TGCT of the shoulder remains a rare and complex condition requiring individualized treatment strategies. Arthroscopic and open resections are the mainstays of therapy, while the role of adjuvant treatments should be carefully considered. Given the risk of recurrence, follow-up is essential. Further studies are needed to establish standardized treatment protocols and evaluate long-term outcomes.

Level of evidence Level IV (Case series with no comparison group).

Keywords Tenosynovial giant cell tumor · Pigmented villonodular synovitis · Shoulder · Open resection · Arthroscopy · Recurrence

✉ Josefa Stadelmeier
josefa.stadelmeier@med.uni-muenchen.de
Filip Bijeljac
Filip.Bijeljac@med.uni-muenchen.de
Alexander Klein
Alexander.Klein@med.uni-muenchen.de
Felix Winden
Felix.Winden@med.uni-muenchen.de

Paul Reidler
Paul.Reidler@med.uni-muenchen.de
Hans Roland Dürr
Hans_Roland.Duerr@med.uni-muenchen.de

¹ Orthopaedic Oncology, Department of Orthopaedics and Trauma Surgery, University Hospital, LMU Munich, Campus Großhadern, Munich, Germany

² Department of Radiology, University Hospital LMU Munich, Campus Großhadern, Munich, Germany

Introduction

Background

Tenosynovial giant cell tumor (TGCT), formerly known as Pigmented Villonodular Synovitis (PVNS), is a rare, benign proliferative disorder of the synovium, the tendon sheath and the bursa [1]. Typically, TGCT presents as a monoarticular process and predominantly affects the knee joint, hip, ankle, shoulder, elbow as hands and feet. Two types of TGCT are recognized: the nodular-type TGCT (N-TGCT) and the diffuse-type TGCT (D-TGCT) [2].

Epidemiology

Epidemiological data on TGCT are limited and varied, making comparisons challenging. In nationwide studies, the incidence rates of N-TGCT ranged around 11 to 34 per million. For D-TGCT, incidence rates were reported between 5 and 8.4 per million [3–5].

There is inconsistency in the data concerning the gender distribution of pigmented villonodular synovitis. While some studies indicate that males and females are affected at similar rates, others suggest a slight predisposition in females, specifically in cases of N-TGCT [3, 6]. TGCT predominantly presents between 35 and 50 years of age [5, 6].

Pathogenesis

The pathogenesis of TGCT remains unclear, with theories ranging from chronic inflammatory processes to neoplastic etiology [7, 8]. Last is at present the commonly accepted pathogenesis, hence the name was changed from PVNS to TGCT [9, 10].

The most frequently suggested associations include chronic recurring trauma, intra-articular hemorrhage, and persistent inflammation [11, 12]. West et al. hypothesized that a chromosomal translocation involving the 1p13 locus may contribute to the underlying pathogenesis [13]. Colony-stimulating factor-1 (CSF-1) overexpression promotes the formation of aberrant cellular aggregates, contributing to synovial soft tissue hyperplasia and thus defining the clinical and radiological disease [14].

Imaging and clinical presentation

TGCT is one of the few soft-tissue conditions with a nearly pathognomonic magnetic resonance imaging (MRI) appearance, characterized by synovial hypertrophy, low signal intensity and a “blooming artifact” [15–17]. This artifact, caused by iron in hemosiderin deposits, appears as notable signal loss on both T1- and T2-weighted MRI [18]. While

N-TGCT presents most commonly with a painless swollen joint and MRI shows a well-circumscribed soft tissue mass [19], D-TGCT presents with a painful swollen joint, often with reduced mobility. On MRI an ill-defined soft-tissue mass is visualized [15]. In some cases impingement is the leading symptom [20].

Clinical, histological and molecular profile

Clinically, N-TGCT typically presents as a well-circumscribed, nodular mass causing painless swelling or mechanical symptoms (e.g., locking, impingement), with slow growth and low risk of local invasion. In contrast, D-TGCT manifests as an infiltrative synovial process with painful swelling, effusions, stiffness, and progressive joint destruction over time, often requiring repeated interventions if untreated. N-TGCT rarely progresses to diffuse form, while D-TGCT may extend intra- and extra-articularly [5].

N-TGCT typically shows mononuclear histiocyte-like cells, multinucleate giant cells, lymphocytes, and hemosiderin deposits, with moderate mitotic activity [15, 21]. D-TGCT displays a more infiltrative pattern with higher mitotic rates, a variable presence of giant cells, and occasional sarcoma-like features [22].

Molecularly, both forms are clonal neoplasms driven by CSF1 overexpression, often due to genomic rearrangements such as COL6A3::CSF1. This overexpression promotes tumor growth via paracrine recruitment of inflammatory cells and forms the basis for targeted therapy with CSF1R inhibitors [23–26].

Treatment strategies

In both N-TGCT and D-TGCT, the decision to pursue active treatment should carefully weigh the potential for cure against the typically benign nature of TGCT, its sometimes slow progression, the risk of local recurrence (LR) after complete resection, and the possibility of surgery-related complications [5]. Active surveillance should be considered as approach for asymptomatic patients [5].

The standard of care for symptomatic TGCT is surgical intervention, provided it can be performed with acceptable morbidity [5]. For N-TGCT, marginal excision is sufficient, whereas D-TGCT requires extensive synovectomy to achieve local disease control and optimize functional outcomes. Surgical access may be achieved via an open anterior approach or arthroscopically, depending on tumor extent and anatomical considerations [27]. In cases of extensive rotator cuff pathology or advanced joint destruction, rotator cuff repair or shoulder arthroplasty in combination with complete synovectomy may provide effective symptom relief and functional restoration.

Adjuvant therapies

Current evidence is insufficient to support radiotherapy as a standard treatment for TGCT in neoadjuvant, adjuvant, or recurrent settings. Radiation therapy can be in very selected cases an effective supplementary treatment, particularly where complete synovectomy is not feasible. It helps to prevent recurrences and preserves joint function with minimal toxicity [28, 29]. Other adjuvant therapies may include radiosynoviorthesis (RSO) and cryotherapy [30]. Both are at the moment considered as not beneficial.

Systemic treatment

In current trials, CSF1R pathway inhibitors such as Pexidartinib, Imatinib, Nilotinib, Vimseltinib, and monoclonal antibodies such as Emactuzumab have demonstrated effectiveness in TGCT, leading to significant tumor reduction, symptom relief, improved function, and sustained disease control [31–35]. Systemic treatment with CSF1R inhibitors may be considered when symptomatic TGCT cannot be surgically removed with acceptable morbidity and symptoms remain difficult to manage, particularly if disease progression is likely to impair quality of life (QoL) [5].

Study

In 1999, only 25 cases had been described in the English literature, managed by open synovectomy in all cases [36]. In 2017, Noailles et al. provided a literature review including 6 studies with 46 patients [36–42]. In this study, we present a series of four patients with TGCT of the shoulder, illustrating the heterogeneous clinical presentations, diagnostic challenges, and therapeutic outcomes associated with this rare entity. Additionally, we conducted a comprehensive review of the existing literature on shoulder TGCT, including 65 studies comprising 108 patients. Through the analysis of our cases and the available evidence, we aim to expand the limited knowledge on shoulder TGCT by providing insights into its clinical and radiological features, while underscoring the importance of timely diagnosis and appropriate management to optimize patient outcomes.

Materials and methods

Patient selection

Between March 2005 and May 2021, 4 patients (2 female, 2 male; mean age 38.8 ± 21.4 years) were diagnosed with TGCT of the shoulder—three with diffuse-type and one with localized-type (localized-type will be called nodular-type throughout the text). The right (dominant) shoulder was affected in three cases, the left in one. All patients underwent preoperative MRI and intraoperative histopathological confirmation by our Department of Pathology. Symptoms had been present for an average of 32.5 months (range: 12–60 months) before diagnosis. Surgical treatment was performed in all cases, including one rotator cuff repair. Two patients received adjuvant RSO. The mean follow-up period was 90 ± 95 months, with a minimum of 39 months. Patient characteristics are summarized in Table 1.

Surgical technique

Patients 1, 2, and 4 were positioned in a 30° beach-chair setup, while patient 3 was placed supine with the upper body elevated 30°; all underwent general anesthesia. The arm was positioned freely in all cases. Patient 1 initially underwent diagnostic shoulder arthroscopy with supraspinatus tendon repair and bony decompression, followed by open N-TGCT resection via the deltopectoral approach. Patients 2, 3, and 4 underwent primary open resections of D-TGCT using the deltopectoral approach. Patient 4 required a planned two-stage procedure; the second stage involved repositioning in left lateral decubitus and dual access via a medial scapular and subspinal incision.

Pathology

Tissue samples from synovial tissue were stained with hematoxylin and eosin, Elastica-van-Gieson (EvG) and iron stain and reviewed under a light microscope by a pathologist who specialized in musculoskeletal pathology.

Table 1 Patient characteristics

Patient No.	Age/ Sex	Duration of preoperative Symptoms (mo)	TGCT type	Torn Rotator Cuff Tendon	Adjuvant therapy	Recur-rence	Fol- low- Up (mo)
1	54/f	31	nodular	SSP	no	no	40
2	24/f	27	diffuse	-	no	no	39
3	17/m	12	diffuse	-	RSO	yes	48
4	60/m	60	diffuse	-	RT	stable disease	233

(abbreviations: f=female; m=male; mo=months; TGCT=Tenosynovial Giant Cell Tumor; SSP=Supraspinatus Tendon; RSO=radiosynoviorthesis; RT=Radiotherapy)

Postoperative rehabilitation

Drains were removed within 1–3 days postoperatively. Patients 1–3 received interscalene plexus blocks for 4–5 days, after which all patients maintained adequate pain control according to WHO classification levels 1–2. Shoulders were immobilized in an arm sling for 4–6 weeks, with passive glenohumeral mobilization up to 60° in abduction and flexion. From week 4, assisted active movements up to 90° were introduced, excluding external rotation. Beginning in week 7, exercises were progressively intensified beyond 90°, with rotation as tolerated. The goal was full shoulder mobility by 12 weeks, with return to sports permitted no earlier than week 12.

In the 1st year, MRI follow-up checks were carried out every 3 months, every 6 months in the 2nd year, then annually for 5 years.

Outcome instruments

The patient related outcome measures (PROMs) of this study included the Disabilities of Arm, Shoulder and Hand Questionnaire (DASH), the Oxford-Shoulder-Score (OSS) and the Short Form 36 Health Survey (SF-36). The total DASH score ranges from 0 to 100, where a score of 0 indicates an excellent functional outcome. The OSS ranges from 0 to 48, with a score of 48 representing the optimal functional outcome. The SF-36 health survey scores range from 0 to 100, where a score of 100 reflects excellent health and the highest quality of life. These PROMs were collected postoperatively at the time of the last follow-up. The rest of the data were retrieved from the patients' medical records.

Literature search strategy and selection criteria

A comprehensive literature search was conducted to identify all published studies reporting on TGCT of the shoulder. The search was performed using PubMed, google scholar, Web of Science from their inception to 15.02.2025. The following search terms were used in combination with Boolean operators: "tenosynovial giant cell tumor" OR "TGCT" OR "pigmented villonodular synovitis" OR "PVNS" OR "giant cell tumor of the tendon sheath" AND "shoulder".

Inclusion criteria were: (1) studies reporting on TGCT of the shoulder; (2) clinical studies, case series, or case reports; (3) abstracts published in English. Exclusion criteria included: (1) studies focusing on TGCT of other joints without specific mention of the shoulder; (2) review articles without original patient data.

Titles and abstracts were screened by 2 independent reviewers, followed by a full-text assessment for eligibility. Any disagreements were resolved by consensus or

consultation with a third reviewer. References of included articles were manually screened to identify additional relevant studies.

Statistical analytic tools

Analysis was performed with Microsoft Excel 2016. No statistical hypothesis tests were performed due to the small sample size ($n=4$). Results are presented descriptively using means $\pm$ standard deviations, ranges, and confidence intervals where appropriate to characterize the patient cohort and outcomes.

Results

Case 1

A 54-year-old female presented with a 2-year and 7-month history of progressive, movement-dependent left shoulder pain and restricted mobility without prior trauma. The pain was exacerbated by activity and present at rest and during the night. Conservative treatment, including physiotherapy and a corticosteroid injection, was ineffective.

MRI revealed a solid intra-articular mass with hemosiderin deposits in the shoulder recess, suggestive of TGCT (Fig. 1), along with AC joint osteoarthritis, a supraspinatus tendon tear with footprint cyst formation, and early degenerative changes of the glenohumeral joint. Clinically, TGCT appeared asymptomatic, with the symptoms attributed primarily to the rotator cuff pathology. Examination showed tenderness over the AC joint, subacromial space, and bicipital groove. Active range of motion included 160° flexion, 100° abduction, internal rotation to L2, and 30° external rotation. Impingement signs and supraspinatus and AC joint tests were positive.

Arthroscopy revealed articular-sided partial rupture of the SSP (Ellman III), SLAP I lesion, subacromial bursitis, acromioclavicular joint arthrosis, bony subacromial impingement, and a cherry-sized nodular lesion in the lower recess (Fig. 1). Arthroscopic synovectomy, SLAP debridement, subacromial bursectomy, lateral clavicle resection, supraspinatus tendon repair using a SpeedScrew anchor (Smith & Nephew GmbH, Hamburg, Germany) and bony subacromial decompression were performed. In open surgery, the pedunculated, dorsally luxated N-TGCT was removed in total. Histopathology confirmed TGCT. Three years and four months postoperatively, the patient remained free of recurrence with excellent functional outcome and full range of motion (PROMs in Tables 2 and 3).

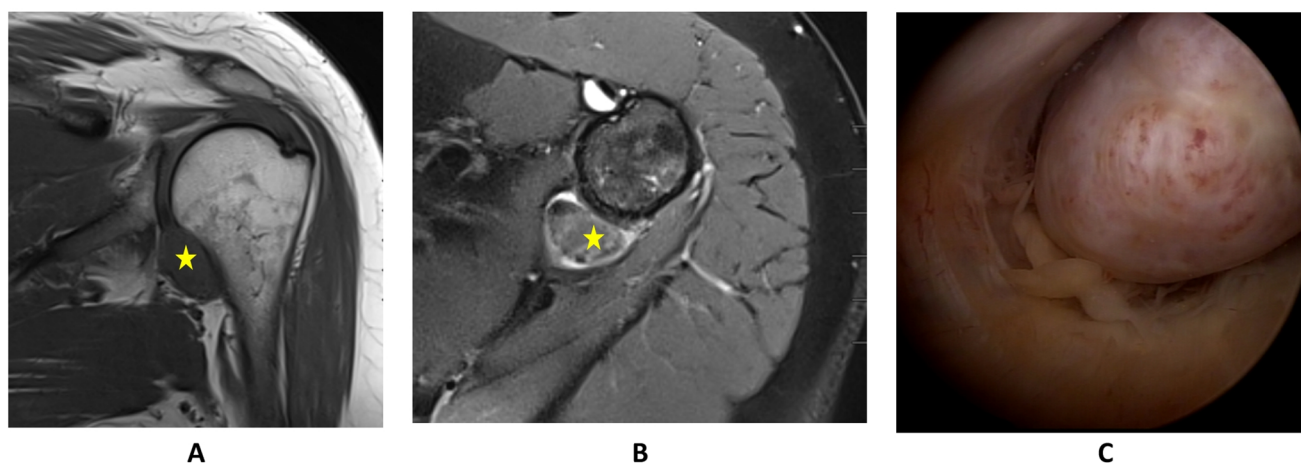


Fig. 1 MRI scans and arthroscopic images of TGCT of the shoulder in patient 1. **A:** T1w coronal view of the left shoulder, depicting a mass in the lower recess. **B:** T2w axial view of the left shoulder, depicting a

mass in the dorsal lower recess. **C:** Arthroscopic view of a mass in the dorsal lower recess of the left shoulder

Table 2 Individual Oxford-Shoulder-Score (OSS) and Score of the Disabilities of Arm, Shoulder and Hand Questionnaire (DASH), including mean and standard deviation (SD) for the patient collective at a median follow-up of 44 months (range 39–233 months)

Patient no.	OSS	DASH	DASH Sport/ Instrument	Sport / Instrument	DASH Work	Work
1	47	0,83	0	Volleyball/ piano	0	Art teacher
2	48	0	0	Running, biking	0	Desk activity
3	47	0,83	25	Strength training	0	Computer scientist
4	41	21,67	12,5	Senior gymnastics	-	Pensioner
Mean	45,75	5,83	9,38		0,00	
SD	3,20	10,57	11,97		0,00	

Table 3 Individual scores of the Short Form 36 Health Survey (SF-36), including mean and standard deviation (SD) for the patient collective at a median follow-up of 44 months (range 39–233 months)

Patient no.	Physical Functioning	Role limitations due to physical health	Role limitations due to emotional problems	Energy/ fatigue	Emotional well-being	Social functioning	Pain	Gen- eral health
1	80	0	0	30	40	88	0	35
2	100	100	100	80	96	100	100	100
3	95	100	100	80	88	100	90	40
4	55	100	100	50	88	75	78	60
Mean	82,50	75,00	75,00	60,00	78,00	90,75	67,00	58,75
SD	20,21	50,00	50,00	24,49	25,61	11,93	45,56	29,55

Case 2

A 24-year-old female patient reported right shoulder pain for two years and three months, treated conservatively. The pain was intermittent and worsened with specific movements. For two months, she noticed a lump in the upper arm accompanied by increased pain, which later subsided. Further diagnostic workup, including MRI was initiated. A diagnosis of diffuse TGCT involving the long biceps tendon and anterior shoulder joint was confirmed (Fig. 2).

She underwent marginal resection of the TGCT lesion. The patient was positioned in a beach chair position with a deltopectoral surgical approach. Intraoperative findings

included extensive TGCT masses along the long head of the biceps tendon, requiring transection and refixation of the subscapularis tendon. A solid TGCT lesion caudally was also noted, pedunculated and attached to the capsule. Histopathology confirmed TGCT.

Three years and three months postoperatively, the patient remained free of recurrence with excellent functional outcome (PROMs in Tables 2 and 3).

Case 3

An 18-year-old male presented with progressive, atraumatic right shoulder pain over one year, localized intra-articularly

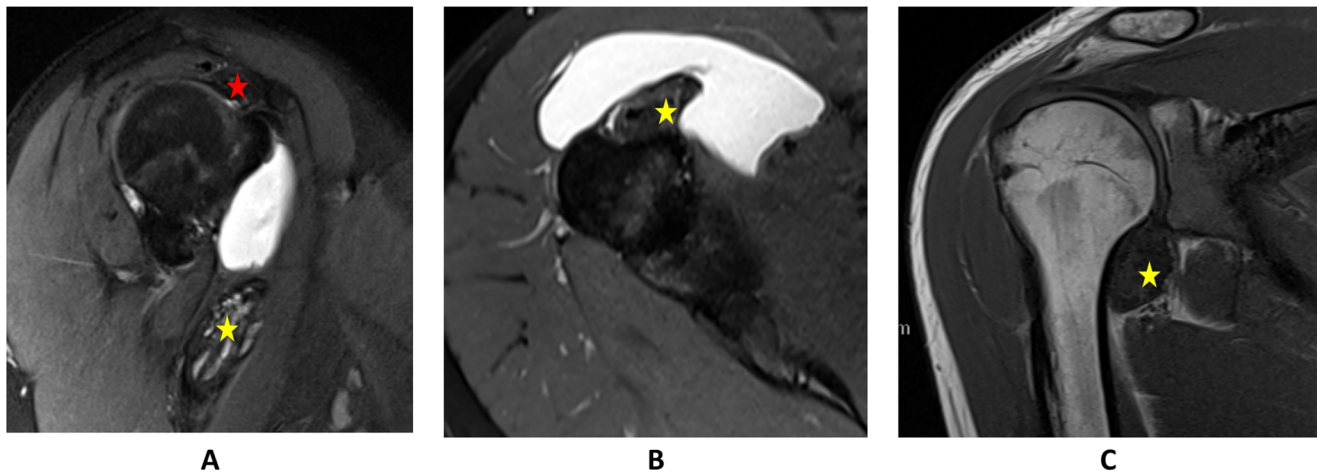


Fig. 2 MRI scans of TGCT of the shoulder in patient 2. **A:** sagittal PD tse fs TE 47 of the right shoulder; **B:** axial PD tse fs TE 47 of the right shoulder; **C:** coronal T1 TSE of the right shoulder; The star marks the masses of TGCT around the biceps tendon (yellow) and subacromial (red)

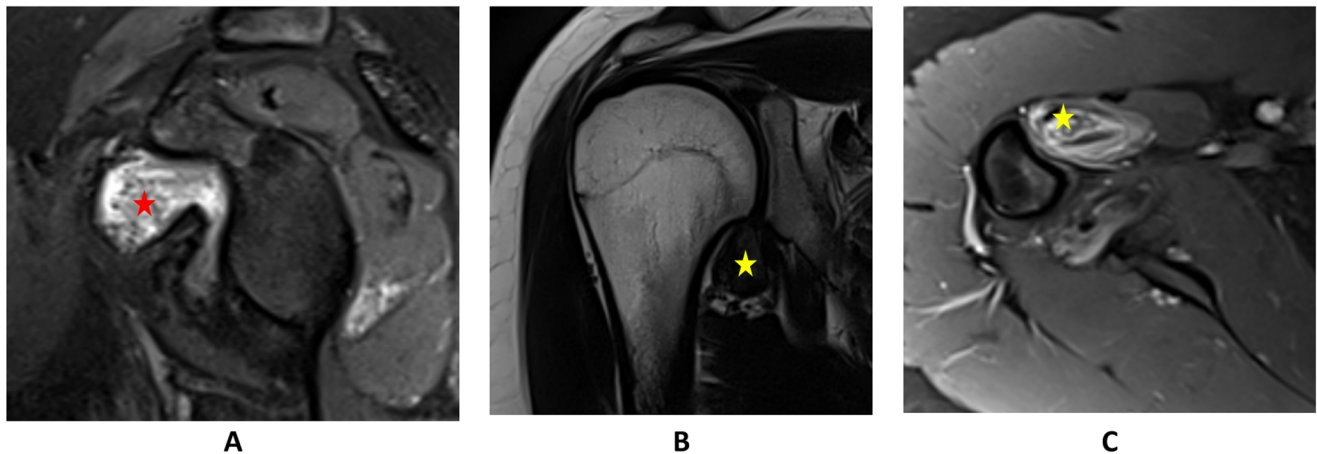


Fig. 3 MRI scans of TGCT of the shoulder in patient 3. **A:** PDFS sagittal of the right shoulder: red star marks the mass below the coracoid. **B:** T2 coronal of the right shoulder: yellow star marks the mass in the

lower recess. **C:** T1FS axial view of the right shoulder: yellow star marks the mass in the lower recess

and radiating to the proximal humerus. The discomfort was described as intra-articular, radiating to the proximal humerus, and unrelated to mechanical load. MRI revealed a suspected TGCT in the inferior recess and below the coracoid process (Fig. 3). Open synovectomy via a deltopectoral approach confirmed TGCT histologically. Postoperative recovery was initially uneventful.

Seven months postoperatively, MRI revealed a LR, necessitating a repeat open synovectomy and subsequent RSO. Despite initially unremarkable follow-up imaging, progressive lesion growth was noted 1.5 years later. The patient was clinically symptom-free. A third surgical intervention—arthroscopic total synovectomy—was performed three years after the initial recurrence, revealing extensive brownish synovitis and mild cartilage damage (grades I–II). The arthroscope was also repositioned ventrally in order to

achieve a dorsal synovectomy of the lower recess. Histopathology again confirmed TGCT.

Postoperative care included free mobilization and a second RSO. The patient remained symptom-free with no radiological evidence of recurrence at one-year follow-up. One year later, there was no evidence of recurrence (PROMs in Tables 2 and 3).

Case 4

A 61-year-old male patient experienced right shoulder pain for at least six years, progressively worsening over the past six months. Imaging, including X-rays and MRI, revealed extensive TGCT extending into the thoracic wall, involving the humeral shaft with intraosseous components, and encasing neurovascular structures without infiltration (Fig. 4).

The patient underwent open anterior synovectomy, tumor resection, and intraosseous curettage. This patient showed very pronounced TGCT findings: a large tumor extending subscapularly, a broad infiltration of the dorsal joint space extending far distally along the humerus. In addition, there was an extensive infiltration of the long head of the biceps tendon on the ventral side, destroyed to such an extent that it tore when pulled with a finger. Besides, the TGCT infiltrated the proximal medullary cavity in the bicipital sulcus. Here, the window was opened with the Luer and extensively curetted. Ultimately, the lesions could be completely removed ventrally, but there were still tumor parts in the area of the subscapularis muscle and the dorsal humerus, which were then removed in a planned second surgery six weeks later. Histopathology confirmed TGCT.

Postoperative care included immobilization in a Gilchrist brace for four weeks, and limited shoulder movement. Postoperative radiation therapy followed due to remaining tumor tissue.

MRI surveillance continued at regular intervals, revealed stable disease without significant progression over subsequent years. 19 years and 5 months after the initial diagnosis, the patient maintained functional mobility with mild restrictions (PROMs in Tables 2 and 3).

Outcome of DASH, OSS and SF-36

All scores for each patient and means are shown in Tables 2 and 3.

Pathologic findings

Macroscopically, the excised tissues were variably orange-brown and gray-glassy.

Patient 1 (N-TGCT) showed a nodular lesion with oval nuclei, scattered disorganized giant cells, and hemosiderin-loaded macrophages, without atypia or necrosis.

Patients 2–4 (D-TGCT) demonstrated cell-rich synovial tissue with irregular giant cells, foam cells, hemosiderin deposits, and signs of old hemorrhage. Focal necrosis and marked bleeding were present, but no cellular atypia was observed.

Literature review

Shoulder TGCT is an exceptionally rare condition, with a total of 107 cases identified in the literature, including the four patients presented in this study. The sex distribution was nearly balanced, with a slight predominance of female patients (46.7%) over male patients (36.4%). The mean age at diagnosis was 50.3 ± 19.7 years, ranging from 5 to 84 years. The average duration of symptoms prior to diagnosis was 23.6 ± 28.1 months. TGCT subtype was specified in only 65 cases, of which 33.8% were classified as N-TGCT and 66.1% as D-TGCT. The affected side was reported in 58 cases and the left shoulder was affected in 34.5% of cases, the right shoulder in 58.6%, and bilateral involvement was reported in 6.9%. A concomitant rotator cuff tear was documented in 32.4% of patients, while bone involvement was reported in 30.6% of cases.

Surgical details were available for 77 cases: 42.9% underwent open surgery, 53.2% arthroscopic surgery, and 3.9% a combined approach. Rotator cuff repair was performed

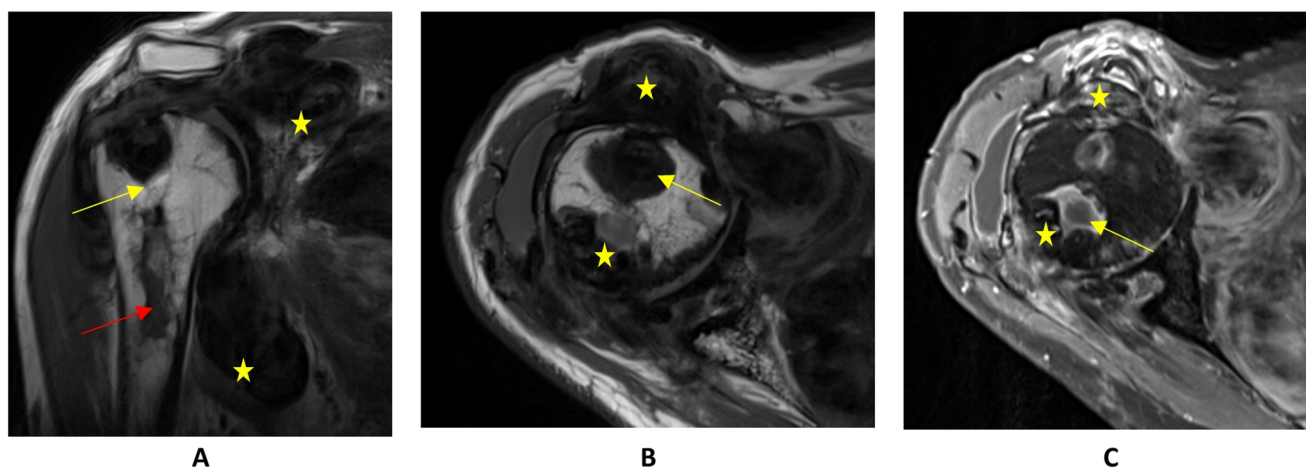


Fig. 4 MRI scans of TGCT of the shoulder in patient 4. **A:** T1 TSE coronal: cystic components (marked by a yellow arrow). Destruction of the humeral head and erosions in the subcapital humeral shaft (marked by a red arrow). The lesions extend subacromial and to the medial thoracic wall (exemplified by yellow stars). **B:** T1 TSE axial: hypoin-

tense nodular structures (exemplified by yellow stars) with individual cystic components (marked by a yellow arrow). **C:** T1 TSE axial with contrast fluid: hypointense nodular structures (exemplified by yellow stars) with individual cystic components (marked by a yellow arrow) with increased absorption of contrast medium

in 20.8% of cases, and rotator cuff debridement in 2.7%. Shoulder arthroplasty was performed in 12 cases (15.6% of surgical cases), including four hemiarthroplasties (HA), six reverse total shoulder arthroplasties (rTSA) (one of the rTSAs in a case of recurrence), one anatomic total shoulder arthroplasty (aTSA), and two cases where the type of prosthesis was not specified.

Adjuvant therapy was administered in 9.3% of cases ($n=10$), including RSO in one patient (0.9%), radiotherapy in eight patients (7.5%), and osmium acid therapy in one patient (0.9%). The mean follow-up duration was 37.6 ± 36.2 months (range 6–233 months) in 67 cases. In those 44.8% were D-TGCT, 19.4% were N-TGCT and in 35.8% type of TGCT was not mentioned. In those 67 cases LR was reported in 10.6% ($n=7$) and residual disease in 4.5% ($n=3$). All reported recurrences and residual diseases were classified as D-TGCT. Details of the literature review are depicted in Table 4.

Discussion

Main findings

This case series demonstrates that shoulder TGCT can be effectively managed with arthroscopic and open synovectomy combined with rotator cuff repair, achieving excellent long-term functional outcomes and quality of life. All patients maintained adequate pain control and returned to work or sports. Even the patient with most extensive intraosseous involvement showed disease stability over 10 years without revision surgery, highlighting the feasibility of conservative management after initial resection.

Epidemiology

With a mean age of 38.8 ± 21.4 years (range 17–60 years) at their initial presentation, patients in this study fit well compared to other studies. It is suggested that the incidence peak is between 30 and 50 years of age [43]. Other literature reviews even suggest 50–80 years [44]. So far, only two case reports mentioned TGCT in the shoulders of adolescents [36, 45]. Females and males are equally affected [43].

Diagnosis

In this case series, the mean duration from symptom onset to diagnosis was 32.5 ± 17.4 months, compared to 23.6 ± 28.4 months in the literature, highlighting a significant diagnostic delay in TGCT. This delay often results in advanced joint involvement, with approximately one-third of patients

already exhibiting bone erosions due to synovial inflammation, mechanical stress from the lesion, or massive rotator cuff tears [41]. Despite this, the majority of the patients achieve symptom resolution after arthroscopic or open surgery [41, 43]. TGCT should be considered in young patients with unexplained shoulder pain and MRI findings suggestive of neoplastic disease [36, 37].

Functional outcomes post-surgical intervention

There is limited literature on PROMs after TGCT surgery of the shoulder. VJ et al. reported on six patients undergoing arthroscopic synovectomy, showing significant improvements: Constant score from 64.8 to 94.5, American Shoulder and Elbow Surgeons (ASES) score from 81.2 to 99.7, and University of California, Los Angeles (UCLA) score from 23.2 to 34.8 at ≥ 36 months follow-up [46].

Quality of life post-surgical intervention

According to our literature research this is the first study reporting quality of life results for patients with TGCT of the shoulder. Most patients reported outcomes equal to or exceeding the sex- and age-adjusted normative data for the German population [47]. Patient 1 developed severe rheumatoid arthritis, which likely accounts for the self-reported impairment in quality of life, despite demonstrating excellent functional outcomes for the shoulder. Patient 4 reported diminished physical functioning (Score = 50) compared to the age- and sex-adjusted German normative data (Mean Score = 76.4; 95%-CI = 73.7–79.1). This reduction may be attributable to his limited shoulder mobility. Patient 3 reported significantly worse general health (Score = 40) relative to the German normative population (Mean Score = 75.3; 95%-CI = 73.7–77.0). As the youngest participant in this study, the impact of impaired functionality may be more pronounced, particularly due to the inability to engage in sports and strength training, as reflected by a DASH sport/instrument score of 25. Furthermore, the most recent revision surgery occurred only 10 months prior to the assessment, suggesting potential for further improvement in personal strength and overall functional outcomes.

Treatment – open surgery or arthroscopy?

There are no standardized guidelines for choosing between open and arthroscopic surgery in the management of shoulder TGCT. Both approaches yield good outcomes. Open synovectomy may be necessary for complete resection,

Table 4 Results of the literature review of TGCT of the shoulder

source	num- ber of patients	sex	age	duration of symp- toms in months	nodular / diffuse	affected side	rota- tor cuff tear	bony involvement	type of surgery	cuff-repair	type of AP	adju- vant therapy	follow- up time (months)	recurrence	treatment of recurrence	resid- ual lesion
Agrawal et al. [59]	1	m	38	4	d	r		yes	open				8	yes	RSO	yes
Broski et al. [60]	1	f	46		d	l			open							
Byers et al. [55]	1								open							
Cheng et al. [52]	1	m	20	36	n		yes		AS				22,4	no		
Chiang et al. [41]]	5	f					yes		AS				22,4	no		
		f					yes		AS				22,4	no		
		m					yes		AS				22,4	no		
		m					yes		AS				22,4	no		
		m					yes		AS				22,4	no		
Cho et al. [61]	1	m	21	3		r			AS+open				18	no		
Colak et al. [62]	1	f	49						AS				9	no		
Costallat et al. [63]	1	f	15	12		r			AS							
Cotten et al. [64]	1	m	52	8	d				open							
Dias et al. [65]	1	f	59	1	n	l			open				24	no		
Dkhissi et al. [66]	1	m	68	36	d		yes	yes	open				6	yes	rTSA	
Dorwart et al. [43]	2	f	65	48	d		yes	yes	open	yes			114	yes		
		m	56	9	d			yes	open		rTSA					
Du et al. [67]	1	f		12	d	l			open							
Elumogo et al. [68]	1	m	65	chronic		l			open							
Flandry and Norwood [69]	1	m	28		d				open				24	no		
Ganel et al. [70]	2	f	74	9	d		yes	yes	open				36	no		
		m	79	8	d		yes	yes	open				24	no		
Giannatos et al. [44]	1	f	50	24		l			AS				12	no		
Golge et al. [54]	1	m	75	chronic			yes	yes	open		rTSA					
Graf et al. [71]	1	f	80		d	b	yes		AS	yes						
Gumina et al. [40]	9	f	65,8	12		r	yes		AS	yes			27			
		f	65,8	12		r	yes		AS	yes			27			
		f	65,8	12		r	yes		AS	yes			27			
		f	65,8	12		r	yes		AS	yes			27			
		f	65,8	12		r	yes		AS	yes			27			
		f	65,8	12		r	yes		AS	yes			27			
		m	65,8	12		r	yes		AS	yes			27			
		m	65,8	12		r	yes		AS	yes			27			
		m	65,8	12		l	yes		AS	yes			27			
		m	65,8	12		l	yes		AS	yes			27			
Ji et al. [51]	2	f	66	36	d	r	yes	yes	AS	yes	RT		24	no		
		f	71	120	d	r	yes	yes	AS	yes			36	no		
Johansson et al. [42]	4		35	72	d			yes		rTSA			24	no		

Table 4 (continued)

source	num- ber of patients	sex	age	duration of symp- toms in months	nodular / diffuse	affected side	rota- tor cuff tear	bony involvement	type of surgery	cuff-repair	type of AP	adju- vant therapy	follow- up time (months)	recurrence	treatment of recurrence	resid- ual lesion
			35	72	d			yes			rTSA		24	no		
Kho et al. [39]	1		35	24	n			yes					180	no		
Konrath et al. [72]	1	m	64	4	d	b	yes	yes	AS				48	no		
Kwon et al. [73]	1	f	71	6	d	r		yes	open		HSA		7	no		
Lee, K. et al. [74]	1	f	27	0	n	l		yes	open				12	no		
Lee, S.-J. et al. [75]	1	m	64		d	b	yes	yes	AS				43	no		
Lee, M. et al. [76]	1	f	40		d	l		yes	open							
Levin and Gannon [77]	2	f	79	2	d			yes	open							
Lewis et al. [78]	1	f	21	6	d			yes	open							
Li et al. [27]	6	m	58	0	n	r	yes		AS				92	yes	2 x AS (progress from N-TGCT to D-TGCT)	
		f	45		n/d	r										
		f	30		n	l			AS			RT	62	no		
Liang et al. [79]	1	f	65		n	r	yes		AS				44	no		
Loh et al. [80]	1	f	62		n	r	yes		AS				40	no		
Mahieu et al. [38]	2	f	38		n	r			AS				28	no		
		m	64		n	r	yes		AS				46	no		
						r										
Liang et al. [79]	1	m	46	6	n	r			AS+open				9	no		
Loh et al. [80]	1	f	30	96	n	l		yes	AS				96	no		
Mahieu et al. [38]	2	f	53	36	d	r	yes	yes	AS			osmium acid	24	no		
Mankin et al. [81]]	4															
Mazabraud et al. [82]	4							yes								
								yes								
Mazabraud et al. [82]	1	f	17		d	r (mf)			conservative				14	residual		yes
Mulier et al. [84]	1	m	64	12	d		yes	yes		yes	RT		6	no		
Muller et al. [36]	1	m	16	4	n	l		yes	open							
Palmerini et al. [85]	2							yes	open		AP					
								yes	open		AP					
Pantazopoulos et al. [53]	1	m	36		d			yes	open		HSA		24	no		
Park et al. [49]	1		29		d		yes	yes	open				12	no		
Pereira et al. [20]	1	f	15	24	n	r			AS		HSA		48	no		
Petsatodis et al. [48]	2	f	60	12	d	r			open		HSA			no		

Table 4 (continued)

source	num- ber of patients	sex	age	duration of symp- toms in months	nodular / diffuse	affected side	rota- tor cuff tear	bony involvement	type of surgery	cuff-repair type	type of AP	adju- vant therapy	follow- up time (months)	recurrence	treatment of recurrence	resid- ual lesion
Popov et al. [86]	1	m	63	6	d	l			open		HSA		60	no		
Rajakulasingam et al. [87]	1	f	63	120	d	l		yes	open			RT	12	no		
Rezaee, A. et al. [88]	1	m	59		n					yes			21	no		
Sawmiller et al. [89]	1	f	57	12			yes									
Sayegh et al. [90]	1	f	34	20	d	l			AS							
Schwartz et al. [56]	2	m	55	36	n			yes	open		rTSA					
		m	58	90	d				open							
Seiler et al. [91]	1	m	55	60					open				60	no		
Selby et al. [92]	1	m	58		n	r		yes	AS			RT				
Serra et al. [29]	1	m	74	8	d			yes								
Sher et al. [93]	1	f	18		n			yes								
Simmer et al. [94]	1	f	58	2	n	l			AS				53	no		
Sipahioğlu et al. [95]	1	m	23	24	d				AS				36	no		
Snook [96]	2	f	82	12	d			yes	open			RT	18	yes		yes
		f	84	1	d			yes	conservative			RT	24	residual		yes
Sotje et al. [97]	1	m	5		n				AS+open	yes			40	no		
Stadelmeier et al.	4	f	54	31	n	l	yes		open				39	no		
		f	24	27	d	r			open			RSO	48	yes	open, AS, RSO	
		m	17	12	d	r			open							
Tang et al. [98]	1	m	60	60	d	r		yes	open			RT	233	residual		yes
Tong et al. [99]	1	f	74		d	r							36	yes		
Toro et al. [50]	1	m	51	6	d		yes									
Vj et al. [46]	6	f	39	6	d	l		yes	open		aTSA		48	no		
		f	42		d	l			AS				36	no		
		f	38		r				AS				36	no		
		m	74		r				AS				36	no		
		f	63		l				AS				36	no		
		f	60		r				AS				36	no		
Zhao et al. [100]	1	m	27		d	r			AS				36	no		
		f	7	6	d	b (mf)	yes		conservative							yes

(Abbreviations: m = male; f = female; n = nodular; d = diffuse; l = left; r = right; b = both shoulders; mf = multifocal; AS = arthroscopy; HSA = hemi shoulder arthroplasty; AP = arthroplasty (not specified); rTSA = reverse total shoulder arthroplasty; aTSA = anatomic total shoulder arthroplasty; RSO = radiosynoviorrhesis; RT = radiotherapy)

especially in D-TGCT [42], while arthroscopic resection can also be effective if total synovectomy is achieved [39].

Arthroscopy offers the advantage of joint-wide visualization and lower morbidity and may be preferable when diagnosis is uncertain [27, 38]. In this series, one patient was successfully treated arthroscopically for re-recurrence. Alternating between open and arthroscopic techniques may help in recurrent cases.

Expert consensus by Stacchiotti et al. recommends macroscopic complete resection for all TGCT types, with arthroscopic or open approaches chosen, based on disease extent. D-TGCT has a high risk of local recurrence and should be discussed in a multidisciplinary tumor board. Extra-articular D-TGCT may require marginal resection through extended incisions due to soft tissue involvement [5].

What to do with rotator cuff tear?

In cases involving the shoulder, a thorough evaluation of the rotator cuff and articular surfaces is essential, as these factors play a critical role in determining clinical outcomes. Rotator cuff tears associated with shoulder TGCT are typically classified as massive, and degenerative changes in the articular surfaces are consistently observed to varying degrees. Consequently, in addition to synovectomy, the following surgical interventions are recommended if needed [40]: arthroscopic debridement and of the cuff tear [39, 41], partial cuff repair [41], HA [48, 49], and TSA [50].

Jong-Hun Ji et al. and Li et al. reported favorable clinical outcomes following arthroscopic or open synovectomy combined with rotator cuff repair, highlighting the therapeutic benefit of early intervention [27, 51]. Notably, Gumina et al. found that patients with D-TGCT who underwent arthroscopic synovectomy and subacromial debridement had significantly worse clinical outcomes compared to a control group without synovitis or osteoarthritis (arthroscopic debridement for irreparable rotator cuff tear alone). These poorer results were attributed to secondary osteoarthritis caused by unrecognized or late-treated TGCT [40]. Importantly, in cases where TGCT was identified early and treated with combined synovectomy and rotator cuff repair, outcomes were satisfactory, and no osteoarthritic changes were observed at follow-up.

Severe joint destruction and arthroplasty

TGCT of the shoulder can lead to severe joint destruction, often presenting with polycystic changes in the humeral head [52]. Elevated intra-articular pressure contributes to localized bone resorption and cyst formation, which may be further invaded by hypertrophic synovium via cortical

breaches, the chondro-osseous junction, or vascular foramina [36, 48, 53]. As the villonodular tissue expands, progressive intraosseous infiltration may occur.

In advanced cases with substantial joint degeneration, various forms of shoulder arthroplasty—including HA, aTSA, and rTSA—have shown favorable outcomes [48, 50, 54]. Case reports demonstrate that these procedures effectively relieve pain, restore joint function, and reduce recurrence risk by enabling more complete dorsal synovectomy after osteotomy of the humeral head [5, 50]. The choice of prosthesis should be guided by the extent of osseous destruction and the presence of rotator cuff pathology. Specifically, rTSA may be advantageous in patients with concomitant rotator cuff tears, while aTSA or HA are appropriate in cases with preserved cuff integrity [42, 43]. Current evidence supports arthroplasty as a viable treatment option in patients with TGCT of the shoulder and advanced joint damage, particularly in elderly individuals with degenerative changes [36, 50].

Adjuvant therapies

Percutaneous radiotherapy can offer some relief of the symptoms and it can be beneficial as adjuvant treatment after surgical resection for residual lesions [28, 29]. However, due to the young age of most patients, it is rarely a primary alternative. For inoperable cases, systemic therapy is preferable, while surgical management with complete macroscopic resection remains the treatment of choice for D-TGCT, and early marginal excision is recommended for the nodular form [5].

Given that patients with TGCT are typically young, and the disease is non-life-threatening, long-term complications such as malignant transformation, fibrosis, joint stiffness, and other sequelae are of particular concern. The use of radiotherapy in selected cases lacking viable alternative treatment options remains a subject of ongoing debate [5].

Radiosynoviorthesis has not demonstrated efficacy in the management of D-TGCT and should not be employed as a substitute for incomplete surgical resection. Prospective studies are warranted to better define its potential therapeutic role in the treatment of TGCT [5].

Recurrence

Reported LR rates of TGCT after treatment vary widely between 9% and 55%, influenced by nodular or diffuse disease, joint location and follow-up duration [55–57]. Most of the recurrences are seen in the first 2 years after surgery. Recurrence in the shoulder is rarely reported, likely due to

the overall low incidence of TGCT at this site [50]. In this literature review, the LR rate for shoulder TGCT was 10.6% ($n = 7$) and the rate for residual disease was 4.5% ($n = 3$) at a mean follow-up of 37.6 ± 36.2 months (range 6–233 months).

D-TGCT has a higher recurrence risk than the localized form, though data specific to the shoulder are limited [57]. Key risk factors include incomplete surgical resection, particularly with extra-articular or intraosseous involvement, as well as delayed diagnosis and long symptom duration. These factors contribute to more extensive synovial disease and increase the likelihood of residual tumor and recurrence. Moreover, revision surgery in recurrent TGCT cases carries a substantially elevated risk of further recurrence [5]. Careful preoperative MRI evaluation is essential to detect occult or discontinuously growing lesions that may be missed during standard surgical approaches [58].

The main limitation of this study is the small sample size, which is inherent to the rarity of TGCT of the shoulder. Nevertheless, the extended follow-up duration and the inclusion of both functional and quality-of-life outcome measures enhance the clinical relevance of the presented findings. Despite these limitations, the present series contributes one of the longest follow-up periods and most comprehensive functional assessments reported to date for this rare entity, thereby expanding the current evidence base. These observations permit cautious but meaningful conclusions regarding the management of shoulder TGCT and underscore the importance of individualized treatment planning and vigilant follow-up.

Conclusion

TGCT of the shoulder is a rare condition that requires an individualized and multidisciplinary approach. Both arthroscopic and open synovectomy can achieve favorable outcomes when complete resection is accomplished. Given the risk of recurrence, structured long-term follow-up remains essential. To further refine treatment algorithms and identify prognostic factors specific to shoulder TGCT, larger multicenter or registry-based studies with standardized outcome reporting are needed.

Author contributions JS and HRD wrote the main manuscript text. FB gathered the patient related outcomes and included the patients. PR reviewed and provided the figures. JS, FB, AK, FW, PR and HRD reviewed the manuscript.

Funding Open Access funding enabled and organized by Projekt DEAL.

Data availability No datasets were generated or analysed during the current study.

Declarations

Conflict of interests The authors declare no competing interests.

Open Access This article is licensed under a Creative Commons Attribution 4.0 International License, which permits use, sharing, adaptation, distribution and reproduction in any medium or format, as long as you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons licence, and indicate if changes were made. The images or other third party material in this article are included in the article's Creative Commons licence, unless indicated otherwise in a credit line to the material. If material is not included in the article's Creative Commons licence and your intended use is not permitted by statutory regulation or exceeds the permitted use, you will need to obtain permission directly from the copyright holder. To view a copy of this licence, visit <http://creativecommons.org/licenses/by/4.0/>.

References

1. Dei Tos A, Somerhausen N, Rijn M (2020) Tenosynovial giant cell tumour. WHO Classification of Tumours Editorial Board (eds). In: Soft Tissue and Bone Tumours, 5th edn. International Agency for Research on Cancer, Lyon (France), pp 133–136
2. Granowitz SP, Mankin HJ (1967) Localized pigmented villonodular synovitis of the knee: report of five cases. *J Bone Joint Surg Am* 49(1):122–128
3. Mastboom MJL, Verspoor FGM, Verschoor AJ, Uittenbogaard D, Nemeth B, Mastboom WJB et al (2017) Higher incidence rates than previously known in tenosynovial giant cell tumors. *Acta Orthop* 88(6):688–694. <https://doi.org/10.1080/17453674.2017.1361126>
4. Ehrenstein V, Andersen SL, Qazi I, Sankar N, Pedersen AB, Sikorski R et al (2017) Tenosynovial Giant Cell Tumor: Incidence, Prevalence, Patient Characteristics, and Recurrence. A Registry-based Cohort Study in Denmark. *J Rheumatol* 44(10):1476–1483. <https://doi.org/10.3899/jrheum.160816>
5. Stacchiotti S, Dürr HR, Schaefer I-M, Woertler K, Haas R, Trama A et al (2023) Best clinical management of tenosynovial giant cell tumour (TGCT): A consensus paper from the community of experts. *Cancer Treat Rev* 112:102491. <https://doi.org/10.1016/j.ctrv.2022.102491>
6. Stephan SR, Shallop B, Lackman R, Kim TWB, Mulcahey MK (2016) Pigmented Villonodular Synovitis: A Comprehensive Review and Proposed Treatment Algorithm. *JBJS Rev* 4(7). <https://doi.org/10.2106/JBJS.RVW.15.00086>
7. Sciort R, Rosai J, Dal Cin P, de Wever I, Fletcher CD, Mandahl N et al (1999) Analysis of 35 cases of localized and diffuse tenosynovial giant cell tumor: a report from the Chromosomes and Morphology (CHAMP) study group. *Mod Pathol* 12(6):576–579
8. Nilsson M, Hoglund M, Panagopoulos I, Sciort R, Dal Cin P, Debiec-Rychter M et al (2002) Molecular cytogenetic mapping of recurrent chromosomal breakpoints in tenosynovial giant cell tumors. *Virchows Arch* 441(5):475–480. <https://doi.org/10.1007/s00428-002-0640-y>
9. Fletcher CDMBJ, Hogendoorn PCW, Mertens F (2013) WHO Classification of Tumours of Soft Tissue and Bone. 4 ed. WHO Classification of Tumours. Lyon: IARC Press
10. Ota T, Nishida Y, Ikuta K, Tsukushi S, Yamada K, Kozawa E et al (2021) Tumor location and type affect local recurrence and joint damage in tenosynovial giant cell tumor: a multi-center study. *Sci Rep* 11(1):17384. <https://doi.org/10.1038/s41598-021-96795-6>

11. Myers BW, Masi AT (1980) Pigmented villonodular synovitis and tenosynovitis: a clinical epidemiologic study of 166 cases and literature review. *Med (Baltim)* 59(3):223–238
12. Falster C, Stockmann Poulsen S, Joergensen U (2017) A rare case of localised pigmented villonodular synovitis in the knee of a 24-year-old female soccer player: diagnosis, management and summary of tenosynovial giant cell tumours. *BMJ Case Rep* 2017(bcr-2017-219549). <https://doi.org/10.1136/bcr-2017-219549>
13. West RB, Rubin BP, Miller MA, Subramanian S, Kaygusuz G, Montgomery K et al (2006) A landscape effect in tenosynovial giant-cell tumor from activation of CSF1 expression by a translocation in a minority of tumor cells. *Proc Natl Acad Sci U S A* 103(3):690–695. <https://doi.org/10.1073/pnas.0507321103>
14. Tap WD, Healey JH (2022) Role of colony-stimulating factor 1 in the neoplastic process of tenosynovial giant cell tumor. *Tumour Biol* 44(1):239–248. <https://doi.org/10.3233/TUB-220005>
15. Friedman T, Chen T, Chang A (2013) MRI diagnosis of recurrent pigmented villonodular synovitis following total joint arthroplasty. *HSS J* 9(1):100–105. <https://doi.org/10.1007/s11420-012-9283-y>
16. Garner HW, Ortiguera CJ, Nakhleh RE (2008) Pigmented villonodular synovitis. *Radiographics* 28(5):1519–1523. <https://doi.org/10.1148/rq.285075190>
17. Murphey MD, Rhee JH, Lewis RB, Fanburg-Smith JC, Flemming DJ, Walker EA (2008) Pigmented villonodular synovitis: radiologic-pathologic correlation. *Radiographics* 28(5):1493–1518. <https://doi.org/10.1148/rq.285085134>
18. Cheng XG, You YH, Liu W, Zhao T, Qu H (2004) MRI features of pigmented villonodular synovitis (PVNS). *Clin Rheumatol* 23(1):31–34. <https://doi.org/10.1007/s10067-003-0827-x>
19. Ushijima M, Hashimoto H, Tsuneyoshi M, Enjoji M (1986) Giant cell tumor of the tendon sheath (nodular tenosynovitis). A study of 207 cases to compare the large joint group with the common digit group. *Cancer* 57(4):875–884
20. Pereira VL, Baldan AR, Andreoli CV, Belangero PS, de Castro Pochini A, Ejnisman B (2021) Subacromial pigmented villonodular synovitis: case report and review. *J Surg Case Rep* 2021(3):rjab019. <https://doi.org/10.1093/jscr/rjab019>
21. Rubin BP (2007) Tenosynovial giant cell tumor and pigmented villonodular synovitis: a proposal for unification of these clinically distinct but histologically and genetically identical lesions. *Skeletal Radiol* 36(4):267–268. <https://doi.org/10.1007/s00256-006-0249-3>
22. de St. Aubain Somerhausen N, van de Rijn M (2013) Tenosynovial giant cell tumour, diffuse type. WHO classification of tumours editorial board (eds). In: WHO classification of tumours of soft tissue and bone. IARC, Lyon (France), pp 133–136
23. Mastboom MJL, Hoek DM, Bovée JVMG, van de Sande MAJ, Suzhai K (2019) Does 1 overexpression or rearrangement influence biological behaviour in tenosynovial giant cell tumours of the knee? *Histopathology* 74(2):332–340. <https://doi.org/10.1111/his.13744>
24. Ho J, Peters T, Dickson BC, Swanson D, Fernandez A, Frova-Seguín A et al (2020) Detection of CSF1 rearrangements deleting the 3' UTR in tenosynovial giant cell tumors. *Genes Chromosomes Cancer* 59(2):96–105. <https://doi.org/10.1002/gcc.22807>
25. Möller E, Mandahl N, Mertens F, Panagopoulos I (2008) Molecular identification of COL6A3-CSF1 fusion transcripts in tenosynovial giant cell tumors. *Genes Chromosomes Cancer* 47(1):21–25. <https://doi.org/10.1002/gcc.20501>
26. Tap WD, Wainberg ZA, Anthony SP, Ibrahim PN, Zhang C, Healey JH et al (2015) Structure-Guided Blockade of CSF1R Kinase in Tenosynovial Giant-Cell Tumor. *N Engl J Med* 373(5):428–437. <https://doi.org/10.1056/NEJMoa1411366>
27. Li Y, Mei L, Li T, Pang L, Tang X, Li J (2022) Clinical outcomes of patients with pigmented villonodular synovitis of the shoulder after arthroscopic synovectomy. *BMC Musculoskelet Disord* 23(1):1023. <https://doi.org/10.1186/s12891-022-05978-3>
28. Heyd R, Micke O, Berger B, Eich HT, Ackermann H, Seegenschmiedt MH et al (2010) Radiation therapy for treatment of pigmented villonodular synovitis: results of a national patterns of care study. *Int J Radiat Oncol Biol Phys* 78(1):199–204. <https://doi.org/10.1016/j.ijrobp.2009.07.1747>
29. Serra TQ, Morais J, Goncalves Z, Agostinho F, Melo G, Henriques M (2017) An unusual case of diffuse pigmented villonodular synovitis of the shoulder: A multidisciplinary approach with arthroscopic synovectomy and adjuvant radiotherapy. *Eur J Rheumatol* 4(2):142–144. <https://doi.org/10.5152/eurjrheum.2016.15084>
30. Dürr HR, Capellen CF, Klein A, Baur-Melnyk A, Birkenmaier C, Jansson V et al (2019) The effects of radiosynoviorrhesis in pigmented villonodular synovitis of the knee. *Arch Orthop Trauma Surg* 139(5):623–627. <https://doi.org/10.1007/s00402-018-3097-4>
31. Tap WD, Gelderblom H, Palmerini E, Desai J, Bauer S, Blay J-Y et al (2019) Pexidartinib versus placebo for advanced tenosynovial giant cell tumour (ENLIVEN): a randomised phase 3 trial. *Lancet* 394(10197):478–487. [https://doi.org/10.1016/S0140-6736\(19\)30764-0](https://doi.org/10.1016/S0140-6736(19)30764-0)
32. Cassier PA, Gelderblom H, Stacchiotti S, Thomas D, Maki RG, Kroep JR et al (2012) Efficacy of imatinib mesylate for the treatment of locally advanced and/or metastatic tenosynovial giant cell tumor/pigmented villonodular synovitis. *Cancer* 118(6):1649–1655. <https://doi.org/10.1002/cncr.26409>
33. Verspoor FGM, Mastboom MJL, Hannink G, Maki RG, Wagner A, Bompas E et al (2019) Long-term efficacy of imatinib mesylate in patients with advanced Tenosynovial Giant Cell Tumor. *Sci Rep* 9(1):14551. <https://doi.org/10.1038/s41598-019-51211-y>
34. Gelderblom H, Cropet C, Chevreau C, Boyle R, Tattersall M, Stacchiotti S et al (2018) Nilotinib in locally advanced pigmented villonodular synovitis: a multicentre, open-label, single-arm, phase 2 trial. *Lancet Oncol* 19(5):639–648. [https://doi.org/10.1016/S1470-2045\(18\)30143-8](https://doi.org/10.1016/S1470-2045(18)30143-8)
35. Spierenburg G, Grimison P, Chevreau C, Stacchiotti S, Piperno-Neumann S, Le Cesne A et al (2022) Long-term follow-up of nilotinib in patients with advanced tenosynovial giant cell tumours: Long-term follow-up of nilotinib in TGCT. *Eur J Cancer* 173:219–228. <https://doi.org/10.1016/j.ejca.2022.06.028>
36. Muller LP, Bitzer M, Degreif J, Rommens PM (1999) Pigmented villonodular synovitis of the shoulder: review and case report. *Knee Surg Sports Traumatol Arthrosc* 7(4):249–256. <https://doi.org/10.1007/s001670050158>
37. Noailles T, Brulefert K, Briand S, Longis PM, Andrieu K, Chalopin A et al (2017) Giant cell tumor of tendon sheath: Open surgery or arthroscopic synovectomy? A systematic review of the literature. *Orthop Traumatology: Surg Res* 103(5):809–814. <https://doi.org/10.1016/j.otsr.2017.03.016>
38. Mahieu X, Chaouat G, Blin JL, Frank A, Hardy P (2001) Arthroscopic treatment of pigmented villonodular synovitis of the shoulder. *Arthroscopy* 17(1):81–87. <https://doi.org/10.1053/jars.2001.7803>
39. Koh KH, Lim KS, Yoo JC (2010) Arthroscopic treatment of pigmented villonodular synovitis involving bilateral shoulders. *Orthopedics* 33(6):442. <https://doi.org/10.3928/01477447-20100429-31>
40. Gumina S, Carbone S, Campagna V, Castagna A, Della Rocca C, Giannicola G (2013) Pigmented villonodular synovitis of the shoulder associated with massive rotator cuff tear treated by arthroscopic synovectomy and debridement. *Musculoskelet Surg* 97(Suppl 1):79–84. <https://doi.org/10.1007/s12306-013-0258-z>

41. Chiang ER, Ma HL, Wang ST, Hung SC, Chen TH (2009) Arthroscopic treatment for pigmented villonodular synovitis of the shoulder associated with massive rotator cuff tear. *Arthroscopy* 25(7):716–721. <https://doi.org/10.1016/j.arthro.2009.01.007>
42. Johansson JE, Ajjoub S, Coughlin LP, Wener JA, Cruess RL (1982) Pigmented villonodular synovitis of joints. *Clin Orthop Relat Res* (163):159–166
43. Dorwart RH, Genant HK, Johnston WH, Morris JM (1984) Pigmented villonodular synovitis of the shoulder: radiologic-pathologic assessment. *AJR Am J Roentgenol* 143(4):886–888. <https://doi.org/10.2214/ajr.143.4.886>
44. Giannatos V, Ierodionou S, Koutas K, Argyropoulou E, Sakellariou E, Kokkalis ZT (2024) Pigmented Villonodular Synovitis of the Shoulder: A Case Report and Literature Review. *Am J Case Rep* 25:e944483. <https://doi.org/10.12659/AJCR.944483>
45. Schumacher HR (2010) A case of villonodular synovitis of the shoulder in an adolescent: imaging and pathologic diagnosis. *Rev Bras Reumatol* 50(4):478 author reply 9–80
46. Vj P, Muruganandam A, Perumal S, Ak S, Sivaraman A (2024) A Successful Arthroscopic Management of a Benign but Locally Aggressive Tenosynovial Giant Cell Tumour in the Shoulder. *Cureus* 16(4):e57492. <https://doi.org/10.7759/cureus.57492>
47. Ellert U, Kurth BM (2013) [Health related quality of life in adults in Germany: results of the German Health Interview and Examination Survey for Adults (DEGS1)]. *Bundesgesundheitsblatt Gesundheitsforschung Gesundheitsschutz* 56(5–6):643–649. <https://doi.org/10.1007/s00103-013-1700-y>
48. Petsatodis G, Karataglis D, Kapoutsis DB, Papadopoulos P, Christodoulou AG (2011) Hemiarthroplasty for pigmented villonodular synovitis of the shoulder: a report of two cases. *J Orthop Surg (Hong Kong)* 19(1):116–119. <https://doi.org/10.1177/230949901101900127>
49. Park JH, Park JW, Shin JS, Lee JM, Lee JI (2012) Hemiarthroplasty in a patient with pigmented villonodular synovitis of the shoulder. *Orthopedics* 35(1):e104–e107. <https://doi.org/10.3928/01477447-20111122-30>
50. Toro FG, Paulos JA, Fuentes DL, Sancy KL (2002) Total shoulder arthroplasty in pigmented villonodular synovitis: a case report. *J Shoulder Elb Surg* 11(2):188–190. <https://doi.org/10.1067/mse.2002.121632>
51. Ji JH, Shafi M, Park SE, Kim WY (2009) Subacromial bony erosion: a rare presentation of pigmented villonodular synovitis of the shoulder. *Knee Surg Sports Traumatol Arthrosc* 17(5):534–538. <https://doi.org/10.1007/s00167-009-0752-x>
52. Cheng JC, Wolf EM, Chapman JE, Johnston JO (1997) Pigmented villonodular synovitis of the shoulder after anterior capsulolabral reconstruction. *Arthroscopy* 13(2):257–261. [https://doi.org/10.1016/s0749-8063\(97\)90166-3](https://doi.org/10.1016/s0749-8063(97)90166-3)
53. Pantazopoulos T, Stavrou Z, Stamos C, Kehayas G, Hartofilakidis-Garofalidis G (1975) Bone lesions in pigmented villonodular synovitis. *Acta Orthop Scand* 46(4):579–592. <https://doi.org/10.3109/17453677508989240>
54. Golge UH, Kuyucu E, Gksel F, Kaymaz B (2015) Glenohumeral Arthrosis Treatment with Reverse Shoulder Prosthesis - Developed for Chronic Villonodular Synovitis. *EC Orthopaedics*
55. Byers PD, Cotton RE, Deacon OW, Lowy M, Newman PH, Sissons HA et al (1968) The diagnosis and treatment of pigmented villonodular synovitis. *J Bone Joint Surg Br* 50(2):290–305
56. Schwartz HS, Unni KK, Pritchard DJ (1989) Pigmented villonodular synovitis. A retrospective review of affected large joints. *Clin Orthop Relat Res* 247:243–255
57. Verspoor FGM, vdGIC M, Erik V, VRP H, vdGW T, Schreuder HWB (2013) Pigmented Villonodular Synovitis: Current Concepts About Diagnosis and Management. *Future Oncol* 9(10):1515–1531. <https://doi.org/10.2217/fon.13.124>
58. Capellen CF, Tiling R, Klein A, Baur-Melnyk A, Knosel T, Birkenmaier C et al (2018) Lowering the recurrence rate in pigmented villonodular synovitis: A series of 120 resections. *Rheumatology (Oxford)* 57(8):1448–1452. <https://doi.org/10.1093/rheumatology/key133>
59. Agrawal AC, Dash RN, Kar BK, Sakale HS (2020) Pigmented Villo-Nodular Synovitis of Shoulder Joint Leading to Humeral Head Erosion. *J Orthop Traumatol Rehabilitation* 12(2):143–146. https://doi.org/10.4103/jotr.jotr_81_20
60. Broski SM, Murdoch NM, Skinner JA, Wenger DE (2016) Pigmented Villonodular Synovitis: Potential Pitfall on Oncologic 18F-FDG PET/CT. *Clin Nucl Med* 41(1):e24–e31. <https://doi.org/10.1097/rlu.0000000000000893>
61. Cho CH, Sohn SW, Song KS, Kang CH, Min BW, Bae KC et al (2008) Extra-articular pigmented villonodular synovitis of the subacromial space. *Orthopedics* 31(12). <https://doi.org/10.3928/01477447-20081201-04>
62. Colak AF, Aksakal MF, Demirel Sarikaya K, Kaynar AF, Yalcinkaya B, Cetin A (2024) A rare cause of shoulder pain: pigmented villonodular synovitis revealed by magnetic resonance imaging. *Pain Med* 25(6):415. <https://doi.org/10.1093/pm/pnae007>
63. Costallat B, Montagner S, Amstalden E, Ferreira D, Filho A, Costallat L (2009) A case of villonodular synovitis of the shoulder in an adolescent: imaging and pathologic diagnosis. *Rev Bras Reumatol* 49(1). <https://doi.org/10.1590/S0482-50042009000100008>
64. Cotten A, Flipo RM, Mestdagh H, Chastanet P (1995) Diffuse pigmented villonodular synovitis of the shoulder. *Skeletal Radiol* 24(4):311–313. <https://doi.org/10.1007/BF00198424>
65. Dias JM, Costa MM, Duarte A, Pereira da Silva JA (2013) Localized Pigmented Villonodular Synovitis of the Shoulder: a Rare Presentation of an Uncommon Pathology. *Acta Med Port* 26(4):459–462
66. Dkhissi M, Aboutaib R, Zryouil B (1992) [Villonodular synovitis of the shoulder. Apropos of a case report]. *Acta Orthop Belg* 58(2):213–215
67. Du Y, Li L, Hu X (2024) Sonographic features of diffuse giant cell tumor of the tendon sheath in the shoulder: A case report. *J Clin Ultrasound* 52(3):338–340. <https://doi.org/10.1002/jcu.23618>
68. Elumogo CO, Kochenderfer JN, Civelek AC, Bluemke DA (2016) Pigmented villonodular synovitis mimics metastases on fluorine 18 fluorodeoxyglucose position emission tomography-computed tomography. *Quant Imaging Med Surg* 6(2):218–223. <https://doi.org/10.21037/qims.2016.01.04>
69. Flandry F, Norwood LA (1989) Pigmented villonodular synovitis of the shoulder. *Orthopedics* 12(5):715–718. <https://doi.org/10.3928/0147-7447-19890501-10>
70. Ganel A, Pritsch M, Yaffe B, Engelberg S, Apter S (1987) Villonodular synovitis of the shoulder in the elderly. *J Am Geriatr Soc* 35(7):692–695. <https://doi.org/10.1111/j.1532-5415.1987.tb04349.x>
71. Graf J, Bernd L, Pauschert R, Niethard FU (1991) Das seltene Auftreten einer villonodulären Synovialitis an beiden Schultergelenken. *Z Rheumatol* 50:46–48
72. Konrath GA, Nahigian K, Kolowich P (1997) Pigmented villonodular synovitis of the subacromial bursa. *J Shoulder Elb Surg* 6(4):400–404. [https://doi.org/10.1016/s1058-2746\(97\)90010-0](https://doi.org/10.1016/s1058-2746(97)90010-0)
73. Kwon M, Bang JY, Nam KH (2020) Rapid destruction of shoulder joint by pigmented villonodular synovitis treated by hemiarthroplasty: A case report. *Int J Surg Case Rep* 77:138–142. <https://doi.org/10.1016/j.ijscr.2020.10.128>
74. Lee K, Kim H, Lee H, Jang IT, Choi S (2021) An abrupt-onset shoulder joint subluxation and pseudoparalysis caused by intra-articular pigmented villonodular synovitis: A case report. *Jt Dis Relat Surg* 32(1):258–261. <https://doi.org/10.5606/ehc.2021.75437>

75. Lee S-J, Yoo J-C, Lim K-S (2007) Arthroscopic Treatment of Pigmented Villonodular Synovitis of the Shoulder-A Case Report. *Clin Shoulder Elb* 10(1):140–145
76. Lee M, Lee SH, Suh JS, Yang W-I, Shin K-H Outcomes of Diffuse-Type Pigmented Villonodular Synovitis (PVNS) after Open Total Synovectomy. *J Korean Bone Joint Tumor Soc.* 2010(16):27–36. <https://doi.org/10.5292/jkbjts.2010.16.1.27>
77. Levin EJ, Gannon W (1963) Diffuse villonodular synovitis of the shoulder. *Am J Roentgenol Radium Ther Nucl Med* 89:1302–1304
78. Lewis S, Edmonds L, Wolin E (2018) Hot shoulder PET/CT lesion: Unusual presentation of tenosynovial giant cell tumor. *Radiol Case Rep* 13(3):559–562. <https://doi.org/10.1016/j.radcr.2018.02.006>
79. Liang Y-x, Fang H-I, Zhong X (2019) Ultrasonic manifestations of pigmented villonodular synovitis on shoulder joint: a case report. *Guoji Yiyao Weisheng Daobao* 25(14):2371–2373
80. Loh RSN, Lim WSR, Lie DTT, Soeharno H (2025) A misdiagnosed case of shoulder pigmented villonodular synovitis – A case report. *J Orthop Rep* 100586. <https://doi.org/10.1016/j.jorep.2025.100586>
81. Mankin H, Trahan C, Hornicek F (2011) Pigmented villonodular synovitis of joints. *J Surg Oncol* 103(5):386–389. <https://doi.org/10.1002/jso.21835>
82. Mazabraud GA, Verdie C, Cheneau J (1974) [Hemopigmented villonodular synovitis of the large joints]. *Rev Chir Orthop Reparatrice Appar Mot* 60(4):265–298
83. Mukhopadhyay K, Smith M, Hughes PM (2006) Multifocal PVNS in a child—followed over 25 years. *Skeletal Radiol* 35(7):539–542. <https://doi.org/10.1007/s00256-005-0013-0>
84. Mulier T, Victor J, Van Den Bergh J, Fabry G (1992) Diffuse pigmented villonodular synovitis of the shoulder. A case report & review of literature. *Acta Orthop Belg* 58(1):93–96
85. Palmerini E, Staals EL, Maki RG, Pengo S, Cioffi A, Gambarotti M et al (2015) Tenosynovial giant cell tumour/pigmented villonodular synovitis: Outcome of 294 patients before the era of kinase inhibitors. *Eur J Cancer* 51(2):210–217. <https://doi.org/10.1016/j.ejca.2014.11.001>
86. Popov HI, Gherman C, Rogojan L, Botar-Jid C, Barna C, Fodor D (2012) Milwaukee shoulder syndrome associated with pigmented villonodular synovitis. Case report. *Med Ultrason* 14(1):67–70
87. Rajakulasingam R, Murphy J, Badreddine I, James S, Botchu R (2019) Pigmented Villonodular Synovitis Masquerading as a Metastasis: Imaging Features and Coaxial Needle Biopsy Technique. *Iowa Orthop J* 39(2):73–75
88. Rezaee A, Chen W, Dilsizian V, Chen Q, Kimball AS (2015) Giant Cell Tumor of the Tendon Sheath With Discordant Metabolism as a False Positive on Staging of Mantle Cell Lymphoma. *Clin Nucl Med* 40(10):814–815. <https://doi.org/10.1097/rlu.0000000000000861>
89. Sawmiller CJ, Turowski GA, Sterling AP, Dudrick SJ (1997) Extraarticular pigmented villonodular synovitis of the shoulder: a case report. *Clin Orthop Relat Res.* 335:262–267
90. Sayegh ET, Wilk RM (2021) Pigmented Villonodular Synovitis of the Glenohumeral Joint and Biceps Tendon Sheath. *Cureus* 13(4):e14529. <https://doi.org/10.7759/cureus.14529>
91. Seiler H, Seeliger H, Tager B, Boos H (1979) [A contribution to pigmented villonodular synovitis of the shoulder joint (author's transl)]. *Z Orthop Ihre Grenzgeb* 117(3):376–382
92. Selby L, Kukar M, Wang J, Beg M, Sullivan J (2014) Pigmented villous nodular synovitis mimicking metastatic melanoma on PET-CT. *Int J Surg Case Rep* 5(5):231–233. <https://doi.org/10.1016/j.ijscr.2014.02.009>
93. Sher M, Lorigan JG, Ayala AG, Libshitz HI (1990) Case report 578: Pigmented villonodular synovitis of the shoulder. *Skeletal Radiol* 19(2):131–133. <https://doi.org/10.1007/BF00197622>
94. Simmer Filho J, Ghidetti THC, Kautsky RM (2024) Localized Pigmented Villonodular Synovitis in the Shoulder: Report of a Case Treated through Arthroscopy. *Rev Bras Ortop (Sao Paulo)* 59(Suppl 2):e188–e93. <https://doi.org/10.1055/s-0044-1779313>
95. Sipahioğlu S, Zehir S, Aşkar H, Ozkanlı U (2011) [Diffuse pigmented villonodular synovitis in the shoulder joint and the biceps tendon: a case report]. *Eklemler Hastalıkları Cerrahisi* 22(3):172–176
96. Snook GA (1963) Pigmented villonodular synovitis with bony invasion. A report of two cases. *JAMA* 184:424–425. <https://doi.org/10.1001/jama.1963.73700180010016b>
97. Sotje G, Jensen H, Pruss H, Baron Y, Janig U (1992) [Pigmented villonodular synovitis of the shoulder joint in childhood]. *Aktuelle Radiol* 2(3):162–165
98. Tang K, Zheng X, Lin J, Wang L (2019) Diffuse-type tenosynovial giant cell tumor of the shoulder evaluated by FDG PET/CT. *Clin Nucl Med* 44(4):310–312
99. Tong KM, Hsu KC, Lee TS, Chang SM (1994) Diffuse pigmented villonodular synovitis of the shoulder: a case report. *Zhonghua Yi Xue Za Zhi (Taipei)* 53(3):188–192
100. Zhao L, Zhou K, Hua Y, Li Y, Mu D (2016) Multifocal pigmented villonodular synovitis in a child: A case report. *Med (Baltimore)* 95(33):e4572. <https://doi.org/10.1097/MD.0000000000004572>

Publisher's note Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.