CASE REPORT

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

Casper Falster,¹ Simon Stockmann Poulsen,² Uffe Joergensen²

¹Department of Molecular Endocrinology and Stem Cell Research Unit (KMEB), Odense University Hospital, Odense, Denmark

²Department of Orthopedic Surgery and Sports Traumatology, Odense University Hospital, Odense, Denmark

Correspondence to

Casper Falster,
cfalster@health.sdu.dk

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SUMMARY

Localised pigmented villonodular synovitis (PVNS) of the knee is a rare diagnosis, with clinical signs and symptoms mimicking meniscal damage or other common knee injuries.

We report the case of a 24-year-old female soccer player, seeking treatment after 7 months of persisting knee pain. Additionally, we present an overview of tenosynovial giant cell tumours.

On examination, the patient was found to have tenderness in the medial joint space of the knee. MRI revealed a heterogeneous formation in the central part of the knee. The formation was completely enucleated arthroscopically, histological analyses confirmed the diagnosis of localised PVNS. The patient was subsequently free of symptoms with no signs of recurrence on MRI and had resumed soccer practice at the 1-year follow-up appointment.

BACKGROUND

Tenosynovial giant cell tumours (TSGCTs) are rare and present themselves with various symptoms which may mimic traumatic injuries during objective assessment. The nomenclature surrounding TSGCT varies from author to author; however, there appears to be a consensus that the term TSGCT encompasses four subtypes (table 1). Although similar in several aspects, the different subtypes are important to acknowledge as they exhibit dissimilar rates of recurrence and may require different treatment regimens according to the examined literature.

CASE PRESENTATION

A 24-year-old female soccer player presented with medially localised pain of the right knee. The pain had appeared in the months following a skiing accident, where the patient lost her balance and fell forward with both skis stuck in the snow; she had not felt any rotation of the knee joint or subsequent looseness. In the following days, the knee felt tender, but she was able to resume skiing the day after the accident. Otherwise, no history of prior trauma to the knee. The pain had progressed in the following months, preventing her from participating in soccer practice.

On examination, the knee presented with discrete swelling but no warmth or redness.

A standard knee examination was performed, including stability testing, palpation and tests to provoke pain. The patient reported tenderness during palpation of the posteromedial joint line along with discomfort during Apley's test, with rotation and compression posteromedially. Additionally, the patient reported a sensation of movement restriction or locking when the knee was fully flexed. Based on these findings, the tentative diagnosis was a meniscal lesion.

INVESTIGATIONS

In order to confirm the diagnosis, the patient was referred to MRI in accordance with the guidelines of the Danish Society of Radiology. The scan revealed an intact meniscus. However, a cystic formation presenting with a heterogeneous signal intensity was found in close contact with the synovial membrane in the central part of the knee, posterior to the posterior cruciate ligament (PCL) (figure 1). The tumour, measuring 2.8×3.2×1.8 cm, was protruding posteriorly, seemingly adding pressure to the contents of the popliteal fossa (figure 2). However, no neurovascular symptoms were observed.

TREATMENT

After tumour conference, the preoperative plan was arthroscopic removal if possible, otherwise conversion to open surgery. Due to its appearance on the MRI, the possibility of the tumour being of malignant nature was considered minimal. No biopsy was taken preoperatively, as this would have no clinical consequence.

Using an intercondylar approach, with an anterolateral and anteromedial arthroscopic entry point for the scope and probe, respectively, the tumour presented itself with a firm, yellow texture and vascularisation (figure 3). The localisation was posteromedially in the joint, situated in its own capsule and exhibiting adherence to the PCL in its entirety, in accordance with the MRI.

A posteromedial entry point was established for the shaver, and the synovium on the anterior part of the tumour was removed, revealing the firmer tumour tissue. Utilising the blunt side of the shaver, the tumour was loosened completely

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Figure 1 T1-weighted MRI showing an intra-articular localised, heterogenous formation and measuring 2.8×3.2×1.8 cm.

from the posterior part of the capsule and could be enucleated without breaking the capsule. The tumour was subsequently sent for pathological examination, where microscopic evaluation revealed a dense fibrous tissue with accumulation of histiocytes and multinucleated giant cells, in addition to scattered depositions of haemosiderin. According to the pathologists examining the tumour, these findings were consistent with the diagnosis of localised TSGCT.

OUTCOME AND FOLLOW-UP

At 6 months and 1-year postsurgery, the patient was seen for follow-up. The recovery had so far been propitious; the patient experienced no pain and walked without a limp. The knee exhibited no passive or active restriction of motion, no locking or popping and was stable in all directions. No palpable mass or tenderness were present and MRI showed no signs of recurrence. Thus, the patient had been able to slowly resume her soccer practice.

DISCUSSION

The following section provides a brief summary following the guidelines of the WHO from 2003,¹ stating that TSGCT presents as a localised or diffuse type. Furthermore, the work of Lucas² is utilised, subcategorising localised and diffuse types of

Figure 2 T2-weighted MRI. The tumour is protruding posteriorly, seemingly adding pressure to the content of the popliteal fossa. However, no neurovascular symptoms were observed.

TSGCT depending on site of origin, being either tendon sheath or intra-articular (table 1).

Subclassifications and clinicopathology of TSGCT

Giant cell tumours of the tendon sheath (localised TSGCT)

First described by Jaffe and Lichtenstein in 1941, the giant cell tumours of the tendon sheath (GCTTS) are localised, benign lesions and the most common of the TSGCTs. These tumours are most likely to occur in individuals between the age of 30 and 50 years, more often in women than men.³ As the name implies, GCTTS arise from synovial-lined tendon sheaths, almost

Figure 3 The tumour, seen arthroscopically, presenting itself with a firm, yellow texture and vascularisation.

Table 1 Classification of tenosynovial giant cell tumours (TSGCTs)

Classification of TSGCTs		
Site of origin	Localised	Diffuse
Tendon sheath/bursa	GCTTS	Diffuse-type GCT
Intra-articular	Localised PVNS	Conventional PVNS

GCT, giant cell tumour; GCTTS, giant cell tumour of the tendon sheath; PVNS, pigmented villonodular synovitis.

exclusively presenting on the digits of the hand.^{3,4} Despite these facts, rare cases occasionally emerge, reporting GCTTS in the knee joint of young patients.^{5–7} The aetiology of GCTTS is uncertain but has been ascribed to trauma, inflammation, aneuploidy and clonal chromosomal abnormalities.^{8,9}

The diagnosis of GCTTS may be difficult, especially in large joints where the symptoms are often non-specific and scarce, and are therefore easily mistaken for common injuries such as meniscal tears or other internal knee derangements. Plain radiographs are usually not contributing to the diagnosis. MRI, however, is effective and highly sensitive for diagnosing GCTTS. T1-weighted and T2-weighted images will often present with a homogenous low signal intensity, due to the dense collagen and haemosiderin-laden macrophages.¹⁰ Histologically, all TSGCT are identical in terms of cellular composition, presenting with a polymorphous population of multinucleated osteoclast-like giant cells, epithelioid histiocytes, mononuclear stromal cells and haemosiderin depositions.²

Treatment consists of careful and complete excision. Additionally, due to frequent recurrence, postoperative radiotherapy has been recommended as adjuvant therapy, reportedly reducing local recurrence considerably from 25%–45% to 4%.¹¹

Localised pigmented villonodular synovitis (localised and intra-articular TSGCT)

The localised pigmented villonodular synovitis (PVNS) is considered benign and morphologically identical to GCTTS. It presents as a circumscribed, occasionally pedunculated, intra-articular mass and arising from the synovium.^{2,12} Reportedly, localised PVNS occurs in individuals of the same age range as GCTTS; however, the incidence has also been described as predominant in older patients, in their fourth to sixth decade of life.¹³

According to Myers and Masi, the incidence of PVNS collectively is 1.8 per million in the general population, the localised form accounting for 15%–25% of the cases.¹⁴

Unlike GCTTS, localised PVNS occurs almost exclusively in the knee, but with equally diffuse symptoms, encompassing discomfort, swelling, pain and locking alike.^{13,15} Utilising MRI, the localised PVNS exhibits heterogenous low signal intensity, owing to high haemosiderin content in xanthoma cells, rich collagen proliferation and a reduction in the T2 relaxation time.¹⁰

There is currently no consensus on optimal treatment of localised PVNS, but it is generally accepted that arthroscopic excision often results in successful treatment.¹³ As localised PVNS is a rare disorder, the recurrence frequency has not been definitively established; however, a review of long-term follow-up studies on arthroscopically treated localised PVNS by Dines *et al.*¹⁶ reported no recurrences in patients followed for up to 4.5 years post-operatively, hinting that arthroscopic treatment alone might be sufficient.

Conventional PVNS (diffuse and intra-articular TSGCT)

In contrast to the localised forms of TSGCT which are circumscribed with a nodular growth pattern, the diffuse forms present as locally aggressive, infiltrative processes with diffuse and villous growth patterns. Also, although very rarely, diffuse-type TSGCT are capable of malignant transformation.¹⁷

The conventional PVNS occurs in the same age range as the localised PVNS, but is more common, constituting 75%–85% of the 1.8 per million incidence in the general population.¹⁴ Any joint can be involved, although the knee is most common and occasionally extra-articular foci are seen. In which cases, the

synovium appears brown and is diffusely covered by villous and coarse nodular outgrowths.¹⁸

Conventional PVNS often presents with knee swelling, diffuse pain and stiffness. The clinical course often consists of recurrent knee effusions without related trauma over a long period.¹⁹

MRI findings are usually identical to the localised form; however, due to the locally aggressive nature of conventional PVNS, subchondral cysts and bone erosions are seen in 25% of affected knees. These findings are thought to be secondary to the hyperplastic villous formations, extending through vascular foramina and into cortical bone.²⁰

Due to the diffuse presentation, treatment of conventional PVNS is complicated with several different treatment combinations. In primary intra-articular only cases, arthroscopic total synovectomy is recommended in order to minimise chance of recurrence. Total synovectomy differs from partial synovectomy in that it includes synovectomy of the posterior compartments. If extra-articular foci are present, combined arthroscopic and open synovectomy is recommended in the thorough work of Kramer *et al.*¹⁹ Following surgery, recurrence rates are reported to range from 8% to 18%. Some authors even report as high as 50%.^{2,21,22} If the recurring conventional PVNS presents with preserved articular surfaces, total synovectomy can be repeated and adjuvant beam radiation therapy should be considered, as evidence suggests reduced recurrence.²³ Lastly, if the recurrence presents with significant articular degeneration, a total knee replacement should be considered.

Diffuse-type giant cell tumours (diffuse and extra-articular TSGCT)

The diffuse-type giant cell tumour (GCT) is defined by the presence of poorly confined tissue mass, either isolated or in association with an articular component, and represents only a small proportion of TSGCT, allegedly 5%–15%.^{4,24} Although defined as extra-articular, arising from tendon sheath or bursa, a significant number of cases are believed to represent extra-articular extensions of primary intra-articular lesions.²⁵

The most commonly involved site is the knee, followed by the foot and ankle. Like its localised counterpart, the GCTTS, diffuse-type GCT tends to occur in relatively young patients with a slight female predominance.²⁵ In contrast to the other described TSGCT, pain is usually not a debut symptom of diffuse-type GCT, in a review of 50 cases by Somerhausen and Fletcher²⁵ pain was only described in 14%. Instead, most patients presented with a painless, slowly growing soft tissue swelling with no apparent adhesion to any articular or tendinous structure.

As both intra-articular and extra-articular sites are often involved, an MRI image demonstrating joint invasion and a concurrent weak signal of T1 and T2, should lead to the consideration of diffuse-type GCT as a probable diagnosis.²⁶

Histologically, diffuse-type GCT exhibits the same polymorphous cell population as other TSGCT, however, unlike the localised GCTTS, diffuse-type GCT aggressively entraps and infiltrates adjacent soft tissue and frequently erodes bone. Also, pseudoalveolar spaces and cellular areas devoid of giant cells are often observed.^{2,25}

The optimal treatment of diffuse-type GCT is unclear. Surgical excision is currently considered the principal method, however, both open surgery and arthroscopy are used, and synovectomies can be either partial or complete. In severe cases, total joint replacement is appropriate. Additionally, both external beam radiotherapy and radiosynovectomy, utilising the

instillation of 90-Yttrium, have been reported to reduce the rate of recurrence.²⁷

Malignant TSGCT

Malignant TSGCT is very rare, with fewer than 50 cases reported in English literature.¹⁷ First, described by Enzinger *et al*²⁴ as uncontrolled growth within a benign lesion or as the continued recurrence of a previously benign lesion.

Even with aggressive treatment, consisting of both surgical resection, chemotherapy and radiation, the prognosis is poor, with a median survival of 22.5 months after diagnosis. Metastasis is seen within 4 months to 5 years, with a median of 11 months. Most common sites are regional lymph nodes and lungs.¹⁷ Malignant TSGCT are often radiation associated, occurring years after radiotherapy for diffuse lesions.² Histologically, they often exhibit large polygonal cells with eosinophilic cytoplasm. Trisomy 5 and 7 have been described.²⁸

CONCLUSION AND FUTURE PERSPECTIVES

The question of whether the tumour reported in the case of the 24-year-old female soccer player was a GCTTS or a localised PVNS still remains.

As the tumour arose from the synovial membrane, showed a heterogeneous signal on MRI and no signs of recurrence were observed at follow-up, localised PVNS seems to be the most plausible diagnosis. Naturally, it is important to keep in mind, that a follow-up period of 1 year is brief and a recurrence might not be detectable at this point.

During our review of the literature surrounding the four subtypes of TSGCT, no definitive aetiology was described, although both trauma, inflammation, neoplastic changes and viral infections have been hypothesised to contribute to the development.⁸⁻⁹ However, the times they are a changing, and most recent findings regarding the aetiology of TSGCT suggest that a majority of tumours contain translocations involving chromosome 1p13, at which breakpoint the gene *CSF1* (*macrophage colony-stimulating factor 1*) is present, resulting in overexpression of *CSF1*.²⁹

In the TSGCT carrying the translocation, it is only present in a minority of intratumoural cells, resulting in overexpression of *CSF1* only in these cells. The bulk of the cells, however, express *CSF1R*, but not *CSF1*.²⁹ This suggests that most TSGCT are in fact clonal neoplastic proliferations driven by overexpression of *CSF1*, leading to recruitment of *CSF1R*-bearing macrophages which make up the bulk of the tumour, through a tumour-landscaping effect.

Researchers have since used this knowledge, in an attempt to treat TSGCT. In 2008, Blay *et al*³⁰ reported a complete remission in a 34-year-old female, with recurrent right elbow TSGCT treated with imatinib. Based on this report, Snoots *et al*³¹ attempted the same approach on a 35-year-old male with recurrent hip TSGCT and observed a stable reduction in tumour size. In 2010, Cheng *et al*³² demonstrated, using a xenograft TSGCT model, that an anti-*CSF1* monoclonal antibody significantly inhibits macrophage infiltration into the tumour, thus supporting clinical trials of humanised agents and anti-*CSF1R* drugs in TSGCT refractory to conventional treatment. At present time, a number of clinical trials are investigating the use of various *CSF1R* inhibitors for the treatment of TSGCT.²⁷

In conclusion, TSGCT of the knee often present with diffuse symptoms and can easily be misdiagnosed as meniscal damage or tear of a ligament. Thus, if no history of trauma is present, such cases should undergo thorough investigation, both clinically and

radiologically. Also, correct diagnosis of the TSGCT subtype is crucial in order to initiate optimal treatment modalities and follow-up regimen.

Patient's perspective

I first experienced problems bending my right knee in April 2015, I wasn't able to squat all the way down. I thought it had something to do with a skiing accident in March 2015. I ran a half marathon in May 2015 but felt like my right knee started to become thicker than my left knee. So I figured something was wrong and it wasn't going away. Since I was a bit lazy, I didn't get a doctor's appointment until October 2015. She gave me some kind of medicine for 14 days to make my knee become thinner again, and she asked me to take it easy for those days. That didn't help and she referred me to the orthopaedic department at Odense University Hospital. Six weeks later, around December 2015, I was examined at the orthopaedic department by chief physician Uffe Joergensen. He decided that I needed an MRI. Six weeks later, in January 2016, I got an MRI. Four weeks later, in February 2016, I was back with Uffe Joergensen, who explained that I had some tissue behind my knee looking like a little egg, which he'd have to remove. Three weeks later, in March 2016, I got surgery, which went fine. I used crutches for 2–3 days. Since I had to work, I went to work by bike, after a week. Not easily, but possible. After 4 weeks, I started swimming with a swimming flipper around my right foot. After 6 weeks, I started light running. And after 3 months, I started strength workout. After about 8 months, I was able to squat all the way down. Maybe I could have done that before, but my head wasn't there mentally. Today, 10 months after the surgery, I never notice that I've had challenges with my right knee.

I've had a good experience with the service of being operated in. I'm satisfied with the good result, and happy that I could get surgery within 3 weeks.

Learning points

- Consider MRI if a suspected diagnosis of meniscal damage or tear of ligament presents with no history of trauma.
- Arthroscopic removal of tumour with a favourable outcome is possible. However, due to high rates of recurrence in some subtypes, adjuvant radiation therapy should be considered.
- Correct diagnosis of tenosynovial giant cell tumour subtype is crucial, as treatment regimens and recurrence rates differ.
- All tenosynovial giant cell tumours share the same histological composition.
- New treatment options, utilising anti-*CSF1* monoclonal antibodies, are being investigated in clinical trials.

Contributors The authors declare that all authors have been equally involved in conceiving and writing this case report. As the primary author, CF has been in charge of contact with the patient in question; in addition, he has written the majority of the case report and has examined the literature in conjunction with second author, SSP. The last author, UJ, has provided support and guidance regarding literature search and scientific writing. All authors have approved the final version for publishing and agreed to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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