



## A CASE REPORT: AN INTRICATE RADIOLOGICAL POTRAIT OF PIGMENTED VILLONODULAR SYNOVITIS: CROSS- SECTIONAL INSIGHT

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### ABSTRACT

Pigmented Villonodular Synovitis is an infrequent, benign proliferative disease of the synovial membrane that mainly affects large joints such as the knee. This case report presents a 46-year-old male with advanced right knee pain, swelling, and mechanical symptoms over more than a few months. Initial clinical assessment recommended intra-articular pathology without systemic involvement. MRI exposed diffuse synovial thickening with characteristic low signal intensity on T1- and T2-weighted images and blooming on GRE sequences due to hemosiderin deposition, confirming the diagnosis of diffuse PVNS. Arthroscopic synovectomy was performed using multiple portals to access the anterior and posterior compartments. This case importance of early MRI in diagnosing PVNS, the utility of arthroscopy for minimally invasive management, and the need for long-term follow-up due to recurrence risk. Advances in targeted therapy, such as CSF1R inhibitors, offer promise for recurrent or inoperable cases.

**KEYWORDS:** Pigmented Villonodular Synovitis, Knee Joint, Arthroscopic Synovectomy, Synovial Proliferation, MRI, CSF1R Inhibitors, Hemosiderin, Recurrence.

### Introduction

Pigmented Villonodular Synovitis (PVNS), currently referred to as tenosynovial giant cell tumor, is an uncommon proliferative disorder of the synovium affecting joints, bursae, and tendon sheaths. Histologically, it is characterized by synovial hyperplasia, hemosiderin deposition, lipid-laden macrophages, and multinucleated giant cells. Although considered a benign entity, PVNS demonstrates locally aggressive behavior with a significant risk of recurrence, particularly in its diffuse form. The knee joint is most commonly involved, accounting for approximately 80% of cases, followed by the hip, ankle, shoulder, and elbow joints[5].

The term PVNS was first introduced by Henry L. Jaffe

in 1941[1]. Two main forms are recognized: localized and diffuse. The localized form typically presents as a well-circumscribed intra-articular or extra-articular lesion, whereas the diffuse form involves extensive synovial proliferation and may lead to progressive joint destruction if untreated[2,3].

The exact etiology remains uncertain, with proposed mechanisms including inflammatory, reactive, and neoplastic processes. Recent molecular studies have demonstrated overexpression of colony-stimulating factor 1 (CSF1) due to chromosomal translocations, supporting a neoplastic origin[4].

PVNS most commonly affects individuals between 20 and 50 years of age. Epidemiological studies suggest no strong

gender predilection overall, although slight variations exist between localized and diffuse forms[2]. Clinically, patients present with insidious onset of joint pain, swelling, stiffness, and reduced range of motion. These nonspecific features often mimic other joint pathologies such as osteoarthritis, meniscal injury, rheumatoid arthritis, and septic arthritis, leading to delayed diagnosis[6].

Magnetic Resonance Imaging (MRI) is the imaging modality of choice for PVNS. It demonstrates characteristic findings including synovial thickening, joint effusion, and low signal intensity on both T1- and T2-weighted images due to hemosiderin deposition, with blooming artifacts on gradient-echo sequences[7].

Management depends on disease extent. Localized lesions are usually treated with arthroscopic excision, while diffuse disease often requires total synovectomy via arthroscopic, open, or combined approaches. Recurrence remains a challenge, particularly in diffuse PVNS, with reported rates up to 30–45%[8,9]. Advances in molecular biology have led to targeted therapies such as CSF1 receptor inhibitors, notably pexidartinib, offering new treatment options in unresectable or recurrent disease[10].

We present a case of diffuse PVNS of the knee, highlighting the role of MRI in diagnosis and management.

## Case Report

A 46-year-old male accessible to the orthopaedic outpatient health centre with a primary complaint of determined pain and swelling in the right knee, which had increasingly deteriorated over 7 to 8 months. The patient, otherwise healthy with no important comorbidities, reported that the discomfort began unkindly, without any identifiable traumatic incident. The activity-induced pain was first irregular, mild. However, over time, it became more determined and simpler, mainly during activities involving continued standing or knee flexion, such as climbing stairs or sitting on your heels. The patient also reported a clear decrease in knee flexibility, occasional joint locking, and a sense of fullness around the joint.

There systemic illness was no history of fever, chills, night sweats, weight loss, or signs suggestive of infection. There was no history of joint disorders, tuberculosis, or previous surgeries. The patient had no similar complaints in other joints, and there was no family history of arthritis or connective tissue disease. He was a non-smoker and did not consume alcohol. He worked in an administrative job and was otherwise sedentary.

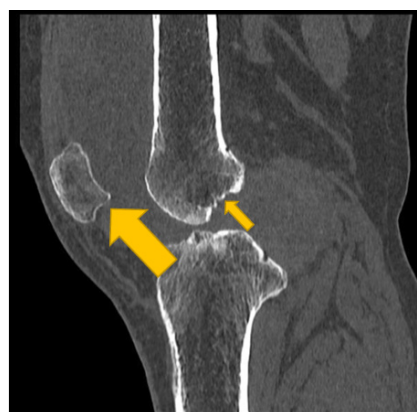
On physical investigation, the patient was afebrile and hemodynamically stable. Inspection of the right knee exposed a mild to moderate effusion and fullness over the suprapatellar region. The covering skin was normal, with no erythema or warmth. Palpation established the presence of effusion, and the patellar tap test was positive. There was sensitivity over the medial joint line and the posterior

knee region. The knee exhibited limited range of motion, mainly at the terminal flexion arc, and mild allowance lag. Ligamentous stability tests, including Lachman, anterior/posterior drawer, and varus-valgus stress, were negative. The right lower limb was normal in the Neurovascular examination. The contralateral knee or other joints showed no abnormalities.

The initial normal results of CBC (Complete Blood Count), Rh factor, ESR (Erythrocyte Sedimentation Rate), C-Reactive Protein and anti-cyclic citrullinated peptide antibodies and negative signs and serologic markers implied reduced inflammation. The absence of systemic signs and negative serologic markers made inflammatory arthritis less probable. An MRI of the right knee joint was completed to assess the amount of joint pathology on clinical examination.

The MRI established a well-preserved joint arrangement with no fractures or dislocations. Multiple marginal osteophytes were noted in the visualised femur, tibia, and patella, suggesting early osteoarthritic changes. There was diffuse, irregular thickening of the synovial membrane, particularly in the posterior compartment and suprapatellar pouch. The thickened synovium exhibited multiple villous and nodular projections. These T1-weighted image areas had low signal intensity, and T2-weighted sequences had intermediate signal. Blooming artefacts were seen on gradient echo sequences, suggestive of hemosiderin deposition. A large joint effusion was observed extending into the suprapatellar bursa, displacing Hoffa's fat pad and causing mass effect.

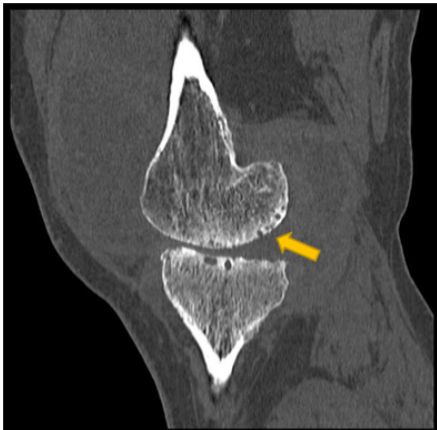
Figure1 demonstrates well-defined sub-chondral pressure erosions with sclerotic margins involving the posterior aspect of the medial femoral condyle as shown by the small yellow arrow, consistent with extrinsic cortical erosion due to chronic synovial proliferation. Associated adjacent sub-chondral sclerosis is noted. An osteophyte is seen along the inner (articular) surface of the patella as shown in three big yellow arrow, suggestive of secondary degenerative changes. There is mild anterior displacement of the patella likely due to joint effusion.



**Figure 1.** Sagittal bone window CT image of the knee

Numerous nodular synovial masses were seen impinging on the adjacent articular cartilage of the distal femur and proximal tibia. Cortical erosions were noted in these areas, mainly posteriorly. A concurrent CT scan revealed multiple subchondral cysts and areas of sclerosis along the articular margins, consistent with chronic degenerative changes.

Figure 2 demonstrates well-defined subchondral pressure erosions with sclerotic margins involving the medial femoral condyle and medial tibial plateau, consistent with extrinsic cortical erosions due to chronic synovial proliferation as shown in the yellow arrow. Associated adjacent sub-chondral sclerosis is noted.



**Figure 2. Parasagittal bone window CT image of the knee**

Figure 3 demonstrates well-defined sub-chondral pressure erosions with sclerotic margins involving the medial compartment, encircled in the yellow circle, including the medial femoral condyle and medial tibial plateau, consistent with extrinsic cortical erosions due to chronic synovial proliferation. Associated adjacent sub-chondral sclerosis is noted.

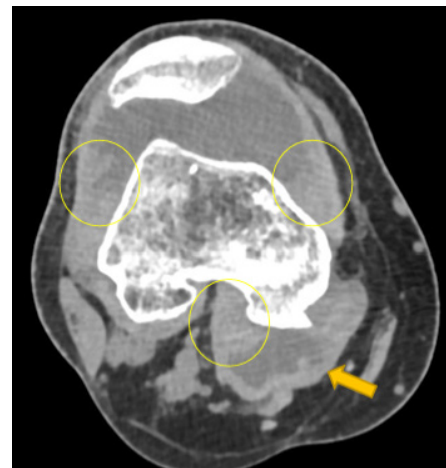


**Figure 3. Coronal reformatted CT image of the knee**

The anterior cruciate ligament established intra-substance hyperintensity at the tibial attachment, but the femoral origin could not be assessed due to overlying synovial tissue. The medial and lateral collateral ligaments appeared normal and the posterior cruciform ligament was intact, even though a small fluid collection was noted adjacent

to the medial collateral ligament. The visualisation of the medial and lateral menisci was limited due to extensive synovial overgrowth, but no definite meniscal tears could be appreciated.

Figure 4 demonstrates hyperdense synovial proliferation within the bilateral parapatellar recesses and posterior joint recess as shown by the yellow circles, with mild diffuse synovial thickening as shown by the yellow arrow. No internal calcifications or mineralization are identified within the proliferative tissue. Findings are suggestive of synovial proliferative pathology, in keeping with pigmented villonodular synovitis (PVNS).



**Figure 4. Axial Non-contrast CT image, soft tissue window of the knee at the level of the mid-patella**

Soft tissue examination in the surrounding muscles exposed mild oedema. The popliteal tendon, neurovascular bundle, and lymph nodes were average. The hyperintense signals in the subcutaneous tissues are suggestive of localised oedema. Based on a Pigmented Villonodular Synovitis diagnosis, of long-winded condition of the right knee was made on the radiological results. Secondary degenerative osteoarthritic changes were also noted.

The patient was advised to continue regular follow-up every 6 months. He was informed about the potential for recurrence, especially given the diffuse nature of the disease. This case highlights the classical features and successful management of diffuse PVNS, underscoring the status of early diagnosis and complete treatment planning.

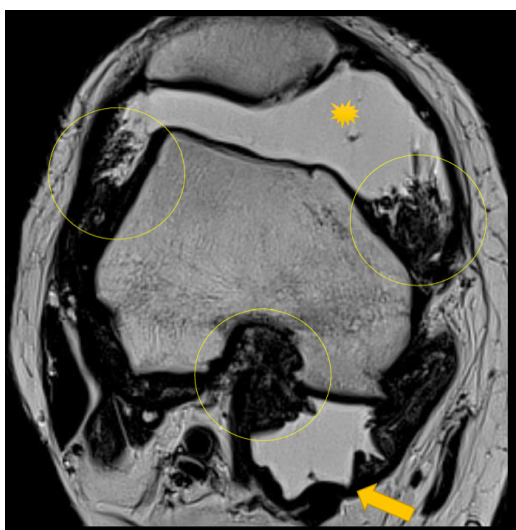
## Discussion

Pigmented Villonodular Synovitis is a rare, benign but locally destructive proliferative disorder of the synovial membrane. It most frequently affects large joints such as the knee, followed by the hip, ankle, and shoulder. The condition is characterised histologically by synovial hyperplasia, hemosiderin deposition, foamy histiocytes, and multinucleated giant cells. The aetiology remains indeterminate, but several theories have been proposed, including long-lasting inflammation, trauma-induced proliferation, and, more recently, neoplastic origins involving

chromosomal translocations and CSF1 gene overexpression [11]. Our case exemplifies a typical presentation of diffuse PVNS of the knee, manifesting with non-specific symptoms such as progressive pain, swelling, and restricted range of motion.

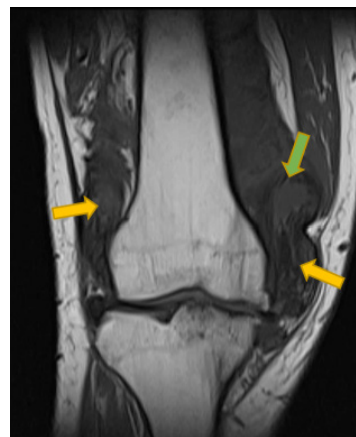
The frequency of PVNS is estimated at 1.8 per million per year, with the knee being the most frequently involved site. Diffuse PVNS tends to affect a younger demographic, typically in the third to fifth decades of life. Clinically, PVNS frequently mimics other joint disorders, such as internal derangement, meniscal injury, or inflammatory arthropathy, which can delay diagnosis. In our patient, the absence of systemic symptoms and normal serological markers helped exclude rheumatoid arthritis and infectious etiologies. The chronicity of symptoms and the presence of mechanical joint locking prompted advanced imaging [12].

MRI is the imaging sensory system of choice for diagnosing PVNS. It proposes outstanding soft tissue contrast and can establish the pathognomonic features of the disease. In this case, the MRI results of diffuse synovial thickening, low T1 and T2 signal strengths due to hemosiderin deposition, and blooming artefacts on GRE sequences were strongly suggestive of PVNS. The presence of erosions and pressure-related bone changes further supported the diagnosis of the diffuse form. These imaging characteristics not only aid in diagnosis but also assist in preoperative planning by defining the amount of synovial involvement [13]. In our study, the figure 5 demonstrates diffuse synovial proliferation within the bilateral parapatellar recesses and posterior joint recess, appearing predominantly low to intermediate signal intensity as shown in the yellow circles. Associated mild synovial thickening is noted as shown in the yellow arrow. There is moderate joint effusion as shown by the yellow asterisk. The low signal intensity is attributable to hemosiderin deposition, characteristic of pigmented villonodular synovitis (PVNS).



**Figure 5. MRI Knee Axial T2weighted non-fat-suppressed image of the knee at the level of the mid-patella**

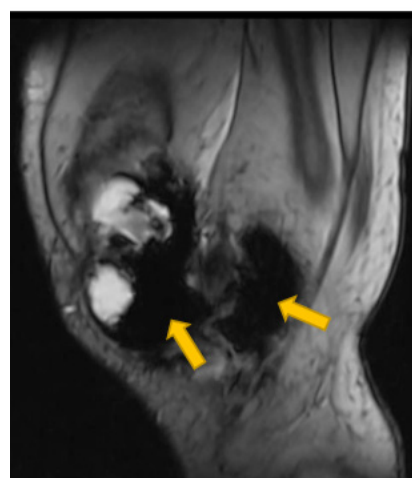
Figure 6, showed the diffuse synovial proliferation involving the medial compartment as well as the parapatellar and posterior joint recesses, appearing predominantly isointense to skeletal muscle as shown by yellow arrows. Associated mild synovial thickening is noted as shown by green arrow. Findings are consistent with synovial proliferative pathology, in keeping with pigmented villonodular synovitis (PVNS).



**Figure 6. MRI Knee Coronal T1-weighted non-fat suppressed image of the knee**

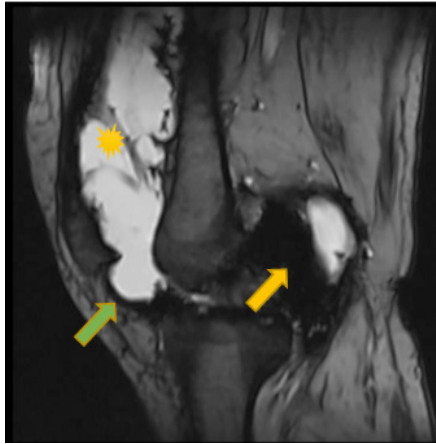
Histopathological examination remains the gold standard for definitive diagnosis. In our case, histology confirmed diffuse PVNS with characteristic features such as papillary and nodular synovial architecture, hemosiderin-laden macrophages, multinucleated giant cells, and chronic inflammatory cells. No malignant features were noted. The absence of granulomatous inflammation helped to rule out tuberculous synovitis, which is an applicable differential in endemic regions [14].

Figure 7 demonstrates marked blooming within the intrasynovial proliferative mass, corresponding to hemosiderin deposition as shown by the yellow arrows. This susceptibility effect is characteristic of pigmented villonodular synovitis (PVNS) and supports the diagnosis.



**Figure 7. MRI Knee Parasagittal gradient-echo (GRE)/susceptibility weighted image**

Figure 8 demonstrates marked blooming within the intra-synovial proliferative mass, corresponding to hemosiderin deposition as shown by the yellow arrow and diffuse synovial thickening shown by the green arrow. There is moderate joint effusion as shown by the yellow asterisk. This susceptibility effect is characteristic of pigmented villo-nodular synovitis (PVNS) and supports the diagnosis.



**Figure 8. MRI Knee Parasagittal gradient-echo (GRE)/susceptibility**

Treatment of PVNS primarily involves surgical synovectomy, either through an open or arthroscopic approach. Arthroscopic synovectomy is generally preferred for knee involvement due to its minimally invasive nature, faster recovery, and better cosmetic outcomes. However, achieving complete synovial excision arthroscopically can be stimulating, especially in the posterior compartments of the joint[15]. In our case, a combined anterior and posterior portal approach allowed for near-total excision of diseased tissue, resulting in good symptomatic relief and functional improvement.

In spite of surgical management, PVNS is dishonourable for its high recurrence rates, mainly in the diffuse form. Recurrence rates range between 30% and 45%, depending on the extent of synovial involvement and completeness of excision. For this reason, adjuvant therapies such as radiotherapy have been considered in cases of incomplete excision or recurrence. External beam radiation or radio synovectomy using isotopes like Yttrium-90 has shown some promise in reducing recurrence rates, although long-term safety, especially in younger patients, remains an apprehension[2].

Current advances in molecular biology have led to the development of targeted therapies for PVNS. CSF1R inhibitors, such as pericarditis, have shown efficacy in patients with unresectable or recurrent diffuse PVNS. These drugs inhibit the CSF1 pathway, which is aberrantly activated in PVNS due to translocations involving the CSF1 gene. While promising, these therapies are typically reserved for cases that are refractory to surgical treatment or in patients where surgery poses important dangers[16].

Our patient's postoperative course was ordinary, with good pain relief and functional recovery noted at follow-up. At six months, there were no signs of recurrence, although he remains under surveillance. Regular follow-up with clinical examination and periodic imaging is essential for early detection of recurrence. Lasting studies are necessary to assess functional consequences and disease-free endurance in PVNS patients treated with arthroscopic synovectomy alone[1].

PVNS remains an analytic task due to its non-specific clinical presentation. A high index of suspicion, especially in young to middle-aged patients with chronic monoarthritic conditions, is serious for early detection. MRI is irreplaceable for diagnosis and surgical planning, while histopathology approves the disease. Arthroscopic synovectomy proposes a dependable treatment sensory system for diffuse PVNS of the knee, though vigilance for reappearance is mandatory. Continuing investigation into adjuvant and molecular therapies may in addition improve management consequences in the upcoming[3].

## Conclusion

Pigmented Villonodular Synovitis is a rare but important cause of chronic monoarticular joint symptoms, predominantly in the knee. Our case shows the typical presentation of diffuse PVNS, with classical MRI features including synovial thickening, hemosiderin deposition, joint effusion, and bone erosions. Early diagnosis through imaging is critical to prevent secondary joint degeneration and functional impairment. Arthroscopic synovectomy remains the mainstay of treatment, with favourable results when combined with vigilant postoperative monitoring. In this case, the diagnostic value of MRI and the need for histopathological confirmation to guide treatment are considered. Clinicians should maintain a high index of doubt for PVNS in patients with persistent knee swelling and atypical degenerative changes on imaging.

## Author Contribution

Dr. Guru Yogendra M conceptualized the study, supervised the work, and finalized the manuscript. Dr. Anil Kumar Sakalecha contributed to study design and critical revision of the manuscript. Dr. Hina Akhtar was involved in data collection and literature review. Dr. Sneha D N and Dr. Meghana N assisted in radiological data acquisition and interpretation. Dr. Rakesh Bhixavatimath and Dr. Anant Shubham Singh contributed to case documentation and drafting of the manuscript. Dr. Segu Yamini supported data analysis and manuscript editing. All authors reviewed and approved the final manuscript.

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## Conflict of Interest

None

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