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Radiology

MRI in Action: Detecting Diffuse Pigmented Villonodular Synovitis of the Knee – A Case Report

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Abstract | **Case Report**

Pigmented villonodular synovitis (PVNS) is a rare, benign but locally aggressive proliferative disorder of the synovial membrane, most commonly affecting large joints such as the knee. We report the case of a 65-year-old male presenting with progressive bilateral knee pain, limited mobility, and joint cracking. MRI revealed diffuse synovial thickening with villous and nodular projections, hemosiderin deposition, and marked blooming on T2* sequences, consistent with PVNS, in addition to complete ACL rupture, medial meniscus tear, tricompartmental osteoarthritis, and a Baker's cyst. This case highlights the pivotal role of MRI in diagnosis, assessment of associated joint damage, and surgical planning.

Keywords: Pigmented villonodular synovitis, Knee, MRI, Tricompartmental osteoarthritis, Synovectomy.

INTRODUCTION

Pigmented villonodular synovitis (PVNS), also known as diffuse-type tenosynovial giant cell tumor, is a rare, benign but locally aggressive proliferative disorder of the synovial membrane. It is characterized by synovial thickening, hemosiderin deposition, and multinucleated giant cells, which confer its distinctive pigmented appearance [1]. Its estimated incidence is approximately 1.8 cases per million per year [2].

Although it can affect any synovial joint, PVNS shows a predilection for large joints, especially the knee, and typically affects young adults in their second to fourth decades of life [3]. Clinically, it presents with progressive joint swelling, stiffness, pain, and sometimes recurrent hemarthrosis. Two forms are recognized: localized and diffuse, with the latter being more aggressive and associated with higher recurrence rates after treatment [4].

Magnetic Resonance Imaging (MRI) plays a crucial role in the diagnosis and assessment of PVNS. It allows detailed evaluation of synovial hypertrophy, hemosiderin deposition, joint effusion, and possible bone erosions, making it the imaging modality of choice before surgical planning [5]. Histopathological confirmation remains the gold standard, but MRI findings are highly suggestive and essential in guiding management [6].

The aim of our study is to report a case of pigmented villonodular synovitis in a young patient, highlighting the contribution of MRI in its diagnosis and therapeutic approach.

PATIENTS AND METHODS

Case Report:

Mr. B. M., a 65-year-old male patient, with no significant past medical history, presented with progressive bilateral knee pain associated with walking difficulties and joint cracking noises. The symptoms had been evolving gradually, causing increasing functional impairment.

On physical examination, the patient exhibited limited range of motion of both knees, painful mobilization, and swelling without signs of acute inflammation.

Magnetic Resonance Imaging (MRI) of both knees revealed the following findings:

Diffuse synovial thickening with nodular intra-articular proliferations and areas of hemosiderin deposition, strongly suggestive of pigmented villonodular synovitis (PVNS) (bilateral). **Figure 1.**

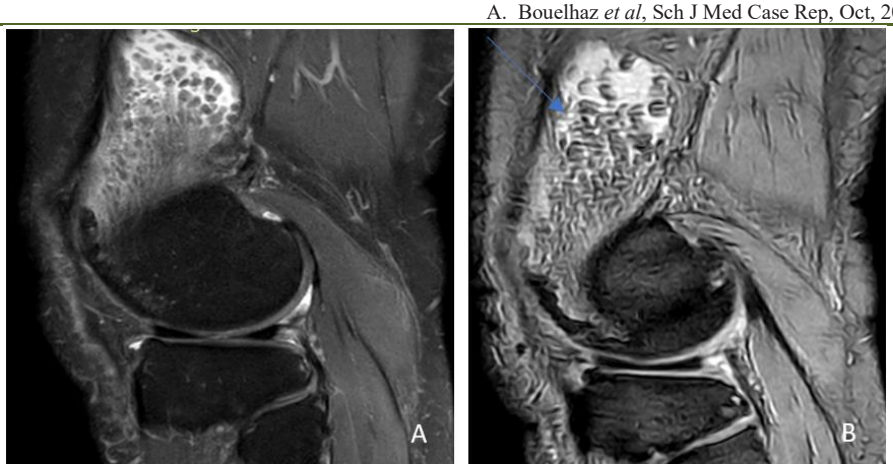

Figure 1. Sagittal PD Fat Sat (A) and T2* (B) sequences demonstrating irregular synovial hypertrophy with nodular intra-articular masses and blooming artifacts consistent with hemosiderin deposits (blue arrow).

Widened anterior cruciate ligaments (ACL) with hyperintense signal on DP sequences, consistent with mucoid degeneration (bilateral). Posterior cruciate ligaments (PCL) were intact.

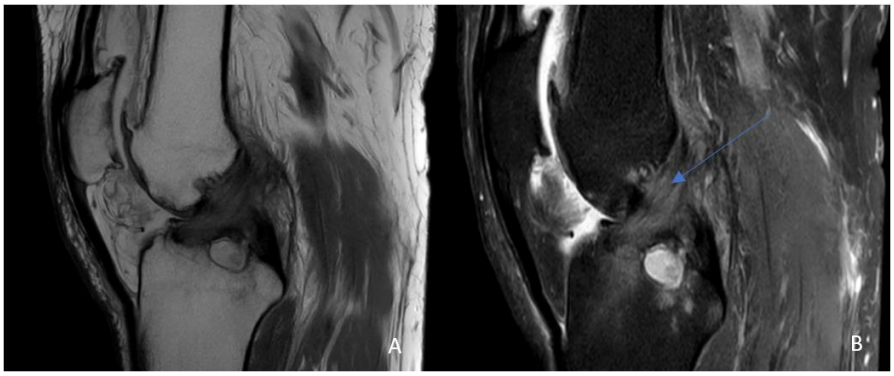

Figure 2. Sagittal T2 (A) and sagittal PD Fat Sat (B) sequences demonstrating fusiform ACL thickening with heterogeneous signal intensity (blue arrow).

Complex tear of the medial meniscus posterior horn with a detached flap forming a bucket-handle configuration (bilateral).

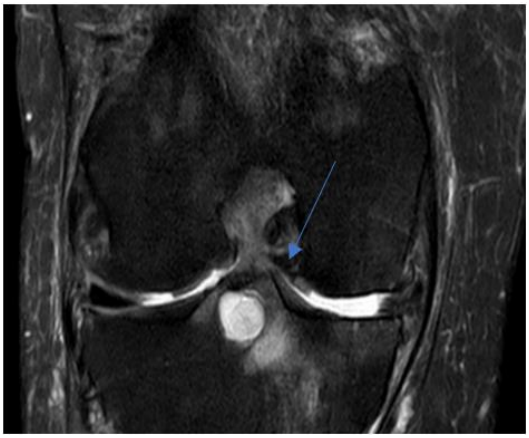

Figure 3. Coronal PD Fat Sat view illustrating a bucket-handle tear of the medial meniscus posterior horn (blue arrow)

Tricompartmental osteoarthritis with marginal osteophytes, joint space narrowing, and subchondral erosions (bilateral).

Advanced chondropathy: femorotibial and patellofemoral compartments grade IV, associated with subchondral bone marrow edema (bilateral). Complex tear of the medial meniscus posterior horn with a detached flap forming a bucket-handle configuration (bilateral).

Well-defined subcortical bone cysts (geodes) in the tibial intercondylar eminence and femoral intercondylar notch, consistent with degenerative changes (bilateral).

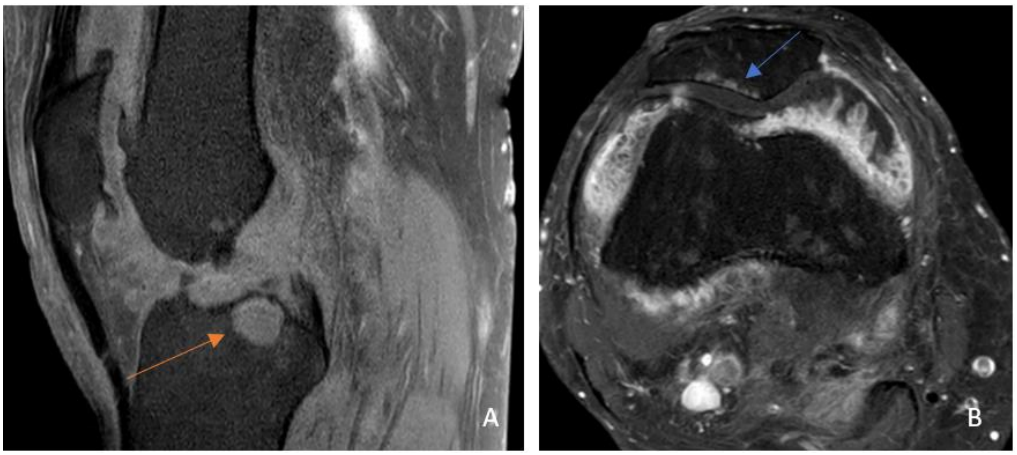

Figure 4. Sagittal PD Fat Sat (A) illustrating tricompartmental osteoarthritis with osteophytes, joint narrowing, and tibial subcortical cysts (orange arrow). Axial T1 injected (B) demonstrating severe chondropathy (grade IV, blue arrow) with subchondral marrow edema.

Medial popliteal cysts (Baker's cysts), right measuring 65 × 34 mm and left 26 × 33.5 mm, communicating with the joint space.

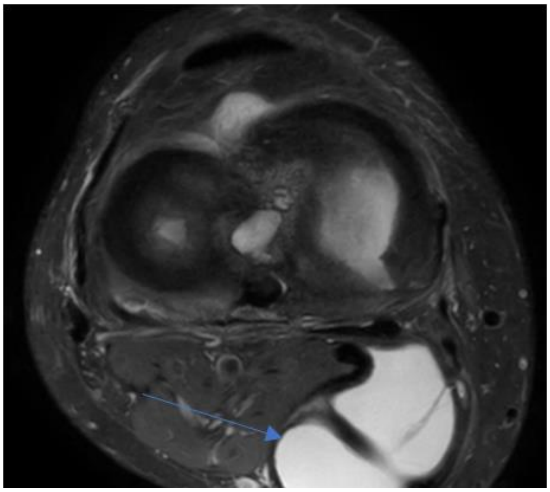

Figure 5: Axial PD Fat Sat demonstrating Baker's cysts (blue arrow) with joint space communication

Moderate joint effusion (bilateral)

In summary, MRI findings were consistent with bilateral pigmented villonodular synovitis, associated with advanced tricompartmental osteoarthritis (grade IV chondropathy), bilateral medial meniscus tears with bucket-handle configuration, mucoid degeneration of the ACLs, and bilateral Baker's cysts.

DISCUSSION

Pigmented villonodular synovitis (PVNS) is a rare benign proliferative disease of the synovial membrane, characterized by villous or nodular synovial hyperplasia and hemosiderin deposition [7]. It was first described by Jaffe in 1941, who distinguished three clinical forms: pigmented villonodular synovitis, bursitis, and tenosynovitis [8]. According to the World Health Organization classification, the localized forms are now grouped under giant cell tumor of the tendon sheath, whereas the diffuse intra-articular form is referred to as diffuse-type tenosynovial giant cell tumor [9].

The incidence of PVNS is estimated at 1.8 cases per million inhabitants per year [10]. It generally occurs in young adults between the third and fifth decades of life, with no clear sex predominance [11]. The disease typically affects a single large joint, most commonly the knee (66–80% of cases), followed by the hip and ankle [12]. Bilateral involvement, as observed in our patient, remains exceptional and has been only rarely reported in the literature [13].

Clinically, PVNS evolves insidiously, with

patients often presenting with progressive joint swelling, pain, stiffness, and sometimes mechanical symptoms such as cracking or locking. Hemarthrosis may also occur due to hemosiderin deposits within the synovium [14]. In our case, the patient consulted for bilateral gonalgia associated with limited mobility and joint noises, which are consistent with the typical clinical presentation.

Imaging is central to diagnosis and management. Plain radiographs are often nonspecific, showing only soft tissue swelling or, in advanced stages, joint space narrowing and subchondral erosions [15]. CT can depict bone erosions but lacks sensitivity for soft tissue characterization.

MRI is considered the gold standard for the diagnosis and staging of pigmented villonodular synovitis (PVNS). It typically demonstrates a lobulated intra-articular soft tissue mass that appears iso- to hypointense relative to muscle on T1-weighted sequences. On T2-weighted and proton density images, the lesion shows marked heterogeneity with multiple areas of low signal intensity, reflecting hemosiderin deposition. Gradient-echo (T2*) sequences often reveal a characteristic blooming artifact, which is regarded as pathognomonic for PVNS [16]. After contrast administration, moderate and heterogeneous synovial enhancement is usually observed. Additional associated findings may include joint effusion, bone erosions, chondropathy, and, in long-standing disease, degenerative joint changes.

In our patient, MRI revealed a diffuse synovial thickening with villous and nodular projections, inhomogeneous signal intensity with hemosiderin deposits, and marked blooming on T2*, strongly suggestive of PVNS. Additional degenerative changes such as tricompartmental osteoarthritis and grade IV chondropathy were also demonstrated, illustrating the destructive potential of the disease when diagnosis is delayed.

The main differential diagnoses of PVNS include hemophilic arthropathy, synovial hemangioma, and synovial chondromatosis. Clinical history and MRI findings usually allow these entities to be distinguished [17]. Definitive diagnosis, however, relies on histopathological confirmation, which demonstrates hemosiderin-laden macrophages, multinucleated giant cells, and a fibrous stroma [8].

Management of PVNS aims to prevent joint destruction and preserve function. Surgical synovectomy remains the gold standard, either by open or arthroscopic approach depending on the extent of disease. However, recurrence rates remain significant, ranging from 8% to 56% in diffuse forms [18]. In advanced cases with severe degenerative changes, as seen in our patient with tricompartmental osteoarthritis, joint replacement may be considered. Adjuvant treatments such as external radiotherapy or radiosynoviorthesis have been proposed in cases of recurrence or incomplete resection, but their use remains controversial due to potential complications [19].

CONCLUSION

Pigmented villonodular synovitis (PVNS) is a rare, benign but locally aggressive disorder of the synovium, most frequently affecting large joints such as the knee. MRI is the key diagnostic tool, revealing villous or nodular synovial proliferation, hemosiderin deposition, joint effusion, and bone or cartilage involvement. It is essential for surgical planning, particularly synovectomy, and for monitoring recurrence, which is higher in diffuse forms. Early diagnosis and comprehensive management are critical to prevent joint destruction, preserve function, and guide optimal treatment strategies, as demonstrated in this case.

Conflicts of Interest: The authors declare no conflicts of interest.

Contributions of the Authors: All authors contributed to the conduct of this work. They have read and approved the final version of the manuscript.

REFERENCE

1. Myers BW, Masi AT. Pigmented villonodular synovitis and tenosynovitis: a clinical epidemiologic study of 166 cases and literature review. *Medicine (Baltimore)*. 1980 ;59(3):223-238.
2. Myers BW, Masi AT, Feigenbaum SL. Incidence and prevalence of pigmented villonodular synovitis: a critical review of the literature. *Semin Arthritis Rheum*. 1980 ;10(2):69-83.
3. Rao AS, Vigorita VJ. Pigmented villonodular synovitis (a review of seventy cases). *J Bone Joint Surg Am*. 1984 ;66(1):76-94.
4. Verspoor FG, Zee AA, Hannink G, van der Geest IC, Veth RP, Schreuder HW. Long-term follow-up results of primary and recurrent pigmented villonodular synovitis. *Rheumatology (Oxford)*. 2014 ;53(11):2063-2070.
5. Murphey MD, Rhee JH, Lewis RB, Fanburg-Smith JC, Flemming DJ, Walker EA. Pigmented villonodular synovitis: radiologic-pathologic correlation. *Radiographics*. 2008 ;28(5):1493-1518.
6. Aurégan JC, Klouche S, Bohu Y, Lefèvre N, Herman S, Hardy P. Treatment of pigmented villonodular synovitis of the knee. *Arthroscopy*. 2014 ;30(10):1327-1341.
7. Myers BW, Masi AT. Pigmented villonodular synovitis and tenosynovitis: a clinical epidemiologic study of 166 cases and literature review. *Medicine (Baltimore)*. 1980 ;59(3):223-238.
8. Jaffe HL. Pigmented villonodular synovitis, bursitis and tenosynovitis. *Arch Pathol*. 1941 ;31:731–765.

9. Fletcher CDM, Bridge JA, Hogendoorn P, Mertens F. WHO Classification of Tumours of Soft Tissue and Bone. 4th ed. Lyon : IARC ; 2013.
10. Myers BW, Masi AT, Feigenbaum SL. Incidence and prevalence of pigmented villonodular synovitis: a critical review of the literature. *Semin Arthritis Rheum*. 1980 ;10(2):69-83.
11. Rao AS, Vigorita VJ. Pigmented villonodular synovitis (a review of seventy cases). *J Bone Joint Surg Am*. 1984 ;66(1):76-94.
12. Aurégan JC, Klouche S, Bohu Y, Lefèvre N, Herman S, Hardy P. Treatment of pigmented villonodular synovitis of the knee. *Arthroscopy*. 2014 ;30(10):1327–1341.
13. Ofluoglu O. Pigmented villonodular synovitis. *Orthop Clin North Am*. 2006 ;37(1):23–33.
14. Dines JS, DeBerardino TM, Wells JL, et al. Long-term follow-up of surgically treated localized pigmented villonodular synovitis of the knee. *Arthroscopy*. 2007 ;23(9):930–937.
15. Dorwart RH, Genant HK, Johnston WH, Morris JM. Pigmented villonodular synovitis of synovial joints : clinical, pathologic, and radiologic features. *AJR Am J Roentgenol*. 1984 ;143(4):877–885.
16. Murphey MD, Rhee JH, Lewis RB, et al. Pigmented villonodular synovitis: radiologic-pathologic correlation. *Radiographics*. 2008 ;28(5):1493–1518.
17. Ottaviani S, Ayral X, Dougados M, Gossec L. Pigmented villonodular synovitis: a retrospective single-center study of 122 cases and review of the literature. *Semin Arthritis Rheum*. 2011 ;40(6):539–546.
18. Verspoor FG, Zee AA, Hannink G, et al. Long-term follow-up results of primary and recurrent pigmented villonodular synovitis. *Rheumatology (Oxford)*. 2014 ;53(11):2063–2070.
19. Mollon B, Lee A, Busse JW, et al. The effect of radiosynoviorthesis on local recurrence of diffuse pigmented villonodular synovitis: a systematic review and meta-analysis. *BMC Musculoskelet Disord*. 2015 ;16:36.