Loosening of Total Knee Arthroplasty Associated with Pigmented Villonodular Synovitis: Case Presentation and Literature Review

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ABSTRACT
Introduction: Pigmented villonodular synovitis (PVNS) in patients with total knee arthroplasty (TKA) is a rare condition with an unclear etiology that can cause pain, hemarthrosis, or, less commonly, prosthetic loosening. We present the case of a 54-year-old male patient with a left TKA who developed pain and joint effusion three months postoperatively. Radiographs showed signs of loosening, and due to suspicion of infection, a two-stage revision surgery was performed one year after the initial procedure. Pathological synovial tissue was identified, and a radical synovectomy was carried out during the first stage of revision. Histopathological analysis confirmed PVNS. At three years postoperatively, the patient exhibited satisfactory functional recovery with no signs of recurrence. **Conclusion:** Considering PVNS as a differential diagnosis in cases of postoperative pain and hemarthrosis is crucial for early diagnosis and appropriate treatment.
Keywords: Pigmented villonodular synovitis; total knee arthroplasty; revision total knee arthroplasty; hemarthrosis.
Level of Evidence: IV

Aflojamiento de la artroplastia total de rodilla asociado a sinovitis vellonodular pigmentada. Presentación de un caso y revisión bibliográfica

RESUMEN
Introducción: La sinovitis vellonodular pigmentada en pacientes sometidos a una artroplastia total de rodilla es un cuadro muy raro, de causa poco clara, que puede provocar dolor, hemartrosis o, con menos frecuencia, aflojamiento de la prótesis. Presentamos el caso de un hombre de 54 años sometido a una artroplastia total de rodilla izquierda, que evolucionó con dolor y derrame articular a los tres meses de la operación. En las radiografías, se observaron signos de aflojamiento y, ante la sospecha de infección, se indicó la revisión en dos tiempos al año de la cirugía. Se detectó alteración del tejido sinovial y se procedió a la sinovectomía radical durante el primer tiempo quirúrgico. El análisis histopatológico confirmó una sinovitis vellonodular pigmentada. A los tres años de la cirugía, la recuperación funcional y clínica era satisfactoria, sin recurrencias. **Conclusión:** Es esencial sospechar una sinovitis vellonodular pigmentada como alternativa diagnóstica en casos de dolor y hemartrosis, para llegar a un diagnóstico precoz y brindar un tratamiento apropiado.
Palabras clave: Sinovitis vellonodular pigmentada; artroplastia total de rodilla; revisión de artroplastia total de rodilla; hemartrosis.
Nivel de Evidencia: IV

INTRODUCTION

Pigmented villonodular synovitis (PVNS) was first described by Chassignac in 1852 as a nodular lesion in the sheath of a flexor tendon of the hand.[1] In 1864, its localized form was described in the knee.[2] PVNS is a benign disease of the synovial tissue, characterized by low incidence but local aggressive potential. It can be localized either intra- or extra-articularly, with the intra-articular form further classified into localized or diffuse types.[1,2] The knee is the most commonly affected joint, followed by the hip, ankle, and shoulder.[1] The true etiology of PVNS remains unclear, though hypotheses suggest chronic synovial inflammation triggered by microtrauma, recurrent

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hemarthrosis, or metabolic changes as potential causes.[1] In recent years, increasing evidence supports the possibility of a neoplastic origin for PVNS.[1,3] Genetic studies have identified chromosomal translocations that enhance cytokine expression, promoting the proliferation and differentiation of cells involved in PVNS, such as macrophages and multinucleated giant cells, which synthesize tissue-destructive molecules.[1] Clinically, PVNS typically presents with pain, joint effusion, and, less commonly, stiffness or a palpable mass.[1,2] MRI is the imaging modality of choice, while histopathological analysis confirms the diagnosis, revealing papillary synovial hyperplasia composed of multinucleated and mononucleated histiocyte-like giant cells with hemosiderin deposits.[1,3] Treatment varies depending on the form, clinical presentation, and disease progression, but synovectomy—either open or arthroscopic—is the standard intervention, often supplemented by radiotherapy or biological therapies that target cell signaling pathways.[1]

In patients with joint replacements, chronic synovitis may develop due to a reaction to polymer particles from polyethylene, cement, metal, ceramic, or corrosion, mimicking PVNS.[3-5] However, PVNS following total knee arthroplasty (TKA) is exceedingly rare, and its etiology remains unclear.[6-9] Several hypotheses have been proposed: some suggest that PVNS arises spontaneously, with no direct relation to the prosthetic implant, thus presenting the same risk as a native knee; others propose that an irritative stimulus may induce chronic synovitis, leading to recurrent hemorrhage and hemosiderin deposition.[6,7,9-11]

In these patients, PVNS may manifest as pain, hemarthrosis, or prosthetic loosening. The recommended treatment is radical synovectomy to minimize the risk of recurrence.[6-8]

Evidence regarding PVNS in patients with TKA is limited to case reports. We present a case of early prosthetic loosening following TKA due to PVNS, along with a review of the literature.

CLINICAL CASE

A 54-year-old man who had undergone a right TKA one year earlier required mobilization under anesthesia one month post-surgery at our center. He initially presented with severe left knee pain, which severely limited his daily activities and had not improved despite six months of conservative treatment. On physical examination, his range of motion was 0° to 100°, with no coronal or sagittal instability. Preoperative pain, assessed using the visual analog scale (VAS), was 9/10, and his preoperative Knee Society Score (KSS) was 44/60.[12] Anteroposterior and lateral radiographs of the knee confirmed a diagnosis of genu varum osteoarthritis (Figure 1).

A left TKA was performed using a standard anterior approach with medial parapatellar arthrotomy. No macroscopic abnormalities of the synovial tissue were observed, so no resection was performed. A cemented posterior-stabilized prosthesis was implanted without patellar resurfacing (FHK®, FH ORTHO, France). Proper alignment and stability in extension, flexion, and mid-flexion were achieved (Figure 2).

Three months postoperatively, the patient experienced pain both at rest and during activity, recurrent joint effusion, a mass in the subquadricipital recess, and limited flexion to 90° (Figure 3). The surgical wound had healed, with no signs of erythema or increased temperature, and no coronal or sagittal plane instability was detected. Given the failure of analgesics and physical therapy, arthrocentesis was performed six months after surgery, yielding hemorrhagic fluid without bacterial growth. Seven months postoperatively, follow-up radiographs revealed a radiolucent area in zones 1-2 of the Knee Society classification, suggestive of early prosthetic loosening.[13] (Figure 4).

The patient subsequently experienced intermittent low-grade fever (up to 37.5°C), and laboratory tests showed elevated acute phase reactants: erythrocyte sedimentation rate (ESR) of 35 mm/h (normal range: 2-20) and C-reactive protein (CRP) of 22 mg/L (normal value: <7). Considering the mechanical pain, joint effusion, low-grade fever, radiolucent findings, and altered infection markers, the case was interpreted as possible septic loosening. A two-stage revision surgery was planned. The first stage was performed one year after the initial TKA. Intraoperatively, hypertrophic, hyperemic, and brownish-pigmented synovial tissue was observed (Figure 5). Both prosthetic components were loose, but there were no signs of polyethylene or prosthetic material wear. With improved exposure, radical synovectomy was completed, and multiple bone, periprosthetic tissue, and interface samples were taken for culture and histopathologic analysis. A handmade articulating spacer composed of antibiotic-loaded cement (tobramycin and vancomycin) was placed. *Staphylococcus epidermidis* was isolated from one of the culture samples. Despite this finding, in consultation with the infectious disease team, antibiotic therapy was administered per the periprosthetic infection protocol. Three weeks later, the histopathological study confirmed a diagnosis of PVNS (Figure 6).

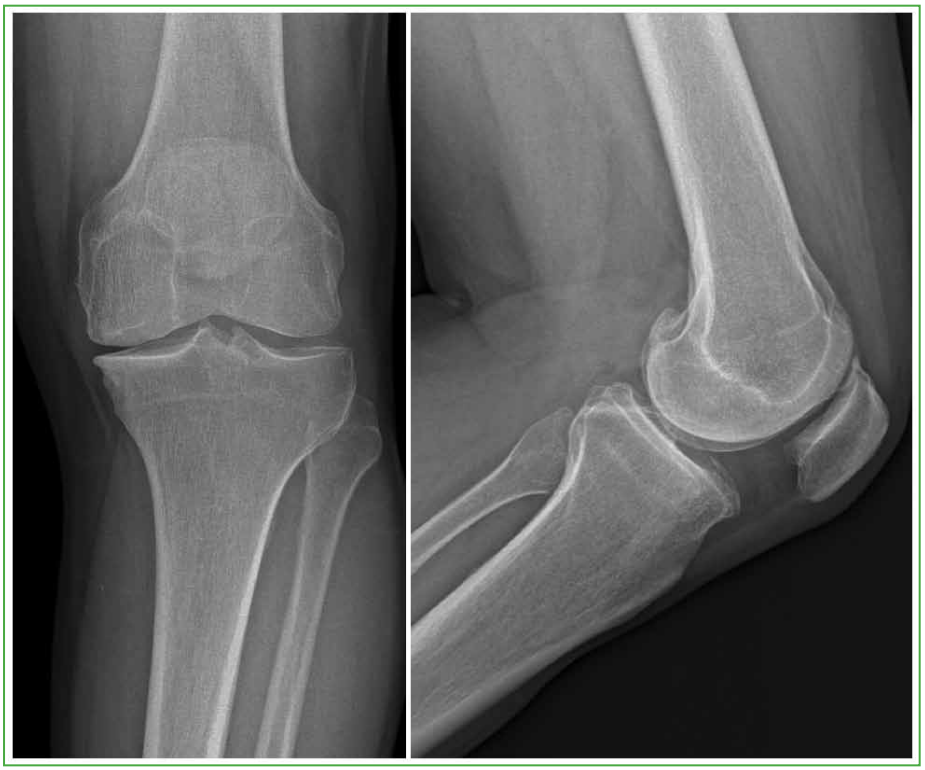

Figure 1. Anteroposterior and lateral radiographs of the left knee. Knee osteoarthritis is observed with greater involvement of the medial compartment.

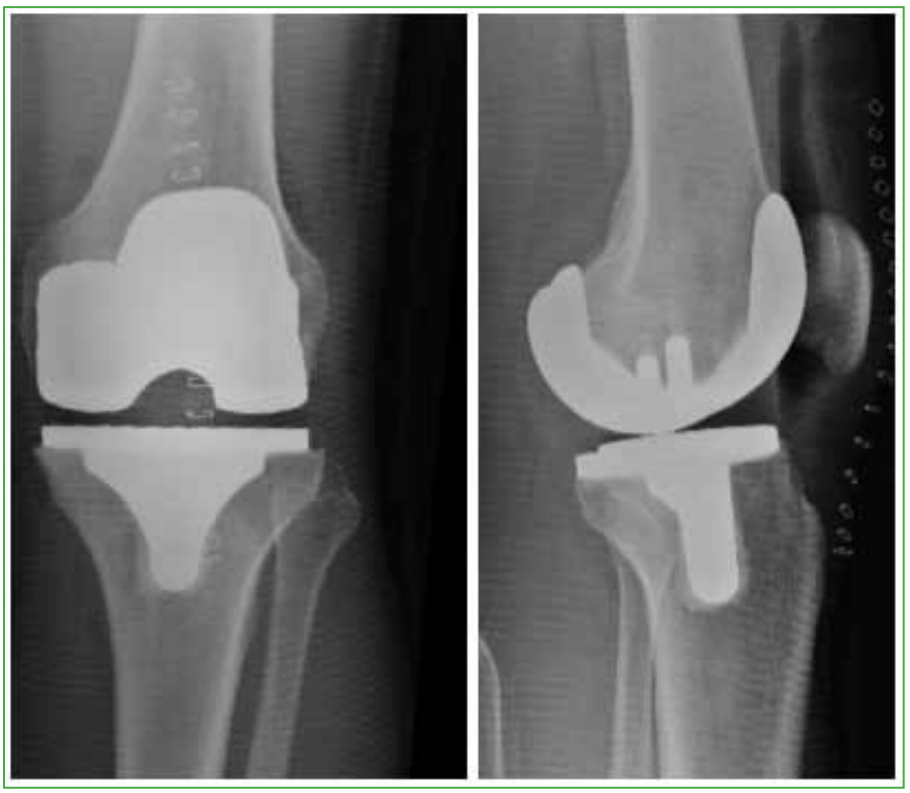

Figure 2. Anteroposterior and lateral radiographs of the left knee in the immediate postoperative period.

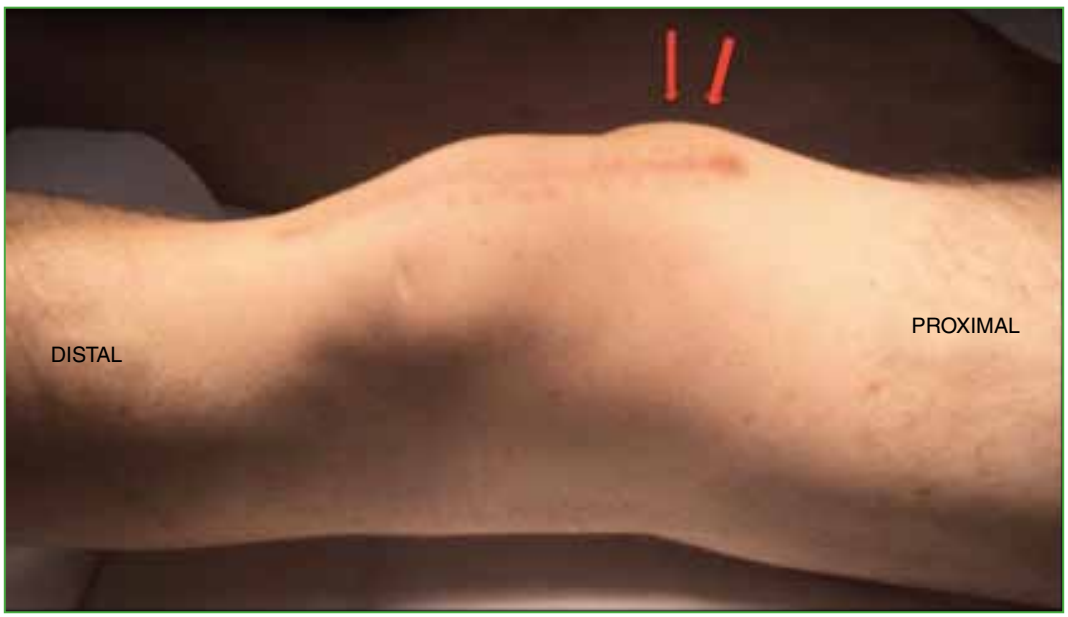

Figure 3. Clinical image three months after surgery. The arrows show the joint effusion in the subquadricipital recess.

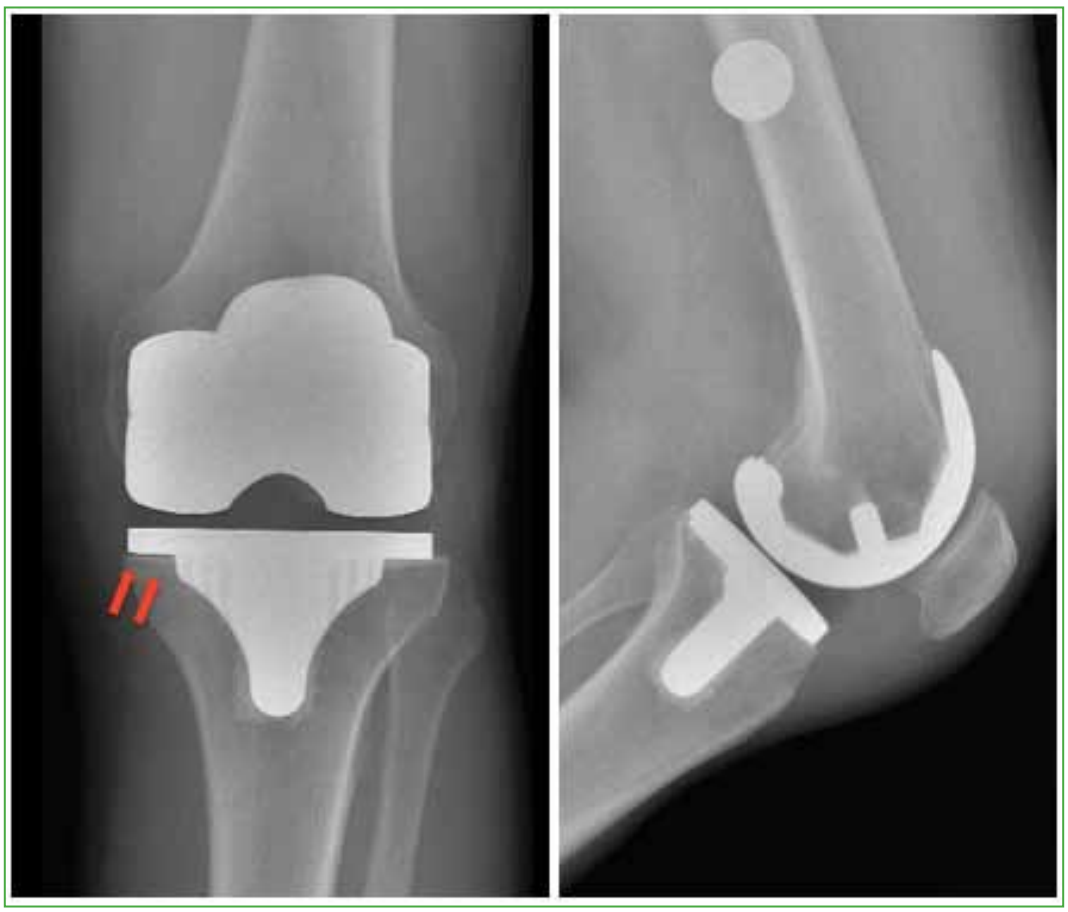

Figure 4. Anteroposterior and lateral radiographs of the left knee. A radiolucent image is observed, in the AP projection, in zones 1 and 2 of the Knee Society classification.

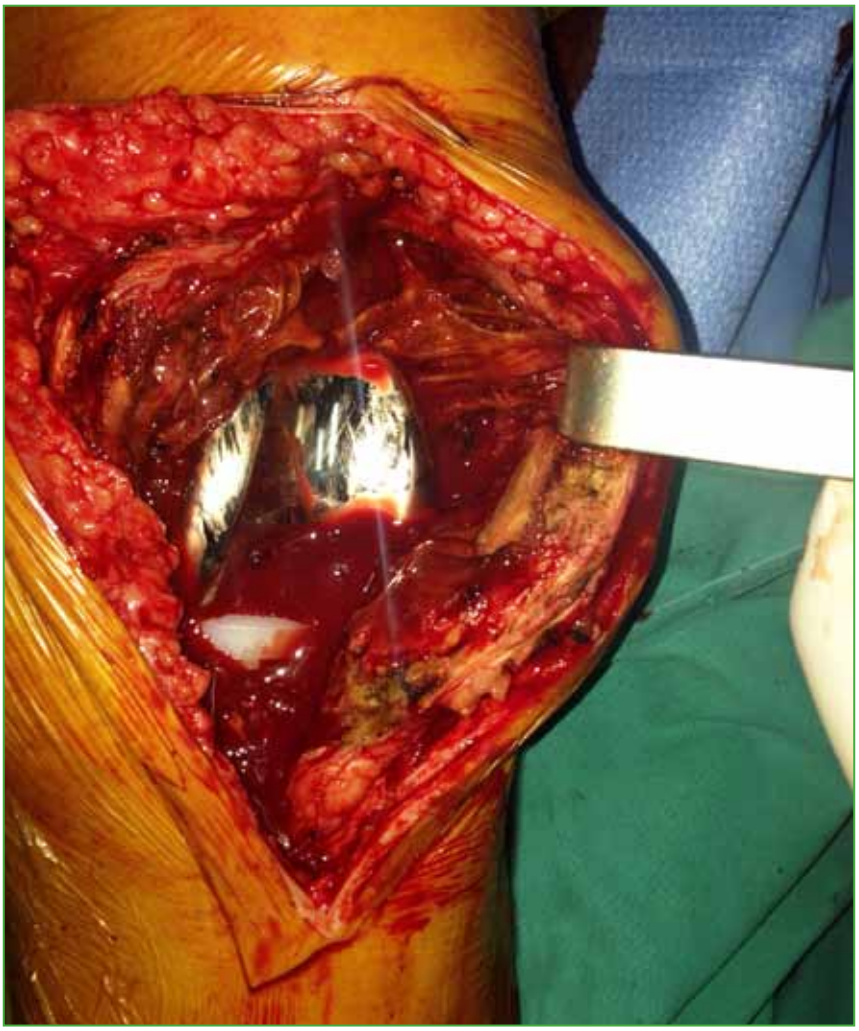

Figure 5. Intraoperative image during the first stage of revision. Hypertrophic and brownish synovial tissue is observed.

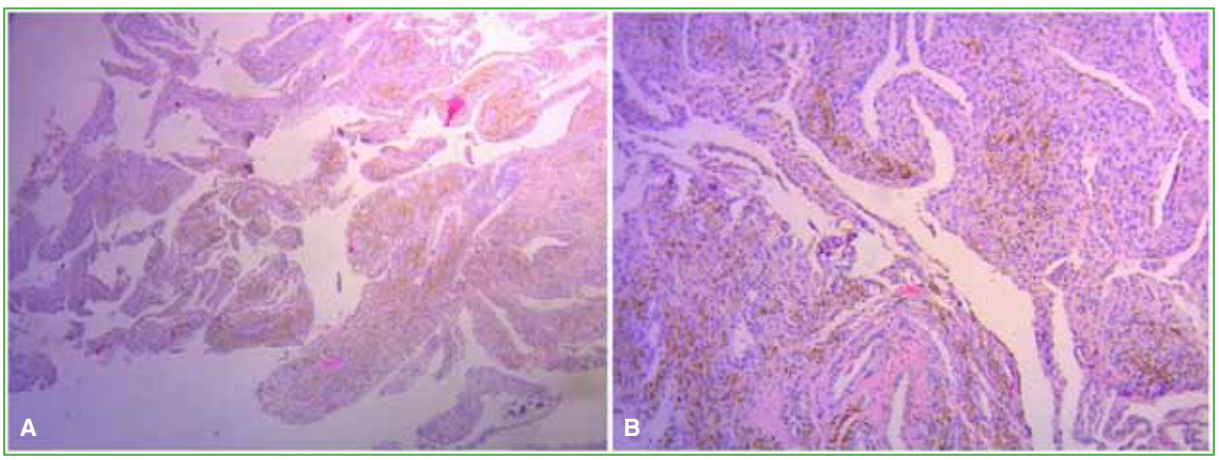

Figure 6. Images of the histological study. The villonodular architecture is observed (**A**) composed of polygonal synovial cells, macrophages and multinucleated giant cells loaded with hemosiderin, and fibrous stroma (**B**). Hematoxylin-eosin staining at x4 (**A**) and x10 (**B**) magnification.

The patient continued antibiotic therapy for seven weeks. At week 10, given the favorable clinical course and reduced infection markers, the second stage of revision surgery was performed. No pathological changes were observed in the periprosthetic tissue, as confirmed by histopathological analysis. The spacer was removed, and a cemented revision prosthesis with a constrained liner and stems was implanted in both the femur and tibia (Triathlon®, Stryker, USA) (Figure 7).

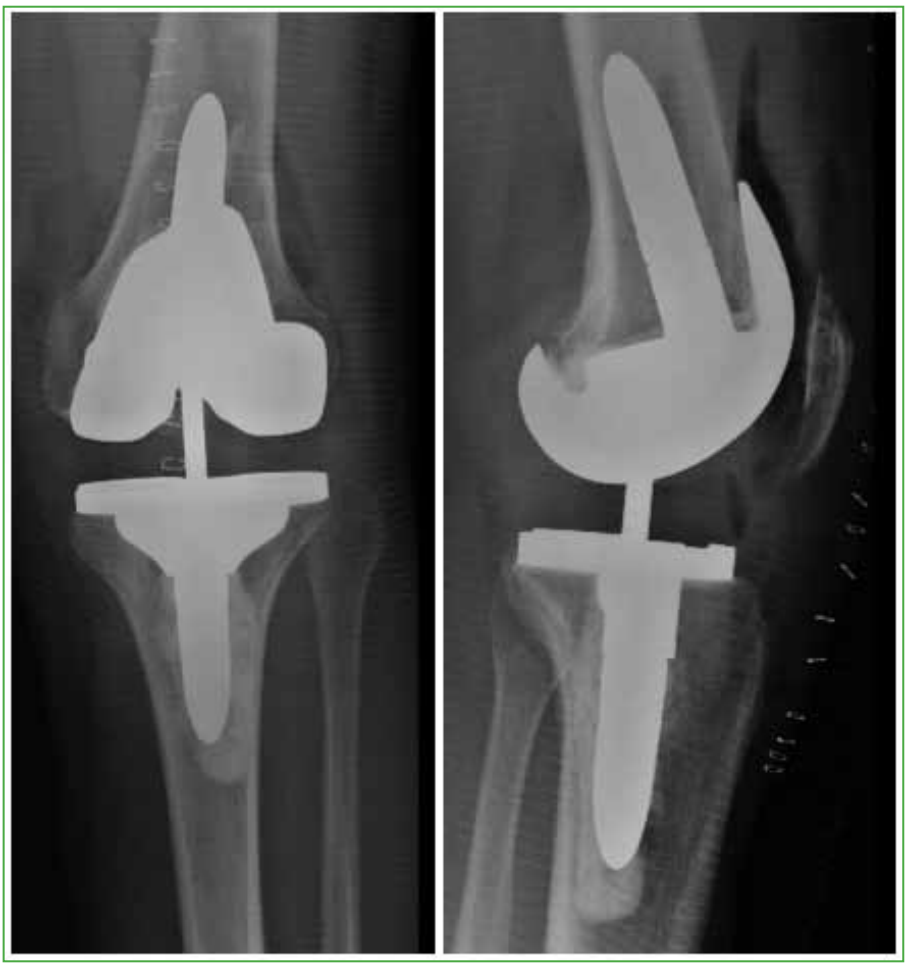

Figure 7. Anteroposterior and lateral radiographs of the left knee after the second stage of revision.

The patient's postoperative course was satisfactory, and he resumed daily activities without pain. At the three-year follow-up, he exhibited mild chronic swelling in the subquadricipital recess, which was not painful. His range of motion was 0° to 110°, with a VAS pain score of 2/10 and a Knee Society Score of 92/90. Radiographs showed no signs of prosthetic loosening.

DISCUSSION

In our patient, PVNS after TKA presented clinically with recurrent joint pain and effusion three months postoperatively. Radiographs revealed a new radiolucent area around the tibial baseplate, interpreted as prosthetic loosening. One year after the initial surgery, the patient underwent a radical synovectomy and two-stage revision for suspected septic loosening, with histological analysis confirming the diagnosis of PVNS.

To our knowledge, publications on PVNS in knee replacement patients are limited to case reports. Hypotheses regarding the occurrence of PVNS in joint replacement patients are varied. Some suggest that its appearance is spontaneous, unrelated to the TKA, while others propose that the trauma of surgery may predispose the patient to PVNS.[10,11,14] The most widely accepted—though debated—hypothesis posits that chronic synovitis forms the basis for PVNS, exacerbated by inflammatory, immunological, or toxic reactions, along with recurrent hemorrhages caused by microtrauma. These microtraumas may result from soft tissue friction due to clinical or subclinical instability, poor implant positioning, or oversized implants.[6-8,15]

In 2004, a consensus classification of periprosthetic and neo-synovial membranes (formerly referred to as "synovial-like interface membranes") was established in Germany. This classification categorizes aseptic and septic implant failures based on easily reproducible histopathological criteria.[16] It has gained international recognition and has evolved through successive updates to the version known today, summarized in Table 1.[4]

Table 1. Types of periprosthetic tissue pathology recognized in conventional histological examination.[4]

Type I	Neo-synovial/periprosthetic membrane of particle-induced type
Type II	Neo-synovial/periprosthetic membrane of infectious type
Type III	Neo-synovial/periprosthetic membrane of combined type
Type IV	Neo-synovial/periprosthetic membrane of fibrous type without particles
Type V	Endoprosthesis-induced arthrofibrosis
Type VI	Bone diseases

The most common type of periprosthetic membrane is particle-induced (type I), characterized by synovial hyperplasia with macrophages and multinucleated cells containing polyethylene, cement, metal, or wear debris particles, along with variable lymphocytic infiltrates and particle-induced necrosis.[4] Another type is the PVNS-like membrane, which is distinguished by the presence of wear particles, villonodular histological architecture, and giant multinucleated cells from foreign body reactions, with minimal or no hemosiderin deposits.[3] Conversely, true PVNS membranes exhibit villonodular architecture, contain multinucleated and mononucleated cells and a fibroblastic stroma, lack wear particles, and show clear hemosiderin deposits.[3,6,7] Despite clear histopathological differences, these membranes can be clinically indistinguishable, all presenting with pain, joint effusion, and limited range of motion.[5,6,17] They can also cause aseptic loosening due to macrophage activation and the release of proinflammatory cytokines, which stimulate osteoclasts and other inflammatory cells. Distinguishing between these membranes is crucial because PVNS has a higher recurrence risk (up to 50%) compared to other types, which are more manageable by addressing the underlying stimuli.[1,18]

The published cases share similarities with ours. In all, the synovium appeared benign during the initial TKA or unicompartmental replacement surgery, supporting the theory that PVNS develops postoperatively.[6-11,15,17,18] Clinical manifestations were similar, characterized by pain of insidious onset over several months, joint effusion, limited range of motion, and occasionally a palpable mass.[6,7,11] However, unlike our case, where symptoms began three months postoperatively, other reported cases developed symptoms between one and nine years after surgery. Many patients, including ours, were initially evaluated for suspected periprosthetic infection, underwent arthrocentesis, and hemorrhagic fluid was collected without bacterial identification.[6,9,10] Due to poor response to conservative treatment, surgery was performed in all cases, either arthroscopically or through open surgery. Localized or diffuse brown or yellow hypertrophic synovial tissue was resected, and in some cases, prosthetic revision was carried out based on intraoperative findings.[6,7,9,17,18] Notably, no polyethylene wear was

observed in almost all cases, supporting the concept that PVNS does not involve wear particles in histological studies.[6,8,9,11,15] Histopathological confirmation of PVNS was achieved in all cases, with no recurrences reported, although follow-up periods varied. The key findings from published cases are summarized in Table 2.

Table 2. Summary of case reports of pigmented villonodular synovitis in patients with knee joint replacement.

Author (year)	Patient	Clinical manifestations/Studies	Treatment/Results
Ballard et al.[10] (1993).	- 67-year-old man - Right primary cemented TKA, 9 years before	- Diffuse knee pain and joint effusion of 2 months of evolution. - Hemorrhagic arthrocentesis without germ isolation	- Extensive open synovectomy - Diffuse PVNS - Good evolution, no recurrence six months after surgery.
Bunting et al.[11] (2007).	- 72-year-old woman - Right primary cemented TKA, 2 years before	- Pain and joint effusion of 7 months of evolution	- Arthroscopic synovectomy - Localized PVNS - Good evolution, no recurrence in the postoperative period.
Mohanlal et al.[17] (2009).	- 69 year old man - Right UKA, 5 years before	- Anterior knee pain and joint effusion of 1 year of evolution.	- Arthroscopic synovectomy, then revision to TKA - PVNS - Good evolution, no recurrence in the postoperative period.
Oni and Cavallo[9] (2011).	- 74 year old man - Left primary cemented TKA, 18 months before	- Anterior knee pain and joint effusion of 1 month of evolution. - Hemorrhagic arthrocentesis without germ isolation	- Extensive arthroscopic synovectomy - Diffuse PVNS - Good evolution, with no recurrence 6 months after surgery.
Chung and Park[8] (2011)	- 74-year-old woman - Left primary cemented TKA, 5 years before	- Knee pain and joint effusion of 1 month of evolution. - Radiographic signs of osteolysis	- Single-stage revision with radical synovectomy - Localized PVNS - Good evolution, no recurrence in the postoperative period.
Onodera et al.[15] (2012).	- 61-year-old woman - Right UKA, 5 years before	- Knee pain and joint effusion of 3 years of evolution. - Hemorrhagic arthrocentesis without germ isolation and MRI compatible with PVNS.	- Arthroscopic synovectomy - Localized PVNS - Good evolution, no recurrence one year after surgery.
Camp et al.[7] (2016).	- 64-year-old woman - Right primary cemented TKA, 9 years before - Antiphospholipid syndrome anticoagulation	- Generalized knee pain of 1 year of evolution - Instability in flexion - Polyethylene wear and loosening (osteolysis) - Pseudarthrosis of patellar fracture	- Extensive synovectomy, partial patellectomy and TKA revision - Diffuse PVNS - Good evolution, no recurrence 18 months after surgery.
Zhang et al.[18] (2016).	- 67-year-old man - Left primary cemented TKA, 6 years before - Pulmonary sarcoidosis of 10 years of evolution	- Revision with polyethylene replacement, 3 years after index surgery - Diffuse knee pain and joint effusion	- Two-stage revision with radical synovectomy - Diffuse PVNS and sarcoid granuloma - Good evolution, no recurrence 10 months after surgery.
Kia et al.[6] (2018).	- 62-year-old woman - Right primary cemented TKA, 4 years before	- Recurrent joint effusion and chronic anterior pain - One year after surgery, arthroscopic resection of Hoffa's fat pad, without synovial alterations. - Hemorrhagic arthrocentesis without germ isolation and MRI compatible with PVNS.	- Extensive synovectomy and polyethylene replacement - Diffuse PVNS - Good evolution, no recurrence one year after surgery.
Our study (2024)	- 54 year-old man - Left primary cemented TKA, 1 year before	- Diffuse pain and joint effusion of 9 months of evolution. - Hemorrhagic arthrocentesis without germ isolation	- Two-stage revision with radical synovectomy - Diffuse PVNS - Good evolution, with no recurrence 3 months after surgery.

TKA = total knee arthroplasty; UKA = unicompartmental knee arthroplasty; MRI = magnetic resonance imaging; PVNS = pigmented villonodular synovitis.

The main concern with PVNS is its potential for recurrence, which can present more aggressively, as well as the complications associated with treating PVNS in joint replacement patients. Recurrence rates for PVNS in native knees vary depending on whether the disease is diffuse (8-30%) or localized (18-60%).[1,18] To prevent recurrence, complete synovectomy is crucial and can be complemented by radiotherapy or biological therapies, particularly in cases of prior recurrence or high recurrence risk.[1,7] In our case, a radical synovectomy was performed alongside a two-stage revision for suspected early septic loosening, with no recurrences over a three-year follow-up.

Camp et al. performed a single-stage revision for instability and aseptic loosening, incorporating a radical synovectomy and partial patellectomy with quadricipital tendon reinsertion for a previous patellar pseudarthrosis.[7] These authors emphasized that component revision allows for a more thorough synovectomy. However, some cases were treated with arthroscopic synovectomy, achieving good disease control.[9,11,17]

Due to the limited evidence available, published studies on PVNS in TKA patients can help guide the management of cases like ours. In such patients, TKA is indicated to control the disease in severe or recurrent cases and to restore joint function lost due to PVNS-induced damage.[19,20] Surgical goals include resecting all pathological synovial tissue, which may adhere to ligaments or tendons, often necessitating the use of more constrained prostheses.[21] Some studies also suggest radiotherapy or biological therapies to prevent recurrence, with a reported recurrence rate of 11-13% over an average six-year follow-up.[20] From a clinical and functional point of view, patients typically show marked improvement after surgery, although there is an elevated risk of stiffness, infection, and, in some cases, aseptic loosening.[22]

Although this paper presents only one case, we believe it is crucial to recognize the possibility of PVNS in patients with TKA. PVNS should always be suspected in patients with pain associated with recurrent joint effusion, and it is essential to differentiate it from low-virulence infections. We recommend evaluating these cases with radiography and arthrocentesis, where findings may include osteolysis and hemorrhagic synovial fluid without germ isolation. If diagnostic uncertainty persists, MRI with a metal artifact reduction sequence can be used, which may reveal hypointense signal synovitis. Another option is diagnostic arthroscopy, which allows for sampling and debridement of pathological synovial tissue. However, in cases where loosening or infection is suspected, a revision surgery with radical synovectomy should be performed in the same surgical procedure.

CONCLUSION

PVNS in TKA patients is an exceedingly rare condition of uncertain etiology that negatively affects patient function and satisfaction. It is crucial to consider PVNS as a differential diagnosis in cases of pain and hemarthrosis to enable early diagnosis and timely treatment.

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