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Introduction

The synovium is a specialized tissue derived from mesenchymal cell lineage that is essential for the proper function of joints. Synovial membranes are comprised of two layers, the synovial intima and subsynovial tissue. The synovial intima is one to four cell layers thick and is composed of synovio-cytes, macrophages, and fibroblasts [1]. This overlies the subsynovium, which consists of loosely organized connective tissue, adipocytes, macrophages, lymphatics, and blood vessels [2, 3]. Beneath this connective tissue lies the dense, fibrous joint capsule. The synovium functions as a mechanical shock absorber and filter system to secrete hyaluronic acid and synovial fluid to lubricate and nourish the articular surface [4]. It lines intra-articular structures, including tendons, ligaments, and the bare, intra-capsular periosteal surfaces not covered by cartilage.

The synovium of the knee is the most extensive and complex in the body. Anteriorly, the synovium attaches to the articular borders of the patella. At the upper border of the patella, it extends on each side of the patella posterior to the aponeuroses of the vastus medialis, vastus lateralis, and quadriceps tendon to attach to the anterior femoral shaft, forming the suprapatellar bursa. From the inferior patella, it extends posterior to the infrapatellar fat pad to its insertion on the anterior tibia. Medially and laterally from the patella,

the membrane travels inferiorly, forming redundant alar folds (plicae) of synovium that may project into the joint [5]. The infrapatellar plica (ligamentum mucosum) is the most common plica in the knee and extends from the inferior pole of the patella through the infrapatellar fat pad, anterior to the anterior cruciate ligament (ACL) to insert in the anterior intercondylar notch [6]. The synovial membrane lines the anterior, medial and lateral aspects of the ACL and posterior cruciate ligament (PCL), before reflecting posteriorly to join the posterior fibrous capsule. This functionally divides the knee into medial and lateral compartments [7]. The medial and lateral borders of the joint are defined as the synovial membrane passes inferiorly from the femur to the medial and lateral attachments of the menisci, leaving the peripheral borders of the menisci devoid of synovial membrane [8]. The synovium then extends from the inferior portion of the meniscal attachments to form the medial and lateral perimeniscal recesses. Between the lateral meniscus and the popliteus tendon, the synovial membrane forms the popliteal recess. The synovial membrane extends posteriorly from the femur to the proximal origins of the lateral and medial heads of the gastrocnemius, thus forming the posterior femoral recess [9].

Pathology of the synovium can be part of a systemic process or primary synovial disease affecting a single articulation. Degenerative, traumatic, inflammatory, infectious, or neoplastic processes can affect the synovium. The most common primary synovial disorders are pigmented villonodular synovitis, synovial chondromatosis, synovial hemangioma, and lipoma arborescens [4, 10].

Magnetic resonance (MR) imaging provides excellent soft tissue contrast with multi-planar capabilities by noninvasive means to effectively evaluate the synovium. MR imaging with intravenous (IV) gadolinium-based contrast is most beneficial when characterizing synovial disease, as the synovial membrane is difficult to distinguish from adjacent joint effusion on T1-weighted imaging (both low in signal intensity) and T2-weighted imaging (both high in signal intensity) [4, 11]. The normal synovium avidly enhances on

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post-gadolinium images and this enhancement aids evaluation of synovial disorders.

The primary focus of this chapter is to present the most common synovial disorders affecting the knee, review characteristic MR imaging findings, and discuss surgical treatment options.

Case 1: Pigmented Villonodular Synovitis

A 36-year-old male presents with complaints of progressively worsening right knee pain over the past 2–3 years with no history of antecedent trauma. He describes mechanical symptoms and intermittent effusions without antecedent trauma. The pain is not clearly localized and affects the anterior as well as the posterior aspect of the knee. Nonsteroidal anti-inflammatory medications and physical therapy did not improve the patient's symptoms and advanced imaging was obtained.

Introduction

Pigmented villonodular synovitis (PVNS) is a benign but disabling proliferative disorder of the synovial lining of joints first described by Jaffe et al. in 1941 [12]. It is characterized by the development of villi and nodular thickening of the synovial membrane and deposition of hemosiderin-laden macrophages [13–15]. The etiology of PVNS remains controversial, but recent studies support the theory that a combination of reactive inflammatory disease and chromosomal translocation result in neoplastic proliferation of the synovium [16–18]. It is a rare disease with an incidence of 1.8 per 1,000,000 people, affecting males and females with equal prevalence and most commonly occurs between the ages of 20 and 50 years [19, 20].

Clinical Presentation

PVNS can affect any synovial joint but has a predilection to impact the knee (75%), hip (15%), ankle, and shoulder [20]. Monoarticular involvement of the knee occurs in 66–80% of cases, and involvement of more than one joint is rare [21]. Synovial involvement can be focal; however, diffuse involvement of the affected joint is more common.

Patients with localized PVNS may present with a tender, confined bulging of the synovium. Mechanical symptoms of catching and locking are frequently reported (38%) and can often lead to a misdiagnosis of meniscal pathology [22]. Diffuse involvement of the synovium typically presents with generalized joint effusion, limited range of motion, and tenderness to palpation. Relevant laboratory studies (ESR, CRP,

WBC) are usually within normal limits. Joint fluid aspiration can aid in the diagnosis, as the aspirate often contains an elevated cholesterol content in the setting of normal blood cholesterol levels, and it is serosanguinous in nature with a prevalence of hemarthrosis in 75% of PVNS cases [21–25].

Histopathology

The gold standard for diagnosis of PVNS is synovial biopsy. The predominant lesion-defining cells are proliferating, polyhedral, mononuclear synovial cells that contain vesicular nuclei, abundant cytoplasm, and hemosiderin pigment. The cell population within the lesion contains foamy histiocytes, mononuclear cells, and giant cells [20, 26].

Imaging

Conventional radiographs alone are often nondiagnostic when evaluating a patient for PVNS. Joint effusion and dense soft tissue swelling are common but nonspecific. Few exhibit cystic changes or osteophyte formation, and there is usually an absence of periarticular osteopenia, which is helpful to differentiate PVNS from an inflammatory arthritis [15, 17, 27]. Advanced disease may result in joint space narrowing and periarticular erosion of bone particularly in the hip and ankle. Although less common in the knee secondary to the relatively lower intra-articular pressure, bony change may occur in response to the soft tissue proliferation (Fig. 10.1) [24, 28–31].

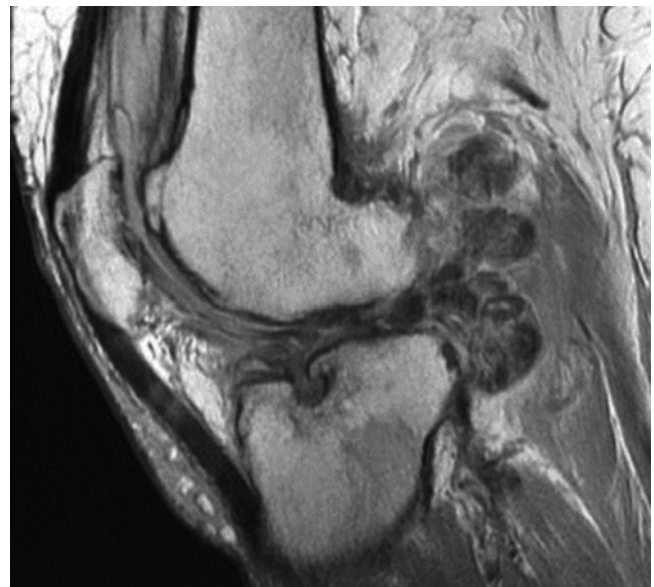


Fig. 10.1 Sagittal proton-density magnetic resonance image without fat saturation illustrating pigmented villonodular synovitis with bony erosion of medial tibial plateau

MR imaging is the method of choice for diagnosis, surgical planning, and evaluation of PVNS [14, 15, 32, 33]. Characteristic MR imaging features of PVNS enable a diagnosis to be made in 83–95 % of cases [24, 32].

In localized or focal PVNS, the classic MR image appearance is a solitary ovoid lesion with low signal intensity on both T1- and T2-weighted imaging. The most common location is the infrapatellar fat pad but areas may also include the suprapatellar pouch, intercondylar notch, and lateral synovial gutter [15, 22, 32].

In diffuse PVNS, MR images of the knee reveal synovial thickening, hypertrophy, and irregularity most commonly in the regions of the suprapatellar bursa and posterior joint recess. The most reliable diagnostic feature is the deposition of hemosiderin-laden macrophages viewed on T1- and T2-weighted images and on echo gradient imaging. As seen in Fig. 10.2, the synovial thickening can produce diffuse low-signal-intensity masses. On gradient echo imaging, these demonstrate a characteristic “blooming” artifact of hemosiderin deposition due to the paramagnetic effects of the iron [15, 32]. Diffuse PVNS exhibits an extensive pattern of hemosiderin deposition within the synovium resulting in frond-like low signal irregularities within the joint capsule. Enhancement is seen on T1-weighted images after IV gadolinium injection. Diffuse disease can extend beyond the joint capsule, and common extra-articular sites of involvement around the knee include the semimembranosus bursa, the popliteus tendon sheath, and previous arthroscopy portals.

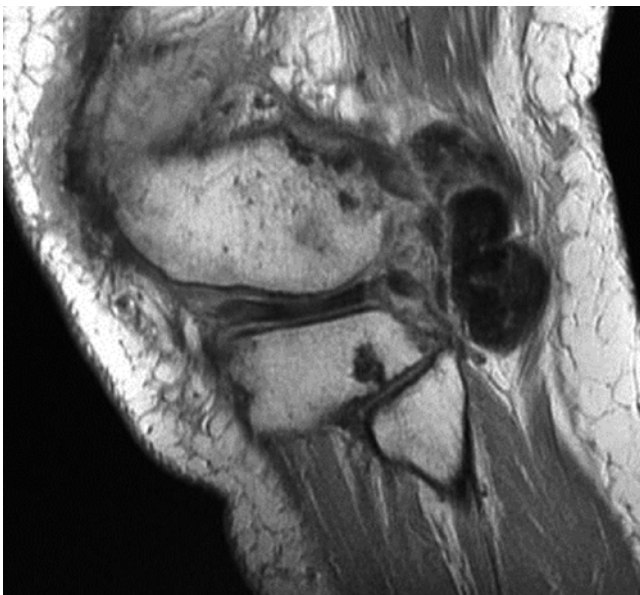


Fig. 10.2 Sagittal proton-density magnetic resonance image without fat saturation reveals low-signal mass extending from the posterior joint space of the knee

Treatment

Localized PVNS

Lesions can be sessile or pedunculated and are often limited to a single location, most commonly intra-articular in location. Nonoperative management may include physical therapy and intra-articular corticosteroid injections; however, these are of limited utility in transiently improving mechanical symptoms and offer no benefit for definitive treatment.

Nodular excision with partial synovectomy (Fig. 10.3) can often be performed arthroscopically with relief of symptoms and 0 % recurrence rate at short- and mid-term follow-up [22, 27, 34–37]. Open local excision may be performed for extra-articular involvement and for inaccessible intra-articular disease such as the posterior knee or fixed synovial disease [17].

Diffuse PVNS

Although considered to be a benign disease, diffuse PVNS can be locally aggressive and cause joint damage. Like local PVNS, nonoperative management may include physical therapy and corticosteroid injections to temporarily improve localized pain and swelling; however, this will not treat the underlying disease pathology. External beam radiation therapy has been described as primary treatment for unresectable lesions or in nonoperative candidates with recurrence rates of 7–67 % [38–40].

Surgical treatment for diffuse PVNS remains controversial and depends on the extent of the disease. Some authors advocate arthroscopic excision with complete synovectomy as the treatment of choice [34, 35, 41]. Arthroscopic treatment



Fig. 10.3 Arthroscopic view of focal, localized pigmented villonodular synovitis lesion

offers the patient a faster postoperative recovery to baseline function and decreased likelihood of stiffness [17, 27]. However, incomplete excision is not uncommon and often leads to recurrence. Recurrence rates approach 25 % for intra-articular involvement and 50 % for extra-articular disease, depending on the extent of the primary excision [20, 42]. For this reason, many surgeons prefer open arthroscopy excision with complete synovectomy, particularly for large-volume diffuse PVNS [17, 26, 42–44]. An open, posterior approach may also be necessary to excise extra-articular extension along tendons within the popliteal fossa. Adjuvant radiosynovectomy with intra-articular injection of yttrium-90 can be used after synovectomy to decrease recurrence rates of intra-articular lesions; however, there is a paucity of evidence and limited case series [45–47].

Case 2: Synovial Chondromatosis

A 46-year-old male presents with insidious-onset pain, swelling, crepitus, and decreased range of motion in his right knee for the past 2 years. He denies any history of trauma. On physical examination, an effusion is present and there is a palpable suprapatellar mass.

Introduction

Primary synovial chondromatosis is a benign disease first described by Leannac in 1813 but not formally named until 1958 by Jaffe [48–51]. It is rare and has an incidence of 1 per 100,000 people [52, 53]. It is two to four times more likely to develop in men and most commonly presents in the third to fifth decades of life [48, 49, 54]. Although clonal abnormalities and rare malignant changes have been reported in synovial chondromatosis, the disease process is generally categorized as a metaplastic rather than neoplastic disease of synovial cells [55–59]. It is characterized by chondroid metaplasia of synovial mesenchymal cells with resultant formation of lobulated, pedunculated intra-articular chondral bodies. These often shed into the joint, which may then ossify (osteochondromatosis). In 1977, Milgram described a clinical progression of this disease process by delineating its course into three phases: (1) active intrasynovial disease only with no loose bodies; (2) transitional lesions with both active, intrasynovial proliferation and free loose bodies; and (3) multiple, free osteochondral bodies with no demonstrable intrasynovial disease [60].

Secondary synovial chondromatosis is associated with mechanical or arthritic joint abnormalities, which result in the formation of loose, intra-articular chondral bodies [61].

Clinical Presentation

Synovial chondromatosis is classically a monoarticular disease, although polyarticular involvement has been described in up to 5 % of cases [53, 62]. The knee is the most commonly involved joint (50–65 %), followed by the hip, elbow, and shoulder [49, 53, 63]. Bilateral knee involvement has been reported in up to 10 % of cases; however, most cases are likely a representation of secondary synovial chondromatosis [54]. Primary synovial chondromatosis is most commonly diffuse, involving the entire synovium of the affected joint; however, localized disease has been described [64, 65].

The most common sites of involvement within the knee are the suprapatellar pouch, infrapatellar fat pad, and the anterior interval between the ACL and the infrapatellar fat pad [66, 67]. Less commonly, the posterior compartment (posterior to the PCL) may be involved [65, 66, 68, 69].

Patients present with a subacute onset of pain (85–100 %), swelling (40–58 %), and limited range of motion (35–55 %). Patients seldom recall an antecedent trauma to the knee and have no apparent systemic signs of infection or illness. On physical exam, patients often have an effusion, tenderness to palpation, articular crepitus (20–33 %), locking (5–10 %), palpable nodules, or a distinct mass (5–20 %) [54, 64, 70–72].

Histopathology

The gross appearance of synovial chondromatosis consists of hyperplastic synovium overlying white, nodular projections of hyaline cartilage diffusely scattered across the entire joint surface [49, 54, 73–75]. This often gives the synovium a “cobblestone” appearance. The nodules may detach from the synovium, thus creating free chondral bodies within the affected joint. The number of nodules can range from a few to thousands, depending on the stage of the disease. They vary in size from a few millimeters to a few centimeters in diameter [49, 54]. As the nodules increase in size, the central zone can undergo calcification and, rarely, endochondral ossification. Multiple detached nodules may coalesce to form a large mass termed giant synovial chondromas, though this is rare [54, 76].

Imaging

As seen in Fig. 10.4, conventional roentgenograms reveal intra-articular ossified bodies in 70–95 % of cases of primary synovial chondromatosis. Characteristically, they are innumerable, similar in size and shape, and evenly dispersed throughout the synovial lining of the affected joint



Fig. 10.4 Lateral radiograph of the knee, revealing multiple ossified bodies in the posterior compartment

[48, 49, 54, 73, 75, 77]. Periarticular bony erosions are common in more constrained joints such as the wrist, elbow, and hip; however, these are less likely in the knee and shoulder [78].

Computed tomography (CT) imaging of the knee can be useful in differentiating primary synovial chondromatosis from other causes of soft tissue lesions, particularly when conventional roentgenograms are equivocal. Hyaline cartilage has a high water content and therefore low attenuation on CT imaging, which can be appreciated in the nonmineralized synovial thickening caused by synovial chondromatosis. Furthermore, the majority of nodules caused by synovial chondromatosis contain central and peripheral ossification which CT imaging delineates as a “ring-and-arc” pattern of mineralization or a target appearance [53, 54]. However, purely chondral lesions are better defined by magnetic resonance imaging.

MR imaging of the knee provides the optimal modality to aid in the diagnosis and treatment plan for primary synovial chondromatosis. Due to the heterogeneous makeup of calcification and ossification of the nodules, variable signal characteristics are identified, as illustrated in Figs. 10.5 and 10.6 [54, 76, 79–81]. The most common pattern (77 %) demonstrates low/intermediate signal intensity on T1-weighted sequences and high signal intensity on T2-weighted sequences. This correlates clinically with the Milgram phase 2 lesions, as described above [54, 76].

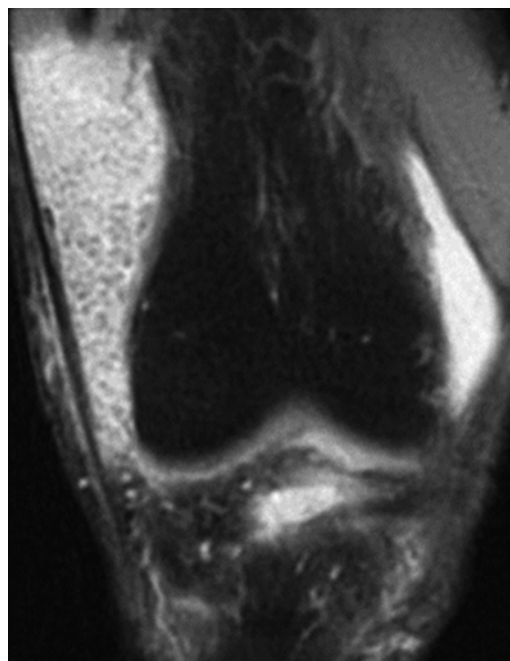


Fig. 10.5 Coronal fat-saturated T2-weighted magnetic resonance image revealing numerous low-signal osteochondral bodies

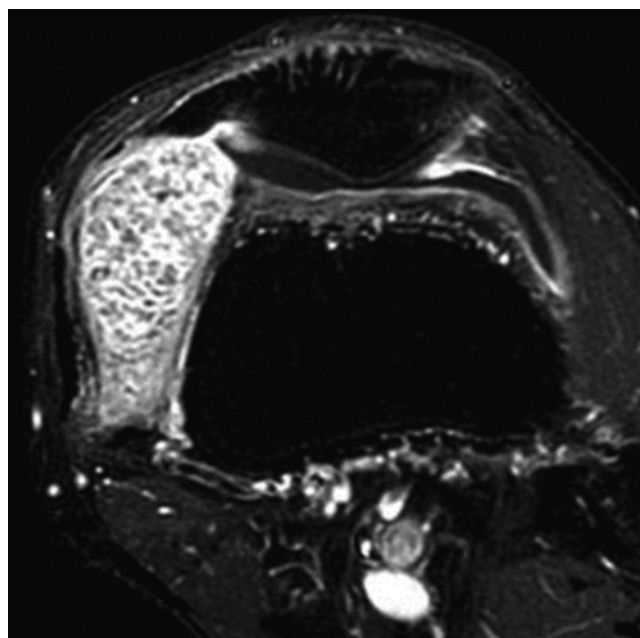


Fig. 10.6 Axial fat-saturated T1-weighted magnetic resonance image post IV gadolinium demonstrates multiple low-signal osteochondral bodies within the enhancing synovium

Treatment

Patients presenting with recurrent pain, swelling, and mechanical symptoms refractory to nonoperative management are candidates for surgical intervention. Primary synovial chondromatosis

is generally a benign and self-limiting disease; however, it may be progressive and complicated by osteoarthritis. On rare occasions, synovial chondrosarcoma can arise in the setting of synovial chondromatosis (5 % incidence) [54, 75, 82]. Therefore, the treatment of choice is surgical excision of the osteochondral nodules with or without partial or complete synovectomy, depending on the extent of the disease. Surgical excision can be performed by open or arthroscopic approach, and the technique should be selected based upon ability to safely access and thoroughly remove all the diseased synovium and nodules.

Arthroscopic excision offers the surgeon better visualization of the intra-articular synovium and decreased pain, stiffness, and postoperative rehabilitation course for the patient [65, 69, 83]. The need for partial or complete synovectomy for treatment of primary synovial chondromatosis remains controversial. Milgram recommended treating patients with phase 1 primary synovial chondromatosis (active intrasynovial disease only with no loose bodies) with synovectomy. For phase 2 (transitional form with active intrasynovial disease and chondral bodies), he recommended synovectomy with chondral body retrieval. Lastly, for phase 3 (late inactive intrasynovial disease with chondral bodies but no synovial abnormality), he recommended chondral body retrieval alone [60]. With this treatment regimen, Milgram observed recurrence rates of 12.5 %, 10 %, and 0 % for phases 1, 2, and 3, respectively [54, 60]. The recurrence rate for larger series ranges from 3 to 23 % and recurrence is most likely related to incomplete primary excision [63, 64, 70]. Ogilvie-Harris and Saleh reported nearly a 60 % recurrence rate when treating patients with loose chondral body retrieval alone [84]. Therefore, arthroscopic retrieval of chondral loose bodies and complete synovectomy is the treatment of choice for primary synovial chondromatosis. This was performed in our patient, as seen in Figs. 10.7 and 10.8.

Case 3: Synovial Hemangioma

An otherwise healthy 13-year-old female with no history of trauma presents with recurrent effusions and decreased range of motion in her right knee.

Introduction

Hemangiomas and hemangiohamartomas are types of rare, benign vascular tumors that may affect the musculoskeletal system. Hemangiohamartomas, more commonly known as arteriovenous malformations, are differentiated from hemangiomas in that they contain fat, connective tissue, and peripheral nerve structures [29]. Hemangiomas, however,

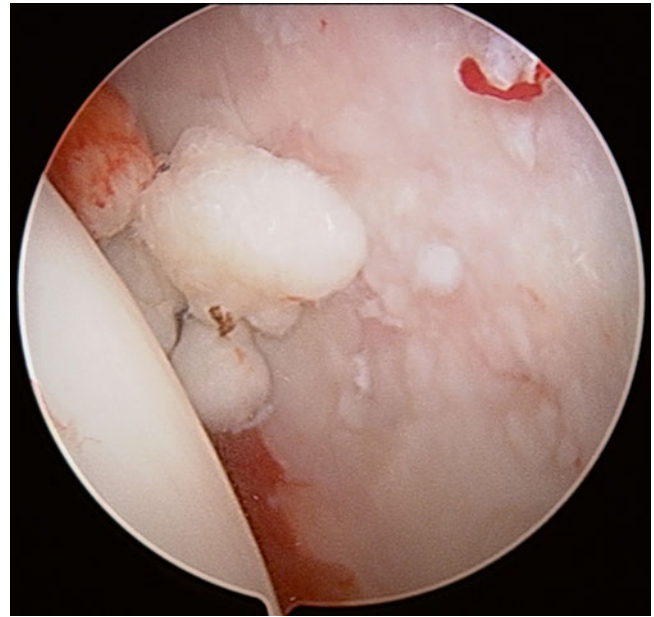


Fig. 10.7 Arthroscopic view of numerous osteochondral bodies embedded within the synovium



Fig. 10.8 Arthroscopic view of multiple osteochondral bodies and inflamed synovium

are composed primarily of closely packed thin-walled capillaries. Depending on their location in relation to the joint, hemangiomas may be described as juxta-articular (outside the joint capsule but in relation to it), intra-articular, extra-articular, or intermediate (both intra- and extra-articular) [85–87]. Both the intra-articular and intermediate types

generally involve the synovial membrane. Synovial hemangiomas were first described by Bouchut in 1856 and further categorized as being either localized or diffuse in character by Bennett and Cobey in 1939 [88, 89]. They are exceedingly rare, with approximately 200 reported cases [90, 91]. Most commonly, they occur in children and adolescents with the average age of onset of 10.9 years in females and 12.5 years in males [92, 93]. Synovial hemangiomas are slightly more common in females (53 %) [91, 94, 95].

Clinical Presentation

Synovial hemangiomas have been reported in the wrist, ankle, and elbow, but they most commonly involve the knee [87, 91, 92, 96, 97]. The tumor may be localized and well circumscribed or diffuse in character. Patients often present with an insidious onset of knee pain and recurrent effusions with associated limitation in range of motion. Atrophy of the quadriceps muscle is common in patients with a synovial hemangioma [87, 95, 98]. Patients with localized and pedunculated synovial hemangiomas may have a palpable soft tissue mass and often experience mechanical symptoms. Diffuse involvement can lead to more pronounced synovial venous congestion and hemorrhagic synovitis. Patients with both localized and diffuse subtypes often have a diagnostic delay, because of the rarity of synovial hemangiomas [99–102]. This delay leads to many patients having a history of spontaneous swelling and recurrent hemarthrosis in the absence of coagulopathy. In up to 40 % of cases, the patient may present with overlying cutaneous hemangiomas and venous congestion of the knee [91, 93, 103, 104].

Histopathology

Synovial hemangiomas can be classified as cavernous, capillary, mixed, or arteriovenous [91, 105]. Most commonly, they exhibit features of cavernous hemangiomas with lobulated proliferation of capillary-sized vascular channels. Inflammatory cells and hemosiderin-laden macrophages are frequently identified, thus resembling pigmented villonodular synovitis. However, the existence of dilated, thin-walled capillaries with cavernous appearance differentiates PVNS from synovial hemangiomas [91, 97].

Imaging

Conventional roentgenograms may reveal the presence of an effusion, soft tissue mass, phleboliths, advanced maturation of the epiphysis, or periosteal reaction [95, 100, 103, 106].

Frequently, however, radiographs show no abnormalities and further imaging is necessary. CT imaging can aid in the diagnosis to rule out bony abnormality; however, the demarcation between soft tissue mass and muscle is limited.

MR imaging does not use ionizing radiation and provides better soft tissue differentiation to demonstrate the extent of involvement of the synovial hemangioma [105, 107–109]. In conventional MR imaging without contrast, the lesions are lobulated with well-defined margins and exhibit homogeneous, low-to-iso-signal intensity on T1-weighted imaging and heterogeneous, high signal intensity on T2-weighted imaging, as seen in Figs. 10.9 and 10.10 [90, 100, 103, 106, 110, 111]. The use of gadolinium contrast has been advocated by some authors to differentiate synovial hemangiomas from ganglion cysts, cystic synovial hyperplasia, synovial sarcoma, leiomyoma, and synovial chondromatosis [90]. With Gd-enhancement, synovial hemangiomas exhibit heterogeneous enhancement (Fig. 10.11). This has been useful to differentiate synovial hemangiomas from other intra-articular synovial lesions with the exception of synovial sarcomas which can display similar characteristics. However, these malignant lesions most commonly arise extra-articularly [90, 112]. MR imaging allows for accurate preoperative assessment in the classification of the lesion, thus guiding a definitive treatment strategy.

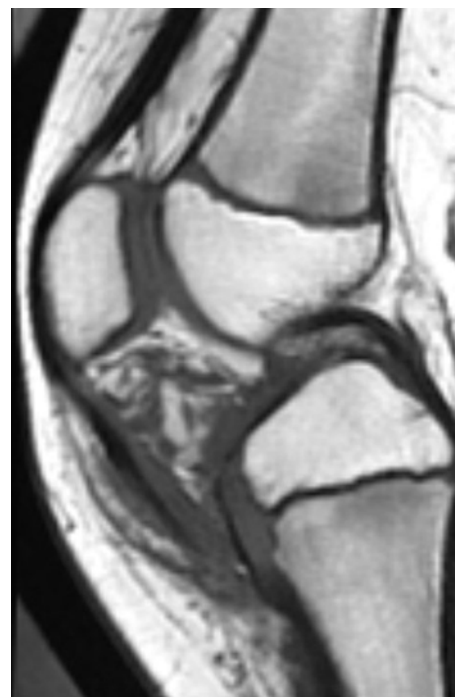


Fig. 10.9 Sagittal T1-weighted magnetic resonance image revealing infrapatellar lesion with internal fat signal



Fig. 10.10 Sagittal T2-weighted magnetic resonance image revealing infrapatellar lesion with internal high T2 signal and suggestion of peripheral vessels



Fig. 10.11 Sagittal fat-saturated T1-weighted magnetic resonance image post IV gadolinium enhancement demonstrates discrete vessels and strong enhancement consistent with synovial hemangioma

Treatment

Aside from pain, mechanical symptoms, and decreased range of motion, synovial hemangiomas frequently cause recurrent hemarthrosis, which can lead to early destruction of the articular surface of the knee. Therefore, several treatment options have been described for synovial hemangiomas including embolization and open or arthroscopic surgical excision with and without partial synovectomy. Like other synovial disorders, the treatment of choice depends on the classification and extent of involvement.

Localized synovial hemangiomas can often be treated successfully with arthroscopic surgical excision, especially when the lesion is pedunculated and well circumscribed [91, 105, 113, 114]. Frequently, localized lesions will have a single vascular supply that can be managed arthroscopically with minimal bleeding.

Diffusely involved synovial hemangiomas in a single compartment of the knee are better treated with open arthrotomy, surgical excision, and partial synovectomy [95, 98, 115, 116]. They often have multiple feeding vessels; therefore, preoperative angiography with embolization may improve surgical results and decrease postoperative bleeding [91, 99, 101]. However, in diffuse synovial hemangiomas that involve multiple compartments of the knee, nonoperative management with repeat imaging to evaluate progression has been encouraged [91, 98].

Case 4: Lipoma Arborescens

A 45-year-old male with diabetes mellitus presents with complaints of mechanical symptoms in his right knee and a palpable, tender mass over the superior aspect of his patella.

Introduction

Lipoma arborescens (diffuse articular lipomatosis) is a rare, benign intra-articular disease affecting the synovial membrane. First described by in 1957 by Arzimanoglu, it is characterized as a diffuse substitution of the joint's subsynovial layer by mature adipocytes, resulting in the formation of villous projections [117–125]. Numerous villous lipomatous synovial proliferations differentiate lipoma arborescens from an intra-articular lipoma [120, 121, 126]. The etiology of lipoma arborescens remains unclear; however, developmental, traumatic, inflammatory, and neoplastic origins have been postulated [118, 123, 124]. Since its first description, there have been approximately 75 case reports presented in the literature [127]. There is a slight male preponderance (56–70 %) and the age range of disease onset

is 10–90 years with a mean age of 37 years [127–129]. Associated conditions include osteoarthritis, rheumatoid arthritis, psoriatic arthritis, gout, joint trauma, and diabetes mellitus [118, 128, 130–133].

Clinical Presentation

Lipoma arborescens is a rare disorder that can affect the hip, shoulder, wrist, elbow, and ankle, but it most commonly affects the knee [117, 119, 124, 128, 129, 134]. Most cases are monoarticular; however, bilateral knee involvement has been described in up to 16 % of cases [127, 129]. The suprapatellar pouch is invariably involved. Other common sites include the condylar gutters and the premeniscal regions [117]. Patients with lipoma arborescens typically present with painless swelling of the knee, which leads to progressive restriction of range of motion. They often have intermittent exacerbations with resultant mechanical symptoms, as the villous projections can become trapped within the joint resulting in catching and locking [124]. Laboratory testing (ESR, CRP, WBC, uric acid, rheumatoid factor, HLA-B27) in patients with lipoma arborescens is normal [123, 129, 135]. Joint fluid aspiration reveals a serous appearance, is negative for cells and crystals, and is sterile on culture [124].

Histopathology

Macroscopically, lipoma arborescens is a fatty tissue mass with frond-like appearance. The finger-shaped synovial projections are numerous with broad bases [123, 127, 129, 136, 137]. Histologically, the disease is characterized by diffuse replacement of the subsynovial layer with mature adipocytes. Focal infiltration of perivascular mononuclear inflammatory cells is often appreciated [118, 119, 129].

Imaging

Conventional roentgenogram is of limited utility in the diagnosis of lipoma arborescens but may indicate the presence of a soft tissue mass and help to rule out bony involvement. CT imaging often demonstrates a soft tissue synovial mass and the characteristic synovial fronds may be outlined by an adjacent effusion. Low attenuation measurements are consistent with fat, and there is little or no enhancement after contrast administration [123, 130, 135, 138].

MR imaging is the modality of choice when evaluating a patient for lipoma arborescens. MR imaging reveals villous synovial thickening with fatty frond-like projections, usually



Fig. 10.12 Sagittal T1-weighted magnetic resonance image showing frond-like, villous proliferation of fat within the suprapatellar synovium, consistent with lipoma arborescens

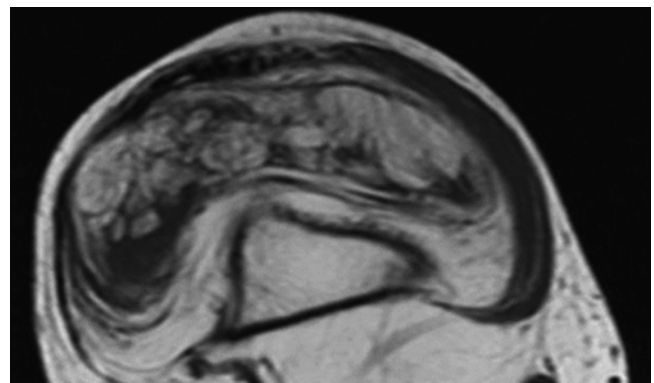


Fig. 10.13 Axial T1-weighted magnetic resonance image reveals frond-like, villous proliferation of fat within suprapatellar synovium, consistent with lipoma arborescens

with an effusion. The uniform fat signal and lack of hemosiderin aid diagnosis and differentiation from other lesions. (Fig. 10.12) [117, 133, 134, 139, 140]. Associated findings on MR imaging include chondral degenerative changes (87 %) and meniscal tears (72 %) [141]. Similar to that of subcutaneous fat, lipoma arborescens has high signal intensity on T1 (Fig. 10.13) and low signal on fat-saturated and STIR sequences (Fig. 10.14) [127, 141].

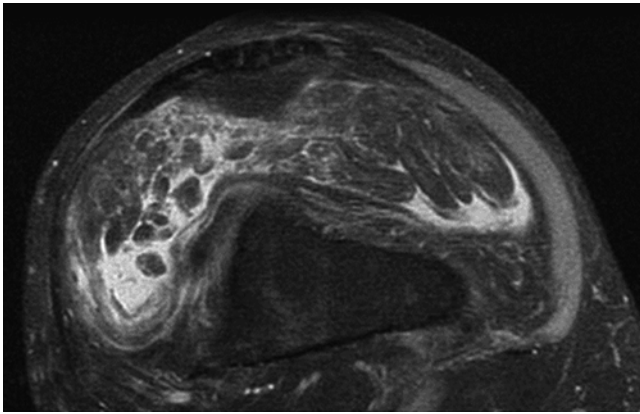


Fig. 10.14 Axial fat-saturated T2-weighted magnetic resonance image shows complete saturation of the signal forming the villous structures, therefore confirming their fatty composition

Treatment

Lipoma arborescens causes recurrent painless swelling of the knee that often leads to mechanical symptoms. Nonoperative management may consist of physical therapy and corticosteroid injections to abate frequent exacerbations. Asymptomatic and incidentally identified lesions do not require a surgical intervention.

Our patient presented with symptoms unresponsive to nonoperative treatment and elected to undergo an excision with partial synovectomy, as is the treatment of choice [123, 124]. Prior to 1998, reported lesions were excised via arthrotomy with partial or total synovectomy leading to low rates of recurrence. Sola and colleagues were the first to report successful arthroscopic excision of a lipoma arborescens lesion without recurrence [123]. Currently, the preferred treatment is arthroscopic excision of the mass with anterior synovectomy [117, 127, 142, 143]. Although some patients may have recurrent effusions postoperatively, likely secondary to arthritic changes, actual recurrence of lipoma arborescens is rare [142, 144].

References

- Smith MD. The normal synovium. *Open Rheumatol J.* 2011; 5:100–6.
- Cohen MJ, Kaplan L. Histology and ultrastructure of the human flexor tendon sheath. *J Hand Surg Am.* 1987;12(1):25–9.
- Schmidt D, Mackay B. Ultrastructure of human tendon sheath and synovium: implications for tumor histogenesis. *Ultrastruct Pathol.* 1982;3(3):269–83.
- Chung CB, Boucher R, Resnick D. MR imaging of synovial disorders of the knee. *Semin Musculoskelet Radiol.* 2009;13(4): 303–25.
- Garcia-Valtuille R, Abascal F, Cerezal L, Garcia-Valtuille A, Pereda T, Canga A, et al. Anatomy and MR imaging appearances

- of synovial plicae of the knee. *Radiographics.* 2002;22(4): 775–84.
- Kosarek FJ, Helms CA. The MR appearance of the infrapatellar plica. *AJR Am J Roentgenol.* 1999;172(2):481–4.
- Lee SH, Petersilge CA, Trudell DJ, Haghighi P, Resnick DL. Extrasynovial spaces of the cruciate ligaments: anatomy, MR imaging, and diagnostic implications. *AJR Am J Roentgenol.* 1996;166(6):1433–7.
- Fenn S, Datir A, Saifuddin A. Synovial recesses of the knee: MR imaging review of anatomical and pathological features. *Skeletal Radiol.* 2009;38(4):317–28.
- De Maeseneer M, Van Roy P, Shahabpour M, Gosselin R, De Ridder F, Osteaux M. Normal anatomy and pathology of the posterior capsular area of the knee: findings in cadaveric specimens and in patients. *AJR Am J Roentgenol.* 2004;182(4):955–62.
- O’Connell JX. Pathology of the synovium. *Am J Clin Pathol.* 2000;114(5):773–84.
- Boegard T, Johansson A, Rudling O, Petersson I, Forslind K, Jonsson K. Gadolinium-DTPA-enhanced MR imaging in asymptomatic knees. *Acta Radiol.* 1996;37(6):877–82.
- Jaffe HL, Lichtenstein L, Sutro CJ. Pigmented villonodular synovitis, bursitis and tenosynovitis. *Arch Pathol.* 1941;31:731–65.
- Barile A, Sabatini M, Iannesi F, Di Cesare E, Splendiani A, Calvisi V, et al. Pigmented villonodular synovitis (PVNS) of the knee joint: magnetic resonance imaging (MRI) using standard and dynamic paramagnetic contrast media. Report of 52 cases surgically and histologically controlled. *Radiol Med.* 2004;107(4):356–66.
- Hughes TH, Sartoris DJ, Schweitzer ME, Resnick DL. Pigmented villonodular synovitis: MRI characteristics. *Skeletal Radiol.* 1995;24(1):7–12.
- Masih S, Antebi A. Imaging of pigmented villonodular synovitis. *Semin Musculoskelet Radiol.* 2003;7(3):205–16.
- West RB, Rubin BP, Miller MA, Subramanian S, Kaygusuz G, Montgomery K, et al. 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.* 2006;103(3):690–5.
- van der Heijden L, Gibbons CL, Dijkstra PD, Kroep JR, van Rijswijk CS, Nout RA, et al. The management of diffuse-type giant cell tumour (pigmented villonodular synovitis) and giant cell tumour of tendon sheath (nodular tenosynovitis). *J Bone Joint Surg Br.* 2012;94(7):882–8.
- Nilsson M, Hoglund M, Panagopoulos I, Sciort R, Dal Cin P, Debiec-Rychter M, et al. Molecular cytogenetic mapping of recurrent chromosomal breakpoints in tenosynovial giant cell tumors. *Virchows Arch.* 2002;441(5):475–80.
- Beguin J, Locker B, Vielpeau C, Souquieres G. Pigmented villonodular synovitis of the knee: results from 13 cases. *Arthroscopy.* 1989;5(1):62–4.
- de St. Aubain Somerhausen N. Diffuse-type giant cell tumour. In: Fletcher C, Unni K, Mertens F, editors. *Pathology and genetics of tumours of soft tissue and bone (Series).* Lyon: IARC; 2002.
- Dorwart RH, Genant HK, Johnston WH, Morris JM. Pigmented villonodular synovitis of the shoulder: radiologic-pathologic assessment. *AJR Am J Roentgenol.* 1984;143(4):886–8.
- Dines JS, DeBerardino TM, Wells JL, Dodson CC, Shindle M, DiCarlo EF, et al. Long-term follow-up of surgically treated localized pigmented villonodular synovitis of the knee. *Arthroscopy.* 2007;23(9):930–7.
- Breimer CW, Freiburger RH. Bone lesions associated with villonodular synovitis. *Am J Roentgenol Radium Ther Nucl Med.* 1958;79(4):618–29.
- Ottaviani S, Ayril 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): 539–46.

25. Zwass A, Abdelwahab IF, Klein MJ. Case report 463: pigmented villonodular synovitis (PVNS) of knee. *Skeletal Radiol.* 1988; 17(1):81–4.
26. Sharma H, Rana B, Mahendra A, Jane MJ, Reid R. Outcome of 17 pigmented villonodular synovitis (PVNS) of the knee at 6 years mean follow-up. *Knee.* 2007;14(5):390–4.
27. Flandry F, McCann SB, Hughston JC, Kurtz DM. Roentgenographic findings in pigmented villonodular synovitis of the knee. *Clin Orthop Relat Res.* 1989;247:208–19.
28. Merry P, Williams R, Cox N, King JB, Blake DR. Comparative study of intra-articular pressure dynamics in joints with acute traumatic and chronic inflammatory effusions: potential implications for hypoxic-reperfusion injury. *Ann Rheum Dis.* 1991; 50(12):917–20.
29. Visuri T, Kiviluoto O. Arthroscopic volume of the knee joint in young male adults. *Scand J Rheumatol.* 1986;15(3):251–4.
30. Draeger RW, Singh B, Parekh SG. Quantifying normal ankle joint volume: an anatomic study. *Indian J Orthop.* 2009;43(1):72–5.
31. Yen CH, Leung HB, Tse PY. Effects of hip joint position and intra-capsular volume on hip joint intra-capsular pressure: a human cadaveric model. *J Orthop Surg Res.* 2009;4:8.
32. Cheng XG, You YH, Liu W, Zhao T, Qu H. MRI features of pigmented villonodular synovitis (PVNS). *Clin Rheumatol.* 2004;23(1):31–4.
33. Steinbach LS, Neumann CH, Stoller DW, Mills CM, Crues 3rd JV, Lipman JK, et al. MRI of the knee in diffuse pigmented villonodular synovitis. *Clin Imaging.* 1989;13(4):305–16.
34. Ogilvie-Harris DJ, McLean J, Zarnett ME. Pigmented villonodular synovitis of the knee. The results of total arthroscopic synovectomy, partial, arthroscopic synovectomy, and arthroscopic local excision. *J Bone Joint Surg Am.* 1992;74(1):119–23.
35. De Ponti A, Sansone V, Malchere M. Result of arthroscopic treatment of pigmented villonodular synovitis of the knee. *Arthroscopy.* 2003;19(6):602–7.
36. Kim SJ, Choi NH, Lee SC. Tenosynovial giant-cell tumor in the knee joint. *Arthroscopy.* 1995;11(2):213–5.
37. Ozalay M, Tandogan RN, Akpınar S, Cesur N, Hersekli MA, Ozkoc G, et al. Arthroscopic treatment of solitary benign intra-articular lesions of the knee that cause mechanical symptoms. *Arthroscopy.* 2005;21(1):12–8.
38. Berger B, Ganswindt U, Bamberg M, Hehr T. External beam radiotherapy as postoperative treatment of diffuse pigmented villonodular synovitis. *Int J Radiat Oncol Biol Phys.* 2007;67(4): 1130–4.
39. Horoschak M, Tran PT, Bachireddy P, West RB, Mohler D, Beaulieu CF, et al. External beam radiation therapy enhances local control in pigmented villonodular synovitis. *Int J Radiat Oncol Biol Phys.* 2009;75(1):183–7.
40. O'Sullivan B, Cummings B, Catton C, Bell R, Davis A, Fornasier V, et al. Outcome following radiation treatment for high-risk pigmented villonodular synovitis. *Int J Radiat Oncol Biol Phys.* 1995;32(3):777–86.
41. Blanco CE, Leon HO, Guthrie TB. Combined partial arthroscopic synovectomy and radiation therapy for diffuse pigmented villonodular synovitis of the knee. *Arthroscopy.* 2001;17(5):527–31.
42. Schwartz HS, Unni KK, Pritchard DJ. Pigmented villonodular synovitis. A retrospective review of affected large joints. *Clin Orthop Relat Res.* 1989;247:243–55.
43. Akinci O, Akalin Y, Incesu M, Eren A. Long-term results of surgical treatment of pigmented villonodular synovitis of the knee. *Acta Orthop Traumatol Turc.* 2011;45(3):149–55.
44. Mankin H, Trahan C, Hornicek F. Pigmented villonodular synovitis of joints. *J Surg Oncol.* 2011;103(5):386–9.
45. Ozturk H, Bulut O, Oztemur Z, Bulut S. Pigmented villonodular synovitis managed by Yttrium 90 after debulking surgery. *Saudi Med J.* 2008;29(8):1197–200.
46. Shabat S, Kollender Y, Merimsky O, Isakov J, Flusser G, Nyska M, et al. The use of surgery and yttrium 90 in the management of extensive and diffuse pigmented villonodular synovitis of large joints. *Rheumatology (Oxford).* 2002;41(10):1113–8.
47. Kat S, Kutz R, Elbracht T, Weseloh G, Kuwert T. Radiosynovectomy in pigmented villonodular synovitis. *Nuklearmedizin.* 2000; 39(7):209–13.
48. Crotty JM, Monu JU, Pope Jr TL. Synovial osteochondromatosis. *Radiol Clin North Am.* 1996;34(2):327–42, xi.
49. Dorfman HD, Czerniak B. Synovial lesions. In: Dorfman HD, Czerniak B, editors. *Bone tumors (Series).* St. Louis, MO: Mosby; 1998.
50. Fanburg-Smith JC. Cartilage and bone-forming tumors and tumor-like lesions. In: Miettinen M, editor. *Diagnostic soft tissue pathology (Series).* Philadelphia, PA: Churchill-Livingstone; 2003.
51. Jaffe HL. *Tumours and tumorous conditions of bones and joints.* 1st ed. Philadelphia, PA: Lea & Fabiger; 1958.
52. Felbel J, Gresser U, Lohmoller G, Zollner N. Familial synovial chondromatosis combined with dwarfism. *Hum Genet.* 1992;88(3):351–4.
53. McKenzie G, Raby N, Ritchie D. A pictorial review of primary synovial osteochondromatosis. *Eur Radiol.* 2008;18(11):2662–9.
54. Murphey MD, Vidal JA, Fanburg-Smith JC, Gajewski DA. Imaging of synovial chondromatosis with radiologic-pathologic correlation. *Radiographics.* 2007;27(5):1465–88.
55. Nakanishi S, Sakamoto K, Yoshitake H, Kino K, Amagasa T, Yamaguchi A. Bone morphogenetic proteins are involved in the pathobiology of synovial chondromatosis. *Biochem Biophys Res Commun.* 2009;379(4):914–9.
56. Mertens F, Jonsson K, Willen H, Rydholm A, Kreicbergs A, Eriksson L, et al. Chromosome rearrangements in synovial chondromatous lesions. *Br J Cancer.* 1996;74(2):251–4.
57. Sciot R, Dal Cin P, Bellemans J, Samson I, Van den Berghe H, Van Damme B. Synovial chondromatosis: clonal chromosome changes provide further evidence for a neoplastic disorder. *Virchows Arch.* 1998;433(2):189–91.
58. Sperling BL, Angel S, Stoneham G, Chow V, McFadden A, Chibbar R. Synovial chondromatosis and chondrosarcoma: a diagnostic dilemma. *Sarcoma.* 2003;7(2):69–73.
59. Davis RI, Foster H, Arthur K, Trewin S, Hamilton PW, Biggart DJ. Cell proliferation studies in primary synovial chondromatosis. *J Pathol.* 1998;184(1):18–23.
60. Milgram JW. Synovial osteochondromatosis: a histopathological study of thirty cases. *J Bone Joint Surg Am.* 1977;59(6): 792–801.
61. Villacin AB, Brigham LN, Bullough PG. Primary and secondary synovial chondrometaplasia: histopathologic and clinicoradiologic differences. *Hum Pathol.* 1979;10(4):439–51.
62. Seckley J, Anderson SG, Snow TM, Benjamin M. A rare case of polyarticular synovial osteochondromatosis. *J Anat.* 2002; 200(5):524.
63. Maurice H, Crone M, Watt I. Synovial chondromatosis. *J Bone Joint Surg Br.* 1988;70(5):807–11.
64. Murphy FP, Dahlin DC, Sullivan CR. Articular synovial chondromatosis. *J Bone Joint Surg.* 1962;44(1):77–86.
65. Jesalpura JP, Chung HW, Patnaik S, Choi HW, Kim JI, Nha KW. Arthroscopic treatment of localized synovial chondromatosis of the posterior knee joint. *Orthopedics.* 2010;33(1):49.
66. Kyung BS, Lee SH, Han SB, Park JH, Kim CH, Lee DH. Arthroscopic treatment of synovial chondromatosis at the knee posterior septum using a trans-septal approach: report of two cases. *Knee.* 2012;19(5):732–5.
67. Bozkurt M, Ugurlu M, Dogan M, Tosun N. Synovial chondromatosis of four compartments of the knee: medial and lateral tibiofemoral spaces, patellofemoral joint and proximal tibiofibular joint. *Knee Surg Sports Traumatol Arthrosc.* 2007;15(6):753–5.

68. Church JS, Breidahl WH, Janes GC. Recurrent synovial chondromatosis of the knee after radical synovectomy and arthrodesis. *J Bone Joint Surg Br.* 2006;88(5):673–5.
69. Pengatteeeri YH, Park SE, Lee HK, Lee YS, Gopinathan P, Han CW. Synovial chondromatosis of the posterior cruciate ligament managed by a posterior-posterior triangulation technique. *Knee Surg Sports Traumatol Arthrosc.* 2007;15(9):1121–4.
70. Roulot E, Le Viet D. Primary synovial osteochondromatosis of the hand and wrist. Report of a series of 21 cases and literature review. *Rev Rhum Engl Ed.* 1999;66(5):256–66.
71. Trias A, Quintana O. Synovial chondrometaplasia: review of world literature and a study of 18 Canadian cases. *Can J Surg.* 1976;19(2):151–8.
72. Butt SH, Muthukumar T, Cassar-Pullicino VN, Mangham DC. Primary synovial osteochondromatosis presenting as constrictive capsulitis. *Skeletal Radiol.* 2005;34(11):707–13.
73. Resnick D. Tumors and tumor-like lesions of soft tissues. In: Resnick D, editor. *Diagnosis of bone and joint disorders (Series).* 4th ed. Philadelphia, PA: Saunders; 2002.
74. Unni KK, Inwards CY, Bridge JA, Kindblom LG, Wold LE. Synovial tumors. In (Editor) Unni K. Krishnan, published by the American Registry of Pathology, ISBN #9781881041931. *Tumors of the bone and joints (Series).* 4th ed. Silver Spring, MD: ARP; 2005.
75. Weiss SW, Goldblum JR. Cartilaginous soft tissue tumors. In (Editor) Sharon W. Weiss, published by Mosby, ISBN #0323012000. *Enzinger and Weiss's soft tissue tumors (Series).* 4th ed. Philadelphia, PA: Mosby; 2001.
76. Garner HW, Bestic JM. Benign synovial tumors and proliferative processes. *Semin Musculoskelet Radiol.* 2013;17(2):177–8.
77. Dunn EJ, McGavran MH, Nelson P, Greer 3rd RB. Synovial chondrosarcoma. Report of a case. *J Bone Joint Surg Am.* 1974;56(4):811–3.
78. Norman A, Steiner GC. Bone erosion in synovial chondromatosis. *Radiology.* 1986;161(3):749–52.
79. Sheldon PJ, Forrester DM, Leach TJ. Imaging of intraarticular masses. *Radiographics.* 2005;25(1):105–19.
80. Kramer J, Recht M, Deely DM, Schweitzer M, Pathria MN, Gentili A, et al. MR appearance of idiopathic synovial osteochondromatosis. *J Comput Assist Tomogr.* 1993;17(5):772–6.
81. Jaganathan S, Goyal A, Gadodia A, Rastogi S, Mittal R, Gamanagatti S. Spectrum of synovial pathologies: a pictorial assay. *Curr Probl Diagn Radiol.* 2012;41(1):30–42.
82. Kransdorf MJ, Murphey MD. *Imaging of soft tissue tumors.* 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2006.
83. Mubashir A, Bickerstaff DR. Synovial osteochondromatosis of the cruciate ligament. *Arthroscopy.* 1998;14(6):627–9.
84. Ogilvie-Harris DJ, Saleh K. Generalized synovial chondromatosis of the knee: a comparison of removal of the loose bodies alone with arthroscopic synovectomy. *Arthroscopy.* 1994;10(2):166–70.
85. DePalma AF, Mauler GG. Hemangioma of synovial membrane. *Clin Orthop Relat Res.* 1964;32:93–9.
86. Mastragostino S, Fares GC. [Synovial hemangioma of the knee]. *Riv Anat Patol Oncol.* 1959;15:824–36.
87. Halborg A, Hansen H, Sneppen HO. Haemangioma of the knee joint. *Acta Orthop Scand.* 1968;39(2):209–16.
88. Bouchut ME. Tumeur erectile de l'articulation du genou. *Gaz Hop (Paris).* 1856;29:379.
89. Bennett GE, Cobey MC. Hemangioma of joints. *Arch Surg.* 1939;38:487.
90. Sasho T, Nakagawa K, Matsuki K, Hoshi H, Saito M, Ikegawa N, et al. Two cases of synovial haemangioma of the knee joint: Gd-enhanced image features on MRI and arthroscopic excision. *Knee.* 2011;18(6):509–11.
91. Akgun I, Kesmezacar H, Ogut T, Dervisoglu S. Intra-articular hemangioma of the knee. *Arthroscopy.* 2003;19(3):E17.
92. Moon NF. Synovial hemangioma of the knee joint. A review of previously reported cases and inclusion of two new cases. *Clin Orthop Relat Res.* 1973 Jan–Feb;(90):183–90.
93. Ramseier LE, Exner GU. Arthropathy of the knee joint caused by synovial hemangioma. *J Pediatr Orthop.* 2004;24(1):83–6.
94. Rogalski R, Hensinger R, Loder R. Vascular abnormalities of the extremities: clinical findings and management. *J Pediatr Orthop.* 1993;13(1):9–14.
95. Suh JT, Cheon SJ, Choi SJ. Synovial hemangioma of the knee. *Arthroscopy.* 2003;19(7):E27–30.
96. Atkinson TJ, Wolf S, Anavi Y, Wesley R. Synovial hemangioma of the temporomandibular joint: report of a case and review of the literature. *J Oral Maxillofac Surg.* 1988;46(9):804–8.
97. Winzenberg T, Ma D, Taplin P, Parker A, Jones G. Synovial haemangioma of the knee: a case report. *Clin Rheumatol.* 2006;25(5):753–5.
98. Yilmaz E, Karakurt L, Ozdemir H, Serin E, Incesu M. [Diffuse synovial hemangioma of the knee: a case report]. *Acta Orthop Traumatol Turc.* 2004;38(3):224–8.
99. Aalberg JR. Synovial hemangioma of the knee. A case report. *Acta Orthop Scand.* 1990;61(1):88–9.
100. Cotten A, Flipo RM, Herbaux B, Gougeon F, Lecomte-Houcke M, Chastanet P. Synovial haemangioma of the knee: a frequently misdiagnosed lesion. *Skeletal Radiol.* 1995;24(4):257–61.
101. Ryd L, Stenstrom A. Hemangioma mimicking meniscal injury. A report on 10 years of knee pain. *Acta Orthop Scand.* 1989;60(2):230–1.
102. Devaney K, Vinh TN, Sweet DE. Synovial hemangioma: a report of 20 cases with differential diagnostic considerations. *Hum Pathol.* 1993;24(7):737–45.
103. Greenspan A, Azouz EM, Matthews 2nd J, Decarie JC. Synovial hemangioma: imaging features in eight histologically proven cases, review of the literature, and differential diagnosis. *Skeletal Radiol.* 1995;24(8):583–90.
104. Reutter G, Klug S, Rompel O. Synoviales Hämangiome als seltene Ursache eines Hämarthros. *Monatsschr Kinderheilkd.* 2001;149(2):150–3.
105. Price NJ, Cundy PJ. Synovial hemangioma of the knee. *J Pediatr Orthop.* 1997;17(1):74–7.
106. Llauger J, Monill JM, Palmer J, Clotet M. Synovial hemangioma of the knee: MRI findings in two cases. *Skeletal Radiol.* 1995;24(8):579–81.
107. Cohen JM, Weinreb JC, Redman HC. Arteriovenous malformations of the extremities: MR imaging. *Radiology.* 1986;158(2):475–9.
108. Levine E, Wetzel LH, Neff JR. MR imaging and CT of extrahepatic cavernous hemangiomas. *AJR Am J Roentgenol.* 1986;147(6):1299–304.
109. Petasnick JP, Turner DA, Charters JR, Gitelis S, Zacharias CE. Soft-tissue masses of the locomotor system: comparison of MR imaging with CT. *Radiology.* 1986;160(1):125–33.
110. De Filippo M, Rovani C, Sudberry JJ, Rossi F, Pogliacomi F, Zompatori M. Magnetic resonance imaging comparison of intra-articular cavernous synovial hemangioma and cystic synovial hyperplasia of the knee. *Acta Radiol.* 2006;47(6):581–4.
111. Okahashi K, Sugimoto K, Iwai M, Tanaka M, Fujisawa Y, Takakura Y. Intra-articular synovial hemangioma; a rare cause of knee pain and swelling. *Arch Orthop Trauma Surg.* 2004;124(8):571–3.
112. Tuncbilek N, Karakas HM, Okten OO. Dynamic contrast enhanced MRI in the differential diagnosis of soft tissue tumors. *Eur J Radiol.* 2005;53(3):500–5.
113. Kroner K, Fruensgaard S. Synovial venous hemangioma of the knee joint. *Arch Orthop Trauma Surg.* 1989;108(4):253–4.
114. Wirth T, Rauch G, Ruschoff J, Griss P. Synovial haemangioma of the knee joint. *Int Orthop.* 1992;16(2):130–2.

115. Aynaci O, Ahmetoglu A, Reis A, Turhan AU. Synovial hemangioma in Hoffa's fat pad (case report). *Knee Surg Sports Traumatol Arthrosc.* 2001;9(6):355–7.
116. Neel MD, Toy PC, Kaste SC, Jenkins JJ, Daw N, Rao BN. Painful limp in a 10-year-old boy. *Clin Orthop Relat Res.* 2003; 410:326–33.
117. Franco M, Puch JM, Carayon MJ, Bortolotti D, Albano L, Lallemand A. Lipoma arborescens of the knee: report of a case managed by arthroscopic synovectomy. *Joint Bone Spine.* 2004;71(1):73–5.
118. Arzimanoglu A. Bilateral lateral arborescent lipoma of the knee—a case report. *J Bone Joint Surg.* 1957;39(4):976–9.
119. Bernstein AD, Jazrawi LM, Rose DJ. Arthroscopic treatment of an intra-articular lipoma of the knee joint. *Arthroscopy.* 2001; 17(5):539–41.
120. Hill JA, Martin 3rd WR, Milgram JW. Unusual arthroscopic knee lesions: case report of an intra-articular lipoma. *J Natl Med Assoc.* 1993;85(9):697–9.
121. Gao J, Gillquist J, Messner K. An unusual case of extraarticular lipoma of the knee joint. *Knee Surg Sports Traumatol Arthrosc.* 1996;4(3):164–6.
122. Margheritini F, Villar RN, Rees D. Intra-articular lipoma of the hip. A case report. *Int Orthop.* 1998;22(5):328–9.
123. Sola JB, Wright RW. Arthroscopic treatment for lipoma arborescens of the knee—a case report. *J Bone Joint Surg.* 1998; 80A(1):99–103.
124. Hallel T, Lew S, Bansal M. Villous lipomatous proliferation of the synovial membrane (lipoma arborescens). *J Bone Joint Surg.* 1988;70A(2):264–70.
125. Blais RE, LaPrade RF, Chaljub G, Adesokan A. The arthroscopic appearance of lipoma arborescens of the knee. *Arthroscopy.* 1995;11(5):623–7.
126. Pudlowski RM, Gilula LA, Kyriakos M. Intraarticular lipoma with osseous metaplasia: radiographic-pathologic correlation. *AJR Am J Roentgenol.* 1979;132(3):471–3.
127. Xue J, Alario AJ, Nelson SD, Wu H. Progressive bilateral lipoma arborescens of the knee complicated by juvenile spondyloarthritis: a case report and review of the literature. *Semin Arthritis Rheum.* 2013;43(2):259–63.
128. Siva C, Brasington R, Totty W, Sotelo A, Atkinson J. Synovial lipomatosis (Lipoma arborescens) affecting multiple joints in a patient with congenital short bowel syndrome. *J Rheumatol.* 2002;29(5):1088–92.
129. Davies AP, Blewitt N. Lipoma arborescens of the knee. *Knee.* 2005;12(5):394–6.
130. Armstrong SJ, Watt I. Lipoma arborescens of the knee. *Br J Radiol.* 1989;62(734):178–80.
131. Chaljub G, Johnson PR. In vivo MRI characteristics of lipoma arborescens utilizing fat suppression and contrast administration. *J Comput Assist Tomogr.* 1996;20(1):85–7.
132. Hubscher O, Costanza E, Elsner B. Chronic monoarthritis due to lipoma arborescens. *J Rheumatol.* 1990;17(6):861–2.
133. Soler R, Rodriguez E, Bargiela A, Da Riba M. Lipoma arborescens of the knee: MR characteristics in 13 joints. *J Comput Assist Tomogr.* 1998;22(4):605–9.
134. Erol B, Ozyurek S, Guler F, Kose O. Lipoma arborescens of the knee joint. *BMJ Case Rep.* 2013;2013. pii:bcr2013009271.
135. Grieten M, Buckwalter KA, Cardinal E, Rougraff B. Case report 873. *Skeletal Radiol.* 1994;23(8):652–5.
136. Kloen P, Keel SB, Chandler HP, Geiger RH, Zarins B, Rosenberg AE. Lipoma arborescens of the knee. *J Bone Joint Surg Br.* 1998;80(2):298–301.
137. Hirano K, Deguchi M, Kanamono T. Intra-articular synovial lipoma of the knee joint (located in the lateral recess): a case report and review of the literature. *Knee.* 2007;14(1):63–7.
138. Martinez D, Millner PA, Coral A, Newman RJ, Hardy GJ, Butt WP. Case report 745—synovial lipoma arborescens. *Skeletal Radiol.* 1992;21(6):393–5.
139. Ryu KN, Jaovisidha S, Schweitzer M, Motta AO, Resnick D. MR imaging of lipoma arborescens of the knee joint. *AJR Am J Roentgenol.* 1996;167(5):1229–32.
140. Kim RS, Song JS, Park SW, Kim L, Park SR, Jung JH, et al. Lipoma arborescens of the knee. *Arthroscopy.* 2004;20(8): e95–9.
141. Vilanova JC, Barcelo J, Villalon M, Aldoma J, Delgado E, Zapater I. MR imaging of lipoma arborescens and the associated lesions. *Skeletal Radiol.* 2003;32(9):504–9.
142. Yan CH, Wong JW, Yip DK. Bilateral knee lipoma arborescens: a case report. *J Orthop Surg (Hong Kong).* 2008;16(1):107–10.
143. Ji JH, Lee YS, Shafi M. Spontaneous recurrent hemarthrosis of the knee joint in elderly patients with osteoarthritis: an infrequent presentation of synovial lipoma arborescens. *Knee Surg Sports Traumatol Arthrosc.* 2010;18(10):1352–5.
144. Coventry MB, Harrison Jr EG, Martin JF. Benign synovial tumors of the knee: a diagnostic problem. *J Bone Joint Surg Am.* 1966; 48(7):1350–8.