Management of Synovial Disorders

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Contents

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Abstract

Synovium lines the capsule of joints, tendon sheathes and bursae. It is seen to differ from the outer thick layer of the capsule both macro- and microscopically. This capsular layer contains a rich network of blood vessels, lymphatic vessels and nerves, which penetrate the inner synovial membrane. There are numerous capillaries on the surface of the synovial membrane and delicate capillary loops extend into the margins of the articular cartilage at the site of the insertion of the capsule. These are part of the vascular border of the joint which was termed by Hunter the "circulus articuli vasculosus" (Jaffe HL. Metabolic degenerative and inflammatory diseases of bone and joints. Philadelphia: Lea and Febiger. 1972. p. 92). Synovium also covers intra-articular ligaments and tendons and intracapsular areas of bone which are not covered by articular cartilage. The synovial membrane is involved in all diseases of joints and tendons. Villi are projections of the surface of the synovial membrane which consist of fibrils of collagen with lining cells. They can be identified microscopically and vary in size, shape, and composition according to the underlying pathological conditions which are considered here.

Keywords

Arthroscopic • Disorders • Foreign body synovitis • Haemophilic Synovitis • Pigmented villonodular synovits • Plica syndrome • Post-traumatic synovitits • Rheumatiod arthritis synovitis • Synovectomy • Synovial • Synovial chondromatosis • Synovial haemangioma • Synovial lipoma • Total joint replacement • Treatment-conservative • Tuberculous synovitis

Pigmented Villonodular Synovitis (PVNS)

This is a rare disease of joints. The knee is most commonly affected (80 %), followed by the hip (15 %). The shoulder, ankle, elbow, and other synovial joints are also sometimes involved [1]. It is a monoarticular joint disease and synovial bursae and tendon sheaths are also affected. Men and women are equally affected. The patients are usually adolescent with involvement of the lower limbs. Bony involvement is rare and usually affects the hip. Generally, in all types the fingers (tendons and sheaths) are affected in 60 % of the patients and the knee in 30 % [1].

The disease was probably first described by Chassaignac who, in 1852, reported the nodular form affecting the index and middle fingers [2]. It was considered to be a form of synovial sarcoma. Dowd in 1912 considered it to be a benign form (villous arthritis) [3]. There has been some confusion with terminology and several names have been ascribed to the condition such as xanthoma, myeloxanthoma, giant-cell tumour, villous arthritis and benign synovioma. Two patients in his series had recurrence and repeated operations were necessary, which led to amputation. These two patients were still alive 6 years after operation. It is well known that synovial sarcoma has a poor prognosis if diagnosed late. Lichtenstein doubted that these two patients had a synovial sarcoma. As a result of his histological examination he stated that no case of PVNS developed malignant change. Several theories have been proposed regarding the aetiology of PVNS. In 1941 Jaffe, Lichtenstein and Sutro [4] termed it pigmented villonodular synovitis implying a benign inflammatory aetiology. Some authors considered the aetiology to be repeated microtrauma. Some of the changes in PVNS are similar to the synovial changes which are seen in haemophilia. Another theory has suggested that it is caused by changes in the concentration of lipids in the blood. However, it is not reproduced by injecting lipid into joints. The aetiology therefore remains unknown although many authors have reported it as a benign neoplastic disorder which often occurs in conjunction with abnormalities of chromosome 1 p11-13 [5, 6]. More so there are immunophenotypic differences in giant cells between PVNS and Haemosiderotic Synovitis and the expression of CD51 in PVNS giant cells only as well as the higher ki-67 index

in PVS, effectively distinguishes these two conditions [7]. Immunophenotype, also, differs comparing to other inflammatory diseases. Compared to Rheumatoid Arthritis with which PVNS has histologically homogeneous appearance, proliferating synovial cells display heterogeneous immunophenotype in both RA and PVNS indicating functional properties of both macrophages and fibroblasts. Aneuploidy seems to be a special feature of diffuse PVNS [8]. Furthermore malignant transformation and metastasis appear in some reports initially diagnosed as PVNS [9–11].

Several theories have been proposed to explain the formation of cysts in PVNS. These are rare but it is postulated that in joints with limited possibility of expansion such as the hip, the synovium extrudes at the junction between the articular cartilage and bone, or through pressure within the bone where atrophy allows cysts to form within the Haversian systems. In several studies giant cells in PVNS have been shown to express all the phenotypic features of osteoclasts including the ability to induce lacunar resorption which may account for the bony lesions seen in this condition [12, 13].

In conclusion the aetiology remains obscure. It seems as though that it is of inflammatory origin with a destructive course and high rate of recurrence in the diffuse form and possibility to metastasize very rarely to another joint or to the lungs [12].

Incidence

After its onset PVNS progresses slowly. Flandry et al [14] have estimated an incidence of between 1 and 3 per million of population. Dorfman and Czerniak [2] consider it to represent 5 % of benign soft-tissue tumours. Biopsy of tissue from 1388 total hip and knee replacements revealed one case of PVNS and 12 of malignancy [15].

Classification

At operation the lesion presents as villous or nodular tissue which is yellow brown because of the deposition of haemosiderin. It can be classified as PVN synovitis, PVN bursitis or PVN tenosynovitis according to the site, and all may be nodular (localized or diffuse). The first is more common in joints, and the last in tendon sheaths.

Histology

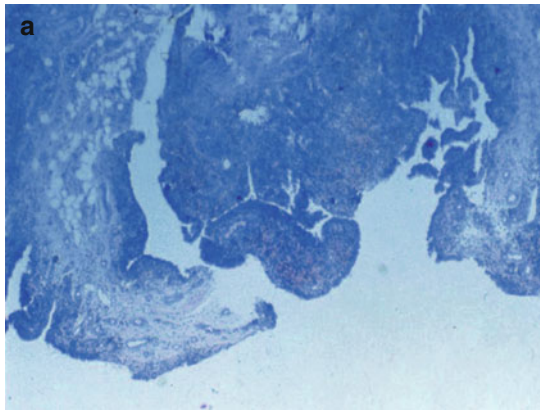
Histological examination shows villous hypertrophy of the synovial membrane in both types with active proliferation of the synovial cells and variable fibrosis. There are stromal cells among which can be found multinuclear giant cells and cells containing lipids. Deposits of haemosiderin are prominent and may be either extracellular or within histiocytes. The synovial membrane is fairly vascular (Fig. 1a, b). When there is bony involvement, similar tissue can be found nearby, forming cysts (Fig. 2).

Differential Diagnosis

Cases have been reported which were misdiagnosed as malignant synovioma and treated by amputation [16]. The presence of multinuclear giant cells, cells containing haemosiderin and the absence of spindle cells in active proliferation, distinguish the disease from malignant conditions. The clinical, microscopic and histological appearances also distinguish the condition from other active inflammatory disorders such as rheumatoid arthritis, traumatic haemarthrosis and haemophilia.

Symptoms

These include persistent pain which gradually increases, possible locking of the joint in the nodular form and stiffness. When the shoulder is involved, there may be extension of the synovium into the subdeltoid bursa. In the knee and ankle, haemarthrosis and swelling are the usual characteristic findings. In the fingers and toes there is irregular swelling extending outside of the tendon sheath.



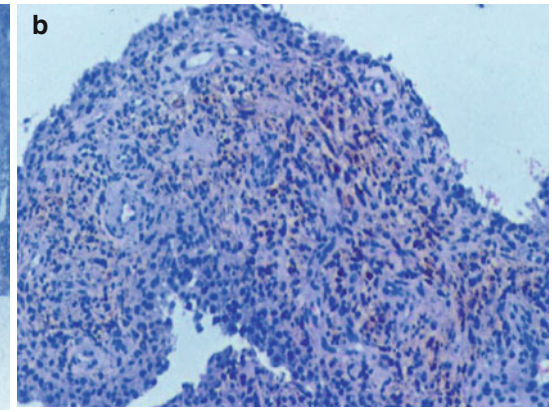


Fig. 1 (**a**, **b**) Villous formation of the synovial membrane can be seen on histology with active proliferation of the synovial cells and variable fibrosis. On higher magnification multi-nuclear giant cells and lipid-bearing cells can be seen. Haemosiderin can be seen extra- or intra-cellular, while the synovium looks vascular

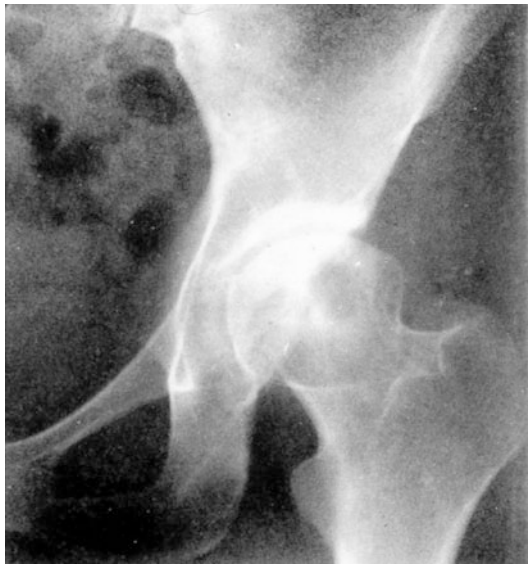


Fig. 2 Bone cysts in PVNS can be seen away from the articular surface

Radiological Findings

If there is no bony involvement there will be no radiological abnormalities. In advanced cases there may be cysts at some distance from the articular surface; they may be well-defined and the joint space relatively preserved (Fig. 2). In more advanced cases there may be secondary degenerative changes. In cases affecting the fingers, pressure indentation of bone may be seen.

Echo and MRI Findings

Both techniques may delineate the lesion. This is especially so with MRI in which multiple synovial lesions with low or intermediate signal intensity on T1-weighted images or low signal intensity on T2-weighted and gradient echo images can be seen (Fig. 3). The use of contrast medium can enhance the lesions, being non-diagnostic.

Treatment

Surgical treatment may be open or arthroscopic. Open treatment applies to the diffuse type of the disease affecting the joint (more so if there are exra-articular masses), synovial bursae and tendon sheaths.

Since the knee is the major joint affected, the surgical approach to the knee will be described.

The skin is prepared and the tourniquet is inflated. The joint is approached through a straight midline incision to the skin and a medial parapatellar incision. The capsule is separated from the synovium (Fig. 4) and the hypertrophied synovium can be seen protruding on incising the synovial membrane (Fig. 5). Subsequently the synovial suprapatellar pouch is resected *en bloc* (Fig. 6) with the remaining synovium to the margins of the articular cartilage

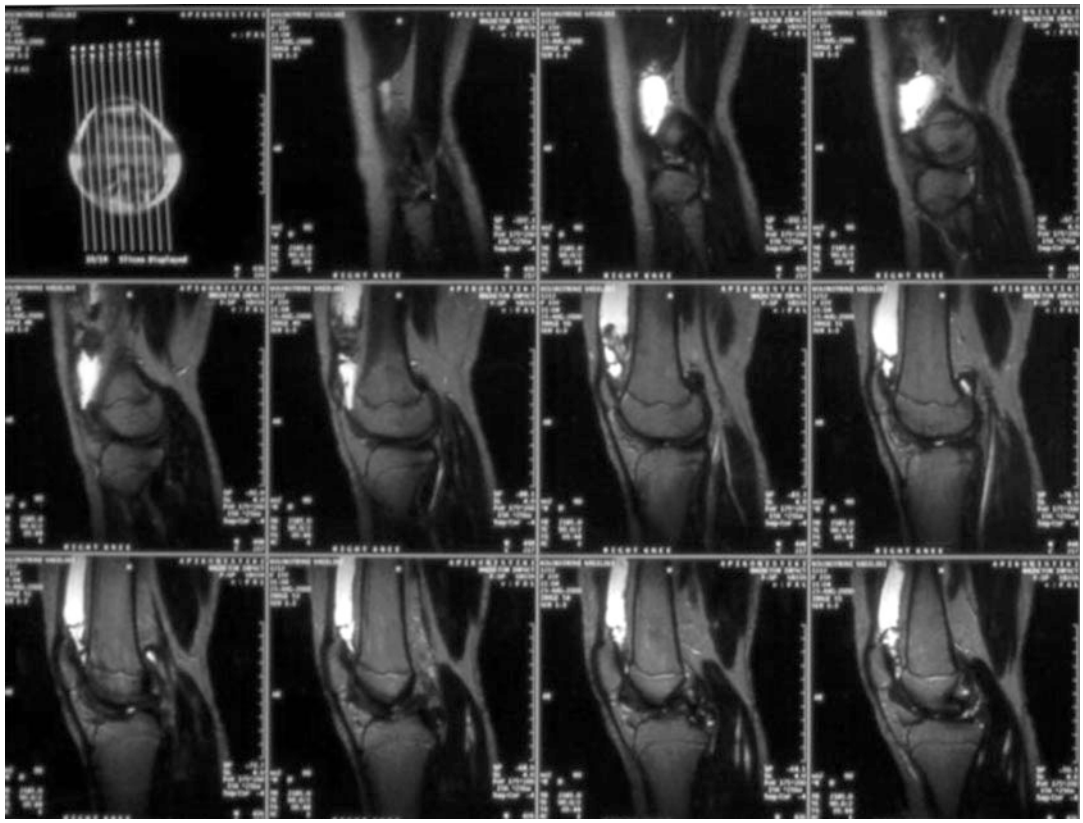


Fig. 3 MRI scans showing hypertrophied lobular synovium

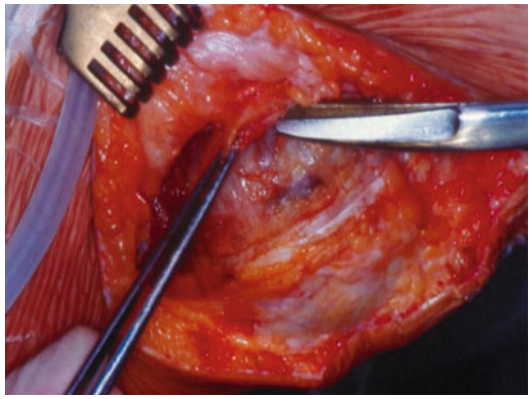


Fig. 4 Synovium can be clearly dissected from the outer fibrous or other structures

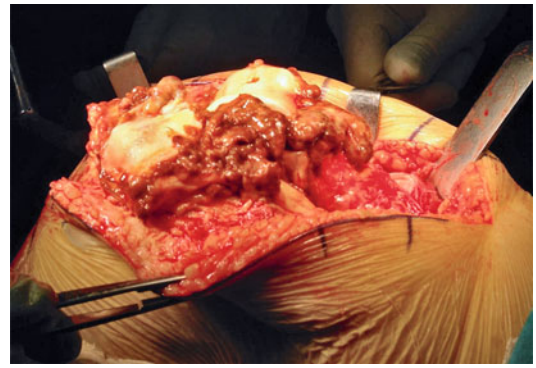


Fig. 5 Hypertrophied synovium can be seen protruding on incising the synovial membrane with villus formation

which is meticulously excised (Figs. 7, 8a, b). The synovial remnants which invade the cruciate ligaments are also removed. Then the menisci are detached from the periphery and the possibly underlying remnants of the synovium are curetted. The detached menisci are repaired with non-absorbable sutures. If there are any bony cysts, these are evacuated by curettage and

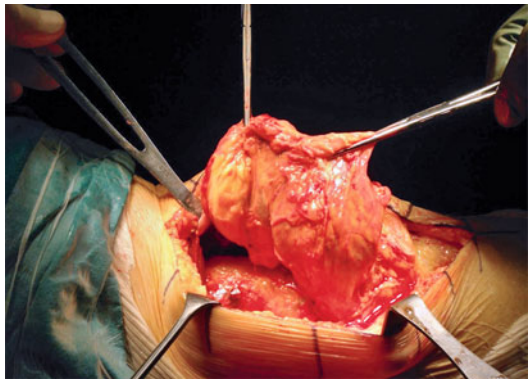


Fig. 6 Most of the synovial membrane including the supracondylar pouch can be excised en-bloc

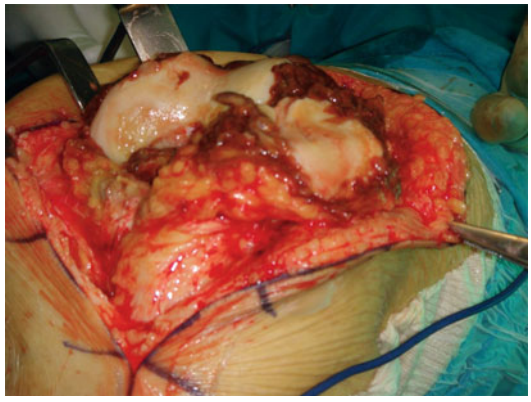


Fig. 7 The remnants of the excised synovium can be meticulously be removed up to the borders of the articular cartilage

filled with bone graft. After suturing and vacuum suction placement, if there are lesions in the back, either extra- or intra-articular, the patient is turned over. The tourniquet is reinflated and the posterior lesions are approached through an S incision. The extra-articular lesions are excised and the posterior joint is approached after dissecting the peroneal nerve and detaching the two heads of the gastrocnemius protecting the neurovascular structures. The capsule is incised with medial and lateral incisions and the inside tissue is excised. After suturing and vacuum suction placement the leg is immobilized in a Robert Jones bandage and continuous passive movement and physiotherapy are started from the first postoperative day with isometric exercises followed by active assisted exercises and gradual weight-bearing. Postop pain is controlled in the first 2 days by epidural medication or PCA.

Arthroscopic treatment of the knee is indicated for the nodular (localized) form and the node or nodes are removed through medial or lateral portals depending on their site. This treatment gives equally good results as the open procedure and better rehabilitation. Postoperative rehabilitation is simple, as for other minor arthroscopic procedures.

Arthroscopic synovectomy for the diffuse form of PVNS has a high incidence of recurrence, 14 % in 42 months [17]. Recurrence-free survival is 95 % for open synovectomy and 62 % for arthroscopic synovectomy at 2 years and at 5 years

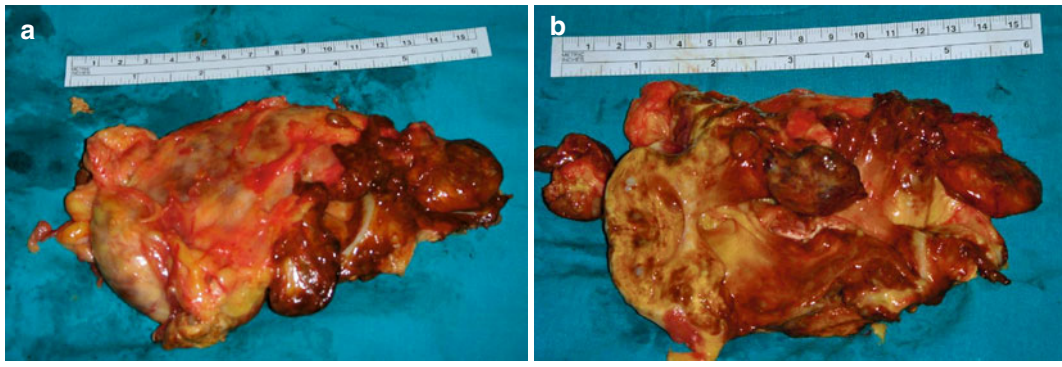


Fig. 8 (**a**) The main part of the excised synovium with the supracondylar pouch. (**b**) The inside of the pouch with the nodular appearance

73 % for open and 48 % for arthroscopic [18]. Arthroscopic total synovectomy gives equally good results as open total synovectomy, according to some authors, [19] but it is a technically demanding procedure and requires posterior portals and experience with 70° scope. The same authors reported five clinical recurrences in nine patients in 1.8 years [20]. On the other hand, De Ponti et al presented better results for the defuse PVNS with extended arthroscopic synovectomy and the recurrence rate was lower in comparison to the partial synovectomy group [20].

In conclusion open total surgical synovectomy, as described, remains the most reliable and consistent method of treating all anatomic variations of diffuse PVNS, especially in the extra-articular form [12, 21]. Safe comparison between the different studies is difficult because many authors do not use MRI for the diagnosis of recurrences [12].

Total knee replacement (TKR) is indicated in more advanced cases in conjunction with synovectomy when secondary O.A. changes have been established. The results are encouraging. In 18 patients followed for a mean of 10, 3 years after TKR and synovectomy there was one case of recurrence and three of aseptic loosening without recurrence [22]. Arthrodesis with synovectomy was the treatment of choice for advanced case previously before TKR became an established successful procedure.

As mentioned before, hip joint is affected much less by diffuse PVNS. Synovectomy, as it is generally accepted, is indicated in patients with preserved articular cartilage. Arthrotomy with subsequent dislocation of the hip is necessary to complete maximal synovectomy. Vastel et al presented 16 patients, mean age 35 and 16 years follow-up. All had synovectomy and in addition 3 cup arthroplasty, 4 total hip replacement and 1 monopolar replacement. Nine patients needed repeat surgery, but only one had recurrent synovitis 14 years after treatment with synovectomy and cup arthroplasty. Secondary osteoarthritis developed in all 8 patients who had been treated with synovectomy alone and 4 of them required total hip arthroplasty within the follow-up period. They concluded that synovectomy prevents recurrence of the synovitis, but does not prevent the development of secondary osteoarthritis [23].

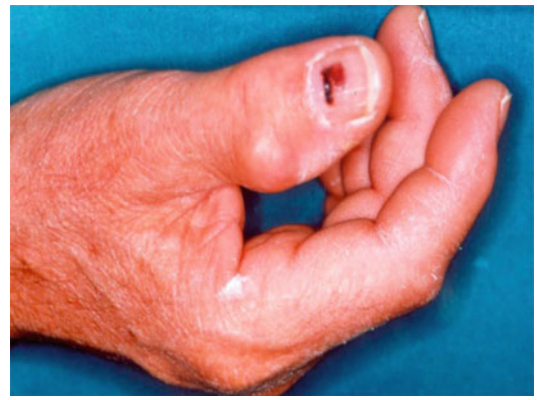


Fig. 9 Defuse PVNS of the extensor tendons of the thumb, totally excised

Total hip replacement in diffuse PVNS is indicated when there are advanced O.A. changes. Gonzalez Della Vale et al reviewed 117 cases from the literature and presented 7 new cases. Among the new cases, 4 underwent synovectomy and primary total hip replacement with no recurrences detected after an average follow-up of 13 years. One patient, who underwent synovectomy, had a recurrence 9 years later, requiring a total hip replacement. Regarding the reviewed cases of PVNS of the hip, 53 % did not have enough information for analysis. Of the remaining cases 10 had recurrence, 1 in the arthroplasty group (24 patients) and 9 in the synovectomy group (26 patients) [24]. Recently Yoo et al reviewed 8 patients for 8.9 years following cementless total hip arthroplasty (THA) combined with synovectomy. None of the patients had clinical or radiographic evidence of PVS. Osteolysis occurred in 4 hips and two revision surgeries were performed [25].

Open surgical treatment is the only treatment for PV bursitis and tenosynovitis. In the former, excision of the affected bursa and synovectomy are the recommended procedures. In the latter, total resection of the hypertrophic synovium is indicated (Figs. 9–11). Recurrence is the main complication. In the diffuse form a rate of recurrence of between 30 % and 46 % has been

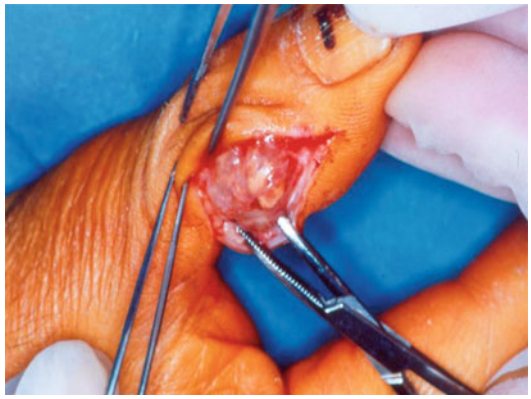


Fig. 10 Defuse PVNS of the extensor tendons of the thumb, totally excised

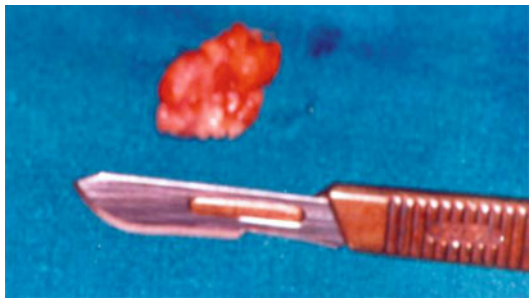


Fig. 11 Defuse PVNS of the extensor tendons of the thumb, totally excised

reported, and in the nodular form of between 27 % and 48 %.

Radiation therapy in PVNS has been used as adjuvant external beam therapy with no significant advantage over surgical synovectomy alone. Arthroscopic synovectomy with adjuvant low dose radiotherapy showed recurrence rate 14 % same as open synovectomy [26]. Possible complications are reported; joint stiffness, skin reactions, poor wound healing and possibly sarcomatous transformation. The results are variable [27–29].

Synovial Chondromatosis

This is a metaplastic disorder involving the synovial membrane of a joint, the tendon sheath or the bursa, producing nodules of cartilage which are

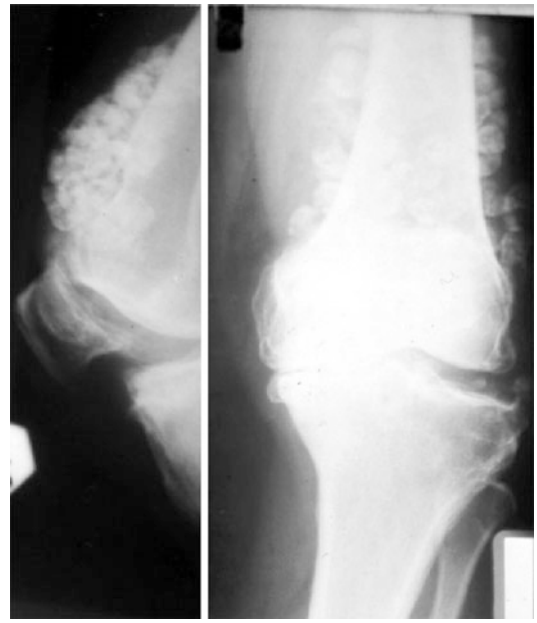


Fig. 12 X-Rays of the knee of a 60 years-old woman with Synovial Chondromatosis

gradually detached from the synovium becoming loose. Although rare it presents between the ages of 20 and 50 years, usually occurring in men [30]. Approximately 70 % of cases involve the knee. Other areas such as the hip, shoulder, elbow, and the temporomandibular joint may be affected. In contrast to PVNS, in 10 % of cases it may be bilateral [2].

Symptoms

Pain and swelling are the main presenting features with occasional locking. They are usually mild, insidious and chronic until there is limitation of movement of the affected joint.

Radiological Findings

There may be multiple loose bodies, which if mineralized, can be seen on plain radiographs particularly in longstanding cases (Fig. 12). In 10 % of the cases loose bodies cannot be identified on plain radiographs [2]. MRI is of value in early cases.

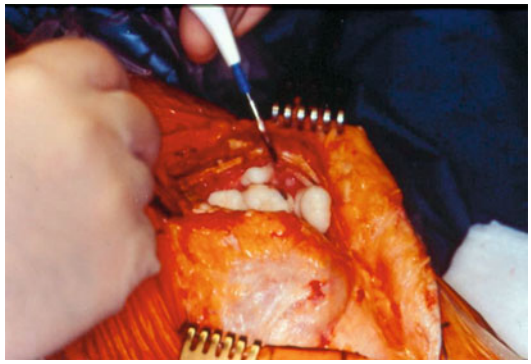


Fig. 13 Same patient as Fig. 12. Multiple loose bodies popping out of the joint on incising the synovium

Fig. 14 Hypertrophyed synovium with no pigmentation can be seen

Operative Findings

These depend on the stage of the disease. In late cases on opening the joint, multiple loose bodies are seen (Fig. 13). The synovium resembles that of PVS without pigmentation or villi (Fig. 14). The loose bodies may be of many different sizes (Fig. 15).

Arthroscopic Findings

In early stages, loose bodies may be identified and can be excised (Fig. 16). No macroscopic synovial changes are seen in the early cases.

Histology

The synovium in fully developed cases contains multiple nodules of hyaline cartilage of myxoid

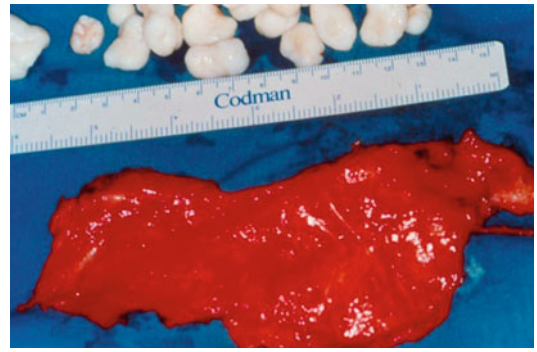


Fig. 15 The excised synovium and loose bodies

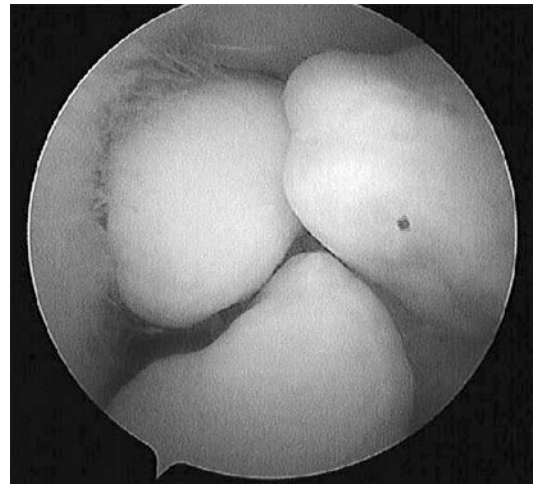


Fig. 16 Arthroscopic picture in an early case of synovial chondromatosis

features (Fig. 17a). The cellularity of cartilage nodules is often increased. The loose bodies resemble hyaline cartilage (Fig. 17b).

Treatment

Open surgical treatment is indicated in longstanding cases in which there are multiple chondromatous loose bodies and the synovium appears to be proliferative. All loose bodies are removed and a synovectomy is carried out as previously described. After operation, the same regime is followed as for PVS. If there are secondary symptomatic degenerative changes, TKR may be required.

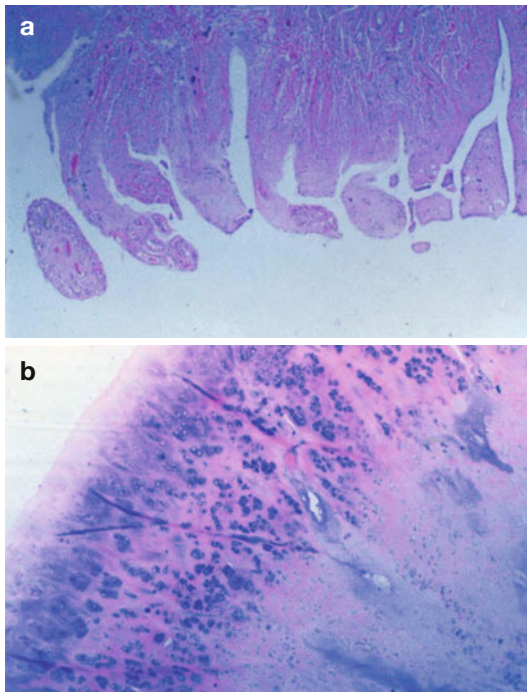


Fig. 17 (**a**) The synovium in synovial chondromatosis looks nodular on histology before the top ends become loose bodies. (**b**) Histologic picture of a loose body at the same patient with hyaline cartilage formation

Arthroscopic treatment is recommended in early cases in which there are a few loose bodies without proliferative synovial changes requiring synovectomy. Synovial chondromata are those cases in which there is a single cartilaginous nodule within the synovial membrane. There is controversy as to whether it exists or reflects synovial metaplasia.

Synovial chondromatosis of the hip appears in the literature in a few reports regarding the outcomes. Schoeniger R. et al reviewed 8 patients who had joint debridement and total synovectomy performed through open surgery dislocating the hip and flip osteotomy of the greater trochanter. The mean follow-up was 6.5 years. No patient had recurrence of the disease at follow-up. Finally at 5 and 10 years, 2 patients had developed O.A. requiring total hip arthroplasty. Even these 2 patients did not show recurrence of the disease on histologic examination of the synovial membrane. They concluded that synovectomy prevents recurrence of the disease with no morbidity [31].

Arthroscopic removal of loose bodies has higher recurrence rate than if it were combined with arthroscopic synovectomy ($p = 0.02$) [32]. In 5 cases of shoulder arthroscopy, removal of loose bodies and partial synovectomy, performed due to synovial chondromatosis, clinical results were very good, whereas radiological signs of chondromata were observed in 2 patients [33].

If secondary O.A. develops in conjunction with synovial chondromatosis total knee or total hip arthroplasty is required. Ackerman D. et al from the Mayo Clinic reviewed 11 patients treated with total knee or total hip arthroplasty with mean follow-up time after surgery of 10.8 years. Pain and functional scores improved in all patients. There was only one recurrence of the disease for the knee and one for the hip group, 25 % and 14 % respectively [34].

Plica Syndrome

Plicae in the knee are some of the normal synovial structures. They are remnants of the mesenchymal tissue that occupy the space between the distal femoral and proximal tibial epiphyses in the 8 weeks-old embryo. Under some circumstances the incomplete resorption leaves synovial pleats in most of the knee. Plicae in the knee are classified based on their anatomical location; the infrapatellar plica or ligamentum mucosum, the suprapatellar plica and the medial patellar plica or medial shelf. Lateral plicae exist but rarely [35]. Dandy described several variations of the most common medial plicae [36].

Plicae become pathological when thickening and fibrosis occurs with subsequent inelasticity that can lead in snapping over the femoral condyle causing synovitis, chondral damage and pain [37].

Pain is the most common symptom. Swelling, pseudo-locking, and a feeling of snapping are common symptoms as well. Arthroscopy is of value in diagnosis and the reason for the symptoms may be attributed to a plica in the absence of other pathology such as meniscal lesions, loose bodies etc.

Post-traumatic synovitis after injury to a plica may cause symptoms and a plica may also cause recurrent haemarthrosis [38].

The treatment of synovial plicae is conservative in the first instance with administration of non-steroid anti-inflammatory drugs, isometric quadriceps and hamstring exercises. If there is no symptomatic improvement arthroscopic removal of the plica after inspection of the entire joint is the treatment of choice [39]. Postoperatively, an intense programme of physiotherapy is required. The syndrome may recur and further arthroscopy may be needed if a new plica forms, which must be removed. The routine medial and lateral arthroscopic portals are used.

Post-Traumatic Synovitis

This refers to the reaction of the synovial membrane after trauma. It can be at the site of injury to the synovium or secondary to an associated injury to ligament or bone. Immediately after the injury local haemorrhage or rupture of the synovial membrane may be distinguished arthroscopically. Haematoma at the insertion of a ligament gives the suspicion or rupture. If trauma to the synovium is longstanding the reaction is generalized. The knee is the most commonly affected joint.

Immediately after injury parts of the traumatized synovium may protrude inside the joint and if devascularized may become loose and cause locking. Subsynovial haematoma, hydrarthrosis and haemarthrosis may resolve with the passage of time whereas deposits of haemosiderin may continue for many months and be mistaken for PVS. They differ, however, in that the changes in post-traumatic synovitis are superficial. In case of recurrent haemarthrosis a thick plica-like appearance of the synovium can be seen either arthroscopically or histologically. Chronic synovial tears may become fibrotic and lead to arthrofibrosis and stiffness.

The indication for surgery is severe mechanical type of pain after injury non-responding to 3 months of conservative treatment [40]. In primary synovial injuries arthroscopy is of value for removal of pieces of the injured synovium and for washing-out the joint to avoid synovial inflammation. Arthroscopy is decided after MRI exclusion of other internal disorders of the joint. The sensitivity, specificity and accuracy of MR imaging compared to arthroscopy is 88 %, 95 % and 95 % respectively [41]. In late cases arthroscopy can be difficult because of lack of space for expansion of the joint, and several portals may be needed.

Haemophilic Synovitis

Haemophilia is a sex-linked inherited disorder, transmitted as a recessive Mendelian trait. It is expressed by males and transmitted by females, who are not affected. One of the main presenting symptoms is repeated haemarthroses due to lack of a clotting factor. Successive haemorrhages cause proliferation of the synovial membrane, reactive inflammation and eventually destructive changes in the joint.

The initial symptoms are pain, heamarthrosis and impaired function. The knee is the most commonly involved joint. Repeated haemarthroses cause stiffness due to initially reactive synovitis and subsequently to arthrofibrosis. Contractures of the knee or other affected joints usually appear as the condition progresses and the development of secondary degenerative changes is inevitable.

Initially, radiographs are normal. Gradually, due to disuse, bone atrophy becomes evident and later there are secondary degenerative changes.

MRI may show hypertrophy of the synovium as in PVNS. Histologically the synovial villi are plump and matted together. The cells of the synovial lining contain haemosiderin and are mostly macrophages [42].

Open synovectomy is the established surgical procedure after initial conservative treatment with the administration of the missing clotting factor, immobilisation and physiotherapy. Radioactive synovectomy is indicated in patients who have inhibitors to the clotting factor, immune deficiency or advance hepatitis [43, 44].

Arthroscopic synovectomy offers many advantages, since the disease is not proliferative, and is preferable to an open procedure in order to avoid postoperative haemorrhage and fibrosis. Synovectomy, by either method, does

not arrest the development of degenerative changes [45].

With the availability of activated recombinant factor VIII the possibility of total joint arthroplasty was expanded in haemophilic patients with inhibitors [46]. Total hip and knee replacement in post-haemophilic degenerative arthritis have encouraging results [47, 48]. Latest techniques of continuous infusion of clotting factor have significantly helped to reduce the complication rates and have achieved results which match to those of the non haemophilic population undergoing arthroplasties [49].

Synovial Lipoma

A lipoma very rarely rises from the synovial membrane [2, 50]. Clinically it can cause locking, swelling, mass effect and synovitis. In rare cases, concerning the knee, it can displace the patella due to the mass-effect [51]. A lipoma also may arise from tendon sheaths of the hand. When found in the knee it should not be mistaken for hyperplasia of Hoffa's fat pad. Histologically, it consists of adipose tissue and is identical to those found elsewhere. Arthroscopic excision is recommended.

Synovial Haemangioma

This is another rare lesion affecting the knee, elbow and tendon sheaths. It may be either localised or diffuse. The symptoms in the localised form consist of pain, swelling and occasionally locking. In the diffuse form it can cause a haemarthroses and even destruction of the joint [52]. Histologically, it may be capillary or mixed type (capillary and cavernous haemangioma) [2]. According to Campanacci [31] the diffuse form may be confused with PVNS if there are repeated haemarthroses. The two conditions can be distinguished histologically. MRI is recommended. Arthroscopic treatment should be used for the localised form whereas open synovectomy is needed for the diffuse type [53, 54].

Synovial Sarcoma

Synovial sarcoma is a tumour the cells of which resemble those of normal synovium. It accounts for 5–10% of soft-tissue tumours. It is usually located in par-articular or extra-articular tissues; 5–10% are intra-articular. The knee is the most commonly-affected joint [31].

The tumour grows slowly accompanied by pain and tenderness. On palpation a deeply located round or lobulated mass can be identified or in rare cases it may present as chronic synovitis or an internal derangement.

Radiography shows calcification or ossification within the tumour in 25% of cases. On CT or MRI, the tumour appears inhomogeneous. T2-weighted images show intermediate and high- intensity signals. Histologically the tumour consists of epithelial-like and fibroblast-like cells [31].

Arthroscopy has a limited indication and only in rare cases in which the tumour is intra-articular. It is used only for diagnostic biopsy. Wide excision gives results similar to those of amputation with a survival at 5 years of 25–60 % depending on the stage of the tumour [55].

Foreign-Body Synovitis

This condition is identified as a result of wear mainly in joint replacement. The synovium reacts to wear particles by an inflammatory mechanism with central necrotic hypertrophic synovium infiltrated by various cells. Eventually, the implant becomes loose and revision is necessary (Figs. 18–20).

Tuberculous Synovitis

The condition was common before the pasteurisation of milk. Nowadays, it is seen in old cases of pulmonary tuberculosis which has been either untreated or incompletely managed or in immuno-suppressed patients. Synovial joints, tendon sheaths or bursae can be affected as a manifestation of the tertiary stage of the disease or in post-primary re-infection.

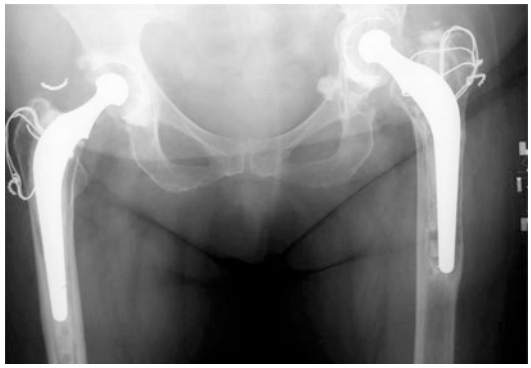


Fig. 18 Loose Charnley total hip replacement in 82 years-old patient on the left side, after 18 years

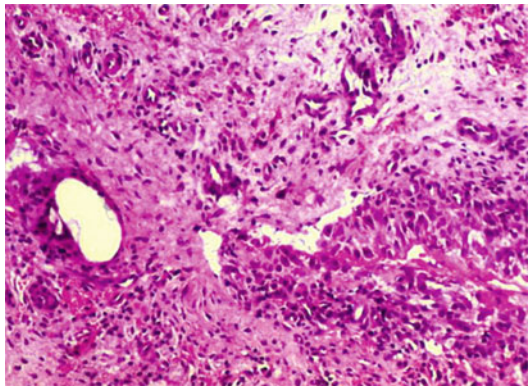


Fig. 19 Foreign particle inflammatory reaction with infiltration by macrophages and histiocytes around the plastic particle (same patient)

The condition can affect the spine in 25–40 % of cases, the hip in 25 % and the knee in 20 % [42].

The presenting symptoms are pain which is usually mild, effusion, synovial or peri-articular thickening, local mild heat but not redness (as in non-specific infections), limitation of movement, muscle spasm and eventually atrophy of the muscles and contractures with inability to walk if the hip, knee or ankle are affected. In untreated cases cold abscesses with chronic sinuses may develop.

The radiological findings in the early stages are demineralisation of the joint resembling transient osteoporosis, bony erosions in late cases and subluxation if a synovial joint is affected (Fig. 21a, b).

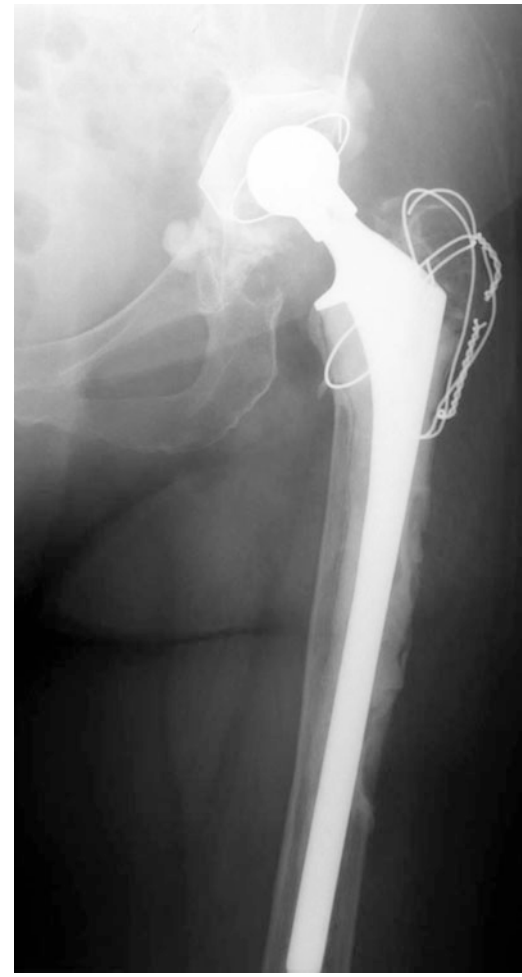


Fig. 20 After revision surgery (same patient)

MRI, echo studies and CT may show synovial hypertrophy and bony erosions or atrophy. A bone scan may show increased uptake. The ESR and the level of C-reactive protein are increased. The tuberculin test is of value although if negative it does not exclude tuberculosis. PCR examination of the joint fluid can confirm the diagnosis. Nowadays, guinea-pig inoculation is not used because of the delay in obtaining the result. Acid-fast bacilli may not always be observed under microscopy.

Arthroscopy is of value at the onset of the disease when it has not eroded the bone and the diagnosis has not been established. Biopsy will indicate villous proliferation in the synovial membrane and infiltration by epithelial-like cells, Langhans-type giant cells and lymphocytes (Fig. 22).

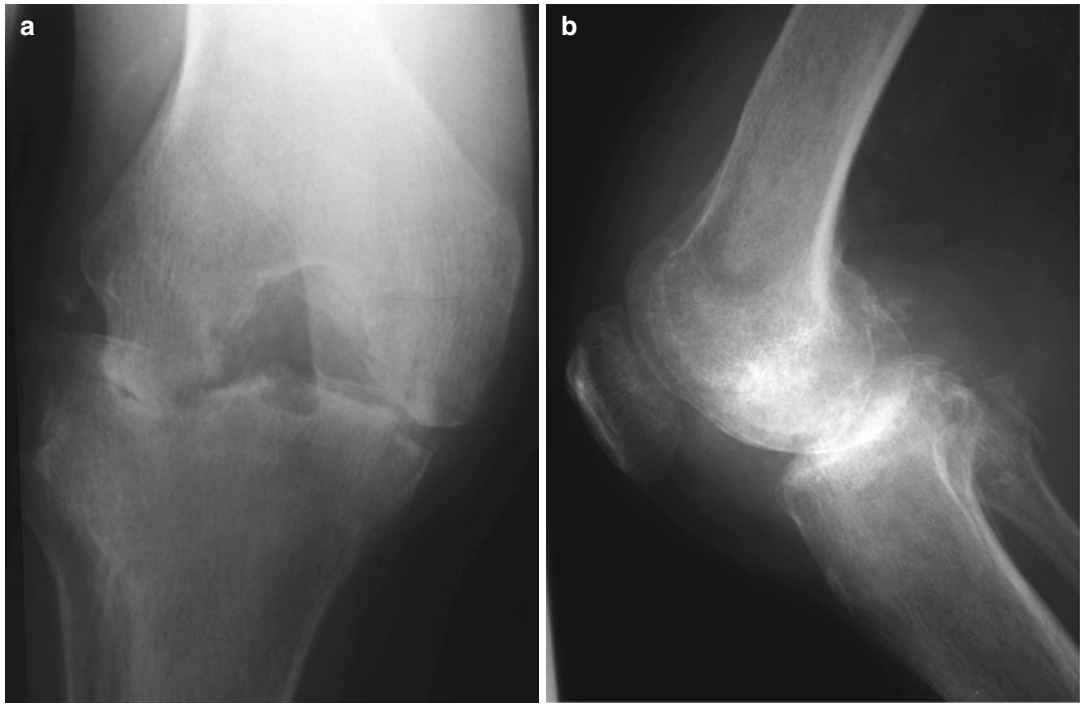


Fig. 21 (**a**, **b**) The X-rays of a neglected case of tuberculosis of the right knee with bone erosions and subluxation

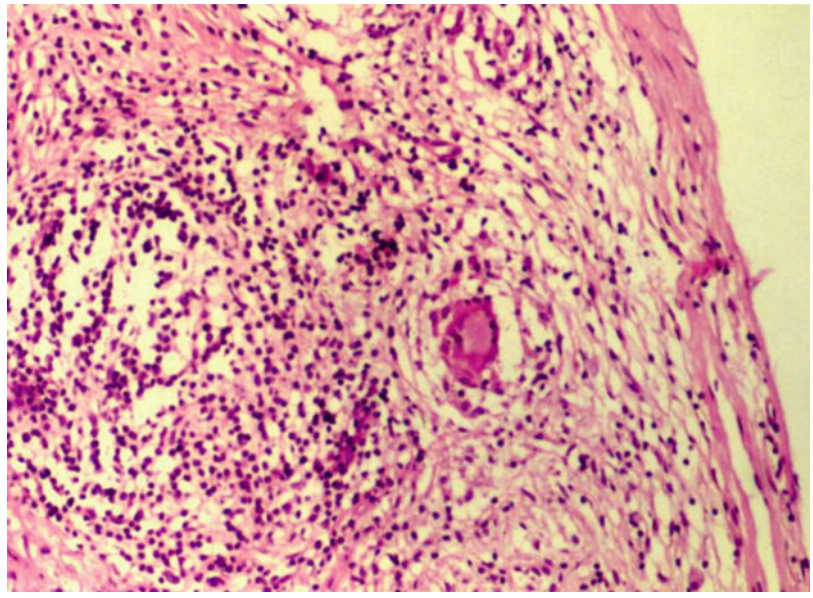


Fig. 22 Microscopic view of the previous patient, showing Langhans-type cells with infiltration of the synovium by epithelial like cells and lymphocytes

Open synovectomy may be performed in the early stages in order to prevent the progress of the disease locally. If this happens, the patient can undergo joint replacement at a later stage and age. This is combined with anti-TB drugs for the appropriate time (Fig. 23).

In late cases open surgery is the only treatment for removal of all infected synovium, bursae or

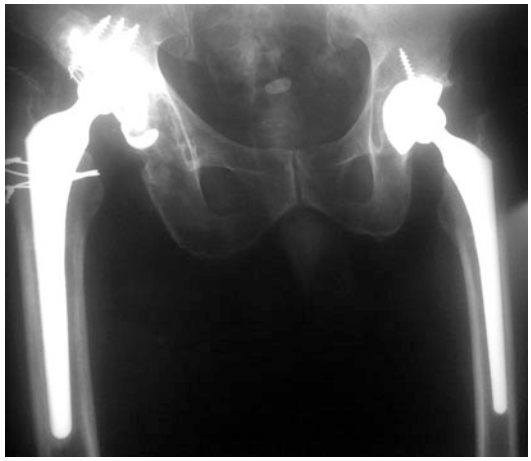


Fig. 23 Fifty years-old patient with bilateral avascular necrosis of both hips following bone marrow transplantation for acute lymphocytic leukemia. The right hip was complicated by tuberculosis. Reconstruction of the right hip was undertaken in a two-stage procedure using allograft, cage and cemented acetabulum. Six years post-op X-ray

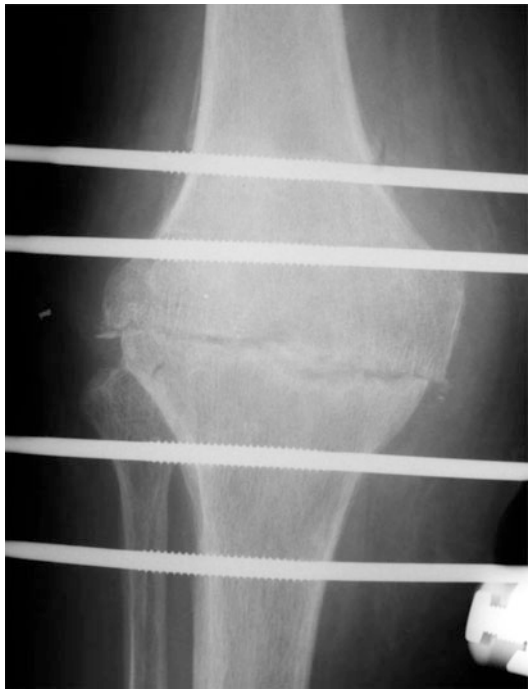


Fig. 24 Post-operative X-ray of the performed arthrodesis of the knee

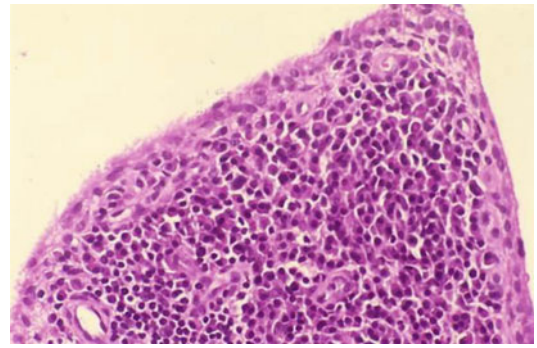


Fig. 25 Synovium in rheumatoid arthritis infiltrated by plasma cells, lymphocytes and macrophages

cysts with curettage of bony erosions and eventually arthrodesis (Fig. 24).

Rheumatoid Arthritis Synovitis

The aetiology of this chronic systemic inflammatory disease is multi-factorial with genetic disposition and immunological reactions leading to generation of cytokines due to an immune system defect and inflammatory reaction of the synovial membrane. There is a predilection for the involvement of joints.

The arthritis is initiated by non-suppurative inflammation of the synovial membrane. This produces an effusion and synovial hypertrophy extends to the margin of the articular cartilage creating the so-called pannus which gradually erodes bone and ligaments leading to distortion of the joint. Extra-articular manifestations are characteristic such as rheumatoid nodules, arteritis, scleritis, pericarditis and splenomegaly.

Swelling, heat, pain, morning stiffness and deformities are the usual symptoms. Women are twice as often affected as men. There are also musculo-skeletal, haematological, lymphatic, pulmonary, cardiovascular, immunological and neurological manifestations.

Laboratory tests show anaemia to some extend depending on the stage of the disease and a raised ESR. Rheumatoid factor is found in 80 % (non specific) of the cases and ANA is present in 20–30%. Anti-CCP antibodies are also found

with specificity of 95 % and in combination with RF of almost 100 %.

Histological examination shows that the synovium is infiltrated by lymphocytes, plasma cells and macrophages. There is hyperplasia of the synovial cells (Fig. 25).

Radiographs do not show specific indications of the disease, but in early stages some osteoporosis may be seen as in the early stages of other inflammatory conditions. As the process of the disease continues there is marginal erosion of bone leading to joint destruction and secondary osteoarthritis. In the early stages the arthritis is atrophic and MRI and CT findings are not specific.

Medication to control inflammation includes disease modifying anti-rheumatic drugs, biologics, NSAID's, including COX-2 inhibitors, and corticosteroids.

If the joint does not respond to the selected drugs, synovectomy may be necessary. It is generally agreed in multi-centred studies that early synovectomy in the first 6 months can prevent or extend the time of appearance of bony erosions, although this remains a matter of debate and there may be no long-term benefit.

Open synovectomy of the knee as described in the section on Pigmented Villonodular Synovitis (PVNS) is a well-accepted procedure for rheumatoid arthritis, and it may be used in other joints such as the elbow, wrist or MCP and PIPJs. Open surgical synovectomy is the treatment of choice providing that there are no erosions of the articular cartilage. Post-operative physiotherapy is important to maintain the function of the joint.

Arthroscopic synovectomy has been introduced in recent years and seems to give better early results with regard rehabilitation and mobility of the joint. A disadvantage of the technique is that hypertrophied synovium may impede the view of the joint. Multiple portals may be used [56]. The operating time is extended [57] and care must be taken to remove as much of the synovium without damaging ligaments and other structures. It is not recommended to remove synovium from the popliteal space because of the possible damage to the vessels and nerves in the popliteal fossa [58]. According to other authors and as it was mentioned in the Pigmented Villonodular Synovitis (PVNS) section, this method is technically demanding and requires posterior portals and 70° scopes [20]. There are no long-term results available for arthroscopic synovectomy. The short-term results are compatible with those of open synovectomy. In a recent comparative study (13 years follow-up) in 53 patients and 58 rheumatoid elbows, of which 23 had been selected to be treated by arthroscopic synovectomy (group 1) and another 23 by open synovectomy (group 2). 11 of the 23 elbows of group 1 and 16 of the 23 elbows of group 2 were mildly or not painful at latest follow up examination and also there was no significant difference in the overall clinical results with both methods used. Open synovectomy provides persistent improvement in pain relief and function, provided that pre-operative flexion is $\geq$ than 90 degrees. If patients have pre-operative stiffness they have higher risk of post-operative stiffness with open surgery. Recurrent synovitis was noticed in 21 % in the arthroscopic group and 10 % in the open group. Fibrous ankylosis is contra-indication for arthroscopy [59]. Although a real joint-preserving effect has not been demonstrated, pain reduction and improvement of joint function recommend arthroscopic synovectomy as a substantial treatment option as described [60]. Open synovectomy of the hip gives 85 % improvement of function and 94 % survival rate with 4 years mean follow-up. In 65 hip synovectomies (nine required dislocation), five hips required total hip arthroplasty during follow up. None showed avascular necrosis [61].

After some years the joint will become eroded and unstable with secondary osteoarthritis and replacement is necessary. The results of total hip and total knee arthroplasties differ from osteoarthritis, due to younger age, osteoporosis and risk factors. In a recent study, the overall infection rate was 3.7 % in 657 hip and knee replacements (follow-up 4.3 +/− 2.4 years) [62].

References

1. Dorfman HD, Czerniak B. Bone tumors. St. Louis: Mosby; 1998.
2. Chassaignac M. Cancer de la gaine des tendons. Gaz Hop Civ Milit. 1852;47:185–6.
3. Dowd CN. Villous arthritis of the knee (sarcoma). Ann Surg. 1912;56:363–5.
4. Jaffe HL, Lichtenstein L, Sutro CJ. Pigmented villonodular synovitis, bursitis and tenosynovitis. Arch Pathol. 1941;31:731.
5. Sciot R, Rosai J, Dal Cin P, et al. Analysis of 35 cases of localized and diffuse tenosynovial giant cell tumor: a report from the Chromosomes and Morphology (CHAMP) study group. Mod Pathol. 1999;12:576–9.
6. Gonzalez-Campora R, Sales Herrero E, Otal-Slaverri C, et al. Diffuse tenosynovial giant cell tumor of soft tissues: report of a case with cytologic and cytogenetic findings. Acta Cytol. 1995;39:770–6.
7. Mahendra G, Kliskey K, Athanasou NA. Immunophenotypic distinction between pigmented villonodular synovitis and haemosiderotic synovitis. J Clin Pathol. 2010;63(1):75–8.
8. Berger I, Weckauf H, Helmchen B, Ehemann V, Penzel R, Fink B, Bernd L, Autschbach F. Rheumatoid arthritis and pigmented villonodular synovitis: comparative analysis of cell polyploidy, cell cycle phases and expression of macrophage and fibroblast markers in proliferating synovial cells. Histopathology. 2005; 46(5):490–7.
9. Bertoni F, Unni KK, Beabout JW, Sim FH. Malignant giant cell tumor of the tendon sheaths and joints (malignant pigmented villonodular synovitis). Am J Surg Pathol. 1997;21:153,163.
10. Perka C, Labs K, Zippel H, Buttgereit F. Localized pigmented villonodular synovitis of the knee joint: Neoplasm of reactive granuloma? A review of 18 cases. Rheumatology (Oxford). 2000;39:172–8.
11. Chin KR, Barr SJ, Winalski C, Zurakowski D, Brick GW. Treatment of advanced primary and recurrent diffuse pigmented villonodular synovitis of the knee. J Bone Joint Surg Am. 2002;84:2192–202.
12. Neale SD, Kristelly R, Gundle R, Quinn JM, Athanasou NA. Giant cells in pigmented villonodular synovitis express an osteoclast phenotype. J Clin Pathol. 1997;50:605–8.
13. Darling JM, Goldring SR, Harada Y, et al. Multinucleated cells in pigmented villonodular synovitis and giant cell tumor of tendon sheath express features of osteoclasts. Am J Pathol. 1997;150:1383–93.
14. Flandry F, Hungston JC, Mc Cann SB, Kurtz DM. Diagnostic features of pigmented villonodular synovitis of the knee. Clin Orthop. 1994;928:212–20.
15. Lawrence T, Moskai JT, Diduch DR. Analysis of routine histological evaluation of tissues removed during primary hip and knee arthroplasty. J Bone Joint Surg Am. 1999;81-A:926–31.
16. Byers PD, Cotton RE, Deacon OW, et al. The diagnosis and treatment of pigmented villonodular synovitis. J Bone Joint Surg Am. 1968;36-A:1007.
17. Zvijac JE, Lau AC, Hechtman KS, et al. Arthroscopic treatment of pigmented villonodular synovitis of the knee. Arthroscopy. 1999;15:613–7.
18. Sharma V, Cheng EY. Outcomes after excision of pigmented villonodular synovitis of the knee. Clin Orthop Relat Res. 2009;467(11):2852–8.
19. Ogilvie-Harris DJ, McLean J, Zamett 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.
20. De Ponti A, Sansone V, Malcherè M. Result of arthroscopic treatment of pigmented villonodular synovitis of the knee. Arthroscopy. 2003;19(6):602–7.
21. Tyler W, Vidal A, Williams R, Healey J. Pigmented Villonodular Synovitis. J Am Acad Orthop Surg. 2006;14(6):376–85.
22. Hamlin BR, Duffy GP, Trousdale RT, Bf M. Total knee arthroplasty in patients who have pigmented villonodular synovitis. J Bone Joint Surg Am. 1998;80(1):76–82.
23. Vastel L, Lambert P, De Pinieux G, Charrois O, Kerboull M, Courpied JP. Surgical treatment of pigmented villonodular synovitis of the hip. J Bone Joint Surg Am. 2005;87(5):1019–24.
24. Gonzalez Della Vale A, Piccaluga F, Potter HG, Salvati EA, Pusso R. Pigmented villonodular synovitis of the hip: 2- to 23-year followup study. Clin Orthop Relat Res. 2001;388:187–99.
25. Yoo JJ, Kwon YS, et al. Cementless total hip arthroplasty performed in patients with pigmented villonodular synovitis. J Arthroplasty. 2010;25(4):552–7.
26. Flundry FC, Hughston JC, Jacobson KE, Barrack RL, McCann SB, Kurtz DM. Surgical treatment of pigmented villonodular synovitis of the knee. Clin Orthop Relat Res. 1994;300:183–92.
27. Mendenhall WM, Mendenhall CM, Reith JD, Scarborough MT, Gibbs CP, Mendenhall NP. Pigmented Villonodular Synovitis. Am J Clin Oncol. 2006;29(6):548–50.
28. Bickels J, Isaakov J, Kollender Y, Meller I. Unacceptable complications following intra-articular injection of yttrium 90 in the ankle joint for diffuse pigmented villonodular synovitis. J Bone Joint Surg Am. 2008;90(2):326–8.
29. Heyd R, Micke O, Berger B, et al. Radiation therapy for treatment of pigmented villonodular synovitis: results of a national patterns of care study. Int J Radiat Oncol Biol Phys. 2010;78(1):199–204.
30. Campanacci M. Bone and soft tissue tumors. Padova/New York: Piccin Nuova Libraria/Springer; 1999.

31. Schoeniger R, Naudie DD, Siebenrock KA, Trousdale RT, Genz R. Modified complete synovectomy prevents recurrence in synovial chondromatosis of the hip. Clin Orthop Relat Res. 2006;451:195–200.
32. 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.
33. Urbach D, McGuigan FX, John M, Neumann W, Ender SA. Long-term results after arthroscopic treatment of synovial chondromatosis of the shoulder. Arthroscopy. 2008;24(3):318–23.
34. Ackerman D, Lett P, Galat Jr DD, Parvizi J, Stuart MJ. Results of total hip and total knee arthroplasties in patients with synovial chondromatosis. J Arthroplasty. 2008;23(3):395–400.
35. Dupont JY. Synovial plicae of the knee. Controversies and review. Clin Sports Med. 1997;16(1):87–122.
36. Dandy DJ. Anatomy of the medial suprapatellar plica and medial synovial shelf. Arthroscopy. 1990;6(2):79–85.
37. Hardaker WT, Whipple TL, Bassett III FH. Diagnosis and treatment of the plica syndrome of the knee. J Bone Joint Surg Am. 1980;62(2):221–5.
38. Yamamoto Z, Fujita A, Minami G, Ishida R, Abe M. A traumatic hemarthrosis caused by a large mediopatellar plica. Arthroscopy. 2001;17:415–7.
39. McGinty JB. Operative arthroscopy. Philadelphia: Lippincott Williams & Wilkins; 2003.
40. Comin JA, Rodriuez-Merchán EC. Arthroscopic synovectomy in the management of painful localized post-traumatic synovitis of the knee joint. Arthroscopy. 1997;13(5):606–8.
41. Bredella MA, Tirman PF, Wischer TK, Belzer J, Taylor A, Genant HK. Reactive synovitis of the knee joint: MR imaging appearance with arthroscopic correlation. Skeletal Radiol. 2000;29(10):577–82.
42. Jaffe HL. Metabolic degenerative and inflammatory diseases of bone and joints. Philadelphia: Lea and Febiger; 1972. p. 726.
43. Gilbert MB, Radomisli TE. Therapeutic options in management of hemophilic synovitis. Clin Orthop. 1997;343:88–92.
44. Thomas S, Gabriel MB, Assi PE, Barboza M, Perri ML, Land MG, DA Costa ES. Radioactive synovectomy with Ytrium citrate in haemophilic synovitis: Brazilian experience. Haemophilia. 2011;17:211–6.
45. Rodriguez-Merchan EC, Megallon M, Galindo E, Lopez CC. Hemophilic synovitis of the knee and the elbow. Clin Orthop. 1977;343:47–53.
46. Solimeno LP, Perfetto OS, Pasta G, Santagostino E. Total joint replacement in patients with inhibitors. Haemophilia. 2006;12 Suppl 3:113–6.
47. Rodriguez-Merchan EC. Total knee replacement in haemophilic arthropathy. J Bone Joint Surg Br. 2007;89(2):186–8.
48. Yoo MC, Cho YJ, Kim KL, Ramteke A, Chun YS. The outcome of cementless total hip arthroplasty in haemophilic hip arthopathy. Haemophilia. 2009;15(3):766–73.
49. Goddard NJ, Mann HA, Lee CA. Total knee replacement in patients with end-stage haemophilic arthropathy: 25-year results. J Bone Joint Surg Br. 2010;92(8):1085–9.
50. Hill JA, Milgram JW, Martin WR. Unusual arthroscopic knee lesions: case report of an intra-articular lipoma. J Natl Med Assoc. 1993;85:697–9.
51. Min KD, Yoo JH, Song HS, Bl L. A case of intra-articular synovial lipoma of the knee joint causing patellar dislocation. Knee Surg Sports Traumatol Arthrosc. 2010;18(8):1094–7.
52. Kalson NS, et al. Destructive synovial haemangioma of the hip resembling pigmented villonodular synovitis. J Arthroplasty. 2011;26:339.e15–20.
53. Aynaci O, Ahmetoglu A, Reis A, Turhan AU. Synovial hemangioma in Hoffa's fat pad: case report. Knee Surg Sports Traumatol Arthrosc. 2001;9:355–7.
54. Price NJ, Cundy PJ. Synovial hemangioma of the knee. J Pediatr Orthop. 1997;17:74–7.
55. Singer S, Bartolini EH, Demetri GD, et al. Synovial sarcoma: prognostic significance of tumor size, margin of resection, and mitotic activity for survival. J Clin Oncol. 1996;14:1201–8.
56. Sculco TP. The knee joint in rheumatoid arthritis. Rheum Dis Clin North Am. 1998;24:143–56.
57. Klug S, Wittmann G, Weselob G. Arthroscopic synovectomy of the knee joint in early cases of rheumatoid arthritis: follow-up results of a multicenter study. Arthroscopy. 2000;16:262–7.
58. Ayral X, Bonvarlet JP, Simmonet J, Amor B, Dougados M. Arthroscopy-assisted synovectomy in the treatment of chronic synovitis of the knee. Rev Rhum Engl Ed. 1997;64:215–26.
59. Tanaka N, Sakahashi H, Hirose K, Ishima T, Ishii S. Arthroscopic and open synovectomy of the elbow in rheumatoid arthritis. J Bone Joint Surg Am. 2006;88(3):521–5.
60. Carl HD, Swoboda B. Effectiveness of arthroscopic synovectomy in rheumatoid arthritis. Z Rheumatol. 2008;67(6):485–90.
61. Carl HD, Schraml A, Swoboda B, Hohenberger G. Synovectomy of the hip in patients with juvenile rheumatoid arthritis. J Bone Joint Surg Am. 2007;89(9):1986–92.
62. Bongartz T, Halligan CS, Osmon DR, Reinalda MS, Bamlet WR, Crowson CS, Hanssen AD, Matteson EL. Incidence and risk factors of prosthetic joint infection after total hip or knee replacement in patients with rheumatoid arthritis. Arthritis Rheum. 2008;59(12):1713–20.