



Spontaneous Osteonecrosis of the Knee (Ahlbäck's disease) in Children, and Overview

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Abstract

Background: Many treatments for Spontaneous Osteonecrosis of the Knee have been described, but there is little guidance on the use of joint-preserving treatments or the use of Biphosphonate for different stages of the disease.

Methods: A systematic review with narrative synthesis of four bibliographic databases was performed to identify studies evaluating efficacy (clinical, radiological outcomes, and failure rates of joint preservation treatment for SONK).

Results: Identified studies: described non-surgical measures and surgical interventions (Study on one patient and described several treatment methods). One study was a randomized trial evaluating conventional treatment with pain relief, use of biphosphonates, and physical therapy. Treatment is supportive with analgesics and weight-bearing restrictions, and most other joint-preserving surgical interventions. has brought the expected results

Conclusions: Rest and analgesia with or without weight-bearing restrictions appear to be appropriate and often effective first-line treatments for stage I or II or III disease. Arthroplasty, rather than joint-preserving therapy, is the definitive treatment for late-stage disease. For children, this issue needs careful consideration. However, current research is limited and higher-level evidence is needed before it can be said with certainty which joint-preserving treatments are most effective for SONK at different stages

Keywords: Avascular Necrosis; Knee Pain; Arthroplasty; Osteonecrosis; Spontaneous Osteonecrosis of the Knee, (SONK); Postarthroscopic Osteonecrosis

Introduction

First described by Ahlbäck, et al. [1] in 1968, osteonecrosis of the knee can be a devastating disease leading to end-stage knee arthritis. Spontaneous osteonecrosis of the knee (SONK) was described as a distinct entity in 1968 [1]. Subsequently several papers have been devoted to this condition but only a few [2-4].

Osteonecrosis of the knee is the second most commonly affected site, after the hip [5]. This remains a rare cause of knee pain for which treatment options and recommendations continue to evolve as we learn more about the cause and pathophysiology of this disease [1]. (Figure 1).

Osteonecrosis of the knee has been classified into three types: idiopathic also known as primary osteonecrosis or osteonecrosis of the knee (SONK)], secondary (also known as traumatic osteonecrosis, ischemic osteonecrosis progressive or idiopathic) and after

endoscopy [5]. SONK is considered the most common form of knee osteonecrosis, with a higher incidence in patients over 50 years of age [6]. Recent studies have reported a prevalence of SONK of 3.4% and 9.4% in people over 50 and 65 years of age, respectively [6]. In contrast, secondary osteonecrosis is more common in younger patients and is associated with certain medical conditions and risk factors such as sickle cell disease, myeloproliferative disorders, alcohol, corticosteroids and smoking.

Postarthroscopic osteonecrosis is the rarest form of osteonecrosis affecting the knee, however, Cetik, et al. [7]. reported that it affects 4% of patients (n = 50) after arthroscopic knee surgery, most commonly after meniscectomy.

Because of the paucity of studies describing different alternatives in patients with idiopathic, secondary, and post-endoscopically determined osteonecrosis of the knee, we have attempted to

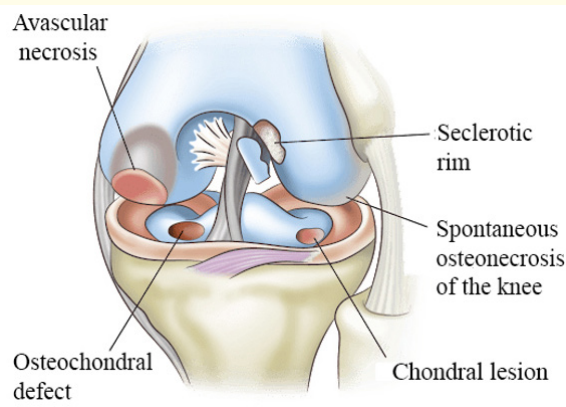


Figure 1: Injury of Spontaneous osteonecrosis Knee.

provide an overview of the etiology. causes, clinical presentation, radiological findings, and most current treatment recommendations for each of these cases. three entities.

Material and Methods

Anatomy

Your knees are the largest and strongest joints in your body. It is made up of the lower end of the femur (femur), the upper end of the tibia (shinbone), and the patella (patella). The ends of these three bones where they meet are covered by articular cartilage, a smooth, slippery substance that protects the bones and makes it easier for them to glide against each other when you move your legs (Figure 2).

Osteonecrosis of the knee most commonly occurs on the inside of the knee (medial femoral canal). However, it can also occur on the outside of the knee (lateral femoral condyle) or on the plane of the shin bone (tibial plateau). Osteonecrosis of the knee occurs most often in the medial femoral condyle, a segment of bone located at the lower end of the femur.

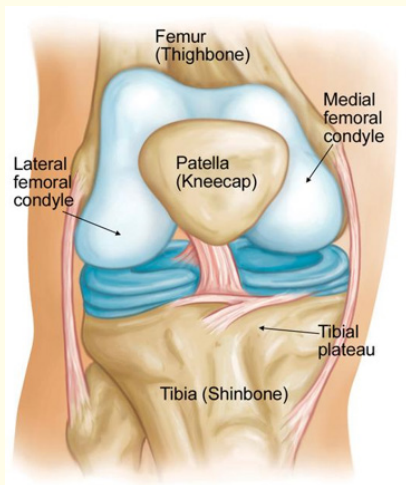


Figure 2: Anatomy of the Knee.

Symptoms

Osteonecrosis develops in stages (Figure 2).

- The first symptom is usually pain on the inside of the knee. This pain can occur suddenly and be triggered by a specific activity or a minor injury.
- As the disease progresses, standing and putting weight on the affected knee becomes more difficult and moving the knee joint becomes painful.

Other symptoms may include

- Swelling in the front and inside of the knee
- Sensitivity to touch around the knee
- Limited range of motion in joints

The disease can take several months to more than a year to progress. It is important to diagnose osteonecrosis early because some studies show that early treatment has better results (Figure 3).

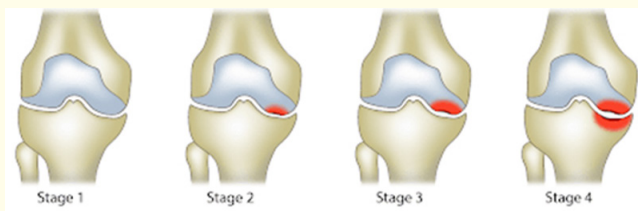


Figure 3: The four stages of osteonecrosis of the knee. The disease can progress from a normal, healthy knee (Stage 1) to the collapse of the bone and severe osteoarthritis (Stage 4).

Physical Examination

To perform a careful examination of the knee, looking for:

- Joints are swollen, hot or red
- Tender
- Passive (assisted) and active (self-directed) range of motion
- Joint instability
- Pain when putting weight on the knee
- Any signs of injury to the muscles, tendons and ligaments around the knee

During the exam, to look for painful areas in your knee. Imaging studies will help to confirm the diagnosis.

If you have osteonecrosis, may take your medical history and perform a physical exam, may also order one or more imaging tests to see which bones are affected and evaluate the extent of bone or joint damage. This information helps determine the stage of the disease. Early diagnosis increases the chance of successful treatment.



Figure 4: Examination of the Knee.

Imaging study
X-ray

X-rays are often the first type of imaging study a doctor will order because they can also help rule out other causes of joint pain, such as fractures or arthritis (Figure 5,6). However, X-rays are not sensitive enough to detect bone changes in the early stages of osteonecrosis, so your doctor may order further tests if your X-ray results are normal. If you are diagnosed with the disease, X-rays are often used to monitor bones at a later stage.



Figure 5: X-ray shows osteonecrosis at the lower end of the femur in the medial femoral condyle.



Figure 6: As osteonecrosis progresses, narrowing of the joint space can occur due to loss of articular cartilage.

Magnetic Resonance Imaging (MRI)

MRI is a highly sensitive tool for visualizing bones and joints and can detect early signs of osteonecrosis before they are seen on X-rays (Figure 7). An MRI scan can even detect signs of osteonecrosis before you experience symptoms. MRI can help provide doctors with information about the extent of bone damage and the overall structure of the joint.

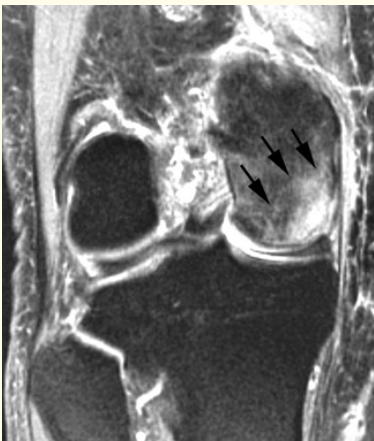


Figure 7: This MRI scan shows osteonecrosis in the medial femoral condyle. An MRI is often used to detect early.

Computed Tomography (CT) Scan

A CT scan produces clearer images than a regular X-ray (Figure 8). Doctors can use CT scans to evaluate the extent of bone damage, but they are less used than MRIs because they are less sensitive.



Figure 8: CT scan knee joint of Spontaneous osteonecrosis Knee.

Clinical Presentation

SONK is a relatively rare disease, usually occurring in middle-aged to elderly patients. It affects three times more women than men [1,8-10]. There is little published epidemiological data on the incidence and prevalence of SONK; one study described an incidence of 3.4% in patients under 50 years of age, increasing to 9.4% in those over 65 years of age [6].

This condition typically presents with acute severe pain, often localized to the affected area, often without a history of trauma [9].

Patients often complain of increased pain when carrying heavy objects, along with persistent pain at rest [10]. Examination of the knee may reveal evidence of effusion with localized tenderness over the affected area, usually the medial femoral condyle [9,11]. In the early, acute stages of the disease, the patient may have limited range of motion compared to the normal knee. In more permanent cases, permanent flexural deformation may occur [12].

Diagnosis

The diagnosis of SONK can often be made based on the clinical history and radiographic changes.

However, routine radiographs often give normal results in the early stages of the disease. Magnetic resonance imaging (MRI) may be required [13], which can detect subtle changes in the bone marrow as early as 72 hours after symptom onset [14]. It can provide more detailed information about the extent and distribution of bone marrow involvement as well as the presence of cartilage damage due to bone collapse, with regard to prognosis [14,15]. MRI has significantly improved the early diagnosis of disease and is the imaging modality of choice for diagnosis, staging, and monitoring of disease [14,16,17].

Differential diagnosis

Knee osteonecrosis can be divided into four groups: idiopathic osteonecrosis of the knee (SONK, also described as primary osteonecrosis), secondary osteonecrosis (also called idiopathic osteonecrosis, ischemic or non-traumatic), post-arthroscopic and post-traumatic osteonecrosis [5,13]. SONK is the most common type of osteonecrosis and affects older age groups [6], compared to secondary osteonecrosis, which is associated with potential medical or drug problems. hidden otherwise. Alcohol, steroid use, sickle cell disease, myeloproliferative disorders, and kidney disease have all been linked to the development of secondary osteonecrosis [9]. Postarthroscopic osteonecrosis is considered the rarest form, with onset after arthroscopic knee surgery, more specifically after meniscectomy [7,9]. In posttraumatic osteonecrosis, as the name suggests, there is a history of trauma or surgery before the onset of symptoms, leading to bone death, often in a discrete area of the knee [5].

In addition to the various forms of osteonecrosis, there are other intra-articular pathologies of the knee that need to be considered when diagnosing SONK: osteochondritis dissecans, transient osteoporosis, and traumatic bone marrow injury must be ruled out. except because they often have common clinical and pathological signs [5]. It can sometimes be difficult to differentiate between these conditions based on history and physical examination alone, and radiological findings can be confusingly similar. Certain

characteristics, such as patient age, lesion location, clinical symptoms, histology, lack of intra-articular laxity, and relatively slow appearance of radiographic changes Routine radiology can help distinguish SONK from other conditions [8].

Osteochondritis dissecans (OD) is a condition that affects the articular cartilage and subchondral bone. It leads to various pathological abnormalities, starting with softening of the articular cartilage, premature separation of the cartilage and partial or, in some cases, complete separation of an osteochondral fragment [6]. It affects the posterolateral aspect of the medial femoral condyle in most cases. OD typically affects adolescents and young adults, whereas SONK is most common in middle-aged and elderly patients [9].

Transient osteoporosis is another differential diagnosis. This is an uncommon, self-limiting syndrome of unknown cause, characterized by joint pain and osteoporosis [20]. Unlike SONK, it is usually located on the lateral femoral condyle and has characteristic manifestations of focal osteoporosis within 8 weeks of symptom onset [21,22].

Posttraumatic bone marrow lesions and edema should also sometimes be considered as a possible diagnosis; this may be associated with acute injury or with more subacute injuries related to overload, such as stress fractures [23].

Specific features on images can be used to aid in the diagnosis of SONK. One of the most important differences between early SONK and bone marrow edema (as seen in osteoporosis or transient trauma) is the presence of focal subchondral lesions on MRI [24,25]. The localized nature of the lesions in early SONK suggests that a pathologic process is underway at this location, rather than a disease process that is diffuse throughout the femoral condyle, as seen in marrow edema. bones associated with osteoporosis or transient trauma [14].

The location of the disease also helps distinguish between conditions; The lateral femoral condyle is the most commonly affected site in transient osteoporosis, the lateral aspect of the medial femoral condyle is often affected in OD, while the medial aspect of the femoral condyle and tibial plateau are often involved. most related to SONK [5]. Border flattening is also seen more frequently inSONK [26].

Treatment

Treatment of osteonecrosis depends on several factors, including:

- Stage of the disease
- The amount of bone affected
- The underlying cause of the disease

Non-surgical treatment

In the early stages of osteonecrosis, treatment is nonsurgical. If the affected knee area is small, nonsurgical treatment may be sufficient.

Nonsurgical treatment may include:

- **Medicine**
 - Nonsteroidal anti-inflammatory drugs (NSAIDs), such as ibuprofen and naproxen, can help reduce pain and swelling in your knee.
 - Your doctor may also talk to you about bisphosphonates for treatment, especially if you are diagnosed at a younger age.
- **Reduced weight bearing capacity:** For some patients, removing weight from the affected knee can slow damage from osteonecrosis and aid healing. Your doctor may recommend that you use crutches for a while to take the weight off your knee. In some cases, wearing an “unloading” brace can help relieve pressure on the joint surfaces by shifting weight away from the affected part of the knee.
- **Exercise:** Your doctor or physical therapist can provide you with an exercise program designed to help strengthen your thigh muscles and maintain range of motion in the affected joint. In some cases, you may want to exercise in water to avoid stress on your knee joints.
- **Modify operations:** Your doctor may recommend that you avoid certain activities that cause painful symptoms.

Surgical treatment

If most of the bone surface is affected or if your pain does not improve with nonsurgical treatment, your doctor may recommend surgery. There are several different procedures used to treat osteonecrosis of the knee.

Arthroscopic surgery and microfractures. During debridement (cleaning), your doctor will use a small camera and miniature surgical tools to remove loose pieces of bone or damaged cartilage from inside the joint space. For small lesions, the surgeon may also drill multiple holes or microfractures in the underlying bone to help promote blood flow and induce a healing response.

Core decompression

This procedure involves drilling a larger hole or several smaller holes into the bone to relieve pressure on the bone surface and create channels for new blood vessels to nourish the affected areas of the knee (Figure 9).

When osteonecrosis of the knee is diagnosed early, core decompression is often successful in preventing bone collapse and the development of arthritis.



Figure 9: As osteonecrosis progresses, narrowing of the joint space can occur due to loss of articular cartilage.

Osteochondral (bone and cartilage) grafting

Core decompression is often combined with bone and cartilage grafting to help regenerate healthy bone and cartilage support in the knee joint. A bone graft is healthy bone tissue transplanted into a needed area of the body. This tissue can be taken from a donor (allograft) or from another bone in your body (autograft).

Currently there are also some types of artificial bone grafts.

Autologous chondrocyte implantation (ACI)

This is a two-stage procedure. In the first stage, your doctor performs an arthroscopy to remove a small number of cartilage-producing cells (chondrocytes) from your knee. These cartilage cells are sent to a laboratory where they are cultured (multiplied) for up to 6 weeks to obtain more cells.

In the second stage, the doctor performs another procedure to transplant cartilage cells into the area of your knee that has lost cartilage. Cells then grow in the joint, replacing the damaged cartilage with healthy cartilage.

Bone cutting

In an osteotomy, the tibia (shin bone) or femur (thigh bone) is cut and then reshaped. The surgeon will remove a portion of bone or insert a bone graft or synthetic bone to help shift weight away from the damaged area of your knee. Shifting your weight away from the damaged side of the joint will help reduce pain and improve function.

Total knee replacement or partial (part) knee replacement. If the disease has progressed to the point where the bones have collapsed, you may need surgery to replace damaged parts of your knee. During a knee replacement, the doctor removes the damaged bone and cartilage, then places new metal or plastic joint surfaces to restore knee function.

Outcome

For most patients, treatment of osteonecrosis is successful in reducing pain and improving function. However, results may vary depending on the stage of the disease at diagnosis and the type of treatment.

Case Report

A 6-year-old boy presented with gradually sharp pain in the inner half of his knee, turning into stabbing pain when bearing weight. He had no previous health complaints, did not take any medications, and did not smoke. Physical examination was unremarkable, only mild swelling and effusion.

The last four weeks he has been walking a lot on hard surfaces. MRI showed nonspecific bone marrow edema. All MR examinations were performed on an Achieva 1.5 T scanner (Philips, Best, Netherlands) with a standard knee protocol. At follow-up MRI after six weeks, bone marrow edema had increased in both intensity and extent and was suggestive of osteonecrosis. A small subchondral sclerosis was also present in the medial femoral condyle on T1-weighted (T1-weighted) images. X-rays were unremarkable. However, combined, the two MRI studies were interpreted as suggesting a possible deficiency fracture. Treatment has now been initiated with non-steroidal anti-inflammatory drugs (NSAIDs) and preservation of weight-bearing capacity.

MRI at three months showed unchanged distribution but further progression of signal intensity of bone marrow edema. Knee pain did not decrease in previous pain intensity according to the patient's own assessment. However, pain and swelling increased when carrying heavy objects, and the patient had difficulty moving due to knee pain and mild effusion. Physical therapy treatment was initiated and protected weight bearing continued.

At six months, MRI showed no change in overall regression of bone marrow attenuation. The findings were now interpreted as osteonecrosis. Treatment with a bisphosphonate (alendronic acid 50 mg orally/week) was initiated, while protected weight bearing was continued. MRI at seven months showed mild progression of the subchondral cystic area and reappearance of sporadic bone marrow edema. Clinical outcomes are unstable. At 10 months, MRI showed progressive bone marrow edema but the cyst was not filled. Bisphosphonate treatment has now been changed to zoledronic acid (5 mg IV/6 months). At the clinical examination after one year of follow-up, there were no changes in clinical outcomes.

After one year and six months, the minimal subchondral lucency was not present on radiographs, while MRI showed that the cyst was still progressing. Knee pain persists, which increases with

weight bearing and knee bending. On physical examination, there was some swelling and effusion. Treatment was continued with zoledronic acid (total duration of treatment with bisphosphonates was 1.5 years) and weight bearing was maintained. At one year and ten months, an MRI scan showed that there was still subchondral edema. The pain remained but moderate muscle atrophy had developed. The final Clinical (Figure 10), MRI (Figure 11), CT Scan (Figure 12), and Radiographic (Figure 13), Imaging Diagnosis were Stages IV [1] are results after two years showed more severe osteonecrosis and limited joint mobility. Patient is still conservatively treated and observed.



Figure 10: A-C. Clinical Images: A. Mild swelling around Knee, B. Knee Flexion 134.6°, and C. Extension 35.2°.

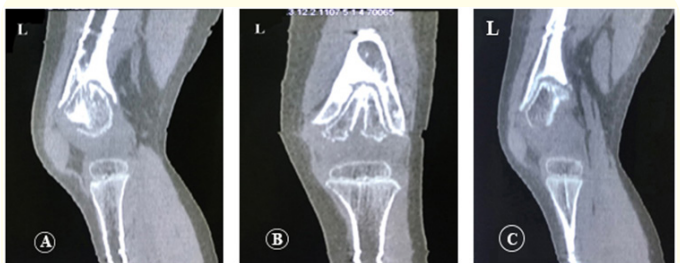


Figure 11: A-C. Injury of Condylar and Knee show on MRI.



Figure 12: A-C. Injury of Condylar and Knee show on CT Scan.

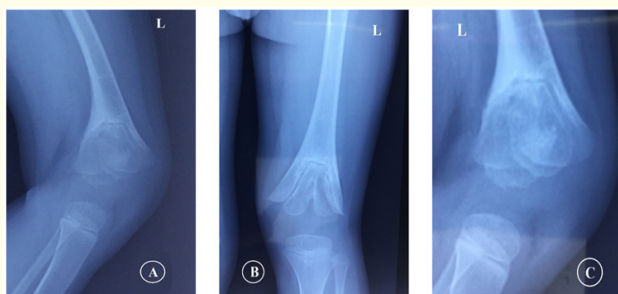


Figure 13: A-C. Injury of condylar show on X- ray.

Discussion

Since idiopathic osteonecrosis (ON) of the knee was described by Ahlback in 1968, [1] many studies have been published that have helped define the natural history of the disease, allowing its classification into stages. progression and comparison of treatment options. A form of secondary osteonecrosis has been identified that typically involves larger areas of bone. Currently, the treatment of ON knee is still controversial and depends on many factors including: the patient's age, the stage of the disease as well as the size and location of the lesion.

The rate of ON in the distal femur is second only to the femoral head, but still only accounts for 10% of the total cases of avascular necrosis. As a result, most studies have had small numbers of patients, making firm conclusions about the natural history and appropriate treatment difficult. Early stages are usually treated non-surgically while more advanced stages often require surgery.

Blood supply to the knee

The knee is the largest and shallowest synovial joint in the body and is made up of three joints; two femoral joints between the femoral condyle and the lateral and medial tibia, and one patellofemoral joint between the femur and patella [27].

The knee has a rich arterial blood supply (Figure 14), mainly originating from the five main branches of the popliteal artery. The superior medial and lateral geniculate arteries, the medial (posterior) artery, and the inferior medial and lateral geniculate arteries form a rich anastomosis to supply blood to the knee joint, [28]. The highly vascular structure of the knee is involved in all aspects of growth, repair, and metabolism [29]. There is increasing evidence that abnormalities in the blood supply around the knee may be directly related to the initiation or progression of many disease processes and have been implicated in the development of osteoarthritis as well. and osteonecrosis [30].

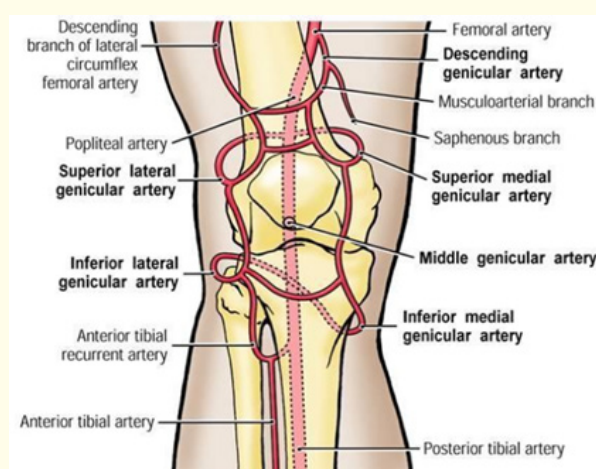


Figure 14: Genicular anastomosis, providing blood supply to the knee joint [31].

Etiology

Primary spontaneous ON has been hypothesized to have a traumatic etiology. It is thought that microfractures in the subchondral bone are the result of an accumulation of microtrauma. This allows synovial fluid to enter the bone, increasing edema and marrow pressure and ultimately leading to ischemia. This theory is speculative, as only a minority of patients reported any prior trauma. Currently, there are two main theories regarding the cause of secondary ON, neither of which has been proven. Disruption of microvascular circulation in the subchondral bone may result from fat embolism or microthrombosis, which then causes edema and increased bone marrow pressure. This increased pressure further impedes circulation, leading to ischemia and necrosis. Steroid treatment is associated with secondary ON. Corticosteroids may increase the size of fat cells in the marrow. This factor can increase spinal pressure leading to ischemia. Many other conditions are associated with ON including: systemic lupus erythematosus (SLE), kidney transplant, alcoholism, Gaucher disease, hemoglobinopathy, and Caisson decompression sickness.

Arthroscopic surgery is rarely associated with ON knee. Whether arthroscopy can be considered a factor in the pathogenesis of ON remains controversial. Several studies have reported "post-endoscopic" ON diagnosed by MRI. Unfortunately, most cases are diagnosed retrospectively and a pre-existing condition of ON cannot be excluded.

Reddy and Frederick [32] identified distinct differences between the lateral and medial femoral condyles with respect to intraosseous and extraosseous blood supply in a cadaveric dye injection study. They hypothesized that this difference could explain the increased frequency of femoral osteonecrosis in the femoral condyle. The superior lateral geniculate artery and inferior lateral geniculate artery combine to supply the lateral femoral condyle, while the superior medial geniculate artery and other small branches of the popliteal artery supply the medial femoral condyle. The intraosseous supply to the lateral condyle consists of a multibranched bypass supplying the subchondral bone, in contrast to the medial femoral condyle, which has a single trophic vessel to the subchondral bone and an area of potential divides at the anterior femoral condyle. They noted that the femoral tunnel used in PCL reconstruction is located very close to the main extra-cellular blood supply to the medial femoral condyle and may be at risk during surgery.

Staging of disease

Koshino, *et al.* 1982 [33] described a four-tiered radiological classification of SONK based on plain radiographic appearances, which was later modified by Aglietti, *et al.* 1983 [8] to include a fifth stage of disease, describing the appearance of degenerative changes of the knee. This is the classification system still widely in use to stage SONK (Table 1).

Stage	Radiographic changes
1	No changes on plain radiographs
2	Flattening of the weight bearing portion of the femoral condyle
3	Flattening of the femoral condyle with sclerotic halo around area of disease
4	Sclerotic ring becomes more defined, with associated subchondral collapse
5	Narrowing of the joint space, osteophyte formation ± tibial subchondral sclerosis

Table 1: Stages of SONK as described by Koshino (1982) [3] and later modified by Aglietti (1983) [8].

In stage 1 disease, also described as the incipient stage, patients usually describe significant knee pain which can last for several weeks. Plain radiographs are usually normal. After a variable period, the pain may spontaneously resolve, and patients become asymptomatic, or the disease may progress to subsequent stages [3].

Stage 2 disease is characterized by the appearance of flattening of the medial femoral condyle on plain radiographs, with MRI scanning able to provide additional information about the overall size of the area of osteonecrosis [33]. (Figure 15).

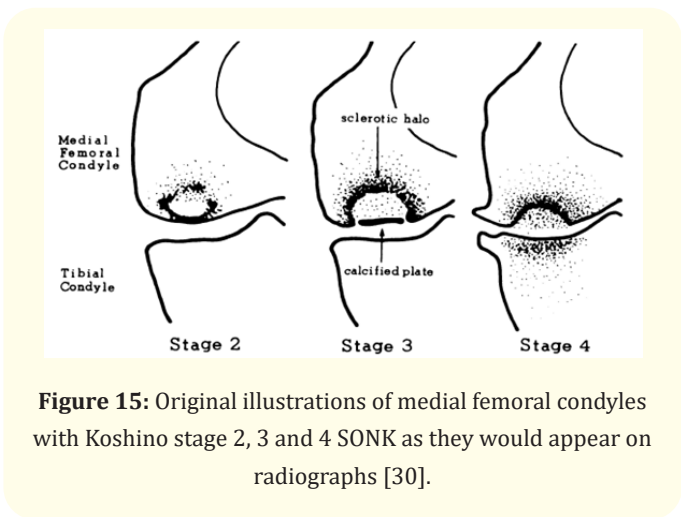


Figure 15: Original illustrations of medial femoral condyles with Koshino stage 2, 3 and 4 SONK as they would appear on radiographs [30].

The onset of stage 3 disease is determined by the appearance of a ring of sclerosis on radiographs. This lesion represents partial necrosis of the subchondral bone accompanied by sloughing of the overlying articular cartilage [33]. Further sclerosis with destruction of articular cartilage and subchondral collapse represents progression to stage 4 disease, which may extend across the width of the femoral condyle leading to loss of joint uniformity and joint collapse. This can lead to significant knee misalignment [33].

Patients with stage 5 disease exhibit characteristic features of osteoarthritis. Plain radiographs show narrowing of the joint space, bone spur formation with subchondral sclerosis in some cases [8].

For cases of SONK that have progressed to degenerative changes, the OA classification system, described by Ahlbäck (1968) [1] or Ficat and Arlet (1980) can also be used to classify type of disease severity (Table 2).

Stage	Ahlbäck classification [1]	Ficat and Arlet classification [34]
0	No signs of arthrosis	
I	Diminished joint space	Knees with normal appearance
II	Obliteration of joint space	Cystic or sclerotic lesions, or both. Normal joint contour, no subchondral fractures
III	Erosion <5mm	Crescent sign & subchondral collapse
IV	Erosion between 5-10mm	Reduced joint space, subchondral cysts and osteophytes
V	Erosion >10mm and lateral subluxation	

Table 2: Classification systems used to describe degenerative changes associated with SONK [1,34].

Clinical presentation and diagnosis

Patients with ONK often present with acute pain in one knee without injury. The output kneeling on the inner thigh is the most common sign when performing surgery. Patients often feel pain that is worse at night and when carrying heavy objects, often similar to the pain after a torn meniscus. In the early stages of the disease, anteroposterior (AP), lateral, and treatment radiographs are recommended, although in the early stages of the disease they are often negative and in some cases remain calculated for the entire duration. clinical appearance of symptoms.

Typical radiographic signs of later-stage disease include radiolucent or flattening of the condyle. Magnetic resonance imaging (MRI) is recommended for early detection of disease stages due to its high sensitivity in detecting fits. Initial MRI features of early-stage pterosaurs include low-signal-intensity submental areas on T2-weighted images, ambulatory epiphyseal contours, and deep low-signal-intensity contours in the request for influence. Bone scintigraphy may show increased uptake in the affected condyle, however, this method is more sensitive in identifying disease than MRI.

As a result, bone scans are not indicated for lizards, which are exceptionally sensitive and friendly, with one study demonstrating that bone scans detected only 56% of injuries previously detected by MRI. in which the disease setting is multifocal, histologically confirmed. The Koshino classification was the first classification for SONK described in 1979 [3]. There are four stages in the Koshino staging system, based on clinical and radiological findings. A patient who has symptoms in the knee but has normal radiographs is classified as stage I. Stage II represents an area of strength tolerance with nutrients in the form of a splint and surrounding protection with stiffness. Stage III can realize the expansion of the optic zone around the affected area and collapse under the flesh. Stage IV is a degenerative stage with bone hardening and spur formation around the condyle.

Treatment

Nonoperative treatment

Nonsurgical treatments are most appropriate for asymptomatic patients and those in the early stages of the disease. Weight protection, analgesics, nonsteroidal anti-inflammatory drugs (NSAIDs), and physical therapy for quadriceps and hamstring strengthening were all used. Idiopathic ON may respond well to nonsurgical treatment. Lotke and colleagues reported 35 of 36 knees that functioned well when treated nonoperatively in spontaneous stage I [11]. Lesions can sometimes heal on their own. This is more likely to happen in the early stages and with smaller

lesions. Ahlback, *et al.* [1]. found that lesions smaller than 0.24 [cm. sup.2] can resolve, even after several months.

Uchio and colleagues [11], in a randomized controlled trial, concluded that the use of a lateral wedge base is an effective treatment for early idiopathic osteonecrosis affecting the medial femoral condyle. They found a significant improvement in the size of the necrotic area as well as pain when using the lateral wedge. They cautioned that results were not affected in knees with progressive joint changes or significant varus alignment. Secondary ON may not be as effective as nonsurgical treatment. Mont., *et al.* [5]. found that 59% of knees treated nonoperatively for secondary ON had good or excellent results after only 2 years of follow-up. By 6 years, only 18% of knees survive nonsurgical treatment, and disease stage is thought to contribute little to the final outcome.

Operative treatment

A variety of surgical techniques have been performed for ON including arthroscopic debridement, osteotomies, core decompression, bone grafting, and prosthetic replacement. Indications for surgery are based on the stage and extent of the disease as well as the patient's age and needs.

Arthroscopy

Wiedel [35] reported the successful treatment of mechanical symptoms and knee pain associated with steroid-induced ON over a mean follow-up period of 40 months through arthroscopic resection of the articular cartilage flap, loose body and torn meniscus due to degeneration. In a small series, Miller concluded that tissue excision does not alter the natural course of the disease but may reduce the mechanical symptoms. If fluid escape through subchondral cracks occurs, intraosseous pressure may increase, possibly worsening ischemia.

High tibial osteotomy

High tibial osteotomy (HTO) can be used to shift the mechanical axis of the limb so that the affected femoral condyle is offloaded. Use of HTO requires only one condyle to be affected, so it is more commonly used in cases of spontaneous ON. Koshino [3] compared the results of drilling or bone grafting for osteonecrotic lesions of the femoral condyle with or without high tibial osteotomy to correct angular deformity. In this study, both radiographic and clinical improvement were greater in knees that had concomitant tibial osteotomy. Because the results of varus osteotomy for medial condyle involvement are best in knees with a postoperative femoral angle of 164° to 173° and 7° to 16° of anatomic valgus, Koshino recommends a goal of 10° varus. Postoperative knee score is inversely correlated with preoperative Koshino stage and therefore earlier

surgical intervention is recommended. During an average follow-up of 5 years, only one patient required total knee replacement, while 95% experienced pain relief and improved function. Aglietti, *et al.* reported 87% good to excellent results after 6.2 years using HTO with or without additional bone grafting. In their study, necrosis improved in 58% while osteoarthritis was seen in 29%. Soucacos and colleagues, in a retrospective review of 32 patients, recommended HTO in patients younger than 60 years of age with stage III primary osteonecrosis with an area of less than 50% of the condyle. On the other hand, prosthetic replacement provides better results. The use of HTO in secondary ON is generally not recommended because the patient may have bicondylar or more extensive lesions.

Core decompression

Core decompression is thought to reduce pressure within the bones, allowing for improved vascularity and possibly slowing the progression of the disease. In a procedure similar to that used in the hip, a guide wire can be placed into the necrotic bone in a retrograde direction. A drill with a 6 mm or 8 mm tube is then used to decompress the bone without damaging the joint surface. Multiple smaller burrs, forward or backward, can be used to decompress and stimulate healing of osteonecrotic lesions. 0.62 Kirschner wire can be used for this technique.

Lotke, *et al.* [36] do not recommend core decompression; it did not alter the natural history in a series of 79 patients with stage I osteonecrosis of the medial femoral condyle.

An extra-articular drilling procedure was used to achieve core decompression in 16 patients with primary ON in a study by Forst and colleagues. Fifteen of these patients had stage I disease. This resulted in immediate pain relief in all cases and complete resolution of the pulpal signal in all but one case at 6 months postoperatively. Patients treated nonoperatively continued to have pain for several months. These investigators concluded that extra-articular drilling of the early ON of the femoral condyle reduces pain, but once the condyle collapses, progression of the disease cannot be prevented.

Jacobs, *et al.* [37] reported their results on 28 core decompressions performed primarily for secondary AVN (26 of 28 cases, 2 idiopathic). Their results depend on the stage. All patients with Ficat stages I and II had good outcomes after a median follow-up of 54 months. People with stage III osteonecrosis have a more unpredictable outcome; however, despite the 52% success rate, they suggest that the procedure may have a role in younger patients, as the alternative may be total knee replacement. They made the important point that core decompression should not be considered a substitute for femoral or tibial osteotomy to correct malalignment.

In a retrospective analysis of 102 knees with ON secondary to steroid use, Mont, *et al.* [5] initially treated all patients nonoperatively; Forty-seven knees that remained symptomatic after a minimum of three months of protected weight bearing underwent core decompression. Nonsurgical treatment yielded poor results with only 18% of knees classified as good or excellent by the Knee Society score at 6 years, while the core decompression group had 73% results from good to excellent after 11 years of follow-up. This difference was even more dramatic as Ficat stage increased (86% in the core group vs. 15% in the nonsurgical group). They also reported reduced radiographic progression in the core group (35% progressed to stage III or IV in the core group vs. 75% progressed in the nonsurgical group) [38]. has recommended the addition of a calcium sulfate-based bone lubricant that injects and aspirates bone marrow into the defect created by core decompression in an attempt to hasten the filling of the defect and prevent bone collapse. Extracting the core is a relatively minor procedure. Patients often feel immediate pain relief and it can slow the progression of the disease. This should be done before the joint collapses. Rescue operations are not affected by this procedure.

Autologous bone grafting

Perez, *et al.* [39] reported an excellent outcome in one patient at 2 years of follow-up after the use of an autologous iliac crest plug, followed by CPM and 8 weeks of non-weight bearing in one patient. Grade 3 AVN on the Skeletal Joint side.

Fukui, *et al.* [40] used autologous iliac crest bone graft in the treatment of femoral condyle secondary to steroid use. In their series, 90% of knees achieved good pain relief and maintained good range of motion after a mean follow-up of 79 months. All seven patients in this study were able to walk an unlimited distance after post-procedural rehabilitation. The osteochondral graft was taken from the ipsilateral iliac crest and pressed tightly into the osteochondral defect at the site of osteonecrosis. The shape and contour of the bone is chosen to match as closely as possible the area of the affected femoral condyle. The periosteum is left intact and continuous passive motion is used to stimulate chondrogenic potential after surgery. Whether this actually led to cartilage formation remains to be determined, but the investigators noted that no functional decline occurred at the most recent follow-up examination. One patient in this study who had a poor outcome had an extensive necrotic area that did not provide a stable foundation for the graft, which subsequently migrated. These investigators recommended revision of realignment if mechanical axial malalignment existed and required careful planning for defects wider than 3 cm, as graft width is limited. They concluded that this technique is useful in the treatment of osteonecrosis because it can allow normal function and maintenance of bone mass, even if definitive surgery

is required in the future. Kotani and colleagues [41] reported on 16 patients, all of whom had positive results after 5 years using autologous bone grafting with the osteoarthritic transfer system (OATS) for Lotke lesions. 2-3. They emphasize the point that these patients do not have varus alignment.

Osteochondral Allograft

The use of allogeneic bone graft to replace necrotic bone has had mixed results in the literature. Bayne and colleagues reported 66% good or excellent results in posttraumatic ON, whereas spontaneous or secondary osteonecrotic knees had poor results, with all but one case. They felt that elderly patients with idiopathic ON were unable to comply with postoperative weight-bearing restrictions leading to subsidence and fragmentation of the grafts while poor vascularization in secondary ON was observed. Prevents the joining of pieces.

Meyers and colleagues reported on the use of novel osteochondral allografts for a variety of diagnoses. [42] All three patients treated with new osteochondral grafts for osteonecrosis had excellent results after 3 to 4 years of follow-up. They identified eight necessary conditions to achieve successful results using allogeneic bone and cartilage grafting.

- The osteochondral allograft must be fresh and include no more than 0.5 to 1 cm of subchondral bone.
- The dimensions of the donor and recipient must match exactly to achieve uniformity of the joint surface.
- The graft must be "covered" with autologous bone to ensure uniform contact.
- Must have a firm compression fit.
- The joint must be stable.
- The synovial membrane must be closed to contain synovial fluid that provides nutrients.
- Non-weight bearing must be strictly enforced until complete grafting; And
- Joint movement must be started early.

Flynn, *et al.* reported on the use of fresh frozen allograft in secondary ON in patients younger than 50 years of age. On average, after 4.2 years, 70% of knee joints have good or excellent results. The allografts in their study contained a significant amount of bone, as they were used as replacements for large osteonecrotic lesions. Frozen allografts may be less immunogenic than fresh bone allografts, however, postmortem histological examination of fresh osteochondral allografts has shown that human chondrocytes can last up to 8 years. This technique may be best used as an adjunct to another procedure such as HTO, which can preserve the graft by shifting the mechanical axis of the limb, especially in young patients requiring leg replacement.

Unicompartmental knee arthroplasty

When considering prosthetic replacement, unicompartmental arthroplasty is an attractive option as there may be less blood loss, less bony resection, quicker rehabilitation, and more normal knee kinematics. It is important to recognize that unicompartmental arthroplasty is unable to make large corrections to the mechanical axis. If there is significant malalignment, a total knee replacement would be indicated. Atsui and coworkers [43] found unicompartmental knee arthroplasty to be a useful procedure in ON with the following indications: unicompartmental ON, body weight less than 155 pounds, age greater than 60 with low expected activity level, flexion contracture less than 30[degrees], less than 10° of angular knee deformity, and intact cruciate and collateral ligaments. These investigators aimed for a femoral tibial angle of 175°, 5° of valgus, and reported good to excellent results in 100% of ten patients at an average of 42 months follow-up.

In a review of 34 unicompartmental arthroplasties performed for ON of the medial femoral condyle at an average of 5.5 years follow up, Marmor [44] reported 89% good to excellent results. Two of the four failures in his series were due to development of lateral condyle ON requiring revision to TKA. Cartier and colleagues [63] found that 95% of patients maintained good and excellent results at 54 months following unicompartmental replacement for osteonecrosis.

Lotke and associates [4] recommend treatment of their stage II-III lesions with arthroplasty. Unicompartmental arthroplasty may be adequate if the lesion is confined to a single condyle. Larger lesions may develop fixed deformities if surgical intervention is delayed and may require total knee replacement. Unicompartmental knee replacement is probably best used in spontaneous ON, affecting only one compartment in a patient who is older and lighter. It is appropriate to order a preoperative MRI when considering unicompartmental replacement to rule out the presence of osteonecrotic lesions in the opposite condyle. If the other compartment is affected, or in cases of secondary osteonecrosis, total knee replacement may provide a more predictable result.

Total knee arthroplasty

Several studies have focused on total knee replacement for ON and the results are mixed. Ritter and colleagues [45] compared the survival of total knee replacement (TKR) for primary ON with that of osteoarthritis and reported no statistical difference in severity, pain, correction, or radiolucency among these groups. Limitations of this study include a small study group consisting of only females and a high dropout rate. Their revision rate was 17% after seven years. Aglietti and colleagues [46] recommend TKR for ON stage III or higher. This procedure yielded 95% positive results after 4.4 years. They caution against the use of monocondylar replacement because they found a 28% correction rate after initial good

results. Radke and colleagues [47] also concluded that although single knee replacement may provide better short-term results, total knee replacement is better in the long term. Bergman and Rand [48] reported 87% good and excellent outcomes with TKR for secondary ON at 4 years, with a predicted survival rate of 85% at 5 years. In this study, there were no failures due to aseptic loosening, and the authors noted that necrotic bone did not pose any management problems.

Seldes and colleagues [49] reported a 5-year survival rate of 84% in steroid-induced ON knee. Three failures were due to loosening, while two were due to infection. They also found a minor complication rate of 19%. They hypothesized that this complication rate may be due to the fact that many patients with secondary ON have chronic diseases and are therefore physiologically weaker.

Mont and colleagues [5] reported a success rate of 55% and a loosening rate of 37% in a group of patients with ON secondary to lupus and steroid use. In a more recent study, they reported a discouraging success rate of 71% using a variety of knee prostheses for secondary ON over a 24-year period. A subsequent study using modern cemented prostheses and stems as needed yielded a 97% success rate after a minimum follow-up of 4 years.

Although the results of TKA for ON are not quite as good as those performed for osteoarthritis, it appears that addressing bone deficiency with additional cement and stem injection may narrow the difference. Total knee arthroplasty remains the most predictable surgical treatment modality, especially in older patients with ON.

Physical therapy management

While it has long been known that the basic process in Legg-Calvé-Perthes disease and Ahlbeck is one of bone necrosis and some Osteonecrosis in Children, the actual causal mechanisms are unknown [50]. Catterall researched Perthes-Legg-Calvé conservative treatment in 1981 [51] with good results of 57% in Group 1 and 2. We didn't find outcome in the literature on conservative treatment of SONK but hope to have good results for this type of disease.

Appropriate treatment for avascular necrosis is necessary to prevent further deterioration of the joint. If untreated, most patients will experience severe pain and limitation of movement within two years. Although physical therapy cannot cure avascular necrosis, it can slow down the progression of the disease and decrease associated pain. It is suggested that patients with Stage 1 and 2 osteonecrosis could benefit from a physical therapy pro-

gram [52]. Most patients will eventually need surgical treatment, such as core decompression or arthroplasty [53].

Nonoperative treatment involves three main goals [52].

- Relief of symptoms
- Prevention of disease progression
- Improvement of functionality
- Nonoperative treatment begins with patient education and addressing known risk factors, such as smoking and alcohol abuse. In addition, corticosteroids should be avoided [51].

To assist the patient in regaining function and relieve painful symptoms, crutches or other gait aids can be introduced. The physical therapist should instruct the patient on how to properly use these devices. By using crutches (Figure 16), the load that the knee joint bears, will be decreased. This weight-bearing restriction is an important conservative treatment. In literature, it is considered that weight-bearing restriction as a stand alone therapy is insufficient in preventing disease progression, but it's a reasonable treatment option when combined with pharmacological agents or surgery [54]. Physical therapy treatment focuses on exercises to maintain joint mobility and strengthen the muscles around the affected joint. During physical therapy, excessive compressive and shear forces on the joint should be avoided. The outcome depends on the lesion's



Figure 16: Walking with Crutches.

size and stage at the initiation of the treatment.

In order to maintain joint mobility, both passive and active exercises should be initiated. Passive exercises contain passive movements of the hip and stretching exercises. Active exercises consist of 3 dimensional motions of the knee joint and can be applied during standing, sitting on a chair or while lying down. In the next stage, strengthening exercises are added. These exercises will focus on the muscles of the hip and thigh but will also include exercises for the core area as they play a large supporting role. To improve functionality it is important to implement endurance training and coordination training in a more advanced stage of the therapy. Endurance can be trained by walking on a treadmill or cycling on a home trainer. To improve coordination, walking exercises with increased complexity and balancing exercises can be adopted in physical therapy sessions [55]. Physical therapy after surgery is also a key component for recovery. It starts immediately the day after surgery. They prepare the patients for discharge by showing them how to do their everyday activities like getting in and out of bed and walking with a walker or crutches [56].

In a more advanced stage of the therapy, the therapist instructs the patient on exercises to strengthen their muscles, improve range of motion, work on balance and gait speed. The patient specifically learns how to move, while maintaining hip precautions. The rehabilitation protocol is combined with a home exercise program. Below is an example of a training program in an advanced phase of recovery [57].

Biphosphonate

Discussion By definition, osteonecrosis means local death of osteocytes, osteocytes, hematopoietic progenitor cells, and adipose marrow, and it is commonly attributed to bone marrow pressure [58] or due to an occult subchondral fracture [59]. Although the cells in the necrotic lesion die, the inorganic bone matrix is essentially unaffected and the bone initially retains its load-bearing capacity [60]. Regardless of the cause, blood vessels develop into the necrotic bone from the surrounding spine and osteoclasts are introduced into the lesion to destroy the necrotic bone matrix. A remodeling process begins, with concomitant bone resorption and bone formation. The mechanical strength of regenerated bone may be temporarily reduced, either by resorption of necrotic bone or by fatigue fractures in unregenerate necrotic bone.

If necrosis occurs in the subchondral load-bearing bone, the ultimate result may be partial joint collapse and secondary osteoarthritis [51,62]. In a previous study of 40 patients at our hospital [63] with a series of similar but untreated knee osteonecrosis, the patients had a higher Lotke index. had worse outcomes with a higher frequency of articular surface collapse and subsequent

surgery than those patients in the present series treated. Only 10 of 40 patients had a good outcome in the original study. with complete radiographic recovery, compared with 10 of 17 patients in the present series ($p = 0.03$, Fisher's exact test). Eight of 40 treated patients received considered intermediate due to the development of osteoarthritis, but had minor symptoms compared with the present study, in which 4 of 17 bisphosphonate-treated patients had mild osteoarthritis. acute attacks appeared to be less with bisphosphonates. 22 of 40 patients were considered to have failed due to osteoarthritis and major symptoms.

Of these patients, 13 patients underwent osteotomy, which compression or prosthetic fitting. In the present series, 2 of 17 patents failed because of the need for surgery ($p = 0.2$, Fisher's exact test). It is worth noting that none of these patients were able to complete the planned minimum 6 months of bisphosphonate treatment, and they were the only patients who failed to do so. Although surgery was not performed, another patient in the bisphosphonate treatment group was considered a failure because he had severe osteoarthritis (Ahlbäck grade 3) and collapse of the joint surface. In the original series, lesion size was found to be associated with poorer outcome but in the current series no such association was found. In our series, lesions larger than the cutoff point in the Lotke index healed 30% without osteoarthritis, but lesions smaller in size also collapsed. Additionally, the time from symptom onset to treatment initiation did not appear to be related to outcome. In the only randomized trial using bisphosphonates in osteonecrosis (of the hip) [62], a fixed treatment duration of 6 months was set. In our study, treatment was continued until the central region began to increase in radiographic density, indicating osteoblastic activity in the center, and mechanical strength was hypothetically regained.

It is not known how long the treatment will last. In conclusion, the outcome for our patients in the present series appears to be better than that in a previous series of untreated patients [62] and calls for a randomized study. prospective study to determine whether bisphosphonates have the potential to alter outcomes. Perhaps partial weight bearing for long periods of time would be effective, or perhaps both is needed. In the future, other drugs may also be used such as RANK ligand antibodies-which, in addition to being shorter-acting, have also been shown to be more effective than bisphosphonates in experimentally induced osteonecrosis [64].

Risk factors

It's not always known what causes anemia, but have identified several risk factors that make someone more susceptible to osteonecrosis.

- **Injury:** Knee injuries such as stress fractures or dislocations combined with certain types of trauma to the knee, can damage blood vessels and reduce blood flow to the affected bone.
- **Oral corticosteroids:** Many diseases such as asthma and rheumatoid arthritis are treated with oral steroid medications. Although it is not known exactly why these drugs may lead to osteonecrosis, research suggests there is a link between the disease and long-term steroid use. Steroid-induced osteonecrosis frequently affects many joints in the body.
- **Medical conditions:** Osteonecrosis of the knee is associated with medical conditions, such as obesity, sickle cell anemia, and lupus. Osteonecrosis is also commonly seen in HIV-infected patients due to the use of HIV medications.
- **Implant:** Organ transplantation, especially kidney transplantation, is associated with osteonecrosis.
- **Excessive alcohol use:** Consuming too much alcohol over time can cause fat to build up in the blood vessels as well as increased cortisone levels, leading to reduced blood supply to the bones.

Regardless of the cause, if osteonecrosis is not identified and treated early, it can develop into severe osteoarthritis.

Conclusion

Because osteonecrosis of the knee is a relatively rare disease, there is little literature on treatment. There are no prospective, randomized trials comparing treatment options. Most investigators recommend nonsurgical treatment for small, asymptomatic lesions. Core decompression may provide pain relief if performed before collapse, but it is unclear whether it alters the natural course of the disease. Prosthetic replacement remains the most predictable way to treat progressive disease, however, compared with osteoarthritis, the complication rate may be higher and the ultimate success rate somewhat lower.

Limitation

This study has limitations as a retrospective study and a small sample size. The patient is still in the treatment phase and has not yet had a final diagnosis of the disease. Larger data and longer follow-up periods are needed. It is necessary to compare the results of treatment methods, especially to compare the results between surgical and non-surgical treatments.

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