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Review Article

AN OVERVIEW ON OSTEONECROSIS AND TOTAL HIP ARTHROPLASTY: THE OPTIMAL TREATMENT MEASURE FOR ADVANCED STAGE OSTEONECROSIS POST JOINT PRESERVING PROCEDURES

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ABSTRACT

Osteonecrosis, also known as Avascular necrosis of the hip is not a specific disease but a final common pathway of various pathological processes leading to vascular impairment of the femoral head. A number of factors including hip fractures, high dose corticosteroid administration and alcohol abuse have been implicated in the process. Total hip arthroplasty, or surgical replacement of the hip joint with an artificial prosthesis, is a reconstructive procedure that has improved the management of Osteonecrosis. In this review we briefly summarize Osteonecrosis, its etiology, staging's and the evolution of total hip arthroplasty, the design and development of prosthetic hip components, and the possible complications of total hip arthroplasty are also discussed.

Keywords: * Osteonecosis/Avascular necrosis (AVN) * Osteonecrosis of femora head (ONFH) *Total hip replacement/Arthroplasty (THA)/(THR) *Prosthesis

INTRODUCTION

Osteonecrosis:

Osteonecrosis also known as avascular necrosis (AVN) is the death of bony tissue from causes other than infection usually adjacent to the bone surface caused by loss of blood supply as a result of trauma or any another event. The lack of blood supply may cause the surface of the bone to collapse, and arthritis will result. The femoral head is the commonest site of avascular necrosis as its blood supply renders it vulnerable to ischemia. Multiple hypothesis remain to explain the development of AVN, which include ischemia, direct cellular toxicity and altered differentiation of mesenchymal stem cells.[1] Most recently idiopathic AVN has been linked to anatomical abnormalities including decreased femoral neck angle, increased femoral anteversion, reduced centre edge angle and reduced acetabular anteversion. Atraumatic osteonecrosis in adults most commonly presents in the hip. The specific etiology and pathogenesis is often uncertain.[2]

Anatomy:

The hip is one of the body's largest joint. It is a ball and socket joint. The socket is formed by the acetabulum, which is part of the pelvis bone. The ball is the femoral head, which is the upper end of the femur. The bone surface of the ball and socket are covered with articular cartilage, a smooth tissue that cushions the end of the bones and enables them to move easily. A thin tissue called synovial membrane surrounds the hip joint. In a healthy hip, this membrane makes a small amount of fluid that lubricates the cartilage and eliminates almost all friction during hip movement. Bands of tissues called ligaments connect the ball to the socket and provide stability to the joint. All of the blood supply comes into the ball that forms the hip joint through the neck of the femur (*the femoral neck*), a thinner area of bone that connects the ball to the shaft. If this blood supply is damaged, there is no backup. Damage to the blood supply can cause death of the bone that makes up the ball portion of the femur. Once this occurs, the bone is no longer able to maintain itself. When AVN occurs in the hip joint, the top of the femoral head (the ball portion) collapses and begins to flatten. This occurs because this is where most of the weight is concentrated. The flattening creates a situation where the ball no longer fits perfectly inside the socket. Like two pieces of a mismatched piece of machinery, the joint begins to wear itself out. This leads to osteoarthritis of the hip joint, and pain.

Etiology:

It can be thought of as traumatic or non-traumatic. In non-traumatic cases, it is bilateral in 40% of the cases. Avascular necrosis of bone (AVN) occurs as two main variants, local and systemic. Local AVN is usually caused by trauma or microtrauma where as systemic AVN manifests as epiphyseal necrosis or bone infarction, which is often multifocal.[3].Hip dislocations, hip fractures, and other injuries to the hip can damage the blood vessels and impair circulation to the femoral head. A number of factors may predispose to postfracture hip

necrosis, i.e. the type of fracture, the quality of reduction and union of the fracture .[4, 5] Overconsumption of alcohol over time can cause fatty deposits to form in the blood vessels and can elevate cortisone levels, resulting in a decreased blood supply to the bone. Many diseases, including asthma, rheumatoid arthritis, systemic lupus erythematosus and many dermatological diseases are treated with steroid medications.[6] Corticosteroid-induced osteonecrosis of the femoral head (ONFH) often affects both femoral heads. Such bilateral ONFH cases are generally detected concurrently on magnetic resonance imaging (MRI). On the other hand, in unilateral cases, it is rare that contralateral ONFH is subsequently detected.[7, 8] Recent studies have also shown elevated cryofibrinogen levels in atraumatic osteonecrosis.[9] Another possible mechanism is intraluminal obliteration of blood vessels by microscopic fat emboli, sickle cells, nitrogen bubbles (caisson disease), or focal clotting due to procoagulant abnormalities Osteonecrosis is associated with diseases, including Caisson disease (diver's disease or "the bends"), sickle cell disease, myeloproliferative disorders, Gaucher's disease, systemic lupus erythematosus, Crohn's disease, arterial embolism, thrombosis, and vasculitis. It may be related to enlargement of space-occupying marrow fat cells, which leads to ischemia of the adjacent tissues. Vascular insults and other factors may also be significant. Intramedullary haemorrhage at the silent stage is probably related to the pathogenesis of idiopathic avascular necrosis of the femoral head. [10] Idiopathic osteonecrosis is diagnosed when no other cause can be identified. Other causes include Chandlers disease which is the osteonecrosis of the femoral head of adults. Medial femoral condyle osteonecrosis is most common in women older than 60 years.

Histo-pathological changes:

Grossly necrotic bone, fibrous tissue and subchondral collapse is seen. Early changes involve autolysis of osteocytes and necrotic marrow followed by inflammation with invasion of buds of primitive mesenchymal tissue and capillaries. Newly woven bone is laid down on top of dead trabecular bone, followed by resorption of dead trabeculae and remodeling through “creeping substitution”. The bone is weakest during resorption and remodeling and collapse and fragmentation can occur.

Staging:

Ficat classification

- Stage 0- no pain, normal radiographic finding, abnormal bone scan or MRI findings.
- Stage I- Pain,normal x-ray findings, abnormal bone scan or MRI findings
- Stage IIa - Pain, cysts and/or sclerosis visible on x-ray,abnormal bone scan or MRI findings, without subchondral fracture
- Stage III- Pain, femoral head collapse visible on x-ray, abnormal bone scan on MRI findings, crescent sign (subchondral collapse) and/or step off in contour of subchondral bone

- Stage IV- Pain, acetabular disease with joint space narrowing and arthritis (osteoarthrosis) visible on x-ray, abnormal MRI or bone scan findings.

Steinberg staging system [11]

- Stage 0- Normal or nondiagnostic radiograph, bone scan, and MRI findings
- Stage I- Normal radiograph findings, abnormal bone scan and.or MRI findings
    - -A- Mild <15% of head affected
    - -B- Moderate: 15-300%
    - -C- Severe >30%
- Stage II- Lucent and sclerotic changes in femoral head
    - -A-Mild <15%
    - -B-- Moderate: 15-300%
    - -C- Severe >30%
- Stage III- Subchondral collapse (crescent sign) without flattening
    - -A- Mild <15% of articular surface
    - -B- Moderate: 15-300%
    - -C- Severe >30%
- Stage IV- Flattening of femoral head
    - -A-Mild: <15% of surface of <2mm depression
    - -B- Moderate: 15-30% of surface of 2-4mm depression
    - -C-Severe >Severe:>30%of surface or >4mm depression
- Stage V – Joint narrowing and/or acetabular change
    - -A-Mild:Average of femoral head involvement as in stage IV and estimated acteabular
    - -B-Moderate involvement
    - -C- Severe
- Stage VI- Advanced degenerative changes

International classification of osteonecrosis of femoral head (Association Research Circulation osseus[ARCO])[12]

- Stage 0 – Bone biopsy results consistent with osteonecrosis; other test results normal
- Stage I- positive finding on bone scan, MRI or both
    - -A-<15% involvement of femoral head (MRI)
    - -B- 15-30% involvement
    - -C->30% involvement

- Stage II- Mottled appearance of femoral head, osteoscleorsis, cyst formation, and osteopenia on radiographs; no signs of collapse of femoral head on radiographic or CT study; positive findings on bone scan and MRI; no changes in acetabulum
    - -A-<15% involvement of the femoral head(MRI)
    - -B- 15-30% involvement
    - -C->30%involvement
- Stage III- Presence of crescent sign lesions classified on basis of appearance on AP and Lateral radiographs
    - -A-<15% crescent sign or <2mm depression of femoral head
    - -B-15-30% crescent sign or 2-mm depression
    - -C->30% crescent sign or >4mm depression
- Stage IV – Articular surface flattened; joint space shows narrowing; changes in acetabulum with evidence of osteosclerosis, cyst formation, and marginal osteophytes.

Examination:

Initially, the physical examination findings of AVN may be unrevealing. Abnormal physical findings depend on the location and severity of disease. With progression of AVN of the hip, joint function deteriorates and the patient may walk with a limp. AVN of smaller, non–weight-bearing joints typically does not cause significant disability. Findings may include the following:

- Patients with AVN may have tenderness around the affected bone.
- Both active and passive joint movements may be restricted and painful.
- A neurologic deficit may be present if a nerve is affected (compressed) because of necrosis and compression deformity of affected bones.
- Advanced AVN can result in joint deformity and muscle wasting.

No laboratory test findings specifically suggest or confirm the presence of avascular necrosis (AVN). During the early stages plain radiographs show normal findings and pathologic condition can be detected by other techniques, especially Magnetic Resonance Imaging (MRI)[13] If radiographs are normal or show femoral head lucencies suspicious for osteonecrosis, magnetic resonance imaging (MRI) of the hips without contrast is most appropriate. The ACR advises that MRI is the most sensitive and specific imaging modality for diagnosis and provides optimal evaluation of the likelihood of articular collapse. Involvement of greater than 30-50% of the femoral head, often in the sagittal plane, indicates significantly increased risk of articular collapse.

Treatment:

The treatment of osteonecrosis of the femoral head remains a challenging problem because both hips are commonly affected and, often, patients have systemic illness with persisting underlying disease or risk

factors. One treatment option for stage I (the shape of the joint is unaltered, the *x* ray findings are normal, and the diagnosis is made with magnetic resonance imaging (MRI) only) and for stage II (the shape of the joint is still preserved and the articular surface is intact, but the diagnosis can be done with an *x*-ray examination) is conservative management, usually core decompression. Once collapse has occurred (stage III), arthroplasty is often indicated as the disease has reached the stage of no return because the joint surface is altered as a result of collapse in the necrotic region.[14-16]

Nonsurgical Treatment:

If AVN is caught early, keeping weight off the sore-side foot when standing and walking may be helpful. Patients are shown how to use a walker or crutches to protect the hip. The idea is to permit healing and to prevent further damage to the hip. Patients are shown stretches to avoid a loss of range of motion in the hip. Anti-inflammatory medicine is often used to ease pain. Bisphosphonates are other groups of medications that can be helpful. One particular bisphosphonate (fosamax normally used for the treatment of osteoporosis) has been shown effective in reducing the risk of femoral head collapse in patients with avascular necrosis.[17] In some cases, biophysical treatment modalities are prescribed such as electrical stimulation or extracorporeal shock wave therapy in an attempt to treat early disease by promoting angiogenesis and bone modeling.[18, 19] Sometimes these measures may help delay the need for surgery, but they rarely reverse the problem.

Surgery:

If the femoral head has not begun to collapse, an operation to try to increase the blood supply to the femoral head can be done. Several operations have been designed to do this. Treatment must be directed to relieving symptoms whether done by surgical of non-surgical means.[20]. The choice of surgery is based in preoperative staging. Core decompression and cancellous and cortical bone grafting procedure usually are indicated in Ficat stage IIa or earlier stages. The trapdoor procedure and allograt procedures are indicated for stage IIb or stage III lesions. Osteotomies are used to stage II and stage III disease. Arthrodesis and arthroplasty are utilized primarily for stages III and IV but occasionally are used for Stage I and II.

Decompressing the Femoral Head:

The simplest operation is to drill one or several holes through the femoral neck and into the femoral head, trying to reach the area that lacks blood supply. The drill bores out a plug of bone within the femoral head. This operation is thought to do two things: (1) it creates a channel for new blood vessels to quickly form into the area that lacks blood supply, and (2) it relieves some of the pressure inside the bone of the femoral head. Relieving this pressure seems to help decrease the pain patients experience from AVN.

Decompression is often accompanied by the use of bone grafts with or without growth factors, a procedure designed to stimulate bone growth at the site of the defect. The donated bone comes from the patient (taken from the pelvic bone or lower leg). The bone is crushed up into tiny pieces and applied to the hole or defect caused by the necrotic process.

The decompression operation (with or without bone grafting) is done through a very small incision in the side of the thigh. The surgeon watches on a fluoroscope as a drill is used. (A fluoroscope is a type of X-ray that shows the bones on a TV screen. The surgeon uses the fluoroscope to guide the drill where it needs to go.) This operation is usually done as an outpatient procedure, and the patient will be able to go home with crutches the same day.

Fibular Bone Graft:

A more complicated procedure is to try to increase the blood supply to the femoral head by a vascularized fibular bone graft procedure. This is a tissue transplant procedure in which the graft is taken from the fibula (the thin bone that runs next to the shin bone). The graft is vascularized, meaning it has a blood supply of its own. Because it supports the femoral head, the graft is also referred to as a strut graft. A piece of the small bone is removed from the lower leg (the fibula) along with the blood vessels to the bone. A hole is then drilled through the side of the femur and into the femoral head. The blood vessel is then attached from the fibula to one of the blood vessels around the hip. This creates instant blood flow into the bone graft and into the head of the femur. This operation does two things: (1) it brings blood flow to the femoral head through the bone graft, and (2) the fibular bone graft is strong and keeps the femoral head from collapsing as the bone heals itself. This procedure is an inpatient procedure and will require the patient to stay in the hospital for several days. This is a very complicated operation and is not commonly done. It is not always successful because the blood supply to the graft is fragile and may not form completely.

Rotational Osteotomy:

In cases of small lesions involving less than one-third of the surface of the femoral head, rotational osteotomy has been very successful. The procedure involves making a cut through the bone and turning the head of the femur so that the necrotic bone is no longer bearing any weight

Hemiarthroplasty:

Hemi arthroscopy may be:

- Unipolar hemiarthroplasty - replacement of the femoral head and neck.

- Bipolar hemiarthroplasty - replacement of the femoral head and neck plus an addition of an acetabular cup that is not attached to the pelvis (i.e. fits into the existing acetabular cup).
- Resurfacing hemiarthroplasty - replacement of the surface of the femoral head.

A number of surgical approaches to the hip joint are used, each with different advantages and disadvantages. The most commonly used approaches include the direct anterior, direct lateral and posterior approaches[21]

Total hip artroplasty (replacement): (THA/THR):

Total hip arthroplasty (THA) is one of the most successful and effective prosthetic replacement procedures performed today. It is a cost-effective surgical procedure undertaken to relieve pain and restore function to the arthritic hip joint. More than 1 million arthroplasties are done every year worldwide, and this number is projected to double within the next two decades.[22, 23] About 250000 patients are operated on annually for hip arthroplasty in the USA alone. [1] Two thirds of these are performed in patients over 65 years of age.[24] Ninety to ninety five per cent of patients can expect to have their total hip replacement functioning at 10 years,[24] and in 85% they will still be functioning at 20 years.[25]For patients with hip pain due to a variety of conditions, THA can relieve pain, can restore function, and can improve quality of life. Sir John Charnley, a British orthopedic surgeon, developed the fundamental principles of the artificial hip and is credited as the father of THA. He designed a hip prosthesis in the mid to late 1960s that still sees use today. Since 1960, improvements in joint replacement techniques and technology have greatly increased the effectiveness of total hip replacement. In total hip replacement, the damaged bone and cartilage is removed and replaced with prosthetic components. Rest pain, pain with activity and functional limitations are the most important criteria for THA[26] Arthroplasty results in a lower rate of subsequent reoperation at mid- and long-term follow up, and better mid-term functional recovery.[27]**.** Improvement in physical activity and quality of life solely depends on the patient and his families efforts towards rehabilitation and changes in sedentary lifestyle should be made.[28]

Prosthesis design:

Bearing surfaces are the surfaces which articulate in the prosthetic joint. The femoral head and the acetabular liner can be used in different combinations. These will give different appearance on radiograph depending on the configuration. Options for bearing surfaces include:

- metal on metal (metal head and a metal acetabular component)
- metal on polyethylene (metal head with a polyethylene acetabular liner)
- ceramic on ceramic (ceramic head with a ceramic acetabular liner)

Femoral component or stem: This refers to the prosthesis which is implanted into the femur. They can be described by length, taper and presence of collar. Attached to the femoral component is the neck and head which in most prostheses can be altered in size to create a stable joint.

Acetabular component: main variations in acetabular component on radiograph are related to fixation method and position.

Prosthesis position:

Stability of the prosthetic joint is the primary goal in choosing the position of implanted prosthesis. Multiple patient and surgical factors can alter the ideal position of the components. The overall position of the prosthetic hip replacement should ideally be in a mild degree of valgus, but should not exceed 140 degrees. Excessive valgus creates strain on the knee, can lengthen the leg and can lead to superior dislocation with adduction of the leg. A hip positioned in varus can cause increased loosening and stem failure and can also lead to dislocation. The acetabular component should be positioned in 20 degrees of anteversion with an inclination of 45 degrees. Femoral component position should be in 10-15degrees of anteversion. Excessive anteversion or retroversion can lead to dislocation.

Prosthesis fixation:

Femoral stem fixation can be either cemented or noncemented (biological) fixation. There is a tendency to use noncemented femoral stems in younger patients, due to higher reported rates of loosening of cemented stems in long term follow-up. Most common fixation for the acetabular component is noncemented.

Biologic fixation uses either porous coated metallic surface to stimulate bone in growth or grit blasted surface to allow bone on growth. Prosthesis can also be coated in hydroxyapatite, which is an osteoconductive agent.

Grading of cement technique on radiograph for femoral stem:

- **A:** medullary canal completely filled with cement
- **B:** a slight radiolucency exists at the bone cement interface
- **C:** radiolucency > 50% of bone cement interface
- **D:** radiolucency involving 100% of the interface between bone and cement in any projection, including absence of cement distal to the stem tip

Gruen zones are around the femoral stem from zone 1-7 from the proximal lateral aspect anticlockwise with zone 4 distal to the tip.

Charnley zones are around the acetabular component. There are 3 zones indicating the superior, middle and inferior third of the acetabulum.[29]

Surgical approach for total hip arthroplasty:

Most common surgical approach for total hip arthroplasty is a posterior approach to the hip. Skin incision is made 10-15cm centered on the posterior aspect of the greater trochanter. Dissection includes splitting fascia lata and gluteus maximus in line with its fibres. This will uncover the short external rotators, which are dissected off the femur and retracted back over the sciatic nerve to protect the nerve throughout the operation. A capsulotomy is then performed and the hip dislocated. The damaged femoral head is removed and replaced with a metal stem that is placed into the hollow center of the femur. The femora stem may be either cemented or press fit into the bone. A metal or ceramic ball is placed on the upper part of the stem. This ball replaces the damaged femoral head that was removed. The damaged cartilage of the acetabulum is removed and replaced with a metal socket. Screws or cement are sometimes used to hold the socket in place. A plastic, ceramic, or metal spacer is inserted between the new ball and the socket to allow for smooth gliding surface

Protocols post-surgery:

Good analgesia after total hip arthroplasty/replacement (THA) is an important factor in postoperative recovery.[30]

Total hip precautions after surgery should be followed for 3 months and include:

- Avoid hip flexion past 90 degrees
- Avoid internal rotation of the lower extremity
- Avoid crossing the midline of the body
- Avoid sitting on low, soft surfaces
- Use raised toilet seats for 6 weeks-3 months (discussed with the surgeon at the 6 week visit)

Patient will be weight bearing as tolerated with a rolling walking or other piece of medical equipment as seen fit by the surgeon. The patient may progress from a walker to a straight cane when they can demonstrate equal weight distribution, adequate balance and limited Tredelenburg gait or limp. Patients may feel uneven in their gait; this is typically due to an adductor spasm and resolves over time. Driving is prohibited for the first 6 weeks or until off pain medication and walking without a cane. TED hose should be worn during walking hours and removed at night time. They need to be worn for 6 weeks post operatively. Sutures will be removed 10-14 days post-operatively. The patient may shower with a water proof bandage but a shower stool or tub bench is recommended for the first 3 months. No soaking in the bathtub. Do not use weight machines until at least 3 months after your surgery. Patients will attend physical therapy 2-3x/week for the first 6 weeks or until patient returns for follow up. Three distinct but interrelated processes constitute the physical,

psychological, and social recovery process: reclaiming physical ability, reestablishing roles and relationships, and refocusing self. Intervening conditions affecting the recovery process include co morbid conditions, the personal outlook of the patient, patients' relationships, and social support. The recovery process can lead to changes in personal and social functioning that patients might not anticipate. Awareness of potential changes will inform patient education and enable clinicians to develop strategies that facilitate THA patients' return to health.[31-33]

Possible Complications of Surgery:

Most complications in total hip arthroplasty are infrequent and can be prevented or treated readily if anticipated and recognized. Complications associated with any major surgical procedure, including those related to anesthesia, co morbid medical conditions, medications, and allergic reactions, may also occur. Potential complications known to occur during or following total hip arthroplasty can be categorized as intraoperative or postoperative. The complication rate following hip replacement surgery is low. Serious complications, such as joint infection, occur in less than 2% of patients. Major medical complications, such as heart attack or stroke, occur even less frequently. However, chronic illnesses may increase the potential for complications. Although uncommon, when these complications occur they can prolong or limit full recovery. Prolonged physical impairments in range of movement, postural stability and walking speed are commonly reported following total hip arthroplasty (THA)[34]

Intraoperative complication:

Fracture

The incidence of fracture during total hip arthroplasty ranges from 0.1 to 1 percent for cemented components and from 3 to 18 percent for uncemented components. Most intraoperative fractures occur on the femoral side during stem insertion. Minor fractures may be left alone or treated with wires or cables. More extensive fractures may require more complex solutions including revision of components, bone grafting, and/or supplementary hardware (e.g., plates and screws). Factors that increase the risk of fracture during primary arthroplasty include female sex, osteopenia, inflammatory arthropathies, and cementless stem fixation. The use of longer-stem cementless implants during revision surgery is an additional risk factor for fracture.[35]

Nerve and vessel injury

The incidence of nerve injury in primary total hip arthroplasty ranges from 0 to 3 percent. Injury to the sciatic nerve is most common, but the femoral, obturator, and superior gluteal nerves may also be injured. The peroneal division of the sciatic nerve is more susceptible to injury than the tibial division. The incidence of

vascular injuries associated with total hip arthroplasty/replacement (THA) is low. However, several vascular structures are at risk of injury within the pelvis. These include the external iliac, femoral, and obturator vessels. Both reaming of the acetabulum and drilling of the acetabular screw holes may place these structures at risk.[36]

Postoperative complication:

Infection

Infection may occur superficially in the wound or deep around the prosthesis. It may happen while in the hospital or at home. It may even occur year's later. Minor infections of the wound are generally treated with antibiotics. Major or deep infections may require more surgery and removal of the prosthesis. Any infection in your body can spread to your joint replacement.

Blood Clots

Compared with other surgical procedures, joint replacement is associated with a high risk of deep venous thrombosis (DVT) and pulmonary embolism (PE). Without prophylaxis, the incidence of DVT after total knee arthroplasty (TKA) is 50-84%, and after total hip arthroplasty (THA), 47-64%. With prophylaxis that uses anticoagulation therapies, the incidence is reduced 22-57% after TKA and 6-24% after THA. Blood clots may form in one of the deep veins of the body. While blood clots can occur in any deep vein, they most commonly form in the veins of the pelvis, calf, or thigh. A prevention program which may include blood thinning medications, support hose, inflatable leg coverings, ankle pump exercises, and early mobilization are planned. Venous thromboembolism (VTE) prophylaxis is recommended for all patients undergoing total hip arthroplasty. The selection of an appropriate prophylaxis regimen represents a balance between efficacy and safety.[37]

Leg-length Inequality

Sometimes after a hip replacement, one leg may feel longer or shorter than the other. An orthopedic surgeon will make every effort to make leg lengths even, but may lengthen or shorten your leg slightly in order to maximize the stability and biomechanics of the hip. Some patients may feel more comfortable with a shoe lift after surgery.

Dislocation

Hip implant dislocation occurs when the ball comes out of the socket. The risk for dislocation is greatest in the first few months after surgery while the tissues are healing although dislocation is uncommon. If the ball does come out of the socket, a closed reduction usually can put it back into place without the need

for more surgery. In situations in which the hip continues to dislocate, further surgery may be necessary. When setting a hip prosthesis, the anteversion angle of the cup is the most important factor to be taken into consideration, but it is also essential to adjust the antetorsion angle of the stem to match the shape of individual bones to create a more stable hip joint. Finally, the tension in the soft tissues, should also be borne in mind to avoid postoperative dislocation after total hip arthroplasty.[38, 39]

Loosening and Implant Wear

Over years, the hip prosthesis may wear out or loosen. This is most often due to everyday activity. It can also result from a biologic thinning of the bone called osteolysis. If loosening is painful, a second surgery called a revision may be necessary.

Immunological response:

To date, THA has been highly successful in relieving pain and restoring the functionality of patients' joints, and has significantly improved their quality of life. However, these implants are expected to eventually fail after 15–25 years in situ due to slow progressive inflammatory responses at the bone-implant interface. Such inflammatory responses are primarily mediated by immune cells such as macrophages, triggered by implant wear particles. As a result, aseptic loosening is the main cause for revision surgery over the mid and long-term and is responsible for more than 70% of hip revisions.[40]

CONCLUSION

Osteonecrosis is a bone disease that results from the loss of blood supply to the bone. Without blood, the bone tissue dies. This causes the bone to collapse and may also cause the joints that surround the bone to collapse. Osteonecrosis presents with pain and limitation in physical activity. Arthroplasty has an excellent prognosis and is one of the most successful advancement of surgical techniques used to treat osteonecrosis. THA can be cost saving or cost effective. Patients' needs and characteristics should be carefully assessed while providing post-operative care and support.

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