Core decompression in treatment of aavascular necrosis of head of femur

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English summaryOsteonecrosis of the femoral head is a pathologic process resulting from the death of living elements of bone. It is not a specific disease but rather the end result of various conditions, ultimately, with impairment of blood supply to the femoral head. Mechanical and biological factors contribute toosteonecrosis.Osteonecrosis may affect young individuals, in whom the incidence of bilateral disease is surprisingly high. Osteonecrosis of the hip may progress through various stages and terminate with degenerative arthritis of the hip joint. This process may take 3 to 5 years from its onset or may neverprogress past an early stage.Many causes contribute to osteonecrosis of the femoral head. Most cases of osteonecrosis of the femoral head are caused by trauma. Transcervical fractures, complete hip dislocations, and compression fractures of the femoral head are common. Many drugs and diseases have been linked to osteonecrosis of the femoral head. There is an increasing incidence of femoral head osteonecrosis associated with steroid intake for the treatment of medical illnesses (18% to 57%) or transplantation (13%). The association of alcoholism with osteonecrosis of the femoral head is well known and is thought to be caused by fat emboli. Bone changes owing to a hemoglobinopathy (particularly sickle cell disease) are caused by mechanical interruption to the blood supply of the superior weight-bearing portion of the femoral head. (Plancher & Razi 1997)193Several studies have shown that the major cause of osteonecrosis of the femoral head is an increase in the intraosseous pressure of the proximal femur. It is hypothesized that because the femoral head is a closed compartment, any ischemic episode produces venous obstruction that results in increased intraosseous pressure. Similar to a compartment syndrome, this increase in pressure produces a cycle of progressive ischemia that eventuallyleads to bone death.The onset of symptoms is usually insidious, but an acute attack of pain may happen with minor traumatic injuries. Pain is almost always unilateral; however, in approximately 55% of cases, the opposite hip becomes involved within 2 years. Range of motion is well preserved in the beginning of the disease but gradually deteriorates. Decreased motion and limp develop asdegenerative changes progress.Clinical and pathologic changes may not appear until months or years after the insult; however, histologic and metabolic changes occur within hours. These two types of changes are divided into the early and late stages ofosteonecrosis.In the early stage, cells lose their viability 12 hours after a vascular insult. Histologic changes are not evident until 48 hours later, and

radiologic changes are not seen before 2 months. The death of bony trabeculae is not evident until 2 or sometimes 4 weeks post insult. Because of its nourishment from the synovial fluid, no changes occur in articular cartilage.Ingrowths of vascular fibrous tissue occurs during the first few weeks as a repair process occurs in the base of the femoral head. (Catto 1965) The earliest sign of repair is an increase in density found on radiography in the194superolateral area of the femoral head. Thickened trabeculae are noted, and, on occasion, diffuse osteopenia may be the first radiographic abnormality. In the late pathologic stage, repair is blocked because of the advancement of fibrous tissue, which becomes a dense avascular wall. Gross segmental collapse leads to increased density of mechanically compressed bone. Areas of radiolucency evolve within the head as the result of a reparative process. Frequently, a radiolucent line or crescent sign is seen beneath the subchondral bone of the superior portion of the femoral head in the anteroposterior or lateral view. This is a result of the collapse of dead cancellous bone that separates from the articular cartilage. Ficat and Arlet1980 defined the appearance of the crescent sign as a transitional stagebetween a spherical and a flattened femoral head. Gradually, flattening ofthe weight-bearing area occurs, and the crescent sign gradually becomesobliterated with a radiograph that reveals dispersed regions of radiolucency.The acetabulum and articular cartilage remain grossly normal until the verylate stage of the disease; however, some surface irregularities and cell injurymay be seen histologically. Changes in the acetabulum, such as sclerosis,cystic degeneration, and osteophyte formation, begin late.Evaluating the degree or stage of involvement in the femoral head is important. Staging the disease determines the prognosis as well as the optimal method of treatment. Ficat and Arlet classification system is one ofthe most popular used.Diagnosis in early stages provides the opportunity to prevent collapse of the femoral head. A thorough history must be taken, including past and present diseases, hip trauma, excessive drug intake, and exposure to sporting activities, such as diving. The complete physical examination should include195an evaluation of the hip with documentation of the range of motion. The radiologic evaluation should include an anteroposterior view of the pelvis with the legs internally rotated and a lateral view of both hips. Frog leg lateral views can be substituted for a shoot through the lateral view if the patient can flex the hip to 90 degrees with some abduction. Early diagnosis is important to prevent segmental collapse and to help select the best treatment modality. Special imaging techniques can be used to confirm the suspected clinical diagnosis when plain films are normal. Bone scanning with technetium-99m diphosphate is a sensitive but not specific test. CT isuseful once structural changes have evolved in the bone. MR imaging is thebest technique for the early diagnosis of osteonecrosis of the femoral head.(Hungerford & Zizk 1978)Osteonecrosis of the femoral head remains a difficult therapeutic problem. The stage of disease at the time of diagnosis determines the best treatment options, although other factors, such as etiology, the duration of symptoms including pain and impediment, age, general health, and unilateral orbilateral involvement, are also important factors.The ideal approach is to preserve rather than to replace the intact femoral head. Nonoperative measures, such as observation, analgesics, and limited weight bearing, may be successful when the

femoral head is minimally involved. nonoperative measures should be recommended for pain relief, when the diagnosis is not confirmed, or for conditions that have not progressed to a point at which there is a need for reconstruction.Core decompression has become a widely used therapeutic procedure for the treatment of osteonecrosis (ON). Ficat realized that the goal of core196decompression was to decrease the high intramedullary pressure in an attempt to relieve pain, improve venous drainage, and a Several different bone-grafting techniques have been advocated in the literature. These include the addition of autogenous or allograft cancellous bone to a core decompression, osteochondral grafts, muscle-pedicle bone grafts, and free vascularized bone grafts with iliac or fibular bone amplify vascularingrowth.Various types of femoral osteotomies have been performed to preserve the femoral head in young individuals with stage III disease by redirecting theforces on the femoral head.Reconstructive measures are preferred once the femoral head has collapsed or acetabulum degenerative changes have occurred.Hip fusion is infrequently recommended for osteonecrosis because of its high failure rate. Patients are not usually satisfied with a fused hip. Consideration should always be given to the opposite hip because there is a high incidence of bilateral involvement. (Plancher & Razi 1997)