CURRENT CONTROVERSIES IN RHEUMATOLOGY

RESPONSE: THE ROLE OF CORE DECOMPRESSION IN THE TREATMENT OF ISCHEMIC NECROSIS OF THE FEMORAL HEAD

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Nontraumatic ischemic necrosis of the femoral head remains a source of much controversy. Nonetheless, it is a disease of major importance to both rheumatologists and orthopedists. Most patients first present to a physician during the advanced stages of the disease. The early stage of the disease does not produce any radiologic abnormalities, so that in those patients who do present early, it is frequently not diagnosed. The disease appears most frequently in young adults, and presentation after the age of 50 is uncommon. The average age of the patients in our series was 38 years. No satisfactory nonsurgical treatment methods have been reported as yet; thus, most patients simply wait for the symptoms to progress and the destruction of the femoral head to become sufficient to justify total hip replacement.

The etiology of this disease is also controversial. All the diverse hypotheses have their adherents. Examples of proposed etiologies are that (a) the cell is sick, attacked by toxic agents such as alcohol and steroids (1,2); (b) the intracellular lipocytes hypertrophy, creating intraosseous hypertension, which produces a compartment syndrome (3–6); or (c) circulating fat globules embolize the microcirculation of the metaphysis, causing osseous infarction (7,8).

In spite of the diversity of these hypotheses and the diversity of the conditions that are associated with an increased incidence of necrosis of the femoral head, the actual presentation and course of the disease are remarkably similar for all patients. In the earliest stage of the disease, radiographic findings are normal. Biopsy at this stage shows marrow necrosis and fibrosis as well as some trabecular death (9). Mottled sclerosis, trabecular hypertrophy, and cysts in the femoral head, usually localized to the anterolateral portion, represent the first radiographic signs. These changes may progress to a well-demarcated, anterolateral wedge-shaped infarct, which is evident just prior to collapse of the femoral head. Many patients present with clinical symptoms at the time of collapse, when a subchondral radiolucency or step-off can be seen. This represents structural failure of the femoral head, and progression is then usually only a matter of months or 1–2 years before joint space narrowing and secondary degenerative changes occur (10,11).

The purpose of this article is to present the author's perspective on the current role of core decompression in the treatment of ischemic necrosis of the femoral head. In order to do that, 3 issues need to be addressed: 1) the scientific rationale of core decompression, 2) the historical background for core decompression, and (3) the sources of the current controversy.

THE SCIENTIFIC RATIONALE OF CORE DECOMPRESSION

This author has previously published several extensive treatises on the pathogenesis of ischemic necrosis of the femoral head (12–14). It is important to distinguish pathogenesis, i.e., the mechanisms occurring in the *development* of disease, from etiology, i.e.,

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the factors that *cause* disease. There is considerable evidence to implicate the compartmental nature of bone and the development of intraosseous hypertension as the principal pathogenetic mechanisms for the progression of this type of necrosis. Intraosseous hypertension has been measured and recorded by numerous investigators (5,15–19). While it might not be present at all stages of the disease in every case, it is present during the earliest stages of the disease and with sufficient frequency to likely play an important *pathogenetic* role.

There can be little doubt as to the compartmental nature of bone. Michelsen is credited with the first observations of pressure relationships in the bone marrow bed (20). The elegant physiologic studies of Wilkes and Visscher confirm the compartmental nature of bone circulation (21). Bone functions as a Starling resistor, consisting of a rigid canister (the cortex) through which pass flexible thin-walled tubes (the blood vessels). The flow in the traversing tubes is inversely proportional to the pressure within the rigid canister and outside the vessels. Any increase in the driving pressure outside the canister will result in a proportional increase in flow, but any pressure within the canister will tend to collapse the tube and proportionally decrease flow. Therefore, any of the extravascular intraosseous tissues, by rapidly increasing their volume, could have a negative effect on bone blood flow through the creation of intraosseous hypertension. These tissues would include the normal hematopoietic elements, intraosseous lipocytes, Gaucher's cells, and even edema fluid. The intraosseous hypertension might be primary, such as that caused by intraosseous lipocyte hypertrophy effected by steroids, or secondary, as in that caused by extravascular edema fluid following a period of ischemia due to another direct primary cause such as microemboli. Certainly, other vascular episodes of ischemia are known to produce secondary compartment syndromes.

If one accepts this compartmental nature of bone, which seems to have been incontrovertibly proved, and if one accepts the potential role of increased intraosseous hypertension in the pathogenesis and progression of ischemic necrosis of the femoral head, then core decompression becomes a logical and rational treatment. As with all compartment syndromes, the success of the decompression will depend upon the magnitude of the original ischemic insult and the degree of cell death caused by the compartment syndrome that has occurred at the time of treatment. If all of the cells have already died, then the decompression will be ineffective, as it is in other compartment syndromes. However, if the disease is in the process of evolution, as opposed to being thoroughly established and in the end stages, core decompression may result either in reversal of the circulatory deficit or in arrest of its progression.

HISTORICAL BACKGROUND OF CORE DECOMPRESSION

Ficat and Arlet began to investigate the puzzling phenomenon of the patient with a painful hip and normal radiographic findings in 1962. In 1968, they published the results of their functional evaluation of bone to define the preradiologic stage of ischemic necrosis of the femoral head (9). This evaluation consisted of the measurement of intraosseous pressure, a stress test performed by injecting 5 ml of saline intraosseously, a venogram of the intraosseous venous drainage, and a biopsy of the bone (9). In the majority of the patients, intramedullary baseline pressures and/or pressures shown by stress test were elevated, and the majority had abnormal venous drainage patterns evident on venography. In 1971, these authors published their results of core decompression performed in 100 patients, whose progress was followed from 1 to 5 years. The results for those with stage I disease (n = 40) were good or excellent in 88% of the patients. The results for those with stage II disease (radiographic changes without collapse; n = 16) were good or excellent in 69% (22).

I became aware of the techniques of Ficat and Arlet during a study tour of France in 1973 and began applying them immediately, with similar, but not quite so good, results. There were significant differences in the patient population that could explain the differences in results; specifically, in my series, nearly 45% of the patients had steroid-associated avascular necrosis, including a particularly high number with systemic lupus erythematosus (SLE), a group that was notably absent from Ficat and Arlet's patient population.

In 1975, the results of my initial experience were reported (23). The term "core decompression" was coined at that time as an alternative to "forage," the term used by Ficat and Arlet, which had assumed negative connotations from its application to the drilling of femoral heads in patients with osteoarthritis of the hip. That procedure provided only a short-term positive benefit.

In the mid-1970s, when the concept of core

decompression was introduced to the English-speaking orthopedic community, there was no satisfactory treatment for ischemic necrosis of the femoral head. The procedure was applied enthusiastically to all patients with the disease who did not have massive femoral head collapse and joint line destruction. In 1978, Dr. T. Zizic and I published the results of our experience in treating patients with alcohol-associated femoral head necrosis (24). Of 13 patients with stage I and stage II disease, only 1 had radiologically evident progression, and none had had further surgery during an average followup period of 2 years. However, 18 of 25 patients with stage III disease had progressed radiologically, and 8 of these had further surgery. Of the 17 patients who had not undergone further surgery, only 8 remained asymptomatic. In 1980, we also presented the results a study of core decompression in a group of patients with SLE (25). The reports of 1975, 1978, and 1980 stimulated a degree of enthusiasm for the treatment of ischemic necrosis of the femoral head by core decompression.

In 1983, Dr. P. Ficat, who was the foreign guest speaker for the Eleventh Open Scientific Meeting of the Hip Society, presented the results of a long-term study of core decompression, which involved a minimum followup period of 5 years and maximum follow-up period of 18 years (mean 9.6 years) (26). He reported good or excellent clinical results in 93.9% of the patients with stage I disease and the continuation of virtually normal radiographic findings in 86.5% of that group (n = 82). Among those with stage II disease, good and excellent clinical results were found in 82.3%, and radiologically excellent results occurred in 66.6% (n = 51). Ficat's report of another study in a subsequent publication showed similar results (27).

SOURCES OF THE CURRENT CONTROVERSY

In 1986, Camp and Colwell reported the results of 40 core decompressions performed on 31 patients by 13 orthopedic surgeons (17). The group experienced a 15% rate of major complications, which included 1 femoral head blowout, 1 femoral head perforation, and 4 proximal femoral fractures through the core decompression site. The patients were divided into biopsy-positive (n = 25) and biopsy-negative (n = 15) groups. The authors considered the result of the biopsy to constitute the establishment of the diagnosis, despite the fact that 6 of the 15 patients in the biopsy-negative group had radiographic findings typical of avascular necrosis, and 9 of the 15 had characteristic radiologic changes at the time of latest follow-up visit. In the biopsy-positive group, 11 of 18 patients who underwent the procedure in the precollapse stage had clinical or radiographic progression. Because these authors used 1 single item from the diagnostic armamentarium as the criterion for diagnosis, it is very difficult to interpret the results.

It is clear that those patients who had radiographic findings typical of avascular necrosis, or whose results became consistent with such necrosis during the followup period probably represent a sampling error rather than the absence of the disease. However, it is possible that those patients who had negative radiographic results preoperatively and who continued to have negative results postoperatively, despite the fact that their symptoms were unchanged by the surgical procedure, might not have had avascular necrosis at all. Camp and Colwell concluded that core decompression was "an ineffective procedure with significant morbidity because of the complication of fracture" (17). I do not agree with this conclusion because of the way their surgery was done and the way their results were analyzed. A detailed critique of their article was published as a letter to the editor of that journal in 1988 (28).

In 1987, Warner et al reported their experience with core decompression in patients at Brigham and Women's Hospital in Boston (18). They cored 39 hips in 25 patients, most of whom had steroid-associated necrosis. After a minimum of 2 years followup, the disease had progressed in only 2 of 12 hips with stage I disease (17%), whereas 7 of 12 hips with stage IIA disease (predominantly sclerotic), 4 of 4 hips with stage IIB disease, and 9 of 11 hips with stage III disease had radiographically and/or clinically evident progression. The authors' classification of stage IIB disease, which included flattening and a crescent sign, would correspond to Ficat's stage III. They concluded that core decompression was indicated for stages 0, I, and IIA disease.

In 1988, Tooke et al reported the results of their experience with core decompression in patients at the University of California at Los Angeles (19). After an average 3-year followup period, none of the stage I lesions (n = 10) had progressed, whereas 11 of the 26 stage II lesions had progressed to stage III after an average of 14 months following the core decompression. Four of 9 patients with stage III disease did not have radiographic or clinical deterioration during an average 4-year followup period. On the basis of their experience, the authors concluded that "core decom-

pression is recommended for all stage I and II cases and for stage III hips in which the patient will not accept a major joint replacement reconstruction" (19).

Finally, Hopson and Siverhus reported only a 40% success rate after an average followup period of 39 months for 17 patients with stage I or II ischemic necrosis of the femoral head (29). All but 2 of their patients had steroid-associated disease. One of their 17 patients experienced a fracture through the core tract. No mention of the postoperative management is made in the report.

CURRENT PERSPECTIVE ON CORE DECOMPRESSION

Core decompression, in my view, remains a safe and effective surgical technique for the treatment of stage I and stage II ischemic necrosis of the femoral head. Even those investigators reporting or interpreting their results in a negative light (~40% success rate) have a significantly higher success rate than that reported for patients in whom the necrosis is not treated (10,11,30). A femoral head blowout like that reported by Camp and Colwell comes from not clearing the coring device. The specimen becomes wedged in the core biopsy trocar, converting the trocar from a hollow core tube to a battering ram. This is an avoidable technical error. Likewise, femoral perforation comes from not using adequate radiographic controls, another avoidable error. We have noted only 2 femoral fractures in 15 years of experience using this procedure on more than 200 patients. Both of these instances involved patients who slipped and fell in the early postoperative period while on crutches. We have observed no spontaneous fractures through the core tract.

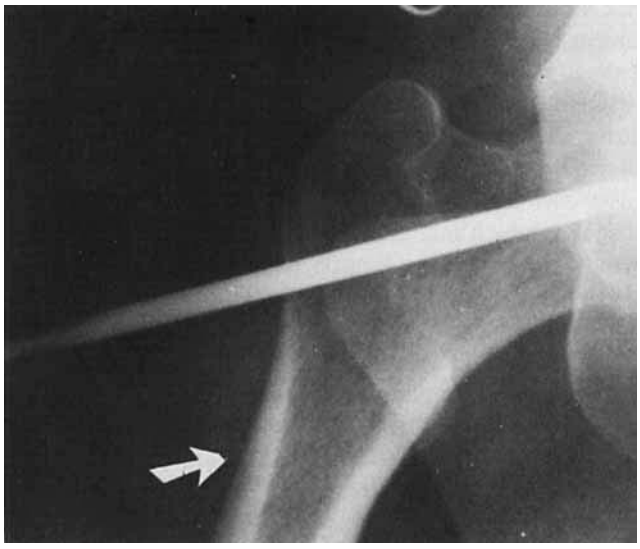

Figure 1. Intraoperative radiograph showing the correct position of the lateral cortical entry hole for core decompression through the thin cortex at the trochanteric flare. An entry window through the thick cortex more distally (**arrow**), as recommended by Camp and Colwell (17), may predispose to fracture.

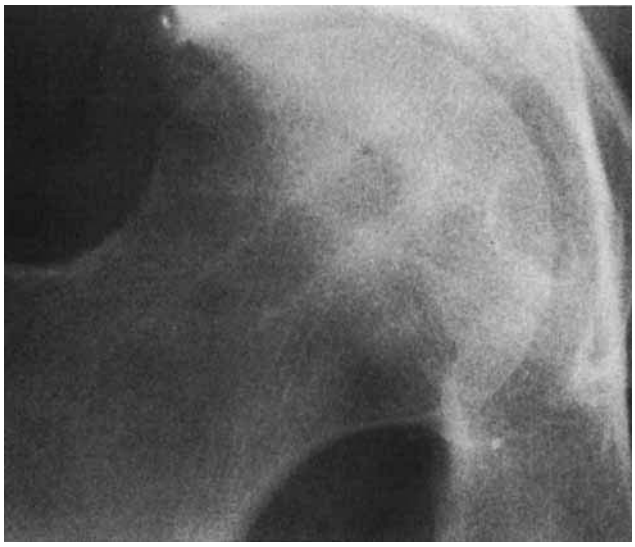

Figure 2. A 14-year followup radiograph of a 43-year-old man with alcohol-associated ischemic necrosis of the femoral head. He has remained asymptomatic. A biopsy indicated the presence of dead bone. During the same followup period, he had 1 primary and 2 revision total hip arthroplastic procedures on the contralateral side.

One possible explanation for the difference between our fracture rate and those reported by Camp and Colwell (10%) and Hobson and Siverhus (6%) is the location of the lateral cortical entry hole. The drawing of the surgical technique in the article by Camp and Colwell shows the entry hole through the thick lateral cortex distal to the lesser trochanter, and the radiograph of 1 of their patients who sustained a fracture through the core tract shows the same distal entry hole. We, like Ficat and Arlet, recommend that the entry hole be placed at the base of the trochanter, where the lateral cortex is relatively thin and less subject to the bending stresses from a more distally placed entry hole (Figure 1). In addition, we insist that the patient use 2 crutches and sustain minimal weight-bearing for 6 weeks. However, despite the risk, we have had many patients who had bilateral core decompressions or core decompressions associated with femoral osteotomy or total hip replacement on the

contralateral side, and none of these patients has experienced fracture.

Most of the American reports of this disease noted a high percentage of patients with steroid-associated avascular necrosis. This type of necrosis may progress to stage III without the occurrence of a great deal of reactive bony change. Steroid-associated avascular necrosis is extremely unusual in France and did not constitute a significant part of the series reported by Ficat and Arlet. Part of the problem in the past, in assessing stage I and early stage II ischemic necrosis, is that there has been no reliable mechanism for determining the extent of the lesion or the status of the living moiety of bone and bone marrow. It is likely that stage I includes a wide variety of degrees of involvement. With the more widespread use of magnetic resonance imaging (MRI), it may be possible to distinguish those stage I cases that have a uniformly high success rate from core decompression from those cases that have a uniformly low success rate. Certainly, we have seen stage I bone biopsy samples that showed that trabecular death had been minimal, and other cases that were radiologically identical in which bone death had been extensive. It is therefore, perhaps, not surprising that the disease does not behave in the same way in all cases.

Before the advent of MRI, establishment of the diagnosis of avascular necrosis in the preradiologic stage depended, to a certain extent, on the evaluation of the bone biopsy. In fact, we have reported 5 cases in which the patient was suspected to have ischemic necrosis of the femoral head and actually had a primary or secondary bone tumor (31). Therefore, in the past, bone biopsy was an important part of the diagnostic procedure. With the advent of MRI, biopsy became much less important, and it should now be possible to carry out a randomized study of the effectiveness of core decompression in the preradiologic and precollapse stages of ischemic necrosis. Moreover, MRI is identifying patients as positive who are radiologically and symptomatically negative for the disease. These patients are usually detected at the time of presentation for symptomatic and more advanced disease on the contralateral side. This group, too, would form a very valuable patient subset for determining the capacity of core decompression to prevent the development of symptomatic and structurally damaging ischemic necrosis.

Our current recommendation is that unilateral or bilateral core decompressions be carried out in all patients presenting with the precollapse stages of ischemic necrosis of the femoral head (Figure 2). For those with radiologically advanced stage II lesions, this procedure is often performed in conjunction with a repositioning femoral intertrochanteric osteotomy. For patients with unilateral stage III disease who are willing to accept significantly reduced activities and who have control of their physical environment and wish to avoid total hip arthroplasty for a period of time, core decompression can also be recommended. The patient, however, must understand that the procedure is done for palliation and not for cure, and its purpose is to postpone immediate total hip arthroplasty. For stage III disease, the procedure is most effective in reducing night and rest pain and is less effective in producing activity-related pain.

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