Core Decompression for Juvenile Osteonecrosis

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KEYWORDS

- Core decompression • Avascular necrosis
- Legg-Calvé-Perthes disease • Femoral head
- Osteonecrosis

When Legg-Calvé-Perthes disease (LCPD) develops at age 12 years or older, the results are generally poor, with or without current methods of treatment.[1–5] These juvenile patients are more likely to become Stulberg class V even when the necrotic segment is less than 50% of the femoral head.[1,4] Joseph and colleagues[1] reported 62 patients aged 12 years or older at onset. In their series, neither the Catterall group nor the Herring grade correlated with final outcome and none benefited from containment treatment.

We propose the term idiopathic juvenile osteonecrosis to distinguish this age group from younger children with Perthes disease and from adults with osteonecrosis of the femoral head. One difference is that the femoral head in the older pediatric age group does not reconstitute as rapidly as in younger children with LCPD.[6] Another difference between juvenile osteonecrosis and younger children with Perthes disease it that the older age group has less remodeling capacity of the femoral head or acetabulum.[1,2,5,7]

Compared with adults, juvenile osteonecrosis may have a slightly more favorable prognosis. In adults, the rate of bone resorption exceeds the rate of bone deposition.[8] This difference causes a weakness in the structural integrity of the bone followed by subchondral fracture and collapse.[9] Ficat[10] identified that stage III osteonecrosis in adults is associated with a break in the articular surface. However, there is a transitional stage in which a subchondral fracture may be present without segmental collapse.[10] Young children have thick articular cartilage that may be more resistant to deformation and disruption than that of older children. Adolescents may also tolerate some subsidence of the necrotic fragment without disruption of the articular surface. Restoring support to the joint surface before cartilage fracture may preserve joint function.[11]

The treatment rationale of the femoral head should be tailored to address these differences between adult and childhood osteonecrosis of the femoral head. Joseph and colleagues[1] suggested that an alternative method of treatment of juvenile osteonecrosis should be investigated. He concluded that the success of treatment depends on necrotic bone resorption (elimination), new bone formation in its place, and a remodeling process that is protected from further deformation by adequate containment. It is our opinion that early core decompression combined with containment in the form of a shelf acetabuloplasty can meet these objectives and may improve outcomes compared with the natural history and with previous treatment methods for this age group. Core

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decompression and shelf acetabuloplasty (labral support) for idiopathic juvenile osteonecrosis is the focus of this article.

CORE DECOMPRESSION

Core decompression was reported as a possible solution for LCPD in the 1930s.[12,13] Details and examples of cases were not described, but Ferguson and Howorth[13] indicated that drilling in the active stage resulted in earlier and more complete healing.

The objectives of core decompression are to remove necrotic bone, reduce venous congestion, and encourage revascularization.[14] Removal of necrotic bone has been identified as a key component for success of core decompression.[15,16] Some investigators advocate arthroscopic inspection to ensure removal of necrotic bone.[17,18]

The principle objective of decompression is to reduce intraosseous pressure to decrease venous congestion and improve capillary blood flow. The bone marrow pressure in the femoral head and neck is increased in patients who develop osteonecrosis.[10,19] The pressure is reduced and venous flow improved in some patients following decompression by drilling or by proximal femoral osteotomy.[19,20] Vascularity of the proximal femur is also enhanced by the angiogenesis caused by the trephine opening new vascular channels. In children, the growth plate of the proximal femur is a barrier to revascularization.[21] Reossification of the femoral head is facilitated by removal or fenestration of the epiphyseal plate.[21] Thus, core decompression has several beneficial effects for revascularization of the femoral head.

Numerous investigators have compared core decompression with nonoperative management

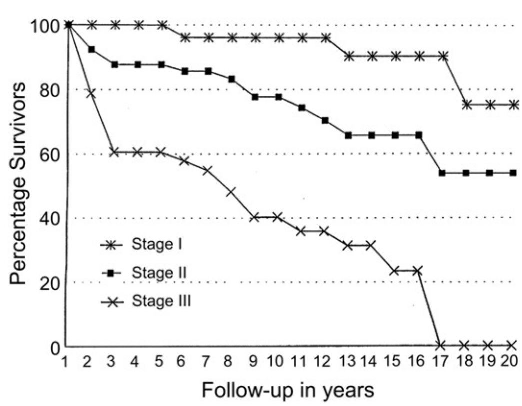

Fig. 1. Survival curves for each Ficat stage. (*Reproduced from* Fairbank AC, Bhatia D, Jennah RH, et al. Long-term results of core decompression for ischaemic necrosis of the femoral head. J Bone Joint Surg Br 1995;77:47; with permission.)

for osteonecrosis of the femoral head. Improved outcomes have been noted following core decompression in the early stages of necrosis.[15,16,22–27] Fairbank and colleagues[25] reported satisfactory long-term results of core decompression performed in Ficat stages I and II (**Fig. 1**). Ficat and Arlet[28] classified patients with a crescent sign as stage II. Stage III indicates flattening and collapse of the femoral head. Other investigators have included the crescent sign as stage III with or without femoral head collapse.[29]

Techniques for core decompression have ranged from multiple small drillings with 3.2-mm Steinmann pins[30,31] to expanding reamers and trapdoor procedures that remove large amounts of necrotic bone.[14] Small pin drillings have been less effective for larger lesions with higher intraosseous pressures.[19] More complete removal of necrotic bone has been recommended for larger lesions in later stages of disease, including stage III, as long as the articular cartilage is intact.[14]

Maintaining a sturdy structural support in the decompressed area during the revascularization process helps prevent disruption of the articular cartilage.[15,16,32] Gradual substitution of necrotic bone or implant material by living bone keeps the decline of the bone's mechanical properties to a minimum.[33] Various graft materials and adjunctive techniques have been used following core decompression.[16,34,35] Liebermann and colleagues[36] and Chang and colleagues[37] performed core decompressions with bone grafting and partially purified bone morphogenic proteins as adjuncts. Keizer and colleagues[16] noted improved outcomes with autograft compared with allograft. For patients with Ficat stage III necrosis, free vascularized fibular grafting is beneficial.[35] Free vascularized fibular grafting in children and adolescents for stage III osteonecrosis has better reported outcomes than those performed in adults.[38]

In the adult population, the prognosis depends on several factors, including the size of the lesion, the location, and the bone quality of the uninvolved portion of the femoral head.[31,39–45] The smaller the lesion, the better the result, regardless of whether it is Ficat stage I or II.[31,39–41] Koo and Kim[43] reported that patients with less than 30% of femoral head involvement did not develop progression of the osteonecrosis, whereas all the hips with more than 40% involvement collapsed. The location of involvement of hip osteonecrosis is also an important factor.[41] Lateral lesions fare worse compared with those with medial or lateral involvement.[31] These factors also apply to juvenile osteonecrosis but the adolescent age group with open growth plates may have a more favorable prognosis when treated later in the course of disease.