



IFSSH Scientific Committee on Kienböck's Disease

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Kienböck's Disease

One century after 1910 Kienböck's first publication of the disease, the pathogenesis for the so-called lunatomalacia still remains unknown. Mechanical factors, acute trauma or repetitive stress do not seem to be a primary cause, but factors that explain symptom aggravation of an already present Kienböck's disease (KD). A biological, more than a mechanical cause, is likely to induce focal intraosseous vascular deprivation with minor bone marrow infarctions as the initiating mechanism of bone weakening, fracture and collapse.

There is an unbalanced bone remodelling, with increased bone resorption by osteoclasts not being counteracted by increased new bone formation by osteoblasts. The reason why osteoclast action overpasses osteoblastic activity in the repair process is still not known. The role of genetic predisposition to the disease is a suggested possibility in selected cases, but certainly it does not play a role in most avascular necrosis. Indeed, no specific gene has been found in association with KD so far. Yet, it is not unlikely that the genetic background of the host may have an influence in the intensity of the reaction after infection and/or immune reactions, and the hypothesis that some sub-populations may be more susceptible to develop KD than others is worth consideration. Another thought provoking possibility is provided by some researchers who suggest KD to be secondary to reactive arthritis, based on increased polymerase chain reaction (PCR arrays) and viral RNA analysis in some patients with KD. Certainly, there is a need to reinforce such evidences with further research in this regard.

While diagnostic techniques have improved in recent years, significant questions remain unanswered about the treatment choices and timing. Most surgeons act under the conviction that surgical interventions appreciably improve the natural history of KD (considering each stage independently). In fact, some appropriately powered, randomized, prospective studies comparing operative vs non-operative treatment in patients with early stages of the disease appear to point in that direction, but a definitive response in this regard is not yet available. Indeed, early diagnosis of KD could allow more efficient treatments, especially in young patients with high functional requirements.

Revascularization and/or radial shortening is the most common surgical preference. The exception is in the infantile, or even in the juvenile lunatomalacia (the so-called "teenböck's disease") where the prognosis is good with conservative measures. Finally, as Litchman pointed out in the centennial celebration held in Vienna, Austria, in May 2010, the natural history and true outcomes of treatment must be determined by

cooperative, multicenter data based on modern research techniques that have been proven to provide consistent, patient oriented results. Only through such a cooperative effort will we ever definitively arrive at a consensus in the classification and treatment of

Kienböck's disease.

REFERENCES:

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