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Arthrosis of the knee in chronic anterior laxity☆

H. Dejour*, G. Walch , G. Deschamps , P. Chambat

Clinique de chirurgie orthopedique et traumatologique, centre hospitalier Lyon-Sud, 69310 Pierre-Bénite, France

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ABSTRACT

Arthrosis following rupture of the anterior cruciate ligament has been analysed in two series. The first series was derived from a review of 150 cases of reconstruction of the anterior cruciate ligament with a follow-up of 3 years or more. Arthrosis was seen to have developed in 13.3%. The second series was concerned with 64 cases of unilateral arthrosis treated by upper tibial valgus osteotomy in whom there had been a previous rupture of the anterior cruciate ligament. The 'tolerance interval' – that is the time between the original ligamentous injury and the time of osteotomy – for the development of arthrosis was very variable, ranging in the natural-history cases from 10 to 50 years, with a mean of 35 years. It is important to recognise the radiological signs of the onset of arthrosis. These are osteophytosis of the intercondylar notch, osteophyte formation at the posterior part of the medial tibial plateau, and, in particular, narrowing of the medial joint line with posterior subluxation of the medial femoral condyle, well seen in lateral radiographs whilst standing on one lower limb. Early arthroses, appearing after 10 years, may occur as a 'natural arthrosis', but it develops much more frequently after surgical treatment that had failed to correct anterior laxity and particularly when it had been performed on knees that were already pre-arthrotic. The main factor in arthrosis is anterior laxity measured radiologically by an 'active Lachman' radiograph. Removal of the medial meniscus which in itself, is liable to produce arthrosis, is even more harmful in anterior cruciate laxity since it doubles the degree of anterior subluxation of the tibia seen on unilateral weight-bearing. The development of varus deformity, which characterises progressive arthrosis, has its origin in wear of the posterior part of the medial tibial plateau caused by anterior cruciate laxity. Other factors play an important part such as associated lateral laxity, constitutional genu varum and weakness of the hamstring muscles, which oppose the subluxating action of the quadriceps.

1. Introduction

Although the risk of knee osteoarthritis after injury to the anterior cruciate ligament (ACL) has been well documented by both experimental and clinical studies [1,3,4,6,8,9], the frequency and course of this complication remain unclear. Its importance, however, has been demonstrated by J.-C. Imbert [5], who studied the natural history of ACL injury, and by J.-H. Aubriot and P. Rivat [2], who investigated post-operative osteoarthritis. Statistics for revision surgery after ligament injury often fail to provide information on osteoarthritis, and details are often insufficient on the type of radiograph used to establish the diagnosis. The objectives of our work are to describe the early stages of osteoarthritis, to identify causative biomechanical factors, and to shed light on the crucial issue of lesion potential for progression.

2. Study material

Our study relies on two very different sources of statistical data.

1 – *The first source* is a study of *osteoarthritis detected during re-evaluation in 150 cases of chronic anterior knee laxity treated* with ACL reconstruction using a free transplant harvested from the middle third of the patellar tendon, combined routinely with an antero-lateral fascia lata graft as described by Lemaire [7]. All these patients were re-evaluated, after 3 to 6 years, the mean follow-up being 4 years.

In this study, osteoarthritis was defined chiefly according to the radiographic findings. At re-evaluation, the work-up included comparative radiographs of both knees with an antero-posterior single-leg-stance view, a lateral single-leg-stance view with the knee flexed at 30°, and a 30° axial view of the patellae. Osteoarthritis was defined as joint space narrowing by 50% or more on at least one radiograph combined with osteophytes. We defined pre-osteoarthritis as joint space narrowing by less than 50% and

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* Corresponding author.
E-mail address: rco@sofcot.fr (H. Dejour).

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Fig. 1. *Sav. . . osteoarthritis during the natural history of ACL injury*. 49-year-old woman an ACL tear at 17 years of age.

osteoarthritic remodelling as presence of osteophytes with no detectable cartilage damage. The distribution was as follows: normal radiographs, $n = 90$; osteoarthritic remodelling, $n = 40$; pre-osteoarthritis, $n = 7$; and definite osteoarthritis, $n = 13$. Thus, overall, osteoarthritis developed in 20 knees, *i.e.*, 13.3%.

2 – *The second source of data* is very different: in *64 cases of medial tibio-femoral osteoarthritis* treated with valgus high tibial osteotomy, *ACL injury was diagnosed retrospectively* with a very high degree of probability. There was an immutable clinical pattern with a history of a specific injury described as a sprain and often treated with cast immobilisation. The knee never returned to normal; instead, the patient experienced recurrent knee instability responsible for functional impairment that often required the discontinuation of all sports. The pain related to osteoarthritis developed only many years later. These criteria for prior ACL injury were met in 20 patients. We believe these patients illustrate the development of osteoarthritis as part of the natural history of ACL injury. These 20 cases constitute our natural-history group (Fig. 1).

In 24 other patients, meniscal signs prompted resection of the medial meniscus. After this procedure, the episodes of knee locking and pain resolved but the knee remained weak, tending to give way, although a return to a measured level of sports participation was possible in some patients. We believe these patients illustrate the natural history of ACL injury with medial meniscectomy. The remaining 20 patients had been diagnosed with ACL injury and treated with a number of procedures on the ligaments before our evaluation at the stage of osteoarthritis.

In the 44 patients providing information on the natural history of ACL injuries with or without meniscectomy, we found convincing evidence of ACL injury. A palpable clunk was identified as the cause of the long-standing instability in 42 of these patients, and the remaining two knees had marked anterior translation in both flexion and extension: a: antero-posterior single-leg-stance view; b: lateral view in the supine position; c: lateral single-leg-stance view; note the posterior subluxation of the medial condyle; d: active Lachman radiograph: 13 mm.

Our classification of our patients may seem open to criticism, as osteoarthritis can cause injury to the ACL. To avoid this pitfall, we did not include patients with highly advanced osteoarthritis and irreducible subluxation.

In the included patients, we believe that the very long history of clinical symptoms constitutes definitive evidence of a traumatic ACL injury in the distant past. In all likelihood, the number of cases of osteoarthritis induced by chronic anterior laxity is far greater. Indeed, we did not include patients with global osteoarthritis, as involvement of the lateral tibio-femoral compartment eliminates the palpable clunk. Neither did we consider patients with bilateral osteoarthritis, as we wanted to conduct a detailed evaluation of the contralateral healthy knee. Nevertheless, ACL injury is bilateral in a noticeable proportion of cases (9%) (Fig. 2).

This second statistical study in 64 patients seen at the stage of advanced osteoarthritis and having a history of ACL injury in the remote past is of particular interest, as it provides information on the tolerance interval, which we define as the time from the sprain to the tibial osteotomy.

3. Radiological findings in osteoarthritis secondary to anterior cruciate ligament injury

Two patterns of osteoarthritis progression were observed: