Clin Orthop Relat Res (2016) 474:1810–1811 / DOI 10.1007/s11999-016-4945-6

Clinical Orthopaedics and Related Research®
A Publication of The Association of Bone and Joint Surgeons®

Published online: 21 June 2016

CORR Insights

CORR Insights®: The Natural History of Osteoarthritis: What Happens to the Other Hip?

Hugo Armando Rodriguez MD

Where Are We Now?

Osteoarthritis (OA) is the most common joint disease, and is among the most frequent health issues associated with older individuals [1–4]. In fact, OA is the leading indication for joint replacement surgery [6].

Following unilateral hip arthroplasty, a patient will usually ask about his or her other hip. "Will my other hip deteriorate as well? Will I need another operation?" There are no easy answers. Although patient characteristics and radiographic findings can offer clues, the pathophysiology of OA is poorly understood, as the effort to prevent OA is based more on assumptions than real science. Even with modern technology and research at our disposal, preventing, diagnosing, and treating OA remains a major challenge.

Twenty years before the current study, Ritter and colleagues [9], reported that 37% of contralateral hips diagnosed as normal would likely have OA develop within 10 years, and 8% would undergo total hip replacement.

Amstutz and Le Duff determined the incidence of contralateral OA damage in a group of patients who were treated with unilateral hip arthroplasty. They found that the proportion of patients developing OA in the contralateral hip in patients with unilateral OA was 41% at 10 years, with 19% undergoing hip arthroplasty. This information will help surgeons know more about the fate of their patients' contralateral hips.

Where Do We Need To Go?

Although Amstutz and Le Duff provided us with valuable information, several questions remain. For example, a shallow, dysplastic acetabulum is believed to predispose to OA [7]. But how do we explain that it is common for only one hip to develop OA when the dysplasia is similar in both hips? Why do some individuals with advanced joint damage have minimal pain and dysfunction? Why do most patients with OA frequently experience periods of remission with symptoms that virtually disappear [3]? Why does primary OA rarely occur in the ankle and wrist, but is common in hip and knee [8]?

How Do We Get There?

The pathogenesis of articular cartilage failure is multifactorial, and includes

This CORR Insights® *is a commentary on the article* "The Natural History of Osteoarthritis: What Happens to the Other Hip?" *by Amstutz and LeDuff available at:* DOI: 10.1007/s11999-016-4888-y.

The author certifies that he, or a member of his immediate family, has no funding or commercial associations (eg, consultancies, stock ownership, equity interest, patent/licensing arrangements, etc.) that might pose a conflict of interest in connection with the submitted article.

All ICMJE Conflict of Interest Forms for authors and *Clinical Orthopaedics and Related Research*® editors and board members are on file with the publication and can be viewed on request.

The opinions expressed are those of the writers, and do not reflect the opinion or policy of *CORR*® or The Association of Bone and Joint Surgeons®.

This *CORR* Insights® comment refers to the article available at DOI: 10.1007/s11999-016-4888-y.

H. A. Rodriguez MD (✉)
Hospital Infantil Universitario de San Jose, Cra 16A no 82-46 Cons 401, Bogota, Cund 1, Colombia
e-mail: harodriguez1@fucsalud.edu.co; hugortopedista@yahoo.com

CORR Insights

various mechanisms: Biology, genetics, biomechanics, and inflammation. If we really want to treat the disease, we need to understand them all, instead of trying to think about the pathogenesis of end-stage arthritis only in mechanical terms. It is important to integrate basic, experimental, and clinical research in the study of OA.

In order to properly study "the other hip," we will need a better understanding of the initiating events in cartilage damage, the connection between different pathologic influences, and the role of the chondrocyte in sustaining matrix homeostasis.

We also need to investigate if metabolic differences in articular cartilage across different joints may explain the lower incidence of OA in some joints like the ankle [2]. Our research should focus on examining how aging and mechanical loading can potentially produce cartilage damage.

References

1. Brooks PM. Impact of osteoarthritis on individuals and society: How much disability? Social consequences and health economic implications. *Curr Opin Rheumatol*. 2002;14:573–577.
2. Chubinskaya S, Huch K, Mikecz K, Cs-Szabo G, Hasty KA, Kuettner KE, Cole AA. Chondrocyte matrix metalloproteinase-8: Up-regulation of neutrophil collagenase by interleukin-1 beta in human cartilage from knee and ankle joints. *Lab Invest*. 1996;74:232–240.
3. Cooper C, Dennison E. The natural history and Prognosis of Osteoarthritis. In: Brandt KD, Doherty M, Lohmander LS, eds. *Osteoarthritis*. Oxford, England: Oxford University Press; 1998:237–249.
4. Dennison E, Cooper C. Osteoarthritis: Epidemiology and classification. In: Hochberg MC, Silman AJ, Smolen JS, Weinblatt ME, Weisman MH, eds. *Rheumatology*. London, England: Mosby; 2003:1781–1791.
5. Felson DT. The epidemiology of osteoarthritis: Prevalence and risk factors. In: Kuettner KE, Goldberg VM, eds. *Osteoarthritic Disorders*. Rosemont, IL: American Academy of Orthopedic Surgeons; 1995:229–237.
6. Murphy L, Helmick CG. The impact of osteoarthritis in the United States: A population-health perspective. *Am J Nurs*. 2012;112:S13–19.
7. Ponseti IV. Morphology of the acetabulum in congenital dislocation of the hip. Gross, histological and roentgenographic studies. *J Bone Joint Surg Am*. 1978;60A:586–599.
8. Praemer AP, Furner S, Rice DP. Musculoskeletal Conditions in the United States. Rosemont, IL: American Academy of Orthopaedic Surgeons; 1999:182.
9. Ritter M, Carr K, Herbst S, Eizember L, Keating E, Faris P, Meding J. Outcome of the contralateral hip following total hip arthroplasty for osteoarthritis. *J Arthroplasty*. 1996;11:242–246.