The rise of osteoarthritis

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Osteoarthritis was seen as a stepchild of rheumatology until very recently. Veterinarians took an interest in it because they had racehorses and racing greyhounds to take care of, whose economic value to their owners made it worthwhile. But medicine had little to offer, especially as osteoarthritis was thought to be part of ageing, like the greying of hair, and few distinctions were made in the clinical presentations. The terminology of "degenerative" or "wear-and-tear" arthritis reflected this view. It held little interest for most clinicians.

The name, osteoarthritis, implicitly acknowledges that inflammation plays a part in the presentation, and the nodal deformities of interphalangeal joints have aroused some curiosity since the early 18th century; but little else has been done to distinguish osteoarthritis from other forms of arthritis. For therapy, there were thermal spas for those who could afford to go to them, and surgeons performed osteotomies on the lower extremities of some crippled by hip and knee arthritis.

What has changed to attract clinicians, biologists, geneticists, pathologists and orthopaedists now? For one thing, there is the recognition that osteoarthritis represents a final common pathway for all insults to the joints (*1*). Also, we now know that osteoarthritis does not arise de novo but has antecedents that may be remote in time or, because of their severity (as in athletic injuries), more recent. That characterizes osteoarthritis as a dynamic process worthy of study, and has led to therapeutic advances that may not yet be able to halt the progression but can afford relief from pain and disability. To that end, scales and tests permit clinical assessment of severity, and molecular biology offers explanations of pathogenesis. The clinical definitions that emphasize changes made visible by X-ray — such as joint (space) narrowing owing to cartilage loss and osteophytes (or spurs, which are reparative processes that increase the articular surface), buttressing, increased density of juxta-articular bone, and eburnation — cannot distinguish between symptomatic and relatively asymptomatic osteoarthritis. The majority of people have relatively mild symptoms, if any at all, with perhaps some sensitivity to weather changes, occasional limitations of motion, and mild-to-moderate pain. As they do not seek medical consultation, a vast industry caters for them, with, for example, herbals, omega fatty acids from fish oils, simple analgesics for self-medication, and topical products such as capsaicin. Imports from Asia include the Ayurvedic pharmacopaeia (mostly herb-derived), yoga, and acupuncture. For the majority, moral support and time spent — more difficult to provide in today's hectic medical environment — bear fruit.

For an appreciable minority, however, osteoarthritis behaves as a true disease. Fraying and fibrillation of cartilage result from the elaboration of proteolytic and collagenolytic enzymes by the chondrocytes that initially attack the matrix within which they are embedded (*2*). Synovitis develops, with elaboration of cytokines that further attack the cartilage. Oedema and increased vascularity develop in response. Much of the more severe pain in osteoarthritis probably results from synovitis. This is certainly true of the variety known as erosive interphalangeal osteoarthritis (*3*), which occurs in women around the time of menopause and entails the symmetrical involvement of distal and proximal interphalangeal joints and the base of the thumb. Frequently in inflammatory osteoarthritis, crystals of calcium pyrophosphate dihydrate or of hydroxy apatite incite the inflammation. Cartilage has no nerve fibres and therefore cannot hurt, but synovium, periosteum, and the contiguous ligaments and muscles can produce pain.

Some short-term studies suggested that simple analgesics, like paracetamol offered sufficient pain relief, and the 1995 guidelines developed for the American College of Rheumatology (ACR) reflected this view (*4*). But most subsequent studies confirmed patient preferences for nonsteroidal anti-inflammatory drugs (*5*), reflected in a revision in 2000 (*6*). Thus, cures are elusive, as osteoarthritis begins long before it is clinically apparent, and we have not yet learnt how to recognize those who will develop severely symptomatic disease, or benefit from any preventive treatment that might be found.

Osteoarthritis must be looked at not only as the final common pathway for all the afflictions, abuse, and injuries of the joint but also as an active joint disease, and perhaps the former dichotomy of osteoarthritis and arthrosis was appropriate after all. As the population of the world grows older and medical advances lengthen average life expectancy, osteoarthritis will become a larger public health problem — not because it is a manifestation of ageing but because it usually takes years to reach clinical relevance. An older population lives on through those years, so physicians, surgeons, architects, and city planners, as well as designers of furniture and cars, will have to take notice. ■

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