Leader

Osteoarthritis: definitions and criteria

With the aim of promoting consistency in osteoarthritis (OA) research the American Rheumatism Association (ARA) has recently published a proposed system of classification and a set of criteria for the reporting of osteoarthritis of the knee.¹ Preliminary findings of the criteria committee's work on OA of the hip and hand have also been reported.² These publications raise many fundamental questions, ranging from the value and use of diagnostic criteria to the definition and nature of OA.³

Definitions and classification of OA

Historically, evidence of bone hypertrophy accompanying focal cartilage damage led to pathologists being able to differentiate between OA and other forms of arthritis.⁴ Radiology is the rheumatologists surrogate for pathology, and the combination of joint space narrowing, subchondral bone changes, and osteophytosis on the x ray soon became the centrepiece of definitions of OA.⁵ The fact that some cases of OA appeared to be caused by previous injury or disease then led to the division of OA into primary and secondary types,⁶ as well as its classification by joint site. It was then shown by Lawrence and others that symptoms correlated poorly with anatomical changes in individual joints,⁷ but no satisfactory clinical definition of OA was forthcoming.

With this background the ARA has considered the definition and classification of OA. Their working definition of OA is: 'a heterogeneous group of conditions that lead to joint symptoms and signs which are associated with defective integrity of cartilage, in addition to the related changes in underlying bone and at the joint margin.'¹ This is a cumbersome and woolly definition, but one that is certainly no worse than any others that have been suggested.⁸ It is worth noting that it demands the presence of symptoms and stresses focal defects of articular cartilage. Loss of cartilage is the one feature that is present in all attempted definitions of OA known to us.

The ARA classification of subsets of OA by joint site and into 'idiopathic' or 'secondary' is not dissimilar to the established systems. The term

idiopathic, however, replaces 'primary' in the recognition that specific aetiologies may in time be found for subsets of idiopathic OA. Calcium pyrophosphate deposition has been included, they admit arbitrarily, in the secondary group. This approach presumes that OA is a group of heterogeneous diseases with a similar end point. The alternative construct, that OA represents the variable, age related reaction pattern of damaged joints, is not considered, though this approach is favoured by some investigators.⁹ It is an important point as calcium pyrophosphate deposits, for example, may be the marker of a certain type of joint reaction rather than a disease subset.¹⁰

Criteria for reporting knee OA

The ARA has tried to develop criteria for reporting knee OA using commonly available diagnostic techniques. Twenty three historical, clinical, and laboratory features were chosen, and their sensitivity and specificity for disease detection evaluated by a 'Delphi' technique of opinion sampling. These variables were subsequently expanded to include 85 features to be tested prospectively in a multicentre study.¹

Subjects included in the study had a history of knee pain thought to arise from the joint, currently available radiographs, and no evidence of secondary OA. The standard against which the classification criteria were judged was the physicians' clinical diagnosis of idiopathic knee OA, which was reviewed by three members of the committee. The criteria were evaluated on 264 patients from 14 centres. After a change in diagnosis in 27 cases 130 were considered to have idiopathic knee OA. The remaining 107, who were a younger group with a variety of conditions (mostly rheumatoid arthritis (RA)), were used as controls. After the initial univariate analysis, features showing some discriminatory power were considered for further examination. Combinations of these variables were subjected to a technique known as recursive partitioning, from which were derived three classification trees, or algorithms: one for clinical criteria alone, and one each for a combination of clinical and

laboratory, or clinical, radiographic, and laboratory features. These criteria appeared to have a high sensitivity and specificity when applied to the study groups and performed well in clinical practice.

Features that emerged as having the most discriminatory power included age over 50, crepitus, bony enlargement, morning stiffness of less than 30 minutes, and osteophytosis, which was the only radiographic predictor. The classification trees would allow a diagnosis of OA in someone with knee pain if they were over 40, and had crepitus or bony enlargement without prolonged morning stiffness. Similarly, the combination of knee pain and osteophytes emerged as highly discriminatory for OA if radiographs were available.

It is important to recognise the shortcomings of the study before considering the application of these criteria. The controls were not matched for age or sex, were younger than the patients with OA, and included many patients with RA. At its simplest then, the criteria have only been shown to perform well in the differentiation of OA from younger people with RA. This may account for the emergence of age and osteophytosis as important discriminators and the absence of features such as joint space narrowing. Osteophytes may be an independent, age related variable,¹¹ and the incidence of features such as crepitus in populations is unknown. It remains to be seen how the criteria will perform in older subjects, or in differentiating OA from other causes of knee pain. The study also suffers from circularity. The criteria were derived from a consensus view of what constitutes OA, and the diagnosis was made by committee members. In the absence of any definition or test for OA it could be said that the exercise had only described what the members of the committee think are the features of an osteoarthritic knee joint. Finally, the features to emerge as important in the recognition of OA are largely subjective and have not been validated.

The ARA states that criteria of this sort should not be used for the diagnosis of knee OA. Many of the problems highlighted in this article are also acknowledged by the authors of the ARA reports. Why, then, are OA criteria being developed? They could help, it is suggested, in the description of patients involved in studies, or in epidemiological work. The hope is that they will aid communication by providing a descriptive framework for studies of OA. It will be important, however, to validate the reproducibility and prevalence of chosen features of OA joints if that aim is to be achieved. Unless the criticisms outlined above can be answered by suitable investigations it is probably unwise to try to apply the published criteria.

Conclusions

Any studies that help consistent reporting of the features of OA and improve communication are useful. There is still a huge problem in OA research caused by the lack of any clear understanding of its clinical outcomes. The ARA publications reviewed here have stimulated us to rethink our system of disease classification and to test the validity of several clinical and radiographic features. Any attempt, however, to classify and find criteria for a heterogeneous condition, which defies definition and for which there is no test, is bound to flounder. The ARA has ended up with criteria for knee OA which exclude mention of cartilage damage, the indispensable condition of most definitions, including its own. There should be better ways forward.

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