CONCISE REPORTS

Hypermobility associated with osteoarthritis of the thumb base: a clinical and radiological subset of hand osteoarthritis

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Abstract

Objectives—To study the impact of articular hypermobility on the clinical and radiological features of hand osteoarthritis (OA) and to investigate whether hand osteoarthritis associated with hypermobility should be considered a separate subset of hand OA.

Methods—Fifty consecutive female patients with clinical hand OA and thumb base symptoms were examined for hypermobility according to the Beighton criteria.

Results—Thirty one of the 50 patients had hypermobility features (Beighton score 22) and 17 patients fulfilled four or more Beighton criteria. Corresponding figures for 94 control patients were 30 (p < 0.05) and nine (p < 0.001) respectively. Patients with hypermobility features were characterised clinically and radiologically by fewer and less severely involved interphalangeal joints. Radiologically, two fairly distinct subsets could be identified: Severe interphalangeal OA in which the prevalence of hypermobility was similar to controls, and patients with predominant involvement of the first carpometacarpal joint (CMC 1), most of whom had evidence of hypermobility.

Conclusion—A causal relation exists between articular hypermobility and development of thumb base OA, and hypermobility associated hand OA constitutes a definite clinical and radiological subset of hand OA. In the clinical setting, the easily applied hypermobility criterion of passive dorsiflexion of the fifth finger >90° is useful in identifying most patients with hand OA and hypermobility.

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Osteoarthritis (OA) is a very heterogeneous condition, representing a common pathway from many causes. It is likely that further understanding of the pathophysiological mechanisms of OA will lead to a division into definite subsets.

Benign articular hypermobility has been implicated in the development of OA, particularly of spine and knee joints,1–3 and we have recently shown that articular hypermobility is related to the severity and disability of clinical thumb base OA in a mixed group of patients with hand OA.4 Hand OA is a common condition mostly affecting postmenopausal women and causing considerable morbidity. The severity of involvement of the first carpometacarpal joint (CMC 1) is a major determinant of disability.4 Radiographic evaluation of the CMC 1 joint can be difficult, particularly in the early stages when instability may be present in the absence of other changes.5–7 There is evidence that laxity of the volar ligaments with secondary joint instability plays an important part in the development of CMC 1 OA,8–9 but no connection has been found with generalised articular hypermobility.

The aim of this study was to investigate the impact of hypermobility on clinical and radiological findings in patients with clinical thumb base OA, and to investigate whether hand OA associated with hypermobility constitutes a definite radiological subset of hand OA.

Patients and methods

PATIENTS

As part of an ongoing study of hand OA in Iceland, fifty three consecutive female patients who had been referred for ergotherapy treatment with a diagnosis of hand OA and had symptoms from the thumb base were included in the study. Three patients were excluded at our initial examination due to other diseases (inflammatory arthritis two, diabetic or arthropathy one). The remaining 50 patients all fulfilled the American College of Rheumatology clinical criteria for hand OA.10 Median age was 64, range 47–87. Hypermobility features and affected joint distribution and severity for each patient were assessed clinically by two of us.4 The Beighton criteria for assessment of hypermobility are based on the following tests: (1) passive dorsiflexion of the fifth finger >90°; (2) passive apposition of thumb to forearm; (3) hyperextension of elbows ≥10°; (4) hyperextension of knees ≥10°; (5) resting palms on floor on forward flexion with straight knees. The first four are bilateral, giving a numerical score from 0 to 9.11 As well as clinical examination, unequivocal anamnestic criteria were accepted.
There was a reasonable correlation between clinical examination and radiological scores (r, 0-7, p < 0.001 for number of affected joints) and the number of clinically affected joints was comparable with the number of joints with a radiological grade of ≥2 (table). Two patients had no radiological evidence of CMC involvement and 13 had only slight involvement (radiological scores 1-2). Grade ≥3 radiological involvement of the trapeziocapitap joint was seen in 16 patients, 10 of whom had a Beighton score of 0-1 (p < 0.05). Cumulative radiological scores correlated with age and duration of disease: (interphalangeal score v age r, 0.46, p < 0.01; v duration r, 0.61, p < 0.001. CMC1 score v age r, 0.33, p < 0.05; v duration r, 0.38, p < 0.01). Interestingly, however, there was no correlation between interphalangeal and CMC1 scores (r, 0.06, NS).

Figure 1 shows the relation between radiological subsets of hand OA and hypermobility when patients are grouped into two categories; one with severe bilateral interphalangeal OA, and one without severe interphalangeal disease and more pronounced CMC1 disease. Hypermobility features (Beighton score ≥2) were present in 16 of 22 of those with predominant CMC1 disease compared with 8 of 21 of those with severe interphalangeal disease (p < 0.01). Seven patients with mild radiological changes could not be classified either way.

Figure 2 shows two examples of patients from the extremes of the hand OA range. One patient has severe classic Heberden's disease and CMC1 involvement and the other (who fulfilled five Beighton criteria) has very little interphalangeal OA, but severe CMC1 OA with subluxation.

Discussion
In this study of 50 consecutive female patients with clinical thumb base OA, hypermobility
features were much more prevalent than in age matched controls. Patients with hypermobility features also seemed to constitute a definite subset, characterised clinically and radiologically by mild interphalangeal joint changes and predominantly CMC1 involvement.

From the present findings it is easy to envisage at least two main pathophysiological pathways in the development of hand OA: one a systemic disorder which causes a generalised disease of hand joints, and another in which ligament laxity leads primarily to joint instability and secondarily to cartilage damage and OA in the CMC1 joint. The discordance between radiological changes in the interphalangeal and CMC1 joints despite correlation with age and duration of disease seems to support different pathogenetic mechanisms. A similar discordance has been described by McCarthy et al in a longitudinal study.13
Several mechanisms have been suggested for the development of OA in hypermobile subjects, but when the present findings are viewed in the light of the work done by Pellegrini, the most likely pathway is through laxity of the palmar ligament in hypermobile subjects, causing joint instability and excessive shear forces which initiate osteoarthritic changes in cartilage in adjacent areas of the joint. Even moderate laxity (Beighton scores of 2–3) seems to be of importance, a finding analogous to that of Diaz et al. Although laxity of the thumb base ligaments may be the most relevant, the thumb apposition criterion is impractical in the clinical setting for obvious reasons. Instead, the criterion involving ≥90° dorsiflexion of the fifth finger seemed the most useful, identifying most hypermobile patients. The little finger criterion also has the advantage of being readily apparent on clinical examination whereas some of the other criteria involve anamnestic evaluation. It could be argued from the present findings that hypermobility might be protective for development of interphalangeal OA. Although the exact relation can only be clarified through longitudinal studies, it seems that the “normal” prevalence of hypermobility features in patients with severe radiological interphalangeal OA, and the similarity of the radiological findings in the trapeziometacarpal and the interphalangeal joints make this explanation less likely.

Apparently, hand OA associated with hypermobility is very prevalent in Icelandic patients submitted to ergotherapy. In our previous study of 100 such patients including males and patients without thumb base involvement, we found a direct relation between hypermobility on the one hand and the clinical severity of CMC1 OA and disability on the other. This was not apparent in the present study, probably due to selection criteria and the impact of symptoms at the thumb base on disability. Patients receiving ergotherapy are a heterogenous group with regard to disease subsets, but they probably constitute a severely symptomatic group of patients with hand OA. Thus hypermobile patients are almost certainly underrepresented in this group in relation to the total prevalence of hand OA in Iceland. The prevalence of hand OA associated with hypermobility has yet to be shown in other populations, but the available evidence suggests that the prevalence of hypermobility and hand OA in Iceland is similar to that found in Sweden and England.

The present findings seem to call for reconsideration of current views of the pathogenesis of hand OA. Although the exact relation between hypermobility and hand OA can only be determined through longitudinal studies, this study raises questions regarding current treatment, identification of patients at risk, and prevention. Certainly, hand OA associated with hypermobility should be taken into consideration in future studies.

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