Heberden’s nodes and osteoarthrosis of the hip

Sir, I have read the interesting report by McGoldrick and O’Brien studying the relation between various patterns of osteoarthritis (OA) of the hip and Heberden’s nodes. Their paper also challenges our earlier report, which found no significant association between osteoarthrosis of the weight bearing joints (hips and knees) and Heberden’s nodes in a controlled study.²

My main criticism of the McGoldrick and O’Brien paper is that they do not compare like with like. The prevalence and severity of the nodes in their axillary, superomedial, and protrusion groups combined (representing primary OA) is indeed higher than in the congenital dislocation of the hip, dysplasia, and superolateral groups combined (representing secondary OA). On the other hand, the combined female to male ratio is 32/18 among the probands in the first three groups and 15/22 among the probands in the last three groups. As is also pointed out by the authors this represents a clear difference in favour of women in the groups representing primary OA.

It is universally acknowledged that Heberden’s nodes by themselves are more common among women, regardless of the presence of OA in other joints. Thus it would have been better if the authors had analysed their data by considering the prevalence of the nodes among the two genders separately in order to eliminate the sex bias.

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References

Osteomalacia and coeliac disease presenting as isolated dactylitis

Sir, We read with interest the case report ‘Osteomalacia and coeliac disease presenting as isolated dactylitis’ by Jawad et al.¹ We believe that the dactylitis was related to secondary parathyroid overactivity rather than a primary manifestation of osteomalacia.

We have a patient, an Asian vegetarian woman aged 46 years, who presented with pain and swelling in her right index and middle fingers. An x-ray examination was suggestive of a dactylitis. Her investigations excluded tuberculosis, sarcoidosis, sickle cell disease, syphilis, and inflammatory arthritis.

Serum calcium was low (2.06 mmol/l), phosphate normal (0.95 mmol/l), alkaline phosphatase raised (212 I/UL), and serum vitamin D very low (3 mmol/l). Parathyroid hormone was 1.5 ng/l (normal up to 0.5). She was treated with 3000 U calciferol daily. Three months later her dactylitis had
settled and the biochemical abnormalities came back to normal, including parathyroid hormone of 0.48 ng/l.

Our patient had dietary osteomalacia and secondary hyperparathyroidism which settled completely with calciferol treatment.

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Reference


Sir, The case report presented by Drs Sultan and Bruckner concluding that the dactylitis seen in their patient was a manifestation of secondary hyperparathyroidism is in keeping with conclusions we published previously.1

We pointed out that the radiological bone changes in our patient’s hand were confined to the middle phalanx, and although they may well have been caused directly by the osteomalacia, it seemed more likely that they were a manifestation of secondary hyperparathyroidism. Clearly the finding of a raised parathyroid hormone is not unexpected and does not prove that the radiological changes were due to hyperparathyroidism, particularly as in our case the histology was non-specific, though tetracycline labelling was not performed.

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J D PERRY
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Reference


Inhibition of neutrophil myeloperoxidase by rabbit anti-(human myeloperoxidase)

Sir, In the paper by Nurcombe and Edwards published in the Annals1 the authors claim to have shown inhibition of fluid phase neutrophil myeloperoxidase by the IgG fraction of an anti-myeloperoxidase antibody in a chemiluminescence system. But incredibly for such a reputable scientific journal they have been allowed to present their data (Fig. 5) without any reference to the effect of normal rabbit (control) IgG on fluid phase myeloperoxidase in the same system. Even if it had had no effect, this should have been mentioned. We have been using a similar system to evaluate the effect of antibodies to myeloperoxidase on the activity of the fluid phase enzyme, and there is undoubtedly considerable protein quenching by normal immunoglobulin (with apparent inhibition) of the chemiluminescence generated by fluid phase neutrophil myeloperoxidase, compared with activity in the absence of normal IgG. This must be taken into consideration in the interpretation of any ‘inhibition’ occurring in the presence of anti-myeloperoxidase IgG.

Reference


Sir, Drs Thompson and Lee comment on the fact that in Fig. 5 (and 6) of our recent paper ‘Role of myeloperoxidase in intracellular and extracellular chemiluminescence of neutrophils’1 we did not state the effects of equivalent amounts of non-immune IgG on this system. In such experiments we routinely measure the effects of non-immune IgG2 3 as some types of chemiluminescence are susceptible to non-specific quenching by soluble proteins. The problem of non-specific protein quenching by antibodies is greatly reduced when IgG fractions are purified from high titre antisera. In our experiments such non-specific quenching of extracellular chemiluminescence by equivalent amounts of non-immune IgG was only 5-10% of that observed by our anti-(myeloperoxidase) IgG and hence the effects noted in Figs 5 and 6 were due to specific inhibition of myeloperoxidase.

Reference

Osteomalacia and coeliac disease presenting as isolated dactylitis.
A H Sultan and F E Bruckner

doi: 10.1136/ard.48.7.614-b

Updated information and services can be found at:
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