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occurs, measuring creatine kinase concentration may give a better ‘overall’ impression of the muscle damage than do myoglobin levels.

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References


Ankylosing spondylitis and middle ear impairment

Sir, The publication in your journal of a case report by Magaro et al, in which a middle ear conductive impairment was observed in association with ankylosing spondylitis (AS),1 drew our attention to the subject. We studied the auditory function of 94 ears from 48 patients with AS diagnosed according to the New York diagnostic criteria.2 There were nine women and 39 men, with ages ranging from 15 to 58 years, and an average age of 35 years. In all of them otoscopy, Weber’s test, Rinne’s test, pure tone audiometry, verbal audiometry, tympanometry, and a stapedial reflex test were performed. In some patients a brain stem evoked response audiometry (BERA) examination was made. Ears in which previous disease could affect the results were rejected (two ears with chronic otitis media). We did not find any middle ear impairment, but sensorineural hearing loss was found more frequently in patients with AS than in the control population.

We think the results presented by Magaro et al1 do not provide sufficient evidence for concluding that the conductive defect was due to involvement of the fibrocartilaginous articulations between malleus and incus or incus and stapes and not to the stapes fixation seen in otosclerotic ears. The case presented by Magaro et al could be common otosclerosis in a patient with AS. An exploratory tympanotomy should make it clear which structures are responsible.

Our study suggests that middle ear involvement either does not occur in patients with AS or is very uncommon (less than 1% of ears in patients with AS). On the other hand, the sensorineural component deserves further research, and this is being conducted in our hospital.

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References


Thyroid disorders in systemic lupus erythematosus

Sir, The observations by Goh and Wang in the Annals that the prevalence of thyroid disorders is greater in a population of Malaysian patients with systemic lupus erythematosus (SLE) than in the general population are confirmed by our own experience. In a recent survey in Oxford of 64 patients with SLE (61 female, three male) 10 women were found who had also suffered clinical thyroid disease. Seven had had thyrotoxicosis, predating the diagnosis of SLE in six by one to 17 years. All were treated with either carbimazole or Neo-Mercazol, and two required radiolabelled iodine for subsequent flares. Three patients had suffered hypothyroidism, predating the SLE in one, and were treated with thyroxine. Thyroid antibody levels were not available.

The prevalence of overt thyroid disease in our group was 11-5% for thyrotoxicosis and 4-9% for hypothyroidism in women, compared with 1-9 and 1-0% respectively for a British population. There were no clinical features of this subgroup which distinguished them from the total SLE population. Goh and Wang suggest a possible association between the antibodies responsible for the false positive Wassermann reaction and the presence of thyroid disease. None of our thyroid subgroup had a false positive Wassermann reaction, and the frequency of antithyroldipin antibody was no different in these patients from that in the total group. It is possible that the thyroid disorders were related to the presence of thyroid stimulating and inhibiting immunoglobulins which have been demonstrated in patients with SLE.3

Two patients showed a third autoimmune disorder; pernicious anaemia in one and Addison’s disease in the other. Among the SLE patients without overt thyroid disease were one with insulin dependent diabetes mellitus and one with myasthenia gravis, predating the SLE by 10 and eight years respectively. Clearly there is considerable overlap between the organ specific and non-organ specific autoimmune disorders.

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