Further studies are needed to clarify the biological significance of the soluble form of interleukin-2 receptor in systemic sclerosis and, by longitudinal follow up of several patients, its possible use for monitoring the progression of the disease.

PAOLO AIRO MARIAM ALIA BETTINZILO ROBERTO GORLA ROBERTO CATTANEO Servizio di Immunologia Clinica Ospedali Civili 25100 Brescia Italy


Nature of the ossicular joints and their involvement in rheumatoid arthritis

Sir: I read with interest the paper published recently in the Annals1 but must disagree with the description of the ossicular joints as synovial with diarthrodial articular disc. I note the use of the word by Reiter et al2 as a reference for this statement, but examination of his paper shows that his assertion is not based on referenced pathological or anatomical fact. In 1981 the ossicular joints were described by Anson as 'diarthrodial joints surrounded by a thin capsular ligament. From the inner surface of the capsular ligament a wedgeshaped circular rim projects into the joint cavity and incompletely divides it.'3 There was no mention of synovium or a cartilaginous disc. Modern understanding of the physiology of sound conduction through the middle ear emphasises the importance of the ossicles acting only as a piston without significant rotation about the joints.4 In man the incudomalleal joint seems to have no function apart from binding the two bones together as there is no movement at the joint. Indeed in many rodents there is no joint and the malleus and incus are one bone. The most important factor changing the low pressure, high displacement vibrations of air into the low displacement vibrations suitable for driving cochlear fluids is the ratio of the areas of the tympanic membrane and the stapes footplate.

Therefore, the theory that these joints should participate in the rheumatoid process seems far less likely. Interestingly, however, this study and those of Reiter and Moffat all showed altered stiffness of the middle ear conducting mechanism. Goodwill, who examined the ossicles removed at necropsy from three rheumatoid patients, did not find nodules or erosive joint changes but instead fibrous tissue replacement of the long process of the incus.5 This finding is not uncommon in chronic otitis media and is thought to result from the fact that the long process of the incus has the most tenuous blood supply of all the ossicular elements and thus is most at risk from endarteritis obliterans secondary to infection with subsequent resorption of bone and replacement by fibrosis. Probably, the middle ear changes in rheumatoid arthritis are secondary to the associated vasculitis, which is already known to account for most of the other extra-articular manifestations.

A E CAMILLERI Department of Otolaryngology Glasgow Royal Infirmary 86 Castle Street Glasgow G4 0SF

ANKYLOSING SPONDYLITIS AND SELECTIVE IGA DEFICIENCY

SIR: We read with interest the account from Dr Herrero-Beaumont and colleagues of a patient with selective IGA deficiency and severe spondyloarthropathy.1 We report a further case of IGA deficiency in ankylosing spondylitis, a 40 year old woman, first noted inflammatory low back pain and stiffness at the age of 27. Over the following years she also experienced intermittent knee and ankle pain, each episode resolving without intervention or sequelae. Ankylosing spondylitis was diagnosed at 33, after referral to a rheumatologist. Sacroiliac radiographs showed bilateral marginal sclerosis and erosions, and she was HLA-B27 positive. There was no past or family history of uveitis, psoriasis, or other associated disease, and serological tests for antinuclear antibodies and rheumatoid factor were consistently negative. Treatment consisted of non-steroidal anti-inflammatory drugs, analgesics, and exercise. The course of her disease was remarkable and did not feature the destructive peripheral arthropathy noted in the previous reports. At her last assessment her disease activity was subjectively moderate, with no morning stiffness and with lower back pain rated as 26% on maximum on a visual analogue scale. The mirror Schöber index was 5 cm, chest expansion 2 cm, and she had no peripheral synovitis or enthesitis. Erythrocyte sedimentation rate was 43 mm/1 h, C reactive protein 27 mg/l (normal<10).

The patient was otherwise healthy, and IGA deficiency was not suspected on clinical grounds. During serological studies of a sample of patients with ankylosing spondylitis (to be reported separately) she was noted to have barely detectable circulating IgA and IgG antibodies. Serum total IGA concentration was <0.2 g/l (normal range 0.8-3.9) by laser nephelometry, and <50 mg/l by enzyme linked immunosorbent assay (ELISA). By contrast, her IgA, IgG, IgM, IgD, and IgE levels were all normal.

Although selective IGA deficiency has been associated with recurrent mucosal infection and allergy, and with an increased incidence of several autoimmune disorders, most patients are asymptomatic.2 IGA deficiency affects about 0.2% of the adult white population,3 and ankylosing spondylitis up to 1%.4 By chance alone two the disorders will therefore occur together in 0.002% (one in 50 000 people), suggesting that there are around 1000 such patients in the United Kingdom. Unlike the four previously reported cases5 our patient did not have severe ankylosing spondylitis, and we argue against interpreting IGA deficiency as a poor prognostic factor in ankylosing spondylitis until more evidence has been accumulated.

At a mechanistic level the existence of such cases indicates that an intact IGA response is not essential to the pathogenesis of ankylosing spondylitis.

I L McLEAN Division of Immunological Medicine MRC Clinical Research Centre Harrow HA1 3UJ


Ankylosing spondylitis and selective IgA deficiency.

I L McLean, B L Kidd, P W Thompson and M I Cawley

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