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Arthritis of the middle ear in ankylosing spondylitis

Sir, We have read with interest the paper by Magaro, Ceresia, and Frustaci on arthritis of the middle ear in ankylosing spondylitis.1

The joints between the incus and the malleus, and between the incus and the stapes are of the diarthrodial type. The footplate of the stapes articulates with the walls of the oval window in a syndesmotic joint; this articulation is held together by the annular ligament.2 For all practical purposes the ossicular chain acts as a rigid piston which is suspended from the walls of the middle ear by tendons and ligaments. It transmits the excursions of the tympanic membrane in a columnar fashion, and only a gross defect in the tympanic membrane, fixation of the ossicular chain to the walls of the middle ear, or loss of movement of the joint between the footplate of the stapes and the oval window will result in significant conductive deafness. In fact in tympanoplasty surgery all or part of the ossicular chain is substituted by different types of autologous or artificial implants, and the auditory result is good provided that satisfactory continuity is established between the tympanic membrane and the oval window.

The exploratory and audiometric findings in the subject of Magaro, Ceresia, and Frustaci’s report are consistent with ankylosis of the stapediovestibular joint. Usually this condition is diagnosed as otosclerosis until proved otherwise by exploratory tympanotomy. The radiological changes in tomography of the middle ear are seldom conclusive.

Otosclerosis is a familial disease of the bone of the otic capsule, which often produces anomalous bone formation and becomes symptomatic when it encroaches upon the stapediovestibular joint, decreasing its mobility. This new bone has a characteristic histological appearance3 which clearly differs from the inflammatory enthesitic changes observed in ankylosing spondylitis.4 Otosclerosis is the commonest single cause of deafness in active adult life, and its incidence in the general population of the western hemisphere is approximately one in 200.5

Since the hearing loss of the reported1 patient is surgically treatable, it would have been interesting to know the macroscopic and pathological findings of his middle ear, before suggesting ankylosing spondylitis as the cause.

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References


Diabetic cheiroarthropathy in adult non-insulin-dependent diabetes

Sir, We read with interest the paper of Dr Fitzcharles and coworkers1 who reported a high prevalence of limited joint mobility in adult patients with non-insulin-dependent diabetes (NIDD). We also observed the same abnormality but its prevalence was very low. In 1983 we examined 102 patients with NIDD and only four showed limited joint mobility or sclerodactyly, or both.2 From January to July 1984 we found the same features in three out of 63 NIDD patients. All our diabetic patients with limited joint mobility or sclerodactyly, or both were affected by diabetic microangiopathy (retinopathy or nephropathy, or both), had good glycaemic control, and none of them was receiving insulin.

As Dr Fitzcharles and coworkers pointed out, this condition is fairly common in juvenile insulin-dependent diabetes, but it ‘has been described only infrequently in adults’ with either insulin- or non-insulin-dependent diabetes.3

Since the patients were assessed by the same method, the discrepancy between our observations may reflect genetic differences between different populations. It would be interesting to study the HLA system in these patients to determine whether different subsets exist, as well as to know the prevalence of systemic sclerosis in the population studied by Dr Fitzcharles and coworkers. It is apparent that the occurrence of joint contractures and sclerodactyly is similar in diabetes and systemic sclerosis, and microangiopathy is very important in the pathogenesis of both disorders; therefore further studies are necessary in order
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to establish a possible relationship between these two diseases.
Finally, though we did not find the same high prevalence of chieiroarthropathy in NIDD as did Fitzcharles et al., we do agree that there is an association between microvascular derangement and diabetic chieiroarthropathy which is independent of glycaemic control.

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Rupture of the spleen in rheumatoid arthritis

SIR, Haskard et al. reported, in the Annals1 two patients with rheumatoid arthritis, in whom spontaneous rupture of the spleen developed. Two similar cases were subsequently reported.2 The following case was seen in April 1980 and is reported in order to substantiate the relationship between rheumatoid arthritis and spontaneous rupture of the spleen.
The patient was a 56-year-old white female. In the winter of 1978 she developed pain and stiffness of her knees, neck, and shoulders, which lasted several months. She was then well until the winter of 1980, when she developed soreness and stiffness in the small joints of her hands, wrists, knees, ankles, and the small joints of her feet, which was particularly severe in the morning. She did not have fever, chills, night sweats, sicca symptoms, or Raynaud’s disease. She had a 12-year history of insulin-dependent diabetes.
Physical examination at that time disclosed an ill, white female. Her spleen was not palpable. There was tender diffuse swelling of the right second and fourth proximal interphalangeal joints. There was marked synovial thickening of all the metacarpophalangeal joints of the left hand. Both wrists were tender and painful on full flexion. Both knees contained small effusions. The ankles were tender. Her packed cell volume was 36%, leucocyte count 6.8×10⁹/l, platelet count 397×10⁹/l, erythrocyte sedimentation rate 50 mm/h, rheumatoid factor positive 1:320. Antinuclear antibody was positive 1:80 with a homogeneous pattern.

On 23 April 1980 she developed ‘indigestion’. She had not experienced abdominal trauma. At that time her packed cell volume was 28% and leucocyte count 6.4×10⁹/l. The next day she developed diffuse abdominal pain and rectal bleeding. Her packed cell volume was 18%. Sigmoidoscopy showed friable, bleeding rectal mucosa. Above 4-5 cm, the colonic mucosa was normal. Gastrographin upper gastrointestinal series showed displacement of the stomach by a mass in the left upper quadrant.
She underwent emergency laparotomy. At the time of laparotomy it was found that her abdomen was distended with fluid and blood and that there were blood clots in the left upper quadrant of the abdomen. The spleen was actively bleeding and she underwent splenectomy. Microscopic examination of the spleen failed to show fibrinoid necrosis or rheumatoid nodule formation. She then made an uneventful recovery. One month later she underwent repeat coloscopy, which demonstrated mild erythema of the rectum and no other abnormalities. Repeat coloscopy in October 1981 was normal.

This additional case supports the hypothesis that spontaneous rupture of the spleen can occur as a complication of rheumatoid arthritis. Awareness by physicians of this possibility in a patient with rheumatoid arthritis with acute abdominal pain would be of great importance in facilitating prompt diagnosis and surgical treatment.

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References

Note

Basic course in rheumatology

A one day meeting will be held at Guy’s Hospital on 15 March 1985. It is designed principally for junior staff not yet committed to the specialty, but doctors preparing for MRCP or in the early stages of specialty training may also find it useful. Details from Dr T Gibson, Department of Rheumatology, Guy’s Hospital, London, SE1 9RT.