LETTERS

Relation between ectopic ossification after total hip arthroplasty and activity of general inflammation in patients with ankylosing spondylitis

Ectopic ossification (EO) is a well known complication after total hip arthroplasty (THA). Some previous studies have considered ankylosing spondylitis (AS) a risk factor of EO, however other studies have refuted this association.¹ The reported incidence of EO varies widely in different series.²

We retrospectively studied a total of 20 primary THAs in 16 Japanese patients with definite AS. The age of the patients at surgery ranged from 30 to 64 years (mean 45). All THAs apart from three cases were performed using bone cement. Surgical exposure was achieved by the posterolateral approach. The length of follow up averaged 93 months (range 29 to 148). Compared with the incidence of EO in AS patients, we investigated 126 primary THAs (92 osteoarthritis (OA), 26 rheumatoid arthritis (RA) and eight aseptic necrosis of the femoral head (ANF)) in 107 patients (15 male/92 female, mean age 61 years; range 32 to 85, mean follow up period 63 months; range 28 to 128). Bone cement was used in 118 of these 126 cases of THA. We evaluated supine anteroposterior roentgenographic findings of the hips. EO was graded according to Brooker's classification.³

Six of 20 THA cases showed evident radiographic EO after THA in AS patients (Class I: 2 THAs, Class II: 3 THAs, Class III: 1 THA). The incidence of EO was 30.0%. In the control group, the incidence of EO was 32.5% (OA; 38.0%, RA; 15.4%, ANF; 25.0%), and there was no significant difference in the incidence of EO between the AS group and the control group using the χ^2 test ($\chi^2=0.051$, $p>0.01$, NS). Mean age of the ossification group in AS patients (6 THAs) was 42 years, and mean age of the non-ossification group in AS patients (14 THAs) was 46 years. Mean (SEM) erythrocyte sedimentation rate (ESR) was 33.0 (7.7) mm 1st h and mean (SEM) C reactive protein (CRP) was 43.0 (8.9) mg/l in the ossification group, while mean ESR was 19.8 (4.1) mm 1st h and mean CRP was 10.8 (2.7) mg/l in the non-ossification group. There was a significant correlation between CRP values and the formation of EO using the Mann-Whitney U test ($p<0.005$). However, there was no correlation between ESR and EO ($p=0.08$, NS), as previously reported.⁴ In patients with RA, no relation between the formation of EO and acute phase reactants was found.

Although the results of different series are difficult to compare simply because there were other risk factors and different criteria of EO were used, our study did not recognise a high incidence of EO in AS patients compared with that in the controls. Based on this result, we did not conclude that AS is a risk factor of EO. However, we found a significant correlation between the formation of EO and preoperative serum CRP values in

AS patients. Recently, significant increases in serum anti-collagen (type I, II, III and IV) antibody concentrations, especially in the IgA class, have been reported in AS patients.^{5,6} Type I collagen is present predominantly in bones, tendons, and joint capsules. After THA, type I collagen was found in the residue of excised joint capsules and bone fragments around hip joints. A significant correlation between the serum IgA concentration and CRP has been reported in AS patients.⁷ Therefore, this periarticular residue may trigger local inflammations and induce the formation of EO in active general inflammatory phases and at high serum CRP concentrations.

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Diplopia as the first manifestation of relapsing polychondritis

Relapsing polychondritis (RP) is an uncommon systemic disorder, which is characterised by recurrent inflammation and destruction of cartilage structures.¹ Auricular, nasal, ocular, articular, tracheobronchial, and cardiac impairment are well recognised manifestations of RP.¹⁻⁴ Central nervous system involvement is considered to be rare in RP, usually occurring in the course of the disease.¹⁻⁴ We have observed a new case, which is of particular interest, as the patient developed diplopia revealing RP.

A 69 year old man presented with an uveitis of the right eye in November 1997. The patient had no previous medical history, and he was successfully treated with both corticosteroid and antibiotic ointments. He was admitted to our hospital in February 1998 with a two day history of diplopia. Physical examination showed a right VI nerve palsy.

The patient had no fever and general physical examination was otherwise normal. Laboratory findings were: erythrocyte sedimentation rate 60 mm 1st h, C reactive protein 20 mg/l (normal <5), haemoglobin 7.3 mmol/l, white cell count $4.7 \times 10^9/l$, and platelets $580 \times 10^9/l$. Glycaemia, liver, and renal tests were normal. Cerebral computed tomography was within normal limits. Cerebrospinal fluid analysis revealed no abnormality, and notably IgG index was normal without oligoclonal bands; both Gram staining and bacterial cultures remained negative. Other investigations, including chest radiographs, electrocardiogram, transoesophageal echocardiography and cerebral arterial Doppler were also normal. Because of the previous history of uveitis of the right eye, an accessory salivary gland histological examination was carried out, which was normal without evidence of granulomas or vasculitis. Both thoracic computed tomography and abdominal ultrasound were also normal, and notably hepatosplenomegaly and lymphadenopathy were absent. On the ninth day, the patient developed both conjunctivitis and uveitis of the left eye, and then bilateral auricle chondritis and hoarseness of voice within one week. Autoantibody screening tests—that is, complement profile, rheumatoid factors, antinuclear antibodies, anticardiolipin and antiphospholipid antibodies, lupus-like anticoagulant, antineutrophil cytoplasmic antibodies, antichondroitin antibodies, and cryoglobulinaemia, were negative. Otorhinolaryngeal examination showed no abnormality. The diagnosis of RP was made because of the following manifestations: (a) recurrent bilateral uveitis, (b) bilateral auricle chondritis, and (c) hoarseness of voice related to laryngeal cartilage inflammation. The patient was successfully treated with prednisone 1 mg/kg daily, resulting in resolution of diplopia and uveitis of the left eye and in improvement of both left auricular chondritis and dysphonia. After a four month follow up, the patient remains free of ocular, auricular, laryngeal, and neurological features with prednisone at a dose of 45 mg daily.

Central nervous system involvement is rare in RP. In a series of 62 patients with RP, Zeuner *et al*⁵ have therefore found that only six patients (9%) had neurological manifestations. However, neurological impairment may lead to life threatening complications in patients with RP, for example, hemiplegia, cerebellar dysfunction, aseptic meningoencephalitis, rhombencephalitis or cerebral arterial aneurysms, and also to cranial nerve palsy, polyneuritis or myelitis.¹⁻¹⁰ The pathological mechanisms of neurological impairment are still not clearly understood, but it may be related to vasculitis of both small and medium sized vessels.¹⁻³ Our case is original in that neurological involvement—that is, diplopia related to right VI nerve palsy—revealed a typical RP. Because central nervous system manifestations may precede other systemic signs, we suggest therefore that when unexplained neurological symptoms are noted in patients, a clinical evaluation for an underlying and misdiagnosed RP may be done. Our findings further emphasise the importance of recognising such neurological complications at an early stage in RP, resulting in accurate diagnosis and management, and therefore avoiding the risk of severe sequelae. Finally, we postulate that clinical neurological examination should systematically be carried out in patients with RP, as

such an involvement may result in more follow up and aggressive treatment.

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Complete heart block caused by primary Sjögren's syndrome and hypopituitarism

CASE REPORT

Sjögren's syndrome is a systemic autoimmune disease characterised by lymphocytic infiltration of exocrine glands accompanied by a variety of extraglandular manifestations.¹ Endocrinological manifestations, however, are infrequently encountered with the exception of autoimmune thyroiditis.^{2,3}

Here we report a case of combined Sjögren's syndrome and hypopituitarism where the patient presented with complete heart block. To date the only two cases reported were published in Japanese.^{4,5}

A 76 year old woman was brought to the emergency room for syncope. She was diagnosed with bradycardia resulting from atrioventricular block with a ventricular rhythm of 30 bpm and a VVI pacemaker was placed to control the life threatening bradycardia. Her complaints were chronic fatigue, cold intolerance, decreased appetite, xerostomia, and dry eyes over the past five months. There was no history of arrhythmias nor was there any hint that an overdose of medication caused the atrioventricular conduction dis-

turbance. She gave birth to three children and menstruated until age 53 ruling out Sheehan's syndrome as a cause for hypopituitarism. Physical examination demonstrated decreased skin and nipple pigmentation, a hoarse voice, and loss of axillary and pubic hair.

Laboratory tests showed increased erythrocyte sedimentation rate of 66 mm 1st h, normochromic and normocytic anaemia, hyponatraemia, and hyperimmunoglobulinaemia. The leucocyte count was not raised, with a normal white cell differential and normal serum C reactive protein, ruling out acute inflammation.

As the patient's complaints and physical findings pointed towards a complex endocrine disorder, several tests were conducted. Serum concentrations of free thyroxine (7.1 pmol/l (normal 9.0-24.5)), free triiodothyronine (4.8 pmol/l (normal 3.4-7.00)), thyrotropin basal (1.1 mU/l (normal 0.17-2.9)), and basal prolactin (2.1 ng/ml (normal 2-20)) were near normal, whereas the stimulation with thyrotropin releasing hormone was blunted for thyrotropin (1.76 mU/l) and prolactin (2.3 ng/ml), indicating a final insufficiency of these regulatory pathways. Furthermore, basal serum cortisol and adrenocorticotropin concentrations were 4.06 µg/dl (normal 4.4-17.4) and 5.7 pg/ml (normal 7-74) but failed to rise upon appropriate stimulation upon corticotropin releasing hormone testing. Basal serum concentrations of luteinising hormone (< 0.5 mIU/ml (normal 10.8-61)) and follicle stimulating hormone (0.9 mIU/ml (normal 35-151)) were found to be diminished. The serum concentration of growth hormone (0.3 µIU/ml (normal <1-10)) also did not respond to stimulation with arginine. The plasma vasopressin was slightly increased (7.1 pg/ml (normal 1.5-6)). These findings are consistent with hypopituitarism accompanied by inappropriate secretion of vasopressin (SIADH). Autoantibodies against thyroid microsomal antigen, thyroglobulin, and TSH receptor were not detected. However, increased antibody titres against smooth muscle (1:8) and against parietal cells (1:4) were indicative of autoimmune hypophysitis.⁶ Computed tomography did not show any pituitary abnormalities. Because of the implanted pacemaker, magnetic resonance imaging could not be performed.

The diagnosis of Sjögren's syndrome was established by xerostomia, keratoconjunctivitis sicca, positive Schirmer test (right 3 mm/5 min, left 2 mm/5 min) positive ANA (1:64) with anti-60kD Ro(SS-A)/La(SS-B) antibodies meeting the European study group classification criteria.⁷ A salivary gland biopsy was considered but not performed because the patient did not give her consent. Rheumatoid factor was not detectable.

The patient was given a substitution regimen of 7.5 mg/d prednisolone and 75 µg/d L-thyroxine and 400 mg/d hydroxychloroquine for Sjögren's syndrome. Her symptoms improved after four weeks. Her electrocardiographic readings returned to a normal sinus rhythm. Hydroxychloroquine was discontinued later because it did not further improve the clinical symptoms.

Interactions between the neuroendocrine and immune system are increasingly appreciated.⁸ Our case report suggests that the pituitary gland may be another site of lymphocyte infiltration in Sjögren's syndrome. This could eventually cause pituitary dysfunction, possibly because of local im-

mune cell mediated tissue injury or autoantibodies, or both. Both autoantibodies in Sjögren's syndrome⁹ and hypothyroidism¹⁰ resulting from hypopituitarism have been described to cause heart blocks. The condition that was predominant in our case cannot be determined.

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Gut feeling

The gut as a causative factor in rheumatic disease has fascinated many researchers and physicians. We present an unexpected link between gut and arthritis and report an exceptional case of inflammatory seronegative arthritis that happened to be associated with chronic appendicitis.

A 52 year old, otherwise healthy woman presented with three months of migratory arthritis, which turned during the fourth month into an additive course involving symmetrically the wrists, ankles, metacarpophalangeal (MCP) joints, and left shoulder. She also reported morning stiffness and fatigue of more than one hour. She denied that she had had fever, skin eruption, psoriasis, aphtae, gastrointestinal or genitourinary symptoms or a familial history of rheumatic conditions, psoriasis or inflammatory bowel diseases. Physical examination was normal except for symmetric synovitis involving both ankles and wrists, the left 3rd and 4th and the right 2nd MCP joints. Laboratory tests disclosed an erythrocyte sedimentation rate (ESR) of 25 mm 1st h; serum C reactive protein, haemoglobin and thrombocytes count

were normal; white blood cell count was $13\,700/\text{mm}^3$, with 79% neutrophils, 13.8% lymphocytes, 5.6% monocytes, 0.8% eosinophils and 0.8% basophils. The results of blood chemistry tests including uric acid, calcium, phosphorus, liver enzymes, and creatinine were normal. Urine analysis disclosed mild microhaematuria. Rheumatoid factor and antinuclear antibodies were negative. Radiographs of the hands and feet showed soft tissue swelling and a chest radiograph was normal.

The patient was treated with naproxen with partial improvement of her arthritis. Ten days after admission, she complained of severe sudden abdominal pain spreading within an hour from the epigastrium to the entire abdomen. Her fever rose to 38.5°C . The abdomen was slightly more tender in the right lower quadrant without defence or rebound. Normal peristalsis sounds were heard. Abdominal plain radiography was normal. ESR increased to 60 mm 1st h and the white blood cell count to $29\,000/\text{mm}^3$. Urine analysis showed microhaematuria. Blood cultures were later found to be sterile. The surgeon did not diagnose an acute surgical abdomen; gynaecological examination was normal. The patient recalled a similar episode of transient abdominal pain two months previously. Cefuroxime was prescribed. Within 24 hours, her abdominal symptoms and fever resolved completely. An abdominal ultrasound study and a cystoscopy—because of persistent microhaematuria—were normal. Repeated urine cultures were found to be sterile. Beside the above mentioned brief episode, the patient remained completely asymptomatic regarding the abdominal symptoms, but despite naproxen, she still had active polyarthritis. Weekly methotrexate at a dose of 7.5 mg was started with the working diagnosis of seronegative rheumatoid arthritis (RA). After receiving five doses of methotrexate, in a routine outpatient visit, her joint symptoms were slightly improved but she reported another episode of acute abdominal pain two weeks previously that resolved spontaneously within hours. A gastrointestinal barium study was ordered. A week later, the patient was admitted to the emergency ward with severe

abdominal pain and fever of 39°C . On laparotomy a perforated appendix with local adhesions was identified and removed. Pathological studies confirmed acute suppurative appendicitis without evidence of Crohn's disease or granulomatous lesions. The day after the operation, the patient experienced a dramatic resolution of her polyarthritis. Methotrexate was stopped at the time of appendectomy. Since then and during two years of further follow up the patient is still free of any musculoskeletal symptoms.

Although the relation between an acute to chronic appendicitis and polyarthritis in this patient could have been serendipitous, the fact that the established RA-like syndrome resolved immediately, completely, and permanently after the appendectomy strongly suggests a causative link between the two entities.

Retrospectively, the patient had at least four episodes of acute appendicitis—the fourth one evolving into perforation and acute localised appendicitis. During the past 15 years several convincing reviews have established that besides acute appendicitis, the 'appendicitis repertoire' also includes recurrent bouts of acute as well as longstanding appendicitis.^{1,2} Nevertheless, our awareness of atypical forms of appendicitis is still low. Appendicitis as a cause of rheumatic symptoms has been described in two cases.^{3,4} In both of them, the abdominal presentation was atypical while the rheumatic symptoms were prominent. In one case, appendicitis, later found to be granulomatous, was found on abdominal computed tomography examination of a patient with a three month duration migratory arthritis and septicaemia—without abdominal symptoms. The second case was a 10 year old girl with 19 months of episodic colicky abdominal pain and nine months migratory polyarthritis. The underlying process was found to be an acute on chronic appendicitis that finally perforated. Appendectomy in both these cases resulted in prompt resolution of the rheumatic symptoms.

The flora of appendicitis does not qualitatively differ from that of the normal appendix, *Escherichia coli* and *Bacteroides fragilis* being the most commonly encountered organism.⁵

A possible link between appendicitis and arthritis could be *Yersinia*. Although pseudo-appendicular syndrome is a prevalent manifestation of *Yersinia* in adults,⁵ occasional reports of true appendicitis do exist.⁶ On the other hand, a known complication of yersiniosis is reactive arthritis⁷ typically persisting 1–4 months after resolution of the gastrointestinal symptoms.⁸

Sometimes, trivial diseases may offer a diagnostic challenge. Polyarthritis is a frequent medical problem and acute appendicitis is one of the most banal surgical conditions. The lesson to be learnt from this case report is the challenge to the physician of recognising this unexpected yet possible encounter between the two.

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