MATTERS ARISING

Scoliosis and Trendelenburg sign in a painting by P P Rubens

In an article on Rubens' painting "The Three Graces" Dequeker suggests that hypermobility is a medical explanation of the seeming scoliosis and Trendelenburg sign in the middle figure.3 However, this does not exclude the possibility of an artistic phenomenon without medical reference.

Sculptors in classical Greek and Roman periods often used the contrapposto posture. In this, by putting most weight on one leg, the other leg can be shown in a relaxed and semi-flexed position. This undulating between tension and relaxation will animate the figure. A person with normal muscular function and a normal back can perfectly well pose in this way with relaxed hip abductors on the weightbearing side, a descending hip on the opposite side, and a compensating scoliotic posture. This posture is facilitated by support from the arm as in Rubens' painting. If the person takes a forward step, relaxation of the muscles of the weightbearing hip can no longer be maintained, and the positive Trendelenburg sign will disappear.

In the Renaissance period the use of this contrapposto posture was revived. During his stay in Rome Rubens eagerly studied the then recently excavated Laokoon sculpture with its three distorted figures.2 He often used such distorted postures in his paintings to give the impression of vigorous muscular characters capable of performing great tasks. The best example is probably "The Debarcation at Marseilles" in the Maria de Medici cycle from 1622 to 1625 for the Luxembourg Palace in Paris.3 Here, three young women, nereides, with curved muscular backs at the bottom of the picture nearly seem to carry the ship of Maria de Medici.

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References

Author's response

Dr Hansen's remarks about our recent article in the Annals are pertinent and have to be taken as a cautionary explanation for the observed functional scoliosis and positive Trendelenburg sign. I am grateful for this artistic-historical information. However, this does not exclude the possible diagnosis of benign familial hypermobility syndrome. In several other paintings by Rubens, where the three sisters (sisters) of the three graces are represented, clinical signs of hypermobility can be seen. In the painting "The Judgement of Paris" (London National Gallery) a positive Trendelenburg sign and scoliosis can be seen in two blond sisters who are now in a walking position without support. In one of them the right wrist is in 90° hyperflexion. In the painting "Sine Cerere et Baccho friget Venus" (Brussels Koninklijke Musea voor Schone Kunsten), subluxation of the left wrist is seen in the dark blond sister and hyperextension of the distal interphalangeal (DIP) joint of the fourth finger in another sister with brown hair. Hyperextension of a DIP and metacarpophalangeal joint and hyperflexion of a wrist joint is also seen in the brown haired sister of the painting "The Musa and Saints" (Antwerp, Sint-Jacobskerk).

I, as well as Sven Hansen, am fully aware that errors of diagnosis are commonly made either by seeing disease where none exists or by interpreting at face value a pathological appearance that is only the expression of an artistic conception. The observations made in P P Rubens' painting, representing the sitters for "the graces" painting who are Rubens' second wife Helen Froment and her younger sisters, are very suggestive of the diagnosis of benign familial hypermobility syndrome and not a purely artistic phenomenon.

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Comparison of WOMAC with SF-36 for OA of the knee or hip

Angst et al compared WOMAC with the SF-36 as tools to assess the outcome of a three to four week inpatient rehabilitation programme for people with osteoarthritis of the knee or hip.4 They concluded that both instruments capture improvement in pain levels, but functional improvement can be better detected by WOMAC. We have reservations about the use of SF-36 in this context.

We too provide residential musculoskeletal rehabilitation of usually three weeks' duration in our residential rehabilitation programme for people with osteoarthritis of the knee or hip. They concluded that both instruments capture improvement in pain levels, but functional improvement can be better detected by WOMAC. We have reservations about the use of SF-36 in this context.

We provide residential musculoskeletal rehabilitation of usually three weeks' duration and have been searching for a suitable instrument to assess quality of life at the time of discharge from our programme. We have rejected the SF-36 for the following reasons.

A large majority of the questions in the SF-36 relate to the subject's experience over the past four weeks. The condition of most of our patients improves considerably over the three weeks of treatment. It is therefore not appropriate to ask how they have been over the previous four weeks. We note that the period of treatment in the report by Angst et al varies from three to four weeks.

It is not only the length of time which makes the use of the SF-36 inappropriate in this setting, many of the questions assume the subject is living an everyday life. For example, inquiry is made about "both work outside the home and housework", "other activities at home", and "normal social activities with family, friends, neighbours, or groups".

Obviously if a person is devoting time and energy to an inpatient musculoskeletal rehabilitation programme they are in no position to be truly engaged in any of these work or social activities.

While the outcomes of our similar residential rehabilitation programme for people with osteoarthritis are in accordance with those of Angst et al, we do not feel it is appropriate to use the SF-36 to measure improvement at discharge. It is of course quite reasonable to use it before admission and at three or six months' follow up.

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Authors' response

In their letter commenting on our article,1 Jones and Leighton deal with two major problems which might arise in the application of the SF-36 to inpatients. We would like to stimulate discussion about this issue by our following response.

The first problem concerns the fact that many of the SF-36 items ask about subjective health status over the past four weeks at the time of administration of the questionnaire. Jones and Leighton suggest, therefore, that the results at the end of an inpatient rehabilitation (three or four weeks) reflect some kind of an average of the health status during that rehabilitation period in which most of the patients have improved considerably. We agree that this assessment is unlikely to show the maximum of improvement that may be expected at the day of discharge from the clinic or shortly thereafter. However, one can assume that the result overestimates the health status for the time periods close to the day of administration of the questionnaire (for example, at the day of discharge) owing to the fact that the response is based on the patient's memory. The same problem, but in the opposite direction, would arise if we administered the SF-36 two or four weeks after the day of discharge. Thus we possibly miss the maximal effect, which may last only a few days, but we do obtain an assessment of a certain time period, which is likely to be more valid and more clinically important than that of a single day. To take account of this point, we also reported results...
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aβ2-GPI and aCL were measured by enzyme linked immunosorbent assay (ELISA; Inova Diagnostics, Inc, San Diego, CA and Hemagen Diagnostics, Inc Waltham, MA, respectively). Commercially obtained HEp-2 slides (Immuno Concepts, Sacramento, CA) were used for indirect immunofluorescence (IIF). Samples were tested for antibodies to topoisomerase 1 (Scl-70), U1 ribonucleoprotein (U1-RNP), and Sjögren’s syndrome antigen A and B (SS-A/SS-B) by double immunodiffusion.

Student’s t test (two tailed) was used for comparison of means, and Fisher’s exact test (two tailed) for analysis of frequencies. Age distributions were compared with the Mann-Whitney test because healthy controls described their age in decades, not years.

Table 1 summarises the demographics and laboratory data for the study group. The patients with SSc were significantly older than both the healthy controls (p=0.005) and the patients with RP (p=0.02). All mean laboratory values were within the normal range. Figure 1 compares the values for tests among the study groups except aβ2-GPI IgG, where all tests were negative. IgM aβ2-GPI were found in two patients with SSc (8%), one patient with RP (4%), and none of the healthy controls (p>0.05). Three (12%) patients with SSc, five (22%) with RP, and one (5%) of the healthy controls had positive tests for IgG or IgM anti-2GPI (p>0.05). The sera positive for aCL were not the same as those positive for aβ2-GPI.

The two patients with SSc positive for aβ2-GPI had mean disease duration of 19 months; both had cutaneous manifestations and one had hypoa with decreased carbon monoxide transfer factor (TLco). The three patients with SSc and aCL had mean disease duration of 112 months. One had hypoa (with normal TLco and non-restrictive pulmonary function tests), one had restrictive lung disease and digital ulcers, and one had oesophageal hypomotility. None of the study participants had thrombocytopathy or a history of deep venous thrombosis. Twenty two per cent of the group with Raynaud’s disease had aCL, which is higher than the 8.7% reported by Vayssairat et al. Patients with positive tests did not differ from those who had negative clinical manifestations or laboratory values.

All of the patients with SSc and RP and 13% of the healthy controls had positive IF tests on HEp-2 substrates. None of the patients with SSc had antibodies to topoisomerase 1 (Scl-70) or SS-A/SS-B. No IIF pattern correlated with aβ2-GPI or aCL.

In our study we found that the frequency of antibodies to β2-GPI and aCL was low in scleroderma, 8% and 12% respectively. There were no clear clinical or laboratory correlations with a positive test.

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References
Recurrence of reactive arthritis after a booster dose of tetanus toxoid

We report the case of a 24 year old man who developed a recurrence of reactive arthritis after receiving a booster of tetanus toxoid.

Case report

A 24 year old man presented with acute swelling and pain over the right ankle. Two weeks before presentation, he had been given a booster tetanus toxoid vaccination. Within a few days of the injection, he felt pain and noticed swelling of the right ankle. The swelling and pain worsened, such that at presentation he was walking with a pronounced antalgic gait. There was no preceding history of trauma, infection, or any past history of psoriasis, iritis, inflammatory bowel disease, or inflammatory back pain. There was no family history of ankylosing spondylitis.

Three years previously he had presented to another rheumatologist with acute synovitis of the right ankle, metatarso-phalangeal joint of the left big toe, and tenosynovitis of the long flexor of the right middle finger. This was associated with conjunctivitis and chlamydial urethritis, with raised levels of specific IgM. His plasma viscosity was raised at 1.69 mPa·s; haemoglobin was 139 g/l, rheumatoid factor negative, and HLA-B27 positive. Plain radiographs of the left ankle, foot, and right hand showed no abnormalities. He was treated with indometacin and an intra-articular injection of triamcinolone to the left ankle as well as minocycline for himself and his partner. The conjunctivitis settled within a few days, the urethritis within one week, and the arthritis resolved. He was given indometacin as an intra-articular injection at the right ankle, which took six months before becoming quiescent, by which time he was able to stop the indometacin. Chlamydia IgG was negative by this time.

Physical examination at the second presentation showed swelling of the right ankle joint, with tenderness and synovial thickening. The subtaloid, mid-tarsal, and metatarso-phalangeal joints were fully mobile with no swelling. The erythrocyte sedimentation rate was raised at 36 mm/1st h.

Initially, triamcinolone was injected into the right ankle, indometacin 50 mg three times a day was prescribed, and he was given elbow crutches to stop him weight bearing on the right leg. Two weeks later there was partial improvement. Prednisolone (20 mg a day decreasing by 5 mg weekly) and enteric coated sulphasalazine (500 mg twice daily) were added. One month later the ankle synovitis and pain had settled.

Discussion

Several strands of evidence link different vaccine-induced development of a spectrum of arthritides. Often, a close temporal relation between vaccination and the onset of arthritis exists, allowing an inference about the influence of a particular vaccine. On this basis, tetanus toxoid injection has been associated with the development of rheumatoid arthritis, comprising a symmetrical inflammatory polyarthritis and positive rheumatoid factor, with negative hepatitis B vaccine-induced arthritis. However, the mechanisms underlying arthritis associated with vaccination are not yet fully understood. A cross reaction between bacterial lipopolysaccharide epitopes and synovial antigen, leading to an idiotype-anti-idiotype immunological response enhanced by HLA-B27 expression, may provide one model.

The mechanisms underlying arthritis associated with vaccination are not clear, but there is sufficient evidence to suggest that some vaccines may cause joint disease or autoimmune arthritis in a susceptible person. Although the mechanisms of vaccination-induced arthritis are not clear, there is sufficient evidence to suggest that some vaccines may cause joint disease or adversely affect pre-existing joint problems. It would therefore be unwise to warn patients awaiting vaccination about the possible adverse effect on joint symptoms.

The spectrum of arthritis associated with vaccination is illustrated by the induction of large joint monarticular by combined diphtheria, poliomyelitis, and tetanus toxoid vaccine. In one of these cases, synovectomy was curative until a booster vaccination five years later caused recurrence; and, indeed, it has been suggested that rechallenge with vaccine may be associated with more severe symptoms. Further evidence for the role of vaccines in arthritis comes from monitoring of adverse drug reactions, with one survey indicating a causal link between rubella vaccination and acute and chronic arthritis, especially in women.

The spectrum of arthritis associated with vaccination are not yet fully understood. A cross reaction between bacterial lipopolysaccharide epitopes and synovial antigen, leading to an idiotype-anti-idiotype immunological response enhanced by HLA-B27 expression, may provide one model. However, HLA-B27 expression is not a prerequisite for arthritis linked to vaccines although its presence may predict a more prolonged and severe course. Vaccines may also trigger autoimmune responses by binding to critical antigen binding clefts on the major histocompatibility complex class II molecule, thereby triggering T cell proliferation. The impact of vaccination associated with vaccine can be severe, with prolonged and significant morbidity lasting many months. Hassan and Oldham reported Reiter's syndrome with joint pains and conjunctivitis lasting many months, whereas Bracci and Zoppii additionally reported fevers and lymphadenopathy with the hepatitis B surface antigen vaccine (Engerix B). As with our case, appropriate treatment, including non-steroidal anti-inflammatory drugs and intra-articular or oral steroids, can be useful in limiting the duration and degree of symptoms.

Vaccination has also been shown to cause necrotising vasculitis. Leucocytoclastic vasculitis has been induced most often, but polyarteritis nodosa-like and systemic vasculitides have also been reported in a few instances. In children, two other syndromes may occur after rubella vaccination (and natural infection): (a) the "arm syndrome", in which brachial radiculoneuropathy causes arm and hand pain, and arthritis that are worse at night; (b) "catcher's crouch", a lumbar radiculoneuropathy causing pain in the popliteal fossa on arising in the morning, which is exacerbated by knee extension and improves in a "catcher's crouch" position. Both syndromes occur one to two months after vaccination. Although the initial episode may last up to two months, relapses may occur for up to a year, eventually resolving completely without permanent sequelae.

Our case highlights a relationship between vaccination and arthritis and the ability of vaccine to trigger a reactive arthritis in a susceptible person. Although the mechanisms of vaccination-induced arthritis are not clear, there is sufficient evidence to suggest that some vaccines may cause joint disease or adversely affect pre-existing joint problems. It would therefore be unwise to warn patients awaiting vaccination about the possible adverse effect on joint symptoms.

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References


Asymptomatic splenic infarction in Wegener's granulomatosis

Wegener's granulomatosis (WG) is a necrotising, granulomatous vasculitis that classically involves the clinicopathological triad of upper and lower respiratory tracts and the kidney. Less frequently, the disease may affect other organs as well. Serious and occasionally fatal complications are often associated with WG. The spleen was commonly affected: 78–100% of patients had splenic lesions with a combination of necrosis, vasculitis, and granuloma formation. Clinically, such asymptomatic splenic disease is rare, however. We wish to report briefly the case of a 47 year old woman who presented with manifestations of classical WG and radiological evidence of splenic infarcts.
Case report
A 47 year old woman during the past month developed fevers to 38.6°C associated with weight loss, diffuse arthralgias, anaemia, and erythrocyte sedimentation rate of more than 100 mm/1st h. During the past three months she complained of nasal congestion and occasional episodes of epistaxis. A chest x ray examination showed a left upper lobe density, and her family doctor prescribed oral amoxicillin in combination with clarithromycin, without improvement. Ten days before admission gross haematuria was noted and a freshly collected urinary specimen showed the presence of red blood cell casts. Renal function declined rapidly with a serum creatinine level of 600 µmol/l and the patient was referred to the hospital for further investigation. A positive cANCA titre (1/160) was found using the indirect fluorescence technique and a positive antiproteinase 3 result on enzyme linked immunosorbent assay (ELISA; 66 U/ml). WG was considered on the basis of ANCA analysis results and the multisystemic nature of the disease and a percutaneous renal biopsy was performed under computed tomography (CT) guidance and evaluation of the renal biopsy specimens with light microscopy and immunofluorescence showed an acute necrotising segmental pauci-immune glomerulonephritis with crescent formation in more than 50% of the glomeruli. An impressive and rather unexpected CT finding was the presence of well defined areas of low attenuation within the spleen, consistent with splenic infarction (fig 1). A search for lupus anticoagulant and antiphospholipid antibodies was negative and no abnormalities of blood clotting could be detected. A transoesophageal echocardiogram failed to detect cardiac sources of emboli.

Treatment was started with 500 mg methylprednisolone intravenously per day for three consecutive days together with cyclophosphamide at a dose of 2 mg/kg body weight per day orally. After three days methylprednisolone was continued at 48 mg/day orally. Pulmonary and renal function progressively recovered and serum creatinine was 150 µmol/l on discharge.

Today, three years after the initial presentation, the patient is in stable remission and serum creatinine is 125 µmol/l. A recently performed CT scan showed a considerable volume reduction and scarring of the spleen (fig 2).

Discussion
Splenectomy in WG has included such abnormalities as splenomegaly, capsular adhesion, impaired splenic function, and infarcts. Infarction may occur as a result of a distal occlusion of the splenic artery or its branches, because splenic parenchymal arterial end vessels that do not communicate with one another. There are few reports on splenic infarction on post mortem in patients with WG. Histological examination frequently shows massive or multiple areas of splenic necrosis, usually associated with extensive central arteritis, splenic trabeculitis, follicular arterioliitis and necrosis, disseminated visceral granulomata, and capsulitis. On CT, splenic infarcts classically and more commonly appear as peripheral, well defined, wedge shaped areas of low attenuation. However, other patterns of infarction have been recognised. These include multiple heterogeneous low attenuation lesions; regions of normal enhancement centrally with peripheral low attenuation; and large, low attenuation hypodense lesions that may have a rim of enhancing tissue peripherally. Examination with ultrasound in combination with duplex sonography of splenic blood supply permits non-invasive diagnosis of splenic infarction. The diagnosis can be confirmed by magnetic resonance imaging or CT scan, which permits assessment of the extent of splenic infarction.

Splenectomy in WG may be more prevalent than previously believed. Pain in the left upper quadrant and left shoulder and fever may be present after splenic infarction, but many patients remain asymptomatic. Consequently, cross sectional imaging is not often carried out and the lesion may frequently go unrecognised. Unless there are signs of imminent rupture of the spleen or bleeding, a conservative approach is justified. In the long term these patients may be more susceptible to pneumococcal infection because of the functionally asplenic condition. This possibility provides further help in the diagnosis of this rare condition in vivo.

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References

Two cases of Mycobacterium avium septic arthritis
The "unusual and memorable" case reported by Ter Borg and Termette serves as a useful reminder that atypical mycobacterial infections, although uncommon, need to be considered in immunocompromised patients. We present here two case reports of patients with pre-existing rheumatic disease receiving immunosuppressive treatment, who developed septic arthritis due to Mycobacterium avium intracellulare.

Case one
A 51 year old woman presented in 1999 with Raynaud's phenomenon, facial telangiectasia, sclerodactyly, and a positive antinuclear antibody. She complained of exertional dyspnoea, and subsequent high resolution computed tomography of the chest disclosed a ground glass appearance, indicative of active alveolitis. A diagnosis of scleroderma with interstitial lung disease was made, and treatment with prednisolone 30 mg and azathioprine 100 mg daily was started. In August 2000, she complained of pain and stiffness in the left shoulder, and this was treated with an intra-articular steroid. Six months later, she developed a left shoulder effusion, from which Gram stain and initial bacterial cultures of the fluid were sterile. On September 18, 2002, she developed septic arthritis due to Mycobacterium avium intracellulare.
Successful radiosynoviorthesis of an olecranon bursitis in psoriatic arthritis

We describe the case of a 45 year old male patient who for more than 10 years had psoriasis with typical manifestations at knees and elbows. The family recalled psoriasis of the grandfather. Without any trauma or special straining, an olecranon bursitis and an arthritis of the left elbow developed in 1999 as the initial manifestation of psoriatic arthritis. Three months after developing the bursitis, the patient came to the rheumatologic outpatient clinic for his first visit.

The clinical findings showed a patient with good general condition (height 186 cm; weight 93 kg), blood pressure 120/80 mm Hg, rhytmic pulse rate 68 beats/min; psoriatic skin lesions at knees and elbows; no reduction of spine mobility. The left elbow showed an olecranon bursitis with a diameter of 50 mm. The remaining musculoskeletal system was not affected.

The laboratory results were within the normal ranges, HLA-B27 was negative, anti-nuclear antibodies negative, functional tests of liver and kidney were normal.

Radiographic findings showed that sacroiliac joints and the left elbow joint were normal. Sonography showed an olecranon bursitis with a large effusion (fig 1A).

Diclofenac 100 mg twice daily was given for the first two weeks but did not produce any effect. After that, the olecranon bursa was punctured aseptically and a crystal suspension of 10 mg trimacinolone hexacetonide was injected. Two days later, the bursitis relapsed completely. Further therapeutic options were surgical bursectomy or, alternatively, radiation synovectomy. After having received complete information, the patient gave his consent to treatment by radiosynoviorhesis. After aspiration of 9 ml of a serous effusion, 55 MBq thinem-186 was instilled into the olecranon bursa, and then, to avoid radiosynovitis, 5 mg trimacinolone was injected. Radiosynovitis scanning immediately after the injection and three days later showed that the radiouclide was distributed uniformly in the bursa. There were no local signs of an infection.

A physical examination three months after radiation synovectomy of the olecranon bursitis showed regular clinical findings. Arthrosonographic results had also normalised (fig 1B). Even six months later the bursitis was not reactivated.

Radiation synovectomy is often used as an alternative, or in addition to, surgical synovectomy. Definite indications are chronic persisting synovitis, intermittent hydrops, relaxing synovitis after surgical synovectomy, deforming arthroarthropathy, and activated osteoarthritis resistant to other treatments. Some studies have reported successful concomitant treatment of Baker’s cysts in the treatment of goutarthritis, but radiosynovioorthesis with rhenium-186 for the treatment of Baker’s cysts is not usual. It is possible, however, by infusion of a radioisotope into the knee joint, but the popliteal cyst must not be punctured directly. Due notice should be taken of contraindications.

Other reports disagree about the success rates of radiosynovioorthesis in treating psoriatic arthritis compared with rheumatoid arthritis. A few years ago, only patients aged over 40 were treated with radiosynovioorthesis. Today, this treatment is used in an increasing number of younger patients. The success rate for radiosynovioorthesis of olecranon bursitis is between 50 and 80%, depending on the localisation and the amount of inflammatory activity.

Up to now, no studies of the treatment of chronic inflammation of the bursa by radiosynovioorthesis have been reported. In our patient, neither the treatment with a non-steroidal anti-inflammatory drug (200 mg diclofenac daily) nor the local treatment with trimacinolone hexacetonide after a decompression aspiration led to improvement. An alternative to surgical bursectomy, radiosynovioorthesis with rhenium-186 was performed. The patient improved quickly and started working again the following day. The follow up examinations, after intervals of three and nine months, confirmed the continuing success.

As far as we know this is one of the first reports on radiosynovioorthesis in an isolated bursitis. This case gives cause for hope that radiosynovioorthesis represents a successful alternative treatment to operational intervention for chronic inflammation of the bursa.

References


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Figure 1 Sonography of the left elbow (A) showing an olecranon bursitis (58.1 mm; 17.7 mm; 3.3 mm; 2.5 mm) and (B) three months after radiation synovectomy.
D-penicillamine 500 mg daily and methyl-remained stable with treatment with the same hospital for SSc, and her condition three weeks. Since the age of 22, she had been an episode of paraesthesiae of her right leg, diplopia. A year earlier the patient had had an episode with 10 days' history of vertigo and vision that broke up time eye tests were normal. These weakness or sensory loss. There was no evidence for keratoconjunctivitis sicca, as Schirmer's I, rose bengal, and break up time eye tests were normal. These had been performed several times previously before the present admission. Furthermore, the patient had never complained of optic neuropathy, is rather in favour of MS, although this abnormality has been reported in SSc.3 On the other hand, a significant percentage of patients with systemic lupus erythematosus may present with demyelinating disease, and, some of them with oligoclonal banding in the CSF.4 Brain or spinal cord disease, or both, with clinical features and laboratory findings indistinguishable from MS has been reported in Sjögren's syndrome too, although CNS involvement in this syndrome has been a matter of serious debate.5 6

In the absence of guidelines for the management of such patients, we considered our patient as a case of classical MS and, therefore, she was not deprived of the possible benefit of a disease modifying treatment, such as interferon β.

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Multiple sclerosis in the course of systemic sclerosis

We describe the case of a young woman with longstanding systemic sclerosis (SSc), who later developed multiple sclerosis (MS), and discuss the possible explanations for this rare co-occurrence.

A 30 year old white woman was admitted to the department of neurology of our institution with 10 days' history of vertigo and diplopia. A year earlier the patient had had an episode of paraesthesiae of her right leg, which resolved spontaneously within two to three weeks. Since the age of 22, she had been under the care of the rheumatology service of the same hospital for SSc, and her condition remained stable with treatment with d-penicillamine 500 mg daily and methylprednisolone 2 mg daily.

Clinical examination showed an alert woman with normal vital signs and typical appearance of sclerodermat—that is, tightness and atrophy of the skin of her face and hands with contractions of her fingers. Examination of the lungs, heart, and abdomen showed no abnormality. Fundoscopy disclosed temporal pallor bilaterally. There was vertical nystagmus on upward gaze and diplopia on looking to the right, without apparent atheromaoptalmoplegia. Deep tendon reflexes were brisk and abdominal reflexes were absent bilaterally. An extensor plantar response was seen on the right but no muscle weakness or sensory loss.

There was no evidence for keratoconjunctivitis sicca, as Schirmer's I, rose bengal, and break up time eye tests were normal. These had been performed several times previously before the present admission. Furthermore, the patient had never complained of xenostomia.

Routine blood tests were normal. Serology showed positive antinuclear antibodies at a titre of 1/640, of the fine speckled pattern, and positive anti-ScI70 antibodies. Antibodies to cardiolipin and the other extractable nuclear antigens, including Ro/SSA, La/SSB, Sm, and nRNP, were absent, as they had been on several occasions in the past.

Visual evoked potentials were abnormal bilaterally. Cerebrospinal fluid (CSF) analysis disclosed increased intrathecal IgG synthesis (IgG index 0.88, normal <0.66) and oligoclonal bands. Magnetic resonance imaging (MRI) studies showed several abnormalities of the brain and the cervical cord (fig 1).

A five day trial of intravenous methylprednisolone 500 mgday resulted in moderate relief of her symptoms and treatment was started with interferon β to prevent progression of the neurological process. At present, the patient has been receiving interferon β for two years and there is no evidence of any further neurological compromise.

One can suggest three possibilities for the coexistence of the neurological syndrome and the SSc in this patient. Firstly, MS occurring independently from SSc might account for the neurological deficits, given the laboratory findings and the patient's sex and age, and the prevalence of MS in the general population. However, it is also possible that there is an association between the two conditions, because MS, like SSc, is also believed to be autoimmune in nature, and the pathogenic role of T cells is crucial in both processes.7 Furthermore, MS has been increasingly reported in association with other autoimmune diseases not primarily affecting the nervous system.8 If any of the above possibilities is present, the prognosis and therapeutic approach of our patient should match those of typical MS. The coexistence of SSc and MS is rare and, as far as we know, has been described in only four patients. Rapidly progressive and finally gripping MS developed in their early twenties, whereas SSc appeared later in the course of the MS in all four patients.9 10 Interestingly, unlike these cases, our patient presented in her thirties with a mild form of MS, several years after the onset of SSc.

A third possibility exists that, the neurological manifestations of this patient might have been part of her primary disease—that is, SSc. Involvement of the central nervous system (CNS) in this disease is considered uncommon, and secondary to vasculopathic damage.11 12 The fact that our patient had prolonged visual evoked potentials, suggestive of optic neuropathy, is rather in favour of MS, although this abnormality has been reported in SSc.13 On the other hand, a significant percentage of patients with systemic lupus erythematosus may present with CNS disease and, some of them with oligoclonal banding in the CSF.4 Brain or spinal cord disease, or both, with clinical features and laboratory findings indistinguishable from MS has been reported in Sjögren's syndrome too, although CNS involvement in this syndrome has been a matter of serious debate.14 15

In the absence of guidelines for the management of such patients, we considered our patient as a case of classical MS and, therefore, she was not deprived of the possible benefit of a disease modifying treatment, such as interferon β.
CORRECTION

Heavy cigarette smoking and RA
(Masi AT, Aldag JC, Malamet RL.
Ann Rheum Dis 2001;60:1154.)
The authors of this letter, in a further analysis of their data, found that four heavy smokers in the control group were incorrectly included in the 168 subjects matched to the 42 pre-RA cases who had baseline negative rheumatoid factor (RF−) status. They should be correctly reassigned to the 48 matched controls for the 12 pre-RA cases who had baseline positive rheumatoid factor (RF+) status.

The correct assignments place 11 (23%) heavy smokers in the 48 controls for the 12 pre-RA RF+ cases. Those 12 cases include two (17%) heavy smokers. The 168 controls for the 42 pre-RA cases who had baseline negative rheumatoid factor (RF−) status should correctly include eight (5%) heavy smokers. Those 42 cases include 11 (26%) heavy smokers. The new correct figures are shown in bold in the table.

The correct assignments strengthen the findings in this prospective, community based study that baseline heavy cigarette smoking was an independent risk factor from baseline positive rheumatoid factor status.

Corrections printed in the journal also appear on the Annals website www.annrheumdis.com and are linked to the original publication.

Table 1  Numbers of pre-RA cases and matched controls reporting heavy cigarette smoking (CS 30+/day) at baseline by relevant categories and odds ratios (ORs) with 95% confidence intervals (95% CIs) for developing ACR+ rheumatoid arthritis

<table>
<thead>
<tr>
<th>Categories</th>
<th>Pre-RA cases</th>
<th>Respective matched controls</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number</td>
<td>CS 30+/day (%)</td>
</tr>
<tr>
<td>Pre-RA RF+</td>
<td>12</td>
<td>2 (17)*</td>
</tr>
<tr>
<td>Pre-RA RF−</td>
<td>42</td>
<td>11 (26)</td>
</tr>
<tr>
<td>Entry and post-RA RF−</td>
<td>15</td>
<td>4 (27)</td>
</tr>
<tr>
<td>Conversion of pre-RA RF− to RF+†</td>
<td>27</td>
<td>7 (26)</td>
</tr>
</tbody>
</table>

*No association of CS 30+/day with pre-RA RF+ (p=0.99).
†Conversion of RF− at baseline to RF+ after clinical onset of RA.
FORTHCOMING EVENTS

3rd International Congress on Autoimmunity
20–24 Feb 2002; Geneva, Switzerland
Contact: Professor Yehuda Shoenfeld, 3rd International Congress on Autoimmunity, PO Box 50006, Tel Aviv 61500, Israel
Tel: +972 314 0018
Fax: +972 517 5676
Email: autoim02@kenes.com
Website: www.kenes.com

22nd European Workshop for Rheumatology Research
28 Feb–3 Mar 2002; Leiden, The Netherlands
Contact: Professor F C Breedveld, Leiden University Medical Centre, Department of Rheumatology, PO Box 9600, 2300 RC Leiden, The Netherlands
Tel: +31 (0)71 526 3598
Fax: +31 (0)71 526 6752
Email: F.C.Breedveld@lumc.nl
Website: www.eurlr.org

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Contact: Professor F C Breedveld, Leiden University Medical Centre, Department of Rheumatology, PO Box 9600, 2300 RC Leiden, The Netherlands
Tel: +31 (0)71 526 3598
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Website: www.eurlr.org

OMERACT VI
11–14 Apr 2002; Bali
Includes two modules: MRI and economics; and four workshops: patients’ perceptions, imaging (healing), progressive systemic sclerosis, minimally important clinical difference and osteoarthritis
Contact: Ms Lisa McClain, ARC Epidemiology Unit, University of Manchester, Oxford Road, Manchester M13 9PT, UK
Tel: +44 (0)161 275 5993
Fax: +44 (0)161 275 5043
Email: Lisa@fs1.ser.man.ac.uk
Website: www.eurlr.org

British Society for Rheumatology
XXVth AGM
23–25 Apr 2002; Brighton, UK
Contact: BSR, 41 Eagle Street, London WC1R 4TL, UK
Website: www.rheumatology.org.uk

4th EULAR Sonography Course
25–28 Apr 2002; Madrid, Spain
The course is entitled “Practical use of musculoskeletal ultrasonography”
Contact: Esperanza Naredo
Email: enaredo@eresmas.com
Website: www.eular.org/courses and www.sameint.it/eular

10th International Vasculitis and ANCA Workshop
25–28 Apr 2002; Cleveland, Ohio, USA
Contact: Deborah J Bork, The Cleveland Clinic Foundation, Desk A50, Center for Vasculitis Care and Research, 9500 Euclid Avenue, Cleveland, OH 44195, USA

Tel: 216 445 8533
Fax: 216 445 7569
Email: borkd@ccf.org
Website for registration and abstract submission: www.clevelandclinicemed.com/courses/Vasculitis2002.asp

International Congress: New Trends in Osteoarthritis
9–11 May 2002; Milan, Italy
Contact: Organising Secretariat, O.I.C. S.r.l., Via Fatebenefratelli 19, 20121, Milan, Italy
Tel: +39 02 65 71 200
Fax: +39 02 65 71 270
Email: osteoarthritis@oic.it

10F World Congress on Osteoporosis
10–14 May 2002; Lisbon, Portugal
Contact: IOF Secretariat, 71 cours Albert Thomas, F-69003 Lyon, France
Tel: +33 472 91 41 77
Fax: +33 472 36 90 52
Email: info@iof Lyon.org
Website: www.osteofound.org

5th European Conference on Systemic Lupus Erythematosus
26–30 May 2002; Athens, Greece
Chairman Professor HM Moutsopoulos Secretariat: AmphiTrion Congress Organising Bureau
Email: hmoutso@med.uoa.gr
Email: congress@amphiTrion.gr

Annual European Congress of Rheumatology
12–15 June 2002; Stockholm, Sweden
Contact: Fred Wys, Executive Secretary EULAR, Witikonerstrasse 15, CH-8032, Zurich, Switzerland
Tel: +41 1 383 9690
Fax: +41 1 383 9810
Email: eular@bluueyn.ch
Website: www.eular.org

10th International Congress on Behcet’s Disease
27–29 June 2002; Berlin, Germany
Under the auspices of the International Society for Behcet’s Disease
Up to eight young investigator awards, each of $500, will be awarded on the basis of abstracts submitted
Contact: Professor Ch C Zouboulis, Department of Dermatology, University Medical Centre Benjamin Franklin, The Free University of Berlin, Faberstrasse 60–62, 14195 Berlin, Germany
Fax: +49 30 84456908
Email: zoubbere@zedat.fu-berlin.de
URL: www.userpages-fu-berlin.de/~zoubbere
ISBD website: www.behcet.ws

29th Scandinavian Congress of Rheumatology
15–18 Aug 2002; Tromso, Norway
Contact: Hans Nossent, Department of Rheumatology, University Hospital Tromso, Norway
Tel: +47 776 27294
Fax: +47 776 27258
Email: 29scr2002@rito.no or revhan@rito.no

Translational Research in Autoimmunity
21–22 Sep 2002; Pavia, Italy
Contact: Organising secretariat: eventi S.R.L., Corso Cavour, 1820 – 27100 Pavia, Italy
Email: tra@e30pr.com
Website: www.e30pr.com
Congress website: www.medicine.ucsd.edu/albani/2001meeting

OsteoArthritis Research Society International (OARSI) World Congress
22–25 Sep 2002; Sydney, Australia
Contact: OsteoArthritis Research Society International (OARSI), 2025 M Street NW, Suite 800, Washington DC 20036, USA
Tel: +1 202 367 1177
Fax: +1 202 367 2177
Email: oarasi@oarisi.org
Website: www.oarisi.org

10th International Congress on Antiphospholipid Antibodies
29 Sep–3 Oct 2002; Sicily, Italy
Deadline for abstracts 1 April 2002
Contact: Secretariat, 10th International Congress on Antiphospholipid Antibodies, c/o Kenes International, PO Box 50006, Tel Aviv 61500, Israel
Tel: 972 3 5140018/9
Fax: 972 3 5140077 or 972 3 5172484
Email: aps@kenes.com
Website: www.kenes.com/aps

7th International Conference on Eicosanoids and Other Bioactive Lipids in Cancer, Inflammation and Related Diseases
14–17 Oct 2002; Nashville, Tennessee, USA
Contact: Lawrence J Marnett, Biochemistry Department, Vanderbilt University, School of Medicine, Nashville TN 37232-0146, USA
Tel: (615) 343 7329
Fax: (615) 343 7534
Website: www.eicosanoids.science.eaync.edu

66th American College of Rheumatology AGM
23–29 Oct 2002; New Orleans, USA
Contact: ACR, Ronald F Olejko, Director of Conferences and Meetings, 1800 Century Place, Suite 250, Atlanta, Georgia 30045–4300, USA
Tel: +1 404 633 3777
Fax: +1 404 633 1870
Email: acr@aCRheumatology.org
Website: www.aCRheumatology.org

Third International Meeting on Social and Economic Aspects of Osteoporosis and Osteoarthritis
7–9 November, 2002; Barcelona, Spain
Contact: Yolande Piette Communication, Boulevard Kleyer 108, 4000 Liège, Belgium
Tel: 32 4 254 12 25
Fax: 32 4 254 12 90
Email: ypc@compuserve.com

Certifying Examination in Pediatric Rheumatology
18 Nov 2002
Contact: American Board of Pediatrics, 111 Silver Cedar Court, Chapel Hill, NC 27514-1513, USA
Tel: 919 929 0461
Fax: 919 918 7114 or 919 929 9255
Website: www.abp.org

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