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.¹ But the posture of this middle figure should probably be interpreted as 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 tries to take 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 Laokoön 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 Debalation at Marseilles” in the Maria de Medici cycle from 1622 to 1625 for the Luxembourg Palace in Paris.³ Here, three young women, nereids, with curved muscular backs at the bottom of the picture nearly seem to carry the ship of Maria de Medici.

S E Hansen
Clinic of Rheumatology, H S Bispebjerg Hospital, DK-2400 Copenhagen NV, Denmark

References

Author’s response

Dr Hansen’s remarks about our recent article in the *Annals* are pertinent and have to be taken as an alternative 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 the 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 proximal interphalangeal (DIP) joint of the fourth finger in another sister with brown hair. Hyperextension of a DIP and metacarpophalangeal finger joint and hyperflexion of a wrist joint is also seen in the brown haired sisters of the painting “The Madonna 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 convention. The observations made by 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.

J Dequeker
Department of Rheumatology, University Hospitals, K U Leuven, B-3000 Leuven, Belgium

References

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. 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 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. Thus 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.

J G Jones, F Leighton
Queen Elizabeth Hospital, PO Box 1342, Whakauae Street, Rotorua, New Zealand

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 over the past four weeks. We note that 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.

*PostScript*
of the three month follow up (that is, two months after discharge) in our study in order to reflect the course of the effects and whether the different responsiveness of the SF-36 compared with the WOMAC remained consistent. In addition, we will publish further results of three months assessments up to the two year follow up of our patients during the next year.

The second issue deals with the fact that some items ask about activities of daily living and participation which are not demanded or hardly possible to perform during a stay in the clinic. These are mainly the items contained in questions 4 (a4–d4) and five (a5c) comprising the role physical and role emotional subscales. For this reason, we report these two scales as part of the SF-36 for the sake of completeness, but we did not include them in the analysis of the comparison of WOMAC and the SF-36. Nevertheless, item 8, which is the bodily pain scale, is also affected by this problem. Müller et al dealt with this issue recently. The authors created a modified SF-36m, which was adapted in items 4, 5, and 8 to the situation of a clinic stay. They concluded that bodily pain and role emotional did not show significantly different effects from those obtained by the original SF-36, but that the role physical scale was slightly more relevant in the SF-36m.

We used the SF-36 for three reasons. Firstly, the SF-36 assesses health status comprehensively—that is, not only pain and disease-specific scales as physical function, etc but also psychometric dimensions and dimensions of social participation. As a result, it gives an overall assessment of the patient’s health which is compatible with the disease-specific scales as physical function, etc. Secondly, it allows a comparison of the results with those to patients with different diseases, which is not possible with the WOMAC. Consequently, item 8 of the SF-36 can also be administered to “healthy” people and to patients with different diseases, which allows a comparison of the results with those for other patient groups and the general population. Thirdly, the SF-36 is one of the best tested, best known, and most widely used health measure all over the world.

F Angst, A Aeschlimann
Clinic of Rheumatology and Rehabilitation, 5330 Zürich, Switzerland

References

LETTERS

Is pamidronate effective for acute rheumatic pain?

Parenteral pamidronate is licensed in the United Kingdom for the management of Paget’s disease, tumour related hypercalcemia, and metastatic bone pain, where it can rapidly relieve symptoms.1 It is also widely used for the prevention and treatment of osteoporosis, although this represents unlicensed use of the drug, and there is some evidence that it can be rapidly effective for pain relief in patients with osteoporotic vertebral fractures.2,3 It has been used with some effect for the management of ankylosing spondylitis,4 but the full extent of any analgesic properties of the drug has not been fully explored. These properties became apparent to us quite by chance in the three cases described here.

Case reports

Patient A

A 23 year old female nurse with known ankylosing spondylitis was admitted to hospital with worsening pain in the chest and right buttock pain uncontrolled by regular opiate analgesia and a variety of potent non-steroidal anti-inflammatory drugs. Parenteral methylprednisolone was prescribed, followed by pamidronate 30 mg for “bone protection”. In the event, pamidronate was given but not methylprednisolone, deferred owing to unexplained pyrexia. Shortly after receiving her pamidronate, her intractable pain was so greatly improved that methylprednisolone was declared and she was discharged three days later. The improvement seen has been sustained for over six months. The unexpected analgesic effectiveness in this case led to its use in two subsequent cases.

Patient B

A 38 year old housewife with chronic low back pain was admitted with a short history of acute back pain and a modestly raised C reactive protein (14 mg/l). Isotope bone scan showed increased uptake in the fifth lumbar intervertebral disc. Magnetic resonance imaging identified abnormal signal from this disc suggestive of discitis. An infective cause was felt to be unlikely: antibiotics were not prescribed, but in view of her persistent symptoms, pamidronate 30 mg was given by intravenous infusion, with sufficient sustained improvement in her acute back pain to allow discharge two days later.

Patient C

A 33 year old male factory worker with a history of juvenile chronic arthritis since early childhood and spondyloarthropathy was admitted with generalised bone pain despite weekly oral methotrexate, phenylbutazone, and oral analgesia. Intercurrent diarrhoea was investigated but remained unexplained. Parenteral pamidronate 30 mg was given, leading to sustained improvement in his rheumatic pains.

Discussion

We believe these cases represent the first time that sustained analgesic efficacy has been attributed to a single dose of parenteral pamidronate in acute rheumatic pain not related to osteoporosis or neoplasia. The mechanism whereby pamidronate provides rapid onset sustained pain relief for metastatic bone disease or osteoporotic fractures is unknown. Many of the known effects of bisphosphonates on bone structure and cell populations are unlikely to be rapidly analgesic. However, it has been suggested that bones have complex sensory innervation, with nociception mediated by neuropeptides including substance P, prostaglandin E2, and calcitonin gene related peptide which may be influenced by bisphosphonates.5 There is no reason to believe that such an analgesic effect would be confined to bone affected by osteoporosis or neoplasia and might well extend to bone pain due to inflammation. In the three cases described many other factors might have contributed to the apparent effectiveness of parenteral pamidronate, including chance. However, the results suggest that the potential role of pamidronate in the control of acute rheumatic pain warrants further evaluation.

A El-Shafei, T Sheeran, D Mulherin
Department of Rheumatology, Cannock Chase Hospital, Staffordshire, UK

Correspondence to: D Mulherin, Department of Rheumatology, Cannock Chase Hospital, Rd, Cannock, Staffs, WS11 2XY, UK; diamulherin@msghtr.wmids.nhs.uk

References

Antibodies to β, glycoprotein I and cardiolipin in SSc

Systemic sclerosis (SSc) is a multisystem disease in which organ damage is initiated by fibrosis, microvascular occlusion, and proliferation of the vascular intima. The reported prevalence of antiphospholipid antibodies (aCL) in SSc varies from 0 to 25%,1 2 and reports of clinical associations have been variable.1 3 4 To our knowledge, only one study has examined antibodies to β, glycoprotein I (β2GPI) in SSc and shown a correlation with pulmonary hypertension and raised mean pulmonary artery pressure.5 In our study we examined the frequency of β2GPI and aCL in SSc and Raynaud’s phenomenon (RP).

Twenty six patients with SSc (16 diffuse, 10 limited), 23 with RP and 21 healthy volunteers (employees at the research facility) were included in this retrospective study. Informed consent was obtained. All 16 patients with diffuse SSc and one patient with limited SSc patients met American Rheumatism Association (ARA) preliminary criteria for scleroderma.6 The remaining nine with limited SSc had at least three of the following: sclerodactyly, calcinosis, Raynaud’s phenomenon, oesophageal dysmotility, telangiectasia, or positive anticientromere antibodies. The patients with RP had no manifestations of connective tissue disease. Clinical and laboratory assessments were recorded at the initial visit.

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@beta2GPI and aCL were measured by enzyme-linked immunomunosorbent 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 antigens A and B (SS-A/SS-B) by double immunodiffusion. Patients with RP (p=0.02). All mean laboratory values were within the normal range. Figure 1 describes their age in decades, not years. Student’s t test (two tailed) was used for analysis of frequencies. Age distribution 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 aβ2GPI IgG, where all tests were negative. IgM aβ2GPI 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-cardiolipin (p>0.05). The sera positive for aCL were not the same as those positive for aβ2GPI.

The two patients with SSc positive for aβ2GPI had mean disease duration of 19 months; both had cutaneous manifestations and one had hypoxia with decreased carbon monoxide transfer factor (Tlco). The three patients with SSc and aCL had mean disease duration of 112 months. One had hypoxia (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 thrombocytopenia 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 Vossiari 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 IIF 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 aβ2GPI or aCL. In our study we found that the frequency of antibodies to β2GPI and aCL was low in scleroderma, 8% and 12% respectively. There were no clear clinical or laboratory correlations with a positive test.

**Acknowledgments**

This research is supported by the Canadian Institutes for Health Research. Dr Schoenroth is supported by the Alberta Heritage Foundation for Medical Research. Dr Lonzetti is supported by Sclerodermie Québec.

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<tr>
<th>Table 1 Demographics and laboratory results in patients with SSc, RP, and normal controls</th>
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<td><strong>Scleroderma</strong> (n=26)</td>
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BUN, blood urea nitrogen; CK, creatine kinase.
*Comparison of age distribution versus SSc.

![Figure 1](http://www.annrheumdis.com) Comparison of aβ2GPI and aCL antibody levels in patients with SSc, RP, and normal controls. The numbers on the ordinate represent optical density values converted to SMU (standard IgG β2GPI units), MPL (1 MPL unit = the binding of 1 µg/mL IgG aCL), or GPL (1 GPL unit = the binding of 1 µg/mL IgG aCL). The arrows indicate the cut off values for each dataset.

**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 of his 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 second and third metatarsophalangeal 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 C-reactive protein (45 mg/L). 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 urticaria cleared, and he was able to walk without assistance. 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 sulfasalazine (500 mg twice daily) were added. One month after the ankle synovitis and pain had settled.

Discussion

Several strands of evidence link different vaccines to the development of a spectrum of arthritis. Some, 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. Recombinant hepatitis B vaccination has also produced similar pictures. Symmetrical small joint polyarthritis but with negative rheumatoid factor has been described in association with intravesical Bacillus Calmette-Guerin (BCG) vaccine used as immunotherapy for bladder carcinoma.

The spectrum of arthritis associated with vaccination is illustrated by the induction of large joint monarthritis 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 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. 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 vaccination can be severe, with prolonged and significant morbidity lasting many months. Hassan and Oldham reported Reiter’s syndrome with joint pain, and conjunctivitis lasting many months, whereas Bracci and Zoppini additionally reported fevers and lymphadenopathy with the hepatitis B surface antigen vaccine (Engerix B). However, as with our case, antibody 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 arthralgias 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 retrigger a reactive arthritis in a susceptible person. Although the mechanisms of vaccination and 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 behoove physician to warn patients awaiting vaccination about the possible adverse effect on joint symptoms.

Correspondence to: Dr Jawad

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 within the spleen occur in many autoimmune rheumatic diseases, and prompt recognition of these complications is important. In a recent series of patients with WG, the spleen was commonly affected: 78–100% of patients had splenic lesions with a combination of necrosis, vasculitis, and granuloma formation. Clinically apparent 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 renal function progressively recovered 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 60 ml of serosanguinous fluid was aspirated. Gram stain and initial bacterial cultures of the fluid were negative, and Zielh-Neelsen stain did not show acid fast bacilli. Microscopy showed the presence of calcium hydroxyapatite crystals, and hence the shoulder was again injected with corticosteroids. Two months later, M avium was identified from the synovial fluid culture. The patient was treated with clarithromycin and ethambutol, and has made a good clinical response.

References

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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 hydrostasis, relapsing synovitis after surgical synovectomy, destructive mono- or polyarticular arthritis, and activated osteoarthritis resistant to other treatments. Some studies have reported successful concomitant treatment of Baker’s cysts in the treatment of polyarthritis, but radiosynoviorthesis of the right knee 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 radiosynoviorthesis in treating psoriatic arthritis compared with rheumatoid arthritis. A few years ago, only patients aged over 40 years were treated with radiosynoviorthesis. Today, this treatment is used in an increasing number of younger patients. The success rate for radiosynoviorthesis 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 radiosynoviorthesis have been reported. In our patient, neither the treatment with a nonsteroidal anti-inflammatory drug (200 mg diclofenac daily) nor the local treatment with triamcinolone hexacetonide after a decompression and aspiration led to improvement. A possible alternative to surgical bursectomy, radiosynoviorthesis 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 radiosynoviorthesis of an isolated bursitis. This case gives cause for hope that radiosynoviorthesis represents a successful alternative treatment to operational intervention for chronic inflammation of the bursa.

M N Berliner, R G Bretzel
3rd Department of Internal Medicine, University Hospital Giessen, Germany

R Klett
Department of Nuclear Medicine, University Hospital Giessen, Germany

Correspondence to: Dr M N Berliner, Universitätsklinikum Giessen, Medizinische Klinik, III Rheumatologie, Rodlohrtal 6, D-35392 Giessen, Germany; michael.berliner@rheuma.med.uni-giessen.de

References
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 confusion. In particular, she had been experiencing vertigo since the age of 22, 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 scleroderma—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 ophthalmoplegia. 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 1, rose bengal, and break up time eye tests were normal. These had been performed routinely several times previously before the present admission. Furthermore, the patient had never complained of xerostomia.

Routine blood tests were normal. Serology showed positive antinuclear antibodies at a titre of 1:640, of the fine speckled pattern, and positive anti-Scl70 antibodies. Antibodies to cardiolipin and the other extractable nuclear antigens, including Ro(SSA), La(SSB), Sm, and RNP, 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 mg/day 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 pathogenetic role of T cells is crucial in both processes. Furthermore, MS has been increasingly reported in association with other autoimmune diseases not primarily affecting the nervous system. 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. 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. 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. 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. 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.

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.

E Chroni, C Paschalidis, T Stergiou
Department of Neurology, University of Patras, Patras, Greece

C Vlahanastasi, A P Andonopoulos
Division of Rheumatology, Department of Medicine, University of Patras

Correspondence to: Professor A P Andonopoulos, Division of Rheumatology, Department of Medicine, University of Patras School of Medicine, 265 00 Rio, Patras, Greece; andonand@med.uapatras.gr

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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.

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 5674  
Email: autoim02@kenes.com

**22nd European Workshop for Rheumatology Research**
28 Feb–3 Mar 2002; Leiden, The Netherlands  
**Contact:** Professor F C Bredveld, 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.Bredveld@lumc.nl  
Website: www.eurwr.org

**Tenth Intensive Applied Epidemiology Course for Rheumatologists**
11–15 Mar 2002; Manchester, UK  
No previous experience in epidemiology is needed. The course is residential and limited to 25 places.  
**Contact:** Ms Lisa McClair, 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@f1.ser.man.ac.uk

**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:** Conference Organisers Q2O, 7 Swann Street, Old Isleworth, Middlesex TW7 6JR, UK; or Peter Brooks, Faculty of Health Sciences, Level 1, Edith Cavell Building, Royal Brisbane Hospital, Herston 4029, Australia  
Fax: +61 400 208 569  
Email: tony@q2q.co.uk or p.brooks@mailbox.uq.edu.au

**British Society for Rheumatology**
XIXth AGM  
23–26 Apr 2002; Brighton, UK  
**Contact:** BSR, 41 Eagle Street, London WC1R 4TL, UK  
Website: www.rheumatology.org.uk

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

**IOF 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: AmphiVision 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 Wyss, Executive Secretary EULAR, Witkonnerstrasse 15, CH-8032, Zürich, Switzerland  
Tel: +41 1 383 9690  
Fax: +41 1 383 9810  
Email: eular@bluewin.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, Fabekstrasse 60–62, 14195 Berlin, Germany  
Fax: 49 30 84456908  
Email: zoubbere@zedat.fu-berlin.de  
Website: www.userspace-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 27924  
Fax: 47 776 2728  
Email: 29scr2002@rito.no or revhan@rito.no

**Translated Research in Autoimmunity**
21–22 Sep 2002; Pavia, Italy  
**Contact:** Organising secretariat: eventi S.R.L., Corso Cavour, 1820 – 27100 Pavia, Italy  
Email: tra@e20pr.com  
Website: www.e20pr.com  
Congress website: www.medicine.ucsd.edu/albanic/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: oarsi@oarsi.org  
Website: www.oarsi.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 Kennes 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.eayne.edu

**66th American College of Rheumatology AGM**
25–29 Oct 2002; New Orleans, USA  
**Contact:** ACR, Ronald F Olejko, Director of Conferences and Meetings, 1800 Century Place, Suite 230, Atlanta, Georgia 30045–4300, USA  
Tel: +1 404 633 3777  
Fax: +1 404 633 1870  
Email: acr@rheumatology.org  
Website: www.rheumatology.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 90  
Email: yp@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

www.anrheumdis.com
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J G Jones and F Leighton

*Ann Rheum Dis* 2002 61: 182-183
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