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. But the posture of this middle figure should probably be interpreted as an artistic phenomenon without medical reference.

Scultors 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 Laokoon sculpture with its three distorted figures. 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 Debarkation at Marseilles” in the Maria de Medici cycle from 1622 to 1625 for the Luxembourg Palace in Paris. 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 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 Musae 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 finger joint and hyperflexion of a wrist joint is also seen in the brown haired sister of the painting “The Venus and Saints” (Antwerp, Sint-Jacobskerk).

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

Reference

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. The 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, Whiskers Street, Rotorua, New Zealand

Authors’ response
In their letter commenting on our article, 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 may 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...
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 monthly 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 social 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 (a–d) and five (5a-5c) comprising the role physical and role emotional scales. For this reason, we reported 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 responsive in the SF-36m, which was adapted in items 4, 5, and 8.

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 status which is compatible with the WHO's new ICIDH or the future ICF concept defining health. Secondly, the SF-36 can be also administrated 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, known, and most widely used health measure all over the world.

F Angst, A Aeschlimann
Clinic of Rheumatology and Rehabilitation, 5330 Zuzwil, 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. 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. It has been used with some effect for the management of ankylosing spondylitis,6 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 back pain 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 effect of pamidronate 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 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 effect of pamidronate in this case led to its use in two subsequent cases.

Patient C

A 33 year old male factory worker with a history of juvenile chronic arthritis since early childhood and spondylarthropathy 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.7 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.8 There is no reason to believe that such an analgesic effect would be confined to those affected by osteoporosis or neoplasm and might well extend to bone pain due to inflammation. In the three cases described many other factors might have contributed to the apparent analgesic effect 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: Dr D Mulherin, Department of Rheumatology, Cannock Chase Hospital, Drayton Rd, Cannock, Staffordshire, WS11 2XY, UK; diarmuid.mulherin@msgh-tr.wmids.nhs.uk

References

Antibodies to β2 glycoprotein I and cardiolipin in SSC

Systemic sclerosis (SSc) is a multisystem disease in which organ damage is caused by fibrosis, microvascular occlusion, and proliferation of the vascular intima. The reported prevalence of anticardiolipin antibodies (aCL) in SSc varies from 0 to 25%.9,10 Reports of clinical associations have been variable.9,11 To our knowledge, only one study has examined antibodies to β2 glycoprotein I (β2GPI) in SSc and shown a correlation with pulmonary hypertension and raised mean pulmonary artery pressure.6 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) were included, 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.12 The remaining nine with limited SSc had at least three of the following: Raynaud’s phenomenon, calcinosis, esophageal dysmotility, telangiectasia, or positive antitopomere 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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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 antigens 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-cardiolipin (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 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 thrombocytopaenia 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 Voyssairat 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 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.

Acknowledgments

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<table>
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<td>Age groups</td>
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<td>Anticentromere antibodies</td>
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<td>CK (µmol/l)</td>
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<td>BUN (mg/l)</td>
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<td>Creatinine (mg/l)</td>
<td>9.1 (SD 1.3)</td>
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BUN, blood urea nitrogen; CK, creatine kinase. *Comparison of age distribution versus Ssc.

References


L Schoenroth, M Fritzler
Faculty of Medicine, University of Calgary

L Lonetti, J-L Senécal
Department of Medicine, University of Montreal

Correspondence to: Dr M Fritzler, 4108 Heritage Medical Research Building, Faculty of Medicine, University of Calgary, 3330 Hospital Drive NW, Calgary, Alberta, Canada T2N 4N1; fritzler@ucalgary.ca

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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 the right ankle. Two weeks before presentation, he had been given a booster tetanus toxoid vaccine. 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, mid-foot, metatarsal and talonavicular joint, with tenderness and synovial thickening. The subtaloid, mid-tarsal, and metatarso-phalangeal joints were also involved. On presentation he was walking with a pronounced antalgic gait and had been given a booster tetanus toxoid injection. 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.

The spectrum of arthritis associated with vaccination is illustrated by the induction of large joint monarthritis by combined diphtheria, poliomyelitis, and tetanus toxoid vaccine.1 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.2 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.3

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.4 However, HLA-B27 expression is not a prerequisite for arthritis linked to vaccines although its presence may predict a more prolonged and severe disease.5 Vaccines may also trigger autoimmune responses by binding to critical antigen binding clefts on the major histocompatibility complex class I molecule, thereby triggering T cell proliferation and interferon production.6

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.

The impact of vaccination associated with vaccination may be severe, with prolonged and significant morbidity lasting many months. Hassan and Oldham reported Reiter’s syndrome with joint swelling and conjunctivitis lasting many months, whereas Bracci and Zoppini additionally reported fevers and lymphadenopathy with the hepatitis B surface antigen vaccine (Engerix B).7 However, as with our case, arthritis associated with vaccination may 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 polyarthritis nodosa-like and systemic vasculitides have also been reported in a few instances.8 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.9

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 vaccine-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 wise to warn patients awaiting vaccination about the possible adverse effect on joint symptoms.

A Kaul, M Adler, F Alokkay, A S M Jawad
Rheumatology Department, The Royal London Hospital, 275 Bancroft Road, Mile End, London E1 4DG, UK

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.1 Less frequently, the disease may affect other organs as well. Serious and occasionally fatal complications within the spleen occur in many autoimmune rheumatic diseases,2,3 and prompt recognition of these complications is important. In the non-renal type 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.4 Clinical involvement of the spleen with WG disease is rare, however.5 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.

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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 sediment 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. Evaluation of 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 infarction, with small areas of enhancement within the larger hypodense lesions (fig 1). A search for lupus anticoagulant and antiphospholipid antibodies was negative and no abnormalities of blood clotting could be detected. A trans-oesophageal 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

Splenic involvement in WG has included such abnormalities as splenomegaly, capsular adhesion, impaired splenic function, and infarcts.1 Infarction may occur as a result of a distal occlusion of the splenic artery or its branches, because splenic parenchymal arteries are end vessels that do not communicate with one another. There are few reports on splenic infarction on post mortem in patients with WG.2,4 Histological examination frequently shows massive or multiple areas of splenic necrosis, usually associated with extensive central arteritis, splenic trabeculitis, follicular arteriolitis and necrosis, disseminated visceral granulomatata, and capsulitis. On CT, splenic infarcts classically and more commonly appear as peripheral, well defined, wedge shaped areas of low attenuation.5 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.6 Examination with ultrasound in combination with duplex sonography of splenic blood supply permits non-invasive diagnosis of splenic infarction.7 The diagnosis can be confirmed by magnetic resonance imaging or CT scan, which permits assessment of the extent of splenic infarction. Splenic involvement in WG may be more prevalent than previously believed.8,9 Pain in the left upper quadrant and left shoulder and fever may be present after splenic infarction, but many patients remain asymptomatic.10 Consequenously, 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,11 a conservative approach is justified. In the long term these patients may be more susceptible to pneumococcal infection because of the functionally asplenic condition.12 This possibility provides further help in the diagnosis of this rare condition in vivo.

References


Two cases of Mycobacterium avium septic arthritis

The “unusual and memorable” case reported by Ter Borg and Termestee serves as a useful reminder that atypical mycobacterial infections, although uncommon, need to be con- sidered in immunocompromised patients.

We present here two case reports of patients with pre-existing rheumatic disease receiving immuno- suppressive treatment, who developed septic arthritis due to Mycobacterium avium intracellular.

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 50 ml of serosanguinous fluid was aspirated. 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.

Figure 1 Abdominal CT scan showing a normal liver and a spleen with well defined areas of low attenuation, consistent with infarction.

Figure 2 Abdominal CT scan three years later shows volume reduction and scarring of the spleen.
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 strain, 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 rheumatological outpatient clinic for his first visit. The clinical findings showed a patient with good general condition (weight 86 kg), blood pressure 120/80 mm Hg, and without any signs of ulceration or pitting edema of the affected area. The flexion range of the left elbow was limited to 40° in extension. 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 triamcinolone 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 radiosynoviorthesis. After aspiration of 9 ml of a serous effusion, 55 MBq rhenium-186 was instilled into the olecranon bursa, and then, to avoid radiosynoviorthesis, 5 mg triamcinolone was injected. Radiosynoviorthesis of olecranon bursitis is between 50 and 80%, depending on the localization and the amount of inflammatory activity.6,7

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 non-steroidal anti-inflammatory drug (200 mg diclofenac daily) nor the local treatment with triamcinolone hexacetonide after a decompression aspiration led to improvement. An 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 olecranon bursitis. This case gives cause for hope that radiosynoviorthesis represents a successful alternative treatment to operational intervention for chronic inflammation of the bursa.

References


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. Indications are chronic persisting synovitis, intermittent hydroarthrosis, synovectomy after surgical synovectomy, hemophilic arthropathy, and activated osteoarthritis resistant to other treatments. Some studies have reported successful concomitant treatment of Baker's cysts in the treatment of psoriarthritis, but radiosynoviorthesis of the treatment for Baker's cysts is not usual. It is possible, however, by infusion of a radioactive isotope into the knee joint, but the popliteal cyst must not be punctured directly. Due notice should be taken of contraindications.8 Other reports disagree about the success rates of radiosynoviorthesis in treating psoriatic arthritis compared with rheumatoid arthritis.9 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 localization and the amount of inflammatory activity.6,7

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


Figure 1 Sonography of the left elbow (A) showing an olecranon bursitis (58 mm; 17.7 mm; 3.3 mm; 2.5 mm) and (B) three months after radiation synovectomy.


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 5 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 I, rose bengal, and some of them with oligoclonal banding at the C7 level, resulting in focal enlargement of the cord at that level.

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 progressive 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 Sjogren’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 Paschalis, 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; andandon@med.uapatras.gr

References
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.

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.

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.
Two cases of *Mycobacterium avium* septic arthritis

M J Bridges and F McGarry

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