Sonography as a replacement for sialography for the diagnosis of salivary glands affected by Sjögren’s syndrome

Recently, it has been suggested that sonographic evaluation of the salivary glands is useful in the diagnosis of Sjögren’s syndrome. Kawamura et al. and, more recently, Ariji et al., showed that descriptive and quantitative assessment of the salivary glands by sonography efficiently differentiated between diseased and normal glands in patients with Sjögren’s syndrome. They showed that the proposed sonographic gradings correlated well with the sialographic gradings. These findings suggest that sonography might be an alternative diagnostic tool for Sjögren’s syndrome.

Here, we attempted to determine whether sonography can take the place of sialography as an alternative technique for the assessment of salivary gland involvement in Sjögren’s syndrome. Sialography and sonography were performed on 294 patients who presented with sicca syndrome (171 positive and 123 negative for Sjögren’s syndrome). We diagnosed patients with Sjögren’s syndrome on the basis of the criteria of the European Community Study Group. Sonographic features characteristic of Sjögren’s syndrome are heterogeneous echogenicity with hypo- and hyperechoic signals throughout the affected gland (fig 1).1,2

Table 1 shows the performance of each of the diagnostic criteria. Sialography performed best among the five diagnostic criteria—that is, sialography, functional tests (Saxon and Schirmer), and serological tests (SS-A and SS-B). Interestingly, when used instead of sialography, sonography provided a good performance, comparable with that of sialography (McNemar test, p=0.067). In contrast, the other diagnostic criteria did not perform as well as the two imaging criteria.

Logistic regression analysis was performed to identify diagnostic criteria that might be used as predictive indicators for differentiating between patients with and without Sjögren’s syndrome.3 Univariate logistic regression analysis showed that the six diagnostic criteria assessed (sialography, sonography, Saxon’s test, Schirmer test, SS-A, and SS-B) did correlate with a positive diagnosis of Sjögren’s syndrome, indicating that these six criteria, if used alone, could effectively predict the presence of Sjögren’s syndrome (table 1). On multivariate analysis, however, only sialography and sonography showed significant correlations with a positive diagnosis of Sjögren’s syndrome (table 1); when sialography was used together with the functional and serological criteria, only sialography showed a significant correlation. If sonography was used instead of sialography, only sonography displayed a significant correlation with a positive diagnosis of Sjögren’s syndrome (table 1). Collectively, these findings suggest that the sonography performs as well as sialography in differentiating between parotid glands affected by Sjögren’s syndrome and normal glands. In contrast, the other diagnostic criteria did not perform as well as the two imaging criteria.

Some discrepancies were found between the diagnostic performance in the present study and that in previous studies. For example, Schirmer’s test in our study performed poorly compared with the performance reported by Vitali et al.4 SS-A and SS-B displayed high sensitivity and low specificity in our study, whereas low sensitivity and high specificity were found in the previous study.5 These inconsistencies may be due to the differences in patient groups or in techniques, or both. Despite these differences, the performance by sialography was similar, consistent with the notion that the imaging techniques, including sialography, provide reliable results in the diagnosis of Sjögren’s syndrome.

In conclusion, a diagnosis of Sjögren’s syndrome can be made on the basis of a wide range of diagnostic tests, and not merely on fixed combinations of these tests. Evaluation of salivary gland involvement contributes significantly to the performance of the criteria. Thus the availability of different imaging techniques, such as Doppler sonography6 and magnetic resonance imaging,7 to assess salivary gland involvement allows clinicians to classify patients with sicca syndrome correctly.

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**Table 1** Performance and logistic regression analysis of diagnostic criteria for Sjögren’s syndrome

<table>
<thead>
<tr>
<th>Diagnostic Test</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
<th>Accuracy (%)</th>
<th>Univariate Analysis</th>
<th>Multivariate Analysis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sialography</td>
<td>87</td>
<td>98</td>
<td>92</td>
<td>Coefficient 6.02</td>
<td>Coefficient 4.87</td>
</tr>
<tr>
<td>Sonography</td>
<td>76</td>
<td>94</td>
<td>84</td>
<td>SE 0.75</td>
<td>SE 1.06</td>
</tr>
<tr>
<td>Saxon</td>
<td>70</td>
<td>71</td>
<td>71</td>
<td>p Value &lt;0.00001</td>
<td>p Value &lt;0.00001</td>
</tr>
<tr>
<td>Schirmer</td>
<td>59</td>
<td>57</td>
<td>59</td>
<td>0.56</td>
<td>0.67</td>
</tr>
<tr>
<td>SS-A</td>
<td>83</td>
<td>56</td>
<td>70</td>
<td>1.92</td>
<td>1.06</td>
</tr>
<tr>
<td>SS-B</td>
<td>88</td>
<td>42</td>
<td>51</td>
<td>1.66</td>
<td>0.97</td>
</tr>
</tbody>
</table>

NS, not significant.
Radiographs of the hands and feet were normal. There were slight erosions of the sacro-illiac joints and of the symphysis pubis.

The patient was treated with non-steroidal anti-inflammatory drugs (NSAIDs) and on several occasions with local injections of corticosteroids into the knee joints. For the psoriatic nails he took acetretine (Neotigason) at a daily dose of 20 mg, for 12 months, but the nail lesions did not improve. In view of the persistence of the nail deformities, the patient has been treated since January 2000 with sulfasalazine (the dose being progressively increased from 0.3 g daily to 2 g daily), in addition to NSAIDs. Three months later, the nail lesions started to recede and they disappeared progressively (Fig 1B); the improvement has persisted until now. Concomitantly, there was a marked improvement of the arthritis.

Discussion

Nail disease is significantly associated with PsA. It is particularly common in cases with DIP joint involvement and tends to involve more severe PsA. In view of the close chronological relationship between the administration of sulfasalazine and the improvement of the nail lesions, it can be considered that sulfasalazine played a beneficial part in the pathophysiological condition of our patient. Dermatological assessment of patients treated with sulfasalazine for PsA showed signs of cutaneous improvement compared with those receiving placebo. The series of Farr et al reports improved cutaneous lesions in as few as 3/15 patients receiving sulfasalazine for PsA and showed signs of cutaneous improvement compared with those receiving placebo. The improvement in quality of life achieved by patients receiving sulfasalazine for PsA showed signs of cutaneous improvement compared with those receiving placebo. The improvement in quality of life achieved by patients receiving sulfasalazine for PsA showed signs of cutaneous improvement compared with those receiving placebo.

Treatment of PsA with cyclosporin or etanercept is effective for both joint and skin lesions of PsA, again no data about the outcome of psoriatic nail lesions were provided in these clinical studies. Our case report might be the occasion to draw the attention of rheumatologists to the possible beneficial effects of basic treatment such as sulfasalazine not only for PsA but also for treating psoriatic nails.

Nail lesions in psoriatic arthritis: recovery with sulfasalazine treatment

Treatment with sulfasalazine has been reported to be effective in psoriatic arthritis (PsA). However, the role of sulfasalazine in cutaneous lesions has been surrounded by controversies. As far as we know its possible beneficial effect on nail lesions has not been reported.

Case report

A 25 year old man had presented with nail lesions considered to be psoriatic since 1996. During the same period he started to have pain in both knee joints. Since 1998 he had also had pain in the distal interphalangeal (DIP) joints. At the end of the same year the patient consulted a rheumatologist. On clinical examination, both knee joints were swollen and a Baker’s cyst was present at the right side. The 4th and 5th DIP joints of both hands were red, painful, and slightly swollen. Nail deformities were present in both hands (Fig 1A) and feet. Psoriatic lesions of the auditory canals and intergluteal fold were seen, prompting the diagnosis of psoriasis partimae.

Synovial fluid from the right knee joint contained 17.8x10^6 leucocytes (86% polymorphonuclear); no crystals were seen. The erythrocyte sedimentation rate was 33 mm/1st h. Rheumatoid factor was negative, as were cultures of nail specimens for fungi.

References


Home sequential high dose intravenous immunoglobulins in systemic autoimmune disease

The high cost of IV immunoglobulins is often considered to be a disadvantage of this treatment. However, the role of IV immunoglobulins in the treatment of immunodeficiency disorders and autoimmune diseases has been widely acknowledged. This study aimed to evaluate the efficacy and safety of sequential high dose IV immunoglobulins in the treatment of selected autoimmune diseases, particularly systemic lupus erythematosus (SLE), rheumatoid arthritis (RA), and juvenile idiopathic arthritis (JIA). A total of 30 patients (18 women, 12 men) were enrolled, with a mean (SD) age of 44.0 (0.9) for the women and 51.0 (0.9) years for the men (range 21–74). All the patients had received the first two treatments in hospital to ascertain their tolerance. Patients mostly received Tégeïline (314 treatments), Endobulin (81 treatments), and Gammax (81 treatments). All the patients had a corticoid and a non-refractory autoimmune disease (most likely polyarthritis, dermatomyositis, and adult onset Still’s disease).

The dose prescribed for each treatment was generally 2 g/kg. Treatments were carried out monthly and consisted of two days of treatment. The primary aim of the treatments at home remained conventional. The efficacy of the IV immunoglobulins was described by the patients as very good 17% good 33%, modest 3%, nil 47%. The efficacy of the IV immunoglobulins was described by the senior doctor as very good 33%, good 30%, nil 17%. Evaluation of the efficacy described by the patients themselves was based on purely functional criteria (general condition, pain,
invoiced by the hospital administration for years, the savings for the community amount $2701 against $2471 for a treatment at home. The fusion stand at the pharmacy and at home).

better monitoring, who preferred the treatments at the hospital (n=3), better quality of sleep (n=2), activities (n=3), avoiding repeated trips to the hospital (n=10), more occupation (n=6), time gain (n=12), presence of next of kin (n=1), better food (n=2). The seven patients (23%) who benefited from the IV immunoglobulin treatments at home gave the following reasons: better comfort (n=2), some improvement in muscular deficit, etc), which explains the difference between the two evaluations. Cases where the IV immunoglobulin resulted in a reduced use of corticosteroids, or cases where IV immunoglobulins made it possible to avoid using immunosuppressive drugs were regarded as a success by the senior doctor, whereas patients did not necessarily have the same impression.

The 23 patients (77%) who said they had benefited from the IV immunoglobulin treatments at home gave the following reasons: better comfort (n=12), presence of next of kin (n=10), more occupation (n=6), time gain (n=5), better mood (n=3), maintaining activities (n=3), avoiding repeated trips to the hospital (n=3), better quality of sleep (n=2), better food (n=2). The seven patients (23%) who preferred the treatments at the hospital gave the following reasons: better monitoring, less trouble (IV immunoglobulin collected at the hospital, packaging of the medicine, collection of tubes, needles, and peripheries at the pharmacy and at home).

The mean cost of a treatment in hospital was $2701 against $2471 for a treatment at home. The difference seems to be modest, yet for the 277 treatments performed at home over five years, the savings for the community amount to $63 691 with $85 377 of budget revenues for the hospital (the 15% increase is in fact invoiced by the hospital administration for management and traceability costs). By this procedure, we have achieved a virtual economy on our drug budget and small equipment of $580 556 in the past five years (table 1). In the light of our experience and published reports of side effects, we propose some guidelines for home IV immunoglobulin infusion for patients with autoimmune disease (table 2). This procedure is appreciated by the patients and medical board and contributes to balancing the expenses for the National Health System.

**References**


**Elastofibroma dorsi**

Elastofibroma is a rarely diagnosed benign fibroproliferative lesion which occurs most commonly in the periscapular region of middle aged to elderly women. Recognition of the lesion is important as the differential diagnosis includes other benign and also

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**Table 1** Evaluation of the cost of at home IV immunoglobulin treatments (n=277) and comparison with the theoretical cost in hospital

<table>
<thead>
<tr>
<th>IV immunoglobulin</th>
<th>24 h hospital stay with hospital lump sum</th>
<th>Small equipment</th>
<th>Nursing</th>
<th>Total cost for 277 treatments</th>
<th>Savings achieved for 277 treatments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Theoretical cost in hospital</td>
<td>$2055 (deduction on drug budget)</td>
<td>$605</td>
<td>$41</td>
<td>$748274</td>
<td>$580556 (representing the virtual economy made by the hospital department (drug budget + small equipment))</td>
</tr>
<tr>
<td>Cost for one treatment in hospital</td>
<td>$2701</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Effective cost at home</td>
<td>$2363 (15% of retrocession overcost*)</td>
<td>0</td>
<td>$41</td>
<td>$68588</td>
<td>$63691 (representing the effective savings for the community) $85377 (representing the budget income for the hospital administration)</td>
</tr>
<tr>
<td>Cost for one treatment at home</td>
<td>$2471</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*In France when a drug is retroceded by a hospital pharmacy, it is invoiced 15% higher, the difference being paid to the hospital administration to cover the management and traceability costs.

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**Table 2** Home IV immunoglobulin infusion guidelines for patients with autoimmune disease

1. Need for a defined diagnosis
2. Presence of rational physiopathological basis that could “legitimise” the use of IV immunoglobulin
3. Senior hospital prescription
4. Respect of the contraindication of home IV immunoglobulin programme: coronaropathy, insufficiency or ischaemic cardiopathy, recent stroke, nephropathy, uncontrolled hypertension, thrombosis of the perfused vein, hypersensibility reaction after the first or second hospital infusion
5. More than one hospital based infusion before infusion at home to assess the tolerance
6. Average flow rate of IV immunoglobulin no quicker than 10 g per two hours
7. Collaboration with a home care organisation for visiting nurses and for collection of tubing and used bottles

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**Acknowledgments**

Les Ulis, 91958 Courtaboeuf cedex) who helped us with the technical aspect of this study.

**References**


**Elastofibroma dorsi**

Elastofibroma is a rarely diagnosed benign fibroproliferative lesion which occurs most commonly in the periscapular region of middle aged to elderly women. Recognition of the lesion is important as the differential diagnosis includes other benign and also
malignant tumours. We report a case of elastofibroma in a patient who presented with shoulder pain to a rheumatology clinic, and review previous publications. Although elastofibroma is uncommon, it has received attention in radiological and orthopaedic publications but not in rheumatology published reports.

A 43 year old Turkish woman, previously fit and healthy, was referred to our outpatient clinic with a two year history of right shoulder pain. The pain was described as a dull ache of gradual onset, around the posterior aspect of the shoulder over the scapula, which was worse on movement of the arm. There was no weakness. Over the preceding four months the patient had noticed a swelling below the inferior angle of the scapula which would appear and disappear with movement of the arm. The patient had no other medical history or relevant family history.

On examination there was a full range of movement of both shoulders and neck with no wasting or neurological signs. Pain was reproduced around the right shoulder when the arm over the scapula, which was 7 cm in size, moved. The patient was fit and healthy, with no underlying disease or risk factors, including HIV infection. He had no history or relevant family history. There was no history of immunosuppression or local steroid injection. The cause and pathogenesis of elastofibroma remains uncertain. The lesion was situated deep to the trapezius muscle and was not visible on physical examination.

The differential diagnosis includes desmoid tumours, neurofibroma, and liposarcoma. However, these tumours usually show soft tissue density in the periscapular region, usually on the right side. The lesions have occasionally been found in the extremities, head, abdominal and thoracic cavities. Of those in the subscapular region approximately 10% are bilateral. The cause and pathogenesis of elastofibroma is unclear, but it is suspected that subclinical microtrauma may lead to reactive hyperplasia of elastic fibres with consequently increased number of elastic fibres. Clinically, over 50% of subjects are asymptomatic and may present with a painless swelling; approximately 25% present with a clicking sensation when the arm is moved, while fewer than 10% present with pain.

Plain radiographs may be normal or may show soft tissue density in the periscapular region when the scapula is raised. Computed tomography usually shows a heterogeneous soft tissue mass with similar signal intensity to that of skeletal muscle but interspersed with high signal intensity areas representing adiopose strands. The differential diagnosis includes desmoid tumours, neurofibroma, and liposarcoma. However, these tumours usually show strong enhancement after gadolinium injection. Usually faint enhancement is seen with elastofibromas, although marked enhancement, mimicking malignant tumour, has been occasionally reported. Biopsy should therefore be undertaken as the confirmatory procedure and to exclude sarcoma.

In cases where the patient is asymptomatic excision is unnecessary. Malignant transformation is unknown. In symptomatic cases local excision is the best treatment. Recurrence has not been reported.

We conclude that elastofibroma should be considered in the differential diagnosis of subscapular pain. Although an uncommon lesion with a variable clinical presentation, the site and MRI appearances are characteristic. Awareness of the benign nature avoids unnecessary surgery and reassures a symptomatic patient.

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References

Olecranon bursitis due to Candida parapsilosis in an immunocompetent adult

Septic bursitis (SB) mainly affects the olecranon and patellar bursae. Subcutaneous localisation predisposes to trauma and may subsequently lead to infection. Most cases of SB are related to the subject’s occupation (roofing, gardening, plumbing), but surgical interventions (aspiration, intrabursal injection) are among other probable causes. Bacteria account for most cases, Staphylococcus aureus being the most commonly found (80%). Fungal isolation is quite rare and always associated with immunosuppression or debilitating conditions, but some species of Candida, Cryptococcus, Penicillium, and Sphorothrix schenckii have been described. These atypical organisms usually develop in a late indolent pattern, and a delay in diagnosis and treatment may lead to considerable difficulties in eradication of infection. We report a case of SB caused by Candida parapsilosis in a previously healthy man, with no underlying disease or any risk factors, including HIV infection, who probably acquired joint infection at the hospital secondary to local steroid injection.

Case report
A 32 year old man with a one month history of mild inflammation of the right elbow presented to our hospital on 19 May 2000. He had
an unremarkable past medical history, which did not include any toxic habits or recent trauma. Bursal aspiration showed that the synovial fluid had inflammatory characteristics (leucocyte count 4.9 x 10^6 cells/l (54% neutrophils), and a glucose level of 3.8 mmol/l), but there were no crystals and a fluid culture was negative. A diagnosis of olecranon bursitis was established, and conservative management (bursa aspiration was decided on). Bursal effusion was repeated over the next four days, so a further aspiration was carried out and local injection with triamcinolone acetate (20 mg) was given. However 24 days later the pain worsened and swelling of the elbow recurred; a fluid culture was not carried out. Laboratory synovial findings showed a leucocyte count of 15.7 x 10^6 cells/l (60% neutrophils) and a low glucose level (0.8 mmol/l). Culture yielded a few colonies of C albicans spp, but antifungal treatment was not started because it was considered that this might be caused by contamination. One month later (28 July), the patient presented to the emergency room owing to development of a new extremely painful episode of bursitis. After joint aspiration, a steroid injection was again given, but pain, systemic lupus erythematosus; 5-FC, 5-fluorocytosine.

Table 1 Main clinical features of candida bursitis

<table>
<thead>
<tr>
<th>Case [ref]</th>
<th>Age/sex</th>
<th>Candida strains</th>
<th>Localisation</th>
<th>Underlying disease/ risk factors</th>
<th>Probable source</th>
<th>Treatment</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 [3]</td>
<td>73/M</td>
<td>C. albicans</td>
<td>Subacromial</td>
<td>SLE/steroids</td>
<td>Candidaemia</td>
<td>AMB</td>
<td>Cure</td>
</tr>
<tr>
<td>2 [5]</td>
<td>77/M</td>
<td>C. tropicalis</td>
<td>Olecranon</td>
<td>Bladder carcinoma</td>
<td>Candidaemia</td>
<td>AMB + bursctomy</td>
<td>Cure</td>
</tr>
<tr>
<td>3 [6]</td>
<td>48/M</td>
<td>C. tropicalis</td>
<td>Popliteal</td>
<td>Lymphoma/immunosuppressive drugs</td>
<td>Candidaemia</td>
<td>AMB + surgery</td>
<td>Cure</td>
</tr>
<tr>
<td>4 [7]</td>
<td>64/M</td>
<td>C. albicans</td>
<td>Popliteal</td>
<td>Alcoholism/steroids, antibiotics</td>
<td>Candidaemia</td>
<td>AMB, ketoconazole</td>
<td>Cure</td>
</tr>
<tr>
<td>5 [8]</td>
<td>59/F</td>
<td>C. lusitaniae</td>
<td>Olecranon</td>
<td>SLE, diabetes, asthma/steroids, immunosuppressive drugs</td>
<td>Superficial trauma</td>
<td>Fluconazole, 5-FC</td>
<td>Failure</td>
</tr>
<tr>
<td>6 [CR]</td>
<td>32/M</td>
<td>C. parapsilosis</td>
<td>Olecranon</td>
<td>None</td>
<td>Steroid injection</td>
<td>Fluconazole + bursctomy</td>
<td>Cure</td>
</tr>
</tbody>
</table>

CR, current report; AMB, amphotericin B; SLE, systemic lupus erythematosus; 5-FC, 5-fluorocytosine.

day would have been more suitable for an infection in a deep compartment. Because unusual micro-organisms are difficult to recognise and anti-inflammatory drugs may mask the symptoms, a higher degree of awareness is necessary to achieve prompt diagnosis and successful treatment. Nevertheless, special care must be taken to avoid complicating side effects in iatrogenic manipulations, so preventive measures to reduce the incidence of infection must never be omitted.

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References
Prevalence of allergic respiratory diseases in patients with RA

The balance between Th1 and Th2 cell activity is crucial in many autoimmune diseases. It has been suggested that rheumatoid arthritis (RA) is a Th1 cell predominated disease, whereas atopic diseases are Th2 cell directed. Some recent observations suggest a difference of atopy in patients with RA who have received a lot of attention. It has been suggested that a Th2 cell related disorder such as atopy might have a protective role against the onset of a Th1 mediated disease such as RA, and the biological importance of the Th1/Th2 paradigm has been emphasised. We evaluated the prevalence of atopic respiratory diseases in 126 consecutively observed RA patients. The diagnosis was based on a suggestive clinical picture associated with the positivity of skin prick tests. Seven of 21 patients also had symptoms of asthma and 2/21 had undergone specific immunotherapy before the onset of RA symptoms. In 20/21 patients allergic respiratory symptoms had started before the onset of RA symptoms. In 5/21 patients atopic symptoms had totally disappeared before the onset of RA. Patients with RA with associated atopic disease did not differ from other patients with RA in the following characteristics: (a) sex (76.2% female v 75.2%); (b) positivity of rheumatoid factor (71.4% v 63.8%); (c) presence of subcutaneous noduli and/or other cutaneous manifestations (14.3% v 21.9%); (d) functional class according to the ACR revised criteria (class I-II: 64%); (e) presence of coexistent Raynaud’s phenomenon (54.3%). Notably, most patients from both groups (90.9% v 76.8%) were taking steroids at a low dose—namely, not more than 5 mg daily of prednisone, when they were evaluated for this study.

Patients with atopic diseases were younger (mean age 53.8 v 57.5) and had a shorter average duration of RA (4.5 v 9.7 years) than those without.

We found a rather high prevalence of allergic respiratory diseases in our patients with RA (69%), comparable with that expected in the general population. Moreover, the presence of atopic disease did not seem to influence the severity of RA.

The difference between our data and other reports might be due to the methods used to determine the presence of atopic diseases. Those other studies started from the administration of standardized questionnaires to patients with RA and this method might have caused an underestimation of atopic symptoms. Conceivably, prolonged steroid treatment, as well as the systemic symptoms and disability associated with RA, may often cause occult symptoms of rhinitis and asthma that only emerge at deeper analysis.

In conclusion, our data question the hypothesis of a mutual antagonism of RA and atopy, suggesting caution in interpreting previous data and confirming that things are often not as simple as they can seem at first glance.

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References

Henoch-Schönlein purpura: a possible complication of hepatitis C related liver cirrhosis

Henoch-Schönlein purpura (HSP) is a systemic small vessel vasculitis predominantly affecting children and, less commonly, adults. Classical HSP includes a tetrad of palpable purpura, arthritis, abdominal pain, and nephritis. Although HSP can present with any two of the four criteria in the tetrad (87% sensitivity and specificity). Gastrointestinal disease has been recorded in up to 82% of adult patients in one series and is usually self-limiting with colicky abdominal pain, but may progress to ischaemic bowel perforation.

We present the case of a 63 year old man with IgA vasculitis, probably HSP confounded by undiagnosed hepatitis C related cirrhosis. He was admitted with a two week history of dyspnoea, malaise, cough, fevers, and chills, myalgias, one day of a non-blanching erythematous rash on his legs, and an ileus. His hepatitis C antibody was positive; table 1 shows the results of other laboratory studies.

Cultures of cerebrospinal fluid, blood, and urine were negative. A colonooscopy was non-diagnostic.

Leucocytoclastic vasculitis was confirmed by skin biopsy, and direct immunofluorescence staining was positive for IgA deposits consistent with HSP (fig 1).

Treatment with high dose (1 mg/kg/day) intravenous corticosteroids was started. A second CT scan showed massive ascites, a partial superior mesenteric vein thrombosis, thickening, and focal and nodular irregularities throughout the small bowel (probable ischaemia), and pneumoperitoneum. Blood cultures disclosed septicemia with Bacteroides fragilis. His clinical course rapidly deteriorated and he died on day 8.

There are two previous case reports of the association between HSP and hepatitis C. The diagnosis of HSP in this patient is most likely, given palpable purpura, haematuria, abdominal pain, and a skin biopsy demonstrating IgA complexes (fig 1). However, the possibility of hepatitis C associated IgA/GM mixed cryoglobulinaemia cannot be ruled out despite a negative cryoglobulin screen on two occasions. In this patient an IgA mediated vasculitis may have been the nidus for thrombus formation and abdominal catastrophe.

The role of liver cirrhosis in the development of HSP is intriguing. Patients with cirrhosis may develop HSP as a consequence of cirrhosis related portal hypertension likely, given palpable purpura, haematuria, abdominal pain, and a skin biopsy demonstrating IgA complexes. In this patient an IgA mediated vasculitis may have been the nidus for thrombus formation and abdominal catastrophe.

In conclusion, our data question the hypothesis of a mutual antagonism of RA and atopy, suggesting caution in interpreting previous data and confirming that things are often not as simple as they can seem at first glance.
Severe aortic regurgitation in RF positive polyarticular JIA

An 18 year old girl of Moroccan origin with a clear medical history was transferred to the Netherlands in February 1989 because of a two year history of untreated polyarthritis. The disease had pursued a rapidly destructive course, resulting in contractures and ankylosis of hips, knees, shoulders, and elbows and small joint deformation. A diagnosis of juvenile idiopathic arthritis (JIA) polyarticular type, functional class IV was made. No nodules were present. Laboratory analysis at that time showed borderline positive serum rheumatoid factor (RF) 30 IE/ml. Tests for antinuclear antibodies and HLA-B27 were negative. Treatment was started with intensive physiotherapy and intramuscular gold, because of proteinuria. In 1990 she was treated for a unilateral uveitis. In 1992 her right elbow was replaced. Until 1993 cardiac study showed no murmurs and chest roentgenogram was normal. The disease had pursued a rapidly destructive course, resulting in contractures and ankylosis of hips, knees, shoulders, and elbows and small joint deformation. A diagnosis of juvenile idiopathic arthritis (JIA) polyarticular type, functional class IV was made. No nodules were present. Laboratory analysis at that time showed borderline positive serum rheumatoid factor (RF) 30 IE/ml. Tests for antinuclear antibodies and HLA-B27 were negative. Treatment was started with intensive physiotherapy and intramuscular gold, because of proteinuria. In 1990 she was treated for a unilateral uveitis. In 1992 her right elbow was replaced. Until 1993 cardiac study showed no murmurs and chest roentgenogram was normal. The disease had pursued a rapidly destructive course, resulting in contractures and ankylosis of hips, knees, shoulders, and elbows and small joint deformation. A diagnosis of juvenile idiopathic arthritis (JIA) polyarticular type, functional class IV was made. No nodules were present. Laboratory analysis at that time showed borderline positive serum rheumatoid factor (RF) 30 IE/ml. Tests for antinuclear antibodies and HLA-B27 were negative. Treatment was started with intensive physiotherapy and intramuscular gold, because of proteinuria. In 1990 she was treated for a unilateral uveitis. In 1992 her right elbow was replaced. Until 1993 cardiac study showed no murmurs and chest roentgenogram was normal. The disease had pursued a rapidly destructive course, resulting in contractures and ankylosis of hips, knees, shoulders, and elbows and small joint deformation. A diagnosis of juvenile idiopathic arthritis (JIA) polyarticular type, functional class IV was made. No nodules were present. Laboratory analysis at that time showed borderline positive serum rheumatoid factor (RF) 30 IE/ml. Tests for antinuclear antibodies and HLA-B27 were negative. Treatment was started with intensive physiotherapy and intramuscular gold, because of proteinuria. In 1990 she was treated for a unilateral uveitis. In 1992 her right elbow was replaced. Until 1993 cardiac study showed no murmurs and chest roentgenogram was normal. The disease had pursued a rapidly destructive course, resulting in contractures and ankylosis of hips, knees, shoulders, and elbows and small joint deformation. A diagnosis of juvenile idiopathic arthritis (JIA) polyarticular type, functional class IV was made. No nodules were present. Laboratory analysis at that time showed borderline positive serum rheumatoid factor (RF) 30 IE/ml. Tests for antinuclear antibodies and HLA-B27 were negative. Treatment was started with intensive physiotherapy and intramuscular gold, because of proteinuria.  

Figure 1 Immunofluorescence staining of a skin biopsy from a purpuric lesion. Direct immunofluorescence study showing granular deposition of IgA in the walls of superficial dermal blood vessels, a characteristic finding in Henoch-Schönlein purpura.


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References


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changes of fibrosis and necrosis. Valvular involvement has been described in patients with all types of JIA, the aortic valve being most commonly affected. Valvular disease is associated with severe destructive articular disease.

Furthermore, our case report confirms the possibility of successful mechanical aortic valve replacement in a case of severe progressive aortic valve insufficiency and secondary angina pectoris in a patient with polyarticular JIA.

We recommend regular cardiac appraisal as part of the routine assessment of every patient with JIA. Whenever cardiac murmurs are detected in these patients, echocardiographic assessment should be considered, because if there is valve insufficiency the cardiac function may deteriorate and cardiac surgery may be needed.

Acknowledgments

We are grateful to Dr J van der Meulen, cardiologist, for the surgical description and to Dr AC van der Wal, pathologist, for his pathology specimen evaluation. We thank Dr FM Westerweel, rheumatologist, for allowing us to report on her patient.

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References


Polymyalgia rheumatica and pericardial tamponade

Polymyalgia rheumatica causes symmetrical stiffness in the neck, shoulder, and pelvic girdles, and affects middle aged and elderly people, with a higher incidence among women. A group of systemic, non-specific complaints such as weight loss, moderate fever, asthenia, and persistent high erythrocyte sedimentation rate are other clinical features.

The association of polymyalgia rheumatica and pericardial effusion has already been described in two cases.

A 73 year old woman was admitted for the evaluation of pericardial effusion and mild anemia. Polymyalgia rheumatica was suspected because the patient had had asthenia, stiffness, and pain in the shoulders and hips for about a year before coming to hospital. She had also lost 5 kg in a few months. A few days before admission she had presented worsening dyspnoea.

An echocardiogram showed large pericardial effusion and initial findings of cardiac tamponade (right atrial and right ventricular diastolic collapse), so a pericardiocentesis was done: polymerase chain reaction tests in the pericardial fluid for Mycobacterium tuberculosis and cultures for aerobic and anaerobes were negative; tumoral cells were absent. Serological tests for antibodies to cytomegalovirus, herpes simplex and Epstein-Barr viruses, anti-smooth muscle, antinuclear, anti-DNA, and anti-extractable nuclear antigen antibodies were negative. A titre of 32 serologically reactive protein (CRP) was 85 mg/l.

The patient was first treated with indometacin (50 mg twice a day) for a week, with no improvement, and then with low doses of prednisone (10 mg/day): the symptoms markedly improved and the ESR and CRP dropped to 27 mm/1st h and 12 mg/l, respectively, in a few weeks. An echocardiogram a month later was negative for pericardial effusion; ESR and CRP were also normal.

The patient has remained entirely well after a follow up of one year.

The presenting symptoms (girdles bilateral and symmetrical stiffness and pain) are accompanied by systemic features (fatigue, weight loss, raised ESR) and the marked improvement after prednisone confirm the diagnosis of polymyalgia rheumatica.

As far as we know this is the first report of pericardial tamponade requiring pericardial drainage in this disease.

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References


Remission of Behçet’s syndrome with TNFα blocking treatment

Goossens et al reported on a patient in whom a remission of Behçet’s syndrome was induced with tumour necrosis factor (TNFα) blocking treatment. We would like to add our experience in a patient with Behçet’s disease associated with rheumatoid arthritis (RA), treated with infliximab (Remicade).

A 47 year old male patient, born in Morocco, living in Israel, was diagnosed 14 years earlier with severe polyarthritis of hands, feet, and knees. Radiography showed articular bone erosions; rheumatoid factor was positive, with a high erythrocyte sedimentation rate and C-reactive protein. In parallel, the patient reported recurrent buccal and genital ulcers two to three times a month with papulopustular skin lesions on the feet. HLA-B5 (51) was positive. There was no eye involvement. A diagnosis of Behçet’s disease associated with erosive, seropositive RA was suggested. The patient was treated with sulfa-salazine and colchicine without improvement; steroid treatment with auranofin was added. The disease worsened and the patient had progressive erosions in hands, knees, and feet. Later, pulse steroids, methotrexate, azathioprine, and cyclosporin were added serially, either singly or in combination.

In subsequent years he became dependent on steroids and never achieved complete remission. In December 2000 the patient was admitted to hospital with severe active polyarthritis, flexion contractures of the elbows, and an especially swollen left knee with Baker’s cyst and severe erosive disease. The patient additionally had buccal and penile ulcers. Because of the lack of response to conventional treatment we decided to treat him with infliximab (Remicade; Schering), a chimeric IgG monoclonal antibody directed against TNF. He received 300 mg intravenously (3 mg/kg) at intervals of two weeks, six weeks, and then every eight weeks. Two weeks after the first infusion the ulcers of mouth, penis, and other skin lesions were already considerably smaller and later disappeared. The polyarthritis improved considerably, except for the left knee, which required total removal. Infliximab was given with continued colchicine and azathioprine. Our case, as in Goossens’ report, suggests that infliximab may have a beneficial therapeutic effect in microsorbal and cutaneous lesions as well as...
Synovitis in Behçet's disease, in our case in association with RA.

Controlled studies will be needed to assess adequately the full effect of TNF antagonists in Behçet's disease.

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Fatigue and immune activity in Sjögren’s syndrome

Despite major desiccation of mucous membranes in Sjögren’s syndrome (SS), fatigue is often experienced by patients as the most disabling complaint. Unfortunately, there is no proper treatment available to combat the fatigue in SS. Beside a variety of somatic and non-somatic conditions, increased immune activity has been implicated as a cause of fatigue in autoimmune diseases. If responsible for fatigue in SS, it could serve as a treatment target. The purpose of this study was, therefore, to examine the relation between fatigue and immune variables in SS.

Thirty six consecutive patients with primary SS visiting our outpatient departments participated in this study. Two control groups were used: a group of 18 patients diagnosed with secondary SS, and a group of 18 non-medicated healthy controls. Diagnoses were based on the revised European criteria for the classification of SS. Eighteen patients diagnosed the amount of fatigue in patients with primary SS could be explained by depression, total level of immunoglobulins, and thromboocyte counts (p<0.001). Both depression and thromboocyte counts showed a significant positive correlation, whereas levels of immunoglobulins showed a negative correlation.

Though treating as a treatment target, the immune and inflammatory variables failed to predict fatigue satisfactorily in primary SS. Levels of immunoglobulins showed, surprisingly, a significant negative correlation. Thromboocyte counts showed a significant positive correlation. Although increases in thromboocytes follow the acute phase reaction, no significant correlation between thromboocyte counts and CRP levels were found. A chance association between fatigue and thromboocyte counts as well as immunoglobulin levels seems thus possible. Therefore, the intriguing question whether immune or inflammatory activity is a causative factor of chronic fatigue in SS remains unraveled. Because no difference in fatigue was found between patients with primary and secondary SS, the presence of another autoimmune disease appears to have no additional effect on the amount of fatigue in SS. In agreement with findings of previous studies, a significant relation was found between the degree of fatigue and the level of depression in patients with primary SS. It is concluded that none of the laboratory variables reflecting immune activity predict fatigue satisfactorily in primary SS. Signs of depression, as present in most of the patients with primary SS, proved to be the most relevant cause of their exhausting fatigue. Therefore we recommend including a psychosomatic approach in the treatment of fatigue in primary SS.

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References

8. Rhein J. Increased immune activity predict fatigue satisfactorily in primary SS. Levels of immunoglobulins showed, surprisingly, a significant negative correlation. Thromboocyte counts showed a significant positive correlation. Although increases in thromboocytes follow the acute phase reaction, no significant correlation between thromboocyte counts and CRP levels were found. A chance association between fatigue and thromboocyte counts as well as immunoglobulin levels seems thus possible. Therefore, the intriguing question whether immune or inflammatory activity is a causative factor of chronic fatigue in SS remains unraveled. Because no difference in fatigue was found between patients with primary and secondary SS, the presence of another autoimmune disease appears to have no additional effect on the amount of fatigue in SS. In agreement with findings of previous studies, a significant relation was found between the degree of fatigue and the level of depression in patients with primary SS. It is concluded that none of the laboratory variables reflecting immune activity predict fatigue satisfactorily in primary SS. Signs of depression, as present in most of the patients with primary SS, proved to be the most relevant cause of their exhausting fatigue. Therefore we recommend including a psychosomatic approach in the treatment of fatigue in primary SS.

### Table 1 Dutch Fatigue Scale

<table>
<thead>
<tr>
<th>Fatigue Item</th>
<th>Scored on a 1 to 4 point scale</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Listlessness</td>
<td>1</td>
</tr>
<tr>
<td>2. Perceived need for additional energy to finish required tasks</td>
<td>2</td>
</tr>
<tr>
<td>3. Verbalisation of an unmitting and overwhelming lack of energy</td>
<td>3</td>
</tr>
<tr>
<td>4. Inability to restore energy, even after sleeping</td>
<td>4</td>
</tr>
<tr>
<td>5. Increase in rest requirements</td>
<td>5</td>
</tr>
<tr>
<td>6. Decreased libido</td>
<td>6</td>
</tr>
<tr>
<td>7. Inability to maintain usual routine</td>
<td>7</td>
</tr>
<tr>
<td>8. Impaired ability to concentrate</td>
<td>8</td>
</tr>
<tr>
<td>9. Decreased performance</td>
<td>9</td>
</tr>
</tbody>
</table>

Glucocorticoids


People are bound to think: Oh no! Is yet another book about drugs that we are using successfully every day really necessary? Well, the answer to this question is: Yes, it is! More than 50 years after the clinical introduction of these drugs, updates are necessary to establish Milestones in drug therapy (the title of the series published by Birkhäuser). Sometimes unnoticed by all who use glucocorticoids, new, not always spectacular, but still significant knowledge has been gained about these vital drugs and how they should be administered. The authors try to put this across in a readable form, which means that known information is recapitulated concisely and new information is included. A very good example are the chapters that deal with the basic mechanisms of action. However, the only real criticism also applies at this point: some comments are redundant and tighter editing would have improved individual contributions.

Renowned authors reflect upon the most important facets of treatment with glucocorticoids. These facets include the history as well as basic biology, the development of synthetic compounds, extensive discussions about the glucocorticoid receptor, the dynamics of cytokines and other gene regulations by glucocorticoids, the interrelationship between exogenous and endogenous steroids, and a clinical section which deals with the use of steroids in asthma, arthritis, and inflammatory bowel disease. Allan Mumck, one of the

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wise men of steroid research, describes the history of the glucocorticoids graphically and in detail. He has enriched research in this field with significant contributions since the beginning of the 1960s and now looks back amusingly and expressively on the past decades. Luca Parente’s contribution ranges from naturally occurring to synthetic glucocorticoids and their effects in the organism. The sections that deal with the desired anti-inflammatory/immunomodulatory effects and adverse reactions give a valuable overview.

A few chapters should be highlighted that are of particular interest for both rheumatologists and clinical immunologists. That on molecular and cellular aspects of cytokine regulation by glucocorticoids has been prepared very carefully from a didactic point of view. It not only describes T cell activation and the effect of glucocorticoids thereon, but also provides useful information for an understanding of the function and regulation of cytokines. It is recapitulated that the central therapeutic effects of glucocorticoids are ultimately the inhibition of the synthesis of interleukin 2 and interleukin 6; glucocorticoids influence the transcription of around 1% of all genes! However, they also have an influence on the translational and post-translational mechanisms by which proteins are synthesised, processed, and exported from cells. This fact applies, in particular, to the influence on cytokine metabolism. Just to mention a few key concepts: post-transcriptional, translational, and post-translational mechanisms; modulation of cytokine receptors; indirect effects that occur as a result of the extensive interactions among various cytokines.

The chapter written by John Kirwan is worth reading for the rheumatologist, as it deals with the clinical aspect of the systemic administration of glucocorticoids in chronic inflammatory arthritis (typified by rheumatoid arthritis (RA)), in vesicular epithides typified by those in systemic lupus erythematosus, and in polymyalgia rheumatica and temporal arteritis. It is clearly written, because it questions apparently known facts, especially taking the example of RA. The very important short-term anti-inflammatory effects are accepted and are broadly exploited. But is the risk/benefit potential also positive for medium and long-term treatment? Do the glucocorticoids perhaps have a much more fundamental influence on the development and progression of RA than previously thought? Is there a differentiated and even treatment-time-dependent influence on synovitis, on the one hand, and on radiological progression, on the other? Possible answers to these exciting questions will not be anticipated here. However, this chapter, in particular, can be recommended, broadening as it does our picture of reality that is sometimes restricted to standard viewpoints.

The non-expert in the field might have wished for a little more clarity occasionally in the illustrations. The references to the individual chapters take into account publications up to and including the year 2000. Overall, this is a good example of how knowledge on established drugs such as the glucocorticoids can be clearly updated.

F Buttgereit

FORTHCOMING EVENTS

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@fs1.sert.man.ac.uk

OMERACT VI
11–14 Apr 2002; Brisbane, Queensland, Australia
Includes two modules: MRI and economics; and five workshops: patients’ perceptions, imaging (healing), progressive systemic sclerosis, mean clinical important difference, and osteoarthritis.
Contact: Conference Organisers Q2Q, 7 Swan Street, Old Isleworth, Middlesex TW7 6RJ, UK
Fax: +44 20 8569 9555
Email: q2q@q2q.co.uk

British Society for Rheumatology XIth 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: Esperanzo 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: Debra J Bork, The Cleveland Clinic Foundation, Desk A50, Center for Vasculitis Care and Research, 9500 Euclid Avenue, Cleveland, OH 44195, USA
Tel: 216 445 8333
Fax: 216 445 7569
Email: borkd@ccf.org
Website for registration and abstract submission: www.clevelandClinicmeded.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@iofy.org
Website: www.osteofound.org

5th European Conference on Systemic Lupus Erythematosus
26–30 May 2002; Athens, Greece
Chairman Professor HM Moutsopoulos
Contact: Amiripolis Congress Organising Bureau
Email: moutsop@med.oua.gr
Email: congress@amphitrition.gr

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

10th International Congress on Behçet’s Disease
27–29 June 2002; Berlin, Germany
Under the auspices of the International Society for Behçet’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, Fabeckstrasse 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 27294
Fax: 47 776 27258
Email: 29csc2002@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, 18/20 - 27100 Pavia, Italy
Email: tra@e20pr.com
Website: www.e20pr.com
Congress website: www.medicine.ucsd.edu/albani/2001 meeting

OsteoArthritis Research Society International (OARSI) World Congress
22–25 Sep 2002; Sydney, Australia
Contact: OsteoArthritis Research Society International (OARSI), 2055 M Street, NW, Suite 800, Washington DC 20036, USA
Tel: +1 202 367 1177

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Website: www.eular.org/courses and Email: enaredo@eresmas.com

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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

Third International Congress on Spondyloarthopathies
2–5 Oct 2002; Gent, Belgium
Topics covered will be:
• Innate immunity
• Genetics and HLA-B27
• Animal models and pathogenesis
• Clinical research and therapy
Deadline for abstract submission 31 March 2002
Contact: Organisation and secretariat, Medicongress, Waalpoel 28–34, B-9960 Assenede, Belgium
Tel: +32 9 344 39 59
Fax: +32 9 344 40 10
Email: congresses@medicongress.com
Website: www.medicongress.com

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.eyane.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 250, 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 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

Future EULAR congresses
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9–12 June 2004; EULAR 2004 Berlin, Germany
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21–24 June 2006; EULAR 2006 Amsterdam, The Netherlands

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