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).

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. 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. 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. 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 sonography and magnetic resonance imaging, 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 Criteria</th>
<th>Sialography</th>
<th>Sonography</th>
<th>Saxon</th>
<th>Schirmer</th>
<th>SS-A</th>
<th>SS-B</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sensitivity [%]</td>
<td>87</td>
<td>76</td>
<td>70</td>
<td>59</td>
<td>83</td>
<td>88</td>
</tr>
<tr>
<td>Specificity [%]</td>
<td>98</td>
<td>94</td>
<td>71</td>
<td>57</td>
<td>56</td>
<td>42</td>
</tr>
<tr>
<td>Accuracy [%]</td>
<td>92</td>
<td>84</td>
<td>71</td>
<td>59</td>
<td>70</td>
<td>51</td>
</tr>
</tbody>
</table>

Univariate analysis

<table>
<thead>
<tr>
<th>Coefficient</th>
<th>SE</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sialography</td>
<td>6.02</td>
<td>3.69</td>
</tr>
<tr>
<td>Sonography</td>
<td>0.75</td>
<td>0.39</td>
</tr>
<tr>
<td>Saxon</td>
<td>1.67</td>
<td>0.56</td>
</tr>
<tr>
<td>Schirmer</td>
<td>1.92</td>
<td>0.32</td>
</tr>
<tr>
<td>SS-A</td>
<td>1.66</td>
<td>0.51</td>
</tr>
<tr>
<td>SS-B</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Multivariate analysis

<table>
<thead>
<tr>
<th>Coefficient</th>
<th>SE</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sialography</td>
<td>4.87</td>
<td>3.97</td>
</tr>
<tr>
<td>Sonography</td>
<td>1.06</td>
<td>1.07</td>
</tr>
<tr>
<td>Saxon</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Schirmer</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SS-A</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SS-B</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

NS, not significant.
Radiographs of the hands and feet were normal. There were slight erosions of the sacroiliac 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 joint. For the psoriatic nails he took acitretin (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 psoriatic nails the patient was 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 arthritides.

Discussion

Nail disease is significantly associated with PsA.1 It is particularly common in cases with DIP joint involvement and tends to indicate 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 pathiological condition of our patient. Dermatological assessment of patients treated with sulfasalazine for PsA has been reported in two series; according to the report published in the series of Gupta et al., patients treated with sulfasalazine for PsA showed signs of cutaneous improvement compared with those receiving placebo.2 The series of Farr et al. reports improved cutaneous lesions in as few as 3/15 patients treated with sulfasalazine and 1/15 patients receiving placebo.3 However, we could not find any indication of the evolution of possible simultaneous psoriatic nail lesions.

Treatment of PsA with cyclosporin or etanercept is effective for both joint and skin lesions of psoriasis;4 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 canal and interglottal fold were seen, prompting the diagnosis of psoriasis partim inversa.

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, this does not take into account the benefits gained—for example, the savings achieved in the costs of corticosteroids and immunosuppressive drugs and, above all, the improvement in quality of life achieved through functional improvement, as noticed in inflammatory myopathies and Still’s disease.26 It is precisely to minimise the costs of IV immunoglobulin treatments and to enable patients to remain at home that we have developed the administration of IV immunoglobulins at home when sequential treatments are necessary.

Between January 1995 and March 2000 30 patients (18 women, 12 men) were enrolled, with a mean (SD) age of 44 (0.9) for the women and 51 (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 Tégéline (314 treatments), Endobiline (81 treatments), and Gammagard (three treatments). All the patients had a corticosteroid or refractory autoimmune disease (mostly polymyositis, dermatomyositis, and adult onset Still’s disease).

The doses prescribed for each treatment were generally 2 g/kg. Treatments were carried out monthly and consisted of two days when performed in hospital and five days when performed at home. The average flow rate of the IV immunoglobulin perfusions performed at home was 10 g/2 h (extreme values: 30 min–4 h). The secondary effects of the treatments at home remained conventional and minor.

The efficacy of the IV immunoglobulin was determined by the patients as very good in 17% of cases, good in 33%, modest in 3%, nil in 47%. The efficacy of the IV immunoglobulin was described by the senior doctor as very good in 33%, good in 30%, nil in 17%. Evaluation of the efficacy described by the patients themselves was based on purely functional criteria (general condition, pain,
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 pharmacy, calling the nurse at home, stay with the technical aspect of this study).

The mean cost of a treatment in hospital was $2701 against $2471 for a treatment at home.

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>$41 (deduction on small equipment budget)</td>
<td>0</td>
<td>$748274</td>
<td>$580556 (representing the virtual economy made by the hospital department (drug budget + small equipment))</td>
</tr>
<tr>
<td>Effective cost at home</td>
<td>$2363 (15% of retrocession overcost*)</td>
<td>0</td>
<td>$41</td>
<td>$684588</td>
<td>*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.</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>Cost for one treatment at home</td>
<td>$2471</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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.

Acknowledgments
To Monique Tomczak who typed this document; Thomas Rémy, Bernard Dauvergne, and Mazen Sztajzel (Laboratoire français du fractionnement et des biotechnologies, 3 avenue des Tropiques, BP 105, Les Ulis, 91958 Courtaboeuf cedex) who helped us with the technical aspect of this study.

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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

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: coronaryopathy, insufficiency or ischaemic cardiopathy, recent stroke, nephropathy, uncontrolled hypertension, thrombosis of the perfused vein, hypersensitivity 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 |
fibroma. Surgical excision was performed.

The cause and pathogenesis of elastic fibroid tumours is not fully understood and is the subject of much speculation. However, there are several possible explanations.

- **Microtrauma Hypothesis:** This theory suggests that repetitive microtrauma, such as from sports or occupational activities, can lead to the formation of elastofibroma. The trauma may cause injury to the elastic fibres, leading to their degeneration and replacement with fibrous tissue over time.

- **Mechanical Hypothesis:** Some authors propose that the mechanical stress on the shoulder, especially during movements such as lifting or overhead activities, can cause the formation of elastofibroma. The repetitive movement of the arm over the scapula may lead to microtrauma and the development of the lesion.

- **Genetic Hypothesis:** There is also evidence suggesting a genetic component in the development of elastofibroma. Some studies have found a familial incidence of elastofibroma, indicating a possible genetic predisposition.

- **Immunological Hypothesis:** Another theory involves an immunological reaction against the elastic fibres. This could lead to the formation of a tissue reaction around the affected fibres, resulting in the development of the lesion.

- **Hormonal Hypothesis:** Some researchers have suggested that hormonal changes, particularly in women, could contribute to the development of elastofibroma. The hormonal fluctuations during puberty, pregnancy, or menopause may affect the elastic fibres and trigger the formation of the tumour.

**Presentation and Diagnosis**

- **Symptoms:** Elastofibroma typically presents as a slow-growing, painless mass over the scapula. The pain is usually described as a dull ache and may be exacerbated by movement of the arm and shoulder.

- **Signs:** Clinically, elastofibroma is often seen as a firm, well-circumscribed mass on physical examination. It is usually located between the serratus anterior and the thoracic cage, and sometimes involves the infraclavicular region.

- **Investigations:** The diagnosis of elastofibroma is primarily based on clinical examination and imaging studies. Plain radiographs may show soft tissue density in the periscapular region, although MRI is the best non-invasive technique for confirming the diagnosis. MRI can provide detailed images of the lesion, showing areas of high signal intensity that correspond to the fibrous tissue.

**Treatment**

Local excision is the treatment of choice for elastofibroma, as it is a benign lesion that rarely poses a diagnostic challenge. However, the third case presented here highlights the importance of considering elastofibroma in the differential diagnosis of shoulder pain, especially when other conditions have been previously ruled out.

**Conclusion**

Elastofibroma should be considered in the differential diagnosis of shoulder pain in patients presenting with a painless swelling. While it is a rare condition, its recognition is crucial to avoid unnecessary surgery and reassures a symptomatic patient.
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 4.9×10^3 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 (bursectomy and rest) 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 steroid injection was again given, but owing to development of a new extremely painful episode of bursitis. After joint aspiration, a steroid injection was again given, but this time a fluid culture was not carried out.

On 1 August clinical symptoms persisted. Physical examination showed an increase in the size of the olecranon bursa. The patient had never presented with fever, arthralgias, or any general complaints. Laboratory studies, including a test for antibodies to HIV, were normal or negative. Magnetic resonance imaging was performed showing multiseptate bursitis; the adjacent structures were normal. A removal of 10 ml bursa fluid again yielded a positive culture for *Candida* 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 this time a fluid culture was not carried out.

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Prevalence of allergic respiratory diseases in patients with RA

The balance between Th1 and Th2 cell activity is crucial in many autoimmune disorders. It has been suggested that rheumatoid arthritis (RA) is a Th1 cell predominated, whereas atopic diseases are Th2 cell directed. Some recent observations\(^3\) of a decreased presence of atopy in patients with RA 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,\(^2\) and the biological importance of the Th1/Th2 paradigm has been emphasised.

We evaluated the prevalence of atopic respiratory diseases in 126 consecutively observed outpatients with RA in the American College of Rheumatology (ACR) criteria. The presence of allergic respiratory diseases was investigated in all patients by an allergic rhinitis 

EAACI guidelines, of skin prick tests by a trained allergologist. 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 3/21 had undergone specific immunotherapy of skin prick tests. In 20/21 patients allergic respiratory symptoms had started before the onset of RA symptoms. In 5/21 patients atopic symptoms had totally disappeared by the time of this study.

Patients with RA with associated atopic disease did not differ from other patients with RA in the following characteristics: (a) sex (76.2% female vs 73.2%); (b) positivity of rheumatoid factor (71.4% vs 63.8%); (c) presence of subcutaneous noduli and/or other extracutaneous manifestations (14.3% vs 21.9%); (d) functional class according to the ACR revised criteria (class I-II: 64% vs 60%); (e) current treatment with two or more disease modifying antirheumatic drugs in combination (57.1% vs 60.9%); (f) current steroid treatment (57.1% vs 54.3%).

Notably, most patients from both groups (90.9% vs 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 ± 57.5) and had a shorter average duration of RA (4.5 vs 9.7 years) than those without.

We found a rather high prevalence of allergic respiratory diseases in our patients with RA (76.2%), comparable with that expected in the general population.\(^4\) Moreover, the presence of atopic disease did not seem to influence the severity of RA.

The difference between our data and other reported prevalence data might be due to the methods used to determine the presence of atopic diseases. Those other studies started from the administration of standardised questionnaires to patients with RA and this method might have underestimated the prevalence of 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 abdominal hypertension. In 20% of cases, there is a coexistence of 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\(^5\) and is usually self-limiting with colicky abdominal pain, but may progress to ischaemic bowel perforation.\(^6\)

We present the case of a 63 year old man with IgA vasculitis, probably HSP confounded by undiagnosed hepatitis C related liver cirrhosis. He was admitted with a two week history of dyspepsia, malaise, cough, fevers, and chills, myalgias, one day of 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 cerebrosphinal fluid, blood, and urine were negative. A coloscopy 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 INITIATED. A CONSIDERABLE TO MILD IMPROVEMENT WAS OBTAINED; HOWEVER, ON DAY 4 IT BECAME TACHYCARDIC AND DEVELOPED A TENDER ABDOMEN. A SECOND CT SCAN SHOWED MASSIVE ASCITES, A PROSPECTIVE MENSURING VEIN THROMBOSIS, THICKENING, AND LOCAL AND NODULAR IRREGULARITIES THROUGHOUT THE SMALL BOWEL (PROBABLE ISCHAEMIA), AND PNEUMOERTITIS. 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 case was most likely, given palpable purpura, haematuria, abdominal pain, and 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 a defect in the metabolism of IgA, resulting in a decrease in the production and deposition of the nephritogenic IgA subclass.\(^6\)

Henoch and paediatric HSP differ in the incidence and severity of renal involvement, with nephropathy and progression to renal insufficiency being greater in adult HSP \(^7\) which is associated with a poor outcome.\(^7\) Gastrointestinal manifestations vary widely and include abdominal pain, nausea/vomiting, intestinal haemorrhage and, rarely, perforation.\(^8\)

There have been no large clinical trials in adults with complicated HSP. Corticosteroids used in a series of children have been shown to relieve symptoms,\(^7\) but fail to deal prospectively with the prevention of HSP\(^\text{c}\) complications. Adults respond favourably to corticosteroids and may be managed with short courses of treatment,\(^7\) but corticosteroids may also mask severe abdominal catastrophe.

Several important points can be learnt from this case report:

1. Although nephritis is the most important long term prognostic factor in HSP in the short term, gastrointestinal disease can lead to death despite early therapeutic intervention.

2. Liver cirrhosis secondary to hepatitis C may precipitate development of HSP or mixed cryoglobulinaemic vasculitis through the defective metabolism of CICs.

3. Given the increasing incidence of hepatitis C related liver disease world wide, the association of these diagnoses and clinical implications should be considered more often.

Acknowledgments

We thank Drs Karen Stout, Brett Sheppard, Amy Howard, and Sandhya Venugopal for their participation in, and discussions about, this case.

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Table 1 Significant laboratory values on the day of admission

<table>
<thead>
<tr>
<th>Study</th>
<th>Patient's values</th>
<th>Normal values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Haemoglobin (g/l)</td>
<td>114</td>
<td>135–175</td>
</tr>
<tr>
<td>White blood cell count (×10³/l)</td>
<td>14000</td>
<td>3.4–10</td>
</tr>
<tr>
<td>Platelet count (×10³/l)</td>
<td>130</td>
<td>0.15–420</td>
</tr>
<tr>
<td>Complement C3 (mg/l)</td>
<td>400</td>
<td>880–2030</td>
</tr>
<tr>
<td>Complement C4 (mg/l)</td>
<td>&lt;100</td>
<td>160–470</td>
</tr>
<tr>
<td>Serum creatinine (µmol/l)</td>
<td>88</td>
<td>70–110</td>
</tr>
<tr>
<td>Alkaline phosphatase (µ/l)</td>
<td>99</td>
<td>35–105</td>
</tr>
<tr>
<td>Aspartate aminotransferase (U/l)</td>
<td>40</td>
<td>11–32</td>
</tr>
<tr>
<td>Alanine aminotransferase (U/l)</td>
<td>39</td>
<td>5–30</td>
</tr>
<tr>
<td>Lactate dehydrogenase (U/l)</td>
<td>176</td>
<td>110–205</td>
</tr>
<tr>
<td>Total bilirubin (µmol/l)</td>
<td>38</td>
<td>4–20</td>
</tr>
<tr>
<td>Albumin (g/l)</td>
<td>13</td>
<td>36–52</td>
</tr>
<tr>
<td>Urine analysis (RBC/HPF)</td>
<td>20</td>
<td>0–3</td>
</tr>
<tr>
<td>ANA titre</td>
<td>1/40</td>
<td>&lt;1/40</td>
</tr>
</tbody>
</table>

RBC/HPF, red blood cells/high power field; ANA, antinuclear antibody.

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.

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) 50 IE/ml. Tests for antinuclear antibodies and HLA-B27 were negative. Treatment was started with intensive physiotherapy and intramuscular gold, the latter being replaced by sulfasalazine because of proteinuria. In 1990 she was treated for a unilateral uveitis. In 1992 her right eye was replaced. Until 1993 cardiac examination showed no murmurs and aortic regurgitation was normal.

In November 1995 she was admitted because of a six month history of progressive respiratory distress and increasingly frequent attacks of angina pectoris. Her heart rate was 84 beats/min with a blood pressure of 160/0 mm Hg. A grade 3/6 systolic ejection murmur that radiated into the ascending aorta was heard over the cardiac apex as well as a grade 3/6 ejection diastolic de crescendo murmur over the left sternal border. A pericardial friction rub was not present. Examination of the carotid arteries disclosed a murmur and palpable thrill on both sides. An electrocardiogram showed left ventricular hypertrophy and the chest radiograph slight cardiomegaly. An echocardiogram demonstrated left ventricular dilatation (65 mm; normally <55 mm) and an abnormally thickened aortic valve. Colour Doppler echocardiography showed severe aortic regurgitation, a pressure gradient over the aortic valve (maximum pressure gradient 38 mm Hg, mean gradient 24 mm Hg), and diastolic back flow in the abdominal aorta. The diagnosis aortic valve insufficiency and secondary angina pectoris was made.

She underwent surgical replacement of her aortic valve with a Medtronic Hall prosthetic valve No. 21. The postoperative course was uneventful. Pathological evaluation of the excised strongly thickened and fibrotic tri-leaflet aortic valve was performed.

Microscopic findings in one of the rheumatoid leaflets showed granulation tissue with lymphoplasmocellular infiltration and some polymorphonuclear cells around two areas of fibrinoid necrosis surrounded by a palisade of histiocytes (figs 1 and 2). These findings are similar to the description of a developed typical rheumatoid nodule.

At follow up after four years the aortic valve prosthesis still functions well and the patient has no cardiac signs and symptoms.

To our knowledge, this case is the first illustrated report of typical rheumatoid nodules found in an aortic valve removed owing to aortic valve insufficiency in a patient with polyarticular JIA. Our patient never had any nodules on other locations. Valvular disease is rare in patients with JIA and consists of valvulitis with a substrate with non-specific...
We are grateful to Dr J van der Meulen, cardiotho-
diatic function may deteriorate and cardiac
because if there is valve insufficiency the car-
graphic assessment should be considered,
are detected in these patients, echocardio-
patient with JIA. Whenever cardiac murmurs
part of the routine assessment of every
JIA.

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, echocardio-
graphic assessment should be considered,
because if there is valve insufficiency the car-
diac function may deteriorate and cardiac
surgery may be needed.

Acknowledgments

We are grateful to Dr J van der Meulen, cardiotho-
ractic surgeon, for the surgical description and to Dr
AC van der Wal, pathologist, for his pathology spec-
imen evaluation. We thank Dr FM Westerweel, rheu-
matologist, for allowing us to report on her patient.

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Polymyalgia rheumatica and
pericardial tamponade

Polymyalgia rheumatica causes symmetrical
stiffness in the neck, shoulder, and pelvic gird-
les, 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 fea-
tures.

The association of polymyalgia rheumatica and
pericardial effusion has already been described in two cases.7 8

A 73 year old woman was admitted for the
evaluation of pericardial effusion and mild
anemia. Polymyalgia rheumatica was sus-
ppected because the patient had had asthma,
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 pericar-
dial effusion and initial findings of cardiac
tamponade (right atrial and right ventricular
diastolic collapse), so a pericardiocentesis was
done: polymere chain reaction tests in the
pericardial fluid for Mycobacterium tuberculosis
and cultures for aerobes and anaerobes were
negative; tumoral cells were absent. Serologi-
cal tests for antibodies to cytomegalovirus,
herpes simplex and Epstein-Barr viruses,
anti-smooth muscle, antinuclear, anti-DNA,
and anti-extractable nuclear antigen antibod-
ies were negative. The erythrocyte sedimenta-
tion rate and C reactive protein were normal.
The change in C reactive protein was very small
and later disappeared. The erythrocyte
sedimentation rate (ESR) was 130
mg/mm1st h and C reactive protein (CRP) was 85
mg/l.

The patient was first treated with indomet-
acin (50 mg twice a day) for a week, with no
improvement, and then with low doses of
prednisone (10 mg/day): the symptoms mark-
edly improved and the ESR and CRP dropped
to 27 mm/1st h and 12 mg/l, respectively, in
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 accompa-
nied by systemic features (fatigue, weight
loss, raised ESR) and the marked improve-
ment 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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Remission of Behçet’s syndrome
with TNFα blocking treatment

Gooosens et al reported on a patient in whom a
remission of Behçet’s syndrome was in-
duced with tumour necrosis factor (TNF)
blocking treatment.9 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 ago with Behçet’s disease associated with
rheumatoid arthritis. He had fever, weight
thrifts 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 improve-
ment; steroid treatment with auranofin was
added. The disease was poorly controlled with
progressive erosions in hands, knees, and feet.
Later, pulse steroids, methotrexate, azathiop-
rine, 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 polyar-
thritis, 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 conven-
tional 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 consider-
ably smaller and later disappeared. The
polyarthritis improved considerably, except
for the left knee, which required total replace-
ment. Infliximab was given with continued
colchicine and azathioprine. Our case, as in
Gooosens’ report, suggests that infliximab
may have a beneficial therapeutic effect in
microsceral and cutaneous lesions as well as

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Figure 2. Detail of an area of fibrinoid
necrosis surrounded by a palisade of histo-
cytes; infiltration predominantly with
lymphocytes and plasma cells (haematoxylin
and eosin). Bar represents 25 µm.
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 34 non-medicated healthy controls. Diagnoses were based on the revised European criteria for primary SS. Patients with other chronic diseases were excluded from the study. The Dutch Fatigue Scale (DUFs) was used to quantify fatigue. This validated questionnaire poses nine questions about different aspects of fatigue (table 1). Because depression is frequently observed in SS, a standardised psychiatric questionnaire (SCL-90) was used to rule out this potential confounding variable for fatigue. Immunological activity was evaluated by assessing rheumatoid factor, antinuclear antibodies, presence of anti-SS-A and anti-SS-B, levels of immunoglobulins (IgG, IgM, and IgA), haemoglobin levels, leucocytes, thrombocytes, erythrocyte sedimentation rate, and C reactive protein (CRP). After preliminary analysis using correlation tests, the best model to explain fatigue was calculated by using multiple regression with forward selection (SPSS version 8.0). Independent Student t tests were used to compare the studied groups.

Fatigue was equally raised in patients with both primary and secondary SS, and differed significantly from that of healthy controls. Twenty one (58%) patients with primary SS scored “high” or “very high” out of the six categories for depression according to the SCL-90 criteria. These depression scores did not significantly differ from the scores in secondary SS patients. Further analysis showed that 79% of the fatigue in patients with primary SS could be explained by depression, total level of immunoglobulins, and thrombocyte counts (p<0.001). Both depression and thrombocyte counts showed a significant positive correlation, whereas levels of immunoglobulins showed a negative correlation.

Though tempting 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. Thrombocyte counts showed a significant positive correlation. Although increases in thrombocytes follow the acute phase reaction, no significant correlation between thrombocyte counts and CRP levels were found. A chance association between fatigue and thrombocyte 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 un unravelled. 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.

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 cytokine 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 Mumcu, one of the
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 effects 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 vasculitic episodes and in polymyalgia rheumatica and temporal arthritis. It is clearly written, because it questions apparently known facts, especially taking the example of RA. The important very 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.ser.man.ac.uk

British Society for Rheumatology XIXth AGM
23–26 Apr 2002; Brighton, UK

Contact: BSR, 41 Eagle Street, London WC1R 4TL, UK
Website: www.rbm.org.uk

4th EULAR Sonographie Course
25–28 April 2002; Madrid, Spain

The course is entitled “Practical use of musculoskeletal ultrasonography”
Contact: Esperanza Naredo
Email: enaredo@eresmas.com
Website: www.eular.org/courses and www.sameint.it/eular

10th International Vasculitis and ANCA Workshop
25–28 Apr 2002; Cleveland, Ohio, USA

Contact: Debora 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@ioflyon.org
Website: www.ioflyon.org

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

Annual European Congress of Rheumatology
12–15 June 2002; Stockholm, Sweden
Contact: Fred Wyss, Executive Secretary EULAR, Wirikonerstrasse 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
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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 48456908
Email: zoubbere@zedat.fu-berlin.de
Website: www.userspages.fu-berlin.de/~zoubbere
15B3 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: 29cr2002@rit.no or revhan@rit.no

Translational Research in Autoimmunity
21–22 Sep 2002; Pavia, Italy
Contact: Organising secretariat: eventi S.R.L., Corso Cavour, 1820 – 27100 Pavia, Italy
Email: tra@e20pr.com
Website: www.e20pr.com
Congress website: www.medicine.ucsd.edu/albani/2001meeting

Osteoarthritis Research Society International (OARSI) World Congress
22–25 Sep 2002; Sydney, Australia
Contact: Osteoarthritis Research Society International (OARSI), 2025 M Street, NW, Suite 800, Washington DC 20036, USA
Tel: 202 367 1177

www.annrheumdis.com
10th International Congress on Antiphospholipid Antibodies
29 Sep–3 Oct 2002; Sicily, Italy
Deadline for abstracts 1 April 2002
Contact: Secretariat, 10th International Congress on Antiphospholipid Antibodies, c/o Kenes International, PO Box 90006, 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 Spondyloarthropathies
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• Genetics and HLA-B27
• Animal models and pathogenesis
• Clinical research and therapy
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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.cayne.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

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Fax: 919 918 7114 or 919 929 9255
Website: www.abp.org

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18–21 June 2003; EULAR 2003 Lisbon, Portugal
9–12 June 2004; EULAR 2004 Berlin, Germany
8–11 June 2005; EULAR 2005 Vienna, Austria
21–24 June 2006; EULAR 2006 Amsterdam, The Netherlands
Sonography as a replacement for sialography for the diagnosis of salivary glands affected by Sjögren’s syndrome
K Yonetsu, Y Takagi, M Sumi, T Nakamura and K Eguchi

Ann Rheum Dis 2002 61: 276-277
doi: 10.1136/ard.61.3.276

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