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 sonography 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.

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

<table>
<thead>
<tr>
<th></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>
<tr>
<td>Univariate analysis</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Coefficient</td>
<td>6.02</td>
<td>3.69</td>
<td>1.67</td>
<td>0.56</td>
<td>1.92</td>
<td>1.66</td>
</tr>
<tr>
<td>SE</td>
<td>0.75</td>
<td>0.39</td>
<td>0.29</td>
<td>0.32</td>
<td>0.36</td>
<td>0.51</td>
</tr>
<tr>
<td>p Value</td>
<td>&lt;0.00001</td>
<td>&lt;0.00001</td>
<td>0.00006</td>
<td>0.00787</td>
<td>&lt;0.00001</td>
<td>0.000012</td>
</tr>
<tr>
<td>Multivariate analysis</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Coefficient</td>
<td>4.87</td>
<td>3.97</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>SE</td>
<td>1.06</td>
<td>1.07</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>p Value</td>
<td>&lt;0.00001</td>
<td>0.00002</td>
<td></td>
<td></td>
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<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 knee joints. For the psoriatic nails he took acitretine (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 lesions, it can be considered that sulfasalazine played a beneficial part in the pathologic condition of our patient. Dermatological assessments 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. 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. 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 psoriasis6; 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.

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 dermatologist. 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, predicting the diagnosis of psoriasis partime.

Synovial fluid from the right knee joint contained 17.8 x 10^6 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.

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

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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), giving the following reasons: better monitoring, less trouble (IV immunoglobulin collected at the hospital pharmacy, calling the nurse at home).

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

Table 1

<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</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>$63691</td>
</tr>
</tbody>
</table>

Cost for one treatment in hospital: $2701

Cost for one treatment at home: $2471

*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.

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

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

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

Table 2 Home IV immunoglobulin infusion guidelines for patients with autoimmune disease
A 32 year old man with a one month history of right shoulder pain. The pain was described as a dull ache of gradual onset, around the posterior aspect of the arm. The patient had no other medical history or relevant family history.

Of note was a 5 cm mass palpable inferolateral to the relative apex of the right scapula, on the posterior aspect of the arm. On inspection, this mass was firm, poorly circumscribed, and minimally mobile. Plain radiographs were 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 poorly defined margins. MRI is the best non-invasive technique for demonstration of this tumour. Elastofibromas appear as poorly circumscribed soft tissue lesions with similar signal intensity to that of skeletal muscle but interspersed with fibrous tissue. Typically, 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.

The patient had noticed a swelling below the inferior angle of the right scapula which was apparent under pressure. Over the preceding four months the patient had noticed a swelling below the inferior angle of the right scapula which appeared and disappeared with movement of the arm. The patient had no other medical history or relevant family history.

On examination there was 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 was circumducted. In this position a firm, poorly circumscribed, and minimally mobile mass of 5x7 cm was apparent underlying the inferior angle of the scapula. The rest of the examination was normal.

Initial investigations showed a normal full blood count, bone profile, and inflammatory markers, and a normal radiograph of the right shoulder and scapula. Subsequent magnetic resonance imaging (MRI) showed a poorly circumscribed heterogeneous soft tissue mass between the chest wall and the scapula (fig 1). The signal intensity was similar to that of adjacent muscles with interspersed strands of high signals similar to those of fat. No significant contrast enhancement was seen. The lesion was biopsied under computed tomography guidance and a histological examination showed elastic fibres within a collagenous fibrous tissue with entrapped adipose tissue, consistent with a diagnosis of elastofibroma. Surgical excision was performed because the mass was causing pain. Postoperative histology confirmed an elastofibroma. The patient has remained asymptomatic after surgery with no recurrence of the mass. Elastofibromas, first described in 1961, is a benign, slow growing, mesenchymal soft tissue lesion. They usually occur in active subjects above the age of 50 with a male:female ratio of 1:5. Most (99%) occur in the subcapsular region, usually on the right side. The lesions have occasionally been found in the extremities, head, abdominal and thoracic cavities. Of those in the subcapsular region approximately 10% are bilateral. The cause and pathogenesis are unclear, but it is suspected that subclinical microtrauma may lead to reactive hyperplasia of elastic fibres with consequently increased production of fibrous tissue. 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 poorly defined margins. MRI is the best non-invasive technique and most useful for diagnosis. Elastofibromas appear as poorly circumscribed soft tissue lesions with similar signal intensity to that of skeletal muscle but interspersed with high signal intensity areas representing adipose strands. The differential diagnosis includes desmoid tumours, neurinoma, 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.

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 Sphingobacterium 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 × 10⁵ 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 (fluid aspiration and walking) 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; so a further aspiration was carried out and local injection with triamcinolone acetate was given. However 24 days later the pain worsened and swelling of the elbow recurred; so a further aspiration was carried out and local injection with triamcinolone acetate was given. 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Prevalence of allergic respiratory diseases in patients with RA

The balance between Th1 and Th2 cell activity is crucial in both cutaneous and 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 of a decreased level 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, 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 (mean age 53.8 ± 14.9 years, 76.8% female) (7). The study 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 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 disappeared by the time of this study. In 20/21 patients atopic symptoms were associated with an ileus. In 14/21 patients atopic symptoms disappeared by the time of this study. In 20/21 patients atopic symptoms had totally disappeared by the time of this study.

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

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 nephropathy. The diagnostic criteria include 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 liver cirrhosis. He was admitted with a two week history of dyspnoea, malaise, cough, fever, 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 convalescent serological titre of anti-hepatitis C virus of the patient and his abdomen showed portal hypertension, a small cirrhotic liver, small spleen, omental and peri-splenic varices, an atrophic pancreas, and modest ascites. The purpuric lesions and ileus improved. However, on day 4 he became tachycardic and developed a tender abdomen. 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 sepseicaemia 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 our patient was 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 anti-cryoglobulinemia 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 defective liver metabolism of IgA circulat- ing immune complexes (ICs), resulting in tissue deposition, although this is known to occur without overt vasculitis.

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

There have been no laboratory data in adults with complicated HSP. Corticosteroids used in a series of children have been shown to relieve symptoms, but fail to deal prospectively with the prevention of abdominal complications. Adults respond favourably to corticosteroids and may be managed with short courses of treatment, but cortico- steroids may also mask severe abdominal catastrophe.

Several important points can be learnt from this case report:

- 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.
- Liver cirrhosis secondary to hepatitis C may precipitate development of HSP or mixed cryoglobulinaemic vasculitis through the defective metabolism of ICs.
- Given the increasing incidence of hepatitis C related liver disease worldwide, the association of these diseases and their 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.
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 (&lt;10³/l)</td>
<td>14000</td>
<td>3.4–10</td>
</tr>
<tr>
<td>Platelet count (&lt;10⁹/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 (U/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>15</td>
<td>30–52</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.

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 chest roentgenogram 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 diastolic decrescendo 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.1

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 JIA2 and consists of valvulitis with a substrate with non-specific inflammatory infiltrate (c) (haematoxylin and eosin). Bar represents 400 µm.

Figure 1: Section from aortic valve cusp showing a central area of fibrinoid necrosis (a), a palisade of radially arranged histiocytes (b), and a lymphoplasmocytic infiltrate (c) (haematoxylin and eosin). Bar represents 400 µm.

References

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 arterial 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. van der Meulen, cardiothoracic surgeon, for the surgical description and to Dr. van der Wal, pathologist, for his pathology specimen examination. 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 inflammatory disorders such as polymyalgia rheumatica, 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 anaemia. Polymyalgia rheumatica was suspected because the patient had had asthena, 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 pericardiocentesis was done: polymerase chain reaction tests in the pericardial fluid for Mycobacterium tuberculosis and cultures for aerobes and anaerobes were negative; tumoral cells were absent. Serological tests for antibodies to cytomegalovirus, herpes simplex and Epstein-Barr viruses, anti-smooth muscle, antimuclear, anti-DNA, and anti-extractable nuclear antigen antibodies were negative. The polymyalgia rheumatica was treated with colchicine and azathioprine. The patient was treated with sulphasalazine and colchicine without improvement. A diagnosis of Behçet’s disease associated with erosive, seropositive RA was suggested. The patient was treated with sulfasalazine and colchicine without improvement; steroid treatment with auranofin was added. The disease was poorly controlled, with 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 try 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 replacement. Infliximab was given with continued colchicine and azathioprine. Our case, as in Goossens’ report, suggests that infliximab may have a beneficial therapeutic effect in microsural 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.1 Unfortunately, there is no proper treatment available to combat the fatigue in SS. Beside a variety of somatic and non-somatic conditions,2 increased immune activity has been implicated as a cause of fatigue in autoimmune diseases.3 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 the classification of SS.4 Control groups were matched for age and sex. Disease duration or treatment did not differ significantly between patients with primary and secondary SS. Patients with other chronic diseases were excluded from the study. The Dutch Fatigue Scale (DFS) 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,5 a standardised psychiatric questionnaire (SCL-90) was used to rule out this potential confounding variable for fatigue.6 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 thromboocyte counts (p<0.001). Both depression and thromboocyte 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. Thromboocyte counts showed a significant positive correlation. Although increases in thrombocytes 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 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.7 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

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 Munck, 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 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 vasculitis epitomised by those in systemic lupus erythematosus, and in polyarthritis rheumatica and temporal arteritis. It is clearly written, because it questions apparently known facts, amusingly and expressively on the past decades. 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. His 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.

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Email: hmoussop@med.uoa.gr
Email: congress@amphihtion.gr

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Website: www.userspages.fu-berlin.de/~zoubbere
15SBD 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
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Contact: Organising secretariat: eventi S.R.L., Corso Cavour, 18/20 – 27100 Pavia, Italy
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