A successful renal transplantation in Behçet's syndrome

Renal involvement is not frequent in Behçet's syndrome (BS) and consists of occasional reports of patients suffering from glomerulonephritis, IgA nephropathy and renal amyloidosis. We present the successful outcome of a renal transplantation in a patient who had end stage renal failure secondary to glomerulonephritis. To our knowledge, this is the first patient with BS to receive an organ transplantation.

The detailed history of this patient at the time of the diagnosis of glomerulonephritis was the subject of a case report in 1991. In brief, she was 21 years old when she developed recurrent oral and genital ulcers, bilateral uveitis, erythema nodosum, folliculitis, and intermittent arthritis of the knees. Two years later, she was referred to our centre for further evaluation of eye symptoms. She had no active mucocutaneous lesions at that time, the pathergy reaction was positive and she had positive HLA B7. It was decided to prescribe only local drops for her mild eye involvement. Three months later she experienced two ocular episodes resulting in a sharp decline of visual acuity and azathioprine 2.5 mg/kg/day was prescribed. Two weeks later she was admitted to the hospital because of microscopic haematuria. She was ANA negative, the anti-DNA and serum complement levels were normal range. Her glomerular filtration rate was 67 ml/min. An open renal biopsy showed diffuse proliferative glomerulonephritis and weak focal segmental positivity of IgA and IgM. She was treated with three boluses of 1 g methylprednisolone and was discharged prescribed azathioprine 150 mg/day, aspirin 300 mg/day and prednisone 30 mg/day. She was well except for occasional mucocutaneous symptoms and a mild decline in renal function during the next four years. However her renal function deteriorated progressively despite uninterrupted treatment with azathioprine and changing doses of prednisone and she was put on regular haemodialysis twice a week. In the 14th month of haemodialysis, she received a kidney from her mother. The graft function started immediately and she was prescribed maintenance immunosuppression with azathioprine, cyclosporin A and methylprednisolone. An acute interstitial type rejection on the 11th day of transplantation was treated successfully with pulsed corticosteroids. Now 40 months after transplantation, she has normal renal function and is free of any symptoms of BS except for occasional oral ulcers.

We had some hesitation in performing a renal transplantation on our patient initially because of the lack of any previous experience and particularly because of our concern for the heightened inflammatory response of BS patients to simple penetrating trauma that is best characterised by the pathergy reaction. This reaction, however, is not only limited to the skin and development of aneurysms after vascular punctures and episodes of synovitis after arthrocentesis have been observed. Furthermore, postoperative complications leading to a poor outcome such as occlusions of grafts/anastomoses after the surgical treatment of aneurysms or perivalvular leakage and suture breakdown after aortic valve replacement have been reported in BS patients. As these complications are probably related to the pathergy phenomenon reaction, you would also reasonably expect problems after an organ transplantation, an operation with arterial and venous anastomoses. On the other hand, we had previously shown that despite the increased inflammation, wound healing after full thickness skin punch biopsies is not changed in BS. We have not experienced any of the feared complications after the transplantation procedure in this instance. One reason for this favourable outcome might be that our patient was female. It is known that BS runs a milder disease course in women compared with men. Additionally, the rather intensive immunosuppressive/anti-inflammatory post-transplant drug use might also have contributed to the diminished disease activity of our patient as well as to the prevention of a reaction at the site of transplantation. Whatever it might be related to, the outcome in our patient suggests that BS patients can undergo renal transplantation with a satisfactory outcome.

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LYMPHOCYTE PHENOTYPES IN SYSTEMIC SCLEROsis

Although the pathophysiology of systemic sclerosis (SSc) is not fully clarified, there are considerable data implicating abnormalities of microvascular changes, fibroblast activation and immune system abnormalities. Immune system activation may play a part as a stimulus in both fibrotic and vascular damage. To investigate the immune system abnormalities in the pathogenesis of SSc we evaluated lymphocyte phenotypes in patients with SSc and healthy controls by flow cytometry (Epics Profile II) for total T (CD3), T helper (CD4), T suppressor (CD8), B lymphocyte cell surface marker (CD19), activation marker (CD25) and natural NK cell surface marker NKH-1 (CD56).

We studied 29 patients (27 women, two men) 16 limited, 12 diffuse and one overlap who fulfilled preliminary criteria for classification of SSc. Anti-nuclear antibody was positive in 25 (86.2%) and anti-Scl70 antibodies was positive in seven (24.1%) patients. The age range of the patients was 20–63 years (mean (SEM) 40 (5)) and the mean (SEM) disease duration was 5.5 (5.5) years. Patients were receiving no medication nor had received any immunosuppressive agent for at least three months. Controls were 12 aged and sex matched healthy volunteers with an age range from 27–51 years.

Data were compared for significance for Student’s unpaired t test. Table 1 summarises lymphocyte phenotypes in patients with SSc and healthy controls.

We found a higher expression of T cell activation marker CD25+ and NK cell main surface marker CD56+. In lymphocyte phenotypes there was not any difference among disease subsets and CD25+ and CD56+ were not correlated with the disease duration.

Immune system abnormalities have been suspected in the development of SSc because of the presence of autoantibodies, increased cytokine production and evidence of overlap with other autoimmune diseases. It was suggested that immune system changes play the major part in the development of vasculopathy and fibrosis. Previous reports on T lymphocyte subpopulations in SSc are partially conflicting. Melendro et al demonstrated that there was no significant difference in the levels of CD3+, CD4+, CD8+ and CD19+ in 22 SSc patients and control group but in rheumatoid arthritis (RA) CD3+ and CD8+, in Sjögren’s syndrome CD3+, CD4+ and CD8+ levels were significantly decreased compared with those of controls and they suggested that the abnormalities in immune regulatory T cell circuits leading to autoimmunity are different in each connective tissue disease.

Table 1 Lymphocyte phenotypes in patients with SSc and healthy controls

<table>
<thead>
<tr>
<th>Serum</th>
<th>CD3 (%)</th>
<th>CD4 (%)</th>
<th>CD8 (%)</th>
<th>CD25 (%)</th>
<th>CD56 (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>SSc</td>
<td>71 (1)</td>
<td>44 (1)</td>
<td>31 (1)</td>
<td>31 (9)</td>
<td>22 (2)</td>
</tr>
<tr>
<td>Control</td>
<td>69 (9)</td>
<td>45 (9)</td>
<td>25 (6)</td>
<td>19 (14)</td>
<td>14 (5)</td>
</tr>
<tr>
<td>t value</td>
<td>0.660 &gt;0.05</td>
<td>0.110 &gt;0.05</td>
<td>1.914 &gt;0.05</td>
<td>1.339 &gt;0.05</td>
<td>2.691 &gt;0.05</td>
</tr>
</tbody>
</table>

*Unpaired Student’s t test. Data shown as mean (SD).
Lymphocyte populations and cytokine concentrations in pericardial fluid from a systemic lupus erythematosus patient with cardiac tamponade

Pericardial involvement is the most common cardiovascular complication in systemic lupus erythematosus (SLE). The clinical picture varies from subclinical pericardial effusion and classic acute pericarditis to cardiac tamponade. In immunological studies of pericardial fluid (PF) have been limited to determination of autoantibodies, complements and immune complexes. To further study the pathogenetic mechanisms involved in lupus pericarditis we examined the lymphocytic populations and cytokine concentration pattern in PF and peripheral blood (PB) from a SLE patient with cardiac tamponade.

We report a case of a 38 year old man with SLE diagnosed in December 1995 when he presented with polyarthralgia, photosensitivity, oral ulcers, nephritis, non-hemolytic anemia, positive ANA, increase of anti-dsDNA antibodies, positive anti-SSA, anti-SSB, proteinuria, and hypocomplementaemia. The patient improved with corticosteroid and intravenous cyclophosphamide treatment. However, on 18 June 1997 he presented with syncope, hypotension (80/40 mm Hg), a tachycardia, jugular vein distension and cardiomegaly. Almost daily over a three month period. After a 22 month follow up, he remained clinically stable without recurrence of pericardial involvement or SLE exacerbations.

Before starting immunosuppressive treatment, PF and PB were obtained simultaneously for immunological analysis. Mononuclear cells from both sources were isolated by gradient centrifugation and the frequency of lymphocyte populations expressing natural killer cell phenotypes in patients with progressive systemic sclerosis. Arthritis Rheum 1983;26:841–7.

The decreased pericardial lymphocyte count and fluid characteristics observed here are in agreement with other studies. The higher frequency of CD4+ T cells and NK cells in PF could be associated with the observed cytokine concentration pattern. For example, CD4+ memory T cells from SLE patients highly secrete IL10 compared with PB. IL6 concentration was much higher in PF than in PB. IL2 concentration was reduced in PF compared with PB.

The decreased pericardial lymphocyte count and fluid characteristics observed here are in agreement with other studies. The higher frequency of CD4+ T cells and NK cells in PB could be associated with the observed cytokine concentration pattern. For example, CD4+ memory T cells from SLE patients highly secrete IL10 compared with PB. Further studies would be required to confirm the results presented here. In addition, immunocytochemical studies of pericardial fluid of patients with systemic sclerosis are in agreement with these results. Immunocytochemical studies of pericardial fluid of patients with systemic sclerosis are in agreement with these results.

Table 1 Frequency of lymphocyte populations and cytokine concentrations in peripheral blood and pericardial fluid

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Peripheral Blood</th>
<th>Pericardial Fluid</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lymphocyte population (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CD4+ T cells</td>
<td>57.8</td>
<td>50.0</td>
</tr>
<tr>
<td>CD4+ T cells</td>
<td>17.6</td>
<td>25.0</td>
</tr>
<tr>
<td>CD8+ T cells</td>
<td>34.3</td>
<td>25.0</td>
</tr>
<tr>
<td>B cells</td>
<td>7.8</td>
<td>8.3</td>
</tr>
<tr>
<td>NK cells</td>
<td>34.3</td>
<td>41.7</td>
</tr>
</tbody>
</table>

Cytokine concentration* (pg/ml)

\[ \text{IL2} 201.8 <4.0 \]
\[ \text{IL4} <6.0 <6.0 \]
\[ \text{IL6} <6.0 <6.0 \]
\[ \text{TNF} \alpha 13.0 13.98 \]
\[ \text{IFN} \gamma 1.5 32.8 \]

*Manufacturer (Genzyme, Boston, MA) detection limits: 3 pg/ml for IL1, INF, and TNF, and 4 pg/ml for IL2. 6 pg/ml for IL3, and 5 pg/ml for IL10.

tissue are necessary as the composition of lymphocyte and cytokine profiles may differ between pericardial fluid and tissue.

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11 Al-Janadi M, al-Dalaan A, Harigai M, Suzuki Y, et al. Antinuclear antibodies in patients with AS (HLA-B27+ AU with SA), and 29 (19.9%) did not have HLA-B27+ AU. No significant differences were found in clinical features of AU between the three study groups. Erythrocyte sedimentation rate, C reactive protein and IgA were found to be more increased in patients than in control.

Table 1 Lymphocyte populations in AU patients and controls

<table>
<thead>
<tr>
<th>AU patients (n=146)</th>
<th>Controls (n=31)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lymphocytes (no/mm³)</td>
<td>CD3 (no/mm³)</td>
<td>CD4 (no/mm³)</td>
</tr>
<tr>
<td>2425.60 (964.44)</td>
<td>1734.20 (726.67)</td>
<td>1023.91 (489.16)</td>
</tr>
<tr>
<td>(964.44)</td>
<td>(726.67)</td>
<td>(489.16)</td>
</tr>
<tr>
<td>2567.74 (820.72)</td>
<td>1835.64 (586.68)</td>
<td>71.27 (4.28)</td>
</tr>
<tr>
<td>(820.72)</td>
<td>(586.68)</td>
<td>(4.28)</td>
</tr>
<tr>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
</tbody>
</table>

AU = anterior uveitis, SA= spondyloarthropathy, IAU= idiopathic anterior uveitis; AU= anterior uveitis; SA= spondyloarthritis.

Figure 1 Absolute values of CD4CD45R+ cells. Patients with IAU had absolute values lower than the control group, and percentages less than those of SA patients (p<0.001). IAU= idiopathic anterior uveitis; AU= anterior uveitis; SA= spondyloarthritis.

Figure 2 Percentages of CD4CD45R− cells. Patients with IAU had higher percentages than the healthy subjects and SA patients (p<0.001). Abbreviations as in figure 1.
We are indebted to Ms E Velasco for assistance in the preparation of the manuscript.

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MATTERS ARISING

RS3PE: six years later

We read with interest the paper by Cantini et al and would like to comment on it.

In 1992 we performed a retrospective multicentre study of 27 patients. We concluded that personal history of polymyalgia rheumatica (two patients), presence of erosions (one patient) and evolution to haematological diseases (two patients concomitantly developed a T lymphoma and one a myelodysplastic syndrome) suggested that RS3PE syndrome might not be a distinct clinical entity. At that moment 12 patients were asymptomatic and 12 required treatment. This was reported elsewhere.

Now, six years later, we have reviewed the original cohort of patients with the RS3PE syndrome. A questionnaire was sent to the participating rheumatologists. The survey focused on articular symptoms, treatment and evolution. The current cohort was composed of 22 patients (16 male; 6 female; mean age:77.9; range 64–91). Four patients died (the three with haematological diseases, one stroke) and one was not located. Thirteen patients were asymptomatic and without treatment, in contrast nine required treatment, namely corticosteroids (6), gold salts (1), clo- roquine (1) and NSAID (1). Interestingly, two of the patients were identified by their rheumatologist as having a seronegative rheumatoid arthritis, another patient had a chronic disease with separate corticosteroid responsive episodes of bilateral hand oedema and polyynalgic syndromes at different times. Last but not least one patient developed Ray- naud’s phenomena, both hands had sclero- dactyly. A nifluid capillary microscopy showed a decreased number of capillary loops, which were widened, suggesting systemic sclerosis.

Our results suggest that RS3PE syndrome has a good prognosis as more than half of the patients are asymptomatic and without treat- ment six years later. However, there is a sub- set of patients that have other diseases. Although pure RS3PE syndrome does exist the evolution should be carefully monitored.

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Authors’ reply

We appreciate the comment by Olivi et al on our article on RS3PE. They reviewed 27 previ- ously described RS3PE patients after a fol- low up of six years.

As we suggested in a previous report, they confirm that RS3PE syndrome should be considered a heterogeneous condition associ- ated with different inflammatory rheumatic diseases and also with neoplastic disorders.

In our study none of the 23 patients with RS3PE syndrome developed clinical find- ings supporting the diagnosis for another disease. The different study design and selec- tion of patients may in part explain the subset of patients with other diseases and with a worse prognosis observed by Olivi et al.

We designed a prospective follow up study excluding patients satisfying the criteria for the diagnosis of polymyalgia rheumatica, rheumatoid arthritis and seronegative spondylarthropathies. Moreover, patients with a clinical history of cancer were excluded from the study. In their original report these authors performed a retrospec- tive study including all patients with remit- ting distal extremity swelling with pitting oedema. They recruited also patients not evaluated for spondylarthropathies, which may be associated with distal extremity swell- ing with pitting oedema.

However, in their retrospective evaluation Olivi et al found that 13 of 22 (59%) patients were asymptomatic and drug free over a six year follow up period, confirming that RS3PE is not associated with other conditions and with a good prognosis does exist.

The problem is how to best label this clinical picture. As discussed in our article, the simi- larities of demographic, clinical and MRI findings between patients with “pure” RS3PE syndrome and those with polymyalgia rheumatica and the concurrence of the two syndromes suggest that these conditions may be part of the clinical spectrum of the same disease. In the series of Olivi et al the patient with a clinical course characterised by alternating relapses of HLA-B27 and pitting oedema or polymyalgic symptoms further supports our hypothesis. Even those RS3PE patients successively diagnosed as having seronegative rheumatoid arthritis (elderly onset seronegative arthritis) do not conflict with our conclusions. Healey described pa- tients who developed episodes of polymyalgia rheumatica and seronegative rheumatoid arthritis at different times during follow up. Similar clinical characteristics have been recently described in a population based cohort of patients with giant cell arteritis fol- lowed up over a 42 year period. Four of the six patients who fulfilled the criteria for the diagnosis of rheumatoid arthritis during the follow up experienced multiple separate episodes of symmetrical arthritis, proximal symptoms of polymyalgia rheumatica and distal extremity swelling with pitting oedema.

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5 Healey LA. Polymyalgia rheumatica and seronegative rheumatoid arthritis may be the same entity. J Rheumatol 1999;22:89–903.


Cryataly in arthritis: new age nonsense or novel therapeutic target?

Apatite crystals are present in up to 70% of fluid from degenerated joints. Their presence correlates strongly with radiographic evidence of cartilage degeneration and is associated with larger joint effusions than compared with joints without crystals. Whether the presence of apatite crystals is a cause of cartilage damage or an effect of cartilage damage is unclear. Several lines of evidence suggest that apatite crystals cause joint destruction. For example, apatite crystals induce both mitogenic and proinflammatory synovial fibroblasts and chondrocytes in vitro. They also induce matrix metalloprotease (MMP) synthesis and secretion, thus promoting tissue destruction. The cellular mechanisms whereby apatite crystals induce such responses are currently under investigation. Like many other growth promoting agents, apatite crystals induce a variety of transcription factors such as nuclear factor-κB (NF-κB) and activator protein 1 (AP-1). They also induce mitogen activated protein kinases (MAPK) and protein kinase C (PKC). Furthermore, such activation is specific as the crystals do not activate protein tyrosine kinases (PTK) or phosphatidylinositol 3-kinases (PI3K). If the crystals were present simply as a consequence of joint destruction, we would expect them to be present in other arthropathies characterised by cartilage degradation and synovial lining proliferation such as rheumatoid arthritis (RA). However, apatite crystals are rarely found in RA joint fluids. Thus, current data support the view that apatite crystals cause joint destruction.

On the other hand, the clinical significance of apatite crystals in joint degeneration continues to be questioned. Dieppe and Swan doubt that apatite crystals are of pathogenic importance, but they fail to refute the hypothesis that it is a sign not of apatite crystals but of excessive joint destruction. If so, why should we expect to see apatite crystals in joints with joint destruction? It is also unclear whether apatite crystals are causally related to joint destruction or are a result of joint destruction. For example, apatite crystals may be present simply as a consequence of joint destruction, but are not related to increased concentrations of keratan sulfate or interleukin 1b. J Rheumatol 1999;58:961–6.


3 Carroll GJ, Stuart RA, Armstrong JA, Breidahl PD, Laining BA. Hydroxyapatite crystals are a frequent finding in osteoarthritic synovial fluid, but are not related to increased concentrations of keratan sulfate or interleukin 1b. J Rheumatol 1997;20:1861–6.


Author’s reply

We agree with all the points made by Dr McCarthy. Basic calcium phosphates (BCPs) in synovial fluids may be important, and it may be that their identification will be valuable in relation to future treatments. However, she seems to agree with the only two points made about BCPs in our article (which is about the identification of urate and pyrophosphate crystals): that is, that on the basis of current understanding BCPs are of “doubtful significance”, and that their identification should have no influence on contemplatory therapeutic decisions.

PAUL DIEPPE

MRC Health Services Research Collaboration, University of Bristol, Bristol

Mortality in rheumatoid arthritis patients

The paper “Mortality in rheumatoid arthritis patients with disease onset in the 1980s” of considerable interest.1 A decrease in mortality risk for rheumatoid arthritis (RA) patients in more recent years would be important, even if only in the first 10 years of RA. However, this inception cohort differs from those previously published so that no direct comparison is possible. As (earlier and older and larger) studies have shown standardised mortality ratios of two to three, a finding of “normal” mortality might imply that more recently used treatment strategies are reversing the excessive mortality in RA previously described.2

Yet, even at first perusal, there are a lot of deaths in this series of relatively young people. In the 10 years after a mean age of 51, 18 patients (10%) had died. Over 20 deaths were said to be “expected”. However, using US mortality rates for a population mean aged the same, projected over 10 years, two thirds women, and white, one would expect only 11 deaths using 1996 mortality rates and 12 deaths using 1985 rates, over the 1710 patients years of follow up. While we did not have the age distribution of this RA cohort to calculate precise expectations, these figures should be conservative. Female mortality rates in the US white female popula-

Letters, Matters arising

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tion, at 3.4 per 1000 per year at age 51 and 9.0 per 1000 per year at age 61 are presumably higher than those in long lived Malmöhus County, Sweden.

Of interest, in our own larger study with meticulously computed “expected” values in four different populations we also had an “expected” death rate of about 10% to 15% over 10 years. But, these were not inception cohorts and their age at start of follow up was 60.4, 62.6, 59.8, and 69.1 years. Thus, they were much older cohorts. Given the expected doubling of mortality rates each eight years (Gompertz’s law), expected deaths should have been two to three times more in our cohorts than in a cohort beginning at age 51.

Finally, recent studies have not suggested that “rheumatoid” deaths in themselves are the cause of the increased mortality in RA. The observed “excess” deaths are spread around in multiple disease categories, with accelerated atherosclerosis numerically the largest problem and only a slight relative increase in systemic RA complications, gastrointestinal haemorrhage, and infections.

JAMES F FRIES
Daniel A Bloch
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Authors’ reply

We were pleased to notice the interest in our paper shown by Drs Fries and Bloch. In reply to their comments we do not consider the death rate of 10% in the cohort as an excessive one compared with the age and sex matched general population. It is not possible to calculate more precise figures of expected deaths knowing the mean age of the cohort only. To clarify this and make comparison possible we enclose a table of the age distribution in our cohort in five year intervals giving the number of observed and expected deaths for each age interval separately.

Women do live longer in Malmöhus County, Sweden than in the US. Female mortality rates in Malmöhus County were 3.76 per 1000 at age 51 and 7.32 per 1000 at age 61 in 1985. In 1996 the corresponding figures were 2.03 per 1000 at age 51 and 3.39 per 1000 at age 61.

We agree that the main cause of death in RA patients very seldom is the rheumatoid disease in itself. This was true also for our study where no certain connection between RA and death was found in any of the cases.

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KERSTIN EBERHARDT
Department of Rheumatology, Lund University Hospital, Sweden

<table>
<thead>
<tr>
<th>Age group</th>
<th>Number of patients</th>
<th>Expected mortality</th>
<th>Observed mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>15-19</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>20-24</td>
<td>3</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>25-29</td>
<td>7</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>30-34</td>
<td>3</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>35-39</td>
<td>15</td>
<td>0</td>
<td>0</td>
</tr>
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<td>40-44</td>
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<td>45-49</td>
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