polyarthritis in a 39-yr-old female associated with giardiasis. Arman [6] reports two cases of chronic relapsing polyarthritis in a 30-yr-old female and a 26-yr-old male. Barton et al. [7] report a 29-yr-old HLA B27-positive female with a family history of ankylosing spondylitis who developed an asymmetrical oligoarthritis following giardiasis, but who had no signs of sacroiliitis. All cases responded to antimicrobial therapy.

There are no reports in the literature of axial involvement in the arthropathy associated with giardiasis. Other than as specified the patients were HLA B27 negative or, in the case of the original series, not determined. The patient reported here appears to have developed an axial reactive arthritis with clinical evidence of enthesisitis following giardiasis. Although this is a common presentation of ReA, we believe it is the first report secondary to giardiasis. The clinical picture is complicated by the course of treatment with sulphasalazine; however, after stopping this treatment, there has been no recurrence of the arthropathy and the CRP continued to fall and remains normal. Furthermore, the clinical findings of enthesisitis resolved after metronidazole, whilst appearing to worsen on treatment with sulphasalazine. It is interesting that our patient is HLA B27 negative; however, at present, there is insufficient information available to determine the relationship between HLA status and the risk of developing ReA following giardiasis.

From a clinical viewpoint, we feel that this case highlights the necessity of taking a detailed history. In the specific case of giardiasis, the intestinal infection may be asymptomatic and the diagnosis may be overlooked in a patient presenting with ReA. This is important as antimicrobial chemotherapy appears to be curative. In our patient, the history of travel to Eastern Europe, where giardiasis is relatively common, might have suggested the diagnosis. We conclude that chronic giardiasis needs to be excluded in patients presenting with a spondyloarthritis, abdominal symptoms and a suspicious travel history.

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Accepted 24 October 1997
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Nailfold Capillary Microscopy in Patients with Raynaud’s Phenomenon: Experience in a District General Hospital

Sir—Raynaud’s phenomenon (RP) is a common disorder affecting ~10% of the adult population [1]. The criteria for primary RP are absence of digital pitting or ulcerations, normal ESR, negative ANA and normal nailfold capillary microscopy (NCM) [2]. NCM has been shown to be the best prognostic indicator for connective tissue disease (CTD) [3–5]. Enlarged and deformed capillaries, often surrounded by avascular areas, are associated with systemic sclerosis (SSc) and tortuous capillaries with SLE [6]. This technique is well established as a research investigation, but informal discussion suggests that NCM is rarely performed outside specialist referral centres. We therefore set out to establish whether it was feasible to organize a service at a district general hospital, and to assess its value in the diagnosis and management of patients presenting with RP.

Seventy-five consecutive patients who attended the rheumatology clinics with RP were included in this study. All had a full clinical examination and blood count, ESR, ANA, RF, thyroid function, and X-rays of the chest and hands (RF was regarded as positive at a titre of 1/80 and ANA at 1/40). The patients were divided into three groups prior to NCM. RP only (26) had no other objective abnormality. Possible CTD (36) had positive laboratory or clinical findings (Sjögren’s 5, digital scars or ulcers 5, telangiectasia 3, sclerodactyly 1, dysphagia 1). Thirteen had established diagnoses: SSc (5), SLE (5), RA (2), dermatomyositis (DM) (1).

The technique used for NCM was based on that described by Maricq et al. [7]. The ring finger of the non-dominant hand was examined using a Vickers dissecting microscope (×30 magnification) with an incident light source. Clear nail varnish was applied to the nail/skin interface and the capillaries examined by two observers. The results of NCM in the three groups of patients are shown in Table I.

NCM proved to be a simple test with no intra-observer error. We found it most useful in the management of the RP-only group. Normal NCM has enabled us to diagnose primary RP in 21 patients and the 14 who have had RP for >4 yr will not be reviewed routinely. A patient in this group who had tortuous capillaries has subsequently developed metastatic gastric carcinoma.

In the possible CTD group, the 10 with dilated capillaries will be closely monitored for evidence of SSc. They include nine with negative antibodies and five with no skin abnormalities, and highlight the importance of NCM in monitoring this group. Follow-up of similar patients over 2.1–6.5 yr [3, 4, 5, 8] suggests that between three and seven of our 11 patients with abnormal NCM will develop CTD.

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Accepted 26 January 1997
TABLE I

<table>
<thead>
<tr>
<th>Group</th>
<th>Total</th>
<th>Female</th>
<th>Duration of RP (yr)</th>
<th>Positive ANA</th>
<th>Positive RF</th>
<th>Abnormal NCM</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>&lt;4</td>
<td>4–9</td>
<td>10+</td>
<td></td>
</tr>
<tr>
<td>RP only</td>
<td>26</td>
<td>22</td>
<td>39.4 (21–74)</td>
<td>8</td>
<td>7</td>
<td>11</td>
</tr>
<tr>
<td>Possible CTD</td>
<td>36</td>
<td>33</td>
<td>52.2 (28–79)</td>
<td>14</td>
<td>6</td>
<td>16</td>
</tr>
<tr>
<td>Definite CTD</td>
<td>13</td>
<td>11</td>
<td>48 (28–69)</td>
<td>3</td>
<td>2</td>
<td>8</td>
</tr>
<tr>
<td>SSc 5</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SLE 5</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RA 2</td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DM 1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>75</td>
<td>66</td>
<td>47</td>
<td>25</td>
<td>15</td>
<td>35</td>
</tr>
</tbody>
</table>

DL, dilated loops; TL, tortuous loops.

CD134/OX40 Expression by Synovial Fluid CD4+ T Lymphocytes in Chronic Synovitis

Sir—Pitzalis [1] has recently reviewed the role of adhesion mechanisms in the pathogenesis of chronic synovitis. The processes leading to the accumulation of CD45R0+ T lymphocytes in synovial fluid (SF) of chronic synovitis were illustrated focusing on the selective adhesion to and migration through the endothelium by activated T cells. The main role in these mechanisms is played by LFA-1 and VLA integrins, but a proportion of the binding of T cells to endothelial cells cannot be inhibited by the addition of combinations of blocking antibodies against known adhesion molecules.

Recently, a new molecule has been demonstrated to play an important role in the process of adhesion of activated T cells to the endothelium [2], and it has been identified as the previously described OX40 antigen (now, also termed CD134), a member of the tumour necrosis factor receptor family [3–5]. Anti-OX40 antibodies inhibit the binding of phytohaemagglutinin-activated CD4+ T cells to human vascular endothelial cells, which were found to express OX40-ligand [2]. Moreover, OX40-transfected T cells were shown to bind in an OX40-system-specific way to vascular endothelial cells [2].

In humans, OX40 is detectable in non-malignant tissues only on very rare lymphocytes in the T-cell compartment of lymphoid organs [6] and is virtually absent on peripheral blood resting lymphocytes [6, 7]. However, its expression can be induced upon stimulation in vitro with mitogens, mainly on CD4+ cells, but also on a small number of CD8+ cells [6, 7].

We have studied by flow cytometry the expression of OX40 using the monoclonal antibody L106 (provided by the VI International Workshop for Leucocyte Differentiation Antigens) in eight patients with rheumatoid arthritis (RA) and in six age-comparable normal individuals. On CD4+ cells purified from peripheral blood by means of immunomagnetic beads (Dynal, Oslo, Norway; purity was always >96%), only a moderate difference between the two groups was observed, as far as the number of cells co-expressing OX40 was concerned (mean ± 1 S.D. in RA 13 ± 3% vs 6 ± 2% in healthy controls; P = 0.053; Wilcoxon’s rank sum test).

However, high levels of OX40 expression were observed on CD4+ cells purified from SF of eight patients with RA (49 ± 25%; P < 0.01 vs peripheral blood CD4+ cells from healthy controls), in a patient with psoriatic arthritis (PsA) (29%), and even in two patients with osteoarthritis (OA) of the knee, from whom lymphocytic SF was aspirated (median 30%). On the other hand, only a small number of purified