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Accepted 6 December 2003

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Visceral leishmaniasis resembling systemic lupus erythematosus

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Ann Rheum Dis 2004;**63**:1348–1349. doi: 10.1136/ard.2003.014480

Systemic lupus erythematosus (SLE) is a chronic autoimmune disease of unknown aetiology. B cell hyperactivity with production of multiple autoantibodies is the hallmark of the disease.[1] On the other hand, such polyclonal B cell activation may occur in chronic infectious diseases. In this report we present a patient with visceral leishmaniasis who was diagnosed as having SLE and we discuss the clinical and laboratory findings which may discriminate between these two entities.

CASE REPORT

A 50 year old man presented in October 2001 with arthralgias, fatigue, weight loss, and low grade fever. Laboratory evaluation revealed haemoglobin 110 g/l, white blood cells 3.9×10^9/l with normal differential count, platelets 90×10^9/l, and erythrocyte sedimentation rate (ESR) 50 mm/1st h.

He was admitted to the hospital where physical examination disclosed mild splenomegaly. A laboratory investigation confirmed anaemia, leucopenia, and thrombocytopenia, increased ESR and C reactive protein (CRP) (table 1). Renal, liver and thyroid function tests, as well as urine analysis were within normal limits or negative. Serum electrophoresis showed moderate diffuse hypergammaglobulinaemia with no monoclonal bands. A stool specimen for occult blood was negative. Repeated blood, urine, throat, and bone marrow cultures were negative. Serological tests for viral hepatitis B and C, cytomegalovirus, Epstein-Barr virus, and human immunodeficiency virus, as well as for toxoplasma and brucella infections were negative. Immunological tests showed positive antinuclear antibodies (ANA) at a titre of 1/1280 with a fine speckled pattern, positive IgM rheumatoid factor at a titre of 1/640, positive anti-Sm antibodies, positive Venereal Disease Research Laboratory (VDRL) test and positive lupus anticoagulant test (table 1). A chest radiograph was normal and a purified protein derivative test was negative. Finally, bone marrow biopsy showed no abnormalities. A diagnosis of SLE was made.

Two months later, the patient experienced high spike fever, fatigue, and weight loss. He was treated with small doses of steroids without improvement and he was admitted for further evaluation. Physical examination showed a body temperature of 39°C. The patient was sweating, anxious, and pallid. The rest of physical examination disclosed moderate splenomegaly. A computed tomography scan of the abdomen confirmed the presence of splenomegaly. Laboratory and immunological tests were similar to those done previously (table 1). However, a high titre of antibodies directed against *Leishmania donovani* was detected (1/1280 by immunofluorescence assay). A repeated bone marrow biopsy disclosed the presence of parasites in the macrophages (fig 1). Sodium antimony gluconate was given intramuscularly for 4 weeks, with excellent results.

DISCUSSION

The haematological abnormalities of SLE include haemolytic anaemia, leucopenia or lymphopenia, and thrombocytopenia, due to the presence of autoantibodies directed against erythrocytes, leucocytes, and platelets.[1] The diagnosis of SLE requires four or more of the American College of Rheumatology criteria.[2] In our patient the diagnosis of SLE was based on the following criteria: arthralgias, haematological abnormalities, positive ANA, and positive VDRL and anti-Sm antibodies. However, this patient also had splenomegaly and high titres of CRP. Splenomegaly is not a common sign of SLE, unless there is lymphoma development or concurrent infection. High titres of CRP are not a common laboratory finding in SLE, unless a concurrent infection occurs. However, high titres of CRP in lupus have been associated with symmetrical polyarthritis and the presence of pleurisy.[3]

Table 1 Laboratory and immunological features

Variables	October 2001	February 2002
Haemoglobin (g/l)	110	105
White blood cells ($\times10^9$/l)	3.9	3.65
Platelets ($\times10^9$/l)	90	75
ESR (mm/1st h)	50	65
C reactive protein (mg/l)	56	75
Antinuclear antibodies (titre)	1/1280	1/1280
IgM rheumatoid factor (titre)	1/640	1/640
Anti-Sm antibodies	Positive	Negative
VDRL test	Positive	Not done
Lupus anticoagulant	Positive	Not done
Direct Coombs test	Positive	Negative

VDRL, Venereal Disease Research Laboratory.

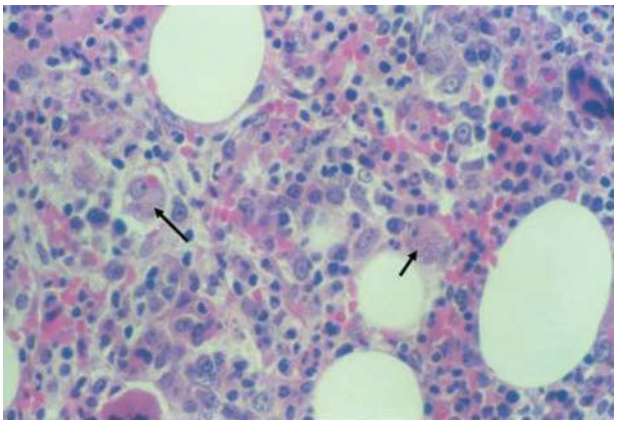

Figure 1 Bone marrow biopsy. Leishmania parasites are phagocytosed by macrophages (arrows). Haematoxylin and eosin ×400.

On the other hand, splenomegaly is a common finding in chronic infections, and especially in parasitic ones. Visceral leishmaniasis is caused by *Leishmania donovani*. It is characterised by fever, sweating, cytopenias and may be associated with many immunological abnormalities. Haematological abnormalities expressed as leucopenia, thrombocytopenia, or anaemia are mainly due to splenomegaly and hypersplenism.

Leishmania donovani is an intracellular parasite which attaches to macrophage receptors, and is phagocytosed and multiplies. In addition, *Leishmania donovani* infection induces a non-specific, as well as a specific antibody production, much of which is probably due to the parasite-released substances, which act as B cell mitogens.[4 5] As a consequence of the B cell hyperactivity, *Leishmania donovani* may cause hypergammaglobulinaemia and the production of autoantibodies such as ANA, and others.[6] On the other hand, the prolonged saturation of the reticuloendothelial system infected by parasites contributes to organomegaly and mainly to splenomegaly, causing cytopenias.

We conclude that:

- All patients with positive ANA do not have SLE
- Splenomegaly is not a common sign in patients with SLE
- High titres of CRP are not a common laboratory finding in lupus patients and may discriminate SLE from infections
- Visceral leismhaniasis may present with cytopenias and the production of autoantibodies mimicking SLE.

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Accepted 18 November 2003

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Neurovascular mechanisms as a possible cause of remission of rheumatoid arthritis in hemiparetic limbs

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Ann Rheum Dis 2004;**63**:1349–1351. doi: 10.1136/ard.2003.016410

In patients with rheumatoid arthritis (RA), ischaemic stroke frequently leads to an unexplained remission of the arthritis in the paretic limb. Here we present two cases which suggest that neurovascular mechanisms contribute to the asymmetry of inflammation by impairing the microcirculation in the paretic extremity.

CASE REPORTS

A 47 year old man developed RA in 1988. In 1990, he had an apoplectic insult, resulting in a complete, left sided hemiplegia. The right hand had a marked ulnar drift of the metacarpophalangeal (MCP) joints and an inflamed wrist with impaired motion, whereas the left hand showed no inflammation or deformity. An *x* ray analysis of the right hand demonstrated carpal ankylosis and subluxation and erosion of all MCP joints. The left hand showed no erosive changes (fig 1A). Thermal imaging indicated marked temperature differences between both hands, most obvious at the wrists (fig 1B). Duplex sonography measured no detectable flow of the left radial artery. Electrophysiological investigation suggested demyelination that was slightly more pronounced on the paralytic side. The sympathetic skin response was negative.

The second case involved a 66 year old woman who had had RA since 1982. In 1988 she had an ischaemic stroke with right sided hemiparesis that later recovered, leaving merely somewhat diminished muscle strength and minor hyperaesthesia. The left wrist and all MCP joints had active arthritis, whereas on the right, only two MCP joints were inflamed. An *x* ray analysis of the left hand disclosed carpal ankylosis, subluxation of all MCP joints, and erosions of all carpometacarpal (CMC), MCP, and proximal interphalangeal