tightness aggravated by deep respiration as well as pain in ankles and knees. Physical examination showed a pericardial friction rub and polyarthritits affecting ankles and knees. The electrocardiogram showed widespread elevation of ST segments and chest x-ray examination revealed an increase in heart size compatible with pericardial effusion. Laboratory tests showed haemoglobin 73 g/l, packed cell volume 0.22, reticulocytes 3%, platelets 119 x 10^9/l, and white blood cell count 3.78 x 10^9/l (56% neutrophils, 14% eosinophils, 2% basophils, 34% lymphocytes, 6% monocytes, 1% myelocytes). Erythrocyte sedimentation rate was 118 mm/h. Blood and urine chemistries were within normal limits. A thick smear for malaria was negative. The Coombs direct test (polyspecific anti-globulin, anti-IgG, anti-C3b/C3d) was negative and Coombs indirect test was also negative. Antinuclear antibody test was positive at a titre of 1/800 (spotted pattern), dsDNA antibodies (Farr's technique) were not detected, and complement levels were normal. Cardiolipin antibodies (enzyme linked immunosorbent assay (ELISA)) were detected (IgG isolated at high level and IgM at moderate level) and lupus anticoagulant was negative. Tissue antibodies were present (antiparietal cells 1/100, antismooth muscle 1/200). Antinuclear cytoplasmic and histone antibodies were negative. Circulating immune complexes were 76 µg/ml. The same day, treatment with quinine was withdrawn with a prompt relief of symptoms within 24–48 hours and laboratory tests returned to normal within two weeks. After another year follow up the patient remained asymptomatic and no autoantibodies are detected in her serum.

Multiple quinine dependent antibodies have been reported as being responsible for a variety of clinical syndromes associated with anaemia, leucopenia, thrombocytopenia, coagulopathies, renal failure, and disseminated intravascular coagulation. 8 However, a lupus-like syndrome and production of cardiolipin antibodies induced by this drug have not been previously described. Interestingly, histone antibodies, which are often found in other drug induced lupus-like syndromes, were not detected in our patient. On the other hand, although malaria itself may produce a number of immunological abnormalities—including the production of cardiolipin antibodies, 9 it is unlikely that this infection was the cause of the lupus-like syndrome and the cardiolipin antibodies because these appeared when the patient was afebrile and with a thick smear negative for Plasmodium falciparum, and disappeared after withdrawal of quinine.

Quinine should be considered as the potential cause when patients receiving this drug present with either a lupus-like syndrome or warning signs of a coagulation disorder. Failure to consider quinine as a possible cause for the symptoms will lead to delay in diagnosis and prolonged morbidity, whereas both syndromes will disappear quickly when quinine treatment is stopped.

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Scleroderma and the watermelon stomach

The watermelon stomach is an unusual cause of gastrointestinal blood loss and iron deficiency anaemia. We report on two patients who presented with the watermelon stomach and subsequently developed typical features of scleroderma.

A previously healthy 72 year old white woman was found to have a haemoglobin of 62 g/l. Colonoscopy evaluation disclosed non-blooding rectal arteriovenous malfor-


The watermelon stomach has been reported to occur in association with autoimmune diseases. 3 Patients with gastric antral vascular ectasia and scleroderma have been

Watermelon stomach with erythema (patient No 2).
Improvement of severe pulmonary hypertension in a patient with SLE

The development of pulmonary hypertension during the course of connective tissue diseases is a prognostic severity marker. The CREST syndrome is a variant of limited cutaneous systemic sclerosis. We report on the case of a patient with advanced pulmonary hypertension arising in the course of systemic lupus erythematosus (SLE) successfully treated with vasodilator and immunosuppressive drugs.

Our patient had several of the variables associated with poor survival rates in patients with pulmonary hypertension: 1. New York Heart Association functional class IV, presence of Raynaud’s phenomenon, raised mean pulmonary arterial pressure, decreased cardiac index. Immunosuppressive treatment may be warranted early in the course of the disease, as no treatment has shown sustained efficacy. Heart-lung transplantation may be undertaken, but experience in patients with systemic disease is limited, costly, and disappointing. We report on a patient with advanced pulmonary hypertension arising in the course of systemic lupus erythematosus (SLE) successfully treated with vasodilator and immunosuppressive drugs.

The patient was a 22-year-old woman who presented arthralgias, myalgias, pericarditis, Raynaud’s phenomenon, hypocomplementaemia, high titre anti-nuclear antibodies with speckled pattern (anti-Sm, anti-RNP) without DNA or phospholipid antibodies. Low dose prednisone (10 mg/d) was started. Exertional dyspnoea, syncope and signs of right side heart insufficiency appeared before January 1991, related to the early pulmonary capillary wedge pressure 7 mmHg pulmonary hypertension (table). Neither thromboembolism—that is, normal pulmonary angiography, nor any post-transplantation was performed. Heart-lung transplantation was considered, but not performed because no donors were available at that time.

In January 1992 corticosteroid treatment (500 mg pulse methylprednisolone for three days, followed by oral prednisone 0.5 mg/kg/d) in combination with monthly pulse cyclophosphamide (750 mg) and mesna were instituted. After two months a dramatic haemodynamic improvement was observed: the patient had significant resolution of her dyspnoea and was able to walk. Thus in January 1993 prednisone was tapered and pulse cyclophosphamide was given once every three months. At that time the patient returned to work. In September 1994, when the patient had received a total of 20 cyclophosphamide courses over a period of 32 months without any side effects, pre-capillary (pulmonary capillary wedge pressure 3 mmHg) pulmonary hypertension (table) showed an improvement in pulmonary hypertension with both an increase in cardiac index (+73%) and a decrease in mean pulmonary artery pressure (−44%).

Evolution of haemodynamic findings before (January 91 and 92) and after two (March 92) and June 93 and September 95 cyclophosphamide infusions

<table>
<thead>
<tr>
<th>Treatment</th>
<th>PAP (mmHg)</th>
<th>Mean PAP (mmHg)</th>
<th>PVR (mmHg min/l)</th>
<th>CI (l/min/m²)</th>
<th>Pao2 (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>January 91</td>
<td>Prednisone 5 mg/d</td>
<td>94/41</td>
<td>57</td>
<td>26</td>
<td>2</td>
</tr>
<tr>
<td>January 92</td>
<td>Prednisone 30 mg/d</td>
<td>85/40</td>
<td>56</td>
<td>ND</td>
<td>ND</td>
</tr>
<tr>
<td>March 92</td>
<td>Prednisone 30 mg/d</td>
<td>66/27</td>
<td>ND</td>
<td>ND</td>
<td>78</td>
</tr>
<tr>
<td>June 93</td>
<td>Prednisone 15 mg/d</td>
<td>72/23</td>
<td>48</td>
<td>13</td>
<td>5</td>
</tr>
<tr>
<td>September 95</td>
<td>Prednisone 5 mg/d</td>
<td>54/18</td>
<td>32</td>
<td>9</td>
<td>3</td>
</tr>
</tbody>
</table>

*PAP = pulmonary artery pressure, systolic/diastolic; PVR = pulmonary vascular resistance; CI = cardiac index; Pao2 = arterial O2 tension, breathing room air. 1PAP calculated with echocardiography.