structure is under the control of germ-line genes, the sharing of idiotypes implies a restriction in these genes amongst the population sharing the idiotypes. The patient was demonstrated to have one of the common anti-DNA antibody idiotypes (32/15) already identified amongst monoclonal anti-DNA antibodies[7] and serum autoantibodies[15] as well as tissue bound autoantibodies[8]. That his rash manifested itself in a clinically unusual way is noteworthy, given that the DNA antibody idiotype identified has previously been found in patients with typical lupus rashes.

The inhibition of the binding of anti 32/15-R to its homologous idiotype 32/15 by both ssDNA and dsDNA supports the idea that in this patient the 32/15 idiotype is present on DNA antibody molecules which have been deposited at the dermoepidermal junction. It should be noted that an idiotype first identified on an antibody of given isotype and antigenic specificity is not necessarily confined either to that isotype or antigen specificity[15].

There appears to be a variable response to therapy in vesiculobullous LE. Good responses to both dapsone and oral steroids have been reported. From the limited reports which are available it appears that the response to oral steroids may be greater if the skin eruption is associated with a flare of the systemic disease. If it is not associated with significant systemic activity, dapsone may be the more successful treatment[2]. In our patient a combination of dapsone, prednisolone and azathioprine appears to be the most effective.

We conclude that the patient reported here meets the criteria of Camisa and Sharma[1] for the diagnosis of vesiculobullous SLE. The idiotype and inhibition studies provide strong circumstantial evidence suggesting that anti-DNA antibodies are deposited at the DEJ in this entity.

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Pulmonary embolism complicating murine typhus

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We report a patient suffering from murine typhus (MT) who developed deep vein thrombophlebitis which was further complicated by acute pulmonary embolism (PE). PE in MT has not previously been reported. The mechanism responsible for this hazardous complication of MT is discussed. Attention should be paid to patients with MT who present with pulmonary symptoms.

Case report

A 50-year-old man, previously in good health, was admitted with a 10-day history of fever, chills, profuse sweating, diffuse articular pains, myalgia, severe headaches and a dry cough. Two days prior to admission nausea and vomiting had commenced. His family physician had prescribed a combination of ampicillin and cloxacillin which was later replaced by co-trimoxazole; neither regimen had had a favourable response.

On examination the patient was pyrexial (39°C) with a tachycardia of 120/min. The lungs were normal on percussion and auscultation. A 1/6 systolic murmur was detected over the cardiac apex. The edge of the liver was palpable 3 cm below the costal margin. Tenderness was elicited on squeezing the left calf but Homan's sign was negative. The rest of the physical examination was normal.

The primary laboratory tests revealed: haemoglobin 13.1 g/dl, white blood count 7600 with a marked shift to the left, SGOT 55 units, SGPT 62 units. Several blood and sputum cultures were negative. The serological tests were as follows:

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C-reactive protein + 2, Weil-Felix, Widal, antinuclear antibodies, were negative; C_3, C_4 levels were normal. Chest X-ray did not reveal any abnormality but the electrocardiogram demonstrated a diffuse flattening of the T waves. Both intravenous pyelography and liver-spleen scan were within normal limits.

On the fourth hospital day the patient had a syncopal episode. On immediate examination a blood pressure of 100/70 and a pulse rate of 108/min were recorded. About 3 hours later the patient had a bout of coughing with streaks of blood in the sputum. This time fine, dry, basal crepitations and a pleural friction rub could be heard bilaterally. Arterial pH was 7.49; P_{CO_2} 35 mmHg; P_{O_2} 68 mmHg; and bicarbonate 28 mEq/l. The electrocardiogram showed an $S_1Q_3T_3$ pattern. Lung scan demonstrated multiple perfusion defects at the lung bases. A diagnosis of acute pulmonary embolism was established and treatment with heparin 30 000 units/day was instituted. The fever curve showed some improvement but five days later it rose again. Heparin was gradually replaced by a coumarin derivative. A few days later the results of the indirect immunofluorescence antibody (IFA) test to rickettsial antigens (relating to the blood specimen drawn on the second day of hospitalization) were available. Antibodies to MT of both IgM and IgG fractions at a titre of at least 1:40 were detected (further titrations are not performed), with a fluorescence of +3. Serial IFA determinations performed at 2-week intervals revealed a rise (to +4) and fall (to + 2) in the degree of fluorescence of IgM antibodies, while the IgG remained constant at + 4. Accordingly, the patient was given tetracycline and recovered completely. Three months later the patient still complained of weakness; a tachycardia of 100/min and a mild pleural rub were present. A second lung scan demonstrated a marked improvement.

Discussion

Pulmonary embolism (PE) is a leading cause of morbidity and mortality. It usually commences with the formation of a thrombus in the deep calf veins in conditions such as the postoperative period, pregnancy, congestive heart failure, chronic obstructive lung disease and fractures of the lower extremities[1]. An inflammatory process which damages the vessel wall is among the mechanisms initiating thrombus formation[2]. Such inflammatory abnormalities have been described in polyarteritis nodosa, Buerger's disease, anaerobic bacterial infections, rickettsial and meningococcal infections, arsenic and mercury intoxications as well as following snake bites[2,3]. However, the risk of developing venous thrombosis and subsequently PE during the course of an infectious disease does not seem to have been emphasized.

Although our patient did not exhibit a maculopapular rash, which may occur in murine typhus (MT)[4], and had a negative antibody response to the proteus OX-19 antigen[5], the entire clinical picture was compatible with MT. Moreover, the IFA test, which is currently the most sensitive and specific of the established techniques for detection and characterization of MT[6,7], was strongly positive.

MT is a rare disease today, and in the era of effective antibiotic therapy the complications of this disease are even more infrequently seen[8]. The documented complications include central nervous system disturbances[9-11], myocardial injury[12], gangrene of the extremities[8], impairment of kidney function[9,12], phlebitis[12] and cephalic vein thrombosis[9]. In the late stages of the disease pneumonitis and bronchopneumonia may also occur[9]. A thorough search of the literature for the last 50 years revealed no reports on PE as a complication of MT. This may stem from the fact that accurate diagnostic means for the detection of PE were scarce at times when MT was more prevalent. Nevertheless, it is interesting that haemoptysis has been recorded in MT, but was attributed to bronchial erosions[10].

The pathophysiology of the rickettsial diseases could well explain the formation of clots within blood vessels with subsequent seeding of emboli. Rickettsiae are known to invade the endothelium of blood vessels – arterial, capillary, as well as venous[13]. The damage to the integrity of the endothlial lining may thus begin the clotting cascade.

We suggest that patients with MT and possibly other rickettsial diseases should be monitored for the occurrence of deep vein thrombosis. The development of pulmonary symptoms in these patients might indicate that PE has occurred.

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