antisocial, and had intellectual difficulties. A psychotherapist she saw regularly diagnosed a narcissistic personality disorder. After four years of self bleeding she stopped for one year, and her haemoglobin concentration became 120-130 g/l.

Comment

Our patients had typical features of factitious disease,[1 2] three of them developing anorexia nervosa. They had been bleeding themselves for four to 11 years and were well adapted to severe chronic anaemia with haemoglobin concentrations rarely above 60 g/l. Their physical fitness was remarkable, and, notably, they tailored their bleeding such that it did not jeopardise an active life.

Although our patients showed little overt psychopathological behaviour, all had a borderline personality disorder.[2] Psychotherapy seems to be of little value in such disorders, and only one patient with factitious anaemia has been successfully treated with psychotherapy.[4] Nevertheless, such patients should be offered regular medical and psychiatric care; psychiatric consultation at an early stage in somatisation disorders reduces the costs of health care considerably, although it does not improve mental, physical, or social health.[5] Similarly, our patients appreciated regular meetings with doctors or psychotherapists to discuss their problems, although such meetings did not result in a cure.

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Central Haematology Laboratory and Department of Medicine, Inselspital, University of Berne, Berne, Switzerland
MARTIN F FEY, MD, registrar
ANDREAS RADVILA, MD, senior lecturer

Correspondence to: Dr A Radvila, Department of Medicine, Inselspital, CH-3010 Berne, Switzerland.

Fatal cardiac failure due to myocardial microthrombi in systemic lupus erythematosus

Systemic lupus erythematosus has a variable clinical presentation and a high mortality. We report on a patient with the condition whose death was caused by acute circulatory failure resulting from diffuse thrombotic occlusion of the myocardial microcirculation.

Case report

A 22 year old woman with a six month history of "poor circulation" and exertional dyspnoea was admitted to hospital because of digital ischaemia. Examination showed lip ulceration, cervical lymphadenopathy, and vasculitic lesions on her hands and feet. She had a sinus tachycardia of 110 beats/minute, blood pressure of 130/100 mm Hg, and an apical mid-diastolic murmur.

Investigations showed that the haemoglobin concentration was 121 g/l, white cell count $24{\cdot}2\times10^9$/l, platelets 77×10^9/l, blood urea 13·8 mmol/l, and serum creatinine 135 μmol/l. Dipstick testing of urine for blood and protein yielded positive results, and red cell casts were seen on microscopy. An electrocardiogram showed generalised S-T segment depression of 1 mm, and a chest *x* ray film showed slight cardiomegaly with no pulmonary congestion. Echocardiography confirmed a moderately severe mitral stenosis. The left atrium, right atrium, and right ventricle were enlarged, but the left ventricle was of normal size. Serum titres of antinuclear antibody were 1/640 (IgG) and 1/80 (IgM), and the concentration of antibody to double stranded DNA was 80 units/ml (normal range 0-25). Serum was strongly positive for IgG and weakly positive for IgM anticardiolipin antibodies on enzyme linked immunosorbent assay (ELISA) with Loizou's method. Serum C3 concentration was 0·58 g/l (normal range 0·75-1·50). A coagulation screen was within normal limits; the test for lupus anticoagulant was not done.

Her condition deteriorated with confusion, oliguria, and rising blood urea concentrations. Aggressive systemic lupus erythematosus was diagnosed and methylprednisolone and cyclophosphamide started, the intention being to start plasma exchange and dialysis the next day. Within 24 hours, however, she developed profound hypotension and peripheral circulatory failure and died.

At necropsy the kidneys showed focal glomerulosclerosis, proliferative glomerulonephritis, and a superimposed necrotising glomerulitis. The glomerulitis was related to afferent arteriole microthrombi and to fibrinoid vasculitis, which was also present in the lungs, liver, and pancreas. No abnormality of the brain was seen and no source of infection found. There was moderately severe mitral stenosis with fusion of the valve cusps due to warty, erythematous endocardial nodules. Aggregates of fibrin, inflammatory cells, and DNA debris were attached to the valve surface and adjacent endocardium. The main coronary arteries were mildly atheromatous. Many of the intramyocardial arteries contained occlusive thrombi unaccompanied by vasculitis but surrounded by extensive recent myocardial necrosis.

The findings at necropsy were consistent with systemic lupus erythematosus but also showed mitral stenosis caused by Libman-Sacks endocarditis. Death resulted from diffuse acute myocardial necrosis due to extensive thrombi in the microvasculature.

Comment

Though renal failure is one of the major causes of death in systemic lupus erythematosus and our patient had severe lupus nephritis, the degree of uraemia would not explain the fatal circulatory failure. There was no evidence of overwhelming infection, of lupus in the cerebrum, or of appreciable atheroma in the large vessels. Libman-Sacks endocarditis rarely causes severe valvular dysfunction, although isolated reports have documented mitral regurgitation and combined stenosis and regurgitation similar to that in our patient.[2] In our case, however, there was no clinical or radiological evidence of pulmonary oedema or central cyanosis, and the mitral stenosis is thus unlikely to have led to the fatal circulatory failure.

Death from fulminant cardiac necrosis resulting from thrombotic occlusion of the myocardial microcirculation has not been described previously.[1 3] Circulating antiphospholipid antibody may have played a part, as the lupus anticoagulant has been associated with arterial and venous thromboses.[4] Thus thrombolytic or fibrinolytic treatment, or both, might be beneficial. Kant has reported resolution of microvascular thrombosis in patients with systemic lupus erythematosus treated by ancrod, the defibrinogenating enzyme obtained from the venom of the Malayan pit viper.[5]

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Belfast City Hospital, Belfast BT9 7AB
J H BROWN, MB, MRCP, registrar, renal unit
C C DOHERTY, MD, MRCP, consultant nephrologist
D C ALLEN, MB, MRCPATH, senior registrar, department of pathology
P MORTON, MD, FRCP, consultant cardiologist

Correspondence to: Dr Doherty.

Effect of low dose heparin on blood loss at caesarean section

There is good evidence for the efficacy of low dose heparin in preventing venous thrombosis and pulmonary embolism after surgery,[1] but prophylactic heparin is not usually given before caesarean section. The main reason is the fear of increasing the amount of bleeding in an operation which has a high operative blood loss, but there is also the difficulty of predicting the effect of heparin in pregnancy, where the coagulation mechanism may already be disturbed. We decided to measure the effect of heparin on blood loss during caesarean section.

Patients, methods, and results

Fifty patients were given a subcutaneous injection of either 5000 units of sodium heparin or isotonic saline one hour before the operation and similar injections twice daily for five days after the operation. There were no significant differences in age, parity, blood pressure, height, weight, and gestation between the two groups. Patients were excluded from the study if they had a placenta praevia, multiple pregnancy, pregnancy induced hypertension, antepartum