Technetium-99m-labelled pertechnetate is 90% bound to intravascular proteins and diffuses readily to extravascular spaces (Hays and Green, 1972). In normal subjects an equilibrium is reached in muscles 6-8 minutes between intravascular and extravascular pertechnetate. This time was more than doubled in our patient at the beginning of the disease, most probably because of the inflammatory processes involved in the angiitis, but it returned to normal under treatment.

Furthermore, the scintigraphic changes correlated well with the clinical state and the changes in the biological values. The scan pattern and the delay in the equilibrium of the curve were still abnormal when the second biopsy was normal though one could infer from the clinical state and the E.S.R. that the disease was still active. After 13 weeks the only abnormalities were a raised E.S.R. and the scan pattern.

Whether the appearance of the scan is pathognomic for periarteritis nodosa, as stated by Mintz *et al.* (1970), remains to be proved, but we feel that the combination of dynamic studies and scintigraphy provided a valuable and objective means of evaluating the extent of this disease and its response to treatment. The possibilities offered by this simple method in other collagen and muscle diseases are under investigation.

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Termination of Pregnancy with Utus Paste: Report of a Fatal Case

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Termination of a mid-trimester pregnancy when the uterus is already scarred from a previous hysterotomy has until recently posed a problem of management. If a second hysterotomy is performed subsequent pregnancies may require delivery by ceasarean section to avoid a risk of the uterus rupturing during labour. Hence pharmacological methods of termination have been used instead of a second hysterotomy. One such abortifacient agent which has been available for a number of years is Utus paste and many reports of its efficacy have appeared.

Utus paste is a proprietary preparation consisting of a semi-solid soap mixed with potassium iodide and astringents. It is administered after warming to 37°C by injection through a sterile metal cannula which has previously been introduced through the uterine cervix. Expulsion of the uterine contents usually occurs within 24 hours and should be complete. If uterine evacuation is incomplete or if it is followed by persistent bleeding curettage is performed.

Reports of serious and sometimes fatal reactions to the use of intrauterine pastes have appeared in the literature during the past 30 years. We report such a case which occurred immediately before the prostaglandins became available.

Case Report

The patient, aged 19 years, was undergoing therapeutic termination of a pregnancy of 14 weeks' gestation for social reasons. Three years previously she had undergone a hysterotomy for the termination of a pregnancy of 20 weeks' gestation. On the present occasion the uterus was thought to be too large to allow suction termination and it was therefore decided to use Utus paste. Preoperatively her haemoglobin was 13·4 g/100 ml and there was no previous history of serious illness or allergies. She was not taking any medication.

Premedication with papaveretum 15 mg and atropine 0·6 mg was given about one hour preoperatively. Anaesthesia was induced with 70 mg methohexitone intravenously and maintained with 70% nitrous oxide, 30% oxygen, and 0·5% halothane. The cannula was introduced into the uterus and 25 ml of Utus paste was injected.

Anaesthesia was discontinued and the patient opened her eyes almost immediately. She was lifted on to a trolley and turned to the right lateral position. Within seconds she became apnoeic, cyanosed, and pulseless. She was intubated immediately and ventilated with 100% oxygen and external cardiac massage was begun. A catheter was passed to the right side of the heart and aspiration of blood from the catheter showed no obvious evidence of air embolism. Atropine 0·6 mg, isoprenaline 0·1 mg, and sodium bicarbonate 200 mEq were infused through the catheter. E.C.G. showed asystole but injection of 1 ml of adrenaline 1/1,000 and 10 ml of 20% calcium chloride produced ventricular fibrillation. This was converted to sinus rhythm by external defibrillation and the peripheral pulses were found to be regular and of good volume. Cyanosis persisted however, though the lungs were easy to inflate, and after two or three minutes ventricular fibrillation recurred. External cardiac massage was resumed, mannitol infusion was begun, and a urinary catheter was inserted into the bladder. Uniformly blood-stained urine was obtained and because of the possibility of haemolysis blood samples were sent for full haematological analysis. About 45 minutes after the initial cardiac arrest the patient's colour improved and she reverted to sinus rhythm spontaneously. She was then transferred to the intensive care unit.

The chest *x*-ray picture was normal. The whole blood haemoglobin was 13·2 g/100 ml but 1·1 g of this was accounted for by the plasma haemoglobin. Also the urine was positive for haemoglobin. Intravascular haemolysis was diagnosed from this evidence. The peripheral blood film showed entirely normal red cell morphology. Because of the severe degree of red cell breakdown the serum potassium was estimated and found to be 4·1 mEq/l. (normal). A diagnosis of "defibrination" syndrome appeared to be confirmed by the following results of test performed on the appropriate serum and citrated blood samples: thrombin clotting time 40 seconds (normal control value 14 seconds), corrected by the addition of equal parts of normal plasma to 15 seconds; fibrinogen titre 1 in 4 (normal value 1 in 128 or greater); and fibrin-fibrinogen degradation products (latex agglutination kit; Diagen) a normal level of less than 8 μm/ml. These results were interpreted as showing severe consumption coagulopathy without evidence of enhanced fibrinolytic activity.

In view of this diagnosis anticoagulant therapy was instituted. Heparin was administered by constant intravenous infusion at a rate of 1,600 units/hour in an attempt to prevent progression of the consumption coagulopathy. About six hours later the patient's condition deteriorated and despite intermittent positive-pressure ventilation, blood transfusions, mannitol, and dexamethasone she died 13 hours after her initial cardiac arrest.

Necropsy showed evidence of massive haemolysis, the blood being almost completely fluid throughout the circulatory system. There were extensive subcutaneous haemorrhages, subdural haemorrhages, and a retroperitoneal haemorrhage. No pulmonary embolism was evident though the lungs were oedematous and showed extensive intrapulmonary haemorrhage. There was also haemorrhage into the myometrium and broad ligament but no evidence of uterine trauma and the uterine membranes were intact.

Comment

This patient developed three complications immediately after

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the insertion of Utus paste into her uterus—intravascular haemolysis, consumption coagulopathy, and cardiac arrest. There can be little doubt that part of the injected Utus paste entered the blood draining the uterus. The high intravascular concentration of paste would result in dissolution of the red cell membrane. After this haemolysis coagulation would be triggered leading to fibrin formation. The haemolysis was unlikely to have been provoked by the deposition of fibrin because fragmented red cells were not seen in peripheral blood films.

The multiple fibrin emboli which would occur would bring about widespread obstruction within the microcirculation causing acute circulatory failure and large-scale shunting of blood within the lung. Good oxygenation of the lung would not then relieve gross hypoxaemia. This would explain the protracted cyanosis. Eventually the patient's own fibrinolytic system would become active and would lyse the fibrin occluding the pulmonary vessels, her colour improving as hypoxaemia was relieved.

Close comparison can be drawn between the above postulate and amniotic fluid embolism. In the latter condition, however, residual non-lysable evidence of previous embolism persists in the form of fetal squames, mucus, and sometimes meconium.

Various complications have been reported after the use of intrauterine pastes; haemolysis was initially reported by Weilerstein (1944) and later by Williams *et al.* (1955). These workers also described causes of collapse similar to those in our own case, as did Dutra *et al.* (1950). The latest report of the D.H.S.S. (1972) on maternal deaths in England and Wales included the case of a 38-year-old patient, 18 weeks pregnant, who died 10 minutes after the injection of 25 ml of Utus paste into the uterus. The occurrence of consumption coagulopathy complicating the intrauterine injection of Utus paste has not, to our knowledge, previously been reported. The pathogenesis is uncertain but several potential mechanisms are possible. They include lysis of red cells with thromboplastin release, platelet lysis with thromboplastin formation, a direct action on the plasma coagulation mechanism, indirect initiation of coagulation of complement activation, or some synergy between several of these systems. Further studies have been undertaken by us into both the haemolytic and coagulation-inducing properties of Utus paste and the results will be reported elsewhere.

It is likely that the use of intrauterine pastes will be superseded by safer methods in the near future. This report adds yet another cause of consumption coagulopathy to the long list of medical and surgical conditions which are complicated by this syndrome.

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Glomerulonephritis Associated with Coxiella burnetii Endocarditis

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A patient with endocarditis associated with chronic *Coxiella burnetii* infection is described in whom glomerulonephritis developed with granular deposits containing immunoglobulins and complement in the glomeruli. The serum was notable for the variety of circulating antibodies detected, which included antibodies directed against native DNA.

Case Report

The patient, a man, had had rheumatic fever at the age of 12 years and at age 40 (1955) was found to have mitral stenosis which required mitral valvotomy. In 1964 the signs of mitral regurgitation and aortic stenosis and regurgitation were documented and two years later he developed intermittent fever, myalgia, and purpura and began to lose weight. Repeated blood cultures were sterile and antinuclear factor was not detected. In 1970 replacement of the mitral and aortic valves was performed using fascia lata prostheses and at operation the aortic valve was found to contain a defect suggesting endocarditis. After six months of symptomatic improvement he again lost weight and developed exertional dyspnoea and a catheter study showed recurrence of mitral stenosis. No further surgery was undertaken. In February 1973 (age 58) he developed oliguria and haematuria and was admitted to hospital.

On admission he was in atrial fibrillation and was normotensive (blood pressure 130/70 mm Hg). He had the signs of mitral stenosis and regurgitation and aortic stenosis, splenomegaly, and purpura on the trunk and lower limbs. The blood urea was 230 mg/100 ml, serum creatinine 11·4 mg/100 ml, and haemoglobulin 9·5 g/100 ml with a normochromic blood film. His platelet count was 79,000/mm^3 and platelet antibodies were detected. Repeated blood cultures were sterile and the antistreptolysin-O titre was normal (<200 IU/ml). Serum immunoglobulins were raised (IgG 1,900 mg, IgA 700 mg, IgM 420 mg/100 ml). Tests for antinuclear and rheumatoid factors gave titres of 1/250 and 1/64 respectively and the direct Coombs test was negative. Anti-DNA antibody was found to be raised on two occasions, at 60% on 25 March and 57% on 11 May (normal range 0-30%). Complement fixation tests were negative for brucella but positive for anti-*Cox. burnetii* antibodies (titres for phases I and II were both 1/640) and hepatitis-B antigen (HBAg) was detected in the serum. Serum complement (C'3) was reduced (range of values 38-54 mg/100 ml; normal range 81-150 mg/100 ml) and no cryoglobulins were detected. The L.E. cell phenomenon was absent. Urine microscopy showed red cells and granular casts and urine culture was sterile. A high-dose infusion pyelogram showed normal renal size.

Because of the oliguria at the time of admission peritoneal dialysis was carried out. Renal biopsy showed the glomeruli to be either hypercellular owing to proliferation of endothelial and mesangial cells, with an increase in the mesangial matrix (fig. 1), or sclerotic. Electron microscopy showed granular deposits close to the mesangial region with variable fusion of foot processes of the epithelial cells (fig. 2). Immunofluorescence examination showed IgG, IgA, and C'3 in the glomeruli in a granular pattern (fig. 3).

After peritoneal dialysis his renal function improved and in view of the high anti-*Cox. burnetii* antibody titres, which suggested chronic infection with this organism, he was treated with co-trimoxazole. His renal function subsequently deteriorated, however, and he died in May 1973. Permission for necropsy was not obtained.

Since his illness had been prolonged several previous sera were available and these were examined for anti-*Cox. burnetii* and anti-DNA antibodies (see table).

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