Comment

The sex distribution of patients in the series (five women to one man) shows a female preponderance in contrast to the equal sex distribution in previous series of sarcoidosis in scar tissue (James, 1959, Scadding, 1967). The mean age of onset (37 years) and age range (28-53 years) agree closely with other figures for scar sarcoid involvement (Scadding, 1967).

Scar infiltration by sarcoid tissue has been thought to result either from previous contamination by foreign material in a favourable matrix (Löfgren *et al.*, 1955) or as a hypersensitivity reaction akin to erythema nodosum (James, 1959) occurring at the time of sarcoid activity elsewhere in the body. There was no histological evidence of foreign material contamination in any of the present six patients but the venesection sites did become infiltrated at the time of lung sarcoid involvement in five cases, suggesting that the scar tissue provided a favourable matrix when the sarcoidosis was most active. The venepuncture needles used in all cases, so far as could be ascertained, did not contain metals, such as beryllium and zirconium, likely to give granulomatous reaction.

All these patients exhibited features typical of sarcoid involvement in scars (Scadding, 1967) in that infiltration occurred early in the disease before major lung parenchymal involvement, and that in the four earlier cases the skin changes tended to follow the course of lung changes with good prognosis for complete recovery.

I should like to thank Dr. I. B. Sneddon for his helpful criticism, and Dr. Sneddon, Dr. R. E. Church, Dr. S. S. Bleehen, and Dr. J. J. Daly for permission to report details of patients under their care.

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MEDICAL MEMORANDA

Mononucleosis and the Miniskirt: An Incompatible Combination

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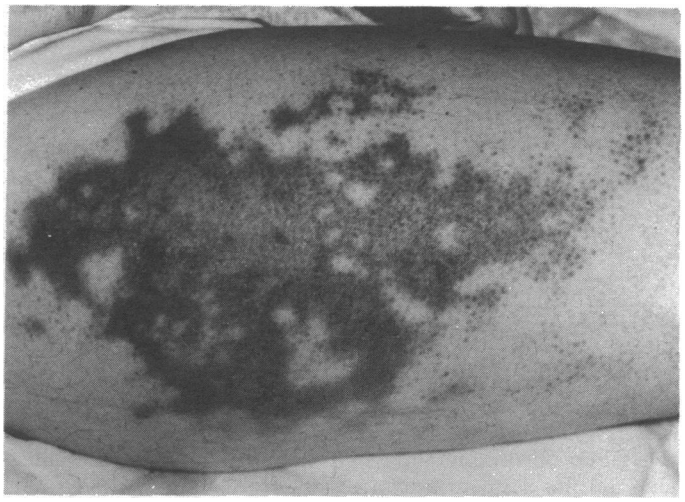
The dangers of being out on a spring night in a miniskirt are not the only ones that leap immediately to mind.

Case History

An attractive 19-year-old girl presented in April 1970 wearing an extremely short miniskirt barely covering the buttocks. She gave a one-week history of pruritus and tenderness over both legs. For three or four days she had complained of slight malaise. On rising on the morning before admission she had discovered blackened areas over the lateral aspect of both thighs, which were painful and tender and gradually turned blue and red. She had not noticed any changes in her fingertips, toes, or the tip of her nose. Apart from childhood exanthemata her only previous rash had been after receiving penicillin seven years previously, and she had not received penicillin before this admission.

When in hospital she had large areas of discoloration on the outer aspect of both thighs measuring about 25 by 15 cm, the upper edges close to the hem of her miniskirt. They had a reticular pattern, being navy-blue in the centre and becoming purple, red, and rust-coloured moving outwards towards the edge (see Fig.). The lesions were shiny, raised above the surrounding skin, not ulcerated, but tender and painful. There were similar but much smaller patches on the outer aspect of each arm. She was apyrexial and had no other abnormal signs; in particular her fauces were normal and there was no lymphadenopathy or splenomegaly.

On the day of admission the haemoglobin was 13·7 g/100 ml, the W.B.C. was 8,350/mm³, platelets were 126,000/mm³, and the E.S.R. was 4 mm in the first hour. The blood film showed spontaneous agglutination of red cells which obscured their morphology, although no fragmented cells were noted. Many atypical mononuclear cells were seen. A screening test for infectious mononucleosis was positive. Next day the haemoglobin was 14·3 g/100 ml, the W.B.C. was 8,900/mm³, 72% abnormal mononuclears, platelets were 94,000/mm³, reticulocytes were 1·5%, and urea and electrolytes were normal. Bilirubin was 1 mg/100 ml, alkaline phosphatase was 32 K.A. units, and alanine transferase was 60 units. Schum's test for methaemalbumin was negative. The prothrombin ratio was 1-3 (control 10 sec.), and the activated partial thromboplastin time 68 sec. (control 31 sec.). L. E. cells were negative. Rheumatoid factor was positive. Paul-Bunnell-Davidsohn test showed antibody against sheep red blood cells present to a titre of 512. The antibody was removed after absorption with ox erythrocytes but not after absorption with guinea-pig kidney. Cold agglutinin titres at various temperatures are given in the Table.



Patient's right thigh on day after admission.

Titres of Adult and Cord Cells at Various Temperatures

	4°C	20°C	32°C	37°C
Adult cells	512	64	8	0
Cord cells	64	4	0	0

Urinary urobilinogen was increased, urinary bilirubin was absent. Chest *x*-ray appearances were normal.

Meteorological information for the period before the illness was obtained from the Long Ashton Agricultural Research Station. It showed that the air temperature had fallen to 1·5°C on the night before the rash appeared and that the grass temperature had fallen to −3°C. Nearly ½ in (1·3 cm) of rain had fallen during the day.

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The patient was kept warm in bed and the rash gradually faded during the course of a week. The discoloration lightened through red and brown, and on discharge from hospital only slight residual staining remained. There was no evidence of a falling haemoglobin, and after discharge she suffered no further symptoms. The cold agglutinins, however, were still present after a month. When reviewed three months later the haemoglobin was normal and cold agglutinins were absent.

Comment

Skin eruptions occur in about 5% of patients with infectious mononucleosis and these have been reviewed (McCarthy and Hoagland, 1964). A reticular discoloration of this kind seems not to have been previously reported. It is likely that this rash was related, firstly, to the cold agglutinins present, producing an area of particular stasis on exposure to the cold night, and, secondly to a bleeding tendency which caused the rash to become haemorrhagic.

Although red cell autoantibodies acting in the cold are the rule in infectious mononucleosis they do not normally give rise to symptoms. Capra *et al.*, (1969) found an indirect acting IgG anti-i antibody in 90% of patients with this disorder, and an IgM anti-i cold agglutinin may be present in between 7% (Jenkins *et al.*, 1965) and 26% (Worlledge and Dacie, 1969) depending on the criteria used. These antibodies being directed mainly against fetal rather than adult red cells rarely cause a haemolytic anaemia. Anti-I, which is directed against adult rather than fetal red cells, does occur in infectious mononucleosis occasionally, and Worlledge and Dacie (1969) referred to 12 such cases with haemolytic anaemia reported in the literature. The present patient undoubtedly had a high titre anti-I, but there was no evidence of haemolytic anaemia.

Mild thrombocytopenia is a fairly common manifestation of infectious mononucleosis. Carter (1965) found counts of less than 100,000/mm^3 in 8 out of 57 patients. Haemorrhagic complications are very rare. The cause of the thrombocytopenia is not clear but is likely to be abnormal consumption.

Abnormalities of coagulation are very rare. The prolonged activated partial thromboplastin time was not further investigated, but there are at least three possible causes for it.

It might have been caused by liver damage, as in the case described by Schumacher and Barcay (1962). This is unlikely since this patient's liver damage was very minor, and factor VII levels, which are usually the most sensitive to liver damage, were, on the evidence of the prothrombin time, unaffected.

Intravascular coagulation might account for a fall in platelets and of clotting factors of the intrinsic system while leaving the extrinsic system unaltered. There are two cases in the literature in which this may have occurred. Wintrobe (1967) referred to a patient with afibrinogenaemia, and Dodsworth and Burns (1971) reported on a patient with hypofibrinogenaemia and thrombocytopenia who responded to treatment with heparin. In neither case was an increased level of fibrin degradation products reported. I cannot exclude this as a cause, but the typical red cell changes were not seen on the blood film.

In infectious mononucleosis a wide array of irregular autoantibodies appear, and it is possible to conceive of antibodies to clotting factors causing a prolonged partial thromboplastin time. I cannot exclude this, but no such case has been previously reported.

I am grateful to Dr. I. S. Bailey for permission to report the case of a patient under his care, and to the Long Ashton Agricultural Research Station for meteorological information.

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Severe Hyponatraemia in Hyperlipaemic Diabetic Ketosis

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The occurrence of spuriously low measured plasma electrolyte values in the presence of hyperlipaemia has been recognized for some time (Albrink *et al.*, 1955). This is due to displacement of the plasma water by lipid so that the water content of a given volume of plasma is depressed below its normal value of 94·5%. By conventional flame photometry techniques the electrolyte content of a measured volume of plasma is determined irrespective of its water content. Therefore in the presence of high concentrations of a displacing substance there will be important and variable discrepancies between the measurements obtained and the true concentrations of electrolytes in the aqueous phase, which is what determines their biological effects. Measurement of osmolality by depression of freezing point is not influenced by the presence of displacing substances and reflects the concentration of solutes in the plasma water. It is recognized that uncontrolled diabetes, especially when of gradual onset, may be accompanied by severe hypertriglyceridaemia (Bagdade *et al.*, 1967). We report here an extreme example of such a case with consequent complications in the management of fluid and electrolyte balance.

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Case Report

In 1969 the patient, a 35-year-old married housewife, presented with tiredness and urinary frequency. She was found to have a urinary infection and glycosuria, with a typical diabetic glucose tolerance curve. At that time she weighed 80 kg and she was started on a 130-g carbohydrate diet; however, she failed to attend for follow-up.

In 1971 she was admitted to hospital with a three-day history of malaise and an eight-hour history of vomiting, confusion, and breathlessness. She was stuporose and clinically dehydrated. The heart rate was 140/min with sinus rhythm, the blood pressure 140/100 mm Hg, and the extremities cold, with vasoconstriction. The respiratory system was normal apart from a respiratory rate of 40/min. The abdomen was generally tender and the liver edge was palpable. There were no localizing neurological signs, but her optic fundi showed intense lipaemia retinalis. There were no eruptive xanthomata.

Laboratory investigations on admission gave the following results: blood glucose 600 mg/100 ml; plasma sodium 86 mEq/l., potassium 2·2 mEq/l., bicarbonate 5 mEq/l., urea 40 mg/100 ml,