Medical Memoranda

Sulphaemoglobinuria Associated with Pyelitis

Sulphaemoglobin is usually an intracorporeal pigment, therefore sulphaemoglobinuria would not be expected. Extracorporeal sulphaemoglobinaemia has occasionally been reported, but even so sulphaemoglobinuria has not been described (Harrison, 1947). This paper describes the identification of sulphaemoglobin in urine.

A dark brown fresh catheter specimen of urine was received in the laboratory from a female patient who was suffering from severe haemolytic anaemia and who had symptoms of pyelitis. The urine contained large quantities of oxyhaemoglobin, and sulphaemoglobin was present at a concentration of about 150 mg. per 100 ml. Sulphaemoglobin was identified by spectroscopic and chemical tests (Sunderman *et al.*, 1953). No methaemoglobin was detected. Microscopical examination of the urine showed occasional erythrocytes and pus cells, and many organisms. On culture a profuse growth of an obligatory anaerobic streptococcus was obtained. Examination of the blood showed an active haemolytic process, and free oxyhaemoglobin was present in the plasma. No other abnormal pigments were detected in the erythrocytes or plasma.

The patient was treated with chloramphenicol, and the sulphaemoglobin disappeared as the urinary infection was cured. The presumptive conclusion reached was that the streptococci had converted oxyhaemoglobin to sulphaemoglobin within the patient's urinary tract. Despite repeated attempts it was not found possible to repeat this conversion *in vitro*: only methaemoglobin was produced when the streptococci were cultured in a medium containing urine and blood.

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Sudden Collapse in Infantile Eczema Treated with Cortisone

The admission to hospital of very young children suffering from infantile eczema has been condemned because of the fear of sudden collapse and death occurring during the first few days after admission (Davies, 1940). The aetiology of the condition is uncertain, and in view of the rapidity with which the collapse develops there are few descriptions of its onset, although the phenomenon is fairly well recognized (Gray *et al.*, 1926).

The following report of a case treated with cortisone is of interest.

CASE REPORT

A 5-months-old boy was admitted to hospital on February 15, 1955, with a history that from the age of 3 months he had had infantile eczema affecting the cheeks, chin, and left leg. Before admission he had been treated with penicillin ointment and Lassar's paste. Six weeks before admission here he had been admitted to another hospital with an acute upper respiratory tract infection, and had remained there for three weeks. During that time there was no record of any untoward developments, but the eczema had spread to involve the head, chest, arms, and legs. The child was admitted here for the treatment of the eczema because his mother was in hospital suffering from a puerperal psychosis, and his aunt who cared for him was distressed by the worsening of his eczema.

On examination he was a well-nourished child who demonstrated no abnormal physical signs apart from a dry,

crusted eczema of the scalp, face, chest, and legs. His temperature, pulse rate, and respirations were normal. Treatment was begun with an ointment containing ammoniated mercury and tar. At 2 a.m. on February 18—that is, 56 hours after admission—his temperature was found to be 104.6° F. (40.3° C.), pulse 146, and respirations 80. However, by 10 a.m. these had fallen to temperature 100.2° F. (37.9° C.), pulse 136, and respirations 44. No abnormality was found in the chest or in other systems on general physical examination, and the skin condition showed no change. At 2.30 p.m., however (68 hours after admission), it was suddenly noted that the skin had cleared and that the child was pale and very quiet. His temperature was taken and found to be 107° F. (41.7° C.); the pulse was of poor volume, with a rate of approximately 172; and the respirations were shallow and at the rate of 90. Fontanelle pulsation was not palpable, and the appearance of the infant suggested a peripheral vascular failure and that he was *in extremis*. Examination of the chest showed no abnormality. A diagnosis of "eczema collapse" was made.

The rapidity of onset and the degree of hyperpyrexia, with absence of other physical signs except those indicative of peripheral vascular collapse, suggested the possibility that the child had developed an acute adrenal failure. It was therefore decided to treat him with cortisone. Tepid sponging had reduced his pyrexia to 100° F. (37.8° C.) and pulse to 146, but his respiratory rate fell only to 70.

He received cortisone in the following dosage:

February 18	3 p.m. . . .	12.5 mg. intramuscularly
	4 p.m. . . .	12.5 mg. orally
	6 p.m. . . .	12.5 mg. "
	8 p.m. . . .	12.5 mg. "
	12 mdt. . . .	12.5 mg. "
February 19	12 noon . . .	12.5 mg. "
	6 p.m. . . .	6.25 mg. "
	12 mdt. . . .	6.25 mg. "

An hourly temperature, pulse, and respiration chart was instituted and all local treatment discontinued with the onset of the collapse. In addition to cortisone, he was given sodium bicarbonate, 1 dr. per pint of water (7 g. per litre) in 2-oz. (57-ml.) doses two-hourly, as had been previously suggested in this condition (Marsden, 1948). His condition improved rapidly, and the temperature and the pulse and respiratory rates returned to normal, while the eczema became clinically apparent once more.

He remained well until the ninth day after admission, when he again became pale and collapsed, though less severely than before (temperature 102° F. (38.9° C.), pulse 150, and respirations 64). Cortisone therapy was again instituted, 12.5 mg. being given orally at six-hourly intervals for six doses, and sodium bicarbonate administered as before. He improved speedily again, and, although the temperature and pulse rate did not subside to normal levels, the fall in the respiratory rate paralleled the clinical improvement in his condition. A further slight rise in the temperature and the pulse and respiratory rates at the time of discontinuing cortisone suggests that therapy may have been stopped too soon.

After this the child gave no further cause for alarm, although at the end of his stay in hospital he developed an attack of rubella. He was finally discharged on March 6.

COMMENT

The dramatic improvement in the clinical picture (from a moribund state to normality within 24 hours) following the administration of cortisone in this instance of collapse in a case of infantile eczema lends some support to the view that the condition may be dependent upon an acute adrenal failure. (This is an explanation already proposed by Marsden, unpublished.) This hypothesis will accord with the characteristic appearance of the condition within a few days of admission to hospital—that is, when the acute additional stress of removal of the child from its normal home surroundings is at its maximum and is acting upon a subject already demonstrating an "allergic" eczematous reaction which itself bears a relation to physiological and environmental stress.