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MEDICAL MEMORANDA

Epidermal Necrolysis from Clothing Impregnated with Paraffin

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Toxic epidermal necrolysis was first described by Lyell (1956) in Great Britain and Lang and Walker (1956) in South Africa. The explosive nature of the eruption, which often occurs in young subjects, and the characteristic "sliding away" of the epidermis over a red and sharply demarcated base usually allow easy recognition.

In a review of 128 cases Lyell (1967) found staphylococci (particularly of phage type 71) and drugs responsible for just over half the cases in children under 10 years of age. Drugs were responsible for most cases in early adult life. In his "miscellaneous" group—occurring at all ages—he mentioned "poisons" as a cause in five cases. One of these was due to externally encountered paraffin (kerosene) in an epileptic. The following case history draws attention to this cause of an acute necrolytic eruption which may occur more often than is apparent from the sparse records.

Case History

A 12-year-old boy was brought to the casualty department of Wycombe General Hospital on a Sunday morning in February 1972, having developed a rash of rather startling appearance on the second day of a camping weekend. The eruption was characteristically that of toxic epidermal necrolysis and he was admitted to the dermatology ward. On examination he was found to be a cheerful and obviously healthy boy. Temperature and pulse rate were normal and his only complaint was of pain, discomfort, and limitation of movement associated with the eruption. This consisted of an extensive area of intensely erythematous skin over which the epidermis had either become detached or could be detached by slight movement of the fingers. At the lower level of epidermal loosening there was a collection of apparently purulent fluid. The eruption, involving an area of about 150 cm², extended upwards on each side of the chest to include the axillary folds and the inner aspects of the upper arms as far as the antecubital fossae. The axillary apices were spared. Two smaller patches were present on the front of the neck. There were no mucosal lesions and the rest of the skin was clear.

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Progress was uneventful with bland treatment and he was discharged a week later. A nasal swab grew *Staphylococcus aureus*, phage type 55. Swabs from the throat and the purulent skin sac were sterile.

On the day after admission a fuller history was obtained from his parents who confirmed the absence of any preceding illness, medicines, or drugs. The boy had left for a weekend camping expedition with other members of a club on the Friday night. The clothes he carried in his knapsack had been packed in a polyethylene bag, separate from other camping equipment, which included a plastic one-litre bottle containing paraffin (kerosene). The first day passed without incident and the boy noticed the rash only on waking on the Sunday morning. He had slept in pyjamas over which he wore a heavy sweater, the weather being somewhat cold. It is probable that he had not replaced his clothes in the polyethylene bag on the Saturday morning and it is certain that they came in contact with the paraffin because the smell was noticed strongly on his clothes by his parents when they unpacked his bags.

A plastic container similar to the type used by him was half filled with paraffin and subjected to only a light shaking associated with an upright position on a car journey. After some minutes it was apparent that paraffin was leaking out from the sides of the cap, even though this had been reasonably firmly screwed on.

Comment

Both petrol and paraffin (kerosene) are known to produce skin lesions exactly simulating those of toxic epidermal necrolysis, and such cases have, in fact, been included in accounts of this disease (Lyell, 1967, Merien *et al.*, 1961, Walker, 1962). Kerosene fuel caused extensive lesions on the bodies recovered from the sea after the Comet aircraft disaster in 1954 (Armstrong *et al.*, 1955). The lesions are usually confined to areas covered by clothing and have well-demarcated edges. A similar picture occurred in a boy of 3 years whose head had been immersed in a pool of petrol (Ainsworth, 1960), and Aidin (1958) described the same changes in another boy killed in a vat of petrol. Absorption of petrol vapour from petrol lighters in aircraft causing similar burns on the thigh was the subject of correspondence in this journal over 20 years ago.

The present case is noteworthy, as it occurred in circumstances which are likely to be encountered more often. The combination of a plastic type of container (which is not ideal for carrying volatile liquids) and failure to keep the container separated from clothing in the knapsack were the cause of contamination. The wearing of a heavy pullover on top of the pyjamas obviously enhanced prolonged contact with the skin. The pyjama trousers were not so covered and, as they had been rolled up on top, were probably less contaminated.

It is not difficult to envisage that this combination of circumstances might occur again. We feel that careful instructions should be given by club leaders and medical officers concerned with camping on the correct packing of paraffin and similar fluids.

As this is a true chemical "burn" we have thought it best to acknowledge the similarity with Lyell's syndrome but to stress its separate identity by omission of the word "toxic", used by Lyell to describe the prodromal toxæmia occurring in his cases.

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Ventricular Arrhythmias Associated with Use of Thioridazine Hydrochloride in Alcohol Withdrawal

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Thioridazine hydrochloride (Melleril) is a widely used phenothiazine with a low incidence of extrapyramidal side effects (Kelly *et al.*, 1963). It is known to cause E.C.G. changes, which are sometimes reversed by potassium supplements (Kelly *et al.*, 1963; Alexander *et al.*, 1967). Deaths from ventricular tachycardia and fibrillation have been reported after large doses of thioridazine (Kelly, *et al.*, 1963; Alexander, *et al.*, 1967; Giles and Modlin, 1968). In the two cases reported ordinary doses of the drug in the context of alcohol withdrawal and hypokalaemia were associated with the development of ventricular fibrillation.

Case 4

A 41-year-old male alcoholic developed delirium tremens after alcohol withdrawal. He was given haloperidol 10 mg twice a day, procyclidine hydrochloride 10 mg twice a day, and thioridazine hydrochloride in doses of up to 600 mg daily. Two days later he collapsed and was found to be in ventricular fibrillation. He was defibrillated and transferred to this hospital. On arrival he was unconscious and areflexic, with sluggish pupils. His blood pressure was 110/70 mm Hg. Investigations showed a serum K⁺ of 2.6 mEq/l., with normal urea and other electrolytes. He was acidotic (pH 7.32) with a base deficit of 10 mEq/l. He continued to have numerous ventricular ectopic beats, with runs of ventricular tachycardia and fibrillation. He was given KCl in 5% dextrose intravenously, 10 mEq/hr, with intravenous lignocaine at 1 to 4 mg/min and defibrillated as necessary. After 48 hours the rhythm stabilized. The serum K⁺ was 4.7 mEq/l. and the infusion was stopped. Unfortunately he remained unconscious, and after four days an E.E.G. showed a "flat" recording. Necropsy showed terminal bronchopneumonia, a fatty liver, and an area of necrosis in the posterior myocardium, without any evidence of coronary artery occlusion.

Case 2

A 41-year-old woman was admitted to hospital for alcohol withdrawal. She was given thioridazine 600 mg daily. Two days later she collapsed with a tachycardia and an unrecordable blood pressure. She became cyanosed and semiconscious and was transferred to this hospital. On arrival she was noted to be in ventricular

tachycardia, which reverted to sinus rhythm after a D.C. shock of 100 joules. Her serum K⁺ on arrival was 2.3 mEq/l., and she was acidotic (pH 7.26) with a base deficit of 14.5 mEq/l. During the next two hours she had 12 episodes of ventricular fibrillation, which were reversed by D.C. shock. She received 50 mEq sodium bicarbonate intravenously, followed by an infusion of lignocaine at 2 mg/min. An injection of 10 mEq KCl in dextrose was given directly into the subclavian vein, followed by 60 mEq KCl in dextrose over the next four hours. At this time her condition had improved. Blood gases were now normal, but the serum K⁺ remained low at 2.7 mEq/l. The infusion of KCl was continued at 10 mEq/hr, and she was started on effervescent potassium chloride, four tablets four times a day. Nine further episodes of ventricular fibrillation were aborted by D.C. shocks of 100-200 joules. Twenty-four hours after admission she was in sinus rhythm. Serum K⁺ was 3.4 mEq/l. The E.C.G. showed prominent U waves, but no evidence of infarction. The patient recovered, and was discharged with a normal E.C.G., and a serum K⁺ of 4.6 mEq/l.

Comment

E.C.G. changes reported in patients taking more than 200 mg of thioridazine daily include abnormal Q-T intervals, U waves, and T wave changes (Kelly, *et al.*, 1963; Wendkoss, 1964; Huston and Bell, 1966; Alexander, *et al.*, 1967). Fatal ventricular fibrillation has occurred in patients taking over 2 g of the drug daily (Giles and Modlin, 1968; Shoonmaker *et al.*, 1966). Atrophy of the myocardium, with fragmentation of the cell cytoplasm, has been noted at necropsy in these cases and may be an effect specific to phenothiazines (Kelly, *et al.*, 1963; Giles and Modlin, 1968).

The cases reported here were treated with thioridazine to ameliorate withdrawal symptoms. Both patients developed ventricular fibrillation after modest doses of the drug, and both were found subsequently to be severely depleted of potassium. Hypokalaemia is a recognized complication of alcoholism and is probably due to a poor diet. Replacement by intravenous potassium chloride improved the arrhythmia in both cases. The area of myocardial necrosis, without any coronary artery occlusion, noted at necropsy in the first case may represent a specific phenothiazine-induced lesion.

It is important to identify situations in which the use of a normally safe drug may be hazardous. Thioridazine has found a place in the treatment of alcohol withdrawal. Some of these patients may be potassium depleted, and its use in this context is potentially dangerous. Determination of the serum K⁺, an E.C.G., and possibly potassium supplements should accompany the use of the drug in alcohol withdrawal.

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