REPORT ON THREE EXTENSIVE INDUSTRIAL CHEMICAL BURNS

BY

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Extensive chemical burns are fortunately rare, which speaks well for the safety precautions taken to protect personnel in industry. At the Birmingham Burns Unit, which serves a population of about four million, we have seen only three cases in the last five years.

But when chemical burns of the skin occur they are often disfiguring and disabling, and sometimes fatal. The risk to life depends on the amount of skin destruction, the degree of absorption, and the toxicity of the chemical to the kidneys and liver. The three cases reported here illustrate some of the clinical problems involved in diagnosis and management.

Case 1. Cresylic Acid Burn

Whilst at work a man aged 47 fell into a vat of "ardrox" (a cresylic acid derivative). His clothes were removed quickly and he was washed thoroughly under a shower before being transferred to hospital, where an intravenous infusion of 5% dextrose in saline was started. Six hours later he was transferred to the burns unit, where he was found to have burns amounting to 15% of the body surface (excluding erythema), involving the buttocks, genitalia, lower limbs, and forearms. The burns consisted of brown staining of the skin without blistering. All the areas were hypersensitive to pin-prick and the burns were apparently superficial partial skin loss. There were certainly at that time no areas of full-thickness skin destruction.

Three hours after admission he began to vomit, and oral fluids were stopped, these being replaced by 4.3% dextrose in 0.18% saline intravenously.

Thirty-six hours after the injury it became apparent that anuria had developed, and conservative measures comprising use of glucose, arachis oil, and a restricted fluid intake were

started (Bull *et al.*, 1949). This mixture was not tolerated, and 50% intravenous dextrose was substituted through a "polythene" catheter into the superior vena cava. Calcium gluconate, glucose, and insulin were used to control the serum potassium (Meroney, 1956), and, in view of the anuria, only a single dose of oxytetracycline 500 mg. was given to combat infection (Wilkinson, 1955). However, the blood urea rose steadily to 528 mg./100 ml. and the serum potassium to 9.3 mEq/l., and the patient died on the tenth day.

The patient's condition, apart from renal insufficiency, had appeared satisfactory until the seventh day, when he developed generalized rhonchi in both lungs, a pericardial rub, and precordial pain. The precordial pain disappeared on the ninth day. On the tenth day he became unconscious and developed congestive heart failure, dying within 18 hours of becoming comatose. During these last hours his heart failure was treated with intravenous digoxin and the acidosis with intravenous 1/6-molar sodium lactate. Electrocardiograms showed changes consistent with hyperkalaemia (Fig. 1), and, although a dramatic change towards normality occurred after he was given digoxin (Fig. 2), a fatal outcome was not prevented. Unfortunately, permission for a necropsy was not granted.

Case 2. Chromic Acid Burn

A man aged 47 slipped at work and plunged his arm into a vat of chromic acid. He received an extensive burn of the whole of the right upper limb. The burn was immediately washed with water, and 45 minutes after the accident he was admitted to the burns unit, where the arm was again generously washed in "buffer solution." This is a mixture of monobasic and dibasic phosphate which has the following composition: monobasic potassium phosphate (KH_2PO_4), 70 g., and dibasic sodium phosphate ($\text{Na}_2\text{HPO}_4 \cdot 12\text{H}_2\text{O}$), 180 g., in 850 ml. of water; this makes 1,000 ml. of solution (Bull and Jackson, 1952).

The area of the burn was 9% of the body surface. The skin of the arm and forearm was stained yellow, and the appearance and texture were consistent with a diagnosis of full-thickness destruction. The skin was also analgesic to pin-prick. In contrast, some skin on the hand and in the axilla was sensitive to pin-prick, and skin loss appeared to be partial only.

FIG. 1.—Case 1. E.C.G. leads I, II, and III when the serum potassium was 7.5 mEq/l.

FIG. 2.—Case 1. E.C.G. leads I, II, and III after intravenous digoxin.

Seventy-five minutes after the accident, and as quickly as could be arranged, the patient was anaesthetized and the dead skin of the arm and forearm excised. The skin and most of the subcutaneous fat were removed, leaving only the larger veins and some areolar tissue. In spite of this deep excision, many small thrombosed veins were clearly seen in the deep fascia at the first few dressings after operation—that is, the acid had already penetrated deeply into the fat before excision. The partial skin burn on the hand and in the axilla, amounting to a loss of 2%, was not excised.

It was not possible to graft the resulting raw area until about three weeks later owing to a surface layer of necrotic fatty tissue and small thrombosed veins, but after three weeks' treatment with penicillin cream (1,000 units/g.) the "clean" granulating area was successfully grafted in one operation. The patient, who was discharged from hospital 44 days after injury, developed no renal or liver damage (cf. Pascale *et al.*, 1952; Samitz, 1955), and no other toxic effects could be found.

Seventeen hours after injury his urine had a specific gravity of 1028, with a trace of protein. There was no sugar in it; the benzidine test for blood was negative, and there were no absorption bands on spectroscopy. Microscopy showed an occasional hyaline cast and red cell only.

Microscopical examination of the excised skin showed the epidermis to be absent and there was coagulative necrosis of most of the dermis, particularly the upper half or two-thirds involving dermal epithelium. In the deeper third of the dermis the dilated congested capillary network suggested capillary stasis. Here coagulation of the sweat coils had not occurred, but some showed evidence of early disintegrative necrosis.

Blood and urine examination four months after injury showed no evidence of renal or liver damage.

Case 3. Nickel Sulphate and Acid Burn

A man aged 62 slipped and fell into a vat of hot acid nickel-plating solution; he managed to climb out quickly, and first-aid at the factory consisted in application of sodium bicarbonate. The hot solution was a mixture of nickel chloride (8 oz. to a gallon; 50 g. to a litre), nickel sulphate (50 oz. to a gallon; 320 g. to a litre), boric acid (6 oz. to a gallon; 38 g. to a litre), and cumarin (5 lb./100 gallon; 500 g./100 l.). It was at a temperature of 178° F. (81° C.) and had a pH of 3.5. He was admitted to hospital 40 minutes after the accident and the burns were treated again with a "buffer solution" compress. The burn covered 40% of the body surface and had the appearance of only partial destruction of the skin.

Shock was treated by plasma transfusion, using serial haematocrits, and blood-volume estimations, using radioactive isotopes (³²P and ⁵¹Cr). His breathing on admission was rapid, deep, and sighing, and in view of the acidity of the solution into which he had fallen 450 ml. of 1/6 molar lactate solution was given intravenously, followed by plasma. The respiratory rate slowed with this treatment. The CO₂ combining power on admission was later reported as having been 22 mEq/l.

Nickel was detected in the urine and serum, and, in view of the possibility of toxic complications, dimercaprol was given (Gilman and Goodman, 1956).

Owing to vomiting within three hours of admission fluids could not be taken orally, and the fluid requirement had to be given intravenously through a polythene catheter into the inferior vena cava.

Urine output and osmolarity readings showed that the solute cleared by the kidney varied from 1,270 to 630 mEq a day until the fifth day, when the patient developed hypotension and pre-renal oliguria. Daily serum and urine electrolyte and urea measurements were taken, and deficiencies of sodium and potassium treated accordingly. Hyponatraemia occurring on the fifth and final day responded dramatically to intravenous 5% saline, an

unconscious and apparently moribund patient being rendered conscious and rational for a few hours at a time. Noradrenaline was tried as treatment for the hypotension of the fifth day. Though the blood pressure responded to a continuous graded intravenous administration, the urine output and general condition did not improve. He finally succumbed to pneumonia. Penicillin and tetracycline were used to combat infection. No nickel was found in the urine after the third day.

At necropsy, lobar pneumonia and oedema of the lung were the only significant findings apart from the burned area of skin, most of which, in contrast to its initial appearance, turned out to be mostly full-thickness skin loss.

Discussion

From the point of view of extent and depth, extensive chemical burns presumably carry the same risk to life as thermal injuries: sometimes, also, the burning agent is both hot and caustic and the burn is mixed. In either event the patient may die from shock, ischaemic renal failure, infection, or exhaustion. Provided the toxic chemical is not absorbed sufficiently to damage vital processes the burn is a surface lesion only, and as soon as it is neutralized therapeutically or by tissue protein it ceases to differ appreciably from a similar thermal injury.

The peculiar risks of some extensive chemical burns, however, depend upon the absorption and toxicity of the chemical, which may damage the kidneys or liver enough to be fatal apart from the local skin lesion.

Measures to prevent absorption of the chemical or to remove it fall into four groups—namely, washing from the surface, neutralization, removal of the tissue reservoir, and facilitation of detoxication or excretion.

Immediate Removal of Clothing and Washing

These are the most effective first-aid measures, done under a shower if necessary, and good washing facilities should always be available when men work in proximity to toxic chemicals. The immediate removal of contaminated clothing cannot be unduly stressed, as fatal absorption of chemical may occur very rapidly. A case has been described where a man did not remove his clothing contaminated with nitrobenzene: he died within an hour, and his blood was "like chocolate" owing to methaemoglobinaemia (Hamilton, 1919). Men who have neglected to remove clothing contaminated with methyl alcohol have been blinded (Hunter, 1957).

Heat is evolved when concentrated sulphuric acid is mixed with water and may produce a thermal burn of the skin; in such a case the acid should be washed off first with anhydrous ethyl alcohol (Hunter, 1957). Washing off is made easier sometimes by adding another chemical, as in the case of trinitrophenylmethyl nitramine (tetryl): the hands should be washed in running water with the addition of 5% sodium sulphite to convert it into a water-soluble substance (Silver, 1938).

Neutralization of the Chemical

In different circumstances this can be applied on, into, or under the skin, and it may be done by local or systemic application. Local treatment may be carried out by applying the antidote on the surface of the skin or by subcutaneous injection—for example, compresses of monobasic and dibasic phosphate buffer solution may be applied to the skin surface after washing for acid or alkali burns. Hydrofluoric acid burns may be treated by surface application of magnesium oxide paste and subcutaneous injection of 10% calcium gluconate,

magnesium, and calcium fluoride, being relatively harmless and insoluble (Jones, 1939). Again, oxidation by means of local application of sodium hypochlorite is one of the ways of neutralizing aromatic arsines.

An example of systemic administration of an antidote is the use of atropine for organic phosphorus insecticides. Here atropine is used parenterally because these insecticides are powerful cholinesterase inhibitors.

Surgical Removal of Toxic Chemical in Tissues

This means excision of skin and fat in the case of chemical burns, to remove the reservoir of chemical within the body (Case 2).

The management of a burn with a toxic chemical is simplified if the case is suitable for surgical excision. Washing, neutralization, and excision can then all be completed with the greatest speed. If the burn is on the limbs there is probably a place for the immediate application of a tourniquet until after the burn is excised, but this should be done under medical supervision. Copious fluids, unless and until there is evidence of oliguria due to renal damage, are easy to provide, orally or intravenously. Where the site of the burn excludes the use of a tourniquet, the operation blood loss, and consequent transfusion, may even result in some benefit from part exchange of the blood volume.

Surgical excision is eminently suitable for full-thickness burns, but a difficult problem is presented by burns of doubtful depth. Should they be excised or not? On the one hand, excision may be the patient's only chance of survival; on the other hand, it costs him an operation, a large permanent scar, a longer healing-time—and all these may be unnecessary if the dose of chemical which could be absorbed is sublethal. The difficulty is not a technical one: it is not very hard to excise a large area of skin and subcutaneous tissue provided enough blood is available (Topley and Jackson, 1957). The problem turns on our inability to know if there is a lethal dose of chemical in the burned skin, and if it will be, or has already been, absorbed.

In some cases—for example, poisoning by the aromatic nitro- and amino-derivatives—spectrophotometric estimation of the concentration of methaemoglobin will indicate the severity of the poisoning. Heinz bodies, punctate basophilia, and polychromatophilia are valuable early signs of poisoning by dinitrobenzene and aniline. Coproporphyrinuria is an early sign in trinitrotoluene poisoning, and may be fifty times greater than normal in a fatal case (Hunter, 1957). Even in these cases, where it is possible to investigate poisoning with simple tests, the surgeon still never knows whether there is a further risk from the chemical remaining in the skin.

To be at all confident of making the right decision in these circumstances, the surgeon needs to know whether previous similar patients with burns of that size, caused by that strength of that particular chemical, lived or died. These cases are rare, and reports of them are virtually non-existent in medical literature.

The diagnosis of the depth of burning is always difficult, and this difficulty is increased in burns with some chemicals such as phenol (Jackson, 1953). But sensitivity to pin-prick always indicates partial-thickness skin loss at the time of examination, although this may later become whole skin loss owing to the action of the chemical (Case 3). Analgesia to pin-prick does not necessarily imply full-thickness loss.

Facilitation of Detoxication or Excretion of Toxic Chemical

If a large dose of the chemical is absorbed in spite of these measures, survival will depend largely upon increasing its excretion in a copious dilute urine. Possibly, exchange transfusions or the artificial kidney may be useful for this purpose, but we have no experience of this form of treatment.

Dimercaprol has been found extremely useful in treating intoxication by arsenical, mercurial, cadmium, and heavy metals drugs. It destroys the complexes formed between the metal and body proteins and increases the rate of excretion of the metal in its detoxified form (Gilman and Goodman, 1955). In Case 3 the dimercaprol probably protected the enzyme systems against any ill effect of nickel (Gilman and Goodman, 1955) and also produced a beneficial diuresis ("acetazolamide effect"), incidentally increasing sodium excretion at a time when a burned patient would be expected to have sodium retention.

Summary and Conclusions

Three cases of extensive chemical burns are presented.

The various methods of treating these burns are surveyed.

The problems involved in the diagnosis of chemical burns requiring surgical excision are discussed.

Publication of short histories of such cases, treated successfully and unsuccessfully, with details of the solution, burn, timing, treatment, and outcome, would be the first step towards more rational management. Meanwhile one must bear in mind the potentially serious nature of these injuries and be correspondingly bold and thorough in efforts to save life.

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"Many sick people need no more than medical care to enable them to return to their old way of life. But many live among difficulties whose complications cannot be escaped easily, and for them effective help in illness may require the efforts of several professions working together. The almoner's particular sphere of knowledge is largely that common to caseworkers, of human responses in the face of difficulty and pressure, and of ways of supporting a man's individuality and integrity against odds. She must in addition have special understanding of the personal effects of illness and of the associations between social problems and illness; and she must appreciate the place and the responsibilities of social work in the medical world and its relationship with social service outside."—*Annual Report of the Institute of Almoners, 1957-58.*