

Skin Trauma in Patients Receiving Systemic Corticosteroid Therapy

DAVID JOHN DAVID

British Medical Journal, 1972, 2, 614-616

Summary

Surprisingly extensive skin damage may result from trivial accidents in patients treated with systemic corticosteroids. In one year 21 such patients were admitted to a plastic surgical unit. Skin necrosis and delayed healing are problems in these patients, and skin grafting may be necessary.

Introduction

Of the many side effects of treatment with corticosteroids the least well documented is the major skin damage which can result from minor knocks (Rook *et al.*, 1968). The present study examines the nature of the lesions produced against the background of the underlying disease for which the corticosteroid therapy was administered and highlights the morbidity that results. As these lesions are notoriously difficult to assess and treat the principles of surgical management are outlined.

Patients and Analysis

Seventeen patients with this complication were admitted to the South-Western Regional Plastic Surgical Unit at Frenchay Hospital during the 12 months from 1 February 1971 to 31 January 1972 (see Table). They accounted for a total of 21 admissions, four people being admitted on two separate occasions. The total inpatient stay was 298 days with a mean duration of 14 days for each admission. Four of these patients had a total of eight additional admissions for the same reason before February 1971.

In every case the injuring force was such as would not be expected to damage normal skin seriously—for example, in Case 4 the patient knocked her leg against a cardboard box and raised a large flap of non-viable skin and subcutaneous tissue from the pretibial region. Without exception each patient had a

lesion of the lower limb below the thigh. In addition Cases 6 and 14 degloved the dorsum of a hand. The commonest site was the pretibial region of the leg (15 admissions). Other sites were the knee (three admissions), the dorsum of the foot (two admissions), and the calf (one admission). It is notable that in every case the injury was sustained at home.

The diseases for which the systemic corticosteroid therapy was administered were asthma (nine cases), rheumatoid arthritis (seven cases), and disseminated lupus erythematosus (one case).

Ten patients were in the seventh and eighth decades of life, six patients were in the fifth and sixth decades, and one was in the first decade. There were only three males in the group studied. With one exception all cases had been treated with systemic corticosteroids for at least five years. Case 5 had had only a four-month course of therapy. Every patient had at least one other "Cushingoid" feature in addition to skin atrophy.

Diagnosis and Treatment

The history of a trivial injuring force is characteristic—for example, knocking against furniture, tripping on steps, or being jumped on by a dog. The possibility of corticosteroid therapy should be sought in people with this type of soft-tissue injury. The associated history or clinical appearance of rheumatoid arthritis or asthma confirms the disease complex. The physical findings of the moonface, obesity, and hypertension are often associated with skin which looks and feels atrophic.

Assessment of the amount of skin loss and the viability of flaps was often found to be difficult. Underestimation of the amount of skin damage was particularly common. The leg lesions usually consisted of lacerations which raised one or more flaps of skin and subcutaneous tissue from the deep fascia (Fig. 1). Haematoma usually extended around the laceration, dissecting in a plane deep to the atrophic subcutaneous fat and devitalizing a much wider area than was initially apparent (Fig. 2).

Local anaesthesia was preferred in the older, sicker patients with small lesions. Plain lignocaine was used before debridement of the lesion to avoid the further tissue anoxia caused by adrenaline and to make assessment of tissue vascularization easier (Grabb and Smith, 1968). Infiltration with adrenaline was used when taking the split skin graft to prevent rapid absorption of the local anaesthetic agent. Where local anaesthesia was impracticable (nine cases), general anaesthesia was used with

South-Western Regional Plastic and Jaw Unit, Frenchay Hospital, Bristol

DAVID JOHN DAVID, M.B., B.S., F.R.C.S., Senior Registrar

Details of Patients Admitted with Skin Damage 1 February 1971 to 31 January 1972

Case No.	Age and Sex	Disease	Steroid Dosage (Duration)	Days in Hospital (No. of Admissions)
1	48 F.	Asthma	Prednisolone 5 mg twice daily (8 years)	27 (2)
2	61 F.	Rheumatoid arthritis	Betamethasone 0.5 mg twice daily (6 years)	16 (1)
3	42 F.	Asthma	Prednisolone 5 mg twice daily, corticotrophin 40 mg 2 weekly (7 years)	7 (2)
4	52 F.	Asthma	Prednisolone 5 mg/day (10 years)	44 (2)
5	9 M.	Asthma	Prednisolone 5 mg twice daily (4 months)	3 (1)
6	50 M.	Asthma, psoriasis	Triamcinolone 4 mg three times daily, betamethasone cream (5 years)	21 (2)
7	62 F.	Rheumatoid arthritis	Methylprednisolone acetate 40 mg daily (6 years)	20 (1)
8	74 F.	Rheumatoid arthritis	Prednisolone 5 mg twice daily (5 years)	13 (1)
9	71 F.	Asthma	Prednisolone 5 mg twice daily (15 years)	14 (1)
10	72 F.	Asthma	Prednisolone 5 mg/day (8 years)	17 (1)
11	44 F.	Rheumatoid arthritis	Prednisolone 5 mg/day (20 years)	2 (1)
12	78 F.	Rheumatoid arthritis	Triamcinolone 4 mg/day (10 years)	32 (1)
13	60 F.	Asthma	Dexamethasone 0.5 mg twice daily (11 years)	2 (1)
14	68 F.	Disseminated lupus erythematosus	Prednisolone 10 mg four times daily (12 years)	21 (1)
15	53 F.	Rheumatoid arthritis	Methylprednisolone 4 mg/day, corticotrophin 40 mg/month (5 years)	17 (1)
16	72 M.	Asthma	Paramethasone 2 mg twice daily (7 years)	23 (1)
17	63 F.	Rheumatoid arthritis	Prednisolone 7.5 mg/day, methylprednisolone 4 mg/day, corticotrophin 40 mg 2 weekly (7 years)	19 (1)

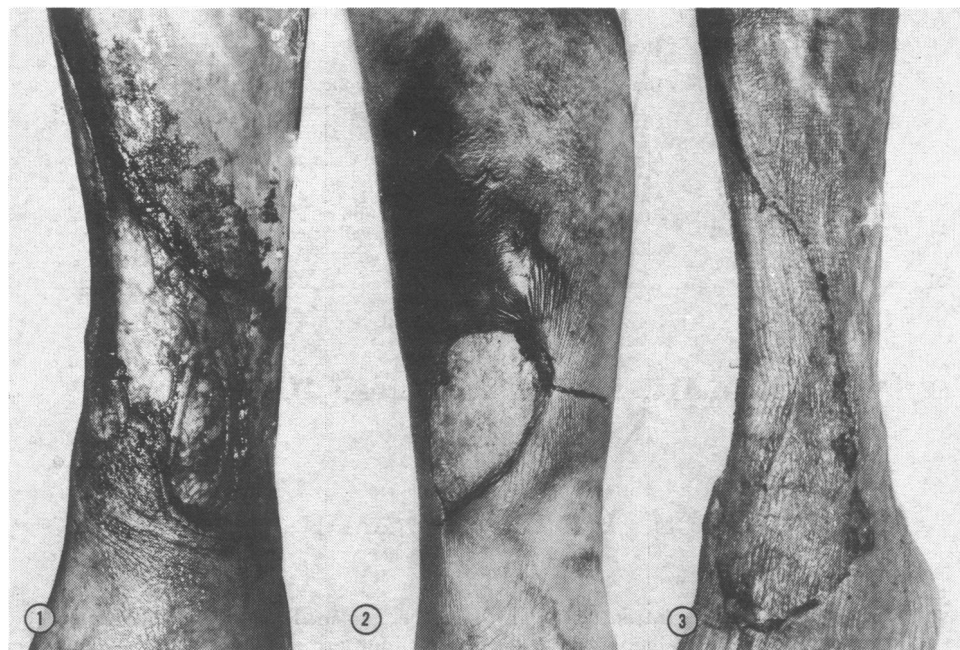


FIG. 1—Case 14. Typical laceration of leg with non-viable flaps of skin and subcutaneous tissue raised from deep fascia.

FIG. 2—Case 12. Lower part of wound has been treated, but extending above it is a haematoma with overlying dead soft tissue.

FIG. 3—Case 14. Appearances after debridement and grafting.

corticosteroid supplements to cover the procedure and post-operative period (seven cases) or left at maintenance levels without temporary increments (two cases).

It is essential for debridement to be thorough, the best criterion of viability being arteriolar bleeding. All haematoma and surrounding grey devitalized fat must be removed. If there has been delay in treatment cleaning must be even more thorough. Attempts to return the whole of a flap to its bed under tension should be resisted. Obviously non-viable and doubtfully-viable skin flaps should be excised. A healthy bed is prepared for grafting by complete excision of all devitalized fat leaving only vascularized soft tissue.

Split skin grafts are taken from the medial side of the thigh. This is often found to be technically difficult because of the thin atrophic skin. Great care has to be exercised to cut the graft thinly so as to avoid creating as big a lesion at the donor site as the one being treated.

The technique of delayed application of skin grafts (Matthews, 1945) is advised (1) because it reduces the time the patient spends on the operating table and (2) because it gives time for the wound to stop bleeding and hence a better chance of graft "take."

Discussion

The atrophic skin seen in people receiving systemic and local corticosteroid therapy has been investigated by Mancini *et al.* (1960). They showed that systemic corticotrophin, cortisone, and prednisolone and topical hydrocortisone produced progressive atrophy of the collagen bundles and fibres, which lose their normal cross-connexions and appear thin and dissociated. The reticular and elastic fibres look thin and fragmented. Many of the fibroblasts lose their extensions. The basement membrane appears thin and discontinuous. Local application tends to affect the more superficial parts of the skin, whereas systemic therapy affects the deeper parts of the corium.

Local corticosteroids had been used only in Case 6, and then in conjunction with systemic therapy. It is of interest that in spite of the wide use of topical corticosteroids no example of

skin trauma occurred where skin atrophy was a result of topical therapy alone. With this type of therapy the atrophy is severe only in the flexures or on the face, and these areas are less exposed to trauma than the legs and hands.

All the patients in this series were emergency admissions from the Bristol area. None was seen from the more distant parts of the catchment area. It appears, however, that all the major centres in the South-Western Region are seeing this problem.

The liberal prescribing of corticosteroids for these conditions was found to be unjustified for several patients. Two patients (Cases 2 and 12) with rheumatoid arthritis had burnt-out disease for more than two years and were successfully weaned from steroid therapy while in hospital. In two of the asthmatic patients (Cases 3 and 13) dosage was in excess of that required to control their disease.

Patients on long-term corticosteroid treatment are at great risk of serious injury from small accidents. As skin structure rapidly returns to normal after discontinuance of steroid therapy no patient was seen with skin trauma who was not currently taking corticosteroids. Housewives especially may suffer many injuries for which long periods in hospital may be needed, repeated anaesthetics given, painful graft donor areas created, and prolonged aftercare required. One patient (Case 4) had five admissions, required five operations, and had spent 119 days in hospital since starting corticosteroid therapy 10 years previously.

Attempts to inset flaps totally and to sew up lacerations under tension in patients receiving systemic corticosteroids should be avoided. It almost always results in skin necrosis, delayed slough separation, and a greatly prolonged healing time. The rate of healing is not obviously affected when treatment is adequate although the hypertrophic phase of scar production is suppressed by the corticosteroid therapy (Griffith, 1966). Because of this suppression and the fact that contour defects from injury are not very great in legs with atrophic skin and subcutaneous tissue, the cosmetic results after grafting are good (Fig. 3).

Length of treatment appears to be more important than total daily dosage in producing these problems. This is probably due to increased time exposure to the chance of injury rather than an increase in soft-tissue morbidity.

Conclusion

Skin trauma is a notable complication of systemic corticosteroid therapy and further threatens the ideal that the doctor should "at least do no harm" by his treatment.

I wish to thank Mr. D. C. Bodenham, Mr. R. T. Routledge, Mr. R. W. Hiles, and Mr. R. W. Pigott for access to their patients and their advice; Mr. J. Lendrum for reading the manuscript;

Mr. L. Banham for his photography; and Mrs. S. Bassett for her clerical assistance.

References

- Grabb, W. C., and Smith, J. W. (editors) (1968). *Plastic Surgery, A Concise Guide to Clinical Practice*. Boston, Little, Brown.
 Griffith, B. H. (1966). *Plastic and Reconstructive Surgery and the Transplantation Bulletin*, 38, 202.
 Mancini, R. E., Stringa, S. G., and Canepa, L. (1960). *Journal of Investigative Dermatology*, 34, 393.
 Matthews, D. N. (1945). *Lancet*, 1, 775.
 Rook, A., Wilkinson, D. S., and Ebling, F. J. G. (1968). *Textbook of Dermatology*, vol. 2. Oxford, Blackwell Scientific.

Gastroenteritis with Necrotizing Enterocolitis in Premature Babies

HARRY STEIN, JOHN BECK, ALBERT SOLOMON, ARTHUR SCHMAMAN

British Medical Journal, 1972, 2, 616-619

Summary

Eleven premature babies developed necrotizing enterocolitis in an epidemic of gastroenteritis and salmonella infection. This occurred in one of two premature baby wards over a period of 10 weeks. All affected babies had severe gastroenteritis and six had salmonella infection. No cases of necrotizing enterocolitis occurred in the unaffected ward during the same period. All other possible factors which might have predisposed to necrotizing enterocolitis occurred with equal frequency in both wards.

There seems little doubt that infection was the significant factor in the pathogenesis.

Introduction

Necrotizing enterocolitis in neonates has aroused great interest in the past decade. Its increasing frequency is thought to be due to the fact that many neonates, particularly premature babies, who would previously have succumbed are now surviving (Fetterman, 1971). An association with exchange transfusion is well described (Castor, 1968; Corkery *et al.*, 1968; Orme and Eades, 1968; Beck *et al.*, 1971). For the rest a multiplicity of associations occurs. It seems to be more common in premature babies (Rossier, *et al.*, 1959; Berdon *et al.*, 1964; Mizrahi *et al.*, 1965) and particularly when they have suffered from asphyxia, apnoeic attacks, hyaline membrane disease, or hypothermia. These stress conditions may give rise to intestinal ischaemia with devitalization, infection, and necrosis (Lloyd, 1969; Stevenson *et al.*, 1969; Fetterman, 1971). Stasis of intestinal contents seems important in some cases. To many authors the role of infection seems paramount in the pathogenesis (Blanc, 1952; Waldhausen *et al.*, 1963; Mizrahi *et al.*, 1965). This has been thought to act by promoting a Schwartzman reaction (Rossier *et al.*, 1959; Hermann, 1965). The evidence for infection is somewhat tenuous, and local infection and particularly infective diarrhoea are considered rare (*British*

Medical Journal, 1970; Fetterman, 1971). In fact, though blood-staining of stools is a common presenting feature there has been very little evidence of a true preceding gastroenteritis and none of salmonella infection.

We report 11 cases in premature babies all associated with frank diarrhoea, and in six of these salmonellae were cultured from the stools. Two of the cases had exchange transfusions.

Subjects

During a 10-week period in the spring of 1971 an epidemic of gastroenteritis with a high incidence of salmonella cultures, obtained from the stools, occurred in one of two identical premature infant wards at Baragwanath Hospital. This necessitated the closing down of the infected ward. In all, 63 babies had diarrhoea, 22 of whom were infected with either *Salmonella thompson* or *Salmonella johannesburg*. Eleven cases of necrotizing enterocolitis occurred in this period; all had diarrhoea and six had proved salmonella infection. During the same period there were no cases at all of necrotizing enterocolitis in the uninfected ward. The babies admitted to the latter ward were identical in all other respects in terms of weight, pregnancy, birth history, and neonatal state to those in the infected ward. The only variable, in fact, was the great discrepancy in the rate of infection.

All babies were Bantu (Negro). Seven were males and four females. All were premature with birth weights ranging from 1,050 to 1,900 g. Nine of the pregnancies were normal, one mother had hypertension, and another was syphilitic. Antenatal care varied a great deal, being adequate in six cases and poor or non-existent in five (Table I). Four babies were born in hospital and the remaining seven "on district"; 10 were vertex presentations and one was breech.

TABLE I—Relevant Aspects of Pregnancy and Delivery

Case No.	Sex	Antenatal Care	Place of Birth (Hospital or "On District")
1	M.	Yes	District
2	M.	Yes	Hospital
3	M.	Yes	Hospital
4	M.	No	District
5	M.	No	District
6	F.	Yes	District
7	M.	Yes	Hospital
8	F.	No	District
9	F.	No	District
10	M.	No	Hospital
11	F.	Yes	District

Baragwanath Hospital and University of the Witwatersrand and the South African Institute for Medical Research, Johannesburg

HARRY STEIN, M.B., F.R.C.P., Paediatrician

JOHN BECK, M.B., F.R.C.S., Paediatric Surgeon

ALBERT SOLOMON, M.B., M.MED.RAD., Radiologist

ARTHUR SCHMAMAN, M.B., M.R.C.PATH., Pathologist