British Journal of Plastic Surgery (1980), **33**, 251–255 0007-1226/80/00550251$02.00

SKIN LOSS IN MENINGOCOCCAL SEPTICAEMIA

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Meningococcal septicaemia continues to be an exceptionally severe illness with a very high mortality. Children surviving this disease often present extensive necrosis of the soft tissues which can present major problems of skin cover in the acute phase and in the reconstruction of the residual deformities.

We wish to present our experiences in the treatment of children with skin loss following meningococcal septicaemia and to show how our plan of management of these cases has evolved.

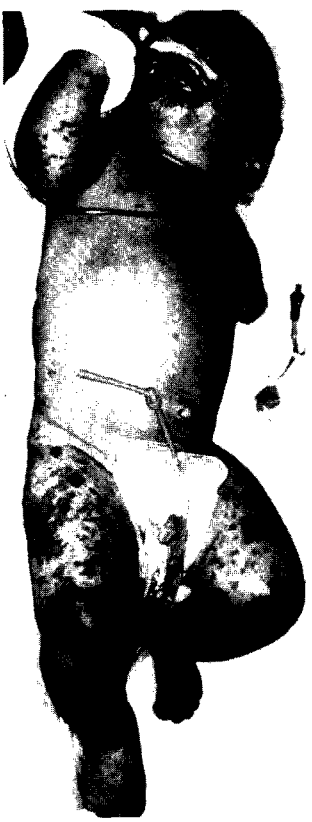

FIG. 1. Extensive purpura and ecchymoses developing rapidly within hours of the onset of the disease and showing a characteristic peripheral distribution.

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During the period January to June 1977, 15 severely ill children with meningococcal septicaemia were admitted to the Respiratory Intensive Care Unit at the Red Cross War Memorial Children's Hospital in Cape Town. Of these 15 children, four were referred to the Plastic Surgical Service and one other child was referred from the Infectious Diseases Hospital in the city, to give a total of 5 children with skin loss following meningococcal septicaemia.

Clinical Observations

Age of Child. There was a wide variation in ages ranging from 5 months to 6 years.

Types of Skin lesions. Fourteen of the children developed in a matter of hours, extensive purpura and ecchymoses in the skin (Fig. 1). These lesions invariably became more extensive in size and depth. One child presented with petechial spots only and these lesions did not progress.

Mortality. Of the 15 children, 8 died—a mortality of the order of 50 per cent.

Outcome in the survivors. In three of the remaining 7 survivors, spontaneous healing occurred following separation of the necrotic tissues. Four children developed large skin defects and were referred to the Plastic Surgical Service for further treatment after recovery from the acute stage of the systemic illness.

Observations on the Five Children with Extensive Skin Loss

Site of skin lesions. In all 5 children the areas that required grafting usually involved the limbs (especially the lower limbs) and the buttocks (Fig. 1).

Initial wound care. In 4 children the sloughing tissues were deliberately excised as soon as possible after recovery from the acute phase of the illness. In one child the sloughs were allowed to separate spontaneously before referral to the Plastic Surgical Unit.

Amputation. One child required partial amputation of a big toe.

Primary graft "take" and wound healing. In only one child did the skin graft take successfully at the first operation. In the remaining four children repeated grafting procedures were required (Fig. 2). In all the children spontaneous healing occurred to a greater or lesser degree and without exception produced unsightly scars (Fig. 3). The inability to achieve successful primary skin graft cover has occurred in 4 more children subsequently managed in this unit since June 1977.

Discussion

Long before the isolation of the causative organism (Neisseria Meningitidis), it was well recognised that the skin could be seriously affected: indeed another term for the disease was spotted fever as most patients had haemorrhagic spots on the skin. The skin lesions may be either petechial; maculo-papular; or purpuric and ecchymotic in nature. According to Warren *et al.* (1974) only 14 per cent of patients with meningococcal infections are reported to have no skin lesions.

FIG. 2. Appearance of wound after failure of the first split skin graft. The wound presents a healing edge and a healthy base.

FIG. 3. An example of healed wounds to show the unsightly scars with loss of contour of the limbs, due to destruction of the subcutaneous and superficial fatty tissue.

Although it is estimated that only 1 : 10,000 infections by Neisseria Meningitidis develops into a significant illness, (Godl, 1972) when severe acute meningococcal infection does occur one of the two well defined clinical patterns appears. In the majority following a preceding illness, clinical meningitis occurs without any manifestation of disseminated intravascular coagulation or endotoxic shock. In these cases, with appropriate treatment, the mortality is reported to be less than 10 per cent. In a minority of cases the clinical picture is that of an overwhelming septicaemia with endotoxic shock and disseminated intravascular coagulation. Despite the most intensive care available the mortality of this group of patients is high, varying from 40-80 per cent.

A common feature in meningococcal septicaemia is rapid progression of the skin lesions from petechial spots to large purpuric areas and ecchymoses. This pattern of progression was seen in all but one of the 15 children reported in this series. Warren *et al.* (1974) believe that the extent and nature of the skin lesions can be used as a prognostic index. This and other aspects of the disease are at present the object of a critical investigation at this hospital by the physicians involved.

In meningococcal septicaemia, the basic pathological effect upon the skin is diffuse vascular damage, the exact nature of which is not yet clear (Sotto *et al.*, 1976). Evans *et al.* (1969) believed that favourable growth conditions in the skin led to rapid bacterial

multiplication causing vasculitis, thrombosis and haemorrhage by direct bacterial invasion, possibly supplemented by endotoxin.

Davis and Arnold (1974) investigated the problem of whether the meningococcal endotoxin has greater skin potency than other endotoxins. Comparison of the biological potencies of endotoxins from several pathogenic organisms has shown that the endotoxin from Neisseria Meningitidis is unique in its effect upon the skin and must be regarded as responsible for the cutaneous skin lesions in meningococcal septicaemia (Warren *et al.*, 1974).

Whether the mode of action is due to a local Schwartzman reaction as described by Sotto *et al.* (1976) or due to hypersensitivity as postulated by Whittle *et al.* (1973) the end result is a specific injury of the vessels of the skin by the meningococcal endotoxin producing oedema, capillary thrombosis with extravasation of blood into the extravascular tissues and subsequent patchy necrosis. The extent of the skin damage would appear to be related to the severity of the infection and the amount of endotoxin present.

In the light of this knowledge, we believe that these necrotic skin areas should be excised as soon as the children recover from their systemic illness to prevent and or control secondary infection. Unless the organisms beneath the eschar are known and their sensitivity to antibiotics established, immediate skin grafting at the time of slough excision is probably unwise. Furthermore the base of the wound, in our experience, is usually unsatisfactory for primary grafting. Following adequate bacteriological control and after allowing a good layer of granulation tissue to appear, we have successfully undertaken split skin grafting in the 5 children recently referred to us.

We initially believed that the explanation for our poor skin grafting results was due to the basic pathology of this disease and in 3 of these children, biopsies taken from the base of the wound were examined histologically, but no difference in the structure of this granulation tissue was observed, when compared to granulation tissue removed from other wounds such as burns.

With the probable elimination of "inadequate" granulation tissue as the cause of the skin graft failure, two other factors may be contributory. Firstly accurate bacteriological control of these wounds is necessary and quantitative assessment of their bacterial flora is recommended before skin grafting is undertaken. Secondly, it is possible that following the debilitating nature of this disease there may be a deficiency of a so-far unidentified substance necessary for skin-graft success.

Despite the likelihood of a poor "take" of primary skin grafts, this procedure is not necessarily contraindicated. O'Donaghue and Zarem, (1971) have reported that autogenous, and to a lesser degree lyophilised skin grafts can stimulate vascularisation of a wound bed. Nathan *et al.* (1973) have also shown that skin grafts are leucotaxic and a major reduction in bacterial colonisation of a burn wound in rats occurs within 24 hours of skin grafting. We thus advocate early skin grafting under bacteriological control accepting that multiple grafting procedures may be necessary. The initial use of homograft or xenografts is an attractive alternative in preparation for final autografting.

Although we have not had cause to use more complicated methods of skin closure such as skin flaps in the early stages of convalescence, we believe that extreme caution should be exercised, in view of the basic pathophysiology of the skin lesions.

Once healing was finally achieved, severe residual scarring has been noted in all our children. Scar revisions and surgical repair of the deformities can be a major problem, an observation that was made in the paper by Gaze and Murray (1976).

We wish to thank Dr Max Klein and his staff of the Respiratory Intensive Care Unit for their help and co-operation: Dr D. Beattie for his advice on the immunological aspects of this disease and Dr D. Diedtrich for the histological studies.

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