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# Loss of split thickness skin grafts due to non-group A β-haemolytic streptococci

GEOFFREY R WILSON MB FRCS
*Registrar*
GORDON W FRENCH MB ChB
*Senior House Officer*
LANCE SULLY MB FRCS
*Consultant*
*Burns Unit, City Hospital, Nottingham*

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**Summary**

*Over a 17-month period 77 patients requiring a split skin graft for a burn injury have suffered loss of previously well taken graft due to the growth of a β-haemolytic streptococcus. Of these only 42 were streptococci of Lancefield group A (*Streptococcus pyogenes*); 16 were group B, 3 group C and 16 group G. Some strains of groups B, C and G produce cytopathic and spreading factors capable of destroying the new skin graft and regenerating epithelium. We suggest that the non-group A streptococci may be more pathogenic than previously recognised in this particular respect.*

## Introduction

It has been accepted for many years that burn wounds are frequently colonised by haemolytic streptococci *(1)*. The α- and non-haemolytic, as well as the β-haemolytic, are common in burns *(2)*, but the α- and non-haemolytic strains are not associated with any clinical problems.

In 1933 Lancefield *(3)* classified the β-haemolytic streptococci according to the polysaccharide antigen found in their cell wall. Various of these Lancefield groups are pathogenic to man and other mammals. Those particularly recognised as human pathogens are groups A, B, C, D and G *(4)*.

It has long been recognised that the group A β-haemolytic streptococcus (*Streptococcus pyogenes*) is very destructive to healing epithelium and the new skin graft, and it is recommended that a grafting procedure is not performed in the presence of a positive wound swab *(1,5)*.

We have failed to find any reference to skin graft loss from β-haemolytic streptococci other than group A. Jackson *et al. (6)* stated that "there is no evidence that streptococci of Lancefield groups C and G contribute to graft failure".

Most streptococcal wound infections are associated with carriage in the nasopharynx of the patient *(7)*, so that part of the management of a positive wound swab is to isolate him or her from other patients. This is distressing for the patient and relatives, and costly, both in terms of nursing time and equipment. The Nottingham Burns Unit dresses all but facial burns as a general policy. The patients are not isolated on admission. Hands, ears and obviously full thickness burns are dressed with silver sulphadiazine, and all other burns with vaseline gauze. Prophylactic antibiotics are not used. Patients with a positive swab for a β-haemolytic streptococcus are treated by isolation, oral phenoxymethylpenicillin and flucloxacillin, with povidone-iodine to the wound.

This study reports a number of cases where non-group A streptococci have caused clinical problems, and discusses which factors there are in common between Lancefield groups A, B, C and G that might be responsible for wound problems.

## Results

During the period 1 November 1985 to 31 March 1987 the Nottingham Burns Unit had 472 inpatient admissions and 232 patients attending outpatients only. There were 77 cases where a positive swab for a β-haemolytic

TABLE I *For the period 1 November 1985 to 31 March 1987*

| | | |
|---|---|---|
| Total inpatient admissions | 472 | |
| Outpatient attendance only | 232 | |
| Positive swab for β-haemolytic streptococcus with loss of skin graft ($n$=77) | | |
| Lancefield group A | 42 | (55%) |
| group B | 16 | (20%) |
| group C | 3 | (5%) |
| group G | 16 | (20%) |

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Correspondence to: G R Wilson MB FRCS, Plastic Surgery Unit, City Hospital, Hucknall Road, Nottingham NG5 1PB

streptococcus was linked with loss of skin graft. The clinical course was very characteristic. A skin graft that was taking well (ie pink and adherent), would at subsequent dressings show increasing areas of epithelial loss. The wound bases were a bright, haemorrhagic red, with a glareous sheen. Sometimes there was a peripheral erythema to the grafted area. Occasionally this appearance was seen at the first dressing of a graft that would have been expected to have taken well. The wound was then slow to heal and on occasions needed regrafting.

Of the 77 cases, 42 were Lancefield group A, 16 group B, 3 group C and 16 group G (Table I).

A questionnaire was circulated to all the burns units in the UK to see if any had had problems with non-group A streptococci. Of the 19 replies 7 had noticed loss of skin graft and/or delayed healing, particularly with groups C and G (Table II).

During the period of study, two patients have been referred to the plastic surgery department from other departments in the hospital with skin loss on the lower limb following trauma and subsequent cellulitis. A streptococcus of group G was cultured from the blood and the wound of one patient, and a group C streptococcus from the wound of the other patient.

## Discussion

Can we be confident that the organism cultured from the wound swab equals infection and is not just colonisation? Confirmatory biopsies were not performed as Lawrence *(8)* has shown that quantitative methods of bacterial isolation from a burn wound that has been dressed gives no more useful information than surface swabbing. Jones *et al. (9)* found that surface swabbing of leg ulcers in diabetics yielded the same bacteria as swabs taken from the ulcer base when any slough and exudate had been removed.

TABLE II *Results of questionnaire sent to burns units in the UK*

| *Burns unit* | *No. of admissions/ annum* | *Dress* | *Surgery* | *Prophylactic antibiotics* | *Non-group A streptococcus* | *Groups* |
|---|---|---|---|---|---|---|
| 1 | 120 | Y | E | N | N | — |
| 2 | 350 | Y | E | Y (flu) | N | — |
| 3 | 155 | Y | E | N | N | — |
| 4 | 200 | Y | E/DE/L | N | N | — |
| 5 | 120 | Y | E | Y (pen) | N | — |
| 6 | 120 | Y/N | DE/L | N | N | — |
| 7 | 70 | Y | DE/L | N | N | — |
| 8 | 470 | Y | E/DE/L | Y (pen) | N | — |
| 9 | 440 | Y | E | N | N | — |
| 10 | 580 | Y | E | Y (flu) | N | — |
| 11 | 14 | Y | E | N | N | — |
| 12 | 80 | Y | DE | N | N | — |
| 13 | 190 | Y | E/DE/L | N | Y | B,C,G |
| 14 | 300 | Y | E/L | N | Y | B,C,G |
| 15 | 120 | N | DE | Y (pen) | Y | C,G |
| 16 | 400 | Y | E | N | Y | C,G |
| 17 | ? | Y | DE | N | Y | B,G |
| 18 | 310 | Y | E/DE | N | Y | C,G |
| 19 | 60 | Y | L | N | Y | B,C,G |

Dress: Y=yes, N=no, ie exposure method. Surgery: E=early, ie within first week; DE=delayed early, ie at 2 weeks; L=late, ie after separation of eschar. Pen=phenoxymethylpenicillin; flu=flucloxacillin.

TABLE III *Cell wall toxins and exotoxins produced by the streptococci*

| *Lancefield group* | *A* | *B* | *C* | *G* |
|---|---|---|---|---|
| Polysaccharide | *N*-acetyl-glucosamine +rhamnose | *N*-acetyl-glucosamine +rhamnose | *N*-acetyl-glucosamine +rhamnose | Rhamnose |
| M protein | M 1–80 | — | M7 M20 M21 | M16 |
| T protein | Numerous | — | T2 T4 T8 T25 | T16 |
| Fc reactive factor | + | — | + | + |
| Haemolysin | streptolysin O streptolysin S | (haemolysin O) (haemolysin S) | streptolysin O (streptolysin S) | streptolysin O |
| Streptokinase | + | — | + | — |
| Hyaluronidase | + | + | + | + |
| DNAase | + | + | + | — |

(Bergey's Manual of Systematic Bacteriology vol. 2, 1986 *(10)*)

The T protein is one of the cell wall antigens used in Griffiths typing of streptococci.

The group A streptococcus has various cell wall associated toxins and exotoxins responsible for its pathogenicity. β-haemolytic streptococci other than group A produce many of these factors.

PATHOGENIC FACTORS

The cell wall has four constituents: protein, polysaccharide and peptidoglycan that are interwoven and lipoteichoic acid. The outer layer is a fringe of fimbriae.

The M protein is a heat stable, trypsin sensitive constituent of the wall considered the principal factor of virulence. It precipitates fibrinogen, causes clumping of platelets and leucocytes and inhibits migration of the leucocytes—it thus renders the organism resistant to phagocytosis. The group A streptococcus has at least 80 antigenic variants of the M protein, and there is overlap with group C (M7, M20 and M21) and group G (M16) (Table III).

The peptidoglycan/polysaccharide complexes of the cell wall hinder degradation by lysozyme.

The Fc reactive factor binds the Fc fragment of heavy chain IgG—it is found in group A's and some strains of groups C and G.

Lipoteichoic acid is a constituent of the fimbriae and is responsible for adherence to epithelial cells of mucosa.

EXTRACELLULAR PRODUCTS

Haemolysins are cytopathic for mammalian cells. They block phagocytosis by impairing chemotaxis and ingestion by leucocytes and by disrupting lysosomes. Group A streptococci produce two haemolysins: streptolysin O ($O_2$ labile) and streptolysin S ($O_2$ stable). Groups C and G produce streptolysin O. Group C also produces a variant of streptolysin S. Group B produces haemolysins, but different to both O and S. Streptokinase is an activator of the fibronolytic system—it acts by lysing clots and fibrin precipitates. It is produced by groups A and C.

Hyaluronidase is produced by all four groups. It breaks down hyaluronic acid, and with streptokinase allows the organism to spread rapidly through the tissues. Thus the tendency for streptococci to produce a spreading cellulitis.

A series of degrading enzymes is produced by some strains of all the groups. These include DNAase, RNAase and various proteinases that act on several naturally occurring proteins.

Thus all four groups of streptococci produce spreading factors and cytopathic factors capable of destroying skin grafts and regenerating epithelium (Table III).

Why then has this problem not been reported before? Some units have recently noticed clinical problems associated with a growth of groups B, C or G, but attempts to 'blame' the loss of skin graft on these organisms have met with disbelief. The reason given for the loss of graft being poor surgical technique, bed preparation or postoperative care. Other burns units are culturing non-group A streptococci but have not reported any clinical problems. We feel that there may have been a change in the pathogenicity of some strains of groups B, C and G which is responsible for the loss of split skin graft.

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