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Staphylococcal sepsis in a burns unit

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SUMMARY

An outbreak of staphylococcal sepsis in a burns unit occurred between January 1976 and May 1978. Many patients and members of staff had boils, and a number of patients also developed septicaemia. Most of the boils in the early period of the trial and a large proportion of boils in patients during the later period yielded *Staphylococcus aureus* resistant to penicillin, tetracycline and erythromycin only (PTE), and were shown to be of phage type 95 in the early period while strains were phage typed. From blood cultures, most strains in the early period were of resistance pattern PTE and phage type 95, but in the later period other resistance patterns were predominant. Strains from burns were usually multiresistant (PTEKNML) and of the phage pattern 29/77, which had been endemic in the Unit, but during the early period of the outbreak there was an increased proportion of strains in burns with the resistance pattern PTE and of phage type 95.

Staphylococcal sepsis has for many years been very infrequent in the burns unit. This outbreak seems to have been initiated by a strain of phage type 95 and resistance pattern PTE, but during the course of the outbreak the endemic strain of type 29/77 and some other staphylococci seem to have developed enhanced ability to cause clinical infections, conceivably by transduction from the epidemic strain of phage type 95.

INTRODUCTION

Staphylococcus aureus is isolated more often than other pathogens from burn lesions (Lowbury, 1960, 1972; Wickman, 1970; Thomsen, 1970), but in this Unit it has usually caused less clinical infection than *Pseudomonas aeruginosa* and other opportunistic gram-negative bacilli. In this respect burns differ from clean operation wounds, in which *Staph. aureus* is a frequent cause of sepsis, while sepsis due to *Ps. aeruginosa* is relatively rare (Williams *et al.* 1966; Ayliffe *et al.* 1977). At most times few, if any, of the patients whose burns show heavy colonization with *Staph. aureus* develop overt septic lesions or septicaemia, though less severe degrees of sepsis in burns are hard to recognize, and there is evidence that staphylococci may sometimes interfere with the 'take' of skin grafts (Cason & Lowbury, 1960).

In 1976 and 1977 there was an outbreak of clinical *Staph. aureus* infection in this Unit. We report here some details of the outbreak and of the bacteria that caused it.

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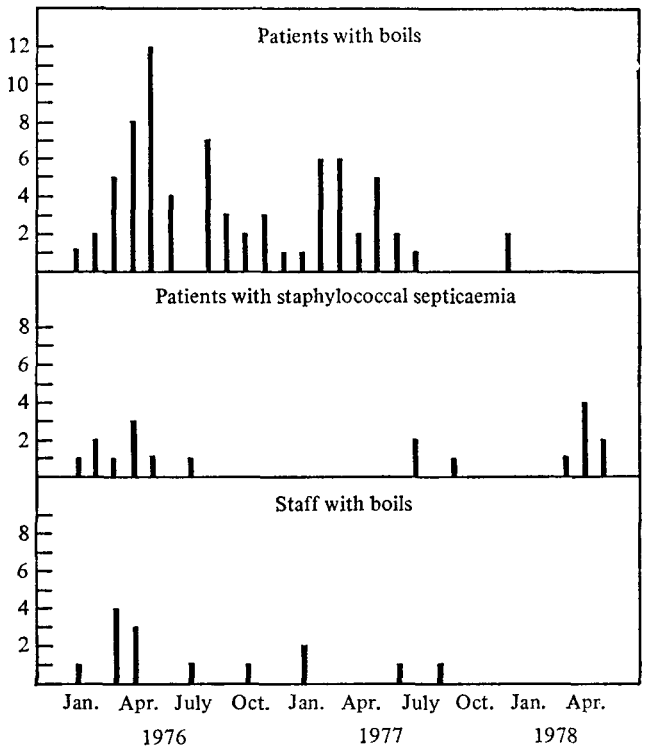

Fig. 1. The numbers of patients and of staff with boils, and the numbers of patients with staphylococcal septicaemia during the outbreak (January 1976 to May 1978).

THE OUTBREAK

Figure 1 shows the numbers of patients in the Burns Unit in each month from from January 1976 to May 1978 who had boils or staphylococcal septicaemia, and of staff (mainly nurses) in each month who had boils. The columns record the total numbers of infections in each month, and the same person sometimes appears in the records for 2 or more months. There was a progressive rise in the numbers of patients with boils to a peak of 12 in May 1976; in each month from January to May one or more patients had staphylococcal septicaemia. The months in which the largest number of staff members had boils were March (4) and April (3) 1976. After July 1976 there were no further cases of staphylococcal septicaemia until August 1977, and there was a fluctuating decline in the incidence of boils in patients and staff. From January 1977 there was a new wave of infections (boils), reaching a level of 5–6 between February and May, and then declining. After August 1977 there was no further incidence of boils except in January 1978, when two patients had them. Between August 1977 and May 1978, however, there were several cases of staphylococcal septicaemia. Over the whole period, 55 patients had boils and 19 had septicaemia; among members of staff 11 nurses and 2 doctors developed boils.

Table 1. *Antibiotic resistance groups of* Staph. aureus *from boils*

		Numbers of *Staph. aureus* strains isolated which were			
		PTE	Multi-resistant	Other patterns*	Total
Patients	1976	58 (67%)	16 (19%)	12 (14%)	86
	1977–8	26 (49%)	8 (15%)	19 (36%)	53
Staff	1976	7 (54%)	2 (15%)	4 (31%)	13
	1977–8	1 (20%)	0 (0%)	4 (80%)	5
Total	1976	65 (66%)	18 (18%)	16 (16%)	99
	1977–8	27 (47%)	8 (14%)	23 (40%)	58

* Mostly Pen + Er resistant with occasional resistance to Tet, Kan, Linc, fucidin, rifamycin.

Strains of Staph. aureus *from infections and from burns*

Methods

Burns were sampled on admission, at change of dressings and operations, and daily if treated by exposure, with swabs moistened in peptone water. Swabs were inoculated with minimum delay (usually not more than 30 min) on horse blood agar (containing 4% New Zealand agar to prevent swarming of Proteus spp.), in Robertson's cooked meat broth and on special media for isolation of the main groups of bacteria. Cultures were examined and strains isolated and identified as described elsewhere (Lowbury *et al.* 1971). Blood cultures were obtained from patients when they had pyrexia with a temperature of over 103 °F. Single colonies on primary blood agar plate cultures from burns, and also from suppurative boils and from blood cultures, which resembled *Staph. aureus*, were picked and examined for Gram stain morphology and by a tube coagulase test, for identification of *Staph. aureus*. Coagulase-positive strains were tested for sensitivity to a range of antibiotics (penicillin (P), methicillin (M), cephaloridine (C), tetracycline (T), erythromycin (E), lincomycin (L), kanamycin (K), gentamicin (G), fusidic acid (F), novobiocin (N), rifamycin (R) and trimethoprim (Tr)) by a ditch plate method (Topley, Lowbury & Hurst, 1951), and a selection of strains was phage typed with a standard set of staphylococcal typing phages kindly supplied by the Central Public Health Laboratory, Colindale.

Antibiotic resistance patterns

Tables 1, 2 and 3 show the numbers of strains of *Staph. aureus* isolated from boils, blood cultures and burns, respectively, during the outbreak and (in selected periods) from burns before and during the outbreak, which were (*a*) resistant to penicillin, tetracycline and erythromycin (PTE), (*b*) multi-resistant (i.e. resistant to PTEKNML), and (*c*) showing other resistance patterns – mostly penicillin and erythromycin resistance, and occasional resistance to K, L, F, R, and T, alone or in combination.

The majority of the boils in 1976, both in patients and in staff members,

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Table 2. *Antibiotic resistance groups of* Staph. aureus *from blood cultures*

Period	Number of *Staph. aureus* isolates which were: PTE	Multi-resistant	Other	Total
1976	4	1	2	7
1977–8	0	8	3	11
Total	4	9	5	18

Table 3. *Antibiotic resistance groups of* Staph. aureus *from burns*

Period	Numbers of *Staph. aureus* isolates which were: PTE	Multi-resistant	Other	Total
April 1975	20 (5%)	359 (91%)	16 (4·1%)	395
December 1975	24 (10%)	109 (46%)	106 (44·4%)	239
January 1976	122 (17·5%)	350 (50%)	225 (32·3%)	697
April 1976	102 (15%)	424 (62%)	159 (23%)	685
April 1977	28 (6%)	91 (18%)	376 (76%)	495
April 1978	1 (0·2%)	216 (45·5%)	258 (54%)	475

yielded *Staph. aureus* strains which were resistant to PTE only (Table 1). In the period 1977–8 PTE was still the commonest resistance pattern of strains from boils in patients, but there were more strains with other resistance patterns, and only one of the five infections in members of staff at that time was with a strain of resistance pattern PTE. Multi-resistant strains were uncommon in boils (less than 20%) in both periods of the outbreak.

In 1976, 4/7 blood cultures yielding *Staph. aureus* (Table 2) had a strain of resistance pattern PTE, but in 1977–8 none of the 11 patients with *Staph. aureus* septicaemia had strains of resistance pattern PTE in their blood cultures; most (8/11) were multi-resistant.

A large proportion of the *Staph. aureus* from burns (Table 3) before and during 1976 were multi-resistant. Strains showing the resistance pattern PTE increased from 5% in 1975, before the outbreak, to 15% at the height of the first wave of the outbreak (in April 1976). There was a large fall in the proportion of multi-resistant *Staph. aureus* between April 1976 and April 1977, from 62% to 18%, a change associated with stopping the routine prophylactic use of erythromycin in October 1976 (Lilly & Lowbury, 1978). The proportion of strains showing the resistance pattern PTE also fell sharply, and in April 1978 only 1 out of 475 (0·2%) *Staph. aureus* strains isolated from burns had that resistance pattern. The predominance of multi-resistant strains and strains showing other resistance patterns in burns paralleled that of strains isolated in the same period from blood culture.

Table 4. *Phage types of* Staph. aureus (1976)

	Number of strains of *Staph. aureus* of type			
	95	29/77	Other*	Total
Boils, staff and patients	13	3	3	19
Blood culture	3	2	4	9
Burns	31	81	36	148

* Mostly Group III or non-typable strains.

Table 5. *Associaton of phage types and resistance patterns* (1976)

	Number of strains of phage type		
Resistance patterns	95	29/77 etc.	Other*
PTE	40	4	22
Multi-resistant	10	78	36
Other†	3	41	23

* Mostly Group III or non-typable strains.
‡ Mostly Pen + Er resistant with occasional resistance to Tet, Kan, Linc, fucidin, rifamycin.

Phage patterns

Table 4 shows the main phage patterns of *Staph. aureus* isolated from boils, blood cultures and burns during the initial peak outbreak in 1976. Strains isolated in the later period were not available for typing. Type 95 strains were the ones most commonly isolated from boils, and type 29/77 strains were the ones most often found on burns. Blood culture strains were more often of type 29/77 or of other types (mostly Group III) or non-typable than of type 95, though the ratio of type 95 to type 29/77 strains was much higher in blood cultures than it was in burns.

Table 5 shows the association of phage patterns and resistance patterns in *Staph. aureus* isolated from boils, blood cultures and burns. Type 95 strains most often showed the resistance pattern PTE, and type 29/77 were most often multi-resistant.

DISCUSSION

Most of the infections, both in patients and in members of staff, during 1976 were caused by strains of *Staph. aureus* resistant to penicillin, tetracycline and erythromycin (PTE) only and of phage type 95. At the same time the majority of strains of *Staph. aureus* isolated from burns were multi-resistant and showed the phage pattern 29/77, or related patterns. During the outbreak of 1976, strains of resistance pattern PTE and phage type 95 became commoner in burns, but were still in the minority. A majority of blood culture strains (4/7), however, were of resistance pattern PTE. During the continuation of the outbreak in 1977 there were more infections caused by strains of different resistance patterns, and in the spring of 1978 several septicaemic infections occurred in severely ill patients, all but one

being caused by strains of resistance patterns different from those which were causing most of the infections in 1976; in this period, too, there was no associated incidence of boils and septicaemia, as there had been in the period of January to May 1976. The later outbreak therefore appears to be distinct from the earlier one. A number of patients in February to July 1977 had boils due to the epidemic type of 1976, and this appears to have been a continuation of the 1976 outbreak, but most of the infections between August 1977 and May 1978 were caused by strains with resistance patterns different from the epidemic type of 1976. The sharp fall in multi-resistant staphylococci in 1977 appears to have been an effect of stopping the routine use of erythromycin as a prophylactic in many patients not known to be actively immune to tetanus (Lilly & Lowbury, 1978).

Staphylococci of resistance pattern PTE and phage type 95 appeared to be the epidemic strain causing both septicaemic infections and boils in 1976, but multi-resistant strains, which have for many years been the predominant staphylococci of the burn flora in this Unit, were also causing some infections in the earlier period, and a larger proportion (though fewer boils) in the period after July 1977. Although strains of phage type 95 and resistance pattern PTE seem to have been exceptionally virulent, no difference could be shown between these strains and the multi-resistant strains of type 29/77 in mouse leg virulence tests (Selbie & Simon, 1952).

It is surprising that the rare occurrence of an outbreak of clinical infection with *Staphylococcus aureus* in the Burns Unit should have been associated with more than one phage type and resistance pattern of staphylococci. This phenomenon might, conceivably, have been due to the transduction of virulence by a phage from a type 95 strain. In the later part of 1978 staphylococci of phage type 95 and resistance pattern PTE (or resistant to penicillin only) were still quite often found in burns; as neither patients nor members of staff had boils or other forms of clinical staphyloccocal infection during this period, it would seem that this epidemic strain had shed its virulence or disappeared from the Unit.

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