TWO OUTBREAKS OF NEONATAL SKIN SEPSIS CAUSED BY STAPHYLOCOCCUS AUREUS, PHAGE TYPE 71

BY

C. H. L. HOWELLS and H. EVERLEY JONES

From The Royal Hospital, Wolverhampton

(RECEIVED FOR PUBLICATION MAY 12, 1960)

Specific epidemic types of *Staphylococcus aureus* have been shown to be responsible for several outbreaks of pemphigus neonatorum (Elliott, Gillespie and Holland, 1941; Hobbs, 1944; Allison and Hobbs, 1947; Williams, Sims-Roberts and Cook, 1947; Parker and Kennedy, 1949; Miller, 1950; Rountree, Heseltine, Rheuben and Shearmann, 1956; Gillespie, Pope and Simpson, 1957).

In this communication two further outbreaks in separate hospitals caused by *Staphylococcus aureus* phage type 71 are reported. The organisms were also isolated from a member of the nursing staff in each hospital.

Methods and Materials

Swabs were taken from the skin lesions of all the affected babies and from the anterior nares of both mothers and nursing staff. The swabs were cultured on blood agar anaerobically and aerobically. Saline moistened swabs were used for sampling the environment of the affected babies.

The Outbreaks

Hospital I. This hospital contains a maternity ward of 25 beds, eight of which are in one ward, and the remaining 17 are in small rooms of two to four beds. The unit deals, in the main, with emergencies, and some of the patients are discharged within 48 hours of delivery owing to pressure on beds. Normally the babies are with the mothers but, in view of the emergency nature of much of the work, some infants are often moved into a nursery.

For some time before the outbreak under discussion, all babies were dusted daily with hexachlorophene powder, and the incidence of sepsis among the babies had been low. During the two weeks preceding the first case, there had been two trivial examples of 'sticky eyes' and on May 13, Case 1, then aged 8 days, developed mild ophthalmia and two days later mastitis which proceeded to an abscess requiring incision. Mother and child were isolated and no further cases occurred until May 20, when Case 2, 12 days old, delivered by caesarian section, developed a septic blister behind the right ear. This was complicated by a submental abscess which required incision.

The department was closed to further admissions, but subsequent infections occurred as follows:

May 22. Case 3, 5 days old; sticky left eye; two blisters on right arm, one behind right ear, and one in left axilla.

May 23. Case 4, aged 5 days; paronychia right thumb; blister left thumb; septic spot left cheek.

Case 5, 3 days old; blister adjacent to umbilicus.

May 24. Case 6, 6 days old; small blister under chin.

May 25. Case 7, aged 4 days; umbilicus moist with adjacent blister.

Case 8, aged 6 days; blister left little finger; two days later, blister left nostril.

Case 9, aged 7 days; blister right index finger.

May 27. Case 10, aged 10 days; septic spot left leg, followed by several others on right leg and groin.

Case 11, twin of infant 10; oedema of left lower eyelid with associated blister; blister on forehead.

Case 12, 4 days old; blister left lower eyelid and lower lip, followed by another on cheek.

May 28. Case 13, 4 days old; multiple small blisters and spots on chin, blister on left hand; later, blister on right shoulder and middle and ring fingers of right hand.

Case 14, 4 days old; blister on right ear; later, blisters in both axillae, left groin, on the neck and left ring finger.

(The maximum extent of the disease is recorded in each case.)

None of the babies affected was seriously ill, and all responded well to treatment.

Hospital II. This hospital has a maternity department of 40 beds, divided into three wards of (a) 19 beds, (b) nine beds, and (c) 14 beds. The second of these, ward (b) has two labour wards, and patients from ward (c) are brought down to (b) to be delivered. As soon as they are fit enough, they and their babies are taken back to their original ward on the floor above. Thus ward (b) is exposed to a good deal of traffic, and this ward was the only one in which infection occurred. All babies are nursed with their mothers, and each ward is of the open type.

There had been no example of neonatal infection in ward (b) for some weeks before August 1959. The ward had, however, been unusually busy just before the first case appeared (on August 5 and 6, 17 women had
been delivered) and, for some time, the average duration of stay had been reduced to about four days. Infant care is much the same as in hospital I, but there are fewer trained staff available.

On August 7, Case T (born by caesarian section seven days before, birth weight 4 lb. 13 oz.) developed a blister on the right forearm. She was removed from the ward and, on the next day, a fresh blister appeared behind the left ear. On August 9, despite treatment, multiple blisters of the trunk and limbs developed, and were associated with widespread desquamation, resembling Ritter's disease. Nikolski's sign was present. At first the disease appeared to respond to treatment (penicillin, followed by aureomycin and erythromycin). On August 13, deterioration set in, the temperature rose for the first time and signs developed in the lungs. She died quite suddenly on the same day aged 13 days. Necropsy revealed confluent bronchopneumonia from which *Staphylococcus aureus* was isolated.

On August 7, Case R (also born by caesarian section on July 31, birth weight 8 lb.) developed paronychia of the right ring and both little fingers. He was removed to the isolation ward. On August 10, a small blister appeared beside the umbilicus. No further lesions were seen and he made a good recovery.

After the occurrence of the second case, ward (b) was closed. All patients at risk were followed, but no other cases of infection were seen.

The sources and sites of the infection in the two Hospitals are given in the Table.

**TABLE**

**Sources of *Staphylococcus aureus*, Phage Type 71**

<table>
<thead>
<tr>
<th>Source</th>
<th>Site</th>
<th>Hospital</th>
</tr>
</thead>
<tbody>
<tr>
<td>Case 3</td>
<td>Skin bullae</td>
<td>I</td>
</tr>
<tr>
<td>Case 4</td>
<td>Skin bullae</td>
<td>I</td>
</tr>
<tr>
<td>Case 5</td>
<td>Skin bullae</td>
<td>I</td>
</tr>
<tr>
<td>Case 7</td>
<td>Skin bullae</td>
<td>I</td>
</tr>
<tr>
<td>Case 12</td>
<td>Skin bullae</td>
<td>I</td>
</tr>
<tr>
<td>Case 13</td>
<td>Skin bullae</td>
<td>I</td>
</tr>
<tr>
<td>Case 14</td>
<td>Skin bullae</td>
<td>I</td>
</tr>
<tr>
<td>Sister W,</td>
<td>Nasal mucosa</td>
<td>I</td>
</tr>
<tr>
<td>Saline swab</td>
<td>Window ledge</td>
<td>I</td>
</tr>
<tr>
<td>Saline swab</td>
<td>Partition</td>
<td>I</td>
</tr>
</tbody>
</table>

**Management**

The organism isolated was found to be penicillin sensitive in all except one infant. Penicillin was therefore given to all except the most mildly affected. The skin lesions were treated with local applications of flavine or gentian violet in spirit. The latter seemed to be rather more effective. The carriers were treated with graneodin applied to the anterior nares, and excluded from duty until their nasal swabs were clear. When the second cases occurred, the practitioners attending infants discharged subsequently were asked to notify the hospital if any infection developed in either mothers or their infants. Only one such came to light, a single spot which rapidly disappeared. The wards concerned were closed to further admissions and the walls thoroughly cleaned, using 1:30 'savlon'. Following this, redecoration was undertaken.

The blankets and mattresses used were stoved. Subsequently, cotton cellular blankets were introduced. The use of hand creams and soaps containing chlorohexidine was encouraged. It is our impression that, following these outbreaks, there is greater awareness of the possible dangers amongst the nursing staff, and there have been no further cases.

**Results**

The first infant to be delivered in Hospital I was the only sufferer who developed no skin lesions. There is no proof that this baby was infected by the same strain of *Staphylococcus* as that cultured from many of the others. All the other infants showed the remarkably similar clinical picture described later.

In Cases 2, 8, 9, 10, *Staphylococcus aureus* phage type 71 was not obtained, possibly due to the habit, subsequently discouraged, of discarding the plates after one colony had been picked. In Cases 12 and 13, the strains were lysed by other dilute phages of Group II at 1,000 × R.T.D. Parker, Tomlinson and Williams (1955) regard these as 'normal 71' strains.

**Discussion**

In both these outbreaks, the majority of the staphylococci isolated from the lesions were of the same phage type. In Hospital I, a single carrier was detected. In Hospital II, the mother of Case T who, together with one of the ward sisters alone among the adults at risk, was found to be harbouring the particular strain of *Staphylococcus aureus*, may have been the culprit. Nasal swabs from the affected sister had never previously yielded *Staphylococcus aureus* phage 71. Subsequent swabs have been negative. Two points require emphasis. First, phage type 71 had never previously been found despite the occurrence of minor staphylococcal infection and the previous isolation of the *Staphylococcus aureus* from nasal swabs, blankets and dust and, second, the great similarity in the clinical appearance. The skin lesions were very alike and started as a small superficial pustule which spread to form a bleb which quickly desquamated, leaving a raw, red, slightly moist area. In some instances the initial finding was a minute spot beside the finger nail, which soon became a paronychia, often spreading to form quite a large blister. Sometimes more
than one finger was involved simultaneously. None was seen on the toes.

There can be little doubt that *Staphylococcus aureus* phage type 71, was responsible for the cases of pemphigus. Though Gillespie *et al.* (1957) reported similar findings in Bristol, it would be unwise to regard this particular phage type as being responsible always for the clinical condition described. Elek (1959) regards phage susceptibility and pemphigus-producing quality as being two independent biological entities. This association might be entirely fortuitous. Indeed, other phage types, particularly 80, have been responsible on other occasions, both in Australia (McLean, 1956; Clarke, McGeoch and Sippe, 1956; McCartney and Yates, 1956) and also in England where it has been 'frequently identified' (Elek, 1959). Nevertheless, it seemed to us of interest to record two further examples in which *Staphylococcus aureus* phage type 71 was once more an aetiological agent.

**Summary**

Two outbreaks of pemphigus neonatorum due to *Staphylococcus aureus* phage type 71 are described. In both outbreaks carriers in the nursing staff were incriminated. Some methods of management are detailed.

Our thanks are due to Dr. Keith Thompson for help with phage typing and to the staff of the two maternity departments for their co-operation.

We are indebted to the Technical Staff of the Bacteriology Department, The Royal Hospital, for their help.

**REFERENCES**


Two Outbreaks of Neonatal Skin Sepsis Caused by Staphylococcus aureus, Phage Type 71
C. H. L. Howells and H. Everley Jones

Arch Dis Child 1961 36: 214-216
doi: 10.1136/adc.36.186.214

Updated information and services can be found at:
http://adc.bmj.com/content/36/186/214.citation

These include:

Email alerting service
Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Notes

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/