

also, that no antigenic likenesses exist between strains of streptococci from acute throat infections similar to that observed between scarlatinal strains.

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**Unusual instances of infection with streptococcus scarlatinae.**

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In the preceding study we noted that infections of the throat with scarlatinal streptococci may occur during an epidemic of scarlet fever, and that these infections are not accompanied by a rash. There is also clinical and epidemiological evidence that these cases may be responsible for cases of clinical scarlet fever among contacts. Williams has recently found this streptococcus in osteomyelitis, endocarditis and in chronically inflamed tonsils, so that we may be assured that *Streptococcus scarlatinae* is a rather widely distributed organism and is not confined in its distribution to the cases diagnosed as clinical scarlet fever.

We know little concerning the clinical manifestations of *Streptococcus scarlatinae* in conditions other than the usual angina with the scarlatina-form rash because the methods of identification are so recent that few observations have accumulated. Some of the infections are associated with a rash as in wound scarlet fever, yet we know that the infection may take place without cutaneous manifestations. The original strain for the toxin made by Dick and Dick was obtained from the infected finger of a nurse caring for a scarlet fever patient. In this instance no rash was reported.

The study of such atypical infections is important from the epidermiologic standpoint. Unfortunately, there is but little opportunity to study such cases in infectious hospitals because they are seldom recognized unless they occur among known scarlatinal contacts, or unless they are uncovered in an attempt to account

for a case of scarlet fever without known exposure. During the past two years we have been able to collect data on six cases of infection with *Streptococcus scarlatinæ* which did not present the usual rash, yet occurred among contacts of scarlatina or were apparently the source of scarlatinal infection in others. The identification of the strains was made by their toxin which was neutralized with antiscarlatinal serum. These data are presented briefly in the following paragraphs:

CASE I. The head of a family returned home after an extended vacation. He developed an acute coryza soon after his arrival. A few days later his wife developed a mild pharyngitis and antrum infection with considerable nasal discharge. The daughter developed clinical scarlet fever a few days after the mother was taken ill. She responded to antiscarlatinal serum. Cultures from the nasal passages of the mother showed streptococci producing scarlatinal toxin. The Dick test was faintly positive. Serum was given as a prophylactic and the pharyngitis subsided immediately. She had had no rash. She was probably the source of the scarlatinal infection in the child.

CASE II. The head of a family was severely prostrated with an acute angina. Throat cultures showed two types of hemolytic streptococci morphologically different on blood agar plates. One of these streptococci was later found to be a *Streptococcus scarlatinæ*. The father had showed no rash. His Dick reaction was negative on the fourth day. Previous reactions were not done. Three days after he became ill a daughter, aged three, developed clinical scarlet fever. She had not been exposed to scarlet fever, since she had been in contact with no one except the mother and a nurse for the previous two weeks. The father apparently had a mixed streptococcus throat infection. What part the scarlatinal streptococcus played in the infection we are not certain. We must conclude, however, that he was responsible for his daughter's infection.

CASE III. Two children developed scarlet fever within a period of 48 hours. They were given antiscarlatinal serum on the fourth day with marked therapeutic benefit. The mother who was Dick negative and had refused prophylactic serum developed an acute streptococcus pharyngitis seven days after the children were first taken ill. The culture of this streptococcus was

lost in the laboratory before the toxin production could be determined. The bacteriologic evidence is not complete in this instance since we did not determine that the strain from the mother was a scarlatinal strain; however, she had had no outside contact for seven days previous to her illness and we conclude she had scarlatina without a rash.

CASE IV. Two children were in bed two weeks with an acute bronchitis. After the second week one of the children developed an acute otitis media with discharge. Two days later the sister vomited and twenty-four hours later showed a typical scarlatinal throat, tongue and rash. Scarlatinal streptococci were recovered from the ear of the first patient and from the throat of the second patient. Since there had been no known scarlatinal contacts, it was concluded that the first child had a scarlatinal otitis media without a rash, and that she was responsible for the infection of her sister. No skin reactions were done to determine if she developed an immunity subsequently.

CASE V. A child of three developed an acute otitis media following an upper respiratory infection (acute coryza). The ear was incised and drained freely for several days, when it suddenly ceased to discharge. The following morning the child had fever, vomited, and had a typical scarlatinal rash, without angina. The ear was reopened, and the rash and symptoms immediately subsided. The Dick reaction on the following day was questionably positive. Cultures from the ear showed a scarlatinal streptococcus as judged by toxin production. In this instance there was probably almost complete immunity to the toxin, yet when confined there was sufficient toxin in the discharge from the ear to cause a rash.

CASE VI. A child of six developed scarlet fever in May. She had been in contact with no other children except her brothers and sisters for the previous week. Scarlet fever was not prevalent. The mother had had an acute antrum two weeks previously. When the child was taken ill this antrum infection had subsided, but cultures from the nose showed streptococci which produced scarlatinal toxin. The mother had apparently had a bacterial infection of the nose with *Streptococcus scarlatinae* and was the source of the daughter's infection. As far as she knew she had had no rash.