Epidem. Inf. (1987), 99, 249–255
Printed in Great Britain

Food-borne outbreak of group G streptococcal sore throat in an Israeli military base

By D. COHEN
Medical Corps, Israel Defence Forces
M. FERNE
National Streptococcal Reference Center, Israel
T. ROUACH
Medical Corps, Israel Defence Forces
AND S. BERGNER-RABINOWITZ
National Streptococcal Reference Center, Israel

(*Accepted 20 January 1987*)

SUMMARY

A food-borne outbreak of sore throat caused by Lancefield group G β-haemolytic streptococci and involving 50 persons occurred in May 1983 in an Israeli military camp. All of the patients available for clinical examination had sore throat and difficulty in swallowing. Exudative tonsillitis occurred in 46% of the patients and the body temperature was above 37·5 °C in 81%. The pattern of attack was uniform over the base and 37 became ill during the night and morning of the 5 May. Thirty-two (84%) of the throat cultures taken from 37 patients grew group G β-haemolytic streptococci. Eight of 29 contacts were positive for group G β-haemolytic streptococci and 6 of the 28 foodhandlers examined had positive cultures of the same group. The organism was also isolated from one food sample. The epidemiological and laboratory investigations indicated that a food handler, a convalescent carrier of group G streptococci, might have been the source of infection. Assumptions on the potential of non-group A streptococci to cause epidemics are discussed.

INTRODUCTION

Non-group A β-haemolytic streptococci have only occasionally been shown to cause outbreaks of acute sore throat (Duca *et al.* 1969; Isaesohn *et al.* 1980; Cohen *et al.* 1981; Dinari *et al.* 1982; Hill *et al.* 1980; Stryker, Fraser & Facklam, 1982). All of these outbreaks but one (Isaesohn *et al* 1980) were food-borne. We report another food-borne outbreak of streptococcal pharyngitis among male recruits in an Israel Defence Force (IDF) camp. Fifty soldiers became ill suddenly and Lancefield group G β-haemolytic streptococci were found to be responsible.

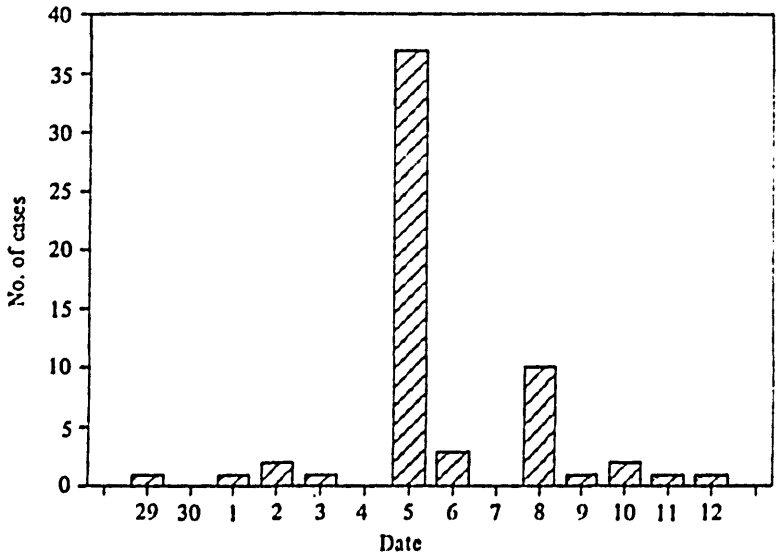

Fig. 1. Cases of acute pharyngitis between 20 April 1983 and 12 May 1983.

THE OUTBREAK

On a single day, in May 1983, 37 cases of acute pharyngitis were seen at the unit medical clinic. No further cases were found among the companies present in the base on the same day and only isolated cases had been seen in the same clinic during the 7 previous days (Fig. 1). Thirty-two of the patients were available for complete clinical examination. All of them had sore throat and difficulty in swallowing, 60% had swollen cervical lymph glands, 46% had pharyngeal exudate and 81% a temperature above 37·5 °C. Three days later, a second group of cases of acute pharyngitis was noted in the unit clinic. This was apparently due either to secondary respiratory spread (6 cases) or delayed visits of patients who initially fell sick on 5 May (4 cases). Five days after the outbreak had begun, the number of new cases abruptly declined.

Systematic clinical and serological follow-up was not feasible because of the subsequent dispersal of recruits to many different training camps.

MATERIALS AND METHODS

Epidemiological and laboratory investigations

The investigating team came to the training base after the report of many cases of acute pharyngitis was received. Epidemiological and laboratory investigations were carried out the same day. Unit medical records were examined for diagnoses related to sore throat. Sixty-one soldiers (both sick and healthy) were questioned about personal details, living and working conditions at the base, duties, date of onset of the illness and symptoms. In addition they were asked about contact with sick people inside or outside the base. In particular their attendance at meals in the base dining-room and details of food eaten in the 3 days preceding the outbreak were noted. The 61 soldiers questioned included 28 of the patients seen at the clinic

https://doi.org/10.1017/S0950268800067716 Published online by Cambridge University Press

in the first day of the outbreak and 33 healthy soldiers who had not complained of sore throat or any other symptoms of upper respiratory infection. The healthy soldiers served in the same base subunits and used the same living accommodation as the patients. They were evenly chosen and sent by their staff to the unit medical clinic for investigation.

Throat swabs were taken from 37 patients and 53 asymptomatic soldiers including food handlers. Streptococci from throat swabs were identified using standard laboratory methods. After plating on tryptic soy agar plus defibrinated sheep blood (5% v/v) (Hy-Labs, Rehovot) the Lancefield groups of haemolytic streptococci were defined using both slide agglutination (Griffith, 1934) and precipitation tests (Swift, Wilson & Lancefield, 1943). The food samples preserved until the arrival of the investigators were inoculated on the same media supplemented with nalidixic acid (10 mg/l). Several strains of group G isolated from patients together with the strain isolated from one of the food items were sent to the Division of Hospital Infection of the Central Public Health Laboratory, London, for serotyping (Efstratiou, 1983). The MIC (minimum inhibitory concentrations) and MBC (minimum bactericidal concentration) were examined in eight strains using doubling dilutions of the organism in Todd Hewitt Broth (Goebens, Fontijne & Michel, 1982). Streptolysin O (SLO) and DNase were determined quantitatively in the supernatant of 10 strains (Williams, 1958; Nelson, Ayoub & Wannamaker, 1968) and hyaluronidase activity was measured by the turbidity test (Watanabe, 1976). Virulence of the epidemic strain was tested by injecting 0·5 ml of three different dilutions of the organism into mice (Horne & Thomasz, 1980).

RESULTS

Thirty-one of the patients (84%) grew group G β-haemolytic streptococci as did 8 (28%) of 29 contacts (Table 1). Twenty-eight food handlers, 4 suffering from acute pharyngitis and 24 asymptomatic, were tested for the presence of group G streptococci in throat cultures. The 4 cases with sore throat grew group G streptococci while only 2 of the 24 'healthy' food handlers were found positive for the same group pf streptococci (Table 1). All the patients and positive contacts harbouring group G streptococci were given a 10-day course of oral penicillin V–K. The same organism was found in the minced meat balls in cold sauce served at dinner the day before the onset of the outbreak. Cultures of other food as well as of water sampled did not yield β-haemolytic streptococci. The strains were sensitive to penicillin. They did not produce high levels of SLO, DNase or hyaluronidase. None of the six mice died after they were given 5×10^6 and 5×10^8 organisms. All of the strains were reported to lack defined T-protein antigens. The simultaneous occurrence of the clinical symptoms in most of the patients (in the evening of 4 May) suggested a common source epidemic. The patients were evenly distributed between the base subunits and among staff and recruits. No meetings or other common activities happened during the days prior to the outbreak. The only common areas for all the personnel were two dining halls, one for recruits and the other for staff, both served by the same kitchen. These findings together with the isolation of group G β-haemolytic streptococci from one of the food samples,

Table 1. *The results of the throat cultures taken from 37 patients, 29 contacts and 28 food handlers*

	No.	Positive to β-haemolytic streptococci (%)	Positive to group G β-haemolytic streptococci (%)
Patients	37	32(86)	31(84)
Contacts	29	9(31)	8(28)
Sick food handlers	4	4(100)	4(100)
Asymptomatic food handlers	24	2(8)	2(8)

Table 2. *The probabilities of eating the food items served during the suspected meals, on 3 May, in cases and controls*

	Cases ($n = 28$)	Controls ($n = 33$)	Odds ratio	P*
Lunch				
Bean soup	9(32)†	14(42)	1·5	N.S.
Roast chicken	18(64)	19(58)	1·3	N.S.
Rice	20(71)	17(52)	2·4	N.S.
Houmous	8(29)	14(42)	1·8	N.S.
Dinner				
Cucumber salad	21(75)	17(52)	2·8	0·051
Cake	19(68)	19(68)	1·5	N.S.
Vegetables	21(75)	19(68)	2·2	N.S.
Egg salad	20(71)	11(33)	5·0	0·0031

* P, values determined using Fisher's exact test.
† Figures in parentheses are percentages.
N.S., not significant.

suggested that the outbreak probably was food-borne. Only two meals shared by all the patients at times compatible with the streptococcal incubation period (1–3 days) were suspected to be the source of the epidemic contamination. We ruled out the possibility that the minced meat balls in cold sauce which had grown group G streptococci could have been the vehicle of the epidemic agent. This food item was served to the soldiers only very few hours before the appearance of the first clinical symptoms in most of the patients.

The case-control study performed on all the food items served at the suspected meals, lunch and dinner 2 days before the onset of the outbreak, suggested that boiled egg salad served cold at dinner that day was the vehicle of the epidemic agent (Table 2). Further one of the food handlers had developed acute pharyngitis 10 days prior to the outbreak and was given a full course of penicillin V–K at the unit medical clinic, but he did not comply with the regime. Group G β-haemolytic streptococci were cultured in large numbers from his throat. This food handler had thus become an asymptomatic carrier of group G streptococci and could well have been the source of the epidemic agent which was transmitted to the food either directly or by the contamination of other food handlers.

DISCUSSION

Air-borne epidemics of streptococcal sore throat sometimes followed by non-suppurative complications, are well described. They are all due to group A streptococci and have also occurred in military populations (Coburn & Young, 1949; Rammelkamp, 1956; Schneider *et al.* 1964). Prevalence of 30% or more group A streptococci in the population at risk, a 50% or greater typability of the epidemic strain accounting for at least one third of cases and possible occurrence of streptococcal non-suppurative complications, are the main features of a streptococcal air-borne epidemic (Zimmerman, Cross & Miller, 1966). The streptococcal food-borne epidemics, which are still not widely recognized, are characterized by different features, such as abrupt onset and termination, appearance of most of the cases within only one incubation period and few complications (Horwitz, 1977).

Milk-borne epidemics which occurred prior to the regular use of pasteurization were mostly due to contamination from cows suffering from mastitis and were the first indications that there was a route of transmission of streptococci other than the respiratory one (Taylor & McDonald, 1959). Later, other food items, particularly cold hard-boiled eggs were suspected to be involved in food-borne epidemics caused by group A β-haemolytic streptococci (Hill *et al.* 1969).

Reports dealing with food-borne epidemics of streptococcal pharyngitis due to streptococci other than those of group A are rare. As a result of faulty handling of milk products, 85 Rumanian peasants developed streptococcal pharyngitis due to group C streptococci. They were not treated with antibiotics during the primary infection and a third of them developed glomerulonephritis (Duca *et al.* 1969). Group C streptococci of T-type 4 were responsible for another food-borne outbreak of acute sore throat in an Israel Defence Force camp (Dinari *et al.* 1982). One hundred and seventy-six students in a college in the eastern United States (Hill *et al.* 1980) and 72 visitors of a hotel in Florida developed severe symptoms of acute pharyngitis due to β-haemolytic streptococci of group G transmitted by egg salad and chicken salad respectively (Stryker, Fraser & Fackham, 1982). These reports were based on epidemiological findings including the results of case control studies. In none was the isolation of the epidemic agent from the food itself reported. It seems that either the food samples were not available for bacteriological tests, or if they were, the overgrowth of a variety of organisms made the isolation of streptococci impossible. In our outbreak, immediately after the first report of an unusually large number of cases of acute sore throat seen in the unit medical clinic was received, we ordered the preservation of food samples covering the last 48 h. The food samples were inoculated on selective media the same day. One of eight food samples (minced meat balls in cold sauce) grew the same group G streptococcus as isolated in the throat cultures of the patients. The food item was thought not to be responsible for the epidemic because it was served only a few hours before most of the patients had begun to suffer from the first symptoms. Nevertheless this finding showed that contamination of food with the epidemic strain did occur. This is, in our opinion, strong evidence for a food-borne outbreak. The findings of the case-control study showed that the egg salad was probably the specific vehicle of the epidemic agent. Unfortunately we did not succeed in isolating streptococci in the egg salad but it is possible that contamination of the egg

salad happened after it was taken from the pots and served. Samples kept in case of possible outbreaks are taken directly from the pots and not from the dishes themselves. In most of the reported food-borne epidemics of streptococcal pharyngitis, cold egg salad was the food item suspected to be the vehicle of the epidemic agent (Cohen *et al.* 1981; Hill *et al.* 1969; 1980; Cohen, Rouach & Dinari, 1981) and a very simple laboratory experiment demonstrated that egg salad was a good medium for the multiplication and growth of streptococci (Furer *et al.* 1976). We assumed that a food handler who was an asymptomatic carrier of group G streptococci was the source of infection. It is well known that nose and throat carriers of streptococci may inoculate food by sneezing or by handling food with hands contaminated by respiratory secretions (Hamburger, Green & Hamburger, 1945). It is generally believed that pyogenic streptococci of group C or group G may cause occasional sporadic and mild infections. There are however a few reports of the development of rheumatic fever or glomerulonephritis after primary upper respiratory infection with group G and group C streptococci (Koshi & Mamman, 1967; Reid *et al.* 1985). Because of the dispersal of recruits to several IDF camps, it was not possible to undertake a systematic serological and clinical follow up but no reports of late complications were received from the camps. Epidemics of streptococcal pharyngitis due to streptococci of group C and G do occur however and the clinical spectrum is similar to that due to group A streptococci. Two possibilities can be advanced for the unusual epidemic potential of these streptococci: the appearance of new and virulent group C or G streptococci to which the population was not previously in contact or exposure to a massive inoculum transmitted by food. The result of the tests performed in order to examine the features of the group G epidemic strain (SLO, DNase, hyaluronidase quantitative determinations, MIC and MBC evaluation and virulence to mice) showed that we dealt with a strain of low virulence. In another report on two outbreaks of streptococcal pharyngitis that occurred in one IDF base 7 days, one after the other, due to group C and group A M-type 49 streptococci, we had the opportunity to compare the symptomatic to asymptomatic ratio among soldiers who had positive throat cultures for group C and group A streptococci. The rate of symptomatic infection with group A M-type 49 was significantly higher than with group C, 45 of 56 (80%) *vs* 28 of 74 (38%) respectively (Cohen *et al.* 1981; Dinari *et al.* 1982). We assume that both in that case as well as in the present one, ingestion of a large amount of inoculum was necessary for the epidemic spread of organisms of relatively low virulence such as group C and group G streptococci.

Thanks are due to Dr J. Kark for the helpful criticism of the manuscript.

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