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REVIEW ARTICLE
Streptococcal contamination of food: an unusual cause of epidemic pharyngitis

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SUMMARY

The purpose of this article is to define the distinguishing characteristics of food-borne streptococcal pharyngitis by reviewing the literature. The main cause of this infection lies in poor handling and preservation of cold salads, usually those which contain eggs and are prepared some hours before serving. A shorter incubation period and a higher attack rate (51–90%) than in transmission by droplets was noted. The epidemics tend to occur in warm climates and in the hottest months of the year. *Streptococcus pyogenes* seems to originate from the pharynx or hand lesions of a food handler. In comparison to airborne transmission symptoms such as sore throat, pharyngeal erythema, and enlarged tonsils, submandibular lymphadenopathy are more frequent than coughing and coryza. Seven out of 17 reports revealed an M-untypeable serotype, which may possess virulent characteristics. Penicillin prophylaxis was shown to limit additional spread of the infection. There were no non-suppurative sequels, and suppurative sequels were very rare. We assume that the guidelines for the prevention of food poisoning would apply to food-borne streptococcal pharyngitis. Food handlers should be supervised to ensure they comply with strict rules of preparation and storage of food. Cold salads, especially those containing eggs, should not be left overnight before serving.

INTRODUCTION

Respiratory droplets constitute the most common means by which streptococcal pharyngitis is spread [1]. Until the 1940s, the main sources of food-borne streptococcal pharyngitis were milk and milk products. Since pasteurization has become a common practice and the storage conditions of food have improved, food-borne spread of streptococcal pharyngitis has become rare, and milk has ceased to be the main vector for the infection [2]. A system of serotyping group A streptococci was developed by Lancefield on the basis of M-protein precipitin reaction, and by Griffith on the basis of T-protein agglutination reaction. M protein is a major factor in determining the virulence of streptococci. Streptococci, which are rich in M protein, are more virulent. Immunity can develop for a serotype based on antibody reaction to M protein. The T protein has no known role in virulence.

This article reviews the data from the reported food-borne outbreaks of streptococcal pharyngitis in the last four decades, in order to characterize the epidemiological, bacteriological and clinical features of the disease.

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Table 1. *Seasonal distribution, place and number of people affected by food-borne streptococcal epidemics*

Reference number	Date	Place of the epidemic	Number of patients
1	February 1991	Israel	55
2	August 1974	Florida	91
3	June 1980	Israel, a military camp	41
4	May 1983	Israel, a military camp	50
5	April 1968	United States Air Force base	1200
6	April 1990	Israel	61
7	May–June 1984	Missouri	60
8	July 1973	Arizona	121
9	March 1975	Israel, a factory	447
10	December 1997	Greece, residential students	154
11	May 1991	Louisiana	75
12	August 1984	Puerto-Rico	23
14	April 1992	Israel, a military camp	197
15	November 1983	Georgia	20
16	May 1981	Oregon	60
17	June 1979	Florida	72
18	July 1982	New Hampshire	34

EPIDEMIOLOGY

Environmental factors

Epidemics are more common in warm climates. Table 1 summarizes the location and seasonal distribution of the epidemics reviewed. Eleven out of 16 reported epidemics took place between April and August. We presume that the reason is that high temperatures enhance multiplication of the germ.

Vector

Table 2 shows that cold salads are the main vector in the reported epidemics. Eggs appear as the most common ingredient [2–10], but mayonnaise, tuna, potatoes, cheese and conch were also reported as ingredients in the dishes which transmitted the germ in epidemics. An experiment was conducted in which streptococci extracted from throats of patients with pharyngitis were grown at room temperature on a medium containing eggs. The streptococci number multiplied by 10^8 in 40 h. This demonstrates that eggs are a very good growth medium for streptococci, especially when kept at room temperature [9, 11].

Attack rate

Table 2 demonstrates that the attack rates ranged between 50 % and 91 % among the people who ate the contaminated food. One article [8] demonstrated a statistically significant lower attack rate among children under 10 years of age. Secondary attack rates were low [2, 12, 13] probably thanks to vigorous antibiotic treatment.

Reservoir

Some of the food handlers who prepared the salads causing the epidemics (Table 3) were found to harbour the streptococci in their pharynx or on skin lesions [1–5, 11, 13–17]. Investigations suggest that substantial amounts of streptococci are disseminated when nasal carriers sneeze or cough. Inoculation by hands contaminated with respiratory secretions is possible in these cases too [5].

Food preparation

Table 3 shows that almost all the salads that caused the epidemics were prepared 24 h before they were served [2, 5, 6, 8, 11, 14, 15, 17]. The time that passed between inoculation and infection allowed the germ to multiply to an infective dose. Foods, which were neither boiled during preparation, nor kept in proper refrigerating facilities [2, 7, 9–12, 17], were prone to develop an infective inoculum of the germ. The fact that some of the foods were kept out of the refrigerator several hours before they were served contributed to the multiplication of the germ and evolution of the epidemics [6, 7, 10–12, 15].

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Table 2. *Source of food-borne streptococcal infections and the attack rates*

Reference number	Source	Attack rate among the people who ate the dish with the streptococcus
1	Cabbage salad	91%
2	Egg salad	77·9%
3	Egg salad	No data
4	Egg salad served cold	64·5%
5	Tuna salad with mayonnaise and eggs	No data
6	Egg salad with mayonnaise	90%
7	Mousse with cream, eggs and gelatine	62·8%
8	Potato salad with mayonnaise and eggs	59%
9	Egg salad	No data
10	Vegetable salad dressing made of mayonnaise and raw eggs	64% of students
11	Macaroni with cheese	50%
12	'Carrucho' conch salad	70%
14	White cheese	No data
15	Rice souse	60%
16	Unknown	No data
17	Chicken salad with mayonnaise	53%
18	Non-conclusive, onion dip, clamp dip or potato salad	No data

IDENTIFICATION OF FOOD-BORNE OUTBREAK OF PHARYNGITIS

Food-borne epidemics of streptococcal pharyngitis differ from airborne epidemics in that they begin abruptly and have a low complication rate [3, 14]. The character of the epidemic curve suggests that the outbreaks have a common-source [10, 15]. The most reliable way of defining food-borne streptococcal pharyngitis is by obtaining an identical positive culture from the throat of the patient and from the food. Some of the epidemiological investigations did not succeed in obtaining a food sample for bacteriological culturing, and in recovering the germ from the food. The use of food-history questionnaires was the alternative method used to establish that a certain food was the source of an epidemic [5, 6]. In those cases, the epidemic was considered as food-borne when an epidemiological investigation showed a significant correlation between having eaten a certain dish, and having streptococcal pharyngitis [6–8, 12, 18].

CLINICAL MANIFESTATIONS

The clinical features of food-borne streptococcal pharyngitis were typically concentrated in the pharynx, more so than with air-borne streptococcal pharyngitis [19]. The food-borne disease was characterized by sore throat, pharyngeal erythema, enlarged tonsils and submandibular lymphadenopathy, and was described as more acute than the air-borne disease, of which the symptoms were coughing and coryza [1, 5, 14]. Secondary air-borne transmission in a food-borne epidemic bore the clinical characteristics of airborne disease [19]. Involvement of the nasal mucosa and the bronchial tree was less common than in pharyngitis transmitted through the respiratory system. It was suggested that the difference between the physiology of swallowing and the physiology of respiration, is the reason for the confinement of the symptoms to the pharynx. The direct exposure of the pharyngeal mucosa to the pathogen by the swallowed food may cause the confinement of the symptoms and signs to the pharynx in this mode of transmission. Small particles of saliva and aerosol contaminated by streptococci, which are inhaled into the respiratory tract, expose the nasal and bronchial mucosa more easily to the pathogen. Therefore, coryza and coughing are more common in the patients with air-borne streptococcal pharyngitis than in those with food-borne infection [19].

LABORATORY INVESTIGATION

When pharyngitis appears sporadically, throat cultures are essential for the diagnosis of streptococcal pharyngitis. The high rate of positive throat cultures suggests that, in an epidemic condition, clinical criteria may be sufficient for diagnosis [3].

In one study conducted in an air force academy [5] where 1200 cadets were infected with food-borne streptococcal pharyngitis, eight cadets were found to

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Table 3. *Epidemiological characteristics of streptococcal food-borne outbreaks*

Reference number	Food handler	Preparation of the food
1	A food handler who had streptococcal pharyngitis and a positive culture for the germ prepared the salad	No data
2	The prisoner who peeled the eggs had pharyngitis with a fever of 39 °C. A throat culture was positive for the germ	The salad was prepared a day in advance and stored in a refrigerator, which was out of order
3	The cook who made the salad had a sore throat 3 days before. A throat culture was positive for the germ	No data
4	A food handler had pharyngitis 10 days before the outbreak. He was prescribed penicillin but did not take it regularly. A throat culture proved that he was a carrier of the germ during the outbreak	No data
5	A food-handler with a positive culture for the germ and no symptoms made the salad	Kept in the refrigerator for 24 h before served
6	The dish was prepared by food-handlers with pharyngitis	The dish was prepared 2 days before it was served. It was taken out of the refrigerator several hours before serving
7	One food handler had a sore throat when preparing the mouse. All cultures were negative for the germ	The mouse was served in the same day that it was prepared, but it was only half an hour in the refrigerator during the whole day
8	One of the food handlers had a son with pharyngitis	The salad was prepared a day before, and kept in the refrigerator
9	Eight food-handlers with a positive culture for the germ	The food was not kept in the refrigerator before serving
10	No data	The dressing was kept in room temperature for 15 h before served
11	A lesion on the back of the hand of the food handler who prepared the dish had a positive culture for the germ	The dish was not boiled. It was kept in the refrigerator for 24 h, and then it was served under a lamp for warming
12	Cultures from the food handlers were negative. They claim to be asymptomatic	The salad was made in an unlicensed restaurant. The conch was from conspicuous sources. The salad was 3 h in the car
14	A food handler with a positive throat culture for the pathogen prepared the cheese	The food handler mixed the cheese with his bare hands and put it in the refrigerator for 24 h
15	The germ was found in a throat culture taken from the woman preparing the souse. She was symptomatic 3 weeks before	The sauce was pre-made a day before and kept in the refrigerator. It was taken out of the refrigerator 4 h before served
16	Four food handlers had a positive throat culture and three had positive cultures from lesions on their hands for the germ	No data
17	A food handler with group G streptococcal pharyngitis proven by culture	The salad was prepared a day before serving. It was kept in a 20 cm deep pot in the refrigerator. It was taken twice out of the refrigerator for an hour
18	The food handler had a son with pharyngitis	No data

have haematuria. None of these cadets had a decline in the C3. The absence of glomerulonephritis in this study indicated that C3 is a useful screening method for the exclusion of glomerulonephritis. In other

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studies, ASO titres and anti-DNase B titres were also found to be high [13, 17]. In the same study [5], the antibody response was shown to be higher in cadets treated with oral erythromycin, than in cadets treated with long acting parenteral penicillin. Asymptomatic carriage was found to be rare [13]. Except from one case in which streptococcus group G was cultured [17], all cases revealed streptococcus group A. Seven out of 17 reports revealed an M-nontypable streptococcus. Since the M-protein is a major factor influencing the virulence of the streptococcus, there could be a specific serotype, which cannot yet be characterized, that influenced the epidemiology and clinical manifestation of these outbreaks. Five of the reports revealed a T/12 serotype and three a T/9 serotype. The T serotype has no clinical importance, but the question remains whether the T or M types influence the streptococcal ability to be transmitted by food.

TREATMENT

The treatment regimen used in the articles reviewed was either a 10 day course of oral penicillin or erythromycin, or intramuscular benzathine penicillin. Streptococci involved in food-borne infections were found to be sensitive to penicillin. In one report [13], 18% of patients treated with erythromycin had a positive culture for the pathogen after the treatment, compared to only 1·5% of the patients treated with penicillin. This demonstrates the advantage of intramuscular penicillin over oral erythromycin in preventing treatment failure in food-borne *Streptococcus pyogenes* infection.

Penicillin prophylaxis was shown to limit additional spread and non-suppurative sequels [5, 12, 14, 17]. Penicillin prophylaxis was effective in preventing secondary transmission during outbreaks of food-borne streptococcal pharyngitis, but it is not yet clear whether this is advisable, in light of adverse effects of penicillin on penicillin-allergic individuals [2]. Low isolation rate of the pathogen from asymptomatic exposed people, suggest that prompt treatment of infected individuals may suffice to prevent the disease from spreading [2].

SEQUELS

Apart from prevention of secondary infection, the major benefit of penicillin treatment lies in the prevention of acute rheumatic fever and glomerulonephritis [2, 17]. In the 5·5 week period of the follow-up study, no evidence of glomerulonephritis or rheumatic fever was found [13]. There were no reports of non-suppurative sequels of streptococcal pharyngitis in the other articles reviewed. Suppurative sequels were very rare and included a few cases of peritonsillar abscesses. This could be attributed to the aggressive antibiotic treatment given liberally in those outbreaks.

PREVENTION

The methods used to control potential respiratory outbreaks do not prevent extensive food-borne epidemics, because of the suddenness with which the majority of cases appear after a common exposure [5]. There is no prospective data correlating between specific kitchen habits of food handlers, and the prevention of food-borne streptococcal pharyngitis. It has been shown that nasal carriers of streptococci may contaminate food by sneezing, or by handling food with hands contaminated by respiratory secretion [3]. Based on Table 3 we assume that the following guidelines for the prevention of food poisoning would apply to food-borne streptococcal pharyngitis too: food handlers who are household contacts of people with acute pharyngitis, should be considered as posing an increased risk of spreading streptococcal pharyngitis, even if they are asymptomatic. They should therefore be excluded from food handling [18]. Food handlers should be supervised to ensure that they comply with strict rules of hygiene and in particular that their hands are clean. The use of bare hands should be banned. The use of barriers or utensils should be enforced. Food handlers with skin lesions should be excluded from handling food. Food handlers who acquire a cold and sneeze or cough should be excluded from food handling or should wear a protecting mask. The storage and preparation of food should be carefully monitored. Food must be properly cooked, especially when prepared in large quantities. Food must not be kept at room temperature unless it is served immediately. Temperatures in refrigerators should be monitored. Cold salads, especially those containing eggs, should not be left overnight. Unpasteurized milk or milk products should not be used. Food handlers should be trained and educated to comply with these recommendations. Administering prophylactic antibiotics to medical personnel should be considered, because of a high secondary attack rate within the medical staff [14].

CONCLUSION

The improvement in the preservation and processing of food in the second half of the last century did not eliminate food-borne streptococcal pharyngitis. Immediate diagnosis of the disease, isolation of infected individuals and propagation of antibiotic treatment, are necessary for the prevention of secondary infection, as well as suppurative and non-suppurative sequels. Food handling procedures and food handlers should be strictly supervised to prevent food-borne streptococcal pharyngitis, as well as other food borne epidemics. Exclusion of food-handlers who have streptococcal pharyngitis should be considered. Further study of the growth and survival of streptococci in food is needed in order to prepare recommendations for prevention of food-borne streptococcal pharyngitis. The importance of the M and T proteins in food-borne streptococcal pharyngitis still remains to be evaluated.

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