

Should young adults with sore throat be treated with antibiotics?

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Summary

The diagnosis of streptococcal sore throat on clinical grounds remains a problem. In this study the clinical prediction in a group of young adults corresponded with laboratory findings indicative of a streptococcal (group A or non-A) infection in 23% of cases.

The culture of throat swabs was of little value, as the only group A culture-positive patient did not show an antibody response, indicating a carrier state.

In 5 cases a streptococcal infection was diagnosed on rising antibody titres only, as culture remained negative. The value of rising antibody titres as a diagnostic tool is also questioned, since they occurred more frequently in the healthy controls than in the sore-throat group.

Antibiotic treatment for sore throat was rarely supported by laboratory findings in the young adult population studied.

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One cannot but agree with Bisno that 'after all these years, the "simple sore throat" remains a surprisingly complex problem'.¹

Current practice comprises clinical assessment complemented by bacteriological culture of a throat swab. In the event of clinical suspicion that *Streptococcus pyogenes* (group A β -haemolytic streptococcus) or other bacterial organisms may be involved, or if the culture confirms its presence or that of other pathogenic organisms, therapy with penicillin V is prescribed for a period of 10 days in an attempt to prevent non-suppurative complications such as acute rheumatic fever.²

This is often the approach of the general practitioner, but there are three further alternatives for the handling of the problem: (i) antibiotic treatment may be given to all patients presenting with the symptom of sore throat;² (ii) the patients may be treated symptomatically;² and (iii) selective cultures and appropriate treatment may be considered.

The purpose of the present study was to evaluate the correctness of the clinical differentiation between streptococcal and non-streptococcal sore throat by means of laboratory studies such as throat-swab cultures, antibody response and full blood counts. Results were compared with those of a paired healthy control group.

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Subjects and methods

Participants in this study were medical and dental students who attended our clinic with the complaint of sore throat. Each was matched with a healthy control within a day. At the time of the initial visit a history regarding the present illness was obtained and a clinical examination performed.

The following specimens were obtained and immediately sent off to the adjacent Department of Medical Microbiology for culture and serological examination: throat swabs (tonsils or tonsillar fossae and posterior pharynx) for culture of streptococcus and adenovirus, and 10-25 ml venous blood for antibody response to streptococcus, *Mycoplasma pneumoniae* and adenovirus. Antibodies tested were antistreptolysin O (ASO) (normal < 200 Todd units/ml, elevated \geq 200 Todd units/ml) and streptokinase haemagglutination (normal < 1:1280; elevated > 2 dilution rise). Since acute-phase titre studies as such are relatively meaningless, it was decided to perform follow-up titre assays after 14 days.

The patients with a history of contact, duration of sore throat longer than 5 days, abnormal cervical lymph nodes, an abnormal pharynx or an elevated temperature were diagnosed as having streptococcal sore throat and treated immediately without waiting for laboratory results. All the cases were given an appointment for a return visit after a fortnight for follow-up antibody titre assays.

Results

A total of 64 patients with sore throat and 64 matched controls were enrolled in this study from January 1981 to February 1982. Only 2 patients did not return for their follow-up visit and were therefore not taken into consideration. The mean age of the group with sore throat was 23,0 years and that of the control group 22,2 years.

The agreement between the clinical prediction and the laboratory findings of the presence of streptococcus for the 62 patients with sore throat is depicted in Table I, which shows that the clinical predictions were correct in 26 cases (42%) and wrong in 36 cases (58%). There is no statistically significant agreement between the clinical predictions and the laboratory findings (χ^2 test for a 2 x 2 table).

Cases taken as being laboratory-positive were those in which laboratory tests revealed one of the following: (i) culture positive

TABLE I. AGREEMENT BETWEEN CLINICAL PREDICTION OF STREPTOCOCCUS AS THE CAUSE OF SORE THROAT AND LABORATORY FINDINGS

Clinical prediction	Laboratory findings	
	Strep. present	Strep. not present
Strep. present	14 (23%)	31 (50%)
Strep. not present	5 (8%)	12 (19%)

TABLE II. LABORATORY COMPARISON OF CONTROLS AND SORE-THROAT PATIENTS

	Sore-throat group		Healthy control group	
	No.	%	No.	%
Culture positive only				
Group A β -haemolytic streptococcus	1	2	3	5
Non-A	12	19	11	18
Positive culture plus antibody response				
Group A	0	0	3	5
Non-A	1	2	1	2
Antibody response only	<u>5</u>	<u>8</u>	<u>2</u>	<u>3</u>
Total of laboratory-positive cases	19	31	20	33
Laboratory-negative cases	<u>43</u>	<u>69</u>	<u>44</u>	<u>67</u>
Total	62	100	64	100

for streptococci; (ii) positive culture plus an antibody response in either or both of the streptococcal antibody assays; and (iii) an antibody response without a positive culture. Laboratory tests were also performed on the control group. An analysis of the cases with a positive laboratory finding in both the sore-throat and control groups is given in Table II.

Discussion

Of the 19 laboratory-positive cases in the sore-throat group only 6 were accompanied by an antibody response, of which only 3 were of real importance (because of possible late sequelae). Of the 3 cases considered to be important 1 was negative on culture with an antibody response in both assays, 1 showed a rise in ASO titre only, and in 1 an organism other than group A streptococcus was cultured although both antibody titres were raised. The antibody response in the other 3 cases consisted of a rise in streptokinase only, which implies that it could be a response to a streptococcus other than group A. In the 13 patients with no

antibody response, 1 was positive on culture for *Strept. pyogenes* (and was therefore only a carrier^{3,4}) and 12 were non-A-positive.

In the control group 6 of the 20 laboratory-positive cases were of interest. In 3 cultures were positive for group A streptococcus and showed an antibody response in both assays. Two had negative cultures, one showed an ASO response only and the other a response in both assays. One subject was positive on culture for a streptococcus other than group A and both titres were raised.

None of the sore-throat or control groups showed a rise in adenovirus antibody titres. Only 5% of the sore-throat cases (3 out of 62) exhibited a rise in *Myc. pneumoniae* antibodies.

In both groups the highest incidence of positive findings was during the autumn, and the clinical findings present in almost two-thirds of the laboratory-positive group were abnormal cervical lymph nodes, an abnormal pharynx and rhinitis.

Conclusion

We conclude that in young adults the clinical and laboratory detection methods for *Strept. pyogenes* as used in this study are unhelpful. Only 3 of the 62 sore-throat patients were actually liable to develop the non-suppurative complications of streptococcal pharyngitis. If sore throat treatment is primarily aimed at the prevention of rheumatic fever (peak incidence being between the ages of 5 and 15 years),⁵ antibiotic therapy is rarely necessary.

The fact that 6 asymptomatic individuals in the control group had either a positive culture plus an antibody response or only an antibody response, which are accepted as the laboratory stigmata of group A β -haemolytic streptococcal infection, is noted as an additional confusing factor.

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