

Group G Streptococcal Infections on a Dermatological Ward

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Groups A, B, C and G streptococci were cultured from 63 consecutive in-patients recruited between November 1987 and April 1988 and monitored until the end of July 1988. Chronic leg ulcers were present in 34 patients. Group G was found in 34 patients, 25 of whom had pyoderma and 3 had sepsis. Six of the patients had no signs of clinical infection, and treatment with antibiotics was therefore withheld. Recurrent phlegmon or erysipelas developed in 2 of 28 patients with clinical Group G infections. Erysipelas developed some 1–7 months later in 3 of the 6 patients who were not initially treated. No significant difference in severity or additional medical conditions was found between the patients with either Group G or Group A streptococci. In comparison, data on all streptococcal cultures at the Department indicated that Group G was isolated 2.6 times as often as Group A streptococci for the in-patients, compared with 1.1 for all patients seen. It is concluded that Group G streptococcal skin infections must be regarded with the same clinical vigilance as Group A infections. **Key words:** Group G streptococcus; Skin infection.

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INTRODUCTION

Group G streptococci were isolated for the first time in 1935, from the vagina in patients with puerperal sepsis (1). Other severe infections such as endocarditis, sepsis and cellulitis have since been reported to be due to Group G streptococci (2–11). In healthy persons, colonization with Group G streptococci has been observed in the pharynx, intestine, vagina and in the skin. In 1–5% of the population living in areas with a temperate climate are pharyngeal carriers, in tropical regions, higher frequencies have been recorded. Fifty-two per cent of 52 children with pharyngitis caused by Group G streptococcus still harboured the organism after observation for 2–4 months (total initial material 134 cases) (12). The incidence of Group G streptococcal infection or pharyngeal carriage shows no seasonal variations (11). Faecal carriage was observed in 2% of pregnant women, but vaginal carriage is uncommon in both pregnant and in non-pregnant women (11). In moist skin areas such as in the toe webs and in the perineal area, carriage was observed in about 2% (11). Contaminated food and domestic animals are other sources of infections in humans (11). Except for cellulitis and erysipelas, reports of skin infections caused by Group G streptococci are uncommon. The present investigation was prompted by an increase in Group G streptococcal skin infections in our Department. A clinical material was collected over a 6-month period in order to study these

infections and compare them with Group A streptococcal infections. The results are reported here.

MATERIAL AND METHODS

Sixty-three consecutive in-patients with a positive streptococcal skin culture were included in the study. They were admitted to the Department of Dermatology, Karolinska Hospital during the period November 1st, 1987 to April 12th, 1988 and observed until the end of July 1988 (Table I). The principal reason for admittance was skin infection or ulcers, old age, or sociomedical causes. The patients were recruited from a 1.5 million urban area. The files of the hospital's Microbiological Unit provided data on all cultures made in the hospital during November 1987–December 1988. Altogether 18,729 specimens were cultured.

Microbiological techniques

Swabs from ulcers or infected areas (skin, throat and intertriginous areas) were cultivated anaerobically on Oxoid Blood Agar Base supplemented with 5% sheep erythrocytes for 18–24 h at 37°C. Transportation time never exceeded 8 h. Isolates were serologically typed with the Streptex Rapid Latex testing system (Wellcome Diagnostics, Dartford, Kent, England). The streptococcal strains were not tested for sensitivity to penicillins.

Treatment

Fever, infected exudative eczema, ulcers, erysipelas, cellulitis or sepsis were regarded as indications for treatment with systemic antibiotics. Penicillin was used in 7 cases, dicloxacillin in 10 and both in combination in 16 cases. Other treatments included erythromycin, cephalosporins or clindamycin. Besides the antibiotics, skin lesions were either left in the open, or were treated topically with solutions of 0.05–0.25% potassium permanganate or 0.9% sodium chloride, eczema was treated with topical corticosteroids. Control cultures were made after discontinuing antibiotics, to ensure elimination of the streptococci.

Scoring and tests

The severity of the Group G streptococcal infections was compared with the Group A infections by scoring sepsis and phlegmon/gangrene as 3, erysipelas as 2 and impetiginous infection as 1. Non-parametric

Table I. Streptococcal species (*str spp*) obtained in 35 males (*m*) and 28 females (*f*)

Upper panel gives data for patients; bottom panel, numbers of cultures. In 4 cultures, Groups A & G were both found (summarized in column A). In 1 culture, Groups G & B were both found (included in column G). Co-cultures with Gram-negative rods (Gram-neg) or *S. aureus* are indicated

Group	A	B	C	G
Mean age	58	66	55	64
Range	29–91	22–92	23–76	19–84
f/m	1/12	7/6	0/3	20/14
<i>str spp</i>	35	35	7	72
+ Gram-neg	0	7	3	11
+ <i>S. aureus</i> ^a	15	14	3	45

^a*S. aureus* strains produced β-lactamase.

Table II. Localization of streptococcal skin infections

	Streptococcal Group				
	A	B	C	G	Sum
Lower extremities	8	3	1	25	37
Trunk lesions	1	3	—	5	9
Arms and hands	4	2	—	1	7
Genital region	—	4	2	3	9
Face	—	1	—	—	1
Total	13	13	3	34	63

methods using the χ^2 -test with Yates' correction, or Mantel-Haenszel or Kruskal-Wallis' methods were applied to test significance.

RESULTS

In 18,729 specimens, cultured at the Microbiological Unit of the hospital during November 1987–December 1988, positive streptococcal cultures were obtained in 885 cases (4.7%). In cultures from the Department of Dermatology, the incidence of Group G streptococci was 2.3-fold higher than in cultures originating from other departments of the hospital. The corresponding figures for Group A and Group C streptococci were 5.9-fold and 4.3-fold, respectively. At the Department of Dermatology the ratio of Group G/A streptococci in all patients treated was 1.1. In the patients admitted to the dermatological wards, Group G streptococci were isolated 2.6 times as often as Group A (Table II). This was significantly different from the overall ratio, 1.1 ($p < 0.003$).

In the whole material, 34 of the 63 patients had leg ulcers of various etiology. Lesions on the lower legs comprised the major source of the isolates in the 34 patients with Group G streptococci, leg ulcers being present in 21 of these patients (Table II). The ulcers were port of entry in the complicating infections (see Table III). The severity of Group G streptococcal infections was scored and compared with that of Group A infections, but no significant difference was found between them ($p = 0.14$).

The Group G streptococcal infections presented as pyodermas in 25 cases and sepsis in 3 (Table III). Two of the 28 patients had recurrences with a new phlegmonous infection or erysipelas during the survey period. Seventy-four percent of patients with Group G and 77% of those with Group A streptococcal infections were treated with β -lactamase stable drugs. Antibiotic treatment was withheld in 6 patients with group G streptococci who had no signs of clinical infection at the time of admittance. However, erysipelas developed in 3 of these 6 patients within 1 to 7 months thereafter.

Other medical conditions are summarized in Table IV. Concomitant disease was recorded in 38–67% of the patients, more often affecting patients with Group G streptococci (19/34) than those infected by the other streptococci (14/29). However, the ratios were not statistically different.

Table III. Clinical condition of the streptococcal skin infections

Category	Streptococcal Group				
	A	B	C	G	Sum
Total number	13	13	3	34	63
<i>Cases treated with antibiotics:</i>					
Phlegmon, gangrene	0	0	0	4	4
Erysipelas	4	0	0	6	10
Impetiginous skin infection	2	0	0	4	6
<i>Dermatoses</i>					
Eczema	4	3	0	5	12
Leg ulcers	3	3	1	6	13
Total pyodermas	13	6	1	25	45
Sepsis	0	0	0	3	3
<i>Cases not treated with antibiotics:</i>					
Eczema	0	2	2	2	6
Psoriasis	0	3	0	1	4
Leg ulcers	0	2	0	3	5
Total dermatoses	0	7	2	6	15

DISCUSSION

In this material, the clinical presentation of skin infections caused by Group G and A streptococci did not differ. A patient especially in old age with a leg ulcer and skin infection gained admission.

Risk groups

The current literature draws attention to risk factors for the acquiring of streptococcal infections, especially in patients with chronic leg ulcers (9, 11). Nosocomial contamination and spreading of strains place such patients at risk. In addition pet animals may be a risk seldom thought of in the clinical setting (5, 6, 11). Gaunt & Seal argued for a concomitant disease in order to promote clinical infection with Group G streptococci (11). Sepsis due to Group G streptococcal infection was seen especially in patients who had a debilitating disease and/or were of advanced age (4). Yet, the ratio between Group G/A streptococcal infections was only 0.46 among patients in a cancer hospital (10). One possible reason for this low propor-

Table IV. Concomitant medical conditions

Mammary cancer and mycosis fungoides were included in the malignant group. In the autoimmune group, cases of systemic lupus erythematosus, rheumatoid arthritis, pemphigus vulgaris, pemphigoid and leukocytoclastic vasculitis were combined. The peripheral vascular diseases included combined arterial and venous insufficiency with ulcerations

Category	Streptococcal Group			
	A	B	C	G
Malignancy	0	1	0	2
Autoimmune disease	3	0	1	3
Diabetes	0	1	0	5
Vascular disease	3	5	1	9

tion was thought to be the low virulence of Group G streptococci in general at that time (1960–68).

In our material, 28 of 34 patients with Group G streptococci had signs of clinical infections. However, this proportion was not significantly different from 13 of 13 patients with Group A infections ($p = 0.09$). Therefore, we could not state that in our patients virulence factors differed depending on the type of streptococci present.

Importance of double infections

Staphylococcus aureus and streptococci were often co-cultivated in samples obtained from our patients (Table I). In an animal model, spreading of Group G streptococcal infection was promoted by proteolytic enzymes produced by *S. aureus* (13, 14). *S. aureus* was also found in 5 of 7 patients with sepsis, phlegmon and gangrene, a ratio not significantly different from that for the total material, 77/132 cultures, presented in Table I.

Treatment

The literature emphasizes the high sensitivity of Group G streptococci to penicillins. However, a decreased sensitivity to these drugs in 60% of the strains has been reported (3, 15, 16). Recommendations often suggest a combination of penicillin and β -lactamase stable antibiotics (2). The presence of *S. aureus* in conjunction with streptococci in many of our cases (Table I) was the reason for following this recommendation. Resistance to tetracycline is commonly encountered (11).

It is concluded that Group G streptococci share the same clinical spectrum as Group A and that they may create a serious condition leading to sepsis, gangrene or erysipelas. Leg ulcers often serve as a port of entry for these infections. We endorse the recommendations for treatment alluded to above and possibly invariably treat patients with Group G streptococci even if an infection is not clinically observed.

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REFERENCES

1. Lancefield RC, Hare R. The serological differentiation of pathogenic and non-pathogenic strains of haemolytic streptococcae from parturient women. *J Exp Med* 1935; 61: 335–349.
2. Tuazon CU. Group G streptococcus. *Am J Med Sci* 1980; 279: 121–124.
3. Finch RG, Aveline A. Group G streptococcal septicemia: clinical observations and laboratory studies. *J Infection* 1984; 9: 126–133.
4. Nordlander IM, Thal E, Tunevall G. Occurrence and significance of hemolytic streptococci Group B-U infectious disease. *Scand J Infect Disease* 1975; 7: 35–37.
5. Rolston KVI, Chandrasekar PH, LeFrock JL. Clinical features and antimicrobial therapy of infections caused by group G streptococci. *Infection* 1985; 5: 203–206.
6. Gaunt N, Rogers K, Seal D, Denham M, Lewis J. Necrotizing fasciitis due to Group C and G haemolytic streptococcus after chiropody. *Lancet* 1984; i: 516.
7. Rolston KVI. Susceptibility of Group B and Group G streptococci to newer antimicrobial agents. *Eur J Clin Microbiol* 1986; 5: 534–536.
8. Dickie AS, Bremner DA, Say PJ. Group G streptococcal septicemia: report of six cases. *J Infection* 1984; 8: 173–176.
9. Lin AN, Karasik A, Salit IE, Fam AG. Group G streptococcal arthritis. *J Rheumatol* 1982; 9: 424–427.
10. Armstrong D, Blevins A, Luoria DB, Henkel JS, Moody MD, Sukany M. Group B, C and G streptococcal infections in a cancer hospital. *Ann NY Acad Sci* 1970; 174: 511–522.
11. Gaunt PN, Seal DV. Group G streptococcal infections. Review. *J Infection* 1987; 15: 5–20.
12. Breese B, Breese Hall C. Beta hemolytic streptococcal diseases. Boston: Houghton Mifflin, 1978: 145.
13. Vartian C, Lerner PI, Shlaes DM, Gopalakrishna KV. Infections due to Lancefield group G streptococci. *Medicine (Baltimore)* 1985; 64: 75–88.
14. Hladny J, Metz H. Mischinfektionen mit β -hämolisierenden Streptokokken im klinischen Untersuchungsmaterial. *Zentral Bakteriell Mikrobiol Hyg [A]* 1976; 234: 177–188.
15. Rolston KVI, Chandrasekar PH, LeFrock JL. Antimicrobial tolerance in Group C and Group G streptococci. *J Antimicrob Chemother* 1984; 13: 389–392.
16. Noble JT, Tyburski MB, Berman M, Greenspan J, Tenenbaum MJ. Antimicrobial tolerance in Group G streptococci. *Lancet* 1980; ii: 982.