Group G Streptococcal Infections

The role of group G β-hemolytic streptococci (GGS) as significant human pathogens has been firmly established during the past 15 years. These organisms are normal inhabitants of the skin, oropharynx, and gastrointestinal and female genital tracts. Although cutaneous infections and pharyngitis are encountered most often, a wide variety of infections—including potentially life-threatening ones, such as septicemia, endocarditis, meningitis, peritonitis, pneumonitis, empyema, and septic arthritis—have been described.1

Asymptomatic pharyngeal carriage of GGS occurs in up to 23% of humans.2 Symptoms of pharyngitis range from a mild upper respiratory tract infection with coryza to exudative pharyngitis accompanied by fever and lymphadenopathy. The illness is indistinguishable from that produced by group A streptococci, and there is no evidence that antimicrobial therapy modifies either the severity or the duration of symptoms.3 Acute rheumatic fever following GGS pharyngitis has not been described. Type 12 M protein antigen, identical to the nephritogenic antigen of group A streptococci, has been isolated from GGS. However, the role of GGS in precipitating acute poststreptococcal glomerulonephritis is uncertain.

Colonization of the human skin with GGS is frequent, and these organisms are responsible for various cutaneous and subcutaneous infections, including cellulitis, wound infections, and pyoderma. Lymphangitis may accompany such infections, and breaches in the integrity of the skin may provide a portal of entry for bacteremia. A rapid response to antimicrobial therapy is the usual outcome, although surgical intervention might be required in the event of a large collection of pus.

Approximately 5% of asymptomatic puerperal women harbor GGS in their genital tracts, and puerperal sepsis and endometritis have been described.4 The incidence of these infections has declined in recent years. However, GGS have been recognized as a cause of neonatal sepsis.4-8 This infection often occurs in premature or low-birthweight infants, and in the setting of prolonged rupture of membranes. Colonization of the birth canal is the most likely source. The outcome is variable, and complications such as progressive respiratory distress, shock, and disseminated intravascular coagulation are invariably fatal.

Septic arthritis due to GGS usually occurs in joints previously damaged by trauma or underlying diseases such as rheumatoid arthritis, or in prostatic joints. The clinical course is often protracted, with recurrent sterile joint effusions requiring frequent drainage and a slow response to antimicrobial therapy despite in vitro susceptibility.9,10

Approximately 8% to 11% of streptococcal bacteremias are caused by GGS. Several authors11-13 have noticed an association of GGS bacteremia with malignant neoplasms, with the frequency ranging from 21% to 65%. Polymicrobial bacteremias are not uncommon, with Staphylococcus aureus being the most frequently isolated copathogen, suggesting a common cutaneous source. Patients with primary infections such as pneumonitis, septic arthritis, ophthalmitis, and meningitis are also often bacteremic. Infections of the bloodstream alone respond favorably to antimicrobial therapy.

Group G streptococcal endocarditis tends to occur in older patients with multiple underlying disorders. Both native and prosthetic valves can be infected, and left-sided involvement is more common. The onset is generally abrupt, and the disease appears to progress more rapidly than that caused by other streptococcal species. Valvular destruction and perivalvular infection may occur, and metastatic foci of infection are not infrequent. Response to single-agent therapy with β-lactams has been poor despite in vitro susceptibility. The reasons for this lack of response are not clear, but host factors should be considered, in addition to failure of antimicrobial therapy.13 Although no comparative data are available, a review of the literature and my personal experience favor the use of a bactericidal combination of antibiotics (eg, penicillin plus gentamicin) in preference to single-agent therapy. This might be particularly important in patients with impaired host defense mechanisms, since they tend to rely more heavily on antimicrobial agents for eradication of infections than do their healthier counterparts.

Group G streptococci are susceptible in vitro to various antimicrobial agents, including penicillin G, the ureidopenicillins, most cephalosporins, vanco-
mycin, and erythromycin.\textsuperscript{14} Combinations of gentamicin with penicillin, cefotaxime, and vancomycin have been reported to be synergistic against 80% to 90% of isolates.\textsuperscript{15} Varying degrees of tolerance (minimal bactericidal concentration to minimal inhibitory concentration ratio of $\geq$32) to $\beta$-lactam agents and vancomycin have been reported.\textsuperscript{16,17} Combinations of gentamicin with $\beta$-lactams and of gentamicin or rifampin with vancomycin are bactericidal against tolerant isolates.\textsuperscript{16} The clinical significance of tolerance is not fully understood, and no serious infections with tolerant GGS have yet been described. Should such infections occur, it would be prudent to use a bactericidal combination as therapy.

In conclusion, GGS are an infrequent but important cause of human infections. In recent years a large number of laboratories have started grouping streptococcal isolates, and GGS are being recognized more frequently, but there seems also to have been a genuine increase in the incidence of these infections. Most infections caused by GGS respond rapidly to antimicrobial therapy, usually with penicillin G or other $\beta$-lactams. However, deep-seated infections, such as endocarditis and septic arthritis, probably require more aggressive therapy with bactericidal antimicrobial combinations.

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**References**


**The Conundrum of Cushing's Syndrome**

Cushing's syndrome has presented clinicians with fascinating diagnostic and therapeutic difficulties for more than half a century. Additional causative mechanisms and different means of treatment continue to be advanced. Although the syndrome usually results from a disordered function originating in the pituitary or the adrenal glands, pathologic hypercortisolism is also well known to result from ectopic secretions. The ectopic corticotropin (ACTH) syndrome, which is characteristic of the latter disorders, is usually associated with very high levels of ACTH secretion, great excesses of cortisol, bilateral adrenal hypertrophy, pigmentation, hypokalemia, hyperglycemia, and hypertension. The very high levels of cortisol accelerate the pace of the disorder, and the underlying malignancy modifies the clinical expression of the endocrine disease. Instead of a

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See also p 929.