JOURNAL OF CLINICAL MICROBIOLOGY, Oct. 1999, p. 3380–3383 Vol. 37, No. 10
0095-1137/99/$04.00+0

Antibiotic Susceptibilities of Group C and Group G Streptococci Isolated from Patients with Invasive Infections: Evidence of Vancomycin Tolerance among Group G Serotypes

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Received 8 February 1999/Returned for modification 2 May 1999/Accepted 10 July 1999

A retrospective review of medical records for 32 patients with invasive group C streptococcus (GCS) or group G streptococcus (GGS) infections was performed. MICs and minimum bactericidal concentrations (MBCs) of penicillin, erythromycin, and vancomycin for all isolates were obtained. Tolerance of vancomycin, defined as an MBC 32 or more times higher than the MIC, was exhibited by 18 GGS isolates (54%). The identification of tolerance in clinical isolates of GGS and GCS may have clinical implications in treating these seriously ill patients.

There is increasing interest in the role of Lancefield group C streptococci (GCS) and group G streptococci (GGS) as emerging nosocomial and opportunistic pathogens (31, 35). The spectrum of human infection caused by these organisms includes primary and secondary bacteremia in normal and immunocompromised hosts, as well as cellulitis, endocarditis, skin and wound infections, meningitis, arthritis, osteomyelitis, pneumonia, abscesses, puerperal infections, and pharyngitis (2, 4–11, 13–16, 19, 20, 26, 31, 35).

Besides being classified by the Lancefield group carbohydrate, the β-hemolytic streptococci are subdivided on the basis of whether they form large colonies or small colonies on sheep blood agar plates (BAP) (6, 7, 10, 13, 15). The large-colony phenotypes of group A and group B are associated with the pathogenic species *Streptococcus pyogenes* and *Streptococcus agalactiae*. Similarly, GCS and GGS large-colony phenotypes are those usually associated with human infection. GCS and GGS are classified in the same subspecies, *Streptococcus dysgalactiae* subsp. *equisimilis* subsp. nov. (34), and are termed *S. pyogenes*-like because these species share a number of virulence factors with group A streptococci (*S. pyogenes*). Small-colony-forming species are placed in the *Streptococcus anginosus* group (formerly known as *Streptococcus milleri*) and are less common causes of abscess formation and bacteremia (12, 15, 32).

The majority of GCS and GGS strains demonstrate in vitro susceptibility to penicillins, vancomycin, erythromycin, and cephalosporins (3, 30). Antimicrobial tolerance, defined as a minimum bactericidal concentration (MBC) 32 or more times higher than the MIC, among GCS and GGS has been reported for penicillin and other agents (24, 27, 29). Only a few clinical isolates have been reported to exhibit tolerance of vancomycin (24, 29). We previously reported tolerance of vancomycin among pharyngeal isolates of non-group A β-hemolytic streptococci (mostly GCS and GGS) from children (36). We chose to investigate further these antibiotic susceptibility patterns among GCS and GGS isolated from patients with invasive infections (bacteremia and meningitis, etc.), for whom similar findings of tolerance may have clinical implications.

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(The study was performed at the Alfred I. duPont Hospital for Children, Wilmington, Del. This work was presented in part at the 97th General Meeting of the American Society for Microbiology held in May 1997 in Miami Beach, Fla. [37].)

At Christiana Care Health Systems a retrospective chart review was performed with 32 patients from whom GCS and GGS were isolated from sterile sites between December 1991 and March 1996. Clinical data were collected on all patients. Bacterial isolates were recovered from frozen storage (−70°C) for further evaluation. Isolate identification was performed with the API 20S Strep Strip (bioMerieux Vitek, Hazelwood, Mo.). Serotyping for GCS and GGS was performed with the PathoDx agglutination kit (Remel, Lenexa, Kans.).

MICs of penicillin, erythromycin, and vancomycin were performed by using National Committee for Clinical Laboratory Standards (NCCLS) broth microdilution methods (22). Tests were performed in cation-adjusted Mueller-Hinton broth with lysed horse blood (Remel; lot 5517). Dilutions tested ranged from 16 to 0.016 μg/ml for all drugs. Plates were prepared on-site (100 μl per well), and antibiotic powders were supplied by the respective manufacturers. Microtiter plates were prepared to include a positive growth control well and a medium sterility well. Plates were stored at −70°C until use and thawed completely at room temperature before inoculation. Organisms were grown in Trypticase soy broth (Becton Dickinson, Cockeysville, Md.; lot 100K7DEJS) for 2 h and then maintained at a 0.5 McFarland standard. Microtiter plates were inoculated with 0.01 ml of the standardized, diluted organism suspension and then incubated at 35°C in 6% carbon dioxide for 20 h. The MIC was interpreted as the lowest concentration of drug at which no growth was visible in the microtiter well. The NCCLS breakpoints for streptococci were used to interpret MIC results (23).

Wells with no visible growth were subcultured on BAP to determine the MBC (50 μl from each well). The BAP were incubated for 24 h at 35°C in carbon dioxide. The MBC was interpreted as the lowest concentration of drug at which fewer than five colonies were observed on the BAP.

All MIC and MBC assays were performed in duplicate for reliability. Broth macrodilution methods according to NCCLS standard procedures were used to confirm MIC and MBC broth microdilution results (21, 22).

Streptococcus pneumoniae ATCC 49619 was used for quality control for all antimicrobials and was tested with each batch of

TABLE 1. Clinical data for patients from whom GCS and GGS streptococci were isolated[a]

Patient	Age (yr)	Sex	Underlying disease(s) and/or condition	Diagnosis	Isolation site(s)	Outcome
1	NA	NA	NA	NA	NA	NA
2	65	F	Cirrhosis, leg ulcers	Cellulitis/sepsis	Blood	Died
3	88	F	CAD, CVA, HTN	Pneumonia	Blood	Survived
4	NA	NA	NA	NA	NA	NA
5	NA	NA	NA	NA	NA	NA
6	82	M	CAD, NIDDM	Cellulitis	Blood	Survived
7	67	M	IDDM, COPD	Foot ulcer	Blood	Died
8	40	M	HIV, IVDA	Endocarditis	Blood	Survived
9	81	M	CAD, CVA, CHF	Sepsis	Blood	Survived
10	82	M	CA, IDDM	Cellulitis	Blood, wound	Died
11	71	M	Knee replacement	Arthritis	Synovial fluid, blood	Survived
12	34	F	None	Meningitis	Cerebrospinal fluid	Survived
13	34	M	IVDA	Upper extremity, abscess	Abscess	Survived
14	84	F	CLL, CVA	Cellulitis	Blood	Died
15	88	M	IDDM, HTN	Sepsis	Blood	Survived
16	37	M	EtOH	Sepsis	Blood	Survived
17	59	M	CLL, TB, HIV	Sepsis	Blood	Died
18	69	M	NIDDM, CAD	Cellulitis	Blood	Survived
19	50	M	None	Patellar bursitis	Bursal fluid	Survived
20	58	F	SLE, NIDDM, HTN	Cellulitis	Blood	Survived
21	55	M	EtOH, seizures	Cellulitis	Blood	Survived
22	76	F	IDDM, HTN, dialysis	Sepsis	Blood, urine, peritoneal fluid	Died
23	55	M	NIDDM	Cellulitis, osteomyelitis	Blood, bone, skin wound	Survived
24	NA	NA	NA	NA	NA	NA
25	NA	NA	NA	NA	NA	NA
26	67	M	COPD, HTN, NIDDM	Lower extremity, ulcers	Blood	Survived
27	71	M	HTN, NIDDM	Cellulitis	Blood	Survived
28	38	M	HTN, obesity	Cellulitis	Blood	Survived
29	85	F	Breast CA, Crohn's	Cellulitis	Blood	Survived
30	32	M	Appendicitis	Abscess	Blood	Survived
31	70	F	Diverticulitis	Abscess	Abscess fluid	Survived
32	69	M	Prostate CA, DVT	Sepsis, polyarthritis	Blood	Survived

[a] Abbreviations: CAD, coronary artery disease; CVA, cerebrovascular accident; HTN, hypertension; NIDDM, non-insulin-dependent diabetes mellitus; IDDM, insulin-dependent diabetes mellitus; COPD, chronic obstructive pulmonary disease; HIV, human immunodeficiency virus infection; CHF, congestive heart failure; IVDA, intravenous drug abuse; CLL, chronic lymphocytic leukemia; CA, cancer; TB, tuberculosis; EtOH, alcohol abuse; DVT, deep-vein thrombosis; SLE, systemic lupus erythematosus; NA, not available; M, male; F, female.

microtiter plates. The results obtained were consistently within acceptable ranges for all drugs.

Between December 1991 and March 1996, 32 sterile-site isolates, 27 GGS and five GCS, were identified and retrieved for study. The demographic and clinical characteristics of the 27 patients for whom data were available are shown in Table 1.

The microbiological and antibiotic susceptibility data, including MIC and MBC broth microdilution results, are summarized in Table 2. Of the 27 GGS isolates, 23 were identified to species level as *S. dysgalactiae* subsp. *equisimilis* (large-colony phenotype), three were *S. anginosus*, and one isolate became nonviable prior to completion of species identification. Among the five GCG isolates, one was *S. dysgalactiae* subsp. *equisimilis* (large-colony phenotype) and four were *S. anginosus*. All MIC and MBC results obtained by broth macrodilution methods were nearly identical to the broth microdilution results presented in Table 2.

All isolates were susceptible to penicillin, and their MICs ranged from ≤0.016 to 0.06 μg/ml. The MBCs ranged between ≤0.016 and 0.5 μg/ml, with no evidence of tolerance. Three isolates, two GGS (large-colony phenotype) and one GCS (large-colony phenotype), were resistant to erythromycin (MICs > 16 μg/ml). The range of erythromycin MICs was ≤0.016 to >16 μg/ml. All isolates were susceptible to vancomycin (MICs between 0.12 and 0.5 μg/ml). Eighteen isolates of GGS exhibited tolerance of vancomycin (MBCs 32 or more times higher than the MICs [Table 2]).

The purpose of this study was to characterize the antibiotic susceptibility patterns of GCS and GGS isolated from sterile clinical sites. The characteristics of patients with GCS and GGS infections (predominantly bacteremia) in our study are consistent with previous reports linking these infections with underlying malignancy or immune system compromise (2, 4, 5, 9, 19, 31, 35). Given the retrospective nature of this study, no conclusions on the relationship between patient outcome and the presence of a tolerant organism can be made, because the majority of patients were at high risk and were not uniformly treated with vancomycin alone.

Our in vitro findings support the use of penicillin G as the antimicrobial agent of choice for GCS and GGS infections. All MICs were less than 0.03 μg/ml, and tolerance was not identified. All isolates in our study were susceptible to vancomycin (MICs ranging between 0.12 and 0.5 μg/ml), but 18 of 32 (54%) GGS demonstrated tolerance. No GCS isolates exhibited tolerance. Since there are few reports in the literature of GCS isolates examined for vancomycin tolerance, the significance of this difference between GCS and GGS is unclear.

Noble et al., in one of the most widely cited reports of vancomycin tolerance among GGS, reported eight of nine clinical isolates that were tolerant of vancomycin (24). Rolston et

TABLE 2. MICs and MBCs of penicillin, erythromycin, and vancomycin for sterile-site isolates

Isolate	Serotype/species	Penicillin		Erythromycin		Vancomycin	
		MIC (μg/ml)	MBC (μg/ml)	MIC (μg/ml)	MBC (μg/ml)	MIC (μg/ml)	MBC (μg/ml)
1	G/group G[a]	≤0.016	≤0.016	0.12	0.16	0.25	0.25
2	G/group G	≤0.016	≤0.016	0.12	8	0.25	8[b]
3	G/group G	≤0.016	≤0.016	0.12	0.25	0.25	8[b]
4	C/*S. anginosus*	0.06	0.06	≤0.016	2	0.5	0.5
5	C/*S. anginosus*	0.03	0.03	≤0.016	0.12	0.5	0.5
6	G/NA[c]	≤0.016	≤0.016	0.12	0.5	0.12	0.25
7	G/group G	≤0.016	≤0.016	0.12	0.5	0.12	8[b]
8	G/group G	≤0.016	≤0.016	>16	>16	0.25	8[b]
9	G/group G	≤0.016	≤0.016	0.12	0.5	0.25	8[b]
10	G/NA	≤0.016	≤0.016	0.12	0.25	0.25	0.25
11	G/group G	≤0.016	≤0.016	0.12	16	0.5	8
12	G/group G	≤0.016	≤0.016	0.12	0.5	0.25	8[b]
13	C/*S. anginosus*	≤0.016	0.03	≤0.016	4	0.5	0.5
14	G/group G	≤0.016	≤0.016	0.12	1	0.25	8[b]
15	C/*S. anginosus*	0.06	0.06	≤0.016	0.12	0.5	1
16	G/group G	≤0.016	0.06	0.12	0.25	0.25	16[b]
17	G/group G	≤0.016	≤0.016	0.12	16	0.25	4
18	G/group G	≤0.016	≤0.016	0.12	0.25	0.25	8[b]
19	G/group G	≤0.016	≤0.016	0.12	0.5	0.12	8[b]
20	G/group G	≤0.016	≤0.016	0.12	1	0.25	4
21	G/group G	≤0.016	≤0.016	0.12	1	0.12	8[b]
22	G/group G	≤0.016	≤0.016	0.12	8	0.25	8[b]
23	G/group G	≤0.016	≤0.016	0.12	4	0.12	16[b]
24	G/*S. anginosus*	0.06	0.06	0.06	0.25	0.5	16[b]
25	G/group G	≤0.016	≤0.016	0.12	4	0.25	2
26	G/group G	≤0.016	≤0.016	0.12	8	0.25	8[b]
27	G/group G	≤0.016	≤0.016	0.12	0.5	0.25	8[b]
28	C/group C[a]	≤0.016	≤0.016	4	>16	0.25	4
29	G/group G	≤0.016	≤0.016	8	>16	0.25	4
30	G/*S. anginosus*	0.06	0.5	≤0.016	2	0.5	16[b]
31	G/*S. anginosus*	0.03	0.06	≤0.016	0.25	0.5	2
32	G/group G	≤0.016	≤0.016	0.12	4	0.12	8[b]

[a] Unless otherwise specified, species listed as group G or group C belong to *S. dysgalactiae* subsp. *equisimilis* or large-colony phenotype.
[b] Vancomycin-tolerant strains.
[c] NA, not available.

al. examined the in vitro activity of nine antimicrobial agents against 35 GGS and 26 GCS isolates from various clinical sites (29). One GGS isolate exhibited tolerance of vancomycin. The two reports in the literature of tolerance to vancomycin have shown significant variability in the percentage of tolerant GGS (eight of nine in Noble's study and one of 35 in Rolston's study). The causes of variability are hypothetical, given the small amount of previous data available, but may include the year of collection, geography, source of the isolate, and previous antibiotic use.

The significance of in vitro vancomycin tolerance is uncertain, and our findings do not necessarily reflect clinical efficacy. Recent evidence presented by Novak et al. demonstrates a molecular mechanism for vancomycin tolerance in *S. pneumoniae*. A rabbit meningitis model utilized in their studies indicated the failure of vancomycin therapy to eradicate tolerant organisms from the cerebrospinal fluid (25). Concerns about potential antibiotic tolerance in GCS and GGS and reports of clinical failures in patients with severe infections have led many authors to recommend combination therapy for synergy (aminoglycoside plus a cell wall-active agent) in the initial treatment of these patients (1, 17, 18, 27, 28, 31, 33, 35).

Our in vitro findings suggest that among high-risk patients with invasive GCS and GGS infections who cannot be treated with penicillin, tolerance of other antimicrobial agents, including vancomycin, should be closely monitored.

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