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Brief report

Identification and characterization of catheter-related bloodstream infections due to viridans group streptococci in patients with cancer

Samuel A. Shelburne III MD, PhD [a,b], Anne-Marie Chaftari MD [a], Mohamed Jamal PhD, MS, BS [a], Iba Al Wohoush MD [a], Ying Jiang MS [a], Shaadi Abughazaleh BS [a], Javier Cairo MD [a], Sammy Raad BS [a], Labib Debiane MD [a], Issam Raad MD [a,*]

[a] Department of Infectious Diseases, M.D. Anderson Cancer Center, Houston, TX
[b] Department of Genomic Medicine, M.D. Anderson Cancer Center, Houston, TX

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Viridans group streptococci (VGS), a leading cause of bloodstream infection (BSI) in cancer patients, are thought to arise from the gastrointestinal tract. We sought to determine whether central venous catheters may serve as the source of VGS BSI, and to compare the ability of the newly proposed mucosal barrier injury laboratory-confirmed BSI definition to assign a VGS BSI source compared with the catheter-related BSI definition.

Gram-positive viridans group streptococci (VGS) are leading causative agents of bloodstream infection (BSI) in neutropenic cancer patients.[1-4] It has long been presumed that the source of VGS BSI is the gastrointestinal tract, especially the oral cavity[1,5,6]; however, given that VGS bacteremia occurs predominantly in cancer patients receiving chemotherapy,[1,5,7] the vast majority of patients with VGS BSI have a central venous catheter (CVC) in place. Presently, there are no data available regarding CVCs as a source of VGS BSI.

In an attempt to recognize the possibility of a gastrointestinal source for certain BSIs, the Centers for Disease Control and Prevention (CDC) recently introduced the concept of mucosal barrier injury laboratory-confirmed BSI (MBI-LCBI) for particular organisms, including VGS.[8] After caring for several patients with VGS bacteremia that appeared to have originated from a CVC but met the CDC's criteria for MBI-LCBI, we conducted the present study to test 2 hypotheses: VGS are capable of causing catheter-related BSI (CRBSI), and the current MBI-LCBI definition does not accurately classify the source of VGS BSI.

METHODS

Settings and definitions

This Institutional Review Board–approved study was performed at the University of Texas M.D. Anderson Cancer Center between January 2000 and April 2010. The medical records of patients with monomicrobial VGS bacteremia were reviewed to identify patients who met the CDC's definition of primary laboratory-confirmed BSI. Such patients who also had available quantitative blood culture data were then assessed for CRBSI based on the Infectious Diseases Society of America (IDSA) guidelines (Fig 1).[9] Patients who lacked a CVC were classified as non-CRBSI. The patients with an indwelling CVC were then classified as follows: patients with a CVC:peripheral colony-forming unit (CFU) ratio of $\geq$3:1 were diagnosed with CRBSI, those with a CFU ratio of $\leq$1:1 were considered non-CRBSI, and those with a CFU ratio between 3:1 and 1:1 were excluded from the analysis, because such patients may or may not have CRBSI.[10] Cases were defined as central line–associated BSI (CLABSI) or MBI-LCBI based on the CDC criteria (http://www.cdc.gov/nhsn/pdfs/pscmanual/4psc_clabscurrent.pdf).

RESULTS

VGS are capable of causing CRBSI

During the study period, we identified 82 patients who met all of our inclusion criteria. Patient characteristics are

* Address correspondence to Dr Issam Raad, MD, Chair, Department of Infectious Diseases, M.D. Anderson Cancer Center, Unit 1460, 1515 Holcombe Blvd, Houston, TX 77030.
E-mail address: iraad@mdanderson.org (I. Raad).
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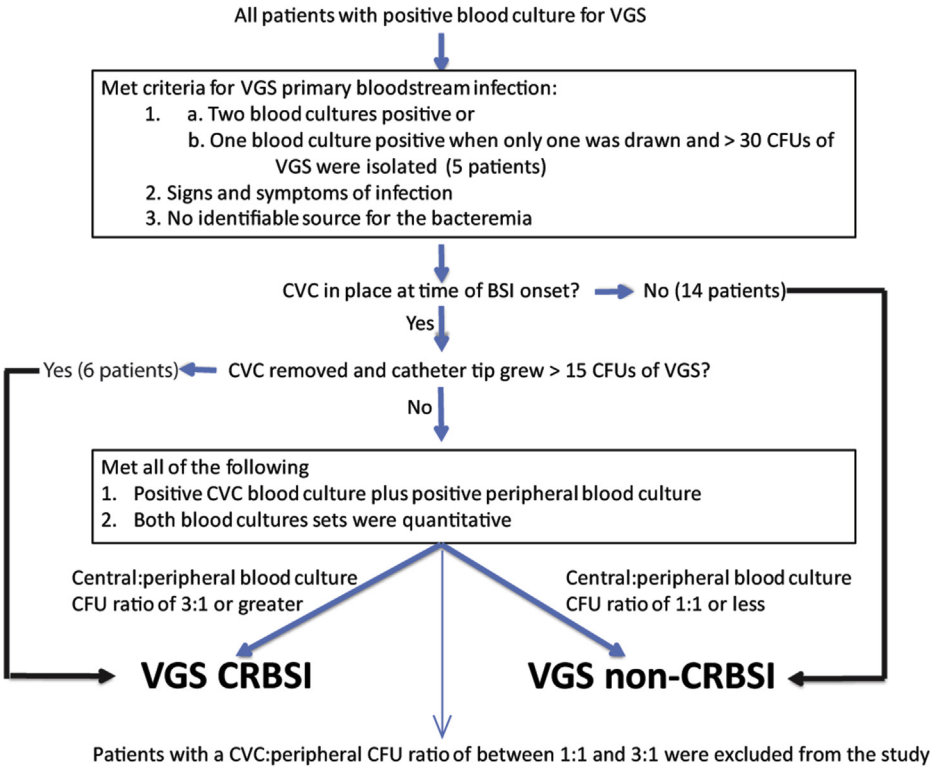

Fig 1. Schematic of patient inclusion.

presented in Table 1. Based on the IDSA criteria, 27 patients were classified as definite VGS CRBSI, and 55 patients were classified as non-CRBSI.[9] Compared with the non-CRBSI patients, patients with VGS CRBSI were more likely to be neutropenic, to be thrombocytopenic, and to be receiving a fluoroquinolone antibiotic as prophylaxis (Table 1). They also were more likely to be infected with fluoroquinolone-resistant and ceftriaxone-susceptible VGS isolates. Consistent with our CRBSI designations, patients with VGS CRBSI were more likely to have their catheter removed within 7 days of BSI onset ($P < .01$). Although there was a trend toward a higher response rate in the VGS CRBSI group ($P = .07$), there was no significant difference in the rate of ICU admission or death between the 2 patient groups (Table 1).

Table 1
Comparison of the CRBSI and non-CRBSI groups

Characteristic	All patients (n = 82)	CRBSI (n = 27)	Non-CRBSI (n = 55)	*P* value
Age, years, mean (SD)	45 (18)	48 (18)	45 (20)	.45
Male sex, n (%)	47 (57)	14 (52)	33 (60)	.64
Hematologic malignancy, n (%)	64 (78)	24 (89)	40 (73)	.16
Bone marrow transplant, n (%)	24 (29)	10 (37)	14 (26)	.30
Neutrophil count <500/μL, n (%)	68 (83)	26 (96)	42 (76)	.03
Platelet count (K/μL), median (IQR)	21 (12-43)	15 (7-27)	23 (13-54)	.03
Platelet count <10,000/μL, n (%)	17 (21)	10 (37)	7 (13)	.02
Duration of neutropenia, d, mean (SD)*	11 (17)	11 (13)	11 (18)	.87
Receipt of fluoroquinolone prophylaxis, n (%)	48 (59)	21 (78)	27 (49)	.01
Receipt of chemotherapy, n (%)†	70 (85)	25 (93)	45 (82)	.40
Interval from CVC insertion to positive blood culture, d, mean (SD)	65 (82)	63 (66)	67 (102)	.84
Susceptibility to fluoroquinolone, n (%)	31 (37)	5 (19)	26 (46)	.01
Susceptibility to ceftriaxone, n (%)	71 (86)	27 (100)	44 (83)	.03
Susceptibility to penicillin, n (%)	47 (57)	19 (70)	28 (54)	.23
Admitted to intensive care unit, n (%)	10 (12)	3 (11)	7 (13)	1.0
CVC removal, n (%)‡	7 (8)	6 (22)	1 (2)	<.01
Appropriate antibiotic treatment, n (%)§	74 (90)	26 (96)	48 (87)	.26
Clinical response within 96 hr, n (%)	58 (70)	23 (85)	35 (63)	.07
30-day mortality, n (%)	11 (13)	3 (8)	8 (15)	.51

*Before BSI onset.
†Within 30 days of BSI onset.
‡Within 7 days of BSI onset.
§Receipt of an effective antimicrobial within 24 hours of patient presentation.

The MBI-LCBI definition does not accurately identify VGS CRBSI

We next sought to compare how the CLABSI and MBI-LCBI definitions would assign a BSI source for the patients in our cohort, using our CRBSI definition as the standard. Among our 82 patients, 68 (83%) were classified with CLABSI, whereas 71 (87%) met the criteria for MBI-LCBI. All of the 71 patients who met the criteria for the MBI-LCBI definition did so because they were neutropenic at the time of blood culture positivity. By definition, all of the CRBSI patients were classified as having CLABSI, but 41 of the 55 patients (75%) who met the non-CRBSI definition also were classified as having CLABSI.

Because MBI-LCBI was designed to identify a noncatheter BSI source,[8] we considered the MBI-LCBI and CRBSI designations to be in agreement when a patient was classified differently by the 2 schemes (eg, "yes" for CRBSI, "no" for MBI-LCBI). Using this approach, we found that the MBI-LCBI definition was in good agreement with the CRBSI definition in non-CRBSI patients (45 of 55; 82%), but that 26 of the 27 patients (96%) with CRBSI were classified as having MBI-LCBI. Thus, in our VGS BSI cohort, both