Clinical and Epidemiological Features of Enterococcus casseliflavus/flavescens and Enterococcus gallinarum Bacteremia: A Report of 20 Cases

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The clinical significance of intrinsically vancomycin-resistant enterococci is not yet fully established, as these organisms are infrequently recovered from clinical specimens. We report our experience with 20 cases of Enterococcus gallinarum and Enterococcus casseliflavus/flavescens bacteremia in humans from 1992 through 1998. Sixteen cases of bacteremia were caused by E. gallinarum. Underlying conditions were present in 19 (95%) of the patients and included malignancy, receipt of transplant, and Caroli’s disease. Polymicrobial bacteremia was present in 9 patients (45%). E. gallinarum and E. casseliflavus/flavescens, although they are infrequently isolated from clinical specimens, may cause serious invasive infections.

Enterococci have emerged as increasingly important nosocomial and community-acquired pathogens. Although they are generally considered to be of low pathogenic potential, it is now well recognized that these organisms can cause serious invasive infections, including endocarditis, bacteremia, urinary tract infection, and pelvic infection [1–5]. According to the most recent National Nosocomial Infections Surveillance System survey, enterococci are the fourth most common cause of nosocomial infections; from 1990 through 1996, they accounted for 10% of recovered isolates overall and 9% of isolates recovered from the bloodstream [6], and from 1986 through 1997, they accounted for 12.8% of isolates recovered from the bloodstream of patients in intensive care units (ICUs) [7].

The third most common type of infection caused by enterococci is bacteremia, including both primary bacteremia, which presumably has a gastrointestinal source, and bacteremia secondary to urinary tract and intra-abdominal infections or the use of intravascular devices [1]. Enterococcal bacteremia is associated with high mortality rates [8]; case-fatality rates range from 12% to 68%, and death due to enterococcal sepsis occurs in 4%–50% of patients [9]. Enterococci’s role as a cause of infection has become increasingly important, not only because of their documented pathogenic potential but also because of increasing antimicrobial resistance (especially resistance to glycopeptide) in some strains [1, 10, 11]. Data from the Centers for Disease Control and Prevention’s National Nosocomial Infections Surveillance System have shown an increase in the percentage of enterococcal infections caused by vancomycin-resistant enterococci (VRE); from 1989 through 1997, the percentage of infections caused by VRE increased from 0.4% to 23.2% among patients in...
the ICU and from 0.3% to 15.4% among patients who are not in the ICU [12, 13].

Glycopeptide resistance in enterococci is associated with diverse phenotypes [14]. Intrinsic low-level vancomycin resistance is characteristic of Enterococcus gallinarum and Enterococcus casseliflavus/flavescens [15, 16]. The VanC-1 ligase is specific for E. gallinarum, and the VanC-2/3 ligase is specific for E. casseliflavus/flavescens [10, 14, 15, 17–22]. The VanC enzymes participate in the synthesis of pentapeptide peptidoglycan precursors ending in D-alanyl-D-serine, which display reduced affinity for vancomycin. Organisms with resistance to VanC remain susceptible to teicoplanin. This naturally occurring vancomycin resistance has not been shown to be transferable, and the related genes are chromosomally encoded in the members of these species [10, 20, 21].

The clinical significance of the enterococci that are intrinsically resistant to vancomycin is not yet fully established, because these phenotypes have not been frequently recovered from clinical specimens, and it was not until 1989 that a test scheme to routinely identify these species was described [23]. Moreover, these organisms have low-level vancomycin resistance, which is often in the “susceptible” range, as defined by current criteria, which are provided by the National Committee for Clinical Laboratory Standards (NCCLS) [24], although typically the MICs of vancomycin are higher than the MICs of non-vanC enterococci. It is estimated that 80%–90% of the enterococcal infections that occur in humans are caused by Enterococcus faecalis, 10%–15% are caused by Enterococcus faecium, and <5% are caused by other species [25].

In a study of 302 consecutive enterococcal isolates recovered from routine cultures, Ruoff et al. [26] observed that E. gallinarum and E. casseliflavus each accounted for only 1% of the isolates, a finding that has been confirmed by other studies [27, 28]. E. gallinarum and E. casseliflavus/flavescens have been implicated in a wide variety of infections in humans, especially immunocompromised persons. These species of enterococci have also been shown to colonize the intestinal tracts of both hospitalized and nonhospitalized individuals, with overall rates of colonization that range from 5.7% [15] to 12.1% [29].

We report our experience with 20 cases of E. gallinarum or E. casseliflavus/flavescens bacteremia seen at the Mayo Clinic from 1992 through 1998. We performed a retrospective analysis of the clinical and epidemiological aspects of bacteremia caused by these species of enterococci.

MATERIALS AND METHODS

Clinical definitions. “Definitely” clinically significant bacteremia due to E. gallinarum or E. casseliflavus/flavescens was defined by isolation of either species from ≥2 blood cultures or from a single blood culture if there was a clinically apparent and/or culture-positive source of infection. One case that did not meet the definition of definitely clinically significant bacteremia but that was considered by the authors to be of “probable” clinical significance was also included. An episode of polymicrobial bacteremia was defined as bacteremia in which >1 organism was isolated from a culture of a single blood sample. All other blood samples that tested positive on culture that were obtained at the same time were included in the single episode [8]. “Nosocomial bacteremia” was defined as bacteremia that was not present or incubating at the time of admission to the hospital [30].

Case ascertainment. Cases were identified by review of the computerized blood culture records of the Mayo Clinic microbiology laboratory (Rochester, MN) from 1992 through 1998. For each infected patient, the complete inpatient and outpatient records were reviewed and clinical and microbiological data were obtained, including age, sex, clinical presentation, presence of polymicrobial bacteremia, comorbid conditions, predisposing factors, potential source of bacteremia, treatment, and outcome.

Blood cultures. Blood specimens were obtained for culture and processed as described elsewhere [31, 32], except that, beginning in May 1996, the 9240 BACTEC blood culture system (Becton Dickinson Diagnostic Instrument Systems) for processing samples was introduced. For each blood culture processed since May 1996, 10 mL of blood was inoculated into each of the following: one 10-mL Isolator (Wampole Laboratories), one 10-mL Plus Aerobic/F resin bottle (aerobic culture; Becton Dickinson Microbiology Systems), and one Lytic/10 anaerobic/F bottle (anaerobic culture; Becton Dickinson).

Identification and antimicrobial susceptibility testing of enterococci. Enterococci were identified by 6.5% NaCl tolerance and growth on bile-esculin agar with esculin hydrolysis. Species-level identification was made on the basis of arginine hydrolysis, motility, pigmentation, growth on tellurite, and formation of acid in mannitol, sorbitol, sucrose, arabinose, raffinose, pyruvate, and sorbose broths. Susceptibility testing was performed by use of an agar dilution method with Mueller-Hinton agar (penicillin was supplemented with lysed horse blood) with a spot inoculum of 10,000 cfu, according to the guidelines of the NCCLS [33]. Mueller-Hinton agar with vancomycin concentrations of 2, 4, 8, and 16 μg/mL was used. MICs were determined after incubation for 16–18 h at 35°C in room air. Since 1996, vancomycin, high-level gentamicin, and high-level streptomycin resistance have been tested by use of brain-heart-infusion (BHI) agar, with incubation of vancomycin and gentamicin BHI agar for 24 h and streptomycin BHI agar for 48 h. Cultures of specimens obtained from other sites that yielded Enterococcus species were also reviewed. Isolates of colonization that range from 5.7% [15] to 12.1% [29].
lates recovered from sources other than blood were not routinely identified to the species level.

RESULTS

During the study period from 1992 through 1998, a total of 252,266 blood cultures were performed at the Mayo Clinic, and the results of 1459 were positive for Enterococcus species; 662 enterococcal bacteremia cases accounted for these positive blood culture results, and of these cases, 16 (2.4%) were due to E. gallinarum, 3 were due to E. casseliflavus, and 1 was due to E. flavescens/gallinarum.

Of the 20 cases, 19 were considered definitely clinically significant. In 5 cases, enterococci were identified from other sources, including urine (in 1 case), liver (in 2), central venous catheter (in 1), and surgical wound (in 1); these enterococci were not identified to the species level. There was 1 case of E. gallinarum endocarditis in a 66-year-old man with a bicuspid aortic valve who had undergone a radical prostatectomy with bilateral ureterointerostomies 2 years earlier. He was cured with aortic valve replacement and administration of IV penicillin and gentamicin.

The patients ranged from 4 years to 84 years of age, and 4 patients were <18 years of age. Seven patients were female. The 1 case that was of probable clinical significance occurred in a patient with acute lymphocytic leukemia who had undergone chemotherapy and had prolonged neutropenia, fever, and multiple episodes of gram-negative bacteremia prior to the emergence of E. gallinarum bacteremia. This patient had been hospitalized for almost 2 months prior to the emergence of E. gallinarum bacteremia. Despite receiving treatment with only trimethoprim/sulfamethoxazole and ceftazidime at the time of the E. gallinarum bacteremia, the patient became afebrile and the neutropenia and bacteremia resolved. This patient was also colonized with vancomycin-resistant E. faecium (MIC of vancomycin, ≥16 μg/mL) and had an episode of vancomycin-resistant E. faecium bacteremia 11 days prior to the episode of E. gallinarum bacteremia. None of the patients were transferred to our institution from outside hospitals.

Underlying conditions were present in 19 (95%) of the patients; these conditions included malignancy in 14 patients (70%), receipt of solid organ transplant in 3 (15%), receipt of bone marrow transplant in 2 (10%), diabetes mellitus in 4 (20%), cholecystolithiasis with cholecystocolic fistula and common bile duct obstruction in 1 (5%), and Caroli’s disease with congenital hepatic fibrosis and polycystic kidney disease in 1 (5%).

There were 8 cases (40%) of community-acquired bacteremia, 7 of which occurred in patients with underlying conditions. Types of infection in the patients with community-acquired bacteremia included hepatic abscesses in 2 patients, endocarditis in 1, cholangitis in 2, primary bacteremia in 1, and wound infection in 1. There was 1 case of community-acquired E. gallinarum bacteremia that involved a patient who had undergone liver transplantation 17 months prior to onset of bacteremia, who presented with fever, nausea, vomiting, and epigastric pain and was found to have biliary dilation and hepatic artery obstruction. The patient died before a definitive diagnosis was made. The patient had Enterococcus species recovered from the urine. The 1 patient without underlying disease was a 12-year-old child who developed a wound infection associated with E. gallinarum bacteremia after sustaining an injury from a farm implement, a power-take-off machine, with avulsion of the left upper limb.

The cases of cholangitis occurred in a 37-year-old man with Caroli’s disease and E. gallinarum bacteremia and in an 84-year-old man with recurrent adenocarcinoma of the head of the pancreas and E. casseliflavus bacteremia. One patient with metastatic cholangiocarcinoma developed hepatic abscesses and subsequently had E. faecium bacteremia, for which a course of vancomycin therapy was administered prior to the development of E. gallinarum bacteremia (MIC of vancomycin, 4 μg/mL).

The other case of hepatic abscess occurred in a patient who had undergone liver transplantation for primary sclerosing cholangitis and who presented 6 weeks after undergoing the transplantation with cytomegalovirus viremia and E. gallinarum bacteremia. Despite having received appropriate treatment for the viremia and 3 days of vancomycin therapy, the patient remained febrile and had recurrent E. gallinarum bacteremia 14 days after onset, at which time hepatic abscess secondary to hepatic artery thrombosis was diagnosed. The single case of community-acquired primary E. gallinarum bacteremia occurred in a patient with lymphoma who had been treated with chemotherapy twice during the month that preceded the episode of bacteremia. Three of the 8 patients with community-acquired bacteremia were hospitalized during the 1–3 months that preceded the episode of bacteremia: 1 patient was hospitalized for enlarging hepatic abscesses, 1 for chemotherapy, and 1 for liver transplantation.

Twelve cases of bacteremia (60%) were nosocomial. Of these 12 nosocomial cases, 1 occurred in a patient with end-stage liver disease secondary to sclerosing cholangitis. This patient had fever and chills and was found to have bacteremia with E. casseliflavus/flavescens. Two cases of E. gallinarum bacteremia occurred following surgery in patients with metastatic pancreatic adenocarcinoma; one patient was readmitted 15 days after discharge with a retroperitoneal abscess following distal pancreatectomy and splenectomy that had been performed 22 days earlier, and the other was readmitted within 2 days of discharge with cholangitis following laparotomy. Five cases of E. gallinarum bacteremia and 1 case of E. casseliflavus bacteremia involved oncology patients with chemotherapy-induced neutropenia (1 of these cases was related to a central venous catheter). One case
of E. gallinarum bacteremia occurred in a patient who had undergone liver transplantation. One case of E. flavescens/gallinarum bacteremia occurred in a patient who had undergone hepatic artery embolization for metastatic islet cell carcinoma. The final case occurred in a patient who had been hospitalized for weight loss and diarrhea and who was found to have intrahepatic duct dilatation and common bile duct obstruction with a cholecysto-tocal fistula, choledocholithiasis, and cholelithiasis. The patient underwent percutaneous transhepatic cholangiography 1 day after admission, and 5 days later she developed a fever associated with E. gallinarum bacteremia.

There were 9 polymicrobial bloodstream infections (45%), 3 of which were community acquired. Three of these infections involved patients with cholangitis, 3 involved patients with hepatic abscesses, and 3 involved patients with hematologic malignancies. Seven of the cases of polymicrobial bacteremia involved E. gallinarum bacteremia and 2 involved E. casseliflavus/flavescens bacteremia. A variety of organisms were isolated concomitantly with E. gallinarum or E. casseliflavus/flavescens, including Escherichia coli, E. faecium, E. faecalis, Stenotrophomonas maltophilia, Clostridium tertium, and Candida albicans. In some instances, bacteria were isolated from blood culture specimens obtained within a few days of the isolation of E. gallinarum or E. casseliflavus/flavescens; these bacteria included Klebsiella species, Enterobacter cloacae, Pseudomonas aeruginosa, Bacteroides ovatus, and coagulase-negative staphylococcus. One patient had concomitant cytomegalovirus viremia.

MICs of vancomycin against E. gallinarum and E. casseliflavus/flavescens isolates ranged from 2 µg/mL to 8 µg/mL; the MIC was 8 µg/mL for only 7 E. gallinarum isolates and for none of the E. casseliflavus isolates (among a total of 42 blood culture isolates obtained from 20 patients). MICs of vancomycin of 2 µg/mL were noted for isolates of both E. gallinarum and E. casseliflavus/flavescens. MICs of penicillin ranged from 0.12 µg/mL to 2 µg/mL, and all the isolates had MICs of ampicillin of 2 µg/mL. Two E. gallinarum isolates exhibited high-level resistance to gentamicin; none of the isolates exhibited high-level resistance to streptomycin. All of the isolates were β-lactamase negative.

Eighteen (90%) of 20 patients had received antibiotics during the 2–69 days that preceded the onset of E. gallinarum or E. casseliflavus/flavescens bacteremia. All of the patients with nosocomial bacteremia and 6 of the patients with community-acquired bacteremia had been treated with antibiotics during the preceding 2 months. In some instances, antibiotic therapy had been started a few days prior to the episode of E. casseliflavus/flavescens bacteremia or E. gallinarum bacteremia. One patient had been receiving long-term antibiotic therapy with “rotating” oral doxycycline, ciprofloxacin, and trimethoprim/sulfamethoxazole to manage recurrent episodes of cholangitis. Another patient had received multiple short courses of ciprofloxacin prior to the diagnosis of E. gallinarum infective endocarditis.

Overall, antibiotics administered prior to the onset of either E. gallinarum bacteremia or E. casseliflavus bacteremia included third-generation cephalosporins, quinolones, penicillins, carbapenems, aminoglycosides, and vancomycin. Five patients with E. gallinarum and 2 patients with E. casseliflavus/flavescens bacteremia received vancomycin for 1–24 days prior to the emergence of bacteremia. In one instance, bacteremia due to E. gallinarum (MIC of vancomycin, 4 µg/mL) emerged while the patient was receiving vancomycin for prior E. faecium bacteremia and hepatic abscess, an occurrence that suggests that vancomycin may not effectively treat patients with E. gallinarum (or E. casseliflavus/flavescens) bacteremia.

Vancomycin therapy was administered to 9 patients; ampicillin, ampicillin/sulbactam, or a combination of piperacillin/tazobactam and gentamicin was substituted for 4 patients, because of either persistent fever or bacteremia. Vancomycin was given to 1 patient concurrently with ampicillin/sulbactam and gentamicin, followed by ampicillin; the patient had polymicrobial bacteremia with E. faecalis, E. coli, Acinetobacter species, a coagulase-negative staphylococcus, and CDC group VB-3. One patient received vancomycin therapy for only 3 days because the E. gallinarum isolate (MIC of vancomycin, 4 µg/mL) was considered to be a contaminant, but bacteremia recurred after 14 days, at which time a hepatic abscess was diagnosed. An Enterococcus species was identified in a specimen of the abscess. Administration of vancomycin, along with gentamicin and ciproxofloxacin, was then initiated, but the patient required liver retransplantation because of persistent, progressive liver abscess. Overall, the duration of antibiotic therapy ranged from 0 to 42 days.

**DISCUSSION**

Although motile enterococci were described 6 decades ago [34], criteria for classification of E. gallinarum and E. casseliflavus/flavescens were described only recently [23, 35–42]; therefore, clinical experience with such strains has been limited. E. gallinarum and E. casseliflavus/flavescens are not frequently isolated from clinical specimens but can cause serious invasive infection. A review of the literature reveals that E. gallinarum or E. casseliflavus/flavescens may be isolated from a variety of clinical specimens and from patients who are either chronically ill or immunosuppressed [15, 23, 26, 28, 43–47]. The majority of cases of bacteremia due to these organisms involved patients with underlying conditions such as renal failure [43, 48], diabetes mellitus [43], hematologic malignancy [49, 50], receipt of solid organ transplant [50], receipt of bone marrow transplant [50], antithrombin III deficiency [50], astrocytoma [50], and chronic osteomyelitis [27]. Cases of vascular and iv line-
related infection have also been described [27, 43, 46]. Not infrequently, bacteremias due to \textit{E. gallinarum} and \textit{E. casseliflavus/flavescens} are polymicrobial [27, 50, 51]. Antibiotic susceptibility patterns indicate that most isolates are penicillin- and ampicillin-susceptible; MICs of vancomycin against \textit{E. gallinarum} are in the range of 2–16 µg/mL, and those against \textit{E. casseliflavus/flavescens} are in the range of 2–8 µg/mL.

Overall, bacteremia due to \textit{E. gallinarum} or \textit{E. casseliflavus/flavescens} accounted for 3% of all cases of enterococcal bacteremia in our study, which is similar to the findings of other studies [15, 50]. Primary bacteremia accounted for 7 (35%) of 20 cases, 6 of which were nosocomially acquired and occurred in patients with malignancies who had received chemotherapy. This may be a result of a bowel source and antimicrobial selection [27]. Enterococci have been shown to “translocate” from the gastrointestinal tract to mesenteric lymph nodes in experimental animals under various conditions [4]. Serious underlying conditions were present in 95% of our patients, including malignancy and receipt of liver or bone marrow transplants, as has been noted in other studies [3, 52–56]. A large proportion (45%) of our patients with \textit{E. gallinarum} and \textit{E. casseliflavus/flavescens} bacteremia had polymicrobial bacteremia. This is similar to the incidence of polymicrobial bacteremia reported in previous studies—50% in the study by Ratanasuwon et al. [50] and 67% in the study by Nauschuetz et al. [51]. As was found in these studies, aerobic gram-negative bacilli were the predominant blood coisolates recovered in our study; other coisolates recovered included \textit{Corynebacterium} species, staphylococci, and other \textit{Enterococcus} species.

The MICs of vancomycin against the \textit{E. gallinarum} and \textit{E. casseliflavus/flavescens} isolates in our study ranged from 2 µg/mL to 8 µg/mL. MICs of vancomycin of 2 µg/mL were noted against both \textit{E. gallinarum} and \textit{E. casseliflavus/flavescens} isolates, but MICs of 8 µg/mL were noted only against \textit{E. gallinarum} isolates. This is in accordance with the findings of previous studies, in which, on average, MICs for \textit{E. gallinarum} are higher than those for \textit{E. casseliflavus} [57–59]. An enterococcus for which the MIC of vancomycin is ≤4 µg/mL is categorized as “susceptible to vancomycin” [24]; however, as was made evident in our study, this does not mean that vancomycin will be active in vivo. Some of our patients’ vancomycin therapy failed despite an MIC of vancomycin that indicated in vitro susceptibility.

Accurate detection of vancomycin resistance in enterococci is important so that appropriate therapy and infection control measures may be instituted in a timely manner [60]. Preventing the spread of VRE requires isolation of patients colonized or infected with VanA and VanB VRE. It is necessary to identify enterococcal species that express low-level vancomycin resistance but that may be misidentified as VanA or VanB VRE by means of screening methodologies to prevent institution of costly, unwarranted infection control measures for patients who are colonized or infected with \textit{E. gallinarum} or \textit{E. casseliflavus/flavescens} [61]. \textit{E. gallinarum} and \textit{E. casseliflavus/flavescens} have not yet been associated with nosocomial outbreaks of infection and, therefore, are not considered to be part of an infection control problem. Because \textit{E. gallinarum} and \textit{E. casseliflavus/flavescens} may be detected among clinical isolates (as shown in our study) and in stool surveillance studies for VRE, accurate identification of such isolates is especially important.

Species-level identification of enterococci is based on physiological studies. \textit{E. faecium} and \textit{E. gallinarum} have similar characteristics but can usually be differentiated from each other by means of the motility test. \textit{E. gallinarum} and \textit{E. casseliflavus/flavescens} are typically motile. \textit{E. gallinarum} can usually be distinguished from \textit{E. casseliflavus/flavescens} by its lack of pigmentation [23, 42]. Some investigators have reported nonmotile \textit{E. gallinarum} and \textit{E. casseliflavus} isolates, as well as nonpigmented \textit{E. casseliflavus} isolates [22, 58, 62, 63], which calls into question the reliability of these properties as a means of distinguishing these species from one other and from other enterococcal species. The absence of these traits can lead to misidentification of enterococcal clinical isolates.

Furthermore, high-level vancomycin resistance of the VanA and VanB phenotypes is transferable and has been noted in rare isolates of \textit{E. casseliflavus} and \textit{E. gallinarum} [16, 17, 64]. The use of molecular techniques specific for VanC has been advocated as a means of identifying enterococci that have low levels of vancomycin resistance [14, 22, 64, 65]. PCR has been used for detection of VRE directly in fecal samples and in colonies on culture plates [16, 60, 66]. Such assays may be used to differentiate VanA, VanB, and VanC genes.

Several studies have demonstrated that \textit{E. gallinarum} and \textit{E. casseliflavus} colonize the gastrointestinal tracts of both hospitalized individuals and nonhospitalized, healthy individuals [15, 29, 46, 67–69]; however, despite higher rates of colonization in some hospitalized individuals, no definite risk factors for colonization or infection have been identified. \textit{E. gallinarum} and \textit{E. casseliflavus/flavescens} are part of the normal stool flora of the general population; this has perhaps impacted the ability of researchers to detect specific risk factors [15]. Therapy with various antimicrobial agents, including cephalosporins and vancomycin, may play a role in increasing colonization with these organisms [15, 29, 69, 70]. Edlund et al. [71] reported a significant increase in the emergence of \textit{E. gallinarum} and \textit{E. casseliflavus} in healthy subjects who were administered oral vancomycin.

In our current series, we describe 20 cases of \textit{E. gallinarum} or \textit{E. casseliflavus/flavescens} bacteremia. This is the largest series reported to date. \textit{E. casseliflavus} and \textit{E. gallinarum} were accorded species status in 1979 and 1982, respectively, and have been routinely identified only since 1989. In our study, \textit{E. gallinarum} bacteremia predominated, although the characteristics
of patients infected with *E. gallinarum* or *E. casseliflavus* were similar: the majority of patients had underlying conditions such as malignancy or receipt of solid organ or bone marrow transplant. Most of the patients in our series had a gastrointestinal source of infection. Endocarditis was documented in 1 case.

In our study, 4 of 20 patients died within 6 days of admission to the hospital; 3 had *E. gallinarum* bacteremia and 1 had *E. casseliflavus* bacteremia. Four patients died 1–2 months after the episode of bacteremia. Because these patients all had serious underlying diseases, it is difficult to attribute their mortality directly to infection with *E. gallinarum* and *E. casseliflavus/flavescens*. This is often true with enterococcal bacteremia, in which high mortality is associated with a high prevalence of underlying conditions; it is difficult to determine whether death is the result of the bacteremia or the underlying condition [52, 53].

In summary, although they are not frequently recovered from clinical specimens, *E. gallinarum* and *E. casseliflavus* may cause serious invasive disease. These organisms may be isolated from a variety of sources, and *E. gallinarum* and *E. casseliflavus/flavescens* bacteremia may be polymicrobial. It is important that all bloodstream isolates of enterococci and those isolates obtained from other sources that are related to serious infection be identified to the species level, not only because of infection control issues but also so that appropriate antibiotic therapy may be initiated. Clinicians need to be alerted to the possibility that vancomycin may not be effective against *E. gallinarum* and *E. casseliflavus/flavescens*, despite in vitro results that indicate vancomycin susceptibility.

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### References


