**When are Oral Antibiotics a Safe and Effective Choice for Bacterial Bloodstream Infections? An Evidence-Based Narrative Review**

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Bacterial bloodstream infections (BSIs) are a major cause of morbidity and mortality in the United States. Traditionally, BSIs have been managed with intravenous antimicrobials. However, whether intravenous antimicrobials are necessary for the entirety of the treatment course in BSIs, especially for uncomplicated episodes, is a more controversial matter. Patients that are clinically stable, without signs of shock, or have been stabilized after an initial septic presentation, may be appropriate candidates for treatment of BSIs with oral antimicrobials. There are risks and costs associated with extended courses of intravenous agents, such as the necessity for long-term intravenous catheters, which entail risks for procedural complications, secondary infections, and thrombosis. Oral antimicrobial therapy for bacterial BSIs offers several potential benefits. When selected appropriately, oral antibiotics offer lower cost, fewer side effects, promote antimicrobial stewardship, and are easier for patients. The decision to use oral versus intravenous antibiotics must consider the characteristics of the pathogen, the patient, and the drug. In this narrative review, the authors highlight areas where oral therapy is a safe and effective choice to treat bloodstream infection, and offer guidance and cautions to clinicians managing patients experiencing BSI.

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as offer guidance to clinicians managing patients experiencing BSI. Given the lack of robust clinical trials on this subject, the evidence for performing a systematic review was insufficient. Thus, the articles and recommendations cited in this review were selected based on the authors’ experiences to represent the best available evidence.

**INFECTION SOURCE CONTROL**

Diagnosing the source of a patient’s BSI is vital to successful treatment for 2 reasons. First, without achieving source control, antimicrobial therapy of any sort is more likely to fail. 7 For example, patients with *Staphylococcus aureus* abscess and persistently positive blood cultures despite intravenous antimicrobials require drainage. Similarly, patients with a CLABSI typically benefit from removal of the foreign body. 11 Second, particular oral antibiotics have different penetration levels into various tissues (Table 1). 12 For instance, if a patient has meningitis due to *Streptococcus pneumoniae* with concurrent BSI, doxycycline would be an inferior choice, despite having good bioavailability and achieving high blood concentrations, because it poorly penetrates the central nervous system. An oral regimen must adequately penetrate the source of infection.

**PATHOGEN AND ANTIMICROBIAL FACTORS**

Several important factors regarding the BSI pathogen should be considered when deciding on oral versus intravenous therapy, as follows: 1) organism speciation and susceptibilities should be available; 2) the pathogen should be susceptible to an oral antimicrobial with high bioavailability that achieves adequate blood and source-tissue concentrations; 3) the candidate antibiotic should have a high barrier to acquired resistance for the pathogen. For example, although *S. aureus* is often susceptible to rifampin, it has a low genetic barrier to resistance; thus, rifampin monotherapy is not recommended; and 4) the selected agent should generally be well-tolerated and have an acceptable safety profile. Table 2 summarizes the characteristics of several key antibiotics.

| TABLE 1. Penetration of Select Oral Antimicrobials to Tissue Sites7,44 |
|---------------------|----------------|----------------|----------------|----------------|----------------|----------------|
| Antimicrobial       | Bloodstream Bioavailability | Lung | Liver | Urinary Tract | Prostate | Bone | GI | Skin | Bile | CSF | Synovial |
| Ciprofloxacin       | 70% | +++ | +++ | +++ | +++ | +++ | +++ | +++ | + | +++ |
| Levofloxacin        | 99% | +++ | +++ | +++ | +++ | +++ | +++ | +++ | + | +++ |
| Moxifloxacin        | 90% | +++ | +++ | +++ | +++ | +++ | +++ | +++ | + | +++ |
| Trimethoprim-Sulfamethoxazole | 90% | ++ | ++ | +++ | + | +++ | +++ | + | + | +++ |
| Doxycycline         | 95% | ++ | ++ | ++ | ++ | ++ | ++ | ++ | + | ++ |
| Minocycline         | 95% | ++ | ++ | ++ | ++ | ++ | ++ | ++ | + | ++ |
| Linezolid           | 99% | +++ | ++ | +++ | + | +++ | +++ | + | + | +++ |
| Metronidazole       | 90% | ++ | +++ | ++ | ++ | ++ | ++ | ++ | + | ++ |
| Clindamycin         | 90% | ++ | ++ | ++ | ++ | ++ | ++ | ++ | + | ++ |
| Ampicillin          | 50% | + | ++ | ++ | + | ++ | ++ | ++ | + | ++ |
| Penicillin V        | 50% | ++ | ++ | ++ | + | ++ | ++ | ++ | + | ++ |
| Amoxicillin         | 85% | + | ++ | ++ | + | ++ | ++ | ++ | + | ++ |
| Cephalexin          | 60% | ++ | ++ | ++ | ++ | ++ | ++ | ++ | + | ++ |

+++ Tissue concentrations equal to or higher than serum concentrations
++ Tissue concentrations at least 50% of the serum concentrations
+ Tissue concentrations less than 50% of the serum concentrations

Bioavailability represents the percentage of the dose that reaches systemic circulation. Tissue penetration reflects the drug movement from the vascular to the interstitial and intracellular compartments of a particular body site. Drugs passively diffuse through fenestrated capillaries into the interstitial compartment of most tissues. However, some tissue sites (eg, the brain and prostate) contain nonfenestrated capillaries and/or active transport pumps that prevent entry or remove the drug. Tissue concentrations are methodologically dependent on the various techniques used in their quantification, and, in some body sites, are influenced by the presence or absence of inflammation (eg, brain tissue). Thus, the values presented here are best approximations.
outpatient.\textsuperscript{13} Finally, the patient should be available for close follow-up. Table 3 summarizes the patient factors to consider.

**EVIDENCE REGARDING BLOODSTREAM INFECTIONS DUE TO GRAM-NEGATIVE RODS**

BSIs due to gram-negative rods (GNRs) are common and cause significant morbidity and mortality. GNRs represent a broad and diverse array of pathogens. We focus on the Enterobacteriaceae family and Pseudomonas aeruginosa, because they are frequently encountered in clinical practice.\textsuperscript{1}

**Gram-Negative Rods, Enterobacteriaceae Family**

The Enterobacteriaceae family includes *Escherichia coli*, *Klebsiella*, *Salmonella*, *Proteus*, *Enterobacter*, *Serratia*, and *Citrobacter* species. The range of illnesses caused by Enterobacteriaceae is as diverse as the family, encompassing most body sites.
Resistance to fluoroquinolones such as ciprofloxacin has been identified as a risk factor for GNR BSI oral treatment failure, highlighting the importance of confirming susceptibilities before committing to an oral treatment plan.\textsuperscript{18,19} Even ESBL Enterobacteriaceae can be considered for treatment with fluoroquinolones if susceptibilities allow.\textsuperscript{20}

The ideal duration of therapy for GNR BSI is an area of active research. A recent retrospective trial showed no difference in all-cause mortality or recurrent BSI in GNR BSI treated for 8 versus 15 days.\textsuperscript{21} A recent meta-analysis suggested that 7 days of therapy was noninferior to a longer duration therapy (10–14 days) for pyelonephritis, in which a subset was bacteremic.\textsuperscript{22} However, another trial reported that short course therapy for GNR BSI (<7 days) is associated with higher risk of treatment failure.\textsuperscript{22} Further data are needed.

**Evidence Regarding Bloodstream Infections Due to Gram-Positive Cocci**

The majority of bloodstream infections in the United States, and the resultant morbidity and mortality, are from gram-positive cocci (GPCs) such as *Staphylococcus*, *Streptococcus*, and *Enterococcus* species.\textsuperscript{1}

**Gram-Positive Cocci, *Pseudomonas aeruginosa***

*Pseudomonas aeruginosa* is a common pathogen, intrinsically resistant to many antimicrobials, and readily develops antimicrobial resistance during therapy. Fluoroquinolones (such as ciprofloxacin, levofloxacin, and delafloxacin) are the only currently available oral agents with antipseudomonal activity. However, fluoroquinolones may not achieve blood concentrations appropriate for *P. aeruginosa* treatment at standard doses, while higher dose regimens may be associated with increased risk for undesirable side effects.\textsuperscript{24,25} Currently, given the minimal trial data comparing oral versus intravenous therapy for *P. aeruginosa* BSIs, and multiple studies indicating increased mortality when *P. aeruginosa* is treated inappropriately,\textsuperscript{26,27} we prefer a conservative approach and consider oral therapy a less-preferred option.

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of knowing the final susceptibility data prior to consolidating to monotherapy with an oral agent, and that macrolides may have beneficial anti-inflammatory effects, though further research is needed.\textsuperscript{34,35}

Although the evidence for treating bacteremic pneumococcal pneumonia using a highly active and absorbable oral agent is fairly robust, \textit{S. pneumoniae} BSI secondary to other sites of infection sites is less well studied and may require a more conservative approach.

**Gram-Positive Cocci, \(\beta\)-hemolytic \textit{Streptococcus} species**

\(\beta\)-Hemolytic \textit{Streptococcus} include groups A to H, of which groups A (\textit{S. pyogenes}) and B (\textit{S. agalactiae}) are the most commonly implicated in BSIs.\textsuperscript{36} Group A \textit{Streptococcus} (GAS) is classically associated with streptococcal pharyngitis and Group B \textit{Streptococcus} (GBS) is associated with postpartum endometritis and neonatal meningitis, though both are virulent organisms with a potential to cause invasive infection throughout the body and in all age-groups. Up to 14\% of GAS and 41\% GBS BSIs have no clear source;\textsuperscript{37,38} given these are skin pathogens, such scenarios likely represent invasion via microabrasion. As \(\beta\)-hemolytic streptococcal BSI is often observed in the context of necrotizing skin and soft tissue infections, surgical source control is particularly important.\textsuperscript{39} GAS remains exquisitely susceptible to penicillin, and intravenous penicillin remains the mainstay for invasive disease; GBS has higher penicillin resistance rates than GAS.\textsuperscript{40} Clindamycin should be added when there is concern for severe disease or toxic shock.\textsuperscript{41} Unfortunately, oral penicillin is poorly bioavailable (approximately 50\%), and there has been recent concern regarding inducible clindamycin resistance in GAS.\textsuperscript{42} Thus, oral penicillin V and/or clindamycin is a potentially risky strategy, with no clinical trials supporting this approach; however, they may be reasonable options in selected patients with source control and stable hemodynamics. Amoxicillin has high bioavailability (85\%) and may be effective; however, there is lack of supporting data. Highly bioavailable agents such as levofloxacin and linezolid have GAS and GBS activity\textsuperscript{43} and might be expected to produce satisfactory outcomes. Because no clinical trials have compared these agents with intravenous therapy for BSI, caution is advised. Although bacteriostatic against \textit{Staphylococcus}, linezolid is bactericidal against \textit{Streptococcus}.\textsuperscript{44} Fluoroquinolone resistance amongst \(\beta\)-hemolytic \textit{Streptococcus} is rare (approximately 0.5\%) but does occur.\textsuperscript{45}

**Gram-Positive Cocci, \textit{Staphylococcus} Species**

\textit{Staphylococcus} species include \textit{S. aureus} (including methicillin susceptible and resistant strains: MSSA and MRSA, respectively) and coagulase-negative species, which include organisms such as \textit{S. epidermidis}. \textit{S. aureus} is the most common cause of BSI mortality in the United States,\textsuperscript{1} with mortality rates estimated at 20\%–40\% per episode.\textsuperscript{46} Infectious disease consultation has been associated with decreased mortality and is recommended.\textsuperscript{47} The guidelines of the Infectious Diseases Society of America for the treatment of MRSA recommend the use of parenteral agents for BSI.\textsuperscript{48} It is important to consider if a patient with \textit{S. aureus} BSI has infective endocarditis.

Oral antibiotic therapy for \textit{S. aureus} BSI is not currently standard practice. Although trimethoprim-sulfamethoxazole (TMP-SMX) has favorable pharmacokinetics and case series of using it successfully for BSI exist,\textsuperscript{49} TMP-SMX showed inferior outcomes in a randomized trial comparing oral TMP-SMX with intravenous vancomycin in a series of 101 \textit{S. aureus} infections.\textsuperscript{50} This observation has been replicated.\textsuperscript{51} Data on doxycycline or clindamycin for \textit{S. aureus} BSI are limited, and IDSA guidelines advise against their use in this setting because they are predominantly bacteriostatic.\textsuperscript{46} Linezolid has favorable pharmacokinetics, with approximately 100\% bioavailability, and \textit{S. aureus} resistance to linezolid is rare.\textsuperscript{52} Several randomized trials have compared oral linezolid with intravenous vancomycin for \textit{S. aureus} BSI; for instance, Stevens et al. randomized 460 patients with \textit{S. aureus} infection (of whom 18\% had BSI) to linezolid versus vancomycin and observed similar clinical cure rates.\textsuperscript{53} A pooled analysis showed oral linezolid was noninferior to vancomycin specifically for \textit{S. aureus} BSI.\textsuperscript{54} However, long-term use is often limited by hematologic toxicity, peripheral or optic neuropathy (which can be permanent), and induced serotonin syndrome. Additionally, linezolid is bacteriostatic, not bactericidal against \textit{S. aureus}. Using oral linezolid as a first-line option for \textit{S. aureus} BSI would not be recommended; however, it may be used as a second-line treatment option in selected cases. Tedizolid has similar pharmacokinetics and spectrum of activity with fewer side effects; however, clinical data on its use for \textit{S. aureus} BSI are lacking.\textsuperscript{55} Fluoroquinolones such as levofloxacin and the newer agent delafloxacin have activity against \textit{S. aureus}, including MRSA, but on-treatment emergence of fluoroquinolone resistance is a concern, and data on delafloxacin for BSI are lacking.\textsuperscript{56,57} Older literature suggested the combination of ciprofloxacin and rifampin was effective against right-sided \textit{S. aureus} endocarditis,\textsuperscript{58} and other oral fluoroquinolone-rifampicin combinations have also been found to be effective.\textsuperscript{59} However, this approach is currently not a standard therapy, nor is it widely used. Decisions on the duration of therapy for \textit{S. aureus} BSI should be made in conjunction with an infectious diseases specialist; 14 days is currently regarded as a minimum.\textsuperscript{47,48}

Published data regarding oral treatment of coagulase-negative \textit{Staphylococcus} (CoNS) BSI are limited. Most CoNS bacteremia and up to 80\% \textit{Staphylococcus epidermidis} bacteremia represent blood culture contamination, though true infection from CoNS is not uncommon, particularly in patients with indwelling catheters.\textsuperscript{60} An exception is the CoNS species \textit{Staphylococcus lugdunensis}, which is more virulent, and bacteremia with this organism usually warrants antibiotics. Oral antimicrobial therapy is currently not a standard treatment practice for CoNS BSI that is felt to represent true infection; however, linezolid has been successfully used in case series.\textsuperscript{51}

**Gram-Positive Cocci, \textit{Enterococcus}**

\textit{E. faecium} and \textit{E. faecalis} are commonly implicated in BSI.\textsuperscript{1} Similar to \textit{S. aureus}, infective endocarditis must be ruled out when treating enterococcal BSI; a scoring system has been
proposed to assist in deciding if such patients require echocardiography. Intravenous ampicillin is a preferred, highly effective agent for enterococci treatment when the organism is susceptible. However, oral ampicillin has poor bioavailability (50%), and data for its use in BSI are lacking. For susceptible strains, amoxicillin has comparable efficacy for enterococci and enhanced bioavailability (85%); high dose oral amoxicillin could be considered, but there is minimal clinical trial data to support this approach. Fluoroquinolones exhibit only modest activity against enterococci and would be an inferior choice for BSI. Although often sensitive to oral tetracyclines, data on their use in enterococcal BSI are insufficient. Nitrofurantoin can be used for susceptible enterococcal urinary tract infection; however, it does not achieve high blood concentrations and should not be used for BSI.

There is significant data comparing oral linezolid with intravenous daptomycin for vancomycin-resistant enterococci (VRE) BSI. In a systematic review including 10 trials using 30-day all-cause mortality as the primary outcome, patients treated with daptomycin demonstrated higher odds of death (OR 1.61, 95% CI 1.08-2.40) compared with those treated with linezolid. However, more recent data suggested that higher daptomycin doses than those used in these earlier trials resulted in improved VRE BSI outcomes. A subsequent study reported that VRE BSI treatment with linezolid is associated with significantly higher treatment failure and mortality compared with daptomycin therapy. Further research is needed, but should the side-effect profile of linezolid be tolerable, it remains an effective option for oral treatment of enterococcal BSIs.

EVIDENCE REGARDING ANAEROBIC BACTERIAL BLOOD STREAM INFECTION

Anaerobic bacteria include Bacteroides, Prevotella, Porphyromonas, Fusobacterium, Peptostreptococcus, Veillonella, and Clostridium. Anaerobes account for approximately 4% of bacterial BSIs, and are often seen in the context of polymicrobial infection. Given that anaerobes are difficult to recover, and that antimicrobial resistance testing is more labor intensive, antibiotic therapy choices are often made empirically. Unfortunately, antibiotic resistance amongst anaerobes is increasing. However, metronidazole remains highly active against a majority of anaerobes, with only a handful of treatment failures reported, and has a highly favorable pharmacokinetic profile for oral treatment. Oral metronidazole remains an effective choice for many anaerobic BSIs. Considering the polymicrobial nature of many anaerobic infections, source control is important, and concomitant GNR infection must be ruled out before using metronidazole monotherapy.

Clindamycin has significant anaerobic activity, but Bacteroides resistance has increased significantly in recent years, as high as 26%-44%. Amoxicillin-clavulanate has good anaerobic coverage, but bioavailability of clavulanate is limited (50%), making it inferior for BSI. Bioavailability is also limited for cephalosporins with anaerobic activity, such as cefturoxime. Moxifloxacin is a fluoroquinolone with some anaerobic coverage and a good oral pharmacokinetic profile, but Bacteroides resistance can be as high as 50%, making it a risky empiric choice.

CONCLUSIONS

Bacterial BSIs are common and result in significant morbidity and mortality, with high associated healthcare costs. Although BSIs are traditionally treated with intravenous antimicrobials, many BSIs can be safely and effectively cured using oral antibiotics. When appropriately selected, oral antibiotics offer lower costs, fewer side effects, promote antimicrobial stewardship, and are easier for patients. Ultimately, the decision to use oral versus intravenous antibiotics must consider the characteristics of the pathogen, patient, and drug.

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References


