Intracranial epidural abscess

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INTRODUCTION

There are three forms of focal intracranial suppuration: brain abscess, subdural empyema, and intracranial epidural abscess. Abscesses that are enclosed within the bony confines of the skull or spinal column can expand to compress the brain or spinal cord and cause severe symptoms, permanent complications, or even death. Prompt diagnosis and proper treatment can avert complications and achieve cure in many cases. Both the diagnosis and management of epidural abscess, which often includes a surgical procedure for aspiration or drainage of the abscess, have been greatly aided by the advent of modern imaging techniques, such as computed tomography and especially magnetic resonance imaging.

The epidemiology, microbiology, clinical manifestations, diagnosis, and treatment of intracranial epidural abscess will be reviewed here. Spinal epidural abscess, brain abscess, and bacterial meningitis are discussed separately. (See "Spinal epidural abscess" and "Pathogenesis, clinical manifestations, and diagnosis of brain abscess" and "Treatment and prognosis of bacterial brain abscess" and "Epidemiology of community-acquired bacterial meningitis in adults" and "Pathogenesis and pathophysiology of bacterial meningitis" and "Clinical features and diagnosis of acute bacterial meningitis in adults" and "Initial therapy and prognosis of community-acquired bacterial meningitis in adults" and "Treatment of bacterial meningitis caused by specific pathogens in adults" and "Neurologic complications of bacterial meningitis in adults".)

TYPES OF EPIDURAL ABSCESS

Two distinct varieties of epidural abscess occur: spinal epidural abscess (SEA) and intracranial epidural abscess (IEA). SEA is more common by a factor of nine to one. The distinction between these two entities is based upon the different anatomy of the two locations within the central nervous system and some differences in symptoms and natural history. IEAs are less common than SEAs and less acute in their evolution. However, like SEAs, IEAs are significant infections requiring optimal therapy to prevent complications.

SEA is discussed in detail separately. (See "Spinal epidural abscess".)

ANATOMY

The intracranial dura mater forms the inner lining of the skull and is directly adherent to bone. Thus, under normal circumstances, there is no actual epidural space. The potential epidural space can be opened by pressure from expanding tumors, blood, inflammatory masses, or pus. This requires that the firmly adherent dura be dissected off the bone; as a result, intracranial epidural abscesses tend to be slow growing, rounded, and well localized (picture 1). In contrast, subdural empyema is a collection of pus between the dura and arachnoid membrane.

PATHOGENESIS AND PATHOLOGY

Organisms usually spread into the potential extradural space by direct extension from a contiguous focus of infection or by inoculation during trauma or neurosurgery. Organisms may also pass through the venous foramina of the frontal bone plate to this space without causing frontal bone osteomyelitis. After reaching this site, the bacteria cause inflammation and the formation of pus or granulation tissue, which gradually dissect the tough and adherent dura away from the inner table of the skull.

Intracranial epidural abscesses usually consist of a localized lesion with a central collection of pus surrounded by a wall of inflammatory reaction, which may calcify. They rarely spread downward (caudally) from inside the skull into the spinal canal because the dura is very tightly attached around the foramen magnum. These findings are different from those of spinal epidural abscesses in which granulation tissue rather than pus is a common finding and longitudinal spread of infection is virtually the rule.

EPIDEMIOLOGY

Intracranial epidural abscess is the third most common focal pyogenic intracranial infection, after brain abscess and subdural empyema [1,2]. It is far less common than spinal epidural abscess. In the past, most cases of intracranial epidural abscesses were associated with sinusitis, otitis, or mastoiditis. Today, many cases arise as a complication of neurosurgical procedures.

MICROBIOLOGY AND PORTALS OF ENTRY

The usual portal of entry is from the exterior. If the inciting infection arises from the paranasal sinuses or ears, the organisms are likely to be microaerophilic or anaerobic streptococci and/or other anaerobes such as *Cutibacterium* (formerly *Propionibacterium*) and *Peptostreptococcus* species. A few cases, particularly those associated with chronic sinusitis, are due to aerobic gram-negative bacilli or fungi.

If infection follows neurosurgery, the most likely organisms are staphylococci, especially *Staphylococcus aureus*, and gram-negative bacteria. Infection can also spread inward from osteomyelitis of the skull or be introduced by fetal monitoring probes applied to the skull during birth.

CLINICAL MANIFESTATIONS

Signs and symptoms develop both as a result of infection and the slowly expanding intracranial mass. The latter can eventually cause raised intracranial pressure, papilledema, and, in some cases, focal neurologic signs. Fever, headache, lethargy, nausea, and vomiting are common. Patients with IEAs complicating sinus infections may have purulent drainage from the nose or ear [3].

Because an IEA usually arises as a complication of another process, the primary infection may be the focus of attention and delay diagnosis of the intracranial extension. As an example, local swelling and tenderness may be attributed to recent trauma, neurosurgery, or sinusitis when in fact it overlies an IEA.

This diagnosis warrants consideration in patients who present with severe headache and fever and a recent history of an intracranial neurosurgical procedure or a known or suspected contiguous source of infection, such as sphenoid sinusitis or otitis media.

DIAGNOSIS

The key to diagnosis is to consider this rare condition early, then to perform a physical examination followed by appropriate imaging. Magnetic resonance imaging with contrast (eg, gadolinium) (image 1) usually provides more information than computed tomography (CT) with contrast (image 2) [4]. CT-guided needle aspiration or open drainage can provide material for staining and culture for bacteria, mycobacteria, and fungi.

DIFFERENTIAL DIAGNOSIS

An intracranial epidural abscess can mimic any intracranial mass lesion, including parenchymal primary and metastatic tumors, meningiomas, hematomas, brain abscesses, or subdural empyemas. Chronic meningitis, tuberculous meningitis, and cranial arteritis should also be considered in the differential diagnosis.

MANAGEMENT

Approach to therapy — Successful treatment of an IEA usually requires a combination of a drainage procedure and antibiotic therapy. Neurosurgical drainage is most commonly performed via burr holes or a craniotomy. If the dura is macerated or breached, a graft or other occlusive approach may be applied.

Sinus-related IEA in children may be managed without neurosurgical procedures if there is adequate sinus drainage (by a surgical or endoscopic drainage procedure) and minimal mass effect from the abscess. This was illustrated in a study of eight children that compared a neurosurgical drainage procedure to sinus drainage without an intracranial procedure [3]. Patients received antibiotic therapy with activity against the isolated organism(s). All patients had clinical and radiologic resolution of infection following therapy.

Empiric antimicrobial therapy — The optimal timing of initiating antimicrobial therapy has not been established. The decision of when to start antimicrobial therapy should be made on a case-by-case basis, depending upon the clinical circumstances. In immunocompetent patients who are not severely ill (ie, do not have neurologic deficits and are not septic) and for whom a surgical procedure to drain the abscess is planned over the following one to two days, it is reasonable to delay antimicrobial therapy until a specimen of the abscess fluid can be obtained. In such patients, an empiric regimen should be started as soon as a specimen has been collected. In immunocompromised patients, patients who have concerning clinical findings, and

patients for whom surgery cannot be performed within one to two days, empiric therapy should be started without waiting for a specimen.

There are no randomized controlled trials evaluating antibiotic regimens for the treatment of IEA. Empiric antibiotic therapy should be chosen based upon the probable origin of the infection and the most common pathogens. For example, if contiguous infection of the paranasal sinuses, ear, or mastoid is the source, a regimen that is active against streptococci, *Haemophilus* species, and anaerobes should be chosen.

An appropriate regimen for adults with contiguous infection is:

- Metronidazole (7.5 mg/kg [usually 500 mg] IV every six to eight hours)

  PLUS

- **Either** ceftriaxone (2 g IV every 12 hours) **or** cefotaxime (2 g IV every four to six hours)

In most other instances, an empiric regimen with antibiotics active against staphylococci, streptococci, and gram-negative bacilli should be chosen. Antibiotics should be directed against the known pathogen if the culture and/or Gram stain of the aspirate is positive. If an aspirate cannot be obtained, the empiric regimen should be continued.

Appropriate empiric parenteral regimens for adults include:

- Vancomycin (table 1) for empiric coverage of methicillin-resistant *S. aureus* (MRSA) and for use in patients who are allergic to penicillin; if susceptibility testing reveals methicillin-susceptible *S. aureus* (MSSA), vancomycin should be replaced with nafcillin (2 g IV every four hours) or oxacillin (2 g IV every four hours) in patients without allergy to these agents since they have better central nervous system (CNS) penetration than vancomycin and better activity against MSSA.

  PLUS

- Metronidazole (7.5 mg/kg [usually 500 mg] IV every six to eight hours)

  PLUS

- Either cefotaxime (2 g IV every 6 hours), ceftriaxone (2 g IV every 12 hours), ceftazidime (2 g IV every 8 hours), or cefepime (2 g IV every 8 hours). Ceftazidime or cefepime is preferable when *Pseudomonas aeruginosa* is considered a possible or likely pathogen.

An alternative for patients requiring a broad-spectrum regimen that has activity against *P. aeruginosa* is meropenem (2 g IV every 8 hours) **plus** vancomycin.

Subsequent antimicrobial therapy — When the etiologic agent or agents have been identified by culture, treatment regimens should be simplified and directed to that pathogen or those pathogens. Specific regimens for bacterial meningitis are discussed separately; similar antibiotic regimens can generally be used for bacterial meningitis and epidural abscesses, although epidural abscesses require a longer duration of therapy. (See "Treatment of bacterial meningitis caused by specific pathogens in adults".)

Staphylococcus aureus — Given substantial rates of MRSA infections, vancomycin (table 1) should be used as initial therapy when *S. aureus* is suspected or proven [5,6]. If susceptibility testing reveals MSSA, therapy should be changed to nafcillin (2 g IV every four hours) or oxacillin (2 g IV every four hours). Although many experts recommend nafcillin over the choice of a first-generation cephalosporin such as cefazolin in patients with extradural infections such as an epidural intracranial abscess, cefazolin can be used as an alternative in such patients with good efficacy if there is no evidence of coexisting meningitis. Cefazolin (2 g IV every 8 hrs) is also a safe and effective alternative to nafcillin in patients who have an intracranial epidural abscess, no coexisting meningitis, and a type I allergy to penicillin [7]. If the *S. aureus* isolate is methicillin-resistant, vancomycin should be continued and dosed to achieve serum trough concentrations of 15 to 20 mcg/mL. A significant drawback to vancomycin is its poor penetration into the cerebrospinal fluid (CSF) of approximately 1 and 5 percent in uninflamed and inflamed meninges, respectively [6,8-10]. Rifampin can be added to vancomycin at a dose of 600 mg orally once daily or 300 to 450 mg twice daily because it achieves bactericidal concentrations in the CSF regardless of meningeal inflammation [5,6,11], although there are few data to support this [12-14].

Based upon case reports and case series of patients with CNS infections caused by MRSA, alternatives to vancomycin include linezolid (600 mg IV or orally twice daily) [15-18], trimethoprim-sulfamethoxazole (5 mg of the trimethoprim component/kg IV every 8 to 12 hours) [6,19,20], and daptomycin (6 to 10 mg/kg IV once daily) [21,22]. Although there are insufficient data regarding the efficacy of these regimens for the treatment of CNS infections caused by MRSA, these agents are reasonable alternatives when vancomycin cannot be used or is ineffective. Further studies are needed to establish the benefit of these agents for the treatment of CNS infections. This is discussed in greater detail separately. (See "Treatment of bacterial meningitis caused by specific pathogens in adults", section on 'Staphylococcus aureus'.)

Duration — The appropriate duration of antimicrobial therapy should be determined on a case-by-case basis, taking into account the laboratory results (white blood cell count, erythrocyte sedimentation rate, C-reactive protein), the adequacy of surgical drainage, the presumed duration of the infection, and the radiographic and clinical response to therapy. The first follow-up magnetic resonance image is obtained at about four to six weeks if the patient is improving or at any time if clinical deterioration occurs.

The usual duration of antimicrobial therapy is six to eight weeks. We generally treat patients who have undergone surgical drainage and who have responded well to medical and surgical therapy for six weeks. In patients with associated osteomyelitis, patients in whom surgical drainage did not occur, and patients in whom the response to therapy has been slow, we treat for six to eight weeks. (See "Nonvertebral osteomyelitis in adults: Treatment".)

Some clinicians transition patients from intravenous antibiotics to oral antibiotics once the patient has improved clinically; however, there is no evidence that this approach is effective. With the exception of rifampin, linezolid, metronidazole, and trimethoprim-sulfamethoxazole, the concentrations of oral antibiotics penetrating the abscess would be expected to be below the minimal inhibitory concentration for most pathogens [23].

PROGNOSIS

With appropriate antibiotic and surgical management, the prognosis for intracranial epidural abscess (IEA) is good. As an example, in a series of 82 patients with IEA (the majority of whom underwent surgery), 81 patients experienced good neurologic outcomes (Glasgow Outcome Scale score of 4 or 5) and 1 patient died (1.2 percent) [24].

SUMMARY AND RECOMMENDATIONS

- **Overview** – Epidural abscess is a rare but important suppurative infection of the central nervous system (CNS). Abscesses that are enclosed within the bony confines of the skull or spinal column can expand to compress the brain or spinal cord and cause severe symptoms, permanent complications, or even death. Prompt diagnosis and proper treatment can avert complications and achieve cure in many cases. (See 'Introduction' above.)

- **Risk factors** – Organisms usually spread into the potential extradural space by direct extension from a contiguous focus of infection or by inoculation during trauma or neurosurgery. In the past, most cases were associated with sinusitis, otitis, or mastoiditis.

Today, many cases arise as a complication of neurosurgical procedures. (See 'Introduction' above and 'Epidemiology' above.)

- **Microbiology** – If the inciting infection arises from the paranasal sinuses or ears, the organisms are likely to be microaerophilic or anaerobic streptococci and/or other anaerobes such as *Cutibacterium* (formerly *Propionibacterium*) and *Peptostreptococcus* species. If infection follows neurosurgery, the most likely organisms are staphylococci, especially *Staphylococcus aureus*, and gram-negative bacteria. (See 'Microbiology and portals of entry' above.)

- **Clinical manifestations** – Signs and symptoms develop both as a result of infection and the slowly expanding intracranial mass. The latter can eventually cause raised intracranial pressure, papilledema, and, in some cases, focal neurologic signs. Fever, headache, lethargy, nausea, and vomiting are common. (See 'Clinical manifestations' above.)

- **Diagnosis** – The key to diagnosis is to consider this rare condition then to perform a physical examination followed by appropriate imaging. Magnetic resonance imaging (MRI) (image 1) usually provides more information than computed tomography (image 2). (See 'Diagnosis' above.)

- **Management** – Successful treatment of an IEA usually requires a combination of a drainage procedure and antibiotic therapy. Neurosurgical drainage is most commonly performed via burr holes or a craniotomy. (See 'Management' above.)

  - **When to start antibiotics** – The decision of when to start antimicrobial therapy should be made on a case-by-case basis, depending upon the clinical circumstances. In immunocompetent patients who are not severely ill (ie, do not have neurologic deficits and are not septic) and for whom a surgical procedure to drain the abscess is planned over the following one to two days, it is reasonable to delay antimicrobial therapy until a specimen of the abscess fluid can be obtained; in such patients, an empiric regimen should be started as soon as a specimen has been collected. In immunocompromised patients, patients who have concerning clinical findings, and patients for whom surgery cannot be performed within one to two days, empiric therapy should be started without waiting for a specimen. (See 'Empiric antimicrobial therapy' above.)

  - **Empiric antibiotic selection** – Empiric antibiotic therapy should be chosen based upon the probable origin of the infection and the likely causative organisms. For example, if contiguous infection of the paranasal sinuses, ear, or mastoid is the source, a regimen that is active against streptococci, *Haemophilus* species, and anaerobes should be chosen. An appropriate parenteral regimen for adults with contiguous infection is

metronidazole **plus either** ceftriaxone **or** cefotaxime. In most other instances, an empiric regimen with antibiotics active against staphylococci, streptococci, and gram-negative bacilli should be chosen. An appropriate parenteral regimen is vancomycin **plus** metronidazole **plus** either cefotaxime, ceftriaxone, ceftazidime, or cefepime; ceftazidime or cefepime should be the cephalosporin selected if *Pseudomonas aeruginosa* is a possible or likely pathogen. An alternative for patients requiring a broad-spectrum regimen that has activity against *P. aeruginosa* is meropenem **plus** vancomycin. (See 'Empiric antimicrobial therapy' above.)

- **Directed antibiotic therapy** – When the etiologic agent or agents have been identified by culture, treatment regimens should be simplified and directed to that pathogen or those pathogens. (See 'Subsequent antimicrobial therapy' above.)

- **Duration of therapy** – The appropriate duration of antimicrobial therapy should be determined on a case-by-case basis, taking into account the adequacy of surgical drainage, as well as the clinical, laboratory (white blood cell count, erythrocyte sedimentation rate, C-reactive protein), and radiographic response to therapy. The usual duration of therapy is six to eight weeks. The first follow-up MRI is obtained at about four to six weeks if the patient is improving or at any time if clinical deterioration occurs. (See 'Duration' above.)

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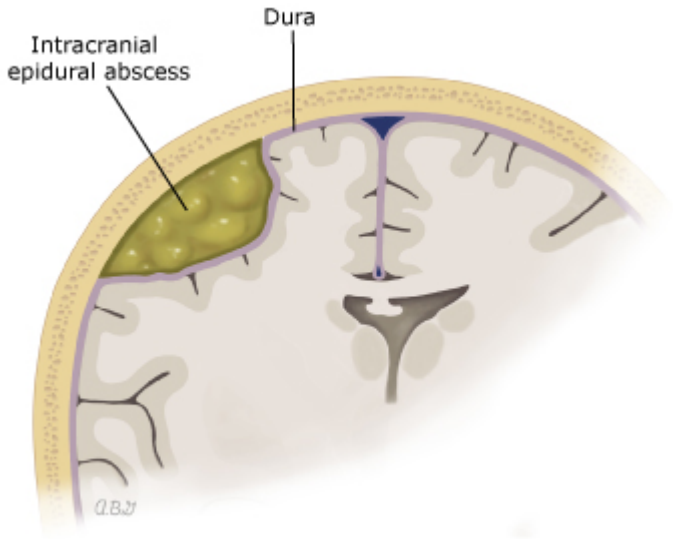
GRAPHICS

Intracranial epidural abscess



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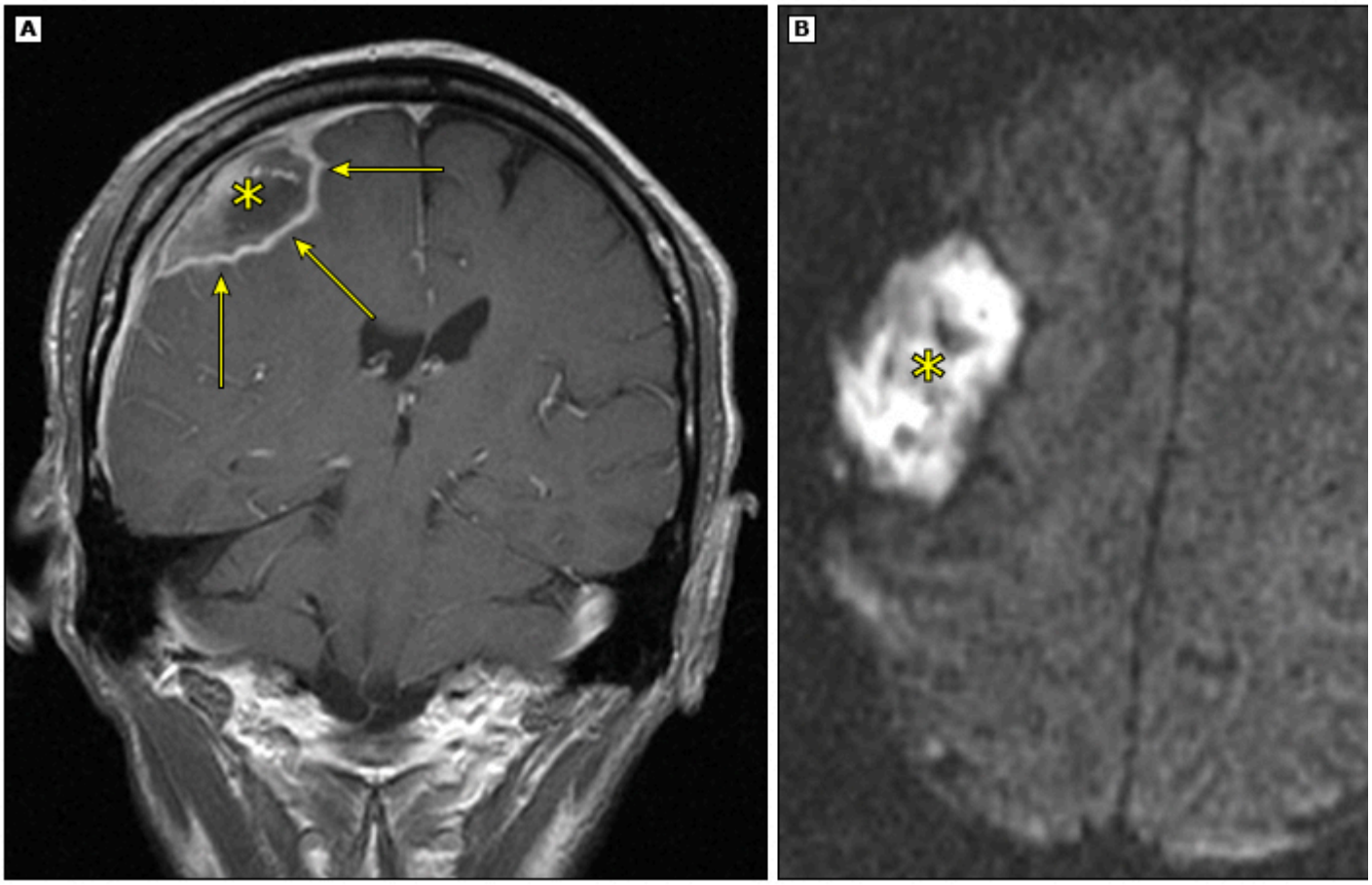
Intracranial epidural abscess by MRI

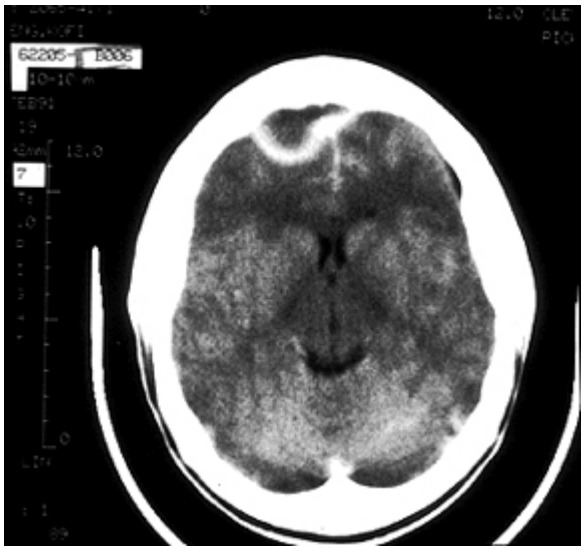


Intracranial epidural abscess in a 55-year-old patient six weeks after surgical intervention for chronic subdural hematoma. (A) T1-weighted MRI showed a contrast-enhancing (arrows) space-occupying lesion (asterisk) with (B) an increased signal intensity on diffusion-weighted imaging (asterisk).

MRI: magnetic resonance imaging.

Graphic 105710 Version 4.0

Intracranial epidural abscess by CT



Axial contrast-enhanced CT scan in a 36-year-old woman with frontal sinusitis who has developed headache and fever. There is a large peripherally enhancing extraaxial fluid collection in the anterior cranial fossa. The biconvex shape is a classic finding with an epidural empyema.

CT: computed tomography.

Courtesy of Andrew L Wagner, MD.

Graphic 69915 Version 4.0

Approach to vancomycin dosing for adults with normal kidney function*

Loading dose (for patients with known or suspected severe *Staphylococcus aureus* infection)¶	Load 20 to 35 mg/kg (based on actual body weight, rounded to the nearest 250 mg increment; not to exceed 3000 mg). Within this range, we use a highe dose for critically ill patients; we use a lower dose for patients who are obese and/or are receiving vancomycin via continuous infusion.
Initial maintenance dose and interval	Typically 15 to 20 mg/kg every 8 to 12 hours for most patients (based on actual body weight, rounded to the nearest 250 mg increment). In general, the approach to establishing the vancomycin dose/interval is guided by a nomogram.Δ
Subsequent dose and interval adjustments	Based on AUC-guided (preferred for severe infection)[1] or trough-guided serum concentration monitoring.◇

AUC: area under the 24-hour time-concentration curve.

* Refer to the UpToDate topic on vancomycin dosing for management of patients with abnormal kidney function.

¶ For patients with known or suspected severe *S. aureus* infection, we suggest administration of a loading dose to reduce the likelihood of suboptimal initial vancomycin exposure. Severe *S. aureus* infections include (but are not limited to) bacteremia, endocarditis, osteomyelitis, prosthetic joint infection, pneumonia warranting hospitalization, infection involving the central nervous system, or infection causing critical illness.

Δ If possible, the nomogram should be developed and validated at the institution where it is used to best reflect the regional patient population. Refer to the UpToDate topic on vancomycin dosing for sample nomogram.

◇ Refer to the UpToDate topic on vancomycin dosing for discussion of AUC-guided and trough-guided vancomycin dosing. For patients with nonsevere infection who receive vancomycin for <3 days (in the setting of stable kidney function and absence of other risk factors for altered vancomycin kinetics), vancomycin concentration monitoring is often omitted; the value of such monitoring prior to achieving steady state (usually around treatment day 2 to 3) is uncertain.

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