Vertebral Osteomyelitis and Epidural Abscess

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KEYWORDS

• Vertebral • Osteomyelitis • Epidural • Abscess • Spinal • Infection

KEY POINTS

- Diagnosis of severe spinal infections requires appropriate index of suspicion, comprehensive assessment of epidemiologic risks, prompt identification of clinical findings, and assessment with advanced imaging.
- Neurologic compromise from a severe spinal infection warrants urgent surgical evaluation.
- Infectious disease consultation assists with navigating complexities of antimicrobial management including choice and duration of therapy, use of adjunctive rifampin, and need for suppressive treatment.

INTRODUCTION

Vertebral osteomyelitis (VO) and spinal epidural abscess (SEA) represent serious musculoskeletal infections associated with functional morbidity, extension to central nervous system (CNS) disease, and risk of mortality.[1] While Morgagni first described SEA in 1761, modern clinicians continue to face many challenges in the diagnosis and management of this potentially devastating condition. Infectious pathogens may cause disease in any of the structures of the spinal cord and its support structures, but certain anatomic locations are much more frequently involved than others. This review will focus on VO and SEAs with or without discitis.

Multiple protective support structures surround the spinal cord and its nerve roots, and an understanding of this anatomy is critical to defining its associated infections. The cord is first surrounded by the meninges, which consist of the pia mater, arachnoid mater, and dura mater moving outward. Pyogenic intramedullary infection, or infection within the spinal cord itself, can occur but is uncommon and beyond the scope of this review. The arachnoid mater is a web-like layer immediately beneath the dura and represents the first potential space outside of the cord itself, known as the subarachnoid space, where a space occupying infectious process can occur. Infections within this subarachnoid space, however, are also rare. Continuing outward,

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Abbreviations	
CI	confidence interval
CNS	central nervous system
CRP	C-reactive protein
CSF	cerebrospinal fluid
CT	computed tomography
ESR	erythrocyte sedimentation rate
HIV	human immunodeficiency virus
MRSA	methicillin-resistant *S aureus*
OR	odds ratio
VO	vertebral osteomyelitis
WBC	white blood cell

the more rigid dura serves as the primary protective soft tissue layer surrounding the cord. External to the dura lies the epidural space, which serves as the potential space of interest in this review. The rigid ligamentum flavum posteriorly, ligamentum denticulatum laterally, and anterior longitudinal ligament anteriorly define the borders of the epidural space. The bony structures of the spinal column surround these ligamentous components including the vertebral bodies anteriorly, lamina and lateral processes laterally, and spinous processes posteriorly. The intervertebral discs sit between the vertebral bodies, serve as a cushioning support, and frequently become concomitantly infected.[1,2]

The different varieties of severe spinal infection share characteristics of evaluation and treatment despite their anatomic distinctions. VO describes infection of the bones comprising the spinal column. SEA is defined as a coalesced space occupying collection within the space between the spinal dura mater and the rigid ligamentous structures and periosteum of the adjacent bony vertebral structures. Infections may meet both definitions concomitantly and, in clinical practice, overlap frequently exists between the two in terms of clinical presentation, epidemiologic risks, diagnostic strategies, and treatment. Furthermore, infection of the gelatinous disc in the intervertebral body spaces and abscess within the adjacent soft tissues including the psoas muscle bodies commonly occurs in conjunction with one or both of the above conditions, and thus management often incorporates their treatment as well.[3,4]

DISCUSSION

Epidemiology

Several reviews on SEAs and VO have been published but data on their incidence, risk factors, and management did not emerge until recent decades. Reihsaus's review of 915 cases of SEA and McHenry's review of 253 cases of VO provide insight into various risk factors[1,3] Traditional risk factors for severe spinal infections include

- Advanced age
- Male sex
- Diabetes mellitus
- Immunosuppression including corticosteroid therapy
- Recent hematogenous infection
- History of underlying spinal column abnormality
- Injection drug use
- Recent traumatic injury to the spinal column.[1,5,6]

Interestingly, approximately half of severe spinal infections had a concomitant or recent infection at another site—44% of SEA and 51% of VO. Nearly a third of those

with SEA recalled recent history of a furuncle or other skin and soft tissue infection.[1,3] A 1999 epidemiologic review noted increasing rates of severe spinal infections over time and subsequent reviews further supported this finding, which stresses the importance of high clinical suspicion for these processes.[7–9] While the previously identified risk factors remain important, other factors including injection drug use associated with the opioid epidemic and prior medical intervention at the affected site, whether surgical or percutaneous treatment of chronic pain associated with the increasing aging population, have become more prevalent. More recently, health care-associated vertebral infections have emerged as complications of neurostimulator devices, central venous access lines, hemodialysis access devices, and even peripheral intravenous lines.[10,11]

Severe spinal infections result from a variety of infectious pathogens including bacteria, mycobacteria, fungi, and parasites. Typical bacterial pathogens comprise the largest number of cases with *Staphylococcus aureus*, whether methicillin-susceptible or methicillin-resistant, representing the most commonly indicted organism. In Reihsaus's review, 751 cases of SEA had an identified organism and 73% of those were *S aureus*. McHenry's review noted similar findings for VO with 69% (123 out of 174) of culture positive cases yielding *S aureus*, and these findings were supported in another large review.[12] These results reflect the association among *S aureus* and skin infections, unintentional or iatrogenic skin barrier penetration, and the virulence of *S aureus* to cause metastatic infection in the setting of bacteremia. Over time, the rates of community-acquired methicillin-resistant *S aureus* (MRSA) have increased, and thus, the rates of severe spinal infection related to MRSA will likely continue to follow this trend.[13] Other skin flora including Streptococci, *Cutibacterium acnes*, *Staphylococcus epidermidis*, and other coagulase-negative Staphylococci are seen to a lesser extent, present more indolently, and typically occur in relation to indwelling devices or recent instrumentation. Enteric organisms including *Escherichia coli*, *Klebsiella pneumoniae*, *Proteus mirabilis*, *Enterobacter* spp, and Enterococci may cause severe spinal infections in those with recent gastrointestinal or urologic illnesses or instrumentation. *Pseudomonas aeruginosa* represents another important pathogen that, while less common, typically relates to recent health care procedures or spinal instrumentation.[1,3]

Unusual bacterial organisms such as *Brucella* and other pathogens including mycobacterial, fungal, and parasitic organisms comprise a small portion of total cases of severe spinal infections. Infection with *Brucella* species tend to occur in those with specific epidemiologic risk, while the different mycobacterial species have various associated risk factors. Fungal musculoskeletal infections most commonly arise due to *Candida* or *Aspergillus* species. Parasitic infections causing spinal disease are exceedingly rare, but *Dracunculus medinensis*, *Schistosoma* species (primarily *Schistosoma mansoni*), *Toxoplasma gondii*, and *Echinococcus granulosus* have been reported.[1,14,15] Neurocysticercosis due to infection with *Taenia* species larvae, while commonly causing intracranial disease in endemic areas, causes spinal lesions extremely infrequently.[16]

Identification of spinal disease due to *Mycobacterium tuberculosis* requires clinical suspicion and recognition of presenting features. With ever-increasing globalization what was considered an infection of only certain parts of the world can now present in immigrants, travelers, and even those without history of travel or exposure to *M tuberculosis*. Extrapulmonary disease may occur with or without concurrent active pulmonary disease and the spine represents the most common site of the 10% of cases who develop musculoskeletal infection. *M tuberculosis* spinal disease, also known as Pott's disease, classically begins with involvement of the anterior portion

of the vertebral body and is characterized by "cold abscesses," or areas of infected material without surrounding inflammation. Neurologic symptoms from *M tuberculosis* disease result from compression of adjacent neurologic structures and bony destruction frequently leading to kyphotic changes of the spinal column. Disease may involve any level of the vertebral column, but the lower thoracic region represents the most common site, and thus, an anterior lesion in the lower thoracic spine in a host with risk factors for *M tuberculosis* should prompt careful consideration of this diagnosis.[17]

Pathogenesis

Infectious pathogens gain access to the epidural space or vertebral bodies via contiguous or hematogenous spread. In many cases, the route of infection acquisition remains unclear, but the risk factors discussed earlier can help form a clinical hypothesis. Local introduction occurs by means of recent surgical instrumentation or direct inoculation after a penetrating injury. Vertebral bodies can occasionally become infected as a result of contiguous spread from retroperitoneal pathology including infected aortic graft material.[3] Conversely, hematogenous seeding by long-standing or even transient bloodstream infections originating from another source may precipitate spinal infection.[1] Disruption of skin or mucosal barriers allows for invasion of colonizing organisms into the bloodstream where they may then seed the epidural space. Additionally, the valveless venous system of the spinal column known as Batson's plexus allows for the spread of infection to and throughout the spinal region with relative ease.[17]

Neurologic compromise due to severe spinal infection results from physical compression of the vital spinal structures. SEAs may cause mass effect within the confined epidural space thus compressing the spinal cord or its roots. VO can cause progressive damage to the bony support structures leading to vertebral body compromise and collapse. This collapse brings with it unpredictable changes to the anatomy of the region and creates substantial risk for direct compression of the spinal cord structures. Additionally, vascular insult related to thrombophlebitis of the associated vessels because of local infection may contribute as well.[1,3]

Clinical Presentation

Clinicians must maintain a high index of suspicion for severe spinal infections given that SEAs and VO may present in any portion of the spinal column with multiple nonspecific symptoms. SEAs most commonly form in regions of larger epidural space with adjacent fat and thus posterior thoracolumbar locations represent the most frequent sites of infections.[11] VO preferentially involves the lumbar spine followed by the thoracic and then cervical spine.[6,12,18] However, clinical experience and the literature highlight the possibility of multifocal disease with or without interspersed unaffected structures. The most common presenting symptoms for spinal infections include

- Neck or back pain (about three-fourths of cases)
- Fever (about one-half of cases)
- New neurologic deficit(s) (about one-third of cases)

Heusner[19] published a description of the stages of symptom progression in 1948, which still helps guide workup and treatment today. This staging system outlined in **Table 1** progresses from Stage 1 to Stage 4 and can be used to guide the need for surgical intervention for epidural abscesses. Suspicion for an abscess with neurologic impacts should prompt urgent evaluation given that progression through Heusner's stages and the associated neurologic deficits can occur at variable rates.[11]

Table 1
Heusner's classification of neurologic symptom progression

Heusner Stage Classification	Clinical Symptoms
Stage 1	Fever, focal back pain with severe point tenderness at the affected site
Stage 2	Back pain with radiation into the extremities, meningeal signs of irritation including neck stiffness, Lhermitte's sign, Kernig's sign, and Brudzinski's reflex
Stage 3	Neurologic deficits including muscular weakness, bowel, or bladder incontinence
Stage 4	Sensory deficits, progression of muscular weakness to paralysis

The combination of fever with new or worsening neck or back pain, especially in the context of concurrent bacteremia, infective endocarditis, or bacteremia within the past 3 months raises high concern. Fever and new neurologic deficits with or without back pain also raises suspicion in the appropriate clinical context.[20] However, the full triad of new back pain, fever, and neurologic deficit(s) occurs in a minority of patients.

Assessment/Evaluation

Clinical evaluation begins with a thorough history and physical examination. History should include details of any prior spinal or systemic infection and, when appropriate, screening questions evaluating for more unusual pathogens associated with travel or specific exposures. Clinicians should consider testing for pathogens including fungal organisms, mycobacteria including tuberculosis, and *Brucella*, among others, in hosts with appropriate epidemiologic risks. See **Table 2** for risk factors to consider which may warrant specialized testing for unusual pathogens.[17,21–27]

Laboratory testing includes hematologic, biochemical, and microbiologic studies that combine to guide severe spinal infection diagnosis. Serum white blood cell (WBC) count may be elevated but lacks sensitivity or specificity for severe spinal infections. Baseline erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) are appropriate in cases where an infectious etiology of clinical or imaging findings is suspected. These inflammatory markers, while nonspecific, provide additional evidence to suggest an infectious etiology when elevated or refute an infectious etiology when within normal limits especially in cases of otherwise equivocal findings.[20] Lumbar puncture is not recommended as part of routine workup of SEA or VO given the small, but clinically relevant, risk of neurologic decompensation or seeding of the cerebrospinal fluid (CSF) while passing through an infected space for access to the CSF for sampling.[28] Additionally, CSF studies are not described in the majority of reviews in the literature so data on their predictive value is limited. In Darouiche's review of SEA cases, 22 of 30 patients in whom CSF was obtained demonstrated CSF pleocytosis (WBCs >5 per mm^3), protein was elevated greater than 45 mg/dL in 26 of 29 cases, glucose was low ($\leq$50 mg/dL) in 14 of 26 cases, and CSF culture was positive in 6 of 24 cases. CSF studies were within normal limits in 3 of 30 cases.[5] However, other reviews note low rates of positive CSF cultures, and those few cases were associated with concomitant positive blood cultures for the same organism.[5,11] Given these mixed and limited data and the fact that treatment of SEA or VO will include an antimicrobial duration that exceeds typical meningitis or ventriculitis treatment durations, the utility of spinal fluid analysis is limited in these cases.

Table 2
Epidemiologic risk factors for unusual causes of severe spinal infections

Pathogen	Endemic Regions(s)	Epidemiologic History and Risk Factors
Brucella spp	Mediterranean and Middle East	Consumption of unpasteurized dairy or raw meat from infected animals Exposure to birth products or waste of infected animals
Tuberculosis	Widespread[a]	Residence in endemic region Advanced human immunodeficiency virus (HIV)
Nontuberculous mycobacteria	Widespread	Trauma, especially with environmental contamination Prior surgical instrumentation
Fungi (*Candida* spp and *Aspergillus* spp)	Widespread	Immunocompromised host Indwelling medical devices or prior surgical instrumentation
Toxoplasma gondii	Widespread	Exposure to cat litter Immunocompromised host • Advanced HIV • Solid organ transplant recipient
Dracunculus medinensis	Tropical Africa and Asia	Poor sanitation
Schistosoma mansoni	South America, Caribbean, Africa, and Middle East	Exposure to water contaminated by snails
Echinococcus granulosus	Temperate climates	Close contact with sheep (direct or indirect via dogs that become infected)

[a] Sub-Saharan Africa and Southeast Asia are classically considered endemic regions for tuberculosis. However, *M tuberculosis* may be seen in travelers from all parts of the world and even in those without history of international travel or known *M tuberculosis* exposure.

Microbiologic identification of a causative organism is critical in treatment success for all forms of spinal infection. Evaluation should include 2 sets of blood cultures (ideally obtained prior to antimicrobial administration).[20] Some cases will have concomitant bacteremia, and blood cultures positive for a typical causative pathogen (*S aureus*, *Staphylococcus lugdunensis*, *or Brucella* species) preclude the need for dedicated invasive testing to establish a microbiologic diagnosis. However, others will have negative blood cultures, which complicates establishing the culprit organism.[12] The Infectious Diseases Society of America (IDSA) guidelines for VO advise obtaining a biopsy of an intervertebral disc space or vertebral endplate sample to be sent for culture. A similar approach has been employed for epidural abscesses in select cases if deemed clinically safe.[11] When tissue sampling is sufficient, pathologic examination of the specimen assessing for histologic changes associated with

osteomyelitis is recommended. A second biopsy should be considered if an initial sample is inconclusive or negative and clinical suspicion remains high.[20] As discussed, additional testing for unusual pathogens should be considered if epidemiologic risks dictate, but the details of these testing modalities including usage, performance, and limitations are beyond the scope of this review.

Radiographic imaging is critical in characterizing extent of infection and assessing for complications. Contrast-enhanced MRI is the imaging modality of choice with sensitivity at or above 90% for both VO and SEA.[4,11] MRI not only provides detailed images that help in differentiating infectious from other invasive processes such as neoplastic disease but also can assist with surgical planning.[29] Computed tomography (CT) after myelography provides detailed images but also involves an invasive procedure and, thus, is not preferred given similar sensitivity of MRI.[30] Standard CT imaging, gallium or technetium-99 bone scan, and PET scan all represent alternative modalities but have downsides including sensitivity, specificity, and availability. Plain radiographs may demonstrate bony destructive changes in long-standing disease but their limited sensitivity in early disease processes make them an inadequate screening modality if severe spinal infection is suspected.[20,31] Clinicians must, however, always consider clinical context when interpreting imaging studies, especially those with highest sensitivity. Common mimickers of infectious etiologies including advanced degenerative changes, acute Schmorl's nodes, ankylosing spondylitis, and advanced neuropathic arthropathy may mislead treating providers and must be considered as alternative diagnoses in equivocal cases.[32]

Treatment

Treatment strategies for VO and SEA center around recommendations made in guidance documents including the 2015 IDSA guideline for native VO and expert opinion and large reviews of VO and SEA.[20] Described treatment approaches include antimicrobial therapy alone, antimicrobial therapy with debridement, or antimicrobial therapy with debridement and spinal instrumentation.[33–36] Consultation with specialists in both spine surgery and infectious diseases helps guide individualized management strategies within the aforementioned framework.

Antimicrobial therapy should be based on in vitro susceptibility testing of the causative pathogen when available. Establishing the causative pathogen with its accompanying susceptibility data helps guide treatment, and thus, clinicians should withhold empiric antimicrobial therapy while obtaining microbiologic specimens in the absence of hemodynamic instability or rapidly progressing neurologic compromise.[20] Many factors including antimicrobial spectrum, tissue penetration and bioavailability, and outpatient feasibility warrant consideration when determining both empiric therapy and definitive treatment. Specifically, antimicrobial penetration of certain agents into the CNS and whether CNS penetration is required in SEA or VO remains a topic of ongoing study and controversy.[37,38] **Table 3** lists reasonable first-line agents, but infectious disease consultation should be sought to best guide therapy.[4,11]

Adjunctive rifampin treatment remains an area of interest and study for various orthopedic infections due to *S aureus* including VO and spinal infections involving hardware. In Europe, rifampin has been used in combination therapy for native *S aureus* orthopedic infections with data supporting its use and an expert consensus statement suggesting rifampin in combination with a fluoroquinolone as an appropriate treatment option.[39] The benefits of rifampin center around its activity against bacterial biofilm. Most typical antimicrobials have limited activity against pathogens within biofilm due to the pathogen's physical protection by a layer of extracellular polymeric substance forming a glycocalyx and the pathogen's decreased replication and protein

Table 3
Reasonable first-line antimicrobial options for common bacterial pathogens causing severe spinal infections

Organism	First-Line Treatment
Methicillin susceptible *Staphylococcus aureus* or coagulase negative staphylococci	Antistaphylococcal penicillin (oxacillin or nafcillin)
Methicillin-resistant *S aureus* or coagulase negative staphylococci	Vancomycin
Streptococci	Penicillin *or* ceftriaxone[a]
Enteric gram-negative organisms (*Escherichia coli*, *Klebsiella* spp, and so forth)	Ceftriaxone *or* cefepime[a]
Pseudomonas aeruginosa	Cefepime *or* ceftazidime[a]
Culture-negative infection or when cultures cannot be safely obtained	Vancomycin *and* ceftriaxone *or* vancomycin *and* cefepime

[a] Antimicrobial selection should be based on in vitro susceptibility data.

synthesis rates.[40] In addition to its propensity to form on hardware, experts believe that damaged or necrotic bone serves as an additional nidus for biofilm formation and thus rifampin utilization may have benefits in cases even without hardware.[41] A recent meta-analysis evaluated the benefit of adjunctive rifampin therapy for native VO and, while noting a statistically significant difference in outcomes (absolute risk reduction of 14%, confidence interval [CI] −19 to −8, $P<.001$) with rifampin use and subgroup analysis favoring rifampin–fluoroquinolone combination over other combinations, the authors note that the overall quality of data on this topic remains weak and limited by a multitude of factors.[42] If utilized, rifampin must always serve as part of a combination regimen and decisions regarding its use require individualization.

Duration of therapy for severe spinal infection is typically at least 6 weeks. Treatment duration is based on various factors including clinical progress, infectious pathogen involved, and the presence of fixation hardware. A multicenter French study noted 6 weeks of therapy is noninferior to 12 weeks in patients with native VO and a microbiologically confirmed organism excluding *Brucella* species, fungal pathogens, or mycobacteria.[43] McHenry's VO review noted significantly worse outcomes for patients treated with less than 4 weeks of definitive therapy although they noted more deaths and presumed higher burden of disease in that group as well.[3] However, patients with risk factors for recurrence including infection with methicillin-resistant *S aureus*, those with an undrained paravertebral or psoas abscess, or those with end-stage renal disease may benefit from a longer course of treatment up to 12 weeks in order to reduce risks of recurrent disease.[44]

Severe spinal infections, particularly SEA, frequently warrant surgical intervention. Operative management of dorsal abscesses typically includes a laminectomy to allow for drainage of the collection and decompression of the epidural space. Anterior abscesses pose additional challenges as access to the site imparts further complexity to surgical treatment.[1] Conservative management with medical therapy alone has been validated in select patient situations. These include

- Those without a defined epidural abscess
- Those with a small abscess (<2 vertebral levels) and no neurologic deficits
- Those with neurologic deficits that have been present for greater than 36 hours prior to presentation without progression[45,46]

Prior clinical experience suggests that abscesses that have been present for greater than 2 weeks likely form granulomatous masses as opposed to liquid pyogenic collections and, thus, have decreased chances of causing progressive disease.[47] However, recurrence or progression despite appropriate antimicrobial therapy can occur in anywhere from 8% to 48% of cases, and many patients treated with antimicrobial therapy alone (16%–73%) will experience residual neurologic deficits.[30,46,48] Urgent consultation with an orthopedic spine surgeon or neurosurgeon should be considered in cases where neurologic deficits are noted as the progression to long-standing deficits including paralysis may be rapid.[49] Additionally, concurrent bacteremia, which fails to clear on appropriate antibacterial therapy warrants surgical evaluation for source control of the infectious process.[20]

Evaluation of the anatomic stability of the spine both before and after any debridement procedure is performed, and it determines whether spinal instrumentation with stabilization hardware may be required. Concerns surrounding the placement of fixation hardware into an actively infected space are well documented in various types of device-associated infections and focus mainly on the concept of biofilm formation on the hardware.[50] While this creates uncertainty and concern about surgical intervention especially with instrumentation, Dennis Hey and colleagues[33] performed a retrospective review of 84 patients, which found that mortality rate was significantly lower for patients who underwent surgical debridement (odds ratio [OR] = 0.80, 95% CI = 0.70 to 1.00, P=.02) and for those who underwent debridement with instrumentation (OR = 0.82, 95% CI = 0.70–0.96, P=.01) compared to those who received antimicrobial therapy alone. The authors noted no statistically significant difference in recurrence or reoperation rates of those who underwent spinal instrumentation compared to those who did not, but they did not discuss details of antimicrobial therapy including whether patients received suppressive therapy. Data support the use of adjunctive biofilm-active agents such as rifampin in implant-associated spinal infections including Köder's 10 year cohort, which noted statistically significant improvement in infection-free survival at 1 and 2 years in cases treated with a biofilm active agent.[51] In cases where hardware is retained or placed at the time of debridement, clinicians may choose to pursue a suppressive antimicrobial regimen after completion of an initial course based on the assessment of risk for recurrence and biofilm formation. Limited data exist to guide this strategy, and individualized assessment dictates the need for suppression. Therefore, such decisions should occur in conjunction with an infectious disease specialist.

Treatment failure in epidural abscess and VO may be challenging to define. Microbiologically confirmed relapse via either repeat operation with consistent positive culture or recurrent bloodstream infection can occur when burden of disease is high or initial debridement of infected bony material was suboptimal. McHenry's review[3] noted relapse in 14% of total cases with 75% of those occurring within the first 12 months and 64% occurring after the completion of the planned antimicrobial regimen. Twenty of 36 of those relapsed cases had undergone surgical debridement further suggesting that combination of adequate debridement with an appropriate duration of optimally dosed antimicrobial therapy is critical. In cases of VO, pain (28%), motor weakness or paresis (16%), or bowel or bladder incontinence (7%) may persist even after appropriate treatment.[12] Persistently elevated ESR and CRP and persistent radiographic changes do not signify treatment failure. ESR and CRP that remain elevated 4 weeks into therapy may provide a clue to ongoing infection but should be interpreted in the context of the patient's clinical progress. Follow-up MRI may show persistent changes that do not correlate with persistent active infection, and thus, MRI should not routinely be repeated in cases of osteomyelitis unless

clinical failure is suspected or there is specific concern about a soft tissue process or epidural abscess.[20]

Avoidance of diagnostic and therapeutic pitfalls in the treatment of severe spinal infections remains of paramount importance. Urgent assessment by the appropriate surgical specialists helps prevent diagnostic and therapeutic delays. Any alternative imaging study including standard CT demonstrating VO should be followed by an MRI to evaluate for epidural abscess as the presence of an abscess may change therapeutic management. Patients with an epidural abscess at 1 vertebral level warrant careful evaluation for an abscess at other levels especially in the context of concomitant bloodstream infection. Given the propensity for severe spinal infections to concomitantly occur with other significant infections like endocarditis appropriate screening must be performed based on subjective and objective data including the organism involved.[52] The duration of antimicrobial therapy depends on various factors as outlined and consultation with an infectious disease specialist should be sought when available.[11]

FUTURE DEVELOPMENTS

The use of oral antimicrobial therapy as a stepdown regimen continues to gain favor in various realms of infection treatment.[53] The current IDSA guidelines recommend intravenous therapy as the standard of care for severe spinal infections.[20] However, given the many potential benefits and risk mitigation of oral regimens, researchers have begun assessing the feasibility of an oral stepdown treatment plan for severe spinal infections, most frequently VO.[54] Passerini and colleagues[55] provided an institutional perspective and systemic review of these data for VO. They noted that between 2019 and 2021 only 2 of 148 cases at their institution were treated with an early switch to an oral regimen defined as within 2 weeks of therapy initiation. Their systematic review, however, included 14 studies totaling 1078 patients and noted no significant difference in outcomes between those with an early switch to oral therapy. These results while promising, must be considered within the context of the limitations of the included studies such as

- Retrospective studies
- Inconsistencies in causative organism(s), antimicrobial utilized, duration of treatment
- Risks for bias including those with “less severe” infections receiving stepdown therapy.

At this time, the data do not yet conclusively allow for a paradigm shift of severe spinal infection management. However, these data hold promise for future developments and permit consideration of oral regimens as stepdown in carefully selected cases.

SUMMARY

Severe spinal infections burden patients with risks of long-standing morbidity and even mortality. Identification of the causative pathogen involves thoughtful assessment of epidemiologic risks and holds paramount importance in guiding management. When the diagnosis of VO or SEA are suspected prompt evaluation with imaging, preferably MRI, helps establish the diagnosis and help prevent long-term sequelae associated with delayed identification of disease. Consultation with experts in surgical management and infectious disease defines the short-term and long-term treatment plans including operative intervention, antimicrobial choice, treatment duration, and follow-up requirements. Intravenous therapy represents the current standard of

antimicrobial management, but with evolving data, oral stepdown therapy may become more commonplace in the future.

CLINICS CARE POINTS

- VO and SEA have overlapping clinical presentations and may exist as concomitant infections.
- Acute and chronic sequelae of severe spinal infections are dictated by their effects on surrounding vital structures.
- Early identification of severe spinal infection is critical in preventing poor outcomes; MRI is the imaging modality of choice and should be obtained in cases of new back pain with fever with or without neurologic deficits.
- Blood cultures should be obtained in all cases of suspected spinal infection; if these are unrevealing, direct sampling via image-guided techniques or surgical intervention should be considered.
- Antimicrobial therapy should be withheld to preserve culture yield except in cases of hemodynamic instability or concern for rapidly progressing neurologic deficits.
- Treatment includes at least 6 weeks of antimicrobial therapy with or without surgical intervention; expert consultation with a surgical specialist and infectious disease specialist helps guide these decisions.
- Follow-up requirements must be assessed on a case-by-case basis as serial inflammatory markers and imaging findings may be misleading.

DISCLOSURE

The author has nothing to disclose.

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