Upper Cervical Epidural Abscess in Clinical Practice: Diagnosis and Management

Khalid Al-Hourani[1] Rami Al-Aref[2] Addisu Mesfin[3]

[1] Department of Orthopaedic Surgery, Bristol Royal Infirmary, Bristol, England, United Kingdom
[2] Wayne State University School of Medicine, Detroit, Michigan, United States
[3] Department of Orthopaedic Surgery, University of Rochester Medical Center, Rochester, New York, United States

Address for correspondence Addisu Mesfin, MD, Department of Orthopaedic Surgery and Oncology, University of Rochester School of Medicine and Dentistry, 601 Elmwood Avenue, Box 665, Rochester, NY 14642 (e-mail: Addisu_mesfin@urmc.rochester.edu).

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Abstract

Study Design Narrative review.

Objective Upper cervical epidural abscess (UCEA) is a rare surgical emergency. Despite increasing incidence, uncertainty remains as to how it should initially be managed. Risk factors for UCEA include immunocompromised hosts, diabetes mellitus, and intravenous drug use. Our objective is to provide a comprehensive overview of the literature including the history, clinical manifestations, diagnosis, and management of UCEA.

Methods Using PubMed, studies published prior to 2015 were analyzed. We used the keywords "Upper cervical epidural abscess," "C1 osteomyelitis," "C2 osteomyelitis," "C1 epidural abscess," "C2 epidural abscess." We excluded cases with tuberculosis.

Results The review addresses epidemiology, etiology, imaging, microbiology, and diagnosis of this condition. We also address the nonoperative and operative management options and the relative indications for each as reviewed in the literature.

Conclusion A high index of suspicion is required to diagnose this rare condition with magnetic resonance imaging being the imaging modality of choice. There has been a shift toward surgical management of this condition in recent times, with favorable outcomes.

Keywords
- spinal epidural abscess
- upper cervical spine
- osteomyelitis
- neurologic deficits
- atlas
- odontoid
- axis

Introduction

Upper cervical (occiput to C2) epidural abscess (UCEA) is an uncommon condition. Spinal epidural abscesses usually are surgical emergencies because of concurrent neurologic deficits. In upper cervical spine infections, degradation of the odontoid ligaments with subsequent atlantoaxial subluxation or dislocation is a risk. The prevalence of osteomyelitis at this level has increased significantly over the past decades primarily due to immunocompromised hosts, intravenous drug use, and infective endocarditis.[1] However, there remains a lack of literature on factors influencing neurologic impairment or the prediction of neurologic and functional recovery.[2]

Epidemiology

UCEAs are a relatively rare condition. To our knowledge, 34 cases were published in the literature since the early 1900s. Although this condition is less common than other spinal epidural abscesses, it is arguably more destructive than its counterparts. Many of the long-term clinical sequelae are secondary to its proximity to both the atlas and axis.

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Spinal epidural abscess in general has an incidence of ~2 to 25 patients per 100,000 admitted to the hospital.[2] Due to the presence of immunocompromised hosts, more invasive procedures, instrumentation, and more accurate imaging, the prevalence has been increasing steadily over the past few decades.[1] The increasing prevalence along with the destructive nature of the pathology signifies the importance of identifying appropriate treatment protocols.

Anatomy

The cervical spine is composed of seven vertebrae (C1–C7), which provide mobility, flexion, extension, and rotatory motion of the neck. The cervical spine is divided into upper, subaxial, and cervicothoracic regions. The *upper cervical spine* refers to the occipitocervical junction, C1 (atlas), and C2 (axis).[3–6] In turn, the *subaxial spine* refers to C3–C6, and C7–T1 is referred to as the *cervicothoracic region*.

The atlas is unique in that it lacks a vertebral body, instead forming a ring that articulates with both the occiput (atlantooccipital joint) and the axis (atlantoaxial joint). The atlantooccipital and the atlantoaxial joints provide the majority of movement associated with the head. The atlantoaxial joint is specifically created by the dens (or odontoid process) articulating with the posterior aspect of the anterior arch of the atlas.[7] The odontoid process is an extension of the C2 vertebral body. Similar to other vertebral bodies, the axis has pedicles and transverse processes. The transverse processes serve as a major point of attachment for muscles and ligaments. Stabilization for the atlantoaxial joint occurs via the transverse ligament at the atlantoaxial joint. Further stabilization is provided by the apical and alar ligaments, which help to prevent the posterior dislocation of the dens.[7]

The development and location of epidural abscesses is in part secondary to the presence of a true epidural space. There is generally adhesion of the dura mater at the foramen magnum superiorly and at the sacrococcygeal membrane inferiorly.[8] Anteriorly, the epidural space is almost virtual as the dura, posterior longitudinal ligament, and periosteum of the vertebral body are in close contact, which results in most spinal epidural abscesses occurring posteriorly.[9] The true epidural spaces occur at the cervical, midthoracic, and lumbosacral regions. The cervical region is a much smaller epidural space and as such is less prone to infection. Generally, spinal epidural abscesses are more common in the lumbar area because it has a larger epidural space with more tissue prone to infection. The cervical region has a smaller epidural space, explaining the relatively rare incidence of UCEAs.

Pathology and Microbiology

The underlying disease (immunocompromised host) and surgical interventions predispose toward the development of spinal epidural abscess.[10] Specifically, the patients with comorbidities such as diabetes, immunodeficiency, obesity, traumatic spinal cord injury, epidural catheter placement, intravenous drug abuse, and surgical instrumentation seem to be at a particularly increased risk.[11–26] In our analysis of the literature, many of the predisposing factors remained the same; the most common factor by far is diabetes mellitus (►**Table 1**). Intravenous drug use and chronic kidney disease also represented a sizeable portion of our cases.

Table 1 Predisposing factors for upper cervical epidural abscess

Predisposing condition	*n*
Diabetes mellitus	11
Intravenous drug use	3
Chronic kidney disease	3
Human immunodeficiency virus	1
Alcohol excess	1

The suggested mechanism of the bacterial invasion into the spinal canal is hypothesized to be mechanical (i.e., invasion through the tissue planes permeating through to the epidural space), hematogenous invasion, or direct contamination from an adjacent infected structure.[17,27,28] Subsets of patients seem predisposed to spontaneous epidural abscess in which there is generally no identified source of infection. We found hematogenous spread and ear, nose, and throat pathology to be the most likely source of infection with some cases having both as a potential cause (►**Table 2**). From the cases reviewed, several patients had more than one source. In contrast, a proportion of patients had no identifiable source. Due to the anatomy of the spine, a bacterial invasion could begin at a specific spinal level and subsequently migrate to different vertebral levels. The development of advanced abscesses leads to a collection of pus within the spinal space. The clinical presentation is generally associated with mechanical compression, with pain and progressive neurologic deficits as the spinal cord is displaced.

Methicillin-sensitive *Staphylococcus aureus* was associated with almost two-thirds of cases of spinal epidural abscesses.[11,15,17,22,29] For UCEA, *S. aureus* was isolated in 60% of cases, and the next most common pathogen was *Streptococcus pneumoniae*. In 20% of cases, no pathogen

Table 2 Likely source of infection upper cervical epidural abscess

Source of infection	*n*
Hematogenous	11
Ear, nose, throat	8
Skin/soft tissue	7
None identified	7
Upper respiratory tract infection	3
Posttonsillectomy	2
Urinary	2
Dental	2
Meningitis	1
Lower respiratory tract infection	1

Note: some cases have more than one source.

Table 3 Isolated pathogen

Pathogen	*n* (%)
Staphylococcus aureus	24 (60)
Not isolated	8 (20)
Streptococcus pneumoniae	2 (5)
Pasteurella	1 (2.5)
Escherichia coli	1 (2.5)
Streptococcus viridians	1 (2.5)
Pseudomonas	1 (2.5)
Alpha-streptococcus	1 (2.5)
Klebsiella pneumoniae	1 (2.5)

was isolated (►**Table 3**). Few cases of anaerobic organisms and fungi including actinomyces and candida were reported for spinal epidural abscess. In our review of UCEA, we can only report one case with pasteurella as the anaerobe.[9,11,16,17,28]

Diagnosis

The classical triad of spinal epidural abscess is pain, fever, and neurologic deficit.[9] Specifically, UCEA seems to initially present with neck pain (33 cases), neck stiffness (18 cases), and/or fever (12 cases) as shown in ►**Table 4**. More insidious presentations included disorientation, headaches, sore throat, and pain on swallowing. The rapidity of symptom onset remains highly variable. The combination of neck pain or stiffness along with fever should raise suspicion for UCEA.

A full neurologic examination including cranial nerves is mandatory and may elicit sensorimotor deficit; however, a normal neurologic examination does not exclude the diagnosis. Respiratory compromise may also ensue. An ear, nose, and throat examination as part of the patient workup is also recommended and may identify a potential etiology for UCEA such as tonsillitis or suppurative otitis.

Table 4 Common signs and symptoms

Signs/symptoms	*n*
Cervical pain	33
Cervical stiffness	18
Fever	12
Motor weakness	5
Malaise	2
Jaundice	2
Cranial nerve weakness/palsy	2
Difficulty swallowing	1
Confusion	1
Headache	1
Back pain	1

As part of the evaluation, inflammatory markers such as erythrocyte sedimentation rate, C-reactive protein, and white blood cell count should be ordered. Although these markers are not specific to UCEA, they remain supportive of a diagnosis if UCEA is in the differential. In the cases we examined, erythrocyte sedimentation rate, C-reactive protein, and white blood cell count were elevated in most of the patients. These laboratory findings can be considered diagnostic only within the context of the complete clinical picture suspicious for UCEA.

Imaging

The initial imaging should include plain radiographs to assess for any common causes of neck pain such as cervical spondylosis or fractures. Additionally, it may show signs of vertebral osteomyelitis such as vertebral collapse or bony erosions. The odontoid view and/or flexion and extension views are indicated if osseous changes in the upper cervical spine are noted.

Magnetic resonance imaging (MRI) remains the modality of choice with the greatest diagnostic accuracy. The reported predictive values include sensitivity up to 95% and specificity over 90%.[9,30,31] Gadolinium enhancement can further increase these values due to its ability to differentiate between abscess and the surrounding neurologic structures. It is useful to compare T1- and T2-weighted images because in T2-weighted images, an epidural abscess will show uptake of signal whereas in T1-weighted images, the epidural abscess and spinal cord have a similar intensity (►**Fig. 1A**, **B**). Computed tomography (CT) is invaluable in the evaluation of vertebral end plate and facet erosions associated with osteomyelitis (►**Fig. 2A, B, C**). CT is also useful for surgical planning because instrumentation and stabilization are needed if there is significant facet and vertebral destruction.

If MRI is contraindicated, then CT myelography would be an option; however, this imaging presents its own risks including introduction of infection, bleeding, and nerve injury as well as the risks associated with radiation. Generally, CT myelography is no longer recommended but is an alternative if MRI is not available or contraindicated.

Cultures

Identifying the causative organism is possible in up to 75% of cases with CT-guided biopsy, which is crucial in the diagnostic pathway.[12] This identification should ideally be done as soon as a diagnosis of epidural abscess is confirmed on imaging. In our review, 27 of 41 cases had cultures obtained in the form of CT-guided aspirate, direct biopsy of tissue at surgery, transoral/retropharyngeal biopsy, or cultures sent following incision and drainage of abscess. Blood cultures are also essential in identifying the organism due to hematogenous spread being a route of infection; however, it has been reported that blood cultures are negative in up to 40% of cases of spinal epidural abscess.[32,33] Of 41 cases, 14 (34%) provided positive blood cultures in our study. Previous antimicrobial therapy is known to decrease the sensitivity of cultures; however, antibiotics should not necessarily be withheld

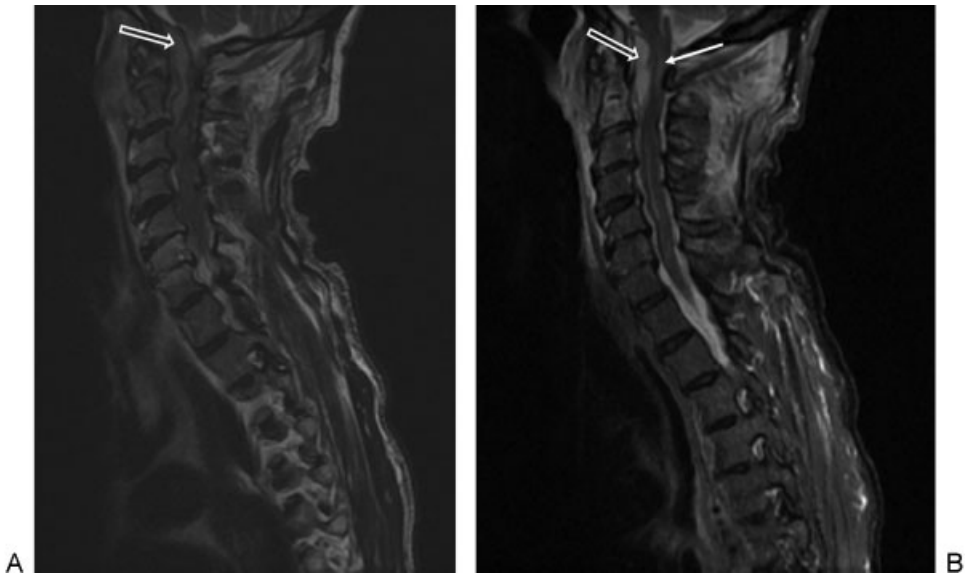

Fig. 1 (A) Sagittal T2-weighted magnetic resonance imaging demonstrating epidural abscess posterior the odontoid (arrow). (B) Sagittal short tau inversion recovery sequence demonstrating epidural abscess (open arrow) and spinal cord signal change in the upper cervical spine (closed arrow).

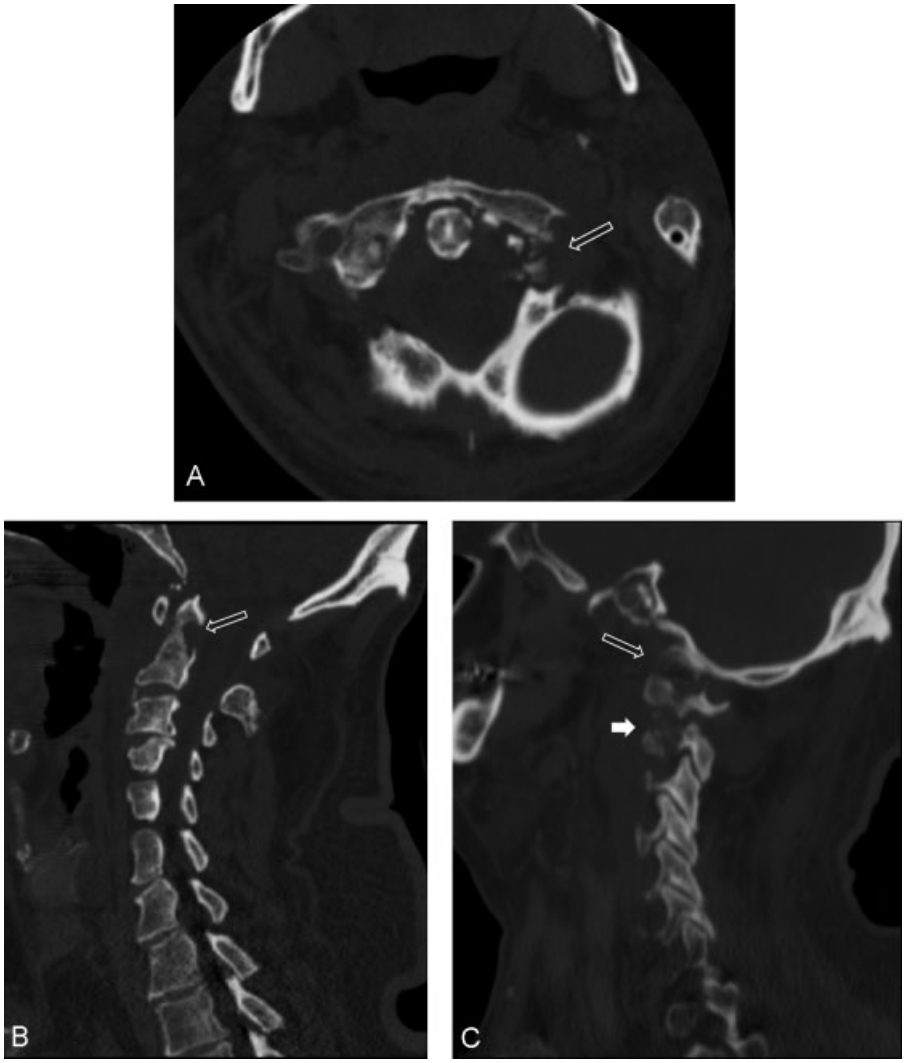

Fig. 2 (A) Axial computed tomography ()CT image of C1–C2 demonstrating left C1 lateral mass erosion (arrow). (B) Sagittal CT demonstrating erosion of the odontoid (arrow). (C) Sagittal CT demonstrating left occipitocervical (open arrow) and atlantoaxial articular destruction (closed arrow).

from the patient to increase culture sensitivity. Therefore, this decision to give or withhold antibiotics should be taken on clinical merit.[9] If another potential source of UCEA is identified such as throat, supportive otitis, or respiratory tract infection, then early appropriate cultures should also be obtained.

Management of UCEA

The treatment options for UCEA include nonoperative or operative management. Nonoperative management consists of immobilization and parenteral antibiotics, and operative management consists of surgical decompression, possibly stabilization and parenteral antibiotics. Nonoperative management with antimicrobials alone may be sufficient in some cases. The type of management largely depends on the case, with medical management alone being reserved for those with significant comorbidities rendering them unfit for surgery, patients with UCEA but no neurologic sequelae, and patients with neurologic deficit lasting more than 48 hours. Patients with rapidly developing neurologic signs and those with worsening inflammatory markers and radiologic signs should be treated operatively if possible. Patients with a destructive osteomyelitis or instability may need further surgery for arthrodesis/instrumentation as part of a combined single-stage (decompression/stabilization) or separate second-stage procedure. From reviewing the cases available to the authors (►**Table 5**), we did note a trend for nonoperative management of these cases certainly up to the 1980s, and thereafter there was a discernible shift toward operative management. Only 2 deaths were noted, with 1 UCEA that was managed nonoperatively and the other case managed operatively. In total, 15 patients were treated with immobilization and antibiotics; 1 of these patients did not survive and 4 developed limited cervical range of motion. Of the rest, Azizi et al described a case with abducens (cranial nerve VI) palsy at the initial presentation, which did not resolve despite antibiotic treatment. None of the patients who were treated nonoperatively had neurologic deficits at presentation, and the majority presented with neck pain and stiffness.[34]

Of the cases we reviewed, 23 were treated operatively mainly in the form of surgical decompression and immobilization with a halo vest. Four patients did not recover favorably: 1 of these patients subsequently died, 2 had limitation of cervical range of movement, and 1 did not recover from a preoperative hemiparesis. The remaining 18 made a full recovery, the earliest at 3-month follow-up and the latest at 2-year follow-up. Of those treated surgically, 3 had neurologic deficits in the form of preoperative tetraparesis, upper extremity numbness, and upper limb 4/5 power, respectively. All 3 made a full neurologic recovery postoperatively. Surgical management seems to be the overwhelming treatment of choice in recent times as it minimizes the neurologic damage and controls sepsis by diminishing the infected tissue burden. In a portion of patients with unstable cervical spines, an instrumented fusion may be required as either a primary or second-stage procedure. CT-guided needle aspiration has been described as an alternative treatment for epidural abscess, particularly reserved for those with a posterior spinal epidural abscess (SEA) and no neurologic deficit or those unable to withstand surgery.[33,35–37] However, in our review we did not encounter any UCEA cases treated in this manner.

Although there remains a discernible lack of evidence on the preference of management of UCEA in particular, recent studies have evaluated operative and nonoperative management of SEA, which can be used to guide our approach. Siddiq et al advocated that medical management alone with or without CT-guided drainage of the abscess is a safe and effective treatment irrespective of age, comorbidities, size of abscess, or even neurologic impairment at the time of presentation.[38] Another proponent of medical treatment alone is Bamberger, who compared the success rates of abscesses in various organs, including epidural, brain, and spine abscesses. Of 44 patients with SEA, 6 had bowel/bladder incontinence, 6 had extremity weakness, 4 had paraplegia or tetraplegia, and 2 had sensory levels. They concluded that of these 44, 40 were successfully treated nonoperatively; however, a limitation to the study was the criteria for success.[39]

Recent studies have suggested that independent risk factors can be used to predict the failure of nonoperative management. Kim et al found that patients with SEA who are over the age of 65, are diagnosed with diabetes, have a MRSA infection, and have a neurologic deficit also have a 99% risk of failing nonoperative management. Patients without these comorbidities can potentially be managed nonoperatively.[40] The duration of antibiotic management is largely dependent on local microbiology protocols; however, we can glean from our review that a prolonged course of parenteral followed by oral antibiotics is often required. Although the duration should be based on clinical improvement, decreasing inflammatory markers, and improvement on interval images (MRI), we did note in our review that at least 6 weeks of antibiotics were administered.

As spinal epidural abscess can occur at various levels within the spine including cervical, thoracic, and lumbar, it is important to note that the management strategies may differ. Although SEA at any level is a serious condition, it is particularly devastating in the upper cervical region due to the fragility of the atlantoaxial joint. Spinal cord compression can impact breathing due to diminished diaphragmatic innervation from C3, C4, and C5. To this effect, there may be a greater margin to consider nonoperative management of the thoracic and lumbar regions as opposed to the upper cervical spine where a large untreated epidural abscess can render the patient ventilator-dependent.

Although there remains a lack of evidence to delineate the indications for the timing of surgical intervention, it remains the consensus that early surgical decompression prevents the progression of neurologic impairment. Patel et al identified that patients who undergo early surgical intervention had improved motor recovery when compared with patients who underwent surgical therapy after failure of nonoperative treatment.[41] The mainstay of surgical treatment continues to be thecal sac decompression, drainage of the epidural abscess, and administration of

Table 5 Cases in the literature from 1931 to 2013 reported to have upper cervical epidural abscess

Authors	No. of patients with UCEA	Age/sex	Relevant comorbidities	Level of infection	Presentation	Organism	Treatment	Outcome	Source of infection	Onset	Aspirate	ESR/CRP/WCC	Antibiotic duration
Odelberg-Johnson et al 1931	1	16 y/F	Measles, whooping cough, rubella	C2	Fever, cervical pain, stiffness, CL	None identified	Plaster of Paris head enclosing head and neck placing the head in hyperextension and traction with the body acting as a counterweight	1.5-y f/u with resolution of neck pain, no limitations with flexion and extension, severe disability with rotation to the right	Posttonsillectomy	1–2 wk postop	–	ESR 58	Not mentioned
Frank et al 1944	1	43 y/M	–	C2	Cervical pain, limited ROM, stiffness in the occipital region; CL; dry tongue; erythematous throat; scattered rhonchi in lungs	*Staphylococcus aureus*	I&D using Hilton's method (multiple staphylococcal abscesses)	Death from meningitis secondary to osteomyelitis of the odontoid process around 15 wk from initial presentation	Cellulitis right hand following spider bite, urinary tract infection	3 wk	–	Raised WCC	None administered
Leach et al 1967	1	49 y/F	Type 1 diabetes mellitus, retinitis proliferans	C1–C2	Cervical pain, stiffness, with limited ROM	*S. aureus*	Cervical collar, oral Abx	Full resolution at 10-mo f/u	Upper respiratory tract infection	Chronic, unclear onset	Open biopsy	ESR 36, WCC 15	3 mo
Rimalovski et al 1968	1	48 y/F	Diabetes mellitus, alcoholic, cervical osteoarthritis	C2	FP: cervical stiffness, TTP, pain with movement; SP: meningitis-like symptoms	*S. aureus*	Penicillin, nitrofurantoin, Staphcillin	Respiratory arrest and death	Positive blood and urine cultures	Acute, days	None	WCC 19.9	3 wk
Ahlback et al 1970	2	(1) 44 y/F; (2) 43 y/M	(1) Diabetes mellitus; (2) –	(1) C1–C2; (2) C1–C2	(1) FP: cervical pain, stiffness; SP: cervical pain, stiffness, limited ROM, Neuro Sx; (2) FP: sudden cervical pain; SP: cervical spine fixed in slight flexion with right rotation, erythematous pharynx	(1) None identified; (2) none identified	(1) FP: I&D of peritonsillar abscess, tonsillectomy; SP: collar, penicillin, streptomycin; (2) FP: no treatment; SP: cloxacillin per os; Crutchfield traction, C1–C2 fusion	(1) Residual cervical stiffness and limited ROM at 7-y f/u; (2) complete recovery with some cervical limitation of ROM	(1) Left otitis media; (2) peritonsillar abscess	(1) 6 wk posttonsillectomy; (2) sudden onset	(1) Epipharynx biopsy; (2) retropharyngeal needle biopsy	(1) ESR 50 WCC 8; (2) ESR 110 WCC 7.9	(1) 12 wk; (2) not mentioned
Vemireddi et al 1978	1	58 y/M	IVDA	C1–C2	Cervical stiffness; weakness in right upper and lower extremity	*S. aureus*	C2 vertebral biopsy, IV nafcillin, halo loop, physical therapy, and dicloxacillin	4-mo f/u: residual cervical stiffness, difficulty turning, no weakness in right upper and lower extremity	None identified	6 d	Biopsy, epidural abscess	WCC 7.8, ESR 74	4 wk IV, 12 wk oral
Venger et al 1986	1	29 y/M	IVDA	C2	Cervical pain, stiffness, limited ROM, TTP, difficulty swallowing, recurrent fevers	*S. aureus*	Hard cervical collar, nafcillin, halo brace	Full recovery at 6-mo f/u	None identified	4 wk	–	WCC 18, ESR 50	6 wk IV
Zigler et al 1987	5	(1) 62 y/F; (2) 66 y/M; (3) 67 y/F; (4) 56 y/F; (5) 72 y/M	(1) Diabetes mellitus, PVD; (2) –; (3) –; (4) chronic renal failure secondary to polycystic disease, congenital aortic	(1) C1–C2; (2) C1–C2; (3) C1–C2; (4) C1–C2; (5) C1–C2	(1) Cervical pain with motion; weakness in lower extremities on ambulation; absent knee jerks; (2) FP: sudden onset cervical pain and fever;	(1) *S. aureus*; (2) *S. aureus*; (3) *S. aureuss*; (4) *Pasteurella multocida*; (5) *S. aureus*	(1) Trans-oral biopsy, IV oxacillin, posterior cervical fusion C1-C3; (2) FP: erythromycin; SP: IV methicillin, halo cast, anterolateral surgical exploration	(1) Full recovery at 4-y f/u; (2) full recovery at 11-y f/u; (3) full recovery at 18-mo f/u; (4) full recovery after arthrodesis, patient died shortly	(1) None identified; (2) post–tooth extraction, positive blood cultures; (3) acute sinusitis; (4) cat scratch left leg, abscess,	(1) Sudden; (2) unknown; (3) acute unknown; (4) 2 wk; (5) unknown	(1) Transoral biopsy; (2) surgical exploration and biopsy; (3) transoral biopsy; (4) –; (5) –	(1) WCC 7.9; (2) WCC 7.5 ESR 108; (3) unknown; (4) WCC 39, ESR 105; (5) unknown	(1) 3 mo total; (2) 7 wk IV, 6 mo oral; (3) 6 wk; (4) 4 wk; (5) unknown

Table 5 (*Continued*)

Authors	No. of patients with UCEA	Age/sex	Relevant comorbidities	Level of infection	Presentation	Organism	Treatment	Outcome	Source of infection	Onset	Aspirate	ESR/CRP/WCC	Antibiotic duration
			stenosis, CHF; (5) –		SP: severe exacerbation with fever; (3) confused, fever, severe occipital and cervical pain radiating to both temporal areas, generalized hyperreflexia; (4) FP: cervical pain and stiffness with movement; SP: upper cervical pain with movement, hyperreflexia, positive Babinski sign; (5) cervical pain on motion; neck held stiffly to the right		with drain placement, posterior cervical arthrodesis, Keflin, oral dicloxacillin; (3) cervical traction, transoral biopsy and debridement of axis and atlas, IV oxacillin, oral oxacillin; (4) FP: IV ampicillin; SP: soft collar, posterior fusion of occiput to axis; (5) IV oxacillin, posterior atlantoaxial arthrodesis, halo jacket	thereafter secondary to CHF and pneumonia; (5) full recovery at 3-mo f/u, at 10-y f/u complained of intermittent discomfort of the neck secondary to spondylosis	septicemia; (5) pneumonia, septicemia, positive blood cultures				
Limbird et al 1988	3	(1) 51 y/M; (2) 62 y/M; (3) 61 y/M	(1) Type 2 diabetes mellitus; (2) BPH; (3) hypertension, renal failure	(1) C1–C2; (2) C1–C2; (3) C1–C2	(1) FP: purulent olecranon bursitis; SP: cervical stiffness, recurrent headaches, malaise, anorexia, fever; TP: severe cervical pain with movement, afebrile, no neurologic deficits; (2) FP: sudden onset severe cervical pain; SP: cervical pain on movement with TTP in the suboccipital region; (3) neck pain and quadriparesis (central cord syndrome with UE worse than LE)	(1) *S. aureus*; (2) *S. aureus*; (3) *Escherichia coli*; GBS	(1) FP: oral antibiotics (resolved); SP: IV oxacillin; oral oxacillin and probenecid (return of cervical stiffness, transferred to different hospital); TP: halo apparatus, IV nafcillin, rifampin; (2) FP: chiropractic manipulation and b/l shoulder injections (failed to resolve cervical pain); IV nafcillin; SP: cervicothoracic orthosis (fall led to posterior displacement); halo vest; surgical debridement from an anterior transpharyngeal approach; posterior atlantoaxial arthrodesis; IV methicillin; (3) halo traction; Abx	(1) Complete resolution at 1-y f/u with mild limitations in flexion and rotation; (2) asymptomatic at 3-y f/u with 50% loss of active cervical rotation; (3) death secondary to two subsequent MIs followed by frank coma	(1) Positive blood cultures; (2) septic shoulder, positive aspirate; (3) none identified	(1) 3 wk; (2) 1 wk; (3) 3 mo	(1) Mastoid culture negative; (2) debridement of tissue; (3) transoral aspirate	(1) ESR 70 WCC 12; (2) WCC 10.7, ESR 102; (3) unknown	(1) 10 d IV, 2 wk oral; (2) 6 wk IV, 2 wk oral; (3) no data
Bartels et al 1990	1	49 y/M	–	C2–C7	Intermittent cervical stiffness	*S. aureus*	Lateral pharyngotomy to drain a large prevertebral abscess; IV Abx	Asymptomatic at f/u	Positive blood cultures	2 wk	Culture on lateral pharyngectomy	WCC 13.6	6 wk IV
Ruskin et al 1992	1	57 y/M	–	C1–C2	Persistent cervical pain, tactile fever, sore throat	*S. aureus*, *Lactobacillus casei*, *Lactobacillus fermentum*	Incision and drainage; IV imipenem	Complete resolution	Upper respiratory tract infection	3 wk	Incision and drainage retropharyngeal abscess	WCC 17.6, ESR 90	3 mo IV

(*Continued*)

Table 5 (*Continued*)

Authors	No. of patients with UCEA	Age/sex	Relevant comorbidities	Level of infection	Presentation	Organism	Treatment	Outcome	Source of infection	Onset	Aspirate	ESR/CRP/WCC	Antibiotic duration
Keogh et al 1992	1	41 y/M	IVDA	C1–C2	Gradually increasing cervical pain radiating to the occiput; generalized malaise, fever, weight loss	*S. aureus*	IV flucloxacillin and fusidic acid; transoral evacuation of extradural pus and excision of eroded odontoid peg; skull traction	Complete resolution at 3-mo f/u	Positive blood cultures	5 wk	Transoral	WCC 17.9	3 mo
Azizi et al 1995	1	65 y/M	Diabetes mellitus, cranial nerve abnormalities, carotid stenosis, headache, PVD, aortofemoral bypass	Clivus–C1	Severe cervical, facial, and shoulder pain; cervical stiffness; indurated cheeks; right ptosis, abducens nerve palsy, left facial weakness	None identified	Halo neck stabilizer; Abx	At f/u complete resolution with residual abducens palsy	Left otitis externa	6 mo symptoms	Transnasopharyngeal biopsy	ESR 132, WCC 6	6 wk
Lam et al 1996	1	58 y/M	–	C1–C2; L1–L3	Diffuse cervical pain and severe lower back pain	*S. aureus*	Laminectomy of L2 and L3; IV Abx; oral Abx	Full resolution at 9-m f/u	None identified	6 wk	Operative	WCC raised	4 wk IV, 8 wk oral
Fukutake et al 1998	1	74 y/M	Cervical spondylosis; BPH	C1–C2	Fever, severe cervical pain, difficulty ambulating, numbness in UE	*Streptococcus pneumoniae*	IV Abx; posterior fixation and autologous bone transplantation	Full resolution at 3 mo	Post-TURP procedure, pneumonia, positive blood, cultures	1 mo	No mention	ESR 127, CRP 31, WCC 21.5	8 wk IV, 4 wk oral
Kurimoto et al 1998	1	72 y/F	Diabetes mellitus	C2	Afebrile, cervical pain and stiffness, right hemiparesis	None identified	Steroids; insulin; IV Abx; transoral surgery; occipitocervical fixation	Right hemiparesis persisted at f/u	None identified	2 wk	Transoral	Normal	No mention
Weidau-Pazos et al 1999	2	(1) 63 y/M; (2) 74 y/F	(1) –; (2) –	(1) C1–C2; (2) C1–C2	(1) Febrile, severe cervical pain with swallowing, difficulty rotating neck; (2) disoriented, encephalopathy, paraparesis, hyperreflexia, positive plantar reflexes b/l	(1) *S. aureus*; (2) none identified	(1) IV Abx; C2 hemilaminectomy with a dorsal approach; epidural abscess removal through transoral surgery 57 d after onset of symptoms; (2) transoral dens resection with placement of halo fixator; IV Abx; posterior fusion	(1) Full resolution at 3-y f/u (patient described fear of rotating more than 70 degrees); (2) full resolution at 3-y f/u	(1) Left hand abscess, positive blood cultures; (2) right gluteal abscess	(1) 1 d; (2) sudden	(1) None; (2) transoral	(1) WCC 13, ESR 38; (2) WCC 10, ESR 85	(1) 4 wk IV; (2) no mention
Anton et al 1999	1	75 y/F	–	C1–C2	Cervical pain, sudden tetraparesis	*Streptococcus. viridians*	Ventral retropharyngeal decompression with second-stage dorsal atlantoaxial spondylodesis	Full resolution at 3-mo f/u	Febrile pharyngitis	8 wk neck pain then sudden tetraparesis	During surgery direct vision	Unknown	No mention
Yuceer et al 2000	1	72 y/M	HIV	C2–C3	Neck pain and 4 limb weakness	*S. aureus*	Decompression and IV Abx	Full resolution by 6 mo	Bilateral pneumonia	20 d	During surgery	WCC 13, ESR 110	8 wk IV
Noguchi et al 2000	1	68 y/M	Type 2 diabetes mellitus, hypertension	C2–C5	Febrile, cervical neck pain and stiffness	*S. pneumoniae*	IV Abx and Philadelphia collar	Full recovery at 2-y f/u	Bacterial meningitis	1 wk	Transoral biopsy	WCC 19.4, ESR 84	3 mo IV

Table 5 (*Continued*)

Authors	No. of patients with UCEA	Age/sex	Relevant comorbidities	Level of infection	Presentation	Organism	Treatment	Outcome	Source of infection	Onset	Aspirate	ESR/CRP/WCC	Antibiotic duration
Suchomel et al 2003	3	(1) 52 y/M; (2) 51 y/F; (3) 50 y/M	(1) None; (2) obese, HTN; (3) type 2 diabetes mellitus, hypertension, previous parotitis/ rhinopharyngitis	(1) C1–C2; (2) C1–C2; (3) C1–C2	(1) Cervical neck pain and stiffness; (2) fever, cervical neck pain/stiffness; (3) fever, neck pain radiating both arms, neck stiffness	(1) *S. aureus;* (2) *S. aureus*; (3) *S. aureus*	(1) Surgical debridement, halo frame, and IV Abx then oral Abx; (2) surgical debridement, halo frame, IV Abx then oral Abx; (3) surgical drainage, halo frame and IV Abx then oral Abx; second-stage stabilization	(1) Full recovery; (2) full recovery 1-y f/u; (3) full recovery 3-mo f/u	(1) ENT cause, infection submandibular duct; (2) laryngitis; (3) previous rhinopharyngitis	(1) 2 mo; (2) 1 wk; (3) sudden onset	(1) Transoral biopsy; (2) CT-guided biopsy; (3) retropharyngeal pus evacuation	(1) ESR 80; (2) WCC/ESR elevated; (3) ESR 90	(1) 3 wk IV, 3 wk oral; (2) 3 wk IV, 3 wk oral; (3) 3 wk IV, 3 wk oral
Hardias et al 2003	1	65 y/M	Chronic renal failure	C1–C2	Febrile, cervical neck pain; progressing neurology	*S. aureus/Proteus mirabilis*	Surgical decompression and halo frame IV Abx	Full resolution focal neurology	Positive blood cultures	2 d	At surgery	Elevated but no figures	2 mo
Paul et al 2005	1	54 y/M	Type 2 diabetes mellitus	Mostly C2 (some C3–C4 involvement)	Neck pain. chronic suppurative otitis media	*Pseudomonas aeruginosa*	Surgical debridement, cervical halo frame, oral Abx	Resolution neck pain 3 mo	Left otitis media	2 wk	Retropharyngeal drainage of abscess	Elevated but no figures	2 wk IV, 4 wk oral
Sasaki et al 2006	1	76 y/F	Type 2 diabetes, liver cirrhosis	C1–C2	Left neck stiffness and pain	None identified	Halo fixation (destructive change atlantoaxial joint) and IV Abx	Full recovery	Positive blood cultures	1 d	None	WCC 10.8, ESR 63	8 wk IV, 4 wk oral
Dimaala et al 2006	1	1 y/M	–	C2	Neck stiffness, malaise, anorexia	None identified	Cervical stabilization, IV Abx	Full recovery	Superficial left thigh abscess	Unknown	None	ESR 94, WCC 6	2 wk IV, 4 wk oral
Curry et al 2007	1	37 y/F	–	C2–C3	Posttonsillectomy	None identified	Debridement, IV Abx	Full recovery	Posttonsillectomy	1 wk	Transcervical drainage	WCC 5.6, ESR 68	8 wk IV
Reid et al 2007	1	58 y/M	Type 2 diabetes mellitus	C1–C2	Cervical neck pain	*S. aureus*	Surgical decompression and halo frame. IV Abx then oral Abx	Full recovery at 6-mo f/u	Positive blood cultures	4 mo	CT-guided	WCC 14.5, ESR 109, CRP 115	3 wk IV, 6 mo oral
Ueda et al 2009	1	37 y/M	Previous conservative treatment mandible 3 months prior	C1	Cervical pain, fever	Alpha-streptococcus	Cervical collar, IV Abx and oral Abx	Full recovery 2-y f/u	Dental extractions and osteomyelitis mandible	2 mo	Transoral biopsy	WCC 20.3, CRP 4.7	3 wk IV, 9 wk oral
Tomaszewski et al 2011	2	(1) 1 wk/M; (2) 1 wk/F	(1) –; (2) –	(1) C2–C3; (2) C2–C4	(1) Restless, jaundiced; (2) jaundice	(1) *S. aureus/Klebsiella pneumoniae*; (2) none identified	(1) Cervical spine immobilization, IV Abx; (2) cervical spine immobilization, IV Abx	(1) Full recovery; (2) full recovery	(1) Positive blood cultures; (2) none identified	(1) 2 wk; (2) 1 wk	(1) Fine needle aspirate; (2) –	(1) ESR 43, CRP 96, WCC 30; (2) ESR 43, CRP 78, WCC 16	(1) 8 wk IV; (2) 6 wk IV
Papp et al 2013	1	4 wk/M	–	C1–C2	Fever, tachycardia, hypotonia	*S. aureus*	Partial hemilaminectomy	Slight restriction neck motion, no neurology	Right mastoid abscess, craniospinal/thoracic abscesses	Acute	Transmastoidal	Unknown	6 wk IV

Abbreviations: Abx, antibiotics; b/l, bilateral; BPH, benign prostatic hypertrophy; CHF, congestive heart failure; CL, cervical lymphadenopathy; CRP, C-reactive protein; CT, computed tomography; ENT, ear, nose, and throat; ESR, erythrocyte sedimentation rate; f/u, follow-up; FP, first presentation; GBS, Guillain-Barré syndrome; HIV, human immunodeficiency virus; HTN, hypertension; I&D, incision and drainage; IV, intravenous; IVDA, intravenous drug abuser; LE, lower extremity; MI, myocardial infarction; Neuro Sx, neurologic symptoms; postop, postoperative; PVD, peripheral vascular disease; ROM, range of motion; SP, second presentation; TP, third presentation; TTP, thrombotic thrombocytopaenia purpura; TURP, transurethral resection of prostate; UCEA, upper cervical epidural abscess; UE, upper extremity; WCC, white blood cell count.

long-term antibiotics. Indications requiring early intervention include acute presentation, evidence of spinal cord compression, and infection-associated spinal instability. Sampath and Rigamonti studied UCEAs and concluded that improved patient outcomes were obtained with rapid identification and aggressive surgical management of patients with SEA. Those patients with poorer outcomes either had several comorbidities or previous spinal surgery or harbored methicillin-resistant species.[42]

Conclusion

UCEA is a rare condition that requires consideration in patients presenting with neck pain and/or stiffness with or without associated fever. A high index of suspicion is required to identify this condition, and MRI remains the imaging modality of choice. Obtaining cultures prior to administration of antibiotics is preferable. The treatment remains controversial with a trend toward surgical decompression and stabilization in modern times, which is supported by favorable patient outcomes.

Disclosures
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References

1 Grewal S, Hocking G, Wildsmith JA. Epidural abscesses. Br J Anaesth 2006;96(3):292–302
2 Gellin BG, Weingarten K, Gamache FW Jr, et al. Epidural abscess. In: Infections of the Central Nervous System. 2nd ed. Philadelphia: Lippincott-Raven; 1997:507
3 Bogduk N, Twomey L. Clinical Anatomy of the Lumbar Spine. 2nd ed. New York, NY: Churchill Livingstone; 1991
4 Malanga GA. The diagnosis and treatment of cervical radiculopathy. Med Sci Sports Exerc 1997;29(7, Suppl):S236–S245
5 Tong HC, Haig AJ, Yamakawa K. The Spurling test and cervical radiculopathy. Spine (Phila Pa 1976) 2002;27(2):156–159
6 Frykholm R. Cervical nerve root compression resulting from disc degeneration and root-sleeve fibrosis. Acta Chir Scand 1951; 160:1–149
7 Tubbs RS, Salter EG, Oakes WJ. The accessory atlantoaxial ligament. Neurosurgery 2004;55(2):400–402, discussion 402–404
8 Richardson J, Groen GJ. Applied epidural anatomy. Contin Educ Anaesth Crit Care Pain 2005;5(3):98–100
9 Sendi P, Bregenzer T, Zimmerli W. Spinal epidural abscess in clinical practice. QJM 2008;101(1):1–12
10 Darouiche RO. Spinal epidural abscess. N Engl J Med 2006;355(19): 2012–2020
11 Nussbaum ES, Rigamonti D, Standiford H, Numaguchi Y, Wolf AL, Robinson WL. Spinal epidural abscess: a report of 40 cases and review. Surg Neurol 1992;38(3):225–231
12 Davis DP, Wold RM, Patel RJ, et al. The clinical presentation and impact of diagnostic delays on emergency department patients with spinal epidural abscess. J Emerg Med 2004; 26(3):285–291
13 Tang H-J, Lin H-J, Liu Y-C, Li C-M. Spinal epidural abscess—experience with 46 patients and evaluation of prognostic factors. J Infect 2002;45(2):76–81
14 Soehle M, Wallenfang T. Spinal epidural abscesses: clinical manifestations, prognostic factors, and outcomes. Neurosurgery 2002; 51(1):79–85, discussion 86–87
15 Hlavin ML, Kaminski HJ, Ross JS, Ganz E. Spinal epidural abscess: a ten-year perspective. Neurosurgery 1990;27(2):177–184
16 Reihsaus E, Waldbaur H, Seeling W. Spinal epidural abscess: a meta-analysis of 915 patients. Neurosurg Rev 2000;23(4): 175–204, discussion 205
17 Huang RC, Shapiro GS, Lim M, Sandhu HS, Lutz GE, Herzog RJ. Cervical epidural abscess after epidural steroid injection. Spine (Phila Pa 1976) 2004;29(1):E7–E9
18 Alcock E, Regaard A, Browne J. Facet joint injection: a rare form cause of epidural abscess formation. Pain 2003;103(1–2):209–210
19 Lin YC, Greco C. Epidural abscess following epidural analgesia in pediatric patients. Paediatr Anaesth 2005;15(9):767–770
20 Philipneri M, Al-Aly Z, Amin K, Gellens ME, Bastani B. Routine replacement of tunneled, cuffed, hemodialysis catheters eliminates paraspinal/vertebral infections in patients with catheter-associated bacteremia. Am J Nephrol 2003;23(4):202–207
21 Bang MS, Lim SH. Paraplegia caused by spinal infection after acupuncture. Spinal Cord 2006;44(4):258–259
22 Chowfin A, Potti A, Paul A, Carson P. Spinal epidural abscess after tattooing. Clin Infect Dis 1999;29(1):225–226
23 Sillevis Smitt P, Tsafka A, van den Bent M, et al. Spinal epidural abscess complicating chronic epidural analgesia in 11 cancer patients: clinical findings and magnetic resonance imaging. J Neurol 1999;246(9):815–820
24 Lechiche C, Le Moing V, Marchandin H, Chanques G, Atoui N, Reynes J. Spondylodiscitis due to *Bacteroides fragilis*: two cases and review. Scand J Infect Dis 2006;38(3):229–231
25 Rigamonti D, Liem L, Sampath P, et al. Spinal epidural abscess: contemporary trends in etiology, evaluation, and management. Surg Neurol 1999;52(2):189–196, discussion 197
26 Khan SH, Hussain MS, Griebel RW, Hattingh S. Title comparison of primary and secondary spinal epidural abscesses: a retrospective analysis of 29 cases. Surg Neurol 2003;59(1):28–33, discussion 33
27 Del Curling O Jr, Gower DJ, McWhorter JM. Changing concepts in spinal epidural abscess: a report of 29 cases. Neurosurgery 1990; 27(2):185–192
28 Kaufman DM, Kaplan JG, Litman N. Infectious agents in spinal epidural abscesses. Neurology 1980;30(8):844–850
29 Lury K, Smith JK, Castillo M. Imaging of spinal infections. Semin Roentgenol 2006;41(4):363–379
30 An HS, Seldomridge JA. Spinal infections: diagnostic tests and imaging studies. Clin Orthop Relat Res 2006;444(444):27–33
31 Cahill DW. Pyogenic infections in the spine. In: Menezes AH, Sonntage VKH, eds. Principles of Spinal Surgery II. New York: McGraw-Hill; 1996:1453–1465
32 Curry WT Jr, Hoh BL, Amin-Hanjani S, Eskandar EN. Spinal epidural abscess: clinical presentation, management, and outcome. Surg Neurol 2005;63(4):364–371, discussion 371
33 Lyu RK, Chen CJ, Tang LM, Chen ST. Spinal epidural abscess successfully treated with percutaneous, computed tomography-guided, needle aspiration and parenteral antibiotic therapy: case report and review of the literature. Neurosurgery 2002;51(2): 509–512, discussion 512
34 Azizi SA, Fayad PB, Fulbright R, Giroux ML, Waxman SG. Clivus and cervical spinal osteomyelitis with epidural abscess presenting with multiple cranial neuropathies. Clin Neurol Neurosurg 1995;97(3):239–244
35 Cwikiel W. Percutaneous drainage of abscess in psoas compartment and epidural space. Case report and review of the literature. Acta Radiol 1991;32(2):159–161
36 Walter RS, King JC Jr, Manley J, Rigamonti D. Spinal epidural abscess in infancy: successful percutaneous drainage in a nine-month-old and review of the literature. Pediatr Infect Dis J 1991; 10(11):860–864

37 Tabo E, Ohkuma Y, Kimura S, Nagaro T, Arai T. Successful percutaneous drainage of epidural abscess with epidural needle and catheter. Anesthesiology 1994;80(6):1393–1395
38 Siddiq F, Chowfin A, Tight R, Sahmoun AE, Smego RA Jr. Medical vs surgical management of spinal epidural abscess. Arch Intern Med 2004;164(22):2409–2412
39 Bamberger DM. Outcome of medical treatment of bacterial abscesses without therapeutic drainage: review of cases reported in the literature. Clin Infect Dis 1996;23(3): 592–603
40 Kim SD, Melikian R, Ju KL, et al. Independent predictors of failure of nonoperative management of spinal epidural abscesses. Spine J 2014;14(8):1673–1679
41 Patel AR, Alton TB, Bransford RJ, Lee MJ, Bellabarba CB, Chapman JR. Spinal epidural abscesses: risk factors, medical versus surgical management, a retrospective review of 128 cases. Spine J 2014; 14(2):326–330
42 Sampath P, Rigamonti D. Spinal epidural abscess: a review of epidemiology, diagnosis, and treatment. J Spinal Disord 1999; 12(2):89–93