Upper Cervical Epidural Abscess in Clinical Practice: Diagnosis and Management

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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.
Spinal epidural abscess in general has an incidence of ~2 to 25 patients per 100,000 admitted to the hospital. 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. 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). 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 (atlan-to-occipital joint) and the axis (atlan-toaxial joint). The atlanto-occipital and 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. 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.

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. 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. The true epidural spaces occur at the cervical, midthoracic, and lumbar-sacral 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. 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. In our analysis of the literature, many of the predisposing factors remained the same; the most common factor by far is diabetes mellitus. Intravenous drug use and chronic kidney disease also represented a sizeable portion of our cases.

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), hematomagenous invasion, or direct contamination from an adjacent infected structure. Subsets of patients seem predisposed to spontaneous epidural abscess in which there is generally no identified source of infection. We found hematomagenous spread and ear, nose, and throat pathology to be the most likely source of infection with some cases having both as a potential cause. 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. 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 was identified.

**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  |

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

Note: some cases have more than one source.
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.

### Diagnosis

The classical triad of spinal epidural abscess is pain, fever, and neurologic deficit. 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.

### 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%. 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. Computed tomography (CT) is invaluable in the evaluation of vertebral end plate and facet erosions associated with 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.

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. 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. 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.

### 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) |

### 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.
**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. If another potential source of UCEA is identi-
ﬁed such as throat, supportive otitis, or respiratory tract infec-
tion, 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 man-
agement 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 sur-
gery, 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 com-
bined 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 nonopera-
tive 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 immobi-
lization 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 antibi-
otic treatment. None of the patients who were treated non-
operatively 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 immobi-
lization 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 tetra-
paresis, 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, particu-
larly reserved for those with a posterior spinal epidural
abscess (SEA) and no neurologic deﬁcit or those unable to
withstand surgery.35,36–37 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 manage-
ment 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; how-
ever, a limitation to the study was the criteria for success.39

Recent studies have suggested that independent risk fac-
tors 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 nonopera-
tively.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, decreas-
ing 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 in-
nervation 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 com-
pared with patients who underwent surgical therapy after
failure of nonoperative treatment.41 The mainstay of sur-
gical 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 |
|--------------------------|---------------------------|------------------|-------------------------------------------------|--------------------|--------------|----------|-----------|---------|-------------------|-------|-----------|-------------|---------------------|
| Odell & Johnson et al. 1931 | 1                         | 46 y/F           | Mumps, whooping cough, rubella                  | C2                 | Fever, cervical pain, stiffness, CL | None identified | Master of Parks head and neck placement; head in hyperextension and traction with the body as a counterweight | 1.5 y/f with resolution of neck pain, no limitations with flexion and extension, severe disability with rotation to the right | Postmortemectomy | 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; erythematosus tissue; scattered rashes in lungs | Staphylococcus aureus | BO-percutaneous needle aspiration (multiple staphylococcal abscesses) | Death from meningitis secondary to osteomyelitis of the odontoid process around 15 wk from initial presentation | Orbital right external approach following spine fracture, urinary tract infection | CPD/IV | Raised WCC | None administered |
| Leach et al. 1967        | 1                         | 49 y/F           | Diabetes mellitus, retroviral proliferations     | C1–C2              | Cervical pain, stiffness, with limited ROM | S. aureus | Cervical collar, oral antibiotics | Full resolution at 10-mo f/u | Upper respiratory tract infection | Chronic, unclear onset | Open biopsy | ESR 36, WCC 15 | 3 mo                  |
| Rimabroski et al. 1968   | 1                         | 48 y/F           | Diabetes mellitus, alcoholic, cervical osteomyelitis | C2                 | PP: cervical stiffness, TTP pain with movement; SP: meningitis-like symptoms | S. aureus | Penicillin, streptomycin, Staphylococcus aureus | Respiratory arrest and death | Posthemorrhagic uremic crisis | Acute, days | None | WCC 119 | 3 wk                  |
| Ahbakk et al. 1970       | 2                         | (1) 44 y/F; (2) 43 y/M | (1) Diabetes mellitus, (2) –                     | (1) C1–C2; (2) C1–C2 | (1) PP: cervical pain, stiffness; SP: cervical pain, stiffness, limited ROM, fever; (2) PP: sudden cervical pain; SP: possible spine fixation in slight flexion with right rotation, erythematous pharynx | S. aureus | (1) None identified; (2) none identified | (1) PP: I&D of peritonsillar abscesses, tonsillotomy; SP: collar, penicillin-streptomycin; (2) PP: I&D; SP: nafcillin; 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 orbit media; (2) pittosporal abscess | (1) 6 wk postcraniectomy; (2) sudden onset | (1) ESR 50 WCC 8; (2) ESR 110 WCC 7.0 | (1) 12 wk, (2) not mentioned |
| Vemireddi et al. 1978    | 1                         | 58 y/M           | MDA                                             | C1–C2              | Cervical stiffness, weakness in right upper and lower extremity | S. aureus | C2 ventral biopsy, nafcillin, halo loop, physical therapy, and dixodcillin | 4-mo f/u: residual cervical stiffness, difficulty turning, no weakness in right upper and lower extremity | None identified | 6 d | Biopsy, epidural abscesses | WCC 7.8, ESR 74 | 4 wk IV, 12 wk oral |
| Venger et al. 1986       | 1                         | 29 y/M           | MDA                                             | C2                 | Cervical pain, stiffness, limited ROM, TTP, difficulty swallowing, recurrent fever | 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 stenosis | (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) PP: sudden onset cervical pain and fever; (3) 133. aureus; (4) S. aureus; (5) 133. aureus; (6) Prosthecitella multifascialis; (7) S. aureus | (1) 133. aureus; (2) S. aureus; (3) S. aureus; (4) Prosthecitella multifascialis; (5) S. aureus | (1) Trans-oral biopsy; IV nafcillin, posterior cervical fusion C1-C2; (2) PP: erythromycin; SP: IV merthiolate, halo brace, anterior lateral surgical exploration | (1) Full recovery at 4-mo f/u; (2) full recovery at 1.5 y f/u; (3) full recovery at 18-mo f/u; (4) full recovery after arthrodesis, patient died shortly | (1) None identified; (2) post-arthrodesis extraction, posterior blood cultures; (3) acute sinusitis; (4) cat scratch left leg, abscesses | (1) Sudden; (2) unknown; (3) acute unknown; (4) 2 wk; (5) unknown | (1) ESR 7.8; (2) WCC 7.5 ESR 108; (3) unknown; (4) WCC 19; ESR 105; (5) unknown | (1) 13.3 mo total; (2) 7 wk IV, 6 mo oral; (3) 16 wk; (4) 4 wk; (5) unknown |

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| 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 |
|--------------------|---------------------------|---------|------------------------|--------------------|--------------|----------|-----------|---------|---------------------|-------|----------|-------------|---------------------|
| Limbird et al 1988 | 3                         | 51 y/M  | (1) Type 2 diabetes mellitus; (2) BPH; (3) Hypertension, renal failure | C1–C2             | C1–C2       | S. aureus | (1) IV nafcillin; (2) Oral antibiotics (resolved); (3) IV oxacillin; (4) IV imipenem; (5) Oral nafcillin, Oral oxacillin | (1) Complete resolution at 3-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 | Positive blood cultures; (2) Staphylococcus aureus; (3) ESR/CRP/WCC: (1) 70; (2) 19.7; (3) 102; (4) Unknown; (5) 10 d IV, 2 wk oral | (2) 6 wk IV | (1) S. aureus; (2) Streptococcus pyogenes; (3) Pseudomonas aeruginosa; (4) Enterobacter cloacae; (5) Unknown | (Continued) |
| Bartels et al 1990 | 1                         | 49 y/M  | –                      | C2–C7             | Intermittent cervical stiffness | S. aureus | Lateral pharyngectomy to drain a large retropharyngeal abscess, IVAbx | 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, tachycardia, sore throat | S. aureus | 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 |

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, cervical spondylitis, diabetes mellitus, pneumonia, hypertension, polymyalgia    | 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               |
| Asl et al 1995   | 1                         | 65 y/M  | Diabetes mellitus, cranial nerve abnormalities, cardiac abnormalities, headache, IV, aortofemoral bypass | Clivo-C1           | Severo cervical, facial, and shoulder pain; cervical stiffness; indurated cheeks, right ptosis, abducens nerve palsy | None identified                   | Halo neck stabilizer; Abx | At f/u complete resolution with residual abducens palsy | Left otitis externa 6 mo symptoms | Transophrangyal biopsy | 6 mo   | Normal   | 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 3 mo f/u | None identified | 6 wk   | Operative | WCC, raised     | 4 wk IV, 8 wk oral |
| Fukutake et al 1998 | 1                        | 74 y/M  | Cervical spondylitis, BPH                                                             | C1–C2              | Fever, severe cervical pain, difficulty ambulating, numbness in UE            | Streptococcus pneumonia          | IV Abx; posterior fixation and autologous bone transplantation | Full resolution at 3 mo | Post-TURP procedure, pneumoectomy, positive blood cultures | 1 mo   | No mention | ESR 127, QRP 35 | 8 wk IV, 4 wk oral |
| Kariyuma et al 1998 | 1                        | 72 y/F  | Diabetes mellitus                                                                     | C2                 | Alkeine, cervical pain and stiffness, right hemiparesis                       | None identified                   | Stents; insulin; IV Abx; transoral surgery; occipitoaxial fusion | 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) –                                                                            | C1–C2; C1–C2      | (1) febrile, severe cervical pain with swallowing, difficulty rotating neck; (2) disorientated, encephalopathy, paranoia, hyperreflexia, positive plantar reflexes | (1) S. aureus, (2) none identified | (1) IV Abx, C2 hemi-laminectomy with a dorsal approach; epidural abscess removal through transoral surgery 57 d after onset of symptoms; (2) transoral hemilaminectomy with placement of halo fixator, IV Abx, posterior fusion | (1) Full resolution at 3 d f/u (patient described fear of rotting 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 95 | (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 spondyloplasty | Full resolution at 3 mo f/u | Febrile pharyngitis | 8 wk neck pain then sudden tetraparesis | During surgery direct vision | Unknown          | No mention          |
| Youssef 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 | Bacterial pneumonia | 30 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. pneumonia                      | 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               |
| Authors                  | No. of patients with UCEA | Age/sex          | Relevant comorbidities                  | Level of infection | Presentation                | Organism                        | Treatment                                                                 | Outcome          | Source of infection | Onset Duration | Aspiration | 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) diabetes, HTN, (3) type 2 diabetes mellitus, hypertension, previous parotitis/rhinoopharyngitis | (1) C3–C2, (2) C5, (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. pneumoniae, (3) S. pyogenes | (1) Surgical decompression and halo frame IV Abx; (2) surgical debridement, halo frame IV Abx; then oral Abx; (3) surgical drainage, halo frame and IV Abx; then oral Abx | (1) Full recovery, (2) full recovery 1 wk/f/u | (1) ENT cause, infection submandibular duct; (2) lymphangitis; (3) previous rhinopharyngitis | (1) 2 mo; (2) 1 wk | (3) sudden onset | (1) Transoral biopsy; (2) C1-guided biopsy; (3) retropharyngeal pus evacuation | (1) ESR 80; (2) WCC/CRP; (3) ESR 90 | (1) 3 wk IV, (2) 3 wk oral, (3) 1 wk IV, (4) 3 wk oral |
| Hanias et al 2003        | 1                         | 65 y/M           | Chronic renal failure                  | C1–C2             | Febrile, cervical neck pain; progressing myelopathy | S. aureus/Proteus mirabilis | Surgical decompression and halo frame IV Abx | Full resolution focal myelopathy | 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 supplicative disease | 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 |
| Suchomel et al 2003      | 1                         | 56 y/F           | Type 2 diabetes, liver cirrhosis      | C1–C2             | Left neck stiffness and pain | None identified | Halo-fracture (destructive change at lumbosacral joint) and IV Abx | Full recovery | Positive blood cultures | 1 d | None | WCC 10.8, ESR 63 | 8 wk IV, 4 wk oral |
| Dimofet al 2006          | 1                         | 1 y/M            | –                                      | C2                | Neck stiffness, malaise, anemia | 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             | Posttranssphenoidal | None identified | Debridement, IV Abx | Full recovery | Ponto-ponsectomy | 1 wk | Transcervical drainage | 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 | C1-guided | WCC 14.5, ESR 90; 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 g/f/u | Dental extractions and intraoral mandible | 2 mo | Transoral biopsy | WCC 20.3, CRP 47 | 3 wk IV, 9 wk oral |
| Tomaszewski et al 2011   | 2                         | (1) 1 wk/M, (2) 1 wk/F | –                                      | (1) C2–C3, (2) C2–C4 | (1) Redness, jaundice; (2) jaundice | (1) S. aureus/Websella pneumonia, (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 aspiration, (2) – | (1) ESR 43, WCC 96; (2) ESR 43, CRP 28, WCC 16 | (1) 3 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 myelopathy | Right mastoid abscess, carotid/thoracic abscesses | Acute | Transmastoideal | 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; CRS, Guillain-Barré syndrome; HIV, human immunodeficiency virus; HTN, hypertension; I&D, incision and drainage; IV, intravenous; IVDA, intravenous drug abuse; UE, upper extremity; MI, myocardial infarction; Neuro Sx, neurologic symptoms; postop, postoperative; PVD, peripheral vascular disease; ROM, range of motion; SP, second presentation; TP, third presentation; TTF, thoracic thoracostomy/pia 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

Khalid Al-Hourani, none
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