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CASE REPORT

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A case report of quadriplegia and acute stroke from tracking retropharyngeal and epidural abscess complicated by necrotizing fasciitis

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Abstract

A 59-year-old male presented to the emergency department complaining of severe posterior neck pain and progressive extremity weakness for 2 weeks. He was found to be quadriplegic with complete sensory and motor deficits at the C5 level and hypotensive. Diagnostic imaging revealed discitis/osteomyelitis at the C5-C6 and C6-C7 vertebral levels with multiple spinal epidural abscesses extending from C5-C7 with resulting severe spinal canal narrowing with cord compression. Imaging also showed a right vertebral artery occlusion, acute right posterior cerebral artery infarct, retropharyngeal abscess, and extensive paraspinal soft tissue myonecrosis.

Vasopressors and broad-spectrum antibiotics were started. He was then transferred to a tertiary medical center where he underwent emergent cervical spine decompression surgery with laminectomy from C3-C7, paraspinal soft tissue debridement, and abscess incision and drainage. He suffered a complicated hospital course and despite aggressive treatment developed worsening infectious myelopathy and died in the hospital. This case involves the rare presentation of quadriplegia and acute cerebral infarction associated with necrotizing fasciitis and spinal epidural abscesses that originated from a retropharyngeal abscess. To date, there have been no cases documenting such a phenomenon, and epidural abscess has not been known to cause adjacent necrotizing fasciitis. Furthermore, vertebral artery thrombosis via mass effect from local infection leading to acute embolic stroke has never been reported. This report sheds light on rare sequela of a tracking retropharyngeal and epidural abscess. Prompt recognition, diagnosis, and treatment are vital to maintain infectious source control and preserve neurological function, although many develop persistent deficits.

KEYWORDS

discitis, epidural abscess, necrotizing fasciitis, osteomyelitis, quadriplegia, retropharyngeal abscess, spinal cord compression, stroke

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1 | INTRODUCTION

Retropharyngeal abscess can lead to many complications, including jugular venous thrombosis, sepsis, esophageal erosion, mediastinitis, pneumonia, and epidural abscess.[1–3] Spinal epidural abscesses generally occur from hematogenous spread and trauma and from adjacent infections such as vertebral osteomyelitis/discitis.[1,4] They can lead to mild to severe neurological deficits and require prompt medical and surgical intervention.[1,4] Two cases of necrotizing fasciitis have been documented causing spinal epidural abscess; however, there are no known cases that have demonstrated a tracking retropharyngeal space infection with spinal epidural abscess formation resulting in paraspinal necrotizing fasciitis. Furthermore, there are no reported cases of vertebral artery thrombosis via suspected mechanism of mass effect from local infection leading to acute embolic cerebral vascular accident.

This case presentation illustrates a unique mechanism that has not yet been documented in the literature. This study will discuss the rare and sometimes disastrous consequences of a posteriorly tracking retropharyngeal abscess leading to spinal epidural abscess formation, necrotizing fasciitis, and acute cerebral vascular accident. Future clinicians must recognize the potential sequela of these infections that require prompt recognition and immediate intervention for potentially life-saving treatment.

2 | CASE PRESENTATION

A 59-year-old man with a history of type II diabetes mellitus, coronary artery disease, tobacco use, cervical stenosis, atrial fibrillation, and medical non-compliance presented to a rural emergency department (ED) with complaints of severe posterior neck pain along with generalized weakness. His symptoms occurred during the past 2 weeks but had progressed 48 hours before presentation. He reported severe weakness in all extremities and the inability to walk. He denied any chest pain, shortness of breath, abdominal pain, nausea, vomiting, diarrhea, sore throat, cough, fevers, chills, or recent illness. He also denied any recent head or neck trauma or falls. There was no history of prior intravenous drug use. He was not taking any of his prescribed medications for the past several months.

In the ED, blood pressure was 84/46 mmHg, heart rate was 115 beats per minute, temperature was 100.1°F, respiratory rate was 28 breaths per minute and SpO2 of 94% on ambient air. His Glasgow Coma Scale was 14 (E4V4M6). Dentition was poor but with no discernable dental or peritonsillar abscess, and the posterior pharynx was clear. Pupils were round and reactive to light bilaterally, and extraocular movements were intact. Nuchal rigidity was present along with tenderness to light touch over the cervical paraspinal area. There was no overlying erythema, drainage, or necrotic-appearing soft tissue changes in the cervical paraspinal region but the area felt "boggy" and warm to the touch. Lungs were clear. Heart sounds were of regular rhythm with tachycardic rate. No murmurs were detected. The abdomen was soft and non-tender. No sensation was detected below the C5 level.

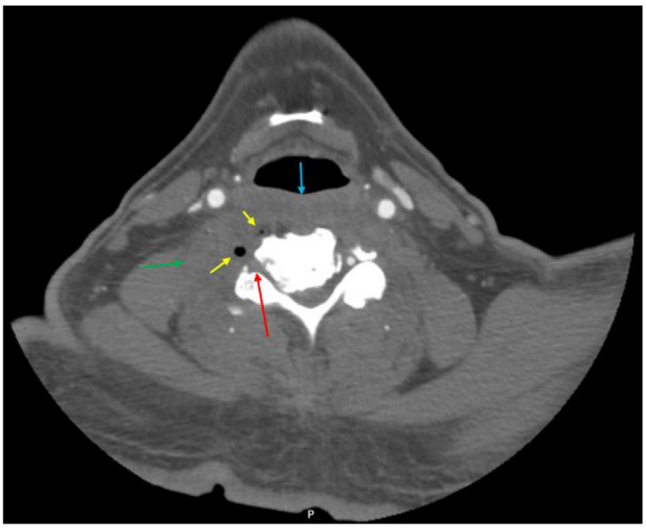

FIGURE 1 Computed tomography angiogram transverse image at the C5 vertebral level. The green arrow indicates right paraspinous edema and myositis, the yellow arrows indicate intramuscular gas in right subarticular recess of C5, the red arrow indicates right vertebral artery thrombosis, and the blue arrow indicates prevertebral soft tissue edema

Strength in all muscle groups in the right upper extremity and bilateral lower extremities was 1 out of 5 intensity. Strength in all muscle groups in the left upper extremity was 2 out of 5 intensity. The biceps, brachioradialis, triceps, patellar, and achilles deep tendon reflexes had a 1 out of 4 response. Distal pulses were intact.

Laboratory results were remarkable for WBC 14.8 k/mcl (4–10 k/mcl) with neutrophilic predominance and 0.7% bands, blood glucose 489 mg/dL (74–106 mg/dL), creatinine 2.4 mg/dL (0.5–1.3 mg/dL) with known baseline creatinine of 1.0 mg/dL, and lactic acid 1.4 mmol/L (0.5–2.0 mmol/L). All other laboratory results, including urinalysis were unremarkable. Chest X-ray had no acute findings. Hemoglobin A1c% was 7.5% (< 5.7%) from 2 weeks prior.

Non-contrast brain computed tomography (CT) scan showed a wedge-shaped hypodensity in the right temporo-occipital region consistent with an acute right posterior cerebral artery ischemic infarct. Subsequent CT angiogram (CTA) of the head and neck were done to evaluate for large vessel occlusion, which revealed vertebral osteomyelitis/discitis at the C5-C6 and C6-C7 vertebral levels with multiple spinal epidural abscesses extending from C5-C7 with resulting severe spinal canal narrowing, cord compression, and cord edema. CTA also showed an acute right long segment vertebral artery occlusion (Figure 1) involving the V1 and V2 segments, acute right posterior cerebral artery infarct, retropharyngeal abscess and extensive bilateral paraspinal soft tissue abscesses, myositis, and subcutaneous/intramuscular gas, consistent with necrotizing fasciitis.

The patient was fluid resuscitated, pan-cultured, given intravenous insulin, and started on intravenous vancomycin, meropenem, and clindamycin. Norepinephrine was required to keep mean arterial pressure at 65 mmHg or greater via a central venous line. The patient was

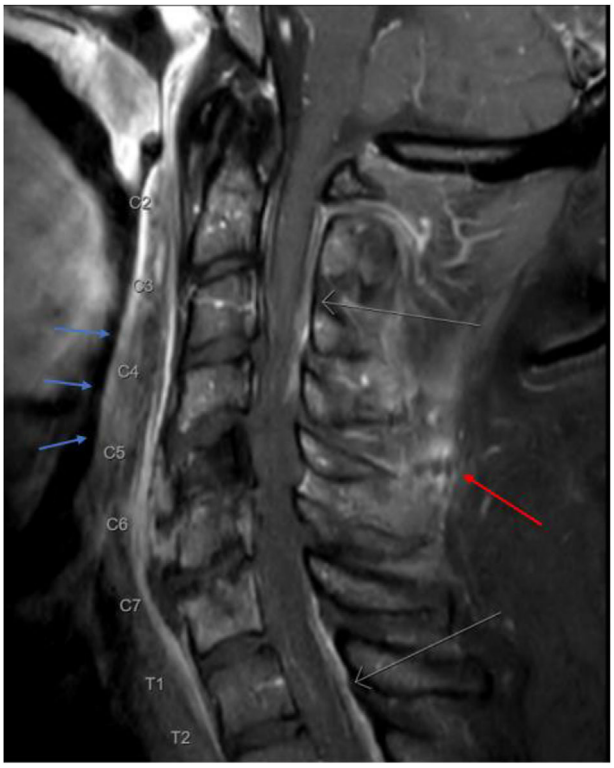

FIGURE 2 T1-weighted magnetic resonance imaging sagittal image of the cervical spine. The blue arrows indicate retropharyngeal abscess, the gray arrows indicate epidural abscesses, and the red arrow indicates area of myonecrosis

intubated for airway protection and concern for possible decompensation enroute before air transfer to a tertiary care facility.

At the accepting facility, contrasted magnetic resonance imaging of the brain, cervical spine (Figure 2), and thoracic spine confirmed the CTA findings and also noted the presence of multifocal epidural phlegmon at the T2-T3 and T7-T8 vertebral levels. The patient went promptly to the operating room with a multidisciplinary surgical team who performed emergent decompressive laminectomies from C3-C7 along with paraspinal soft tissue debridement and abscess incision and drainage. Blood cultures grew *Streptococcus intermedius*. Deep tissue wound culture from paraspinal debridement grew *Streptococcus anginosus*. The patient was not a candidate for a hyperbaric chamber and otolaryngology recommended against further surgical intervention for the retropharyngeal abscess. He also underwent full dental extractions for further infectious source control.

Lower extremity doppler ultrasounds were negative for deep venous thrombosis, and transthoracic echocardiography with agitated saline bubble study showed ejection fraction of 65% to 70% with no anatomical cardiac shunt present.

Despite aggressive treatment the patient developed worsening infectious myelopathy and holocord edema with cord infarct seen on repeat imaging and had persistent quadriplegia. After 15 total hospital days he requested comfort measures and died shortly after removal of life-sustaining treatments.

3 | DISCUSSION

This case elicits the rare complex presentation of quadriplegia and acute cerebral infarction associated with necrotizing fasciitis and spinal epidural abscess that originated from a retropharyngeal abscess. As evidenced by this case, the consequences of an unchecked deep space neck infection are potentially disastrous, especially in those with comorbidities. Our case highlights both known complications of retropharyngeal abscesses as well as several phenomena not previously described in the literature.

Retropharyngeal abscesses are considered a deep space neck infection and occur most commonly following penetrating trauma, such as via a chicken bone or recent instrumentation.[5] They can also develop from local spread from an infection within the oral cavity such as a peritonsillar abscess and occur when the infection travels to the retropharyngeal lymph nodes.[6,7] Typical symptoms include sore throat, odynophagia, fever, and difficulty breathing.[6,7] Most retropharyngeal abscesses are polymicrobial in nature, but the most common isolated organisms are Group A *Streptococcus pyogenes*, *Staphylococcus aureus*, *Fusobacterium*, *Haemophilus* species, and other anaerobic respiratory flora.[3] Spinal epidural abscess originating from retropharyngeal abscess is a documented phenomenon but occurs rarely.[8]

Spinal epidural abscess occurs via several different mechanisms, including hematogenous spread (50%), direct extension from the adjacent infection (33%), inoculation from spinal procedures (15%), and other mechanisms.[1] Classic symptoms of a spinal epidural abscess are back or neck pain, fever, and neurological deficits.[9] They tend to occur more commonly in those with diabetes, abnormalities of the vertebral column, recent trauma to the spine, intravenous drug users, and other immunocompromised states.[9] There have been 2 reported cases of necrotizing fasciitis that have led to the development of a spinal epidural abscess.[10,11] However, there are no documented cases of a tracking retropharyngeal abscess/spinal epidural abscess leading to adjacent necrotizing fasciitis, as in this case. A review article from 2015 reported that spinal epidural abscess carries a 22% risk of permanent paralysis, and mortality rates range from 1.8% to 25% when surgery is delayed.[12] Early surgical intervention within 24 hours substantially reduced morbidity and mortality in this review.[12] Mortality rates may be substantially increased in the setting of concomitant necrotizing fasciitis. Necrotizing fasciitis mortality rates range from 24% to 34%.[13] Both spinal epidural abscess and necrotizing fasciitis require swift recognition and treatment with antibiotics and early surgical intervention, which are paramount to infectious source control and overall survival.[14]

Although rare, there have been several documented cases of V1 and V2 segment vertebral artery occlusions leading to posterior cerebral infarcts via emboli formation,[15] which is the likely mechanism of cerebral vascular accident in this case. There are, however, no documented cases of mass effect from local infection triggering this mechanism. In this case, mass effect attributed to the surrounding tissue infection and adjacent vessel inflammation likely led to the V1/V2 long segment vertebral artery thrombus, followed by embolus and resulting posterior cerebral artery infarct. The patient did have a history of atrial

fibrillation and was not compliant with his anticoagulant; however, given the proximity of the vertebral artery thrombus to the ipsilateral posterior circulation, in the presence of a compressive local infection, this seems a less likely cause. In addition, there was no anatomical cardiac shunt seen on transthoracic echocardiography with bubble study, ruling out paradoxical embolism.

4 | CONCLUSION

This case illustrates the sometimes disastrous complications of a tracking retropharyngeal abscess leading to quadriplegia from compressive spinal epidural abscess, cervical paraspinal necrotizing fasciitis, and acute cerebral vascular accident. Physicians should recognize that, in addition to previously described sequela, deep space neck infections can rarely lead to development of paraspinal necrotizing fasciitis via spinal epidural abscess. In addition, this case illustrates the previously undocumented report of an acute cerebral vascular accident likely stemming from local mass effect and inflammation on the adjacent vertebral artery prompting thrombus and subsequent embolism leading to a posterior circulation infarct. These patients require an aggressive multidisciplinary team approach and likely benefit from care provided at a tertiary academic medical center. Prompt diagnosis, treatment, and infectious source control are vital to patient survival.

CONFLICTS OF INTEREST

The authors declare no conflicts of interest.

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