Acute Hydrocephalus secondary to Holocord Epidural Abscess in an IV drug abuser Presenting with abdominal pain: A Case Report and Literature Review

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Abstract

We present a case of acute hydrocephalus secondary to a cervical, pyogenic spinal epidural abscess in a patient with a history of polysubstance abuse. A 53-year-old female with history of intravenous (IV) substance abuse presented to the hospital with abdominal pain, fever, and chills. On hospital day 2 her mental status deteriorated rapidly and she became acutely quadriplegic. An MRI of the spine revealed a new cervical epidural abscess with cervical cord compression and developing hydrocephalus. External ventriculostomy drainage was immediately performed at bedside with some improvement in her mental status. Hydrocephalus due to spinal cord pathology is a rare entity which has been reported in the literature in cases of trauma and malignancy but not related to an epidural abscess. Acute hydrocephalus although uncommon, should be considered in cases of acute neurological deterioration with known spinal cord pathology.

Keywords

Spinal subdural abscess; Cervical myelopathy induced hydrocephalus; Autonomic dysfunction.

Case report

A 53-year-old female visited the emergency department (ED) with abdominal pain, fevers and chills. Her blood cultures were positive for Staphylococcus aureus. She was started on broad-spectrum antibiotics. A urine Drug screen was tested on admission and was positive for cocaine and opiates. The initial MRI of her cervical and thoracic spine revealed no epidural abscess or paraspinal abscess. She had an elevated white count and elevated lactate levels that went up from 3.4 to 5.3 in 24 hours. She developed worsening mental status on the floor and was transferred to the ICU. She was given narcan 0.4mg and intubated. Her mental status continued to deteriorate and she became hemodynamically unstable. Her head CT revealed no intracranial pathology, but significant edema within the posterior musculature of the neck thought to be due to anasarca. She was obtunded with pin point pupils with sluggish reaction to light and her motor exam was 0/5 in bilateral arms and 1/5 in bilateral legs. Her leg weakness was thought possible to be due to Guillan Barre versus critical care myopathy/neuropathy, although infectious pathology was also highly considered. Her altered mental status was thought to be due to “infectious encephalopathy”. She could not undergo immediate repeat imaging due to profound autonomic disturbance ranging from bradycardia to PEA, requiring multiple codes and pacer pads to be placed. Once she was hemodynamically stable, a repeat MRI of the cervical spine revealed multiple new abscesses in the posterior, paraspinal soft tissues, with the largest at C2-C4 with extension into the spinal canal (Figure 1).

The MRI of her thoracic and lumbar was unremarkable. Due to continued autonomic instability, laminectomy with the patient prone was not advised and she underwent percutaneous cervical epidural abscess drainage. Her exam did not improve after this. In addition, her mental status deteriorated to coma. A repeat head CT showed acute hydrocephalus (Figure 2). An external ventricular drain (EVD) was placed bedside at a right frontal approach. A repeat head CT showed improvement in hydrocephalus and anterior displacement of the poorly seen upper cervical cord and medulla.

Despite maximal treatment, her neurological exam deteriorated with loss of brain stem reflexes and brain death. A neuropathologic evaluation revealed evidence of an extensive purulent inflammation involving the spinal epidural space from the cervical to sacral levels, extending to subdural and parenchymal areas, with total infarction the cord. There was no evidence of an inflammatory process involving the brainstem, cerebellum or the cerebrum.

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Discussion

Hydrocephalus is a common complication of a brain lesion but it is not a common complication of a spinal cord lesion. In the literature, there are cases reported of hydrocephalus secondary to spinal pathology such as spinal tumors, vascular pathology, trauma or congenital abnormalities of the cervico-medullary junction [1-3]. It has been hypothesized that disruption of CSF flow, impaired absorption at arachnoid villi due to elevated CSF protein or fibrinogen, tumor infiltration into the cistern, direct compression of ventricular outlet, or compression of the spinal venous plexus by the tumor itself lead to hydrocephalus [2,4-8]. According to the Williams theory, lesions at the foramen magnum cause a dissociation between the cranial and spinal subarachnoid fluid pressures by obstructing the free flow of CSF [9].

In this case, it was initially hypothesized that compression from the spinal epidural abscess (SEA) caused cord infarction and resultant hydrocephalus. However, pathological findings revealed both abscess and infarction within the spinal cord itself. Intramedullary abscess carries a poor prognosis with permanent neurological deficits and often death, even if diagnosed and treated early and effectively. Diagnosis may be delayed and overlooked because of the nonspecific symptoms, such as fever, back and abdominal pain. MRI is the most sensitive imaging modality to diagnose spinal epidural and medullary abscesses. It is quite uncommon as in our patient to have initial negative MRI imaging then abscess development despite antibiotic treatment in less than 48 hours. In the case of SEA, damage to the spinal cord can be caused by direct compression by the abscess, vascular compromise and infarction, bacterial toxins, and inflammation that could be irreversible [10-12]. Compared to SEA, spinal cord abscesses are exceedingly rare, although reported in IV drug abusers. Due to the rarity, most literature pertains to SEA. Other risk factors include alcoholism, diabetes mellitus, epidural anesthesia, spinal surgery, spinal trauma and immunodeficiency [13-15]. A recent study of 128 cases of SEA revealed a history of IV drug abuse (39.1%) is the most frequent risk factor for SEA followed by diabetes mellitus (21.9%) [14]. Our patient had history of polysubstance IV drug abuse and HIV infection, placing her at a high risk for SEA. SEA typically arises secondary bacteremia, with the common biologic agent S. aureus. Microbiologic evaluation of our patient revealed evidence of Escherichia coli and S. aureus initially in blood cultures. Postmortem cultures from the spinal cord and inflamed meningeal coverings revealed a mixed growth of E. coli, Klebsiella pneumonia and Neisseria subflava. The mainstay of treatment for compressive or symptomatic SEA is antibiotic treatment and surgical debridement [15]. Poor outcome occurs more frequently in patients with SAE at cervical cord levels. Early surgery improves neurologic outcomes compared with surgical treatment delayed by a trial of medical management. More than 41% of patients treated medically failed management and required surgical decompression. Diabetes, C-reactive protein greater than 115, leukocytosis greater than 12.5, and bacteremia predict failure of medical management [16].
Conclusion

We present a case of an IV drug abuser with HIV who presented with bacteremia and abdominal pain who rapidly developed a compressive cervical SAE, cord abscess and infarction with resultant autonomic instability (spinal cord shock) and acute hydrocephalus. Despite treatment she succumbed to her disease. This is the first known case in the literature of this presentation and constellation of complications related to SEA.

References