Posttraumatic Meningitis: A Rare Complication of Open Brachial Plexus Palsy

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Abstract

We herein report a unique case of meningitis resulting from an open brachial plexus injury. The probable mechanism of infection, early diagnostic features that are lifesaving, and successful management are also detailed. A 27-year-old healthy man had a polytrauma, which included a subclavian vessel hematoma, an open wound in the subclavian region, and global brachial plexus palsy on the right side. The open wound was debrided and sutured primarily in the theater while all the other injuries were managed conservatively. His initial normal neurological status worsened on day 6, imaging showed diffuse cerebral edema with meningeal enhancement. His worsening Glasgow coma scale required elective ventilation, and a cerebrospinal fluid tap was done, revealed meningitis, and the culture grew gram-negative coccobacilli (*Acinetobacter*). He was treated with intrathecal colistin and intravenous meropenem and colistin concurrently. Following clinical improvement, he was extubated after 9 days on the ventilator, and antibiotics de-escalated according to the laboratory values. He was discharged with normal neurological status, and at 6 months follow-up showed no residual deficits. The level of evidence is level 5.

Keywords

- meningitis
- brachial plexus palsy
- nerve transfer
- *Acinetobacter*
- cerebrospinal fluid analysis

Introduction

Posttraumatic meningitis (PTM) is considered to be a meningeal infection with craniocerebral edema not restricted by the temporal proximity of the trauma. Anatomical defects following trauma are a major etiological factor in meningitis, as they produce a breach in the complex defense systems which are aimed to guarantee central nervous system integrity and homeostasis.

Here, we report a rare case of a 27-year-old man with open brachial plexus injury leading to PTM.

Case History

A 27-year-old man with no comorbidities was referred to our hospital 1 day after a road traffic accident with a suspected subclavian artery injury. He had monoparesis of the right upper limb with clinical examination suggestive of global brachial plexus injury with total paralysis of the right upper limb. The presence of Horner's sign and paralyzed serratus anterior muscle suggested a preganglionic avulsion of all the roots of the brachial plexus. There was an associated 5-cm deep lacerated wound over the right supraclavicular region. Computed

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tomography (CT) angiography was suggestive of subclavian artery injury with adequate distal vascularity through the collaterals. The limb was clinically well perfused albeit absent palpable radial pulse. In view of the good distal vascularity subclavian artery, the injury did not need any intervention. He also had a closed fracture of the left ulna, grade 2 liver laceration, and right-sided second—fourth rib fractures and underlying lung contusion. He had a normal neurological status (Glasgow coma scale) and the initial CT of the brain showed minimal subarachnoid hemorrhage with a thin interhemispheric bleed. After initial resuscitation, he underwent exploration and debridement of the neck wound with primary suturing and was shifted to the high-dependency unit postoperatively. On a postoperative day 6, he developed generalized irritability and neck pain with no nuchal rigidity for which a repeat CT scan was done which showed a mild increase in cerebral edema. The institution of antiedema measures improved his symptoms for 24 hours. However, he subsequently became irritable and drowsy, with right-sided hemiparesis, ophthalmoplegia, and nuchal rigidity. CT of the brain with contrast showed meningeal enhancement. In view of neurological deterioration, it was decided to electively ventilate the patient. Cerebrospinal fluid (CSF) analysis was done which showed purulent CSF with a total count of 35,000 cells and culture sensitivity grew gram-negative cocobacilli (*Acinetobacter*). He was subsequently started on Inj. meropenem 2 g intravenous (IV) 8th hourly and colistin 4.5 MU IV 12th hourly along with 10 mg 12th hourly dose of intrathecal colistin. In view of the improvement shown in serial CSF analysis and improvement in neurological status, the patient was extubated after 9 days. The antibiotic treatment was continued for 14 days intravenously and 10 days via an epidural catheter. After 2 weeks when his condition stabilized, he underwent left forearm radius plating under regional anesthesia. At the time of discharge, the patient was conscious, oriented, walking, and taking an oral diet.

He was regularly followed up in the outpatient department and subsequently underwent surgery for his brachial plexus palsy under general anesthesia, wherein the global avulsion of all the roots was confirmed, and the patient underwent a spinal accessory to musculocutaneous nerve transfer with an intervening sural nerve graft. At 1 year postbrachial plexus surgery, he presented with good recovery of elbow flexion and is awaiting trapezius transfer to improve his shoulder abduction and free-functioning muscle transfer to restore his finger flexion. At his last presentation, he had perfect mentation, no motor deficit in the lower limbs, and normal bowel and urinary control.

Discussion

Common conditions that are risk factors for PTM include skull fractures, especially basilar skull fractures, facial fractures, CSF leaks, and intracranial pressure monitoring catheters.[1,2] PTM has a very high mortality rate of 65%. As PTM usually occurs in critically ill or polytrauma patients, their diagnosis can be delayed due to various confounding factors such as head injury, acidosis, electrolyte imbalance, etc. Early diagnosis and initiation of antimicrobial therapy, along with supportive care is the main modality in treating these conditions. There should be a high index of suspicion of meningitis in patients with a history of neurosurgery, skull fractures, open vertebral injury, or CSF rhinorrhea when they present with irritability, disorientation, and fever.

Diagnosis of PTM ranges from less than 24 hours to many years.[3] The median injury to infection interval which gives an idea about the disease progression ranges from 5 to 13 days. Causative agents for PTM are varied including ranges of both gram-negative and gram-positive organisms. The commonly reported gram-positive organisms are *Staphylococcus aureus* and streptococcal species.[4] Gram-negative bacteria include *Escherichia coli*, *Klebsiella*, *Neisseria meningitidis*, *Pseudomonas*, and *Haemophilus influenzae*.[5] Antibiotics should be chosen with the kind of organisms expected to cause the disease and their ability to penetrate the blood–brain barrier. PTM with onset less than 3 days and the closed wound was usually pneumococcal while patients with penetrating injuries or prolonged hospitalization or prolonged onset were usually with a gram-negative or resistant organism.

In our case, the route of pathogenic entry is most likely the dural tear caused by avulsion injury of the brachial plexus roots. The preexisting radiating neck pain and the right upper limb palsy due to brachial plexus injury made the signs of nuchal rigidity and right hemiplegia a diagnostic challenge. The patient had associated hyponatremia and a mild edema, which could be explained by the head injury. This made it even more challenging to confirm the cause of the irritability the patient was experiencing. Traditional signs of meningitis such as Kernig's and Brudzinsky's signs are usually not present in these patients, or the symptoms are usually missed due to their critical state.[6]

The extremely purulent CSF sample with a total count of 35,000 in our case with no other previous history of hospitalization in a young adult was extremely alarming. This could be due to the delay in initial debridement of the wound, as he was referred to our hospital 1 day after primary stabilization at a local hospital, or because the vessel hematoma with collaterals was instrumental in causing the hematogenous spread of bacteria along with the dural tear causing a simultaneous spread to leptomeninges. The turbid CSF in an otherwise immunocompetent patient prompted the aggressive antibiotic regimen, intrathecally as well as intravenously. The culture in 24 hours revealed the deadly *Acinetobacter* species which warranted the continuation of antibiotic intrathecally. As per serial CSF analysis, antibiotics were stepped down and stopped (the stoppage of intrathecal antibiotics was guided by the sterile CSF cultures on day 10).

Conclusion

Brachial plexus injuries vary greatly in severity, depending on the type of injury and the amount of force placed on the plexus.[7] The most severe of these types are preganglionic avulsion injuries,[8] where the nerve root covered by the spinal dura mater is avulsed from the spinal cord tearing the dural sleeve. There is an open connection between

the brachial plexus and the spinal cord meningeal layers in brachial plexus injury, and an open brachial plexus injury can result hence in PTM by entry of pathogens. Hence, any open brachial plexus injury with avulsion of roots needs an immediate debridement and high index of suspicion for meningitis, delay of which could have disastrous consequences.

Declaration of Informed Consent

There is no information (names, initials, hospital identification numbers, or photographs) in the submitted manuscript that can be used to identify patients. This case report has been cleared by the hospital's ethical board for reporting.

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None.

Conflict of Interest

None declared.

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