



Transient Horner's Syndrome following Posterior Cervical Spine Instrumentation: A Postoperative Dilemma

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Horner's syndrome is a rare phenomenon associated with a wide variety of medical and surgical conditions. Ipsilateral ptosis and miosis occur in Horner's syndrome due to the sympathetic disruption at any point along the pathway from the posterolateral hypothalamus to the cervical sympathetic trunk.¹ It is a well-recognized but infrequent entity following cervical spine instrumentation through the anterior approach with an incidence ranging from 0.02% to 3.8% during the anterior cervical discectomy and fusion (ACDF) procedure.^{2,3} We report a rare case of transient Horner's syndrome following posterior cervical spine instrumentation for congenital atlantoaxial dislocation (AAD).

A 13-year-old male patient presented with neck pain and weakness in all four limbs for the past 6 months. Neurological examination revealed a motor power of grade 3 in all four limbs with a normal pupillary size and reactivity. A diagnosis of reducible AAD was considered based on the dynamic computed tomographic (CT) imaging without radiological evidence of basilar invagination or Arnold Chiari malformation. The patient was planned for C1–C2 fusion under general anesthesia in the prone position. After induction of anesthesia, the patient was placed in the prone position, the head was fixed with the Mayfield clamp, and the eyes were protected with cotton pads. The C1–C2 fixation was done by pars interarticularis screws under fluoroscopic guidance. Following an uneventful intraoperative course, the patient was turned supine, and anesthesia was reversed. A pupillary examination was performed as eye-opening was absent and there was no motor response to painful stimuli even after 30 minutes following the anesthesia reversal. A pinpoint

non-reactive pupil and incomplete eye closure (ptosis) were noted on the right side while the contralateral eye had no abnormality. The patient was normothermic and blood gas analysis showed no abnormality with normal serum sodium levels. There was no facial edema or cervical swelling on clinical examination. Suspecting an intracranial event, an immediate postoperative CT was performed that was unremarkable. The patient was shifted to the neuro-intensive care unit (NICU) due to a Glasgow coma scale (GCS) of E1VTM1 and was electively ventilated. The patient was started on intravenous dexamethasone 12 mg/day suspecting handling of the cervico-medullary junction during instrumentation. The pupillary examination was performed hourly, and no sedation was given in the NICU considering the poor GCS. A postoperative magnetic resonance imaging scan of the brain and cervical spine on day 1 revealed no abnormality with appropriate placement of the C1–C2 screws. There was resolution of Horner's syndrome (right eye) with return of full GCS (with baseline motor power) score after a period of 36 hours in the NICU and the trachea was extubated successfully.

To the best of our knowledge, this is the first report of transient Horner's syndrome following posterior cervical spine instrumentation in a patient with AAD with normal postoperative imaging. Muller et al have reported a case of Horner's syndrome following the posterior fusion of the thoracic spine, thereby raising awareness of this condition.⁴ The cervical sympathetic chain has an anterolateral location, and a prolonged retraction of the longus coli muscle during the cervical spine instrumentation through

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an anterior approach can be a factor contributing to the dysfunction of cervical sympathetic chain.⁵ The transient occurrence of Horner's syndrome following posterior cervical spine instrumentation is a conundrum that can mislead the diagnosis toward an intracranial event. Even though the etiology of Horner's syndrome is unclear following posterior cervical instrumentation in congenital AAD, it can be possibly attributed to the involvement of the central sympathetic pathway descending through the brain stem. The central sympathetic fibers from the lateral hypothalamus traverse the lateral brain stem to finally terminate at the intermediolateral gray horn of the cervical spinal cord, and insult at any point through this pathway can produce Horner's syndrome, which is typically unilateral.⁶ We ruled out all the possible causes of delayed recovery (metabolic, hypothermia, intracranial bleeding, anesthetic agents) in our patient. The most probable cause of Horner's syndrome and poor neurological status in our case can be attributed to the brain stem handling and edema during the C1-C2 instrumentation that responded well with postoperative intravenous steroids.

To conclude, transient Horner's syndrome can occur as a complication of posterior cervical spine procedures that can mislead towards an intracranial event.

Conflict of Interest

None declared.

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