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Case Report

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Acute Cranial Nerve VI Palsy Following Prolonged Prone Positioning in an Adolescent With Neurofibromatosis Type 1: A Rare Complication of Spinal Surgery in the Prone Position

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Abstract

The abducens or sixth cranial nerve provides motor innervation to the lateral rectus muscle, which abducts the ipsilateral eye with secondary innervation of the contralateral medial rectus muscle to allow for coordinated movement of both eyes. Various acute and chronic pathologic conditions, most importantly pontine infarctions and increased intracranial pressure, can result in acute sixth cranial nerve palsies. We report the uncommon occurrence of acute abducens nerve palsy following spinal fusion surgery in an 18-year-old male patient with a history of multiple neurological and orthopedic conditions. Postoperatively, the patient presented with symptoms that included left diplopia with restricted upward and downward gaze, indicative of abducens nerve palsy. The anatomy of the sixth cranial nerve is discussed, potential etiologies of sixth nerve palsy presented, and a proposed diagnostic workup reviewed. Our report emphasizes the need for comprehensive exploration of ocular symptoms following spinal surgery, given the various potential etiologies of sixth nerve palsy.

Keywords: Sixth nerve palsy; Abducens nerve; Cranial nerves; Posterior spinal fusion; Prone position

Introduction

The abducens or sixth cranial nerve provides motor innervation to the lateral rectus muscle, which abducts the ipsilateral eye with secondary innervation of the contralateral medial rectus muscle resulting in coordinated movement of the eyes. Abducens nerve palsies can result from various pathologic conditions of the central nervous system (CNS), with pontine infarctions and increased intracranial pressure (ICP) being the most significant etiologies [1]. The occurrence of abducens nerve palsy following spinal surgery is extremely rare [2]. Given its potential as a sign of significant CNS pathology, prompt recognition and diagnostic workup to determine the etiology is required. We report the presence of acute abducens nerve palsy following spinal fusion surgery in an 18-year-old male patient with a history of multiple neurological and orthopedic comorbidities. We discuss the anatomical basis of the sixth cranial nerve, potential etiologies of sixth nerve palsy, and review the relevant literature on postoperative occurrences of this condition. Our report emphasizes the need for comprehensive exploration of ocular symptoms following spinal surgery, given the various potential etiologies of sixth nerve palsy.

The review and presentation of this case followed the guidelines set by the Institutional Review Board of Nationwide Children's Hospital (Columbus, Ohio).

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Case Report

Patient history

The patient was an 18-year-old male (34.5 kg) with a history of progressive scoliosis and persistent back pain despite a previous spinal fusion 22 months prior. The patient's surgical history was significant for multiple previous surgical procedures including craniotomy for tumor resection, spinal growing rod interventions, halo application, and repair of orbital wing dysgenesis through a frontal craniotomy. The past medical history was significant for neurofibromatosis type 1, optic nerve glioma affecting the left eye, brain tumor (low-grade glioma) status post resection, growth failure, autism, and attention deficit hyperactivity disorder. There was no past history of previous perioperative or anesthetic complications. Current medications included amlodipine 10 mg and lisinopril 5 mg, both taken once daily for hypertension management. The patient was also prescribed diazepam 5 mg every 8 h as needed, for muscle spasms. Additional medications included methylphenidate extended-release 54 mg every morning, clonidine 0.2 mg

every night, cyproheptadine 8 mg every night, calcium carbonate 1,000 mg once daily, and vitamin D3 (cholecalciferol) 2,000-unit capsule once daily. The only medication allergy reported was hydrocodone.

Diagnosis

Preoperative vital signs revealed a blood pressure of 101/58 mm Hg, pulse rate of 72 beats per minute, respiratory rate of 18 breaths per minute, pulse oxygen saturation (SpO_2) of 97%, and body mass index (BMI) of 15.50 kg/m^2. The patient was alert, cooperative, and exhibited no acute distress. The skin was warm and dry with multiple scattered cafe au lait spots observed. Respiratory and cardiovascular examinations were unremarkable. The abdomen was soft, flat, and nondistended with normal bowel sounds. Neurologic examination revealed symmetrical and full strength in all extremities without dysmetria. There was severe kyphoscoliosis, and a protruding rod was observed on the right side of the patient's back without any surrounding skin breakdown. Preoperative imaging revealed a spinal curvature measured at 120° with the apex on the right side at the T5 level. Additionally, kyphosis of 140° was noted with the apex at T5. Preoperative imaging identified narrowing of the spinal canal at the T5 level, with the anteroposterior dimension measuring 6 mm.

Treatment

The patient underwent a redo spinal fusion procedure consisting of C3 - L3 fusion, T3 - T6 decompression, and T4 - T5 vertebrectomy. The surgery lasted approximately 9 h, during which the patient was positioned in the prone position with Gardner-Wells tongs and traction. Intraoperative concerns included temporary loss of motor somatosensory evoked potentials. Specifically, study of median nerve and posterior tibial nerve revealed intermittent decrease in amplitude, although it remained stable during intraoperative monitoring. Intermittent episodes of hypotension were initially treated with fluid, blood, and intermittent doses of phenylephrine. Ongoing hypotension required the use of a continuous infusion of phenylephrine (0.1 - 4 µg/kg/min), vasopressin (0.5 - 1.5 mIU/kg/min), and norepinephrine (0.05 - 0.2 µg/kg/min). Total fluid administration for the procedure included 6,780 mL with 1,500 mL of 5% albumin, 3,000 mL of Normosol-R, 69 mL of cryoprecipitate, 647 mL of fresh frozen plasma, 994 mL of packed red blood cells, and 570 mL of blood from the cell saver.

The surgery lasted approximately 9 h with an estimated blood loss of 1,400 mL. Dexamethasone and ondansetron were administration for prophylaxis against postoperative nausea and vomiting. Hydromorphone and acetaminophen were administered intravenously for postoperative analgesia. At the completion of the surgical procedure, the patient was positioned supine, and extubated. Following repositioning, all pressors were able to be discontinued. His hemodynamic and respiratory status remained stable during his time in the postanesthesia care unit prior to planned transfer to the pediatric intensive care unit (PICU).

Follow-up and outcomes

The patient was transferred to the PICU in stable condition. During the assessment in the PICU, the patient mentioned experiencing diplopia, but he did not report any ocular pain with eye movements. Additionally, there was swelling and edema on one side of the periorbital area, and his pupils reacted normally to light. A consulting ophthalmologist suspected an acute left-sided cranial nerve VI palsy and recommended close monitoring while maintaining the previously planned postoperative treatment. Additionally, the ophthalmologist suggested that a short course of corticosteroids, specifically dexamethasone, could be considered if the problem did not resolve. Computed tomography imaging of the brain without contrast was ordered and revealed new gas in the basilar cisterns and dorsal soft tissues at the craniocervical junction, presumed to be related to the recent spinal surgery. The consulting ophthalmologist noted that the patient exhibited limited upward and downward gaze. No other abnormality was noted. Within 24 h, the patient exhibited improvement in the ocular symptoms. The left eye demonstrated complete movement towards the midline, although a mild medial deviation persisted at rest. Swelling of the left eyelid decreased, allowing the patient to open the eye independently. At 48 h postoperatively, the cranial nerve VI palsy had resolved, leading to the restoration of normal ocular alignment and vision.

Discussion

The abducens nerve plays a vital role in controlling the extraocular motor functions of the eye [1]. It arises from the brainstem, lacks sensory function, and innervates the lateral rectus muscle for eye abduction [1]. Additionally, it provides secondary innervation to the contralateral medial rectus muscle through the medial longitudinal fasciculus, ensuring coordinated movement of both eyes in a lateral direction [1]. Among all the cranial nerves, the abducens nerve has the second longest intracranial course, traveling through the subarachnoid space and cavernous sinus before entering the orbit through the superior orbital fissure.

The diagnostic evaluation of abducens nerve palsy should be planned according to the suspected etiology. Evaluation of a patient with acute abducens nerve palsy begins with a thorough neurologic examination and exclusion of immediate life-threatening etiologies, including increased ICP and imminent herniation or pontine infarction [1]. Significant intracranial pathology even in the absence of clinical findings on neurologic examination, can easily be ruled out by emergent computed tomography or magnetic resonance. Moreover, a thorough ophthalmological examination should be performed, involving assessment of visual acuity, visual fields, and detailed inspection of the eye movements. Lumbar puncture may be conducted for cases suspecting an infectious or inflammatory cause. [1].

The etiology of abducens nerve palsy is multifactorial including vascular, traumatic, neoplastic, inflammatory, infectious, and congenital causes [1]. Comparing this case with those reported in the existing literature highlights the necessity

Table 1. Summary of Previously Reported Cases of Diplopia Following Spinal Surgery in Prone Position

Study	Procedure	Age (years)	Onset	Presumed etiology	Management	Resolution
Barsoum et al, 1999 [3]	L3 - L5 decompression revision and L2 - L5 fusion	59	Unknown	Abducens nerve traction	Conservative	> 3 months
Nakagawa et al, 2003 [9]	Resection of spinal tumor at C1 - C2	22	3 days	CSF leakage	Conservative	> 3 months
Cho et al, 2009 [5]	L4 - 5 and L5 - S1 posterior fusion	61	2 days	CSF leakage	Conservative	Within 3 months
Abd-Elsayed et al, 2011 [11]	Lumbar posterior fusion	14	1 day	Facial edema	Conservative	Few days
Abd-Elsayed et al, 2011 [11]	Lumbar posterior fusion	34	1 day	Facial edema	Conservative	Few days
Thomas et al, 2012 [2]	L4 - L5 discectomy	53	7 days	CSF leakage	Dural repair	Within a month
Joo et al, 2013 [7]	L5 - S1 discectomy	48	3 days	CSF leakage	Dural repair	Few days
Khurana et al, 2013 [8]	T9 - T10 discectomy	48	Within 3 weeks	CSF leakage	Dural repair	Within 3 months
Khurana et al, 2013 [8]	Thoracotomy with T8 hemivertebrectomy	46	Few days	CSF leakage	Conservative	> 3 months
Sandon et al, 2016 [10]	T6 - 7 discectomy + laminectomy of T6/T7/T8 + T5 - T8 fusion	47	10 days	CSF leakage	Dural repair	Within a month
Kim et al, 2021 [4]	C1 - C2 fusion	71	1 day	Abducens nerve traction	Conservative	Within 3 months
Cordeiro et al, 2022 [12]	C5 - 6 discectomy, fusion	48	1 day	Idiopathic	Conservative	Few days
Hernandez-Mateo et al, 2023 [6]	Laminectomy, fusion	65	60 days	CSF leakage	Conservative	> 3 months
Our report, 2023	C3 - L3 fusion, T3 - T6 decompression, and T4 - T5 vertebrectomy	18	1 day	Abducens nerve traction	Conservative	Few days

CSF: cerebrospinal fluid.

of a thorough assessment for patients presenting with visual changes such as diplopia following prone surgery (Table 1) [2-12]. Ascertaining the underlying cause of these symptoms is crucial for determining appropriate treatment and projecting recovery timelines. We identified six cases where postoperative cranial nerve VI palsy was linked to persistent cerebrospinal fluid (CSF) leakage [2, 5-10]. Treatment in these instances varied between a conservative approach and dural repair, depending on individual clinical circumstances and the extent of the CSF leak. CSF leakage as etiology appears unlikely in our case because the magnetic resonance imaging (MRI) was morphologically normal and did not reveal any subdural fluid collections or hematoma.

In the presented case, the presumed etiology was traction on the abducens nerve due to prolonged prone positioning, similar to cases reported by Barsoum et al in 1999, and Kim et al in 2021 [3, 4]. Interestingly, this patient's head was placed in pins during the case, leading to a potential hypothesis that the pinning may have played a role in the nerve injury. Pinning creates a fixed point of traction and can potentially exert pressure on surrounding soft tissues and neurovascular structures, including cranial nerves with an extensive or delicate intracranial course, such as the abducens nerve. In prolonged surgeries where the head remains immobile, this fixed point could theoretically exacerbate any traction or compression exerted on the nerve due to the prone position. Furthermore, the patient's pre-existing condition of neurofibromatosis type 1 might make the abducens nerve more susceptible to mechanical stress and injury.

Recovery timeframes for cranial nerve VI palsy following spinal surgeries varied widely, ranging from a few days to over 3 months. Our patient's symptoms resolved after a few days, also consistent with the two cases mentioned. The conservative approach to management adopted in these cases includes watchful waiting, without additional invasive interventions. This approach is based on the presumed etiology and the expectation the symptoms would resolve as the nerve recovers from the mechanical stress. The rapid resolution observed in our patient may not necessarily indicate recovery of the abducens nerve itself. Rather, it is plausible that the symptoms were alleviated due to the resolution of a localized edematous process. This suggests that the issue may not be an abducens palsy in the traditional sense, but more an ocular compartment syndrome, explaining the limitations in both upward and downward gaze.

Diplopia is the most specific symptom of cranial nerve VI palsy, as the lateral rectus muscle's impairment prevents the affected eye from abducting properly. In addition to diplopia, the patient had left-sided periorbital swelling, raising the suspicion for other unilateral causes of nerve irritation. However, the predominantly left-sided periorbital irritation may

be explained by prior history of left eye involvement in the setting of neurofibromatosis type 1. Pre-existing anatomical variations might make certain structures more susceptible to mechanical stress, potentially explaining why the patient experienced swelling symptoms primarily on the left side. Understanding the relevant anatomy and considering individual patient characteristics and medical history are essential for accurate diagnosis and management of this rare complication.

Although rare, abducens nerve impairment has been reported previously following prolonged surgical procedures in the prone position (Table 1) [2-12]. Prolonged traction on the intracranial portion of the sixth cranial nerve can result in localized ischemia, resulting in transient postoperative paresis of the nerve. In this report, the patient experienced acute edematous swelling of the left upper and lower eyelids, diplopia, and restricted gaze. However, within 24 h, the patient showed improvement; and by 48 h, ocular symptoms had resolved, and eye movement was fully restored.

In summary, the case of our 18-year-old patient with a history of multiple neurologic and orthopedic comorbidities represents a rare but potential complication of spinal surgery - cranial nerve VI palsy. In our report, the presumed etiology was cranial nerve traction associated with prolonged prone positioning during surgery. Differential diagnosis should consider other etiologies that include CSF leakage, facial edema, and idiopathic causes. Recognizing the anatomy and causes of the sixth nerve palsy, combined with knowledge of prior case studies, can aid in identifying and managing this complication. The quick recovery seen in this case indicates a good prognosis for postoperative cranial nerve VI palsy associated with prolonged prone positioning. However, it is crucial to make a correct diagnosis to rule out structural causes of cranial nerve VI palsy, which could require further imaging studies or even surgical intervention.

Learning points

Abducens nerve palsy should be considered a possible but rare postoperative complication after prolonged prone positioning during spinal procedures. Early detection through comprehensive neurological and ophthalmic assessments is crucial for prompt management, and a conservative approach may be effective if the palsy is presumed to be due to mechanical stress.

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Financial Disclosure

None to declare.

Conflict of Interest

None to declare.

Informed Consent

Review of this case and presentation in this format followed the guidelines of the Institutional Review Board of Nationwide Children's Hospital (Columbus, Ohio). Informed consent was obtained for use of de-identified information for publication.

Author Contributions

CM: case review, preparation and editing of manuscript. VO provided clinical care, manuscript review, and editing. JDT: manuscript preparation, review, and editing.

Data Availability

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

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