CASE REPORT

Isolated Pupillary-sparing Cranial Nerve III Palsy from a Subgaleal Abscess

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ABSTRACT

Cranial nerve (CN) III palsy is rarely caused by intraorbital compression, let alone from a subgaleal abscess.

We present a case of a hypertensive 55-year-old man with an acute isolated pupillary-sparing left CN III palsy from a left subgaleal abscess with associated pterional osteomyelitis and frontotemporal mass. This is the first reported such case and the third reported case of a chronic spontaneous subgaleal abscess. A seemingly routine case of an acute, pupillary-sparing, isolated CN III palsy from hypertension turned out to be a rare case in terms of etiology of the palsy and of the source of the abscess.

Keywords: Cranial Nerve III, palsy, subgaleal abscess, ptosis, ophthalmoplegia

INTRODUCTION

Acute, pupillary-sparing, isolated cranial nerve (CN) III palsy in the middle aged population is often caused by vasculopathic etiologies such as hypertension and diabetes mellitus.[1] However, a compressive etiology still needs to be ruled out as the pupil may be spared early in the disease process.[2] A common cause of compression is a posterior communicating artery aneurysm while compression from aneurysms in the posterior cerebral artery and superior cerebellar were also reported.[3] However, it is rare to develop an isolated CN III palsy from intraorbital compression, let alone from an abscess extending from a subgaleal source with associated pterional osteomyelitis and frontotemporal involvement.

Spontaneous chronic subgaleal abscess formation in the skull is also rare with only 2 reported cases in the literature.[4,5] Acute cases are more common due to a localized source usually thru a skin trauma, injury, or surgical incision. Other identified sources include dental caries, sinusitis, and intravenous inoculation thru injectable recreational drugs. Management for acute cases is more straightforward than for chronic ones since diagnosis is easier to establish.[5] The first reported case did not resolve with directed antibiotics for *Streptococcus pyogenes* and eventually had to be surgically drained. The second case needed to be worked up and was treated for melioidosis.[4] We present a case of a middle aged male with CN III palsy from a frontotemporal mass.

CASE

A 55-year-old man consulted our institution for a left frontotemporal mass and left upper eyelid ptosis. Five weeks

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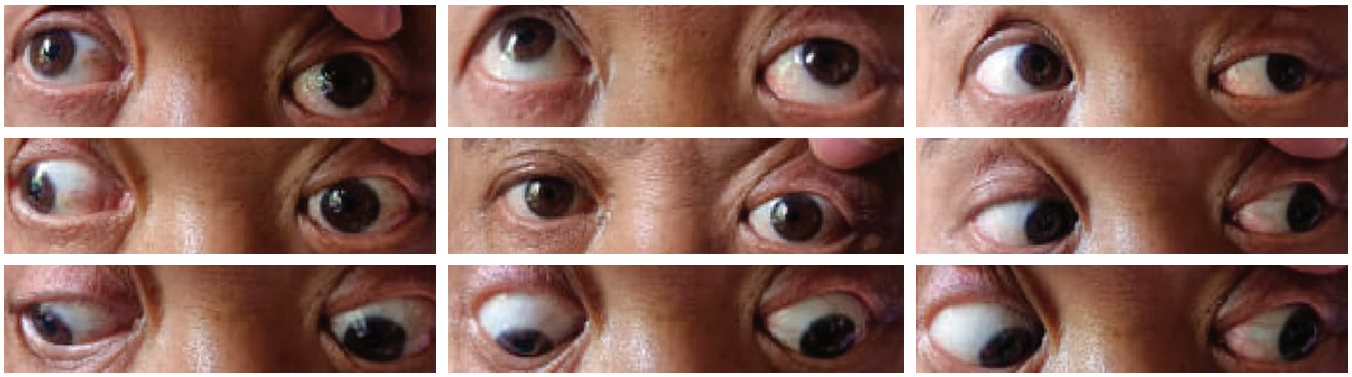

Figure 1. There is intact abduction, full limitation in adduction, and partial limitation of the other gazes in the left eye. There is also ptosis of the left upper eyelid.

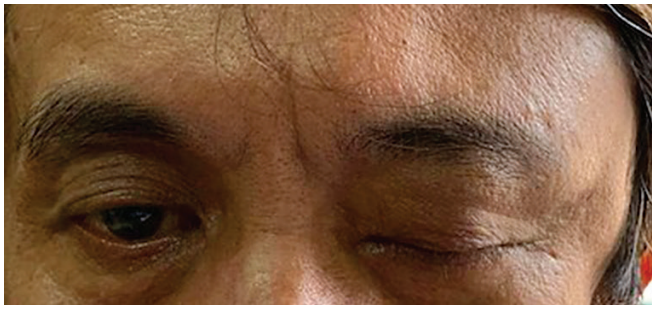

Figure 2. A left frontotemporal mass with associated severe left ptosis.

prior, he saw an ophthalmologist for a 1-week history of diplopia. He was diagnosed with left CN III palsy based on the left eyelid ptosis and extraocular muscle movement limitation (except for abduction) with no relative afferent pupillary defect or anisocoria (Figure 1). He was also diagnosed with hypertensive retinopathy stage 2 for both eyes. The patient was hypertensive with the usual blood pressure of 150/100 mmHg. The patient was referred to Internal Medicine for management of hypertension, but cranial imaging was also requested to rule out an aneurysm. Four weeks before this consult, he developed a left frontotemporal mass (Figure 2) with intermittent non-radiating throbbing pain graded numeric rating scale (NRS) 3/10.

Computed tomography (CT) with contrast and later, magnetic resonance imaging revealed a 10 x 5 x 11-centimeter soft tissue mass with central necrosis affecting the left frontal, parietal and temporal regions and bones (Figure 3A), left frontal process of the sphenoid and zygomatic bones, left lesser wing of the sphenoid bone, and the left lateral orbital rim with associated left globe proptosis (Figure 3B). The mass extended downward into the left masseter (Figure 3C). The left lateral rectus was thickened and encased, extending into the orbital apex. The superior rectus was partially involved anteriorly, and there was an inferior displacement of the left globe (Figure 3D). The cavernous sinus was intact. No sinusitis was seen. On CT angiogram, no aneurysm or other abnormality was seen. Neurosurgery saw the patient and diagnosed him with a subgaleal mass or abscess with an epidural component on the left frontotemporal area and secondary osteomyelitis. The patient was started on antibiotics and was scheduled for craniectomy for possible mass excision and abscess evacuation. Three days before admission, he developed eye redness, fever, and pain on mastication.

On admission, the patient had a left frontotemporal mass with persistent diplopia and ptosis. The visual acuity for both eyes was 20/20, with complete inability to do adduction, up and left, and down and left ductions of the left eye. The rest of the neurological examination was normal. The patient was afebrile but had a slightly elevated white blood cell count at 11.64 10^9/L, with the predominance of neutrophils at 79%. He claimed that he had weight loss and night sweats in the past months. On chest radiograph, nodular densities were seen in the right perihilar area and right middle lung zone with subtle hazy and linear densities in the right middle lung zone pointing to pulmonary tuberculosis. However, acid-fast bacilli were not seen in the sputum, and he tested negative for the GeneXpert MTB/RIF assay. He also had multiple male partners but tested negative on HIV screening (INTEC antibody test) three weeks before admission. He also tested negative for hepatitis A, B, C, and syphilis. He was hypertensive but not diabetic. He had no migraine episodes, no history of trauma, and no dental caries. He was a 5-pack-year smoker and a regular alcohol drinker. He denied the intake of illicit drugs.

He was started on ceftriaxone and azithromycin with no resolution of the mass. He then underwent left pterional craniectomy and abscess evacuation. Intraoperatively, the drained abscess was white and thick. Post-operatively, he was then started on cotrimoxazole and piperacillin-tazobactam. Gram stain of the abscess revealed Gram-positive cocci in singles and chains and Gram-negative bacilli in singles and pairs, but culture studies yielded no bacteria growth. Acid-fast bacilli were likewise not seen in the specimen. Histopathologic study of the excised pterional bone revealed

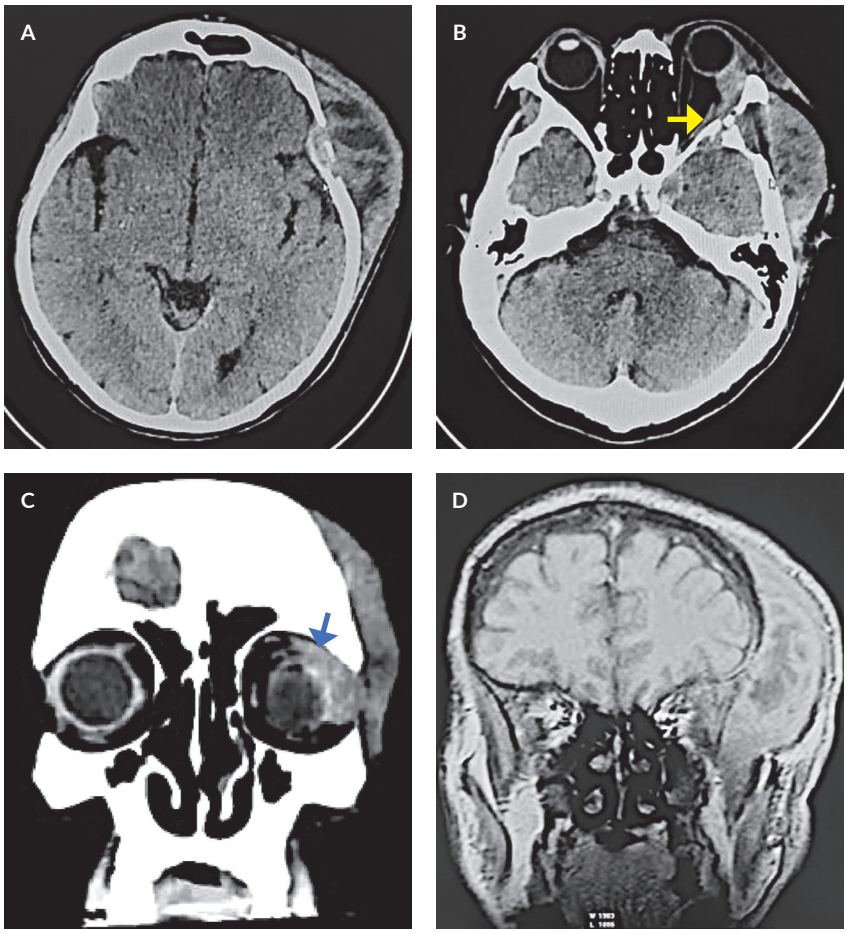

Figure 3. **(A)** An axial view of the CT of the brain shows a soft tissue mass with central necrosis affecting the left frontal, parietal and temporal regions and bones, **(B)** extending into the left frontal process of the sphenoid and zygomatic bones, left lesser wing of the sphenoid bone, and the left lateral orbital rim with associated left proptosis. The left lateral rectus was also involved up to the orbital apex *(yellow arrow)*. **(C)** The left superior rectus was slightly involved *(blue arrow)*, and there was a slight inferior displacement of the left globe. **(D)** A coronal view of the cranial MRI shows the extent of the down into the left masseter.

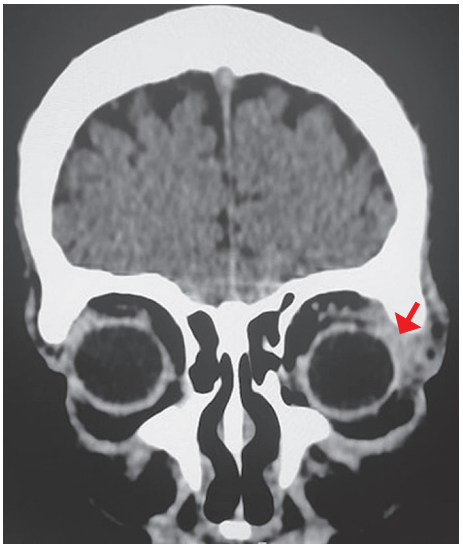

Figure 4. Coronal view of cranial computed tomography shows residual soft tissue mass *(red arrow)* still encasing the lateral rectus and abutting the lateral margin of the superior rectus in the left orbit.

an organizing abscess. Upon discharge, there was still severe ptosis and diplopia from complete limitation in adduction, upgaze, and downgaze. There were 30 degrees of exotropia and a 5-millimeter proptosis of the left eye. A hypoglobus was also noted.

One month after the surgery, there were residual encasements of the lateral rectus on repeat imaging (Figure 4) and a hypoglobus. However, the patient no longer complained of diplopia. He was orthotropic, with no limitations in extraocular movement and with an improvement of the ptosis. No additional intervention was needed except for controlling and monitoring his hypertension.

DISCUSSION

This is the first reported case of an isolated complete CN III palsy from abscess-related intraorbital compression, and the third reported case of a spontaneous subgaleal abscess. A seemingly routine case of an acute, pupillary-sparing, isolated CN III palsy from hypertension turned out to be a rare case in terms of etiology of the palsy and of the source of the abscess.

The localization of the compression to the intraorbital portion of CN III in our patient was based on the presence of lateral rectus involvement extending into the orbital apex and the sparing of the pupil. Involvement of the intraorbital portion of CN III, even from compression, often spares

the pupil as opposed to when compression is located more posterior. Although there was intracranial involvement, it was not severe to cause compression in the supranuclear, nuclear, fascicular, and basilar portions of the CN III tract as there was no evidence of hemorrhage or herniation on cranial imaging. The cavernous sinus was also intact, which was affected in another case report of CN III palsy from an odontogenic abscess.[6]

Subgaleal abscesses result from purulent infections in the space beneath the galea aponeurotica of the scalp. Infection in this site is rare and has been documented from etiologies such as trauma, surgical incisions, adjacent foci of infection, the use of fetal scalp for monitoring, cancers, and hematomas.[5] Schaefer et al., despite isolating *Streptococcus pyogenes* from their patient's aspirated specimens, could not identify any adjacent source of the abscess in the first reported case.[5] They attributed it to a hematogenous spread from a recent pulmonary infection. The second case was a rare presentation of *Burkholderia pseudomallei* infection or melioidosis.[7] These Gram-negative rod bacteria are found in soil and water, are common in the tropics of Southeast Asia and affect patients who have diabetes, heavy alcohol intake, chronic pulmonary and renal disease, steroid use, and cancer.[7] Supputamongko et al.'s patient also presented with weight loss and night sweats but with daily fever spikes.[7]

Our patient only had an ongoing non-tuberculosis pulmonary infection on admission and, as such, can be the most likely source of the subgaleal abscess thru hematogenous spread. He was also immunocompetent and without drug use. Although numerous microorganisms were seen on the gram stain, none were isolated in the culture studies. Our patient also did not have a continuous fever, was not exposed to contaminated soil and water, and did not have the risk factors for melioidosis. Despite the identification of the causative agents, both cases needed surgical drainage, similar to our patient.[5,7] Incision, debridement, and drainage are the recommended surgical procedures for subgaleal abscess, preceded and followed by a long course of directed intravenous antibiotics, especially in the presence of osteomyelitis.[5,7] Our patient did not require a prolonged course of antibiotics despite the presence of osteomyelitis, nor did he require an orbitotomy for the intraorbital and peri-rectal mass. His CN III palsy resolved with no recurrence of the abscess four months after the surgery.

Statement of Authorship

Both authors contributed in the conceptualization of work, acquisition and analysis of data, drafting and revising, and approved the submission of the final manuscript.

Author Disclosure

Both authors declared no conflicts of interest.

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