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Case Report/รายงานผู้ป่วย

Acute Unilateral Isolated Ptosis as a Complication of Frontal Sinusitis: A Case Report

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Abstract

Objective: Acquired upper-eyelid ptosis can be traumatic, mechanical, neurogenic, or myogenic in origin. However, oculomotor nerve or cranial nerve (CN) III palsy caused by frontal sinusitis is extremely rare. The purpose of this report is to document an unusual case of frontal sinusitis in a 15-year-old boy with acute unilateral ptosis as the sole presenting sign of CN III palsy.

Case report: A 15-year-old Thai boy presented with a 2-day history of left-sided upper-eyelid ptosis preceded by a 1-week history of influenza A infection. His pupils were equivalent in size bilaterally, and extraocular muscles exhibited full motility. Magnetic resonance imaging of the brain and orbit revealed fluid opacification and mucosal thickening, predominantly in the left frontal sinus, with no levator palpebrae superioris abnormalities. The case was diagnosed as frontal sinusitis associated with CN III palsy. The ptosis had completely resolved by the fifth day of intravenous levofloxacin.

Conclusions: Frontal sinusitis can lead to palsy of the superior branch of CN III presenting with only isolated ptosis. This case highlights a potential complication of acute sinusitis and raises awareness of a rare differential diagnosis in isolated, unilateral ptosis. Diagnostic neuroimaging may assist in managing such atypical cases.

Keywords: cranial nerve (CN) III palsy, oculomotor nerve palsy, pediatric, ptosis, sinusitis

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Introduction

Upper-eyelid ptosis can be congenital or acquired, and it can be traumatic, mechanical, neurogenic, or myogenic in origin.[1] Aponeurotic ptosis due to levator dehiscence has been most commonly recorded in older people, whereas congenital ptosis, oculomotor nerve or cranial nerve (CN) III palsy, Horner's syndrome, and mechanical ptosis have been widely described in pediatric populations.[1,2] However, it extremely rare for CN III palsy to cause acute unilateral isolated ptosis without limited extraocular muscle motility, afferent pupillary defect, or other neuro-ophthalmological findings. A previous report described that acute ptosis associated with isolated CN III palsy occurred in less than 1% of sphenoid sinusitis cases.[3] This is even rarer in frontal sinusitis, as just one patient with CN III palsy presenting with only isolated ptosis caused by frontal sinusitis has been reported.[4]

The objective of this case report is to highlight an unusual case of frontal sinusitis in a pediatric patient with acute unilateral ptosis as the sole presenting feature of isolated oculomotor nerve or CN III palsy.

Case Presentation

A 15-year-old boy with no past medical or ocular history presented to an ophthalmology clinic in Bangkok, Thailand with a 2-day history of left-sided unilateral isolated ptosis that appeared when he had woken up. He had a 1-week history of low-grade fever, mild frontal headache, and minimal nasal discharge. He had been diagnosed with influenza A infection, for which oseltamivir was prescribed for 5 days. He did not complain of any visual impairment, diplopia, fluctuating ptosis, or orbital pain. He also ruled out any history of trauma, recent vaccination, or treatment of the upper face via injection of botulinum toxin.

Analysis of the vital signs revealed a temperature of 37.1°C, blood pressure of 114/68 mmHg, pulse of 80 beats per minute, and respiratory rate of 20 breaths per minute, with oxygen saturation of 99%. No abnormal head posture or facial anhidrosis was noted. Upon external ophthalmic examination, no periorbital swelling, redness or warmth, or mechanical cause of ptosis was observed; however, there was mild tenderness over the left eyebrow.

His best-corrected visual acuity was 20/25 in both eyes. The intraocular pressure as determined by rebound tonometry was 16 and 19 mmHg in the right and left eyes, respectively. The pupils were reactive to light and equivalent in size bilaterally in bright and dim light, without relative afferent pupillary defect. No remarkable findings of anterior and posterior segments were made upon slit lamp examination. He had partial left upper-eyelid ptosis with the presence of a normal lid crease (Figure 1). The distance from the corneal light reflex to the central upper-eyelid margin, known as margin-reflex distance 1, in the right eye was 3 mm, while in the left it was –2 mm. The vertical palpebral fissure was 8 mm on the right and 3 mm on the left. Levator function was symmetrical and measured 15 mm bilaterally. There were negative findings for all typical myasthenia gravis lid signs, including the fatigability test, Cogan's lid twitch, the curtain sign, and the peek sign. Negativity for Marcus Gunn jaw-winking was also confirmed. Extraocular muscles exhibited full motility in all fields of gaze (Figure 2). Both eyes were orthophoric, and an alternate cover–uncover test did not reveal a phoria. The visual fields were full to confrontation bilaterally. Other cranial nerves revealed no remarkable findings.

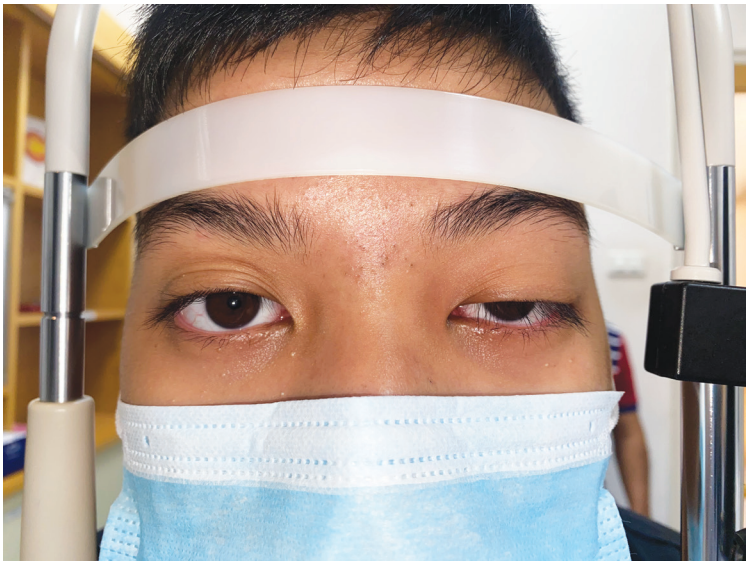

Figure 1 Photograph of the patient. The patient at initial presentation with left upper-eyelid ptosis.

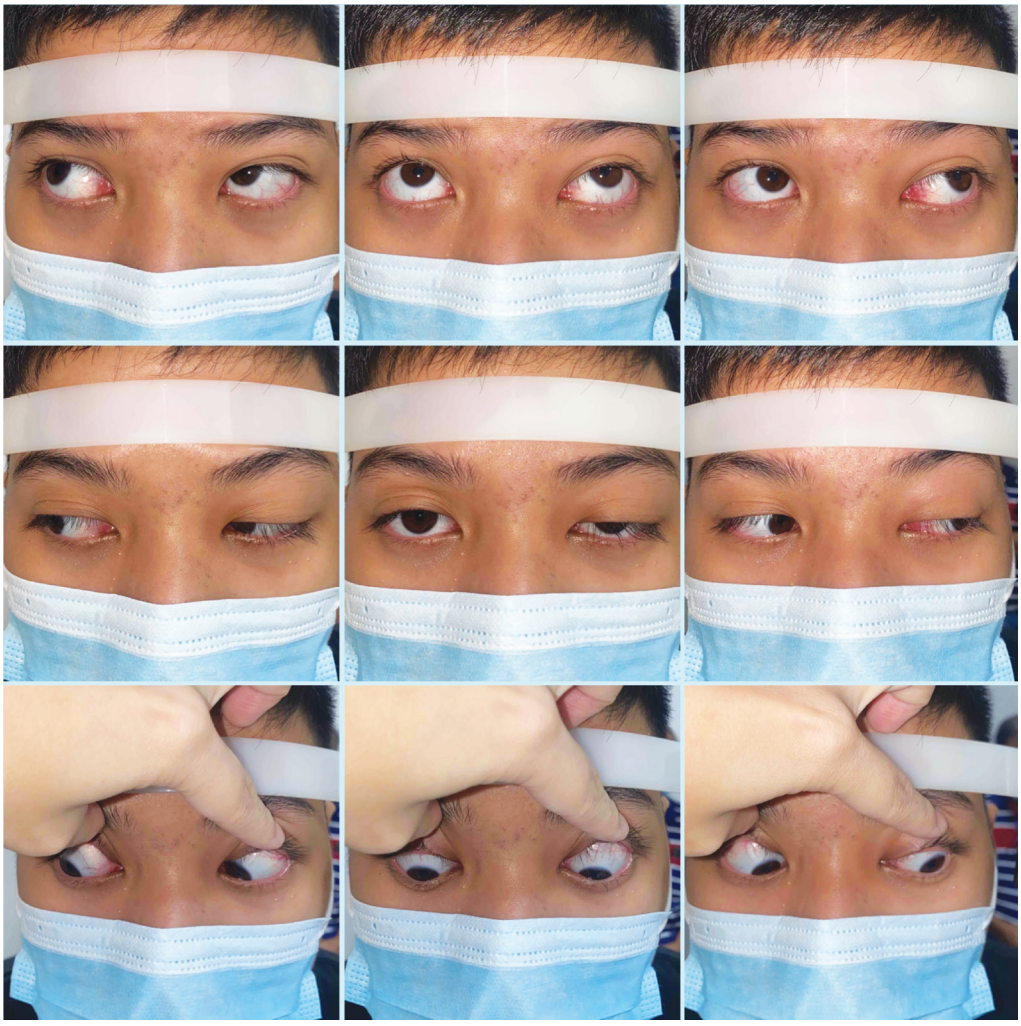

Figure 2 Photographs of the patient. The nine cardinal positions of gaze.

The patient was then referred to the Ear, Nose, and Throat Department after ophthalmic examination because of the suspicion of acute sinusitis. Nasal endoscopy revealed acute bacterial rhinosinusitis, but no nasal polyp, mass, or orbital complication from sinusitis was detected. The patient underwent blood tests, with normal complete blood count and mild elevations of erythrocyte sedimentation rate and high-sensitivity C-reactive protein. He also showed negativity for serum acetylcholine receptor antibodies. Magnetic resonance imaging (MRI) examination of the brain and orbit with contrast was urgently requested to rule out space-occupying lesions and other potential intracranial abnormalities. The MRI revealed no mass, aneurysm, infarction, or hemorrhage; however, it did depict fluid opacification in the left frontal, bilateral ethmoid, and left maxillary sinuses, with no bony erosion. Thin enhancing dura along the left inferior frontal region, adjacent to the left frontal sinus, and mild abnormal enhancing soft-tissue thickening at the superior extraconal space of the left orbit were also noted. There was no evidence of inflammation or injury of bilateral extraocular muscles, including the levator palpebrae superioris (LPS) muscle, and no orbital inflammation or fat stranding were identified (Figure 3).

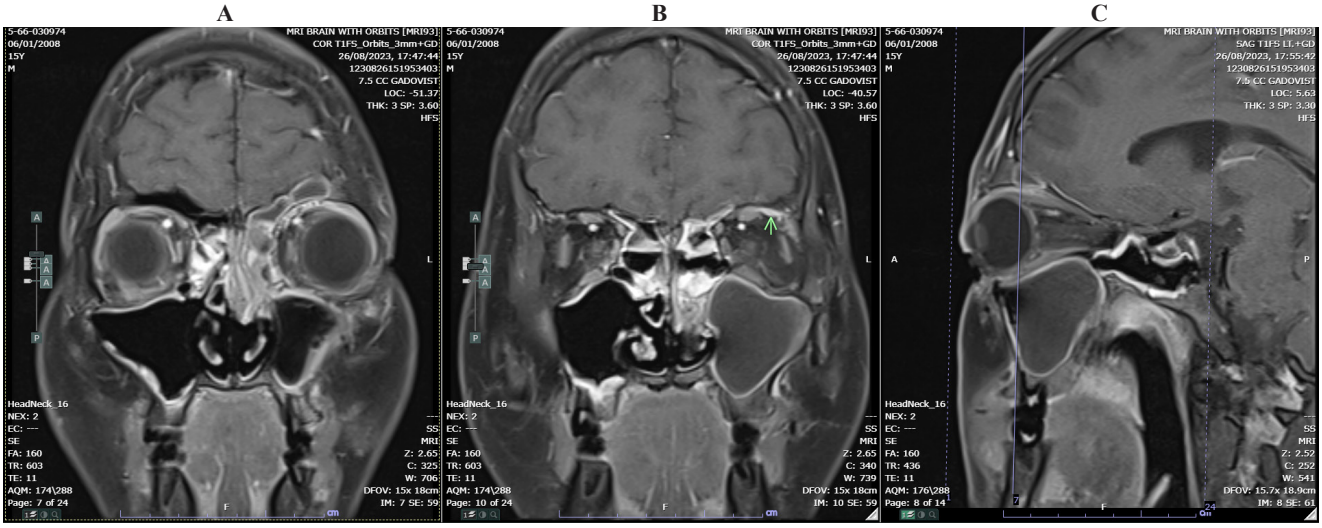

Figure 3 Magnetic resonance imaging (T1 with contrast). (A, B) Coronal views demonstrated left-sided fronto-ethmoidal sinusitis with abnormal contrast enhancing dura along the inferior frontal region and superior extraconal space of the left orbit (arrow). (C) A sagittal view revealed left-sided frontal sinus mucosal thickening with enhancing inflammation.

An otolaryngologist prescribed a once-daily high intravenous dose of levofloxacin (750 mg). The patient achieved complete recovery from the left-sided upper-eyelid ptosis after 5 days of this treatment. In addition, the frontal headache and nasal discharge were ameliorated. The patient was discharged from hospital 7 days after admission.

Discussion

Acquired isolated oculomotor nerve palsy or CN III palsy is typically caused by an aneurysm, brain ischemia, a neoplasm, head trauma, or diabetes mellitus.[5] In addition, Ng et al. proposed that pediatric CN III palsy results most commonly from congenital, traumatic, and tumor-related causes.[2] The proximity

of the facial sinuses and the orbit can lead to orbital complications secondary to sinusitis. However, sinus diseases involving palsy of the superior division of CN III have rarely been reported. Most patients with such palsy have been diagnosed with sphenoid sinusitis.[6-10] To the best of our knowledge, only three cases of palsy of the superior branch of CN III associated with frontal sinus disease have been reported.[4,11,12] Moreover, only one patient has been described as having CN III palsy presenting with only isolated ptosis caused by frontal sinusitis.[4]

Anatomically, the oculomotor nerve or CN III divides into the superior and inferior rami near the anterior cavernous sinus or posterior orbit. Then, the superior ramus passes along the upper part of the lateral wall of the sphenoidal and posterior ethmoidal sinuses.[13] The LPS and superior rectus muscle are supplied by the superior ramus, whereas the medial and inferior recti, inferior oblique, and ciliary ganglion are supplied by the inferior ramus. Clinically, upper-eyelid ptosis and limited supraduction of the eye characterize paresis of the superior division of CN III.

In two case reports, Ajinkya et al.[9] and Yen Nee See et al.[10] stated that isolated ptosis could be explained by partial compression of the superior ramus of CN III by adjacent structures, including inflamed sinuses. The distal branch of this nerve innervates the LPS and superior rectus muscle. Isolated ptosis with no abnormalities of the motility of extraocular muscles can be the result of a compromised LPS supplied by this distal branch. However, both of the above mentioned cases involved patients with sphenoid sinusitis. Meanwhile, Park et al. claimed to have encountered a case in which frontal sinus infection directly spread to the superior orbit through the defective inferior wall of the frontal sinus.[12] In contrast to their findings obtained by computed tomography, no orbital bone erosion or defects were found by MRI in the present case. In addition, their patient presented not only with unilateral ptosis but also with ipsilateral limited supraduction. However, Mirza et al.[4] postulated another mechanism by which isolated ptosis can occur. Specifically, they identified partially localized inflammation of the superior ramus of CN III due to COVID-19 infection. It is thus possible that, in the current case, the patient's influenza A infection could have had similar effects.

Although one report described palsy of the superior ramus of CN III presenting with isolated upper-eyelid ptosis with no ophthalmoplegia, pupillary involvement, or orbital signs from frontal sinusitis, the patient was an adult.[4] With the dramatic resolution of the upper-eyelid ptosis after the initiation of intravenous antibiotics, the authors assumed that the frontal sinusitis found using MRI was the main cause in that case. In addition, the sphenoid sinus was not inflamed in this patient. Given the patient's afebrile status and normal total white blood cell count in the present case, it was supposed that the preceding influenza A infection had caused the frontal sinusitis. MRI was urgently scheduled and performed on this patient to rule out an intracranial cause of the acute ptosis combined with headache, such as intracranial bleeding, the rupture of a posterior communicating artery aneurysm, and mass compression. The MRI also immediately ruled out other concerns, including midbrain infarction and cavernous sinus thrombosis as a complication of sinusitis.

Owing to the absence of facial anhidrosis or pupil constriction, Horner's syndrome could also be ruled out. Another differential diagnosis was myasthenia gravis, but this patient lacked typical myasthenia gravis lid signs, fluctuating weakness, diplopia, and fatigability. Myositis affecting only the LPS was another possible differential diagnosis.[14,15] However, MRI did not reveal any evidence of an inflamed or injured LPS, and the patient was negative for serum acetylcholine receptor antibodies. In addition, he confirmed a lack of any

history of treatment with botulinum toxin on the upper face.

This case should raise awareness of the possibility of sinusitis complications. This case also shows that preceding influenza A infection may contribute to the development of isolated ptosis.

When ptosis is accompanied by other neurological deficits such as ophthalmoplegia, pupillary miosis, or mydriasis suggestive of CN III palsy, Horner's syndrome, imaging of the brain, orbits, or cerebrovascular system should typically be performed on an urgent basis.[16] Computed tomography plays an important role in ruling out serious intracranial abnormalities as a cause of ptosis and achieving a rapid diagnosis of sinusitis when cases have an atypical presentation. However, contrast-enhanced MRI is the imaging modality of choice and provides much more detailed findings of the orbit and its surroundings.

Conclusion

There has been little focus on sinusitis associated with palsy of the superior division of CN III. This report describes an uncommon presentation of a pediatric patient with palsy of the superior branch of CN III presenting with only isolated ptosis caused by frontal sinusitis. It highlights a potential complication of acute sinusitis and raises awareness of a rare differential diagnosis in isolated, unilateral ptosis. Diagnostic neuroimaging may help the management of such atypical cases.

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Ethics Approval

This report was approved by the Institutional Review Board (IRB) of Police General Hospital, Bangkok, Thailand (IRB number: Dh100-66). Informed consent was obtained from the patient and his parents to publish the details of this case. All confidential data were kept in a secured database.

Disclosure

The author declares no conflicts of interest in this work.

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รายงานผู้ป่วย : ภาวะเปลือกตาตกที่เกิดจากโรคไซนัสอักเสบ

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บทคัดย่อ:

วัตถุประสงค์: ภาวะเปลือกตาตกเกิดจากหลายสาเหตุ ได้แก่ เปลือกตาตกหลังอุบัติเหตุ (traumatic) จากการถูกดึงถ่วง (mechanical) จากระบบประสาท (neurogenic) จากภาวะกล้ามเนื้อยกเปลือกตาอ่อนแรง (myogenic) อย่างไรก็ตามภาวะเปลือกตาตกที่เกิดจากประสาทสมองเส้นที่ 3 อัมพาตร่วมกับโพรงไซนัสที่บริเวณหน้าผากอักเสบพบได้น้อยมาก ผู้วิจัยรายงานผู้ป่วยเด็กชายอายุ 15 ปี ที่มีเปลือกตาข้างซ้ายตกจากประสาทสมองเส้นที่ 3 อัมพาต ร่วมกับโพรงไซนัสที่บริเวณหน้าผากอักเสบ

รายงานผู้ป่วย: เด็กชายไทยอายุ 15 ปี มาด้วยเปลือกตาข้างซ้ายตก 2 วัน โดยมีการติดเชื้อไวรัสไข้หวัดใหญ่สายพันธุ์เอนำมาก่อน 1 สัปดาห์ การตรวจการเคลื่อนไหวของลูกตาและรูม่านตาปกติ ผลการตรวจด้วยคลื่นแม่เหล็กไฟฟ้าบริเวณสมองและเบ้าตาพบโพรงไซนัสอักเสบและเนื้อเยื่อรอบๆ บวมโดยเฉพาะโพรงไซนัสหน้าผากด้านซ้าย โดยไม่พบความผิดปกติที่กล้ามเนื้อลืมตา (levator palpabrae superioris) ผู้ป่วยได้รับการวินิจฉัยว่าเป็นประสาทสมองเส้นที่ 3 อัมพาต ร่วมกับโพรงไซนัสที่บริเวณหน้าผากอักเสบ ภาวะเปลือกตาตกหายหลังผู้ป่วยได้รับยาปฏิชีวนะลีโวฟลอกซาซินทางเส้นเลือดเป็นเวลา 5 วัน

สรุป: โพรงไซนัสที่บริเวณหน้าผากอักเสบก่อให้เกิดประสาทสมองเส้นที่ 3 อัมพาตได้ ซึ่งอาจมาด้วยภาวะเปลือกตาตกอย่างเดียว กรณีศึกษานี้ชี้ให้เห็นถึงผลแทรกซ้อนของโพรงไซนัสอักเสบและความสำคัญในการวินิจฉัยผู้ป่วยที่มาด้วยเปลือกตาตกข้างเดียว ซึ่งการตรวจด้วยคลื่นแม่เหล็กไฟฟ้าบริเวณสมองและเบ้าตาสามารถช่วยในการวินิจฉัยในกรณีศึกษานี้

คำสำคัญ: ประสาทสมองเส้นที่ 3, อัมพาต, ประสาทกล้ามเนื้อตาอัมพาต, เด็ก, เปลือกตาตก, ไซนัสอักเสบ

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