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Isolated Third Nerve Cranial Palsy: Complication of Brain Aneurysm

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Abstract

This case report highlights a critical and emergent condition, isolated third nerve cranial palsy due to a brain aneurysm. It emphasizes the importance of differential diagnosis and attentiveness to the physical exam in an emergency setting. The detailed progression from initial symptoms and misinterpretation to final diagnosis offers valuable insight into the dangers of overlooking critical diagnoses. A bilobed intracranial aneurysm arising from the internal carotid artery (ICA) caused symptoms in this patient that could have been attributed to a less malignant etiology, such as a complex migraine. Overlooking the urgency of the situation and missing the diagnosis could have had a grave and irreversible outcome.

Keywords

Migraine, Brain Aneurysm

1. Introduction

Etiologies of oculomotor nerve palsies include structural lesions, cerebrovascular disease, infection/inflammation, and trauma, and vary depending on the location of the insult. Differential diagnoses for 3rd cranial nerve palsy may involve ophthalmic migraine, infection, tumor, brain aneurysm, hemorrhage, or blood flow obstruction to the nerve. The most feared cause of third nerve palsy is compression by an enlarging intracranial aneurysm, commonly located at the posterior communicating artery, though aneurysms at the internal carotid artery and the basilar artery can also induce third nerve palsies [1] [2]. Third nerve palsy resulting from an acutely enlarging intracranial aneurysm represents a surgical emergency due to the imminent risk of rupture and subarachnoid hemorrhage, which can occur within hours to days from the initial palsy signs [3]. About

25% of patients with a ruptured brain aneurysm die within 24 hours, and 50% die within 3 months of rupture due to complications. Patients with acute oculomotor nerve palsy report sudden onset of binocular horizontal, vertical, or oblique diplopia and ptosis, commonly accompanied by pain, except in midbrain lesions, and exhibit clear mydriasis [4] [5]. Chronic oculomotor nerve palsy may present asymptomatically. Imaging studies for diagnosing or ruling out an intracranial aneurysm include contrast-enhanced MRI with MRA, CTA, and cerebral angiography, the latter being the gold standard despite potential procedural complications [6] [7]. The relative risk of an underlying aneurysm is assessed based on the extent of external and internal third nerve dysfunction.

2. Case

A 51-year-old female with a history of migraines presented to the emergency department (ED) with an acute onset of isolated right third cranial nerve palsy. She had visited the same ED about two and a half weeks earlier with complaints of worsening headaches lasting a week. At that time, blood serology was unremarkable, and a non-contrast CT scan of the head showed no intracranial abnormalities. She received treatment with a saline bolus, Toradol, Benadryl, Decadron, Tylenol, and Imitrex, which initially had a good effect, but her symptoms did not completely resolve. She was advised to see a neurologist and was prescribed Topamax, which she discontinued after two days due to nausea, gastrointestinal distress, eye pain, pupillary dilation, and anxiety. Two days after stopping Topamax, she experienced a pulling sensation in her eyelid, described as a muscle spasm, and complained of right upper eyelid twitching for the rest of the week. Two nights before her return to the emergency room, her husband noticed her right pupil was fixed and dilated. The next morning, she had complete paralysis of the upper eyelid. An ophthalmologist performed a dilated optic disk exam of the retina and vessels, which were normal. The same ophthalmologist referred the patient to the ED for further investigation of the isolated right third cranial nerve palsy. On her second ED visit, her temperature was 98˚F, she was hemodynamically stable with a heart rate of 98 beats per minute in normal sinus rhythm and blood pressure of 154/63. She was not in acute respiratory distress, with a respiratory rate of 18 and SpO_2 of 98% on room air. The patient reported blurred vision in her right eye but denied eye pain, migraine headaches, rhinorrhea, sinus pain, facial asymmetry, or diaphoresis. Neurological examination confirmed isolated right third nerve cranial palsy. She could not adduct the right eye, had intact abduction, could extort the eye, but intorsion was not intact. Her pupil was fixed and dilated, with no conjunctival injection, and the globe was non-traumatic without periorbital ecchymosis. An MRI of the brain showed several focal non-enhancing T2 signal abnormalities in the periventricular, deep white matter, and subcortical white matter of the cerebral hemispheres bilaterally, suggesting possibilities of a demyelinating process, vasculopathy, or gliosis from prior trauma. An MRA of the brain revealed a bilobed aneurysm (5 mm, 4

mm) arising from the supraclinoid segment of the right Internal Carotid Artery. The patient was immediately transferred for neurosurgical intervention to a tertiary care center. The diagnosis post-procedure was multilobulated Right Supraclinoid ICA Aneurysm.

3. Discussion

Isolated third nerve palsies often indicate a lesion within the subarachnoid space. Causes include ischemia, aneurysmal compression, infection, inflammation, neoplasia, uncal herniation, and trauma, which may arise from neurosurgical procedures.

Patients with acute oculomotor nerve palsy often report sudden onset of binocular horizontal, vertical, or oblique diplopia and ptosis. Pain is common at the onset of acute palsy, except in cases involving midbrain lesions. Mydriasis may also be present. In contrast, chronic oculomotor nerve palsy may be asymptomatic [3] [4].

Our patient had complete non-pupil sparing third nerve palsy, including ptosis, a fixed dilated pupil, and paralysis of adduction, elevation, depression, and extortion. In this case, the eye's resting position is abduction, intorsion, and slight depression.

Etiologies of oculomotor nerve palsies include structural lesions, cerebrovascular disease, infection/inflammation, and trauma, specific to the location of the insult [3].

The most feared cause of third nerve palsy is compression by an enlarging intracranial aneurysm. The most common site for an aneurysm is the posterior communicating artery, though aneurysms involving the internal carotid artery and the basilar artery have also been reported to cause third nerve palsies [2] [8] [9] [10].

Third nerve palsy resulting from compression by an acutely enlarging intracranial aneurysm is a surgical emergency. This condition poses an imminent risk of rupture and subarachnoid hemorrhage within hours to days after the initial onset of palsy signs [5] [8] [9] [10].

Ischemic third nerve palsies, also known as diabetic third nerve palsies, are the most common cause in adults. The underlying mechanism is believed to be microvascular. Although some isolated ischemic third nerve palsies result from midbrain infarction, most are peripheral.

Most pupillomotor fibers are believed to be located along the superficial periphery of the oculomotor nerve fascicle and remain unaffected in cases of oculomotor palsy due to ischemia. Thus, patients with ischemic third nerve lesions usually maintain intact pupillary function.

However, when aneurysmal compression causes the palsy, the oculomotor nerve fascicle's superficial periphery is the first to be affected. In this case, the pupil will be dilated and unresponsive. Rarely, aneurysms can present without affecting the pupil, occurring when the extraocular muscles are incompletely involved [11] [12].

Traumatic third nerve palsies can result from head injuries with skull fractures. Cranial neuralgia can also lead to third nerve palsy, and sometimes the deficits are permanent. Several reports have shown MRI enhancement of the cisterna in the region traversed by the oculomotor nerve, suggesting a recurrent demyelinating neuropathic pathogenesis [4].

For patients with symptoms, MRI suggests a lesion affecting the nerve's nuclear or fascicular course within the brainstem, including bilateral ptosis, contralateral superior rectus palsy, hemiparesis, ataxia, and tremor.

If meningeal signs or additional cranial nerve deficits are present, especially when bilateral, a lumbar puncture (LP) should be obtained to evaluate for meningitis. The sudden onset of painful oculomotor nerve palsy with associated meningeal signs necessitates an emergent evaluation with non-contrast CT. If the head CT is negative, an LP should be performed to definitively exclude a subarachnoid hemorrhage. In the absence of subarachnoid hemorrhage, imaging should be ordered to exclude an unruptured intracranial aneurysm.

Imaging studies used to diagnose or exclude an intracranial aneurysm include contrast-enhanced MRI with MRA, CTA, and the more invasive cerebral angiography, which is the gold standard diagnostic test. The risk of complications from catheter angiography must be weighed against the risk of missing an aneurysm on an individual basis. This risk depends on institution-specific factors, as well as patient age and comorbidities. The relative risk of an underlying aneurysm is assessed based on the degree of external and internal third nerve dysfunction.

Signs that localize to the cavernous sinus or orbital apex merit an MRI with gadolinium.

Lesions of the third nerve in the cavernous sinus and superior orbital fissure often involve other cranial nerves, including the trochlear nerve, abducens nerve, V1 branch of the trigeminal nerve, and oculo-sympathetic fibers. Lesions within the orbit present with orbital signs such as optic neuropathy, conjunctival injection, and proptosis [3] [5].

Complete internal dysfunction, also known as pupil-involved third nerve palsy, should be presumed to result from aneurysmal compression until proven otherwise [10] [12]. Patients are advised to undergo MRI and MRA (or CTA), but even if these noninvasive studies are negative, a catheter angiogram is strongly recommended to rule out an aneurysm [6] [7].

Once an aneurysm and other mass lesions have been excluded, older patients should be evaluated for giant cell arteritis, and a lumbar puncture (LP) should be considered for those with persistent or worsening deficits when the cause remains unclear.

Other patterns of isolated third nerve deficits include incomplete external dysfunction with no pupil involvement (e.g., divisional palsy) and incomplete or complete external dysfunction with partial internal dysfunction (relative pupil-sparing). Patients should undergo a contrast-enhanced brain MRI to rule out mass lesions, with an MRA or CTA to check for an aneurysm or other mass

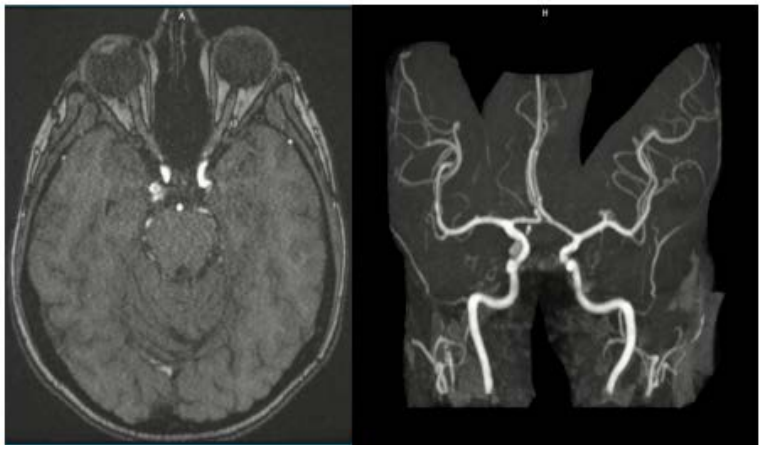

Figure 1. Bilobed Aneurysm arising from R. Supraclinoid segment.

lesions. If negative, catheter angiography should be considered to further investigate the presence of an aneurysm or the less likely possibility of a posterior draining carotid-cavernous sinus fistula. In this intermediate risk category, the choice of testing depends on individual and institution-specific risk-benefit assessments.

A neurologically isolated third nerve palsy with a normal pupillary sphincter and completely paralyzed extraocular muscles (complete external dysfunction) is almost never caused by an aneurysm [11].

In older adults, this presentation is most often caused by ischemic injury. Observation alone is an appropriate diagnostic option for older patients with vascular risk factors (hypertension, diabetes mellitus type II). However, contrast-enhanced MRI and MRA of the brain should be strongly considered in patients without vascular risk factors whose deficits progress or do not improve within six to twelve weeks of follow-up, or in those with signs of aberrant regeneration.

4. Conclusion

4.1. Diagnosis: Multilobulated Right Supraclinoid ICA Aneurysm

An MRI of the brain demonstrated several, focal non enhancing T2 signal abnormalities in the periventricular, deep white matter and subcortical white matter of the cerebral hemispheres bilaterally. The radiologist's differential diagnosis includes a demyelinating process, vasculopathy, or gliosis from prior trauma.

An MRA of the brain was performed and demonstrated a bilobed aneurysm, (5 mm, 4 mm) originating from the supraclinoid segment of the right Internal Carotid Artery (Figure 1). The patient was immediately transferred to a tertiary care center for neurosurgical intervention and definitive treatment.

The patient underwent successful coil embolization of this aneurysm, performed by a neurosurgeon.

On post-operative exam, the patient was extubated and had full strength in all extremities.

4.2. Teaching Points

1) Always remember that a headache can be a sign of a dangerous intracranial process, even in patients with a history of migraines.

2) If the patient's pain does not fully resolve, especially after multiple medications, reassess your differential diagnosis and reconsider your imaging modalities.

3) Neurosurgical repair is the definitive treatment for brain aneurysms.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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