

# รายงานผู้ป่วย: เส้นประสาทสมองที่สามเป็นอัมพาตเนื่องจากภาวะหลอดเลือดคาโรติดโป่งพอง

## A case report: Pupil involving third nerve palsy secondary to ICA aneurysm

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### บทคัดย่อ

ภาวะอัมพาตประสาทสมองเส้นที่สามร่วมกับมีการตอบสนองของรูม่านตาผิดปกติมักมีสาเหตุที่พบได้บ่อยจากการกดทับเนื่องจากลักษณะการเรียงตัวของเส้นใยประสาทที่ทำหน้าที่ตอบสนองของรูม่านตา ภาวะหลอดเลือดแดงโป่งพองเป็นสาเหตุกดทับเส้นประสาทสมองที่สามารถทำให้เกิดอันตรายถึงชีวิตได้ การตรวจร่างกายเพื่อแยกว่าการตอบสนองของรูม่านตาผิดปกติหรือไม่จึงมีความสำคัญทางคลินิก รายงานผู้ป่วยหญิงไทย อายุ 51 ปีมาด้วยอาการหนังตาตกเป็นมากขึ้น ปวดและเห็นภาพซ้อน 3 สัปดาห์ ตรวจตาพบหนังตาขาดมากบังการมองเห็น ไม่สามารถกลอกตาขึ้นบน ลงล่างหรือกลอกเข้าในได้และรูม่านตาขยายขนาดผิดปกติ ไม่ตอบสนองต่อแสงในตาข้างขวา การตรวจร่างกายทางระบบประสาทไม่พบอัมพาตของเส้นประสาทสมองเส้นอื่น ผลตรวจเอกซเรย์คลื่นแม่เหล็กไฟฟ้าสมองและเบ้าตาไม่พบความผิดปกติ ผลตรวจเอกซเรย์คอมพิวเตอร์หลอดเลือดสมองพบหลอดเลือดคาโรติดด้านขวาโป่งพอง ผู้ป่วยได้รับการผ่าตัดวิธีใส่คลิปหนีบที่บริเวณฐานของหลอดเลือดโป่งพองเป็นผลสำเร็จและได้รับการตรวจติดตามเพื่อดูการกลับมาทำงานของเส้นประสาทหลังการรักษา

**คำสำคัญ:** อัมพาตประสาทสมองเส้นที่สาม, หลอดเลือดโป่งพอง, รูม่านตาผิดปกติ

### Abstract

Pupil involving third nerve palsies are commonly related to compressive lesions due to the arrangement of the pupilo-motor fibers of the nerve. Aneurysmal compression can cause life-threatening complications. Distinguishing between pupil involving and pupil sparing third nerve palsies are of great clinical importance. We present a 51 -year-old healthy female complaining of worsening ptosis and painful diplopia for about 3 weeks. Eye examinations

revealed a complete ptosis, limitation of supraduction, infraduction, adduction, and complete mydriasis in the right eye. Neurological examinations revealed no involvement of other cranial nerves. MRI brain and orbit studies were unremarkable. CTA brain revealed a saccular aneurysm from the right internal carotid artery. The aneurysm was successfully treated by a clipping procedure. The patient was followed-up for the recovery of third cranial nerve function.

**Keywords:** Third nerve palsy, Aneurysm, Pupil involving

### Introduction

The third cranial nerve innervates both extraocular muscles (medial rectus, superior rectus, inferior rectus, inferior oblique, and levator palpebrae superioris) and autonomic parasympathetic muscles (ciliary and iris sphincter). Acquired third nerve palsies can present with ptosis, limitation of adduction, supraduction, infraduction and/or pupillary dysfunction. Pupillary dysfunction ranges from varying degrees of pupil dilation to complete mydriasis. Symptoms and signs depend on the affected location of the path between its nucleus in the midbrain and the innervated muscles in the orbit.

On the course in the subarachnoid space, the pupillomotor fibers are located on the dorsomedial surface and more superficial portion of the third nerve, which are susceptible to any compressive lesions. The common etiology is an aneurysm arising from the internal carotid artery (ICA) at the origin of the posterior communicating artery (PCoA) because the PCoA and third nerve are juxtaposed in the subarachnoid space of basal cistern, prior to the nerve entering the cavernous sinus.<sup>1,2</sup>

The majority of third nerve palsy patients suffer pain from first division trigeminal nerve distribution, and the patient may complain of pain for months prior to clinical findings. Thus, if the patient presents with pupil-involving third nerve palsy associated with significant pain in the ipsilateral orbital or frontal area, it will require immediate evaluation for potential aneurysms prior to the occurrence of rupture from aneurysm-related subarachnoid hemorrhage.

### Case Report

A 51-year-old healthy female presented with progressive right ptosis for about 2 weeks. She had new onset of headache, ocular pain, and binocular diplopia for the previous 3 weeks. There was no significant medical and trauma history. The patient underwent magnetic resonance imaging (MRI) of the brain and orbit at a local hospital before being referred to our hospital. Initial best corrected visual acuity was 20/30 OD and 20/20 OS. Intraocular pressures measured 14 mmHg OD and OS. External eye examination revealed a complete right ptosis (Figure 1). Cover testing revealed a constant 90 prism-diopter right exotropia at near and distance. Ocular motility examination revealed a complete deficit in adduction, supraduction, and infraduction in her right eye (Figure 2). Forced duction test was revealed negative. The right pupil was fixed-dilated, non-reactive to light and without reverse afferent pupillary defect. The left pupil was 3 mm in size with normal light reaction. Slit-lamp and dilated fundus examinations revealed no abnormalities of anterior segment and posterior segment in her both eyes. Neurological examinations revealed no involvement of other cranial nerves.



Figure 1 shows complete ptosis in the right eye.



Figure 2 shows ocular motility pattern of complete third nerve palsy with pupil involvement.

This case was diagnosed as an isolated complete right third nerve palsy with pupil involvement. A review of brain and orbit MRI revealed no compressive intracranial lesions. Based on the pupil characteristics of this patient, computerized tomographic angiography (CTA) of the brain was done to exclude the possibility of aneurysm. The CTA brain showed a lobulated saccular aneurysm measuring 0.4x0.8x0.8 cm, arising from the supraclinoid part of right ICA with a postero-inferior fundus point (Figure 3). There was no evidence of aneurysm rupture. The CTA findings in this patient were consistent with the diagnosis of right internal carotid artery aneurysm.

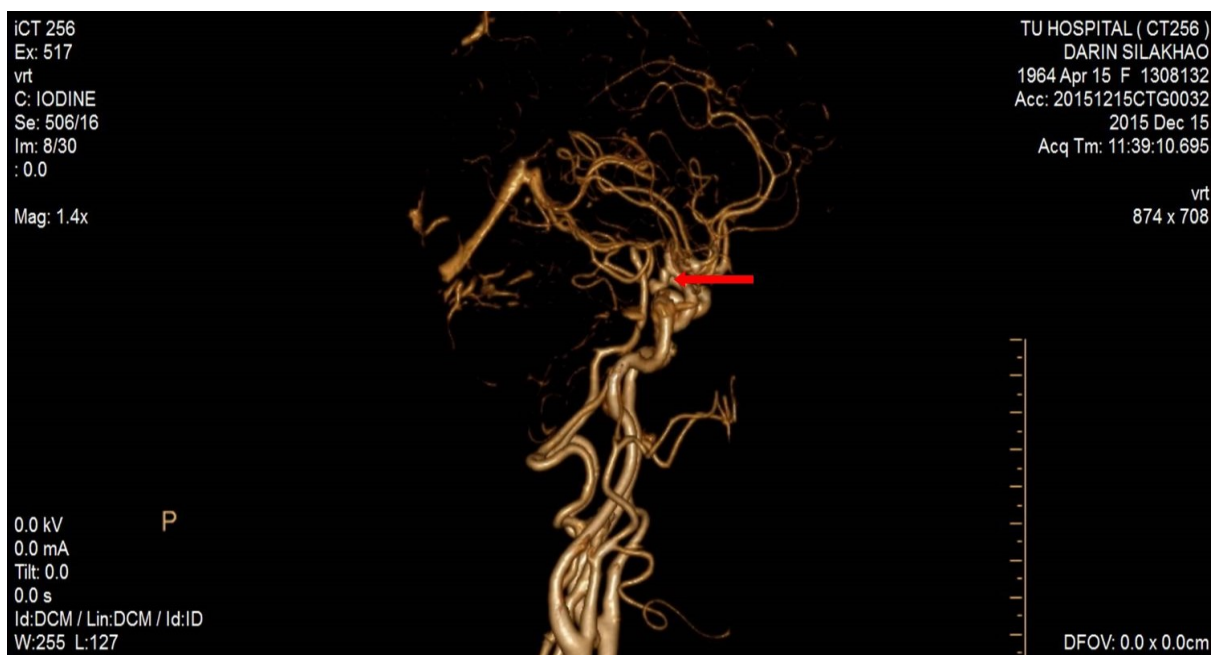


Figure 3 shows lateral view of CTA; a lobulated saccular aneurysm (red arrow), 0.4 cm x 0.8 cm size, postero-inferior to the-supraclinoid part of the right ICA.

The patient was consulted by a neurosurgeon and underwent a surgical clipping procedure. The intraoperative findings showed the aneurysm compressed on the right third nerve. There were no intra or postoperative complications. The clinical symptoms were improved. The patient did not complain of headache or ocular pain. On follow-up at one month, the eye examination revealed complete right ptosis and motility deficit in the same amount. The author recommends that the patient undergo regular follow-up to assess recovery of the third nerve function, because the recovery following aneurysmal clipping takes months to occur.

## Discussion

The third nerve nuclei complexes are located in the midbrain, it projects axons to form fascicles within the brainstem. After exiting the brainstem, the third nerve travels adjacent to the PCoA in the subarachnoid space. When entering the cavernous sinus, the third nerve lies above the fourth and sixth nerves and near the trigeminal and sympathetic nerve fibers. The nerve then bifurcates at the anterior cavernous sinus into superior and inferior division before entering the orbit via the superior orbital fissure to innervate the extraocular muscles and parasympathetic muscle as introduced earlier. Lesions in the cavernous sinus part and orbital part usually presents with multiple cranial nerves palsies rather than isolated nerve palsy. Lesions in the subarachnoid part usually present with isolated third nerve palsy. In the subarachnoid part, The pupillomotor fibers are located more superficially than the central fibers, making them more susceptible to aneurysmal compression.<sup>3</sup> In contrast, the blood supply of the vasa nervorum to the nerve affects the central fibers, sparing the superficial pupillary fibers. The “rule of the pupil” states that normal pupillary function accompanying third nerve palsy is commonly related with vascular etiologies such as diabetes or hypertension.<sup>4</sup> In contrast, pupillary involvement accompanying third nerve palsy is commonly related with compressive etiologies such as aneurysms. This case also highlights the importance of localizing the anatomy, using the appropriate imaging in the management of third nerve palsy.

The management of the third nerve palsy varies according to the age of the patient, the pupil characteristics of the third nerve palsy, and the presence of associated signs and symptoms. Practically, when a patient older than 50 years of age presents with pupil-sparing isolated third nerve palsy, we should investigate for microvascular etiologies and observe closely, because microvascular third nerve palsy usually show improvements within 2-3 months. Neuroimaging should be performed if the patient shows no improvement within that time period, neurological deficit, or other cranial nerve involvement. When adults less than 50 years of age present with isolated third nerve palsy, we should investigate for compressive etiologies and neuroimaging should be performed even for pupil-sparing cases, because compressive third nerve palsy usually occur in this age range.<sup>5</sup> In this case, the history of progressive symptoms, the pupillary involvement, and lack of microvascular risk factor, contributed to the immediate investigations. MRI is the imaging of choice to rule out

non-vascular compressive lesions. In this case, the MRI showed no compressive intracranial lesions. In detecting aneurysms, the cerebral arteriography (CA) is the gold standard diagnostic procedure. However, the use of CA is associated with the possible risk of stroke or death in one to two percent of cases. CTA and magnetic resonance angiography (MRA) are reliable non-invasive vascular imaging which is safer than CA for detecting aneurysms greater than 3-4 mm in size.<sup>6,7</sup> Modern CTA and MRA have increased their sensitivities in detecting intracranial aneurysm.<sup>8</sup> The sensitivity for aneurysms above 4 mm is 95% in CTA and 93%-97% in MRA<sup>9,10</sup> CTA is now viewed as a first line procedure, except in children and pregnant women should undergo MRA to avoid the radiation exposure of CTA. In the present case, the clinical presentations do not support a microvascular ischemic cause and the MRI had been performed to exclude a non-aneurysmal cause, hence this is the rationale for performing CTA to exclude aneurysms causing third nerve palsy. Therefore, patients whom the clinical suspicion of aneurysm is high should undergo vascular imaging.

Intracranial aneurysms can contribute from 18% to 40% of isolated non-traumatic third nerve palsies. Aneurysms involving the junction of ICA and PCoA has commonly been observed up to a rate of 89%.<sup>10</sup> Aneurysmal-related third nerve palsy had complete palsy at 1 week in about 66% of cases. It is frequently associated with ipsilateral facial pain in about 64% of cases, which has been attributed to the compression of pain sensory afferent fibers from the ophthalmic division of the trigeminal nerve that lies within the periphery of the third nerve.<sup>11</sup> Painful third nerve dysfunction may indicate local compression of the aneurysm and the frequency of rupture of an intracranial aneurysm has been reported in approximately 50% of cases.<sup>12</sup> Thus, a third nerve dysfunction requires immediate evaluation and treatment prior to the occurrence of a subarachnoid hemorrhage in association with aneurysmal rupture. Fortunately, the unruptured aneurysm in this patient was successfully treated by a clipping procedure. The patient should be followed-up for the recovery of third nerve function for several months. Resolution of ptosis is usually the first sign of improvement, whereas ocular motility and pupillary dysfunction frequently persist.

### Conclusion

Evaluation of acquired third nerve palsies depend on symptoms, sign, age, and underlying systemic disease. The author presents a case of pupil involving painful third nerve

palsy; which should be considered to have an aneurysm until proven otherwise. Distinguishing between pupil involving and pupil sparing third nerve palsy is of great clinical importance for appropriate investigation and further management. Although the MRI was negative, in light of the manifested clinical signs, the repeated CTA can aid in diagnosis. Similar to CTA, MRA can detect aneurysms more than 4 mm in diameter. These features suggest that cerebral MRI should be performed with MRA for the detection of compressive lesions.

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