PARTIAL CRANIAL NERVE III PALSY DUE TO A POSTERIOR COMMUNICATING ARTERY ANEURYSM: A CASE REPORT

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INTRODUCTION

Cranial nerve III (CN III), the Oculomotor nerve, has central motor fibers, which innervate the levator palpebrae and all the extraocular muscles besides for the superior oblique and the lateral rectus, and outer parasympathetic fibers that innervate the the sphincter pupillae and the ciliary muscle. With a complete CN III palsy, the loss of tone in these muscles classically presents as a unilateral ptosis, mydriasis, and a "down and out" gaze. However, when it is a partial CN III palsy, only a few of the muscles are affected. When the palsy affects the motor fibers and spares the parasympathetic fibers, it is classified as "pupil sparing", because there is ptosis and a "down and out" gaze with no mydriasis. When it affects the parasympathetic fibers and not the motor, it is classified as "non-pupil sparing", because there is mydriasis. "Pupil sparing" occurs from ischemic lesions affecting the central motor fibers, such as from hypertension and diabetes, and "non-pupil sparing" occurs from compression lesions, pressing on the outer parasympathetic fibers.

When there is a "non-pupil sparing" partial CN III palsy, compression from a posterior communicating artery (PCOM) aneurysm must be ruled out. If this aneurysm were to rupture, the prognosis is very poor, with 30% of patients dying outside the hospital, 15% of hospitalized patients who received treatment dying, and those who do survive often have functional disorder, whether it be a hemiplegia or an aphasia.

TIMELINE AND INTERVENTION

Day 1
- 58-year-old African American Male with a PMH of ESRD on dialysis, malignant HTN, CHF, asthma and arthritis presents to the ED for worsening shortness of breath over the past month.
- Vitals: 36.6°C, 109 BPM, 26 RR, 194/124.
- He was hemodialyzed the same day after not receiving any dialysis for the last 5 months. He had limited confusion and CT of the head was done and found to be negative.

Day 2
- Vitals: 36.7°C, 74 BPM, 18 RR, 130/73.

Day 8
- Ophthalmology was consulted due to acute ptosis.
- Neurology was consulted for imaging
- MRI showed multiple scattered punctate foci within frontoparietal lobes

Day 9
- MRA of the head and neck showed a posterior communicating artery aneurysm and the patient was transferred for a catheter angiogram.

EXAMINATION AND DIAGNOSIS

Ophthalmologic Exam: The ophthalmology team was consulted on hospital day 8, due to an acute left ptosis. The patient stated that the ptosis occurred 1 week prior and that he has blurred vision in the same eye. A full non-dilated exam was performed with a focus on acute ptosis. Visual acuity was 20/100 OU and pinholed to 20/70 OU. Pupils were round, reactive, with no rAPD, but were unequal in size, OS>OD. Color vision was full OU, extraocular muscles were full in all directions OU, and visual fields were full OU. Sclerae were white and quiet, corneas were clear, anterior chambers were well formed, irises showed no defects, and the lenses had nuclear sclerotic cataracts. There was no evidence of proptosis, ecchymoses, or edema, but there was a 3-4 mm ptosis on the left side.

Due to the ptosis also presenting with anisocoria, the exam focused primarily on the anisocoria (unequal pupil size) following an algorithm (Figure 1). In light, the patient's right and left pupils were 3 mm and 4.5 mm, respectively. In the dark, the patient's right and left pupils were 5 mm and 6 mm, respectively. Upon addition of 1% pilocarpine in the left eye, the left pupil constricted. This algorithm led to the diagnosis of a CN III palsy, in this patient. Due to the extraocular muscles being intact, the patient had a "non-pupil sparing" partial CN III palsy rather than a complete CN III palsy. Due to the "non-pupil sparing" nature of it, the underlying pathology was most likely due to a compressive lesion. With compressive lesions, a PCOM aneurysm must be ruled out. The ophthalmology team recommended an MRI and an MRA and consulted neurology.

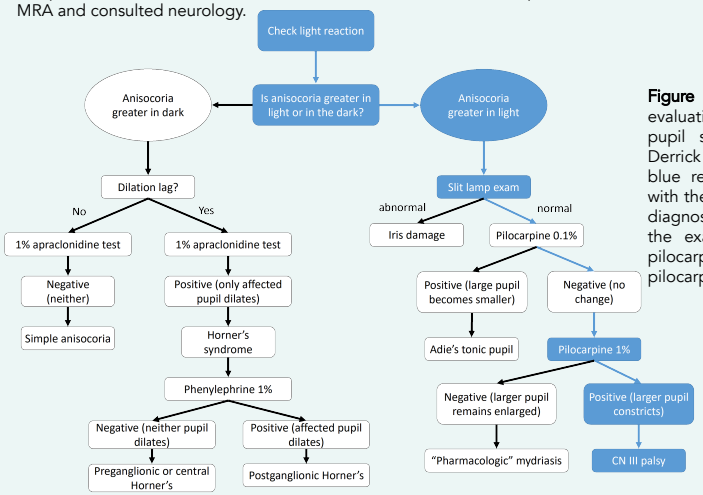

Figure 1: a flowsheet for the evaluation of anisocoria (unequal pupil sizes) (Modified from Smit, Derrick P.) The steps highlighted in blue represents the path we took with the flowsheet to get to our final diagnosis of a CN III palsy. During the exam, we did not use 0.1% pilocarpine and went straight to 1% pilocarpine.

MRI and MRA of Head and Neck: Neurology was consulted for an MRI and and MRA of the head to rule out a possible PCOM aneurysm. The MRI showed multiple scattered punctate foci within frontoparietal lobes. The MRA showed a left PCOM aneurysm that measured 5 x 4 mm (Figure 2). The patient was transferred for a catheter angiogram.

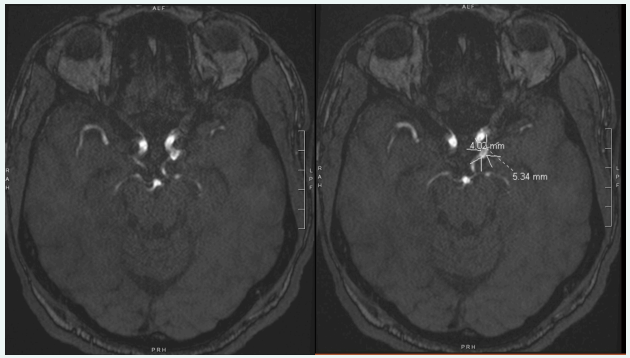

Figure 2: the MRA that was done on hospital day 9, with and without the measurements. The MRA findings were consistent with a PCOM aneurysm that measured 5 x 4 mm.

DISCUSSION

CN III palsy may be uncommon, affecting up to 4 in every 100,000 individuals per year, it is an important concept to understand due to the array of conditions that can induce it. These conditions can range from hypertension to something deadly, such as a brain aneurysm. Due to the potential of a CN III palsy being caused by a deadly condition, it is important to be able to differentiate a non-deadly cause from a deadly one. An important step in determining the pathophysiology of a CN III palsy is by determining if it is "pupil-sparing" or "non-pupil sparing". If the CN III palsy is "non-pupil sparing", the algorithm for anisocoria ("non-pupil sparing") (Figure 1) can be used to determine if the anisocoria is from something as harmless as a physiologic variant or as deadly as a compressive lesion from a PCOM aneurysm.

This case showcased the anisocoria algorithm; one that many have not used in practice. This algorithm is a very effective way to determine the cause of an anisocoria and even if you do not have to use it in practice, understanding each step will play two major roles as a practicing physician. The first role would be to be able to diagnose the cause of an anisocoria. The second role is to understand the pathophysiology of the different causes of an anisocoria. The first step in this algorithm is comparing the pupil sizes in light and in dark to determine if the anisocoria is from a constricted pupil or a dilated one. The pupils dilate in darkness and constrict in light. If the pupil is unable to constrict, it will be dilated in light, which will even out with the other pupil in the dark. The opposite is true when there is a pupil unable to dilate. Dilated pupils are caused by lesions affecting the sphincter pupillae muscle through CN III's parasympathetic fibers and can be caused by an aneurysm. This was the case with this patient whose anisocoria had a difference of 1.5 mm in light and 1 mm in the dark, due to a PCOM aneurysm. Constricted pupils are caused by lesions affecting the pupillary dilator muscle through the sympathetic nervous system, such as from Horner's syndrome. The sphincter pupillae muscle is mostly innervated by M3 receptors, so when pilocarpine (a muscarinic agonist) is used, the muscle will constrict, making the pupil smaller. If the pupil is dilated because an antimuscarinic was used, the pupil will not constrict because the pilocarpine is unable to overcome the antimuscarinic action. If the pupil is dilated due to a CN III palsy, the dilation is from the muscle not being innervated, so when a muscarinic agonist is added, the pupil will constrict.

Conclusion

The clinical implications of CN III palsy do have drastic variations with some being benign and others carrying detrimental outcomes if missed. Our case shows the importance of taking a stepwise (algorithmic) approach to this often-difficult clinical presentation in order to determine which patients have the factors that may suggest more and thus to be able to properly work up and treat those patients.

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