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CASE REPORT

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Pupil-sparing cranial nerve III palsy after intranasal cocaine treatment for cluster headache

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Abstract

This case report discusses a cranial nerve III palsy in a 47-year-old, type II diabetic man that originally presented with a cluster headache that was treated with 100% oxygen by nasal cannula, 975 mg Tylenol, and 100 mg of Imitrex without any symptom relief. He then received a sphenopalatine fossa block using 1 ampule of 4% cocaine. Three days after medicinal, intranasal cocaine for treatment of a cluster headache, the patient presented with a cranial nerve III palsy with spontaneous resolution in 4 months without any intervention. Previously, intranasal cocaine had been standard treatment for cluster headaches; however, recently lidocaine has come into favor for the sphenopalatine block. Intranasal cocaine has been associated with cerebrovascular accidents, even in young adults. The time from cocaine use to cerebrovascular accident can range from hours to years. This known side effect of intranasal cocaine in young, otherwise healthy individuals should be considered when this medication is being used to treat headache, especially in higher risk patients. Therefore, when considering a sphenopalatine nerve block for treatment of headaches, both cluster and migraine, lidocaine should be the preferential treatment over intra-nasal cocaine.

KEYWORDS

cocaine, cranial nerve III palsy, cluster headache treatment, oculomotor nerve

1 | INTRODUCTION

Patients usually present with complaints of binocular diplopia and eyelid dysfunction (drooping, inability to completely close the eye, etc) and, depending on the etiology of the lesion, may have other associated complaints and neurologic findings. Common causes of cranial nerve III palsy include infection, mass lesion, aneurysm, trauma, and ischemic or thrombotic events.[1,2] Each of these processes are diagnosed in their own way, and the diagnostic pathway is particular to each patient based on history and physical and neurologic exam findings. In general, most pupil-sparing cranial nerve III palsies are due to microvascular disease and usually have no associated symptoms. These generally do not require an extensive workup. Patients who present with cranial nerve III palsy and systemic neurologic complaints, or associated pupil dysfunction, usually require further workup including labs and advanced imaging, because these symptoms can be due to more serious disease processes (eg, aneurysms and mass lesions).

2 | CASE REPORT

A 47-year-old male with diabetes mellitus type II and hypertension presented to the emergency department (ED) with a 3-day history of a left-sided cluster headache. The patient had a history of cluster

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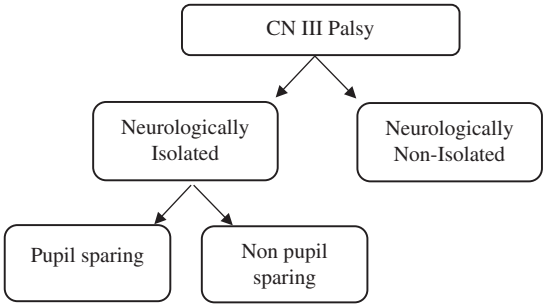

FIGURE 1 Classifications of cranial nerve III palsy

headaches, diagnosed by his primary care physician, that were usually successfully treated with oxygen therapy and oral pain medications. On arrival, the patient had left-sided retro-orbital pain, left rhinorrhea, and tearing of the left eye. These symptoms were similar to his previous cluster headaches. Initial treatment included 100% oxygen by nasal cannula, 975 mg Tylenol, and 100 mg of Imitrex without any symptom relief, subsequently a sphenopalatine nerve block using 1 ampule of 4% cocaine hydrochloride was administered to the left nostril using a cotton tip applicator under direct visualization with the aid of a nasal speculum. The applicator was left in place for 5 minutes with immediate resolution of his headache. The patient was discharged with strict return precautions and planned follow-up with his primary care provider. Three days following discharge, the patient returned to the emergency department with complaints of diplopia and ptosis that he woke up with the previous day and that did not resolve within 24 hours. Physical examination demonstrated an eye resting in abduction and a depressed position (Figure 1A). There was an inability to completely close the left eyelid. Extra ocular muscle testing revealed the left eye was unable to be adducted past midline, but looking to the left produced a normal exam finding (Figures 2C and 1D). Visual acuity tested in each eye separately was 20/20 for near and distant vision. The left pupil was equal in size to the right, round and reactive to light (Figure 1B).

These findings were significant for pupil-sparing cranial nerve III palsy. Laboratory results were significant for blood glucose of 250 mg/dL. The patient was admitted to internal medicine, and neurology/ophthalmology consults were obtained. A computed tomographic (CT) scan and magnetic resonance (MR) imaging of the brain, along with CT angiogram of the head and neck, and MR angiogram of the head were normal and negative for any intracranial pathology. An echocardiogram demonstrated no significant abnormalities, with normal cardiac function and structure. Ophthalmology recommended conservative treatment and an eye patch for diplopia. Four months after initial evaluation, without further intervention, the patient had complete resolution of the diplopia and cranial nerve III palsy.

3 | DISCUSSION

Based on history and physical exam, our patient exhibited signs of neurologically isolated, pupil-sparing palsy. This type of deficit has been associated with peripheral vascular disease, brainstem infarcts, neoplasms, and aneurysms. According to a prospective study in ocular motor mononeuropathies, any common vascular risk factor (ie, diabetes, hypertension, hyperlipidemia, or coronary artery disease) was significantly associated with a peripheral microvascular etiology[3] as opposed to an aneurysm or mass. Another study noted that most diabetic cranial nerve III palsies are peripheral, and the pupillary sparing is due to the sparing of the circumferential fibers.[4] Our patient was noted to have a history of diabetes; however, given the timing of onset of

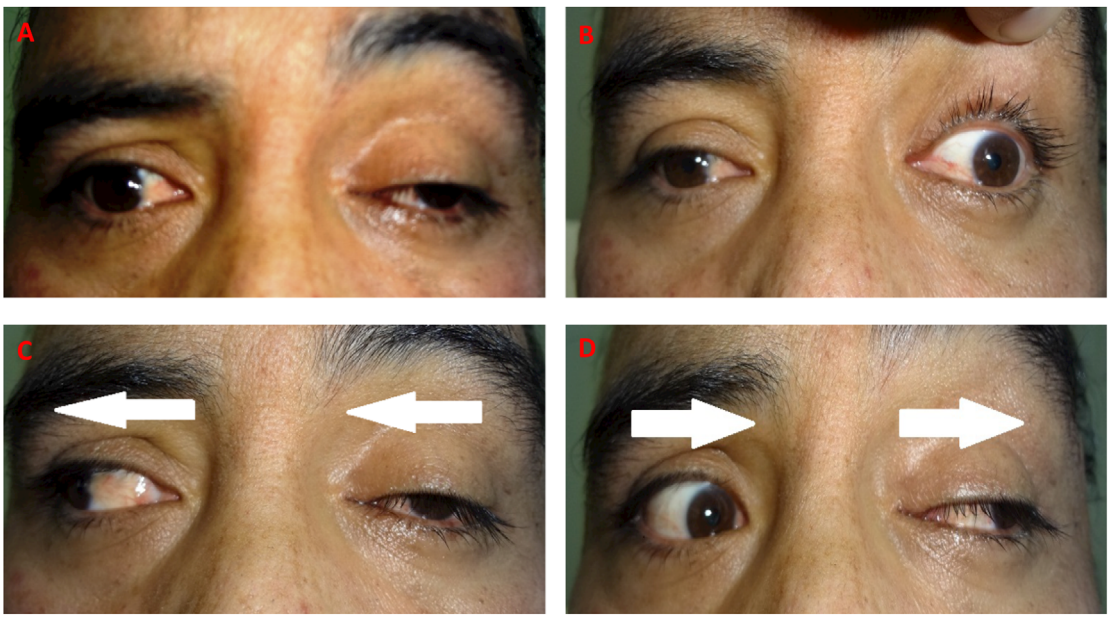

FIGURE 2 (A) Patient looking straight. (B) Pupil comparison with physician holding left eyelid. (C) Patient looking right (adducting left eye). (D) Patient looking right (abducting left eye)

symptoms with the administration of intranasal cocaine, the treatment used cannot be excluded as a cause. In a case report by Migita et al,[5] a man presented with right-sided cranial nerve III palsy the day after inhalation use of cocaine, and the timing of resolution of symptoms was similar to that of our patient. In strokes related to cocaine use, mechanisms of vasospasm include sympathomimetic action preventing reuptake of noradrenaline, serotonin, and dopamine. Another mechanism by which cocaine induces vasospasm is by increaing release of intracelluar calcium.[5] This influx in calcium may produce cerebrovasospasm by direct action on the cerebral blood vessels.[6] These 2 mechanisms of action, in association with a patient who likely already had microvascular disease secondary to his diabetes, could suggest a causation of the cranial nerve III palsy with the administration of cocaine instead of diabetes alone. Headache is a common complaint in the ED and can be challenging to manage. A study done by Doretti et al[8] showed that non-steroidal anti-inflammatories, followed by acetaminophen, were more common in headache treatment than other modalities. Interestingly, a prior study published in April 2018 showed increased efficacy if these medications were used in conjunction with anti-dopaminergic medications and also steroids for reduced rebound headaches.[9] Other treatment modalities that are continuing to be studied include nerve blocks and ketamine for treatment of refractory headaches. As previously discussed, sphenopalatine nerve blocks have been used to successfully treat both cluster and migraine headaches. Cocaine was the original medicinal agent used for this procedure, but recently lidocaine has become the drug of choice, because both of the medications have similar efficacy, with lidocaine having far fewer side effects, especially in patients with high risk for microvascular disease.

4 | CONCLUSIONS

Headaches can be difficult to treat, and emergency physicians have many modalities at their disposal to treat patients. Co-morbidities of the individual patient should be taken into account when using certain treatment options. Hypertension and diabetes, which our patient had, have been shown to be associated with pupil-sparing cranial nerve III palsies, and it is unclear whether the medicinal cocaine used caused the patient's symptoms. Patients who present with this complaint, with a history of either medicinal or recreational cocaine use, should have a standard stroke/ischemic workup including imaging, to ensure no other organic/vascular causes exist. When using a sphenopalatine nerve block for headache treatment, lidocaine should be preferentially selected over cocaine due to the increased potential risk of cocaine, especially in a high-risk patient population.

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