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Prostate cancer metastasis to clivus causing cranial nerve VI palsy

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KEYWORDS
CN VI palsy;
Abduction deficit;
Prostate;
Metastasis;
Clivus

Abstract

BACKGROUND: An abduction deficit can have many potential etiologies. Clinical testing can help distinguish a neurogenic from a restrictive process. For any patient with a current or past history of cancer, even in the setting of vasculopathic risk factors, a further workup is necessary to rule out a metastatic process.

CASE REPORT: A 66-year-old man reported sudden blurry vision but did not describe a definite diplopia. Clinical evaluation found left cranial nerve (CN) VI palsy. Although he did have vasculopathic risk factors, neuroimaging found prostate cancer metastasis to the mid to left clivus, extending to the left cavernous sinus region as well as a smaller metastasis to the left temporal lobe. The patient underwent radiation treatment with improvement in his clinical presentation and symptoms. His prostate cancer was subsequently treated more aggressively, and 2 years later, despite spinal metastases, he was doing relatively well.

CONCLUSION: Prostate cancer commonly metastasizes, with a high propensity to invade bone. CN VI runs along the midline-structured bony clivus, between the pons and the cavernous sinuses. Therefore, a metastatic lesion to the clivus can be responsible for unilateral or bilateral CN VI palsy. In men, a common primary site of cancer metastasis to the clivus is the prostate. Eye doctors must look closely for evidence of even subtle abduction deficits in all patients with a history of prostate cancer. Early detection can lead to improved medical treatment and extended life expectancy.

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Initial presentation

A 66-year-old man presented emergently for a neuro-ophthalmic disease consultation because of a left abduction deficit. Four days before this visit, he noticed a sudden onset of blurred vision. He described this blur as a glare from the sun and car headlights, which seemed to be worse when he looked to the left. He did not specifically report a complaint of diplopia, even when questioned. Although he did note that things looked "blurred and confused" mainly when he looked to the left, he just could not appreciate that things were actually double. Consistent with diplopia was the fact that he noted improvement in vision with either eye occluded. His wife noticed that recently his left eye, at times, appeared to be turned in. The patient denied any associated eye pain or headache as well as any other visual, ocular, or neurologic symptoms. He specifically denied jaw claudication, scalp tenderness, fever, decreased appetite, joint pain, or any other symptoms consistent with giant cell arteritis. Also, all symptoms of myasthenia gravis as well as thyroid dysfunction were denied.

He went to a local hospital emergency room 2 days before this examination because of his vision disturbance. At that time, he was told that his blood pressure was

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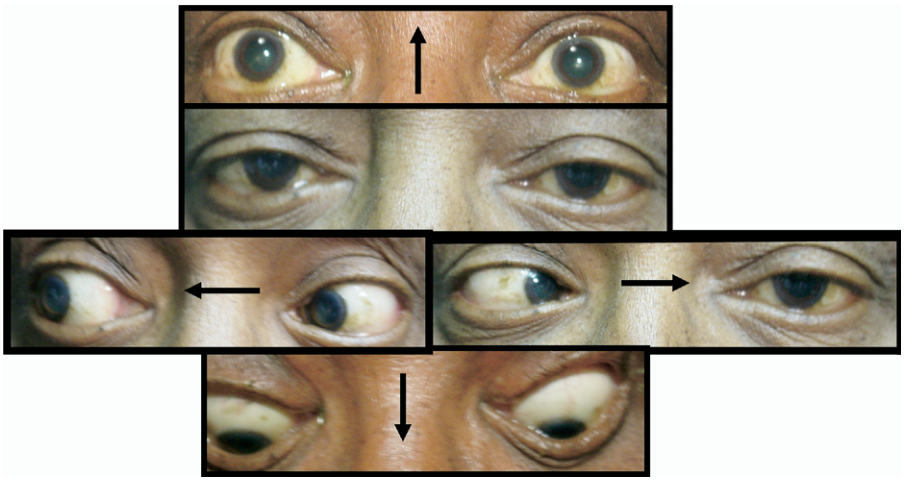

Figure 1 Ocular motility testing. Note left abduction deficit. Although there appear to be evident upgaze restrictions, this was not the case. Unfortunately, a good photo in upgaze was not obtained.

elevated at 190/90 and that this may have been the cause of his symptoms. His ocular history was otherwise unremarkable. His last eye examination was 20 years before this visit. He had no history of trauma to his eyes or head. Systemic history was remarkable for hypertension for the past 10 years as well as a more recent diagnosis of prostate cancer, for which he was being treated with an unspecified chemotherapeutic agent every 3 months. He was seeing a urologist for this treatment and had not undergone surgery or radiation therapy. Otherwise, he was medicated on hydrochlorothiazide and nifedipine.

Best-corrected visual acuities were 20/30 in the right eye (O.D.) and 20/25 in the left eye (O.S.). Color vision testing found 7/7 Ishihara plates correctly identified in both the right and left eyes. Pupils were isocoric, without evidence of a relative afferent pupillary defect. Confrontation visual fields were full to finger counting, simultaneous finger counting, simultaneous hand comparison, and red targets, bilaterally. Ocular motility testing found a left abduction deficit with the left eye obtaining only approximately 5% normal abducting capacity. There were no restrictions noted in any other gazes (*see* Figure 1). Cover testing at distance found a 30–prism diopter esodeviation (eso) in primary gaze, which decreased to 20 eso in right gaze and increased to greater than 45 eso in left gaze. This pattern stayed consistent in superior and inferior gazes, and there were no noted vertical components (*see* Figure 2). Maddox rod testing was performed at distance with no indication of suppression and similar magnitudes of esodeviation in all positions. Cover testing at near (50 cm) found a 25–prism diopter esodeviation in primary gaze, which decreased to 2 eso in right gaze and increased to greater than 45 eso in left gaze. Optokinetic nystagmus testing was symmetric between the 2 eyes when the flag was pulled to the left. However, when the flag was pulled to right, slowed saccades were noted in the left eye, indicating that this abduction deficit was likely neurogenic in origin. A forced duction test was performed and was found to be negative in that the left eye could easily be moved into abduction. Eyelids were properly positioned, without evidence of ptosis. Hertel exophthalmometry measurements were found to be 23 mm O.D. and 22 mm O.S. with a base of 114 mm.

Slit lamp examination was remarkable only for corneal arcus and mild nuclear sclerotic and posterior subcapsular lens changes bilaterally. Applanation tensions were 15 mmHg O.D. and 18 mmHg O.S. Blood pressure was 150/84 mmHg in the right and left arms sitting. Dilated fundus examination found optic discs with distinct margins and no evidence of edema. There was .30/.30 cupping bilaterally. The neuroretinal rim was intact and pink with no evident pallor in either eye. Neurologic examination found CNs V and VII through XII to be intact. Motor, sensory, and coordination testing were unremarkable. There was no evident weakness of the orbicularis oculi muscle in either eye.

The assessment was that the patient presented with a left abduction deficit in which only approximately 5% normal abducting capacity was obtained in the left eye. Because of the negative forced duction test as well as the slowed saccades and asymmetric optokinetic nystagmus, it was likely that this was not a restrictive process, but rather of a neurogenic etiology, specifically, left CN VI palsy. It was possible that this finding could be related to a microvascular compromise to CN VI, related to hypertension; however, this remained a diagnosis of exclusion. It was necessary to

Cover test @ Distance				
	20eso	30eso	>45eso	
Right gaze	20eso	30eso	>45eso	Left gaze
	20eso	30eso	>45eso	

Figure 2 Results of cover testing at distance on initial presentation. Maddox Rod testing yielded similar results.