Transient Isolated Trochlear Nerve Palsy Associated with Rathke's Cleft Cyst

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We report the case of a 52-year-old woman who developed vertical diplopia of 1-days duration. Neuro-ophthal-mological testing revealed left trochlear nerve palsy, and sellar MRI revealed a 1.5 cm-sized pituitary mass lesion, a Rathke's cleft cyst. The diplopia disappeared spontaneously after 6 days.

Key Words: Trochlear nerve, diplopia, transient palsy, isolated cranial nerve palsy, Rathke's cleft cyst, case report

INTRODUCTION

In adulthood, superior oblique palsy is a common cause of acquired vertical diplopia. Trochlear palsy can develop in rare cases of increased intracranial pressure or due to indirect compression from a mass, but not nearly as often as it develops from oculomotor or abducens palsy.

Ocular-motor nerve dysfunction due to a parasellar mass occurs in 1% to 14% of patients with pituitary lesions, usually as isolated third or sixth nerve palsies, or both. We describe a patient who suffered transient isolated left trochlear nerve palsy of 1 week's duration in association with a Rathke's cleft cyst.

CASE REPORT

A 52-year-old woman presented with diplopia

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of 1 days duration. She complained of vertical double vision when she looked in the right lower direction. Other directions of eye movement produced no diplopia. She had experienced a mild, dull headache for 7 days, but denied head or facial trauma, infection or a recent surgical procedure.

Her history, showed no hypertension, diabetes, atherosclerosis or other specific diseases. By physical examination, her blood pressure was 120/70. Her visual acuities were 20/25 bilaterally and no visual field defects were found in the Goldman perimeter. Her pupils were isocoric, and prompt light reflexes were present bilaterally. A red glass test and Bielschowsky's Three-Step head-tilt test revealed isolated superior oblique palsy. The double Maddox rod test showed 2 degrees of left excyclotorsion and right side downward gaze causing left hypertropia of 1 prism diopter. A forced duction test showed no restriction.

MRI of the brain stem and orbits were normal, but sellar MRI showed a 1.5 cm mass lesion. The mass expanded beyond the sella, and was hyperintense on the T1 weighted image but was not enhanced by gadolium, and therefore, was compatible with a Rathke's cleft cyst.

Blood tests were performed during the admission period. Anti-nuclear antibody and acetylcholine receptor antibody were negative. ESR was 22 mm/hr, thyroid function testing and combined pituitary hormone testing were normal.

Repetitive nerve stimulation testing and visually evoked potentials were also normal. On the fourth admission day, her diplopia occurred intermittently and subsided completely the next day. In addition, the double Maddox rod test no

longer showed excyclotorsion.

After a 1 month's follow-up, the diplopia did not recur and MRI showed no interval change in the cyst.

DISCUSSION

The trochlear nerve is vulnerable to head trau-

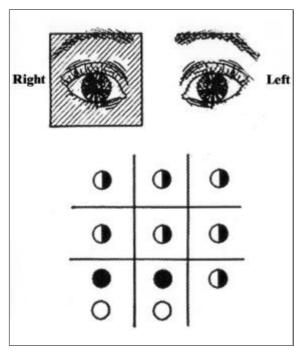


Fig 1. The red glass test showed left isolated trochear nerve palsy. The red glass is in the front of the right eye, and the fields are projected as the sees the images.

ma. Its fascicles are thin and run for some distance in the peduncular cistern. Moreover, the cisternal part of the nerve is closely related to the tentorium cerebelli. After traveling the undersurface of the tentorial edge, it pierces the dura at a point below the point of entry of the oculomotor nerve, and travels into the cavernous sinus along the lateral of the clivus and below the petroclinoid ligament.²

Rathke's cleft cyst is a remnant of the squamous epithelium from Rathke's cleft invagination within the sella. The cyst is usually hyperintense in all pulse sequences, as it contains mucoid or cellular debris. When large and extending beyond the boundaries of the sella, this character of persistent hyperintensity helps in the differentiation of cysts and craniopharyngiomas. Also, the capsule or contents of the cyst are not enhanced on gadolium as occurs with craniopharyngioma.³

Direct invasion of the cyst seems unlikely. In rare cases, pituitary macroadenoma has been reported to cause trochlear palsy, but usually such trochlear palsies are permanent and imaging has show tumor invading the lateral wall of the cavernous sinus.⁴ In the present case, the cyst showed no malignant character and no invasion of the lateral wall of the cavernous sinus.

Another possible mechanism of palsy is indirect traction injury by the cyst upon the trochlear nerve. Jacobson et al. reported transient trochlear palsy after anterior lobectomy.⁵ The volume loss after lobectomy influences the lateral wall of the cavernous sinus by indirect mechanical distortion,

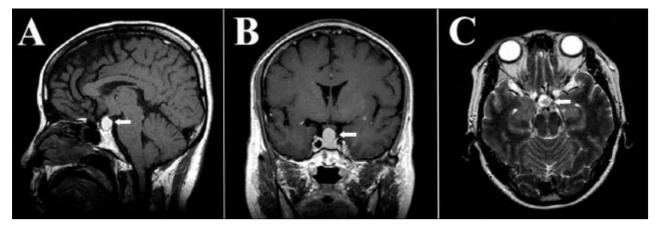


Fig 2. MRI of the brain showed a 1.5 cm mass lesion, which expand beyond the sella, with hyperintense on the T1 (A) and T2 (B) weighted image and not enhanced with gadolium (C).

and it may cause trochlear palsy for several weeks. The trochlear nerve is the thinnest nerve attached to and residing within the lateral wall of the cavernous sinus and is vulnerable to mechanical distortion.

There are other examples of the relative vulnerability of the trochlear nerve. During pregnancy, the volume of the extracellular fluid compartment increase by as much as 50%, and causes expansion of the cavernous sinus. It also exerts compression or traction on the laterally located trochlear nerve. In one case, the diplopia resolved abruptly 5 hours after delivery, but in other cases, it persisted from several weeks to months.⁶

The posterior cavernous sinus has a large space with a venous drainage route from the pterygoid plexus and the superior and inferior petrosal sinus. The oculomotor nerve is separated from the venous channel by a thin meningeal lining. The abducens nerve passes through the Dorello's canal and travels between the carotid artery and Meckel's cave. These cranial nerves are in a relatively free-floating position and the trochlear nerve is vulnerable to the effect of indirect mechanical distortion from the lateral wall of the cavernous sinus.⁷

We suggest that the Rathke's cyst involvement, more so on the left side of the pituitary fossa in our patient, caused indirect mechanical compression on the trochlear nerve at the lateral side of the cavernous sinus. In this case, the cystic size and the degree of deviation by neuro-opthalmologic testing were small. These facts explain

why vertical diplopia was transient. The trochlear nerve was probably not damaged directly by the cyst. Rather, the indirect mechanical distortion changed the relative position of trochlear nerve and secondary damage to the trochlear nerve caused transient palsy, which was resolved soon after decompensation.

This case and other superior oblique palsy by tractional mechanisms showed transient duration and good prognosis. Sufficient follow-up is needed if the cause of the trochlear palsy is thought to tractional injury. Although rare, pituitary tumor or cyst must be included in the differential diagnosis of the trochlear nerve palsy.

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