A 42 year-old white women was referred with ptosis OS and diplopia. Three weeks prior to referral, she noted drooping of her left eyelid. The drooping was worse in the evenings, and had progressively worsened since onset. One week prior to referral, she noted binocular oblique diplopia, which also was worse in the evenings and had progressively worsened since onset. She complained of a dull retrobulbar pain OS of several weeks duration. This was exacerbated by elevation and abduction OS.

Past history was remarkable for asthma and chronic frontal headaches. Medications were Proventil, Azmacort, Slo-bid, Ativan and Prozac.

Examination revealed 20/20 vision OU. There was 2mm of proptosis OS with soft retropulsion OU. There was 5mm of ptosis OS with fatigue on sustained upgaze and a Cogan's lid twitch sign. Ocular motility revealed full ductions OD and a 25% deficit in elevation and adduction OS. In primary position, she had a 25 prism diopter XT that increased to 35 prism diopters on right gaze and she was orthophoric on left gaze. Pupils were equal and reactive with no RAPD. Visual fields, fundus exam and the remainder of the neurologic exam were normal. A tensilon test produced no change in her examination. Examination four days later was unchanged.

MRI revealed an enhancing left cavernous sinus mass. A diagnostic procedure was performed.

After the procedure, she was symptomatically unchanged until three weeks later when she noted that her diplopia spontaneously resolved and the left lid elevated over the course of one day. Exam at that time revealed 2mm of ptosis OS. Ocular motility revealed full ductions OD and a 5% deficit in elevation and adduction OS. In primary position, she had a 16 prism diopter exophoria. There was 0.5 mm anisocoria (left greater than right). There were no signs of aberrant regeneration. The remainder of the exam was normal.
Title of Presentation: Here today, gone tomorrow

Hos: Paul Phillips, M.D. and Nancy J. Newman, M.D.

Final Diagnosis: Meningioma

Summary of case including pathology and references (200-300 words):

**Biopsy Results:** Meningotheliomatous Meningioma

**Course:** Five weeks after the biopsy, the patient was treated with radiation; exam one month post treatment revealed 2mm of ptosis and a 5% deficit in elevation OS. Acetylcholine receptor antibodies were not present.

Cavernous sinus masses typically present with progressive ophthalmoplegia. Spontaneous improvement is considered evidence against a neoplasm as an underlying etiology. However, there are numerous reports of mass lesions presenting with cranial nerve dysfunction which subsequently improves.

Volpe and Lessell\(^1\) described seven patients with remitting sixth nerve palsies due to skull base tumors. All of their patients recovered completely at least once without any intervention. The skull based tumors included one meningioma, three chordomas, two chondrosarcomas and one of unknown etiology. Two of these tumors involved the cavernous sinus. Thomas and Yoss\(^2\) described 70 patients with palsies of two or more cranial nerves due to parasellar neoplasms. 12 (17%) of the patients had neurologic symptoms or signs that remitted spontaneously one or more times. Moorthy et. al.\(^3\) reported eight patients with variable ophthalmoplegia due to intracranial masses who were initially diagnosed as having myasthenia gravis. The intracranial masses included six meningiomas, a chondrosarcoma and an aneurysm. Three of these masses involved the cavernous sinus. Three patients had Cogan's lid twitch sign and seven had positive responses to edrophonium. It was concluded that four of these patients had only an intracranial mass and four may have had both myasthenia gravis and an intracranial mass.

Spontaneous improvement of neurologic deficits due to mass lesions is not limited to ophthalmoplegia. Pless and Lessell\(^4\) described three patients with proved or presumed orbital apex meningiomas involving the optic nerve who had spontaneous improvement in visual acuity and visual fields. Spontaneous visual improvement has been described in patients with chiasmal gliomas.\(^5\)

The mechanisms responsible for the spontaneous improvement of neurologic deficits due to mass lesions are unknown. A reduction in the size of the tumor secondary to auto-infarction or host immune/hormonal factors as well as variable tumor associated edema are postulated mechanisms.\(^4\)

Spontaneous improvement of neurologic deficits does not rule out the presence of a tumor as the underlying etiology.
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