**Sixth Nerve Palsy**
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**CASE HISTORY:**

74-year-old male presented to the clinic with acute onset bilateral diplopia, symptomatic for two days. It began as a blurred vision and now images are completely separate. The diplopia is worse at distance than at near. The patient had gone to the emergency department the morning of examination. They performed a CT scan and bloodwork with no evidence of lesions, stroke, or apparent cause for diplopia. Blood pressures at the ED was 140/78.

Based on symptoms and imaging, GCA and a space occupying lesion were ruled out as likely causes of diplopia.

**Ocular History:**
- Early Cataracts OU
- Corneal Acanthas SIS OU
- Myopia, Presbyopia OU

**Medical History:**
- Hypertension (Atheros, 81mg Aspirin)
- Hyperlipidemia (Pravastatin)
- Allergic rhinitis
- Recent shoulder surgery
- Recently began taking glucocorticosteroids
- (VHO Cancer

**Visual Acuity:**
- OD 20/20-2
- OS 20/20-2

**Pupils:**
- PERRL, (2)
- APD, (2)

**CILIUS:** mild restriction with abduction OS diplopia in left gaze

**Confrontations:**
- Full to finger counting OU

**Slit Lamp Exam:**
- Tonometry: 16mmHg OU
- UiscLashere: 6 Hertanous OU
- Comas: acous OU
- Lens: G+1 NS
- (-) iritis

**Uncapped Fundus Exam:**
- Nerve: C-0 0.25 OU, 54, good color, healthy rim tissue

**Sixth Nerve Palsy Overview**

**Sigs/Symptoms:**
- Binocular horizontal diplopia
- Diplopia worse at distance than at near
- Diplopia worse in direction of parietal muscle
- Limited lateral movement of the eye
- Negative forced duction testing
- Head turn to side of parietal muscle

**Differential Diagnosis:**
- Thyroid eye disease
- Myasthenia gravis
- Herpes Zoster Ophthalmicus
- Idiopathic intracranial hypertension
- Duane's syndrome type I
- Spasm of the near rectus
- Delayed break in fusion
- Old blowout fracture of the orbit
- Giant Cell Arteritis
- Foville's syndrome
- Millard-Gubler syndrome

**Signs of Lesion:**
- New onset headache
- Optic nerve head swelling
- Hemifacial pain, numbness or tingling
- Facial drooping
- Proptosis
- Corneal sensitivity
- Unilateral reduction in tear prism
- Concurrent EOM palsies
- Bilateral palayia

**Treatment:**
- Observation
- Botulism of medial rectus
- Medial rectus recession
- Prism
- Treatment of underlying condition

**DISCUSSION**

Microvascular ischemia is the likely cause of this patient's sixth nerve palsy. Blockage of small arteries secondary to hypertension interrupt blood flow to the nerve resulting in oxygen deprivation of the nerve. The nerve is weak and therefore has limited ability to innervate the lateral rectus muscle.

Typically, sixth nerve palsies resolve without intervention in approximately three to six months and observation is a reasonable course of management. Recovery rates are highest in patients with traumatic and vascular causes. However, one study showed a recurrence in 31% of patients with sixth nerve palsy due to vascular cause. In the case of a sixth nerve palsy, imaging should always be performed if more than one nerve is involved, there is pupil involvement, examination shows proptosis or disc swelling, the patient is young, or if the patient has a history of cancer, or if there is not recovery in the expected timeframe.

**CONCLUSION**

The sixth nerve has a long course from the pons to the lateral rectus muscle and can be affected by trauma at any location throughout its course. The likelihood of an intracranial lesion causing the sixth nerve palsy can be evaluated by looking for signs of involvement of affected adjacent structures. Although isolated sixth nerve palsies are most commonly caused by vasculopathic conditions, it is essential to monitor this condition closely.

**BIBLIOGRAPHY**