*İnönü Üniversitesi Tıp Fakültesi Dergisi* *15 (1) 35-37 (2008)*

# Intracavernous Carotid Artery Aneurysm in a Case of Tolosa-Hunt Syndrome

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Purpose: To report a case presented with the clinical features of both orbital apex and cavernous sinus syndrome.
Methods: Review of clinical, orbital magnetic resonance imaging and magnetic resonance angiography findings.
Results: A 82-year-old man admitted to our clinic with left CN III, CN IV and CN VI nerve palsy and involvement of optic nerve (CN II), ophthalmic ($V_1$) and maxillary ($V_2$) branch of the trigeminal nerve. With the help of orbital magnetic resonance imaging and magnetic resonance angiography the diagnosis of intracavernous carotid artery aneurysm in Tolosa-Hunt Syndrome was made. There is dramatic recovery in visual acuity and pain after the initiation of steroid therapy.
Conclusion: Neuroimaging studies may be helpful to explain the symptomatology and to localize the place of orbital and cavernous lesions.

**Key Words:** Intracavernous carotid artery aneurysm, Tolosa-Hunt Syndrome, Neuroimaging

**Tolosa-Hunt Sendromlu Bir Olguda İntracavernöz Karotid Arter Anevrizması**

Amaç: Orbital apeks ve kavernöz sinus sendrom klinik özelliklerinin taşıyan bir vakayı sunmak.
Yöntem: Hastanın klinik, orbital manyetik rezonans ve manyetik rezonans anjiografi bulgularının tartışılması.
Bulgular: 82 yaşında erkek hasta sol CN II, CN III, CN IV, CN $V_1$, CN $V_2$ and CN VI sinir tutulumları ile başvurdu. Orbital manyetik rezonans görüntüleme yardımı ile Tolosa-Hunt sendrom ve intracavernöz karotid arter anevrizması tanısı kondu. Steriod tedavisi başlanması sonrasında hastanın görme keskinliği ve ağrısında belirgin düzelme oldu.
Sonuç: Nörogörüntüleme yöntemleri orbital ve kavernöz bölge lezyonlarında lezyonun yerinin tespiti ve semptomların açıklanmasında faydalıdır.

**Anahtar Kelimeler:** Intracavernöz karotid arter anevrizması, Tolosa-Hunt sendromu, Nörogörüntüleme

Tolosa-Hunt syndrome (THS) is caused by a non-specific inflammation of the cavernous sinus or orbital apex characterized by painful ophthalmoplegia. We report a case of THS with intracavernous carotid artery aneurysm (ICCAA).

## CASE REPORT

An 82-year-old man with a history of ptosis and visual loss on the left eye was referred to our institution. The patient described recurrent gnawing boring left retroorbital pain at about six-month period. Medical history disclosed a left frontotemporal subdural hematoma operation due to trauma 3 years ago. On ophthalmological examination visual acuity were 20/60 in the right eye (Snellen Chart) and hand motions in the left eye. The both anterior segment and fundus examinations were normal except bilateral nuclear cataract. Intraocular pressures were 16 mm Hg in the right and 18 mm Hg in the left eye. The right pupil was measured as 3 mm and reacted normally to light whereas the left pupil was 5 mm, did not react, and showed an afferent pupillary defect. Ocular movement examination showed a left oculomotor (CN III), trochlear (CN IV), and abducens (CN VI) nerve plegia (total ophthalmoplegia) (Figure 1). The neurological examination revealed the involvement of the ophthalmic ($V_1$) and maxillary ($V_2$) branch of the trigeminal nerve. Orbital magnetic resonance imaging (MRI) weighted in T1 with GD-DTPA demonstrated enhanced signal in the left optic nerve, extra ocular muscles and retroorbital fat (Figure 2a). Magnetic resonance

angiography (MRA) revealed an aneurysm of the $C_3$ and $C_4$ intracavernous part of left internal carotid artery (ICA) measured 20X19X18 mm (Figure 2b). The patient refused angiography, endovascular treatment and biopsy. After the initiation of steroid therapy (oral fluocortolone, 60 mg/day), there was a dramatic reduction of pain within 24 hours. At the third day of the steroid therapy visual acuity was 20/120 on the left eye. The steroid dose was decreased to 40 mg/day after the second week. However ophthalmoplegia did not regressed till the end of one-month. The patient refused to attain our follow-up procedure.

**Figure 1.** Ocular movement examination showed total ophthalmoplegia.

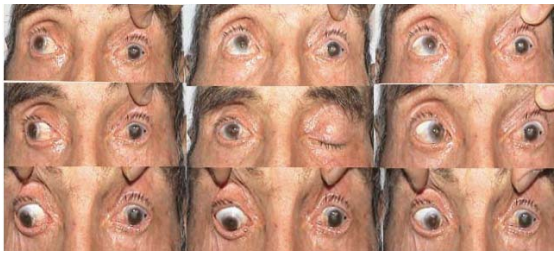


**Figure 2a:** GD-DTPA enhanced T1- weighted axial magnetic resonance imaging shows enhanced signal at left optic nerve (open arrow), extra ocular muscles (solid arrow) and retroorbital fat (arrow head).

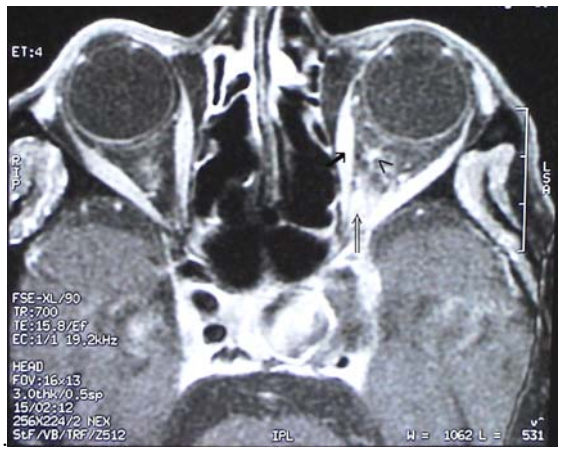
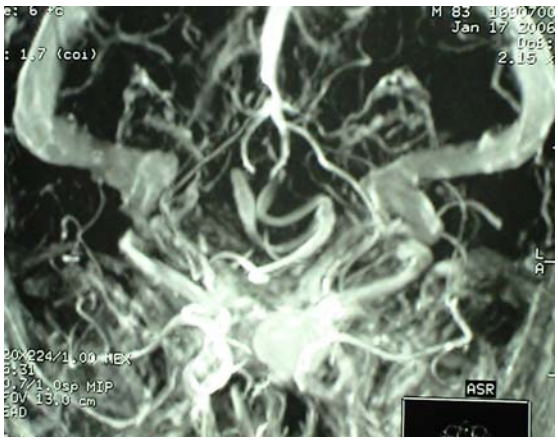



**Figure 2b:** Magnetic resonance angiography shows an aneurysm of the $C_3$ and $C_4$ intracavernous part of left internal carotid artery (ICA) measured 20X19X18 mm.




## DISCUSSION

Tolosa-Hunt syndrome is one of the causes of orbital apex syndrome (OAS). OAS is characterized by variable deficits of CN III, CN IV, CN VI, and ophthalmic branch of the trigeminal nerve ($V_1$) in association with optic nerve dysfunction. Cavernous sinus syndrome (CSS) may include the features of an OAS with added involvement of the maxillary branch of the trigeminal nerve ($V_2$).[1]

Our patient presented with both the clinical features of orbital apex and CSS. After the initiation of the steroid therapy the optic nerve dysfunction, pain and hypoesthesia at the tributary of the ophthalmic branch of the trigeminal nerve ($V_1$) reversed but the symptoms due to involvement of the other cranial nerves persist. The involvement of the CN III, CN IV, CN VI, maxillary branch of the trigeminal nerve ($V_2$) and optic nerve, ophthalmic branch of the trigeminal nerve ($V_1$) were due to ICCAA and orbital apex inflammation, respectively.

Intracavernous carotid artery aneurysm due to inflammatory infiltration was reported previously in a Tolosa-Hunt syndrome patient.[2] In our patient inflammation did not extend up to cavernous sinus so the probable mechanism of the aneurysm might be previous head trauma that was reported as an etiological factor.[3]

Before modern computed tomographic (CT) or MRI, radiographic evaluation for THS consisted of angiography and plain films. Angiographic features in

THS include narrowing of the carotid siphon, occlusion of the superior ophthalmic vein, nonvisualization of the cavernous sinus.[4] However, a normal orbital venogram or arterogram does not exclude THS.[5] Although CT may occasionally reveal enhancing lesions in the cavernous sinus or orbital apex, the appearances are nonspecific. Contrary to other neuroradiological studies which may be normal, MRl are very sensitive tools for the diagnosis of THS.[6]

The symptomatology of OAS and CSS interdigitate to each other. All cranial nerves must be examined carefully in order to localize the place of the lesion. Neuroimaging studies especially MRI may be helpful to explain the symptomatology.

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