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# Horner Syndrome Due to Intrathoracic Multinodular Goiter (Case Report)

## *İntratorasik Multinodüler Guatrın Neden Olduğu Horner Sendromu (Olgu Sunumu)*

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**Summary**

Horner syndrome is characterized by miosis, ptosis, facial anhydrosis and enophtalmus and is caused by a lesion along the oculosymphathetic pathway starting from hypothalamus and ending in the eye. The long course of the oculosympathetic pathway predisposes it to a wide variety of pathologic processes, ranging from harmless vascular headaches to life-threatening conditions such as carotid artery dissection or malignancy. Thyroid neoplasms, benign or malign, are among unusual causes of Horner syndrome and represent 1.3-7.8% of cases. Especially, early diagnosis of benign thyroid neoplasms such as multinodular goiter has particular importance because of its reversibility with appropriate treatment. We report a case of Horner syndrome which has been developed as a complication of intrathoracic multinodular goiter. (Turkish Journal of Neurology 2013; 19:104-6)

**Key Words:** Horner syndrome, multinodular goiter, sympathic system

**Özet**

Horner sendromu, miyozis, ptoz, fasyal anhidroz ve enoftalmus ile karakterizedir. Sendromun nedeni, hipotalamustan başlayıp göze ulaşan sempatik yolaktaki bir hasarlanmadır. Okülosempatik yolağın uzun seyri, onu bir dizi patolojik sürecin hasarına yatkın hale getirir. Bu patolojik süreçler, zararsız vasküler tipte bir baş ağrısı olabileceği gibi, arter diseksiyonu ya da malign karakterde bir tümör gibi yaşamı tehdit eden bir durum da olabilir. Benign ya da malign karakterde tiroid tümörleri %1,3-7,8 gibi nadir bir sıklıkta Horner sendromuna neden olmaktadır. Multinoduler guatr gibi benign karakterli tiroid tümörlerinin erken tanısı, uygun tedavi ile geri dönüşümlü bir durum olmasından dolayı özellikle önemlidir. Burada intratorasik multinodüler guatrın neden olduğu Horner sendromu olgusu sunulmuştur. (Türk Nöroloji Dergisi 2013; 19:104-6)

**Anahtar Kelimeler:** Horner sendromu, multinodüler guatr, sempatik sistem

## Introduction

Horner syndrome is characterized by ptosis, miosis and anhidrosis. The syndrome develops as results of the damage to the sympathetic pathway in any location between the hypothalamus and the eyes (1). The causes of the Horner syndrome are summarized in the Table 1. This syndrome may also occur as a result of the cervical or mediastinal mass lesions of malign character. Timely diagnosis and treatment are crucially important because benign lesions, in addition to malign ones, may cause Horner Syndrome. Thyroid lesions are one of the rare causes of Horner syndrome and may be of malign or benign character (2, 3). The literature showed instances of positive outcome after surgical intervention in Horner cases caused by thyroid pathologies (4, 5, 6). Here we report a case where a thyroid mass lesion caused Horner syndrome.

## The case

The 58-year-old male patient consulted in our clinic with eyelid drooping that had been ongoing for 2 months. He had no other muscular complaints about his body. The drooping was not changing over the course of a day but it was reported that it

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got better in the lying position. The patient was diagnosed with myasthenia gravis on the basis of the clinical findings but this diagnosis was not supported by electromyography or other tests. The patient consulted in our clinic when he was on pyridostigmine treatment. He reported that despite having used pyridostigmine for 180 mg/day for the duration of 2 months, the treatment did not provide any improvement.

The neurological examination showed ptosis in the right eye and miosis in the right pupil (Figure 1). Other neurological findings were normal.

His magnetic resonance imaging (MRI) and electroneuromyography studies were found to be normal. The repetitive neural stimulation assessment did not show any findings supporting the myasthenia gravis disorder. Neck MRI and thoracic computerized tomography (CT) was performed to investigate the presence of Horner syndrome. Multinodular goiters were found in both thyroid lobules extending to posterior mediastinum in both of these imaging studies (Figure 2). The general surgery department performed thyroidectomy on the patient. The pathological inspection results of the biopsy material were congruent with nodular hyperplasia. The patient was re-evaluated after 2 months. The drooping on the right eyelid improved mildly after the surgery.

## Discussion

Horner syndrome includes ipsilateral miosis, ptosis and anhidrosis. In some cases anhidrosis does not accompany the syndrome.

The first sympathetic afferents to the eye are localized in the posterolateral hypothalamus. The nerve fibers originating from here travel as far as the T1 segment of the spinal cord after making connections with brainstem and cerebellum. The second order

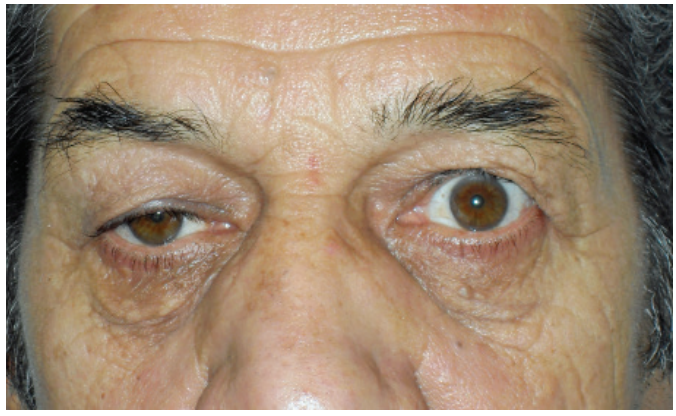

**Figure 1:** Ptosis and miosis in the patient.

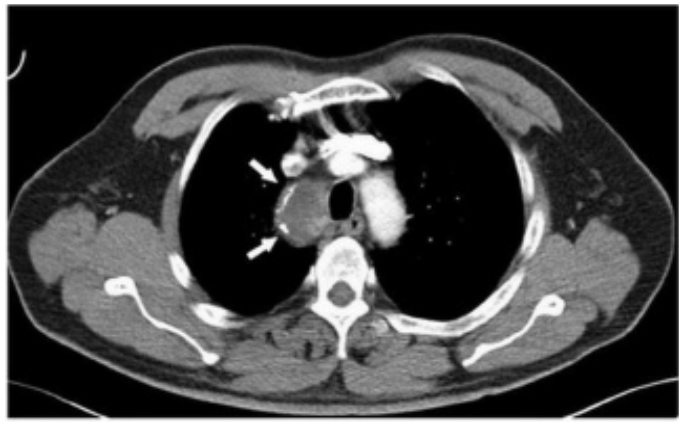

**Figure 2:** Computerized tomography of the patient. The region indicated by the arrows show the retrosternally located thyroid tissue with calcified borders.

**Table 1. Causes of Horner syndrome**

| Central | Preganglionic | Postganglionic |
|---|---|---|
| Hypothalamus/thalamus/brainstem | Cervicothoracic spinal cord | Superior cervical ganglion |
| Ischemia | Trauma | Trauma |
| Hemorrhage | Tumor | Ganglionectomy |
| Demyelination | Syrinx | Cervical adenopathy |
| Tumor | AVM | Jugular venous ectasia |
| Cervical spinal cord | Cervical disc herniation | Internal Carotid Artery |
| Tumor | Epidural spinal anesthesia | Dissection |
| Trauma | Brachial plexus | Trauma |
| Syrinx | Trauma | Thrombosis |
| AVM | Pleural apex | Tumor |
| | Apical lung tumor | Cavernous sinus |
| | Surgical trauma | Tumor |
| | Front neck | Carotid-cavernous fistula |
| | Trauma | Carotid aneurism |
| | Tumor (thyroid) | Inflammation |
| | | Infection |
| | | Thrombosis |
| | | Vascular headache |
| | | Migraine |
| | | Cluster headache |

AVM: Arterovenous malformation

neurons are localized to T1 segment. The nerve fibers of these neurons travel to superior cervical ganglion and synapse with the third order neurons. The nerve fibers of the third order neurons enter the skull in close proximity to internal carotid artery and then get distributed to middle ear, orbital vasomotor fibers, lacrimal gland, accessory levator muscle of the upper eyelid (Müller muscle) and its analog in the lower eyelid (7, 8).

Horner syndrome may present with different subtypes depending on which one of these 3 neuronal groups in sympathetic stimulation pathway coming to the eye are affected. The involvement of the first order neuron group gives rise to central Horner syndrome. It is rarely seen. Thalamus, hypothalamus, cerebellum and brainstem lesions are the main pathologies. The most typical example is presented with lateral bulbar infarction (9). It is easy to clinically diagnose Horner syndrome because it is generally accompanied by other neurological findings. The involvement of the second order neuron group gives rise to preganglionic Horner syndrome. This is the most common form of Horner syndrome and it also is the most common cause of lung apex pathologies (2, 3). Other reasons include vascular lesions, tumors and trauma. Thyroid pathologies are rarely the cause of Horner syndrome of the preganglionic type. The fundamental lesion is located at the sympathetic pathway extending from T1 spinal cord level to the superior cervical ganglion. The involvement of the 3rd order neuron group causes postganglionic Horner syndrome. It is the second most common subtype of Horner syndrome after the preganglionic one (3, 10, 11). It occurs due to vascular, traumatic and infectious causes. The primary pathology is located at the superior cervical ganglion or after.

Horner syndrome is often secondary to an underlying malignant lesion and these are often bronchial cancers of the lung. The benign or malignant tumoral lesions of the thyroid are 1.3-7.8% likely to cause Horner syndrome (6, 10). The syndrome occurs as a result of the pressure applied by the thyroid based tumoral lesions on the second order neuron and its fiber after extending to mediastinum retrosternally. Even though most of the thyroid tumors that cause Horner syndrome were argued to be malignant, some studies argued that benign tumors are actually more frequent (4,5,11, 12,13,14,15,16,17,18,19,20). In our case, the causal link between Horner syndrome and the multinodular goiter was shown by the pathological examination.

Another important point about our case is the pre-existing myasthenia gravis diagnosis at the beginning of the clinical evaluation. In the cases where Horner syndrome is seen in combination with thyroid tumors, the patients often seek help because of the mass on their necks. Our case, on the other hand, consulted to our clinic with miosis and ptosis, and without an apparent mass lesion. The fact that there was no apparent mass caused the patient to be evaluated for muscular and neuromuscular junction disorders and a general omission of the neck and mediastinal lesions in the discriminative diagnosis.

Based on the presented case and the literature, it should be remembered that the benign thyroid pathologies are treatable causes of Horner syndrome. Early diagnosis and proper treatment in the cases of Horner syndrome due to thyroid pathologies and some other treatable conditions may prove to be life saving.

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