Horner Syndrome Following Internal Jugular Vein Catheter Insertion: Report of Two Cases

İnternal Juguler Ven Kateter Takılmasına Bağlı Gelişen Horner Sendromu: İki Olgu Sunumu

ABSTRACT

Horner syndrome is a rare complication which may occur following the insertion of a catheter in to the internal jugular vein. We present two cases of Horner syndrome (HS) which occured following internal jugular venous cannulation.

KEY WORDS: Horner syndrome, Internal jugular vein, Catheter

ÖZ

Horner sendromu internal juguler ven kateter takılmasına bağlı olarak gelişmesi nadir bir komplikasyondur. Bu yazıda internal juguler ven kateterizasyonu takiben gelişen iki horner vakası sunulmuştur.

ANAHTAR SÖZCÜKLER: Horner sendromu, İnternal juguler ven, Kateter

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INTRODUCTION

Horner Syndrome is a triad of unilateral blepharoptosis, pupillary miosis and anhidrosis which results from the functional interruption of sympathetic innervations to the eye. The usual causes of acquired Horner Syndrome are trauma, brain mass lesions or infarction, carotid dissection, tuberculosis, Pancoast tumor and epidural anesthesia injection (1-4). The internal jugular vein (IJV) is the most common site used for insertion of hemodialysis catheters for vascular access due to high success and low complication rates. Damage to the oculosympathetic pathway following central line catheterization is rare but sometimes may occur due to repeated access failures and multiple recannulations (5-6). The most common complications that can occur following IJV cannulation include internal carotid artery puncture, pneumothorax, vessel erosion, airway obstruction and infection. We report two cases of Horner syndrome following right internal jugular vein catheterization.

Case 1

A 29 year old male patient was admitted with peripheral edema and 16 gram proteinuria. According to the patient's history he had become paraplegic due to gunshot injury wounds to the L2 vertebra 4 years before. In the following period renal amyloidosis had developed due to femur shaft fracture and chronic osteomyelitis. On admission the patient was suffering from chronic renal failure and was dependent on renal replacement treatment. The patient's blood pressure was very low because of that peritoneal dialysis was as though before all else. The insertion of a tenchoff catheter was performed for peritoneal dialysis but a short time later peritonitis developed. The catheter was withdrawn and a temporary right internal jugular vein catheter was inserted with multiple attempts. Following catheterization a chest x-ray was taken which revealed no pathology or hematoma formation. This venous catheter was used for hemodialysis and a full dose of heparin was administered. Four days later, the

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patient noticed a right sided ptosis. Subsequent ophthalmic examination by an ophthalmologist revealed a 2 mm ptosis of the right upper lid and constricted pupil (Figure 1). His visual acuity was 6/6 in both eyes. The right pupil was 2 mm in diameter in bright illumination while the left pupil was 3.5 mm. Magnetic resonance imaging and diffusion was performed and no pathology was established. No other neurological symptoms were present such as abnormal eye movements, ipsilateral limb ataxia or dissociated sensory loss. According to this finding a diagnosis of HS was made. In the following month the patient's ptosis cleared up.

Case 2

A 44 year old hypertensive female presented for renal transplantation. She had been treated with chronic hemodialysis three times a week since 2006 through a left brachiocephalic arteriovenous fistula. The cause of end-stage renal disease was hypertensive nephropathy. Kidney transplantion was performed from her mother in 2011. Tacrolimus, mycophenolate mofetil, methylprednisolone, valgancyclovir, and trimethoprim-sulfomethaxozole treatments were iniated after transplantation. Prior to surgery a central line was inserted via the right internal jugular vein by an anesthesiologist in the operating room. One week later, the patient noticed a right sided ptosis. On examination there was not sign of hematoma formation or neurological abnormalities. Further investigation included cranial magnetic resonance imaging, diffusion was performed and no pathology was established. The patient was referred for neurologic and ophthalmic assessment. In dim light the right pupil diameter was measured as 2 mm and the left as 5 mm.

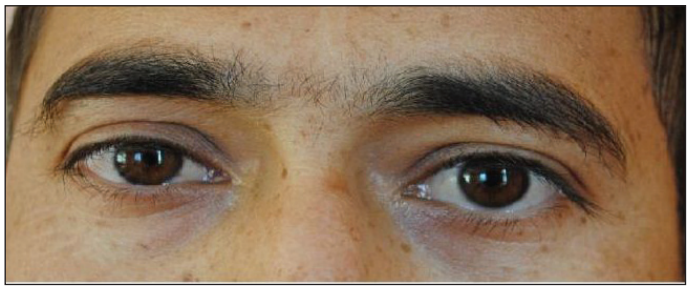

Figure 1: *Right Horner syndrome following cannulation of right internal jugular vein.*

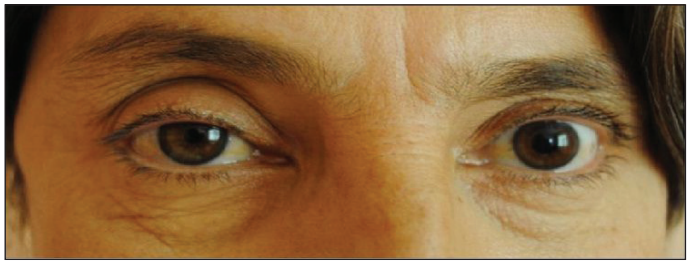

Figure 2: *Right Horner syndrome following cannulation of right internal jugular vein.*

Both pupils were equally reactive to light and no heterochromia was detected. Examination of the patient showed right sided partial ptosis and meiosis and a diagnosis of Horner syndrome was made (Figure 2). In the following month the patient's ptosis was still present but a slight improvement.

DISCUSSION

Horner syndrome, which was first described by Johan Friedrich Horner in 1929, can result from a lesion anywhere along the three-neuron sympathetic pathway that originates in the hypothalamus. Based on localization of the nerve pathway interruption, Horner syndrome is often classified as central, pre or postganglionic. Sympathetic fibers from the hypothalamus descend to the first synapse in the cervical spinal cord (also called the ciliospinal center of Budge). The preganglionic fibers travel from the sympathetic trunk, over the lung apex. They then ascend to the superior cervical ganglion, located at the bifurcation of the common carotid artery. The fibers of the third neuron which are known as postganglionic fibers, then ascend within the adventitia of the internal carotid artery. These fibers enter the orbit to supply the smooth muscle of the levator palpebrae superior is (Muller's muscle) and the dilator pupillae muscle.

Horner syndrome may occur due to lesions of the cerebral hemispheres, hypothalamus, brain stem, cervical spinal cord or the carotid plexus. Associated neurological symptoms and signs can be useful in localizing the origin of Horner syndrome. For example diplopia, vertigo and ataxia suggest brainstem localization. Ipsilateral extraocular paresis in particular a sixth nerve palsy in the absence of other brainstem signs localizes the lesion to the cavernous sinus. In our patients the cranial images were normal and were not in keeping with a brain lesion.

Preganglionic Horner's syndrome lesions can occur as a result trauma or surgery involving the spinal cord, thoracic outlet or lung apex (7). Direct spinal cord trauma or traction on the brachial plexus may distort the ventral roots and cause interruption of the sympathetic innervations, for instance in the newborn. Arm pain or hand weakness typical of brachial plexus lesions suggests a lesion in the lung apex (pancoast tm). Bilateral or ipsilateral weakness, long tract signs, sensory level bowel and bladder impairment suggest involvement of the cervicothoracic cord. Other iatrogenic causes of preganglionic Horner's syndrome include lumbar epidural anesthesia (8) and chest tube placement (9).

The right IJV is preferred as a central venous cannulation access site because it has predictable anatomy, low risk of pneumothorax and high success rate in children and adults. The most common complications to occur following IJV catheterization include carotid artery aneurysm in 2-8% of cases, AV fistula formation, pseudoaneurysm, aortic dissection, and thrombosis following prolonged catheter use. Proximity of the cervical sympathic trunk to the IJV may predispose the trunk to injury by direct

trauma from the needle or from the pressure of the hematoma after an inadvertent puncture of the carotid artery. The choice of insertion site of a central line depends on operation's experience, local practice and the patient. Many techniques for insertion of the catheter into the internal jugular vein have been described (10) with a complication rate of less than 4%. The most common complication is hematoma formation often from damage to the carotid artery. The risk of hematoma to the cervical sympathetic trunk may also be higher when a posterior approach is used to access the vein. In the posterior approach to IJV cannulation, the needle is introduced under the sternocleidomastoid muscle just above the point where the external jugular vein crosses this border, directed towards the suprasternal notch and advanced until it enters the IJV. If the needle angle is too steep, the needle may advance behind the common carotid artery and reach the sympathic trunk, even without perforating the artery. ln a series of 1000 attempted cannulations of the internal jugular vein in patients with coagulapathies, despite an arterial puncture rate of approximately 7%, a severe hematoma was observed in one patient (11). Two patients in this series developed transient HS, which resolved within one month.

The cause of HS in our patients are unknown. While the first patient's catheter was fitted in the nephrology department, the second patient's catheter was fitted in the anesthesia department. We considered HS may have been due to repeated attempts at IJV cannulation and may have been resulted from direct trauma from the needle because our patients had no hematoma formation after the procedure. The development of transient HS in association with anesthesia is an early complication and generally occurs on the first postoperative day. Therefore the second patient's HS was not related to be anesthetic agent. HS may also develop following excessive rotation of the head and neck. When the head is turned to the left more than 40º, the risk of the right IJV overlapping the right carotid artery is significantly increased, leading to greater risk of damage to the sympathetic trunk (12). Patients may have experinced excessive movement of the head and neck.

In conclusion, HS should be recognized as one of the possible complications of percutaneous hemodialysis catheterization via IJV. Physicians should be aware of this complication and avoid repeated manipulations. Catheterization, whenever possible, should be performed under ultrasound guidance.

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