Horner syndrome after unsuccessful venous port implantation by cannulation of the right internal jugular vein

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Abstract

Background: Horner syndrome is a rare but likely underdiagnosed complication of internal jugular vein cannulation.

Case report: We present a case of a young woman undergoing chemotherapy for gestational trophoblastic disease for whom venous port implantation was attempted due to poor peripheral vein access. Despite ultrasound guidance, the procedure was unsuccessful and complicated by a local haematoma, causing compression of the sympathetic nerves with Horner syndrome. The symptoms subsided within 3 weeks without treatment. The possible pathomechanisms of Horner syndrome after central venous cannulation are presented with suggested diagnostic and therapeutic approaches. Special emphasis must be placed on excluding carotid artery dissection because it carries the risk of subsequent cerebral vascular incidents. In the event of a carotid dissection, a multidisciplinary team must choose a pharmacological (antiplatelet drugs/anticoagulation) or interventional approach.

Conclusion: Even with ultrasonography, central venous cannulation is not free of serious risks. In case of anisocoria following an uneventful procedure, diagnostic imaging of the vascular structures in the neck is mandatory for the exclusion of potentially serious complications, such as carotid dissection or venous thrombosis.

Key words: central venous cannulation, complications; sympathetic nervous system, Horner syndrome

Horner syndrome results from damage to the sympathetic nerves that supply the structures of the eye socket. It is characterized by the following classic symptoms: drooping of the upper eyelid (ptosis), constriction of the pupil (miosis), loss of facial sweating on the affected side (anhidrosis), and dilation of the blood vessels, especially within the conjunctiva of the eye. The majority of cases of iatrogenic Horner syndrome have been described after neck local blockade [1] and high epidural anaesthesia [2]. The very first case report of Horner syndrome after cannulation of the internal jugular vein was published in 1972 [3].

CASE DESCRIPTION

Venous port implantation was attempted in a 32-year-old female patient who was undergoing treatment for persistent gestational trophoblastic disease (GTD). At first, she received methotrexate (MTX) alone, but due to the increase in her serum beta-subunit of human chorionic gonadotropin (β-HCG) concentration and suspicious focal change in the right lung detected by computed tomography (CT), her treatment team decided to introduce combination chemotherapy according to the EMA-CO regimen (etoposide, MTX and dactinomycin, alternating with cyclophosphamide and vincristine). Due to difficulties in cannulation of peripheral veins the patient was scheduled for the implantation of a venous port. There were no comorbidities beyond the underlying disease.

After usual preparations under conscious intravenous analgesedation (midazolam 4 mg in fractionated doses and fentanyl 100 µg in fractionated doses), the right internal jugular vein was identified using an ultrasound-guided procedure. The patient’s skin was anesthetized with a 1% solution of lidocaine, and venous cannulation was attempted with the Seldinger method (in-plane technique) using high-
medial access. Despite obtaining venous blood outflow, it was impossible to insert the guide wire deeper than a few centimetres. In this case, ultrasound-guided cannulation was repeated from a low access point. The internal jugular vein was identified and a steady outflow of dark venous blood was observed from the needle after syringe disconnection. The guide wire, dilator and catheter (Smart Port CT; 7.5 Fr; AngioDynamics Inc., USA) were inserted without direct ultrasound control. No unexpected resistance was met. The patient reported only local discomfort. Nevertheless, no blood outflow was obtained, an x-ray examination revealed extravascular catheter placement, within soft tissues of the neck. The catheter was removed and the procedure was not repeated. A few hours later, the patient reported a feeling of drooping of her right upper eyelid. Physical examination revealed the following signs of Horner syndrome: anisocoria (the right pupil diameter was of approximately 1 mm smaller than the left one) and drooping of right upper eyelid. Both pupils reacted normally to light, but there was sustained anisocoria. There were no other symptoms of Horner syndrome. Swelling and bruising of the tissues surrounding the procedure location were observed. Neurological examination did not reveal any damage to other neural structures. No symptoms of local infection were present. X-ray of the patient’s chest was normal. Ultrasound examination of the neck was performed. No signs of vascular injury were found and only “small effacement of muscle structure in the right supraclavicular region with features of a haematoma” was described. During the next few days, neurological and ultrasound examinations were repeatedly performed. The symptoms of Horner syndrome gradually reversed; drooping of the upper eyelid and anisocoria completely disappeared after 3 weeks.

**DISCUSSION**

The anatomy of the sympathetic nervous system supplying the eye is complex. First-order neurons begin in the posterior lateral hypothalamic area and then descend without crossing the midline to the ciliospinal centre between the cervical and thoracic spinal cords, where they form synapses. Preganglionic neurons from this centre cross the stellate ganglion and then, with the cervical part of sympathetic system, reach the superior cervical ganglion. From there, postganglionic neurons travel to the eye socket. Damage to this path on any level may cause the symptoms of Horner syndrome. At present, it is believed that eyeball collapse (*enophthalmos*), previously classified as a symptom of Horner syndrome, is only a delusion resulting from the narrowing of the eyelid aperture [4].

Horner syndrome is a rare complication of central vein cannulation, especially of the internal jugular vein. The incidence of this complication ranges between 0 to 5%, according to various sources [5, 6]. In the majority of the publications dedicated the cannulation of the internal jugular vein, this complication is not described, and the 5% prevalence was present only in one study comprising 40 patients [7]. This is in line with our experience, wherein the case presented in this report is the first such case we have ever observed. However, Horner syndrome can go unnoticed, especially in unconscious patients. Moreover, the sudden onset of the Horner syndrome in such a patient could suggest an intracranial disease and direct diagnostic procedures towards imaging studies of the brain. It is worth noting that in some cases the Horner’s syndrome was described after apparently uncomplicated central venous cannulation. Table 1 summarizes the possible mechanisms of Horner syndrome after central vein patomechanisms, mainly of the internal jugular vein.

<table>
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<tr>
<th>Mechanism of damage</th>
<th>Additional remarks</th>
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<tr>
<td>Direct damage of the stellate ganglion or sympathetic</td>
<td>More likely with high access points, lack of specific treatment; symptoms may</td>
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<td>neurons supplying the eyeball within their cervical</td>
<td>partially or entirely reverse within a few months though cases of persistent Horner</td>
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<td>portions</td>
<td>syndrome have been reported</td>
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<td>Dissection of the common or internal carotid artery</td>
<td>Threat of cerebrovascular complications; depending on type, anti-platelet or</td>
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<td></td>
<td>anticoagulation treatment, as well as intravascular procedures, may be necessary</td>
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<tr>
<td>Compression by a haematoma at the cannulation site</td>
<td>In the majority of cases, symptoms resolve without intervention over the course</td>
</tr>
<tr>
<td></td>
<td>of a few weeks or months, though cases of persistent Horner syndrome have been</td>
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<td>reported</td>
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<tr>
<td>Thrombosis of the internal jugular vein</td>
<td>Delayed complication, a rare cause of Horner syndrome that needs to be treated as</td>
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<tr>
<td></td>
<td>other forms of central vein thrombosis related to central venous cannulation; has</td>
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<td></td>
<td>also been described after insertion of catheters to central vessels from peripheral</td>
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<tr>
<td></td>
<td>access</td>
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<td>Toxic side effects of the drugs administered via a</td>
<td>A late and very rare complication; an important preventive measure is an</td>
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<td>catheter that has partially migrated out of the vein</td>
<td>appropriate depth of catheter insertion and observation of the cannulation site,</td>
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<tr>
<td></td>
<td>which is more likely observed when using drugs with enhanced local toxicity</td>
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<td>(e.g., chemotherapy)</td>
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Carotid artery dissection can lead to a stroke. Therefore it is essential to exclude it as a cause of a Horner’s syndrome following internal jugular vein cannulation [14]. Ultrasonography is a easily accesible diagnostic method. Repeated ultrasound examination during follow-up, assessing not only the morphology but also the flow with spectral Doppler with flow Doppler sonography, has a good specificity. In case of doubt, especially when the imaging quality is not sufficient, or when other neurological signs and symptoms are observed, additional imaging is suggested, including computed tomography, digital subtraction angiography, or magnetic resonance. Repeated neurological examination with the aim of excluding damage of other neural structures and symptoms of central nervous system (CNS) ischemia is of particular importance. Carotid artery dissection requires the consultation of a vascular surgery specialist and interventional radiologist because, depending on the clinical features and the type and burden of changes, anti-platelet drugs or anticoagulation treatment and intra-vessel procedures could be essential [15].

The most likely cause of Horner syndrome in the present patient was compression of on the sympathetic neurons (and possibly stellate ganglion) resulting from the haematoma, which arisen during the attempted internal jugular vein cannulation. The direct cause of the haematoma can be attributed either to the soft tissue damage or an injury of the venous wall. Another possible cause, but less likely — as the procedure was performed under ultrasound guidance, could be an injury of the common carotid artery. The fast and complete symptom reversal argues against direct damage to the sympathetic system.

Complications of the internal jugular vein cannulation are among the possible causes of Horner’s syndrome. In every patient with Horner syndrome after cannulation of the jugular vein, common and internal carotid artery dissection needs to be excluded as this pathology is linked to high morbidity. In this particular case it might have been possible to avoid the complication if the whole procedure of guidewire and dilator insertion had been performed under real-time ultrasound guidance, or if the guidewire position had been checked with radiography before dilator insertion.

ACKNOWLEDGEMENTS
1. The authors declare no financial disclosure.
2. The authors declare no conflict of interest.

References:

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Received: 25.08.2014
Accepted: 3.03.2015