



A rare presentation of Horner's syndrome following cervical epidural steroid injection

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ABSTRACT

Horner's syndrome is a clinically significant condition caused by disruption of the oculosympathetic nerve pathway, typically presenting with ptosis, miosis, and facial anhidrosis. It can result from serious, life-threatening conditions such as trauma, carotid artery dissection, or malignancy; however, it may also arise from iatrogenic reasons, including stellate ganglion blocks, by other surgical interventions of the head and neck. While exceedingly rare, Horner's syndrome has been reported to occur after a cervical epidural steroid injection. Given the various potential etiologies of Horner's syndrome, prompt evaluation is required to rule out life-threatening conditions in the setting of an acute and unexpected presentation.

This case study describes a presentation of Horner's syndrome following a cervical epidural steroid injection (ESI) for a cervical radiculopathy. Due to the unusual nature, a serious cerebrovascular event was initially considered and urgent evaluation was advised. Fortunately, all symptoms resolved fully within two hours without medical intervention. It has been suggested that local anesthetic diffusion to the preganglionic neurons caused pharmacologic disruption of the sympathetic fibers. This case provides additional evidence to the limited reports of Horner's syndrome after cervical epidurals. It also highlights the importance of minimizing or forgoing local anesthetics, considering non-particulate steroids, and conducting emergent evaluation for new onset of neurological deficits during or after cervical ESIs.

1. Introduction

Horner's syndrome is a disruption of the oculosympathetic nerve pathway. It can present as ptosis, miosis, and facial anhidrosis. Potential life-threatening causes of Horner's syndrome may include head and neck trauma, carotid artery dissection, and malignancy. Meanwhile, stellate ganglion block, an interventional pain procedure, is known to cause a benign, transient presentation. Horner syndrome has also been reported as a complication following lumbar epidural anesthesia, especially in the obstetric population [1]. Given the various potential etiologies, it is important to recognize acute and unanticipated Horner's syndrome within the clinical context to facilitate the appropriate medical treatment.

Cervical epidural steroid injections (ESI) are one of the most commonly performed interventional pain procedures. Typically performed under fluoroscopic guidance, it can treat chronic pain caused by a herniated disc, spinal stenosis, and cervical radiculopathy. The most widely reported side effects are neck pain, headache, insomnia, and

vasovagal reaction, while serious complications include dural puncture, subdural hematoma, epidural abscess, spinal cord injury, and death [2]. Meanwhile, Horner's syndrome is a seldomly reported adverse event. Here we report a presentation of Horner's syndrome after a cervical ESI.

2. Case presentation

31-year-old male without significant medical history presented with acute on chronic neck and upper extremity pain consistent with left cervical radiculopathy. Cervical spine magnetic resonance imaging showed a large disc osteophyte at the entrance of the C6-7 foramen. Despite conservative treatment, the patient's pain continued to interfere with occupation and activities of daily living. Therefore, the patient decided to proceed with a cervical ESI to help with pain control and potentially avoid surgical intervention.

The patient was placed in the prone position on the fluoroscopy table. The skin entry point was identified under fluoroscopy and cleaned with chlorhexidine preparation. 3 mL of 1 % lidocaine was applied

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subcutaneously, after entry site was determined under fluoroscopic guidance. Next, an 18-gauge Tuohy needle was placed targeting the left C6-C7 interlaminar space, utilizing multiple anteroposterior and 50-degree contralateral oblique fluoroscopic views. Upon correct needle placement with the use of loss of resistance technique, aspiration confirmed absence of blood or cerebrospinal fluid through the needle. The patient denied any paresthesias. 1 mL of iohexol 240 contrast was injected and confirmed clear epidural spread with fluoroscopy in anteroposterior and oblique views. Afterwards, 80 mg (1 mL) methylprednisolone with 1 mL 1 % lidocaine was injected slowly. This was followed by 1 mL of 1 % lidocaine to flush the steroid out of the needle. Vital signs were stable throughout the procedure. The patient was provided post-procedure instruction and discharged.

Upon returning home approximately thirty minutes after the procedure, the patient reported new left sided miosis and ptosis with associated blurry vision (Fig. 1a). The patient denied left arm paresthesias, neck pain or other focal neurological deficits. Patient was recommended urgent evaluation at the emergency department for new neurological findings. Patient elected for close observation of worsening symptoms which was advised by a neuro-ophthalmologist that he knew as a colleague. Within two hours, all symptoms resolved, and the patient was evaluated by a neuro-ophthalmologist the next day for follow up (Fig. 1b).

3. Discussion

An epidural steroid injection is usually performed under fluoroscopic guidance to ensure accurate needle placement and to minimize risk of complications. Potential adverse effects are uncommon but vary widely, and include nerve damage, infection, bleeding, and other systemic reactions [3]. Although most of these events are well documented, even more unusual complications can occur. To our knowledge, Horner's syndrome following a cervical epidural injection has been reported once in literature [4].

Horner's syndrome results from a disruption of the oculosympathetic nerve pathway innervating the eye and surrounding tissues. The oculosympathetic chain consists of three main neurons: central (first order), preganglionic (second order), post-ganglionic (third order). These neurons correspond to nerves traversing from the hypothalamus to the

upper thoracic spinal cord (C8-T2), superior cervical ganglion, and the eye at the trigeminal nerve [5,6]. Horner's syndrome is a disruption of this pathway from lesions such as tumors, trauma, surgical sympathectomy, or neural blockade [5,7,8]. Loss of sympathetic tone produces the characteristic signs of ptosis, miosis, anhidrosis, and enophthalmos.

It has been postulated that cervical ESI may cause second-order, preganglionic Horner's syndrome by pharmacologic mechanisms. Variations in local anesthetic formula, mechanism of delivery, or patient constitution may increase the local diffusion of medication [6]. These pharmaceutical agents may disturb the action of the preganglionic sympathetic fibers, which exit from the ventral nerve roots (C8-T2) before passing through and ascending from the white rami communicantes [6]. This is largely due to their small size and thus increased sensitivity to drugs [4,6]. While Horner's syndrome after a cervical ESI is transient and benign, its presence should warrant the consideration for other catastrophic etiologies.

As Horner's syndrome presents with unilateral findings, it is critical to consider more serious underlying causes. One major concern is an ischemic event, as particulate steroids injected intravascularly can cause emboli formation leading to ischemia and infarction [9-12]. Scanlon et al. (2007) surveyed 30 cases of brain or spinal cord infarction, including four cases where methylprednisolone were used without local anesthetic [11]. Furthermore, the FDA's Adverse Event Reporting System (FAERS) includes 27 cases of brain infarction, ischemia, or stroke following use of methylprednisolone, triamcinolone, or betamethasone for the primary purpose of treating pain [13]. Despite fluoroscopic guidance, multiple negative aspirations for blood, and contrast usage, fatal infarction from spinal cord injury after an epidural steroid injection has been documented [14].

Hemorrhage is another acute, serious complication to consider when new neurological deficits present. This may arise from aberrant needle placement while performing epidurals [9,15]. Depending on the mechanism and severity of injury, the time course of these bleeding events may vary from hours to days after the procedure. For example, epidural hematomas, while usually self-limiting and clinically insignificant, may lead to spinal cord compression and progressive neurological dysfunction over many hours if left untreated [3]. Risk of hemorrhagic complications increase with age, coagulopathy, hypertension, and other systemic conditions [9].

Given the rare and unexpected presentation of Horner's syndrome after the cervical ESI, we advised the patient for an emergent evaluation to assess for the most concerning condition: an ischemic event. This would include a thorough neurological exam and immediate radiological testing. The current consensus for stroke evaluation relies on the time since onset. A non-contrast computed tomography (NCCT) scan of the brain is preferred at most emergency departments for rapid evaluation to exclude hemorrhage [16,17]. With the absence of bleeding and other exclusion criteria, intravenous thrombolytic therapy may begin while other diagnostic tests are obtained. In general, a computed tomography angiogram (CTA) is obtained next to illuminate any large vessel occlusion, if not done already [16]. Additionally, magnetic resonance imaging (MRI) may be useful for more precise assessment of the brain, spine and associated vasculature, although this is not always necessary [16,17]. The final decision for imaging modalities should be made case-by-case based on patient history, physical examination, and hospital resources [16]. Regardless, a neurologist should be consulted as soon as possible when stroke is suspected. Fortunately, in our case, a neuro-ophthalmologist was available to reassure the patient of the most likely benign diagnosis before further testing was performed.

4. Conclusion

In this reported case, Horner's syndrome may have been caused by the disruption of preganglionic neurons by regional flow of local anesthetics. The transient symptoms fully resolved within hours without medical intervention. A few months later, the patient underwent a



Fig. 1. Patient with left-sided Horner's syndrome is shown. These images were taken 30 minutes after left paramedian cervical ESI with miosis and mild ptosis (a), and 2 hours after cervical ESI with isocoria and symmetric eyelids (b).

second cervical ESI without use of any local anesthetics—resulting in no further complications. The steroid was also switched to dexamethasone—a non-particulate steroid. The ESI provided significant benefits in helping with pain and function, avoiding surgical intervention. While Horner's syndrome from a cervical ESI appears to be a benign, transient process, we continue to recommend emergent evaluation as it may represent a life-threatening condition.

Our practice has implemented the following protocol:

We recommend the continued use of real-time fluoroscopy, loss of resistance technique, and contrast to visualize and confirm accurate placement. Minimal local anesthetics should be used to avoid potential second-order, preganglionic Horner's syndrome. Finally, we advise caution with the use of particulate steroids to minimize risk of an embolic or ischemic event, as there is no reported benefit of utilizing particulate over non-particulate steroids in cervical ESI [18–20].

Informed consent

Informed consent to include photographs for publication was obtained from the patient, and it is retained in his medical record.

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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