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Review Article

Horner's syndrome and epidural anesthesia in labor and cesarean section

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Summary

Horner's syndrome (HS) is based on dysfunction of symphathetic nervous system at the cervical canal. The signs of the syndrome occur on the same side as the lesion of the sympathetic trunk and include: a constricted pupil, a weak-droopy eyelid, apparent decreased sweating, and with or without inset eyeball. HS has been observed as a rare complication of epidural anaesthesia in obstetrics during labour or cesarean section. In parturients, it warrants further investigation as other serious causes must be excluded, such as pancoast tumours, thoracic aortic aneurysms, carotid dissection, neuroblastoma, and brainstem vascular malformation. Management involves early diagnosis of the underlying benign condition while treatment is based on appropriate conservative observation, as most often the syndrome resolves spontaneously. However in very rare cases immediate medical or surgical management is needed.

Key words: Horner's syndrome; Epidural anaesthesia; Vaginal labor; Cesarean section.

Introduction

Pain during labor is one of the most painful experiences in life and is a result of multiple and complicated interactions (physiological, psychological, stimulative, and inhibitory) [1]. There are many factors that can have a positive or negative effect on the normal progress of labor [2]. Endogenous opiates and their main representative beta-endorphins, play a pivotal role in pain sensation [3]. Cervix dilatation and descent of the embryo's head during labor, are the cause of severe pain that stimulates the release of beta-endorphins as a defensive mechanism. Beta-endorphins concentration during labor may increase three to six times [3].

Various methods have been tested from ancient times in a fruitless effort to reduce or suppress pain during labor [4]. Epidural anesthesia is one of the most effective techniques of regional anesthesia used for pain relief, leading to excellent analgesia [5, 6]. However, epidural anesthesia is not devoid of side effects [7]. Horner's syndrome (HS) which was first reported by Kerpes *et al.* and consists of ptosis resulting from the denervation of Müller's muscle in the upper lid, miosis, and anhydrosis associated to enophthalmos and facial flushing on the affected side, is a recognized but rare complication of epidural anesthesia [8]. The exact incidence of HS is unknown. It is higher in parturients compared with the general population. It is also estimated to occur in 0.4% of vaginal laboring patients and in 4% of pregnant women undergoing cesarean section and having epidural anesthesia [8-10]. If HS occurs in non-obstetric women after epidural lumbar anesthesia, some of the clinical signs suggestive of subdural anesthesia shall be investigated [10].

The aim of this study was to investigate the incidence of HS after regional anesthesia, antepartum and postpartum, and the subsequent management and prognosis according to the current literature.

Literature research

A PubMed search covering a period of 20 years (1995-2015) using the terms "Horner's syndrome", "labor", "cesarean section", and "epidural anesthesia (EA)" revealed 30 case reports. In five of the cases, trigeminal nerve palsy and in one glossopharyngeal nerve palsy associated with HS and EA were reported.

Pathophysiology of obstetrical pain treatment

Labor pain has a neuronal mechanism with similar characteristics to other forms of acute pain, as pain signal information is transferred from peripheral receptors via a-delta and c-fibers to the posterior horn neurons of the spinal cord. From this point, those signals can follow three pathways: 1) they ascend via the spino-thalamic tract to the brain and can be perceived as pain; 2) they conduce to the anterior horns of the spinal cord and can lead to local reflexes (skeletal muscle contractions and sympathetic nerv-

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ous system stimulation); 3) they are modified in the spinal cord by descending pain suppression fibers resulting in analgesia [11, 12].

Obstetrical pain can be managed with administration of medication on three targets: 1) neurotaxis originating from the dorsal horns or the peripheral sensory nerves; 2) spinal cord neurons; 3) several described brain sites.

Regional anesthetics (bupivacaine, chloroprocaine, lidocaine, ropivacaine, and levobupivacaine) administrated via epidural anesthesia, are the most effective pain inhibitors and have a remarkable and effective action on the neuroaxis level [13-15].

Pathophysiology of HS

The sympathetic innervation of the eye is characterized by a three-neuron arc. Primary central sympathetic fibers arise from the posterolateral hypothalamus, descend uncrossed through the midbrain and pons, and terminate in the intermediolateral spinal cord at the C8-T2 level (ciliospinal center of Budge) [16, 17].

Secondary pre-ganglionic pupillomotor fibers exit the spinal cord at T1 level and enter the cervical sympathetic chain, proximal to the pulmonary apex, and the subclavian artery. The fibers ascend through the sympathetic chain and synapse in the superior cervical ganglion at the level of the bifurcation of the common carotid artery (C3-C4). Post-ganglionic pupillomotor fibers exit the superior cervical ganglion and ascend along the internal carotid artery. Shortly after, the postganglionic fibers leave the superior cervical ganglion, vasomotor, and sudomotor fibers branch off, traveling along the external carotid artery to innervate the blood vessels and sweat glands of the face [16, 17].

The tertiary pupillomotor fibers ascend along the internal carotid artery entering the cavernous sinus. The fibers then leave the carotid plexus to join the abducens nerve (cranial nerve [CN] VI) in the cavernous sinus and enter the orbit through the superior orbital fissure along with the ophthalmic branch (V1) of the trigeminal nerve (CN V) via the long ciliary nerves. The long ciliary nerves then innervate the iris dilator and the Müller muscle.

It is easy now to understand that HS is caused by a deficiency of the sympathetic activity, caused by a lesion at the ipsilateral side of the symptomatology-affected side, resulting in mild to moderate ptosis, slight elevation of the lower lid, miosis, and dilation lag. Furthermore sweating disorders can be noted ipsilaterally: disorder of a primary neuron due to lesion of the hypothalamo-spinal tract, resulting in anhidrosis of the ipsilateral body side.

Disorder of a secondary neuron due to pre-ganglionic lesions is characterized by anhidrosis of the ipsilateral face side. Disorder of a tertiary neuron due to post-ganglionic lesions, is characterized by absent or limited anhydrosis above the ipsilateral brow [16, 17].

Concerning HS, due to epidural anesthesia during labor or especially in a cesarean section, it has been reported to be caused by cephalad spread of the local anesthetic after a top-up injection of local anesthesia through an epidural catheter [8]. In this manner, the sympathetic chain of neurons at C8-T1 level is interrupted before the entrance to the superior cervical ganglion [8].

This cephalad spread, is a result of epidural veins distension and epidural space volume decrease due to pregnancy as there is partial occlusion of the vena cava inferior that diverts the blood through the epidural plexus venosus, and increases pressure by uterine contractions (spontaneous or due to medical agents), lateral decubitus of the patient during epidural administration, hypobaric local anesthetics, and miscannulation [8, 10, 18-20]. Low dose of local anesthetic concentration inhibits the development of HS [8].

Aside from epidural administration, HS is associated to stroke, face-neck trauma, tumor, vascular headache syndromes, trigeminal autonomic cephalalgias, intraoral anaesthesia, carotid dissection, brachial plexus block, cervical plexus block, while in pediatric patients it might appear as a result of forceps/vacuum delivery or traction, and neuroblastoma [21, 22]. There is no HS case reported during the Valsalva manoeuvre during labor, although there is an increased risk of stroke from paradoxical emboli. HS is not associated to hypotension that could lead to side effects concerning both the mother and the embryo [23]. HS cases subsequent to carotid dissection, which occurs in a variety of incidents like minor trauma, sneezing, and coughing was rarely reported [24].

Discussion

The most cited articles in the literature research are summarized in Table 1. Rabinovich *et al.* found a low HS incidence of 0.13%. Moreover, HS was not confirmed as a subsequent side effect of EA [25-28] in most of the studies. According to current literature, the appearance of HS is unpredicted and is considered to be a benign and transient condition that does not usually require extensive investigation. However, increased vigilance is advised as high sympathetic blockade can lead to cardiovascular collapse and ocular neurologic deficits. These, may cause anxiety and immediate obligatory diagnostic and treatment management may be needed [21]. The duration of the symptoms range from few minutes (25 minutes) to several hours, with a mean time of 125 minutes and often spontaneously resolving within a mean of 215 minutes [21]. The typical symptoms usually occur unilaterally, in some cases bilateral in 11.5% [29]. The reasons are the following: slow injection of small volumes, pregnant positioning, baricity of local anesthetic, congenital transverse septum in the epidural space, and tip of the epidural catheter [23, 30]. The most commonly HS associated symptoms include nasal stuffiness, blurry vision, strange feeling of the eye, and maternal hypotension dyspnea depending on interruption of the symphathetic chain of neurons at C8-T1. Lastly, in some cases,

Table 1. — *Horner syndrome in pregnant women and labour modus.*

Jeret *et al.*, 1995 [36]	Postpartum	Vaginal delivery
Paw *et al.*, 1998 [37]	Antepartum	Vaginal delivery
Biousse *et al.*, 1998 [38]	Antepartum	Vaginal delivery
De la Gala *et al.*, 2007 [54]	Antepartum	Cesarean section
Zahn *et al.*, 2002 [39]	Antepartum	Cesarean section
Robert *et al.*, 2002 [40]	Antepartum	Cesarean section
Holzman *et al.*, 2002 [56]	Antepartum	Vaginal delivery
Vareala *et al.*, 2007 [41]	Antepartum	Cesarean section
Linch *et al.*, 2006 [35]	Antepartum	Vaginal delivery
De la Calle *et al.*, 2004 [43]	Antepartum	Cesarean section
Jadon *et al.*, 2014 [42]	Antepartum	Cesarean section
Sinha *et al.*, 2010 [44]	Antepartum	Cesarean section
Al Mustafa *et al.*, 2010 [29]	Antepartum	Vaginal delivery
Sharma *et al.*, 2010 [23]	Antepartum	Vaginal delivery
Rabinovich *et al.*, 2010 [25]	Antepartum	Vaginal delivery
Jiménez-Caballero *et al.*, 2009 [49]	Antepartum	Cesarean section
Rohner *et al.*, 2008 [45]	Antepartum	Cesarean section
Collier *et al.*, 2008 [46]	Antepartum	Vaginal delivery
Theodosiadis *et al.*, 2006 [47]	Antepartum	Cesarean section
Rowleyb *et al.*,2009 [48]	Antepartum	Cesarean section
Ballabriga *et al.*, 2011 [55]	Antepartum	Cesarean section
Barbara *et al.*, 2011 [8]	Antepartum	Vaginal delivery
Goel *et al.*, 2011 [50]	Antepartum	Cesarean section
Wong *et al.*, 2003 [21]	Antepartum	Cesarean section
Merrison *et al.*, 2004 [24]	Postpartum	Vaginal delivery
Choudhari *et al.*, 2004 [51]	Postpartum	Vaginal delivery
Chandrasekhar *et al.*, 2003 [9]	Antepartum	Vaginal delivery
Narouze *et al.*, 2002 [32]	Antepartum	Vaginal delivery
Friedman et al 2002 [52]	Antepartum	Cesarean section
Anadon et al 1999 [53]	Antepartum	Cesarean section

cranial nerve palsies have been noted; in 10% particularly hypoglossal oculomotor, trigeminal, and facial nerves [8, 28, 31, 32].

Postpartal HS might also occur by unilateral spontaneous carotid arterial dissection without association to labour, with symptoms like head or neck pain, partial HS, subsequent cerebral or retinal ischemia [33-34]. Partial HS includes miosis, ptosis, but not facial anhidrosis. The reason is that facial sweat glands receive their innervation from the symphathetic plexus that surrounds the external carotid artery [8, 33, 34].

Although HS is a benign condition, there are reports which confirm recurrence and persistence of HS, possibly depending on hemodynamic instability subsequent from a high sympathetic block. However the exact reason is not clear and should be object for further investigations in the future [35]. Surgical intervention was required in only one reported case of persistent HS to correct ptosis six months later [8].

According to a recently published report, the epidural infusion technique plays the main role and influences HS occurrence. The previously reported factors like body mass index, position during EA, agent EA solution concentration are not risk factors for HS [24].

Generally the therapeutical management after the HS diagnosis is only conservative. In cases in which a high sympathetic block is suspected, the epidural medication administration must be immediately stopped [8]. A subdural location of the catheter from EA and a carotid dissection must be excluded [8]. An extensive diagnostic workup is not necessary [8]. HS is an unpredictable and unpreventable situation [25].

Conclusion

The early recognition of HS protects the pregnant women from unnecessary anxiety and leads to satisfying perinatal results. The significance and treatment of HS associated with EA in obstetrics and in ante- and postpartum deserve further multicentric future studies.

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