Horner’s syndrome in the prone position – a case report

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Abstract: We report the case of a patient who developed a post-operative Horner’s syndrome after non-cervical surgery. Part of the surgery was in the prone position. A hypothesis incriminating the prone position as being a possible cause of this syndrome is suggested.

Key words: Horner’s syndrome; post-operative; prone position.

A 42 year-old female patient, with a height of 165 cm and weighing 77 kg (BMI 28.8 kg/m2), was admitted to the hospital for a bodylift (circular abdominoplasty and lipposuction) procedure. She had a history of mood disorders and had undergone previous surgery for appendectomy and gastric-bypass. Her medications consisted only of daily escitalopram.

Sufentanil, xylocaine and propofol were used for induction of anesthesia. Muscle relaxation was achieved using atracurium and the patient was successfully intubated at first attempt (Cormak grade: I). Surgery consisted of 3 phases, starting in the prone position, then switching to the supine position, and finishing in the sitting position to complete the abdominoplasty. It was uneventful and the necessary precautions and padding were used at each step for patient positioning. The procedure lasted 6 hours. No incidents occurred during extubation or in the recovery room. During the procedure, head and neck position were regularly checked. A neutral position was maintained at all times in the theatre, as well as in the recovery room.

On postoperative day 2, the patient complained of headache and blurred vision. On clinical examination, right-sided ptosis, myosis and ipsilateral facial anhydrosis were noted. Ptosis was not present before surgery. No other symptoms were present. Clinical examination was normal with regard to cranial nerves, and no anomaly was noticed on examination of the neck. At ophthalmological examination, visual and oculomotor functions, as well as facial sensitivity were normal. Pharmacological testing using cocaine 10% showed a lack of dilation of the right pupil. A diagnosis of Horner’s syndrome was made. Magnetic resonance imaging of the head and neck, as well as computed tomography angiography of the neck, were also normal. These imaging studies covered the pulmonary apex, and no lesions such as a Pancoast tumor were visible. A neurological advice was obtained, and the neurologist confirmed the Horner’s syndrome.

Four weeks later, the patient had partially recovered, and a favorable prognosis was expected.

Discussion

Horner’s syndrome, or oculosympathetic paresis, is the result of the interruption of the sympathetic transmission to the eye, and surrounding structures. The syndrome was initially described by Claude Bernard in 1852, and later by Horner in 1862. The syndrome is often called Horner’s syndrome in English speaking countries, while, in French speaking countries, the term of Claude Bernard-Horner is preferred.

The sympathetic transmission to the eye (oculosympathetic pathways) is a three-neuron pathway (Fig. 1). The first-order neuron originates from the hypothalamus. It descends uncrossed through the brain stem, and synapses in the cervical spinal cord at the C8-T2 level, in the so-called ciliospinal center of Budge. The second-order neuron leaves the cervical spinal cord, through the brachial plexus, over the ipsilateral lung apex and then ascends to the superior cervical ganglion (SCG). The SCG is situated close to the angle of the mandible and the bifurcation of the common carotid artery. The third-order
The diagnosis of the syndrome is mainly a clinical one, and does not require any biological or imaging studies. However, owing to life-threatening causes that might be present, neurovascular imaging is advised to exclude them (2). Pharmacological testing might be helpful to localize the lesion, or evaluate the degree of myosis.

Horner’s syndrome after surgery is a rare complication. However, surgical trauma is one of the most common etiologies. Carotid artery endarterectomy (3) and cervical spine surgery (4) through an anterior approach carry the highest incidence. Gonzalez-Aguado and colleagues (1) have also described several cases of Horner’s syndrome following different cervical and otolaryngological procedures. The syndrome may originate either from a direct trauma along the oculosympathetic pathway, or from an indirect stretching of the nerve structures secondary to inflammation and hematoma formation.

Some common anesthesia techniques can also be at the origin of a Horner’s syndrome. Local anesthetic diffusion during an interscalenic brachial plexus blockade may be one cause. High sympathetic blockade following thoracic or lumbar epidural analgesia (even during labor) have also been described as resulting in reversible Horner’s syndrome (5). A neuraxial anesthesia-induced Horner’s syndrome might be a warning sign for a high level blockade. In one study, Claydon (6) found a Horner’s syndrome incidence of 1.33% with epidural analgesia for labor. Indeed, in pregnant women, an epidural venous engorgement is responsible for a high cephalic spread of the anesthetic agent.

Direct trauma to the sympathetic pathway can also result from an internal jugular vein cannulation. In that case, the lesion may be irreversible, but transient cases with full recovery following a three-month period have also been reported (7). Placement of chest-tubes, particularly if placed on the upper part of the thorax, can also disrupt the oculosympathetic pathway. In a case described by Levy et al., the tip of a chest tube was found to be close to the stellate ganglion, and its removal lead to complete reversal of the syndrome (8).
Position-induced Horner’s syndrome secondary to lateral decubitus position during general anesthesia has also been described (9, 10). In the lateral decubitus position, the cervical spine and the head might be misaligned with respect to the rest of the body, leading to excessive stretch on the non-dependent oculosympathetic pathway. Thompson described a case of Horner’s syndrome secondary to prolonged and unchanged lateral flexion position during an alcohol-induced coma (9). More recently, Atkinson et al. (10) reported another case of Horner’s syndrome following general anesthesia for gynecological surgery in the supine position. The latter was attributed to stretch injury following prolonged head tilt. These cases stress upon the importance of head positioning during general anesthesia. In our case, surgery occurred through three phases; first in the prone position lasting for two hours, second in the supine position lasting for three more hours, and finally in the sitting position for abdominal closure during one supplementary hour. The head was maintained in a neutral position at all times, respecting the alignment of both the cervical and thoracic vertebral columns. In the semi-sitting position, the head was maintained using a special head-rest, and no head rotation was allowed. In our patient, the cause of the syndrome can therefore not be attributed to head and neck rotation, but rather to the prone position. It seems likely that the syndrome involved the 3rd order neuron. However, specialized pharmacological testing would be necessary to prove that the 3rd order neuron is involved. These tests were proposed to the patient, but were refused.

Conclusions

Cases of position-induced Horner’s syndrome have been previously reported. It has always been attributed to a stretch mechanism secondary to rotation or malposition of the neck. However, in our case, patient positioning was checked regularly. The head and neck remained aligned with respect to the rest of the body, in such a way that the axis of the vertebral column was maintained, and no head rotation was allowed. One possible explanation of this complication is probably stretch and compression of the oculosympathetic pathway in the upper part of the thorax during the prone position. The oculosympathetic pathway passes over the apex of the lung, and prolonged excessive pressure on the thorax could result in its compromise. Furthermore, the abduction of the shoulders causes a certain degree of stretch on the upper thorax. Last, a greater pressure is exerted on the thorax in the prone position than in the supine position. All these conditions might be similar to those seen in case of thoracic outlet syndrome. For example, unusual shoulder posture and elevation of the arms can be responsible for the occurrence of symptoms related to a thoracic outlet syndrome. These conditions are grouped in the prone position. Therefore, these factors together with the length of surgery might have contributed to the development of the complication. However, at distance from the intervention, the patient was tested negative for signs of thoracic outlet syndrome. Our hypothesis of a prone position-induced Horner’s syndrome is therefore made after exclusion of all other possible causes. An anatomical variation, for example third order neuron having a superficial trajectory that renders it susceptible to compression might be another hypothesis. Unfortunately, the latter is difficult to verify.

Anesthesiologists need to be aware of Horner’s syndrome. They must know that it can be linked with life-threatening causes, and that it can occur after non-cervical surgery in the prone position.

References