Unilateral blindness following superior laryngeal nerve block for awake tracheal intubation in a case of posterior cervical spine surgery

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INTRODUCTION

Superior laryngeal nerve block (SUPLANEB) is a popular airway anesthetic technique utilized to facilitate awake endotracheal intubation in patients with cervical spine instability.¹⁰,¹¹ SUPLANEB and translaryngeal injection are typically performed easily and safely. However, if not performed by an expert, SUPLANEB carries the risk of subcutaneous emphysema, pneumomediastinum, pneumothorax, hemotoma formation, and laryngospasm.¹⁵ In addition, the local anesthetic...
toxicity of SUPLANE is includes local neurologic complications including laryngeal anesthesia, dysphagia, and dysphonia (e.g., they usually recover within a few hours). Rare central nervous system toxicity following SUPLANE can include convulsions attributed to the accidental injection of local anesthetic directly into the carotid artery.

Here, we present a patient undergoing atlantoaxial fusion who required an awake endotracheal intubation for general anesthesia. Postoperatively, the patient was immediately blind in the left eye and also complained of complete trigeminal anesthesia and ipsilateral hearing loss. This case and the possible mechanisms for these deficits are reviewed.

CASE REPORT

A 25-year-old neurologically intact male was admitted for elective fusion of a nonunion C2 fracture attributed to a diving accident 9 months earlier. Four months later, when neck pain appeared and worsened, both CT and MR studies demonstrated a type II odontoid fracture with a chronic nonunion accompanied by C1-C2 subluxation but without cord compression [Figure 1].

Surgery

Before the C1-C2 posterior arthrodesis, the patient underwent an awake endotracheal intubation requiring SUPLANE. The patient received hydroxyzine 50 mg orally, and the anesthesiologist performed a superior laryngeal nerve block. A 21-gauge needle (1 ½ inch) attached to a syringe containing 5 mL of 1% lidocaine was used once on each side (total injection of no more 10 mL of lidocaine), following which the patient was intubated using a video laryngoscope. The posterior cervical C1-C2 fusion was uneventful.

Nevertheless, after recovering from the general anesthesia, the patient complained of the left eye blindness without ocular pain, periorbital swelling, or oculomotor paresis; he also demonstrated left-sided hearing loss and facial numbness (all trigeminal territories). The direct eye examination revealed the left side anterior segment and fundus was normal, but confirmed complete left-sided visual loss, isolated direct mydriasis, and intact consensual light reflex.

Diagnostic studies

The STAT cranio-orbital computed tomography scan showed no acute intracranial or oculo-orbital lesions, but demonstrated mild bifrontal, interhemispheric, parasellar, sellar, and prepontic pons/midbrain; there was no skull base lesion [Figure 2]. The subsequent CT angiogram documented air in the left carotid sheath, near and along the carotid bifurcation, the internal carotid artery (ICA), and all around the internal jugular vein (IJV) [Figure 3]. There was also diffuse subcutaneous emphysema in the left paralaryngeal area and in the posterior cervicothoracic regions [Figure 4]. No intracranial hematomas or extracranial vascular lesions (no vasospasm) were present.

Treatment

The patient was placed in strict horizontal position and treated with inspired pure oxygen plus systemic steroid pulse therapy (120 mg of methylprednisolone each 6 h) and nimodipine (30 mg each 6 h). The first postoperative day, the visual acuity remained unchanged, but nearly completely recovered the left hearing loss and facial hypoesthesia in the V2 distribution. Brain and orbital MR studies were normal. Optical coherence tomography (OCT) demonstrated postischemic retinal edema [Figure 5] with permeability of the intraocular blood vessels while retinal angiography showed delayed choroidal filling [Figure 6]. Flash visual evoked potentials were absent, while the electroretinogram revealed decreased potentials amplitudes.

Figure 1: Spinal cervical lateral plain radiography (a), sagittal computed tomography scan (CT scan) (b), and sagittal magnetic resonance imaging on T2-weighted image (c) showing type II nonunion odontoid fracture and C1-C2 subluxation without bulbo-medullar compression. Postoperative sagittal CT scan following C1-C2 posterior cervical spine fusion (bicortical iliac crest allograft with laminar wiring were used).
The pneumocephalus and subcutaneous emphysema completely resolved on postoperative day 3. At the time of discharge on the 4th postoperative day, there was a complete resolution of symptoms excepting for the left blindness and the V1 trigeminal hypoesthesia; the patient was sent home on nimodipine (120 mg/day) and oral steroid therapy (60 mg prednisolone) with a tapering dose over the next 4 weeks. Two months later, he remained blind in the left eye without any return of function.

DISCUSSION

Postoperative visual loss following prone spinal surgery is rare. When it occurs, most anesthesiologists accuse the surgeon of direct external mechanical compression of the eye while other etiologies include ischemic optic neuropathy, central retinal artery occlusion, central retinal vein occlusion, cortical blindness, low blood pressure, excessive blood loss, blood dyscrasias, hypothermia, coagulopathic disorders, and microvascular embolism.

Left-sided blindness

After an extensive ophthalmological exploration, it seems that our patient had postoperative ischemic retinal edema secondary to central retinal artery occlusion. The iatrogenic intravascular infusion was likely the cause of pneumocephalus. Subcutaneous emphysema following a translaryngeal injection of local anesthetic has only rarely been reported in literature. As there was a big air bubble in the left
paralaryngeal area in this patient, our hypothesis was that the air leaked into the subcutaneous tissue through a left lateral needle track created following the superior laryngeal injection. It was then driven into the subcutaneous tissue of the cervicothoracic region and also in deep cervical and parapharyngeal areas. Another possibility was that the air was injected directly into the left carotid sheath, and it extended along the ICA and the IJV to the skull base. Thus, the pneumocephalus was due to intracranial gas extension through the carotid canal, jugular foramen, foramen lacerum, or foramen oval through the skull base.[4]

Upper cranial neuropathies

The upper cranial neuropathies (II, V1, V2, V3, and VIII cranial nerves) were likely attributed to pneumocephalus in the sellar and laterosellar regions, the abnormal extracranial air into the left carotid sheath, and the extensive cervicothoracic subcutaneous emphysema. They were also likely due to the accidental injection of the local anesthetic inside/around the ICA and/or the IJV (e.g., extending into the left cavernous sinus). This would explain the partial cavernous sinus syndrome presented by the patient. Another hypothesis is that the SUPLANEB resulted in transient vasospasm, emboli, or hypoperfusion of the ICA, thus directly resulting in occlusion of the blood supply to the left optic nerve.[3]

Future recommendations

When SUPLANEB is performed, the anesthesiologist should take care not to push the needle too far or too lateral to avoid reaching the carotid sheath.[10] It is also mandatory to draw back on the syringe and aspirate before anesthetic drugs injection. Furthermore, for patients with difficult surface anatomic landmarks, using ultrasound can be useful for performing SUPLANEB.[13]

CONCLUSION

Blindness and upper cranial nerve neuropathies must be considered as potential complications of the SUPLANEB utilized for endotracheal awake intubation for spinal neurosurgical/other procedures.

Acknowledgments

The authors thank Pr. Youssef Qamous (Department of Anesthesiology) and Pr. Younes Aissaoui (Department of Intensive Care Medicine) for their valuable suggestions in the management of this patient.

Declarations of patient consent

The authors certify that they have obtained all appropriate patient consent.

Financial support and sponsorship

Publication of this article was made possible by the James I. and Carolyn R. Ausman Educational Foundation.

Conflicts of interest

There are no conflicts of interest.

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How to cite this article: Akhaddar A, Baallal H, Hammoune N, Bouabadi S, AdraouiA, Belfquih H. Unilateral blindness following superior laryngeal nerve block for awake tracheal intubation in a case of posterior cervical spine surgery. Surg Neurol Int 2020;11:277.