Case Report

Reversible Respiratory Arrest without Anesthesiology Assistance after Retrobulbar Injection-A Case Report

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1. Introduction

Retrobulbar anesthesia is associated with local adverse events, including scleral perforation [1], endophthalmitis, central retinal artery occlusion, central retinal vein occlusion, combined retinal artery/vein occlusion [2] and contralateral amaurosis [1-4]. Potential systemic adverse events include convulsions, hemiparesis [3], and brainstem anesthesia, the latter noted in 0.006% (1/18,000) of patients undergoing retrobulbar/peribulbar anesthesia [1]. Brainstem anesthesia can result in respiratory depression and, if untreated, death [5-6].

2. Case Report

An 88-year-old man presented to the Wills Eye Hospital Retina Service with right eye pain for two weeks and vision loss for six months. Baseline best-corrected vision was 20/1600 in the right eye, and 20/20 in the left eye. The intraocular pressures were 22 mm Hg in the right eye and 16mm Hg in the left eye. The right eye had a hemorrhagic central retinal vein occlusion with pronounced secondary iris neovascularization and partial angle closure by fibrovascular tissue. The left anterior segment and fundus exams were normal. Full-scatter panretinal photocoagulation was proposed for the right eye. A 4cc retrobulbar injection of 1% lidocaine was given through the right inferotemporal lid with the globe in primary position using a 25-gauge, 1.5-inch, sharp needle. A "pop" was felt, presumably upon entering the extraocular muscle cone. A second "pop" causing temporary discomfort was

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encountered just prior to injection. Injection resistance was judged to be greater than usual. Approximately 5 minutes after injection, the patient stated his fellow eye vision was blurred, suggesting the retrobulbar lidocaine injection had gained access to the subarachnoid space via perforation of the vaginal sheaths surrounding the optic nerve He was transported by wheelchair to a connected Ambulatory Surgical Center (ASC). During the two-minute trip, he developed increasing somnolence, confusion and apnea. Arriving after hours at the ASC, anesthesiology services were unavailable.

The apneic episodes lasted at least a minute, the longest time the patient was permitted to remain without stimulation. With verbal encouragement and mild tactile stimulation every 30 seconds thereafter, however, the patient would awaken and breathe, then immediately fall back to sleep. He was placed on a stretcher and transferred to the Emergency Room at Pennsylvania Hospital. By 20 minutes after the injection, he developed bilateral facial nerve paralysis and was unable to move his hands or legs, suggesting the lidocaine had traveled farther down the brainstem. Despite marked somnolence and lack of spontaneous respirations, he continued to breathe with verbal and tactile stimuli. Breathing became normal by 90 minutes post-injection and cognitive and motor faculties improved. By six hours post-injection, he returned to his pre-injection mental and physical condition. One day later, the patient could not recall events occurring within two hours after retrobulbar injection. Long-term sequelae were absent.

3. Discussion

This patient experienced a lidocaine injection into the subarachnoid space within the meningeal sheaths surrounding the optic nerve posterior to the globe [4]. This occurs in approximately 1/18,000 cases of retrobulbar anesthetic injection [5]. The authors were unable to find another report showing that persistent tactile and verbal stimulation alone can allow a patient to survive the resultant respiratory arrest. Often intubation is required [5-6], which we did not have available initially. We are confident that, without external stimulation, our patient would have experienced a complete respiratory arrest leading to death. In the absence of resuscitation equipment, our case suggests that frequent arousal can prevent complete apnea and death. Nonetheless, we believe intubation and assisted respiration until the retrobulbar anesthetic agent wears off is a reasonable and, likely preferable, alternative [4-6]. Early recognition is key to a favorable outcome. The first symptom, as herein, is often loss of vision in the fellow eye within minutes following the retrobulbar injection [4]. This occurs because the anesthetic agent diffuses within the subarachnoid space surrounding the optic nerve to the chiasmatic cistern where the optic chiasm containing nerve fibers to the fellow eye also becomes anesthetized [6]. The drug then travels to the pons/medullary respiratory center 4-5 cm beyond within minutes or less, causing respiratory depression due to anesthesia of the central nervous system tissue.

Wang et al. [6] demonstrated that injection into an optic nerve sheath requires 138 mm Hg pressure, versus 35 mm Hg pressure when injecting into retrobulbar adipose tissue. Thus, greater resistance than normal should suggest the possibility of an intra-nerve sheath subarachnoid injection. Inadvertent subarachnoid anesthetic injection, if

recognized and treated, typically results in complete recovery. Failure to recognize and treat it can result in respiratory arrest and death. Even if resuscitation equipment and/or anesthesia assistance are unavailable, persistent verbal and tactile stimulation until the anesthetic effect upon the respiratory center resolves may keep a patient's respirations active and save his or her life.

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Conflict of Interest

The authors have no conflicts of interest with the information presented in the manuscript.

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