Pulmonary edema following retrobulbar block

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ABSTRACT

We report a patient requiring keratoplasty who developed acute pulmonary edema following the administration of a retrobulbar block for anesthesia. A variety of factors that may have been implicated in the genesis of this complication are discussed.

Key Words: local anesthetic, pulmonary edema, retrobulbar block

Local anesthesia is frequently used by ophthalmic surgeons to reduce anesthetic-related complications and length of hospitalization. However, local anesthesia is not without risk. A case report of a patient who developed acute pulmonary edema following a retrobulbar block is presented. No previous reference to this complication has been documented.

CASE REPORT

A 55-year-old female was admitted to the hospital for a penetrating keratoplasty. Past medical history was unremarkable and physical examination and laboratory studies were within normal limits. She was scheduled to have the procedure performed under retrobulbar block.

Immediately prior to the administration of the block, an 18-gauge intravenous catheter was inserted in her left forearm. Electrocardiographic monitor and blood pressure cuff were attached. Five milligrams of diazepam were then administered intravenously. Five minutes later the retrobulbar block was performed using an Atkinson needle; 3.5 ml of 0.5% bupivacaine and 3.5 ml of 2% lidocaine with 150 international units of hyaluronidase was administered.

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Three minutes later the patient complained of lightheadedness and dizziness. She then lost consciousness and became apneic. The patient was immediately ventilated with a face mask and 100% oxygen. Over the next three minutes she developed a supraventricular tachycardia of 140 beats per minute and her blood pressure rose to 170/100. Eight minutes after administration of the block, the patient had a focal seizure involving the right arm. An additional 5 mg of diazepam was administered and the seizure ceased.

After 30 minutes of artificial ventilation, the patient began to awaken and started to breathe spontaneously. At this stage, it was noted that she was tachypneic with a respiratory rate of 25 breaths per minute. The blood pressure had dropped to 125/80 and the heart rate to 80 beats per minute. Chest examination revealed bilateral basal crackles. There was no gallop rhythm or raised jugular venous pressure. Blood gases revealed a pH of 7.25, PaO_2 of 58 mm Hg, $PaCO_2$ of 49 mm Hg, base deficit of six, and standard bicarbonate of 19. A chest x-ray demonstrated evidence of acute pulmonary edema.

Ten milligrams of furosemide were administered. Surgery was postponed and the patient was thereafter transferred to the ICU where she received a further 20 mg of furosemide. By the following morning the patient showed no evidence of clinical pulmonary edema. Blood gases at this time revealed a pH of 7.36, PaO_2 of 98 mm Hg, $PaCO_2$ of 36 mm Hg, base deficit of 0, and standard bicarbonate of 24 on room air. Electrocardiogram and cardiac enzymes showed no evidence of myocardial infarction. She was discharged two days after admission with no complications.

A keratoplasty was successfully performed on the patient one month later under retrobulbar block.

DISCUSSION

In recent years there have been a number of reports of complications occurring with the administration of retrobulbar blocks in ophthalmic surgery.[1-5] The most common manifestation has been loss of consciousness associated with respiratory arrest. The cause of this has been elucidated by Drysdale.[6] He showed that the complications arise from direct injection of the local anesthetic agent into the subarachnoid space, affecting the brain stem. Invariably, with the institution of resuscitative measures, the outcome has been good. The period of coma and apnea has generally been less than 45 minutes. As far as is known, patients have suffered no untoward sequelae.

In our patient, the sequence of events was initially similar, with the development of coma and respiratory arrest because of subarachnoid injection. But the patient also developed unexplained hypertension, tachycardia, and acute pulmonary edema.

It is possible that the local anesthetic solution reached the fourth ventricle, depressing the area postrema, the vagal nucleus, and possibly the nucleus solitarius. The resultant parasympathetic inhibition led to hypertension, tachycardia, sympathetic discharge, and pulmonary edema. But it is unlikely that the local anesthetic solution selectively affected the vagal area of the brain stem alone.

Another hypothesis is that the lidocaine used for the block inadvertently contained epinephrine. This may explain the sympathetic reactions that occurred and the development of pulmonary edema. However, the amount of epinephrine contained in 3.5 ml of 1% lidocaine with epinephrine (1:200,000) should not give rise to acute pulmonary edema.

A third explanation may be that the hyaluronidase used with the local anesthetic caused the problem. It has been stated that hyaluronidase is nontoxic and without side effects.[7] But allergic responses to the drug have been described.[8] Effects of hyaluronidase instillation directly into the subdural space have not been studied.

Cardiogenic pulmonary edema secondary to acute myocardial infarction could be hypothesized, but physical examination, laboratory tests, and the electrocardiogram revealed no evidence of this abnormality.

Finally, the patient may have aspirated during the period of unconsciousness although no gastric aspirate was noted in her pharynx or obtained from the tracheal aspirate.

It would seem that the local anesthetic was injected into the subarachnoid space. This produced the convulsions, apnea, and coma. Despite the various hypotheses, we do not have an adequate explanation for the development of pulmonary edema in this patient. This case serves to reiterate that retrobulbar injection of local anesthetic agents in ophthalmic surgery is not without hazard. Full monitoring and resuscitative equipment should be on hand to deal with any untoward complications that may occur.

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