Venous Air Embolism During Air/Fluid Exchange: A Potentially Fatal Complication

Intraoperative venous air embolism (VAE) has been described in a number of surgical specialties. It occurs if air is drawn into open veins when the operative field is above the level of the heart, a situation traditionally associated with neurosurgical procedures performed with the patient in the sitting position. Venous air embolism can also occur when air is forced into the venous system under pressure. Depending on the amount of air and the rate at which it enters, there can be profound effects on the patient’s hemodynamics. Venous air embolism had not been described in ophthalmology until recently, with 3 cases of intraoperative venous air embolism occurring during air/fluid exchange, 1 of which resulted in death. All of these cases have been described individually in the anesthesia literature. This article is designed to create awareness of the complication in the ophthalmic literature.

Report of Cases. Two of the VAE cases occurred during vitrectomy for removal of an intraocular foreign body (IOFB). In the first case, a 17-month-old child underwent surgery to remove an IOFB that had impaled the optic nerve. To avoid hemorrhage from the central retinal vessels, the infusion pressure was raised to 88 mm Hg prior to extracting the IOFB. Once the IOFB was removed, an air/fluid exchange was performed at the same pressure. Within a few minutes of beginning the exchange, the child became hypotensive, with decreasing oxygen saturation and a drop in end-tidal carbon dioxide from 37 to 10 mm Hg. There were no other identifiable causes of VAE such as air in the intravenous tubing. The air/fluid exchange was halted, and the eye was returned to a fluid-filled state. The patient recovered within 2 hours without any identifiable complications.

The second case involved a 51-year-old man who underwent surgical removal of an IOFB in the right eye. The IOFB was embedded in the ciliary body, and attempts to remove it resulted in hemorrhage that precluded visualization. An air/fluid exchange was performed at 30 mm Hg, which allowed better visualization, and the IOFB was extracted. However, 5 minutes after the air/fluid exchange began, the patient’s end-tidal carbon dioxide dropped to 18 mm Hg, along with progressive desaturation and hypotension. The patient was noted to have a millwheel murmur, which is associated with large amounts of intracardiac air. The air infusion was halted, and the eye was closed but the patient developed cardiac arrest. The patient was subsequently identified as having a patent foramen ovale, which allowed air into the arterial circulatory system, resulting in multiple arterial emboli. The patient developed a myocardial infarction and multiple organ failure and died 4 weeks later.

In the third case, a 55-year-old man underwent a vitrectomy for retinal detachment. As the air/fluid exchange was initiated, the patient developed a large choroidal detachment suggesting that the infusion cannula had migrated into the choroidal space. The patient then developed a millwheel murmur, along with a drop in oxygen saturation, systemic blood pressure, and end-tidal carbon dioxide. Once the air infusion was discontinued, the patient’s hemodynamic status returned to normal over 20 minutes. The surgery was halted, and the patient had no systemic complications. Two weeks later he underwent uneventful repair of the retinal detachment.

Comment. Venous air embolism is a potentially devastating complication that occurs most commonly in neurosurgical procedures that involve operating with the patient in a seated position, which increases the risk of air being drawn into the intracranial venous sinuses. It has also been recognized in procedures that involve using air or other gases to insufflate the surgical site, such as with intraabdominal laparoscopic surgery. It is for this reason that the gas used for laparoscopic surgery is carbon dioxide—it is absorbed into the blood stream much faster than air and the risk of VAE is reduced.

If air enters the circulatory system in small amounts or at a slow rate, it is possible for it to be absorbed into the blood or removed via the alveoli without causing significant problems. When the amount of air is large, circulatory obstruction occurs, as the air blocks the right ventricular outflow tract. The lack of pulmonary perfusion results in increasing functional enlargement of the airway dead space, which causes the characteristic drop in oxygenation and end-tidal carbon dioxide. The absolute amount of gas that can be tolerated in the venous system is unknown but accidental injections of air between 100 to 300 mL have been reported to be fatal. It has been estimated that the air flow through a 20-gauge infusion cannula can reach 1.6 L/min, assuming a pressure differential of 20 mm Hg between the infusion pressure and venous pressure. It would take very little time for a potentially fatal amount of air to enter the circula-
tory system during air/fluid exchange if the air had direct access to the venous circulatory system.

The clinical situation is further complicated by a patent foramen ovale (which is present in 23%-45% of adults, based on autopsy studies). A patent foramen ovale allows trapped air to access the arterial circulatory system through the opening between the right and left atrium. This can result in air emboli throughout the systemic arterial circulatory system, causing cerebral, cardiac, and visceral infarction, as occurred in the second case.

Treatment of VAE consists of immediately flooding the operative field with fluid to stop entry of the air. Additional measures would be at the discretion of the anesthesiologist and include increasing inspired oxygen, discontinuing nitrous oxide, if it is being used, inotropic support, and aspiration of air if a right atrial catheter has been placed. Although this complication is rare, ophthalmologists should be aware that it can occur during an air/fluid exchange, especially in the setting of significant trauma. During retinal cases, the anesthesiologist may not be fully aware of maneuvers happening within the eye, and the operating surgeon may be the first to recognize this problem if a patient becomes hemodynamically unstable after an air/fluid exchange is initiated. Promptly terminating the air infusion and returning the eye to a fluid filled state may help avoid systemic morbidity.

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Clinicopathologic Review of Enucleated Eyes After Intra-arterial Chemotherapy With Melphalan for Advanced Retinoblastoma

Retinoblastoma is a rare disease with only 250 to 300 cases diagnosed per year in the United States. Over the last 15 to 20 years, the long-term survival rates have been up to 99% in the developed world with aggressive treatment including systemic chemotherapy combined with focal laser therapy. Newer treatment techniques are focused on globe conservation while minimizing toxic systemic adverse effects such as myelosuppression, need for blood transfusions, infections, and increased incidence of secondary tumors. One of these newer treatment techniques includes intra-arterial chemotherapy infusion of melphalan. This supraselective intra-ophthalmic artery chemotherapeutic drug delivery has been shown to be successful by Yamane et al1 and Abramson et al2 in advanced intraocular retinoblastoma (Reese-Ellsworth group V) cases. Herein, we report the clinicopathologic finding of 3 eyes of 3 patients diagnosed with advanced retinoblastoma, Reese-Ellsworth group Vb, or International Classification of Retinoblastoma group D, treated with supraselective intra-ophthalmic artery infusion of melphalan at our institution by a technique previously described by Abramson et al.3 The patients underwent enucleation for evidence of tumor progression.

Report of Cases. Case 1. A 21-month-old girl was referred for worsening exotropia. On clinical examination, she was able to fix and follow objects with her right eye, and there was no response with the left eye. Leukocoria, a trace afferent pupillary defect, and intermittent exotropia were noted in the left eye. On dilated funduscopic examination, a peripapillary tumor extending through the fovea with secondary exudative retinal detachment was noted in the left eye. Subretinal and fine vitreous seeding was also noted (Figure 1A). The right eye was unremarkable. The patient was diagnosed with Reese-Ellsworth group Vb retinoblastoma in the left eye, with a normal systemic workup. Systemic chemotherapy was initiated with 4 agents (carboplatin, vincristine, etoposide, and cyclosporine) combined with local laser therapy initially planned for 9 cycles but extended to 11 cycles based on tumor nonresponse. Subsequently, 6 cycles of pericentral carboplatin injections (20-mg dose) were administered. Despite aggressive globe-conserving treatment, the tumor progressed (Figure 1B). Salvage treatment with intra-arterial melphalan infusion was administered at a 3-mg dose initially and then a 7.5-mg dose. On follow-up examination, vitreous hemorrhage was noted obscuring the tumor, with tumor progression evident on echographic imaging. The left eye was enucleated (Figure 1C).

Histopathologic examination disclosed an undifferentiated tumor present in primarily an exophytic configuration arising from the neural retina. The tumor was staged pT2c (pTNM staging) because it extended into the optic nerve to the level of the lamina cribrosa (Figure 1D), focally into the choroid (Figure 1E), and into the vitreous cavity. No tumor was present within the anterior chamber or at the

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