Editorial

Korean J Anesthesiol 2014 November 67(5): 297-298
http://dx.doi.org/10.4097/kjae.2014.67.5.297

Venous air embolism during vitrectomy: a rare but potentially fatal complication

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Over 4,000 articles have been published on intraoperative venous air embolism (VAE) since it was first described in the 19th century [1]. Although its pathological mechanisms, preventative measures, and treatment methods have been established, it still appears consistently. VAE occurs when the pressure in an open vein is lower than atmospheric pressure. Thus, it is caused when a wound is at a higher level than the heart. The pathophysiological consequences of VAE depend on the velocity and quantity of air entry. Air bubbles flowing into the venous system lodge in the pulmonary circulation and are then diffused to alveoli to be exhaled during expiration. In most patients, a small quantity of air bubbles is tolerable. However, if the amount of air exceeds the pulmonary clearance capacity, increasing pulmonary artery pressure and, thus, increased right ventricular afterload, which may cause cardiac arrest. In particular, in patients with patent foramen ovale, paradoxical air embolism can result in a stroke or coronary occlusion, which may be fatal.

General management of VAE includes notifying the operating surgeon, procedure discontinuance, use of 100% oxygen, aspiration of entrained air through a central venous catheter, aggressive intravascular volume infusion, use of a vasopressor, bilateral jugular vein compression, placing the patient in a head-down position and, if necessary, cardiac pulmonary resuscitation (CPR).

It is known that VAE occurs most commonly during sitting craniotomy [1]. However, suitable conditions for it can occur whenever a wound is above the level of the heart. In this issue of the *Korean Journal of Anesthesiology*, Shin et al. [2] report VAE occurring during vitrectomy with airfluid exchange. One patient who was under mechanical ventilation with an oxygen/air mixture suddenly showed findings of cardiac arrest after starting air-fluid exchange. Although the surgical procedure was stopped quickly and CPR was performed, vital signs did not recover and thus percutaneous cardiopulmonary support was applied. Postoperative transesophageal echocardiogram revealed a patent foramen ovale.

VAE can occur during various surgical procedures [1] but it is rare in ophthalmic surgery. The first case report was published by anesthesiologists in 2005 [3] and it was then first introduced in an ophthalmology journal in 2010 [4]. In sitting craniotomy, where the incidence of VAE is among the highest, neurosurgeons are well aware of VAE but ophthalmic operating surgeons tend not to be. Highlighting this, an interesting study was recently conducted by ophthalmologists [5]. Four donor eyes unsuitable for tissue donation were used for simulated vitrectomies. This experiment was performed with 23- or 25-gauge cannulas under several intraocular pressures (30–60 mmHg). They measured air flow against atmospheric pressure to be ~350 ml/min under 40 mmHg infusion pressure through a 25-gauge line. Considering that an air volume of 200 ml (35 ml/kg) may be fatal [1], both ophthalmologists and anesthetists should be aware that ocular air fluid exchange is not completely safe and may even be fatal, especially for patients with patent foramen ovale.

Fortunately, not all air bubbles flowing in the venous system during vitrectomy surgery are potentially fatal. However, like the case described in this issue of the *Korean Journal of Anesthesiology*, complications in patients with other risk factors may be fatal. Thus, continuous preoperative and intraoperative attention by operating surgeons and anesthetists is required to prevent it.

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