LETTER TO THE EDITOR

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The use of Sedaconda in suspected venous gas embolism after emergency cesarean section: a case report

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Dear Editor,

The use of volatile anesthetic (VA) agents for sedation in intensive care units (ICUs) is gaining popularity as a means of reducing reliance on intravenous sedatives such as benzodiazepines and opioids [1]. In addition to their sedative effects, VA agents offer organ-protective properties by mitigating pro-inflammatory cytokine release and ischemia–reperfusion-related cellular injury. They also act as potent bronchodilators and vasoactive agents, promoting both arterial and venous dilation [1].

Inhaled sedation requires the use of anesthesia conservation devices (ACDs), typically reserved for ICU patients with severe respiratory failure [2, 3]. We report a case involving a 36-year-old pregnant woman who experienced a suspected venous air embolism (VAE) during caesarean section, treated with VA. Her medical history included asthma and a prior myomectomy complicated by hemorrhagic shock, requiring ICU admission. At 36 weeks of gestation, she presented with abrupt vaginal bleeding due to placental abruption, necessitating an emergency intervention. Informed consent for publication was obtained and documented in the electronic medical record.

Under general anesthesia, a postpartum hemorrhage protocol was initiated, including administration of tranexamic acid (1 g), fibrinogen (2 g), and blood transfusions. Norepinephrine infusion (up to 0.2 µg kg^{-1} min^{-1}) was used to maintain adequate perfusion. The caesarean delivery was complicated by adhesions from her previous surgery and an anteriorly located placenta. Immediately after delivery, the patient experienced a sudden and severe drop in oxygen saturation, bronchospasm, and hypotension. Norepinephrine was increased to 0.5 µg kg^{-1} min^{-1} to sustain a mean arterial pressure above 65 mmHg.

Arterial blood gas analysis collected a few minutes after the onset of hypotension, while under general anaesthesia (tidal volume 7 mL kg^{-1} body weight and FiO_2 100%), revealed respiratory alkalosis (pH 7.45), profound hypoxemia (PaO_2 18 mmHg), mild hypocapnia ($PaCO_2$ 34 mmHg), an increased arterial–end-tidal CO_2 gradient ($PaCO_2$–$EtCO_2$ > 10 mmHg), and elevated lactate (2.2 mmol L^{-1}). Bronchospasm, refractory to standard beta-adrenergic, anticholinergic, and corticosteroid therapy, only improved following VA administration, with oxygen saturation (SpO_2) progressively recovering to > 94%.

Considering the specific risk factors (placenta abruption, uterine exteriorization, and sudden, persistent desaturation despite relatively rapid recovery of adequate blood pressure values), submassive VAE was hypothesized [4, 5]. In addition to the severe hypotension that, on its own, could not justify severe hypoxemia, our hypothesis was also supported by a wider $PaCO_2$–$EtCO_2$ gradient, indicating impaired ventilation–perfusion (V/Q) matching or increased physiological dead space.

Unfortunately, confirmatory diagnostic investigations for the presumed diagnosis were not performed because of the patient's intraoperative

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hemodynamic instability and the lack of additional acute therapeutic options once the acute clinical condition had resolved.

Furthermore, given the deterioration of gas exchange after discontinuing VA in the operating room, we opted to continue VA sedation in the ICU using the Sedaconda ACD (Sedana Medical, Danderyd, Sweden). The infusion rate was increased to 20 mL h^{-1}, achieving an end-tidal sevoflurane concentration of approximately 1.4%. The patient's respiratory status improved progressively, with complete resolution of symptoms within five hours.

In this case, the diagnosis of VAE was eventually made through exclusion, guided by hallmark features: hypoxia, hypotension, and decreased $EtCO_2$ [4, 5]. In addition, the signs and symptoms that led to the suspected diagnosis of submassive VAE were also corroborated by the exclusion of other clinical conditions with similar manifestations, such as amniotic fluid embolism, pulmonary thromboembolism, pneumothorax, or myocardial infarction, through precordial and pulmonary Doppler evaluation. Specifically, the patient's gas exchange probably reflected both hemorrhagic shock (elevated lactate) and embolic respiratory failure (hypoxemia and hypocapnia).

In this case, VA appeared effective both intraoperatively and as an ICU sedative. VA agents may have played a therapeutic role by improving V/Q mismatch related to air trapping, alleviating bronchospasm and pulmonary vasoconstriction through modulation of vasoactive mediators, thereby potentially mitigating embolic effects [7].

Specifically, while the potential of VA to exacerbate V/Q mismatch in pulmonary embolism is a known concern – also relevant for vasodilators such as sildenafil – in our case, no such adverse effect occurred [8]. We hypothesize that despite the embolic event affecting a large portion of the pulmonary vasculature, once the bronchospasm resolved, the remaining healthy lung units could compensate. This may have enhanced VA uptake and supported oxygenation.

Additionally, we could hypothesize that VA, administered via inhalation, also reached the pulmonary circulation downstream of the vessels occluded by the sub-massive embolic event. The vasodilation at the alveolar level of the incompletely perfused airways could, albeit with reduced absorption, have favored the dissolution of the air bubble.

Specifically, hypoxic vasoconstriction during embolism, triggered by an increase in dead space, represents a physiological protective mechanism aimed at diverting pulmonary blood flow toward better-ventilated areas. In our case, the use of VA may have initially reduced this physiological redistribution of flow. However, the rapid resolution of the gas embolism – facilitated by VA-induced pulmonary vasodilation – likely enabled a prompt restoration of circulation to the regions previously affected by the embolic event. Overall, the beneficial effects of VA on bronchospasm, leading to improved ventilation, combined with enhanced perfusion, may have contributed to optimizing the ventilation–perfusion ratio and ultimately supporting the recovery of adequate SpO_2.

Although the use of sevoflurane during VAE has been reported [6], its efficacy remains under investigation. VA agents may beneficially reduce right ventricular afterload in pulmonary hypertension [8, 9], yet their effect on pulmonary gas-filtering capacity is still not fully understood [9, 10]. Moreover, inhaled anesthetics can potentially alter the pulmonary vessel diameter, potentially facilitating embolus clearance. At the same time, embolic severity also depends on the air bubble volume and infusion rate [11]. Moreover, VA's anti-inflammatory properties may have further supported recovery.

However, the precise mechanism underlying the observed improvement in oxygenation following sevoflurane administration cannot be definitively established from our data, and proposed effects on V/Q matching and hypoxic pulmonary vasoconstriction remain speculative. Rather than implying a direct causal mechanism, we frame the observed improvement in oxygenation as an empirical finding that may reflect a combination of altered pulmonary perfusion distribution, changes in ventilatory mechanics, or reduced patient–ventilator dyssynchrony, all of which have been reported in association with VAs.

In conclusion, VA agents were used in this case during an obstetric emergency and ICU sedation in the context of suspected submassive VAE. Observationally, their use was associated with improvements in hypoxemia and hemodynamic stability. Based on this single experience, VA agents may represent a potential alternative; in this case, their use was associated with improved oxygenation and was hemodynamically well tolerated. Although this finding is purely hypothesis-generating and does not imply a therapeutic recommendation, further studies are needed to better understand the pathophysiology of VAE and to cautiously explore the possible role and mechanisms of VA agents in this setting.

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