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Acute Pulmonary Edema with Paradoxical Desaturation after Salbutamol due to Venous Air Embolism during an Awake Craniotomy: A Diagnostic Challenge

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A 75-year-old non-smoker with good past health underwent an awake craniotomy for motor mapping and glioblastoma resection. During the procedure, she was sedated by intravenous propofol and remifentanil using target-controlled infusion (TCI) with bispectral index monitoring (target: 70–80). The effect-site drug concentrations were titrated between 1 and 2 µg/mL and 0 to 1 ng/mL, respectively. The patient was placed in a semi-sitting position (30-degree head up).

The patient's systolic blood pressure dropped slightly after the start of sedation; her other vital signs remained normal (►**Fig. 1**). Within 10 minutes after bone flap removal, the patient coughed briefly followed by a transient drop in SpO_2 to 78% and end tidal CO_2 ($EtCO_2$) to 1.7 kPa. Because her SpO_2 and $EtCO_2$ promptly improved after applying jaw thrust and nasopharyngeal airway, this episode was attributed to deep sedation. Sedation was stopped and the patient was asymptomatic after regaining consciousness.

Sixty minutes later, during brain mapping, a gradual decline in SpO_2 to 92% was observed with no reduction in $EtCO_2$. The patient remained asymptomatic with no clinical seizures and no epileptogenic activity noted during electrocorticography.

Toward the end of tumor resection, a second precipitous drop in SpO_2 to 80% associated with tachypnea occurred. The patient remained conscious and denied any chest pain. Chest auscultation revealed bilateral expiratory wheezing. Despite increasing her oxygen supplementation and administering salbutamol, her SpO_2 further decreased to 70%. Her SpO_2 improved to 90% only when 100% oxygen was delivered via a non-rebreathing mask. Her arterial blood gas (FiO_2: 1.0) showed that the PaO_2 and $PaCO_2$ levels were 7.7 kPa and 5.05 kPa, respectively. This episode was accompanied by transient hypotension (SBP: 80 mmHg) that responded to intravenous crystalloid bolus infusion. Intraoperatively, 1700 mL of crystalloids was administered with an urine output of 1200 mL over 6 hours and blood loss of 200 mL.

The operation was rapidly completed after this latest third episode of oxygen desaturation. A postoperative ECG showed normal sinus rhythm without acute changes. Transthoracic echocardiography showed an undilated right ventricle with a collapsed inferior vena cava, reflecting the absence of pulmonary hypertension. A chest X-ray (►**Fig. 2**) showed diffuse pulmonary infiltrates. Her serum troponin T level was slightly elevated (41 ng/L) and was normalized within 24 hours. An urgent CT pulmonary angiogram (►**Fig. 2**) showed diffuse pulmonary edema and bilateral pleural effusion without evidence of arterial embolism.

Postoperatively, the patient experienced full recovery with only supportive management, i.e., oxygen supplementation and chest physiotherapy, and was discharged home 8 days later.

Discussion

This case illustrates the unique diagnostic challenges of determining the cause for oxygen desaturation during an awake craniotomy. At first, this was attributed to deep

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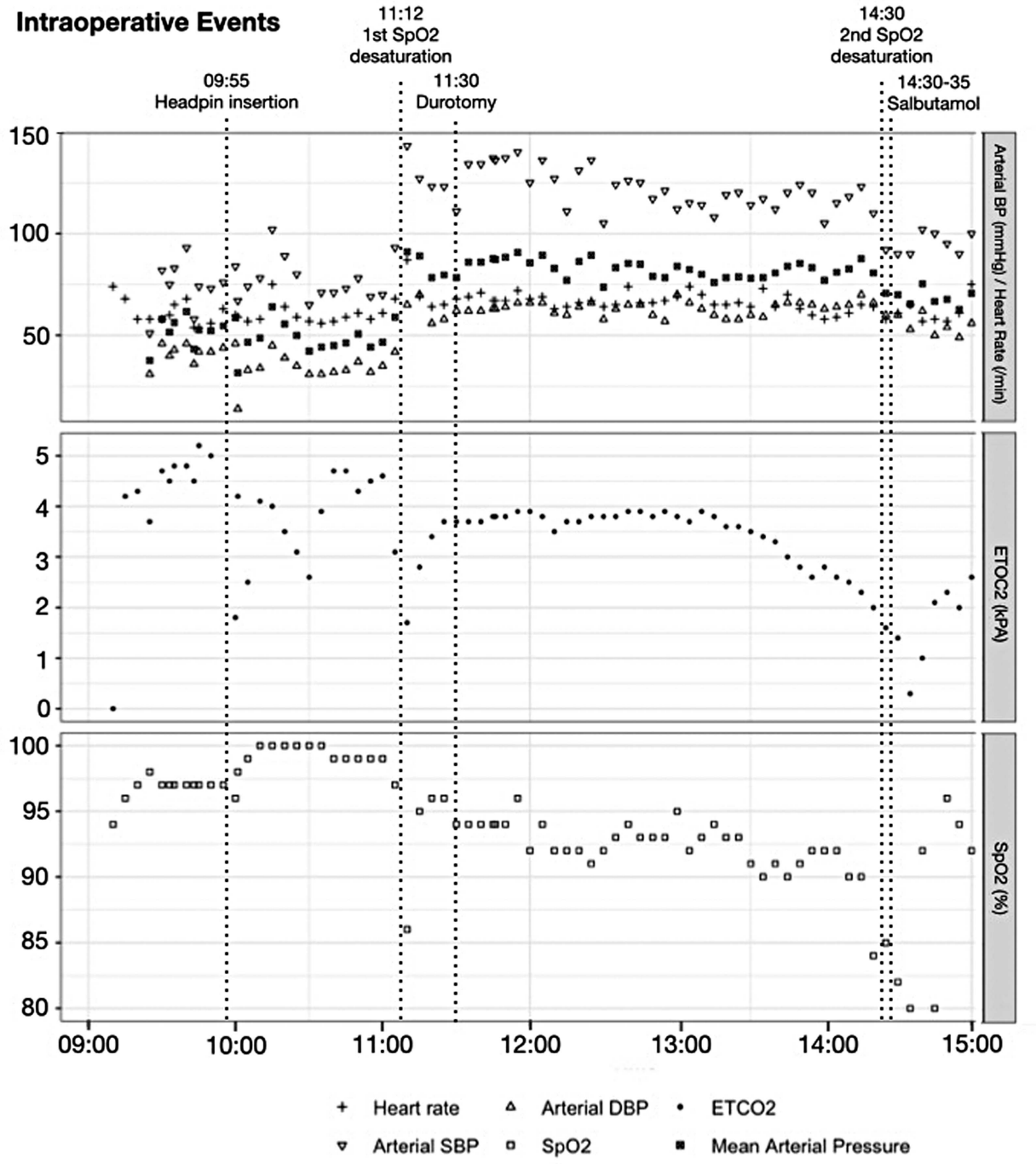

Fig. 1 Intraoperative vitals.

sedation as improvement was observed after upper airway maneuvers were applied coupled with sedative cessation. However, when desaturation recurred, we turned our attention to other causes, especially when the situation was exacerbated after the administration of salbutamol.

Negative pressure pulmonary edema due to airway obstruction was first considered; however, it was ruled out due to the patient's initial rapid clinical recovery. Neurogenic pulmonary edema, acute lung injury due to aspiration or hospital-acquired pneumonia, anaphylaxis, acute asthmatic attack, and cardiogenic pulmonary edema due to diastolic heart failure were differential diagnoses; however, these were excluded due to the absence of associated clinical signs and unsupportive investigation results (►**Fig. 3**). In addition, none of these diagnoses could explain the paradoxical oxygen desaturation observed after salbutamol administration.

Therefore, through a process of elimination, we believe the patient's intraoperative deterioration was due to persistent low-level venous air embolism (VAE). VAE presenting with bronchospasm is uncommon.[1] It was hypothesized that bronchoconstriction develops due to low-level persistent air emboli, resulting in regional alveolar hypocapnia and vagal reflex stimulation caused by inflammatory mediators.[2] Bronchoconstriction resulting from unilateral pulmonary

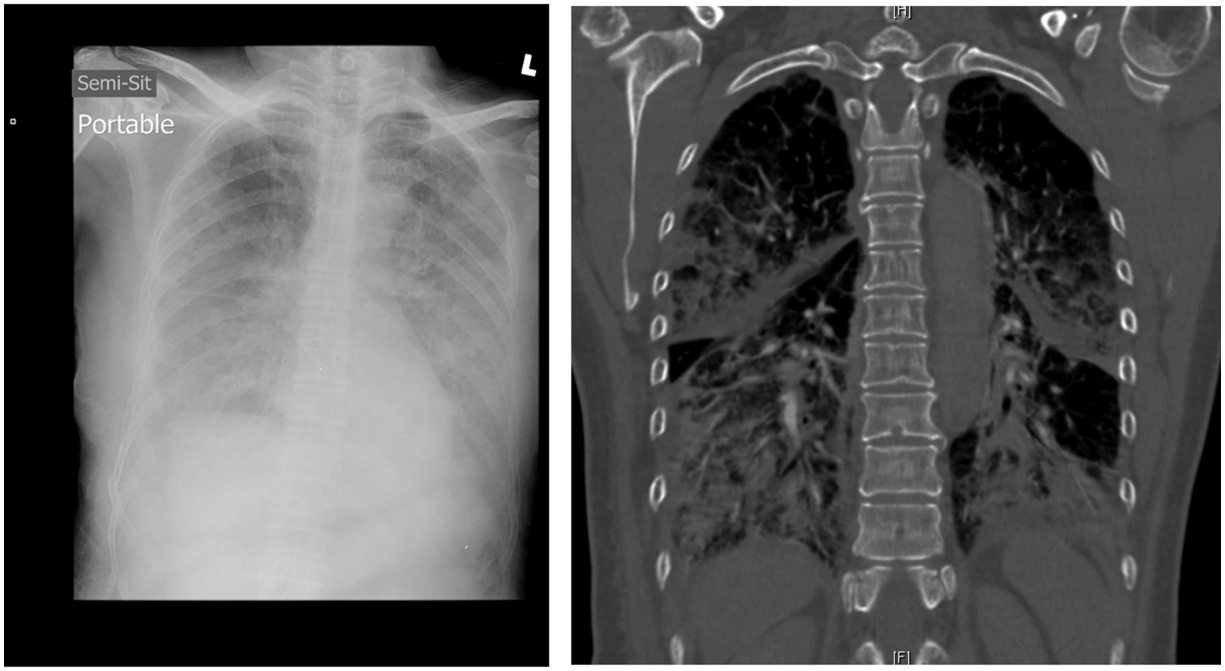

Fig. 2 CXR and CT angiogram.

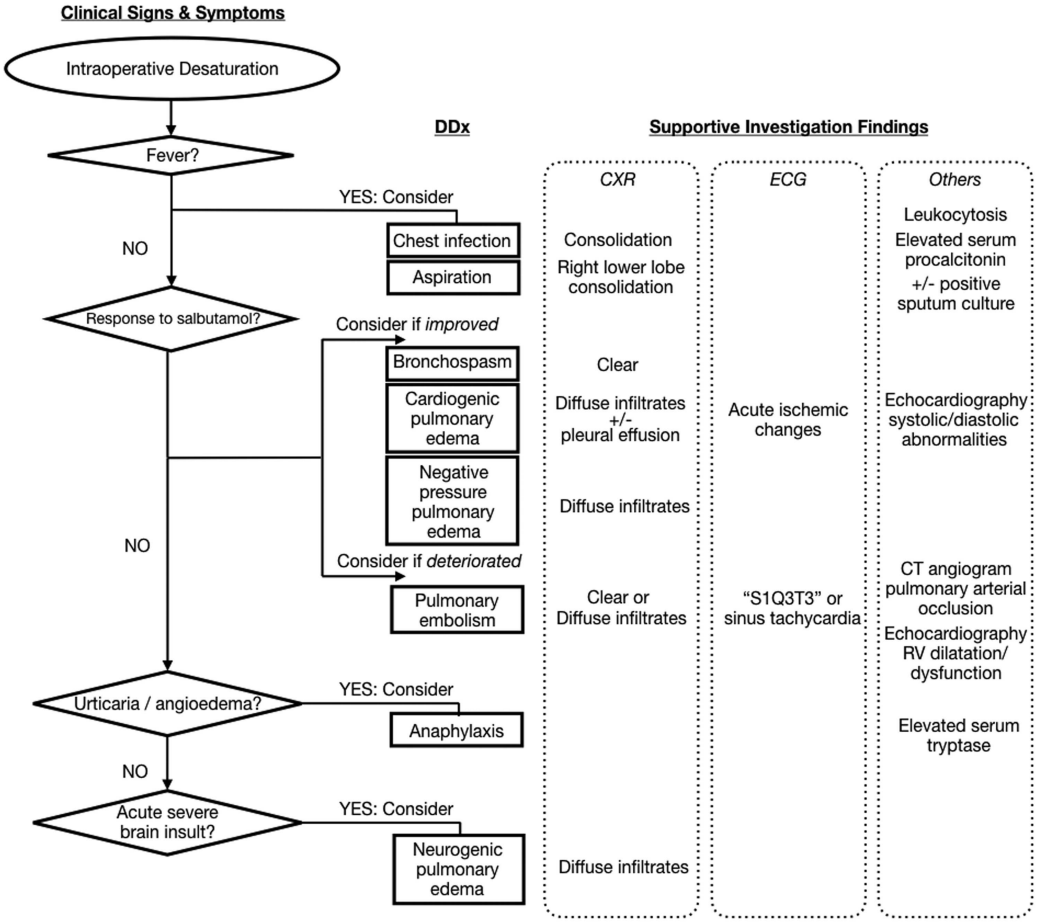

Fig. 3 Flowchart for diagnosis.

artery occlusion could improve V/Q matching, which may explain why salbutamol produced a paradoxical drop in SpO_2 due to bronchodilation of hypocapnic regions.[3] The pathophysiology of VAE-induced pulmonary edema is incompletely understood; however, it was proposed that similar to bronchoconstriction, the production of inflammatory mediators induces acute respiratory distress syndrome.[4] The continued evolution of this post-embolism inflammatory cascade could also be the reason for the patient's second and more severe deterioration.

Typical VAE presenting symptoms include coughing associated with chest discomfort, hypoxemia, tachypnea, hypotension, and tachycardia; however, brief coughing alone is seldom reported.[5] Although the exact mechanism is yet to be elucidated, coughing and tachypnea are postulated to be secondary to systemic hypercapnia that stimulates central medullary chemoreceptors.[4] A considerable number of intraoperative VAE events remain undetected with most patients being asymptomatic.[6] Risk factors for VAE in this patient included spontaneous ventilation and the adoption of a head-up position. Precordial Doppler monitoring is considered highly sensitive; however, due to potential noise disruption during awake brain mapping, only nasal cannula capnography was used. In light of this experience, we suggest that precordial Doppler could still be utilized during the initial sedation phase of this procedure if patients are placed in a semi-sitting or sitting position. Early VAE detection might facilitate salvage treatment; however, it has been reported that if low-level embolization occurs, precordial Doppler may be insufficiently sensitive with most patients presenting only with a drop in $EtCO_2$ and oxygen desaturation.[7] Neuro-anesthetists should have a lower threshold of suspicion for VAE should a patient unexpectedly cough during an awake craniotomy, especially when there is an associated drop in SpO_2 and $EtCO_2$.[4]

Conflict of Interest
None declared.

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