

Management of a sudden massive venous air embolism during emergent craniotomy

BACKGROUND

Severe venous air embolism (VAE) is a rare event (3% incidence) with up to 28% rate of mortality. It occurs in intracranial surgery when air enters the circulation via noncollapsible venous sinuses and reaches the right ventricle. It causes obstruction, pulmonary hypertension via vasoconstriction and air lock that can lead to RV failure. It can be detected early with precordial Doppler or TEE.

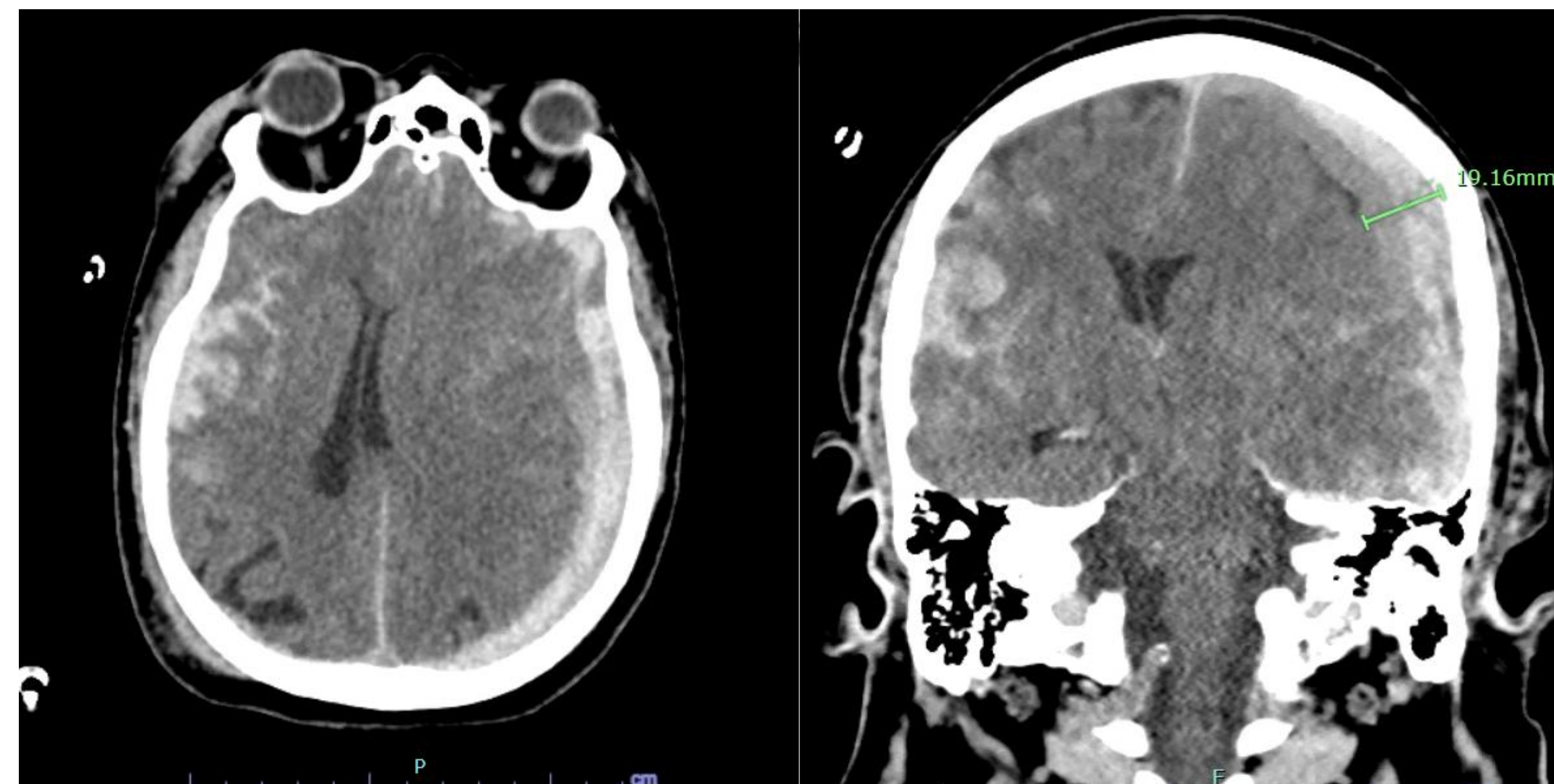
VAE can present in different settings including neurosurgery, interventional radiology, laparoscopic, obstetric interventions and many others. Many cases are subclinical and therefore unreported.

CASE PRESENTATION

- 77M with a history of CAD s/p 3-vessel coronary stenting 4 months prior, on DAPT, HTN, HLD and Afib presented after fall with head strike while walking his dogs resulting in bilateral subdural and epidural hematomas with midline shift. Intubated at OSH, given Keppra and 1 unit of platelets, then transferred to Westchester Medical Center.
- Head CT with findings of left holohemispheric extra-axial hematoma measuring up to approximately 19 cm in thickness with 9-10 mm left to right midline shift with subfalcine herniation; hematoma along the falx and left temporal lobe intraparenchymal hematoma 1.6 cm.
- Other injuries include right 1st, 3-6th rib fractures with resultant hemothorax, right orbital roof and temporal bone fractures, right apical subpleural lung hematoma.
- Prior to intubation mental status was GCS 3T, was transferred sedated on propofol. At the CT was reevaluated off sedation by neurosurgery – localizing LUE, GCS 6T.
- Was given 1 unit of platelets, Keppra and cefazolin in ED.
- Decision made to perform left hemicraniectomy as a life saving measure.

INTRA-OPERATIVE MANAGEMENT

- Standard ASA monitors, Aline, induction with propofol, fentanyl and rocuronium. Phenylephrine infusion started to maintain CPP.
- Positioning with 30 degree head elevation.
- 15 minutes after incision: EtCO₂ decreased from 32 to 18, hypotension to MAP 35 mmHg despite phenylephrine infusion, tachycardia to 120s.
- VAE suspected: surgeons flooded the field with normal saline, patient placed in Trendelenburg position, FiO₂ increased to 100%, PEEP turned off.
- Boluses of ephedrine, epinephrine and vasopressin with minimal improvement of hemodynamics. Calcium given to improve cardiac contractility.
- ABG demonstrated acute anemia, volume depletion and severe acidosis. 4 units of PRBCs transfused and bicarbonate given to improve response to pressors.
- Surgery was terminated 25 minutes after the event, RIJ CVC placed with attempts of aspiration of air. TTE demonstrated improvement in the amount of air seen in RV.
- Patient placed in left lateral position, vitals stabilized and patient transferred to the ICU.



DISCUSSION

Initial diagnosis and treatment is based on clinical signs. Precordial or transcranial doppler can be used in a controlled setting to monitor and detect bubbles with high sensitivity. TEE is the most sensitive method but requires expertise and located close to the surgical field.

Emergent change of position is crucial: initially it was changed to Trendelenburg and as soon as the surgery was aborted, patient moved into a left lateral decubitus, which allowed air to move away from the right ventricle to the right atrium, IVC and SVC, which improved hemodynamics.

Hemodynamic support was initially done with epinephrine due to imminent risk of cardiac arrest, then boluses of vasopressin with the goal of supporting systemic BP without increasing pulmonary vascular resistance. PEEP changes are controversial.

CONCLUSIONS

VAE may be detected by EtCO₂ monitoring. Prevention (hydration, positioning) and prompt recognition of VAE are critical. Having fluids and inotropic agents, consideration for doppler monitoring or a central line placement is important part of the plan for a case with moderate or high risk for VAE.

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