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Delayed Aspiration of Air Emboli from the Central Venous Catheter in a Case of Suspected Massive Venous Air Embolism: A Therapeutic Success

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Abstract

Venous air embolism (VAE), though, clinically benign in majority of cases, the significant ones can lead to life-threatening cardiopulmonary and neurological consequences. Though studies mention the success rate of only 6 to 16% in aspirating air from the central venous catheter (CVC) during VAE, the technique is very specific for diagnosing VAE and has high therapeutic significance. We report a case in which delayed aspiration of air emboli from the CVC in suspected massive VAE during decompressive craniectomy resulted in rapid resolution of hemodynamic instability. If not inserted previously, CVC may be considered in a hemodynamically unstable patient with suspected VAE.

Keywords
- air embolism
- central venous catheters
- hemodynamic
- neurosurgery

Introduction

Venous air embolism (VAE) has been reported widely during neurosurgical procedures with an incidence between 16 and 86%.[1] The incidence is variable owing to the difference in surgical and anesthetic techniques, patient's position during surgery, and diagnostic methods used. The two most important factors that affect the clinical impact of VAE are the volume and the rate of air entrainment.[2] While studies mention the success rate of only 6 to 16% in aspirating air from the central venous catheter (CVC) during VAE, the technique is very specific for diagnosing VAE and has high therapeutic significance.[3] We report a case in which delayed aspiration of air emboli from the CVC in suspected massive VAE during decompressive craniectomy resulted in rapid resolution of hemodynamic instability. Written informed consent was taken from the patient for reporting this case for publication.

Case Report

A 40 year male weighing 70 kg encountered a road traffic accident and suffered head injury. He was taken to a nearby hospital where a noncontrast computed tomography of brain

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was done that showed hemorrhagic contusions in right fronto-temporo-parietal (FTP) lobe with midline shift and mass effect. His initial Glasgow Coma Scale (GCS) on admission was E2V3M5 (E: eye opening, V: verbal response, M: motor response). Intubation of trachea was done and he was treated conservatively for 7 days in the same hospital. However, following deterioration of GCS on 8th day and expansion of hematoma, he was advised to undergo right FTP decompressive craniectomy and hematoma evacuation. The patient's family refused for any further intervention in the same hospital and shifted the patient to our hospital. His GCS on admission to our hospital was E1VTM4. We immediately scheduled the patient for right FTP decompressive craniectomy. In the operation theater, his endotracheal tube was replaced with a new sterile one of same size (8 mm ID). His preoperative vital parameters included blood pressure (BP)—129/83 mm Hg, heart rate (HR)—102 beats/min, and percentage of oxygen saturation (SpO_2)—98% (fraction of inspired concentration, $FiO_2 = 0.4$). We secured two wide bore 16 G intravenous (IV) cannula. Left radial artery was cannulated for beat-to-beat BP monitoring. CVC was not inserted. Induction of anesthesia was done with IV fentanyl—150 µg and IV propofol—120 mg. A loading dose of 8 mg cisatracurium was administered for muscle relaxation. Oxygen:air mixture (FiO_2: 50%) along with total IV anesthesia with fentanyl (75 µg/h), cisatracurium (2 mg/h), and propofol titrated maintain bispectral index of 40 to 50 that was incorporated to maintain intraoperative anesthesia. A small degree of reverse Trendelenburg positioning was done to facilitate decrease in intracranial pressure (ICP) and optimizing surgical visualization. The initial part of the surgery was uneventful. However, during the process of removal of the bone flap, there was injury to superior sagittal sinus (SSS) following which there was a sudden and sustained decline in end-tidal carbon dioxide ($EtCO_2$) from 32 to 22 mm Hg, BP from 126/75 to 64/38 mm Hg, and rapid progressive decline of SpO_2 from 98 to 72%. His HR increased from 92 to 130 beats/min associated with premature ventricular complexes (PVCs). There was associated blood loss of ~200 mL. An arterial blood gas analysis was done that showed partial pressure of carbon dioxide ($PaCO_2$) and $EtCO_2$ gradient of 15 mm Hg. Suspecting it to be VAE, the surgeon was informed immediately. The surgical field was flooded with normal saline and bony edges were sealed with wax. Simultaneously, we ventilated the lungs with 100% oxygen, lowered the head-end of the bed, infused IV fluids, and gave intermittent dose (3 mg) of mephentermine to avoid prolonged hypotension. After ~5 minutes of resuscitation, the $EtCO_2$ increased to 29 mm Hg, BP increased to 110/65 mm Hg, and HR decreased to 110/min. However, the SpO_2 increased only up to 95% at $FiO_2 = 1$ and occasional PVCs. The surgery was resumed. The FiO_2 was kept at 0.8 targeted to $SpO_2 \geq 94\%$. Another similar episode occurred during dural repair that was managed in the same way. However, after the second event, the BP remained in a lower range of 100/68 mm Hg and SpO_2 of 91 to 92% at $FiO_2 = 0.8$ and frequent PVCs. The surgery completed in ~150 minutes. During the surgery, the total blood loss amounted to around 600 mL. We transfused two units of packed red blood cells and two units of fresh frozen plasma. Considering the possibility of ongoing VAE (due to low BP and SpO_2), we inserted Electrocardiogram (ECG) guided CVC (7F, triple lumen) in the right subclavian vein immediately after the completion of the surgery. The tip of the catheter was targeted 2 cm below the superior vena cava (SVC) and the right atrium (RA) junction. To our surprise, 80 mL of air could be aspirated from the CVC (►**Fig. 1**). The BP and SpO_2 started to increase within next 20 minutes and within an hour, the patient was hemodynamically stable with SpO_2 of 97% at $FiO_2 = 0.5$. The patient was electively ventilated after the surgery. On 4th postoperative day (POD 4), tracheostomy was done. Gradually, he started to improve and on POD 9, the patient was E3VTM6. The patient was gradually weaned off from the mechanical ventilation. Decannulation of the tracheostomy tube was done on POD 17 and the patient was discharged from the hospital on POD 20.

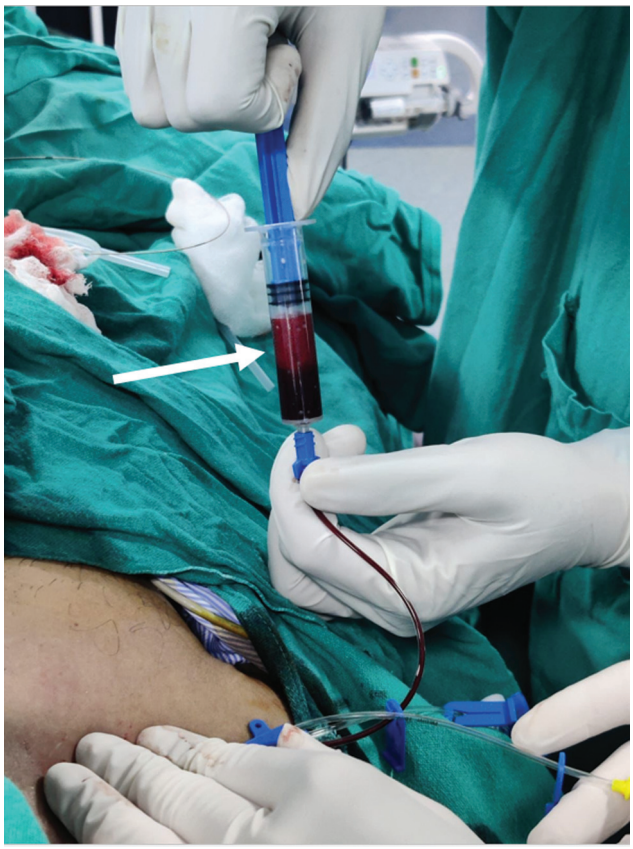

Fig. 1 Aspiration of air emboli from central venous catheter (arrow).

Discussion

In this case, the predominant inciting event for the occurrence of VAE was injury to the SSS. Open and noncollapsible veins form the major predisposing factor for entrainment of atmospheric air.[4] Moreover, the slight head up position in our case probably aggravated the air entrainment. The above presentation could be attributed to manifestation of systemic hypotension due to blood loss as a result of SSS injury. However, there was sudden and sustained fall in $EtCO_2$.

Moreover, the oxygen desaturation (from 98% to 72%), ECG changes of PVCs, $PaCO_2$, and $ETCO_2$ gradient of 15 mm Hg. and the requirement of high FiO_2 (0.8) so as to maintain $SpO_2 \geq 94\%$ even postresuscitation excluded the possibility of systemic hypotension as the primary cause of the event. Other probable causes could be trigeminocardiac reflex and raised ICP. However, in both cases, bradycardia is a predominant feature. Moreover, the timing of SSS injury and consequent decrease in $EtCO_2$ along with hypotension and oxygen desaturation show a definite association with VAE. Again, aspiration of air from the CVC is highly suggestive of the diagnosis of VAE.

This case report primarily brings forth the importance of CVC in the management of massive or significant VAE. Previous literatures have described air aspiration from the CVC leading to prompt resolution of hemodynamic perturbations in suspected VAE.[5,6] In these reports, CVC was in situ during the time of VAE episode. Delayed aspiration of air via CVC is uncommon. In our case, the CVC was inserted at the end of the surgery due to persistence of hemodynamic instability. The CVC was inserted in our case as per the recommendation, that is, tip of the catheter was targeted 2 cm below the SVC and the RA junction that improves the success rate of aspiration of air emboli.[3] In the delayed phase of VAE, cardiovascular support and frequent neurological monitoring are warranted till hemodynamic parameters and neurological integrity are restored.

ECG-guided central venous catheterization has been reported to be accurate and successful. The success rate has been reported be around 96%.[7,8] The technique is easy and inexpensive. Fluoroscopy/C-arm-guided CVC tip placement is also an alternative; however, it involves radiation. Use of transesophageal echocardiography (TEE) has been shown to have 100% accuracy, but the technique is invasive and it is not available in all centers.[9]

Early detection and management of VAE are the sine qua non to prevent potentially fatal complications. A battery of monitors is in use for its early detection before hemodynamic changes occur. TEE and Doppler ultrasound are highly sensitive. However, both have their own limitations and drawbacks. $EtCO_2$ tension monitoring, though intermediate in sensitivity, has the advantage of being noninvasive and semiquantitative. Capnography detects clinically significant VAE with reasonable precision.[10]

Conclusions

The significant VAE episodes can lead to poorer outcome, albeit the fact that majority of the VAE episodes are clinically innocuous. Considering the diagnostic and therapeutic effectiveness of CVC, it may be inserted in a hemodynamically unstable patient with suspected VAE, if hitherto not introduced.

Conflict of Interest
None declared.

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