

CEREBRAL VENOUS AIR EMBOLISM IN A PATIENT WITH SUPERIOR VENA CAVA OCCLUSION

Radhiana Hassan^{1*}, Intan Bazilah Abu Bakar¹

¹Department of Radiology, Kulliyyah of Medicine, International Islamic University Malaysia (IIUM), Kuantan, Pahang, Malaysia.

***Corresponding author:**

Radhiana Hassan, Department of Radiology, Kulliyyah of Medicine, International Islamic University Malaysia (IIUM), Kuantan, Pahang, Malaysia.

Email: radhianahassan@iium.edu.my

DOI: <https://doi.org/10.32896/cvns.v7n4.9-13>

Received: 16.11.2025

Revised: 23.12.2025

Accepted: 24.12.2025

Published: 31.12.2025

ABSTRACT

Introduction: Large cerebral venous air embolism (CVAE) is a rare but serious complication generally attributed to invasive central venous instrumentation or neurosurgical procedures. However, in patients with significant hemodynamic alterations, such as superior vena cava (SVC) occlusion, CVAE can develop through unique retrograde mechanisms following routine care.

Case Report: A 58-year-old male with Stage 4 non-small cell lung carcinoma presented with new-onset headache. Thoracic computed tomography (CT) demonstrated neoplastic occlusion of the SVC by mediastinal lymphadenopathy. Subsequent brain CT revealed multiple air pockets within the superior sagittal sinus and left cavernous sinus. The patient was managed conservatively, with complete resolution of symptoms and air emboli on follow-up imaging 10 days later.

Discussion: In the setting of SVC occlusion, elevated central venous pressure can reverse the physiological pressure gradient. Consequently, air introduced during routine upper extremity peripheral cannulation may migrate cephalad through the valveless internal jugular veins into the cerebral venous system, rather than flowing antegrade to the right heart. This phenomenon is often exacerbated by the presence of collateral vessels which provide a low-resistance pathway.

Conclusion: This case highlights that patients with SVC occlusion are at risk for iatrogenic CVAE even from simple peripheral venous access. Clinicians must be aware of these abnormal hemodynamics and should consider using lower extremity intravenous access in this population to mitigate the risk of retrograde embolization.

Keywords: Air Embolism, Superior Vena Cava Occlusion,

INTRODUCTION:

Large Cerebral venous air embolism is a rare but potentially serious complication that may occur in patients with altered venous hemodynamics [1]. CVAE can result from the retrograde ascent of air bubbles against the direction of venous blood flow. This retrograde migration is facilitated by the absence of valves in the jugular venous system. Once the air bubbles lodge in the cerebral venous sinuses, they can obstruct venous outflow, precipitating severe neurological sequelae including intracranial hypertension, cerebral edema, and venous infarction.

CASE REPORT:

A 58 year old man who was recently diagnosed with Stage 4 non-small cell lung carcinoma and admitted for further work up prior to immunotherapy complained of headache. Otherwise, no symptoms of increased intracranial pressure and no body weakness. Review of his CT thorax showed lung mass at right upper lobe with huge matted mediastinal nodes enlargement causing occlusion of the superior vena cava (SVC) with multiple enlarged collateral vessels at upper thoracic region (Figure 1). A contrasted-enhanced CT brain shows no evidence of cerebral metastasis. However, multiple small air pockets were noted within the superior sagittal sinus and left cavernous sinus (Figure 2). No other significant finding. Patient was treated conservatively for his headache which was completely subsided. A subsequent CT brain 10 days later showed complete resolution of the cerebral venous air.

DISCUSSION:

Cerebral venous air embolism is a rare iatrogenic complication that typically arises from invasive procedures such as central venous catheter access, hemodialysis, defibrillator placement and various neurosurgical interventions [1]. None of these procedures were performed in our patient. In our patient, SVC obstruction

most likely causes abnormal hemodynamic flows which allow retrograde air from peripheral intravenous cannulation. Usually, intravenous injectate administered in the upper extremity travels through the axillary, subclavian and brachiocephalic veins to empty into superior vena cava. However, SVC obstruction with increased venous pressure allows cephalad flow through the jugular veins into the cerebral venous system [1,2]. Presence of collateral vessels also provide low resistance route for cephalad migration. Air bubbles, being lighter than blood thus accumulate in the highest area which is the superior cavernous sinus. This case underscores the increased risk of iatrogenic cerebral venous air embolism in patients with SVC obstruction. Awareness of this entity is crucial to avoid this rare but potentially catastrophic complication and clinicians are advised to exercise extra caution during peripheral intravenous cannulation or consider using lower extremity IV access in these patients [3].

CONCLUSION:

While CVAE is traditionally associated with central venous instrumentation or neurosurgical procedures, this report demonstrates that the altered hemodynamics and elevated venous pressure inherent to SVC obstruction can predispose patients to retrograde embolization even during routine peripheral cannulation. Clinicians must maintain a high index of suspicion for CVAE in oncologic patients with SVC obstruction who present with acute neurological symptoms, such as headache, following intravenous access. To mitigate this specific iatrogenic risk, we recommend considering lower extremity intravenous access as a safer alternative in patients with known SVC obstruction. Prompt recognition and appropriate supportive care are essential to ensure favourable outcomes.

DATA AVAILABILITY:

Further information regarding the data used for this work can be obtained from the corresponding author upon reasonable request.

FUNDING:

This work received no external funding.

CONFLICT OF INTEREST:

The authors have no conflicts of interest to declare and is in agreement with the contents of the manuscript.

REFERENCES:

1. Červeňák V, Všianský V, Cviková M, Brichta J, Vinklárek J, Štefela J, *et al.* Cerebral air embolism: neurologic manifestations, prognosis, and outcome. *Frontiers in neurology* 2024; 15: 1417006.
<https://doi.org/10.3389/fneur.2024.1417006>
2. Carneiro AC, Diaz P, Vieira M, Silva M, Silva I, Custodio M, *et al.* Cerebral Venous Air Embolism: A Rare Phenomenon. *Eur J Case Rep Intern Med.* 2019; 23;6(1):001011.
https://doi.org/10.12890/2019_001011
3. Chuang DY, Sundararajan S, Sundararajan VA, Feldman DI, Xiong W. Accidental Air Embolism. *Stroke*, 2019; 50(7): e183–e186.
<https://doi.org/10.1161/STROKE.AHA.119.025340>

FIGURE LEGEND:



Figure 1: Contrast-enhanced CT thorax, MPR image in coronal plane, soft tissue window demonstrates the SVC occlusion (solid arrow) by enlarged mediastinal nodes and tumour thrombus. Right upper lobe mass (dashed arrow) is the primary lung cancer.

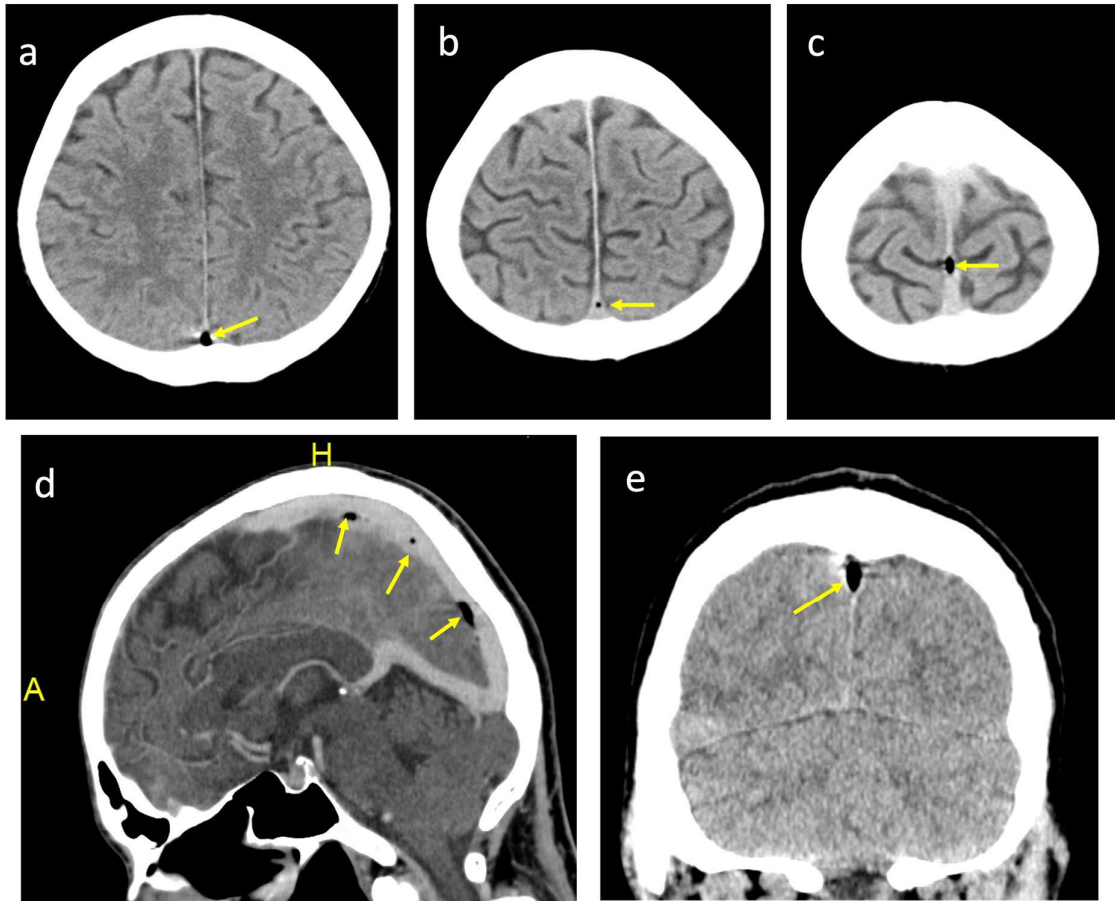


Figure 2: Contrast-enhanced CT brain in soft tissue window (a-c) axial plane, (d, e) sagittal and coronal reformatted image. Multiple air pockets within the superior sagittal sinus (yellow arrows).