

2. Robeson K, Blondin N, Szekely A. Frontotemporal brain sagging syndrome (FBSS) due to minor head trauma. *Neurology*. 2016.
3. Wicklund MR, Mokri B, Drubach DA, Boeve BF, Parisi JE, Josephs KA. Frontotemporal brain sagging syndrome An SIH-like presentation mimicking FTD. *Neurology*. 2011.
4. Schvievink WI, Maya MM, Barnard ZR, Moser FG, Stacey JP, Waxman AD, et al. Behavioral variant frontotemporal dementia as a serious complication of spontaneous intracranial hypotension. *Oper Neurosurg*. 2018.
5. Hong M, Shah GV, Adams KM, Turner RS, Foster NL. Spontaneous intracranial hypotension causing reversible frontotemporal dementia. *Neurology*. 2002.
6. Hong M, Shah GV, Adams KM, Turner RS, Foster NL. Spontaneous intracranial hypotension causing reversible frontotemporal dementia. *Neurology*. 2002;58:1285–7.

^b Department of Neurosurgery, University Hospital Doctor Josep Trueta, Girona, Spain

^c Department of Neurology, University Hospital Doctor Josep Trueta, Girona, Spain

^d Cognitive Impairment Unit, Department of Neurology, University Hospital Doctor Josep Trueta, Girona, Spain

* Corresponding author.

E-mail address: dani89ld@hotmail.com (D.L. Domínguez).

<https://doi.org/10.1016/j.nrl.2022.04.002>
0213-4853/

© 2022 Sociedad Española de Neurología. Published by Elsevier España, S.L.U. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

D.L. Domínguez^{a,*}, C.C. Reinoso^b, J.G. Fulla^c, I.P. Nierga^d

^a Movement Disorder Unit. Ataxia Unit. Department of Neurology, University Hospital Doctor Josep Trueta, Girona, Spain

Position is important: retrograde air embolism after central venous catheter removal



La posición importa: embolismo aéreo retrogrado tras retirada de catéter venoso central

Dear Editor:

Retrograde venous air embolism (RVAE) occurs when central venous pressure (CVP) is lower than atmospheric pressure, as is the case with deep inhalation, vertical positions above 45°, and hypovolaemia. The pressure gradient favours the entry of air into venous circulation, travelling to the right ventricle and pulmonary artery, and potentially even leading to an obstructive shock and right ventricular dysfunction.^{1,2} Some studies show that the air may retrogradely ascend to the cerebral venous circulation when the patient is in a vertical position, due to the lower specific weight of air in comparison with blood. This phenomenon will depend on the size of the bubble, the diameter of the vein, and the patient's cardiac output.^{3,4} Causes of RVAE include trauma, vascular surgery, diving, barotrauma due to mechanical ventilation, and insertion and extraction of central venous catheters. Incidence is difficult to determine, ranging from 1.6% to 55.3%; it is an underestimated entity due to the difficulty of establishing a diagnosis, which requires presence of a known risk factor, compatible clinical signs, no right-to-left shunting in the echocardiography, and imaging studies showing the presence of air in the intravascular space. The most frequent neurological complications are altered level of consciousness, coma, stroke, and

seizures.⁵ Patients may also present haemodynamic and respiratory alterations including dyspnoea, tachypnoea, chest pain, arterial hypotension, low cardiac output, and even obstructive shock and cardiorespiratory arrest. Electrocardiographic alterations include sinus tachycardia, right ventricular overload signs, non-specific changes in the ST segment/T-wave, and elevated markers of myocardial damage. Definitive diagnosis is established by head CT scan revealing air bubbles in the cerebral intravascular space and parenchyma, sometimes accompanied by diffuse cerebral oedema. In addition to symptomatic treatment with volume therapy, treatment for RVAE includes vasoactive amines, antiepileptics, oxygen therapy with high FiO₂, and placing the patient in the left lateral decubitus position (Durant manoeuvre) or the Trendelenburg position. Hyperbaric oxygen therapy may be considered in severe cases.⁶

Patient 1

Our first patient was a 77-year-old man who was admitted due to perforated sigmoid diverticulitis. The patient had a central venous catheter in the right jugular vein, which was removed with the patient in a seated position. Immediately after removal, he presented arterial hypotension and decreased level of consciousness with spontaneous opening of the eyes, fixed gaze and inability to follow commands, and pain with left hemiparesis. A head CT scan revealed air bubbles in the cavernous sinuses and basal cisterns but no other alterations (Fig. 1A). An electrocardiography (ECG) study showed ST-segment elevation in precordial leads and negative T-wave in leads V5, V6, I, and aVL (Fig. 1B), with elevated markers of myocardial damage. He was transferred to the intensive care unit (ICU) a few hours later due to a generalised tonic-clonic seizure; we ruled out toxic, metabolic, and infectious aetiology. A transthoracic echocardiography study showed no atrial septal defect. We started treatment with fluid replacement, oxygen therapy using a high-flow mask, and antiepileptics, which led to favourable progres-

DOI of original article: <https://doi.org/10.1016/j.nrl.2022.04.001>.

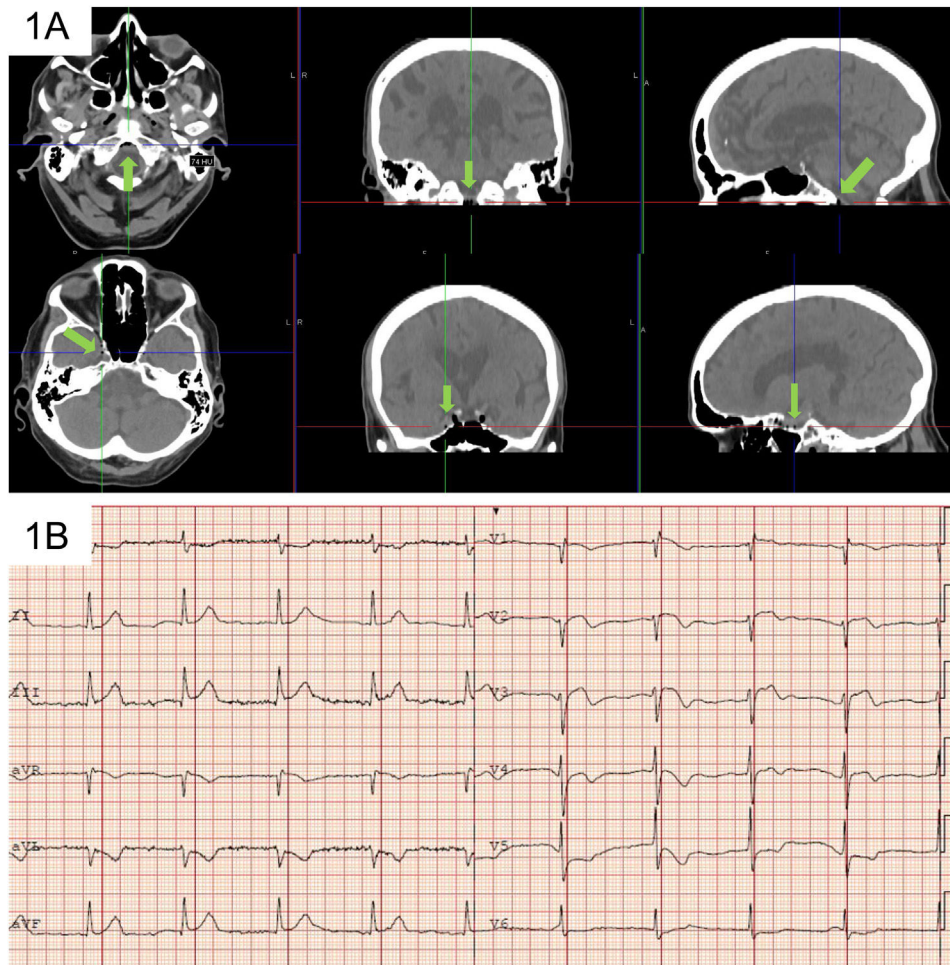


Figure 1 (A) Head CT scan showing air bubbles in the cavernous sinuses and basal cisterns. (B) Electrocardiography revealing ST-segment elevation in precordial leads and negative T-wave in leads V5, V6, I, and aVL.

sion. At 24 hours, ECG findings normalised, myocardial enzyme levels decreased, and a follow-up head CT study confirmed reabsorption of the air bubbles. The patient was discharged from the ICU at 72 hours, presenting normal results in the neurological examination. He was diagnosed with retrograde venous air embolism after removal of the central venous catheter.

Patient 2

The second patient is a 50-year-old man who was admitted to the ICU due to community-acquired bilateral pneumonia. A central venous catheter had been placed in the right subclavian vein and was accidentally removed with the patient standing. His level of consciousness immediately decreased. The neurological examination revealed spontaneous opening of the eyes, with right gaze deviation and decorticate posturing of the upper limbs in response to painful stimuli. We performed orotracheal intubation and started mechanical ventilation. A head CT scan revealed multiple air bubbles in the bilateral frontal and parietal lobes, the left temporal lobe, left cerebellar hemisphere, and both pterygopalatine fossae; these images are compatible with air embolism

(Fig. 2). The patient was treated with oxygen therapy in a hyperbaric chamber. During treatment, he presented tonic-clonic seizures, initially affecting the right upper limb and with subsequent generalisation to the upper hemibody. He was placed under deep sedation for 3 days and received antiepileptics. A follow-up CT scan showed reabsorption of the air bubbles. After discontinuation of sedation, he presented a good level of consciousness with no neurological alterations; clinical progression after extubation was good.

Because RVAE can occur as a result of procedures carried out in nearly all medical specialities, it is important that clinicians remain alert and informed regarding this atypical complication. These 2 clinical cases remind us that, to avoid retrograde air embolism, central venous catheters should always be removed with the patient in a horizontal position.

Author contributions

Salvador Balboa and Dolores Escudero participated in data collection and drafted the manuscript. Rodrigo Albillos and Raquel Yano participated in data collection.

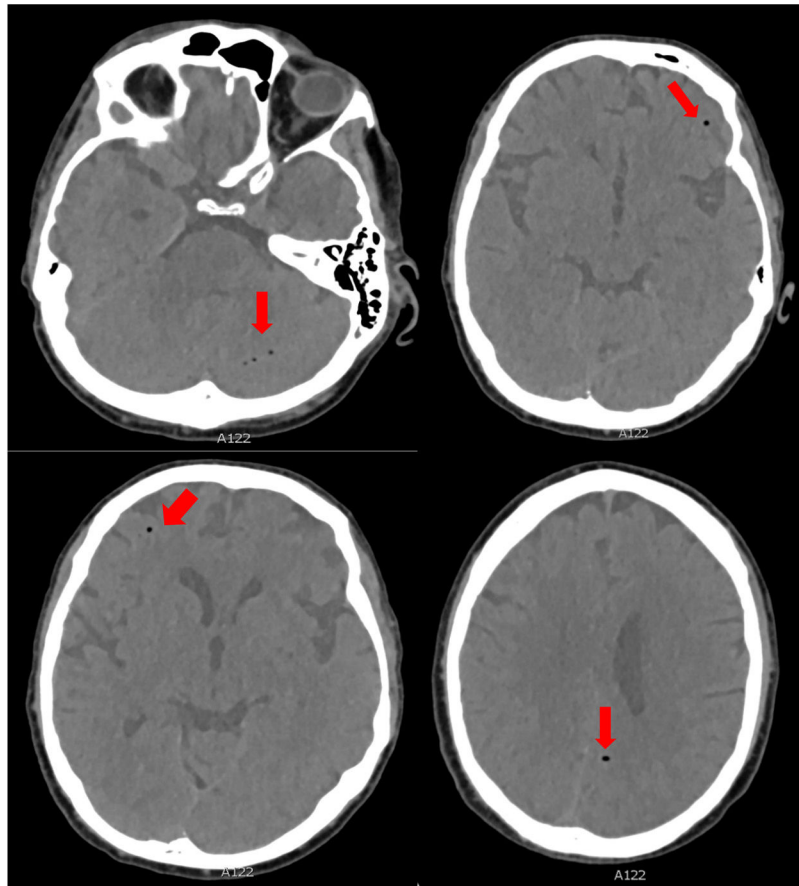


Figure 2 Head CT scan showing multiple air bubbles in both frontal and parietal lobes, the left temporal lobe, left cerebellar hemisphere, and both pterygopalatine fossae; these images are compatible with air embolism.

Funding

This study has received no funding of any kind.

Conflicts of interest

The authors have no conflicts of interest to declare.

References

- Muth CM, Shank ES. Gas embolism. *N Engl J Med.* 2000;342:476–82, <http://dx.doi.org/10.1056/NEJM200002173420706>.
- Schlimp CJ, Lederer W. Factors facilitating retrograde cerebral venous embolism. *J Child Neurol.* 2008;23:973, <http://dx.doi.org/10.1177/0883073808320619>.
- Schlimp CJ, Loimer T, Rieger M, Lederer W, Schmidt MB. The potential of venous air embolism ascending retrograde to the brain. *J Forensic Sci.* 2005;50:906–9, <http://dx.doi.org/10.1520/JFS2005061>.
- Fracasso T, Karger B, Schmidt PF, Reinbold LD, Pfeiffer H. Retrograde venous cerebral air embolism from disconnected central venous catheter: an experimental model. *J Forensic Sci.* 2011;56:S101–104, <http://dx.doi.org/10.1111/j.1556-4029.2010.01572.x>.
- Heckmann JG, Lang CJG, Kindler K, Huk W, Erbguth FJ, Neundorfer B. Neurological manifestations of cerebral air embolism as a complication of central venous catheterization. *Crit Care Med.* 2000;28:1621–5, <http://dx.doi.org/10.1097/00003246-200005000-00061>.
- Schlimp CJ, Loimer T, Rieger M, Schmidts MB, Lederer W. Pathophysiological mechanism and immediate treatment of retrograde cerebral venous air embolism. *Intensive Care Med.* 2006;32:945, <http://dx.doi.org/10.1007/s00134-006-0149-y>.

S. Balboa^{a,b,*}, R. Albillos^{a,b}, R. Yano^{a,b}, D. Escudero^{a,b}

^a Servicio de Medicina Intensiva, Hospital Universitario Central de Asturias, Oviedo, Spain

^b Grupo de Investigación Microbiología Traslacional, Instituto de Investigación Sanitaria del Principado de Asturias, Spain

* Corresponding author.

E-mail address: salvabalboa91@gmail.com (S. Balboa).

<https://doi.org/10.1016/j.nrleng.2022.04.006>

2173-5808 © 2022 Sociedad Española de Neurología. Published by Elsevier España, S.L.U. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).