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C.M. Garnés Sánchez[a,*], G. Parrilla[b], B. García Villalba[b], H. Alarcón Martínez[c], E. Martínez Salcedo[c], S. Reyes Domínguez[d]

[a] *Servicio de Neurología, Hospital Universitario Santa Lucía, Santa Lucía, Cartagena, Murcia, Spain*
[b] *Servicio de Neurorradiología Intervencionista, Hospital Universitario Virgen de la Arrixaca, El Palmar, Murcia, Spain*
[c] *Sección de Neuropediatría, Hospital Universitario Virgen de la Arrixaca, El Palmar, Murcia, Spain*
[d] *Sección UCI pediátrica, Hospital Universitario Virgen de la Arrixaca, El Palmar, Murcia, Spain*

* Corresponding author.
E-mail address: cmgarnes@hotmail.com
(C.M. Garnés Sánchez).

First described case of coma triggered by retrograde venous air embolism: An exceptional but potentially life-threatening situation☆

Primer caso descrito de coma desencadenado por embolismo aéreo venoso retrógrado: una situación excepcional pero potencialmente letal

Dear Editor:

Retrograde venous air embolism (RVAE) occurs when air is introduced into the venous system as a result of a negative pressure gradient, moving in the opposite direction to the normal flow of venous blood, and ultimately reaching the cerebral venous system. It may be secondary to surgery, barotrauma, or invasive procedures such as venous catheterisation, typically central but occasionally through a peripheral intravenous line (PIV). This disorder is underdiagnosed and has an undetermined prevalence and incidence, given that only anecdotal cases have been reported.[1—7] Neurological findings include confusion, amnesia, epileptic seizures, cerebral ischaemic vasculopathy, and/or coma. A cranial CT scan and oxygen therapy (concentrations around 100%) are crucial for the diagnosis and treatment of this complication.[7—9] We present the first reported case of a patient who unexpectedly went into a coma after experiencing an RVAE due to the accidental removal of a PIV.

Our patient was a 79-year-old woman with no relevant medical history who was admitted due to lower limb cellulitis. While hospitalised, she removed her PIV accidentally when she was getting out of bed; as a result, she experienced a sudden drop in arterial blood pressure, tachypnoea, and a decrease in the level of consciousness. During the examination, the patient spoke unintelligibly, did not open her eyes, and showed no withdrawal reflex to nociceptive stimulation (Glasgow Coma Scale 4: E1/V2/M1) and a subcutaneous emphysema in the antebrachial region. The patient was admitted to the intensive care unit where she received intravenous fluid and high-flow oxygen (15 L/min) using a Venturi mask with a reservoir bag. The results of a blood analysis revealed respiratory acidosis, a temporary increase of biomarkers for myocardial necrosis, and D-dimer levels within normal ranges. An ECG showed acute right ventricular overload and pulmonary hypertension (PH) (Fig. 1), while a thoracic radiography revealed incipient signs of heart failure. A cranial CT scan disclosed air bubbles in both cavernous sinuses (Fig. 2A) and in the left infratemporal fossa (Fig. 2B). We ruled out toxic, metabolic, and infectious causes. Within 24-48 hours the patient underwent a helical CT angiography (ruling out pulmonary thromboembolism), transoesophageal echocardiography (ruling out an atrial septal defect) (Fig. 3), right cardiac catheterisation (obtaining a mean pulmonary arterial pressure of 46 mm Hg, indicating moderate PH), and a cranial MRI scan (which showed leukoaraiosis and age-dependent cortico-subcortical atrophy). The patient was diagnosed with coma secondary to a cardiogenic shock brought on by RVAE. Our patient progressed favourably and showed an excellent response to oxygen and saline therapy; in 3 months, she had recovered ad integrum.

The physiopathological mechanism of developing RVAE involves the gas entering the right heart cavities (via the superior vena cava) and the pulmonary circulation (via the pulmonary artery). This affects ventilation/perfusion, promotes right-to-left intrapulmonary shunting, and increases alveolar dead space. In addition, if the volume of air embolism is high, it can also cause PH, right ventricular overload, decreased cardiac output, and eventually low cerebral perfusion pressure and coma.[10—12] Recently, it

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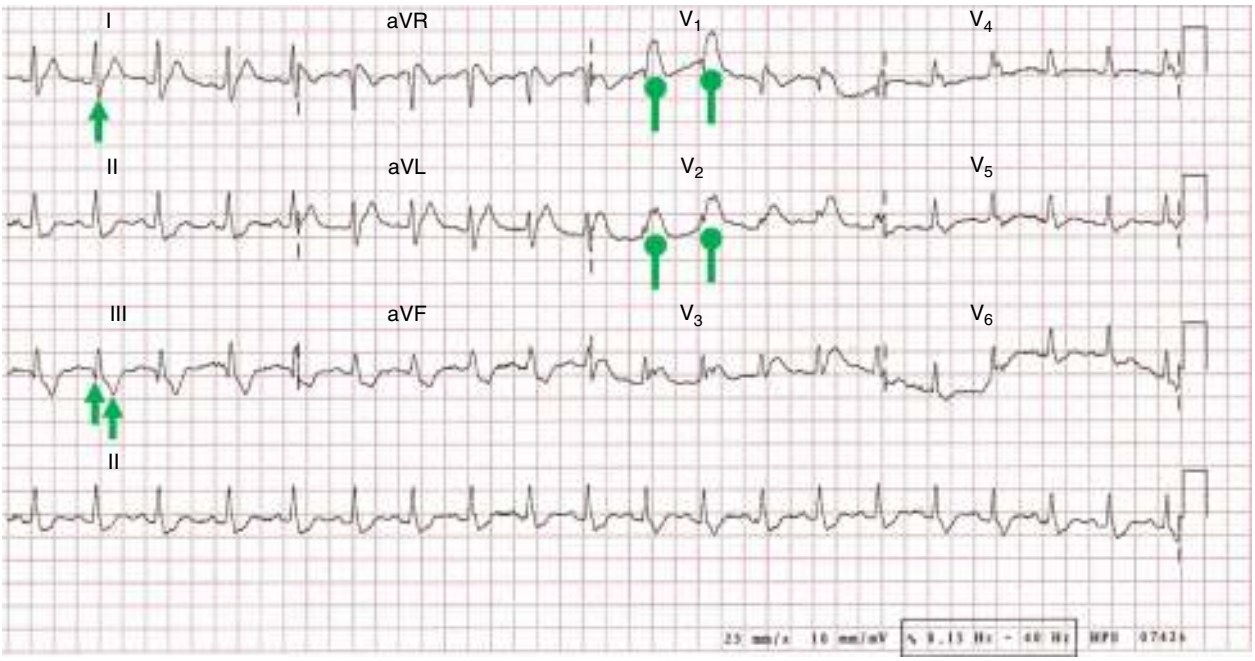

Figure 1 Electrocardiogram (ECG) showing sinus tachycardia at 120 bpm, right axis deviation, $S_1Q_3T_3$ pattern (pointed arrows), and complete right bundle branch block (rounded arrows). All of these findings were new.

was experimentally demonstrated that there is greater likelihood of air reaching the brainstem after PIV manipulation, during Valsalva manoeuvres, and in hypovolaemia (which leads to a decrease in central venous pressure), and particularly when elevating the thorax (angles $\geq 45^{\circ}$ above horizontal), and air flow rates ≥ 0.2 L/min.[13–15]

Diagnosis of RVAE, made by exclusion, is based on a high level of clinical suspicion, the presence of intracranial air in neuroimaging (before it is absorbed by the systemic circulation), and the absence of a right-to-left cardiac shunt in an echocardiogram. Treatment includes: an occlusive dressing over the area to prevent more air entering, the Durant manoeuvre (placing the patient in the left lateral decubitus position and the Trendelenburg position), support measures (volume expansion), inotropes, antiepileptics, and oxygen (100%) (which reduces the size of the air embolism by

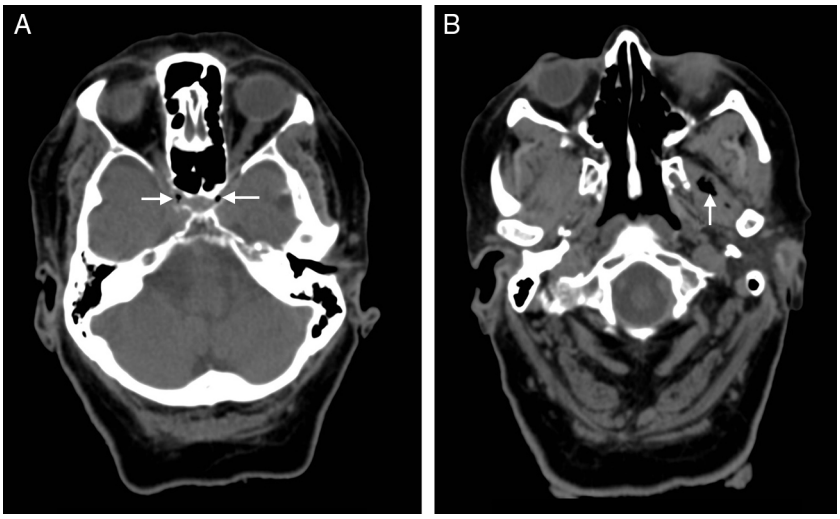

Figure 2 A simple axial CT scan performed 50 minutes after symptom onset revealed air bubbles (arrow heads) inside both cavernous sinuses in the parasellar region (A) and at the level of the left lateral pterygoid muscle (B), adjacent to the trajectory of both internal carotid arteries, which exhibit signs of intracavernous atheromatous calcification.

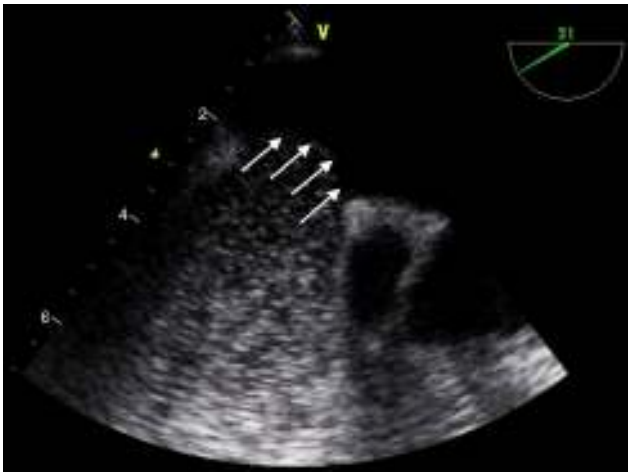

Figure 3 A transoesophageal echocardiogram was performed 24 hours after symptom onset. Spontaneous echo contrast was not seen even during the Valsalva manoeuvre. The image reveals a protrusion of the interatrial septum (indicative of increased pressure in the right cardiac cavities; arrows), but no interatrial connection or air bubbles (probably dissolved in the systemic circulation).

increasing the output gradient of nitrogen inside the air bubbles), and hyperbaric oxygen therapy, if available, in more severe cases.[13,16,17]

Ours is the first reported case of coma secondary to RVAE caused by PIV use and shows the pathophysiological mechanism of RVAE. 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 but potentially devastating complication. Late diagnosis and treatment of RVAE may result in irreversible sequelae. Therefore, this entity should be included in the differential diagnosis of patients with PIV lines in certain clinical contexts who display cardiopulmonary and neurological symptoms not explained by other causes.

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M. León Ruiz[a,*], J. Benito-León[b,c,d], M.A. García-Soldevilla[a], J.A. Arranz Caso[e], E. García-Albea Ristol[a,f]

[a] *Servicio de Neurología, Hospital Universitario Príncipe de Asturias, Alcalá de Henares, Madrid, Spain*
[b] *Servicio de Neurología, Hospital Universitario 12 de Octubre, Madrid, Spain*
[c] *Departamento de Medicina, Facultad de Medicina, Universidad Complutense, Madrid, Spain*

[d] *Centro de Investigación Biomédica en Red sobre Enfermedades Neurodegenerativas (CIBERNED), Madrid, Spain*
[e] *Servicio de Medicina Interna, Hospital Universitario Príncipe de Asturias, Alcalá de Henares, Madrid, Spain*
[f] *Departamento de Medicina, Facultad de Medicina, Universidad de Alcalá, Alcalá de Henares, Madrid, Spain*

* Corresponding author.
E-mail address: pistolpete271285@hotmail.com
(M. León Ruiz).

Acute hydrocephalus secondary to carbon monoxide poisoning☆

Hidrocefalia aguda secundaria a intoxicación por monóxido de carbono

Dear Editor:

Carbon monoxide (CO) poisoning is one of the most common causes of morbidity and mortality by poisoning. This type of toxicity results from tissue hypoxia and CO-mediated damage at the cellular level. Neurological sequelae constitute the principal cause of associated morbidity. The development of acute hydrocephalus is an extremely rare complication. We present a case of CO poisoning in an adult male who developed acute obstructive hydrocephalus secondary to bilateral cerebellar oedema.

The patient was a 38-year-old man with a medical history hypothyroidism treated with thyroid hormone replacement therapy. He also presented personality disorder and had attempted suicide several times. He was found unconscious inside a car inhaling exhaust gases, and beside him were 2 empty blister packs of benzodiazepines. Upon arrival at the emergency department, the patient had a low level of consciousness (Glasgow Coma Scale = 3) and mid-dilated miotic pupils which were poorly reactive; as a result, he underwent orotracheal intubation. A cranial CT (Fig. 1) revealed diffuse hypodensities in both cerebellar hemispheres, basal temporal white matter bilaterally, both internal capsules, and the globus pallidus. The patient was admitted to the intensive care unit (ICU). A urine toxicology test revealed benzodiazepine and methadone, while CO-oximetry showed carboxyhaemoglobin levels of 23.6%. The patient was therefore mechanically ventilated with 100% oxygen until his carboxyhaemoglobin levels decreased to 0.9%, approximately 6 hours later. In the 48 hours after being admitted to the ICU, the patient's neurological symptoms improved to the point where he was able to obey simple instructions, although he remained drowsy. However, 72 hours after admission to the ICU, the patient's state of consciousness deteriorated suddenly. An additional cranial CT (Fig. 1) revealed severe hydrocephalus affecting the lateral ventricles and third ventricle, resulting in a significant mass effect. The patient underwent emergency surgery: first, an external ventricular drain was put in place, from which exuded a clear liquid under high pressure, and then, a decompressive craniectomy of the posterior fossa was performed to relieve cerebellar herniation. After surgery, the patient remained in coma for the next 5 days (flexion—extension of both upper limbs was the only response to painful stimuli). A follow-up cranial MRI scan performed 4 days after surgery (Fig. 2) showed multiple cerebral infarcts in an early subacute stage, extensively affecting the limbic system, hippocampus, fornix, and basal temporal area bilaterally. Patchy areas of small bilateral cortical infarcts could also be observed in the frontal and parietal lobes, as well as extensive infarcts in both cerebellar hemispheres, with the most damage occurring in the territory of the superior and anterior inferior cerebellar arteries; no signs of hydrocephalus were seen. Taken as a whole, these findings suggest multiple anoxic-ischaemic encephalic lesions secondary to CO poisoning. As of the fifth day after surgery, our patient's neurological symptoms had improved progressively, reaching an adequate level of consciousness and showing no focal neurological signs. Three days later, hydrocephalus resolved and the external ventricular drain was removed after remaining closed for 48 hours without visible neurological deterioration.

The clinical presentation and radiological findings of CO poisoning vary greatly. There are 3 mechanisms by which CO damages the central nervous system. Firstly, CO directly causes diffuse hypoxic-ischaemic encephalopathy, which predominantly affects grey matter. Second, although to a lesser extent, it may also cause focal lesions to the cerebral cortex, especially the hippocampus and temporal lobes. Cortical damage may manifest as transient vasogenic oedema or as a necrosis with infarct areas in the absence of cerebral artery occlusion. Third, it may cause white matter demyelination. This finding, normally undetectable in the acute phase of intoxication, is considered the cause of late-onset neuropsychiatric syndrome. This syndrome generally develops after a lucid interval, and the most common symptoms are mental deterioration (amnesia, cognitive dysfunction), emotional disorders (depression, anxiety, mutism), urinary and faecal incontinence, and motor disorders (gait alterations, parkinsonian symptoms). The globus pallidus is the structure most frequently affected by CO poisoning; damage is usually immediate, bilateral, and predominantly affects the anterior 2 thirds of this structure. Occasionally the rest of the basal ganglia are affected (putamen, caudate nucleus, thalamus); in this case, the lesions are typically asymmetrical. The brainstem and the cerebellum are less

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