Cerebral air embolism: the crucial role of imaging.

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Learning objectives

Review the literature with respect to aetiology, diagnosis and current treatment options for cerebral air embolism.

Describe the radiologic findings that define cerebral embolism and ischemia secondary to arterial occlusion.
Background

We can define air embolism as the irruption of gas in the circulation (arterial and / or venous), which will result in varying degrees of tissue ischemia. It could be a fatal event occurring usually iatrogenically and an early diagnosis is essential. Air bubbles (and their effects) can be detected in the cranial vasculature by CT scan or MRI during the early phase of cerebral gas embolism.

When air enters the venous system, it can reach the cerebral vasculature in a number of ways (atrial sepal defect, pulmonary arterial-venous malformation...). For example, we report two cases which showed the presence of a permeable foramen oval that facilitates a paradoxical air embolism into the cerebral arterial tree.

Air in the vessels causes blocking of blood flow and also induces an inflammatory reaction. Cerebral gas bubbles can be detected on the earliest CT scan. Later, may be can see spontaneous resolution of air in the vessels but also an evolving infarct. Neurological manifestations include encephalopathy, seizures and ischemic infarcts with resultant focal neurological deficits.
DEFINITION AND ETIOLOGY OF AIR EMBOLISM.

We can define air embolism, as the entrainment of air in the bloodstream (arterial and/or venous), which will cause various degrees of ischemia and tissue suffering in the affected territory.

The gas that is introduced into the arterial system will reach the central nervous system producing the symptomatology. Although this entity is described among the complications that divers present, this is not the only way to produce an aeroembolism. There is also iatrogenic gas embolism, which we will develop below.

In our center all cases of gas embolism are of an etiology not related to diving. According to the bibliography consulted, the most frequent causes of gas embolism beyond diving are:

- Cardiac surgery with or without extracorporeal circulation.
- Invasive venous or arterial procedures.
- Thoracic, head and neck surgery.
- Penetrating wounds of head and thorax.
- Mechanical respiratory assistance (may cause lung parenchyma rupture and cause gas injection into the bloodstream).
- Injection of gas into tissues or cavities.
- Central venous catheter.

IATROGENIC AEROEMBOLIA: WHAT IS PARADOXICAL EMBOLISM?

Gaseous embolism can occur as a complication of various surgical, diagnostic and therapeutic acts.

The air can enter directly into the arterial circulation, but also the air that reaches the venous circulation will be able to reach the arterial system through, for example, a patent foramen ovale (present according to the bibliography consulted in a 25-35 % of the healthy population [autopsy findings]), which would allow the passage of gas from right to left. Another way of passage would be the existence of a patent intrapulmonary shunt, which allows the gas to pass freely to the arterial system.
This is what we call paradoxical embolism. In the case of permeable foramen ovale (PFO), it is an atrial septal defect that is normally not functioning because there is more pressure in the left cavities.

In order to be possible a hemodynamically active right-to-left shunt has to increase the pressure in the right cavities, as in pulmonary hypertension and during the Valsalva maneuver.

**EFFECTS OF CEREBRAL AIR EMBOLISM**

In cerebral gas embolism, once the gas bounce into the arterial circulation, the bubbles will block the circulation with the consequent tissue ischemia of the parenchyma, accompanied by edema secondary to hypoxia (cytotoxic), which will further compromise the microcirculation.

The gas interface will activate the platelets, which will trigger a disseminated intravascular coagulation (DIC), also producing an endothelial edema that will decrease capillary lumen and a greater affection of the blood flow.

The end result of this process is an acute ischemic stroke.

It is logical to think that the clinical manifestations are going to be identical to another acute ischemic stroke of different etiology, and the neurological deficit will depend on the territory (or territories) affected.

**IMAGE FINDINGS**

Cerebral gas embolism is one of the less frequent causes of pneumocephalus (intracranial gas), which is easily identifiable on CT / MRI (highly sensitive and specific). In order to visualize it in the skull radiograph the amount of gas must be quite high, so it is not the appropriate diagnostic method.

The location of the intracranial air will give us the key to a correct diagnosis: in the case of air embolism it will be located following an arterial / vascular disposition; as opposed to post-traumatic or post-surgical pneumoencephalus where the gas is going to be located in the extraxial space.
In addition to visualizing the air bubbles following the linear pattern of branching of the arterial vessels, with the imaging tests (CT and MRI) we will be able to see the effects caused by the embolism in the cerebral parenchyma.

Thus, in the CT scan we will observe hypodense areas with sulcal effacement and poor differentiation between gray and white matter secondary to ischemia (in RM, they would restrict the signal in the diffusion weighted imaging in the same way as any other acute ischemic injury of any other cause) and edema.

If the diagnostic study is performed very early, we may not see the effects derived from the presence of intraarterial gas in the cerebral parenchyma, because the state of ischemia and tissue edema would not yet have been established.

Next, we provide some images of cases of cerebral gas embolism diagnosed in our center.

**Case 1:**

A 55-year-old woman in the post-operative of a perforated duodenal diverticulum begins with decreased consciousness and decerebrate posture.

Head CT scan without contrast is performed urgently, where it is observed the presence of multiple bilateral air bubbles in relation to the sulcus, especially of the right convexity (where they acquire a linear aspect), suggestive as the first possibility of gas embolism. No lesions are seen at the intraparenchymal level. Fig. 1 on page 9

The next day a new cranial CT of control is realized visualizing the disappearance of practically all the gaseous bubbles except for an isolated bubble in left occipital level. The rest of the study doesn't present relevant radiological findings. Fig. 2 on page 9

Three days after the onset of the condition, the patient continues to present left hemiplegia despite hyperbaric chamber therapy. CT is performed and An asymmetry with sulcal effacement of the right frontoparietal convexity is observed, in relation to early signs of infarction secondary to the gas embolic event. No bubbles are seen in the vessels. Fig. 3 on page 10
Due to this finding a cranial MRI without contrast is performed, where acute ischemic lesions (with significant signal restriction in diffusion weighted imaging) become even more patent.

After a month, a new cranial MRI of control is performed where we appreciate the evolution of these lesions, that no longer present increased DWI signal but are hyperintense on FLAIR images. Fig. 4 on page 11

The echocardiogram confirmed in this patient the existence of a 9x7 mm PFO with right-to-left and left-to-right shunt.

**Case 2:**

A 70-year-old woman with leukemia who is hospitalized for polychemotherapy. She has a central venous catheter with induration and redness. After removal of the catheter, cyanosis of the skin and mucous membranes (without edema or hematoma) was observed, as well as tachypnea and a clinical pattern of disorientation with abrupt reduction of the level of consciousness without response to painful stimuli.

Head CT scan without contrast is performed urgently where it is observed numerous gas bubbles in supratentorial vessels with lineal distribution, associated with slight cerebral edema. Findings compatible with massive cerebral air embolism. Fig. 5 on page 12 Fig. 6 on page 12

Gas bubbles are also observed in the left jugulocarotid space and both masticator spaces. Fig. 7 on page 13

The next day, due to the worsening of the patient, a new cranial CT was performed, where significant diffuse cerebral edema was observed, with obliteration of perimesencephalic cisterns, reduction of supratentorial ventricular system size and loss of sulci.

There are also cortical and subcortical hipodensities at bilateral frontoparietal lobes (with right predominance), compatible with cortical infarctions. There was no longer any presence of intravascular air. Fig. 8 on page 13 Fig. 9 on page 14

In transesophageal echocardiography it is observed that after injecting agitated saline contrast, during Valsalva maneuver, there is contrast passage from right to left cavities through PFO.
Removal of a central venous catheter can cause air suction, causing venous gas embolism. When the patient is placed in a semi-sitting position, the natural tendency of the air is to ascend, causing a cerebral arterial air embolism secondary to the passage of air through the patent foramen ovale (as is the case in our patient).

**TREATMENT**

Treatment should be done early to avoid the spread of tissue ischemia. In addition to life support and patient monitoring (as in any other acute ischemic stroke), the main thing is Hyperbaric oxygen therapy, which favors the decrease in the number and size of the intravascular gaseous bubbles.

Breathing hyperbaric oxygen creates a positive gradient around each bubble, which causes it to decrease in size by diffusion of the gas contained from within it to the outside. In addition, a high oxygen partial pressure is achieved to combat tissue hypoxia.
Images for this section:

Fig. 1: Multiple bilateral air bubbles (<) in relation to the sulcis, mainly in the right cerebral convexity (where they acquire lineal appearance). Findings compatible with gas embolism.

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**Fig. 2:** Same patient, the next day. Only an isolated bubble persists in the left occipital lobe. There are no alterations in the cerebral parenchyma.

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Fig. 3: Same patient who during hyperbaric chamber therapy continues with symptoms (left hemiplegia). We observed an asymmetry with sulcal effacement at the right frontoparietal convexity. Findings suggesting ischaemic stroke in relation to the embolic event. No bubbles are seen in the blood vessels.
**Fig. 4:** MRI (FLAIR): There is improvement of all lesions, with two small right occipital and parietal cortical ischemic lesions persisting.

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**Fig. 5:** Head CT scan without contrast: Numerous gas bubbles with lineal distribution (apparently intravascular). Findings compatible with massive cerebral air embolism.

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Fig. 6: Same patient as in the previous figure.

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Fig. 7: Same patient: Gas bubbles (<) in the left jugulocarotid space and both masticator spaces.

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Fig. 8: Significant diffuse cerebral edema. Loss of sulci. Cortical and subcortical bilateral hipodensities compatible with ischemia.
Fig. 9: Obliteration of perimesencephalic cisterns.

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Conclusion

In the cerebral air embolism the clinical diagnosis may be difficult. Imaging can therefore play a crucial role in revealing the presence of intracranial gas bubbles on CT or MRI scans.

Prevention, as well as early diagnosis and management, may reduce morbidity and mortality.
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