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Letter to Editor

Cerebral Air Embolism: Stroke from an Unseen Enemy

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A cerebral air embolism (CAE) can be a catastrophic cause of ischemic stroke. The true incidence is difficult to track because it is often missed or mistaken, but it may be higher than thought.

Venous emboli can cause cerebral venous stroke and also arterial air embolism through paradoxical embolization, most commonly via a patent foramen ovale, which is present in about 24.2% of the general population. In fact, arterial stroke does not require a patent foramen ovale or arterial septal defect (it can occur due to incomplete filtering of the air in the normal pulmonary capillaries or due to presence of a pulmonary arteriovenous malformation). Contrary to the popular belief, it can occur irrespective of the amount of air (even a small amount of air is enough to cause a neurological event).

The mechanisms of stroke in cerebral embolism are local obstruction of blood flow by air emboli resulting in ischemic infarction or direct endothelial injury with resultant blood–brain barrier breakdown and in situ thrombus formation leading to ischemic infarction.

Cerebral venous air embolism is common and may occur during retrograde flow at the time of inserting, manipulating, or removing a central line or peripheral intravenous line, as well as during hemodialysis line placement, intravenous contrast injection, pacemaker or defibrillator placement, radiofrequency cardiac ablation, hysteroscopy, and, rarely, endoscopy. Other causes are increased intrathoracic pressure, such as during mechanical ventilation, lung tumor resection, percutaneous lung biopsy, posterior fossa/cervical disc surgery, and even during mechanical thrombectomy, especially at the time of contrast injection. The special situations or precipitating factors for these events are the presence of insufficient valves of jugular veins, hypovolemia leading to low venous pressure, and an upright position (>45°) during the procedures causing a lower pressure gradient for the gas bubbles.

The important points regarding cerebral embolism we should remember:

1. The temporal correlation between the precipitating event and the clinical event (air embolism can cause stroke/seizure with or without coinciding air embolisms in the pulmonary and/or cardiac circulation) should be kept in mind—usually it occurs suddenly and shortly after the procedure. However, rare incidence of cerebral air embolism has been reported seven hours after central line removal underscoring requirement of high degree of suspicion for this entity [1].
2. An immediate CT scan rather than an MRI of the brain is the investigation of choice and may include the “minimum intensity projection” (MinIP) modality with higher sensitivity in case of a diagnostic challenge [2].

The gas can be resorbed rather rapidly; the initial CT may not show evidence of air directly but only the consequences of CAE, such as cerebral infarction or brain edema. The rate of air embolism resorption depends on air volume, bubble shape (with an elongated linear bubble taking longer to resorb compared to a spherical one), and blood flow velocity [2]. CAE should resorb completely in minutes to hours given that no more air is entering the circulation.

Findings in MRI are also challenging, as the imaging findings are non-specific (restricted diffusion along the cortical gray matter in a gyriform pattern commonly involving both cerebral hemispheres) and include cortical diffusion restriction (cytotoxic edema) areas and adjacent areas of vasogenic edema (as a result of a regional inflammatory reaction). Venous strokes accompany more cerebral edema compared to arterial strokes.

To make things more challenging, there may be delayed evolution of cortical ischemia with adjacent white matter edema [3]. Hence, a patient may have deficits with normal imaging only to appear later, underscoring the importance of repeated or follow-up imaging if the suspicion is high.

3. In cases of multiple territorial cryptogenic strokes where brain imaging occurred late, we need to seriously consider this entity and retake proper history and do investigations accordingly.

This will allow us to rediscover a percentage of strokes with unknown etiology to have a definite etiology and prevent patients from having unnecessary exposure to bleeding risks secondary to antithrombotics (especially anticoagulants) when we wrongly understand these multiple territorial strokes to be of cardioembolic origin.

4. We need to be more vigilant regarding this procedural complication and keep all departmental staff of the institute well informed so that detection and treatment can be done immediately to avoid catastrophic complications.

Following proper methodology for the procedure and patient positioning can significantly reduce the risk of air embolism. As an example, during mechanical thrombectomy, it is important to use an air filter during the contrast injection, which should be done slowly, as well as priming the catheter to eliminate all air in the circuit.

5. In diagnosed cases, we need to be aware of the issue so that we can do our best to avoid repeated incidences of air embolism and stroke, especially in those cases where the clinical situation will obviously reoccur. As for example, patients on haemodialysis or if there are predisposing factors for air embolism, like the presence of a patent foramen ovale/pulmonary arteriovenous fistula or central origin of the right carotid artery and the right internal jugular vein (chance of CAE higher).

Conflicting Interest: (If present, give more details): Nil

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