Imaging findings in cerebral air embolism after hyperbaric oxygen therapy treatment

Luís Cardoso¹, Ricardo Martins¹, Gonçalo Videira², Catarina Pinto¹, Rui Felgueiras¹, José Pedro R. Pereira¹, and Ângelo Carneiro¹

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Abstract

Introduction: Cerebral air embolism is a rare neurologic complication that can occur in the setting of several medical procedures, with air entering vascular structures. Proposed mechanisms for cerebral lesions induced by air embolism are: ischemic, due to interruption of cerebral arterial flow, or inflammatory, because air microbubbles impair the vascular endothelium, causing a breakdown of the blood–brain barrier (BBB), activation of immune cells and inflammatory proteins and platelet and leucocyte adhesion.

Case Presentation: A transthoracic CT-guided lung biopsy was performed in a 75-year-old woman. After the procedure the patient presented sudden loss of consciousness followed by quick recovery and focal neurologic deficits (left neglect and hemiparesis), NIHSS 10. CT, CTA and CT-perfusion showed no remarkable changes besides minimal amount of air in the cavernous sinus. The patient performed one session of hyperbaric oxygen therapy (HBO) that started 4 hours after symptoms onset. The following days showed progressive recovery and one week later had no neurological deficits. MR two days after event showed right cortico-subcortical fronto-parietal foci of hyperintensity on T2 weighted sequences and in the deeper white matter of centrum semiovale; no diffusion restriction and elevation of centrum semiovale signal on ADC.

Conclusions: Hyperbaric oxygen therapy is associated with improved cerebral oxygenation, reduced (BBB) breakdown, decreased inflammation, reduced cerebral oedema, reduced metabolic derangement and decreased apoptotic cell death. This patient had no restricted diffusion in the acute phase, suggesting beneficial effects of HBO in reducing cytotoxic oedema and possible secondary brain infarction.