



Cerebral Air Embolism with Ischemic Cerebrovascular Stroke, Complicated by Central Venous Line

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Case Presentation

A 79-year old female patient, known with hypertension, diabetes, ischemic heart disease and paroxysmal AF, on regular treatment, admitted to ICU, Bugshan Hospital, Jeddah, KSA, with acute chest infection (bronchopneumonia), dehydration and desaturation, the patient was intubated and ventilated on CPAP mode, then central venous line was inserted for IV fluids and medications including antibiotics, shortly after starting IV medication, the patient became suddenly desaturated, irritable, depressed conscious level and developed repeated generalized seizures and facial myoclonus, neurological assessment at that time revealed GCS: 5/15 (extension to pain), bilaterally irreactive pinpoint pupils, depressed pharyngeal and cough reflexes, bilateral flaccid weakness and equivocal plantar reflexes, the patients was shifted for urgent non contrast CT brain, that showed massive, subcortical, cerebral air embolism (no skull fracture), bilateral PCA territory infarctions and mild sulcal effacement, indicating brain edema (Figure 1A-H), CV catheter was removed directly, leviteracetam 500 mg twice daily were started as antiepileptic, 100% high flow oxygen and general supportive cure. Next day the patient condition improved, no more seizures, her GCS improved to 9/15 (open eyes spontaneously and localizing to pain), the patient became quiet on MV (no more irritability). After stabilization, TEE was done revealed diastolic dysfunction with no PFO, contrast enhanced CT chest showed no hemo or pneumothorax, contrast enhanced CT abdomen showed air in the portal vein and left hepatic lobe but no mesenteric ischemia or occlusion. Two days later, follow up CT brain was done and showed, completely resolved cerebral air embolism and brain edema and some improvement in global cerebral perfusion (Figure 2A-H).

Discussion

For this old patient with multiple co-morbid conditions, her sudden neurological deterioration was explained by massive cerebral air embolism and bilateral parieto-occipital infarction, away from central line source, no source of air embolism was detected, responding well to conservative management (proper positioning, high flow oxygen, antiepileptic medication and general supportive cure), improvement was achieved in both clinical and radiological findings, NCCT brain initially showed extensive and multi located subcortical air embolism, bilateral posterior hemispheric infarctions and mild diffuse brain edema, clinically GCS was 5/15 and repeated convulsions, follow up NCCT brain showed marked improvement, also the patient improved clinically as GCS became 9/15 and no seizures after 2 days from the onset. Air embolism is a serious and life threatening event occurring usually in the ICU and OR settings. Air commonly enters the venous system and sometimes the arterial system with disastrous cardiac, pulmonary or neurological effects and is associated with a high morbidity and mortality [1]. Iatrogenic etiology as a cause of air embolism such as during insertion of central venous catheter, considered as Second etiological reason after surgical procedures [2]. Neurological manifestations are due to two reasons; first the cardiovascular collapse causing reduced cardiac output leading to cerebral hypo-perfusion (as in our case), secondly direct paradoxical cerebral embolism [1]. The goal of treatment in VAE is to prevent further air entry, reduction in volume of air entrained, and haemodynamic support [3]. Administration of 100% oxygen will maximize the patient's oxygenation as well as reduces embolus volume by eliminating nitrogen [4]. Trendelenburg's position as a favorable placement to optimize haemodynamics is now controversial [5].

Conclusion

Symptomatic management and general supportive cure may be enough to improve patient with cerebral air embolism, as in our case here.

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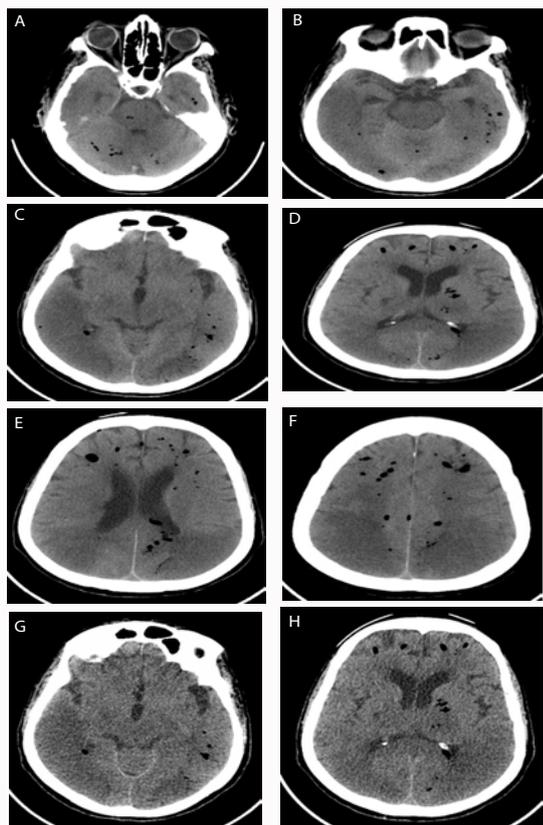


Figure 1A-H: Non contrast CT brain showed extensive and widely distributed cerebral air embolism with right temporal and bilateral occipito-parietal infarctions.

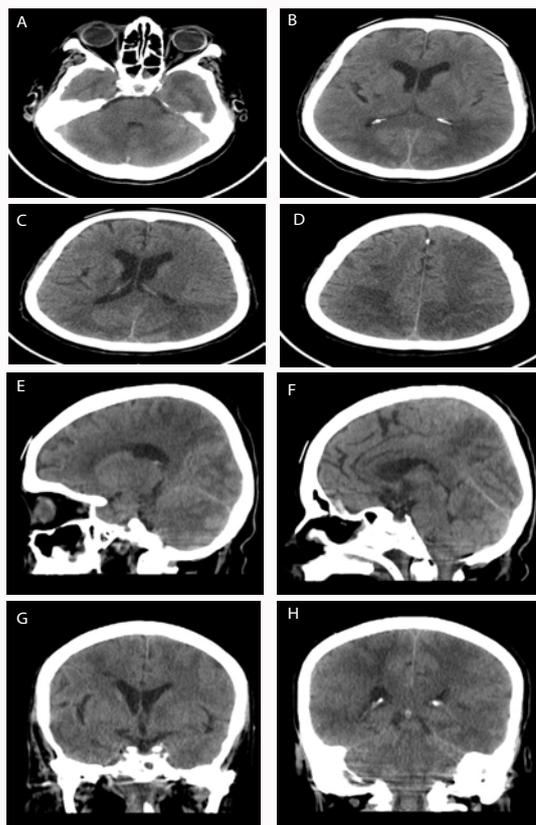


Figure 2A-H: Follow up NCCT brain after 2 days showed, completely resolved cerebral air embolism and some improvement in global cerebral perfusion (through reduction of ischemic area in comparison with initial image).

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