

Successful late treatment of venous air embolism with hyperbaric oxygen

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Summary: A case of haemodialysis-associated venous air embolism is described. The patient commenced hyperbaric oxygen therapy 21 hours after the event when, despite appearing decerebrate, he made a complete recovery. This case underlines the importance of all clinicians being aware of those centres with facilities for hyperbaric therapy and the need to refer all patients with cerebral air embolism even following a prolonged delay.

Introduction

Venous air embolism is a potentially life threatening complication of haemodialysis with an incidence of approximately 1 in 2000 episodes of dialysis.¹ Iatrogenic air embolism may also complicate central venous catheterization,² neurosurgery,³ and cardiac surgery.⁴ Also, many minor occurrences leading to subsequent neurological damage are likely to be missed, for instance during coronary artery bypass surgery.⁵ The incidence in this country is unknown but at least 20,000 cases of air embolism occur annually in the USA.⁶

Following venous air embolism severe haemodynamic and neurological disturbances may occur. The haemodynamic changes are predominantly due to gas in the right side of the heart and pulmonary circulation. Air entering the right atrium and ventricle forms a foam which passes into the pulmonary circulation where occlusion occurs. Failure of the pulmonary circulation and left ventricular filling leads to systemic hypotension and circulatory collapse. Neurological disturbances follow arterialization of the air via a patent foramen ovale⁷ or migration of bubbles through the pulmonary microcirculation.⁸ Thus, gas bubbles entering the cerebral circulation causing ischaemia and cerebral oedema can lead to profound neurological damage. In addition to these direct effects other factors contributing to the pathophysiology include platelet aggregation and activation of Factor XII, and an immediate increase in blood–brain barrier permeability.⁶

Hyperbaric oxygen therapy is the recommended

treatment for the neurological sequelae of air embolism but the completeness of recovery may be directly related to the rapidity in instituting therapy.⁹

We describe a patient who commenced treatment 21 hours after the event at a time when he appeared decerebrate and who made a complete recovery.

Case report

The patient was a 47 year old male with renal failure due to lupus nephritis on hospital based self-supervised haemodialysis for 4 years. While putting himself on dialysis he flushed a large quantity of air (approximately 150 ml) through his venous line and collapsed. He was placed head down on his left side and recovered consciousness immediately with no apparent neurological deficit. During the next 4 hours, despite continuous oxygen by face mask, he became progressively more drowsy and was treated with dexamethasone 4 mg every 6 hours. His conscious level deteriorated further and 15 hours later he was unrousable and exhibited decerebrate posture to painful stimuli. He was mildly fluid overloaded but with no evidence of heart failure. The retinal fundi looked normal.

He was transferred to the Regional Hospital for Infectious Diseases and 21 hours after the air embolism he was placed in a single person hyperbaric chamber (Standard Clinical Vickers RH53) and pressure taken to 2.8 atmospheres with 100% oxygen. This first attempt at hyperbaric treatment was aborted after one hour when he developed pulmonary oedema and convulsions. Hypertonic

peritoneal dialysis was instituted and 2.5 litres of fluid removed over 6 hours. He remained unrousable and decerebrate. He was again placed in the hyperbaric chamber. The pressure was taken to 2.8 atmospheres (100% oxygen) and maintained for 2 hours before slow decompression over 30 minutes. Following removal from the chamber (31 hours after the event) he was conscious and able to answer simple questions but a mild left hemiparesis persisted. Seven hours later, a further 2 hour session of hyperbaric oxygen was undertaken and following this the patient was fully coherent with no detectable neurological deficit. His complete neurological recovery was maintained and eventually he returned to self-supervised haemodialysis in a minimal care unit close to his home. He underwent successful replacement of his incompetent aortic valve and a Hartman's operation for a perforated sigmoid colon 3 years and 3½ years later respectively. He finally died following the development of a gangrenous colon and faecal peritonitis 4½ years after the air embolism. Neuropathological examination failed to elicit any evidence of residual cerebral damage.

Discussion

Following venous air embolism, the recommended methods of maintaining circulation include immediate tipping of the patient into the left lateral decubitus (Durant) position followed by external cardiac massage¹⁰ or aspiration of air from the apex of the right ventricle.¹¹ Oxygen is absorbed but nitrogen bubbles remain and may affect cerebral, coronary or mesenteric circulations.

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