- 30 Ogilvy CS, Carter BS. Stratification of outcome for surgically treated unruptured intracranial aneurysms. *Neurosurgery* 2003;**52**:82–7.
- 31 King JT Jr, Berlin JA, Flamm ES. Morbidity and mortality from elective surgery for asymptomatic, unruptured, intracranial aneurysms: a meta-analysis. *J Neurosurg* 1994;**81**:837–42.
- 32 Taha MM, Nakahara I, Higashi T, et al. Endovascular embolization vs surgical clipping in treatment of cerebral aneurysms: morbidity and mortality with short-term outcome. *Surg Neurol* 2006;**66**:277–84.
- 33 Raaymakers TW, Rinkel GJ, Limburg M, et al. Mortality and morbidity of surgery for unruptured intracranial aneurysms: a meta-analysis. *Stroke* 1998;**29**:1531–8.
- 34 Hempelmann RG, Barth H, Buhl R, et al. Clinical outcome after surgery of intracranial unruptured aneurysms: results of a series between 1991 and 2001. *Acta Neurochir Suppl* 2002;**82**:51–4.
- 35 Tsukahara T, Murakami N, Sakurai Y, et al. Treatment of unruptured cerebral aneurysms; a multi-center study at Japanese national hospitals. *Acta Neurochir Suppl* 2005;**94**:77–85.
- 36 Wirth FP, Laws ER Jr, Piepgras D, et al. Surgical treatment of incidental intracranial aneurysms. *Neurosurgery* 1983;**12**:507–11.
- 37 Deruty R, Pelissou-Guyotat I, Mottolese C, et al. Management of unruptured cerebral aneurysms. *Neurol Res* 1996;**18**:39–44.
- 38 Grunwald IQ, Papanagiotou P, Politi M, et al. Endovascular treatment of unruptured intracranial aneurysms: occurrence of thromboembolic events. *Neurosurgery* 2006;**58**:612–18.
- 39 Niskanen M, Koivisto T, Rinne J, et al. Complications and postoperative care in patients undergoing treatment for unruptured intracranial aneurysms. *J Neurosurg Anesthesiol* 2005;**17**:100–5.

NEUROLOGICAL PICTURE

doi: 10.1136/jnnp.2006.102954

Cerebral air emboli on T2-weighted gradient-echo magnetic resonance imaging

Cerebral air embolism can sometimes be identified on brain CT,¹ and subsequent multiple infarcts can be observed on diffusion-weighted imaging (DWI).² However, air itself on a T2-weighted gradient-echo image (GRE) has not been demonstrated.

A 77-year-old man was hospitalised for pneumonia. Eight days after admission, while sitting upright in bed, he suddenly became unresponsive after violent coughing with a central venous catheter disconnected. He also exhibited conjugate eye deviation to the left, quadriplegia, bilateral Babinski signs and subsequent generalised seizure. A brain CT and GRE revealed multiple lesions, apparently representing air, in the bi-hemispheric corticomedullary junctional area (fig 1A, B). DWI showed multiple infarctions restricted to the cortex nearby the air (fig 1C). The location of acute infarcts on DWI roughly coincided with the location of air emboli on GRE.

We believe that this patient experienced cerebral air embolism as a complication of a central venous catheter. Correspondence of the hypointense lesions on GRE with those on the CT scan supports the presence of air emboli on GRE. Air in the magnetic field has very low magnetic susceptibility and can cause signal loss on MRI, especially on GRE.³ The preferential involvement of the frontal lobe by air emboli may be related to the position of the patient (sitting position) at the time of air entry and the low gravity of air. Air bubbles have the highest chance of being entrapped in the very small sized end arteries (30–60 µm sized small arteries) in the cortical layers.⁴ The entrapped air bubbles can obstruct local blood flow, breakdown the blood–brain barrier, activate inflammatory reactions and then lead to infarcts.

This case report suggests that the differential diagnosis of multiple small foci of marked hypointensity on GRE should include cerebral air embolism, particularly

Figure 1 Brain CT performed 30 min after symptom onset revealed multiple low density lesions (A). A T2-weighted gradient-echo image (GRE) obtained 5 h later showed multiple hypointense lesions in the bi-hemispheric corticomedullary junction (B) consistent with air (arrows). Diffusion-weighted imaging performed 5 days later showed multiple infarcts in the cortex area nearby the air (C).

in patients who have sudden cardiopulmonary or neurological findings in the presence of a central venous catheter. Microbleeds in the areas with injury to the blood–brain barrier caused by air embolism should also be considered as a differential diagnosis. Serial MRI studies, including GRE and DWI in patients with cerebral air embolism, may broaden our understanding of the natural history of air emboli and its impact on tissue injury.

Sang-Beom Jeon, Dong-Wha Kang

Department of Neurology, Asan Medical Centre,
University of Ulsan College of Medicine, Seoul,
South Korea

Correspondence to: Dong-Wha Kang,
Department of Neurology, Asan Medical Centre,
University of Ulsan College of Medicine, 388-1
Pungnap 2-dong, Songpa-gu, Seoul 138-736,
South Korea; dwkang@amc.seoul.kr

References

- 1 Voorhies RM, Fraser RAR. Cerebral air embolism occurring at angiography and diagnosed by computerized tomography. *J Neurosurg* 1984;**60**:177–8.
- 2 Caulfield AF, Lansberg MG, Marks MP, et al. MRI characteristics of cerebral air embolism from a venous source. *Neurology* 2006;**66**:945–6.
- 3 Atlas SW. *Magnetic resonance imaging of the brain and spine*, 3rd edn. Philadelphia: Lippincott Williams & Wilkins, 2002:239–75.
- 4 Muth CM, Shank ES. Gas embolism. *N Engl J Med* 2000;**342**:476–82.