

An unusual cause of hemiplegia post-percutaneous coronary intervention

A 70-year-old man was admitted to hospital with non-ST elevation myocardial infarction. Coronary angiogram revealed a subtotally occluded ostial right coronary artery; percutaneous coronary intervention was performed with successful deployment of drug-eluting stent. As the coronary catheter did not provide sufficient support in cannulation of the right coronary artery, a significant amount of contrast was used (329 ml).

Figure 1. Computed tomography of the brain showing hyperenhancement of the frontal cortex, in keeping with contrast-induced encephalopathy.



At the end of the procedure, the patient developed left hemiparesis. An urgent computed tomography of the brain revealed opacification of the subarachnoid space of both frontal and parietal lobes, in keeping with subarachnoid haemorrhage (*Figure 1*). However, meningeal contrast enhancement could not be excluded; magnetic resonance imaging of the brain was thus performed. This revealed subtle restricted diffusion in the frontal lobes (*Figure 2*) in keeping with contrast-induced encephalopathy.

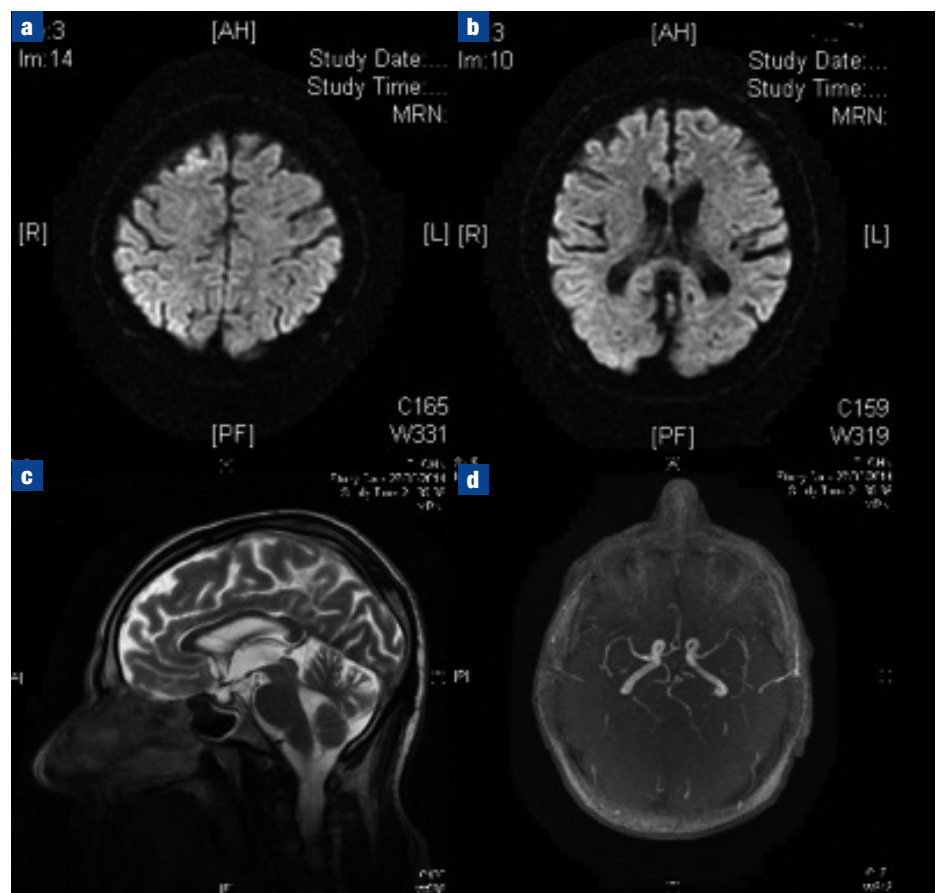
Contrast-induced encephalopathy is a very rare complication following coronary angiography with a reported incidence of 0.06% (Leong and Fanning, 2012). The underlying pathophysiology is thought

to be the result of damage to the cerebral cortex secondary to extravasation of contrast medium through a pathological blood–brain barrier, resulting in changes in oncotic pressure with consequent cerebral oedema (Yu and Dangas, 2011). The patient made an uneventful recovery following regular daily physiotherapy and was discharged home in good general condition. **BJHM**

Leong S, Fanning NF (2012) Persistent neurological deficit from iodinated contrast encephalopathy following intracranial aneurysm coiling: a case report and review of the literature. *Interv Neuroradiol* **18**(1): 33–41 (doi: 10.1177/159101991201800105)

Yu J, Dangas G (2011) New insights into the risk factors of contrast-induced encephalopathy. *J Endovasc Ther* **18**(4): 545–6 (doi: 10.1583/11-3476C.1)

Figure 2. Magnetic resonance imaging showed hyperintensity at the frontal lobes on (a and b) diffusion weighted imaging and (c) T2. d. Magnetic resonance angiography showed no occlusion of the intracranial arteries.



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