Title

Contrast encephalopathy after coiling in the setting of obstructive sleep apnoea

Authors

Liu, Jonathan J
Dahlin, Brian C
Waldau, Ben

Publication Date

2015-09-01

DOI

10.1136/bcr-2014-207503

Peer reviewed

CASE REPORT

Contrast encephalopathy after coiling in the setting of obstructive sleep apnoea

Jonathan J Liu,[1] Brian C Dahlin,[2] Ben Waldau[1]

[1]Department of Neurosurgery, UC Davis, Sacramento, California, USA
[2]Department of Radiology, UC Davis, Sacramento, California, USA

Correspondence to
Dr Ben Waldau, ben.waldau@ucdmc.ucdavis.edu

Accepted 9 September 2015

SUMMARY

Obstructive sleep apnoea (OSA) is increasingly recognised as a source of perioperative morbidity and mortality. We describe a patient with severe OSA who developed transient contrast encephalopathy after elective coiling of an anterior communicating artery aneurysm. Contrast extravasation led to cerebral oedema, seizures and delirium, which eventually completely resolved. OSA is known to be associated with a proinflammatory state that leads to hypertension, impaired endothelial repair capacity and endothelial dysfunction. Further studies are needed to clarify whether OSA increases the risk of endovascular procedures.

BACKGROUND

Obstructive sleep apnoea (OSA) is increasingly recognised as a source of perioperative morbidity and mortality. To the best of our knowledge, we describe the first case of contrast encephalopathy in the setting of severe OSA. This case is important as it can raise awareness among clinicians about the possibility of contrast encephalopathy in patients with endothelial dysfunction.

CASE PRESENTATION

A Caucasian woman with a medical history of morbid obesity (body mass index=43 kg/m^2), severe OSA, oxygen-dependent chronic obstructive pulmonary disease, hypertension and type 2 diabetes, was found, during work up for headaches, to have a 7 mm anterior communicating artery aneurysm. She was offered observation versus treatment, and she opted for treatment of the aneurysm.

A sleep study confirmed severe OSA with an apnoea–hypopnoea index of 110.7 events/hour and oxygen saturation nadir at 75%. She was neurologically intact prior to embolisation.

The endovascular procedure was performed under general anaesthesia. Selective catheterisation of the left common carotid and internal carotid arteries was difficult due to severe tortuosity (figure 1A). The left internal carotid artery was catheterised using an Envoy catheter, allowing for successful coiling of the aneurysm (figure 1B, C). At no point was there a suggestion of aneurysmal rupture. The amount of contrast used for coiling was 240 mL. Total fluoroscopy time was 123.5 min and included catheterisation of the left vertebral artery for assessment of a small left superior cerebellar artery aneurysm.

After extubation, the patient was found to be severely agitated and unable to follow commands. Her neurological status continued to decline, with subsequent respiratory failure and reintubation. A head CT scan was obtained, which showed contrast enhancement and cerebral oedema in the vascular territory of the left internal carotid artery (figure 1D). On postoperative day 2, the patient sustained two focal seizures in her right arm. Her subsequent postoperative course was primarily complicated by delirium. She was eventually extubated and slowly recovered from her delirium.

TREATMENT

Patients with OSA require continuous positive airway pressure (CPAP) to help ameliorate the repeated hypoxic and apnoeic episodes, and to reduce cardiovascular morbidity. In order to improve the endothelial function in our patient, we placed her on daily CPAP at night. Oyama *et al*[1] showed that CPAP therapy improves endothelial dysfunction, and decreases oxidative stress and inflammatory cytokines in patients with OSA.

OUTCOME AND FOLLOW-UP

The patient was discharged from the hospital and was neurologically appropriate, similar to her preoperative status. MRI of the brain 3 months after her

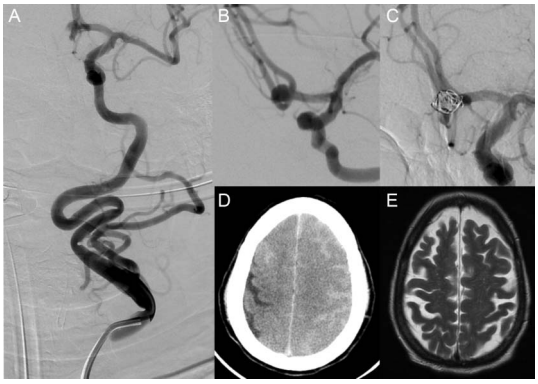

Figure 1 (A) Left common carotid angiography showing marked tortuosity of the left internal carotid artery. (B) The working angle projection delineates a 7 mm left anterior communicating artery aneurysm. (C) Postembolisation images showing obliteration of the aneurysm. (D) Postembolisation head CT scan demonstrating contrast enhancement and cerebral oedema. (E) Brain MRI 3 months after the procedure displays complete resolution of cerebral oedema.

To cite: Liu JJ, Dahlin BC, Waldau B. *BMJ Case Rep* Published online: [*please include* Day Month Year] doi:10.1136/bcr-2014-207503

endovascular procedure showed complete resolution of cerebral oedema and no stroke (figure 1E).

DISCUSSION

We present a case of contrast encephalopathy after cerebral angiography in the setting of OSA. Contrast encephalopathy is supported by cerebral swelling and contrast enhancement exclusively in the distribution of the left internal carotid artery circulation, where, in this case the catheter angiography was performed (figure 2A, B). Since the patient had an absent right A1 segment of the anterior cerebral artery (ACA), contrast enhancement in the right distal ACA territory was also seen, but not in the right middle cerebral artery territory, which is supplied by the contralateral internal carotid artery (figure 2A, B). After 18 h, the contrast material had completely cleared from the convexity (figure 2C, D), Sylvian fissure (figure 2E) and basal cisterns (figure 2F), however, there was residual brain oedema over the convexity. A brain CT scan is very sensitive in detecting subarachnoid haemorrhage; in this case, no subarachnoid haemorrhage was seen.

Contrast encephalopathy associated with non-ionic contrast material has been reported in a few cases after cerebral[2–5] or cardiac[6] angiography. However, it has not been reported in the setting of endothelial dysfunction and OSA.

So far, contrast encephalopathy has been explained as transient disruption of the blood–brain barrier triggered by the hypertonic contrast medium, which draws water out of the endothelial cells of cerebral vessels, causing enlargement of tight junctions and a resultant leaky blood–brain barrier.[7] Other possible mechanisms include microvascular sludging and arterial spasm.[2]

In OSA, it is postulated that hypoxaemia generates reactive oxygen species and proinflammatory molecules.[8] This proinflammatory state leads to an alteration of endothelial-derived factors responsible for vascular tone, leading to hypertension, impaired endothelial repair capacity and other vascular pathologies. The inherent risks of contrast media combined with endothelial dysfunction may predispose patients with OSA to contrast encephalopathy. CPAP therapy improves endothelial dysfunction and decreases oxidative stress and inflammatory cytokines in patients with OSA.[1]

Patients with severe OSA may benefit from assessment of endothelial function in order to estimate the risk of endovascular procedures. Brachial artery flow-mediated dilation is a validated test that measures endothelial dysfunction. Flow-mediated dilation is characteristically severely blunted in patients with OSA.[9]

We have presented the first case of contrast encephalopathy associated with severe OSA following coil embolisation of an unruptured intracranial aneurysm. OSA is increasingly recognised as a source of significant perioperative morbidity and mortality.[10] Patients with OSA requiring endovascular treatment may be at an increased risk for postoperative contrast encephalopathy due to the endothelial dysfunction associated with OSA. While our patient did not obtain a brachial artery flow-mediated dilation study, we would encourage this test to be carried out preoperatively to help risk-stratify patients. Interventions may be postponed until the disease process and endothelial dysfunction are optimally controlled with CPAP prior to administration of contrast media to avoid the development of postoperative contrast encephalopathy.

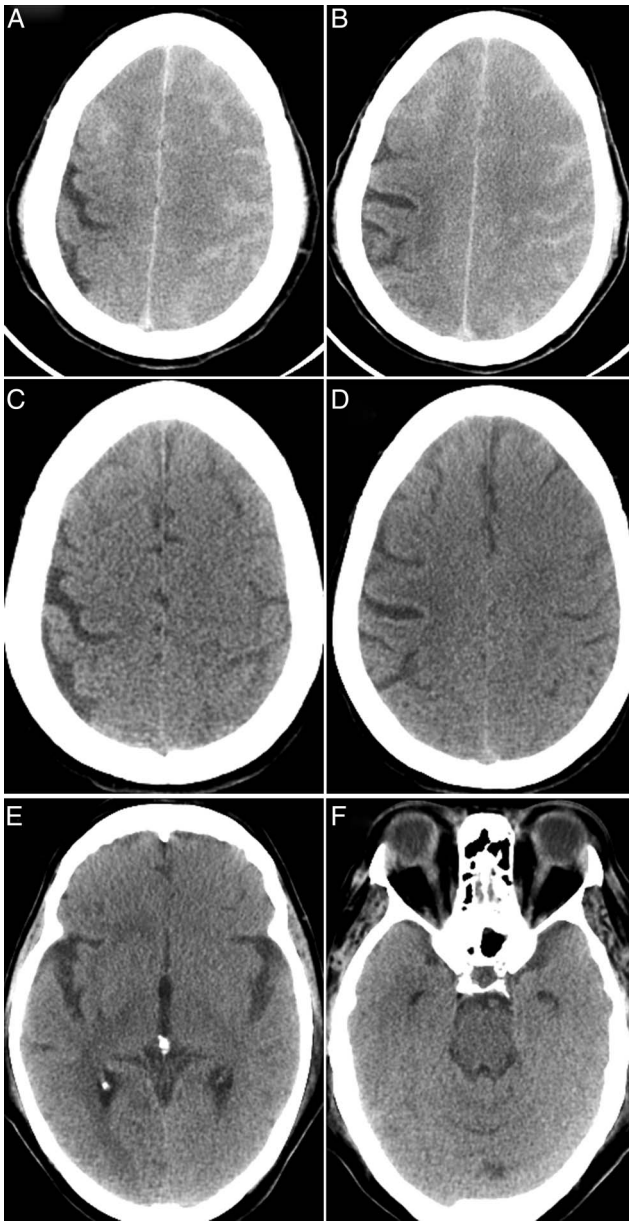

Figure 2 (A and B) A brain CT scan 3 h after coiling showing contrast enhancement in the territory supplied by the left internal carotid artery. There is also contrast enhancement in the distal right anterior cerebral artery (ACA) territory since the right A1 is absent. Therefore, the distal right ACA territory is also supplied by the left internal carotid artery. There is no contrast enhancement in the right middle cerebral artery territory, which is supplied by the contralateral internal carotid artery. (C and D) A brain CT scan 18 h after coiling showing complete resolution of contrast enhancement, but residual brain oedema. No subarachnoid haemorrhage can be seen. (E) No subarachnoid haemorrhage is present in the Sylvian fissures or (F) basal cisterns 18 h after coiling.

While the relationship of contrast encephalopathy and OSA is plausible in our patient, we have no direct proof of causality. Further research is indicated to study whether patients with severe OSA have an increased risk of contrast encephalopathy after neurointerventional procedures.

Learning points

- Severe obstructive sleep apnoea is a likely risk factor for the development of contrast encephalopathy after endovascular procedures, due to the underlying vascular endothelial dysfunction, which merits further research.
- Obstructive sleep apnoea patients may benefit from screening for endothelial dysfunction with the brachial forearm occlusion test for risk stratification.
- Optimal medical management of obstructive sleep apnoea with continuous positive airway pressure prior to endovascular procedures may decrease the degree of endothelial dysfunction and risk of contrast encephalopathy.

Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

1 Oyama J, Yamamoto H, Maeda T, *et al*. Continuous positive airway pressure therapy improves vascular dysfunction and decreases oxidative stress in patients with the metabolic syndrome and obstructive sleep apnea syndrome. *Clin Cardiol* 2012;35:231–6.
2 Horwitz NH, Wener L. Temporary cortical blindness following angiography. *J Neurosurg* 1974;40:583–6.
3 Lee KK, Kang DH, Kim YS, *et al*. Serious blood-brain barrier disruption after coil embolization of unruptured intracranial aneurysm: report of two cases and role of immediate postembolization CT scan. *J Korean Neurosurg Soc* 2011;50:45–7.
4 Shinoda J, Ajimi Y, Yamada M, *et al*. Cortical blindness during coil embolization of an unruptured intracranial aneurysm—case report. *Neurol Med Chir (Tokyo)* 2004;44:416–19.
5 Uchiyama Y, Abe T, Hirohata M, *et al*. Blood brain-barrier disruption of nonionic iodinated contrast medium following coil embolization of a ruptured intracerebral aneurysm. *AJNR Am J Neuroradiol* 2004;25:1783–6.
6 Shrivastava S, Mohan JC, Chopra P. Fatal cerebral edema following angiocardiography: a case report. *Int J Cardiol* 1985;8:490–1.
7 Rapoport SI, Thompson HK, Bidinger JM. Equi-osmolal opening of the blood-brain barrier in the rabbit by different contrast media. *Acta Radiol Diagn (Stockh)* 1974;15:21–32.
8 Stiefel P, Sánchez-Armengol MA, Villar J, *et al*. Obstructive sleep apnea syndrome, vascular pathology, endothelial function and endothelial cells and circulating microparticles. *Arch Med Res* 2013;44:409–14.
9 Panoutsopoulos A, Kallianos A, Kostopoulos K, *et al*. Effect of CPAP treatment on endothelial function and plasma CRP levels in patients with sleep apnea. *Med Sci Monit* 2012;18:CR747–51.
10 Memtsoudis SG, Besculides MC, Mazumdar M. A rude awakening—the perioperative sleep apnea epidemic. *N Engl J Med* 2013;368:2352–3.