Contrast-Induced Encephalopathy – An Unusual Complication Following Endovascular Interventions in the Anterior Circulation

Sir,

Contrast-induced encephalopathy (CIE) is an extremely rare complication which may occur following the use of iodinated contrast media (ICM). Most of the published literature on CIE is based on coronary angiograms and coronary artery interventions.[1] Only 52 cases of CIE have been reported in the literature between 1970 and 2017.[2,3] We report our experience with two cases who were diagnosed with contrast-induced encephalopathy following endovascular intervention in the anterior circulation. These case descriptions highlight the varied clinical and radiological findings in CIE and underscore the favourable outcome in such cases.

Case 1

A 54-year-old lady presented to the emergency department with acute-onset severe headache, vomiting and photophobia. She was known to be hypertensive and non-diabetic. Her renal function tests and liver function tests were normal. Computed tomography (CT) of the brain showed acute subarachnoid haemorrhage (SAH) in the interhemispheric fissure and mild hydrocephalus [Figure 1. Cerebral digital subtraction angiogram (DSA) confirmed the diagnosis of a saccular aneurysm of the aneurysm at the anterior communicating artery [Figure 1]. Endovascular coil embolization of the aneurysm was performed with a near-complete occlusion of the aneurysm [Figure 1]. Approximately, 110 ml of Iohexol (iodine concentration of 300 mg/ml) contrast media was used. Post-extubation, the patient did not have any neurological deficits. The patient was kept fasting for 6 hours post-procedure and intravenous fluids were administered as per protocol. Two hours after the extubation she deteriorated and her GCS score dropped to E3V2M5 with a paucity of right upper and lower limb movements. CT showed left frontoparietal and right frontal lobe sulcal effacement with contrast staining of the sulci. There was no evidence of any fresh haemorrhage or infarct [Figure 2]. Contrast staining of the sulci was evident along the territorial distribution of the left ICA. The contrast staining was evident in right ACA territory also which can be explained by the fact of right A1 was hypoplastic. The

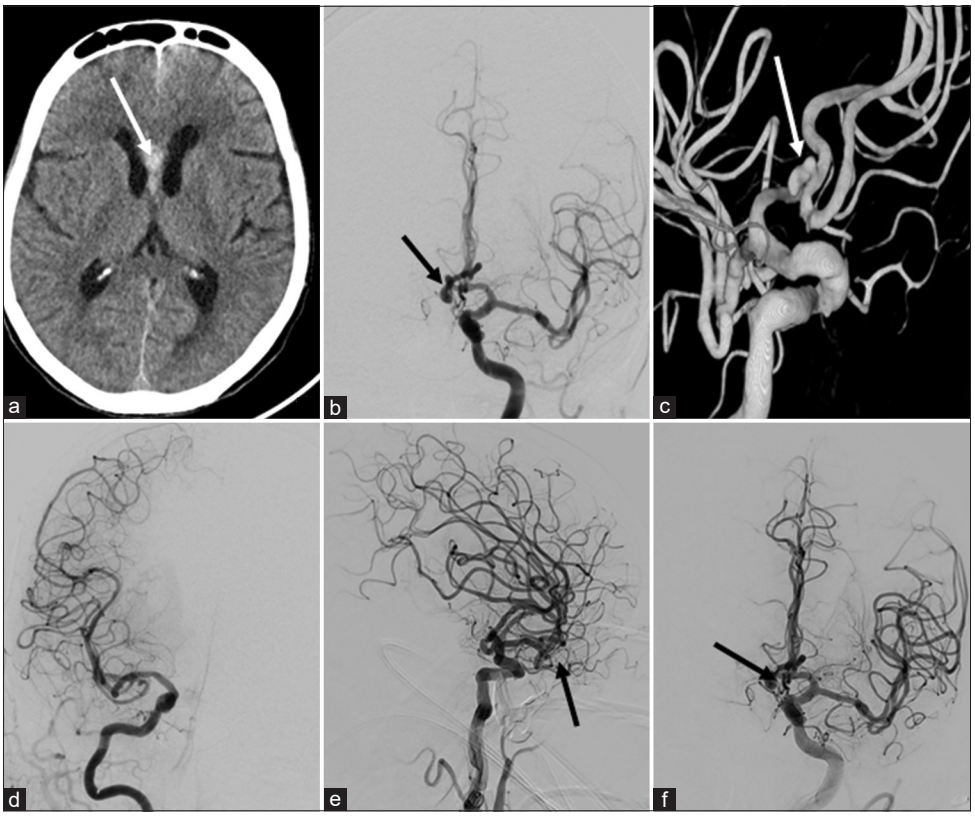

Figure 1: Case 1. Non-enhanced CT scan shows evidence of acute subarachnoid hemorrhage (White arrows, a). Digital subtraction angiogram AP projection (b) and 3D reconstruction of rotational angiogram (c) shows a saccular aneurysm arising from the Anterior communicating artery (Black arrow in b, White arrow in c). The right A1 segment was found to be hypoplastic (d) and both A2 segments were noted to be filling from the left side. Post-coiling check angiograms in AP projection (e) and lateral projection (f) show near-total occlusion of the aneurysm (Black arrows in e and f)

serum electrolytes were normal. Based on typical imaging findings on CT, a provisional diagnosis of CIE was made and the patient was started on intravenous fluids, intravenous dexamethasone q6h and intravenous mannitol 100 ml q6h.

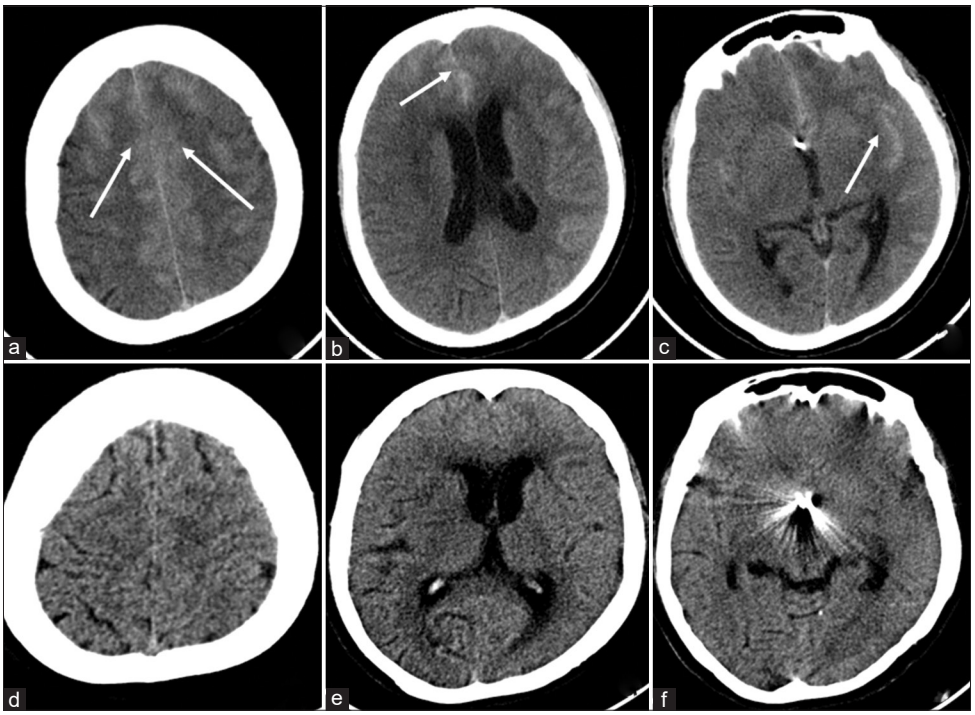

Figure 2: Case 1. Non-enhanced CT obtained immediately after the endovascular coil embolization showed contrast staining along the sulci and gyri of bilateral frontal lobes (White arrows, a), anterior interhemispheric region (white arrow, b) and also along the left sylvian fissure (White arrow, c) corresponding to the vascular territory of the left ACA, right ACA and left MCA. Subsequent follow-up CT after 48 hours shows interval resolution of the gyral and sulcal contrast staining (d, e, f) with resolved gyral edema. Coil mass noted *in situ* (f)

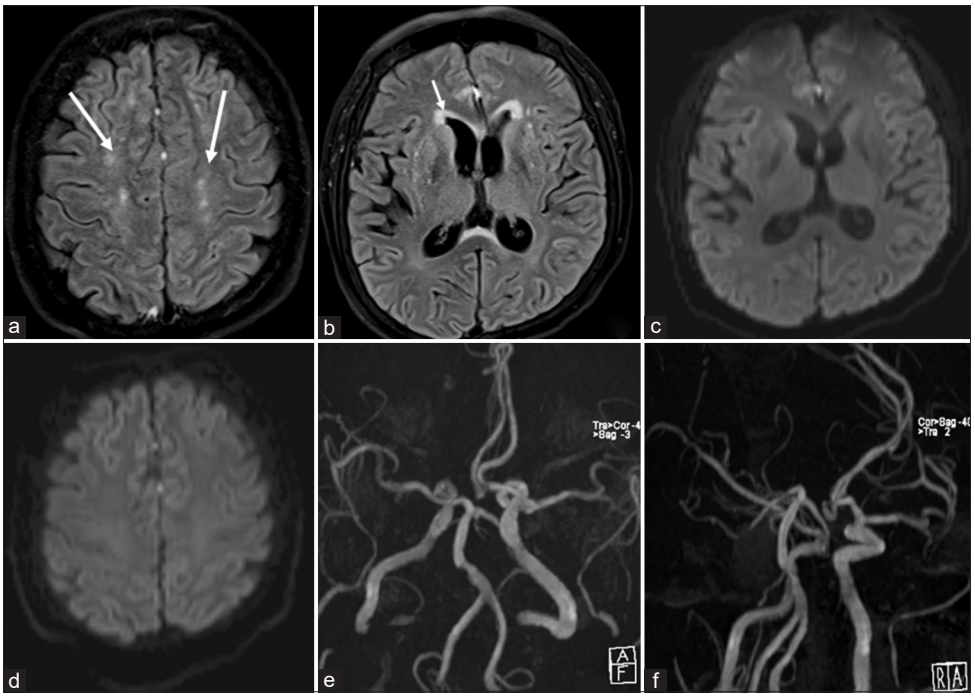

Figure 3: Case 1. MRI done 48 hours after the onset of symptoms. Axial FLAIR images showed patchy hyperintense foci in bilateral frontal white matter (White arrows in a) and the periventricular white matter with presence of ependymitis granularis (white arrow in b). No evidence of new infarcts or hemorrhage seen on Diffusion-weighted images (c, d). Time-of-flight MR angiogram (e, f) showed no evidence of residual aneurysmal filling and no vascular occlusion

Her neurological status improved on day 2 of the treatment. A repeat CT showed resolution of contrast staining and no sulcal effacement was found [Figure 2]. MRI revealed only microvascular ischemic changes with no fresh haemorrhage or acute infarction [Figure 3].

Case 2

A 70-year-old lady, non-diabetic and non-hypertensive presented to the emergency department with right hemiparesis of 5 hours duration. The National Institute of Health Stroke Scale (NIHSS) at the time of presentation was 11. Her renal function tests were normal. The Alberta stroke programme early CT score (ASPECTS) was 9 [Figure 4]. It was decided to proceed with mechanical thrombectomy. Puncture to recanalization time was 15 minutes. The total amount of contrast media used was 25 ml of Iohexol (iodine concentration of 300 mg/ml). The immediate post-procedure MRI showed abnormal sulcal FLAIR hyperintensity in the left frontoparietal region [Figure 5] and no fresh infarct was evident [Figure 5]. Since the patient was under the effect of sedation, neurological status was not accessed in the immediate post-procedure period. The patient was kept fasting for 6 hours post-procedure and IV fluids were administered as per protocol. After weaning of the effect of sedation, her NIHSS was 21 and power in right upper and lower limb was found to be 0/5 (as per the Medical research council grading of muscle power) despite TICI 3 Recanalization. Based on the clinical and imaging findings, a possibility of CIE was raised and she was managed conservatively with intravenous fluid therapy. After 26 hours of treatment, her neurological status improved and NIHSS dropped to 1 and power in right upper and lower limb improved to grade 5/5. MRI imaging at the time discharge showed disappearance of sulcal FLAIR hyperintensity, no haemorrhage and MR angiogram showed patency of left MCA [Figure 5].

Contrast-induced encephalopathy an extremely rare complication of ICM use and has been mostly reported to be associated with cardiac catheterization but the recent literature shows its association with cerebral angiography as well.[4] The incidence of CIE reported in the literature ranges from 0.3 to 2% for vertebral angiography.[5,6] Various risk factors have been postulated to be associated with CIE which include hypertension, diabetes mellitus, chronic renal disease, history of adverse reactions to ICM and a history of stroke or transient ischemic attacks.[3] CIE has been reported to occur with all types of iodinated contrast media irrespective of osmolality.[3,7] Acute cerebral ischemia is an independent risk factor for CIE.[8] A higher dose of intra-arterial contrast media is also found to be more frequently associated with CIE,[9] however, contrast dosage as low as 12 ml have been associated with the development of CIE.[5] The mechanism underlying CIE is unclear, however, it is believed to be caused by chemo-toxicity of the ICM and subsequent osmotic breakdown

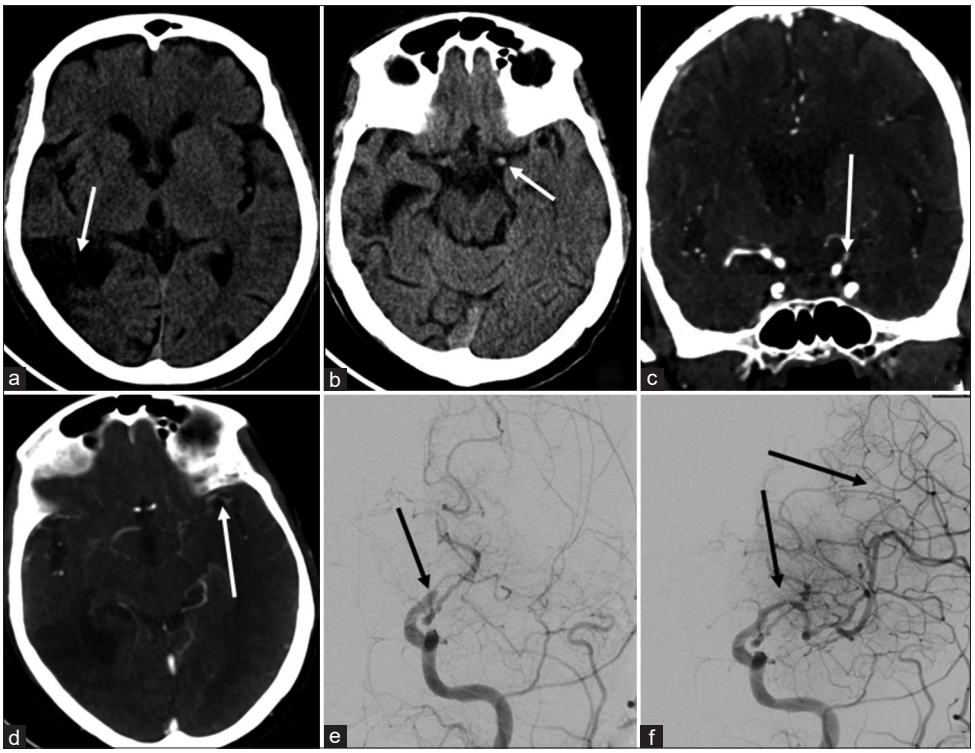

Figure 4: Case 2. Non-enhanced CT shows a chronic infarct involving the right inferior parietal and right posterior temporal lobe (white arrow, a). Hyperdense focus is noted in the left Sylvian fissure (White arrow, b) corresponding to the "hyperdense MCA sign". Terminal ICA occlusion is noted on the left side on CT angiogram (White arrow, c) with good collaterals (White arrow, d). On pre-thrombectomy digital subtraction angiogram (e), occlusion of the left terminal ICA is evident (Black arrow, e). Post-thrombectomy angiogram (f) shows complete recanalization of the left MCA territory (Black arrows, f)

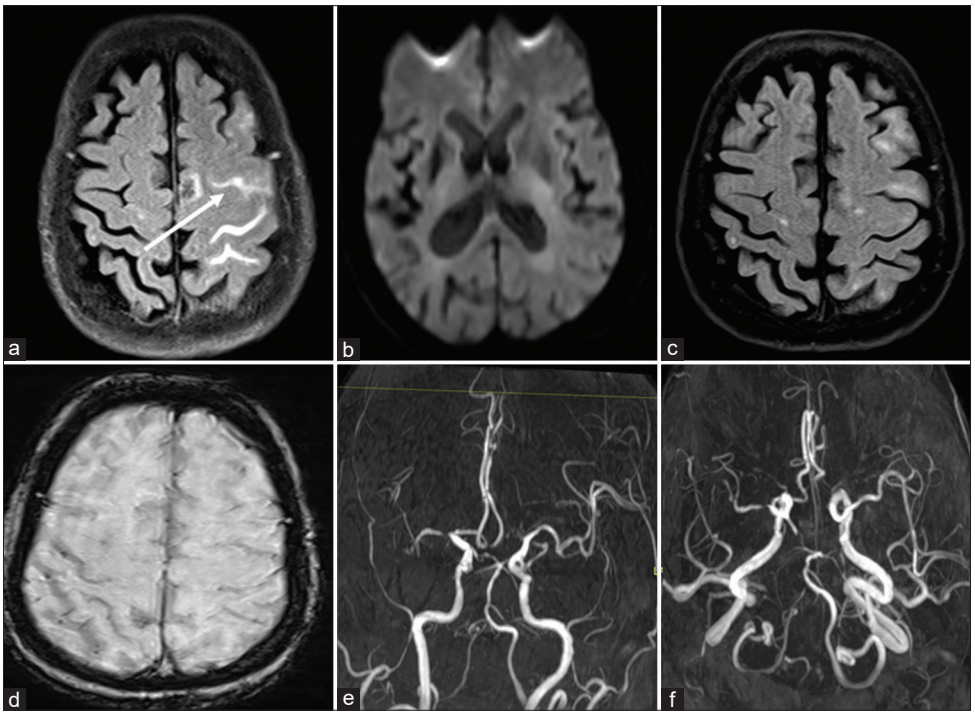

Figure 5: Case 2. Immediate post-thrombectomy MRI shows hyperintense signal along the left superior frontal sulci on axial FLAIR images (White arrow, a). No new infarcts or hemorrgae is seen on Diffusion weighted image (b). Subsequent MRI done 26 hours after the endovascular intervention revealed resolution of the sulcal FLAIR hyperintensities along the left frontal sulci (c). No evidence of hemorrhage was noted on axial Susceptibility weighted image (d). Time-of-flight MR angiogram (e, f) shows patency of the left ICA and left MCA

of the blood-brain barrier. Since the occipital lobe has a higher BBB permeability and a poorly developed autoregulation, it is frequently involved in CIE.[10,11]

Clinical features of CIE include transient cortical blindness, hemiparesis, confusion, focal neurological deficits and coma.[2,7,11] Subarachnoid hemorrhage could be considered as the differential diagnosis of CIE, however, resolution of the sulcal hyperdensity on CT within few hours and absence of xanthochromia, favors the diagnosis of CIE.[3] Alternatively, a dual-energy CT may be useful in differentiating contrast staining from SAH.[12] Rarely neuroimaging can be normal in the face of a florid clinical deterioration.[13] Posterior reversible encephalopathy (PRES) may be considered as a differential diagnosis for CIE in the appropriate clinical scenario. However, the absence of typical neuroimaging features of PRES helped us in ruling out this crucial differential diagnosis in both of our cases.[14]

Management of CIE is conservative and consists of intravenous fluid therapy, antiedema measures such as mannitol infusion, dexamethasone and additionally anticonvulsants may be required if the patient develops seizures.[2,3,9] There is no clear consensus about the treatment strategy for the CIE on account of its rarity.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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