JOURNAL OF NEUROLOGY AND PSYCHOLOGY RESEARCH

CASE REPORT Open Access

Contrast-Induced Encephalopathy After an Aneurysm Treatment by Endovascular Approach

Nitish Kumar (M.D)[1*] Camilo R Gomez (M.D)[1], Adnan I Qureshi (M.D)[1], Farhan Siddiq (M.D)[2]

[1]Department of Neurology, University of Missouri, Colombia

[2]Department of Neurosurgery, University of Missouri, Colombia

Received date: August 06, 2022, **Accepted date:** August 11, 2022, **Published date:** September 01, 2022

***Corresponding Author:** Nitish Kumar (MD), Department of Neurology, University of Missouri, Colombia.

Summary

Contrast-induced encephalopathy (CIE) is an uncommon complication that can present with transient neurologic deficits and is caused by the neurotoxicity of intravascular contrast media. We report a case of an 87-year-old woman who was admitted for an elective aneurysm treatment by endovascular means. The procedure went well without any apparent complications. The total amount of Visipaque contrast media used was 120 ml.

Immediately post-procedure, she was found to have consciousness disturbance, global aphasia, and right-sided weakness while recovering in the neuro-critical care unit. Contrast-induced encephalopathy was suspected. After ruling out differentials like an ischemic stroke, subarachnoid hemorrhage, etc., a diagnosis of contrast-induced encephalopathy was made. The patient recovered completely within 48 hours without any neurological deficits.

Keywords: Contrast Induced, Encepahlopathy, Cerebral Edema, Percutaneous Intervention.

Introduction

Contrast-induced encephalopathy (CIE) after percutaneous cerebral/carotid intervention is a very rare complication caused by the administration of intravascular contrast media. Different kind of contrast media including ionic, non-ionic, hyperosmolar and iso-osmolar have been reported in the literature to induce Contrast-induced encephalopathy [1-5]. *VISIPAQUE*™ (iodixanol) is a dimeric, iso-osmolar, non-ionic, water-soluble, radiographic contrast medium with a molecular weight of 1550.20 (iodine content 49.1%) with an osmolality of 320 mOsm/kg H2O]. One of the earliest cases was reported in 1970 by Fischer [1]. Thereafter, there have been other reports regarding Contrast-induced encephalopathy as well [2-5]. Contrast-induced encephalopathy is characterized by encephalopathy, transient cortical blindness, seizure, or focal neurological deficits [1-6]. This might be attributed to the neurotoxicity of contrast media, which can cause osmotic disruption of the blood-brain barrier. We are reporting a case of an 87-year-old woman who developed Contrast-induced encephalopathy after undergoing an endovascular treatment of an anterior communication artery aneurysm. The quality of life, forcing the patient to make numerous pilgrimages from one specialist to another, often, almost always without real benefit.

Case Presentation

87-year-old female with past medical history of hypertension, hypothyroidism who presented for elective embolization of an anterior communicating artery aneurysm. She initially presented to our hospital with a headache for months and Magnetic resonance imaging was obtained which revealed anterior communicating aneurysm (Figure 1). Computed tomography angiography showed bilateral internal carotid artery proximal segment stenosis with right greater than the left. A 6 mm anterior communicating aneurysm was also found (Figure 1). Digital subtraction angiography confirmed the above findings (Figure 2).

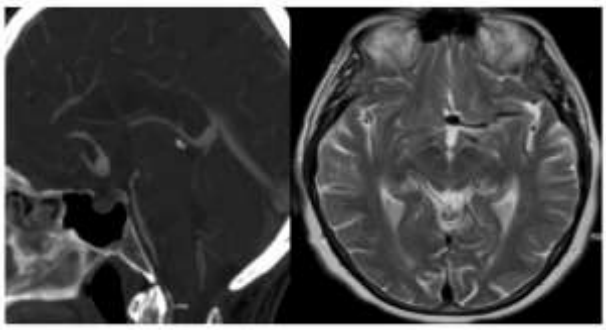


Figure-1. Showing Anterior communicating artery aneurysm on CTA head and MRI brain (T2).

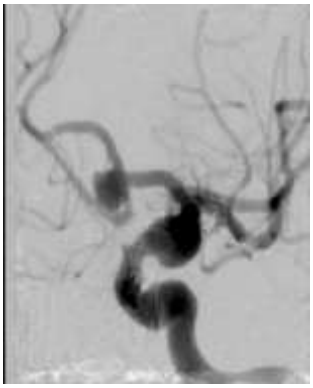


Figure 2. Digital subtraction angiography showing anterior communicating artery aneurysm before treatment.

The patient was discharged at that time and was brought back for an elective aneurysm treatment. She underwent embolization via WEB (Woven Endo-Bridge Device, Microvention/Terumo, Aliso Viejo, California, USA) device. The procedure was done under general anesthesia. Patient was appropriately anticoagulated with heparin throughout the procedure and serials activated clotting time was obtained to ensure that. She received a total of 120 ml of Visipaque (an isosmolar, non-ionic, water-soluble, radiographic contrast medium) with an osmolality of 320 ml/KgH2O.

She was admitted to neurocritical care unit post procedure. Once the patient comes out of the anesthesia, she was noted to have difficulty with speaking and was given 2 mg of Lorazepam due to concerns for seizures and placed on continuous electroencephalogram monitoring and Levetiracetam 750 twice daily. The patient was subsequently noted to have right-sided hemiparesis. An immediate head computed tomography was obtained (Figure 3). Computed tomography showed sulcal effacement in the left cerebral hemisphere with contrast staining in the left frontotemporal areas. Few hours later, she was noted by nursing staff to have reduced pupillary reaction in the right eye and worsening right arm movement. Computed tomography head and perfusion (Figure 4a and 4b) was obtained.

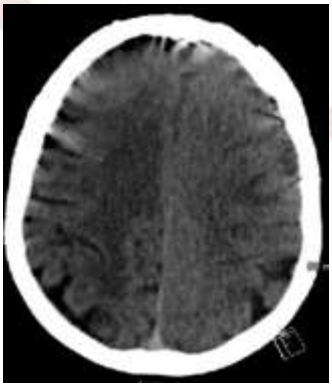
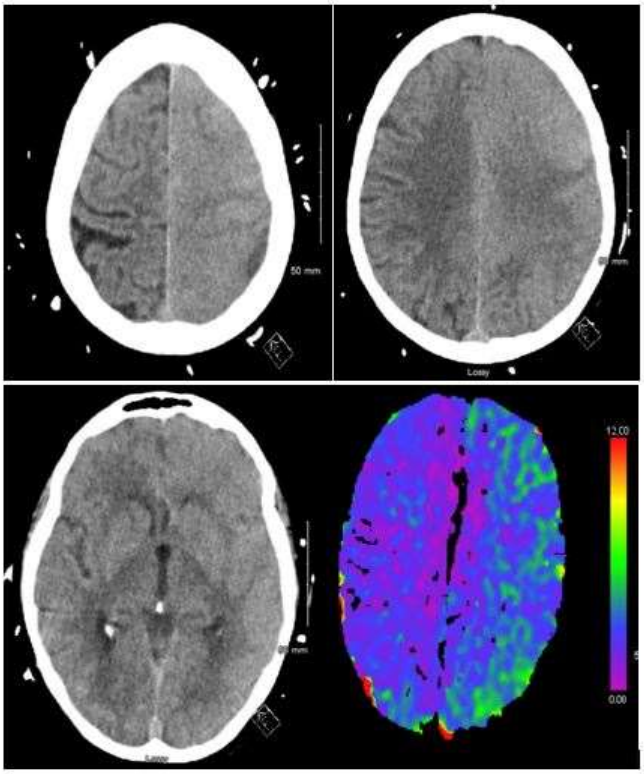


Figure 3. Computed Tomography of the head in immediately after the aneurysm treatment showing signs of left



hemispheric edema. Also note contrast staining as well.

Figure 4a. Computed tomography approximately 12 hours post procedure shows marked ipsilateral cerebral edema. Note sulcal effacement and resolution of the contrast staining noted on previous CT head.

Figure 4b. Computed tomography perfusion head is showing mildly increased Tmax with associated trace decrease in blood flow within the left cerebral hemisphere.

Intravenous hydration with normal saline was continued. Follow-up magnetic resonance imaging (MRI) brain 40 hours after the onset of symptoms showed hyperintensity in the left frontal, left parietal areas on T2, fluid-attenuated inversion recovery (FLAIR) and diffusion-weighted imaging (DWI). There was no change on the apparent diffusion coefficient (ADC) maps (Figure 5), which differentiated contrast induced encephalopathy from cerebral ischemia [7] Patient's electroencephalography revealed diffuse cortical dysfunction. Ultimately, all neurological deficits recovered gradually within 48 hours. Patient was discharged home on day 3 of her admission. She was followed up in outpatient neurology clinic and she continued to have stable neurological examination.

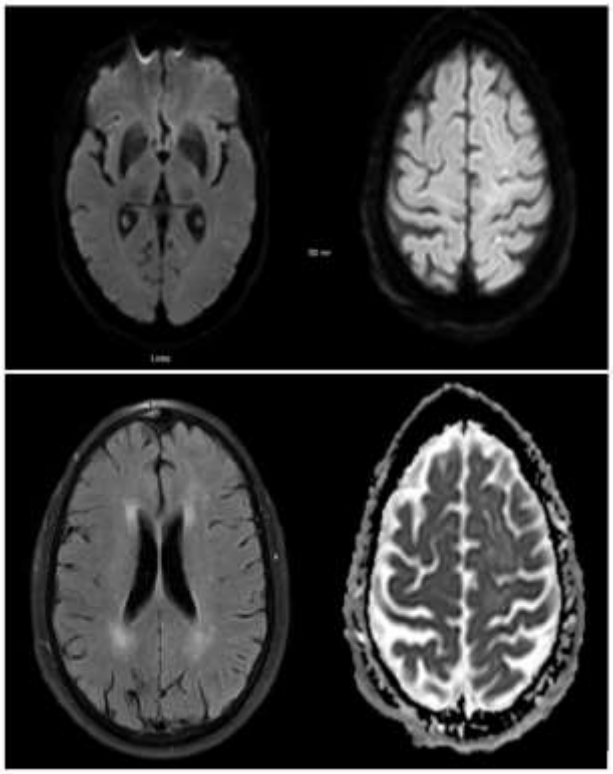


Figure 5. Magnetic resonance imaging showing punctate hyperintensities (on diffusion weighted images) in the left frontal and parietal cortex with no obvious corresponding changes on apparent diffusion coefficient. Also note periventricular changes on T2 FLAIR. Remarkable improvement in the left hemispheric sulcal effacement noted 36 hours ago on CT Head. See Figure 4a and 4b.

Discussion

The first case of Contrast-induced encephalopathy that manifested as transient cortical blindness after coronary angiography was reported in 1970 [1]. The incidence of Contrast-induced encephalopathy after the administration of contrast media has varied, ranging between 0.3%-1.0%, but reaching 4% in some hyperosmolar iodinated contrast media [8]. A retrospective study [6] was done in 2020 that showed the incidence of Contrast-induced encephalopathy was approximately 1.7% in 421 consecutive patients who underwent mechanical thrombectomy.

Patient's neurological symptoms and signs usually develop within hours of exposure to contrast media. However, spontaneous resolution in neurological status usually occurs over a period of days. The diverse manifestations of Contrast-induced

encephalopathy are several, and include cortical blindness, hemiparesis, aphasia, loss of coordination, confusion, seizure, and coma [1-4].

Although the precise mechanism remains unclear, a possible explanation is that the chemical and physical properties of contrast media which could cause osmotic disruption of the blood-brain barrier [7] and the hyper-osmolality and direct neurotoxicity of extravasated contrast media could further result in cerebral edema. This hypothesis is supported by animal studies [4, 9]. The hyper-osmolality of contrast media and the permeability of blood brain barrier can both cause contrast extravasation to cerebral space, since isosmolar contrast medium could also induce Contrast-induced encephalopathy [2]. The occipital cortex is one of the regions with higher permeability of the blood brain barrier [10] Hence, this can explain why it is the most vulnerable region.

The predisposing factors leading to Contrast-induced encephalopathy are chronic hypertension [8] transient ischemia attack (TIA) [10] impaired cerebral autoregulation [10] impaired renal function, large contrast volumes [10], selective vertebral-basilar arteriography [9] and male gender (?). About half of all patients with transient encephalopathy following intra-arterial contrast injection have a history of chronic hypertension [8].

The two independent risk factors for Contrast-induced encephalopathy included renal dysfunction (defined as an estimated glomerular filtration rate <45 mL/min per 1.73 m^2; odds ratio, 5.77 [95% CI, 1.37–24.3]; P=0.02) and history of stroke (odds ratio, 4.96 [95% CI, 1.15–21.3]; P=0.03 [6]. Renal dysfunction impairs the clearance of contrast medium, which may exacerbate the accumulated osmolality and neurotoxicity of contrast (frantz).

The relationship between Contrast-induced encephalopathy and contrast medium volume is somewhat complicated. Previous studies showed that the volume of contrast inducing Contrast-induced encephalopathy ranged from 80 to 400 ml. But one report showed that a local injection of 25 mL contrast medium to the carotid artery can cause Contrast-induced encephalopathy [8]. Selective vertebral- basilar arteriography also had a high risk of Contrast-induced encephalopathy because of the involvement of the arterial supply to the medulla oblongata, brain stem, cerebellum, and basal parts of the temporal and occipital lobes [9].

The diagnosis of Contrast-induced encephalopathy can be challenging, especially in patients with acute ischemic stroke [11]. Numerous post-thrombectomy complications should be considered, including reperfusion injury, hemorrhagic transformation of infarcted area, artery perforation or dissection, and recurrent ischemic stroke.

Consequently, a brain computed tomography without contrast plays the crucial role in diagnosis showing diffuse cortical hyper attenuation similar to Subarachnoid haemorrhage [4]. But normal brain computed tomography did occur in some cases [3] MRI results have previously shown hyper intensity in the cortex on T2, FLAIR, and DWI, which has been described in the literature [7]. A reliable imaging modality that can differentiate Contrast-induced encephalopathy from cerebral ischemia is ADC, which shows no abnormal intensity in patients with Contrast-induced encephalopathy [7]. The ADC provides a quantitative measure of water diffusion. In acute ischemic stroke with cytotoxic edema, decreased water diffusion in infarcted tissue causes a decreased ADC. A significant reduction in ADC lasting for at least 96 hours from stroke onset [12] which revealed delayed MRI examination in our case, may not affect the final result. The susceptibility-weighted imaging of brain MRI can be used to differentiate contrast staining from haemorrhage. Other methods, such as dual-energy computed tomography, measurement of the Hounsfield unit [13], and cerebrospinal fluid analysis, have been proposed to differentiate contrast staining from haemorrhage [11].

The treatment of Contrast-induced encephalopathy is supportive in nature, such as the use of adequate hydration with intravenous crystal- loids and anticonvulsants for seizures. In a few cases, patients had been treated with intravenous steroids and anti-edema agents without adverse consequences [2]. The neurologic deficits lasted 15 minutes to 5 days in a majority of cases. (Frantz) Interestingly, re-injection of contrast medium in such patients might not induce Contrast-induced encephalopathy again [14].

References

1. Fischer-Williams M, Gottschalk PG, Browell JN. Transient cortical blindness. An unusual complication of coronary angiography. *Neurology*. (1970); 20 (4):353-355.

2. Chisci E, Setacci F, de Donato G, Setacci C. A case of contrast-induced encephalopathy using iodixanol. J Endovasc Ther. (2011); 18 (4): 540-544.

3. Sawaya RA, Hammoud R, Arnaout S, Alam S. Contrast-induced encephalopathy following coronary angioplasty with iohexol. *South Med J*. (2007);100 (10): 1054-1055.

4. Sharp S, Stone J, Beach R. Contrast agent neurotoxicity presenting as subarachnoid hemorrhage. *Neurology*. (1999);

52 (7):1503-1505.

5. Spina R, Simon N, Markus R, Muller DW, Kathir K. Contrast-induced encephalopathy following cardiac catheterization. *Catheter Cardiovasc Interv*. (2017); 90 (2): 257-268.

6. Chu YT, Lee KP, Chen CH, Sung PS, Lin YH, Lee CW, *et al.* Contrast-Induced Encephalopathy After Endovascular Thrombectomy for Acute Ischemic Stroke. Stroke. (2020); 51 (12): 3756-3759.

7. Yu J, Dangas G. Commentary: New insights into the risk factors of contrast-induced encephalopathy. *J Endovasc Ther.* (2011); 18 (4): 545-546.

8. Potsi S, Chourmouzi D, Moumtzouoglou A, Nikiforaki A, Gkouvas K, Drevelegas A. Transient contrast encephalopathy after carotid angiography mimicking diffuse subarachnoid haemorrhage. *Neurol Sci*. (2012); 33 (2) :445-448.

9. Haley EC, Jr. Encephalopathy following arteriography: a possible toxic effect of contrast agents. *Ann Neurol*. (1984); 15 (1): 100-102.

10. Muruve DA, Steinman TI. Contrast-induced encephalopathy and seizures in a patient with chronic renal insufficiency. *Clin Nephrol*. (1996); 45 (6) :406-409.

11. Senthilkumaran S, Balamurugan N, Jena NN, Thirumalaikolundusubramanian P. Contrast-induced encephalopathy and diagnostic modalities - Can it make a difference? *Am J Emerg Med*. (2018); 36 (12): 2328.

12. Schlaug G, Siewert B, Benfield A, Edelman RR, Warach S. Time course of the apparent diffusion coefficient (ADC) abnormality in human stroke. *Neurology*. (1997); 49 (1):113-119.

13. Harada Y, Kairamkonda SR, Ilyas U, Pothineni NVK, Samant RS, Shah VA, *et al.* Pearls and Oy-sters: Contrast-induced encephalopathy following coronary angiography: A rare stroke mimic. Neurology. (2020); 94 (23): e2491-e2494.

14. Rama BN, Pagano TV, DelCore M, Knobel KR, Lee J. Cortical blindness after cardiac catheterization: effect of rechallenge with dye. *Cathet Cardiovasc Diagn*. (1993); 28 (2): 149-51.