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Unusual Presentation of Transient Encephalopathy from Angiographic Contrast, After Neurointerventional Procedure

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1. Abstract

Neurotoxicity from contrast media used in angiography is a rare complication from these procedures caused by disruption of the blood–brain-barrier (BBB), most of the time presenting as cortical blindness. The infrequency with which it is encountered makes it a diagnostic challenge. We present the case of a 64-year-old male who developed right hemianopsia, prosopagnosia and hallucinations after embolization of a cervical spinal dural arteriovenous fistula. The neurological deficits which appeared after the procedure, regressed completely after 48 hours. This rare entity should be kept in mind but diagnosed only when all other causes have been ruled out.

2. Keywords: Magnetic Resonance; Spinal cord; Complication; Fistula; Contrast media

3. Introduction

Cerebral angiography is an invasive procedure associated with a very small, but definite risk of neurological morbidity. The incidence of cerebrovascular complications related to cerebral angiography is lower than 1% [1,2].

Numerous studies have demonstrated the capacity of iodinated contrast agents to penetrate the blood brain barrier by opening the intercellular unions and increasing endothelial pinocytosis, affecting the neuronal membrane of the cerebral cortex. The neurotoxicity caused by contrast medium depends on its chemical and physical properties, i.e., osmolarity, lyposolubility and viscosity [3,4]. This event has been described related to transient cortical blindness (TCB), which is a rare but well-known complication of cerebral angiography and is reported to have a higher incidence for vertebral angiography [5]. We would like to report an atypical presentation of neurotoxicity after cerebral angiography who presented with transient hemianopsia, prosopagnosia and hallucinations.

4. Case Presentation

A 64-year-old male with a past medical history significant for smoking presented with tetraplegia, urinary retention, difficulty swallowing and breathing after a transurethral resection of the prostate. The symptoms started eight hours after the surgery. Laboratory testing revealed no abnormalities. Unenhanced MRI of the spine showed abnormal hyperintense T2 signal and hypointense T1 signal extending from the C3 vertebral body level down to the conus medullaris with smooth mild cord expansion. Combined, pre- and post-contrast MRI of the spine revealed enhancement of all the serpiginous

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intradural blood vessels along the posterior aspects of the spinal cord. The findings were compatible with Type I spinal dural arteriovenous fistula (SDAVF).

A spine angiogram was performed to further define specific vessel involvement and to formulate a specific therapeutic treatment. Angiography indicated that the SDAVF had three feeding arteries branching from the left vertebral artery (V2 and V3) (Figure 1).

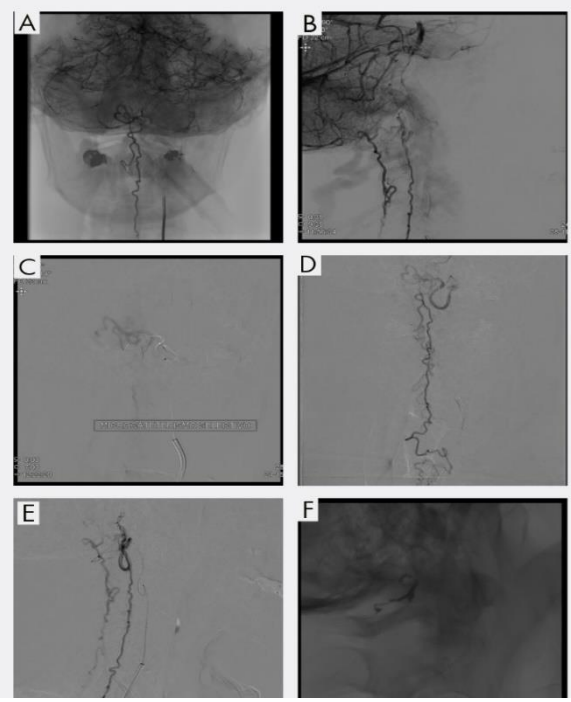

Figure 1: (A) Front and **(B)** lateral projection of macrocatheter injection from left vertebral artery showing one of the three dural arteriovenous fistula and the dilated medullary veins. **(C-D)** Front **(E)** and lateral projections of selective injection from the microcatheter showing the fistula and dilated medullary veins. **(F)** Lateral projection showing NBCA at one of the fistulas.

Embolization with NBCA was performed where by in two feeding arteries and ethanol on the other one with complete occlusion of the fistula. 600 cc of Ultravist -300 were used during the whole procedure.

The patient awoke from the general anesthesia without any new neurological deficit. Approximately 1 hour after the angiography, the patient developed right hemianopsia, prosopagnosia and hallucinations. Stroke code was activated. Urgent non-contrast brain CT and Angio-CT was performed within 2 hours after angiography and showed neither signs of acute infarction nor large vessel occlusion. A big right posterior communicating artery (Figure 2A) and a tiny left posterior communicating artery were evidenced.

After 24 hours, non-contrast MRI using a 1.5-T MR scanner showed gyriform increased signal in the left parieto-occipital cerebral hemisphere on FLAIR and gyriform enhancement on T1 with gadolinium. There were no signs of ischemia on DWI sequence (Figure 2B-F).

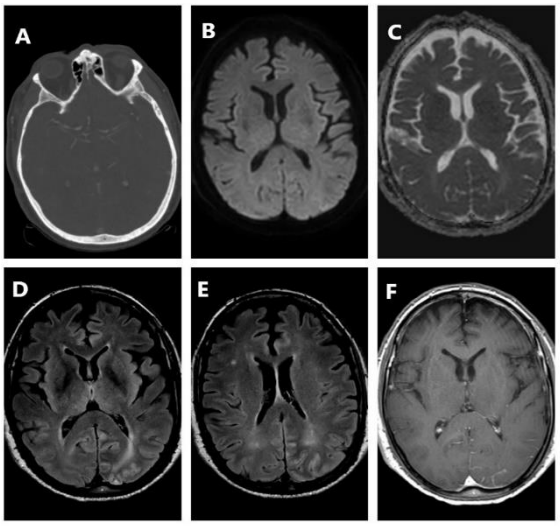

Figure 2: (A) Axial Angio CT showing a prominent right posterior communicating artery. **(B-C)** Axial DWI and ADC showing no diffusion restriction. **(D-E)** Axial FLAIR showing gyri form increased signal and **(F)** gyri form enhancement on T1 with gadolinium on left parieto-occipital cerebral hemisphere.

5. Outcome and Follow Up

There was progressive neurological improvement, with complete resolution of the new symptoms at 48 hours. A new brain MRI was done, which showed less increased signal on FLAIR and no infarct. The patient was discharged on the fifth day with a GCS of 15, he also experienced great improvement in sensorimotor symptoms which progressed with each postoperative recovery day. The event was interpreted as a transitory encephalopathy caused by the contrast agent that was used during the procedures which was directed mostly to the left posterior cerebral artery injected from the vertebral artery, because of the presence of a big right posterior communicating artery. This big right communicating artery is the reason why the right occipital hemisphere is predominantly vascularized by the right internal carotid artery and why we have the atypical

symptoms.

6. Discussion

Transient neurologic deficits following cerebral angiography were previously assumed to result from embolism or factors that impair cortical perfusion, such as SAH or atherosclerotic disease during angiography. However, contrast medium (CM)-induced disruption of the Blood-brain-barrier and neurotoxicity by CM have also been reported as a mechanism of cerebral dysfunction [6]. This event has also been reported in coronary, abdominal angiography or contrast-enhanced CT [6,7]. Symptoms usually start during the procedure or in the following 12 hours [8]. Some authors have reported transient cortical blindness after conventional angiography [6], others reported seizure attacks and transient hemiparesis or aphasia [6,7]. However, there are no reports of patients with neurotoxicity induced by contrast medium who presented with hemianopsia, prosopagnosia and hallucinations. We explain this atypical presentation due to the particular hemodynamic factor of our patient.

Previous reports on patients who developed encephalopathy following angiography have also described CT findings of cortical enhancement and edema, even though in some cases the CT has been normal, as in our case. MRI findings in patients suffering neurotoxicity from contrast agents used in angiography can show areas of increased signal on T2 and FLAIR weighted images located in the cortex. Moreover, enhancement on T1 with gadolinium has recently proved to be capable of detecting the BBB disruption more sensitively and precisely than either MR without enhancement or CT [6], as we saw in our patient. DWI and ADC maps must always be done to exclude a cerebral infarction.

This cortical enhancement is thought to be secondary to contrast extravasation due to disruption of the blood brain barrier following the angiographic procedure [9]. In spite of the BBB being not permeable to CM, transient breakdown of the BBB by CM is well described [6]. This has been explained by hyperosmolality and chemotoxicity of the CM. Hypertonic solutions draw water out of the endothelial cells of cerebral vessels, as the cells shrink and the tight junctions separate out. The severity of the barrier disturbance is questionably related to the ionic and chemical contents of the medium (chemotoxic action). Some factors that could contribute to BBB disruption are iodine concentration, low temperature of the solutions (higher viscosity) and brief injection interval times [6].

The neuronal toxic effect tends to occur predominantly in the parietooccipital cortex, since the posterior circulation is more susceptible to damage by its sympathetic enervation, being more sensitive to blood pressure changes. There is less vascular autoregulatory capacity in this zone since the arteriolar vasoconstriction capacity in the event of an increase in blood pressure is diminished in the posterior circulation [10]. This explains changes observed in posterior reversible encephalopathy syndrome (PRES), seen in patients with hypertensive emergencies and eclampsia.

Some comorbidities, such as renal failure, could contribute to the development of this type of encephalopathy by alteration of contrast clearance. Chronic hypertension is the most important risk factor for the development of this pathology. The cerebral autoregulatory capacity in these patients is usually compromised [1]. There must be very strict control of the blood pressure, since increased pressure aids these hyperosmolar, neurotoxic substances in crossing the blood brain barrier, with the posterior vascular territories being the most sensitive [1]. The lowest possible dose of contrast material should be administered at the lowest iodine concentration which will provide adequate diagnostic information.

We must know that transient encephalopathy needs no treatment because it is self-limiting and reversible [5]; patients progressively recuperate between 6 to 48

hours following the procedure [8]. However, corticoids and diuretics can decrease the duration of symptoms.

7. Learning Points/Take Home Messages

We report an atypical presentation of transient encephalopathy from contrast medium. This is a rare entity that radiologists and clinicians should be aware. The mechanism is more likely related to neurotoxicity of contrast agent injected during angiography with transient disruption of blood-brain barrier. We consider that it must only be diagnosed when all other causes have been ruled out, as more important and frequent causes, which can have devastating and irreversible effects if not identified, must be excluded promptly. It is very important to study hemodynamics of every patient, factor which explained the atypical symptoms of our case.

8. Ethical Approval

All procedures performed in the studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

9. Informed Consent

Informed consent was obtained from the patient included in the study.

References

1. L Guimaraens, Elio Vivas andrés Fonnegra, Teresa Sola, Lluis Soler, Ernest Balaguer, Jaime Medrano, et al. Transient Encephalopathy from Angiographic Contrast. Cardiovasc Intervent Radiol. 2010; 33: 383-388.

2. AA Dawkins, AL Evans, J Wattam, CAJ Romanowski, DJA Connolly, TJ Hodgson, et al. Complications of cerebral angiography: a prospective analysis of 2,924 consecutive procedures. Neuroradiology. 2007; 49: 753-759.

3. Studdard WE, Davis DO, Young SW. Cortical blindness after cerebral angiography. Case Report. J Neurosurg. 1981; 54: 240-244.

4. Utz R, Ekholm SE, Isaac L, M Sands, D Fonte. Local blood–brain barrier penetration following systemic contrast medium administration. Acta Radiol. 1988; 29: 237-242.

5. Wishart DL. Complications in vertebral angiography as compared to non-vertebral cerebral angiography in 447 studies. Am J Roentgenol Radium Ther Nucl Med. 1971; 113: 527-537.

6. Shyn PB, Bell KA. Transient cortical blindness following cerebral angiography. J La State Med Soc. 1989; 141: 35-37.

7. Paúl L, Vicente JM, Pastorín R, A Casasco. A case of temporary nonthrombotic hemiplegia and aphasia due to neurotoxicity from angiographic contrast material? Radiologia. 2009; 51: 614-617.

8. Juan García de Lara, José M.Vázquez-Rodríguez, Jorge Salgado-Fernández, Ramón Calviño-Santos, Nicolás Vázquez-González, Alfonso Castro-Beiras. Transient Cortical Blindness Following Cardiac Catheterization: An Alarming but Infrequent Complication With a Good Prognosis. Rev Esp Cardiol. 2008; 61: 88-90.

9. Lantos G. Cortical blindness due to osmotic disruption of the blood–brain barrier by angiographic contrast material: CT and MRI studies. Neurology. 1989; 39: 567-571.

10. Merchut MP, Richie B. Transient visuospatial disorder from angiographic contrast. Arch Neurol. 2002; 59: 851-854.

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