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CASE REPORT

Transient Cortical Blindness After Cardiac Catheterization: A Case Report and Review of Possible Neurological Etiologies

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Abstract

Cardiac catheterization is a standard procedure performed approximately 1 million times per year. Transient cortical blindness is a rare complication of this procedure. Herein we report a case of complete bilateral vision loss after cardiac catheterization through right radial access, which, to our knowledge, has only been reported once before. Prompt identification of this complication is crucial for patient care. This report provides insight into the diagnostic difficulties, differential diagnosis, imaging findings, and management of transient cortical blindness.

Keywords: Transient cortical blindness, Cardiac catheterization, Posterior reversible encephalopathy syndrome, Contrast-induced neurotoxicity, Hypertensive encephalopathy

1. Introduction

Transient cortical blindness is a rare complication of cardiac catheterization, with 14 reported cases to date in patients who have not undergone coronary artery bypass grafting (CABG); this complication has occurred more frequently in patients following CABG. A single case report has been published on this complication in patients undergoing cardiac catheterization with the right radial approach.[1] The only adequately powered study noting this complication is of Vik-Mo et al. who reported 15 cases of transient visual disturbances and 5 cases of transient cortical blindness.[2] Prompt diagnosis of this complication is crucial to avoid misdiagnosis and provide appropriate patient care.

2. Case description

A 55-year-old woman presented for outpatient cardiac catheterization for staged percutaneous coronary intervention (PCI) of the left anterior descending artery (LAD) and its diagonal branch. Pre-operatively, the patient was clinically stable with a blood pressure of 138/65 mmHg. PCI was performed; one drug-eluting stent (DES) was placed in the LAD, and two were placed in the first diagonal branch. Intra-operatively, the patient became hypertensive with a blood pressure of 190/110 mmHg, for which she was administered labetalol 10 mg intravenously (IV). A total of 300 mL of iohexol contrast was used during the procedure. The patient complained of complete bilateral vision loss in the immediate post-operative period. The patient was anxious, tearful, and subsequently confused. A neurological examination revealed that the patient's pupils were equal, round, and reactive to light, and her speech was slurred. The NIHSS stroke scale was performed, and the patient scored 5 points, 2 points for answering both level of consciousness questions incorrectly and 3 points for bilateral hemianopia. The patient had no loss of

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motor strength or abnormalities in extraocular movements. The patient noted that her vision improved 2 days postoperatively and that her vision had completely returned 3 days postoperatively. Five days postoperatively, the patient's speech was mildly slurred, and she was discharged to an acute rehabilitation facility.

2.1. Past medical history

Four months prior to presentation, the patient had coronary artery disease (CAD) after PCI of the right coronary artery (RCA). In addition, the patient has type 2 diabetes mellitus, for which she is maintained on 56 units of insulin glargine at night and 8 units of insulin lispro with meals. The patient also has a history of controlled hypertension for 7 years on losartan 50 mg daily, hyperlipidemia on atorvastatin 20 mg daily, morbid obesity, chronic obstructive pulmonary disease, sleep apnea, fibromyalgia, depression, anxiety, and a 30 pack-year smoking history with no smoking cessation efforts. She was functional pre-operatively and, apart from CAD, had a stable cardiovascular status with no signs and symptoms of heart failure.

2.2. Differential diagnosis

The differential diagnoses for sudden onset complete bilateral vision loss include bilateral occipital lobe stroke, posterior reversible encephalopathy syndrome, contrast-induced neurotoxicity, occipital epilepsy, reversible cerebral vasoconstriction syndrome, and conversion disorder.

2.3. Diagnostic assessment

Hematology and serum chemistry results were unremarkable, except for elevated glucose levels, 378 mg/dL. Serum creatinine was normal at 1.0 mg/dL. A computed tomography (CT) scan of the head without contrast taken just after the event was limited due to the presence of contrast (Fig. 1). In addition, CT angiogram (CTA) of the head and neck revealed neither evidence of atherosclerosis involving the carotid or vertebral arteries nor evidence of an atherosclerotic plaque or thrombus involving the aortic arch. Magnetic resonance imaging (MRI) at the time of presentation revealed normal findings. Even so, the MRI study was incomplete owing to the low tolerance of the patient to the procedure. An echocardiogram performed the same day showed a left ventricular ejection fraction of 55−60% with no evidence of a thrombus. In addition, there was no evidence of atrial fibrillation before, during, or after the procedure. The next day, a repeat MRI revealed acute punctate infarcts in the bilateral occipital lobes and nonspecific edema in the cerebellar hemispheres. The cerebellar infarct on the right was larger than that in the left hemisphere (Fig. 2). MRI obtained 12 days after the initial presentation showed a less intense but persistently increased T2 signal in the cerebellar hemispheres without occipital infarcts (Fig. 3).

2.4. Therapeutic intervention

The patient was considered to have contrast-induced neurotoxicity (CIN) or posterior reversible encephalopathy syndrome (PRES) subsequent to neurological evaluation, negative CT findings, and initial MRI findings (with bilateral symptoms). Thrombolytic therapy was not administered as a result of the non-stroke diagnosis. She was administered aspirin, ticagrelor, and high-intensity statins. In addition, the patient received physical, occupational, and speech therapies as tolerated.

2.5. Follow up

Outpatient MRI was performed a week after discharge, as previously mentioned. One month after discharge, the patient had normal vision and mildly persistent slurred speech.

3. Discussion

Knowledge of transient cortical blindness is well-established with vertebral and cerebral angiography. Previous studies have reported that transient cortical blindness has a 0.2−1% incidence rate. However, it is a lesser-known complication of cardiac catheterization. The pathophysiology includes disruption of the blood−brain barrier (BBB) due to the hyperosmolar composition of the contrast media relative to the blood. The reason for the predominant involvement of the occipital lobes remains to be elucidated. Even so, it was previously hypothesized that the BBB is uneven and incomplete over the occipital cortex, resulting in its predominant involvement.[3] During cardiac catheterization in a patient with a history of CABG involving the left internal mammary artery (LIMA) to the LAD, failure of selective contrast injection into the LIMA and injection into the vertebral artery is possible and sometimes inevitable.[4] Thus, contrast-associated BBB disruption may be a likely complication of this procedure.

In patients without prior LIMA to LAD graft and cardiac catheterization with selective involvement of

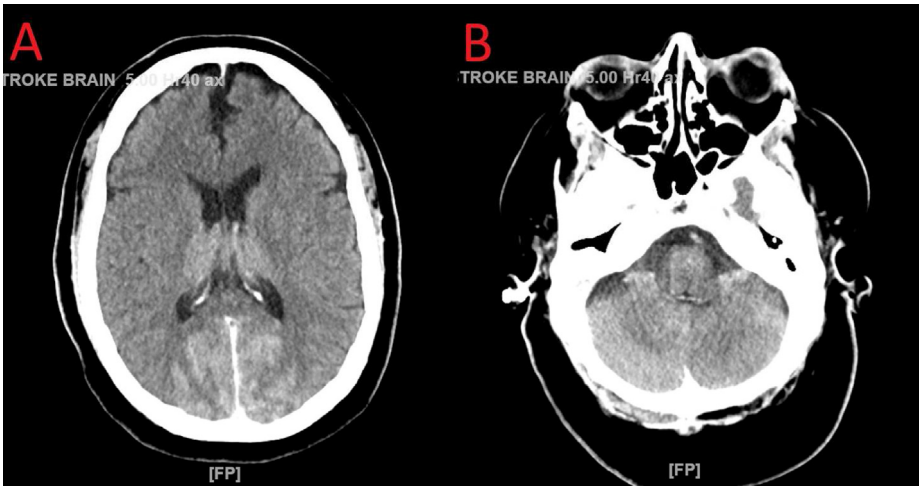

Fig. 1. CT scan of the head without contrast immediately after the procedure. A: Residual contrast from cardiac catheterization noted in bilateral occipital lobes. B: Similar residual contrast noted in bilateral cerebellar lobes.

coronary arteries, as in our case, this mechanism would be possible through inadvertent injection of contrast into the vertebral artery. As noted on the CT scan of the head immediately after the procedure, there was significant contrast material in the cerebellar and occipital regions. CIN is historically noted with the use of high osmolality ionic contrast material. The use of a non-ionic low osmolality contrast material such as iohexol, although infrequently, is associated with CIN.[5] The volume of contrast used during the procedure is not highly associated with the development of CIN, with such a presentation seen when the amount of contrast used is 75 mL–400 mL.[5]

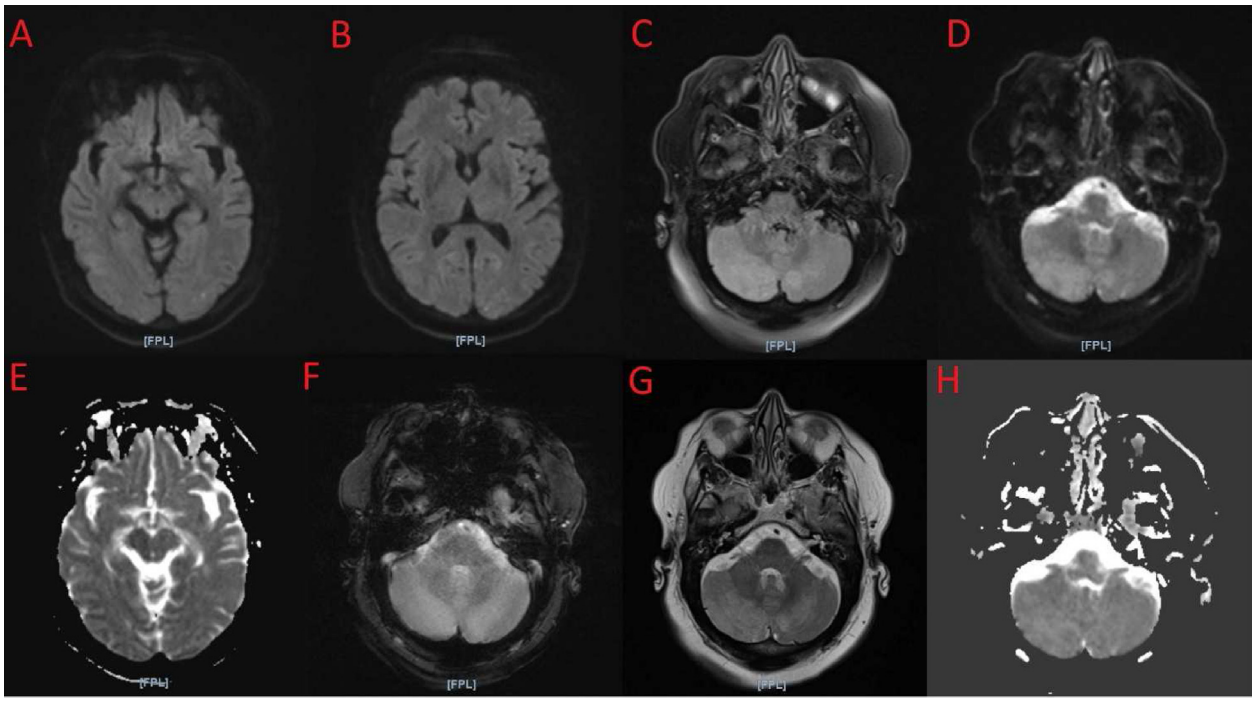

Fig. 2. Magnetic resonance imaging (MRI) on the day of presentation. A, B: Diffusion-weighted images with showing punctate hyperintense lesions in bilateral occipital lobes. C: Fluid-attenuated inversion recovery (FLAIR) showing abnormal hyperintensities in bilateral cerebellum, right greater than left. D: Diffusion-weighted images of the cerebellum not showing any hyperintense abnormalities. E: Apparent diffusion coefficient (ADC) image corresponding to image A showing hypointense punctate lesions. F: Gradient echo (GRE) image with no evidence of hemorrhage. G: T2-weighted image showing findings similar to the FLAIR image. H: ADC image of the cerebellum not showing any hypointense abnormalities.

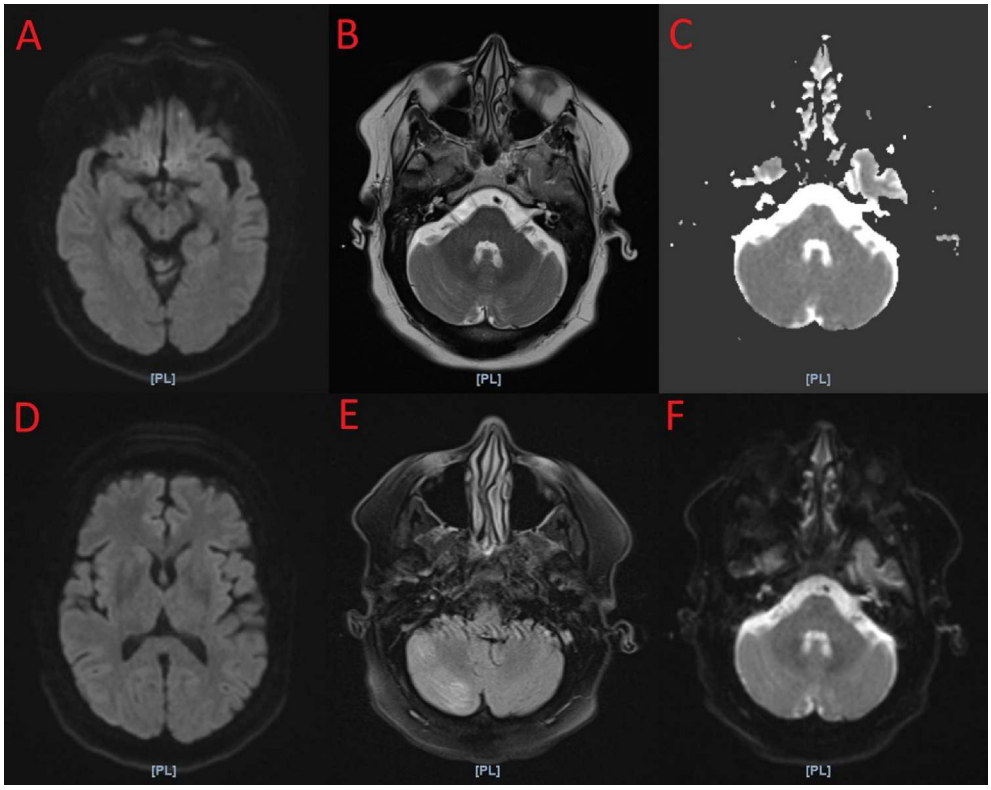

Fig. 3. Magnetic resonance imaging (MRI) performed 12 days after the initial presentation. A, D: Diffusion-weighted images with arrows not showing punctate hyperintense lesions in the occipital lobes noted in Fig. 2. B: FLAIR image of the cerebellum showing improved bilateral hyperintensities. C: ADC image of the cerebellum not showing any hypointense abnormalities. E: T2-weighted image showing similar findings to the FLAIR image. F: Diffusion-weighted images of the cerebellum not showing any hyperintense abnormalities.

Another theory for this phenomenon is a transient bilateral embolic phenomenon through dislodgement and embolization of a thrombus or calcific/cholesterol particle from an atherosclerotic plaque during the advancement of the catheter tip over the guidewire. This is more commonly noted in patients who received cardiac catheterization through femoral access. Dislodgment at any point throughout the course of the catheter from the femoral artery to the aortic arch can cause an embolic phenomenon involving bilateral vertebral arteries, resulting in such a presentation. When radial access is used, the possibility of bilateral embolic phenomenon reduces as dislodgement at any point from the radial artery to the brachiocephalic trunk would cause unilateral vertebral artery emboli. Only when the catheter tip reaches the aortic arch would there be a risk of bilateral vertebral artery involvement. The bilateral embolic phenomenon causing transient cortical blindness has only been described once in the medical literature.[1]

Consequently, another mechanism that is likely for the patient's clinical findings is hypertensive encephalopathy causing PRES. A sudden increase in blood pressure, as documented in our case in the presence of pre-existing hypertension, can result in a loss of the autoregulatory capacity of brain vessels. This results in the formation of areas of vasodilation and vasoconstriction. The outcome is the disruption of the BBB.[6]

Acute cortical blindness after cardiac catheterization is typically managed conservatively through clinical monitoring. Patients subsequently regain their vision within a few hours or days.[7] The need for repeat cardiac intervention rarely arises. In these cases, contrast may be a causative agent, and a contrast rechallenge is best avoided. Rama et al. described three cases in which transient cortical blindness occurred after coronary and repeat angiograms.[8] However, Sadiq et al. described a case in which repeated neurotoxicity was prevented after the administration of 200 mg IV hydrocortisone.[1] As

such, there does not appear to be an increased risk of cortical blindness from repeat exposure to contrast.

4. Conclusions

Transient cortical blindness is a serious complication of cardiac catheterization, with major causes being bilateral embolic phenomenon, CIN, and PRES. As indicated by the name, conservative management improves patient outcomes. However, accompanying symptoms, such as mental status change and speech disturbance, may be noted with possible residual deficits, such as slurred speech, as seen in our patient. Early intervention, including blood pressure control and decreasing the threshold for the administration of IV labetalol, may help to prevent these complications.

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Conflict of interest

The authors declare no conflicts of interest.

Regulatory approval

This is a retrospective report of patient events, and thus no regulatory approval was needed. There are no patient identifiers in the manuscript. However, written informed consent has been taken from the patient for submission of this manuscript.

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References

1. Sadiq MA, al Habsi MS, Nadar SK, Shaikh MM, BaOmar HA. Transient contrast induced neurotoxicity after coronary angiography: a contrast re-challenge case. *Pakistan J Med Sci*. 2020; 36(5). https://doi.org/10.12669/pjms.36.5.2688.
2. Vik-Mo H, Todnem K, Følling M, Rosland GA. Transient visual disturbance during cardiac catheterization with angiography. *Cathet Cardiovasc Diagn*. 1986;12(1):1–4. https://doi.org/10.1002/ccd.1810120102.
3. Kinn RM, Breisblatt WM. Cortical blindness after coronary angiography: a rare but reversible complication. *Cathet Cardiovasc Diagn*. 1991;22(3):177–179. https://doi.org/10.1002/ccd.1810220305.
4. Lee ZV, Arjan Singh RS. Transient cortical blindness after coronary angiography, bypass graft angiography, and coronary angioplasty. *Cureus*. January 2021. https://doi.org/10.7759/cureus.12542.
5. Yazici M, Ozhan H, Kinay O, et al. Transient cortical blindness after cardiac catheterization with iobitridol. *Tex Heart Inst J*. 2007;34(3):373–375.
6. Hinchey J, Chaves C, Appignani B, et al. A reversible posterior leukoencephalopathy syndrome. *N Engl J Med*. 1996;334(8): 494–500. https://doi.org/10.1056/NEJM199602223340803.
7. Horwitz NH, Wener L. Temporary cortical blindness following angiography. *J Neurosurg*. 1974;40(5):583–586. https://doi.org/10.3171/jns.1974.40.5.0583.
8. 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):149–151. https://doi.org/10.1002/ccd.1810280211.