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CASE REPORT

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# Transient cortical blindness, a rare complication during cerebral digital subtraction angiography: A case report and literature review

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**Abstract**

Transient cortical blindness (TCB) is a rare consequence of cerebral angiography with no recognized cause. TCB was observed in a patient with a wide-neck cavernous aneurysm during digital subtraction angiography. One hour after angiography, vision returned spontaneously, with no neurological damage. An MRI was performed three hours after the incident and revealed no abnormalities.

**KEYWORDS**

cerebral angiography, TCB, transient cortical blindness

## 1 | INTRODUCTION

Transient cortical blindness (TCB) is associated with the loss of perceived vision, normal fundi, normal papillary reflexes, and unaltered extraocular movements.[1,2] It is a rare but a known complication of cerebral and coronary angiography following the administration of contrast medium with a reported incidence of 0.3%–1%.[3] Due to the introduction of newer contrast agents, Till et al. recently reported a 0.2% incidence.[4] The incidence of TCB in vertebral angiography is greater relative to the cardiac angiography.[5] Symptoms may appear immediately after a contrast medium injection and disappear within 24 h. We present a case of TCB with DSA and MRI findings.

## 2 | CASE REPORTS

Two episodes of right retro-orbital pain with loss of vision in the right eye were reported by a 42-year-old male patient with a BMI of 25 kg/m$^2$. There has been no previous history of headaches, nystagmus, or strabismus. There has been no loss of consciousness. He had never suffered a head injury before. There is no history of diabetes or high triglycerides. He was a newly diagnosed essential hypertensive who was taking 5 mg amlodipine. His blood pressure at the time of admission was 135/80 mmHg. Due to the previous cerebral flow diverter, he was on daily Plavix 75 mg and Aspirin 75 mg. There is no history of aneurysms in the family. Extraocular muscles were normal, and there was no cranial nerve palsy on physical examination.

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Pupils were equal and reactive to light and accommodation. There was no additional sign of a neurological deficit.

A massive right cavernous saccular aneurysm was diagnosed during his admission magnetic resonance cerebral angiography (Figure 1). To exclude the aneurysm from the parent artery, a 42.5 × 35 mm pipeline flex embolization device (PED) (Intracranial flow diverter stent) was deployed across the cavernous internal carotid artery aneurysmal neck 24 h later. There was no immediate difficulty. For the past 6 months, no reaction to the contrast medium has been reported. After 24 h on prasugrel, the patient was discharged. The patient reported that his symptoms had improved and that he had no new ones. PED stent migration was visible on follow-up skull radiographs at one and 6 months.

A 6-month follow-up digital subtraction angiography of the right internal carotid artery and vertebral artery was performed utilizing a right radial artery approach with a 5F DAV catheter. The angiography was performed with 0.5 ml/kg of Omnipaque 300 mg/ml contrast medium diluted to 50% with normal saline. The internal carotid angiography revealed a proximally migrated stent into the aneurysm sac (Figure 2) with no contrast medium extravasation. The patient became unexpectedly combative during the same procedure, when the right vertebral artery was canulated and contrast medium was injected. He had a brief tonic seizure (lasting about 30 s), followed by horizontal nystagmus, tinnitus, loss of vision, and incomprehensible speech. He remained calm, intelligible in speaking, and progressively recovered his vision over the course of an hour after the seizure. The procedure was canceled. His blood pressure was constant between 140/80 and 145/84 mmHg during and after the surgery. His electrolytes and renal function were both within normal ranges.

The angiograms in the vertebrobasilar and internal carotid artery areas revealed no vascular occlusion or spasm. Within 2 h of angiography, brain magnetic resonance imaging revealed nonspecific patchy T1-weighted hypo-intensities and T2-weighted hyper-intensities in the periventricular areas. On diffusion weighted images and apparent diffusion coefficient, there was no restricted diffusion (Figure 3). Because the non-contrast pictures were non-specific, gadolinium was not used. The authors decided against using a CT scan of the brain because they believed T2-weighted MRI images were more sensitive to fluid-related abnormalities and would expose the patient to less radiation. After a 24-h observation period, the patient was discharged. His 3 months post-event review were uneventful (Table 1).

## 3 | DISCUSSION

Transient cortical blindness is a dramatic complication of coronary and cerebral angiography. It is extremely rare, and our hospital has performed 61 cerebral angiograms over the course of the past 2 years, but this is the very first example of it that we have seen (0.02%). This is in line with the findings of recently published research that can be attributed to more modern contrast agents.[4]

The symptoms of TCB can begin as soon as the contrast agent is injected, and they can take up to 24 h to appear.[5] Bilateral visual loss has been reported in a number of case reports as noted in the current case.[6–8]The index case experienced in addition, tonic seizure, tinnitus, horizontal nystagmus, and incoherent speech. The recovery of normal vision and symptoms may range from few hours, as in the index case of 1 h to about 5 days.[2,7,9]

An embolism is a relatively uncommon complication that can arise from cerebral angiography. It causes cortical ischemia, which is often unilateral. When considering the clinical context of angiography, bilateral cortical blindness is not likely to be due to bilateral embolic infarction. However, it is absolutely necessary to rule out the possibility of a hemorrhagic or embolic infarction by using CT and/or MRI, with negative MRI in this particular case.

After cerebral angiography, there have been two primary hypotheses proposed for TCB, both of which are very speculative and highly contentious. The vast majority of authors are of the opinion that the neurotoxic impact of the contrast agent is responsible for osmotic rupture of

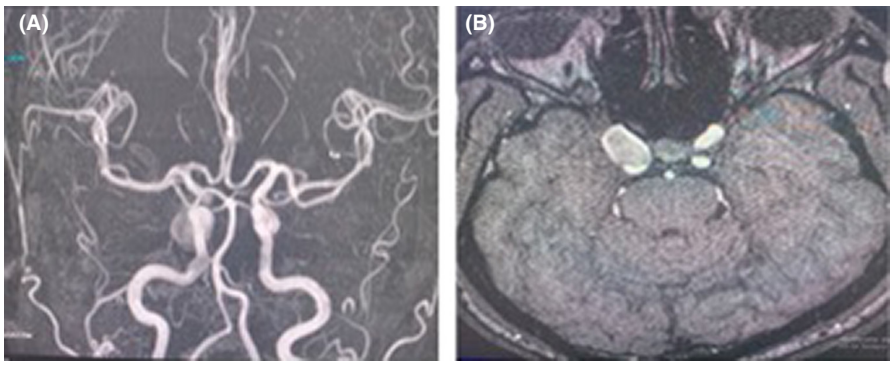


FIGURE 1 Magnetic resonance angiography (A) image showing right cavernous aneurysm (B) T1 weighted image with gadolinium showing the right cavenous aneurysm

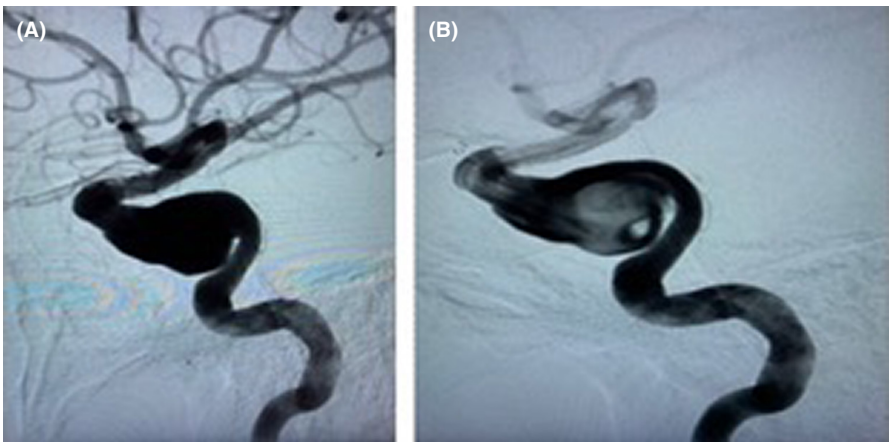


**FIGURE 2** Digital subtraction angiography of the right internal cerebral artery showing (A) a huge cavernous aneurysm (B) migrated PED stent into the aneurysm sac

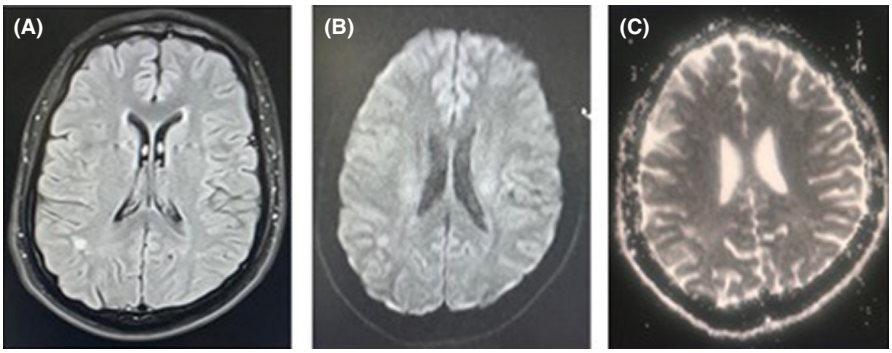


**FIGURE 3** (A) FLAIR (B) DWI (C) images showing patching right parietal lobe hyperintensities

**TABLE 1** Findings of ocular examination 24 h after the event and prior to discharge from the hospital

| OD (Right eye) | Parameter | OD (Left eye) |
|---|---|---|
| 6/6 unaided | VISUAL ACUITY | 6/6 unaided |
| 14 mmHg | INTRAOCULAR PRESSURE (GAT @10AM) | 16 mmHg |
| 15/15 | COLOUR VISION TEST (ISIHARA ) | 15/15 |
| Full and sustained in all directions of gaze | EXTRAOCULAR MOTILITY | Full and sustained in all directions of gaze |
| Normal | EYELIDS | Normal |
| Clear | CONJUNCTIVA | clear |
| White | SCLERA | White |
| Clear with no evidence of epitheliopathy | CORNEA | Clear with no evidence of epitheliopathy |
| Deep and quiet | ANTERIOR CHAMBER | Deep and quiet |
| Pigmented (Brown) | IRIS | Pigmented (Brown) |
| 4 mm in photopic illumination,<br>6 mm in scotopic illumination.<br>Round, centric, regular and reactive to direct and consensual light.<br>No RAPD. | PUPIL | 4 mm in photopic illumination, 6 mm in scotopic illumination.<br>Round, centric, regular and reactive to direct and consensual light.<br>No RAPD. |
| Transparent (no evidence of lens changes) | LENS | Transparent (no cataractous changes) |
| Clear | VITREOUS | Clear |
| Flat retina with areas of chorioretinal degenerative changes (age -related)<br>Well defined disc margin,<br>Pink Neuroretinal Rim,<br>Vertical Cup-dISC ratio: 0.4,<br>Vessels are central.<br>Healthy with normal foveal light reflex | FUNDUS<br>RETINA<br>OPTIC NERVE HEAD<br>MACULA | Flat retina with areas of chorioretinal degenerative changes (age -related)<br>Well defined disc margin,<br>Pink Neuroretinal Rim,<br>Vertical Cup-dISC RATIO: 0.4,<br>Vessels are central.<br>Healthy with normal Foveal Light reflex |

the blood brain barrier (BBB).[10] This hypothesis appears to be supported by the vulnerability of various sympathetic innervation of the posterior circulation to rupture of the blood brain barrier.[11] If a contrast agent such as Omnipaque is not diluted, it will become hyperosmotic to the blood and may exacerbate the breakdown of the blood–brain barrier. It is anticipated that a higher dose of contrast medium may prolong the exposure duration of the cerebrovascular endothelium to the agent by increasing the exposure time. This would ultimately result in a malfunction of the BBB, which would further increase the transfer of contrast material.[2] Table 2 shows previous literature on the type of contrast medium and clinical presentation of patients with TCB during carotid and vertebral angiography.

The second hypothesis is known as PRES, which refers to posterior reversible leukoencephalopathy syndrome.[2] It is a neurotoxic state that develops as a result of the failure of the posterior circulation to auto-regulate in response to sudden shifts in blood pressure. Without infarction, hyperperfusion can occur, which leads to a rupture of the blood–brain barrier (BBB) and vasogenic edema in the periventricular and perivascular areas. This often occurs in the parieto-occipital regions of the brain.[17]

Chronic hypertension could weaken the cerebral arterioles and eventually lead to underperfusion, resulting in brain ischemia and in due course vasogenic edema.[18] Li et al. showed that the rate of hypertension and diabetes were higher in the patients with TCB even though the association does not reach statistical significance.[18] They also showed using logistic regression analysis, patients with low weight who receive higher doses of contrast medium and those with posterior circulation injection have a higher risk of developing TCB than those with lower doses and anterior circulation injection.[18] The index case has a 5-year history of chronic hypertension and TCB occurred during vertebral injection. Yazici et al[19] and Frantz[20] reviewed a total of 33 patients who had transient cortical blindness following coronary angiography and showed that 17 patients had bypass graft and nine patients had chronic arterial hypertension, strengthening the argument of hypertension, and bypass graft as risk factors.

Approximately 50% of people diagnosed with TCB exhibit normal findings on CT scans. Tong et al. discovered, after reviewing 12 cases, that 50% of the patients exhibited extravasation of contrast medium into the subarachnoid spaces and predominantly occipital lobe white matter changes, either unilaterally or bilaterally.[21] The MRI is more sensitive than other imaging modalities, notably the FLAIR (fluid-attenuated inversion recovery) and DWI (diffusion weighted imaging) images. These images show significant signal changes in the parieto-occipital white matter and sometimes patchy contrast enhancement.

**TABLE 2** Contrast encephalopathy following arteriography with adverse clinical outcomes: demographics, contrast agents, presentation, CT findings, and clinical outcome

| Reference | Age/Sex | Arteriography | Indication | Possible risk factor | Previous angiography | Contrast agent/ Volume | Presentation | CT/MRI involved | Clinical resolution | CT/MRI resolution |
|---|---|---|---|---|---|---|---|---|---|---|
| Sarkodie et al (index case) | 42/M | Carotid artery | Post flow diverted insertion follow-up | Hypertension | Yes | Iohexol injection/40 ml diluted to 80 ml | Cortical blindness, Horizontal nystagmus, brief motor seizure | MRI -Periventricular T1 hypo and T2 hyper-intensities | Within 1 h | N/A |
| Guimaraens et al. (2009)[12] | 51/M | Carotid artery | Right ICA aneurysm coiling | Hypertension | No | Iopromide /300 ml | Gerstmann's; left visual field deficit, hemiparesis | MRI - Right fronto-parietal-occipital | 2 days | 5 days |
| Fang et al. (2009)[13] | 80/M | Carotid artery Coronary artery | Coronary and carotid stenting | Hypertension | No | Iohexol /250 ml | Right hemiparesis | left fronto- parietal-occipital | 2 days | 4 days |
| Niimi et al. (2008)[14] | 54/F | Vertebral artery | Coiling basilar apex aneurysm | Non reported | No | Non-ionic/62 ml | Vision loss to light perception | occipital lobe | 30 days | N/A |
| Saigal et al. (2004)[15] | 74/F | Carotid artery Vertebral artery | Coiling basilar tip aneurysm | Hypertension | No | Iohexol /Not stated | Complete bilateral blindness; confusion | left parieto-occipital | 1 day | N/A |
| Dangas et al. (2001)[16] | 82/M | Carotid artery | Carotid artery stenting | Hypertension | Yes | Ioxaglate/50 ml | Confusion; left hemiparesis | Right fronto-parietal | 2 days | 2 days |

Posterior reversible leukoencephalopathy syndrome exhibits the characteristics that are comparable with these.[7] The MRI of the brain performed for the index case within 2 h of the incidence showed patchy periventricular T2-high signal intensities.

Because the condition resolves on its own, medical intervention is rarely necessary. TCB is a diagnosis of exclusion, some authors recommend that steroid treatment, anticoagulation, and hydration be administered before the patient's vision is restored.[21] Steroids will reduce the vasogenic edema and stabilize the BBB. Our patient did not receive any specific treatment; however, we ensured that the airway was patent, blood pressure and pulse were normal, and his random blood sugar was 8 mmol/L. He complained of headache for which 1 g paracetamol was administered orally.

## 4 | CONCLUSION

The diagnosis of TCB is one of exclusion. It is a serious and unusual complication that can occur during cerebral and coronary angiograms. Neurotoxicity of the contrast medium as well as PRES have been proposed as possible causes, despite the fact that this is contentious. Chronic hypertension, receiving a large dosage of contrast medium, and having a vertebral angiography are all independent risk factors. The condition exhibits complete recovery within hours to days, with no vestige of neurological deficit remaining after recovery. It is necessary to treat it with supportive therapy, but there is no evidence to support the use of steroids. Computed tomography, magnetic resonance imaging, and angiography should all be performed immediately in order to rule out the possibility of embolic infarction as a complication of angiography. Radiologists and radiographers need to be aware of TCB, so that they can avoid unnecessary procedures in light of the dramatic presentation of the index case.

## AUTHOR CONTRIBUTIONS

B Sarkodie and E Jackson performed the digital subtraction angiography. B Jimah and D Anim reviewed the MRI. B Jimah prepared the manuscript. E. Brakohiapa critically reviewed and revised the manuscript. All authors read and approved the final version of the manuscript.

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## CONFLICT OF INTEREST

The authors declare no competing interest.

## DATA AVAILABILITY STATEMENT

Data sharing is not applicable to this article as no new data were created or analyzed in this study.

## CONSENT

A consent form was signed by the patient to publish this report in accordance with the journal's patient consent policy.

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