# 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 cerebral angiography complication with unknown etiology. A patient with a wide-neck cavernous aneurysm developed TCB. Vision restored spontaneously one hour after angiography, with no lasting neurological deficit. Three hours after the incident, MRI indicated no abnormalities.

#### Introduction

Transient cortical blindness (TCB) is associated with 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 administration of contrast medium with a reported incidence of 0.3-1% (3). Till et al. reported recently an incidence of 0.2% due to the emergence of newer contrast agent (4). Vertebral angiography has a relative higher incidence (5). The symptoms may start immediately after contrast medium injection and resolve within 24hours. We report a case of TCB during DSA and MRI features.

## CASE REPORTS

A 42-years-old male with body mass index of  $25 \text{kg/m}^2$ , presented with two episodes of right retro-orbital pain with loss of vision in the right eye. No history of headache, nystagmus or strabismus. No loss of consciousness. He had no history of head injury. No history of diabetes or increased triglycerides . He was a newly diagnosed well-controlled essential hypertensive on 5mg amlodipine. His admission blood pressure was 135/80mmHg. He was on Plavix 75mg and Aspirin 75mg daily due to the previous intracranial flow diverter. No family history of aneurysm. Physical examination showed normal extraocular muscles and no cranial nerve palsy. Pupils were equal and reactive to light and accommodation. No other neurological deficit was elicited.

His admission magnetic resonance cerebral angiography demonstrated a huge right cavernous saccular aneurysm (Fig 1). 24hours later, 42.5 x 35mm Pipeline Flex embolization device (PED) (Intracranial flow diverter stent) was deployed across the cavernous internal carotid artery aneurysmal neck to exclude the aneurysm from the parent artery. No immediate complication was noted. No contrast medium reaction was reported for 6months. Patient was discharged after 24hours on prasugrel. Patient reported improvement

in the symptoms with no new symptoms. Follow up skull radiographs at one and six months showed PED stent migration.

A follow-up right internal carotid artery and vertebral artery digital subtraction angiography at 6months was done using a right radial artery approach with a 5F DAV catheter. 0.5ml/kg of Omnipaque 300 mg/ml contrast medium diluted to 50% with normal saline was used for the angiography. The internal carotid angiogram showed proximally migrated stent into the sac of the aneurysm (fig 2) with no extravasation of contrast medium. During the same procedure, when the right vertebral artery was canulated and contrast medium injected, the patient became suddenly aggressive, he experienced brief tonic seizure (lasting about 30seconds), followed by horizontal nystagmus, tinnitus, loss of vision and incoherent speech. Over a period of one hour after the seizure seized, he became calm, coherent in speech and gradually recovered his vision in that order. The procedure was aborted. His blood pressure during and after the procedure were stable between 140/80 - 145/84mmHg. His renal function and electrolytes were within normal limits.

Review of the angiograms did not show any vascular occlusion or spasm in the vertebrobasilar and internal carotid artery territories. Brain Magnetic Resonance Imaging was arranged within two hours after angiography and demonstrated nonspecific patchy T1- weighted hypo-intensities and T2-weighted hyper-intensities in the periventricular areas. No restricted diffusion was seen on diffusion weighted images and apparent diffusion coefficient (Fig. 3). Gadolinium was not used since the non-contrast images were non-specific. Computed Tomography scan of the brain was not done because, the authors were of the opinion that T2 weighted MRI images were more sensitive to fluid related abnormalities and to reduce the radiation to the patient. Patient was discharged after 24hours of observation. Three months follow after discharged was unremarkable.



Fig 1 : Magnetic resonance angiography (a) image showing right cavernous aneurysm.





Fig 2: Digital subtraction angiography of the right internal cerebral artery showing (a) a huge cavernous aneurysm (b) migrated PED stent into the aneurysm sac





## Discussion

Transient cortical blindness is a dramatic complication of coronary and cerebral angiography. It is rare and this is the first case in our institution with 61 cerebral angiograms over a period of 2 years (0.02). This is consistent with recent reports attributable to newer contrast agents (4).

The symptoms of TCB may start immediately after contrast agent injection and upto 24hours (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 one hour to about 5days (7,9,10).

A rare complication of cerebral angiography is embolism which may lead to cortical ischaemia, often unilateral. Bilateral cortical blindness in the clinical context of angiography is unlikely to be bilateral embolic infarction. However, it is essential to rule out hemorrhagic or embolic infarction with CT and /or MRI, which were absent in the current case.

Two main speculative highly debatable hypotheses have been postulated for TCB following cerebral angiography. Most authors believe that the neurotoxic effect of the contrast agent causes osmotic disruption of the blood brain barrier (BBB) (11). The susceptibility of the different sympathetic innervation of the posterior circulation to blood brain barrier disruption appears to support this hypothesis (12). When contrast agent such as Omnipaque is not diluted, it becomes hyperosmotic to blood and may worsen the BBB disruption. It is believed that higher dose of contrast medium may prolong the exposure time of the cerebrovascular endothelium to the agent by increasing the exposure time, thus resulting in BBB malfunction, which would further increase the transfer of contrast material (13). Table 1 shows previous literature on the type of contrast medium and clinical presentation of patients with TCB during carotid and vertebral angiography.

Table 1 Contrast encephalopathy following arteriography with adverse clinical outcomes: demographics, contrast agents, presentation, CT findings and clinical outcome.

Reference	Age/Sex	Arteriography	Indication	Poss
Sarkodie et al. (2021)	42/M	Carotid artery	Post flow diverted insertion follow up	Нуре
Guimaraens et al. $(2009)(14)$	51/M	Carotid artery	Right ICA aneurysm coiling	Hype
Fang et al. $(2009)(15)$	80/M	Carotid artery Coronary artery	Coronary and carotid stenting	Нуре
Niimi et al. $(2008)(16)$	54/F	Vertebral artery	Coiling basilar apex aneurysm	Non
Saigal et al. $(2004)(17)$	74/F	Carotid artery Vertebral artery	Coiling basilar tip aneurysm	Hype
Dangas et al. $(2001)(18)$	82/M	Carotid artery	Carotid artery stenting	Hype

Posterior reversible leukoencephalopathy syndrome (PRES) is the second hypothesis (13). It is a neurotoxic

state that occurs secondary to the inability of the posterior circulation to autoregulate in response to acute changes in blood pressure. Hyperperfusion occurs with resultant disruption of the BBB and vasogenic edema in the periventricular and perivascular spaces, without infarction, most commonly in the parieto-occipital regions (19).

Chronic hypertension could weaken the cerebral arterioles and eventually lead to underperfusion, resulting in brain ischemia and in due course vasogenic edema (20). 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 (20). 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 (20). The index case has a 5-year history of chronic hypertension and TCB occurred during vertebral injection. Yazici et al (21) and Frantz (22) 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.

About 50% of the patients with TCB have normal CT scan features. Tong et al showed after reviewing 12 cases that half of the patients presented with extravasation of contrast medium into the subarachnoid spaces and predominantly occipital lobe white matter changes, either unilateral or bilaterally (23). MRI is more sensitive especially FLAIR (fluid-attenuated inversion recovery) and DWI (diffusion weighted imaging) images and these include high signals in the parieto-occipital white matter and sometimes patchy contrast enhancement. These findings are similar in posterior reversible leukoencephalopathy syndrome (7). The MRI of the brain performed for the index case within two hours of the incidence showed patchy periventricular T2-high signal intensities.

The condition is self-limiting, and often no treatment is required. The condition is diagnosed by exclusion, hence some authors advocate the use of steroids, anticoagulation and hydration prior to the recovery of the vision loss (23). Steroids will reduce the vasogenic oedema 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 8mmol/L. He complained of headache for which 1g paracetamol was administered orally.

## Conclusion

TCB is a diagnosis of exclusion. It is dramatic and rare complication of cerebral and coronary angiography. Though controversial, neurotoxicity of contrast medium and PRES have been suggested as the possible causes. Independent risk factors include chronic hypertension, large dosage of contrast medium and vertebral angiography. The condition shows complete recovery within hours to days with no residual neurological deficit. It requires supportive therapy; no evidence is available for the use of steroids. Embolic infarction as a complication of angiography should be ruled out with computed tomography, magnetic resonance and angiography immediately. Due to the dramatic presentation of the index case, radiologists and radiographers must be aware of TCB to avoid inutile measures.

#### 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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#### AUTHOR CONTRIBUTIONS

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

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Figure Captions

Fig 1 : Magnetic resonance angiography (a) image showing right cavernous aneurysm.

Fig 2: Digital subtraction angiography of the right internal cerebral artery showing (a) a huge cavernous aneurysm (b) migrated PED stent into the aneurysm sac

Fig 3: (a) FLAIR (b) DWI (c) images showing patching right parietal lobe hyperintensities









