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Transient cortical blindness following transfemoral cerebral angiography for giant thrombosed V4 aneurysm: a rare case with diagnostic challenge

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Keywords
Anton syndrome,
Blood-brain barrier,
Cerebral angiography,
Lodixanol,
Transient cortical blindness,
Vertebral aneurysm

Abstract

Transient cortical blindness (TCB) is a rare complication of cerebral angiography, with a reported incidence of 0.3-1%. We report a 46-year-old male with a giant thrombosed left V4 vertebral aneurysm (38 × 42 × 30 mm) who developed acute bilateral total visual loss approximately four hours after transfemoral cerebral angiography (TFCA) using iodixanol 320 mg.

Notably, the patient initially denied any visual loss, raising the consideration of Anton syndrome — a condition characterized by cortical blindness with visual anosognosia and confabulation. Emergency evaluation revealed visual acuity of 1/∞ bilaterally, with normal pupillary reflexes and fundoscopic findings. Post-procedural neuroimaging showed no evidence of acute infarction, posterior reversible encephalopathy syndrome, or contrast extravasation. Treated conservatively with dexamethasone and mannitol, vision recovered fully within 72 hours.

Two principal mechanisms underlie TCB: blood-brain barrier disruption from contrast neurotoxicity, and posterior reversible encephalopathy syndrome from impaired cerebral autoregulation. The giant V4 aneurysm may have augmented posterior circulation vulnerability through local mass effect.

This case illustrates that TCB may be missed when the patient confabulates intact vision, and that Anton syndrome should be considered whenever anosognosia accompanies post-angiographic visual loss.

Introduction

Intracranial aneurysms pose a threat of subarachnoid hemorrhage with potentially devastating outcomes. Imaging and early detection are crucial for diagnosis and prompt management [9]. Despite the wide array of available imaging modalities, digital subtraction angiography (DSA) remains the gold standard for evaluation of intracranial vascular pathology [9,14]. DSA is performed via transfemoral cerebral angiography (TFCA) and, like other invasive procedures, carries procedural risks. Complication rates have progressively

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declined with improvements in catheters, guide wires, and non-ionic contrast agents [14].

DSA complications include thromboembolism, contrast-induced allergic reactions, and angio-site hematoma or infection [14]. Transient cortical blindness (TCB) is a rare complication following cerebral angiography. The reported incidence ranges from 0.3-1%, with some reports citing rates as low as 0.2% with newer contrast agents [10,12,13]. TCB has a higher reported incidence following vertebral angiography [12,13]. Its pathophysiology remains incompletely understood [10,12]. Clinical features include bilateral visual loss with normal fundi, normal pupillary reflexes, and intact extraocular movements. Onset may occur at the time of the procedure or several hours after, with recovery ranging from hours to five days [7].

Clinicians should differentiate TCB from embolic infarction. Bilateral sudden blindness following angiography is more suggestive of TCB than simultaneous bilateral occipital emboli. MRI with diffusion-weighted imaging (DWI) is the key discriminator, as acute infarction demonstrates restricted diffusion absent in TCB [13]. Published diagnostic criteria for TCB require: (1) simultaneous bilateral reversible visual loss unassociated with focal ischemia, epilepsy, migraine, or altered consciousness; (2) normal or near-normal ocular examination; and (3) intact pupillary light reactions [12]. Several case reports of TCB following cerebral angiography have been published [7,10,13,18].

Case Report

We present a 46-year-old past-smoker male with a nine-month history of worsening pulsatile occipital headaches prior to hospital admission. The patient self-medicated with analgesics without significant improvement. He reported prior episodes of transient slurred speech and dysphagia, which had resolved by the time of presentation. No paresis or other focal neurological deficits were noted on examination. Regular medication consisted of amlodipine 10 mg once daily for hypertension.

At a referring hospital, a non-contrast CT scan of the head (Figure 1) revealed a hyperdense lesion between the cerebellum and brainstem, initially suspected as a hemorrhagic lesion. CT angiography (Figure 2) demonstrated a partially thrombosed left V4 vertebral aneurysm proximal to the PICA junction, measuring 38 × 42 × 30 mm. Gadolinium-enhanced MRI-MRA (Figure 3) confirmed a thrombosed left V4 aneurysm of identical dimensions with significant mass effect on the brainstem.

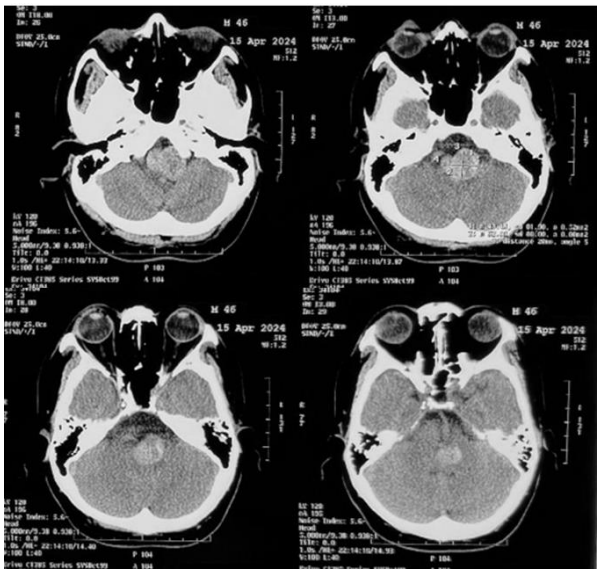

Figure 1. Initial non-contrast CT scan at the referring hospital revealing a hyperdense lesion between the cerebellum and brainstem, initially suspected as a hemorrhagic event.

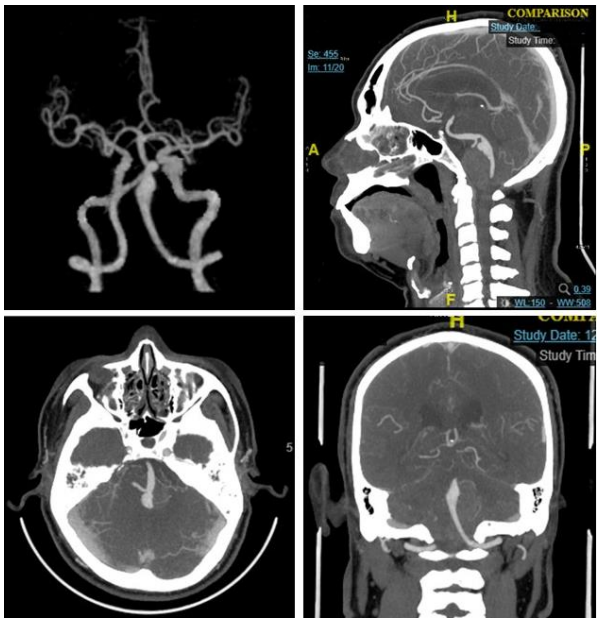

Figure 2. CT angiography demonstrating a partially thrombosed aneurysm at the left V4 vertebral artery.

TFCA was subsequently performed for pre-treatment planning. Six-vessel TFCA (Figure 4) was completed without technical difficulty over approximately 30 minutes using iodixanol 320 mg

(total volume 100-120 cc). Angiography identified a fusiform left vertebral artery aneurysm measuring 8.3 × 15.6 mm and incidentally revealed right A1 artery aplasia.

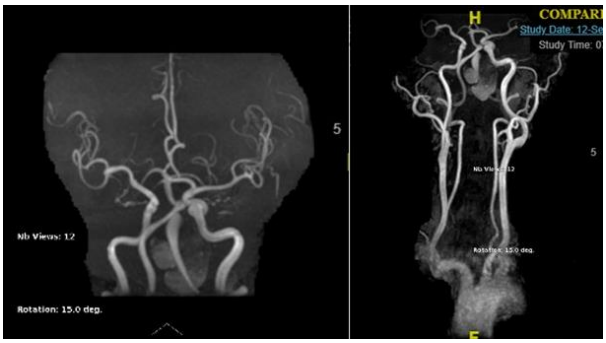

Figure 3. MR angiography revealing an outpouching lesion from the left V4 artery with surrounding thrombosis.

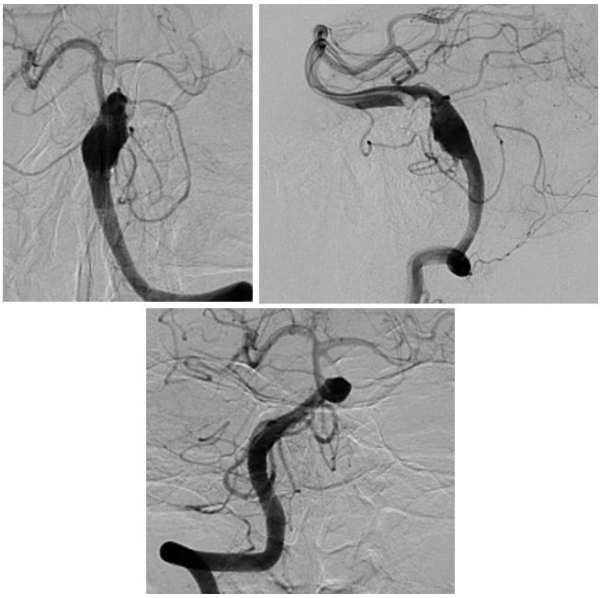

Figure 4. Six-vessel TFCA revealing a fusiform aneurysm at the left vertebral artery (top) along with incidental right A1 artery aplasia (bottom).

Approximately four hours post-TFCA, the patient awoke with acute total bilateral visual loss. Emergency ophthalmological evaluation documented visual acuity of 1/∞ bilaterally. Notably, the patient initially denied any visual loss, instead reporting an inability to locate objects or people — a presentation consistent with visual anosognosia. Fundoscopy revealed mild bilateral blurring of optic disc margins suggesting slight papilledema, with a normal A/V ratio and normal retina. Pupillary light reflexes were intact bilaterally.

Emergency non-contrast CT (Figure 5) and subsequent MRI-MRA (Figure 6), performed several hours post-procedure, showed a stable left V4 aneurysm configuration without significant change in mass effect, no steno-occlusion of cerebral arteries, and no contrast extravasation. The patient received empirical dexamethasone and mannitol (four times daily), given bilateral papilledema and the large aneurysm's posterior circulation mass effect.

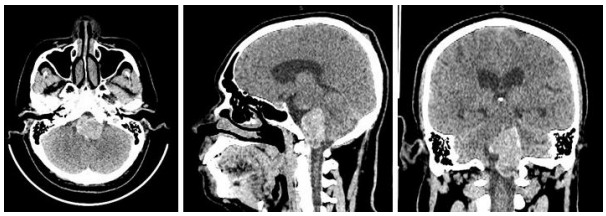

Figure 5. Post-TFCA non-contrast CT scan demonstrating stable left V4 aneurysm configuration without significant change in mass effect.

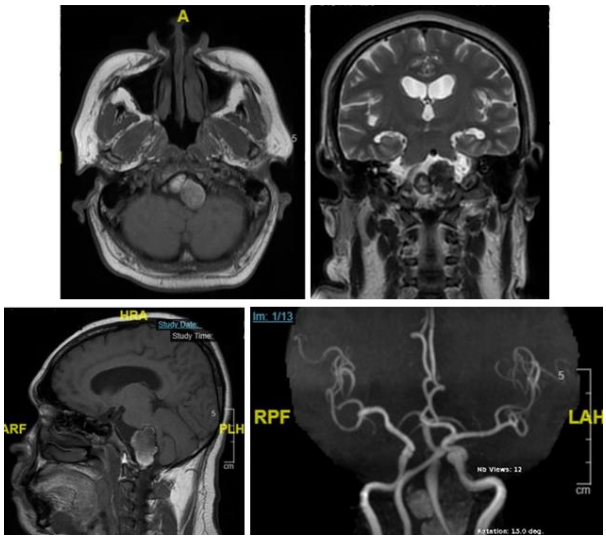

Figure 6. Post-TFCA MRI-MRA showing stable left V4 aneurysm without steno-occlusion.

Visual acuity improved to 1/300 by the following day, with the patient describing flashes of light and moving shadows. By day three post-TFCA, vision had improved substantially, though photosensitivity persisted. Full visual recovery was documented on day four. MRI (Figure 8) confirmed no embolic infarction or PRES-consistent changes on FLAIR or DWI sequences. Three-dimensional reconstruction of the aneurysm (Figure 7) and a clinical timeline (Figure 9) are provided for anatomical context and clarity.

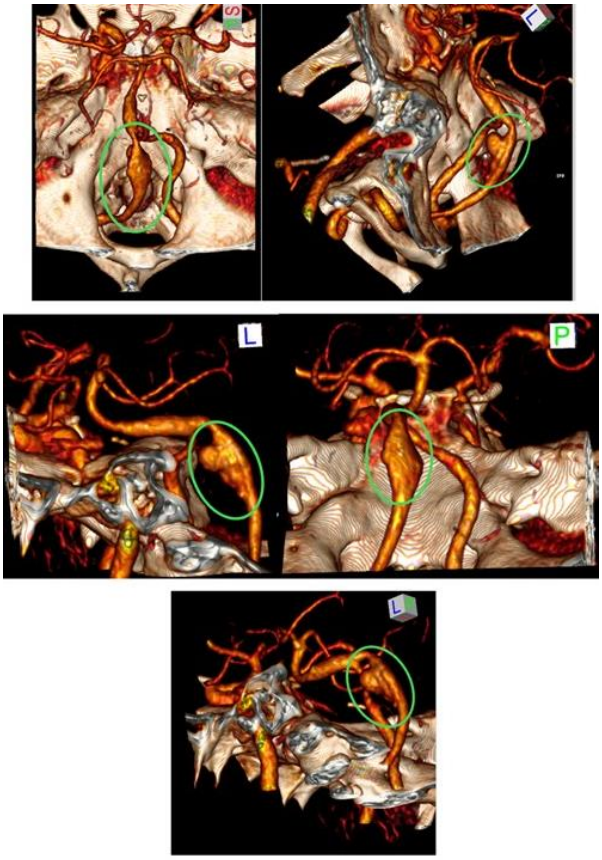

Figure 7. Three-dimensional reconstruction of the left V4 aneurysm demonstrating its morphology and anatomical relationship to the posterior fossa.

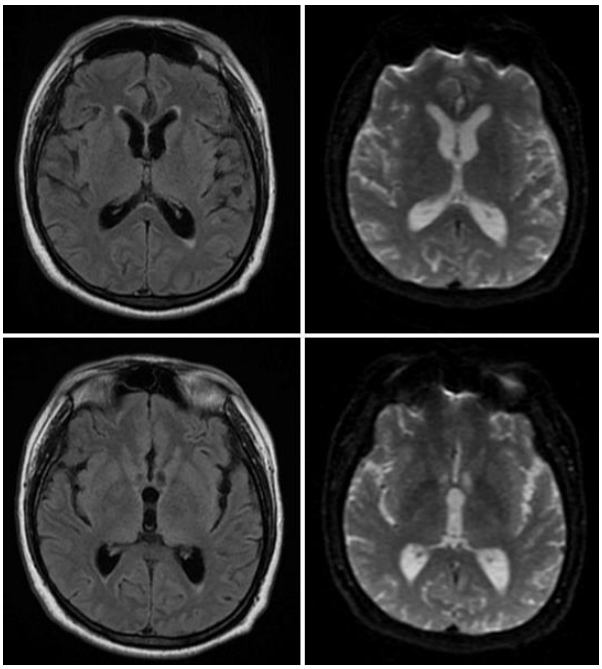

Figure 8. Post-TFCA MRI at the level of the lateral ventricles showing no embolic infarction or PRES-consistent changes. Left: FLAIR sequence. Right: DWI sequence.

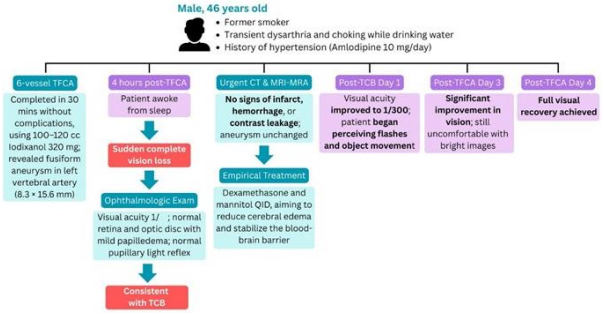

Figure 9. Timeline of clinical events post-TFCA, from procedure completion to full visual recovery on day four.

Discussion

Two principal mechanisms have been proposed for TCB pathophysiology: blood-brain barrier (BBB) disruption from contrast neurotoxicity, and posterior reversible encephalopathy syndrome (PRES) from impaired cerebral autoregulation causing perivascular edema [7,12]. The first mechanism is supported by the comparatively sparse sympathetic innervation of the posterior circulation relative to the anterior circulation, rendering the posterior vasculature more susceptible to contrast-induced endothelial injury and increased permeability [12]. The occipital BBB is relatively thin, facilitating contrast entry into cortical tissue [22].

In this case, iodixanol — a triiodinated, non-ionic isosmolar contrast agent [8,16] — was administered at standard dosage and rate (320 mg, 100-120 cc over 30 minutes), a regimen associated with favorable safety and renal tolerability [6]. Absence of contrast extravasation on post-procedural MRI does not conclusively exclude BBB disruption, as the MRI was performed beyond iodixanol's mean elimination half-life of approximately 2.1-2.2 hours [21].

PRES was considered given the patient's hypertension and posterior-predominant neurological symptoms. PRES is characterized by subcortical posterior edema on MRI, typically reversible with appropriate management [3,5,19,20]. Risk factors include severe hypertension, renal failure, cytotoxic agents, and autoimmune diseases. However, no PRES-consistent radiological findings were identified on post-TFCA MRI in this patient.

The concurrent visual anosognosia raises the diagnosis of Anton syndrome — bilateral cortical blindness with denial of visual loss and visual confabulation [3,4]. The mechanism involves simultaneous impairment of the primary visual

cortex (area 17) and visual association areas (areas 18 and 19), disrupting sensory input and associative integration [4]. Anton syndrome has been reported following cerebral and coronary angiography procedures [2,3,4].

The giant V4 aneurysm (38 × 42 × 30 mm) may have augmented TCB susceptibility through compression of posterior cerebral artery branches supplying the visual cortex [15,17], potentially causing transient hypoperfusion or vascular autoregulation dysregulation [1]. The combination of mechanical aneurysm effects and contrast neurotoxicity likely synergistically increased susceptibility to TCB in this patient.

Conclusion

TCB is a rare but reversible complication following cerebral angiography, with a reported incidence of 0.3-1% [11,12,13]. Standard dosage iodixanol administration precipitated TCB in this patient, suggesting that individual posterior circulation susceptibility — likely amplified by the giant V4 aneurysm's mass effect — may lower the threshold for this complication. When patients deny visual loss following angiography, Anton syndrome should be considered as a concurrent clinical entity [4]. Clinicians should maintain awareness of TCB's diagnostic nuances to enable prompt recognition and appropriate conservative management.

References

1. Chen CY, Chen CJ, Tseng YC. A case report of transient cortical blindness after angiography. Neurologist. 2017;22(3):82-84.
2. Chen JJ, Chang HF, Hsu YC, Chen DL. Anton-Babinski syndrome in an old patient: a case report and literature review. Psychogeriatrics. 2015;15(1):58-61.
3. Das B, Goel G, Mahajan A, Bansal AR, Sapra H, Jha AN. An unusual cause of posterior reversible encephalopathy syndrome. Asian J Neurosurg. 2018;13(4):1254-1256.
4. Das JM, Naqvi IA. Anton syndrome. In: StatPearls [Internet]. StatPearls Publishing; 2024.
5. Geocadin RG. Posterior reversible encephalopathy syndrome. N Engl J Med. 2023;388(23):2171-2178.
6. Geng W, Yang Q, Wang W, Yin Q. The safety of iodixanol in interventional therapy for patients of different ages with cerebrovascular diseases. Am J Transl Res. 2021;13(7):8228-8234.
7. Gomez R, Harris J. Transient cortical blindness following cerebral angiography: a case report (P2-5.015). Neurology. 2023;100(17_supplement_2):2524.
8. Greenberg MS. Greenberg's Handbook of Neurosurgery. 10th ed. Thieme; 2023.
9. Howard BM, Hu R, Barrow JW, Barrow DL. Comprehensive review of imaging of intracranial aneurysms and angiographically negative subarachnoid hemorrhage. Neurosurg Focus. 2019;47(6):E20.
10. Iancu A, Nikoubashman O, Tafelmeier S, Kesseler E, Wiesmann M. Transient cortical blindness after diagnostic cerebral angiography — a single center report. Heliyon. 2024;10(8):e29120.
11. Lagos-Servellon J, Mejia-Raudales B, Yanes-Ponce D. Abstract 065: Acute transient cortical blindness following cerebral angiography: a case report. Stroke Vasc Interv Neurol. 2024;4(S1):e12984_065.
12. Li M, Liang H, Liu C, Liu H, Zheng Y, Shi W, Wang Y, Gu Z, Zhao W, Zhao J. Risk factors of transient cortical blindness after cerebral angiography: a multicenter study. Front Neurol. 2019;10:1005.
13. Lo LW, Chan HF, Ma KF, Cheng LF, Chan TK. Transient cortical blindness following vertebral angiography: a case report. Neurointervention. 2015;10(1):39-42.
14. Nam HH, Jang DK, Cho BR. Complications and risk factors after digital subtraction angiography: 1-year single-center study. J Cerebrovasc Endovasc Neurosurg. 2022;24(4):335-340.
15. Nouh A, Remke J, Ruland S. Ischemic posterior circulation stroke: a review of anatomy, clinical presentations, diagnosis, and current management. Front Neurol. 2014;5:30.
16. PubChem. Iodixanol [Internet]. Cited 2025 Jan 9.
17. Sarkar S, Tripathy K. Cortical blindness. In: StatPearls [Internet]. StatPearls Publishing; 2025.
18. Sarkodie BD, Jimah BB, Anim D, Jackson E, Brakohiapa E, Anaglate AYO. Transient cortical blindness, a rare complication during cerebral digital subtraction angiography: a case report and literature review. Clin Case Rep. 2022;10(7):e6074.
19. Sudulagunta SR, Sodalagunta MB, Kumbhat M, Settikere Nataraju A. Posterior reversible encephalopathy syndrome (PRES). Oxf Med Case Rep. 2017;2017(4):omx011.
20. Triplett JD, Kutlubaev MA, Kermode AG, Hardy T. Posterior reversible encephalopathy syndrome (PRES): diagnosis and management. Pract Neurol. 2022;22(3):183-189.
21. van der Molen AJ, Dekkers IA, Geenen RWF, Bellin MF, Bertolotto M, Brismar TB, Gutierrez JE, Hellstrom M, Sirol M, Zimmer C, Grobner T. Waiting times between examinations with intravascularly administered contrast media: a review of contrast media pharmacokinetics and updated ESUR Contrast Media Safety Committee guidelines. Eur Radiol. 2024;34(4):2512-2523.
22. Yang Y, Zhang J, Li T. The potential risk factors of cortical visual impairment following cerebral angiography: a retrospective study. Ther Clin Risk Manag. 2019;15:1013-1017.