# Accepted Manuscript

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PII: S1878-8750(18)31714-5

DOI: 10.1016/j.wneu.2018.07.234

Reference: WNEU 8799

- To appear in: World Neurosurgery
- Received Date: 26 June 2018

Revised Date: 24 July 2018

Accepted Date: 25 July 2018

Please cite this article as: Tong X, Hu P, Hong T, Li M, Zhang P, Li G, Zhang H, Transient Cortical Blindness Associated with Endovascular Procedures for Intracranial Aneurysms, *World Neurosurgery* (2018), doi: 10.1016/j.wneu.2018.07.234.

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# Transient Cortical Blindness Associated with Endovascular Procedures for Intracranial Aneurysms

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*Correspondence to:* Prof. Guilin Li, Department of Neurosurgery, Xuanwu Hospital, Capital Medical University; International Neuroscience Institute (China-INI), Beijing, China. Email: lgl723@sina.com **Keywords:** transient cortical blindness; cerebral angiography; intracranial aneurysm

# Transient Cortical Blindness Associated with Endovascular Procedures for Intracranial Aneurysms

#### Abstract

We presented three cases of transient cortical blindness secondary to contrast medium toxicity after endovascular procedures for intracranial aneurysms. Two patients noted bilateral blindness 5 or 6 hours respectively of awakening from general anesthesia for aneurysm treatment. The other one noted bilateral blindness during vertebral angiography under local anesthesia. Immediate angiography was performed in one case (Case 1), showing no arterial occlusion. CT was performed in one case that showed brain edema (Case 2). MRI scan was performed in all three cases with abnormal presentation on FLAIR sequences in two patients (Cases 2 and 3). With the use of corticosteroid and intravenous hydration, the cortical blindness resolved within one week in two patients (Cases 1 and 2). The remaining one had only incomplete quadrantanopia three months after blindness onset. Meanwhile, we reviewed the literature and found 12 cases of contrast induced cortical blindness after endovascular procedure for intracranial aneurysms. Based on our experience and the literature reports, we advocate corticosteroid and intravenous hydration for patients with contrast induced cortical blindness after endovascular procedure for intracranial aneurysms.

Keywords: transient cortical blindness; cerebral angiography; intracranial aneurysm

#### Introduction

Transient cortical blindness is a rare but well-known complication after cerebral angiography and neuroendvascular intervention.<sup>1</sup> The reported incidence is 0.3%-1% of patients undergoing vertebral angiography.<sup>2,3</sup> and 2.9% (4/137) of patients undergoing endovascular coiling of posterior circulation aneurysms.<sup>4</sup> The underlying mechanism of this phenomenon has not been proved. Neurotoxicity from the contrast agents may result in cerebral edema and alter neuronal excitability, which may cause such clinical effects as encephalopathy, seizures, cortical blindness and focal neurological deficits. Largely, contrast induced cortical blindness is a self-limiting, transient and reversible phenomenon. However, according to the literature, contrast induced neurologic deficits including cortical blindness may persist after endovascular procedures for intracranial aneurysms. Characteristic CT findings may include abnormal cortical contrast enhancement and oedema, subarachnoid contrast enhancement, and striatal contrast enhancement.<sup>5</sup> However, recent reports on contrast induced cortical blindness have also found normal occipital lobes or no intraparenchymal pathology on CT scan.<sup>4</sup> Most MRI findings may reveal

normal signals or no acute infarction. Some reports may reveal abnormal bright signal intensity on bilateral medial occipital cortex on fluid-attenuated inversion recovery sequences (FLAIR sequences), with or without other cortical abnormality, including small infarctions. In all, current literature support that diagnosis of contrast induced cortical blindness should be based on exclusion of thromboembolic or hemorrhagic complications. It is essential to have a post-treatment angiogram that shows no arterial branch occlusions and a CT or MRI study that shows no acute infarct. Due to the self-limiting nature of contrast induced cortical blindness, there is no consensus on the treatment of this rare complication. Each year, over 2500 patients with intracranial aneurysms receive endovascular treatment or cerebral angiography in our neurosurgical and neurointerventional center. From January 2018 to May 2018, 1120 patients with aneurysms received endovascular embolization (506 patients) or cerebral angiography (614 patients). Cortical blindness occurred in 3 patients, including two patients with posterior circulation aneurysms that received endovascular embolization and 1 patient with internal carotid artery aneurysm that received diagnostic angiography. In this report, we will demonstrate the clinical features, endovascular procedures, image findings, and treatment measures of these three cases with cortical blindness. We will also review the literature of contrast induced cortical blindness after cerebral angiography or endovascular treatment in patients with intracranial aneurysms.

#### **Case illustration**

Case 1. A 64-year-old woman presented with left eye ptosis 1 month before admission to our neurosurgical center. With conservative therapy at local hospital, the symptom of ptosis resolved. The patient had a history of hypertension for 10 years. She suffered from brain infarction three years ago and no sequela was noted on admission, with only small infarctions on head MRI. The patient had no past history of allergy or intoxication and denied any history of sudden headaches associated with intracranial hemorrhage. MRA and CTA at local hospital demonstrated a dissecting aneurysm at the V4 segment of the left vertebral artery. On-admission examination showed that the visual acuity, visual field, optic fundus and ocular movement on both sides were normal. There were no other signs of abnormality. Under local anesthesia, diagnostic DSA showed a dissecting aneurysm at the V4 segment of the left vertebral artery, with the size of 9.2mm×8.1mm×7.7mm (Figure 1 A-B). During the diagnostic angiography procedure, 14 ml of nonionic low-osmolar contrast material -- Ioversol was injected into the vertebral-basilar artery system without development of visual loss or visual field defects. The endovascular treatment procedures were performed under general anesthesia. Lvis

stent-assisted coiling embolization was adopted to occlude the dissecting aneurysm. Ultimately, two Lvis stents and four detachable coils were used. Postprocedural control angiogram demonstrated satisfactory occlusion of the aneurysm, with no signs of artery occlusions (Figure 1 C-D). During the treatment procedure, a total of 300 ml of Ioversol (nonionic contrast) was injected for 1.8 hours into the posterior arterial circulation. The patient was neurologically unchanged immediately after embolization. However, 6 hours of awakening from general anesthesia, the patient noted acute bilateral blindness, having only light perception. The patient could not detect hand-motion in both eyes. Except for that, she had no other signs of neurologic deficits, with normal pupils and ophthalmoscopy findings. Emergent DSA of the left vertebral artery showed no evidence of arterial occlusion or emboli in the vertebral-basilar artery system (Figure 1 E-F). Although no artery occlusion was seen, Tirofiban (Aggrastat) was still used and directly injected into the vertebral-basilar artery system. Compared with the pretreatment brain MRI on FLAIR sequences (Figure 2 A-B), the MRI scan 11 hours after onset of blindness revealed no new infarct on FLAIR sequences (Figure 2 C-D). The patient was treated with Methylprednisolone, 80 mg every 12 hours, tapered for 3 days; subcutaneous injection of low-molecular-weight heparin for 3 days; and intravenous fluid to maintain normal blood pressure. She regained her vision 2 days after blindness-onset. She was discharged 6 days after treatment, with normal visual acuity, visual field and ophthalmoscopy findings.

Case 2. A 53-year-old man presented with sudden acute headache two months before admission. Emergency CT scan at local hospital demonstrated subarachnoid hemorrhage. The patient had a history of hypertension for 6 years. He underwent four times of diagnostic cerebral angiography at the local hospital due to the fact that the results of the first three times of diagnostic angiography were negative. The fourth diagnostic DSA at local hospital showed a small saccular aneurysm at the origin of the P1 segment of the left posterior cerebral artery. The patient denied any symptom during or after diagnostic cerebral angiography at local hospital. Then after two months of conservative therapy and rehabilitation, the patient recovered to his pre-hemorrhage state, with no neurologic deficits. On admission to our neurosurgical center, neurologic examination showed no signs of abnormality. DSA at our neurosurgical center demonstrated a saccular microaneurysm with the size of 1.28mm×1.32mm×0.2 mm at the origin of the left posterior cerebral artery (Figure 3 A-C). Under general anesthesia, 6F guiding catheter was advanced to the V4 segment of the left vertebral artery. Then a microcatheter over a Microvention Traxcess 14 microwire was advanced through the guiding catheter. However, the

microguiding catheter could not be navagated into the intraaneurysmal lumen due to the narrow aneurysmal neck of 0.2 mm. When the microwire was navigated into the aneurysm lumen, the microcatheter was advanced and fixed at the orifice of the aneurysm neck. Then electric coagulation of the aneurysm through a solitaire stent was adopted. The coagulating time lasted for 4 minutes. The treatment was successful and post-treatment control DSA showed disappearance of the aneurysm, with no signs of artery occlusions (Figure 4 A-B). During the treatment procedure, a total of 155 ml of Omnipaque was injected for 2.5 hours into the posterior arterial circulation. The patient showed no signs of neurologic deficits immediately after endovascular treatment. However, five hours of awakening from general anesthesia, the patient noted acute bilateral blindness, having only light perception. The patient was easily agitated. Emergency non-contrast CT scan of the brain showed bilateral brain edema on frontal and occipital lobes (Figure 5 A). FLAR sequences and DWI of MRI one day after blindness onset showed abnormal signal on bilateral frontal and occipital lobes (Figure 5 B-C). The patient was treated with dexamathasone, 5 mg every 8 hours, tapered for 3 days; subcutaneous injection of low-molecular-weight heparin for 3 days; calcium channel blocker for 6 days; and intravenous fluid to maintain normal blood pressure. Diazepam was used to calm the patient when needed. Three days after the blindness onset, the patient slowly regained part of his vision. Five days later, he was discharged with normal vision.

Case 3. A 61-year-old woman, who was neurologically normal, was occasionally found to have an unruptured aneurysm at the supraclinoidal segment of the left internal carotid artery on regular CTA examination. The patient had a history of hypertension for 15 years. On admission, physical examination showed no signs of neurologic deficits. Under local anesthesia, diagnostic cerebral DSA was performed. The patient did not note any discomfort during the diagnostic angiography of the aortic arch, the bilateral internal and external carotid arteries. An aneurysm was found on the left internal carotid artery angiogram (Figure 6 A-D). However, after a total injection of 10 ml Omnipaque into the posterior arterial circulation, the patient noted sudden vision loss with only light perception, companied with left limb numbness. No other neurologic deficits or symptoms were noted. The diagnostic procedure lasted 40 minutes. Then the patient was transferred to neurosurgical ward. The patient was treated with dexamathasone, 5 mg every 8 hours, tapered for 3 days; subcutaneous injection of low-molecular-weight heparin for 3 days; calcium channel blocker for 6 days; and intravenous fluid to maintain normal blood pressure. One day after the initial blindness, the patient slowly regained part of

her visual acuity. Two days later, ophthalmic examination showed left homonymous inferior quadrantanopia. Compared with the prior MRI before angiography (Figure 7 A-C), MRI showed small cerebral infarction of the right occipital lobe (Figure 7 D-F). Limb numbness disappeared two days after blindness-onset. The aneurysm was left untreated due to the initial blindness. Now three months after cerebral angiography, the patient has regained almost all her vision, with only incomplete quadrantanopia that have insignificant effect on her normal life.

#### Literature review

We have reviewed the English language literature for published cases of contrast induced transient cortical blindness after endovascular procedures for intracranial aneurysms (Table 1). Overall, 12 cases were found, with 6 male and 6 female, age ranging from 41 to 74 years old.<sup>4,7-11</sup> In 7 patients, endovascular procedure was performed only in the vertebral basilar system. In 5 patients, the endovascular procedure was performed in both the carotid artery system and the vertebral basilar system. Eight patients underwent endovascular coiling for aneurysms (7 patients with posterior circulation aneurysm and 1 with internal carotid artery aneurysm) and four patients underwent diagnostic angiography (two with anterior circulation aneurysms and two with posterior circulation aneurysms). Of the 12 patients, prior cerebral angiography and/or endovascular embolization was performed in 4 patients, with no contrast induced cortical blindness. All contrast agents were non-ionic contrast materials and the injected volume ranged from 20 to 384 ml (except for 3 patients with unavailable data). The ophthalmic signs were bilateral cortical blindness in 9 patients and visual field defect in 3 patients. As demonstrated in Table 1, 11 out of 12 patients had brain CT scan within hours after blindness-onset, including five patients with normal CT and six with abnormal CT. One patient had only MRI scan, presenting with bilateral occipital abnormality. For the five patients with immediate normal CT presentation, three had abnormal MRI, one had normal MRI and one had no MRI. For the six patients with abnormal CT presentation, two had abnormal MRI, and four had no MRI. Overall, of the 7 patients with available MRI, 6 patients (85.7%) had abnormal MRI presentation in the unilateral or bilateral occipital lobes, especially on the FLAIR sequence. The recovery time ranged from 1 day to 1 month, most within one week. In two patients, visual field defects persisted at 3 weeks and 1 month respectively after blindness-onset.

#### Discussion

We reported three cases of transient cortical blindness after cerebral angiography or endovascular

embolization for intracranial aneurysms. We also reviewed the literature of patients who experienced contrast induced cortical blindness after endovascular procedures for intracranial aneurysms. Regarding literature review, we only included patients with intracranial aneurysms and excluded cases with other pathologies. We found that transient cortical blindness is more likely to be associated with vertebral angiography or endovascular manipulation in the posterior arterial circulation. The exact definition of contrast induced cortical blindness, the underlying pathologic mechanism and the treatment strategies are still making neurosurgeons and neuroradiologists in a dilemma.

#### Diagnosis of contrast induced cortical blindness

In the literature, contrast induced cortical blindness are reported to be self-limiting, resolving within hours to days. However, diagnosis of contrast induced cortical blindness is challenging. It is essential to exclude embolic, hemorrhagic and hemodynamic complications, which may have a similar clinical presentation. Image findings are essential for the diagnosis of contrast induced cortical blindness: with no arterial branch occlusions on post-treatment angiograms and no acute infarct on CT or MRI study. Based on the above-mentioned, diagnosis of contrast induced cortical blindness includes transient cortical blindness and typical CT or MRI findings. Contrast induced cortical blindness is characterized by an acute vision loss after endovascular angiography or procedures. Ophthalmic examination may reveal total blindness, or only light perception, or unilateral or bilateral visual field defects. The pupillary response to light and optic fundus with opthalmoscopy are normal. Regarding our three cases, two patients experienced acute cortical blindness 5 or 6 hours respectively after awakening from general anesthesia for posterior arterial circulation aneurysm embolization. One patient noted bilateral cortical blindness during cerebral angiography under local anesthesia. Except for the patient undergoing electronic coagulation for P1 aneurysm, the other two patients had only cortical blindness, without other presentation.

Typical CT findings include abnormal cortical contrast enhancement and edema, subarachnoid contrast enhancement, striatal contrast enhancement, if the CT is performed soon after presentation.<sup>4</sup> Patient may present with small infarction in the occipital lobe on CT scan several days after blindness presentation.<sup>10</sup> For the 12 cases reviewed in the literature, most had CT scan within hours after blindness onset (except for one patient). Half had normal CT scan and half had abnormal CT presentation in the bilateral or unilateral occipital lobes. Overall, of the 7 patients with available MRI, 6 patients (85.7%) had abnormal MRI presentation in the unilateral or bilateral occipital lobes, especially

on the FLAIR sequence. Patient may even presented with small infarct in the occipital lobe and experienced persistent visual field defects. For our three cases, patients presented with abnormal MRI signals in unilateral or bilateral occipital lobes in two patients and one patient had no change on FLAIR sequences, compared with the pretreatment MRI. Based on our experience and previous findings, patient with contrast induced cortical blindness may present with normal CT or abnormal CT, depending on the timing of CT scanning. However, most patients presented with abnormal MRI signal in bilateral or unilateral occipital lobes, especially on FlAIR or DWI sequences. From this point of view, we could not decide whether the abnormal signal was caused by the neurotoxicity of contrast agent or microembolic agent, especially for Case 3 in our series.

#### Risk factors and underlying mechanism of contrast induced cortical blindness

The mechanism and causes of neurotoxicity remains controversial. Some studies found that transfer of contrast material increases if the blood-brain barrier is disrupted or if contrast material is overdosed or applied intra-arterially.<sup>12-16</sup> However, the occurrence of cortical blindness is not necessarily associated with the amount or the infusion rate of contrast injected. There was also no clear correlation between the occurrence of cortical blindness and the duration of endovascular procedures. According to literature reviewed, the amount of contrast injected ranged from 20 to 384 ml. For our three cases, the volume of contrast injected into the posterior arterial circulation ranged from 10 ml to 314 ml. And the duration of endovascular procedures ranged from 40 minutes to 2.5 hours. Prior cerebral angiograms or endovascular procedures were performed in five of the 12 cases in the literature and in one of our three cases, and did not appear to result in cortical blindness. We agree with the assumption that, even if the total amount of contrast materials is not excessive, repeated injections of contrast materials into a single vessel may contribute to blood-brain barrier breakdown, resulting in contrast neurotoxicity and cortical blindness.<sup>17-18</sup> Studies suggest that contrast media penetrates the blood-brain barrier as a function of dosage, contact time, concentration of anions in the material, and lipophilic characteristics.<sup>4</sup> According to the literature, the risk factors for patients with contrast induced cortical blindness include hypertension and renal failure. All our three cases had a past history of hypertension. Contrast induced cortical blindness has been assumed to be an idiosyncratic reaction, which makes it difficult to avoid contrast induced encephalopathy. Due to the current limited cases in the literature, the underlying mechanism needs further investigation.

#### **Treatment of cortical blindness**

In the literature, contrast induced cortical blindness is a self-limiting process. For the 12 patients with cortical blindness, 10 patients recovered to their pretreatment state within one month and most recovered within one week after blindness-onset. Two patients present with persistent visual field defect at the last follow-up visit. For our three cases, all recovered within one week. Only case 1 underwent cerebral angiography immediately after blindness-onset. Although there was no evidence of vessel occlusion or emboli in the posterior cerebral arteries, Tirofiban (Aggrastat) was still injected into the posterior circulation. It is hard for the doctor to make sure that the blindness was caused only by contrast neurotoxicity, not by microemboli. Our treatment strategy is similar to that of Niimi et al., who used abciximab (ReoPro) intra-arterially injected into the posterior circulation during emergent cerebral angiography even there was no arterial occlusion. In the literature, there is no consensus on the effective therapy for cortical blindness due to its self-limiting nature. However, some authors have advocated the use of aggressive intravenous hydration with a short course of corticosteroids.<sup>4,6,8</sup> All of our three cases received corticosteroids, subcutaneous injection of low-molecular-weight heparin and intravenous hydration. Maybe some or all of our treatment strategies seem to be unneeded intervention for contrast induced cortical blindness. However, even for confirmed contrast induced cortical blindness, no doctor can make sure that the cortical blindness can resolve soon and does not need further treatment. Actually, in most cases, the diagnosis of contrast induced cortical blindness is mostly confirmed during treatment process or after recovery from blindness. Steroids is used to stabilize the blood-brain barrier and thus reduce the the theoretic vasogenic edema.<sup>19</sup>

Therefore, we advocate intravenous hydration and use of steroids for patients with contrast induced cortical blindness. Even if emergency cerebral angiograms showed no evidence of vessel occlusion or emboli in the posterior cerebral arteries, injection of glycoprotein IIb/IIIa inhibitors such as Abciximab (ReoPro) or Tirofiban (Aggrastat) may help to prevent platelet aggregation and thrombus formation.

#### Conclusion

The diagnosis of contrast induced cortical blindness is challenging. CT scan may be normal or abnormal in unilateral or bilateral occipital lobes. Most of these patients may demonstrate abnormal MRI, even with small infarction in the occipital lobes. The underlying pathologic mechanism remains unclear. We advocate immediate cerebral angiogram for such cases to exclude arterial branch occlusions. CT and MRI scan are helpful for diagnosis of this rare complication. We advocate the use

of corticosteroid and intravenous hydration for recovery of cortical blindness.

#### **Disclosure and Acknowledgement**

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper. Author contributions to the study and manuscript preparation include the following. Conception and design: all authors. Acquisition of data: Tong, Hu. Analysis and interpretation of data: Tong, Hu. Drafting the article: Tong. Critically revising the article: all authors. Reviewing submitted version of manuscript: all authors. Approving the final version of the manuscript on behalf of all authors: Peng Zhang. Administrative/technical/material support: Guilin Li and Peng Zhang. Study supervision: Guilin Li and Peng Zhang.

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#### **Figure legends**

Figure 1. Case 1. A-B: diagnostic DSA showing a dissecting aneurysm at the V4 segment of the left vertebral artery; C-D: post-treatment contrast angiography showing complete occlusion of the aneurysm and no arterial occlusion; E-F: immediate DSA after blindness onset showing no occlusion of the arterial branches of posterior circulation.

Figure 2. Case 1. Fluid-attenuated inversion recovery sequences (FLAIR) of MRI before endovascular treatment (A-C) and 11 hours after blindness onset (D-F). The covered areas of abnormal signals were similar on FLAIR sequences at the two different periods. No new infarction being demonstrated after blindness onset.

Figure 3. Case 2. A-B: 3D-DSA showing a microaneurysm at the origin of the P1 segment of the left posterior cerebral artery. C: lateral view of the vertebral angiography showing the microaneurysm. Figure 4. Case 2. A-B: post-treatment DSA showing complete occlusion of the aneurysm with no arterial occlusion.

Figure 5. Case 2. A: emergency CT ten hours after blindness onset showing brain edema on bilateral frontal and occipital lobes; Case B-C: FLAIR (B) and DWI sequences showing abnormal high signals

on bilateral occipital lobes.

Figure 6. Case 3. A-B: diagnostic DSA showing an aneurysm at the left internal carotid artery. C-D: the 3D imaging of the left internal carotid artery aneurysm.

Figure 7. Case 3. compared with the pre-treatment MRI on FLAIR (A), DWI (B) and ADC (C) sequences, the MRI two days after blindness onset showing new infarct at the right occipital lobe, with abnormal signal on FLAIR (D), DWI (E) and ADC (F) sequences.

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References	Sex/	Arteriogra	Indication for study	PA	Contrast agent	Contrast	Volum	Presentation	CT/MRI brain	Clinical
	Age	phy			class	agent	e (ml)		region involved	resolution
Lantos	F/68	CA	PCOM AN	No	Non-ionic	Iohexol	24	Cortical blindness;	CT: bilateral	6 d
$(1989)^{6}$	89) <sup>6</sup> VA (diagnos		(diagnostic)	agnostic)		monomer		confusion; amnesia	occipital	
					low osmolar		2			
Saigal et al.	F/45	CA	Coiling right SCA	No	Non-ionic	Omnipaque	NA	Cortical blindness;	Normal CT;	7 d
$(2004)^7$		VA	AN		monomer			confusion	MR: bilateral	
					high osmolar		$\sim$		occipital	
	F/74	CA	basilar apex AN	No	Non-ionic	Omnipaque	N/A	Cortical blindness;	CT: left	1 d
		VA	(diagnostic)		monomer			confusion	parieto-occipital;	
					high osmolar				MR: left occipital	
	F/73	CA	Left supraclinoid	No	Non-ionic	Omnipaque	N/A	Cortical blindness	Normal CT;	5 d
		VA	AN (diagnostic)		monomer	Y			MR: bilateral	
					high osmolar				occipital	
Shinoda et	F/62	VA	Coiling PICA AN	Yes	Non-ionic	Not	150	Cortical blindness;	CT: bilateral	Persistent
al. (2004) <sup>8</sup>						specified		confusion	occipital, basal	VFD at 3
									ganglia, frontal	weeks
									MR: bilateral	
									occipital	
Niimi et al. (2008) <sup>4</sup>	F/54	VA	Coiling basilar	Yes	Non-ionic	Not	62	Cortical blindness with	MR: bilateral	1 month
			apex AN			specified		only light perception	occipital	
	M/41	VA	Coiling SCA AN	No	Non-ionic	Not	297	Cortical blindness;	CT: right parietal	Mild VFD
						specified		agitation		at 1 month
	M/46	õ VA	Coiling basilar	Yes	Non-ionic	Not	225	Right homonymous	Normal CT	1 month
			apex AN			specified		hemianopia	Normal MRNormal	

Table 1. Literature reported contrast-induced cortical blindness following arteriography and/or embolization in patients with intracranial aneurysm

	M/47	VA	Coiling	basilar	No	Non-ionic	Not	384	Right homonymous	СТ	7 d
			apex AN				specified		hemianopia		
Guimaraens	M/51	VA	Coiling rig	ht ICA	No	Non-ionic	Iopromide	300	Left VFD& hemiparesis;	CT: right	2 d
et al.			AN		monomer			right gaze deviation	frontoparietal-Occi		
$(2010)^9$						low osmolar			0 Y	pital	
Shah et al.	M/58	CA	Coiling rig	tht PICA	Yes	Non-ionic	Iopromide	350	Cortical blindness	CT: cortical	10 d
$(2013)^{10}$		VA	AN			monomer				hyperdensity in the	
						low osmolar		6		territory of PCAs;	
								$\sim$		Vasogenic oedema	
							- K			associated with	
										small occipital	
										infarcts on CT on	
										day 3	
Lo, et al.	M/57	VA	Post-embol	lization	Yes	Non-ionic	Omnipaque	20	Bilateral cortical	Normal CT;	1d
$(2015)^{11}$			check for F	PICA AN		monomer			blindness	MR: abnormal	
					high osmolar				bilateral		
						7			parieto-occipital		
									lobes on FLAIR		
						X				imaging	

\*F: female; M: male; CA: carotid artery; VA: vertebral artery; AN: aneurysm; SCA: superior cerebellar artery; PICA: posterior inferior cerebellar artery; PA: previous angiography; VFD: visual field defect; PCAs: posterior cerebral arteries.

**V** 















## Highlights

Transient cortical blindness is a rare complication after cerebral angiography.

There is no consensus on the treatment of transient cortical blindness.

We advocate the use of corticosteroid and intravenous hydration.

#### Abbreviations

CT: computed tomography

DSA: digital subtraction angiography

DWI: diffusion weighted image

FLAIR: fluid-attenuated inversion recovery sequences

MRI: magnetic resonance imaging