Radiology Case Reports 15 (2020) 1159–1163

Available online at www.sciencedirect.com

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journal homepage: www.elsevier.com/locate/radcr

Case Report

Transient global amnesia following carotid artery stenting: A case report ☆,☆☆

Byung Hoon Lee, MD[1,*]

Department of Radiology, Inje University Ilsan Paik Hospital, 170 Juhwaro, Ilsanseogu, Goyang, Gyeonggido, South Korea

ARTICLE INFO

Article history:
Received 7 April 2020
Revised 12 May 2020
Accepted 14 May 2020

Keywords:
Transient global amnesia
Carotid stenosis
Carotid artery stenting
Stroke

ABSTRACT

Transient global amnesia (TGA) is a neurological disorder characterized by sudden onset of anterograde and retrograde amnesia. TGA following angiography is rare and its possible etiologies have not been determined definitively. Herein, I report a case of TGA after carotid artery stenting with DWI findings, which, to date, has not been reported. The patient was a 71-year-old man who developed TGA following carotid artery stenting. Diffusion-weighted imaging demonstrated a focal restricted diffusion lesion in the left medial temporal lobe. Left carotid artery stenting was performed via the common femoral artery, and the procedural course was uneventful. However, the patient suffered an amnestic event 1 hour after the procedure. DWI performed 1 hour after amnesia onset revealed a small punctate restricted diffusion lesion in the left hippocampus. One day later, his amnesia spontaneously resolved. Although TGA after endovascular procedure is rare and usually has a favorable prognosis, clinicians and interventionists should be aware of this rare complication.

Introduction

Transient global amnesia (TGA) is a benign clinical syndrome characterized by abrupt onset of anterograde amnesia and a variable degree of retrograde amnesia that spontaneously resolves within 24 hours [1,2]. Although many possible causes have been suggested, including ischemia, epilepsy, migraine, emotional stress, and neurotoxicity of contrast media, the underlying etiology of TGA remains unclear [1,3-5]. Herein, I present a rare case of TGA after cerebral angiography and endovascular procedure. To the best of our knowledge, this is the first report describing TGA with diffusion-weighted imaging (DWI) findings after carotid artery stenting.

☆ Competing Interest: The author declares conflict of interest.
☆☆ Funding: There was no funding support for the research.
* Corresponding author.
E-mail address: hoonbeer@hanmail.net
[1] Authors' contribution: Byung Hoon Lee-design of the study, patient enrollment, data acquisition, data analysis/interpretation, and manuscript preparation.
https://doi.org/10.1016/j.radcr.2020.05.028

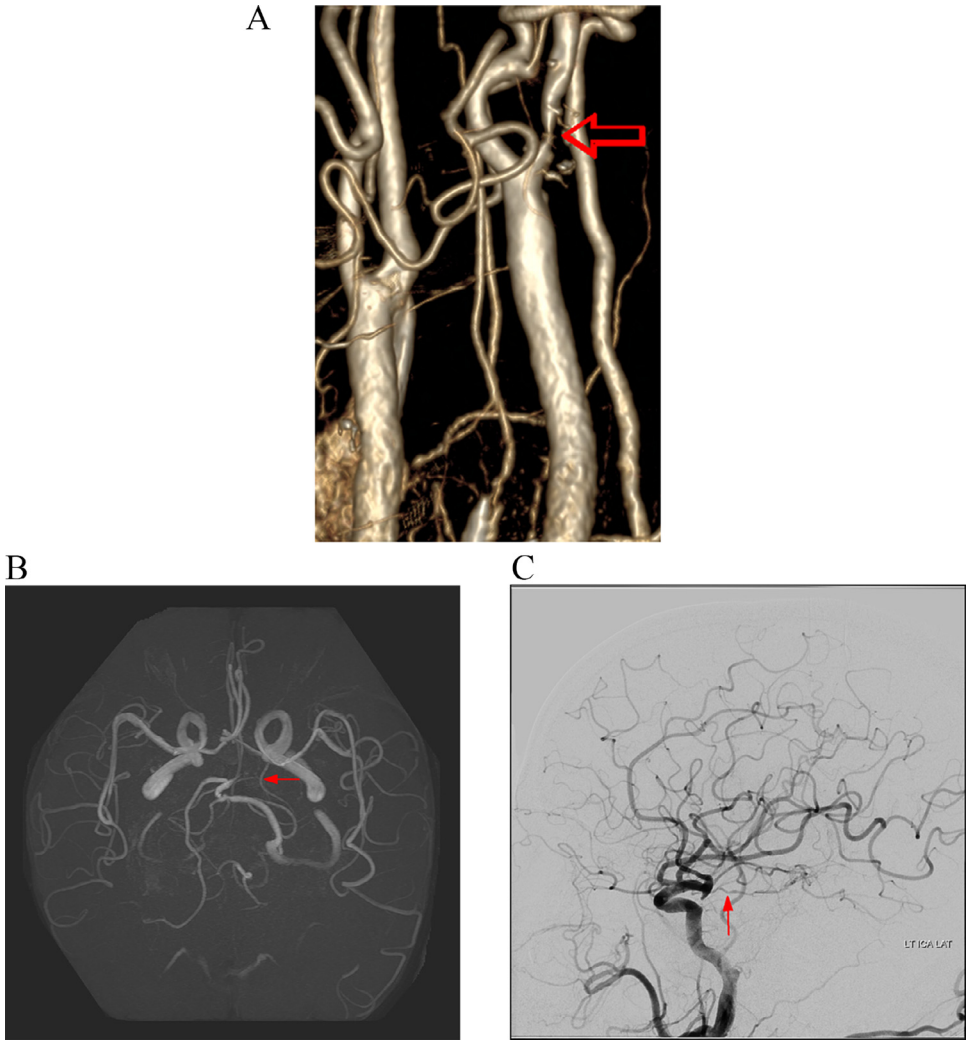

Fig. 1 – A 71-year-old man with transient global amnesia following carotid artery stenting. (A) Three-dimensional volume-rendered computed tomography angiography of the neck showed severe stenosis of the proximal left internal carotid artery (open arrow). (B) Time-of-flight magnetic resonance angiogram maximum intensity projection image showed fetal origin of the left posterior cerebral artery with diffuse luminal narrowing (arrow). (C) Left carotid angiogram after carotid stent implantation in the lateral projection showed no definite occlusion of the left posterior cerebral artery (arrow). (D and E) Coronal diffusion-weighted image (D) and the corresponding apparent diffusion coefficient map (E) taken 1 hour after the amnestic event showed restricted water diffusion in the left hippocampus (arrowheads). The area that contained restricted diffusion was zoomed.

Case report

A 71-year-old man was admitted to our emergency department with sudden painless loss of vision in his left eye of 14-hour duration. His past medical history was relevant for coronary artery bypass surgery 7 years ago, hypertension, and hyperlipidemia. The patient was taking acetylsalicylic acid and a statin.

The diagnosis of branch retinal artery occlusion was made by fundus examination on admission. Shortly thereafter, computed tomography angiography was performed to evaluate for carotid artery stenosis or other vascular abnormalities. Computed tomography angiography revealed severe stenosis of the proximal left internal carotid artery (ICA; Fig. 1A). The left posterior cerebral artery (PCA) originated from the left ICA with multifocal stenosis and luminal narrowing. Initial magnetic resonance (MR) angiography and MR imaging, including DWI, were performed 12 hours after admission. DWI revealed thromboembolic infarction in the left frontal and left occipital cortex. No abnormal signal intensity was detected in the hippocampus. The MR angiogram demonstrated fetal origin of the left PCA with diffuse luminal narrowing (Fig. 1B). Five days after admission, digital subtraction cerebral angiography was performed and showed severe stenosis of the proximal left ICA and luminal narrowing of the left PCA.

Left carotid artery stenting was performed via the common femoral artery using the nonionic contrast medium

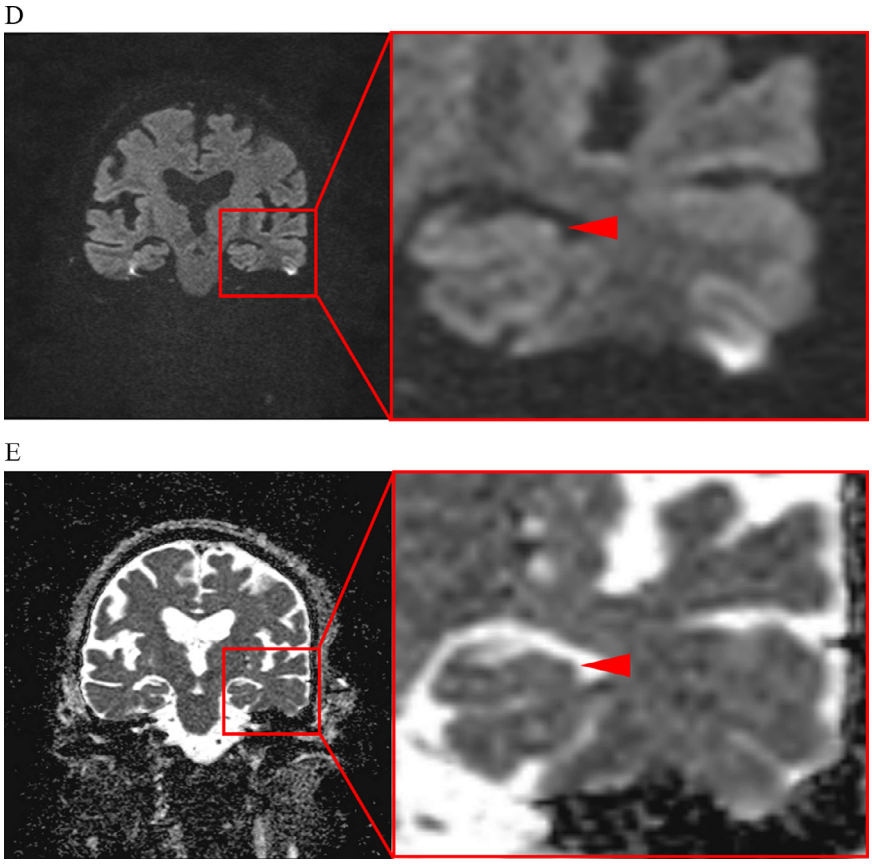

Fig. 1 – Continued

iopamidol. Heparinized saline was used for the flushes during the procedure. Immediately after guiding catheter placement, a bolus of heparin was administered. Cerebral angiography after carotid stent implantation showed no definite occlusion of the left PCA (Fig. 1C). The procedural course was uneventful. However, 1 hour after completion of the endovascular intervention, the patient was unable to remember anything from the time of his admission and repeatedly asked where he was and why he was here. He did not even remember that he had left eye blindness and that he underwent carotid artery stenting. During the amnestic event, however, the patient was alert. DWI performed 1 hour after the amnesia onset revealed a new small punctate restricted diffusion lesion in the left hippocampus (Fig. 1D and E). One day later, his amnesia completely resolved.

Discussion

TGA is a sudden-onset clinical syndrome characterized by loss of memory for recent events without other cognitive impairments or focal neurological deficits that spontaneously resolves within 24 hours [1,5,6]. Our patient was diagnosed with TGA based on the clinical presentation.

TGA following conventional angiography is a rarely reported and possibly underdiagnosed complication of cerebral angiography and endovascular intervention [6]. Most previously reported cases of TGA were following angioplasty or angiography of the posterior circulation [4,6-8]. There are limited number of case reports about TGA after anterior circulation angiography and none include cerebral angiography images or DWI [9,10]. In the present case, left common and ICA angiography was performed, and we demonstrated the angiographic findings and the focal signal abnormality in the left hippocampus seen on DWI.

Although the pathophysiology of TGA is still largely unknown, a variety of possible mechanisms have been suggested, including cerebral ischemia, epilepsy, migraine, and psychological factors [1,4,5]. DWI has become a powerful sequence for the evaluation of acute ischemic stroke and can provide etiologic evidences [11]. Enzinger et al. suggested that DWI abnormalities should be considered a nonspecific finding with several possible underlying mechanisms, probably leading to focal energy failure [11]. However, some investigators suggested that TGA is a reflection of focal acute ischemia as a possible etiology because the punctate restricted diffusion lesions in the medial temporal region showed signal characteristics consistent with cerebral ischemia [12]. Because the maximum level of detection of hippocampal DWI lesions is within 48-72 hours after amnesia onset, early imaging might show a weak signal change or not detect the responsible lesion [12]. In this case, DWI performed 2 hours after carotid artery stenting showed a subtle focal restricted

diffusion area of the left hippocampus, suggestive of acute ischemia.

TGA is a very rare complication of cerebral angiography and several possible etiological mechanisms have been suggested, including ischemia due to embolic occlusion, transient reduction in regional hippocampal blood flow, vasospasm, arterial dissection, and neurotoxic effect of the contrast material [5]. In this case, among the possible etiologies for TGA development are distal thromboembolism, transient hypoperfusion, and neurotoxic effect of contrast media.

One of the proposed causes of TGA following cerebral angiography is ischemia resulting from atherosclerotic plaque, thrombus formation with distal embolism, or particles in the contrast medium [13]. Duan et al. reported that 5 of 4360 patients (0.11%) experienced TGA following cerebral angiography, and only 1 patient showed small acute infarction in the hippocampus on DWI after vertebral artery angioplasty [14]. They suggested that ischemic embolism might be responsible for TGA after angiography because it only occurred in the patients who underwent cerebral and cardiac angiography, but not in those who underwent peripheral angiography beneath the aortic arch [14]. Kim et al. reported TGA following vertebral artery stenting and as PCA occlusion was detected on MR angiography immediately after symptom onset, arterial embolization was suggested as the possible mechanism [4]. However, they did not detect the responsible diffusion abnormality. In this case, thromboembolism might have been induced during the selective catheterization or carotid artery stenting procedure. In addition, a focal restricted diffusion lesion was detected in the left medial temporal lobe on DWI performed 1 hour after amnesia onset. Therefore, in our case, distal embolism of the PCA is one of the possible mechanisms of TGA because the left PCA originated from the left ICA. However, no definite left PCA occlusion was identified on angiography after carotid stent implantation.

Angiography and endovascular procedure may cause arterial injury or induce vasospasm. In the present case, there was no arterial vasospasm or reduced flow during the endovascular procedure. The occurrence of vasospasm affecting only the PCA is unlikely. The underlying severe stenosis of the proximal left ICA or the placement of a distal embolic protection device might have caused decreased arterial flow and transient hypoperfusion of the left PCA territory, leading to hippocampal hypoperfusion. Therefore, hypoperfusion is considered as the cause of TGA after carotid stenting in this case.

Contrast media can be a causative factor of TGA after cerebral angiography [5]. With the use of nonionic contrast media, the rate of angiography-related TGA significantly decreased [5]. In the present case, carotid stenting was performed using the nonionic contrast medium iopamidol. However, impairment of the blood-brain barrier after ischemic stroke may permit entry of contrast media into the brain parenchyma [9,13]. The risk of a neurotoxic effect of contrast media can be exacerbated when the patient has suffered an ischemic stroke before the cerebral angiography procedure because of preexisting damage in the blood-brain barrier [5,6]. Our patient experienced border zone infarcts and branch retinal artery occlusion due to severe stenosis of the proximal left ICA. Therefore, if the blood-brain barrier was already disrupted in the left hippocampus, the contrast agent could have exerted direct neurotoxicity. Hence, in this case, the amnestic event may have been associated with a previously disrupted blood-brain barrier, and the direct neurotoxic effect of the contrast medium can be suggested as a possible cause of TGA after the endovascular procedure.

In conclusion, TGA following carotid artery stenting is extremely rare. Although its definite etiology remains unclear, it may be a combination of multiple factors, such as ischemia due to emboli, hypoperfusion, or contrast medium toxicity. Albeit usually a benign condition, this rare complication should be considered after cerebral angiography of the anterior circulation in patients with fetal origin of the PCA after excluding other causes of amnesia.

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