Paracentral acute middle maculopathy following internal carotid artery stenting due to carotid artery dissection: A case report

Ümit Yaşar Güleser[1], Rashim Thakur[2], Cem Kesim[2], Murat Hasanreisoglu[2]

ABSTRACT

Carotid artery dissection (CAD) is an important cause of cerebral and retinal ischemia especially in young adults. We present a case of paracentral acute middle maculopathy (PAMM) following internal carotid artery stenting (ICAS) in the setting of CAD. A 41-year-old female patient underwent ICAS procedure with the diagnosis of internal CAD after she complained of ptosis in her left eye (OS), blurred vision, numbness in the tongue and headache on the left side two weeks ago. After ICAS, she complained of blackness in the visual field in his left eye, describing a paracentral scotoma. Fundus examination of OS showed four whitish yellow subtle intraretinal lesions superonasal to fovea and along the vascular arcades. Fundus autofluorescence showed two hypofluorescent lesions, spectral domain optical coherence tomography showed hyper-reflective band in the inner and middle retina layers, fundus fluorescein angiography depicted hypofluorescent lesions, optical coherence tomography angiography demonstrated flow deficits in superficial capillary plexus and deep retina capillary plexus consistent with lesion areas in OS. All investigations and ophthalmological examination of her right eye were normal. Based on the history and findings, she was diagnosed with PAMM. Our case underscores the importance to recognize retinal complications in the setting of carotid dissection and its related interventional procedures.

Keywords: Paracentral acute middle maculopathy, internal carotid stenting, carotid dissection, angioplasty

INTRODUCTION

Paracentral acute middle maculopathy (PAMM) is a clinical entity characterized by hyper-reflective band in the inner nuclear layer (INL) and outer plexiform layer (OPL) in spectral domain optical coherence tomography (SD-OCT) and typically evolves into atrophy of these layers in the later stages.[1-3] The underlying mechanism of PAMM is thought to be mainly due to ischemia of the middle retinal layer.[4] With the advent of optical coherence tomography angiography (OCTA), PAMM presents as a flow deficit in the deep capillary plexus.[5]

Internal carotid artery dissection (ICAD) is an important cause of stroke in young patients, accounting for approximately 20% of stroke in patients less than 45 years of age.[6-8] ICAD results from intimal tearing and passage of blood into the false lumen, causing vessel narrowing (subintimal dissection), aneurysm formation (sub adventitia dissection) or both.[9] Conservative treatment with medical therapy may be used in patients who remain asymptomatic.[6-8] However, endovascular treatment with angioplasty and stent placement is now the treatment of choice in symptomatic dissections.[10]

This case report presents an unusual occurrence of PAMM following internal carotid artery stenting (ICAS) in the context of ICAD. We have employed OCTA to depict capillary dropout areas in both the superficial capillary plexus (SCP) and deep retina capillary plexus (DCP), subsequently illustrating the time dependent reperfusion of these plexus with each follow up.

1 Koç Üniversitesi Hastanesi, Oftalmoloji Departmanı, İstanbul, Turkey

2 Koç Üniversitesi Tıp Fakültesi, Göz Hastalıkları Anabilim Dalı, İstanbul, Turkey

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Correspondence author:
Murat Hasanreisoglu
Email: rmurat95@yahoo.com

CASE REPORT

A 41-year-old female patient admitted to the emergency department with left ptosis and blurred vision 5 days ago, which resolved within 24 hours, accompanied by left-sided headache and numbness in the tongue, which developed the day before. Her past medical history was peripheral venous deficiency in lower extremities. She underwent endovascular truncal ablation 4 years ago. She is a non-smoker with no systemic medication. Cranial diffusion MRI was requested from the patient, who had no findings in her systemic examination, and it was found to be normal. Transient ischemic attack (TIA) was considered by the neurologist cranial and cervical magnetic resonance angiography (MRA) was performed and a progressed carotid dissection with aneurysm was diagnosed on left internal carotid artery (ICA). The patient was started on apixaban. Digital subtraction angiography (DSA) was performed which showed dissection extending from ½ distal segment of left internal carotid artery to vertical part of petrous segment which had caused approximately 75% stenosis at narrowest point along with a 6 mm pseudo-aneurysm. On the following day, percutaneous transluminal angioplasty, carotid stenting and aneurysm-dissection embolization were performed, which revealed stenosis and pseudoaneurysm on the background of ICAD. Following 3 days from the procedure, she complained of a black spot in the visual field of her left eye (OS), describing paracentral scotoma. She was consulted to ophthalmology department for further evaluation.

On ophthalmologic examination, best corrected visual acuity (BCVA) was 20/20 on Snellen's chart in her both eyes. Biomicroscopic anterior segment examination, intraocular pressure and pupillary reactions were normal in her both eyes. On dilated fundus examination, right eye (OD) was normal (Fig. 1A). Fundus of OS showed a cotton wool spot (CWS) near the superior arcuate and four whitish yellow intraretinal lesions, the largest of which was half a disc in diameter and was located superonasal to the fovea (Fig. 1B). Fundus autofluorescence (FAF) of OS demonstrated two hypofluorescent areas corresponding to the lesions (Fig. 1D). Spectral domain optical coherence tomography (SD-OCT) (Spectralis HRA+OCT MultiColor,

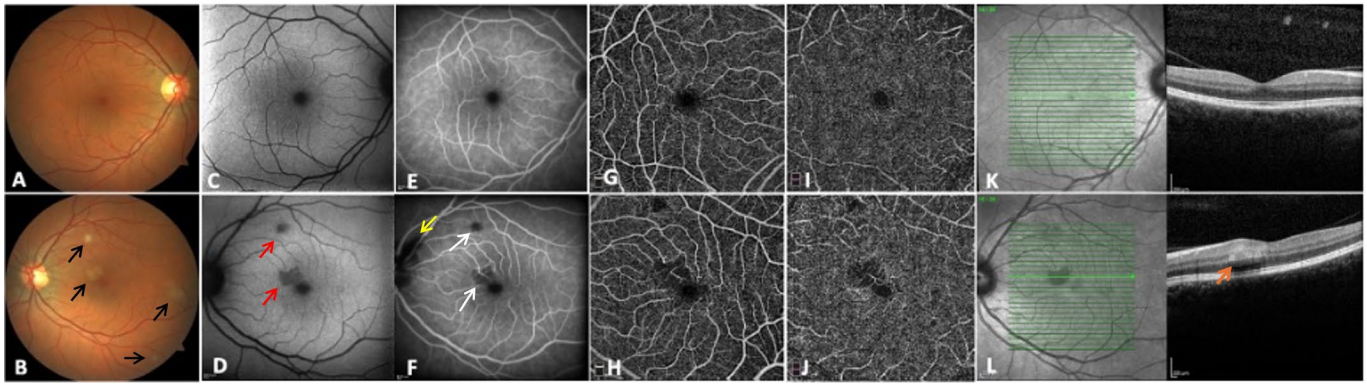

Figure 1: *Multimodal imaging at diagnosis. Color fundus photograph (CFP), Fundus autofluorescence (FAF), Fundus Fluorescein Angiography (FA), Spectral domain optical coherence tomography angiography (OCTA) in superficial retinal capillary plexus (SCP) and deep retinal capillary plexus (DCP) and Spectral domain optical coherence tomography (SD-OCT) of her right eye were normal (Fig. 1A, 1C, 1E, 1G, 1I, and 1K, respectively). 1B: CFP of her left eye (OS) showed four whitish yellow subtle intraretinal lesions located superonasal to fovea, along the inferior arcuate vessels temporally, below the inferior arcuate vessels temporally and below the superior arcuate vessels respectively (black arrows). 1D: FAF of OS demonstrated two hypofluorescent areas corresponding lesions on CFP (red arrows). 1F: FA of OS revealed hypofluorescent lesions corresponding to the lesions (white arrows) and peripapillary retinal hemorrhage (yellow arrow) that developed 2 days later. OCTA of OS revealed flow deficits in SCP and DCP on lesion areas. (1H and 1J, respectively). 1L: SD-OCT of OS showed hyper-reflective band in inner and middle retina (extending from ganglion cell layer to outer plexiform layer) (orange arrow) corresponding to the lesion at macula.*

Heidelberg Engineering, Heidelberg, Germany) of OS revealed a hyper-reflective band in inner and middle retina extending from ganglion cell layer to outer plexiform layer, corresponding to the lesion in the macula (Fig. 1L). After 2 days, retinal hemorrhage was observed next to the optic disc, in addition to the existing lesions. Following the diagnosis, enoxaparin sodium was added to the patient's treatment by interventional radiology. Hemorrhage was thought to be due to coagulopathy. Fundus fluorescein angiography (FA) (Spectralis HRA+OCT MultiColor, Heidelberg Engineering, Heidelberg, Germany) could be performed after 2 days, which depicted hypofluorescent areas in OS with irregular contours corresponding to the lesions (Fig. 1F). OCTA (Angiovue/RTVue-XR Avanti, Optovue Inc., Fremont, USA) of OS demonstrated reduced flow signal in superficial retinal capillary plexus (SCP) and deep retinal capillary plexus (DCP) (Fig. 1H and 1J, respectively) corresponding to the lesions. Paracentral scotoma was detected in OS on visual field test (24-2 SITA-Standard, Humphrey HFA II-i 750i, Carl Zeiss Meditec, Inc., Dublin, CA) (Fig. 2). All investigations were normal in OD (Fig. 1).

The patient was diagnosed with PAMM in OS. She was followed for six months without any additional treatment for PAMM. Lesions in the fundus and FAF disappeared during follow-ups (Fig. 3A and 3B). SD-OCT of OS resulted in atrophy in the paracentral area in 2 months (Fig. 3E2) and 6 months (Fig. 3E3). OCTA demonstrated improvement of flow signal from lesion areas in SCP and DCP compared to presentation (Fig. C and D, respectively). The capillary reperfusion started after one and half month of the presentation and it improved with each follow up. Over the follow up visits we observed a prominent recovery of the SCP and DCP flow signals within the lesion. However, few residual flow deficits persisted at the end of 6 months from the presentation (Fig. C3 and D3). BCVA remained stable as 20/20. The black spot perception of the patient partially improved, however few scotoma persisted on the follow-up visual field test.

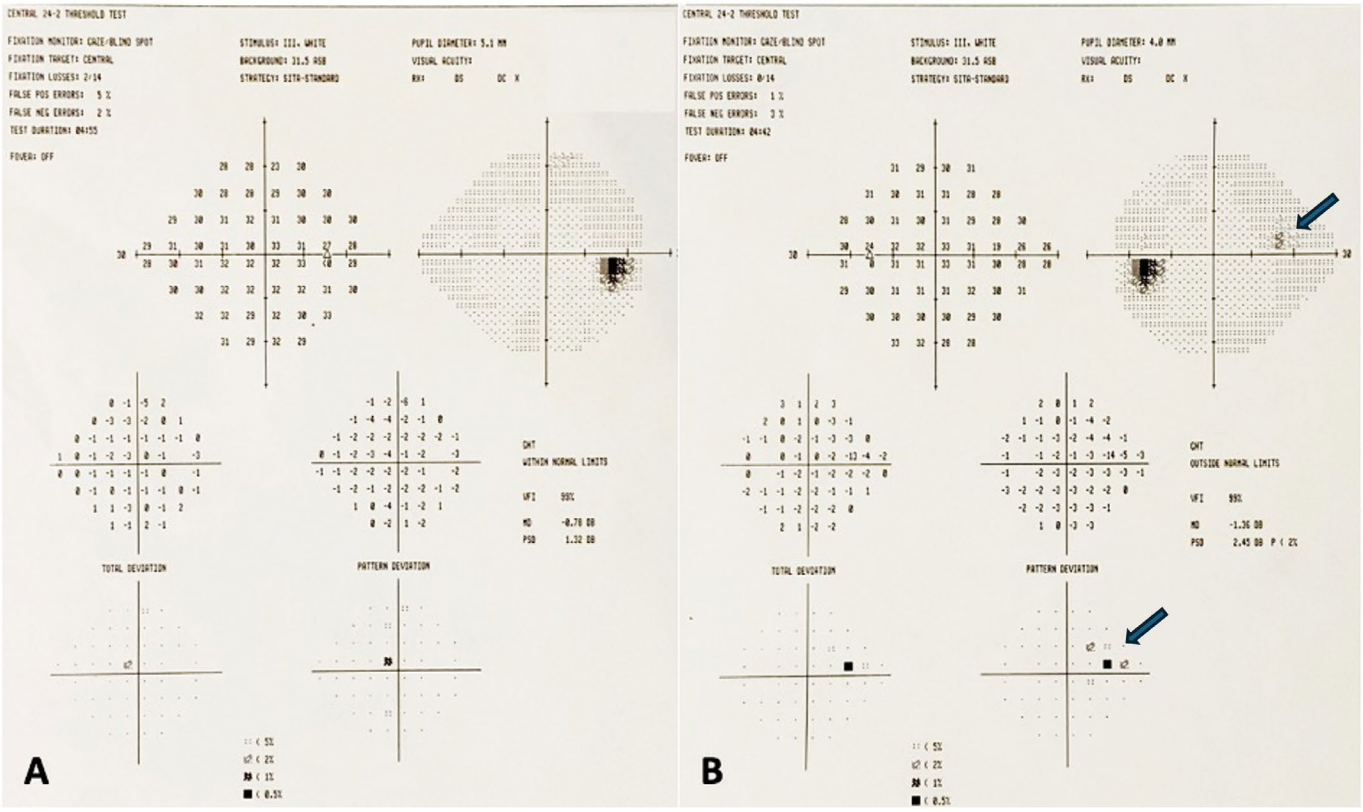

Figure 2: *Visual field test (Humphrey automated perimetry 24-2) was unremarkable in her right eye (A) and demonstrated paracentral scotoma (blue arrows) in her left eye (B) consistent with her complaint at first presentation.*

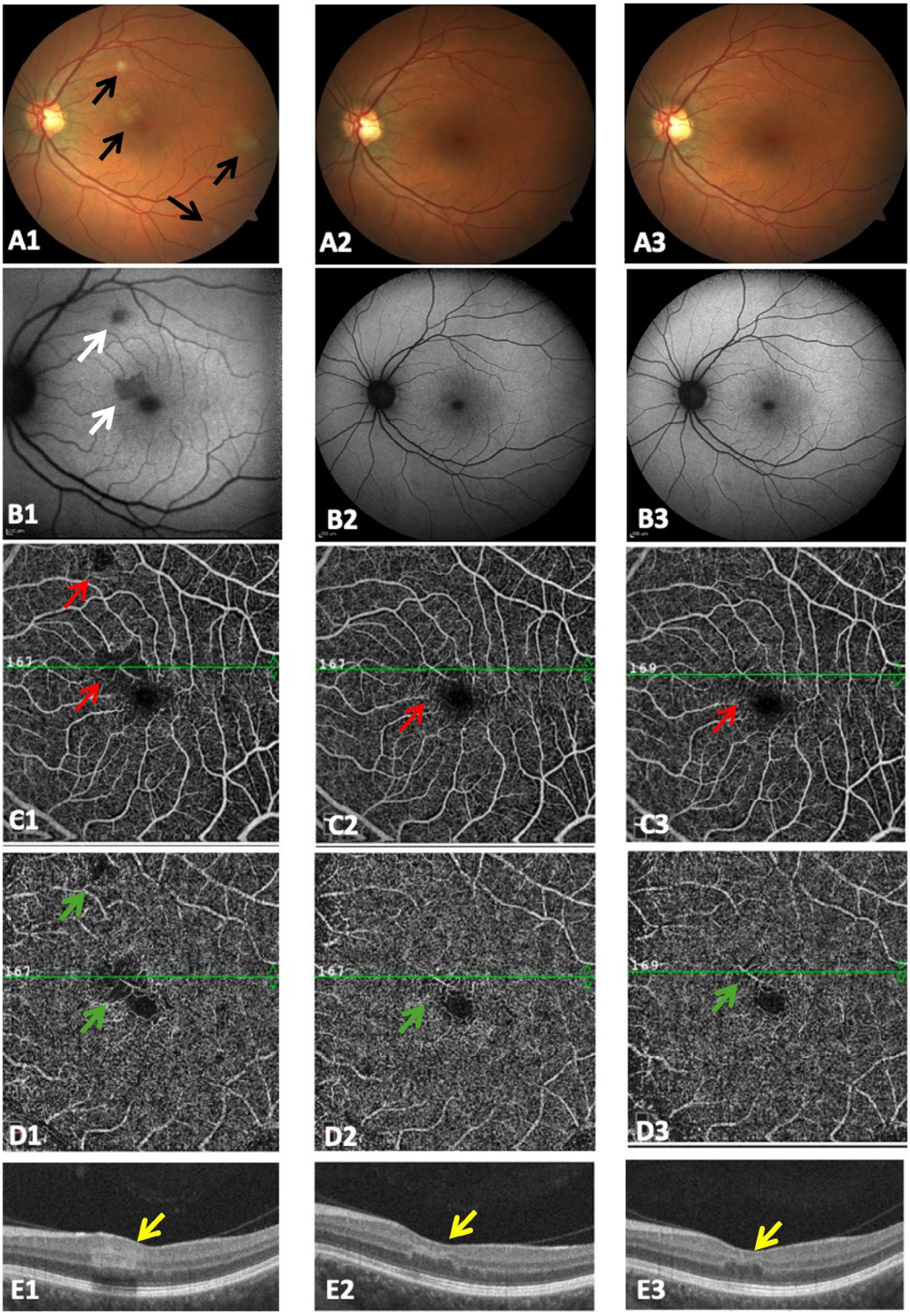

Figure 3: *Follow-up examinations of the left eye for six months. The first column (1) shows the time of diagnosis, the second column (2) shows the 2nd month follow-up, and the third column (3) shows the 6th month follow-up. Multimodal imaging includes color fundus photography (CFP) (A1-3), fundus autofluorescence (FAF) (B1-3), optical coherence tomography angiography (OCTA) in the superficial retinal capillary plexus (SCP) (C1-3) and deep retinal capillary plexus (DCP) (D1-3), and optical coherence tomography (OCT) (E1-3). The lesions disappeared on CFP and FAF at 2- and 6-month follow-up (A2, A3, B2, and B3, respectively) (black and white arrows). OCTA in SCP and DCP illustrates improvement in flow in SCP and DCP compared to presentation. However, capillary ischemic area, dilated capillary vessels and irregular margin of fovea avascular zone persisted (C1, C2, C3, and D1, D2, D3, respectively) (red and green arrows). OCT demonstrates resulting atrophy of the middle retinal layer (E2 and E3) (yellow arrows).*

DISCUSSION

PAMM is an uncommon condition following endovascular interventions. There are few case reports showing PAMM following internal carotid stenting.[11] Other endovascular procedures like coronary angiography, coil embolization, cardiopulmonary bypass, cerebral angiography, aortic aneurysm repair can also lead to PAMM with subsequent compromise in vision.[12-17] In our case report we present a PAMM case following carotid stenting in a young patient in the setting of carotid dissection with multimodal imaging.

Internal carotid artery dissection (ICAD) is one of the most important causes in ischemic stroke in young adults.[18-20] Internal carotid arteries carry blood flow to theeyes bilaterally, therefore ophthalmologic complaints are frequently the initial and sometimes the only presentation of ICAD.[21] Arterial hypoperfusion likely represents the common pathogenic etiology whereby microvascular ischemia within the most distal areas of the retinal arterial system results in localized infarction of the INL and adjacent plexiform layers.[22] The etiology of this patient's PAMM lesion was likely secondary to the procedure of internal carotid stenting, which may have cause the release of micro-emboli into systemic circulation and subsequent partial occlusion of the retinal capillary vasculature or hemodynamic changes, leading to the characteristic anatomic findings of PAMM. Additionally, the location of the PAMM lesion on the same side of the stent placement and shorter duration between the procedure and occurrence of PAMM lesion is also supportive of the release of micro-emboli in systemic circulation during the stent placement procedure. Using embolic protection devices (EPD) during the procedure can prevent embolism.[23,24] However, particles smaller than the filter pores (~100 μm)[24] can pass through the filter and cause retinal capillary (~5 μm) embolization despite EPD.[11] On the other hand, hemodynamic changes can lead to PAMM even if there is no embolism. However, using an EPD device during the ICAS procedure minimizes the risk of embolization and is one of the most important precautions.

PAMM has been associated with reduced blood flow of the SCP and DCP,[25] middle capillary plexus (MCP) alone,[26] or MCP and DCP. We have also employed OCTA to show the decreased perfusion in SCP and DCP and to show the progression of the disease. Our study demonstrates that optical coherence tomography angiography may be useful for not only diagnosis, but also for follow-up evaluations in patients with PAMM.

In conclusion, this case report illuminates a scenario of PAMM following carotid artery stenting in the setting of carotid dissection. The coexistence of carotid artery dissection and the subsequent development of PAMM raises intriguing questions about the vascular dynamics in the setting of carotid interventions. Carotid dissection may predispose patients to altered hemodynamics, potentially contributing to retinal microvascular abnormalities. The complex interplay between vascular interventions and retinal microvasculature warrants heightened awareness among clinicians involved in both ophthalmic and vascular care. Prospective studies are essential to elucidate the underlying mechanisms and optimize management strategies for such cases.

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