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Case Report

Idiopathic Unilateral Foveal Hemorrhage in a Young Woman Without Precipitating Factors: Evaluation with Optical Coherence Tomography Angiography

Oguzhan Kilicarslan,[1] Aslihan Yilmaz Cebi,[2] Didar Ucar[3]

[1]Department of Ophthalmology, Ayancik State Hospital, Sinop, Türkiye
[2]Department of Ophthalmology, Cerkezkoy State Hospital, Tekirdag, Türkiye
[3]Department of Ophthalmology, Istanbul University Cerrahpasa, Cerrahpasa Medical Faculty, İstanbul, Türkiye

Abstract

A 28-year-old young Caucasian female patient without a history of trauma or vascular disease presented with blurred vision and paracentral scotoma in her left eye. Fundus examination showed a small foveal hemorrhage in the superficial retinal layers. Initial visual acuity was 20/50 in the LE. After 2 weeks, visual acuity increased to 20/20, and hemorrhage was resolved in optical coherence tomography angiography (OCT-A) spontaneously. No vascular lesion was seen in any layer of the retina in OCT-A analysis.

Keywords: Fovea, hemorrhage, imaging, optical coherence tomography angiography, retina

Introduction

The patients who have retinal or systemic vascular diseases can develop intraretinal or preretinal focal hemorrhages in the fovea. Mostly, blunt ocular trauma can cause multiple or focal retinal hemorrhages with or without vitreous hemorrhage. Thoracic trauma may trigger Purtscher retinopathy which can present with foveal hemorrhages and retinal whitening. Increased intracranial pressure is another reason for macular hemorrhages such as Terson's syndrome and Valsalva retinopathy (1).

Various hematological diseases may cause fundus changes. Anemia, lymphoproliferative disorders, leukemia, and thrombocytopenia may lead to intraretinal hemorrhages in the macula or periphery (2). There are multiple hemorrhages, especially around the midperiphery in hyperviscosity syndrome. Furthermore, there are other signs such as increased retinal vessel tortuosity, cotton-wool spots, retinal artery or vein occlusion, and retinal neovascularization (3).

There are several case reports about unilateral idiopathic foveal hemorrhage (IFH). Here, we reported an idiopathic foveal intraretinal hemorrhage case which presented subacute decreased vision without any triggering factors or vascular disease. Differently from other cases in the literature, we obtained optical coherence tomography angiography (OCT-A) sections for excluding any abnormal vascular development at the fovea.

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Address for correspondence: Didar Ucar, MD. Department of Ophthalmology, Istanbul University Cerrahpasa, Cerrahpasa Medical Faculty, İstanbul, Türkiye
Phone: +90 537 575 08 95 **E-mail:** didarucar@gmail.com

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Case Report

Here, we presented a 28-year-old Caucasian woman who suffered decreased vision in her left eye (LE) for the past 3 days. There was no history of physical strain, weight lifting, or exercise before the symptoms. She did not have any cardiovascular diseases or atherosclerotic disorders such as systemic arterial hypertension, diabetes mellitus, hyperlipidemia, or metabolic syndrome. Medication anamnesis was unremarkable.

The best-corrected visual acuity in the right eye (RE) was 20/20 and 20/40 in her LE. The corneas were clear, and there was no pathologic feature of the anterior chamber in both eyes. Intraocular pressure was 14 mmHg in the RE and 16 mmHg in the LE. Refractive status was +0.50 D hyperopic in both eyes. Fundus examination of the RE was normal. There was a flat ellipsoid-shaped superficial intraretinal hemorrhage at the foveola in the LE (Fig. 1a). There were no exudates or accompanying vascular anomalies around this hemorrhage. The main retinal vessels and midperipheral and peripheral retina have appeared normal.

There was a hypoautofluorescent area corresponding to foveal hemorrhage (Fig. 1b). The RE was normal in FFA. There was a foveal hypofluorescence corresponding to the hemorrhagic area in the early and late phases of angiography due to the blockage of retinal and choroidal fluorescence at the fovea in the LE (Figs. 1c and d). There was no staining or vascular leakage around the hemorrhage and in other retinal areas.

The foveal hemorrhage appeared as a hyperreflective round area which caused a shadowing effect in the superficial foveola at OCT (Fig. 2a). Any abnormal vascularization area could not be detected at the superficial retinal plexus, deep retinal plexus, outer retina, and choriocapillaris layers with Angio Retina 6.0×6.0 mm scan in spectral domain OCT-A (Fig. 2b).

Complete blood count and coagulation tests were performed for excluding hemoglobinopathies, anemia, and thrombocyte disorders. No therapy was given to the patient in this process. The patient was invited for a control visit after 2 weeks. On the second visit, the foveal hemorrhage completely disappeared (Fig. 1d). Visual acuity improved to

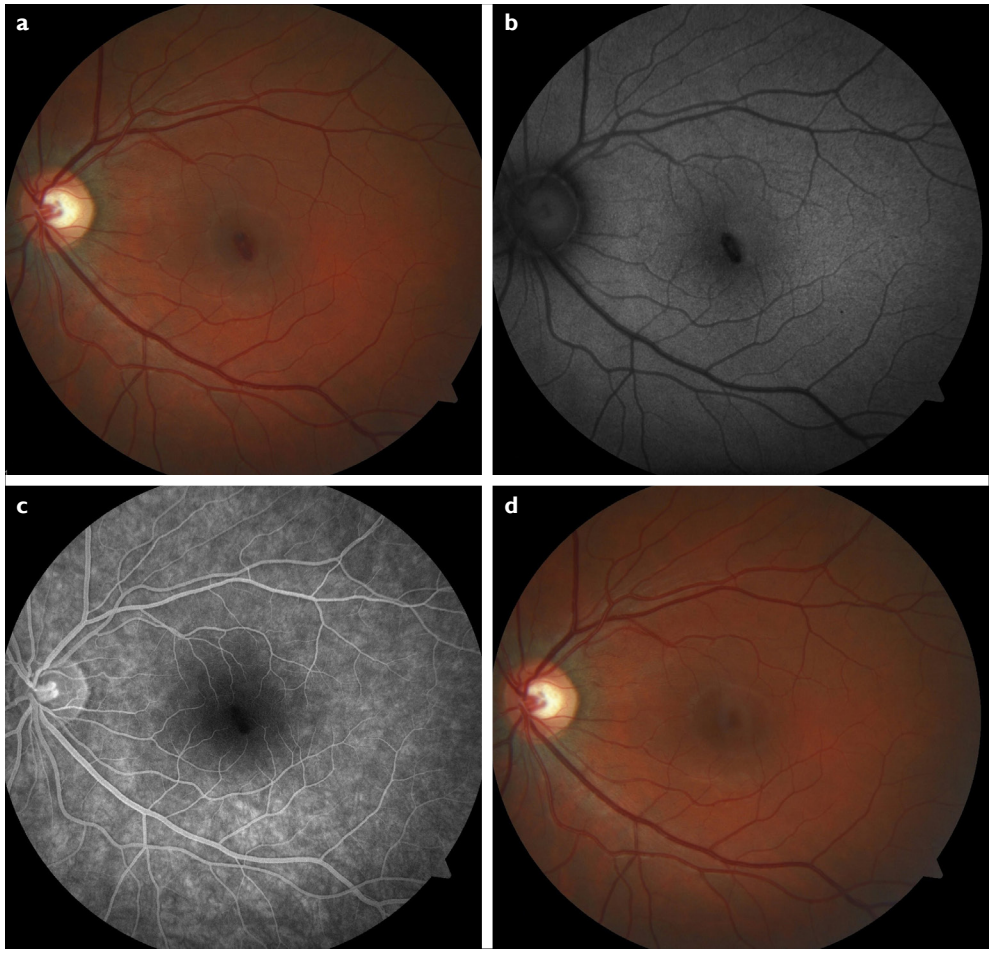

Figure 1. Fundus photograph **(a)**, fundus autofluorescence **(b)**, and fluorescein angiography **(c)** of the left eye at the first visit. Fundus photograph of the left eye after hemorrhage was resolved at the second visit **(d)**.

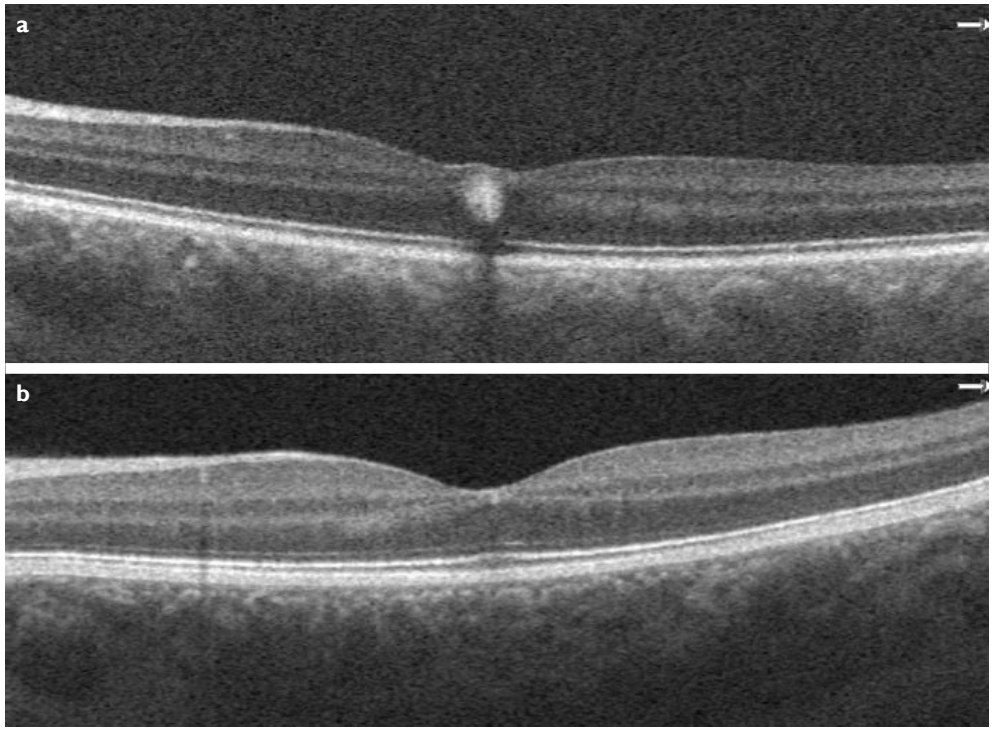

Figure 2. Hyperreflective area at the inner retina corresponding to hemorrhage in SD-OCT in the first visit **(a)**. SD-OCT imaging after hemorrhage was resolved **(b)**.

20/20. Complete resolution of hemorrhage was detected in OCT sections (Fig. 2b). There was not any morphologic disorganization or disruption in the superficial and deep layers of the retina. Any abnormal vascular formation could not be detected at the superficial, deep, outer retina and choriocapillaris in HD Angio Retina 6.0 × 6.0 mm scan (Figs. 3a and b).

In complete blood count and coagulation panel results, there was mild thrombocytosis; however, other results were normal, including PT and APTT. The blood pressure was 110/65 mmHg in the initial examination and 120/70 mmHg in the second examination.

Discussion

In our case, the patient presented with subacute vision loss. Symptoms existed for 10 days and were not progressive. Foveal intraretinal hemorrhages can be seen in diabetic retinopathy, hypertensive retinopathy, Valsalva retinopathy, anemic retinopathy, Terson's syndrome, idiopathic macular telangiectasia, Purtscher retinopathy, and radiation retinopathy.

A sudden increase of ocular intravenous pressure may provoke a hemorrhage in the fovea for instance Valsalva retinopathy or Terson's syndrome. There were no systemic dis-

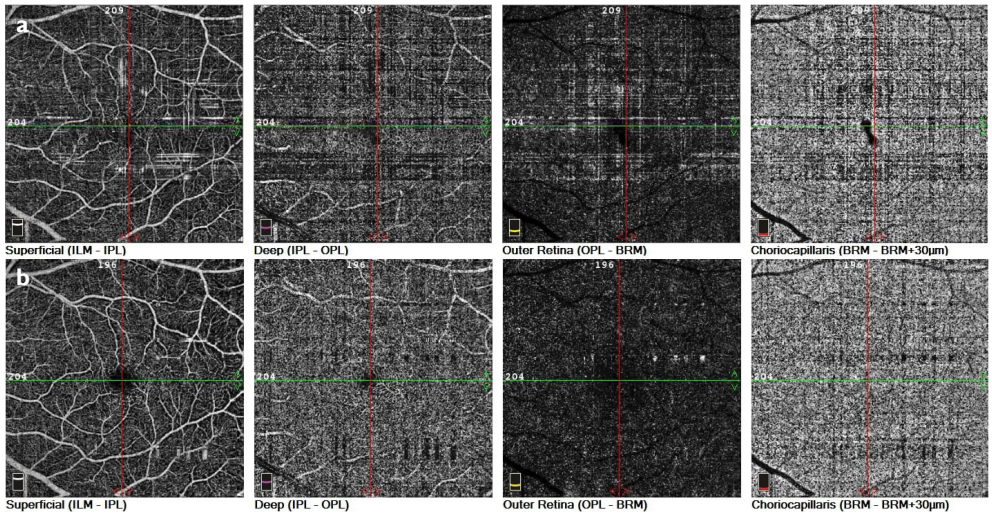

Figure 3. HD Angio Retina 6 mm × 6 mm OCTA slab in the first **(a)** and second **(b)** visits.

eases, history of trauma, or Valsalva in the present case. No usage of anticoagulant or antithrombotic drugs, which could cause hemorrhagic diathesis, was detected.

The main pathogenetic factors of diabetic retinopathy are vascular leakage and capillary occlusion. Microangiopathy can cause intraretinal microhemorrhages at the macula. Vascular leakage causes to form lipid exudates mostly in the posterior pole. Endothelial basement membrane dysfunction due to hyperglycemia can cause microaneurysms resembling foveal hemorrhages (4). Microvascular arteriolar changes in hypertensive retinopathy are generalized or focal arteriolar narrowing and arteriovenous nicking. Advanced retinopathy lesions are cotton-wool spots, intraretinal hemorrhages, and optic disc edema (5). In our case, there was one focal hemorrhage around the foveola incompatible with these pathologies.

Oral contraceptive agents may lead to macular hemorrhages by changing the reproductive hormonal status in women. In general, oral contraceptives are associated with retinal vascular occlusions; however, there are several case series that show these drugs may cause posterior pole hemorrhages. The mechanisms of this condition are explained with thromboembolic status, retinal arterial spasm, and arteriosclerosis (6). Drug history was unremarkable in our case. Furthermore, there is another phenomenon called high-altitude retinopathy, which is seen in workers in high altitudes or climbers, and may resemble this presentation. Hypoxia is the primary factor that contributes to occur retinal hemorrhages in this disease (7).

Valsalva retinopathy was first described by Duane in 1972. Duane presented three cases that had preretinal and intraretinal multiple hemorrhages in the macula. These hemorrhages were explained by a sudden increased intrathoracic pressure related to distal trauma (8).

Messmer et al. used the term solitary intraretinal macular hemorrhage (SIMH) in 1984 for perifoveal focal hemorrhage localized beneath the internal limiting membrane like our case. In this study, Messmer et al. examined 30 patients with SIMH, but only four patients had Valsalva history. The other three patients had a pregnancy, and four patients had an oral contraceptive drug history. All of the cases in this group had complete visual recovery and similar FFA findings to our case. The median resolution time of hemorrhage was calculated as 4 weeks. Hemorrhage disappeared both at fundus examination and optical coherence tomography within 2 weeks in our patient (9).

Pitta et al. described a similar clinical appearance (as a small unilateral foveal hemorrhage) in young adults who did not have any underlying disease. They presented a case series of nine patients, and six of them had physical extrusion history. FFA findings and initial visual status (between 20/200 and 20/20) were similar to the former study of Messmer et al. (9) The complete resolution time of hemorrhage in this case series was between 2 weeks to 1 month similar to our case (1).

Pruett et al. also described a similar spectrum of retinopathy as microhemorrhagic retinopathy (10). They reported 20 patients between 14 and 58 years with monocular, small, macular, round, punctate, or bilobar hemorrhages. Most of the patients had a good initial VA higher than 20/50. Final VA was found as 20/20 or 20/25 at 13 of 15 patients who were regularly followed. Only three of the patients used antihypertensive drugs. Valsalva history was described in three patients, and three patients had impaired platelet aggregation. They could not find enough evidence of one common risk factor for this clinical entity. Hence, many contributing factors were suggested, such as sudden increased intrathoracic pressure, blood cell abnormalities, drugs that impair platelet functions or coagulant factors, and oral contraceptives (10).

The presentation of our case was quite similar to the previous case series about focal foveal hemorrhages that we have mentioned above. Initiation with subacute visual impairment, characteristic solid sub-ILM, and intraretinal hemorrhage in the fovea, FFA findings without leakage and smooth recovery without any sequel are common clinical features with previous cases. Our case had no blood abnormalities except a mild thrombocytosis. In medical history, there was not any situation associated with Valsalva stress such as cough, vomiting, exercise, weight lifting, and any suspicious drug usage. The hemorrhage originated from the perifoveal capillary plexus quite likely. Visual recovery time in our case was similar to previous studies, which varied between 2 weeks and 1 month. Only Pruet et al. reported 1–7 months of complete visual recovery (10). The predilection of young individuals of this pathology was also remarkable.

Multimodal imaging is important in IFH cases. Fundus photography is very crucial to document the regression or progression of hemorrhage during the follow-up period. FFA is useful to detect most abnormal vascular formations (choroidal neovascularization (CNV) and telangiectasia), vascular leakage, and retinal edema or capillary non-perfusion areas. Because IFH is an exclusion diagnosis, findings of any possible vascular abnormality must be excluded before the diagnosis is made. A characteristic FFA finding is a round foveal hypofluorescence without any other pathologic signs. Previous reports also showed similar FFA findings. OCT and OCT-A findings were first reported in the present case. OCT shows a round hyperreflective area in the superficial foveola and its shadow effect. OCT-A also shows the hemorrhage as an ellipsoid non-flow area in the foveola; however, any capillary non-perfusion area or vascular abnormality cannot be seen.

The most related differential diagnosis, in this case, is an idiopathic CNV. CNV-related findings such as subretinal fluid, pigment epithelial detachment, or macular edema did not present in the fundus appearance. Often, ancillary imaging tests are necessary for a definitive CNV diagnosis. Both FFA and OCT-A are very useful devices for detecting CNV in suspicious cases. Because of this, we performed multimodal imaging to exclude a possible idiopathic CNV. In our case, OCT-A did not show any specific vascular growth in the choriocapillaris and retinal layers. The patient's refraction was also excluded, the possibility of a myopic CNV.

Conclusion

Consequently, our case report provided an OCT-A analysis of this clinical entity. OCT-A is a promising technology that analyzes vascular anatomy with detailed segmentation. It is quite successful to detect silent vascularization. Hence, we aimed to exclude any latent vascular abnormality in this clinical entity by performing OCT-A. Even if we could not detect any specific vascular abnormality, further investigation with OCT-A is needed in larger patient groups with IFHs.

Disclosures

Informed consent: Written informed consent was obtained from the patient for the publication of the case report and the accompanying images.

Peer-review: Externally peer-reviewed.

Conflict of Interest: None declared.

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