

Focal choroidal excavation complicated with choroidal neovascularization: a case report and review of the literature

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Dear Editor,

We write to report one case of focal choroidal excavation (FCE) complicated with choroidal neovascularization (CNV), and to review the literature on FCE. FCE is a newly recognized imaging finding with the development of optical coherence tomography (OCT) technology. FCE is defined as an area of macular choroidal excavation without evidence of posterior staphyloma or scleral ectasia^[1]. FCE could be secondary to various diseases^[2-4]. CNV is one of the complications of FCE^[5]. The development of CNV could damage the visual function of patients and thus need to be closely monitored and treated promptly.

A 31-year-old female patient complained of recurrent visual loss in her left eye for 2y and presented to the ophthalmology department. Her best corrected visual acuity (BCVA) was 20/20 in her right eye and 20/30 in her left eye. She was moderately myopic (approximately -5.00 DS) in both eyes. Her slit lamp examination was normal. Fundus examination showed multiple subretinal pigmented scars in her left eye (Figure 1B). The spectral-domain OCT (SD-OCT) scan revealed FCE at the scar areas (Figure 1D-1E). The FCE lesions demonstrated hyperfluorescent changes without leakage in the late phase on fundus fluorescein angiography (FFA) and hypofluorescent spots on indocyanine green angiography (ICGA; Figure 1F-1K). The patient was diagnosed as punctate inner choroidopathy (PIC) of her left eye. Since the lesions

were inactive, she was followed-up regularly.

Three years later, the patient complained of visual loss in her left eye for 4d. Her BCVA was 20/20 in her right eye and 20/300 in her left eye. An SD-OCT scan of her left eye showed a subretinal hyperreflective lesion at the macular fovea, the external limiting membrane (ELM), ellipsoid zone, interdigitation zone, and retinal pigment epithelium (RPE)-Bruch's complex zone were disrupted, along with the thickening of the choroid (Figure 2B). An OCT angiography (OCTA) scan of her left eye revealed CNV (Figure 2C-2D). The patient received 0.5 mg ranibizumab intravitreal injection in her left eye. Two months after the anti-vascular endothelial growth (VEGF) treatment, SD-OCT scan showed the regression of the subretinal hyperreflective CNV tissue (Figure 3A-3C). She received a repeated anti-VEGF treatment, and 1mo after the second injection, the CNV tissue was further resolved (Figure 3D-3F). Her BCVA of the left eye was stable at 20/50 during the follow-up.

FCE was first reported by Jampol *et al*^[6]. Margolis^[1] retrospectively analyzed the clinical and imaging features of 12 FCE cases and proposed the definition and classification of the FCE changes. FCEs can be either bilateral or unilateral^[7], and the number and locations of the FCE lesions varies in different cases^[7]. The FCEs can distribute either in the foveal region or in the extrafoveal region^[8]. FCEs usually appeared as yellowish spots or pigmented mottling, while some of the FCEs may not be discernable by fundus photography^[8]. SD-OCT scan revealed the FCEs as outpouching and disturbance of RPE layer, without changing the border of the choroid and the sclera. The outer nuclear layer usually thickened over the excavation^[4]. The ellipsoid zone and RPE-Bruch's complex zone were intact in more than half of the patients^[7], while in some cases, the RPE layer, ELM and ellipsoid zone are discontinuous and disrupted^[4].

The pathogenesis of FCE has not been fully elucidated. The FCEs were considered to be idiopathic. In early reports referred to FCEs, the lesions were taken as congenital changes^[6], most of the which exhibited no remarkable changes during a long-term follow-up^[7]. Recently, Gan *et al*^[9] pointed out that

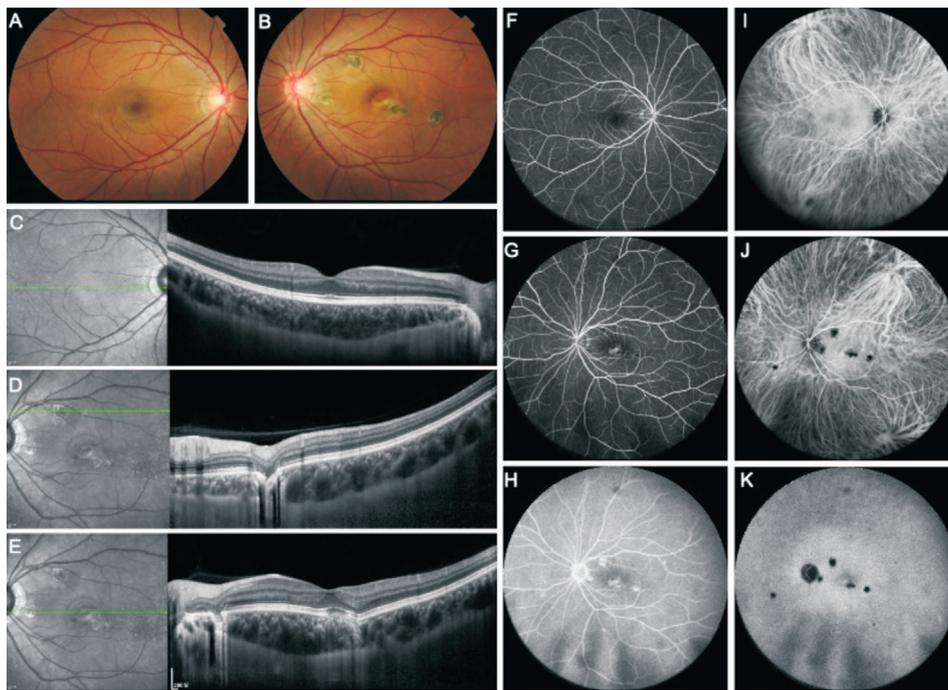


Figure 1 Multimodal imaging of the patient A, B: Fundus photography. The fundus examination showed multiple subretinal pigmented scars in the posterior pole area of her left eye (B). C-E: SD-OCT scan of the patient. The SD-OCT scan revealed FCE at the scar areas in her left eye (D and E). F-H: FFA of the patient. The FCE lesions demonstrated hyperfluorescent changes without leakage. I-K: ICGA of the patient. The FCE lesions showed hypofluorescent spots. SD-OCT: Spectral-domain optical coherence tomography; FCE: Focal choroidal excavation; FFA: Fundus fluorescein angiography; ICGA: Indocyanine green angiography.

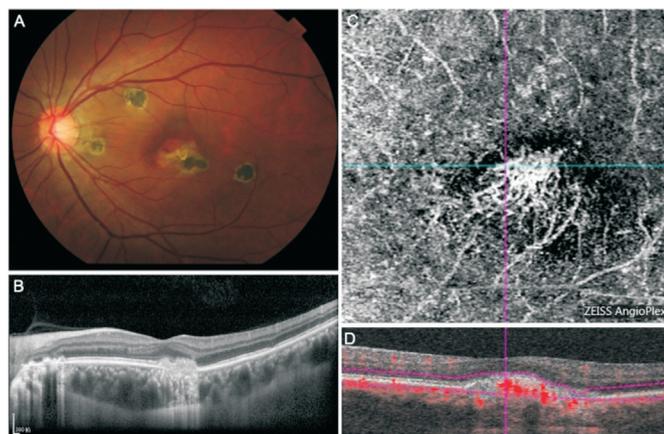


Figure 2 Multimodal imaging of the patient A: Fundus photography showed multiple pigmented scars in the posterior pole area of her left eye; B: SD-OCT scan of her left eye showed a subretinal hyperreflective lesion at the macular fovea, the external limiting membrane, ellipsoid zone, interdigitation zone, and RPE-Bruch's complex zone were disrupted, along with the thickening of the choroid; C, D: OCTA scan of the patient's left eye revealed CNV. SD-OCT: Spectral-domain optical coherence tomography; OCTA: Optical coherence tomography angiography; RPE: Retinal pigment epithelium; CNV: Choroidal neovascularization.

the prevalence of FCE in clinical work is considerable, and FCEs can occur with diverse chorioretinal diseases. Common concomitant diseases include central serous chorioretinopathy (CSC), age-related macular degeneration (AMD), polypoidal

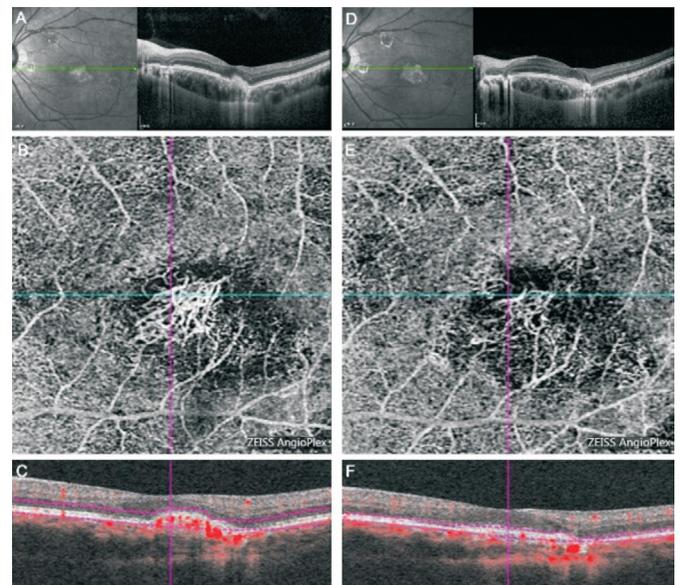


Figure 3 SD-OCT and OCTA images of the patient after anti-VEGF treatment A-C: Two months after anti-VEGF treatment, the SD-OCT scan showed regression of the subretinal hyperreflective CNV tissue. D-F: One month after the second anti-VEGF injection, the CNV tissue was further resolved. SD-OCT: Spectral-domain optical coherence tomography; OCTA: Optical coherence tomography angiography; VEGF: Vascular endothelial growth factor; CNV: Choroidal neovascularization.

choroidal vasculopathy (PCV)^[3,8,10]. Similar to the case reported here, some cases of FCE could be manifestations of choroidal

inflammatory diseases or pachychoroid spectrum diseases^[11]. Kim *et al*^[3] reviewed 33 eyes with multifocal choroiditis and panuveitis (MCP) or PIC, and found that 20% of the patients had FCEs. Haas *et al*^[12] documented a PIC case associated with FCE and CNV. Gan *et al*^[9] demonstrated that there might be common mechanisms in different diseases underlying FCE formation. Their research suggested the processes involving the impairment or tissue loss of the outer retina and inner choroid disrupt the balance of intraocular pressure, and that choroidal pressure may play a role in FCE formation^[9].

Although most lesions are stable, some may develop secondary CNV^[11]. The occurrence of CNV can cause visual loss and thus requires periodic monitoring. Chen *et al*^[7] recently published a long-term observational study showing that approximately 16% of the FCE lesions developed CNV within the FCE region. Xu *et al*^[5] reported that all CNV lesions grew from the bottom or slope of the excavation. Rajabian *et al*^[13] found that 35% of eyes with FCE had CNV. Galvin and Fung^[14] reported that the CNV can also lead to the development of FCE. Focal choroidal ischemia, overlying RPE changes, and occasional separation of the sensory retina from the RPE layer in FCE may serve as a predisposition for CNV development. Recently, OCTA scan showed that the deep capillary plexus and choriocapillaris plexus were significantly altered in FCE patients, and choroidal stroma was significantly reduced in the areas closer to the FCE region, suggesting the weakening of the architectural support^[13]. Mechanical stretching of the retina may cause breakage in the Bruch's membrane, inducing neovascularization of the choroid^[15].

The patient we reported received anti-VEGF intravitreal injection, and the CNV regressed after the treatment. Different research groups^[15-16] found the CNV associated with FCE had a good response to anti-VEGF therapy, and most of the CNV regressed after 1 injection. The lesions in our case remained stable during the follow-up period, which confirmed the efficacy of anti-VEGF injection in the treatment of FCE associated CNV.

In summary, FCE is a lesion detected by OCT scan, which could be either idiopathic or associated with some fundus diseases. CNV is not a common complication of FCE, but can be devastating to the visual function. Thus, caution should be taken with these patients. Anti-VEGF treatment for CNV complicated by FCE can achieve favorable effects. However, the pathogenesis of the FCE and the CNV associated with FCE need further investigation.

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