• Letter to the Editor •

Multimodal imaging findings of focal scleral nodule (solitary idiopathic choroiditis) in three cases

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Dear Editor,

Herein, we report multimodal imaging findings in three cases of focal scleral nodule (FSN). The term "unifocal helioid choroiditis" was initially proposed by Hong[1] in 1997 to describe idiopathic solitary choroidal lesions in 6 cases. In 2002, Shields *et al*[2] reported 60 similar cases, coining the term "solitary idiopathic choroiditis". With the advent of enhanced depth imaging optical coherence tomography (EDI-OCT), these lesions were shown to arise in the sclera rather than the choroid. A 2020 multicenter study proposed renaming solitary idiopathic choroiditis as FSN based on multimodal imaging observations including EDI-OCT in 63 cases[3]. Duignan *et al*[4] reported in their 2021 series encompassing 34 cases that all solitary idiopathic choroiditis lesions were intrascleral and recommended the nomenclature "idiopathic scleroma".

FSN typically presents unilaterally, occurs more frequently in women than men, and most commonly affects individuals between 20 and 50 years of age[3-4]. It is characterized by a well-circumscribed, elevated yellow-white lesion, usually located in the post-equatorial fundus with a predilection for the circumpapillary region. An orange halo may surround the lesion in some cases. Although earlier reports described FSN lesions with signs of active inflammation, including ill-defined margins and a dull yellow appearance, more recent studies suggest that overt inflammatory activity is rarely observed in FSN[2-4].

Ethical Approval This study was conducted in accordance with the Declaration of Helsinki and approved by the Institutional Ethics Committee (No.İ11-1037-25). Written informed consent for publication of clinical details and images was obtained from all patients.

Case 1 A 54-year-old woman was referred with a presumptive diagnosis of intraocular tumor. Her medical history included rheumatoid arthritis and hypothyroidism. Best-corrected visual acuity (BCVA) was 20/20 bilaterally. Anterior segment examination was unremarkable in both eyes. Funduscopic evaluation of the right eye revealed a slightly elevated yellow lesion located inferonasal to the optic disc (Figure 1A). Fundus autofluorescence (FAF) demonstrated a mildly hyperautofluorescent lesion (Figure 1B). B-mode ultrasonography revealed a minimally elevated (<1 mm) acoustically solid irregular lesion (Figure 1C). Swept-source OCT (SS-OCT) identified a dome-shaped intrascleral lesion with thinning of the overlying choroid (Figure 1D). SS-OCT angiography (SS-OCTA) revealed no alterations in the superficial or deep retinal vascular plexuses and showed signal voids in the outer retina and choriocapillaris (Figure 1E–1H).

Case 2 A 46-year-old man underwent a routine ophthalmologic evaluation as part of the preoperative workup for kidney donation. His medical history was unremarkable. BCVA was 20/20 bilaterally. Anterior segment findings and fundus examination of the left eye were normal. The right eye exhibited a slightly elevated yellow lesion located inferior to the optic disc (Figure 1I). EDI-OCT revealed a dome-shaped intrascleral lesion with associated choroidal thinning (Figure 1J). FAF showed mild hyperautofluorescence over the lesion (Figure 1K). The lesion exhibited hyperfluorescent staining on fluorescein angiography (FA), particularly in the mid and late phases (Figure 1L, 1M).

Case 3 A 22-year-old male patient was referred with a prior diagnosis of sclerochoroidal calcification (SCC) in the right eye from an external center. BCVA was 20/20 bilaterally, with unremarkable anterior segment findings. Funduscopic evaluation of the right eye revealed a discrete, slightly elevated yellow lesion located inferonasal to the optic disc (Figure 2A). FAF demonstrated mild hyperautofluorescence (Figure 2B). B-mode ultrasonography revealed a minimally elevated

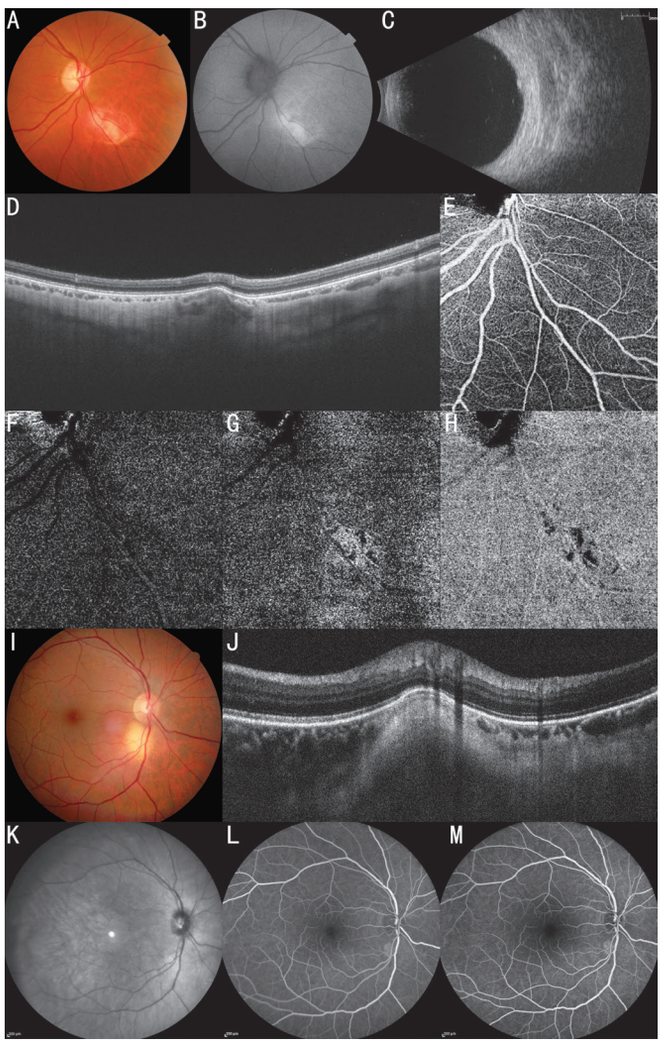

Figure 1 Cases 1 and 2 Case 1: A: Color fundus photograph of the right eye reveals a slightly elevated yellow lesion located inferonasal to the optic disc; B: Fundus autofluorescence demonstrates mild hyperautofluorescence of the lesion; C: B-mode ultrasonogram reveals a minimally elevated (<1 mm) acoustically solid irregular lesion; D: Swept-source optical coherence tomography shows a dome-shaped intrascleral lesion with thinning of the overlying choroid and an intact retinal pigment epithelium contour; E–H: Swept-source optical coherence tomography angiography shows no abnormalities in the superficial (E) and deep (F) retinal capillary plexuses, and reveals flow voids in the outer retina (G) and choriocapillaris (H). Case 2: I: Color fundus photograph demonstrates a slightly elevated yellow lesion inferior to the optic disc in the right eye; J: Enhanced depth optical coherence tomography reveals a dome-shaped intrascleral lesion associated with thinning of the overlying choroid and preservation of retinal pigment epithelium; K: Fundus autofluorescence shows mild hyperautofluorescence corresponding to the lesion; L–M: Fluorescein angiography demonstrated hyperfluorescent staining of the lesion in the mid (L) and late (M) phases.

(<1 mm) and acoustically solid lesion (Figure 2C). SS-OCT identified a dome-shaped, intrascleral lesion with thinning of the overlying choroid and an associated pigment epithelial detachment (Figure 2D). SS-OCTA showed no alterations in the superficial or deep retinal vascular plexuses and flow void areas in the outer retina and choriocapillaris (Figure 2E–2H), indicative of vascular displacement rather than calcific deposits. FA demonstrated progressive hyperfluorescence of the lesion, most notably from the mid to late phases (Figure 2I, 2J). These features are more suggestive of FSN than classic SCC, which typically presents as diffuse, flat calcific plaques.

None of the patients exhibited ocular inflammation. Systemic work-ups for infectious and inflammatory causes including chest radiography, serum angiotensin-converting enzyme levels, tuberculin skin testing, and serologies for *Bartonella*, *Toxoplasma*, *Toxocara*, and *Treponema pallidum* were entirely unremarkable in all patients. Based on clinical and imaging findings, all 3 patients were diagnosed with FSN. Observation was recommended. At a mean follow-up of 10 (6–12)mo, clinical features remained stable in all cases.

DISCUSSION

Multimodal imaging, including EDI-OCT and SS-OCT, is pivotal for accurate diagnosis in FSN. On FAF, FSN lesions commonly display central hyperautofluorescence and peripheral hypoautofluorescence[3-5]. In all our cases, FAF imaging revealed mild to moderate hyperautofluorescence. FA in FSN typically demonstrates early hypofluorescence corresponding to the lesion, followed by late staining without significant leakage. Areas of early hypofluorescence are thought to result from blockage by the elevated lesion and compression of the overlying choriocapillaris[3]. Cases 2 and 3 exhibited similar findings on FA with transition from early hypofluorescence to progressive mid- and late phase hyperfluorescent staining. On B-mode ultrasonography, FSN typically appears as a minimally elevated acoustically solid lesion without posterior shadowing, a finding that was consistent with the imaging features observed in Case 1 and Case 3 of the present study[2-5].

OCT and OCTA play essential roles in identifying the scleral origin and vascular characteristics of FSN lesions. On OCT, FSN typically appears as an elevated scleral mass causing compression of the overlying choroid, with preservation of the adjacent retinal architecture in most cases. OCTA further enables non-invasive assessment of the retinal and choroidal microvasculature by detecting motion contrast generated by erythrocyte flow within blood vessels. SS-OCTA, with its longer wavelength and deeper tissue penetration, allows improved visualization of the choroid compared with conventional spectral-domain systems. In our cases, SS-OCTA demonstrated signal voids at the level of the outer retina and choriocapillaris, consistent with previously reported findings[3]. These flow deficits may be due to compression of

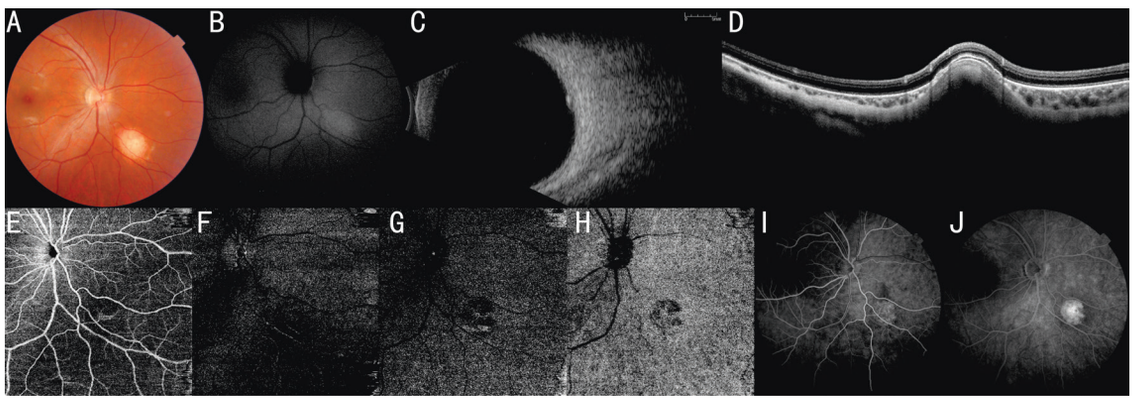

Figure 2 Case 3 A: Color fundus photograph of the right eye reveals a slightly elevated yellow lesion located inferonasal to the optic disc; B: Fundus autofluorescence demonstrates mild hyperautofluorescence of the lesion; C: B-mode ultrasonogram reveals a minimally elevated (<1 mm) acoustically solid irregular lesion; D: Swept-source optical coherence tomography shows a dome-shaped, intrascleral lesion with thinning of the overlying choroid and an associated pigment epithelial detachment; E–H: Swept-source optical coherence tomography angiography shows no abnormalities in the superficial (E) and deep (F) retinal capillary plexuses, and reveals signal voids in the outer retina (G) and choriocapillaris (H); I–J: Fluorescein angiography demonstrated progressive hyperfluorescence of the lesion from mid (I) to late (J) phases.

the choriocapillaris by the underlying scleral lesion, signal attenuation caused by elevation of the lesion, and reduced perfusion secondary to focal inflammatory or structural alterations in the adjacent choroid[3].

Based on initial clinical observations, FSN was considered as an inflammatory condition. However, advancements in multimodal imaging have improved the understanding of FSN as a scleral-based lesion, while its exact pathophysiology and the role of inflammation remain incompletely understood[6]. Recognizing the characteristic imaging features of FSN is essential for distinguishing it from intraocular tumors and inflammatory choroidopathies. Its unknown etiology, absence of clinical signs of inflammation, and negative systemic laboratory workup are critical in differentiating FSN from inflammatory conditions. Whereas inflammatory choroiditis typically presents with multifocal lesions, FSN generally manifests as a solitary, unilateral lesion. Notably, Park *et al*[7] were the first to report a bifocal presentation of FSN.

FSN likely represents a localized, self-limited fibroinflammatory response to an unknown insult (possibly immune-mediated, ischemic, infectious, or parainfectious), originating at the sclera–choroid interface and leading to secondary compression or remodeling of the overlying retinal pigment epithelium (RPE) and choriocapillaris. Kumar *et al*[8] described a case of FSN in a patient with significant exposure to horses, dogs, and wild animals. The authors suggested that cat-related *Bartonella* infection may play a major role in pathogenesis although their patient had negative cat-related *Bartonella henselae* titers[8]. They speculated that *Bartonella*-associated pathogenesis could not be totally ruled out, considering that *Bartonella* antibody titers tend to decline over time[8]. In all our cases, serological tests were negative and no signs of ocular or systemic inflammation were detected. In this context, we are unable to make any further interpretations regarding the pathogenesis of FSN based on our cases.

FSN can mimic a variety of ocular conditions, including choroidal metastasis, choroidal melanoma, retinoblastoma/retinocytoma, astrocytic hamartoma, choroidal scars, white dot syndromes, choroidal osteoma, and SCC. SCC is one of the key entities to consider in the differential diagnosis of FSN. On OCT, FSN appears as a smooth, dome-shaped scleral lesion with no posterior shadowing, mild choroidal compression, and generally preserved RPE contour. In contrast, SCC shows a highly hyperreflective calcified mass with dense posterior shadowing, irregular or rock-like contours, marked choroidal thinning over the lesion, and irregular or atrophic RPE changes. On B-mode ultrasonography, FSN typically appears acoustically solid without posterior shadowing, whereas SCC demonstrates a highly reflective calcified lesion with marked posterior acoustic shadowing. Additionally, FSN tends to occur in the post-equatorial region, while SCC is more commonly located outside the vascular arcades. In cases where multimodal imaging remains inconclusive, orbital computed tomography can be considered to exclude calcified lesions including SCC and choroidal osteoma. Computed tomography involves radiation exposure and may be unnecessary when multimodal imaging findings are clearly concordant with FSN.

Due to its benign nature and limited impact on visual function, FSN generally does not require therapeutic intervention. In cases where the lesion appears active and is accompanied by inflammation, systemic corticosteroids have been proposed as a treatment option, although supporting evidence is limited[2]. Some active lesions may resolve spontaneously or with corticosteroid therapy. Inactive FSN typically appears as a

flat or slightly elevated, well-circumscribed yellow-white lesion surrounded by a halo, without associated features such as intraretinal or subretinal fluid, retinal vascular dilation, or hemorrhage[2,8]. In contrast, active FSN is characterized by a dull-yellow lesion with ill-defined borders and associated exudative changes[2,9-11]. Rarely, choroidal neovascularization has been reported in active FSN[12]. Given the absence of systemic abnormalities and the inactive appearance of the lesions in our patients, a conservative observational approach was adopted. None of the cases demonstrated clinical progression during a mean follow-up period of 10mo. In conclusion, FSN is a benign, non-neoplastic scleral lesion that can clinically mimic intraocular tumors and inflammatory conditions. Accurate diagnosis relies heavily on multimodal imaging including EDI-OCT and SS-OCT. In the absence of clinical or imaging signs of activity, FSN lesions should be observed, as they typically remain stable over time. Further studies are needed to better elucidate the pathogenesis and natural history of FSN.

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