Stellate nonhereditary idiopathic foveomacular retinoschisis and an approach to the differential diagnosis of macular star

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Purpose of review
This review aims to introduce stellate nonhereditary idiopathic foveomacular retinoschisis (SNIFR) and its differential diagnosis. We summarize findings from case reports and series published in the last few years on the clinical and imaging findings in SNIFR.

Recent findings
SNIFR presents as either a unilateral or bilateral macular star on fundus examination without clinical or imaging evidence of exudation or frank vitreomacular traction. optical coherence tomography (OCT) imaging shows schisis cavities in the Henle fibre and outer plexiform layers that correspond to the stellate *en face* findings. Visual acuity is usually minimally affected, and the presence of significant vision loss should prompt high clinical suspicion for alternate diagnoses.

Summary
SNIFR is a recently characterized clinical entity that serves as an important addition to the differential diagnosis of a macular star. It is a diagnosis of exclusion and should be distinguished from other causes of macular star such as neuroretinitis, vitreomacular traction, ocular manifestations of malignant hypertension, congenital juvenile X-linked macular schisis, myopic maculopathy, optic pit maculopathy, nicotinic acid maculopathy or taxane maculopathy among others.

Keywords
case series, differential diagnosis, foveomacular schisis, macular star, retinoschisis

INTRODUCTION

In 2014, a case series of 17 patients was reported by Ober *et al.* [1] in which individuals exhibited a stellate appearance of the macula on fundus examination that correlated to splitting of the Henle fibre layer (HFL) on optical coherence tomography (OCT). In each case, there was no evidence of any underlying genetic, structural, inflammatory or other identifiable cause. This clinical entity was given the descriptive name of stellate nonhereditary idiopathic foveomacular retinoschisis (SNIFR), and as its initial description, at least 18 additional case reports and series have emerged to further characterize this condition. We aim to summarize the most current insights into the pathophysiology, diagnosis and treatment of SNIFR, as well as to provide an overview of the differential diagnosis the clinician must carefully consider before making the diagnosis of SNIFR.

CLINICAL AND IMAGING CHARACTERISTICS OF STELLATE NONHEREDITARY IDIOPATHIC FOVEOMACULAR RETINOSCHISIS

SNIFR is a diagnosis of exclusion, made when classic imaging findings cannot be explained by other known causes of foveomacular retinoschisis and/or stellate maculopathy (discussed below). Reports to date have demonstrated a strong female predominance in SNIFR, though the reason for this has yet to

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KEY POINTS

- Stellate nonhereditary idiopathic foveomacular retinoschisis (SNIFR) is an important new addition to the differential diagnosis of stellate maculopathy.
- SNIFR usually has a benign clinical course with only mild visual impact and can be observed given the lack of evidence for any specific effective treatments.
- Abnormal vitreomacular interface and predisposing glial insufficiency are possible contributors to the pathogenesis of SNIFR, but more study is required.
- SNIFR is a diagnosis of exclusion and can only be finalized after careful consideration of other causes of macular star phenotype, including infectious, inflammatory, exudative, structural and genetic causes.

be elucidated [1,2▪▪]. SNIFR has been described in individuals ranging from 26 to 85 years of age [1,2▪▪,3], though the average patient is usually in their 60 s. Patients are most often asymptomatic on presentation but may report decreased vision or metamorphopsia [1,2▪▪,3–6]. In general, central visual acuity is well preserved. In the absence of other structural change, average visual acuity ranges from 20/20 to 20/30 [1,2▪▪]. In the rare cases of vision worse than 20/50, other factors such as subretinal fluid or exudative maculopathy are usually seen [1,7]. SNIFR can present unilaterally or bilaterally, but the preponderance of cases appears to be unilateral [1,2▪▪,8]. On fundus examination, the foveal reflex may be blunted with apparent elevation and foveal cysts in a stellate configuration (Fig. 1) [1,2▪▪]. This pattern becomes more evident on *en face* infrared imaging [5]. Cross-sectional OCT scans in SNIFR show increased central foveal thickness with foveomacular retinoschisis cavities at the level of the HFL/OPL [1,6,9]. Interestingly, in the original SNIFR report, half of eyes also showed peripheral retinoschisis in inner retinal layers [1]. This finding has been replicated in more recent publications on widefield OCT suggesting an overlapping pathophysiology between foveal and peripheral retinoschisis [2▪▪,10–13]. As is typical of retinoschisis, fluorescein angiography usually shows absence of leakage in the macula (Fig. 2) [1,2▪▪], though several cases of idiopathic foveomacular retinoschisis have noted slight leakage on fluorescein angiography at the disc [3,4]. Care should be taken in these circumstances to extensively rule out other causes, as this does not appear to be a classic finding in SNIFR. Electroretinogram (ERG) can be useful in distinguishing from known genetic or dystrophic conditions, as ERG findings in SNIFR are typically normal [2▪▪,3,6]. Macular perimetry is typically normal; however, recent studies have shown that patients with associated peripheral schisis can show dense far peripheral field defects corresponding to these areas [2▪▪,3]. Finally, by definition, genetic testing for retinal dystrophies is invariably negative in SNIFR.

PATHOPHYSIOLOGY OF STELLATE NONHEREDITARY IDIOPATHIC FOVEOMACULAR RETINOSCHISIS

As the name suggests, the pathophysiology of SNIFR is not yet well understood. At its core, SNIFR results from the development of cystic spaces at the level of the HFL and outer plexiform layer (OPL) [1,9]. Nonleakage on fluorescein angiography and resistance to anti-VEGF therapy support that these cavities are unlikely to be an exudative maculopathy [7]. Instead, recent reports have shown compelling evidence that vitreomacular forces may play a role in schisis development. Although frank evidence of focal vitreomacular traction (VMT) precludes a diagnosis of SNIFR, some authors have hypothesized that a broad, abnormally adherent posterior hyaloid interface may play a causative role. In the original SNIFR cohort, 19 out of 22 eyes showed an attached posterior hyaloid [1]. In the largest SNIFR case series to date, 86% of eyes showed an anomalous or incomplete posterior vitreous detachment (PVD), compared with 42% of unaffected fellow eyes [2▪▪]. Interestingly, multiple reports of surgical intervention for SNIFR or similar forms of foveomacular retinoschisis have noted abnormally adherent posterior hyaloid or internal limiting membrane (ILM) [4,14–16]. This tractional hypothesis is further supported by reports of improvement in retinoschisis after spontaneous release of vitreomacular adherence. Nogueira *et al.* reported a case [17▪] in which posterior hyaloid release from the fovea correlated temporally with improvement of SNIFR findings on OCT. Similarly, Bloch *et al.* [2▪▪] identified two patients with spontaneous improvement after complete PVD. These publications thus suggest that VMT forces may play a role in SNIFR pathogenesis.

There is also evidence that indicates the retinoschisis pattern in SNIFR may be influenced by natural weak points in the foveal architecture. The horizontally directed fibres of the HFL present a mechanical weak point, which may be susceptible to splitting when exposed to vertical tractional forces [2▪▪]. A comparative analysis of swept-source optical coherence tomography angiography (SS-OCTA) imaging in SNIFR and congenital X-linked retinoschisis (CXLR) [18] found that schisis development in SNIFR was primarily located in avascular zones, wherein bridging Muller cells are the only

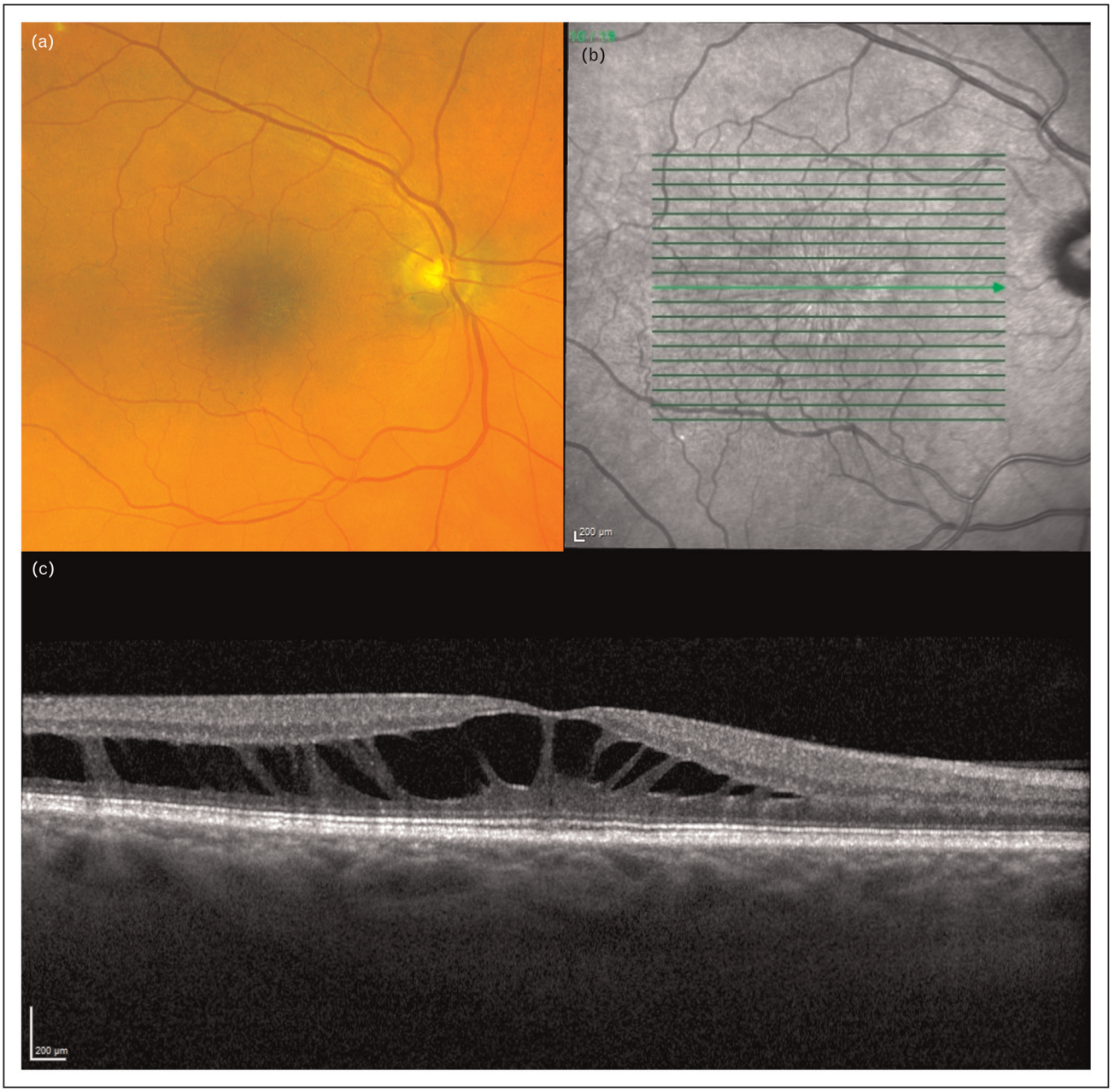

FIGURE 1. Stellate nonhereditary idiopathic foveomacular retinoschisis. (a) Pseudocolour imaging of the right fundus. The stellate striations in the macula radiating outward from the foveal with lack of refractile lipid exudates. (b) En face infrared imaging highlights the stellate findings. (c) Cross-sectional OCT through the fovea reveals schitic cavities in the Henle fibre layer and outer plexiform layer corresponding and there is no frank vitreomacular traction.

support to foveal ultrastructure. This contrasted with CXLR, wherein bridging vessels between the intermediate and deep capillary plexuses span the cavities. It is therefore possible that SNIFR cavities develop in areas of least resistance and may represent dysfunction at the level of Muller cell dendrites. The morphology of the schisis in SNIFR has also been shown to be dynamic, with moment-to-moment changes in retinal thickness being observed with Valsalva efforts [3].

DIFFERENTIAL DIAGNOSIS FOR STELLATE NONHEREDITARY IDIOPATHIC FOVEOMACULAR RETINOSCHISIS

Exudative diseases

Neuroretinitis

One of the most critical entities to rule-out in a patient with suspected SNIFR is neuroretinitis (Fig. 3), first, because of the shared feature of a

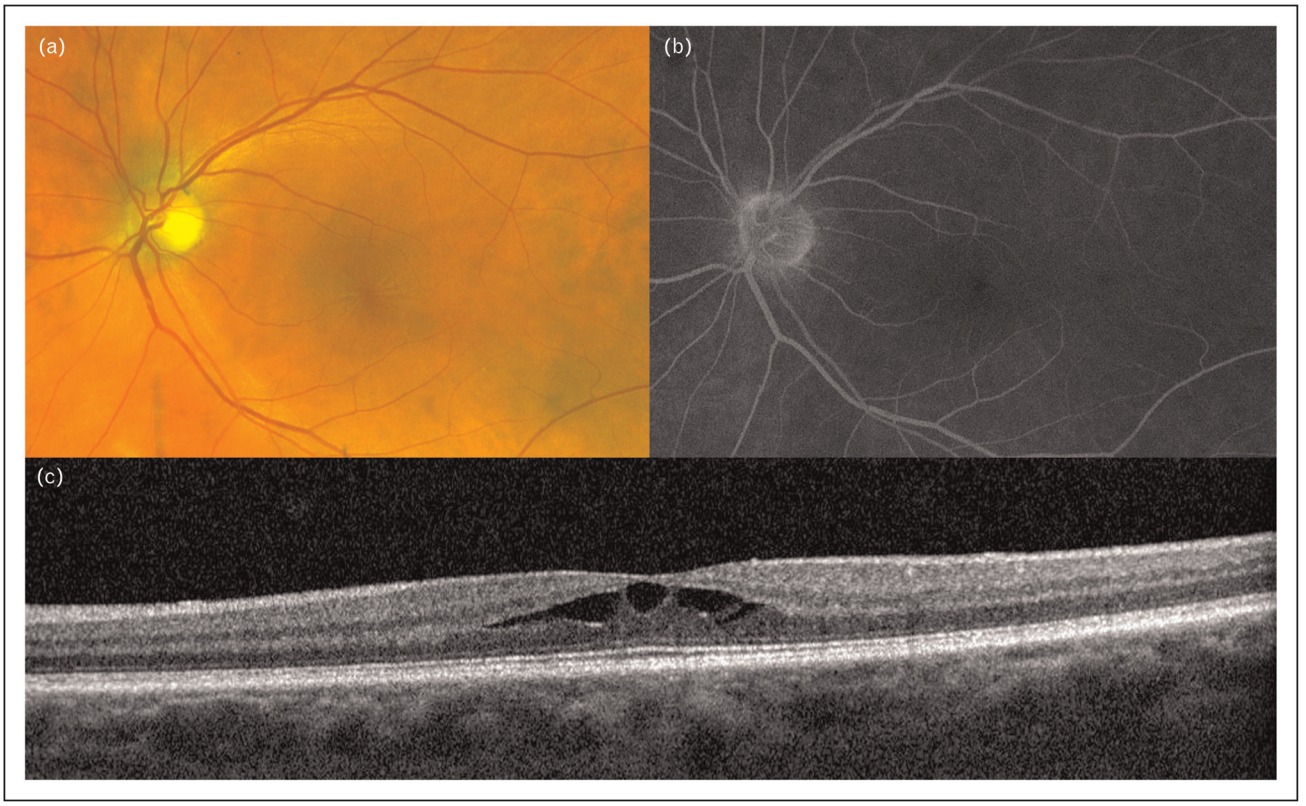

FIGURE 2. Angiographically-silent oedema in SNIFR. (a) Colour fundus photograph showing radial spoking appearance at the macula. (b) The late-phase fluorescein angiography showing no leakage. (c) The optical coherence tomography reveals retinoschisis.

macular star in both these conditions, and second, because of the underlying infectious and inflammatory implications accompanying neuroretinitis that should not be missed by the clinician.

As its name implies, neuroretinitis refers to clinical optic nerve oedema and macular oedema. Because there is a striking appearance of stellate macular oedema, the first description of this clinical entity by Leber used the term 'idiopathic stellate maculopathy' [19]. However, with further investigation undertaken by Gass, the primary and preceding nature of disc inflammation and leakage was revealed, demonstrating that the stellate macular findings in neuroretinitis are a secondary finding, as fluid and lipid seep and deposit into the macular layers of the neurosensory retina [20]. Indeed, the presence of optic disc oedema is thus an immediate clue to the clinician that infectious and/or inflammatory causes are at play and that the clinical picture is inconsistent with SNIFR. Macular lipid exudation is not observed in SNIFR.

An array of infectious causes of neuroretinitis has been described in the literature [21]. Cat-scratch disease secondary to bartonella henselae has been cited as the most-common identifiable cause [22], but other bacterial diseases such a spirochetal (syphilis, Lyme, leptospirosis) and mycobacterial illnesses have also been implicated. In early reports of neuroretinitis, viral prodromes were often noted to precede blurred vision and ophthalmologic manifestations in a sizeable proportion of cases [23]. Numerous viral agents since have been implicated in pathogenesis, including, but not limited to, hepatitis, herpes, influenza and coxsackie viruses [21]. With the right exposure history or risk-factor profile, fungal and parasitic organisms (e.g. diffuse unilateral subacute neuroretinitis) are also possible causes [24].

Several noninfectious inflammatory syndromes have also been known to cause a neuroretinitis phenotype, including idiopathic retinitis, vasculitis and neuroretinitis (IRVAN), sarcoidosis, polyarteritis nodosa, Behcet disease and Vogt-Koyanagi-Harada syndrome. In other cases, an underlying cause is not found and the neuroretinitis is considered idiopathic.

Fluorescein angiography is a useful tool in differentiating neuroretinitis from SNIFR given the hallmark finding of disc staining and leakage in the former and the lack thereof in the latter [23]. Inflammatory chorioretinal lesions may also appear as deep late hyperfluorescence in neuroretinitis and should be absent in SNIFR [21,23]. It is worth noting though that the macular star pattern of exudates in neuroretinitis may persist long after disc oedema has resolved, and therefore excluding a prior episode of

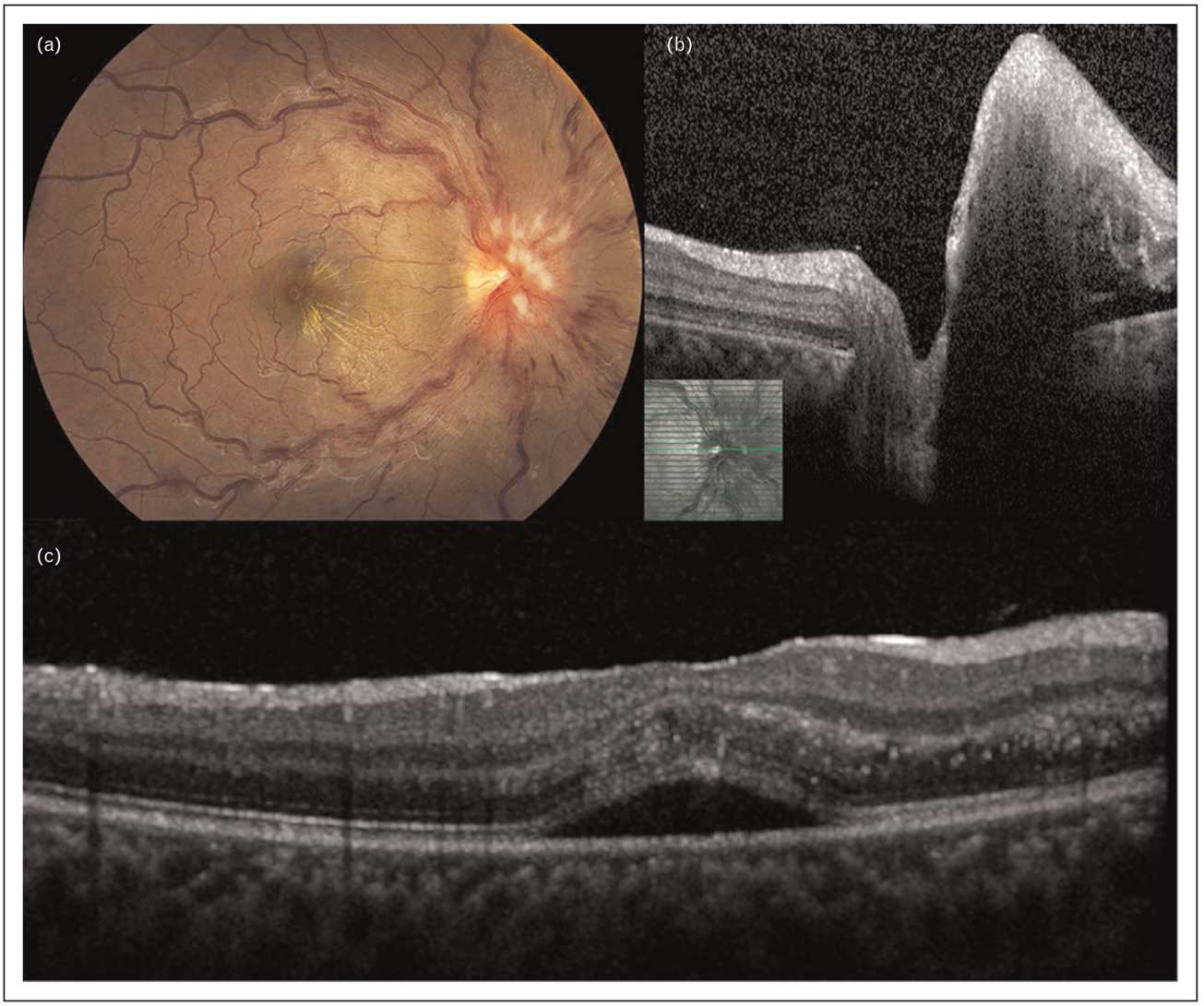

FIGURE 3. Neuroretinitis with prominent disc oedema and lipid exudation. (a) Colour fundus photograph showing stellate macular oedema with exudates. (b) The optical coherence tomography (OCT) at the optic disc showing marked disc oedema. (c) The horizontal OCT at the macula showing subtle macular oedema and subretinal fluid.

neuroretinitis in a case of suspected SNIFR may be more difficult. However, the presence of true refractile exudates is much more indicative of old neuroretinitis, as the stellate morphology of SNIFR results from schisis-change and not true exudation. Subclinical presence of subretinal fluid [25] or more recently described findings of inner-retinal folds and hyperreflective 'epipapillary' deposits [26] are characteristics on OCT studies that favour neuroretinitis over SNIFR as well.

Malignant hypertension

Malignant hypertension is a well known cause of a bilateral macular star. In a retrospective study of 40 patients referred with a diagnosis of neuroretinitis, six patients (15%) were found to have previously unknown malignant systemic hypertension [22]. A previously unknown systemic hypertension is the most common cause of macular star mistaken for neuroretinitis. Some clues can help diagnose this condition, including cotton wool spots, arterial narrowing and superficial peripapillary retinal haemorrhages [27]. Measuring the blood pressure repeatedly should be a part of the initial work-up even in a young patient with macular star and absence of systemic hypertension history. It is also noteworthy that a unilateral macular star can also be a manifestation of malignant hypertension [28].

GENETIC CONDITIONS

Congenital juvenile X-linked retinoschisis

This congenital entity is usually caused by mutations in the *RS1* gene and is inherited in an X-linked

fashion; therefore, it is more common in men than SNIFR, which is a nonhereditary female predominant disease. The CXLR usually presents bilaterally in the first decades of life with vision ranging from 20/60 to 20/120 [29]. In contrast, SNIFR is typically unilateral and adult-onset disease with a mean age of 63 years at presentation with visual acuity of 20/50 or better. Clinical manifestation of CXLR with a stellate pattern of foveal cysts arranged in a spoke-wheel configuration may resemble what could be seen in SNIFR. In addition, the presence of foveomacular schisis on OCT and the absence of leakage in fluorescein angiography could be found in both entities. Another similarity is the presence of peripheral retinoschisis in nearly half of patients. The ERG can help differentiate CXLR from SNIFR, as the former shows decreased b-wave amplitude with preserved a-wave, resulting in a negative pattern, while the latter presents with normal findings on the ERG. Genetic testing is usually normal in SNIFR, however, can reveal mutations in RS1 in CXLR patients [29].

Enhanced S-cone syndrome (Goldman-Favre syndrome)

Goldman-Favre syndrome can present with macular schisis on OCT and lack of fluorescein leakage into the cystic spaces similar to what usually can be seen in patients with SNIFR. The features that can help differentiate Goldman-Favre syndrome from SNIFR are the presence of night blindness, optically empty vitreous and pathognomonic ERG abnormalities [30].

STRUCTURAL DISEASES

Myopic degeneration

Myopic degeneration is characterized by structural changes in the posterior segment of the eye caused by the increased axial length, and it is usually associated with high myopia [31]. Similar to SNIFR, these patients may present with foveomacular retinoschisis on OCT; however, the presence of high refractive error, elongated axial length, posterior staphyloma and dome-shaped macula is more pronounced in myopic degeneration. The development of foveomacular retinoschisis in myopic patients has been shown to occur within the area of posterior staphylomas. In addition, tractional forces from adherent vitreous and epiretinal membrane could lead to development of full-thickness macular hole and foveomacular detachment [31,32].

Optic pit maculopathy

Optic disc pit maculopathy can resemble SNIFR with intraretinal schisis changes on OCT (Fig. 4). The retinoschisis may develop in up to three-quarters of patients with optic disc pit, and the size of the pit is not essentially correlated with the extent of macular detachment [33,34]. A precise clinical examination of the optic nerve head could reveal the greyish, oval excavation at the inferotemporal portion of the optic disc and help differentiate this entity from SNIFR [35]. A study by Theodossiadis *et al.* [36] has shown increased hyperfluorescence in the late phases of fluorescein angiography in patients with optic disc pit maculopathy, suggesting the fluorescein dye's leakage into the schisis cavities, while in most patients with SNIFR, the fluorescein angiography shows no leakage.

Degenerative retinoschisis

Degenerative retinoschisis is essentially an entity confined to peripheral retina and the extension of retinoschisis as far posteriorly as fovea is extremely rare [37]. However, as pointed out above, some

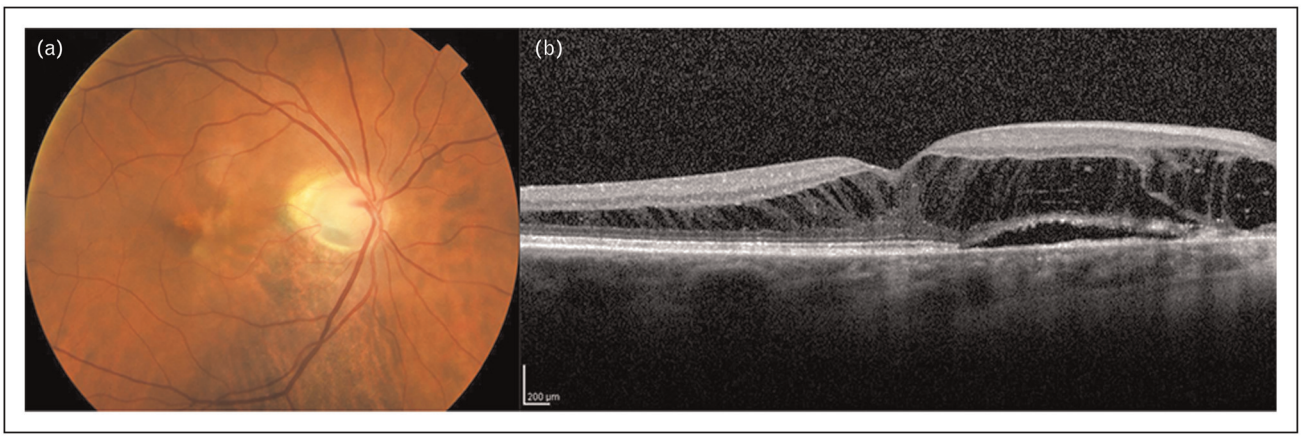

FIGURE 4. Optic pit maculopathy. (a) Colour fundus photograph showing greyish pit at the inferotemporal portion of the optic disc. (b) The horizontal optical coherence tomography showing intraretinal schisis and subretinal fluid.

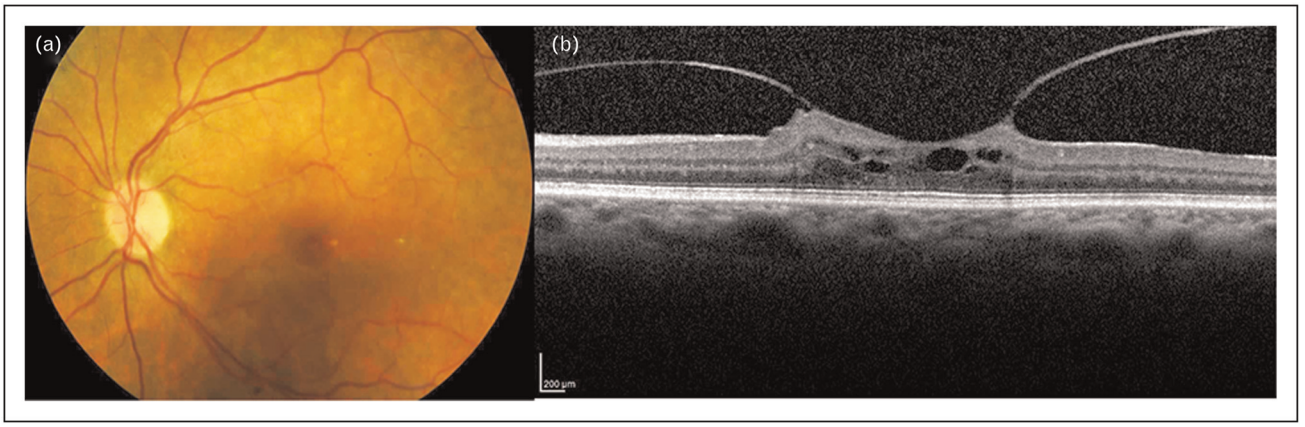

FIGURE 5. Vitreomacular traction. (a) Colour fundus photograph. (b) The horizontal optical coherence tomography showing the vitreomacular traction and associated intraretinal edema.

patients with SNIFR may have concurrent peripheral retinoschisis as well.

Glaucoma

Previous studies have shown a possible relationship between the development of glaucomatous optic disc neuropathy and macular retinoschisis [38,39]. It has been hypothesized that glaucoma can cause structural changes in the optic nerve head and allow vitreous to enter the retina and lead to schisis, similar to optic disc pit maculopathy [40]. A comprehensive workup, including intraocular pressure measurement, optic disc examination and diagnostic modalities such as visual field test and macular and optic nerve head OCT could rule out glaucoma as the underlying disease in a patient with foveomacular retinoschisis.

Vitreomacular traction

The VMT syndrome can cause continuous anterior-posterior traction to the macula and lead to foveomacular retinoschisis (Fig. 5). As discussed above, the presence of traction on OCT as well as other features such as epiretinal membrane can differentiate this entity from SNIFR.

Miscellaneous mimickers

Some other miscellaneous masqueraders can present with macular star and mimic neuroretinitis. These include idiopathic optic perineuritis, branch retinal vein occlusion, diabetic papillopathy and nonarteritic anterior ischaemic optic neuropathy (NA-AION) [41–43]. In a case series of 12 diabetic patients with NA-AION, lipid deposits were present around the macula, forming an incomplete macular star after the optic disk swelling began to resolve. However, they were not typically present during the period of acute vision loss [44]. Niacin (nicotinic acid), a vitamin used to treat lipid disorders, can cause cystoid macular oedema (CME) without fluorescein leakage evidence. Funduscopic examination of the macula typically manifests a bright yellow hue, similar to an exudate and OCT confirms the presence of cystic hyporeflective spaces in inner nuclear layers and outer plexiform. Symptoms usually resolve over 4–8 weeks following discontinuation of the offending agent [45]. Similarly, anticancer agents such as taxanes (docetaxel and paclitaxel) can cause angiographically silent bilateral CME (Fig. 6), which does not form the classic appearance of the macular star and should be differentiated from the typical appearance of SNIFR [46].

Treatment of stellate nonhereditary idiopathic foveomacular retinoschisis

Given that most patients are minimally symptomatic with preserved visual acuity, SNIFR is typically managed with observation. Patients should be monitored regularly with dilated examinations and OCT and be counselled to report vision changes. In cases wherein visual acuity decreases or patients report intolerable symptoms, topical dorzolamide may be a therapeutic option. In one case report, a patient showed total resolution of visual symptoms and SNIFR-related changes on OCT after 1 year of topical dorzolamide [47]. However, a later case series of three patients with SNIFR showed no change after treatment with dorzolamide [8]. At the present time, there is insufficient evidence to support the routine use of dorzolamide in patients with SNIFR. Bevacizumab has been coincidentally used in patients with

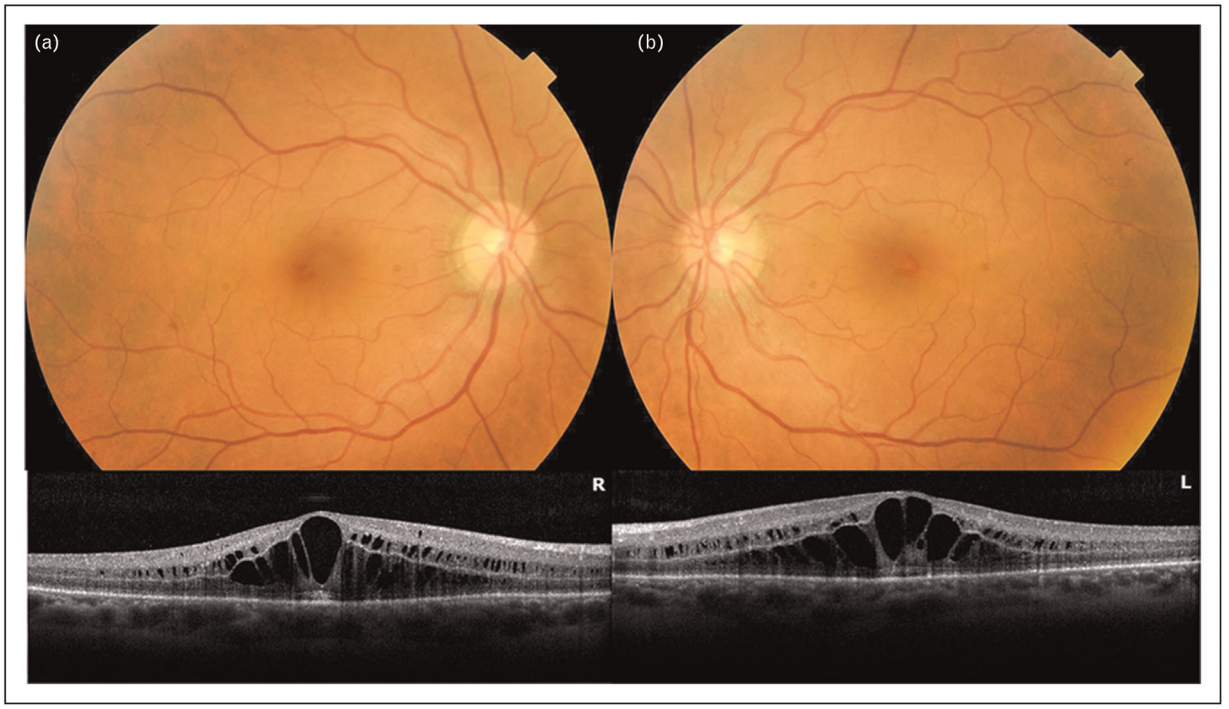

FIGURE 6. Paclitaxel-associated cystoid macular oedema. Colour fundus photographs and the horizontal optical coherence tomography through the fovea showing cystoid macular oedema in the right (a) and left (b) eyes.

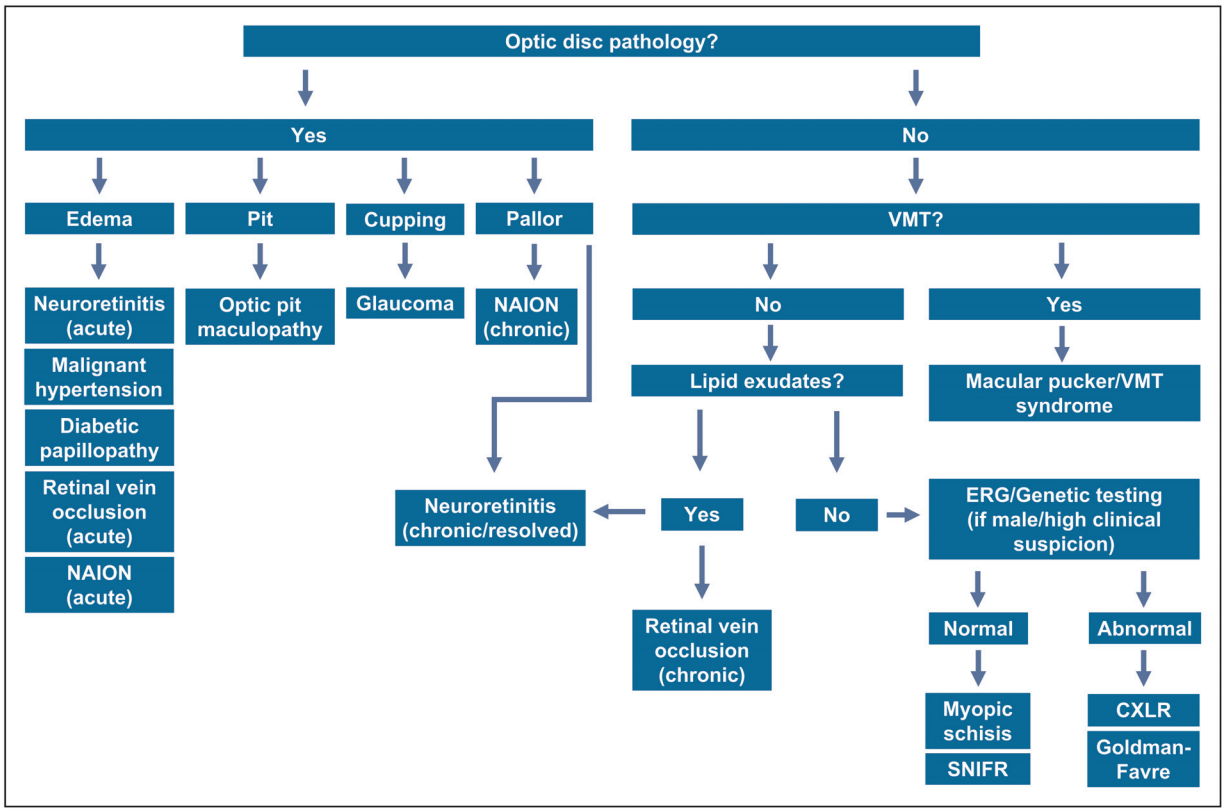

FIGURE 7. Schematic representation of our suggested approach to the differential diagnosis of stellate maculopathy with special focus on the presence or absence of optic disc abnormalities and vitreomacular traction. CXLR, congenital X-linked retinoschisis; ERG, electroretinography; NAION, nonarteritic ischemic optic neuropathy; SNIFR, stellate nonhereditary idiopathic foveomacular retinoschisis; VMT, vitreomacular traction.

SNIFR without significant improvement [7,48,49]. Pars plana vitrectomy with ILM peel has been described in select patients with idiopathic foveomacular retinoschisis [4,14–16]. In each of these patients, visual effects were progressive and more severe than SNIFR and imaging showed evidence of more obvious tractional pathophysiology, arguing more for a diagnosis of VMT rather than true SNIFR. VMT must be ruled out when considering SNIFR. No compelling evidence exists at this time to support surgical intervention in cases purely of SNIFR.

CONCLUSION

SNIFR is a relatively recent addition to the panoply of retinal conditions that can present with findings of a macular star on fundus examination and schisis-like cystic spaces in the macula on OCT imaging. Although thought to be rare, the true prevalence of SNIFR remains yet to be elucidated and thus should remain as a possible diagnosis within the mind of the clinician faced with a patient who has the clinical findings described herein. Ultimate diagnosis of SNIFR hinges on exclusion of other potential causes of similar macular findings (Fig. 7), which typically demonstrate findings of optic disc disease, angiographic leakage or frank VMT. SNIFR typically lacks these additional pathologic findings, carries a good visual prognosis, and as of now is best observed as no treatments have thus far shown definite utility. An improved understanding of the pathophysiology of SNIFR may allow new opportunities for intervention in the future.

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Conflicts of interest

None.

REFERENCES AND RECOMMENDED READING

Papers of particular interest, published within the annual period of review, have been highlighted as:

- ■ of special interest
- ■■ of outstanding interest

1. Ober MD, Freund KB, Shah M, *et al.* Stellate nonhereditary idiopathic foveomacular retinoschisis. Ophthalmology 2014; 121:1406–1413.
2. ■■ Bloch E, Flores-Sánchez B, Georgiadis O, *et al.* An association between stellate nonhereditary idiopathic foveomacular retinoschsis, peripheral retinoschisis, and posterior hyaloid attachment. Retina Phila Pa 2021; 41:2361–2369.

A single site, observational study that identifies the largest case series of SNIFR yet published. In reviewing 28 eyes from 24 individuals, the authors find that visual acuity remains stable (median 20/20) over 17 months and that many patients with SNIFR appear to have concurrent peripheral retinoschisis and a broadly adherent posterior hyaloid.

3. Dolz-Marco R, Kato K, Freund KB, Yannuzzi LA. Unusual case of stellate nonhereditary idiopathic foveomacular retinoschisis. Retin Cases Brief Rep 2017; 11(Suppl 1):S49–S53.
4. Maruko I, Morizane Y, Kimura S, *et al.* Clinical characteristics of idiopathic foveomacular retinoschisis. Retina Phila Pa 2016; 36:1486–1492.
5. Weiss SJ, Adam MK, Hsu J. En face optical coherence tomography of stellate nonhereditary idiopathic foveomacular retinoschisis. JAMA Ophthalmol 2017; 135:e174280.
6. Valikodath NG, Scripsema NK, Lim JI. Bilateral macular schisis in a woman. JAMA Ophthalmol 2021; 139:906–907.
7. Casalino G, Upendran M, Bandello F, Chakravarthy U. Stellate nonhereditary idiopathic foveomacular retinoschisis concomitant to exudative maculopathies. Eye (Lond) 2016; 30:754–757.
8. Schildroth KR, Mititelu M, Etheridge T, *et al.* Stellate nonhereditary idiopathic foveomacular retinoschisis: novel findings and OCT-angiography analysis. Retin Cases Brief Rep 2021. [Online ahead of print]
9. Falb T, Malle EM, Haas A, *et al.* Stellate nonhereditary idiopathic foveomacular retinoschsis in a patient with macular telangiectasia type 2. Retin Cases Brief Rep 2021; 15:335–338.
10. Mandell JB, Kim AY, Shahidzadeh A, *et al.* Widefield OCT findings of a patient with stellate nonhereditary idiopathic foveomacular retinoschisis. Ophthalmic Surg Lasers Imaging Retina 2016; 47:774–777.
11. Javaheri M, Sadda SR. Atypical peripapillary inner retinoschisis in stellate nonhereditary idiopathic foveomacular retinoschisis. Retin Cases Brief Rep 2018; 12(Suppl 1):S92–S97.
12. Ahmed D, Stattin M, Glittenberg C, *et al.* Stellate nonhereditary idiopathic foveomacular retinoschisis accompanied by contralateral peripheral retinoschisis. Retin Cases Brief Rep 2019; 13:135–140.
13. Kaya M, Atas F. Accidentally detected unilateral peripapillary retinoschisis: a case presentation. Eur Eye Res 2021; 1:43–46.
14. Haruta M, Yamakawa R. Vitrectomy for macular retinoschisis without a detectable optic disk pit. Retina Phila Pa 2017; 37:915–920.
15. Sun Z, Gao H, Wang M, *et al.* Rapid progression of foveomacular retinoschisis in young myopics. Retina Phila Pa 2019; 39:1278–1288.
16. Moraes BRM, Ferreira BFA, Nogueira TM, *et al.* Vitrectomy for stellate nonhereditary idiopathic foveomacular retinoschisis associated with outer retinal layer defect. Retin Cases Brief Rep 2020. [Online ahead of print]
17. ■ Machado Nogueira T, de Souza Costa D, Isenberg J, Rezende FA. Stellate nonhereditary idiopathic foveomacular retinoschisis resolution after vitreomacular adhesion release. Am J Ophthalmol Case Rep 2021; 23:101153.

An informative case report wherein the authors identify a patient with SNIFR who shows resolution following vitreomacular adherence release. Timely OCTs convincingly associate the time course of posterior hyaloid release with the resolution of SNIFR imaging findings. This supports the hypothesis that vitreomacular forces play a role in SNIFR pathogenesis.

18. Fragiotta S, Leong BCS, Kaden TR, *et al.* A proposed mechanism influencing structural patterns in X-linked retinoschisis and stellate nonhereditary idiopathic foveomacular retinoschisis. Eye Lond Engl 2019; 33:724–728.
19. Dreyer RF, Hopen G, Gass JDM, Smith JL. Leber's idiopathic stellate neuroretinitis. Arch Ophthalmol 1984; 102:1140–1145.
20. Gass JD. Diseases of the optic nerve that may simulate macular disease. Trans Sect Ophthalmol Am Acad Ophthalmol Otolaryngol 1977; 83:763–770.
21. Abdelhakim A, Rasool N. Neuroretinitis: a review. Curr Opin Ophthalmol 2018; 29:514–519.
22. Kahloun R, Khairallah-Ksiaa I, Abroug N, *et al.* Final diagnosis in patients referred with a diagnosis of neuroretinitis. Neuroophthalmology 2015; 39:266–270.
23. Maitland CG, Miller NR. Neuroretinitis. Arch Ophthalmol 19601984; 102:1146–1150.
24. Ray S, Gragoudas E. Neuroretinitis. Int Ophthalmol Clin 2001; 41:83–102.
25. Habot-Wilner Z, Zur D, Goldstein M, *et al.* Macular findings on optical coherence tomography in cat-scratch disease neuroretinitis. Eye Lond Engl 2011; 25:1064–1068.
26. Zatreanu L, Sibony PA, Kupersmith MJ. Optical coherence tomography in neuroretinitis: epipapillary infiltrates and retinal folds. J Neuroophthalmol 2017; 37:176–178.
27. I-Linn ZL, Long QB. An unusual cause of acute bilateral optic disk swelling with macular star in a 9-year-old girl. J Pediatr Ophthalmol Strabismus 2007; 44:245–247.
28. Chawla R, Tripathy K, Chaudhary S, *et al.* Unilateral macular star in a case of hypertension and retinitis pigmentosa. Semin Ophthalmol 2017; 32:535–536.
29. Rao P, Dedania VS, Drenser KA. Congenital X-linked retinoschisis: an updated clinical review. Asia Pac J Ophthalmol (Phila) 2018; 7: 169–175.
30. Marmor MF, Jacobson SG, Foerster MH, *et al.* Diagnostic clinical findings of a new syndrome with night blindness, maculopathy, and enhanced S cone sensitivity. Am J Ophthalmol 1990; 110:124–134.
31. Shinohara K, Tanaka N, Jonas JB, *et al.* Ultrawide-field OCT to investigate relationships between myopic macular retinoschisis and posterior staphyloma. Ophthalmology 2018; 125:1575–1586.

32. Takahashi H, Tanaka N, Shinohara K, *et al.* Importance of paravascular vitreal adhesions for development of myopic macular retinoschisis detected by ultra-widefield OCT. Ophthalmology 2021; 128:256–265.
33. Theodossiadis GP, Panopoulos M, Kollia AK, Georgopoulos G. Long-term study of patients with congenital pit of the optic nerve and persistent macular detachment. Acta Ophthalmol (Copenh) 1992; 70:495–505.
34. Brown GC, Shields JA, Goldberg RE. Congenital pits of the optic nerve head. II. Clinical studies in humans. Ophthalmology 1980; 87:51–65.
35. Kalogeropoulos D, Ch'ng SW, Lee R, *et al.* Optic disc pit maculopathy: a review. Asia Pac J Ophthalmol (Phila) 2019; 8:247–255.
36. Theodossiadis GP, Ladas ID, Panagiotidis DN, *et al.* Fluorescein and indocyanine green angiographic findings in congenital optic disk pit associated with macular detachment. Retina 1999; 19:6–11.
37. Straatsma BR, Foss RY. Typical and reticular degenerative retinoschisis. Am J Ophthalmol 1973; 75:551–575.
38. Hollander DA, Barricks ME, Duncan JL, Irvine AR. Macular schisis detachment associated with angle-closure glaucoma. Arch Ophthalmol Chic Ill 19602005; 123:270–272.
39. Zhao M, Li X. Macular retinoschisis associated with normal tension glaucoma. Graefes Arch Clin Exp Ophthalmol 2011; 249:1255–1258.
40. Zumbro DS, Jampol LM, Folk JC, *et al.* Macular schisis and detachment associated with presumed acquired enlarged optic nerve head cups. Am J Ophthalmol 2007; 144:70–74.
41. Parodi MB, Iacono P, Ravalico G. Macular star secondary to nasal branch retinal vein occlusion. Ophthalmic Surg Lasers Imaging 2009; 40:504–505.
42. Jastrzembski B, Torun N. A 45-year-old man with unilateral optic disc edema and vision loss. Digit J Ophthalmol 2019; 25:16–20.
43. Friedrich Y, Feiner M, Gawi H, Friedman Z. Diabetic papillopathy with macular star mimicking clinically significant diabetic macular edema. Retina 2001; 21:80–82.
44. Ruiz-Medrano J, Montero JA, Flores-Moreno I, *et al.* Myopic maculopathy: current status and proposal for a new classification and grading system (ATN). Prog Retin Eye Res 2019; 69:80–115.
45. Domanico D, Verboschi F, Altimari S, *et al.* Ocular effects of niacin: a review of the literature. Med Hypothesis Discov Innov Ophthalmol 2015; 4:64–71.
46. Perez JM, Teo K, Ong R, *et al.* Optical coherence tomography characteristics of taxane-induced macular edema and other multimodal imaging findings. Graefes Arch Clin Exp Ophthalmol 2020; 258:1607–1615.
47. Ajlan RS, Hammamji KS. Stellate nonhereditary idiopathic foveomacular retinoschisis: response to topical dorzolamide therapy. Retin Cases Brief Rep 2019; 13:364–366.
48. Amram AL, Kellogg C, Elkeeb A. Nonhereditary idiopathic foveal retinoschisis associated with new-onset proliferative diabetic retinopathy. Retin Cases Brief Rep 2020; 14:232–234.
49. Takkar B, Dube M, Goyal P, *et al.* Stellate nonhereditary idiopathic foveomacular retinoschisis treated as cystoid macular edema. Indian J Ophthalmol Case Rep 2021; 1:121–122.