AN ASSOCIATION BETWEEN STELLATE NONHEREDITARY IDIOPATHIC FOVEOMACULAR RETINOSCHISIS, PERIPHERAL RETINOSCHISIS, AND POSTERIOR HYALOID ATTACHMENT

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Purpose: Stellate nonhereditary idiopathic foveomacular retinoschisis is a disorder characterized by splitting of the retina at the macula, without a known underlying mechanical or inherited cause. This study investigates demographic, anatomical, and functional characteristics of subjects with stellate nonhereditary idiopathic foveomacular retinoschisis, to explore potential underlying mechanisms.

Methods: In this single-site, retrospective, and cross-sectional, observational study, data were collected from 28 eyes from 24 subjects with stellate nonhereditary idiopathic foveomacular retinoschisis. Descriptive statistics were reported, based on the observed anatomico-functional features.

Results: The visual acuity remained stable (median 20/20) in all subjects over a median follow-up of 17 months. All cases demonstrated foveomacular retinoschisis within Henle’s fiber layer, at the junction of the outer plexiform and outer nuclear layers. This schisis cavity extended beyond the limits of the macular OCT temporally in all eyes. In most affected eyes, there were documented features of peripheral retinoschisis and broad attachment of the posterior hyaloid at the macula. Functional testing in a cross-sectional subset demonstrated normal retinal sensitivity centrally but an absolute scotoma peripherally.

Conclusion: Stellate nonhereditary idiopathic foveomacular retinoschisis seems to be associated with peripheral retinoschisis and anomalous or incomplete posterior hyaloid detachment. Despite chronic manifestation, this does not significantly affect central visual function but can manifest with profound loss of peripheral visual function.

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Foveomacular retinoschisis (FRS) describes the presence of a localized separation of retinal layers affecting the central macula. Although FRS is typically associated with congenital X-linked retinoschisis (OMIM #312700),1 it is observed in other inherited disorders, such as enhanced S-cone syndrome (OMIM #268100) and CRB1-associated maculopathy (OMIM #604210).2,3 It is also a recognized manifestation of optic disc pit, myopic traction maculopathy, glaucomatous optic neuropathy, epiretinal membrane, vitreomacular traction (VMT) syndrome, and drug-related cystoid macular edema.4–10 These conditions often seem to have similar morphology but display different anatomico-functional natural histories.

There are several reports of atypical FRS, some of which are suggestive of a possible inherited mechanism,11–15 whereas others remain unexplained.16 In 2014, Ober et al17 coined the term “stellate nonhereditary idiopathic foveomacular retinoschisis” (SNIFR), in an attempt to provide a unifying classification under which to categorize unusual cases, without an explanatory pathophysiological mechanism. Reported cases of SNIFR seem to have favorable functional profiles and share similar anatomical configurations, namely, splitting at the level of HFL,
which is located within the parafoveal retina and comprised of horizontally aligned photoreceptor axons and Müller cell (MC) processes. However, the precise pathoanatomical mechanism through which SNIFR arises remains elusive.

We present a retrospective study of the anatomico-functional characteristics in 28 eyes with presumed SNIFR, of which 7 eyes had additional, cross-sectional multimodal imaging and functional testing, to investigate the retinal function and explore potential underlying mechanisms.

**Methods**

A single site retrospective, observational study was performed to identify subjects with evidence of FRS without a known predisposing disorder. Subjects were included, who presented to a tertiary ophthalmic hospital trust between January 2010 and January 2020, with center-involving macular schisis. Cases were identified through review of electronic case notes, using the search terms “schisis,” “retinoschisis,” “maculoschisis,” and “foveoschisis,” and correlation with historical optical coherence tomography (OCT) imaging. Exclusion criteria included subjects younger than 18 years old, those with significant ocular copathology or alternative predisposing features (such as high myopia or posterior staphyloma, optic nerve anomalies, and epiretinal membrane or focal VMT), and a family history or known genetic abnormality associated with foveomacular retinoschisis. Where documented, the following data were collected: demographic characteristics (age, gender, and ethnicity), Snellen visual acuity at baseline and final visit, spherical equivalent, axial length (AL), reported visual symptoms, and ophthalmic examination findings, including evidence of peripheral retinoschisis (PRS). In cases where data for spherical equivalent or AL were not available, high myopia was excluded if there was an absence of staphyloma on OCT or myopic retinal features on fundus imaging. Serial OCT imaging was reviewed to determine posterior vitreous detachment (PVD) status and peripheral extension of retinoschisis.

A subset of five subjects underwent further, cross-sectional studies, including OCT imaging (Cirrus 5000 HD-OCT; Carl Zeiss Meditec, Dublin, CA or Spectralis SD-OCT, Heidelberg Engineering, Dossenheim, Germany), Optos California widefield scanning laser ophthalmoscopy (Optos, Marlborough, MA), microperimetry (MAIA, CentreVue, Padova, Italy), Humphrey perimeter (Carl Zeiss Meditec), and, where available, biometry (IOLMaster 700, Carl Zeiss Meditec) and autorefraction (ARK-510A; NidekCo, Aichi, Japan). Composite OCT images were created using open-source graphics editing software (GNU Image Manipulation Program). Health Research Authority approval was obtained, and the study was performed according to the tenets of the Declaration of Helsinki.

**Results**

One thousand two hundred nineteen of the subjects identified using the prespecified search terms had macula-involving retinoschisis, of whom 1,194 (98%) were excluded for other predisposing factors (as detailed in Table 1). Thirty-three eyes from the remaining 25 subjects were considered to meet the diagnostic criteria for SNIFR, of which 5 eyes were excluded from further analysis because of the existence of significant ocular copathology, including amblyopia, branch retinal vein occlusion, and focal VMT.

In this group of 28 eyes (from 24 subjects), the mean (±SD) subject age at initial presentation was 63.6 (±11.7) years and 63% were female (Table 2). The median VA at baseline was 20/20, which remained stable over a median follow-up duration of 17 months (range: 2–134 months, in 23 eyes with follow-up data). 15/24 (63%) of subjects were asymptomatic throughout, whereas 9 (31%) reported mild to moderate distortion or blurring. One eye (subject 6) had reported long-standing unexplained poor vision, despite normal electrodiagnostic tests. One subject had a negative genetic test for RS1 mutation, whereas the remainder did not undergo testing based on a lack of anatomical or functional evidence for an inherited retinal disease phenotype, on specialist clinical assessment. 14/24 (58%) had a negative family history documented.
All affected eyes had OCT evidence of FRS, which, in each case, extended beyond the limits of the macular cube scan temporally (Figure 1). 18 affected eyes had a contemporaneous comment regarding examination of the peripheral retina, of which 16 (89%) had recorded features of PRS on examination, one of which had a stable schisis detachment.

24/28 (86%) affected eyes were also noted to have incomplete or anomalous separation of the posterior hyaloid on OCT (Figure 1). Twelve subjects had fellow eyes unaffected by FRS or other macular pathology, 6 (50%) of which also had documented evidence of PRS, whereas only 5/12 (42%) had incomplete separation of the posterior hyaloid on OCT. Of note, two subjects had FTMH and one had a lamellar macular hole in their respective fellow eyes.

A minority of eyes (11/27) had data for refractive spherical equivalent or AL, but in all documented cases, these fell within the normal range. Six eyes also underwent ancillary electrodiagnostic testing, all of which were reported as grossly normal, although two reports mentioned patchy irregularity on eccentric multifocal testing of the temporal retina, presumably in the vicinity of the PRS.

Seven eyes (from subjects 1–5) underwent further cross-sectional examination, multimodal imaging, and functional testing. On biomicroscopic examination, all these subjects demonstrated a stellate appearance at the macula and had peripheral features suggestive of PRS, including microcystoid degeneration or absolute scotoma. On the composite widefield OCT scans, the FRS was evident at the level of Henle’s fiber layer (HFL) and was continuous with PRS or schisis detachment, at which point the schisis cavity appears to widen, involving different or multiple retinal layers (Figures 2–4). All affected eyes had evidence of hyaloid attachment, to varying degrees, at the posterior pole, whereas the fellow eye in the three unilaterally affected subjects showed complete vitreous separation, but in conjunction with features of PRS. This functional peripheral loss was further demonstrated on five eyes with 60-4 ± 30-2 static VF testing. In all cases, microperimetry demonstrated normal macular function, with a mean (±SD) average sensitivity 28.5 (±1.0) dB (Figures 2 and 4).

**Discussion**

We present a combination of retrospective and cross-sectional, observational data from 28 eyes affected with SNIFR, many of which exhibit peripheral retinal manifestations and posterior vitreous hyaloid features that have not been widely reported.

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**Table 1. Pathologies Associated With Foveomacular Retinoschisis**

| Pathology Subgroup   | Precise Pathology               | No. of Subjects |
|----------------------|---------------------------------|-----------------|
| Mechanical           | High myopia                     | 531             |
|                      | Vitreoretinal interface disorders| 243             |
|                      | Optic disc pit                  | 53              |
|                      | Other peripapillary disorders   | 15              |
| Degenerative         | Age-related macular degeneration| 13              |
|                      | Degenerative retinoschisis      | 12              |
|                      | detachment                      |                 |
| Inherited            | X-linked retinoschisis          | 170             |
|                      | Enhanced s-cone syndrome        | 16              |
|                      | Macular dystrophy               | 14              |
|                      | Retinitis pigmentosa            | 5               |
|                      | Best disease                    | 5               |
|                      | Other inherited                 | 20              |
| Inflammatory/vascular| Cystoid macular edema           | 27              |
|                      | Diabetic macular edema          | 13              |
|                      | Central serous chorioretinopathy| 9               |
|                      | Macular telangiectasia          | 5               |
|                      | Other inflammatory/vascular     | 9               |
| Neoplastic           | Melanoma                        | 19              |
|                      | Nevus                           | 7               |
|                      | Other intraocular tumors        | 7               |
| Iatrogenic           | Nicotinic acid maculopathy      | 1               |
| Idiopathic           | Stellate nonhereditary idiopathic| 25              |
|                      | foveomacular retinoschisis      |                 |
| Total                |                                 | 1,219           |
### Table 2. SNIFR Subject Characteristics (28 Eyes)

| No. | Sex | Baseline Age (years) | Eye | Ethnicity | SE (D) | AL (mm) | FRS | PRS | Complete PVD | Baseline VA | Final VA | Follow-up Duration (months) | Symptoms | Comment |
|-----|-----|----------------------|-----|-----------|--------|---------|-----|-----|--------------|-------------|----------|---------------------------|----------|---------|
| 1   | F   | 51                   | OS  | White     | +1.00  | 22.15   | Y   | Y   | N            | 20/16       | 20/20    | 53                        | Mild distortion | Eccentric temporal mfERG abnormality |
| 2   | F   | 58                   | OS  | Black     | +0.50  | 22.55   | Y   | Y   | N            | 20/16       | 20/20    | 22                        | Asymptomatic |                     |
| 3   | M   | 70                   | OS  | White     | +2.00  | 24.12   | Y   | Y   | N            | 20/40       | 20/30    | 20                        | Mild blurring |                     |
| 4   | F   | 54                   | OD  | White     | −5.00  | 24.21   | Y   | Y   | N            | 20/20       | 20/30    | 5                         | Asymptomatic |                     |
| 5   | M   | 53                   | OD  | White     | +2.50  | 24.32   | Y   | Y   | N            | 20/20       | 20/16    | 112                       | Mild distortion |                     |
| 6   | F   | 41                   | OD  | White     | +2.50  | 24.32   | Y   | Y   | N            | 20/20       | 20/16    | 112                       | Mild distortion |                     |
| 7   | M   | 74                   | OS  | Black     | +2.25  | NR      | Y   | Y   | NR           | 20/16       | 20/20    | 10                        | Mild blurring |                     |
| 8   | F   | 60                   | OD  | White     | NR     | 23.27   | Y   | Y   | N            | 20/16       | 20/16    | 117                       | Asymptomatic | Normal ERG  |
| 9   | F   | 65                   | OD  | White     | NR     | 23.27   | Y   | Y   | N            | 20/16       | 20/16    | 77                        | Asymptomatic | Normal ERG  |
| 10  | M   | 70                   | OD  | White     | NR     | 23.27   | Y   | Y   | NR           | 20/20       | 20/16    | 2                         | Difficulty in dim light |                     |
| 11  | M   | 74                   | OS  | NR        | NR     | 23.27   | Y   | Y   | Y            | 20/20       | N/A      | 0                         | Asymptomatic | mfERG abnormalities peripherally |
| 12  | F   | 61                   | OS  | NR        | NR     | 23.27   | Y   | Y   | N            | 20/16       | 20/16    | 47                        | Asymptomatic |                     |
| 13  | F   | 60                   | OD  | Asian     | NR     | 23.27   | Y   | Y   | N            | 20/16       | N/A      | 0                         | Asymptomatic |                     |
| 14  | F   | 37                   | OS  | Chinese   | Emmetropia | 23.27 | Y   | Y   | N            | 20/30       | 20/30    | 4                         | Asymptomatic | Normal ERG  |
| 15  | M   | 70                   | OD  | Black     | Hyperopia | 23.27 | Y   | Y   | N            | 20/20       | 20/20    | 22                       | Mild distortion |                     |
| 16  | F   | 56                   | OD  | White     | +1.50  | NR      | Y   | Y   | N            | 20/20       | N/A      | 0                         | Asymptomatic |                     |
| 17  | F   | 84                   | OD  | White     | NR     | 23.27   | Y   | Y   | N            | 20/40       | 20/40    | 24                       | Blurred vision |                     |
| 18  | M   | 67                   | OD  | NR        | NR     | 23.27   | Y   | Y   | N            | 20/30       | N/A      | 0                         | Asymptomatic |                     |
| 19  | M   | 63                   | OD  | Black     | NR     | 23.27   | Y   | Y   | N            | 20/30       | 20/30    | 3                         | Asymptomatic |                     |
| 20  | F   | 60                   | OD  | White     | NR     | 23.27   | Y   | Y   | N            | 20/20       | 20/20    | 13                       | Asymptomatic |                     |
| 21  | F   | 84                   | OD  | NR        | Pseudophakia | 23.27 | Y   | Y   | N            | 20/40       | 20/60    | 17                       | Mild blurred vision |                     |
| 22  | F   | 71                   | OD  | NR        | Pseudophakia | 23.27 | Y   | Y   | N            | 20/30       | 20/20    | 7                         | Asymptomatic |                     |
| 23  | F   | 77                   | OS  | Asian     | NR     | 23.27   | Y   | Y   | N            | 20/60       | 20/60    | 47                       | Blurred vision |                     |
| 24  | M   | 66                   | OS  | White     | Mild myopia | 23.27 | Y   | Y   | N            | 20/16       | 20/20    | 4                         | Asymptomatic | Normal ERG  |

DRS, degenerative retinoschisis; mfERG, multifocal electroretinogram; NR, not recorded; SE (D), spherical equivalent (diopters).
previously. A mean age at presentation of 63.6 years and slight female preponderance, albeit nonsignificant, is in keeping with previous reports and supports the notion that vitreous liquefaction and anomalous PVD, which is known to occur earlier and more frequently in female subjects, may play a role.17,18

In 2014, Ober et al17 published a retrospective study of 22 eyes from 17 patients, all of which had FRS reported within the outer plexiform and outer nuclear layers, with no alternative predisposing disorder. In this study, six eyes from four patients in this series were demonstrated to have concurrent PRS, whereas a total of 19 eyes (86%) were reported not to have evidence of PVD (despite an average age of 61 in a predominantly myopic cohort). Several subsequent case studies have attributed findings of FRS to SNIFR, some of which have evidence of concurrent extramacular schisis, with associated features suggestive of both vitreoretinal adhesion and PRS (although these was not always considered of primary relevance in these reports).19–22 Considering these factors, along with our findings, we hypothesize that there may be both a tractional element at the vitreoretinal interface resulting in FRS in eyes with SNIFR, as well as an association with PRS.

We found that, of those with contemporaneous documentation, 89% had evidence of PRS in addition to FRS. Moreover, in those who underwent cross-sectional functional testing, a discrepancy was noted between the areas affected by retinoschisis centrally and peripherally. All of our patients’ microperimetric findings support the consensus in the literature that SNIFR does not, for the most part, lead to significant loss of macular function.17 However, it seems that there is a transition zone, in the midperiphery, where both the anatomical and functional characteristics of the retinoschisis changes, from a cavitation solely within HFL to one including the inner nuclear layer and, in some cases, also the nerve fiber layer (Figures 2-4). At approximately this point, the 60-4 static perimetry demonstrates the presence of a dense visual field defect. Here, the retinoschisis is behaving in a functional manner that we would traditionally expect with acquired PRS. Although it is reassuring that the central retina seems to be spared such degeneration, the loss of peripheral field could challenge the purportedly benign course of SNIFR. The precise mechanism by which acquired retinoschisis causes absolute scotoma in the periphery is unclear, but may be attributable to erosion of the neuroretinal and glial support elements during coalescence of microcystoid cavities.23,24 Natural history studies of PRS have previously shown central involvement to be extremely rare; however, these studies pre-date the widespread use of high-resolution OCT.25,26

Another novel observation is the large proportion of eyes with anomalous or incomplete PVD (86%) compared with those unaffected fellow eyes (42%). Furthermore, the presence of vitreomacular interface abnormalities in five excluded or fellow eyes lends further support to the possible role of anomalous PVD in subjects predisposed to developing the features of SNIFR. It was also noted that the unaffected fellow eyes of several subjects had evidence of PRS, but with complete PVD and no FRS. This asymmetric finding has been described previously in a single case by Ahmed et al,21 who ascribed it to possible “early stage of stellate nonhereditary idiopathic retinoschisis without foveal involvement”. In fact, in our study, spontaneous improvement of FRS was observed in two subjects after separation of the posterior hyaloid (Figure 5), and, in one of these cases, residual shallow PRS was detected on OCT. The observation of concurrent PRS and FRS, as in these cases of SNIFR, lends credence to the plausibility of a common pathophysiological mechanism. Although acquired PRS is a common
Fig. 2. Images from subject 1 (OS). Optos widefield SLO imaging (A and B) reveals micr­ocystoid changes in the tempo­ral peripheral retina. Widefield composite OCT (C) demonstrates continuity between the central foveomacular schisis and PRS, with incomplete pos­terior vitreous separation (D). En­face projection of the midretina (E) shows the “spoke-wheel” dis­tribution of the schisis cavity. Microperimetry (F) is normal, with evidence of scotoma in the nasal peripheral visual field (cor­responding to the temporal retinal changes) on 60-4 static perimetry (G). OS, left eye; SLO, scanning laser ophthalmoscopy.

disorder, the concomitant manifesta­tion of FRS might only be rarely observed, because of the necessary co­existence of tightly adherent cortical vitreous at the macula.

The pathoanatomical mechanism by which tractional macular disorders, such as epiretinal membrane or VMT, lead to the formation of foveomacular retinoschisis has previously been explored. It is proposed that, under normal conditions, the combined action of a specialized MC sub­population in the fo­veola (termed the “MC cone”) and “typical” z-shaped MCs in the foveal walls, form and maintain the foveal ultra­structure. The outer processes of the z-shaped MCs run in HFL with the photoreceptor axons in a horizontal orientation, thereby rendering this layer me­chanically vulnerable to separation in response to inward tractional forces. In fact, the morphology of these MCs seem to provide a degree of anatomical compliance, allowing the retention of function in the presence of significant foveal deformation. On OCT, anteroposterior and tangential traction (such as those observed in tractional disorders of the vitreoretinal interface) seem to manifest with progressive beveling of columnar retinal elements (believed to be the MC processes), which obliquely span the schisis cavity. This anatomical phenomenon is believed to be responsible for the radiating “spoke-wheel” pattern, as seen en face imaging. The visual acuity is preserved at the point that the MC processes are in a beveled orientation, only deteriorating once the processes become fully verticalized. Although our cases do not have angiographic data to support an absence of exudative macular edema, the OCT and en face images are highly supportive of a similar pathoanatomical me­chanism in SNIFR. Furthermore, the discrepancy observed between the anatomico-functional behavior of the retina centrally and peripherally could
potentially be explained by a difference in MC morphology. Outside the macula, MCs become verticalized early in response to traction, resulting in the observed multilayer retinoschisis and associated functional decline. This anatomic variability is best observed on the en face images (Figure 4C), where the “spoke-wheel” pattern of the beveled MC processes, centered on the fovea, progressively verticalizes into the perifoveal and midperipheral retina, giving rise to a “speckled” appearance.

In view of this potential mechanism, it is important to distinguish SNIFR from other causes of tractional FRS, which may share morphological characteristics. In particular, the presence of high myopia, VMT, or an epiretinal membrane may indicate an alternative mechanism.7-9; continuity with the optic nerve ought to raise suspicion of optic disc pit maculopathy or glaucoma-associated maculopathy.4-6 Inherited retinal disease should be considered as a possible cause in all young patients with cystoid spaces and inner/outer segment disruption on OCT.1-3 In 2019, Sun et al.35 reported a series of 17 eyes from 10 young, moderately myopic and predominantly female patients. In the first report of its kind, they described a condition, distinct from SNIFR, which manifests with FRS and leads to rapid functional deterioration with the development of subfoveal fluid and FTMH. At the time of surgery, they noted “remarkable liquefaction of the core vitreous” and had difficulty inducing PVD because of tight attachment to the macula.

We agree that this is a distinct entity to SNIFR, but postulate that they may share common features, in particular the lack of complete PVD and tight vitreomacular adherence. Perhaps the difference in subject demographic reflects the premature vitreous liquefaction in Sun et al.’s cohort, compared with the normal age of liquefaction at which those patients with SNIFR seem to be affected. Despite Sun et al.’s description of a lack of PRS in their group, they published two images demonstrating extramacular schisis and it would be interesting to know the results of both anatomical and functional investigations of the peripheral retina in this cohort.

Despite a probable tractional etiology, there is currently no evidence to support surgical intervention in uncomplicated cases of SNIFR. Despite chronicity, most cases seem to maintain good macular function, and subjects are generally not aware of peripheral scotomata. Moreover, it is likely that surgical induction of PVD and removal of the posterior hyaloid would be hampered by both the retinoschisis itself and tight vitreoretinal adhesion, which could increase the rate of intraoperative complication or surgical failure.22

This study is limited by the retrospective design, resulting in incomplete collection of data, such as refractive error, AL or investigations, including genetic testing or fluorescein or OCT angiography. In this regard, we are not in a position to explore certain associations, such as the relationship between refractive error and SNIFR, or confirm a definite...
absence of inherited or exudative pathology. Nevertheless, we have shown that SNIFR seems to be an under-recognized clinical phenomenon, accounting for up to 2% of all recorded cases of FRS. In addition, we have demonstrated an apparent association between SNIFR and both PRS and anomalous or incomplete PVD, in the largest study of this disorder to date. It is conceivable that FRS, and by association, PRS, in SNIFR may represent an acquired mechanical process. In these cases, retinoschisis manifests with different anatomico-functional behavior at the macula to the periphery, exhibiting apparent long-term stability of the visual acuity, despite peripheral absolute scotoma. The reason for this observed discrepancy remains unclear, but may relate to ultrastructural variations in the retina between the macula and the periphery, such as the anatomical conformation of glial support cells (e.g., MCs). Further identification and characterization of such cases using prospective multimodal anatomico-functional testing may shed more light on the causative mechanism of this interesting and unusual disorder.

**Key words:** peripheral retinoschisis, posterior hyaloid attachment, stellate nonhereditary idiopathic foveomacular retinoschisis.

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