


Recognition, Diagnosis and Treatment of Chorioretinal Folds: Current Perspectives

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Abstract: Chorioretinal folds (CRFs) are undulations of the choroid and overlying Bruch's membrane, retinal pigment epithelium and neurosensory retina. CRFs represent a clinical sign that is mandatory to investigate assuming their association with several ocular and extra-ocular disorders. Recent advances in retinal imaging have improved the characterization of CRFs. More importantly, retinal imaging may be useful to detect ocular complications secondary to chronic CRFs, including the development of choroidal neovascularization.

Keywords: chorioretinal folds, maculopathy, choroid, retina

Introduction

Chorioretinal folds (CRFs) are undulations of the choroid, Bruch's membrane (BM), retinal pigment epithelium (RPE), and the overlying neurosensory retina.

CRFs were first described by Nettleship¹ in 1884 in a patient with papilledema secondary to an intracranial mass. The term "choroidal folds" was successively employed by Norton² in 1968. This term was subsequently modified by Gass³ in "chorioretinal folds" as this term was suggested to better describe the anatomical alterations occurring in this disorder. Since their first description, many other diseases have been ascribed to cause CRFs such as choroidal or orbital tumors, infections, immunologic disorder, high hyperopia, thyroid eye disease, posterior scleritis, uveal effusion syndrome, uveitis, ocular hypotony, and age-related macular degeneration (AMD).

Pathogenesis

Many authors have tried to characterize the pathogenesis of CRFs. In details, it was speculated that CRFs are secondary to a congestion of the choroid,⁴ this causing changes in the shape of BM and RPE.⁵ In 1989, Friberg⁶ proposed that CRFs occur because of the stress-and-strain relationship between choroid and sclera, resulting in a buckling force affecting choroid from either scleral thickening or shrinkage.

Diagnosis

Chorioretinal folds are usually diagnosed during routine ophthalmological examinations in patients without symptoms or undergoing visits because of other disorders. Less commonly, CRFs may cause metamorphopsia, photopsia or vision changes and thus represent the "primum movens" bringing patients to the ophthalmology attention.

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At the fundoscopic examination, CRFs usually appear as alternating yellow and dark lines, that are often arranged in parallel. Based on their arrangement, CRF may be classified in horizontal, oblique, vertical, radiating, and concentric.⁷

Fundus fluorescein angiography (FA) has been historically considered as one of the most sensitive imaging tools for the diagnosis of CRFs. The CRFs' characteristics on FA imaging were first described by Norton² as alternating hypofluorescent and hyperfluorescent bands. In details, hyperfluorescent bands are due to the rarefaction of RPE at the CRF peak. Conversely, hypofluorescent bands are secondary to the redundancy of RPE within the CRFs' pits. Uniformly, CRFs are displayed as alternating hyperautofluorescent and hypoautofluorescent bands on fundus autofluorescence (FAF) imaging (Figure 1).⁸

Indocyanine green angiography (ICGA) findings in CRFs were fully described by Haruyama et al.⁹ In CRFs' cases secondary to orbital tumor, the authors described the

presence of choroidal venous tortuosity, as well as dilatation and loop formation in choroidal vascularization with a delay in the colorant filling of the choroidal vessels. Moreover, in cases of CRFs with associated posterior scleritis, the authors detected the presence of delayed choroidal filling, presence of multiple patches of hypofluorescence, and choroidal venous dilatation. Furthermore, the authors were not able to identify specific ICGA findings in idiopathic choroidal folds and AMD-associated CRFs.

Structural optical coherence tomography (OCT) is currently considered as the most specific imaging tool in the diagnosis and characterization of CRFs. Importantly, OCT has granted an improved differential diagnosis between CRF and retinal folds, that are secondary to other causes (eg epiretinal membrane).^{10–12} On OCT images, CRFs appear as typical undulation of the retina, RPE, and choroid that are easier to detect on perpendicular scans (Figure 1).¹³

More recently, OCT angiography (OCTA) characteristics of CRFs have been described in 3 affected eyes.¹⁴

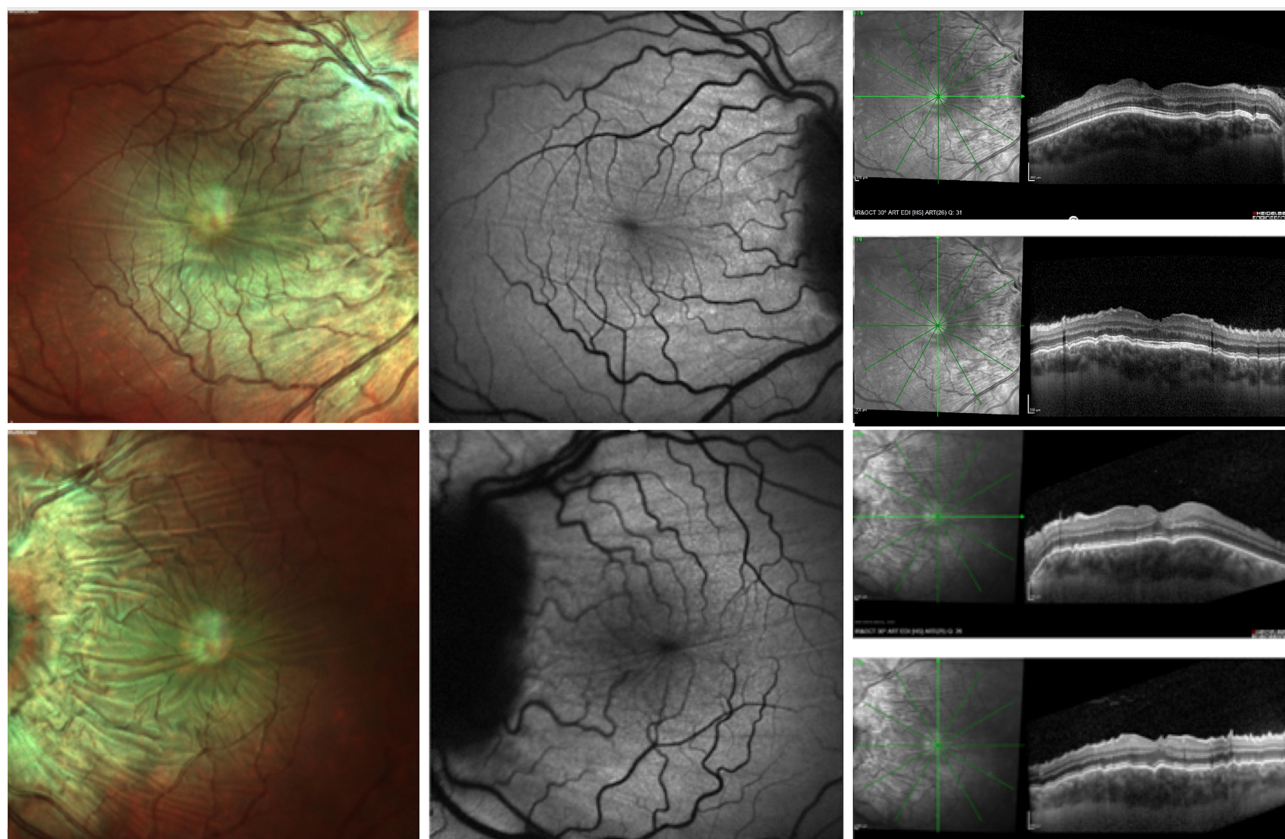


Figure 1 Multimodal imaging from a patient with chorioretinal folds secondary to idiopathic intracranial hypertension. MultiColor fundus images (left) of the right (above) and left (below) eyes demonstrate undulations of the retinal pigment epithelium (RPE) within the macula and around the optic nerve. Blue-fundus autofluorescence (BAF) images (middle) show CRFs as alternating hypoautofluorescent and hyperautofluorescent bands. Structural optical coherence tomography (OCT) images (right) confirm undulations of retina, RPE and choroid that are easier to detect in the perpendicular scans.

Interestingly, the authors identified linear signal decreases in the choriocapillaris perfusion that were topographically associated with the folds (Figure 2). Based on these results, the authors hypothesized that choroidal swelling may cause a stretching of the choriocapillaris and a consequent reduction in perfusion.

Causes of CRFs

Chorioretinal folds may be secondary to ocular and extra-ocular causes.

In 1993, Leahey et al⁷ reviewed 54 patients with CRFs and identified both bilateral and unilateral cases (56% and 44%, respectively). Causes of CRFs were differently represented in unilateral and bilateral cases. Causes as thyroid eye disease, uveitis and choroiditis were more commonly found in bilateral CRFs. Conversely, ocular tumors and trauma were frequent causes of unilateral CRFs. Finally, posterior scleritis, AMD, hypotony and hyperopia were causes of both unilateral and bilateral CRFs.

Ocular Causes

Hyperopia has been identified as one of the main causes of CRFs' formation, as suggested by three important studies with large cohorts.^{7,15,16} In details, hyperopia was identified as the cause of CRFs in 13% to 26% of the total cases, this percentage ranging on the basis of the study cohort.^{7,15,16} Of note, while CRFs may be secondary to primary hyperopia, CRFs may also occur in cases of acquired hyperopia (see below for details).¹⁷

Central serous chorioretinopathy (CSC) has been recently described as an additional cause of CRFs.¹⁸ In the latter study, the authors described a case series of six patients with CSC and CRFs. Importantly, the whole cohort of six eyes had hyperopia. Therefore, the authors concluded that the presence of chorioretinal folds in hyperopic CSC eyes could be associated with an excessive thickness of the choroid occurring in short eyes. Similarly, Corvi et al¹⁹ described a case series of patients with CRFs and choroidal vessel dilatation and hyperpermeability. The

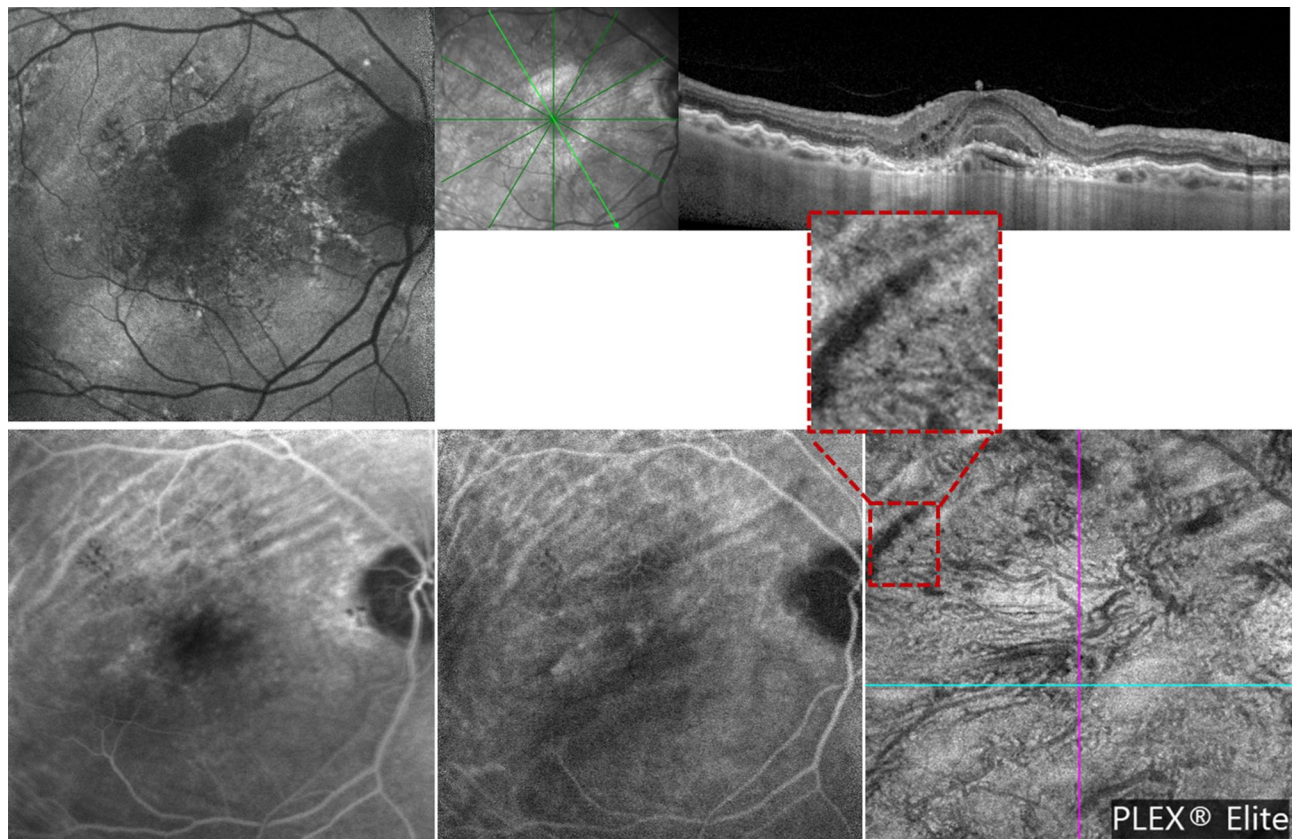


Figure 2 Multimodal imaging from a patient with unilateral chorioretinal folds associated with macular neovascularization in right eye. (Row above): (Left) blue fundus autofluorescence (BAF) image shows linear folds at the macula, easier to detect in the superior region. Structural optical coherence tomography (OCT) image (right) confirms the presence of CFRs. (Row below): The late fluorescein angiography (FA - left) and intermediate indocyanine green angiography (ICGA - middle) images show typical alternating hypofluorescent and hyperfluorescent bands. The optical coherence tomography angiography (OCTA - right) image shows a transversal line of signal reduction corresponding to vascular rarefaction at the choriocapillaris layer.

latter report further suggests the association between CSC – more generally with the pachychoroid spectrum – and the development of CRFs.

Macular neovascularization (MNV)²⁰ represents a well-recognized cause of CRFs. MNV may be secondary to different disorders, including AMD.²¹ The association between CRFs and MNV was first reported by Gass,³ who provided a detailed description of the radial-shaped pattern of MNV-associated CRFs. This pattern of CRFs may be typical in type 1 (or “occult”) MNV and, in the pre-imaging era, CRFs could represent the first sign in presence of MNV. Notably, MNV-associated CRFs were speculated to occur because of the contraction of a fibrovascular membrane adherent to Bruch’s membrane, this resulting in a puckering of the underlying choroid and a series of CRFs spreading outward.³ (Figure 2)

Scleritis is an inflammatory disorder involving the sclera. In cases of involvement of the sclera posteriorly to the insertion of the rectus muscles, the term “posterior scleritis” is usually employed. This form of scleritis has been reported in 2 to 12% of all cases of scleritis.²² Of note, posterior scleritis is usually suspected in presence of symptoms such as periocular pain, blurred vision, and/or headache.²³ Common reported signs include conjunctival chemosis and conjunctival hyperemia.²³ Posterior scleritis may be associated with systemic inflammatory conditions including rheumatoid arthritis and Wegener’s granulomatosis.²³ Also, posterior scleritis may be associated with infectious diseases.²⁴ CRFs may be associated with posterior scleritis as this complication may occur in 28.1 to 35.0% of all cases.^{25–27} Ocular ultrasound should be performed in suspect of posterior scleritis as this examination may display a diffuse or nodular thickening of the sclera, often in presence of retrobulbar and perineural fluid determining the pathognomonic “T-sign”.²⁸ Resolution of CRFs have been reported in several case reports after resolution of scleritis.^{24,29} These findings confirm the stressing role of a thickened sclera to the overlying layers.

Hypotony consists in a transient or permanent reduction in intraocular pressure (IOP) under 5 mmHg.³⁰ Importantly, it may be silent or may be associated with visual loss.³¹ Hypotony may be associated with a reduction in aqueous fluid production such as in cases of iridocyclitis and ciliochoroidal detachment^{32–34} or with an increase in aqueous outflow such as in cases with glaucoma filtering surgery^{35,36} and other surgical procedures (eg vitreoretinal surgeries, corneal transplant, strabismus surgeries, medial canthopexy). The incidence of

hypotony after glaucoma surgery has been reported in 1.3% to 20.0% cases according to different reports. Noteworthy, the use of antimetabolites during filtration surgery, that is aimed at reducing the occurrence of scar,³⁷ has increased the rate of hypotony.^{37,38} Hypotony is an intraoperative and postoperative complication of pars plana vitrectomy. Intraoperatively, the incidence of hypotony can be avoided by limiting the leaks through sclerotomy thanks to cauterization, subconjunctival ophthalmic viscoelastic device (OVD) injection, tissue glue and wound suturing. The postoperative incidence of ocular hypotony is influenced by the underlying pathology. In details, proliferative vitreoretinopathy and inflammatory disorders have a substantial risk of postoperative hypotony.³⁹ In posterior lamellar keratoplasty surgery, hypotony is more a risk factor for graft dislocation than a postoperative complication,^{40,41} mainly in eyes with prior glaucoma surgeries.⁴⁰ Hypotony may clinically result in different complications, including keratopathy, cataract formation, optic nerve edema, irregular astigmatism, choroidal detachment, and hypotony-related maculopathy.^{42–44} CRFs, papilledema and vascular tortuosity are the main features of hypotony-related maculopathy, as described by Dellaporta in 1953.^{42–44} In ocular hypotony, CRFs seem to be secondary to choroidal thickening.⁶

Extra-Ocular Causes

CRFs may also develop in cases of idiopathic or secondary intracranial hypertension. Importantly, CRFs may be the unique sign of idiopathic intracranial hypertension, even in the absence of papilledema. Folds are thought to be secondary to flattening of the posterior pole and dilatation of the optic nerve sheath. It must be emphasized that several papers have reported this association and, therefore, cases of CRFs should be investigated for the presence of idiopathic hypertension.^{45,46} In these cases, a neuroimaging follow-up should be performed.⁴⁷ In absence of imaging suggestive for hypertension, lumbar puncture should be considered in order to further investigate the presence of idiopathic intracranial hypertension.⁴⁸ Remarkably, Kupersmith et al reported the effect of treatment with acetazolamide⁴⁹ on CRFs in patient with idiopathic intracranial hypertension.⁵⁰ The authors did not find significant differences between placebo and treatment groups in terms of CRF prevalence at 6 months. Furthermore, CRFs tend to persist irrespective of the degree of papilledema.

Orbital compression represents another cause of CRFs. Friberg⁶ described the occurrence of CRFs in patients with

orbital tumor. In details, intraconal tumors may displace the optic nerve and the latter may thus compress the choroid and eventually result in CRFs' formation. Conversely, extraconal tumors may directly compress the globe and cause CRFs. Orbital plasmacytoma,⁵¹ enlarged lacrimal gland caused by dacryoadenitis,⁵² frontoethmoidal mucocoele,⁵³ meningioma,^{54,55} cavernous hemangioma,⁵⁶ meningocele,⁴⁶ sinus transverse thrombosis,⁴⁶ orbital pseudotumor^{5,46} are some of the cases reported in the last years. Resolution of CRFs after surgical or medical therapy in orbital masses are unpredictable. In fact, Yeung et al⁵⁴ reported a complete resolution of CRFs after surgical removal of meningioma. On contrary, in case of surgical removal of parasellar meningioma, Taban et al⁵⁵ reported the persistence of CRFs at the one year follow-up.

CRFs may also occur in patients with thyroid eye disease. (TED or Graves' ophthalmopathy). This is an autoimmune disorder of the retrobulbar tissue associated with Graves' disease. The ophthalmic disease is present in up to 50% of all cases of Grave's disease and may have mild to severe manifestations,^{57–60} including CRFs.^{61–66} Mechanisms involved in CRFs' development include exophthalmos, extraocular muscle expansion, and orbital hypertension. Recently, Tran et al⁶⁷ reported on 10 patients (17 eyes) with unilateral or bilateral CRFs secondary to thyroid eye disease. Importantly, treatment of TED resulted in vision improvement in 53% of patients (10/17) and was not correlated with CRFs' resolution or persistence. In agreement, 70% of patients had persistence of CRFs after TED treatment with a mean±SD follow-up of 24.7±23.7 months.

Idiopathic CRFs

Cases without a specific etiology are classified as idiopathic. Recent reports have suggested that idiopathic CRFs represent a percentage of 14–15% of all cases.^{7,15,68} Of note, Olsen et al⁶⁸ speculated that a number of idiopathic CRFs may be actually secondary to silent scleritis, assuming that these patients with idiopathic CRFs are frequently affected by systemic autoimmune disorders.

CRF-Related Maculopathy

Presence of CRFs may eventually result in macular alterations, including ruptures of Bruch's membrane (similar to angiod streaks),⁶ RPE atrophy,⁸ and development of choroidal neovascularization (CNV).⁶⁸ Presence of one of these complications characterizes the CRF-related maculopathy.

The CRF-related maculopathy was characterized by Olsen et al⁶⁸ who described three stages of this

maculopathy on the basis of fluorescein angiography (FA) findings. The stage 3 is characterized by late stippled leakage, this FA pattern resembling presence of type 1 CNV. Assuming that type 1 CNV may characterize the CRF-related maculopathy (in approximately 10% of cases), the differential diagnosis between CNV and stage 3 maculopathy may be challenging as in both cases FA may display the presence of leakage. In these cases, structural OCT may be helpful in order to detect the presence of CNV. Choroidal neovascularizations in CRF-related maculopathy may be treated with anti-VEGF intravitreal injections.¹⁹ Conversely, the stage 3 CRF-related maculopathy, that also may be characterized by subretinal or intraretinal fluid, even in the absence of CNV, is less responsive to anti-VEGF injections.¹⁹ In these cases, the intravitreal injection of dexamethasone may be effective.¹⁹

Conclusions

Chorioretinal folds represent a quite frequent disease of the posterior eye. This disease should not be underestimated by ophthalmologists, as different ocular and extra-ocular causes may result in CRFs' formation. Importantly, the presence of CRFs may eventually result in a secondary maculopathy, that must be recognized and appropriately treated.

Disclosure

Enrico Borrelli is advisor for: Zeiss (Dublin, USA), Centervue (Padua, Italy). Riccardo Sacconi is advisor for: Zeiss (Dublin, USA), Novartis (Basel, Switzerland); reports personal fees from Zeiss and Novartis, outside the submitted work. Francesco Bandello is a consultant for: Alcon (Fort Worth, Texas, USA), Alimera Sciences (Alpharetta, Georgia, USA), Allergan Inc (Irvine, California, USA), Farmila-Thea (Clermont-Ferrand, France), Bayer Shering-Pharma (Berlin, Germany), Bausch And Lomb (Rochester, New York, USA), Genentech (San Francisco, California, USA), Hoffmann-La Roche (Basel, Switzerland), Novagali Pharma (Évry, France), Novartis (Basel, Switzerland), Sanofi-Aventis (Paris, France), Thrombogenics (Heverlee, Belgium), Zeiss (Dublin, USA); reports personal fees from ALLERGAN, outside the submitted work. Giuseppe Querques is a consultant for: Alimera Sciences (Alpharetta, Georgia, USA), Allergan Inc (Irvine, California, USA), Amgen (Thousand Oaks, USA), Bayer Shering-Pharma (Berlin, Germany), Heidelberg (Germany), KBH (Chengdu; China), LEH Pharma (London, UK), Lumithera (Poulsbo; USA), Novartis (Basel, Switzerland), Sandoz (Berlin, Germany),

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