A Retrospective Case Series of Uveal Effusion Syndrome

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Purpose: This study aimed to describe the clinical findings and management of eyes affected by uveal effusion syndrome.

Methods: We retrospectively evaluated the charts of 13 eyes of 8 consecutive patients diagnosed with uveal effusion syndrome attending the Ophthalmology Unit of the University Hospitals Leuven, Belgium, between 2007 and 2018. The presenting features, investigations, management, and outcomes were analyzed for each case.

Results: Cataract surgery was the predisposing factor for uveal effusion in 6 eyes, 2 bilateral uveal effusions (4 eyes) were considered to be medication-induced, and in 3 eyes, the uveal effusion was described as idiopathic. Fundus examination of 5 of 13 eyes showed bullous choroidal detachment, treated with pars plana vitrectomy with super temporal sclerectomy or transscleral puncture. Fundoscopy showed uveal effusion without serous retinal detachment in 3 eyes. Serous retinal detachment accompanied by uveal swelling was observed in 3 eyes and the 2 remaining eyes presented with uveal swelling only. The 8 nonbullous choroidal detachments were treated in a conservative way. A rapid resolution of subretinal fluid and uveal effusion was observed in all cases.

Conclusions: A conservative approach with acetazolamide treatment or just observation was used in our case series in choroidal detachment without substantial visual loss if, over time, slow improvement was documented. However, further studies are needed to verify the effectiveness of the reported therapy.

Key Words: uveal effusion syndrome, choroidal effusion, hyperopia, serous retinal detachment, choroid

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Uveal effusion is an abnormal accumulation of transudative fluid from the choriocapillaris into the suprachoroidal space. This can result in a choroidal swelling, a choroidal detachment, subretinal fluid and a secondary serous retinal detachment, and, finally degeneration of the retinal pigment epithelium (RPE). Uveal effusion is a clinical sign rather than a diagnosis. A uveal effusion due to hypotony, intraocular inflammation, an intraocular tumor, and medication should be described as forms of secondary choroidal detachments. Other terms for this type of uveal effusion have been used conversely: choroidal effusion, ciliochoroidal effusion, ciliochoroidal detachment, and choroidal detachment.

In 1858, Von Graefe,1 and in 1925, Verhoeff and Waite3 described a spontaneous serous detachment of the choroid. Schepens and Brockhurst4 defined the uveal effusion syndrome (UES) as a nanophthalmic disorder with a congenital scleral abnormality causing a thickened sclera that results in congestion of the choroidal venous system due to vortex vein compression.5,6 Gass7 and Gass and Jallo8 hypothesized that, although the abnormality is congenital, aging and hormonal changes result in a further impairment of the permeability of the sclera, causing a decompensation and, as a consequence, an idiopathic uveal effusion. Trelstad et al9 found that the sclera showed histologic and histochemical abnormalities. A proliferation and migration of RPE cells in the subretinal space of patients diagnosed with UES were described by Forrester et al.10 They proposed that the leopard-spot changes in the fundus of UES correlated to the foci of RPE proliferations and multilayering.

Uyama and colleagues divided UES into 3 groups on the basis of pathogenesis, axial length (AXL) of the eye, and scleral thickness (ST). Type 1 UES was a choroidal effusion in a nanophthalmic eye with an AXL <19 mm, high-grade hyperopia, and a thick sclera. Type 2 UES had a thick sclera, but was not associated with nanophthalmos or hyperopia. Histologically, types 1 and 2 demonstrated abnormal sclera with disorganization of collagen fiber bundles and proteoglycan deposits in the matrix. In contrast, type 3 UES showed a normal AXL and a normal sclera. They postulated that a preoperative classification is essential for early surgical management.11

Brockhurst6 introduced the first treatment for UES. He reported an effective surgical procedure for decompression of the vortex veins using sclerectomy. Because isolating the vortex veins is a rather difficult technique with substantial possible complications, Gass7 described good surgical results with sclerectomy and sclerostomy without decompression of the vortex veins.

Case series on UES are sparse in the literature; the clinical findings and subsequent management of affected eyes often vary. We describe the clinical findings of 8 consecutive patients diagnosed with UES and compare their management.

METHODS

This was a retrospective, single-center, observational study carried out in patients attending the Ophthalmology Unit of the University Hospitals Leuven between 2007 and 2018. We reviewed the charts of 13 eyes of 8 consecutive patients diagnosed with UES. Appropriate institutional review board and ethics approval were obtained for the study.

Inclusion criteria were defined as follows: (1) peripheral ciliochoroidal detachment on fundoscopy or ultrasound with or without the presence of a serous retinal detachment; (2) other causes of ciliochoroidal detachment such as hypotony, intraocular inflammation, intraocular tumor, and rhegmatogenous retinal detachment were excluded.

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RESULTS

Relevant clinical features including age, sex, presenting features, best-corrected visual acuity, refractive status, biomicroscopy, intraocular pressure (IOP), AXL, ST, UES classification, treatment, and final visual acuity and refractive error of all 8 reported cases are shown in Supplemental Table 1 (Supplemental Digital Content 1, http://links.lww.com/IJG/A426)12. In our case series, the mean age was 62 years (range, 34 to 77 y) and 4 patients were men (7/13 eyes). Complaints of blurred vision with a myopic shift and anterior displacement of the lens-iris diaphragm were observed in 4 patients, 3 patients presented with symptoms of bilateral acute angle-closure glaucoma (AACG), 1 was referred with suspicion of an intraocular tumor, but ultrasound examination showed a retinal detachment with choroidal swelling and no intraocular masses, and another was referred with a serous retinal detachment after cataract surgery. We observed involvement of both eyes in 5 of 8 patients. Fundus examination of 5 of 13 eyes showed bullous choroidal detachment. Three eyes showed uveal effusion without serous retinal detachment, 3 eyes had serous retinal detachment accompanied by uveal swelling, and 2 eyes presented with uveal swelling only. Leopard-spot changes of chronic serous retinal detachment were seen in 2 patients (Fig. 1). B-scan ultrasonography was used to confirm the diagnosis in all cases (a representative example is shown in Fig. 2).

The uveal effusion was seemingly triggered by cataract surgery in 6 patients. Two uveal effusions were thought to be the result of the recent intake of medication: 1 patient underwent chemotherapy with oxaliplatin (Eloxatin; Sanofi, Paris, France) for her metastasized colon carcinoma and another had recently used topiramate (Topamax; Ortho-McNeil Pharmaceutical, Raritan, NJ) for migraine. In all, 3 eyes were diagnosed with idiopathic uveal effusions.

In our case series, 5 eyes with substantial visual loss were instantly treated with pars plana vitrectomy to manage the retinal detachment in conjunction with internal drainage of subretinal fluid. After introduction of perfluorocarbon liquid (DORC, Zuidland, The Netherlands), disappearance of the choroidal effusion was observed in a myopic patient with normal ST. The other 2 hyperopic patients with increased ST were treated with pars plana vitrectomy in conjunction with a superotemporal sclerotomy or superotemporal transscleral puncture to reduce the choroidal effusion. Resolution of the subretinal fluid and choroidal effusion was rapid with a conservative treatment consisting of acetazolamide or observation in 8 of 13 eyes reported here.

DISCUSSION

UES is an extremely rare disease characterized by choroidal fluid collections, often associated with shifting subretinal fluid and subsequent RPE changes. The causes of primary and secondary choroidal effusions are listed in Table 1 (readapted from Elagouz et al1 with the addition of specific drug-induced choroidal effusions). When the etiology remains unknown, the term idiopathic UES is used.1,8 In our case series, the uveal effusion was seemingly triggered by cataract surgery in 6 patients; choroidal effusion due to hypotony was excluded. Two uveal effusions were considered to be

| TABLE 1. Causes of Uveal Effusion |
|----------------------------------|
| **Inflammatory**                 |
| Trauma and intraocular surgery   |
| Scleritis and infected scleral buckle |
| After cryotherapy and photocoagulation |
| Chronic uveitis, pars planitis, Vogt-Koyanagi-Harada disease, and sympathetic ophthalmia |
| **Hydrostatic**                  |
| Hypotony and wound leak          |
| Hunter syndrome                  |
| Dural arteriovenous fistula      |
| **Drug-induced**                 |
| Topiramate                       |
| Acetazolamide                    |
| Hydrochlorothiazide              |
| Venlafaxine                      |
| Indapamide                       |
| Methazolamide                    |
| Bupropion                        |
| Cabergoline                      |
| XTC                              |
| Escitalopram                     |
| Fluoxacillin                     |
| Sulphasalazine                   |
| Oxaliplatin                      |
| Primary angle closure glaucoma   |
| Uveal effusion syndrome          |
| Hypermetropic or nanophthalmic   |
| Idiopathic                       |

FIGURE 1. Leopard spots.

FIGURE 2. B-scan ultrasound: annular bullous choroidal detachment. Figure 2 can be viewed in color online at www.glaucomajournal.com.
secondary to the recent intake of medication and 1 patient underwent chemotherapy with oxaliplatin for her metastasized colon carcinoma; to our knowledge, this was previously described with cisplatin therapy, another platinum-containing agent.13 Another patient had recently used topiramate for his migraine. Craig et al14 previously reported the mechanism of topiramate-induced acute-onset myopia and angle-closure glaucoma. Murphy et al15 published a comprehensive review of the full spectrum of drugs implicated in drug-induced bilateral AAGC through the uveal effusion mechanism using standardized criteria.

Several hypotheses on the pathogenesis of UES exist. Schepens and Brockhurst4 defined the UES as a nanophathalmic disorder with a congenital scleral abnormality causing a thickened sclera, which results in engorgement of the choroidal venous system.5,6 The eyes of our patients did not fulfill the defined criteria for nanophthalmos and were classified as hyperopic; 2 patients had an increased ST.

Gass7 proposed an alternative for the hydrostatic hypothesis as described above. They postulated that a reduced scleral protein permeability could result in retained fluid in the suprachoroidal space. Bill16 has shown that in healthy individuals, albumin leaves the eye mainly via a transscleral route. For this reason, the colloid osmotic pressure in the suprachoroidal space is effectively zero. If this transscleral route of albumin is somehow impaired, fluid could be retained in the suprachoroidal space. Histologic studies have shown reduced transscleral diffusion in the sclera of patients with UES caused by an abnormal accumulation of glycosaminoglycan-like material expanding the interstitial spaces.9,17 A direct measurement of scleral permeability was performed by Jackson et al.20 They tested transscleral diffusion of high-molecular-weight molecules in scleral tissue removed during surgery from UES patients. Jackson et al21 also tested the hypothesis that UES is the result of a reduced scleral hydraulic conductivity. Their findings suggested that an increased ST is unlikely to significantly impede the scleral permeability to water. Daniele and Schepens22 introduced a fourth hypothesis. They reported 2 cases in which primary hyptony can induce UES in an otherwise normal-sized eye with no morphologic abnormalities. Elagouz et al12 commented on this hypothesis that most patients in UES have normal IOP. The fifth hypothesis, put forward by Kumar et al,23 suggests that an increased permeability of the choroidal vasculature, caused by a nonspecific choroidal inflammation, is the trigger for UES. Finally, Elagouz et al12 concluded that multiple factors can contribute to the pathogenesis of UES, although the relative contribution of each factor may vary in patients affected by UES.

UES usually affects healthy individuals in middle age, with predominance among men, and is usually bilateral. In our case series, men were affected in 7 of the 13 eyes and 3 times the UES was bilateral. Patients frequently present with complaints of loss of visual field or acuity. A myopic shift with anterior displacement of the lens-iris diaphragm is frequently observed, as was the case in our case series in 4 patients. Slit-lamp biomicroscopy generally shows a shallow anterior chamber with or without angle closure. IOP is usually normal, but can also be elevated, mimicking AAGC.1 Areiter et al24 recently reported the spectrum of angle closure, UES, and nanophthalmos. In our case series, 2 patients presented with the symptoms of AAGC. This clinical presentation has been previously described.13,14,25,26 The early signs of uveal effusion are thickening and engorgement of the choroid, ciliochoroidal detachment, and secondary serous retinal detachment. An observation that we made is that no real choroidal detachment was present in 3 patients. Rather, the choroid was swollen as diagnosed by ultrasonography. Other clinical findings may include mild dilatation of the episcleral vessels, blood in the Schlemm canal, and mild inflammation in the vitreous.1 Classic late signs include changes in the RPE, called leopard spots, as were seen in our case series in 2 patients.10 This pattern of pigment clumping in the fundus is typical but not specific, considering it can be a result of other conditions causing chronic choroidal elevation.27

UES is a diagnosis of exclusion and multiple other conditions have been mistaken for UES; therefore, UES still represents a diagnostic challenge. The entire differential diagnosis of serous retinal detachment is shown in Table 2 (readapted from Elagouz et al12).

Brockhurst et al12 described favorable surgical results with vortex vein decompression performed by scleral resection with scleral undermining. Considering that the isolation of vortex veins is a very complicated procedure and the decompression is technically difficult to perform without complications, such as vein rupture, other authors reported surgical techniques without vortex vein decompression. These procedures include performing scleral thinning procedures, called partial sclerectomies, with or without making scleral openings, known as sclerostomies or sclerorhinostomies.7,28–31 The scleral openings were made without considering the anatomic location of the choroidal effusion. Medical treatment including mydriatics, cycloplegics, and steroids before the standard recommended surgical approach has been described several times. However, the treatment is often not successful. A conservative therapy with nonsteroidal anti-inflammatory drugs has also been reported with rare success.23 Kerstetter et al12 hypothesized that prostaglandins analogs could increase the scleral permeability by increasing the uveoscleral outflow. This hypothesis was confirmed by Weinreb.32 Moldow et al34 reported a positive clinical response in UES patients with acetazolamide. In 2014, Derk et al35 reported good results with a conservative therapy consisting of the combination of prostaglandin analogs and acetazolamide. However, acetazolamide can also induce an uveal effusion (Table 1) and should be anamnestically excluded as the causative factor. In our experience, acetazolamide is highly effective. If the UES worsens under therapy with acetazolamide, one should be aware of the potential side effect of this medication. Recently, Park and Lee26 confirmed that medical therapy can be of value in the treatment of UES before surgical treatment. Shields et al37 reported in 2017 that oral, periocular, topical, or a combination of corticosteroids provided control of UES in 95% of cases in 104 eyes with UES. They conclude that corticosteroids can be considered a treatment, in the absence of nanophthalmos and/or ST abnormalities. These patients were, according to the Uyama classification,15 classified as UES type 3 and surgical approaches primarily involving the sclera would not seem to be necessary. It is interesting to note that resolution

### Table 2. Differential Diagnosis of Serous Retinal Detachment

| Condition                                           |
|-----------------------------------------------------|
| Chronic central serous chorioretinopathy            |
| Posterior scleritis                                  |
| Rhegmatogenous retinal detachment with uveal detachment |
| Multifocal choroiditis                               |
| Uveal melanoma                                       |
| Metastatic tumor                                     |
| Severe hypertensive choroidopathy                    |
| Vogt-Koyanagi-Harada disease                         |
| Systemic diseases (myxedema, multiple myeloma)      |

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of the subretinal fluid and choroidal effusion was rapid with a conservative treatment in 8 of 13 eyes reported here. Our case series presents a conservative approach with acetazolamide, that may be considered in UES type 3 without substantial visual loss if no worsening or slow improvement can be documented on subsequent follow-up visits. Nevertheless, further case-series studies with UES patients are needed to determine the safety and effectiveness of this therapy.

In summary, we present the clinical findings and management of eyes affected by UES. UES is a rare disease usually associated with hyperopia and caused by an underlying abnormality of the sclera resulting in the dysfunction of choroidal fluid dynamics. It still represents a diagnostic challenge considering a ciliochoroidal effusion can be the consequence of numerous inflammatory and hydrostatic ocular conditions; therefore, idiopathic UES is a diagnosis of exclusion. The condition is difficult to manage and it can result in severe visual loss due to chronic submacular fluid and secondary RPE changes.

Management of UES is complex and based on disease severity. A conservative approach with acetazolamide treatment or just observation was used in our case series in choroidal detachment without substantial visual loss if, over time, slow improvement was documented. Limitations include that effective subclassification was not possible considering that not all patients had undergone the same diagnostic investigations and we did not perform an internal nor a neurological examination, including magnetic resonance imaging of the brain and orbit, to rule out all possible inflammatory and hydrostatic causes of uveal effusion. Further case-series studies are needed to explore the various causative mechanisms of UES, and to determine whether treatment can be tailored to a given patient.

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