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Clinical features, diagnosis and management of serpiginuos choroiditis

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ABSTRACT

Classic serpiginous choroiditis (CSC) is a rare bilateral, multifocal, recurrent inflammatory disease of the choroid and retina observed mainly in young and middle-aged male patients. We present three cases of otherwise healthy men (ages 30, 52, and 54 years) with the diagnosis of CSC based on clinical and multimodal imaging findings. During the observation period, slow progression of the fundus changes was noted in each patient despite various individualized immunosuppressive treatments (corticosteroids, cyclosporin, cyclophosphamide, azathioprine) used. Progressive macular involvement was associated with a significant irreversible bilateral reduction in visual acuity. The aim of the study is to present the clinical features of CSC, possible diagnostic and therapeutic options, and to familiarize the reader with specific clinical cases.

KEY WORDS: classic serpiginous choroiditis (CSC); fluorescein angiography; indocyanine green angiography; macular involvement; permanent damage of the retina; immunosuppressive treatment

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INTRODUCTION

Classic serpiginous choroiditis (CSC) is a rare, idiopathic chorioretinopathy with the inflammation involving primarily the choroid with secondary retinitis. In very few cases, involvement of the vitreous body or the anterior segment was reported [1].

The entity was first reported in 1932 by Paul Junius, who named it "retinochoroiditis parapapillaris" [2]. In 1983, Gass coined the term "serpiginous choroiditis" to describe the characteristic lesions in the fundus [3]. The etiology of CSC remains unclear. Noninfectious inflammation involving the choriocapillaris with subsequent occlusion and secondary damage to the adjacent structures has been implicated [4]. The postmortem findings of infiltrates of lymphocytes in the choroid, adequate response to immunosuppressive agents, and occasional accompanying inflammation of the anterior segment and the vitreous body have suggested the possible involvement of autoimmune processes [5]. With advances in molecular medicine, an infectious subtype of tuberculosis-related serpiginous-like choroiditis (SLC) has been identified as the genetic material of *Mycobacterium tuberculosis* was isolated from the samples of the choroid body and the aqueous humor from some patients with serpiginous choroiditis [4].

The disease typically affects young and middle-aged patients, more frequently males, usu-

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ally in good general health [6]. Single cases of CSC have been reported in Caucasian women [7], and there is no race predilection nor familial occurrence [8].

The course of CSC is characterized by remissions and exacerbations leading to irreversible retinal damage, resulting in permanent visual acuity deterioration. The time between new episodes of activity varies from a few months to several years [7, 9]. CSC occurs bilaterally and asymmetrically.

Patients present with a sudden, painless deterioration in visual acuity, initially in one eye, experienced as blurry vision, central scotomas, and metamorphopsia. Examination of the anterior segment and the vitreous body usually is unremarkable. Ophthalmoscopy reveals creamy white, ill-defined active lesions that are geographic in shape and commonly located in the posterior pole or the peripapillary region. During subsequent episodes, the lesions progressively spread in a serpentine pattern. Within 2-8 weeks, the acute lesions subside, leaving irreversible changes such as defects of the retinal pigment epithelium, chorioretinal atrophy, and scarring resulting in permanent vision deterioration. With damage to the macular Bruch's membrane, subretinal neovascularization develops in some cases [1, 4, 10].

Visual loss is correlated with the proximity of the lesions to the fovea [4, 9], and numerous active episodes may remain undiagnosed when the central macula is not involved. As a result, in over two-thirds of patients, the first ophthalmoscopy reveals inactive scars in the mid- and far peripheral retina in one or both eyes [10, 11].

Additional examinations that aid in the final diagnosis of CSC include a comparison of color eye fundus photographs, fluorescein angiography (FA) and indocyanine angiography (IA), optical coherence tomography (OCT) of the macula [7], and optical coherence tomography angiography (OCTA) [12].

The first step is to differentiate CSC from SLC and, to rule out the latter, chest radiography, tuberculin skin test, and/or QuantiFERON TB-2G test are performed and, if required, the patient is assessed by a pulmonologist [13]. The clinical picture of SLC differs from that of CSC. The lesions are located not in the peripapillary area but in the peripheral retina. They are multifocal, dispersed, and not confluent. More frequently, inflammatory cells are found in the vitreous. Mainly occurs in younger patients [14].

CSC's etiology is not fully understood, so there is no standard treatment. Systemic (monotherapy or combination therapy) and topical treatments are used. The systemic treatments include glucocorticoids, alkylating and non-alkylating immunosuppressive agents, and biological therapies or their combinations. Intravitreal injections of anti-vascular endothelial growth factor (anti-VEGF) are administered [9, 15–20]. The goal of treatment is to prevent new episodes of activity and further inflammatory involvement of the retina. Our present knowledge of the disease makes it possible to slow but not stop its progression.

In SLC, antituberculosis prophylactic treatment must be initiated before immunosuppressive therapy. However, the effect of antituberculosis treatment on eye disease may be delayed by 10 to 14 weeks. When new active lesions develop during that period, systemic immunosuppressive therapy may be prescribed. Also, topical treatments are considered, e.g., intravitreal methotrexate injections, to avoid the potential risk of tuberculosis reactivation [21].

In CSC, the visual prognosis is poor. With time extensive atrophic scars develop in the problematic areas. Chorioretinal atrophy and damage to the Bruch's membrane increase the risk for subretinal fibrocellular proliferations, which may cause further macular damage. As a result of these complications, most patients suffer significant vision loss in the affected eyes.

CASE PRESENTATIONS

Patient 1 (6-year-long observation)

A 52-year-old otherwise healthy man was initially treated in his local eye clinic for the condition diagnosed as ocular toxoplasmosis and was prescribed clindamycin 300 mg TID, sulfamethoxazole + trimethoprim 960 mg/day, and prednisone 10 mg/day. As the treatment was ineffective and his vision continued to deteriorate, he was referred to the Eye Clinic at the Infant Jesus Clinical Hospital in Warsaw.

On admission, the patient complained of distorted vision and a central scotoma in the left eye. He had experienced an impaired vision in the right eye for many years, but it remained undiagnosed. At presentation, distance best-corrected visual acuity (BCVA) was 0.16 in the right eye and 0.1 in the left eye. Near BCVA was "can't read" in the right eye and 1.25 in the left eye. There were no abnor-

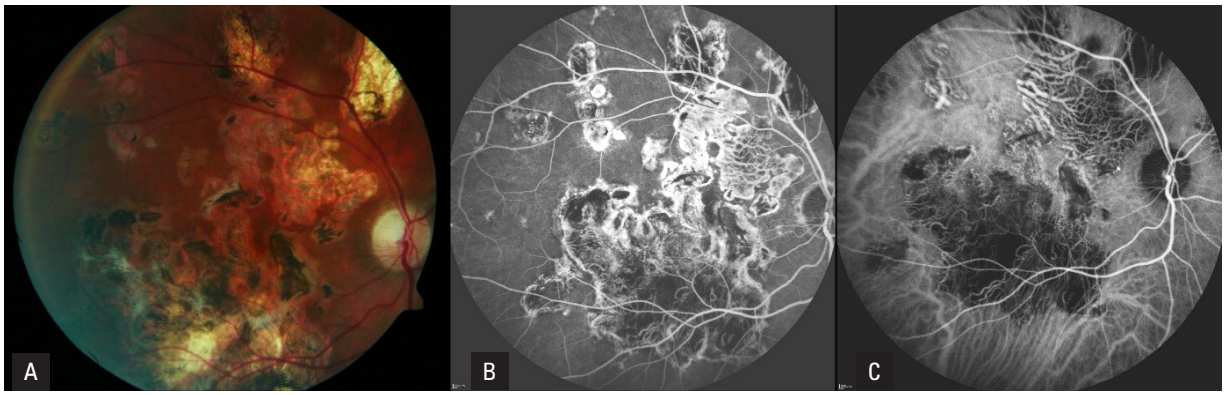

FIGURE 1. Patient 1: Macula of the right eye showing extensive chorioretinal scars with visible large choroidal vessels; **A.** Color photograph; **B.** Fluorescein angiography; **C.** Indocyanine green angiography

malities in the anterior segment. Indirect ophthalmoscopy with a +90-diopter lens demonstrated in the right eye extensive post-inflammatory scars in the posterior pole, serpentine in shape and with preserved continuity (Fig. 1A).

The left eye revealed an ill-defined, irregularly shaped, creamy white lesion in the central macula and post-inflammatory scars in the temporal macula (Fig. 2A).

Fluorescein angiography demonstrated extensive chorioretinal areas of atrophy in the right eye (Fig. 1) and in the macula of the left eye, an expanding area of hypofluorescence with inflammation-associated diffusion of the dye and atrophic scars on the temporal side (Fig. 2CD). Additionally, indocyanine angiography revealed in the left eye hypofluorescence in the macula in the areas of inflammatory activity, temporal to the scarred macula (Fig. 2B).

OCT of the macula revealed the disorganization of the retinal layers in the right eye and small cystic changes indicative of intraretinal edema, scarring, and retinal pigment endothelium defects in the left eye (Fig. 5AB).

Serpiginous choroiditis was diagnosed based on clinical and multimodal imaging findings. A negative QuantiFERON Gold test, chest radiography, and a consultation by a pulmonologist excluded the diagnosis of infectious diseases, including tuberculosis. Initially, the patient was started on oral prednisone 60 mg/day, which he continued for 8 months. As the disease progressed, cyclosporine was additionally prescribed at the starting dose of 250 mg per day, divided into two even doses, and monitoring cyclosporine blood levels was recommended. Vision in the left eye improved to distance BCVA 0.7 with appropriate head positioning and near vision of 0.5. The patient had regular monthly assessments for 2 years for the slow progression of changes in the left eye which ultimately extended beyond the superior temporal vascular arcades (Fig. 4A). Cyclophosphamide was prescribed as add-on therapy. From November 2018 to May 2019, the patient received pulse cyclophosphamide intravenously (800–1000 mg) for 5 doses, and no adverse side effects were observed. However, regular monitoring of cyclophosphamide treatment disclosed raised erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) levels and a suspected lung tumor detected on chest radiography, which was subsequently ruled out by computed tomography (CT). Cyclophosphamide was discontinued for the duration of lung lesion assessment, and prednisone 5 mg was prescribed. With cyclophosphamide discontinuation, the lesions in the left eye progressed toward the fovea, with the resulting deterioration in visual acuity to no better than BCVA 0.16. Another treatment given with prednisone at a starting dose 60 mg, cyclosporine at the starting dose of 250 mg per day, divided into two even doses and monitoring of cyclosporine blood levels, and azathioprine 150 mg/day. All treatment regimens only slowed the progression of lesions in the left eye. The findings in the right eye remained unchanged over 6 years of close monitoring, including BCVA at 0.16. In the left eye, BCVA improved to 0.3 with appropriate head positioning.

Patient 2 (21-year-long observation)

A 54-year-old otherwise healthy man was first admitted to the Clinical Department of Ophthalmology of the Medical Academy of Warsaw (now Warsaw Medical University) in 1999 for a sudden, painless deterioration of vision in the right eye experienced over the past few days. BCVA was 0.4

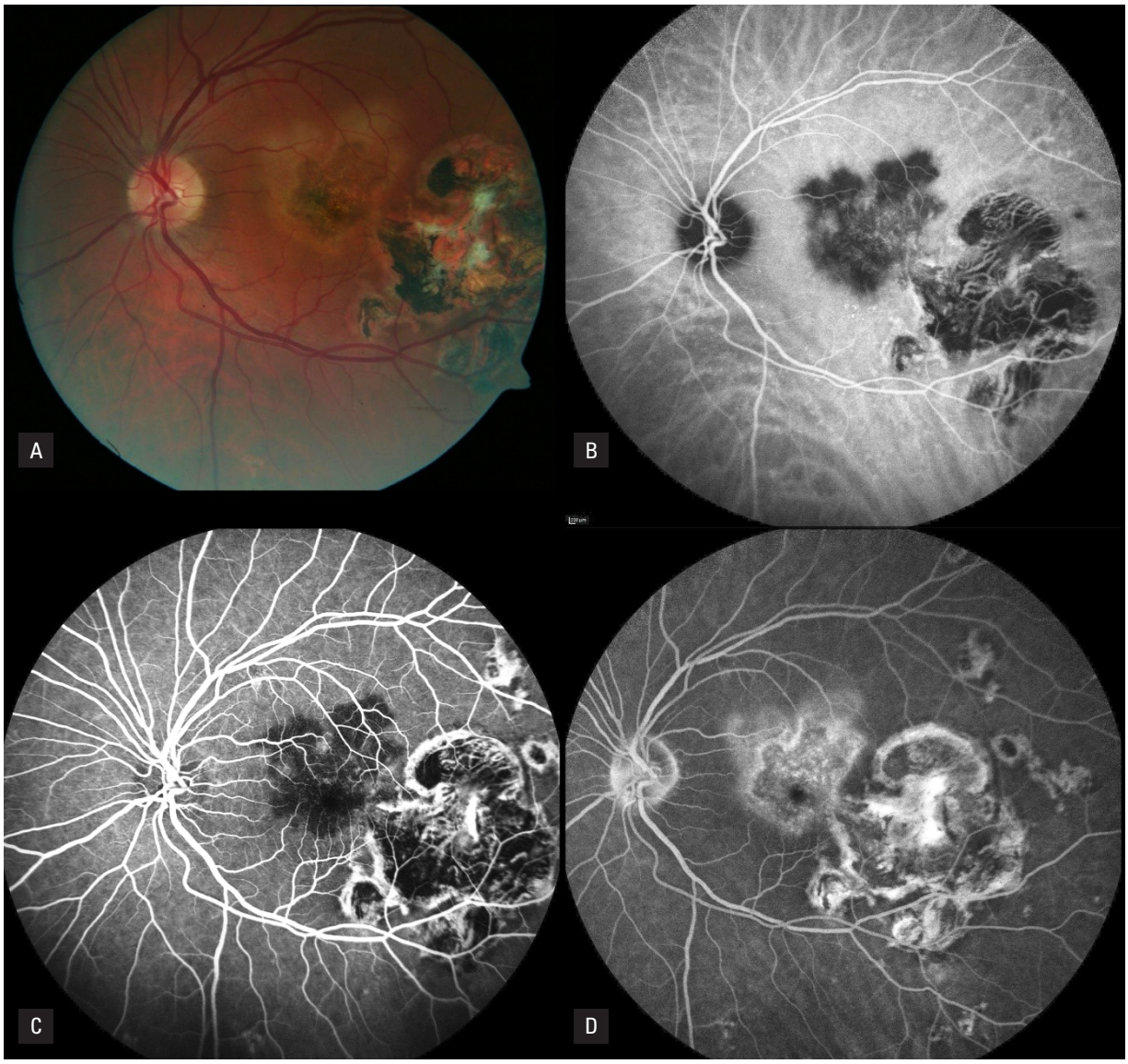

FIGURE 2. Patient 1: Macula of the left eye showing active inflammatory lesions, atrophic scars temporal to the macula with visible large choroidal vessels, and hyperpigmentation in the retina; **A.** color photograph; **B.** Indocyanine green angiography; **C.** Fluorescein angiography (early phase), d) fluorescein angiography (late phase)

in the right eye, and visual acuity in the left eye was normal. The anterior segment and the vitreous chamber were unremarkable. Ophthalmoscopy revealed ill-defined, finger-like creamy white lesions in the posterior pole of the right eye and below the optic disc in the left eye. Bilateral serpiginous choroidopathy was diagnosed, and tuberculosis was excluded. Systemic treatment with prednisone at a starting dose of 60 mg and therapeutic drug monitoring (TDM)-guided cyclosporine dosing were prescribed, and the patient was monitored at his local eye clinic.

In 2002, the patient again presented to the Clinical Department of Ophthalmology with deterioration in visual acuity and "a dark spot" in his vision in his left eye. In the right eye, BCVA 0.4 remained at the same level from 1999. In the left eye, BCVA was found to have deteriorated to 0.4. Ophthalmoscopy demonstrated in both eyes extensive areas of chorioretinal atrophy involving the posterior poles and peripheral retina (Fig. 7A and 8A). OCT of the macula in both eyes revealed generalized macular thinning (Fig. 9AB). The patient had not taken any treatment for a few months. He was prescribed oral prednisone alone at a starting dose of 60 mg/day, but refused retreatment with cyclosporine for self-reported side effects, muscle tremors, and hypertension. As earlier cyclosporine dos-

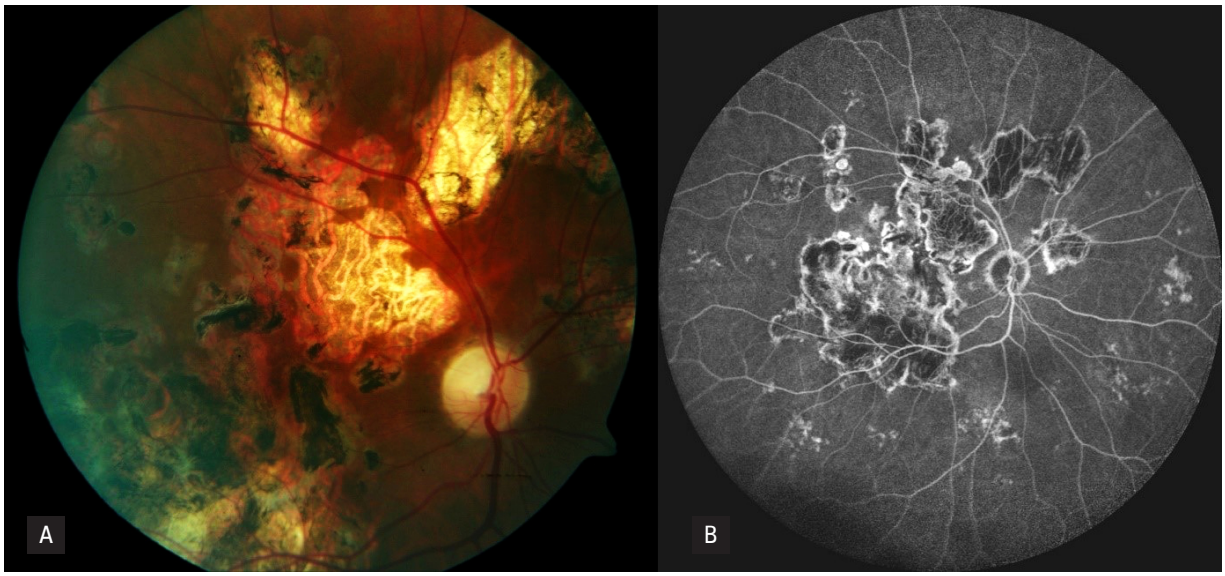

FIGURE 3. Patient 1: The right eye at three after the first assessment; the image remained the same as at presentation; **A.** Color photograph; **B.** Widefield fluorescein angiography taken using a 102° lens

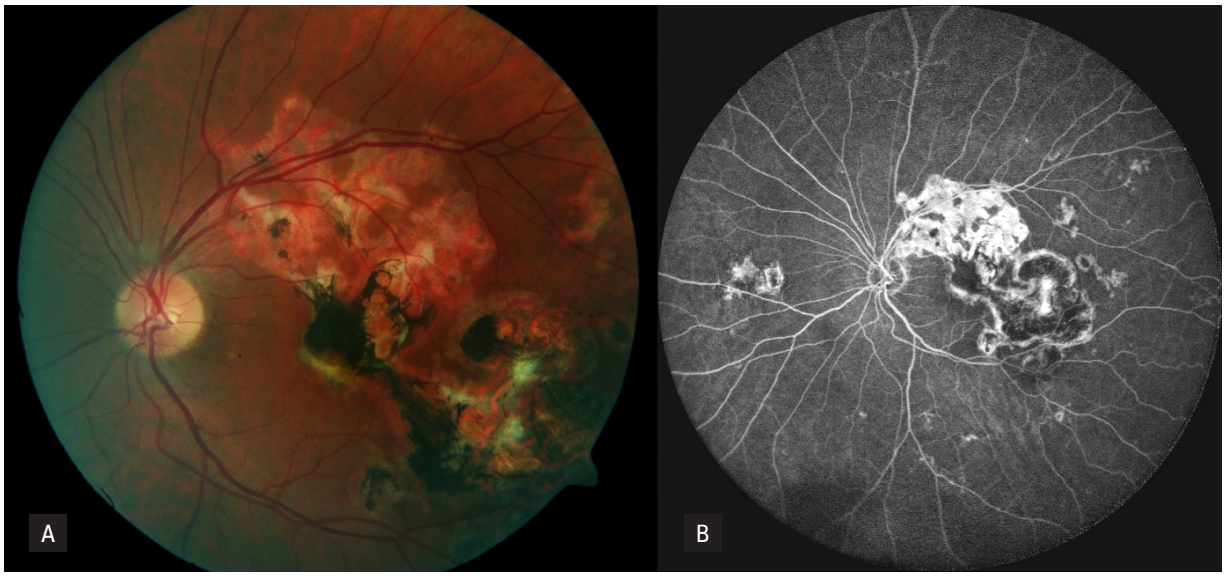

FIGURE 4. Patient 1: The left eye at three years after the first assessment; post-inflammatory scars with pigment in the macula and healed inflammatory lesions which appear as an extensive area of atrophy in the upper part of the macula. **A.** Color photograph; **B.** Widefield fluorescein angiography using a 102° lens

ing was not TDM-guided, the adverse side effects were probably due to cyclosporine overdose.

The patient decided to discontinue prednisone without consulting his doctor because of rapid weight gain. After 6 weeks, he had disease reactivation and presented with progressive deterioration in visual acuity in the right eye. At presentation, BCVA was 0.1 in the right eye and 0.4 in the left eye. The right eye fundus displayed diffuse areas of chorioretinal atrophy and an active inflammatory lesion at the optic disc. A two-drug regimen was prescribed, oral prednisone at a starting dose of 60 mg per day + TDA-guided cyclosporine dosing starting at 300 mg per day, divided into two even doses. Remission was achieved after 3 months with improvement in visual acuity in the right eye to BCVA 0.5 with appropriate head positioning and to BCVA 0.6 with appropriate head positioning in the left eye.

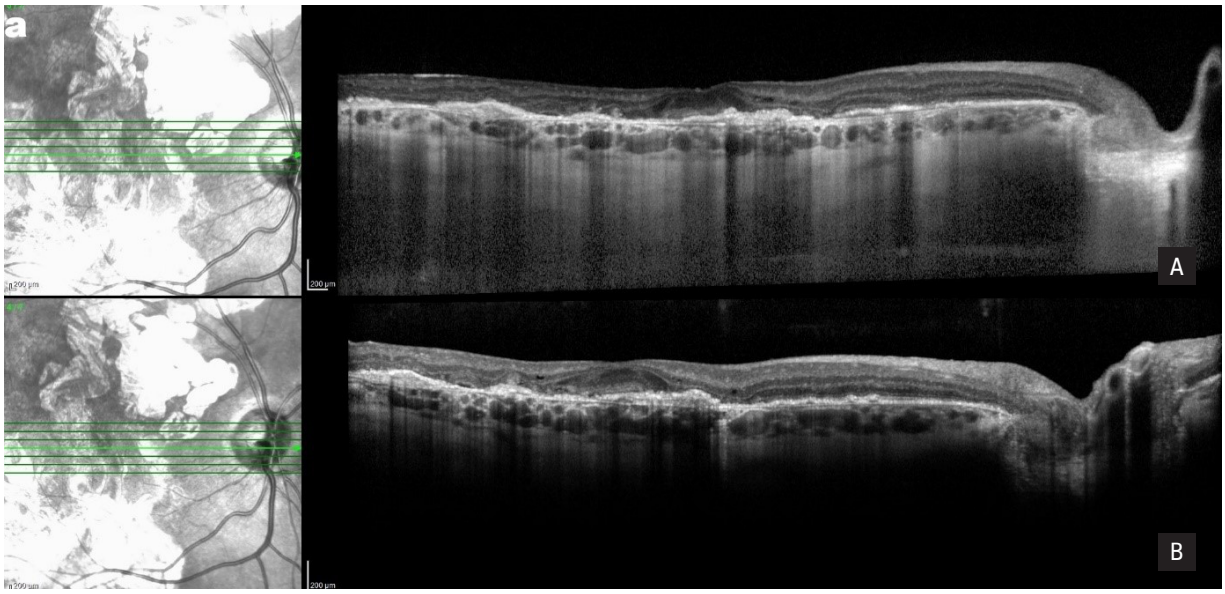

FIGURE 5. Patient 1: Optical coherence tomography (OCT) of the macula in the right eye. **A.** First assessment: Disorganization of retinal morphology with photoreceptor atrophy, defects at the RPE level with post-inflammatory scars, and the absence of intraretinal fluid (inactive changes). **B.** 4 years later: Findings remained the same as at the first assessment

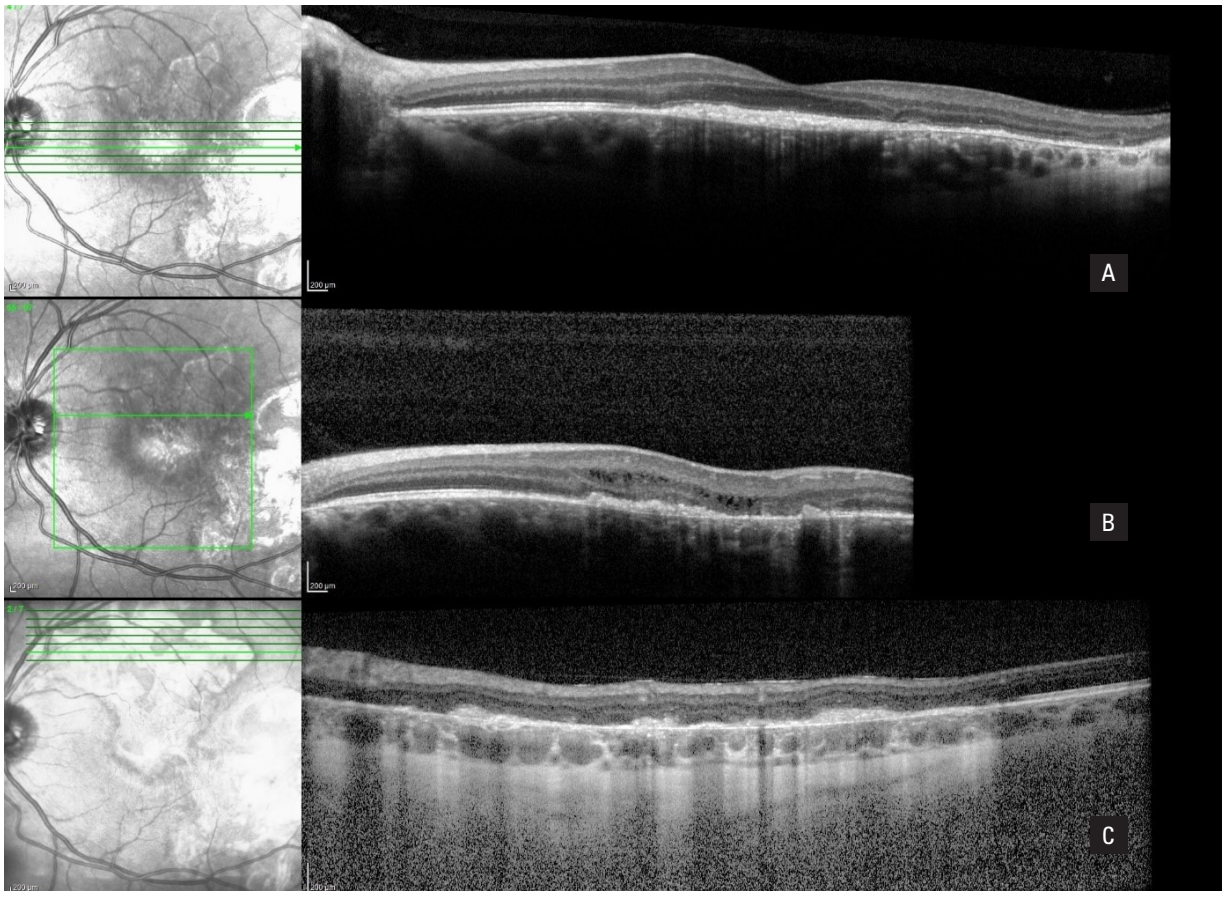

FIGURE 6. Patient 1: Optical coherence tomography (OCT) scans of the macula in the left eye. **A.** The center of the macula, at the retinal pigment epithelium (RPE) level, a fusiform widening corresponding to the scar; above, non-edematous retina, temporal damage to RPE seen as pigment epithelium atrophy and hyperpigmentation; **B.** Areas of active inflammation, visible scars, and atrophy at the RPE level; above, small cysts indicative of intraretinal edema; **C.** 4 years after: atrophic changes with RPE lesions, the absence of intraretinal fluid above the lesions

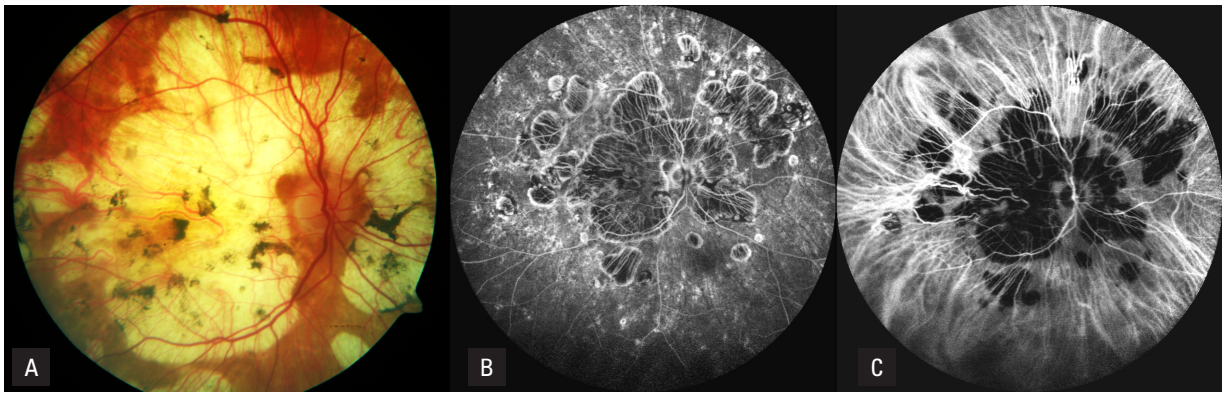

FIGURE 7. Patient 2. The right eye: extensive chorioretinal scars with visible large choroidal vessels. **A.** Color fundus photograph; **B.** Fluorescein angiography; **C.** Indocyanine green angiography. A widefield angiogram was taken using a 102° lens

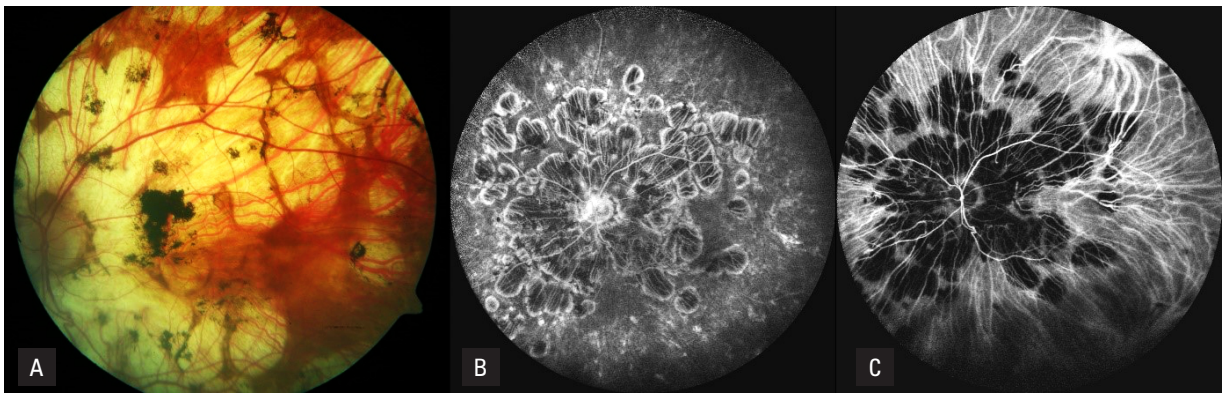

FIGURE 8. Patient 2. The left eye: extensive chorioretinal scars with visible large choroidal vessels. **A.** Color photograph; **B.** Fluorescein angiography, c) indocyanine green angiography. A widefield angiogram was taken using a 102° lens

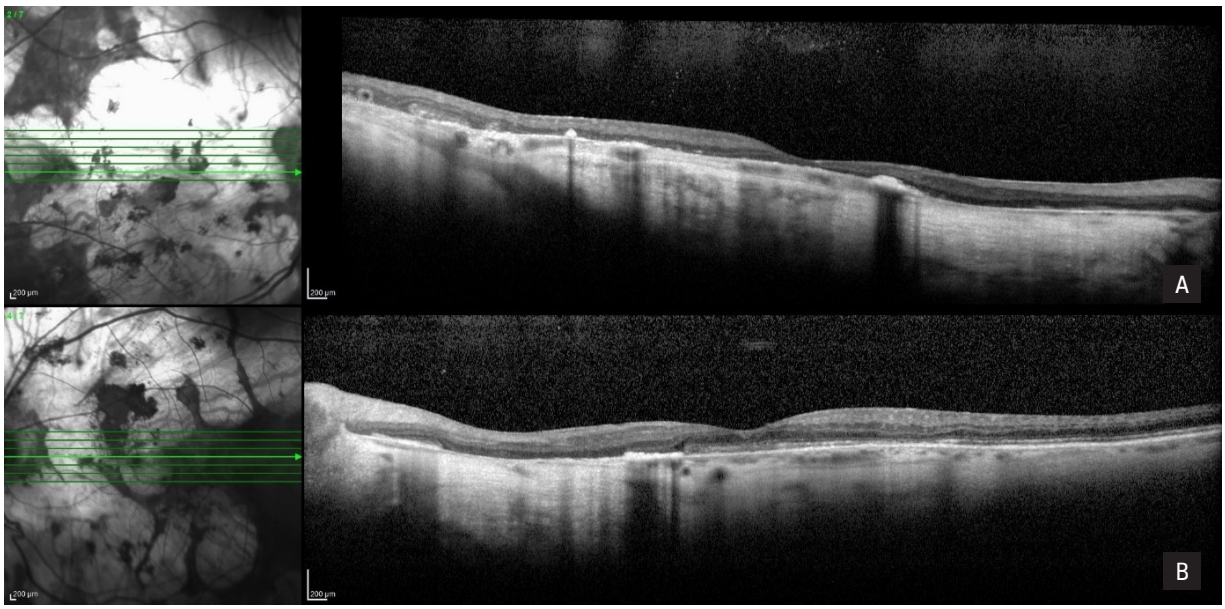

FIGURE 9. Patient 2: optical coherence tomography (OCT) scans of the macula show photoreceptor atrophy, defects, and damage at the RPE level with generalized retinal thinning in the macula as inactive inflammatory lesions. **A.** Right eye; **B.** Left eye

Over the next 3 months, in the right eye, the visual acuity remained the same with BCVA 0.5 with appropriate head positioning, and the inflammatory lesion at the optic disc was healed. Cyclosporine was discontinued after 7 months due to elevated serum creatinine levels of 1.4 mg%. For the next 6 months, the patient took oral prednisone 5 mg/day and remained in CSC remission.

For the entire treatment and follow-up periods, the patient did not adhere fully to the treatment regimen, resulting in recurrence episodes. Without consulting his doctor, he reduced prednisone doses because of weight gain, and when his blood pressure increased, he discontinued cyclosporine. In January 2020, 21 years after the initial presentation, visual acuity was maintained at BCVA 0.5 in the right eye and at 0.7 in the left eye with appropriate head positioning.

Patient 3

A 30-year-old, otherwise healthy man was referred to the Clinical Department of Ophthalmology of the Medical Academy in Warsaw (now Warsaw Medical University) for a single consultation for progressive inflammatory lesions in the retina of both eyes. He was treated with systemic corticosteroids for bilateral changes in the macula and had an earlier history of treatment for ocular toxoplasmosis for about two years. Distance BCVA was 0.3 in both eyes, and near BCVAwas 1.25. The anterior segment and vitreous body examination were unremarkable. The fundus in the right eye displayed an area of peripapillary atrophy with finger-like projections of atrophy and a focus of neovascularization in the macula. In the left eye, there was a small area of peripapillary atrophy and a focus of neovascularization in the macula. The findings were characteristic of serpiginous choroidopathy with dominant macular involvement. The addition of oral cyclosporine to the treatment regimen was advised with regular assessment at the local eye clinic.

DISCUSSION AND CONCLUSION

Classic serpiginous choroiditis is observed mainly in males. It is characterized by bilateral, progressive, multifocal, and recurrent inflammation of the choroid and retina with the resulting scarring, which leads to permanent visual impairment when the macula becomes involved. According to Hooper et al., serpiginous choroiditis usually is not associated with inflammation in the anterior segment and the vitreous cavity [9]. Still, there have been reports of associated inflammation in the vitreous body [1] and the anterior chamber [7, 22]. None of these were observed in the patients reported in this paper.

Serpiginous choroiditis is not difficult to diagnose. Fundoscopy reveals foci of active inflammation coexisting with fibrotic scars in different locations and stages of development. Classic serpiginous choroiditis should be differentiated from tuberculosis-related serpiginous-like choroiditis [7], and chest radiography and testing for tuberculosis infection are performed. According to the guidelines of the Polish Respiratory Society concerning the diagnosis of tuberculosis, the use of TB blood tests (interferon-gamma release assays, IGRAs) such as QuantiFERON TB-Gold (QFT-G) is preferred instead of the tuberculin skin test. QFT-G is considered more reliable because, unlike the tuberculin test, it is not affected by the Bacille-Calmette Guérin (BCG) vaccination [13].

Serpiginous choroiditis is difficult to treat because its etiology has not been fully elucidated, and the optimal treatment has not been established. The visual prognosis is relatively poor, and the 5-year reactivation rate is nearly 50% [23]. Damage to the choroid and retina, including the macular area, may create a risk for the development of choroidal neovascularization (CNV), which actually occurred in approximately 15% to 35% of serpiginous choroiditis patients [24] and was found in Patient 3. Most commonly, long-term immunosuppressive combination therapy is used. Although it usually does not prevent new activation, it may reduce its rate and thus slow retinal damage and visual impairment progression. Discontinuation of the prescribed immunosuppressive treatment by two of the reported patients had impact on their reactivation rates, with a shorter remission duration in Patient 1 or disease progression in Patient 2. Patient 3 had peripapillary lesions and in both eyes, CNV. He had long-term systemic steroid treatment, which at that time (2002) was the only possible treatment modality for CNV associated with recurrent serpiginous choroiditis as anti-VEGF treatments were not available until 2004 [25].

New treatments for serpiginous choroiditis now include biologics such as infliximab and adalimumab [26, 27], but they have not yet been approved for this indication.

Further studies into the etiology of serpiginous choroiditis seem to be crucial for developing an effective therapeutic algorithm.

Conflict of interests

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

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