CURRENT THERAPEUTIC RESEARCH

VOLUME 71, NUMBER 3, JUNE 2010

# Effects of Photodynamic Therapy With Verteporfin for the Treatment of Chronic Central Serous Chorioretinopathy: An Uncontrolled, Open-Label, Observational Study

Yildirim Bayezit Sakalar, MD[1]; Ugur Keklikci, MD[1]; Kaan Unlu, MD[1]; Mehmet Fuat Alakus, MD[1]; and Ismail Hamdi Kara, MD[2]

*[1]Department of Ophthalmology, Dicle University Faculty of Medicine, Diyarbakir, Turkey; and [2]Department of Family Medicine, Duzce University Faculty of Medicine, Duzce, Turkey*

## ABSTRACT

**BACKGROUND:** Central serous chorioretinopathy is an idiopathic disorder that leads to serous neurosensory retinal detachment. The disorder is usually self-limited and resolves spontaneously; however, sometimes neurosensory retinal detachment persists. This form of the disorder is called chronic central serous chorioretinopathy (CCSC).

**OBJECTIVE:** The aim of this study was to assess the effects of photodynamic therapy (PDT) on visual acuity with full-dose verteporfin for CCSC.

**METHODS:** The eyes of patients with CCSC were included in the study. Ophthalmic examination including best-corrected visual acuity (BCVA), fundus examination, fluorescein angiography, and optical coherence tomography was performed before treatment and at 1, 3, 6, 9, and 12 months. PDT with full-dose verteporfin (6 mg/m$^2$ of body surface area) was applied only to areas of active leakage. BCVA was converted to a log of the minimum angle of resolution (logMAR) equivalent for statistical analysis. Central foveal thickness and BCVA between baseline and follow-up were compared.

**RESULTS:** Seventeen eyes of 16 patients (13 males, 3 females; mean [SD] age, 39.75 [7.51] years; mean duration of follow-up, 13.06 [1.82] months) were used in the study. The mean (SEM) logMAR BCVA was 0.26 (0.07) at baseline and 0.04 (0.02) at 12 months. Mean logMAR BCVA values at baseline (0.259) and after treatment (0.112, 0.053, 0.047, 0.041, and 0.041 at 1, 3, 6, 9, and 12 months, respectively) differed significantly ($P = 0.006$, $P = 0.005$, $P = 0.005$, $P = 0.005$, and $P = 0.005$). There was a significant difference in the mean central foveal thickness at the final visit (169 μm) compared with the baseline value (383 μm; $P < 0.001$). BCVA decreased in one eye (20/20 vs 20/25) and persisted during follow-up; in the other 16 eyes, BCVA either increased (n = 10) or remained stable (n = 6).

**CONCLUSIONS:** In this small, open-label study, patients with CCSC treated with a single course of PDT with full-dose verteporfin had significant improvement from baseline in BCVA and resolution of subretinal fluid accumulation and active leakage. Treatment was generally well tolerated, but one patient had worsening in BCVA. (*Curr Ther Res Clin Exp.* 2010;71:173–185) © 2010 Excerpta Medica Inc.

This study was presented in part at the Mediterranean Retina III Meeting, June 13–15, 2008, Istanbul, Turkey.

*Accepted for publication April 2, 2010.*
© 2010 Excerpta Medica Inc. All rights reserved.
doi:10.1016/j.curtheres.2010.04.002
0011-393X/$ - see front matter

**KEY WORDS:** central serous chorioretinopathy, photodynamic therapy, optical coherence tomography, verteporfin.

## INTRODUCTION

Central serous chorioretinopathy (CSC) is an idiopathic disorder, first described by Albrecht von Graefe, that leads to serous neurosensory retinal detachment, with active retinal pigment epithelium leakage in the macular region.[1,2] Patients with CSC typically complain of decreased visual acuity, metamorphopsia, micropsia, and a relatively positive scotoma caused by macular detachment. The precise pathogenesis and treatment of CSC are still poorly understood. It has been reported that the acute form of the disorder is usually self-limited and resolves spontaneously in 4–6 months; recurrence occurs in about one third to one half of all patients.[3–5] Good visual acuity is regained after spontaneous resolution of the detachment. The final visual acuity is better than or equivalent to 20/40 in 91% to 100% of patients who resolve spontaneously.[3–7] Severe visual loss is reported in ~5% of patients with CSC.[3,4,7] Rarely, neurosensory retinal detachment persists and leads to retinal pigment epithelium and photoreceptor damage.[8–10] This form of the disorder is called chronic CSC (CCSC) and can result in severe effects on macular function.[8–11]

The most common treatment for this disease is laser photocoagulation.[3,12] Direct thermal laser photocoagulation has the disadvantages of macular photocoagulation: foveal distortion, scotoma, decreased contrast sensitivity, retinal pigment epithelium damage, and iatrogenic choroidal neovascularization.[12–16] This has led clinicians to investigate newer treatment modalities that might be more effective and less harmful.

It has been reported that photodynamic therapy (PDT) is effective for CSC.[7,17–22] Piccolino et al[17] performed PDT guided by indocyanine green angiography in 16 eyes with CCSC. In that study, the baseline visual acuities were 20/32 to 20/400 and follow-up was 6 to 12 months. They reported that macular exudation resolved completely in 13 eyes (81%) and partially in 3 eyes. Moreover, visual acuity improved 1 to 4 lines in 11 eyes and remained unchanged in 5 eyes. Yannuzzi et al[18] applied PDT guided by indocyanine green angiography in 20 eyes of 15 patients with CCSC. In their study, baseline visual acuity ranged from 20/40 to 20/800 (median, 20/200; mean, 20/275). They observed complete resolution of exudative macular detachments in 12 patients and incomplete resolution in 8 eyes after treatment. Additionally, the vision improved in 6 eyes and remained unchanged in 14 eyes during a mean follow-up of 6.8 months. In a retrospective study, PDT guided by fluorescein angiography was carried out in 9 eyes of 9 patients with acute focal retinal pigment epithelial leaks secondary to CSC. Baseline visual acuity ranged from 20/32 to 20/400 (median, 20/80). The authors found that neurosensory detachment and fluorescein leakage resolved in all patients within 1 month and visual acuity improved from 1 to 6 lines in 7 eyes and remained unchanged in 2 eyes. There was a statistically significant improvement in mean visual acuity ($P = 0.012$), and mean visual acuity improved from 20/80 to 20/40 at 6 months.[19]

The aim of the present study was to assess the effects of PDT with full-dose verteporfin for the treatment of CCSC. Our study differs from earlier studies in that we assessed PDT with full-dose verteporfin in patients with CCSC and better baseline visual acuity.

## PATIENTS AND METHODS

### Patients

This prospective, open-label, uncontrolled observational study was conducted in the Department of Ophthalmology, Faculty of Medicine, Dicle University, Diyarbakir, Turkey. The study included the eyes of consecutive patients who were diagnosed with CCSC and suffered from visual problems. The diagnosis of CCSC was made when subretinal fluid persisted in the macular region for ≥6 months, which was determined by clinical examination and associated with atrophy and decompensation of the retinal pigment epithelium.

Inclusion criteria included active leakage upon fluorescein angiography, presence of subretinal fluid for ≥6 months, which involved the fovea on optical coherence tomography (OCT), lack of choroidal neovascularization or other disease, and 20/200 or better best-corrected visual acuity (BCVA). Exclusion criteria included presence of other maculopathy, choroidal neovascularization, or polypoid choroidal vasculopathy, ocular surgery or another ocular disease, history of allergy to verteporfin or fluorescein, and liver or kidney dysfunction. Patients who previously received PDT or laser photocoagulation for CSC or another condition were also excluded. Other exclusion criteria included choroidal neovascularization or multifocal choroiditis that caused macular detachment alone or with CSC.

The experimental nature of the present study was explained to all patients before treatment, and written informed consent was obtained before study participation. This study was approved by the ethics committee of Dicle University.

### Treatment

All patients underwent a complete eye examination, including BCVA, slit lamp biomicroscopy, dilated fundus examination, color fundus photography, and OCT at baseline, and at 1, 3, 6, 9, and 12 months. Before treatment, active leakage areas were determined by fluorescein angiography. Fluorescein angiography was conducted at baseline and at 1, 3, and 12 months. To determine the presence of subretinal fluid accumulation and measure central foveal thickness before and after treatment at 1, 3, 6, 9, and 12 months, OCT (OCT-3; Zeiss Humphrey, Dublin, California) was used. Ocular examination was performed by a single person (Y.B.S.). PDT with verteporfin (Visudyne, Novartis AG, Basel, Switzerland) was applied in the same manner as the treatment for neovascular age-related macular degeneration in the Treatment of Age-Related Macular Degeneration with Photodynamic Therapy investigation.[23] Briefly, verteporfin (6 mg/m$^2$ of body surface area) was administered via intravenous infusion of 30 mL over 10 minutes. Fifteen minutes after the start of the infusion, a laser light at 689 nm delivered 50 J/cm$^2$ at an intensity of 600 mW/cm$^2$ over 83 seconds using a spot size with a diameter 1000 μm larger than the greatest linear dimension of the

lesion as described by fluorescein angiography. PDT was applied by laser (Visulas 6905, Carl Zeiss Meditech AG, Jena, Germany). PDT was applied to areas of covered active leakage, as determined by fluorescein angiography. However, between 1000 to 1100 μm laser spot size was used to preserve fovea in adjacent lesions.

Repeat treatment criteria were active leakage on fluorescein angiography, together with the presence of subretinal fluid on OCT at 1 month after PDT. Treatment was not applied if there was no active leakage observed on fluorescein angiography and subretinal fluid accumulation on OCT, even if the patient had visual loss.

The main outcome measurements were visual acuity, central foveal thickness, and active areas of leakage on fluorescein angiography. Visual acuity was examined by Snellen testing.

Systemic adverse events (AEs) caused by verteporfin injection were monitored by physical examination and patient interview at each visit. Ocular AEs were monitored and recorded by ocular examination and patient report at each visit.

### Statistical Analysis

SPSS version 11.01 (SPSS Inc., Chicago, Illinois), was used for data analysis and $P \leq 0.05$ was considered statistically significant. Age is reported as mean (SD) and the other data are reported as mean (SEM) and 95% CI. To compare mean log of the minimum angle of resolution (logMAR) BCVA values and central foveal thickness, a nonparametric Wilcoxon-signed rank test and paired-sample *t* test were used, respectively. Visual acuity scores were converted to the logMAR by using a table of equivalent visual acuities for statistical analysis.[24]

## RESULTS

Sixteen consecutive patients (males, 13 [81.3%]; mean [SD] age, 39.75 [7.51] years; median age, 39 years [range, 29–56 years]) with 17 eyes diagnosed with CCSC were approached for enrollment in the study; none were excluded. All 16 patients completed the 12-month follow-up. Three of the 16 patients had a history of steroid usage before presentation; steroid use was stopped ≥6 months before treatment. The mean duration of follow-up was 13.06 (1.82) months. Baseline BCVA values ranged from 20/200 to 20/20. **Table I** summarizes the clinical features of the patients and **Table II** summarizes treatment results.

The active leakage areas were closed within the first month after treatment in all patients, and no recurrence was seen during follow-up (**Figure 1**). PDT was applied only once in all patients and none required a second treatment during follow-up. The mean (SEM) central foveal thickness was 383.35 (29.61) μm at baseline, and 237.82 (18.9), 188.82 (10.66), 172.24 (5.38), 169.76 (5.1), and 169.24 (5.04) μm at 1, 3, 6, 9, and 12 months, respectively (**Figure 2**). The mean central foveal thickness at all postbaseline visits was significantly less than that at baseline (all, $P < 0.001$). There was a significant difference in mean central foveal thickness at the final visit compared with the pretreatment value ($P < 0.001$). Moreover, there was a significant difference in mean central foveal thickness between 1 month and 3 months, 1 month and 6 months, and 3 and 6 months ($P = 0.006$, $P = 0.001$, and $P = 0.01$, respectively).

**Table I. Clinical characteristics of study patients with chronic central serous chorioretinopathy (CCSC) (N = 16).**

| Case | Age, y | Sex | Eye | Eye Color | Duration of CCSC, mo | Treated Areas, no. | PDT spot size, μm |
|---|---|---|---|---|---|---|---|
| 1 | 56 | M | Right | Brown | 8 | 1 | 1050 |
| 2 | 42 | F | Left | Brown | 9 | 1 | 1100 |
| 3 | 41 | M | Right | Green | 12 | 1 | 1000 |
| 4 | 34 | M | Left | Brown | 8 | 1 | 1050 |
| 5 | 43 | M | Right | Brown | 6 | 1 | 1150 |
| 6 | 55 | F | Left | Brown | 7 | 1 | 2700 |
| 7 | 30 | M | Left | Brown | 9 | 1 | 1250 |
| 8 | 37 | M | Right | Brown | 12 | 1 | 2150 |
| 9 | 38 | M | Right | Green | 7 | 1 | 1900 |
| 10 | 44 | M | Right | Brown | 10 | 1 | 1700 |
| 11 | 37 | M | Left | Brown | 6 | 1 | 1650 |
| 12 | 37 | M | Right | Brown | 6 | 1 | 1200 |
| 13 | 40 | F | Left | Brown | 6 | 1 | 1800 |
| 14 | 29 | M | Left | Brown | 6 | 1 | 1100 |
| 15 | 40 | M | Right | Brown | 8 | 1 | 2000 |
| 16 | 33 | M | Right | Brown | 10 | 1 | 2900 |
| | | | Left | Brown | 10 | 2 | 2750 and 1500 |

PDT = photodynamic therapy; M = male; F = female.

The baseline BCVA was 20/20 in 7 of 17 eyes (41.18%). However, these patients had complaints such as metamorphopsia, relative scotoma, or micropsia, and there was subretinal fluid accumulation on OCT. One patient (patient 16) had bilateral CCSC, whereas the other patients had unilateral CCSC. Patient 16 had 1 active leakage area in his right eye and 2 active leakage areas in his left eye (**Figures 3** and 4). PDT was applied to both leakage areas in the left eye and to the single leakage area in the right eye. The other patients had only one leakage area in each eye.

The mean (SEM) logMAR BCVA was 0.26 (0.07) at baseline, 0.11 (0.05) at 1 month, 0.05 (0.03) at 3 and 6 months, and 0.04 (0.02) at 9 and 12 months (**Figure 5**). BCVA values after treatment were significantly lower ($P = 0.006$, $P = 0.005$, $P = 0.005$, $P = 0.005$, and $P = 0.005$ at 1, 3, 6, 9, and 12 months, respectively). There were no significant differences in BCVA values in follow-up after the third month. There was a significant difference between month 1 and month 3 ($P = 0.041$). Visual acuity was

**Table II. The results of photodynamic therapy with verteporfin in patients with chronic central serous chorioretinopathy (N = 16).**

| | BCVA | | CFT, μm | |
|---|---|---|---|---|
| Patient | Pretreatment | Posttreatment | Pretreatment | Posttreatment |
| 1 | 20/32 | 20/20 | 368 | 182 |
| 2 | 20/40 | 20/20 | 473 | 170 |
| 3 | 20/20 | 20/20 | 381 | 176 |
| 4 | 20/20 | 20/20 | 216 | 173 |
| 5 | 20/20 | 20/20 | 340 | 171 |
| 6 | 20/200 | 20/25 | 570 | 200 |
| 7 | 20/63 | 20/20 | 645 | 159 |
| 8 | 20/20 | 20/20 | 194 | 153 |
| 9 | 20/40 | 20/20 | 223 | 150 |
| 10 | 20/63 | 20/20 | 383 | 205 |
| 11 | 20/25 | 20/20 | 290 | 157 |
| 12 | 20/50 | 20/20 | 344 | 191 |
| 13 | 20/50 | 20/25 | 424 | 159 |
| 14 | 20/20 | 20/20 | 415 | 155 |
| 15 | 20/20 | 20/20 | 308 | 194 |
| 16 | 20/100 | 20/50 | 497 | 124 |
| | 20/20 | 20/25 | 446 | 158 |

BCVA = best-corrected visual acuity; CFT = central foveal thickness.

decreased in the first month (20/20 vs 20/25) after treatment in 1 patient (patient 16) and persisted during follow-up.

After the first month, secondary retinal pigment epithelium changes were observed in areas where PDT was applied, but there was no choroidal neovascularization. No patients reported systemic adverse effects related or not related to treatment.

## DISCUSSION

PDT has been used for secondary choroidal neovascularization caused by age-related macular degeneration.[23] Studies have reported the effectiveness of PDT for CCSC.[7,17,18,21,22] The exact mechanism of PDT in treating CCSC has not been fully established, but it is thought to be caused by short-term choriocapillary hypoperfusion and long-term choroidal vascular remodeling, which leads to reduction in choroidal congestion, vascular hyperpermeability, and extravascular leakage.[22,25,26] Retinal pigment epithelium cells damaged by light-activated verteporfin might be

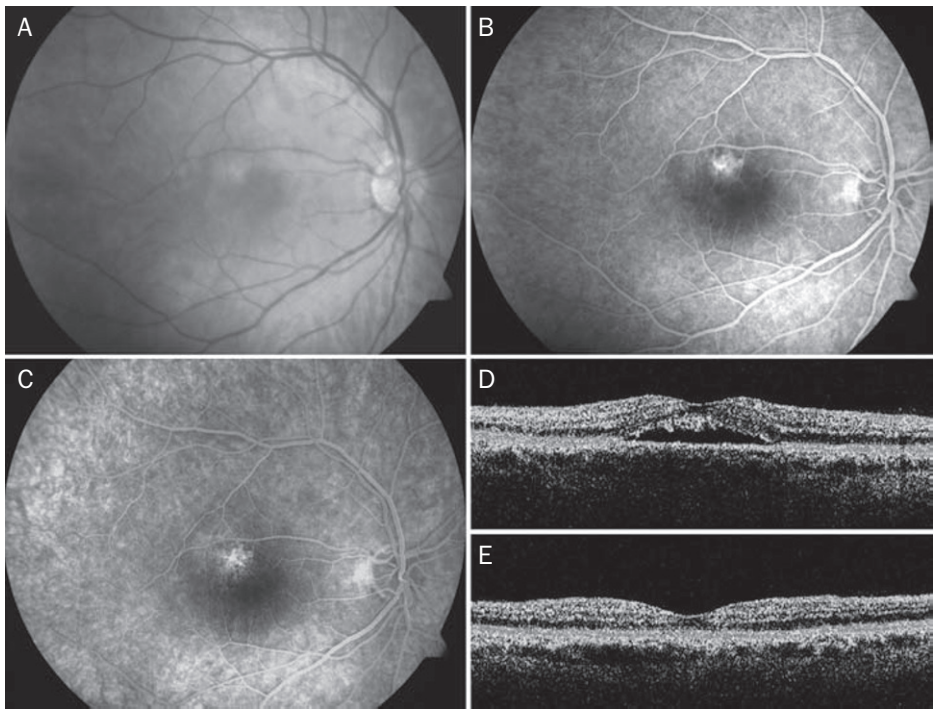


**Figure 1. Patient 1 of 16 patients with chronic central serous chorioretinopathy treated with photodynamic therapy (PDT) and verteporfin. (A) Fundus photography before PDT. (B) Active leakage as seen by fluorescein angiography before PDT. (C) Closure of leakage and retinal pigment epithelium changes at 1 month after PDT. (D) Subretinal fluid accumulation before treatment, as seen by optical coherence tomography (OCT). (E) Complete resolution of subretinal fluid, as seen by OCT at 1-month follow-up.**

replaced by new ones, with possible recovery from metabolic impairment at the retinal pigment epithelium level.[21] Previous studies also have reported that damage induced by PDT with verteporfin is not confined to the endothelium of choroidal neovascularization, but also affects the endothelium of the choriocapillaries.[27,28]

In some previous studies of CCSC, PDT has been found to be effective and visual acuity in almost all cases has increased or remained stable.[7,17,18,21,22] In the present study, visual acuity after PDT with full-dose verteporfin increased in 10 of 17 eyes and remained stable in 6. Visual acuity in the left eye of patient 16, in which both eyes were treated with PDT, decreased from 20/20 to 20/25 at 1 month after treatment, and remained at this level for the duration of follow-up. There was leakage at 2 different areas in this eye, and 2 nonadjacent laser spots of 2750 and 1500 µm were used. In earlier studies,[7,17,18,21,22] decreases in vision without choroidal neovascularization secondary to PDT were not reported after PDT with full-dose verteporfin. Colucciello[29] reported the occurrence of loss of visual acuity and choroidal neovascularization after

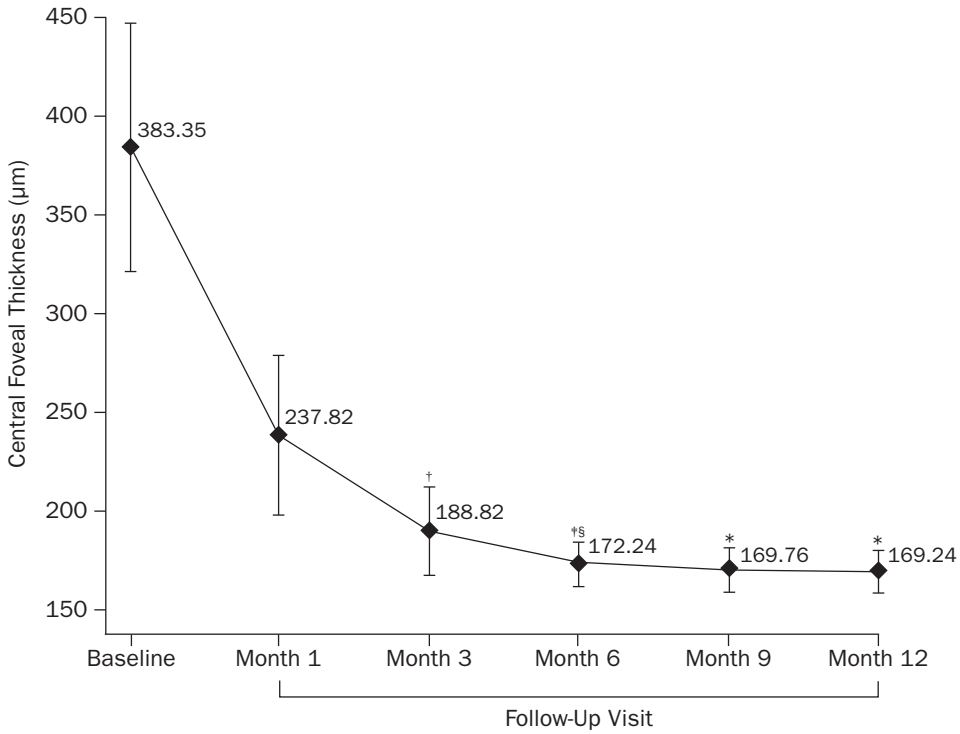


**Figure 2. Changes in mean central foveal thickness before treatment (baseline) and at follow-up in patients with chronic central serous chorioretinopathy treated with photodynamic therapy and verteporfin. **P* < 0.001 versus baseline; †*P* = 0.006 versus month 1; ‡*P* = 0.001 versus month 1; §*P* = 0.001 versus month 3.**

PDT with full-dose verteporfin for CCSC, which might have occurred as a result of choroidal hypoperfusion. Piccolino et al[17] reported that multiple adjacent laser applications can lead to hyper-dosage effects. Adjacent laser applications might enhance the ischemic effect of a singular irradiation and make reperfusion more difficult in certain areas of the choriocapillaris. Wang et al[30] found that foveal atrophy in CSC is associated with reduced visual acuity, despite resolution of the serous detachment. They suggested that foveal attenuation in CSC was associated with >4 months' duration of symptoms and persistent BCVA reduction despite resolution of the serous detachment. These potential adverse effects need further evaluation and might be important considerations for restricting the extensive use of PDT for CCSC until more data is available.

In the present study, in all cases, including the patient with visual acuity decrease, subretinal fluid was resolved in the first month and no recurrence was seen at follow-up. In earlier studies also, subretinal fluid was reported to have been resolved partially or completely after PDT.[7,17,18,21,22] In one of these studies, subretinal fluid was resolved

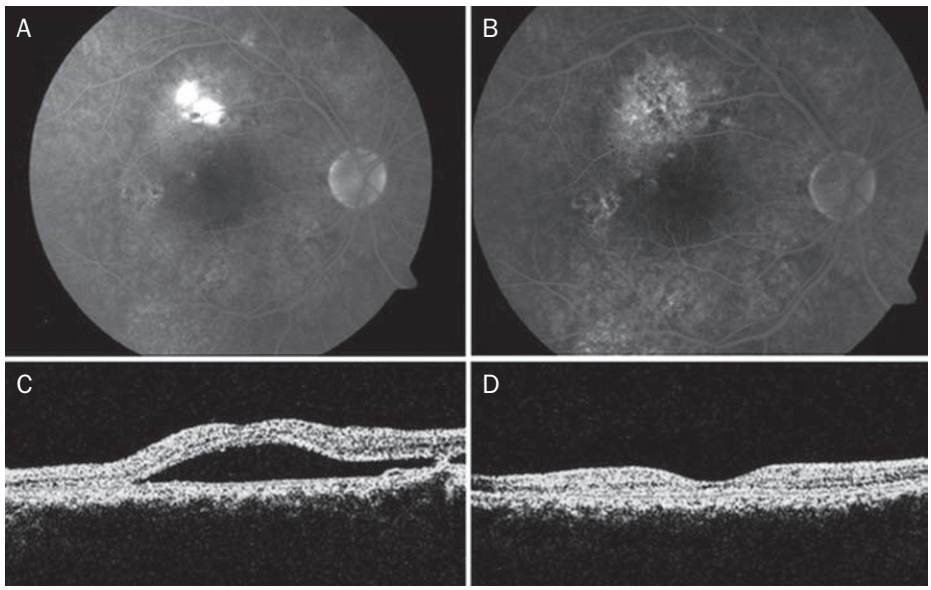


**Figure 3. Right eye of patient 16 of 16 patients with chronic central serous chorioretinopathy treated with photodynamic therapy (PDT) and verteporfin. (A) Area of active leakage, as seen by fluorescein angiography before PDT. (B) Closure of active leakage after PDT, as seen by fluorescein angiography at 1 month. (C) Subretinal fluid and macular detachment, as seen by optical coherence tomography (OCT). (D) Complete resolution of subretinal fluid at 1 month after treatment, as seen by OCT.**

completely in all cases.[7] The resolution of subretinal fluid might be associated with remodeling of the choriocapillaris and absorption of subretinal fluid by intact retinal pigment epithelium cells.[17] Piccolino et al[17] reported that these lasting effects of PDT in the prevention of subretinal exudation might be correlated with hypoperfusion induced in the choriocapillaris. A study by Yannuzzi et al[18] reported that 2 eyes experienced recurrent macular detachments at 4 months after treatment, despite previous resolution of the detachments. Similarly, recurrence was experienced in 2 eyes in the studies by Piccolino et al. However, recurrence was not observed in the study carried out by Taban et al.[7] In the study by Taban et al, 5 eyes of 4 patients with CCSC were administered PDT with full-dose verteporfin and patients were followed for an average of 10 months. In all of the present study's cases, retinal pigment epithelium changes occurred after the first month in the area where PDT was applied, and these changes remained for 12 months. These retinal pigment epithelium changes after PDT might have been caused by choroidal hypoperfusion in areas where PDT was applied. In CSC, another secondary complication of PDT is choroidal neovascularization.[20,29] In the present study, choroidal neovascularization associated with PDT did not develop in any of the patients. In addition, there were no systemic adverse effects associated with verteporfin.

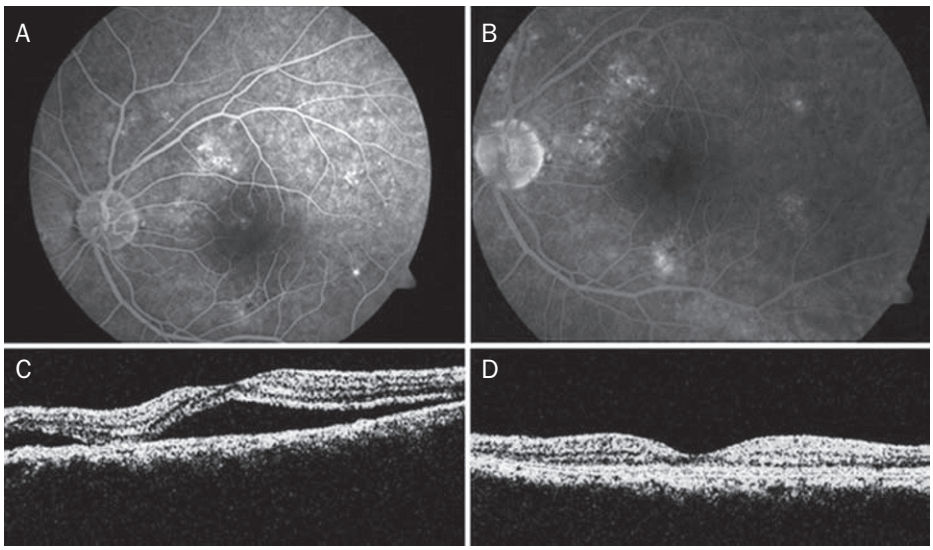


**Figure 4. Left eye of patient 16 of 16 patients with chronic central serous chorioretinopathy treated with photodynamic therapy (PDT) and verteporfin. (A) Two areas of active leakage, as seen by fluorescein angiography before PDT. (B) Closure of leakage and retinal pigment epithelium changes after PDT. (C) Subretinal fluid before treatment, as seen by optical coherence tomography (OCT). (D) Resolution of subretinal fluid, as seen by OCT at 1 month after treatment.**

Our study has several limitations, such as the absence of a control group, the open-label design, small number of patients, and relatively short period of follow-up. The baseline visual acuity of our patients was also better than that in previous studies.[17–19] In this study, no patients required a second application of PDT. This suggests that a single application of PDT might be sufficient for treatment of some cases of CCSC. The present study included 13 months of follow-up. Although this is a relatively short period, it might have been sufficient for assessment of the results. Further studies are needed to determine the efficacy and tolerability of PDT with full-dose verteporfin for treatment of CCSC.

## CONCLUSIONS

In this small, open-label study, patients with CCSC treated with a single course of PDT with full-dose verteporfin had significant improvement from baseline in BCVA and resolution of subretinal fluid accumulation and active leakage. Treatment was generally well tolerated, but one patient had worsening of BCVA after treatment.

## ACKNOWLEDGMENT

The authors have indicated that they have no conflicts of interest regarding the content of this article.

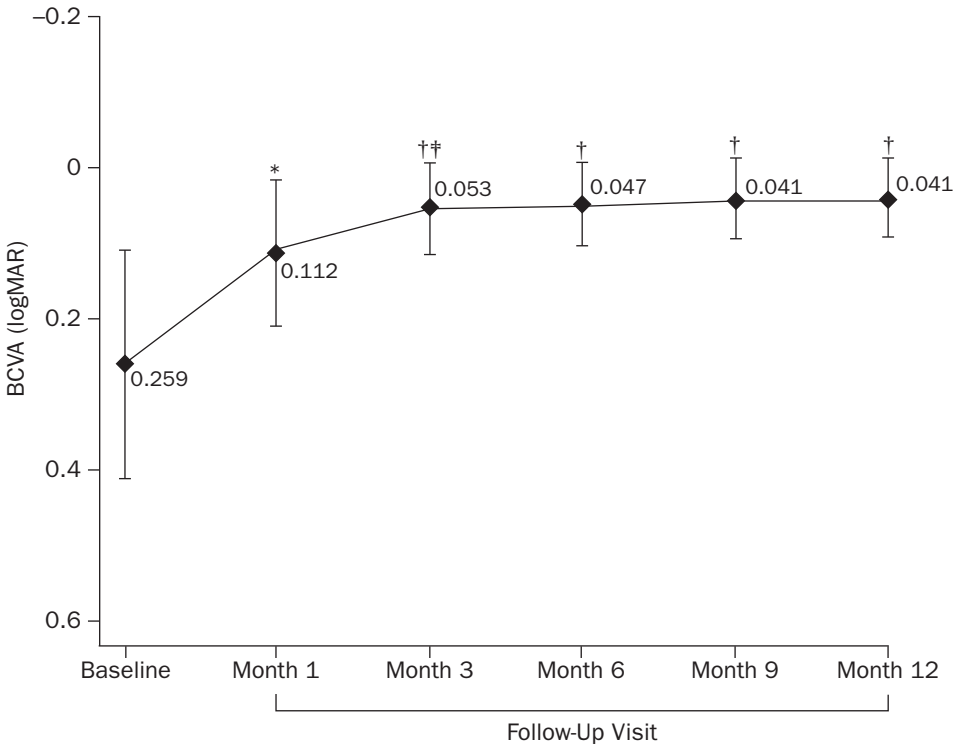


**Figure 5. Changes in the mean log of the minimum angle of resolution (logMAR) best-corrected visual acuity (BCVA) before treatment (baseline) and at follow-up in patients with chronic central serous chorioretinopathy treated with photodynamic therapy and verteporfin. *$P$ = 0.006 versus baseline; †$P$ = 0.005 versus baseline; ‡$P$ = 0.041 versus month 1.**

## REFERENCES


1. Gass JD. Pathogenesis of disciform detachment of the neuroepithelium: II. Idiopathic central serous chorioretinopathy. *Am J Ophthalmol*. 1967;63:587–615.
2. von Graefe A. Ueber centrale reccidivirende retinitis. *Graefes Arch Clin Exp Ophthalmol*. 1866; 12:211–215.
3. Gass JD. *Stereoscopic Atlas of Macular Diseases*. 4th ed. St. Louis, Mo: Mosby–Year Book; 1997: 52–70.
4. Hussain D, Gass JD. Idiopathic central serous chorioretinopathy. *Indian J Ophthalmol*. 1998;46: 131–137.
5. Klein ML, Van Buskirk EM, Friedman E, et al. Experience with nontreatment of central serous chorioretinopathy. *Arch Ophthalmol*. 1974;91:247–250.
6. Castro-Correia J, Coutinho MF, Rosas V, Maia J. Long-term follow-up of central serous chorioretinopathy in 150 patients. *Doc Ophthalmol*. 1992;81:379–386.
7. Taban M, Boyer DS, Thomas EL, Taban M. Chronic central serous chorioretinopathy: Photodynamic therapy. *Am J Ophthalmol*. 2004;137:1073–1080.

8. Yannuzzi LA. Type A behavior and central serous chorioretinopathy. *Trans Am Ophthalmol Soc*. 1986;84:799–845.
9. Yannuzzi LA, Gitter KA, Schatz H. Central serous chorioretinopathy. In: *The Macula: A Comprehensive Text and Atlas*. Baltimore, Md: Williams & Wilkins; 1979.
10. Yannuzzi LA, Shakin JL, Fisher YL, Altomonte MA. Peripheral retinal detachments and retinal pigment epithelial atrophic tracts secondary to central serous pigment epitheliopathy. *Ophthalmology*. 1984;91:1554–1572.
11. Spaide RF, Campeas L, Haas A, et al. Central serous chorioretinopathy in older and younger adults. *Ophthalmology*. 1996;103:2070–2079, discussion 2079–2080.
12. Watzke RC, Burton TC, Woolson RF. Direct and indirect laser photocoagulation of central serous choroidnopathy. *Am J Ophthalmol*. 1979;88:914–918.
13. Robertson DM, Ilstrup D. Direct, indirect, and sham laser photocoagulation in the management of central serous chorioretinopathy. *Am J Ophthalmol*. 1983;95:457–466.
14. Khosla PK, Rana SS, Tewari HK, et al. Evaluation of visual function following argon laser photocoagulation in central serous retinopathy. *Ophthalmic Surg Lasers*. 1997;28:693–697.
15. Simon P, Glacet-Bernard A, Binaghi M, et al. Choroidal neovascularization as a complication following laser treatment of central serous chorioretinopathy [in French]. *J Fr Ophtalmol*. 2001;24:64–68.
16. Schatz H, Yannuzzi LA, Gitter KA. Subretinal neovascularization following argon laser photocoagulation treatment for central serous chorioretinopathy: Complication or misdiagnosis? *Trans Sect Am Acad Ophthalmol Otolaryngol*. 1977;83:893–906.
17. Piccolino F, Eandi CM, Ventre L, et al. Photodynamic therapy for chronic central serous chorioretinopathy. *Retina*. 2003;23:752–763.
18. Yannuzzi LA, Slakter JS, Gross NE, et al. Indocyanine green angiography-guided photodynamic therapy for treatment of chronic central serous chorioretinopathy: A pilot study. *Retina*. 2003;23:288–298.
19. Ober MD, Yannuzzi LA, Do DV, et al. Photodynamic therapy for focal retinal pigment epithelial leaks secondary to central serous chorioretinopathy. *Ophthalmology*. 2005;112:2088–2094.
20. Chan WM, Lam DS, Lai TY, et al. Choroidal vascular remodelling in central serous chorioretinopathy after indocyanine green guided photodynamic therapy with verteporfin: A novel treatment at the primary disease level. *Br J Ophthalmol*. 2003;87:1453–1458.
21. Battaglia Parodi M, Da Pozzo S, Ravalico G. Photodynamic therapy in chronic central serous chorioretinopathy. *Retina*. 2003;23:235–237.
22. Canakis C, Livir-Rallatos C, Panayiotis Z, et al. Ocular photodynamic therapy for serous macular detachment in the diffuse retinal pigment epitheliopathy variant of idiopathic central serous chorioretinopathy. *Am J Ophthalmol*. 2003;136:750–752.
23. Treatment of Age-Related Macular Degeneration with Photodynamic Therapy (TAP) Study Group. Photodynamic therapy of subfoveal choroidal neovascularization in age-related macular degeneration with verteporfin: One-year results of 2 randomized clinical trials—TAP report [published correction appears in *Arch Ophthalmol.* 2000;118:488]. *Arch Ophthalmol*. 1999;117:1329–1345.
24. Ferris FL III, Kassoff A, Bresnick GH, Bailey I. New visual acuity charts for clinical research. *Am J Ophthalmol*. 1982;94:91–96.
25. Schlötzer-Schrehardt U, Viestenz A, Naumann GO, et al. Dose-related structural effects of photodynamic therapy on choroidal and retinal structures of human eyes. *Graefes Arch Clin Exp Ophthalmol*. 2002;240:748–757.
26. Schmidt-Erfurth U, Laqua H, Schlötzer-Schrehard U, et al. Histopathological changes following photodynamic therapy in human eyes. *Arch Ophthalmol*. 2002;120:835–844.

27. Flower RW. Expanded hypothesis on the mechanism of photodynamic therapy action on choroidal neovascularization. *Retina*. 1999;19:365–369.
28. Flower RW, von Kerczek C, Zhu L, et al. Theoretical investigation of the role of choriocapillaris blood flow in treatment of subfoveal choroidal neovascularization associated with age-related macular degeneration. *Am J Ophthalmol*. 2001;132:85–93.
29. Colucciello M. Choroidal neovascularization complicating photodynamic therapy for central serous retinopathy. *Retina*. 2006;26:239–242.
30. Wang MS, Sander B, Larsen M. Retinal atrophy in idiopathic central serous chorioretinopathy. *Am J Ophthalmol*. 2002;133:787–793.

**Address correspondence to:** Yildirim Bayezit Sakalar, MD, Dicle Universitesi Tip Fakultesi Goz Hastaliklari, ABD 21280, Diyarbakir, Turkey. E-mail: ybsakalar@yahoo.com