OUTCOMES OF TREATMENT OF CHOROIDAL NEOVASCULARIZATION ASSOCIATED WITH CENTRAL SEROUS CHORIORETINOPATHY WITH INTRAVITREAL ANTIANGIOGENIC AGENTS

JAY CHHABLANI, MD,* IGOR KOZAK, MD,† FRANCESCO PICHI, MD,‡ MEGAN CHENWORTH, BS,§ MARIA H. BERROCAL, MD,§ RUMNEEK BEDI, BS,¶ RISHI P. SINGH, MD,¶ LIHTEH WU, MD,** CATHERINE MEYERLE, MD,†† ANTONIO MARCELO CASELLA, MD,‡‡ AHMAD MANSOUR, MD,§§¶¶ ZIAD BASHSHUR, MD,§§ ANTONELLA SCORZA, MD,‡ PAOLA CARRAI, MD,‡ PAOLO NUCCI, MD,‡ J. FERNANDO AREVALO, MD, FACS†††; FOR THE KING KHALED EYE SPECIALIST HOSPITAL INTERNATIONAL COLLABORATIVE RETINA STUDY GROUP

Purpose: To report clinical characteristics and treatment outcomes from the largest case series of choroidal neovascularization secondary to central serous chorioretinopathy.

Methods: Retrospective analysis of 46 eyes of 43 consecutive subjects. Collected data included demographic details, history of presenting illness, clinical examination details including visual acuity at presentation and follow-up with imaging and treatment details. Main outcome measures were the proportion of eyes that had improved (3 or more lines), stable (within ±1 line), or decreased (3 or more lines) vision at the final visit as compared with baseline examination. Secondary efficacy outcomes included change in visual acuity at final follow-up, number of injections, treatment-free interval, and adverse events.

Results: Mean age was 57.56 years (range 29–79 years). Mean follow-up duration was 38.3 months ± 58.9 months. More than 3 lines of improvement in 12 eyes (26%), vision was stable (within ±1 line) in 19 eyes (41.3%), and >3 lines of loss was noted in 6 eyes (13%). Mean change in the number of lines was 1.16 ± 3.74. Mean number of anti-vascular endothelial growth factor injections during the follow-up was 4.45 ± 4.1. The longest treatment-free interval was 8.9 months ± 7.5 months. There were no adverse events noted.

Conclusion: Anti-vascular endothelial growth factor therapy as a primary therapy for choroidal neovascularization secondary to central serous chorioretinopathy is safe and efficacious, without any serious adverse events.

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Central serous chorioretinopathy (CSCR) presents as neurosensory detachment secondary to hyperpermeability from choroidal vessels. It can lead to chronic retinal pigment epithelial atrophy, subretinal scarring, cystic changes of the retina, and secondary choroidal neovascularization (CNV).[1,2] Development of CNV secondary to CSCR is a rare entity with an incidence ranging from 2% to 9% during the mean follow-up of 10 years in a study by Loo et al.[3,4] However, studies by Schatz[5] and Mudvari[6] and associates did not report development of CNV in eyes with CSCR during the follow-up of 27 months and 49 months, respectively. Being a rare combination, standard treatment protocols for this condition are not yet established. There are multiple treatment modalities reported for CNV secondary to CSCR, including photodynamic therapy (PDT),[7,8] anti-vascular endothelial growth factor (VEGF) therapy,[9–11] surgical removal of CNV.[12]

Evidence from various randomized trials made anti-VEGF therapy as the standard of care for CNV secondary to age-related macular degeneration (AMD).[13,14] Anti-VEGF therapy has shown great success in various non-AMD CNV cases as well, including idiopathic, pathologic myopia, secondary to chorioretinal inflammation, angioid streaks, and parafoveal telangiectasia.[15–18] However, there are only few case reports on outcome of anti-VEGF therapy in CNV secondary to CSCR without any long-term results.[9–11]

To our knowledge, this study reports clinical characteristics and treatment outcomes from the largest case series of CNV secondary to CSCR.

Methods

This is a retrospective multicenter data collection of interventional case series of eyes with a diagnosis of CNV secondary to chronic CSCR that were treated with intravitreal antiangiogenic therapy. The study duration was from January 2009 to August 2014. The case reports came from many countries of several regions of the world (Europe, North and South America, Middle East, Asia, and Oceania). Institutional review board approval was obtained at each participating center; in addition, the study adhered to the tenets of the Declaration of Helsinki.

Consecutive patients with eyes presenting with acute or chronic CSCR associated with the presence of an active CNV were included. Chronic CSC was defined as a combination of the following: presence of subretinal fluid in the foveal region persisting for 3 months or more, with or without serous pigment epithelial detachment on optical coherence tomography (OCT); presence of widespread retinal pigment epithelium (RPE) changes in the macular region associated with leaks and/or pigment epithelial detachment on fundus fluorescein angiography (FFA); and abnormal, dilated choroidal vasculature and choroidal vascular hyperpermeability with an active leakage area on indocyanine green angiography.

Exclusion criteria included CNV secondary to the laser photocoagulation or PDT performed for CSCR; any history of previous treatment for CNV including laser or submacular surgery or PDT; presence of any other retinal disease that could be a cause for CNV; unavailability of spectral domain optical coherence tomography (SD-OCT) parameters; subjects with follow-up of less than 2 months after the last injection; any other significant concurrent ocular diseases in the study eye, which could be the cause of vision loss.

All patients received comprehensive eye examination, including color fundus images, FFA, SD-OCT, and indocyanine green (depending on physician's discretion). Best-corrected visual acuity was measured using Snellen's charts. Treatment consisted of intravitreal injection of 1.25 mg per 0.05 mL of bevacizumab (Avastin; Genentech, South San Francisco, CA), 0.5 mg per 0.05 mL of ranibizumab (Lucentis; Genentech), or 2 mg per 0.05 mL of aflibercept (Eylea; Regeneron, Tarrytown, NY). After obtaining informed consent, injection was given as per standard protocol. Before the injection, topical anesthesia and ocular surface disinfection with 5% povidone–iodine were performed under topical anesthesia followed by a short course of topical antibiotics. The choice of agent was discussed between the patients and the treating physician. Postoperative period was uneventful in all patients. At each follow-up, SD-OCT scans were repeated. Retreatment was performed in case of persistence of subretinal hemorrhage on clinical examination and/or persistent activity on FFA and/or persistence of subretinal or intraretinal fluid on SD-OCT scan. In case of poor response, angiography was repeated as per physician's discretion.

Data collected included demographic details, history of presenting illness, eye laterality, ocular and systemic comorbidities, treatment history, clinical examination details including visual acuity at presentation and follow-up with imaging, and treatment details.

Main outcome measures in this study were the proportion of eyes that had improved (3 or more lines), stable (within ±1 line), or decreased (3 or more lines) vision at the final visit as compared with baseline examination. Secondary efficacy outcomes included change in visual acuity at final follow-up, number of injections, treatment-free interval, and adverse events.

Statistical Analysis

Snellen visual acuity values were converted into logMAR for statistical analysis at baseline and the last follow-up. Descriptive statistics included mean and

From the *Smt. Kanuri Santhamma Retina Vitreous Centre, L. V. Prasad Eye Institute, Hyderabad, India; †Vitreoretinal Division, King Khaled Eye Specialist Hospital, Riyadh, Saudi Arabia; ‡San Giuseppe Hospital, University Eye Clinic, Milan, Italy; §Department of Ophthalmology, University of Puerto Rico, San Juan, Puerto Rico; ¶Cole Eye Institute, Cleveland Clinic, Cleveland, Ohio; **Instituto de Cirugia Ocular, San José, Costa Rica; ††Retina Division, Wilmer Eye Institute, Johns Hopkins University School of Medicine, Baltimore, Maryland; ‡‡Department of Ophthalmology, Universidade Estadual de Londrina, Londrina, Brazil; §§Department of Ophthalmology, American University of Beirut, Beirut, Lebanon; and ¶¶Department of Ophthalmology, Rafic Hariri University Hospital, Beirut, Lebanon.

None of the authors have any financial/conflicting interests to disclose.

The complete list of participating members of The KKESH International Collaborative Retina Study Group is given in Appendix 1.

Reprint requests: J. Fernando Arevalo, MD, FACS, Vitreoretinal Division, King Khaled Eye Specialist Hospital, Al-Oruba Street, PO Box 7191, Riyadh 11462, Saudi Arabia; e-mail: arevalojf@jhmi.edu

Table 1. Clinical Characteristics of Subjects With CNV Secondary to CSCR

SN	Age, years	Sex	Duration of CSCR, months	CNV Type on OCT	CNV Type on FFA	CNV Location	Baseline VA	Final VA	Baseline CMT, μm	Final CMT, μm	Lines Changed at Last Follow-up	Total Number of Injections	Type of Injection	Total Follow-up, months	Longest Treatment-Free Interval, months
1	64	M	40	1	MC	S	20/40	20/100	213	190	–4	6	6B	64	40
2	63	M	4	2	C	S	20/100	20/40	238	140	+4	3	3B	6	3
3	62	M	28	2	C	S	20/500	HM	356	739	>–5	4	4R	24	6
4*	50	M	48	1	MC	S	20/20	20/20	121	111	0	1	1B	6	4
5*	50	M	48	1	MC	E	20/20	20/20	130	121	0	1	1B	6	4
6	52	F	60	2	C	E	20/25	20/25	129	119	0	1	1B	2	1.5
7	39	M	36	2	C	J	20/100	20/100	167	184	0	2	2R	4	0
8	47	M	6	2	C	J	20/50	20/60	175	129	–1	3	3B	7	4
9	57	M	24	2	MC	S	20/500	20/100	419	364	+7	3	3B	12	9
10	51	M	24	1	C	J	20/50	20/20	211	247	+4	2	2B	15	11
11	44	M	30	2	C	S	20/100	20/100	676	1,182	0	1	1B	12	11
12	61	F	24	2	C	S	20/80	20/100	271	179	–1	2	2B	12	10
13	42	M	2	2	O	E	20/40	20/20	625	249	+3	4	4B	12	7.8
14	63	F	1	2	O	J	20/50	20/20	565	246	+4	7	7B	19	9.2
15	51	M	1.5	2	O	J	20/50	20/20	365	255	+4	2	2B#	12.5	10.5
16	46	F	1	2	C	S	20/160	20/25	452	196	+8	2	2B#	27.6	23.5
17	60	F	0.5	2	C	S	20/200	20/160	354	213	+1	2	2B#	25.3	5.5
18	63	M	18	2	C	S	20/100	20/400	395	257	–6	3	3B	51	12
19	66	M	120	2	C	S	20/400	20/200	566	1,147	+3	11	11B	91	8
20	63	M	23	1	MC	S	20/40	20/400	247	223	–10	4	4B	114	18
21	77	M	26	2	C	S	20/200	20/200	247	241	0	18	18B	55	4
22*	43	M	8	2	C	E	20/25	20/80	200	189	–5	2	2B	230	30
23*	43	M	8	2	C	S	20/1,200	20/400	169	202	>+5	2	2B	230	36
24	59	M	14	2	C	J	20/50	20/200	163	2,889	–6	2	2B	242	9
25	82	M	10	1	O	J	20/100	20/50	259	269	+3	4	3R + 1A	10	4
26	54	F	28	2	C	S	20/60	20/40	269	330	+2	4	3R + 1A	14	8
27	76	F	19	1	O	E	20/30	20/25	395	203	+1	4	4R	24	3
28*	34	M	14	1	O	E	20/2,000	20/20	207	220	>+15	4	3B + 1A	12	8
29*	34	M	14	1	O	E	20/200	20/20	237	278	+10	3	3B	12	9
30	84	M	22	2	C	J	20/60	20/40	382	384	+2	4	4R	15	3
31	75	M	31	1	O	J	20/50	20/20	289	280	+4	7	6R + 1A	28	7
32	50	M	42	2	C	J	20/25	20/25	278	275	0	7	5R + 2A	36	5
33	79	M	18	1	O	E	20/40	20/40	313	326	0	3	2R + 1A	22	10
34	46	M	8	3	C	S	20/100	20/20	329	201	+7	1	1R	9	8
35	57	F	48	1	O	S	20/40	20/40	316	321	0	1	1B	6	6
36	45	M	144	1	O	S	20/20	20/20	272	234	0	9	9R	48	5

(*continued on next page*)

Table 1. (Continued)

SN	Age, years	Sex	Duration of CSCR, months	CNV Type on OCT	CNV Type on FFA	CNV Location	Baseline VA	Final VA	Baseline CMT, μm	Final CMT, μm	Lines Changed at Last Follow-up	Total Number of Injections	Type of Injection	Total Follow-up, months	Longest Treatment-Free Interval, months
37	59	F	24	2	C	S	20/50	20/60	306	236	−1	3	3R	6	6
38	73	M	22	1	O	J	20/60	20/30	328	273	+3	6	5B + 1A	22	16
39	73	F	21	1	O	J	20/70	20/30	375	180	+3	10	3B + 7A	22	4
40	65	M	28	1	O	S	20/50	20/60	291	223	−1	22	22B	27	2
41	89	F	25	2	MC	S	20/70	20/40	269	184	+2	4	4B	24	8
42	52	M	28	1	O	S	20/400	20/125	345	210	+5	5	5B	23	4
43	37	M	24	1	O	S	20/80	20/60	175	124	+1	2	2B	4	2
44	29	M	12	2	O	S	20/60	20/60	305	300	0	1	1B	6	5
45	78	F	72	1	O	J	20/40	20/70	NA	424	−2	5	5B	72	24
46	61	F	72	1	O	J	20/32	20/25	320	286	+1	8	8B	5	5

*Bilateral involvement.

A, Aflibercept (2 mg/0.05 mL); B, Bevacizumab (1.25 mg/0.05 mL); B#, Bevacizumab (2.5 mg/0.05 mL); C, classic; CMT, central macular thickness; E, extrafoveal; F, female; J, juxtafoveal; M, male; MC, minimally classic; O, occult; R, Ranibizumab (0.5 mg/0.05 mL); S, subfoveal; VA, visual acuity.

standard deviation for continuous variables. Student's paired t-test was used for comparison. Statistical analyses were performed using MedCalc for Windows, version 12.5 (MedCalc Software, Ostend, Belgium). The alpha level (Type 1 error) was set at 0.01. Statistical significance was set at $P < 0.05$.

Results

This study included 46 eyes of 43 subjects with CNV secondary to CSCR, treated with intravitreal anti-VEGF therapy. Three subjects (6.5%) had bilateral involvement. There were 30 males and 13 females. Mean age was 57.56 years (range 29–79 years). Six eyes had undergone PDT for nonresolving CSCR at least 2 months (mean: 6 months; range: 2–24 months) before development of CNV. The rest of the eyes did not undergo any treatment for CSCR. Mean and median follow-up duration was 38.3 months ± 58.9 months and 19 months, respectively. None of the patients had history of steroid administration in any form. Among different anti-VEGF injections, 34 eyes (73.9%) were injected with intravitreal bevacizumab and 12 eyes (26.1%) received intravitreal ranibizumab. Because of poor response to intravitreal ranibizumab (5 eyes) and intravitreal bevacizumab (3 eyes), a total of 8 eyes (17.4%) were treated, received intravitreal aflibercept, and responded well.

Mean duration of CSCR before occurrence of CNV was 28.17 months ± 28.26 months. Among the 6 eyes, which underwent PDT for nonresolving CSCR, the mean duration of CSCR before occurrence of CNV was 40.5 months ± 36.9 months. However, in the remaining eyes, the same was 26.2 months ± 26.0 months, with no statistical difference ($P = 0.09$).

Among unilateral cases, fellow eye status was normal in 25 eyes, branch retinal vein occlusion in 1 eye, resolved CSCR in 11 eyes, epiretinal membrane in 1 eye, and macular scar due to scarred CNV in 2 eyes.

On fluorescein angiography, classic CNV was noted in 21 eyes (46%), minimally classic CNV in 6 (1.3%), and occult CNV in 19 (41.3%). Regarding location of CNV, extrafoveal was in 8 eyes (17%), juxtafoveal in 15 (33%), and subfoveal in 23 (50%). On SD-OCT examination, pigment epithelium detachment was noted at baseline in 10 eyes. Type 1 CNV (between the Bruch membrane and the RPE) was present in 20 eyes (43.4%), Type 2 (CNV between the RPE and the retina) in 25 eyes (54.3%), and 1 eye had suspected Type 3 CNV (intraretinal neovascularization).

Mean visual acuity in logMAR at baseline and at final follow-up was 0.59 logMAR ± 0.45 logMAR

(Snellen equivalent: 20/70, range: 20/2,000–20/20) and 0.48 logMAR ± 0.62 logMAR (Snellen equivalent: 20/60, range: hand motions to 20/20), respectively. Median visual acuity in logMAR at baseline and at final follow-up was 0.477 logMAR (Snellen equivalent: 20/60) and 0.3 logMAR (Snellen equivalent: 20/40), respectively. There was no statistically significant change in best-corrected visual acuity at final follow-up compared with baseline ($P = 0.23$). Mean central macular thickness at baseline and at final follow-up was 306.8 μm ± 126.6 μm and 346.2 μm ± 444.12 μm, respectively, without any statistically significant difference ($P = 0.57$). Median (the first and third quartiles) central macular thickness at baseline and at final follow-up was 290 μm (230.5, 358.25 μm) and 241 μm (196, 286 μm), respectively. Clinical characteristics of the patients are shown in Table 1.

Six subjects (7 eyes) were prescribed oral mineralocorticoid antagonist treatment (spironolactone 25 mg for 7 days followed by 50 mg daily) as part of treatment for CSCR, before the development of CNV, with a mean duration of 3.28 months (range: 3 weeks to 8 months).

Twenty-four eyes (52%) had visual improvement during follow-up. In 11 eyes (24%), vision did not change during the follow-up; however, the remaining 11 eyes (24%) had decrease in visual acuity. More than 3 lines of loss was noted in 6 eyes (13%), vision was stable (<3 lines of loss) in 16 eyes (34.5%), and >3 lines of improvement was seen in 12 eyes (26%). Mean number of anti-VEGF injections during the follow-up was 4.45 ± 4.1. Mean change in the number of lines was 1.16 ± 3.74. The longest treatment-free interval was 8.9 months ± 7.5 months. Median treatment-free interval was 7.8 months.

There were no serious systemic adverse events related to treatment during the follow-up. None of the eyes had any ocular complications after anti-VEGF therapy except 1 eye, which had increase in intraocular pressure (27 mmHg), controlled by antiglaucoma medication. Four eyes underwent additional PDT treatment during the follow-up in view of no response to anti-VEGF therapy. None of these 4 eyes had PDT as primary therapy for CSCR (Figures 1–3).

Discussion

The pathogenesis for the development of CNV in patients with CSCR is still not clear. Several theories including chronic decompensation or disruption at the RPE–Bruch membrane complex or increased VEGF expression secondary to choroidal ischemia have been reported to be the cause for formation of CNV.[19–21]

Diagnosis of CNV in eyes with chronic CSCR could be challenging because of the presence of associated pigmentation, RPE atrophy, and scarring. Diffuse leakage and staining on FFA could make the diagnosis and follow-up difficult in the presence of CNV. Cystic changes on SD-OCT are commonly associated with chronic CSCR; therefore, it is important to differentiate these degenerative changes from the changes secondary to active CNV to avoid unnecessary treatment.[22] Because of wide spread involvement of the disease, the treatment protocol for CNV secondary to CSCR may differ from the other causes of CNV. Due to chronic nature of the primary disease, the

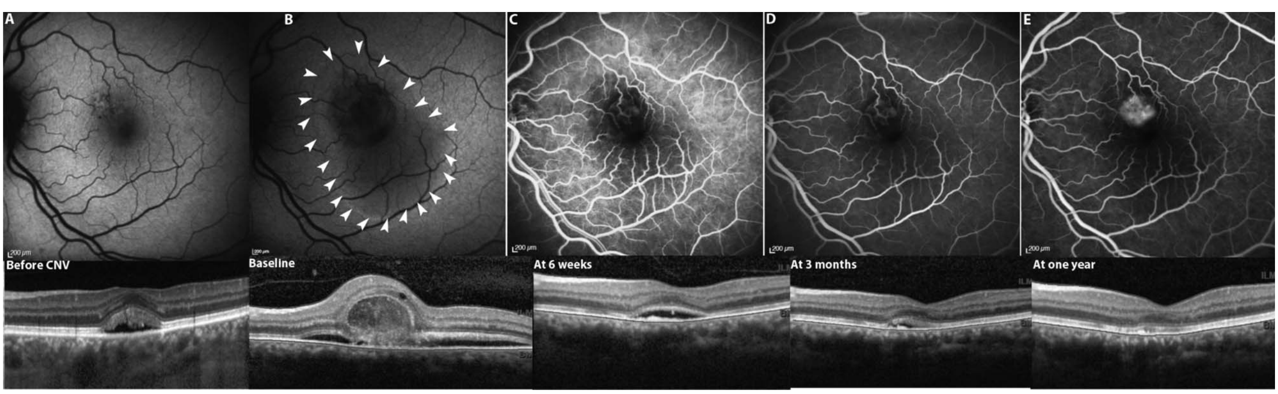

Fig. 1. A 65-year-old woman presented with a chief complaint of decreased visual acuity of her left eye for 3 weeks. On examination, her BCVA was 20/50 and macular examination showed subretinal fluid (SRF) with hyperautofluorescent and hypoautofluorescent areas superonasal to the fovea (**A**). Diagnosis of CSCR was made. The patient was asked to observe. The patient returned after 3 weeks with further loss in vision to 20/100 in the left eye. On examination, SRF with choroidal neovascular membrane was noted. Autofluorescence image (**B**) shows the extent of SRF shown as arrowheads, and central hypoautofluorescence is due to CNV. Fundus fluorescein angiography showed early hyperfluorescence with late leakage (**C–E**) confirming the diagnosis of CNV. Spectral domain optical coherence tomography showed the presence of Type 2 membrane at baseline. The patient was treated with seven intravitreal injections, and serial SD-OCT scans (bottom) showed significant improvement. At last follow-up of one year, her BCVA was 20/20 with no activity noted clinically and on SD-OCT scan. BCVA, best-corrected visual acuity.

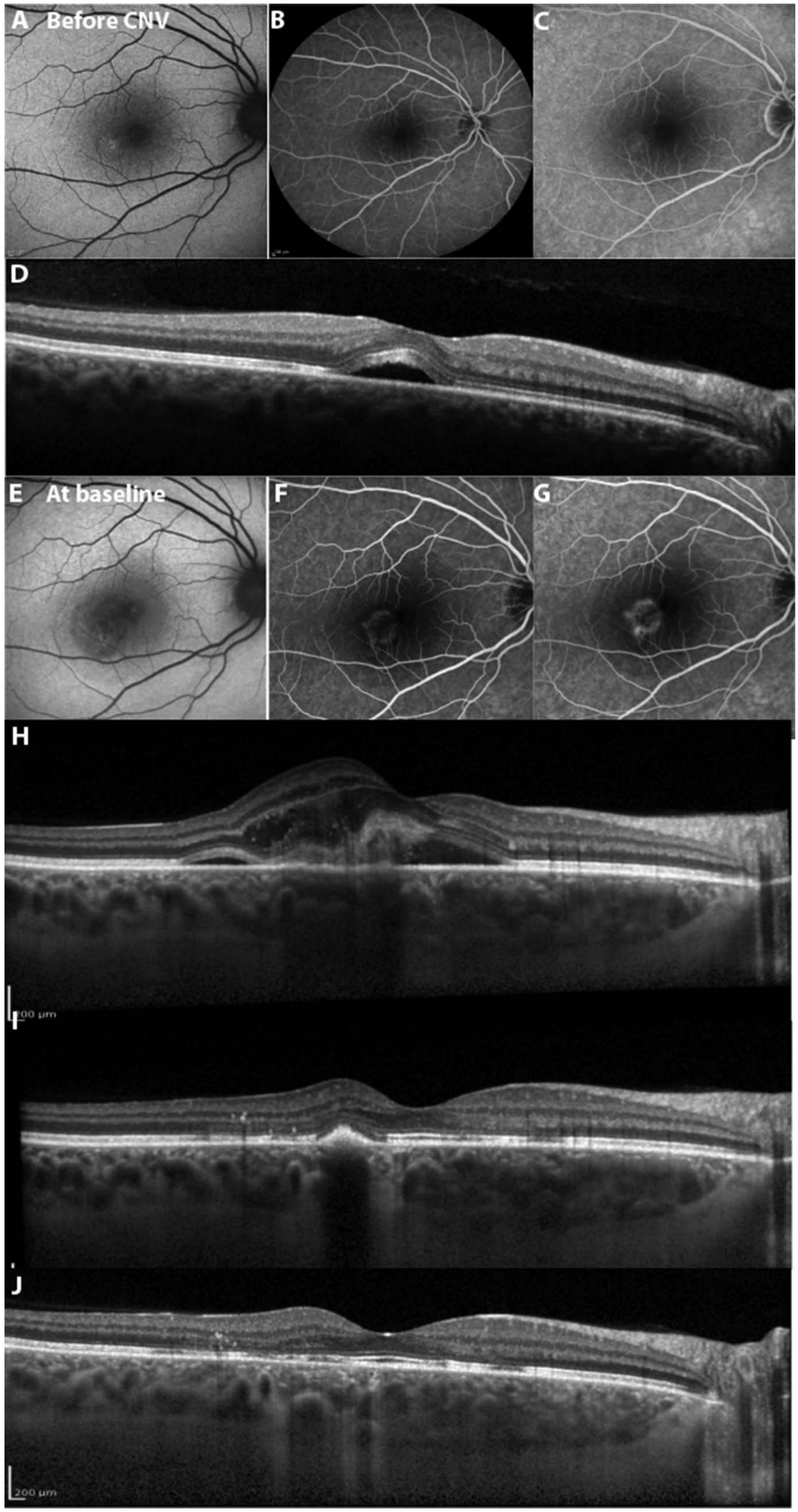

Fig. 2. A 45-year-old man, a known case of CSCR, presented with a chief complaint of significant decrease in visual acuity in his right eye for last 3 months. On examination, BCVA was 20/20 in his right eye and macular examination showed subretinal fluid. Fundus autofluorescence (**A**) shows hyperautofluorescence spot inferotemporal to the fovea with point leak seen on early and late phases of FFA (**B** and **C**). Spectral domain optical coherence tomography (**D**) showed presence of subretinal fluid. The patient was asked to observe. The patient returned back in 3 weeks with further loss in vision. On examination, BCVA was 20/200 in his right eye and macular examination showed subretinal fluid with choroidal neovascular membrane. Autofluorescence (**E**) shows increased area of hypoautofluorescence inferotemporal to the fovea. Fundus fluorescein angiography showed early hyperfluorescence with late leakage (**F** and **G**) confirming the diagnosis of CNV. Spectral domain optical coherence tomography showed the presence of Type 2 membrane at baseline (**H**). The patient was treated with single intravitreal injections, and SD-OCT scan at 2 months (**I**) showed resolution of subretinal and intraretinal fluid with CNV scarring. At 6 months of follow-up, BCVA was 20/20 in his right eye with no signs of activity noted clinically and on SD-OCT scan (**J**). BCVA, best-corrected visual acuity.

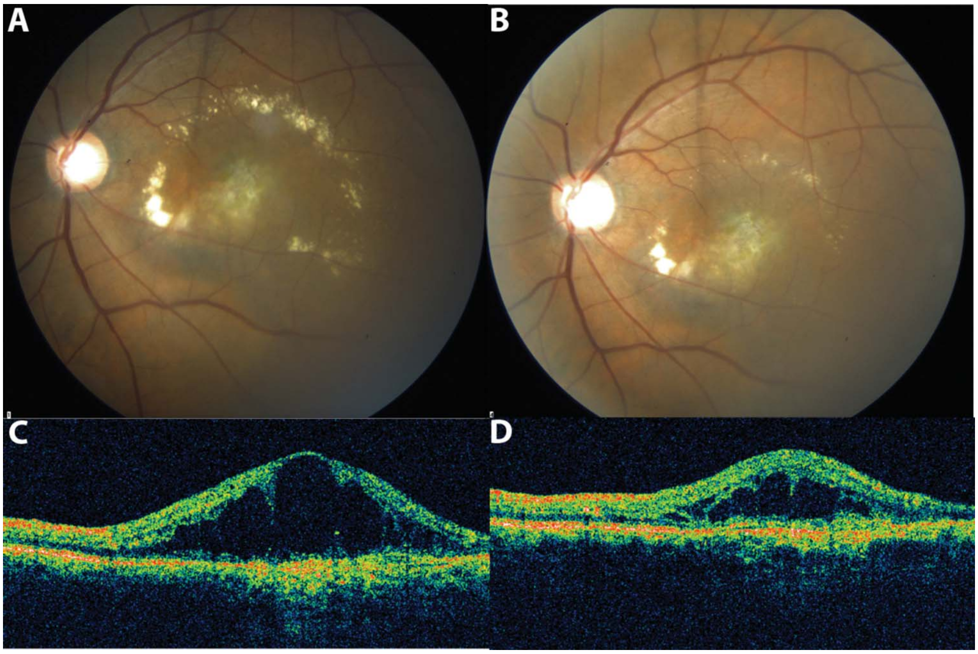

Fig. 3. A 66-year-old man, a known case of CSCR, presented with a chief complaint of significant decrease in visual acuity in his right eye for more than 6 months. On examination, his best-corrected visual acuity was 20/400 in his right eye and macular examination showed subretinal fluid with choroidal neovascular membrane involving the fovea with exudates (**A**). Spectral domain optical coherence tomography (**C**) showed retinal edema with chronic degenerative changes with thickening of the RPE–choriocapillaris complex. The patient was treated with 8 intravitreal injections of bevacizumab during the follow-up of 14 months. At the last follow-up, visual acuity was 20/200 with color fundus photograph (**B**) showed scarring of the membrane. Spectral domain optical coherence tomography showed retinal edema with persistent cystoid changes and scarring changes (**D**).

visual improvement could be limited because of associated RPE atrophy and damaged outer retinal structures.[22]

In our study, 52% eyes had some visual improvement with gain of 3 or more lines in 26% eyes. Previous studies on outcome of PDT for CNV secondary to CSCR report that 60% (6 of 10) eyes gained 2 or more lines of best-corrected visual acuity by Chan et al[7] and 27% (7 of 26) eyes showed 3 lines or more of improvement by Ergun et al.[8] Mean number of line improvement in our study was 1.16 ± 3.74 compared to 1.6 to 2.2 as reported by previous studies on PDT.[7,8] However, previous studies have very small sample size with short follow-up period and PDT-associated complications[23] such that RPE atrophy makes anti-VEGF injections as preferred treatment for this disease.

More than 3 lines of loss was noted in 6 eyes (13%), which is significantly more than that reported previously about CNV secondary to AMD. This could be due to RPE atrophy, pigmentary changes, chronic cystoid changes, scarring associated with primary disease, and CNV per se.

During the median follow-up period of 1.5 years, mean number of injections in our study was 4.4 ± 4.17, irrespective of the type of anti-VEGF therapy. The studies on myopic CNV reported mean number of anti-VEGF injections from 2 to 4.9.[18,24] Studies on inflammatory CNV reported a mean number of 1.3–2.3.[25,26] The limited literature on idiopathic CNV reported mean number of 3.1 injections.[27] In contrast, studies regarding anti-VEGF therapy for CNV secondary to AMD have reported the mean number of 23 injections up to 2 years.[28] Thus, CNV secondary to CSCR requires considerably less number of injections compared with AMD or non-AMD CNVs.

Central serous chorioretinopathy is a common disease in younger age group; however, the occurrence of CNV usually occurs in older age group. In our study, the mean age of study subjects was 57.56 years (range 29–79 years). Reports by Konstantinidis et al[11] and Chan et al[10] also report this disease in older age group with mean age of 66 years and 57.3 years, respectively. This could be due to chronic changes in RPE promoting formation of CNV in chronic CSCR.

Konstantinidis et al[11] reported successful treatment of 5 eyes with CNV secondary to CSCR with intravitreal ranibizumab. Mean number of injections was 10 ± 4.6 with persistence of intraretinal or subretinal fluid till the last follow-up. Chan et al[10] reported successful treatment with three monthly intravitreal bevacizumab injections in 2 eyes with CNV secondary to CSCR with >3 lines of improvement at 6 months of follow-up.

Typically, as in our study, a majority of the cases with CNV present eyes with chronic CSCR; however, Mandal et al[29] reported one case of CNV in an active CSCR. CNV can develop after laser photocoagulation for the leak, usually after few weeks to few months,[30–32] and the cause for this could be the break in the Bruch membrane. Choroidal neovascularization in CSCR can develop after PDT because of its pro-inflammatory effect.[33,34] Because of its focal ailment, we believe that this CNV secondary to laser photocoagulation or PDT in CSCR behaves differently from the CNV secondary to CSCR; therefore, we excluded such cases from this study.

Six subjects in our study were on oral mineralocorticoid receptor antagonist, which is recently being tried for treatment of chronic CSCR with variable results.[35] Because of the small number of subjects, we did not perform any statistical test to evaluate any differences in these subjects.

Previous reports showed that the CNV secondary to CSCR is typically of classic types on FFA with Type 2 features on SD-OCT.[12,36] Recently, Fung et al described Type 1 CNV secondary to CSCR, which could mimic CNV secondary to AMD. In our study, all patients had evidence of CSCR in the form of diffuse RPE atrophy, RPE tracks, and choroidal hyperpermeability on angiography. We had almost equal distribution of Types 1 and 2 CNV on OCT.

Our study has inherent limitations of a retrospective study. Because of multiple sites and physicians, there was no standard protocol for diagnosis, treatment, or follow-up regimen and there could be a selection bias. We did not analyze SD-OCT characteristics in detail; a prospective study may be required to evaluate SD-OCT characteristics as predictors for treatment outcome. There was no statistically significant difference in best-corrected visual acuity and central macular thickness at final follow-up compared with baseline, this could be due to the small sample size. Our study is unable to compare efficacy between the anti-VEGF drugs in treatment of CNV secondary to CSCR.

In conclusion, our study reports the largest case series of intravitreal antiangiogenic therapy for CNV secondary to CSCR and its outcomes. We propose anti-VEGF therapy as a primary therapy for CNV secondary to CSCR because it is safe and efficacious. Because of the rarity and character of this condition, it is unlikely that any clinical trials will soon take place to study this or alternate treatment options.

Key words: CNV, CSCR, choroidal neovascularization, central serous chorioretinopathy, anti-vascular endothelial growth factor.

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Appendix 1. Investigators of the King Khaled Eye Specialist Hospital (KKESH) International Collaborative Retina Study Group

The King Khaled Eye Specialist Hospital (KKESH): J. F. Arevalo, MD, FACS (PI); Eman Al Kahtani, MD; Sawsan Nowilaty, MD; Saba Al Rashaed, MD; Hassan A. Al-Dhibi, MD; Yahya A. Al-Zahrani, MD; I. Kozak, MD; Sulaiman Al-Sulaiman, MD; Abdulelah Al-Abdullah, MD; Ahmad Al-Bar, MD; Yousef Al Dhafiri, MD; Abdullah Al Qahtani, MD; Khalid Al Rubaie, MD; Saeed Al Shahrani, MD; Maha Al Shehri, MD; Badr Al Ahmadi, MD; Abdulaziz Al Hadlaq, MD; Majed Al Harbi, MD; Abdulaziz Al Oreany, MD.

Retina Division, Wilmer Eye Institute, Johns Hopkins University, Baltimore, MD: J. Fernando Arevalo, MD, FACS (PI); Catherine Meyerle, MD.

Smt. Kanuri Santhamma Retina Vitreous Centre, L. V. Prasad Eye Institute, Hyderabad, *India*: Jay Chhablani, MD.

San Giuseppe Hospital, University Eye Clinic, Milan, Italy: Francesco Pichi, MD; Antonella Scorza, MD; Paola Carrai, MD; Paolo Nucci, MD.

Department of Ophthalmology, University of Puezrto Rico, San Juan, Puerto Rico: Maria H. Berrocal, MD; Megan Chenworth, BS.

Cole Eye Institute, Cleveland Clinic, Cleveland, *Ohio*: Rishi P. Singh, MD; Rumneek Bedi, BS.

Instituto de Cirugia Ocular, San José, *Costa Rica*: Lihteh Wu, MD.

Department of Ophthalmology, Universidade Estadual de Londrina, Londrina, *Brazil*: Antonio Marcelo Casella, MD.

American University of Beirut, *Beirut*, *Lebanon and Rafic Hariri University Hospital*, *Beirut*, *Lebanon*: Ahmad Mansour, MD.

Department of Ophthalmology, American University of Beirut, *Beirut*, *Lebanon*: Ziad Bashshur, MD.