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Choroidal neovascularization in the pediatric age group

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Abstract

Purpose: The purpose of the study is to evaluate the clinical features, underlying etiology, and clinical outcomes of choroidal neovascularization (CNV) in the pediatric population.

Methods: This is a retrospective, single-center, interventional case series. A total of 12 eyes of 12 consecutive pediatric patients with CNV with various etiologies were analyzed. The main clinical parameters included the underlying causes, best-corrected visual acuity before and after the treatment, characteristics of the CNV, and the treatment strategies.

Results: There were four girls and eight boys with a median age of 12.3 ± 3 years (range: 7–17 years). Eight of 12 patients have completed the 6-month follow-up. The mean follow-up period was 32.8 ± 41 months (range: 6–132 months) in those 8 patients. Overall, five of them were treated. Four patients were treated with intravitreal anti-vascular endothelial growth factor (anti-VEGF) administration and the remaining patient with photodynamic therapy. Visual acuity improved from $\log\text{MAR } 0.54 \pm 0.2$ (range: $\log\text{MAR } 0.8–0.2$) to $\log\text{MAR } 0.26 \pm 0.18$ (range: $\log\text{MAR } 0.5–0.1$) at the last visit in the treated eyes. All anti-VEGF-treated patients required only a single injection.

Conclusion: CNV, a sight-threatening disease, is rarely seen in the pediatric age group. Families could be hesitant about the intravitreal treatment, but anti-VEGF injections seemed very helpful in our group of treated patients.

Keywords: Aflibercept; anti-vascular endothelial growth factor; choroidal neovascularization; optical coherence tomography angiography; photodynamic therapy; ranibizumab.

Choroidal neovascularization (CNV) is a rare macular disorder that can lead to serious visual impairment in pediatric patients.^[1–4] Although CNV is less common in pediatric patients compared to the adult population, it has more devastating consequences, given that a legally blind child faces significant challenges in education and social development.^[1,5] Pediatric CNVs can be idiopathic, but several underlying disorders, including inflammatory/infectious diseases, degenerative diseases, traumatic events, neoplastic diseases, and hereditary retinal dystrophies, can be present.^[3–5]

The morphological characteristics of pediatric CNV differ from those of adult CNV. While thickening and calcification of Bruch's membrane and diffuse disruption of the retinal pigment epithelium (RPE) are frequently seen in older patients with age-related macular degeneration (AMD), these findings are not commonly seen in children.^[6] Besides, most cases of CNV in older patients with AMD have multiple in-growth sites, whereas CNV in children is more likely to have a solitary in-growth site.^[7,8] It is believed that these morphological features can be the explanation for the possible spontaneous CNV regression in children.^[8]



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The management strategy can be somewhat different than that of adult patients with CNV. Treatment options for pediatric CNV depend on the etiology and include photodynamic therapy (PDT), thermal laser photocoagulation, surgical removal of the membrane, and/or intravitreal anti-vascular endothelial growth factor (anti-VEGF) agents.^[8-10]

Herein, we report a case series of 12 eyes of 12 patients under 18 years of age presenting with CNV and share our experience.

Materials and Methods

Medical records of all patients under 18 years old who presented with CNV to our clinic between 2007 and 2023 were retrospectively analyzed. All of the patients were examined by one of us (AOS). The study was conducted by the ethical standards of the Declaration of Helsinki. Approval was obtained from the Local Ethics Committee (registration code: 8618-GOA, September 09, 2024). Patient demographics, fundus characteristics, treatment approach, and outcomes were examined. Multimodal imaging methods, such as optical coherence tomography angiography (OCTA), fluorescein angiography (FA), and optical coherence tomography (OCT), were used to diagnose CNV.

CNV lesions were classified as follows: Type 1 (a neovascular complex between the Bruch's membrane and RPE, originating from the choroid), type 2 (located above the RPE), and type 3 (intraretinal, retinal angiomatous proliferation).^[3,11-14]

According to the macular photocoagulation study (MPS) protocol, CNV lesions were classified as classic (early lacy hyperfluorescence), occult (late leakage or leakage from an indistinct focus), or minimally classic depending on the pattern of fluorescein findings.^[15]

Active CNV was defined as the presence of subretinal or intraretinal fluid, new or persistent leakage on FA, and/or flow signal corresponding to the CNV complex on OCTA. Regression was defined as the resolution of subretinal/intraretinal fluid and absence of leakage or flow signal on multimodal imaging.

CNV location was defined according to criteria established by the MPS and classified as subfoveal (for lesions within the foveal avascular zone [FAZ]), juxtafoveal (for lesions <200 μ m from the FAZ), or extrafoveal (for lesions >200 μ m from the FAZ) locations. Lesions located adjacent to the disc or within one disc diameter from the optic disc border were considered peripapillary CNVs.

The treatment decision was made on an individual basis, considering the etiology and ocular conditions.

All intravitreal injections were performed under general anesthesia using standard aseptic technique. Retreatment was considered in cases of persistent or recurrent activity on OCT after 4–6 weeks. Patients were monitored every 4 to 6 weeks as possible till the foveal stabilization was obtained, then followed with extended intervals.

Statistical Analysis

IBM Statistical Package for the Social Sciences Statistics Standard Concurrent User, version 26 (IBM Corp., Armonk, New York, USA) was used for analyzing data and worked at a 95% confidence level. Descriptive statistics were presented as the number of units, percentage, mean \pm standard deviation, minimum, and maximum values. Due to the small sample size and the paired nature of the data, changes in best-corrected visual acuity (BCVA) were analyzed using the Wilcoxon signed-rank test. Visual acuities were converted to logMAR equivalents and processed.

Results

Overall, 12 eyes of 12 pediatric patients with CNV were scrutinized. The mean age was 12.3 \pm 3 (range: 7–17 years). There were eight (67%) boys and four (33%) girls. All patients had unilateral CNV.

Patient's characteristics have been shown in Table 1. The follow-up period in 4 patients was <6 months, as they were lost to follow-up after the initial visit. Thus, eight patients could be followed for 6 months or more. In those, the average follow-up was 32.8 \pm 41 months (range: 6–132 months). None of the patients had any concomitant systemic disease.

CNV was associated with several eye diseases: Optic disc drusen in three eyes (25%), laser pointer injury in two eyes (17%), uveitis in two eyes (17%), Bietti crystalline dystrophy in one eye (8%), and best disease in one eye (8%). One of our patients developed CNV after laser injury at the workplace (8%). Idiopathic CNV was diagnosed in two eyes (17%) without any underlying ocular disease (Table 1).

While CNV location was subfoveal in 8 eyes (67%), it was peripapillary in three eyes (25%). In one eye, the CNV lesion was located extrafoveally (8%) (Fig. 1 and 2). Lesion type was Type 2 in eight eyes (67%) and Type 1 in four (33%) eyes.

The mean BCVA at the first visit, in all eyes, was logMAR 0.54 \pm 0.32 (range: logMAR 0–1). The mean BCVA in peripapillary located eyes was logMAR 0.13 \pm 0.1 (range: logMAR 0–0.2) and was found to be better than in subfoveal located eyes. The mean BCVA in eyes with subfoveal CNV was logMAR 0.7 \pm 0.2 (range: logMAR 1–0.5) at initial visit.

Table 1. Demographic and clinical data of 12 children with CNV

Eye no.	Patient age (years)/sex	Etiology	Location	CNV type	CNV status at presentation	BCVA at presentation	Treatment received	Interval between presentation and last visit	CNV status during last visit	BCVA at last visit
1	7/F	Best's disease	Subfoveal	Type 1	Active	20/125	None	10 months	Active	20/125
2	14/M	Bietti crystalline dystrophy	Subfoveal	Type 2	Active	20/100	Ranibizumab x1	24 months	Quiescent	20/25
3	14/F	Uveitis	Subfoveal	Type 1	Inactive	20/63	Aflibercept x1	24 months	Quiescent	20/32
4	9/M	Laser pointer injury	Subfoveal	Type 2	Active	Unavailable	None	None	Active	None
5	13/M	Optic disc drusen	Peripapillary	Type 2	Active	20/32	PDT x1	132 months	Quiescent	20/25
6	14/M	Optic disc drusen	Peripapillary	Type 2	Active	20/32	None	None	Active	None
7	17/F	Uveitis	Subfoveal	Type 1	Active	20/125	Ranibizumab x1	24 months	Quiescent	20/63
8	11/M	Idiopathic	Subfoveal	Type 2	Active	20/63	Aflibercept x1	6 months	Quiescent	20/50
9	9/M	Laser pointer injury	Subfoveal	Type 2	Active	20/200	None	6 months	Active	20/200
10	11/F	Optic disc drusen	Peripapillary	Type 1	Active	20/20	Observation	36 months	Quiescent	20/20
11	15/M	Laser injury at workplace	Subfoveal	Type 2	Active	20/100	None	None	Active	None
12	14/M	Idiopathic	Extrafoveal	Type 2	Active	Unavailable	None	None	Active	None

CNV was considered active in 11 of 12 eyes. Among those with active CNV, 5 eyes underwent treatment. Four eyes of four patients could not receive treatment because they were lost to follow-up, considering the presence of active CNV at the first visit. Parents of 3 patients denied any injection treatment after hearing the possible treatment complications in detail. In one of our uveitic patients, the CNV lesion was inactive at the initial visit, so she was observed. CNV in all eyes remained regressed with a mean BCVA of logMAR 0.38 ± 0.36 (range: logMAR 0–1) at the last visit.

In our clinic, intravitreal anti-VEGF agents were the first treatment of choice for CNV in pediatric patients, and all injections were performed under general anesthesia. An equal number of patients were treated with ranibizumab (Lucentis®; Novartis, Basel, Switzerland; 0.5 mg in 0.05 mL) ($n=2$) and aflibercept (Eylea®; Bayer, Leverkusen, Germany; 2 mg in 0,05 mL) ($n=2$). All of them remained stable with a single injection only, and no recurrences were observed during the follow-ups. One eye was treated successfully with PDT, diagnosed before the anti-VEGF era.

Five eyes had responded well to treatment (4 eyes underwent anti-VEGF injections and 1 eye underwent PDT), showing increased visual acuity from logMAR 0.54 ± 0.2 (range: logMAR 0.8–0.2) to logMAR 0.26 ± 0.18 (range: logMAR 0.5–0.1) at the last visit. Although the Wilcoxon signed-rank test did not reach conventional statistical significance ($p=0.063$), the effect size was large, indicating a clinically meaningful improvement in BCVA despite the small sample size. BCVA remained stable in three eyes of three patients, whose parents did not consent to intravitreal anti-VEGF therapy and remained untreated.

Figure 3 shows the OCT and fundus images of an 11-year-old boy with idiopathic CNV before intravitreal anti-VEGF treatment. After a single dose of intravitreal anti-VEGF treatment, his fundus and OCT findings remained regressed (Fig. 4).

Discussion

In the current study, we evaluated the clinical features of 12 eyes of 12 pediatric patients with CNV. In our case series, the most common

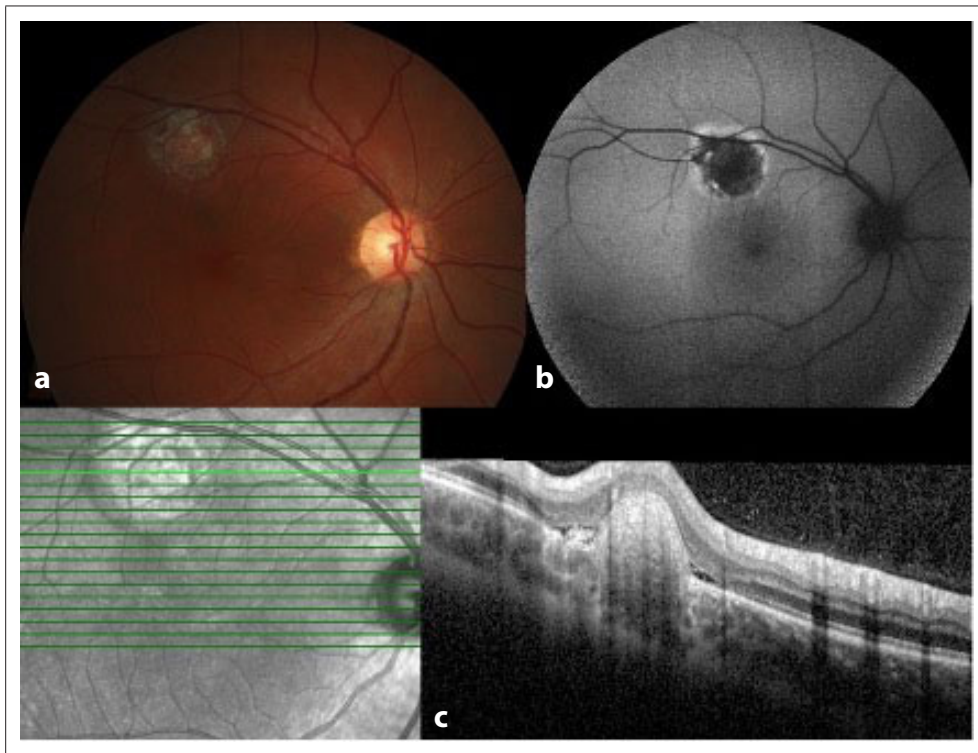


Fig. 1. Right eye, patient 12, at the presentation. **(a)** Color fundus picture revealing an extrafoveal yellowish lesion, together with a tiny intraretinal hemorrhage. **(b)** Fundus autofluorescence imaging showing a hypoautofluorescent lesion surrounded by a hyperautofluorescent rim superior to the fovea. **(c)** Optical coherence tomography exhibiting a minute subretinal fluid with subretinal hyperreflectance.

etiologies were optic disc drusen (25%). Previous large series on pediatric CNV were summarized in Table 2.

In the Intelligent Research in Sight (IRIS) registry, Finn *et al.*^[16] reported the features of 2353 eyes with pediatric CNV. In their study, the most common etiology was posterior uveitis/

inflammatory chorioretinal disease in 19.4% of patients. Other common etiologies were myopia (18.4%) and hereditary dystrophy (5.4%). In 38.2% of eyes, CNV was claimed as idiopathic.

In parallel to the previous studies, CNV was located subfoveally (67%) in most eyes in our group. However, in

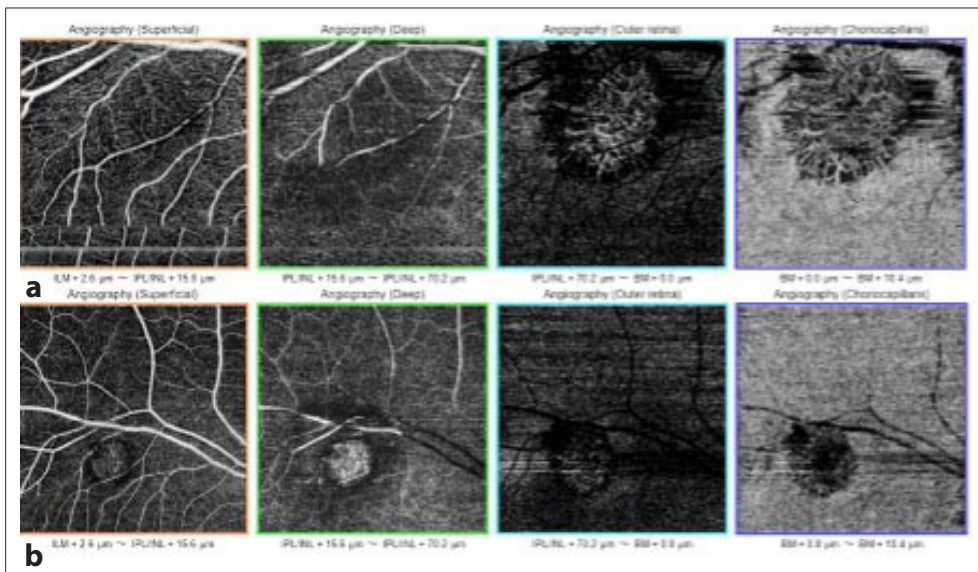


Fig. 2. Right eye, Patient 12, at the presentation. 3 × 3 mm **(a)** and 6 × 6 mm **(b)** Optical coherence tomography angiography slabs demonstrating the neovascular complex exquisitely.

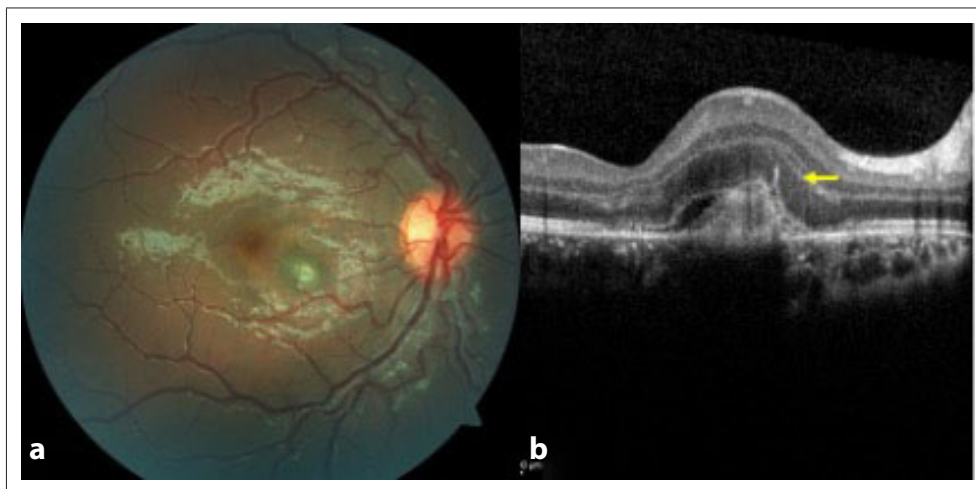


Fig. 3. Right eye, Patient 8. At the presentation, (a) Color fundus picture depicting the extrafoveal. Yellowish lesion, optical coherence tomography section (b) depicting the subretinal hyperreflective area with some subretinal fluid and hyperreflective finger-like projections (pitchfork sign) (yellow arrow).

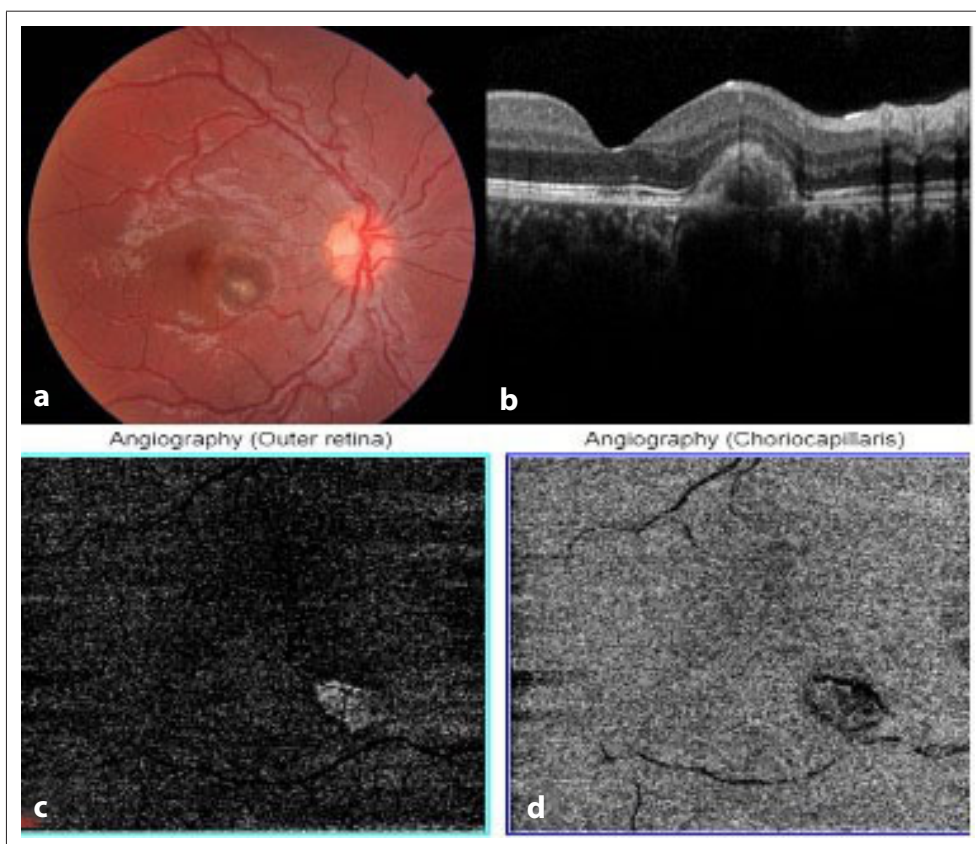


Fig. 4. Right eye, Patient 8, after the anti-vascular endothelial growth factor (anti-VEGF) treatment. (a and b) Optical coherence tomography section and the color fundus. Picture demonstrating the regressed lesion without any subretinal fluid following a single dose of intravitreal anti-VEGF (aflibercept 2 mg) treatment. (c and d) Outer retina and choriocapillaris slabs revealing the neovascular tuft on optical coherence tomography angiography.

three eyes with optic disc drusen, the CNV location was peripapillary. Peripapillary CNV has been reported to be associated with preexisting ocular pathology rather than being idiopathic, like the subfoveal CNVs.^[7,17] Peripapillary

CNV may occur as a consequence of various optic nerve diseases such as idiopathic intracranial hypertension, chronic papilledema, pseudopapilledema, optic disc drusen, malignant hypertension, and optic nerve head

Table 2. Previous large series in the literature on pediatric CNV

Study (year)	Number of eyes (patients)	Nature of study	Average follow-up (months)	Most common etiology (%)	Treatment / no. of eyes	Average injections per eye	BCVA at presentation	BCVA at final visit
Rishi et al. (2013)	36 (27)	Single-centre, retrospective	26 (range: 0.07-111.8)	Inflammatory (41.7)	Anti-VEGF/2 (bevacizumab 1.25 mg) PDT/5 TTT/6 Laser/1	4	20/600	20/400
Kozak et al. (2014)	45 (39)	Multi-centre, retrospective	12.8 (range: 3-60)	Inflammatory (38.4)	Anti-VEGF/45 (ranibizumab 0.5 mg and bevacizumab 1.25 mg)	2.79 (1-13)	20/150	20/100
Barth et al. (2016)	10 (8)	Single-centre, retrospective	35 (range: 6-120)	Choroidal osteoma (30)	Anti-VEGF/7 (afibercept 2 mg, ranibizumab 0.5 mg and bevacizumab 1.25 mg) PDT/3	5.4 (2-10)	logMAR 0.48	logMAR 0.46
Padhi et al. (2017)	43 (35)	Multi-centre, retrospective	Range: 6-84	Retinal dystrophy (39.5)	Anti-VEGF/27 (ranibizumab, bevacizumab and pegaptanib) PDT/1 Laser/1	2.1	Overall visual gain	logMAR 0.43
Ranjan et al. (2021)	26 (23)	Single-centre, retrospective	28.1 (range: 8-72)	Inflammatory (61.5)	Anti-VEGF/19 (ranibizumab 0.5 mg and bevacizumab 1.25 mg)	2	20/125	20/50
Zhang et al. (2021)	33 (30)	Single-centre, prospective	29 (range: 6-60)	Congenital abnormalities (30) Inflammatory (30)	Anti-VEGF/25 (afibercept 2 mg and ranibizumab 0.5 mg) Surgery/1	1.4	logMAR 0.96	logMAR 0.85
Current study	12 (12)	Single-centre, retrospective	32.8 (range: 6-132)	Optic disc drusen (25) Laser point injury (17) Uveitis (17)	Anti-VEGF/4 (afibercept 2 mg and ranibizumab 0.5 mg) PDT/1	1	logMAR 0.54	logMAR 0.38

PDT: Photodynamic therapy; TTT: Transpupillary thermotherapy

anomalies.^[18] Notably, Rishi *et al.*^[7] reported that in their case series, all patients with peripapillary CNV had optic disc drusen.

In our study, a total of 5 eyes underwent treatment. Treatment modalities were tailored individually for each patient. Underlying ocular diseases, CNV location, activation, and systemic conditions were the determining factors.

The initial treatment strategy for pediatric CNV in our clinic was intravitreal anti-VEGF agents. The increasing use of intravitreal anti-VEGF agents has been a major advancement in the management of vitreoretinal diseases in pediatric patients, including CNV. However, it is not currently known whether the short-term suppression of systemic VEGF results in long-term neurodevelopmental outcomes, especially in younger children.^[2,19-23]

Previous reports showed that pediatric CNVs had fewer recurrences and required fewer injections in contrast to CNVs that occurred in adults. Finn *et al.*^[16] mentioned in their IRIS study that CNV was most likely to be managed with anti-VEGF agents in pediatric patients. In their study, 68.4% of eyes treated with anti-VEGF agents required 3 or fewer injections.^[16]

In a subgroup analysis of the MINERVA study – in the open-label, non-randomized study arm – Hykin *et al.*^[24] showed that ranibizumab 0.5 mg treatment for 12 months was beneficial for improving BCVA in adolescent patients with CNV with a relatively small number of injections. Their results were consistent with previously published literature in the young population, as we mentioned before. Table 2 summarizes previous large series in the literature on pediatric CNV treated with anti-VEGF.^[1-4,7,25]

In all patients, injections were performed under general anesthesia. None of them required more than 1 anti-VEGF injection. Among 4 eyes with subfoveal CNV, the mean BCVA for eyes that underwent treatment was logMAR 0.62 ± 0.15 (range: 0.8–0.5) at presentation which improved to mean logMAR 0.3 ± 0.2 (range: 0.5–0.1) on regression, suggesting the favorable impact of intravitreal anti-VEGF treatment for active disease. Therefore, according to the efficacy and favorable prognosis of limited anti-VEGF therapy, it is recommended that timely treatment for CNV should be considered in the pediatric population.^[3,7,25]

One of our patients with peripapillary CNV due to optic disc drusen underwent PDT in 2007. The use of PDT in pediatric CNV has been reported in some case series and reports before. The results of these studies have demonstrated the safety and efficacy of PDT in pediatric CNV.^[26-29] In addition, sometimes unfavorability to PDT may be observed, and intravitreal anti-VEGF injections may be necessary in cases of resistance to PDT.

^[30] In our patient, BCVA increased after he underwent PDT, and intravitreal anti-VEGF treatment was not required afterwards.

In uveitic cases with CNV, if the disease is also active, systemic treatment for the uveitis control is mandatory.^[23] In our patient with intermediate uveitis, oral azathioprine (1.5 mg/kg/day) was administered in association with the intravitreal anti-VEGF treatment. Uveitis activity was monitored by careful clinical examination and FA when deemed necessary. No serious side effects were observed in any patient during the follow-up period.

The limitations of our study are its retrospective design, small number of cases, and the lack of a standard protocol. The small sample size and incomplete follow-up of some patients limit the generalizability of our findings. The loss to follow-up in one-third of cases might have created a selection bias. The study period (2007–2023) spans major advances in imaging modalities and treatment protocols, from the pre-anti-VEGF era to the utilization of OCTA and newer intravitreal agents. This heterogeneity may have affected the diagnostic sensitivity, treatment selection, and thereby the clinical outcome.

Conclusion

CNV in the pediatric population is a serious condition with severe visual consequences. In our series, intravitreal anti-VEGF treatment was an effective, safe option with no serious side effects even with a small number of injections. Since there is no standardized treatment protocol for pediatric patients, every patient should be thoroughly evaluated individually, and appropriate treatment should be selected.

Ethics Committee Approval: This study was approved by The Ethics Committee of Dokuz Eylul University (registration code: 8618-GOA, September 09, 2024).

Informed Consent: Written informed consents were obtained from patient and his family.

Peer-review: Externally peer-reviewed.

Authorship Contributions: Design: T.O., A.O.; Supervision: A.O.S.; Resource: ; Materials: I.K., Z.A., M.K.; Data Collection and/or Processing: I.K., T.O.; Analysis and/or Interpretation: I.K., T.O.; Literature Search: I.K.; Writing: I.K., A.O.S.; Critical Reviews: A.O.S.

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