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Хориоидальная неоваскуляризация у детей: этиология, диагностика и клинические проявления

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АННОТАЦИЯ

Исследования, посвящённые анализу хориоидальной неоваскуляризации (ХНВ) у детей, носят ограниченный характер вследствие низкой частоты развития данного осложнения в офтальмопедиатрии. Однако, учитывая значимое влияние на остроту зрения и трудности диагностики у детей, изучение данного осложнения является актуальным.

Цель. Анализ этиологической структуры и клинических особенностей хориоидальной неоваскуляризации у детей.

Материал и методы. За период 2014–2022 гг. под нашим наблюдением находилось 54 ребёнка (26 девочек и 28 мальчиков), 61 глаз с ХНВ. Помимо стандартного офтальмологического обследования пациентам проводилась оптическая когерентная томография (ОКТ) и ОКТ-ангиография (ОКТА) макулярной зоны и диска зрительного нерва на томографе RS-3000 Advance 2, Nidek (Япония).

Результаты. Возраст детей на момент диагностирования ХНВ составил от 5 до 17 лет (в среднем 11±3 лет). У 30 детей (55,6%) ХНВ сформировалась вследствие воспалительного поражения сетчатки и хориоидеи, из них у 11 — в период ремиссии, у 21 — на фоне патологии сетчатки, хориоидеи и зрительного нерва невоспалительного генеза (у 8 из них — на фоне болезни Беста), у 3 детей ХНВ была расценена как идиопатическая. Срок развития поствоспалительной ХНВ варьировал от 1 месяца до 12 лет, в среднем 7,3±5 месяцев от дебюта заболевания. В большинстве случаев (48 глаз, 78,7%) определялась ХНВ тип 2.

Заключение. Хориоидальная неоваскуляризация является редким осложнением широкого спектра заболеваний глазного дна у детей. В нашей когорте её развитие наиболее часто наблюдалось на фоне воспалительного поражения сетчатки и хориоидеи, в том числе в стадии ремиссии. Разные сроки формирования ХНВ требуют активного мониторинга пациентов с риском её развития.

Ключевые слова: хориоидальная неоваскуляризация; дети; увеит; врождённая и наследственная патология сетчатки и диска зрительного нерва.

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Choroidal neovascularization in children: Etiology, diagnosis, and clinical manifestations

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ABSTRACT

Few studies have analyzed choroidal neovascularization (CNV) in children because of the low incidence of this complication in pediatric ophthalmology. However, given the significant effect on visual acuity and diagnostic difficulties in children, the study of this complication is relevant.

AIM: This study aimed to analyze the etiological structure and clinical features of CNV in children.

MATERIAL AND METHODS: From 2014 to 2022, 61 eyes of 54 children (26 girls and 28 boys) had CNV. The patients underwent standard ophthalmologic examination and optical coherence tomography (OCT) and OCT angiography (OCTA) of the macular zone and optic disc using the RS-3000 Advance 2 Tomograph (Nidek, Japan).

RESULTS: At the time of CNV diagnosis, the children were 5–17 years old, with a mean of 11±3 years. In 30 children (55.6%), CNV was caused by inflammatory lesions of the retina and choroid. Of these cases, 11 occurred during remission, whereas 21 occurred alongside pathologies of the retina, choroid, and optic nerve of noninflammatory genesis (8 of which were associated with Best's disease). In three children, CNV was considered idiopathic. The development time of postinflammatory CNV ranged from 1 month to 12 years, with an average of 7.3±5 months from disease onset. Type 2 CNV was found in most cases (48 eyes, 78.7%).

CONCLUSION: CNV is a rare complication of various ocular diseases in children. In our cohort, it was most frequently observed in children with inflammatory lesions of the retina and choroid, even during remission. Patients at risk of CNV must be actively monitored because it occurs in various forms.

Keywords: choroidal neovascularization; children; uveitis; congenital and hereditary pathology of the retina and optic nerve disc.

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INTRODUCTION

Choroidal neovascularization (CNV) is a serious complication of ocular diseases that disrupts the structure of Bruch's membrane and the retinal pigment epithelium. Age-related macular degeneration is the primary pathology that leads to the development of the choroidal neovascular membrane (CNM) [1]. Myopia complications are the second most common cause of CNV [2].

Studies analyzing CNV in children are limited because of its relatively rare occurrence in pediatric ophthalmology. According to M. Moosajee et al., CNM affects 0.21 per 100,000 pediatric patients [3]. However, analysis of this complication is important because of its significant effect on visual acuity, as it predominantly localizes in the center of the ocular fundus and can be difficult to diagnose in children. In children, CNV has been reported in various ocular pathologies, including myopia, chorioretinitis, congenital anomalies, retinal dystrophies, and ocular trauma [2–16]. To ensure timely diagnosis and appropriate treatment, the etiology of diseases in which CNV can occur in pediatric ophthalmology as well as the clinical manifestations and algorithm of instrumental examination in case of suspected CNV development must be elucidated. **Thus, this study aimed** to analyze the etiologic structure and clinical features of CNV in children.

MATERIAL AND METHODS

A retrospective analysis was conducted on the etiologic structure and clinical manifestations of CNV in 54 children (26 girls and 28 boys, 61 eyes with CNV) who were treated in the Pediatric Surgery Department of the Helmholtz Eye Diseases Research Center between 2014 and 2022.

In addition to a standard ophthalmic examination, optical coherence tomography (OCT) of the macula and optic disc was performed. Since 2016, OCT with angiography function (OCTA) was also used for diagnosis. An RS-3000 Advance 2 Tomograph (Nidek, Japan) was used for these procedures.

RESULTS

At the time of CNV detection, the children were 5–17 years old (mean age, 11±3 years). Only half of the children reported experiencing decreased vision and/or spotting in front of their eyes. In children who were previously diagnosed with ocular fundus pathology but did not report any complaints, CNM was detected during routine examination. In other cases, children visited an ophthalmologist because of strabismus or visual impairment detected accidentally in everyday life. The best-corrected visual acuity ranged from 0.01 to 1.0 (mean, 0.37±0.3). In addition to age-related factors, the absence of complaints in some patients was attributed to the underlying disease, including central chorioretinal foci and dystrophy, partial complicated cataract, glaucoma, and partial vitreous fibrosis in patients with uveitis.

Importantly, 15 out of 54 (27.8%) children were not diagnosed with CNV at their place of residence and were referred to the Helmholtz Eye Diseases Research Center with diagnoses such as active chorioretinitis (11 children), choroidal neoplasm (2 children), macular degeneration (1 child), and Best's disease (1 child). Diagnostic errors often occur because of the lack of medical alertness to potential complications, resulting in insufficient examination of the patients. This can be a significant issue in pediatric patients.

Table 1 presents diseases complicated by CNV development in the group of inflammatory diseases of the retina and choroid and the inflammatory process during its development.

In inflammatory lesions of the retina and optic nerve, the period of CNV development from the onset of the main disease varied widely, averaging 7.3±5 months and ranging from 1 month to 12 years.

CNM was unilateral in 47 children and bilateral in 7 children. One child had uveitis of unclear etiology, 2 had panuveitis associated with Vogt–Koyanagi–Harada disease, 1 had multifocal choroiditis, and 3 had Best's disease.

In 48 children (54 eyes), CNM was localized in the sub- and juxtapapillary zones. Juxtapapillary CNM was found in 7 children (8 eyes), including 1 child with optic disc drusen, 1 with neurochorioretinitis, 1 with chorioretinitis of unclear etiology, 1 with scarring PH, 1 with idiopathic type, and 2 children with panuveitis associated with the Vogt–Koyanagi–Harada disease.

CNM was active in all children. The clinical presentation varied depending on the underlying disease. For example, a grayish subretinal focus with indistinct contours and perifocal hemorrhage, or without hemorrhage, was observed in the chorioretinal/choroidal focus area (Fig. 1). In addition, osteoma (Fig. 2) or hamartoma, and dystrophic changes in the macula in Best's disease, were observed. Patients with myopia (Fig. 3) and idiopathic forms presented with isolated grayish or yellowish foci with indistinct contours and perifocal hemorrhage, or without it. Juxtapapillary CNM was characterized by the formation of a grayish proliferating focus adjacent to the optic disc (Fig. 4).

In this study, 41 children (48 eyes, 78.7%) had type 2 CNM localized between the pigment epithelium and neurosensory retina (Fig. 5). In addition, 13 children (13 eyes) had type 1 CNM developing under the pigment epithelium (Fig. 6). No correlation was established between the CNM type and underlying disease. Type 1 was diagnosed in six children with inflammatory lesions of the retina and choroid with varying activity and localizations and in seven children with congenital and acquired retinal and optic nerve pathologies of noninflammatory genesis.

OCT and OCTA characteristics confirmed the presence and activity of CNM. These characteristics include intra- and subretinal retinal edema surrounding the hyper-reflective subretinal focus and presence of a network of densely

Table 1. Etiologic pattern of choroidal neovascularization in a pediatric cohort

Underlying disease	Number of children
Inflammatory lesions of the retina and choroid:	A total of 30 children
— Central chorioretinitis of unclear etiology and remission	4
— Central chorioretinitis of unclear etiology and unknown activity*	2
— Juxtapapillary chorioretinitis of unclear etiology and remission	1
— Panuveitis associated with the Vogt–Koyanagi–Harada disease	7, of these:
• Active	1
• Sluggish	4
• Subactive	2
— Panuveitis with choroiditis of unclear etiology	3, of these:
• Sluggish	2
• Subactive	2
• Unknown activity*	1
— Toxoplasmosis, retinochoroiditis, and remission	4
— Choroiditis of unclear etiology	6, of these:
• Remission	1
• Unknown activity *	5
— Neurochorioretinitis, remission	1
Congenital and acquired pathology of the retina, choroid, and optic nerve of noninflammatory genesis	A total of 21
— Best's disease	8
— High, moderate, low myopia	7
— Retinal photodamage	2
— Retinopathy of prematurity and scarring phase	1
— Choroidal osteoma	1
— Retinal hamartoma	1
— Optic disc drusen	1
Idiopathic	3

* Whether uveitis was active at the time of CNM formation was unclear because of delayed treatment at the Helmholtz Eye Diseases Research Center and incomplete medical history.

anastomosing newly formed vessels in the area of the focus projection (Fig. 7).

Instrumental diagnostics are crucial for diagnosing CNV and determining its activity, even in the absence of clinical symptoms. OCT and OCTA should be used to exclude the presence of CNV activity when clinical signs are absent (Fig. 8).

Furthermore, cases of subclinical CNV development, such as in Best's disease, where pathological neovascularization

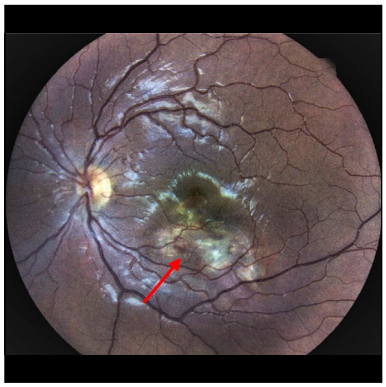

Fig. 1. Ophthalmoscopic picture of an active choroidal neovascularization (arrow) in a child with central chorioretinitis of unclear etiology in remission.

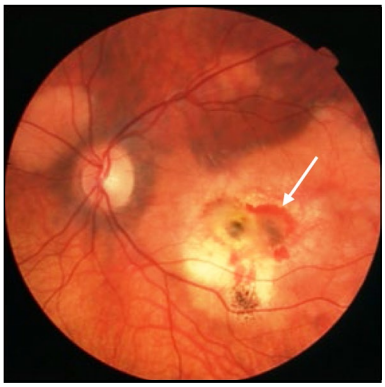

Fig. 2. Ophthalmoscopic picture of active choroidal neovascularization (arrow) in a child with choroidal osteoma. A focus with indistinct contours and perifocal hemorrhage at the edge of the osteoma is shown.

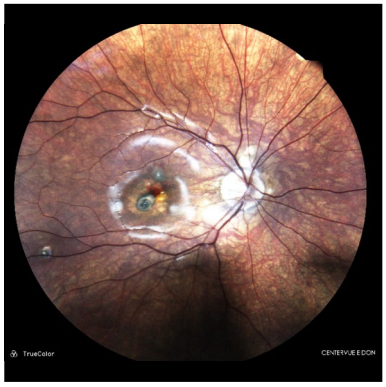

Fig. 3. Ophthalmoscopic image of active choroidal neovascularization in a child with high-degree congenital myopia. A grayish proliferating focus with perifocal hemorrhage is shown.

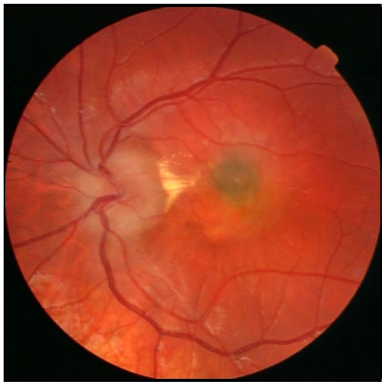

Fig. 4. Ophthalmoscopic image of subfoveolar active choroidal neovascularization in a child with optic disc drusen.

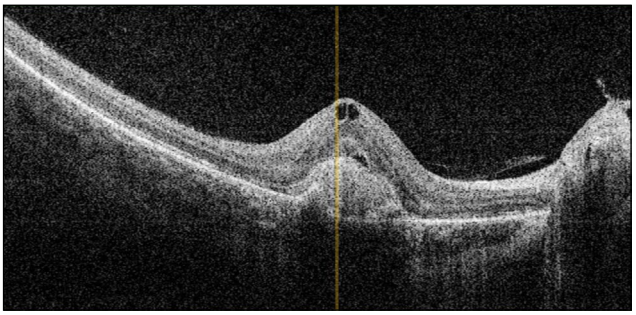

Fig. 5. Optical coherence tomography (OCT) image of choroidal neovascularization type 2 in a child with toxoplasmosis retinochoroiditis in remission.

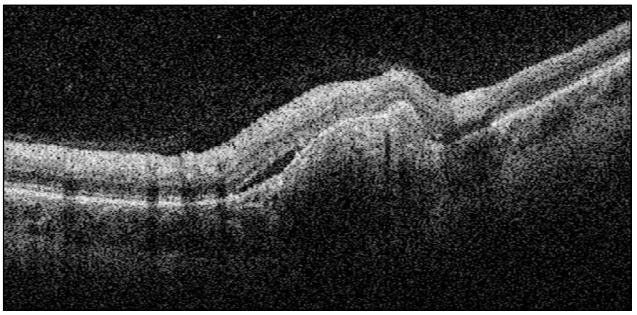

Fig. 6. Optical coherence tomography (OCT) image of choroidal neovascularization type 1 in a child with chorioretinitis of unclear etiology in remission.

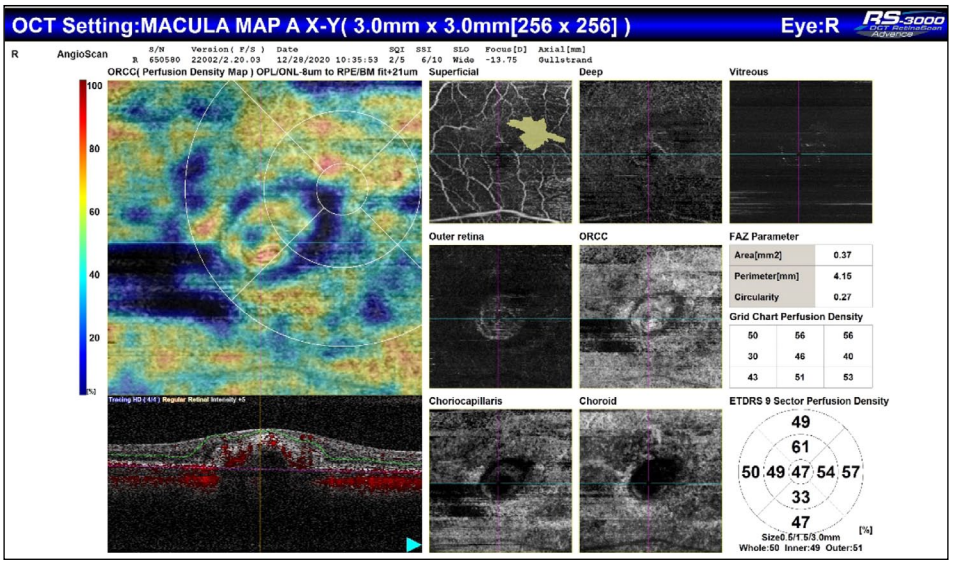

Fig. 7. Optical coherence tomography angiography of active choroidal neovascularization. At the scanning level of the capillary layer of the ocular vasculature, a dense network of anastomosing newly formed vessels is detected.

was concealed by a lipofuscin-like deposit of vitelliform cyst in the resorption stage were observed (Fig. 9).

DISCUSSION

Early diagnosis, timely treatment, and adequate monitoring of CNV are challenging because children, particularly young children, are commonly asymptomatic and pediatric ophthalmologists lack vigilance. Therefore, undiagnosed CNV may be treated as active posterior uveitis, often with prolonged and repeated courses of anti-inflammatory, antiviral, and antiparasitic therapy.

In this study, misdiagnosis was the primary cause in nearly 30% of cases. In addition, OCTA plays an important

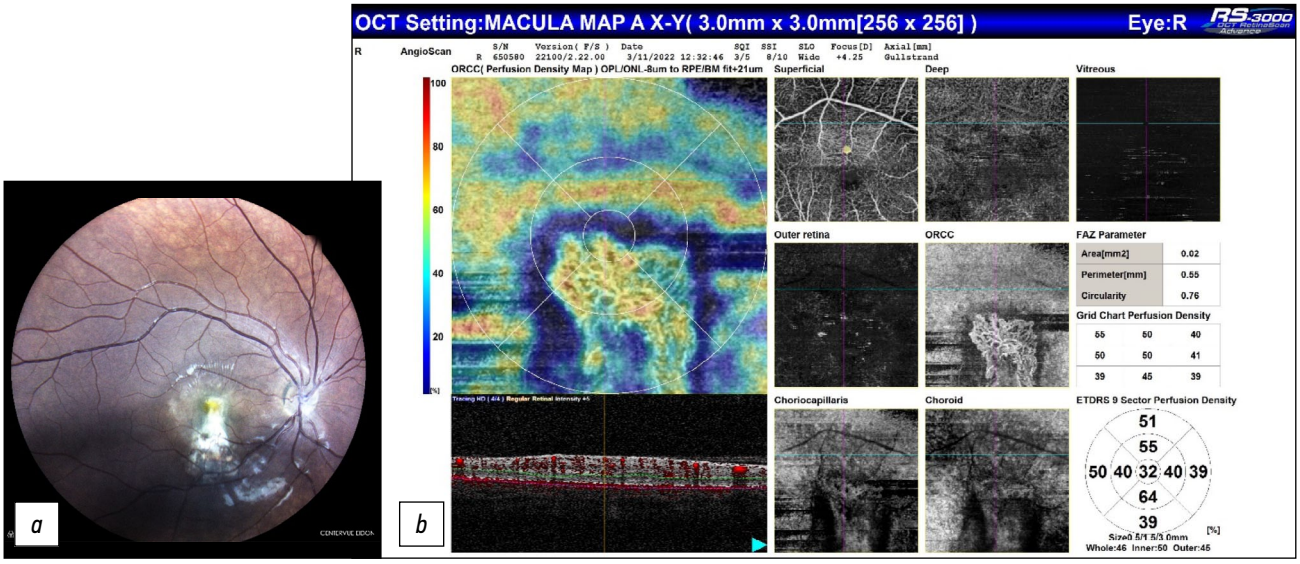

Fig. 8. Choroidal neovascularization: *a*, ophthalmoscopic picture corresponding to chorioretinitis in remission and inactive choroidal neovascularization; *b*, optical coherence tomography angiography showing active choroidal neovascularization in the chorioretinal focus.

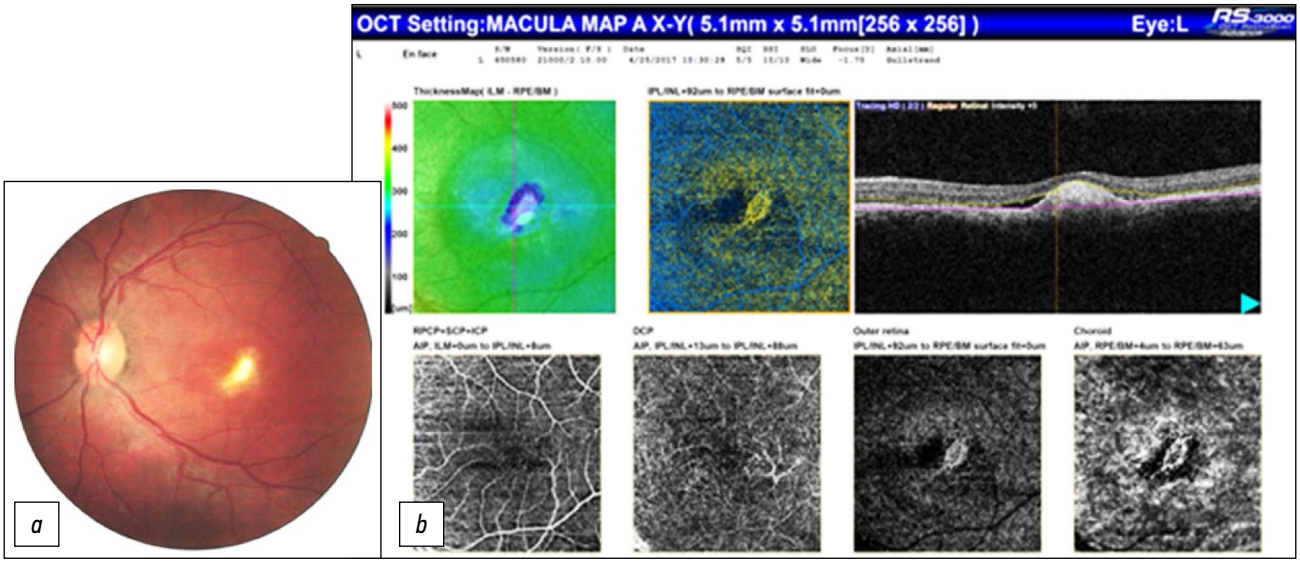

Fig. 9. Best's disease, stage of cyst resorption: *a*, ophthalmoscopic picture; *b*, optical coherence tomography angiography, active choroidal neovascularization in the projection zone of lipofuscin-like substances.

role in the diagnosis and monitoring of CNV, allowing for noninvasive visualization of membrane vessels in addition to the ophthalmoscopic picture.

In our sample, the most frequent pathologies complicated by CNV development in children were inflammatory diseases of the retina and choroid (56% of eyes), followed by Best's disease (14.8%).

The results are consistent with those of other researchers. M. Moosajee et al. found that retinal and choroidal inflammation caused CNV formation in 33.3% of cases, whereas optic disc anomalies caused CNV formation in 33.3% of cases, mainly drusen and less frequently coloboma. Rishi et al. observed postinflammatory CNV in 38% of cases and posttraumatic CNV in 16% of 111 eyes of 96 children with this complication. According to E.B. Goshorn et al., inflammatory diseases were the dominant cause of CNV in 10 of 25 children (4%) [13]. T. Zhang et al. suggested that inflammatory diseases of the retina and choroid were the leading causes of CNV formation in children (30.0%), followed by Best's disease (10%) [6]. T. Padhi et al. observed CNV in 32.5% of 43 eyes of 35 patients with Best's disease [7]. Similarly, T. Barth et al. found CNV in 3 of 9 children with choroidal osteoma [17].

Inflammatory diseases of the retina and choroid are the most common cause of CNV development in children. However, no studies have focused on postinflammatory CNV in children. According to data from the Pediatric Eye Pathology Department of the Helmholtz Eye Diseases Research Center, CNV is observed in 12% of children with posterior and panuveitis, which may be attributed to the concentration of such patients in our center. According to the literature, CNV occurs in only 2% of adult uveitis cases. However, it is much more common in certain nosologic forms, such as pitting

internal choroidopathy (76%–100%), multifocal choroiditis (33%–50%), serpiginous choroiditis (10%–25%), and Vogt–Koyanagi–Harada disease (9%–15%) [18, 19].

In 11 of 30 children (36.7%) with inflammatory lesions of the retina and choroid, CNV developed during the remission stage of the underlying disease. This event may be caused by both subclinical inflammation and chronic ischemia stimulating neoangiogenesis. In another 8 (26.7%) children of this group, inflammation at the time of complication development could not be established because of late treatment and unclear anamnestic data. In our study, the time interval between the onset of uveitis and development of CNM varied from 1 month to 12 years.

Among the noninflammatory congenital and acquired lesions of the retina and choroid, the most common disease was Best's disease, with cyst resorption and scar stages. Bilateral CNM was observed in 3 of 8 children. In our previous study, the formation of CNM in children with Best's disease was not always accompanied by clinical symptoms. Therefore, we recommend performing OCTA in all patients starting from the stage of cyst resorption to detect subclinical CNM. Dynamic follow-up should be conducted depending on the nature of the detected changes [8]. However, CNV may occur less frequently in other retinal dystrophies [4, 6, 9, 20]. The majority (78.7%) of our patients had type 2 CNM, which is more common in childhood and postinflammatory CNM [6, 7, 18].

Therefore, active management of the inflammatory process and prevention of the development of pathological neovascularization in children with posterior and panuveitis are necessary during the early disease stages. In addition, continuous monitoring even after remission is achieved is necessary because visual acuity may be reduced by the underlying disease, even in the absence of complaints. Therefore, regular follow-up examinations are mandatory. For active inflammatory processes, the monitoring scheme is determined by the course of the underlying disease. Children with inactive uveitis and risk of CNV should undergo a comprehensive ophthalmologic examination at least once every 3 months [21]. For those with congenital pathology of the retina and optic disc and a risk of CNV, examinations should be performed at least once every 6 months. Further studies are necessary to determine the risk factors and peculiarities of CNM development in childhood. This approach will enable the development of optimal management techniques for such patients.

CONCLUSIONS

1. CNM is a rare complication of various ocular diseases in children. In our cohort, an inflammatory lesion of the retina and choroid was the leading etiologic factor of CNM development.
2. Instrumental examination methods such as OCT and OCTA play extremely important roles in the diagnosis and monitoring of CNV.
3. Ophthalmologists should be aware of the risk factors and clinical and instrumental signs of CNV in pediatric patients because of its predominantly central localization and rapid, irreversible vision loss if untreated. Thus, early detection and treatment of CNV is important.

ADDITIONAL INFO

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Author contribution. Lyudmila A. Katargina and Ekaterina V. Denisova designed the study; Natalya A. Osipova and Yana A. Kiseleva processed and structured patient's history and examination data, Ekaterina V. Denisova and Natalya A. Osipova examined patients, analyzed data, wrote the manuscript with input from all authors. Thereby, all authors made a substantial contribution to the conception of the work, acquisition, analysis, interpretation of data for the work, drafting and revising the work, final approval of the version to be published and agree to be accountable for all aspects of the work.

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