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Analysis of long-term (34 years) observation of a patient with ophthalmological manifestations of Grenblad–Strandberg syndrome

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ABSTRACT

The prognosis for the life of patients with genetic pathology depends on the interaction between specialists from different areas of medicine for timely detection and selection of treatment tactics. Pseudoxanthoma elasticum (Grenblad–Strandberg syndrome) is a hereditary disease in which elastic fibers of the skin, the cardiovascular system and the retina are affected. Clinical manifestations: skin changes in Grenblad–Strandberg syndrome are represented by flat xanthomatous nodules of yellowish color. Cardiovascular manifestations of pseudoxanthoma elasticum are angina pectoris, decreased pulse amplitude, cardiomyopathy, sudden heart failure, often leading to death. Eye disorders occur in stages. For the early stages, the appearance of angioid streaks is typical, which appear as a result of calcification of elastic fibers of capillaries. The progression of the process leads to neovascularization and hemorrhages from the choriocapillaries, the formation of a subretinal neovascular membrane of foveolar localization, causing a decrease in vision. The late stages are characterized by scarring. Therapy depends on the stage and rate of progression of the disease and is effective at stages I–II (according to Vivaldi). Own clinical observation: Male patient, 71 years old, referred for cataract surgery with the diagnosis “Both eyes: Senile cataract, open-angle glaucoma, (stage I a, under beta-blocker therapy), Grenblad–Strandberg syndrome”. Attention is drawn to the long observation period — 34 years, with documented data from the first examinations in 1989 and all subsequent ones. Of particular interest is the availability of preserved patient documentation for all years of follow-up, including diagnosis and treatment.

Keywords: Grenblad–Strandberg syndrome; skin papules; foveolar region; pseudoxanthoma elastic; angioid streaks; xanthomatous nodules; subretinal neovascular membrane; pigment epithelium; Bruch’s membrane; anti-VEGF therapy.

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Анализ многолетнего (34 года) наблюдения за пациентом с офтальмологическими проявлениями синдрома Гренблада – Страндберга

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АННОТАЦИЯ

Прогноз жизни пациентов с генетической патологией зависит от взаимодействия специалистов из разных областей медицины для своевременного выявления и выбора тактики лечения. Псевдоксантома эластическая (синдром Гренблада – Страндберга) — наследственное заболевание, при котором поражаются эластические волокна кожи, кардиоваскулярная система и сетчатка глаз. Клинические проявления: изменения кожи при синдроме Гренблада – Страндберга представлены плоскими ксантоматозными узелками желтоватого цвета. Сердечно-сосудистыми проявлениями псевдоксантомы эластической являются стенокардия, снижение пульсовой амплитуды, кардиомиопатия, внезапная сердечная недостаточность, часто приводящая к смерти. Со стороны органа зрения: для ранних стадий типично появление ангиоидных полос как результат кальцификации эластических волокон капилляров. Прогрессирование процесса приводит к неоваскуляризации и кровоизлияниям из хориокапилляров, формированию субретинальной неоваскулярной мембраны фовеолярной локализации, вызывающей снижение зрения. Для поздних стадий характерны рубцовые изменения. Терапия зависит от стадии и скорости прогрессирования заболевания и эффективна на I–II стадиях (по Vivaldi). Собственное клиническое наблюдение: Пациент, 71 год, направлен на оперативное лечение катаракты с диагнозом «OU: Сенильная катаракта, О/У IA п/ß-блокаторами глаукома, синдром Гренблада – Страндберга». Обращает на себя внимание уникально длительный период наблюдения — 34 года, с документально подтвержденными данными первых обследований от 1989 г. и всех последующих. Особый интерес вызывает наличие сохранившейся документации на руках у пациента за все годы наблюдения, включающие диагностику и лечение.

Ключевые слова: синдром Гренблада – Страндберга; кожные папулы; фовеолярная область; псевдоксантома эластическая; ангиоидные полосы; ксантоматозные узелки; субретинальная неоваскулярная мембрана; пигменный эпителий; мембрана Бруха; анти-VEGF-терапия.

Как цитировать

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BACKGROUND

Survival prognosis and therapy efficacy in patients with genetic disorders directly depend on collaboration of specialists in different areas for timely diagnosis and selection of adequate treatment strategy.

Pseudoxanthoma elasticum (PXE, Grönblad–Strandberg syndrome) is an inherited disorder that affects the elastic fibers of the skin, cardiovascular system, and retina. The prevalence of PXE is 1 case per 25,000 to 100,000, and the condition is inherited in an autosomal recessive pattern. The disease is caused by mutations in the *ABCC6* gene encoding the MRP6 membrane transporter on chromosome 16p13.1 [1–6]. The ATP-dependent transporter MRP6 is responsible for the active release of glutathione-conjugated low-molecular-weight metabolites from cells, which leads to calcification of the elastic fibers. Because of calcification of the elastic fibers of blood vessels, patients with PXE should avoid injuries (especially head and/or eyes injuries) and limit the use of non-steroidal anti-inflammatory drugs and anticoagulants (due to the increased risk of hemorrhages to the retina, brain, and gastrointestinal tract). Although skin manifestations occur in the vast majority of cases, the outcomes depend on ocular and cardiovascular system changes. Ocular changes associated with this syndrome are characterized by angioid streaks, which represent the breaks in Bruch's membrane caused by destruction of its elastic layer. Central retinal disorders are a significant risk factor for spatial vision loss and disability of a patient of working age [6, 7]. If angioid retinal streaks are detected, a multidisciplinary approach is required, because similar ophthalmoscopic pattern can be observed in Ehlers–Danlos and Marfan syndromes, sickle cell anemia, Paget disease, etc. Timely diagnosis and pathogenesis-directed therapy of these patients increase the chances of long-term preservation of visual function.

HISTORICAL FACTS

French dermatologist D. Rigal first described skin changes characteristic of pseudoxanthoma elasticum in 1881. Later, in 1884, F. Balzer performed histological examinations of the changed skin. However, the term *elastic pseudoxanthoma* was introduced by another researcher Ferdinand-Jean Darier, who managed to differentiate this condition from simple cutaneous xanthoma (yellow papule) [8]. Ophthalmologists Doyne (1889) and Pflange (1892) first reported ocular manifestations in the form of posterior pole hemorrhages leading to dark brown or black pigmented lines and streaks; later US researcher Hermann Knapp proposed the term *angioid streaks* [9]. In 1929, ophthalmologist Esther Grönblad and dermatologist James Strandberg from Sweden first identified a relationship between angioid streaks and PXE, and the condition was named Grönblad–Strandberg syndrome. The term "pseudoxanthoma elasticum" is currently used as a synonym [10, 11].

PATHOMORPHOLOGY AND PATHOGENESIS

Histopathological examinations of eye tissues with angioid streaks, in addition to linear tears with or without fibrovascular tissue ingrowth, showed extensive calcification and thickening of Bruch's membrane with signs of thickening of capillary bridges [12]. The fibrous tissue growth from the choroid causes discoloration, expansion, and blurring of the striae margins. Fibrovascular tissue ingrowth may lead to serous and/or hemorrhagic detachment of the retinal pigment epithelium [13]. No visible changes in the neurosensory retina are observed at early stages. This is followed by atrophy of the choriocapillary, retinal pigment epithelium, and photoreceptors and a decrease in pigment granules in cells near the striae.

Scanning electron microscopy and histochemical examinations of the eye tissue of a patient with homozygous sickle cell anemia and angioid streaks showed severe calcification and breaks in Bruch's membrane. A group of authors proved that calcification, rather than iron deposition, is the main factor leading to Bruch's membrane fragility in patients with hemolytic anemia and angioid streaks [14].

Baccarani-Contri et al. reported calcification and following fragmentation of the elastic fibers. Immunoelectron microscopy proved increased biosynthesis of proteins, such as vitronectin, as well as accumulation of abnormal matrix proteins with high affinity for calcium and calcifying compounds [15]. Quaglino et al. [16] demonstrated the role of fibroblasts in the PXE pathogenesis. A change in glycosaminoglycan metabolism was detected (increased activity of xylosyltransferase I in serum samples from patients with PXE). Xylosyltransferase I is a key enzyme in glycosaminoglycan biosynthesis and has been identified as a serum marker of increased proteoglycan biosynthesis. Considering the above, PXE seems to be a metabolic disorder caused by a decrease or lack of production of the *ABCC6* gene encoding this enzyme, but this suggestion has yet to be proven [17, 18].

Hereditary hemoglobinopathies, such as beta-thalassemia, are known to cause similar ultrastructural changes in the skin, eyes, and cardiovascular system without *ABCC6* mutations [19]. This suggests alternative pathological mechanisms, such as increased chronic oxidative stress, which has also been proven in a study of fibroblasts of patients with PXE [20].

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GENETIC RESEARCH

Direct sequencing found mutations causing PXE only in the *ABCC6* gene, an active transmembrane transporter. The gene contains 31 exons spanning a 75 Kb genomic region, 4.5 Kb of which are encoded by multi-drug resistance-associated protein 6 (MPR6) expressed in the liver and kidneys. However, the tissues primarily affected by PXE (skin, vascular walls, Bruch's membrane) express low concentrations of MRP6 [21–23]. Several authors consider PXE as a systemic metabolic disease with molecular changes associated with abnormal synthesis of the extracellular matrix and defective MRP6 protein transporter [18, 24]. Today, over 110 different mutations of the *ABCC6* gene, which are suggested to cause PXE, have been identified almost in all 31 exons. They include missense and nonsense mutations with fewer splicing mutations, which can be identified in 55%–83% of patients with PXE [25–27]. Failure to identify the mutation can be explained by exon deletion and mutations located further from the coding sequence or other systemic disorders simulating PXE (e.g., sickle cell anemia) should be excluded [28].

CLINICAL MANIFESTATIONS

Skin changes in Grönblad–Strandberg syndrome include flat round or oval yellowish xanthomatous nodules which are arranged linearly or merge into single or diffuse plaques. Early skin manifestations look like papules with a distinct yellowish tint, the size of a millet grain, and a tendency to merge. They have a mottled appearance and are most often localized on the neck and large skin folds (armpits, elbows), less often on other body parts. The affected skin is lemon yellow, somewhat thickened and flabby at the same time, and easily gathers into folds. Skin sagging progresses and causes premature aging. Sometimes papules are found in the inguinal folds, popliteal areas, on the oral ("geographic tongue"), vagina, and rectum mucosa [29]. Cardiovascular manifestations of PXE include angina pectoris, decreased pulse amplitude, arterial hypertension, cardiomyopathy, mitral valve prolapse or stenosis, and sudden cardiac failure often leading to death [30–32]. Ocular changes occur gradually. Angioid streaks are typical for early stages and caused by calcification of the capillary elastic fibers. Progression leads to neovascularization, hemorrhages from the choriocapillaris, and formation of a subretinal neovascular membrane (SNVM) in the fovea area, which decreases visual acuity. Later stages are characterized by severe scarring. The first fundus changes are observed on ophthalmoscopy and include an uneven pigment distribution called *peau d'orange* (orange peel appearance). These changes are most noticeable at the temporal fovea, do not affect visual function, precede angioid streaks, and occur on average within 1–8 years. Angioid streaks are 50–100 μm in diameter, several times larger than retinal vessels, dark or reddish radial lines radiating out from the optic nerve [2, 33, 34]. Patients are asymptomatic until neovascularization develops, which leads to maculopathy with a significant decrease in visual function.

Vivaldi identified 3 stages of fundus changes:

Stage I: grayish or reddish-brown angioid streaks appear in the central retina and its middle periphery, radiate out from the optic nerve, and represent linear breaks in Bruch's membrane;

Stage II: SNVM is formed in the parafoveal area and accompanied by a decrease in visual acuity;

Stage III is characterized by subretinal scarring of the macula, with the development of scotoma and irreversible vision loss. The changes are bilateral, chronic, and progressive [2–4, 35].

Secondary degenerative and hemorrhagic macular changes are found in 73%–86% of cases and often lead to a sharp decrease in visual acuity in adolescents. Even minor injuries can cause diffuse subretinal hemorrhages, especially around angioid streaks. By the age of 50, most patients with PXE have a bilateral decrease in vision to 0.1 or lower [36–41].

TREATMENT

Therapy depends on the stage and rate of disease progression and is effective at stages I–II (according to Vivaldi).

Transpupillary thermotherapy

Transpupillary thermotherapy consists in using a diode laser with a lower threshold to avoid producing a thermal burn and is used to treat macular choroidal neovascularization (CNV) of any origin. A laser beam with an 810 nm wavelength penetrates the transparent eye media well and has a dosed thermal effect due to lower absorption by the retinal pigment epithelium and deeper penetration into the choriocapillaris.

Photodynamic therapy

An analysis of large randomized clinical trials and clinical case reports evaluating the effectiveness of photodynamic therapy (PDT) for CNV secondary to age-related macular degeneration and pathologic myopia showed that the results did not meet expectations. Several studies consider PDT as an adjuvant therapy that does not prevent, but only slows down the natural course of CNV. In some cases, PDT was used in combination with anti-VEGF therapy for the treatment of CNV to enhance the effect. Unfortunately, most of the above treatment methods cannot stop progressive loss of visual function in patients with CNV secondary to angioid streaks.

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Anti-VEGF therapy is the future and shows the most encouraging results [13, 40–47]. When SNVM develops, combined therapy, including intravitreal injections of VEGF inhibitors and focal laser photocoagulation of SNVM, is considered the most appropriate. Currently, treatment for stage III disease is not effective.

Laser photocoagulation

Clarkson et al. [13] reported the treatment results for 6 patients with angioid streaks and macular CNV. They received argon laser photocoagulation targeted directly at the neovascular membrane without any positive effect. Further expansion of the neovascularization area and loss of central vision were reported in all patients. In 1988, Gelisken et al. [42] provided the results from a study of 30 eyes with CNV secondary to angioid streaks. All patients were treated with blue-green argon or krypton laser, and the follow-up period ranged from 2 months to 16 years. The authors concluded that laser treatment was effective in cases of extrafoveolar (200 μm or more from the foveal center) neovascular membranes.

Macular translocation

The method was introduced by R. Machemer and U.H. Steinhorst in 1993. It involves repositioning of the neurosensory retina (macula and various areas of the adjacent retina) further away from the neovascular lesion. Surgery is performed by limited translocation including local retinal detachment and scleral shortening or a 360° retinotomy with rotation of the entire retina. This is a complicated and time-consuming surgical procedure associated with the risk of serious complications (retinal detachment, proliferative vitreoretinopathy, endophthalmitis, etc.) threatening central and peripheral vision [48]. In 2001, Roth et al. described a case of successful inferior macular translocation followed by laser photocoagulation of the CNV area in a patient with angioid streaks [49]. Notably, the encouraging results of these procedures are not statistically significant because of the small number of studies and patients [50–55].

Anti-VEGF therapy

Currently, anti-VEGF therapy is promising, significantly superior to all other CNV treatment methods, and allows stabilizing visual acuity in most patients, especially if early treatment is initiated.

Transplantation of an autologous retinal pigment epithelium flap

The technique involves removal of a neovascular membrane and subretinal fibrosis to further restore the integrity of the retinal pigment epithelium.

MEDICAL GENETIC COUNSELING

All family members should be examined, and the patient should be followed-up by a general practitioner and ophthalmologist. First stage ocular manifestations require constant monitoring and compliance with recommendations to avoid even minimal eye injury and wear protective glasses while working or exercising.

CLINICAL CASE REPORT

Examination and clinical follow-up data of a 71-year-old patient are presented below. The patient was diagnosed with OU age-related cataract, stage IA open-angle glaucoma controlled with beta-blockers, and Grönblad–Strandberg syndrome and referred for cataract surgery to the eye hospital of the I.I. Mechnikov North-Western State Medical University (St. Petersburg). Interestingly, the follow-up period was uniquely long-34 years – with documented data of the first examinations in 1989 and all subsequent test. The available documentation for all years of observation, including diagnosis and treatment, is of particular interest. Medical history states that the patient had complained of decreased visual acuity in both eyes since April 19, 1989 when he was 37 years old (Fig. 1). Primary examination revealed OU central scotoma and retinal degeneration with angioid streaks. Corrected visual acuity: OD = 0.2, sph –1.5D = 0.7; OS = 0.7, sph –0.75D = 1.0. Based on the case conference conclusions (Diagnostic Center No. 7 and Saint-Petersburg Phthisiopulmonology State Research Institute), as well as clinical and instrumental tests, Grönblad–Strandberg syndrome was diagnosed. In 1991, argon laser coagulation of the central lesion in the left eye was performed at the Saint Petersburg Phthisiopulmonology State Research Institute. OU fundus autofluorescence (AF) was performed several times, hypo-autofluorescence in the peripapillary dystrophy area was observed in the optic disk image obtained in 2009. There were an extensive focus of hypo-autofluorescence in the macula and hyper-autofluorescent paramacular paracentral ring. Follow-up examinations (2010–2011) showed no changes. In 2013, AF found an extended atrophy area (Fig. 2).

In 2007, electrophysiology testing (EPT) revealed 3rd order neuron conduction abnormality. Visual evoked potential (VEP) test of OU found that the activity of the 3rd order neuron was reduced, and latency was moderately increased. This finding represented abnormal conduction of visual stimulation along the visual pathways. VEP test showed that OU positive evoked potentials represented IOP regulation abnormality. Glaucoma was diagnosed in both eyes. In 2008, repeated EPT and VEP test demonstrated OU negative evoked potentials and glaucoma stabilization. In 2009, macular examination (multi-

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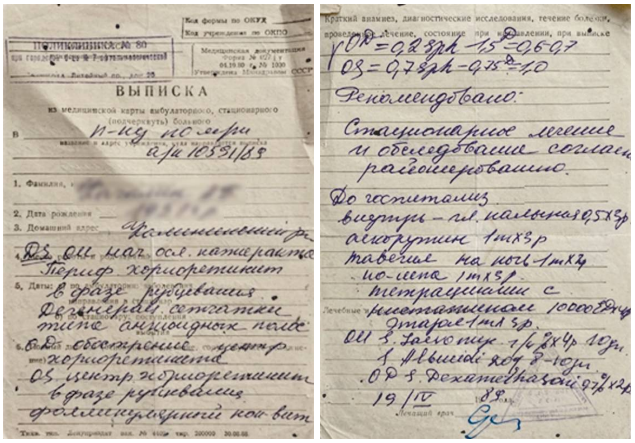


Fig. 1. Ophthalmological examination from 04/19/1989. Both eyes — initial complicated cataract, peripheral chorioretinitis in the scarring phase, retinal degeneration of the "angioid streaks" type
Рис. 1. Осмотр офтальмолога от 19.04.1989. OU — начальная осложненная катаракта, периферический хориоретинит в фазе рубцевания, дегенерация сетчатки типа «ангиоидных полос»

focal electroretinography, mfERG) confirmed damage to the 1st and 2nd order neurons. Macula mfERG revealed signs of abnormal visual conduction in the central retina indicating decreased activity of the outer photoreceptor layers (1st order neuron) and bipolar cells (2nd order neuron) in both eyes. A standard ophthalmic examination during the last hospital stay at the eye hospital of the I.I. Mechnikov North-Western State Medical University in May 2023 provided the following data: OD0.1 non-correctable; OS = 0.04, sph –1.0D cyl –1.0D Ax 65° = 0.1. Bilateral IOP per Maklakov method was 20 mmHg. Kinetic perimetry found bilateral irregular concentric visual field constriction and central scotoma within 15–30° of the fixation point (Fig. 3).

OU biomicroscopy showed normal anterior segment and early-stage age-related cataract. Macular ophthalmoscopy revealed subretinal fibrosis with extensive retinal pigment epithelium atrophy (Fig. 4). Optical coherence tomography indicated increasing disc cupping with average C/D ratios of 0.81 mm and 0.80 mm in the right and left eyes, respectively (Fig. 5). The macular surface was irregular with severe atrophy, destruction of the retinal pigment epithelium–Bruch's membrane complex, and subretinal fibrosis with extensive retinal pigment epithelium atrophy (Fig. 6).

The patient was additionally consulted by a dermatologist-venereologist at the Eye hospital of the I.I. Mechnikov North-Western State Medical University.

Examination on April 24, 2023 revealed a symmetrical pattern and single small oval papules of normal color in the supraclavicular area. Dermoscopy showed no signs of melanocytic tumor. The skin color was yellowish-beige

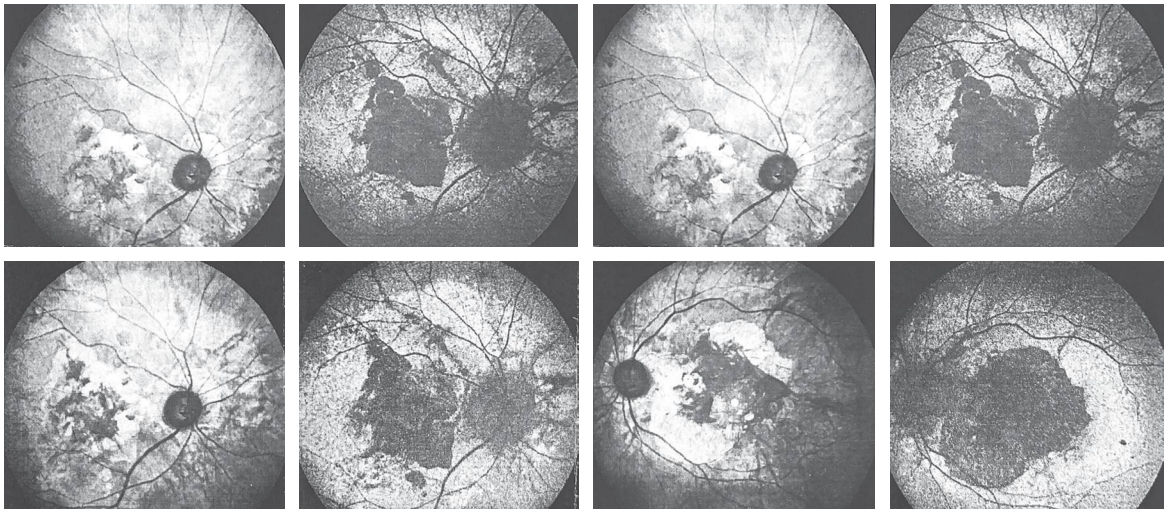


Fig. 2. Images in the IR and AutoFluo modes on the Heidelberg Engineering device. There is an expansion of the area of atrophy. The top row is from 2009, the bottom row is from 2013
Рис. 2. Снимки в режимах IR и AutoFluo на приборе Heidelberg Engineering. Наблюдается расширение площади атрофии. Верхний ряд — 2009 г., нижний ряд — 2013 г.

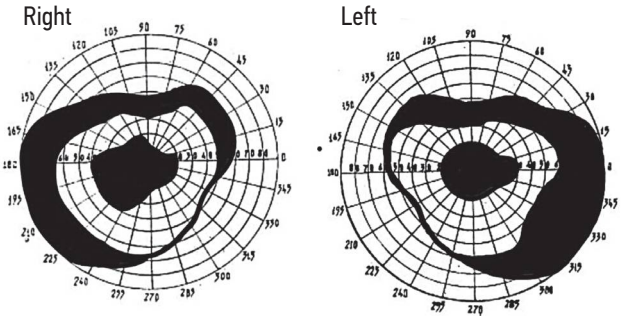


Fig. 3. Kinetic perimetry data, 2023
Рис. 3. Данные кинетической периметрии, 2023 г.

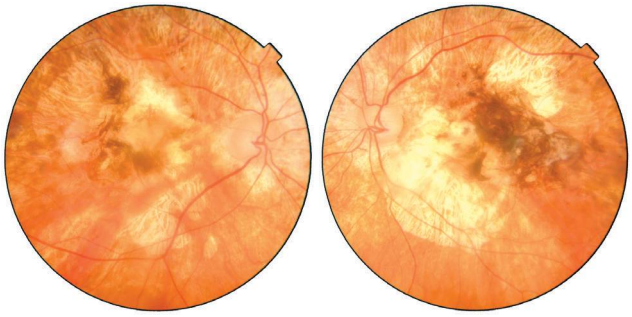


Fig. 4. Color Fundus Photo the right and left eyes, respectively. 2023 Obtained using TOPCON 3D OCT-1 Maestro2
Рис. 4. Цветная фотография глазного дна правого и левого глаза. 2023 г. Выполнено на TOPCON 3D OCT-1 Maestro2

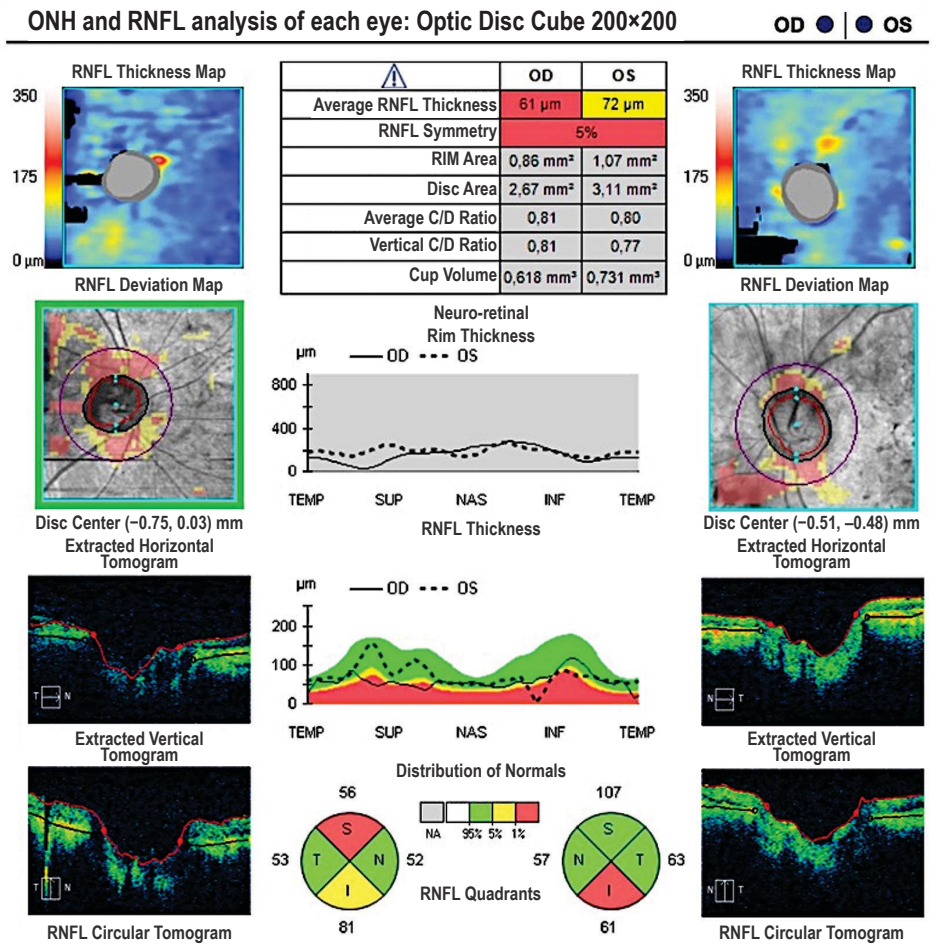
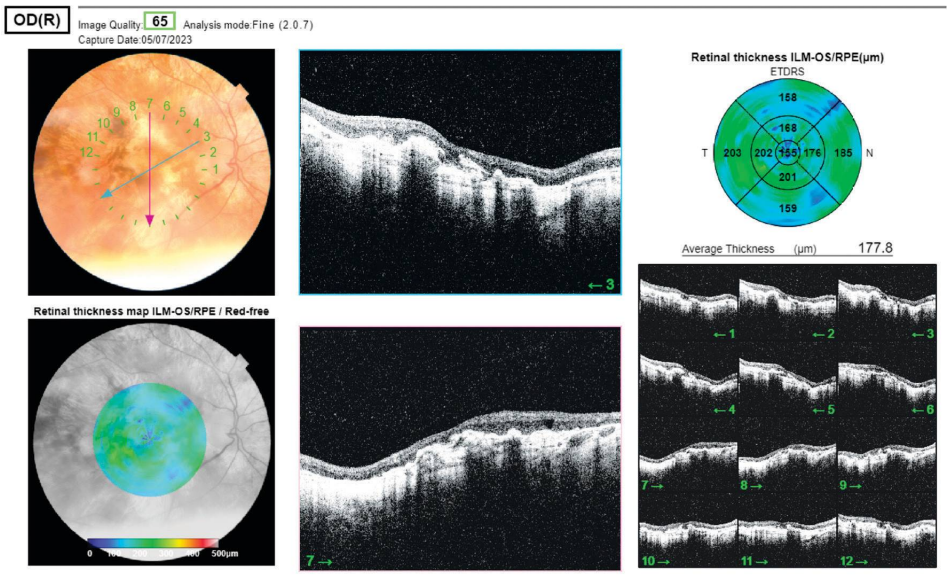


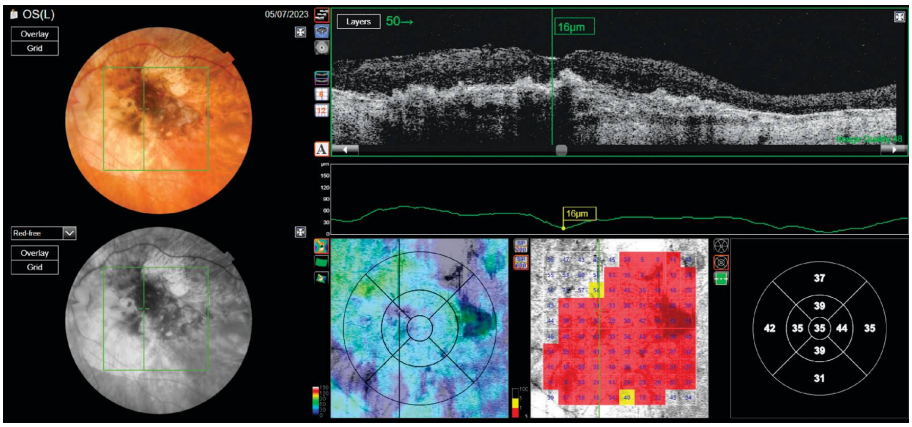
Fig. 5. ONH and RNFL analysis of each eye, Optic Disc Cube 200×200. Extension of the Optic Nerve Head cup of the right eye: average C/D 0.81; left eye: average C/D 0.80. Obtained using ZEISS CIRRUS 5000
Рис. 5. Анализ ONH и RNFL каждого глаза, Optic Disc Cube 200×200. Расширение экскавации диска зрительного нерва на правом глазу: среднее отношение C/D 0,81; на левом: среднее отношение C/D 0,80. Выполнено на ZEISS CIRRUS 5000

and homogeneous. Similar dermoscopy pattern is characteristic of pseudoxanthoma elasticum (Fig. 7).

Bilateral cataract phacoemulsification with intraocular lens implantation 1.5 months apart was performed at the eye hospital of the I.I. Mechnikov North-Western State Medical University. Postoperative visometry and refractometry revealed no changes in visual acuity (OU = 0.1, uncorrectable), the patient reported a subjective significant improvement in vision acuity, which was not enough for self-service. At this stage, all possible conventional treatment methods to preserve or improve the patient's vision acuity were used. The patient reported that they were not enough for self-service, because recovering the transparency of optical media neither improved visual



a



b

Fig. 6. The destroyed complex "pigment epithelium–Bruch's membrane", subretinal fibrosis with extensive atrophy of the Retinal Pigment Epithelium: *a* — right eye; *b* — left eye. Obtained using TOPCON 3D OCT-1 Maestro 2

Рис. 6. Разрушенный комплекс «пигментный эпителий – мембрана Бруха», субретинальный фиброз с обширной атрофией пигментного эпителия сетчатки: *a* — правый глаз; *b* — левый глаз. Выполнено на TOPCON 3D OCT-1 Maestro2

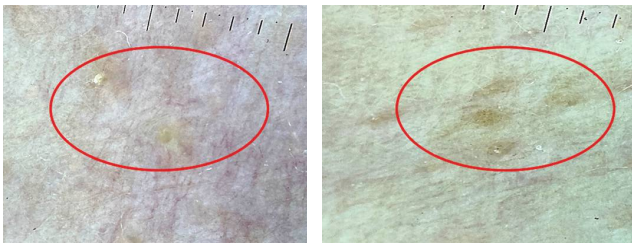


Fig. 7. Photo of the patient's dermatoscopy, 04/24/2023. Check-up by a dermatovenerologist at the Medical and Preventive Center of the North-Western State Medical University named after I.I. Mechnikov, PhD (Med), Assistant Professor A.A. Vashkevich. Isolated small papules of yellowish-beige color and of normal skin color are outlined

Рис. 7. Фото дерматоскопии пациента от 24.04.2023. Осмотр врача-дерматовенеролога Медико-профилактического центра СЗГМУ им. И.И. Мечникова — канд. мед. наук, доцента А.А. Вашкевич. Обведены единичные мелкие папулы желтовато-бежевого окрашивания и цвета нормальной кожи

function nor resulted in central fixation. Currently, transplantation of an autologous retinal pigment epithelium flap is planned.

CONCLUSION

A gradual decrease in visual acuity progressing to irreversible blindness is a common outcome of PXE, especially in patients over 50 years of age. Choroidal neovascularization and following disciform scar with subretinal fibrosis and atrophy lead to loss of spatial vision. The frequency of visual impairments caused by the disease is comparable to that observed in age-related macular degeneration in elderly patients, but they manifest earlier and significantly reduce the quality of life in patients of working age. Patients diagnosed with angioid streaks on ophthalmoscopic examination need regular monitoring to prevent irreversible macular complications.

Moreover, a multidisciplinary approach is extremely important in these patients, as differential diagnosis is required to detect systemic conditions (including life-threatening) in collaboration with specialists from related disciplines.

ADDITIONAL INFORMATION

Authors' contribution. All authors made a substantial contribution to the conception of the study, acquisition, analysis, interpretation of data for the work, drafting and revising the article, final approval of the version to be published and agree to be accountable for all aspects of the study. Personal contribution of each author: A.A. Sharma — writing the text, collecting material and reviewing literature, diagnostic research; N.G. Zumbulidze — writing and editing the text; E.V. Boyko — editing the text; A.V. Kononov — writing the text, diagnostic research

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Competing interests. The authors declare that they have no competing interests.

Consent for publication. Written consent was obtained from the patients for publication of relevant medical information within the manuscript.

ДОПОЛНИТЕЛЬНАЯ ИНФОРМАЦИЯ

Вклад авторов. Все авторы внесли существенный вклад в разработку концепции, проведение исследования и подготовку статьи, прочли и одобрили финальную версию перед публикацией. Личный вклад каждого автора: А.А. Шарма — написание текста, сбор материала и обзор литературы, диагностические исследования; Н.Г. Зумбулидзе — написание и редактирование текста; Э.В. Бойко — редактирование текста; А.В. Кононов — написание текста, диагностические исследования.

Источник финансирования. Авторы заявляют об отсутствии внешнего финансирования при проведении исследования.

Конфликт интересов. Авторы декларируют отсутствие явных и потенциальных конфликтов интересов, связанных с публикацией настоящей статьи.

Информированное согласие на публикацию. Авторы получили письменное согласие пациента на публикацию медицинских данных.

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