ОФТАЛЬМОЛОГИЯ
OPHTALMOLOGY

CORNEAL EDEMA CAUSED BY UNDIAGNOSED IRIDOCORNEAL ENDOTHELIAL SYNDROME AFTER SMILE

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ABSTRACT

The aim. *To present a clinical case of a patient with corneal edema caused by undiagnosed iridocorneal endothelial syndrome after SMILE surgery.*
Iridocorneal endothelial syndrome is characterized by abnormal proliferation of the corneal endothelium beyond the Schwalbe line, blockade of the iridocorneal angle, specific changes in the shape of the pupil and the formation of synechiae between adjacent structures of the anterior chamber angle of the eye, which is accompanied by the development of pretrabecular retention of intraocular aqueous humour, increased intraocular pressure (IOP) and subsequent development of glaucoma in 46–82 % of patients. One of the forms of the disease is Chandler's syndrome, which clinically in the initial stages may be accompanied by the formation of only moderate ectropion. At the same time, endothelial dysfunction is characterized by a significant decrease in the number of cells, a change in their shape and the appearance of epithelioid cells with a hyper-reflective nucleus (the so-called "ICE (iridocorneal endothelial) cells"), with a violation of their natural pumping function. The article presents a clinical case of a patient who underwent a standard examination and symptomatic treatment after laser keratorefractive surgery for myopia correction. Low visual acuity, diffuse corneal edema, IOP decompensation of unclear genesis were observed in one operated eye.
Conclusion. *Keratorefractive operations can act as a trigger that stimulates the transition of the latent form into a clinically developed pathological process, which causes a decrease in corrected visual acuity due to the formation of corneal edema and glaucoma optic neuropathy.*

Key words: *iridocorneal endothelial syndrome, keratorefractive surgery, corneal edema, postoperative complications*

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ОТЁК РОГОВИЦЫ, ВЫЗВАННЫЙ НЕДИАГНОСТИРОВАННЫМ ИРИДОКОРНЕАЛЬНЫМ ЭНДОТЕЛИАЛЬНЫМ СИНДРОМОМ, ПОСЛЕ SMILE

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РЕЗЮМЕ

Цель работы. *Представить клинический случай отёка роговицы, вызванного недиагностированным иридокорнеальным эндотелиальным синдромом, у пациента после SMILE.*

Иридокорнеальный эндотелиальный синдром характеризуется аномальной пролиферацией эндотелия роговицы за пределы линии Швальбе, блокадой иридокорнеального угла, специфическими изменениями формы зрачка и образованием синехий между соседними структурами угла передней камеры глаза, что сопровождается развитием претрабекулярной задержки внутриглазной водянистой влаги, повышением внутриглазного давления (ВГД) и последующим развитием глаукомы у 46–82 % пациентов. Одной из форм заболевания является синдром Чандлера, который клинически на начальных стадиях может сопровождаться формированием лишь умеренного эктропиона. В то же время эндотелиальная дисфункция характеризуется значительным уменьшением количества клеток, изменением их формы и появлением эпителиоидных клеток с гиперрефлексивным ядром (так называемые «клетки ICE» (iridocorneal endothelial)) с нарушением их естественной насосной функции.

В статье представлен клинический случай пациента, прошедшего стандартное обследование и симптоматическое лечение после лазерной кераторефракционной операции по коррекции близорукости. На одном прооперированном глазу наблюдались низкая острота зрения, диффузный отёк роговицы, декомпенсация ВГД неясного генеза.

Вывод. *Кераторефракционные операции могут выступать в качестве триггера, стимулирующего переход скрытой формы в клинически развившийся патологический процесс, который вызывает снижение скорректированной остроты зрения из-за формирования отёка роговицы и глаукомной оптической нейропатии.*

Ключевые слова: *иридокорнеальный эндотелиальный синдром, кераторефракционная хирургия, отёк роговицы, послеоперационные осложнения*

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Refractive surgery is a popular direction in ophthalmology, and its main task is to provide improved quality of life, safety, and high visual acuity. The results of keratorefraction surgeries are usually evaluated according to the following criteria: efficiency, predictability, stability and safety, determined by the resulting visual acuity and the presence of complications. Complications associated with surgery techniques, or with the peculiarities of the early and late postoperative period, become the main cause of patients' disappointment. The most common problems are the development of a dry eye syndrome, inadequate postoperative wound healing with fibroplasia, epithelial ingrowth, insufficient correction of ametropia, various valve adaptation disorders, etc. [1, 2]. The competent intraoperative tactics or adequate postoperative therapy can help to avoid many of the complications mentioned above.

However, in clinical practice there are situations caused by violations of the patients' preoperative examination algorithm. Thus, undiagnosed during the fundus ophthalmoscopy holes and peripheral degeneration of retinal tissue can lead to the development of retinal detachment. Lack of gonioscopy in the examination may lead to untimely detection of pathological changes in the outflow pathways of aqueous humour, the formation of ophthalmic hypertension and glaucoma in patients with congenital and acquired syndromes and diseases accompanied by disorders of the eye hydrodynamics. At the same time, keratorefractive operations may act as a trigger stimulating the transition of the disease from a latent form to a clinically developed pathological process.

The aim of this work is to present a clinical case of the corneal edema formation after SMILE (Small Incision Lenticule Extraction) myopia correction in a patient with an undiagnosed iridocorneal endothelial syndrome.

CLINICAL CASE

Patient B., 35 years old, applied to an ophthalmology clinic for myopia correction. Preoperative diagnostics revealed myopic refraction, SE (spherical equivalence) –4.5 D on both eyes, BCVA (best corrected visual acuity) – 1.0, IOP (intraocular pressure) – 14/14 mmHg. No pathological changes during biomicroscopy and ophthalmoscopy. The patient underwent SMILE surgery on both eyes.

One day after surgery UCVA (uncorrected visual acuity) of the right eye – 1.2, of the left eye – 0.1, the refraction of both eyes was SE = +0.5 D, IOP = 18 mmHg. Insufficient visual acuity of the left eye was explained by the presence of diffuse corneal epithelial edema.

Optical coherence tomography (OCT) of the cornea of the left eye revealed insufficient adhesion of the walls of the intrastromal space that caused the necessity to wash it with a BSS solution and perform a tupfer massage. Positive dynamics has not been achieved.

The patient was consulted in the Irkutsk Branch of the S. Fyodorov Eye Microsurgery Federal State Institution. During biomicroscopy, a local inversion of the pigment border (vascular ectropion) and segmental compaction of the iris stroma were determined at 6 o'clock position (Fig. 1), which could demonstrate the presence of iridocorneal endothelial (ICE) syndrome.

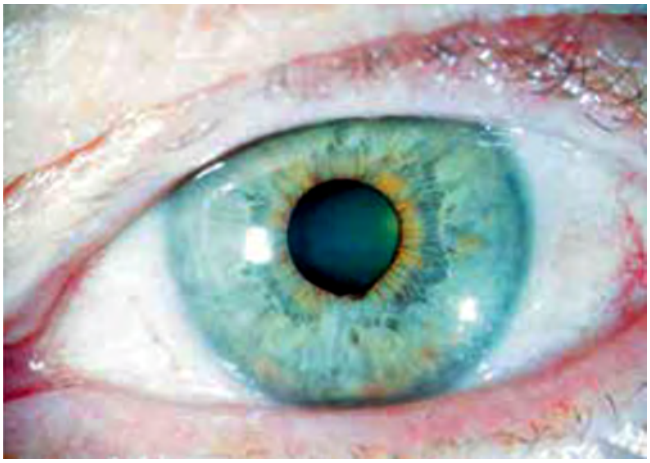


FIG. 1.
Photorecording of the anterior segment of the left eye: vascular ectropion, segmental destruction of the iris stroma at 6 o'clock position

Gonioscopy revealed a high planar goniosynechia in the projection of the inversion of the pigment border, occupying 1 quadrant of the angle of the anterior chamber, confirmed by ultrasound biomicroscopy data.

OCT revealed significant differences in the postoperative corneal intrastromal interface: a linear stroma seal corresponding to the removed lenticule was determined in the right eye in the cornea, additional microcystic spaces, corresponding to the corneal hydration zones in the left eye (Fig. 2).

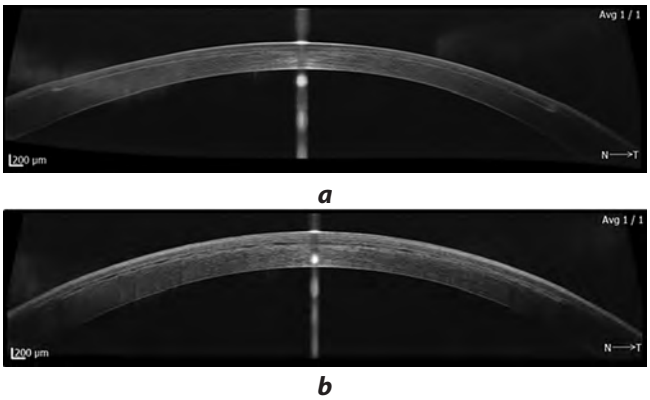
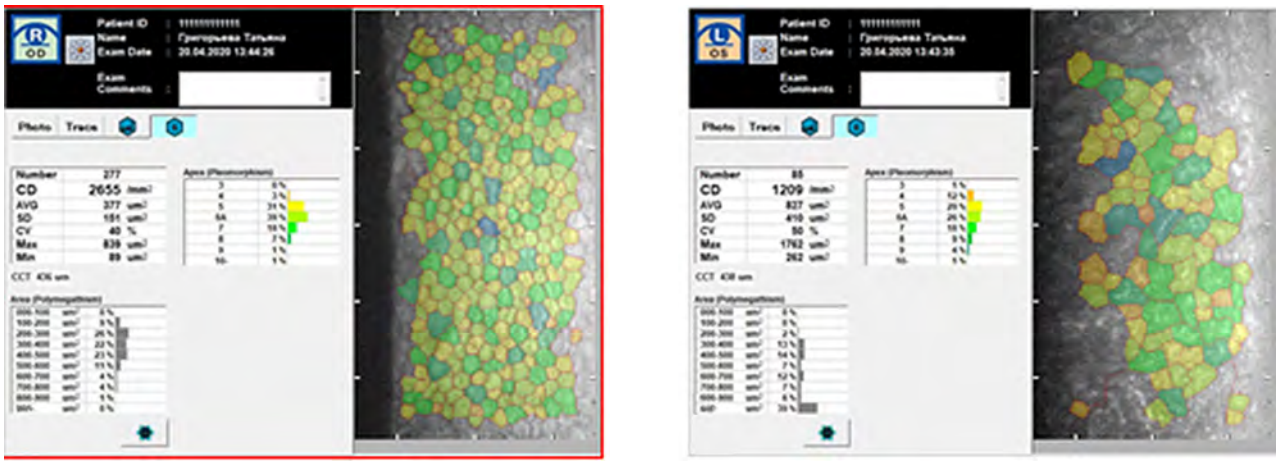


FIG. 2.
*OCT of the cornea: **a** – right eye, linear compression of the stroma corresponding to the removed lenticule; **b** – left eye, insufficient adhesion of the walls of the intrastromal space, small-cystic formations*

Endothelial microscopy of the left eye showed the reduction of endothelial cells number up to 1209/mm^2 (right – 2655/mm^2), a change in their shape and the appearance of epithelioid cells with a hyper-reflective nucle-



a *b*

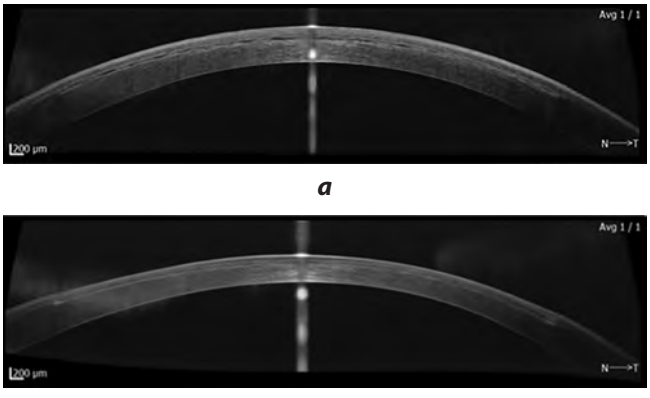
FIG. 3.

*Endothelial microscopy protocol: **a** – right eye, the number of endothelial cells is 2655 per mm^2, the shape and size are not changed; **b** – left eye, the number of endothelial cells is 1209 per mm^2, polymegatism – cells with an area of more than 900 um^2 make up 39 %*

us (Fig. 3), the so-called "ICE cells", which once again confirmed the preliminary diagnosis.

The results of the conducted examination allowed us to state the final diagnosis: iridocorneal endothelial syndrome (Chandler's syndrome), secondary endothelial corneal dystrophy, the condition after SMILE

To reduce the hydrodynamic load on the corneal endothelium hypotensive therapy was prescribed as symptomatic therapy: drops suppressing the production of intraocular aqueous humour – a fixed combination of timolol 0.5 % and dorzolamide 1 % twice a day. After 1 month: IOP = 18/14 mmHg, UCVA = 1.2/1.0, transparent cornea, OCT showed complete adaptation of the walls of the intrastromal space (Fig. 4).



a *b*

FIG. 4.

*OCT of the cornea of the left eye: **a** – before hypotensive therapy, insufficient adhesion of the walls of the intrastromal space; **b** – after hypotensive therapy, complete adaptation of the walls of the intrastromal space, restoration of corneal transparency*

DISCUSSION

In this clinical case, vascular ectropion, fibrous segmental changes in the iris stroma did not alert doctors at the stage of preoperative diagnosis, and laser refractive surgery served as a trigger for the transition of the syndrome into a clinical form of the disease.

It is known that the pathogenetic mechanisms underlying the clinical changes observed in ICD are based on abnormal proliferation of the corneal endothelium [3, 4]. In postnatal age, endothelial cells are postmitotic and do not divide under normal conditions. The pathogenic trigger in IES causes endothelial cells to lose control of the cell cycle. With reduced regulation of expression of cyclin-dependent kinase inhibitors, endothelial cells can proliferate and behave like epithelial cells without undergoing malignant transformation. In this regard, the endothelial cells of the cornea are primarily affected by ICE, their proliferation begins, and the ability to migrate to surrounding tissues also appears [5, 6].

It is known that the appearance of corneal endothelial cells with morphological characteristics resembling the epithelial phenotype, called by E.S. Sherrard and his colleagues in 1985 "ICE cells", which differ from the norm in pleomorphism, polymegatism and the presence of a hyperreflective nucleus, is characteristic of the ICE.

Regardless of the etiological factor, abnormal endothelial cells migrate posteriorly beyond the Schwalbe line, block the iridocorneal angle, move into the anterior chamber of the eye, cover the anterior surface of the iris, form an abnormal basement membrane, which eventually contracts, causing changes in the shape of the pupil, atrophic damage to the iris and the formation of synechiae between neighboring structures. The adhesive process in the corner of the anterior chamber is accompanied by the development of pretrabecular retention of intraocular moisture, an increase in intraocular pressure and the subsequent development of glaucoma in 46–82 % of patients [7].

To date, there are three clinical subtypes of ICE that have a single pathogenetic mechanism, but differ in the nature of pathological changes in the iris. Chandler's syndrome is accompanied by the formation of vascular ectropion (inversion of the pigment border). With progressive atrophy of the iris (essential mesodermal atrophy), the formation of "stretching" or "melting" through ruptures occurs,

with Cogan – Reese syndrome, in addition to ectropion, nevus-like formations of the iris are visualized.

There is no true treatment for iridocorneal endothelial syndrome, so all measures can be attributed to symptomatic therapy [8]:

1) treatment of corneal decompensation and related complications;

2) solving the problem of iris atrophy and its cosmetic and visually significant defects;

3) control of glaucoma associated with ICE.

In the initial stages of the disease, corneal edema can be treated with hypertensive ophthalmic drugs, such as glucose solution 20–40 % or NaCl solution 3 %. A decrease in IOP, even at initially normal values of intraocular pressure, also improves the condition of the cornea. In advanced cases with stable corneal edema with well-controlled IOP, endothelial and descemet membrane transplantation is possible, which is considered today as a procedure of choice to achieve a better functional effect of vision, reduce immunological rejection of the graft and reduce pain [9].

Despite the positive effect of drug and surgical reduction of IOP, the prognosis for the preservation of visual functions is not favorable enough, taking into account the chronic progressive course of iridocorneal endothelial syndrome and its accompanying complications.

CONCLUSION

Manifest changes in the anterior segment of the eye in many syndromes accompanied by the formation of glaucoma make it possible to identify hydrodynamic disorders at the earliest stages and prevent the destructive effect of increased IOP. The diagnosis of a refractive patient should include standard examination approaches: a thorough assessment of changes in the iris, gonioscopy, ophthalmoscopy at maximum mydriasis with an assessment of the state of the optical disc, macular zone and peripheral parts of the retina. The complex and meaningful application of modern imaging methods provides an objective assessment of pathological changes in the eye, and also allows to determine indications and contraindications to refractive surgery.

Conflict of interests

The authors of this article declare the absence of a conflict of interest.

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