# GLAUCOMA OPTIC NEUROPATHY: MODERN METHODS OF EARLY DIAGNOSIS AND PREDICTION OF PROGRESSION

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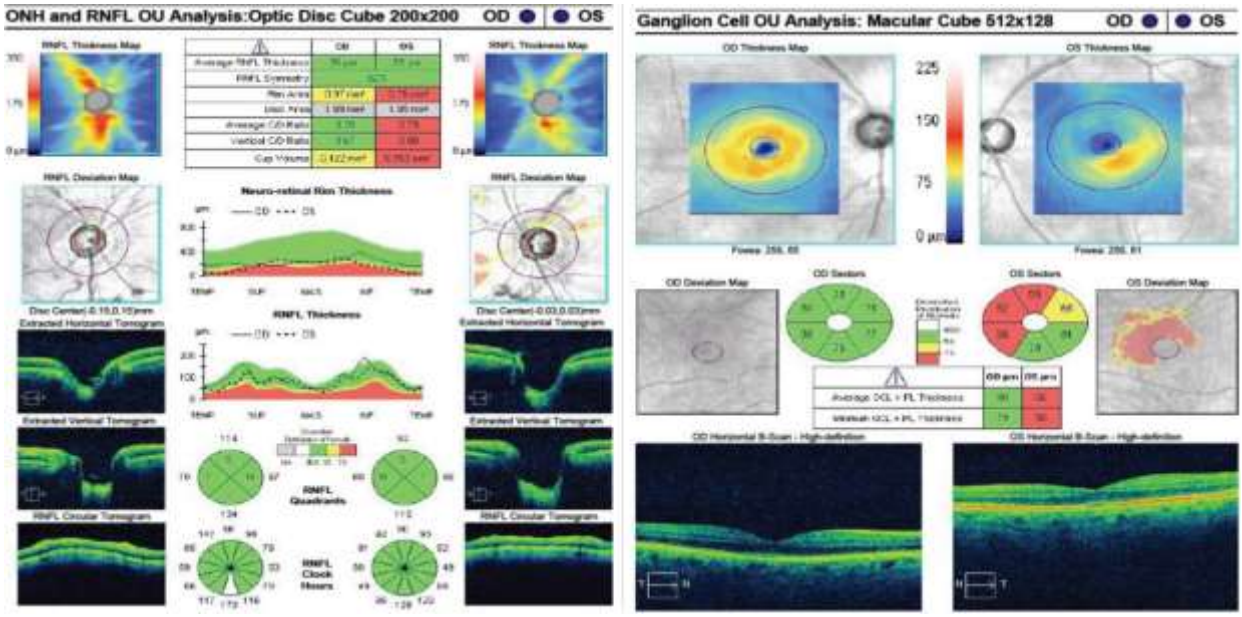
***Abstract.*** *The space between the cornea and the iris is the anterior chamber of the eye, and the space between the iris and the lens is the posterior chamber. Both chambers are filled with intraocular fluid (its second name is "aqueous humor"). This fluid is necessary for the refraction of light, and is also saturated with nutrients necessary for the normal functioning of the organ of vision. Glaucoma is a leading cause of blindness. This eye disease is difficult to diagnose because its symptoms often do not appear until late and is difficult to treat because of the irreversible loss of retinal neurons. Although dead neurons cannot be restored, the progression of glaucoma can be slowed with proper therapy.*

***Keywords:*** *Complications, Diagnosis, Glaucoma, Prevalence, Classification, Causes and progression of the disease, Symptoms.*

**Introduction:** The space between the cornea and the iris is the anterior chamber of the eye, and the space between the iris and the lens is the posterior chamber. Both chambers are filled with intraocular fluid (its second name is "aqueous humor"). This fluid is necessary for the refraction of light, and is also saturated with nutrients necessary for the normal functioning of the organ of vision. The transparent, white outer layer is called the sclera. At the front of the eye, the membrane turns into a transparent convex cornea, which largely controls the refraction of light. The main function of the outer shell is to maintain intraocular pressure and the correct shape of the eye. As we age, the outer shell of all people loses its elasticity and becomes stiffer.

Behind the iris is the ciliary body, which produces intraocular fluid and holds the crystalline lens, a transparent biological lens, capable of changing its curvature, thereby providing a clear image.

The depth of the anterior chamber is approximately 3-3.5 mm, the posterior chamber is significantly narrower. Both cavities communicate with each other through the pupil. Normally, the volume of the eye chambers does not change due to the inflow and outflow of intraocular fluid.




One of the most important structures of the eye is the anterior chamber angle. It is formed where the cornea meets the sclera and the iris meets the ciliary body. It is here that the Schlemm's canal is located, through which moisture flows. This is the drainage system of the eye, the dysfunction of which leads to increased intraocular pressure.

The inner layer (retina) contains visual receptors known as cones and rods. Electrical signals from the receptors are transmitted to the optic nerve, which, through its fibers, enters the cerebral cortex.

The place where the optic nerve exits is called the blind spot - it contains neither rods nor cones.

Under the retina of the eye is the vitreous humor, a transparent, jelly-like substance that maintains the shape and tone of the eye and also participates in the transmission of light rays. In front, the vitreous humor is limited by the lens.

Glaucoma is a general name for a group of eye diseases in which intraocular pressure (IOP) is constantly or periodically increased. As a result, irreversible changes occur in the retina and damage to the optic nerve, which leads to impaired vision and even blindness.

The term "glaucoma" (from the Greek glaukos - "watery blue") was first mentioned in the works of Hippocrates around 400 BC. He called it an eye disease that leads to blindness. As it turned out later, the famous physician did not see the difference between glaucoma and cataracts, so he combined them into one disease.

The disease is named glaucoma because in the later stages, the cornea of the eye may take on a cloudy bluish color.

A "cloudy" eye can be a sign of not only glaucoma, but also other diseases that can only be diagnosed by a doctor.

In the International Classification of Diseases, 10th revision (ICD-10), glaucoma is designated by the code H40. Separate codes are allocated for different variants of the disease:

a. H40.0 - suspected glaucoma;
b. H40.1 - Primary open-angle glaucoma;
c. H40.2 - Primary angle-closure glaucoma;

d. H40.3 - secondary glaucoma after trauma;
e. H40.4 - Glaucoma secondary to inflammatory eye disease;
f. H40.5 - glaucoma secondary to other eye diseases;
g. H40.6 - glaucoma secondary to drug use;
h. H40.8 - Other glaucoma;
i. H40.9 - glaucoma, unspecified;
j. Q15.0 - Congenital glaucoma.

Glaucoma is a serious disease and the main cause of irreversible blindness. In most patients, pathological changes affect both eyes. Every second person with glaucoma does not even suspect that they have the disease.

According to the World Health Organization (WHO), about 600,000 new cases of blindness due to glaucoma are recorded each year, and the total number of people suffering from this disease in the world has already exceeded 100 million.

Every minute, one adult in the world becomes blind from glaucoma, and every 10 minutes, one child becomes blind.

**Research methods and materials:** Glaucoma can be primary (occurs spontaneously and affects only intraocular structures) and secondary (a consequence of injuries and other diseases).

The disease is also classified by origin, course, level of intraocular pressure, and condition of the optic nerve.

Open-angle glaucoma is glaucoma that develops due to problems with the drainage system, meaning the angle of the anterior chamber of the eye is open. If the angle is blocked by something, it is a closed-angle form. There is also a mixed type of glaucoma.

One of the most important indicators in glaucoma is the level of intraocular pressure (IOP). The measurement of IOP is called tonometry. Depending on the measurement method, a distinction is made between tonometric (contact method) and true (non-contact method) intraocular pressure.

**Intraocular pressure level**

a. IOP level
b. Tonometric (Pt),
c. mmHg Art.
d. True (Po),
e. mmHg Art.

**Simple**

a. < 25
b. < 21

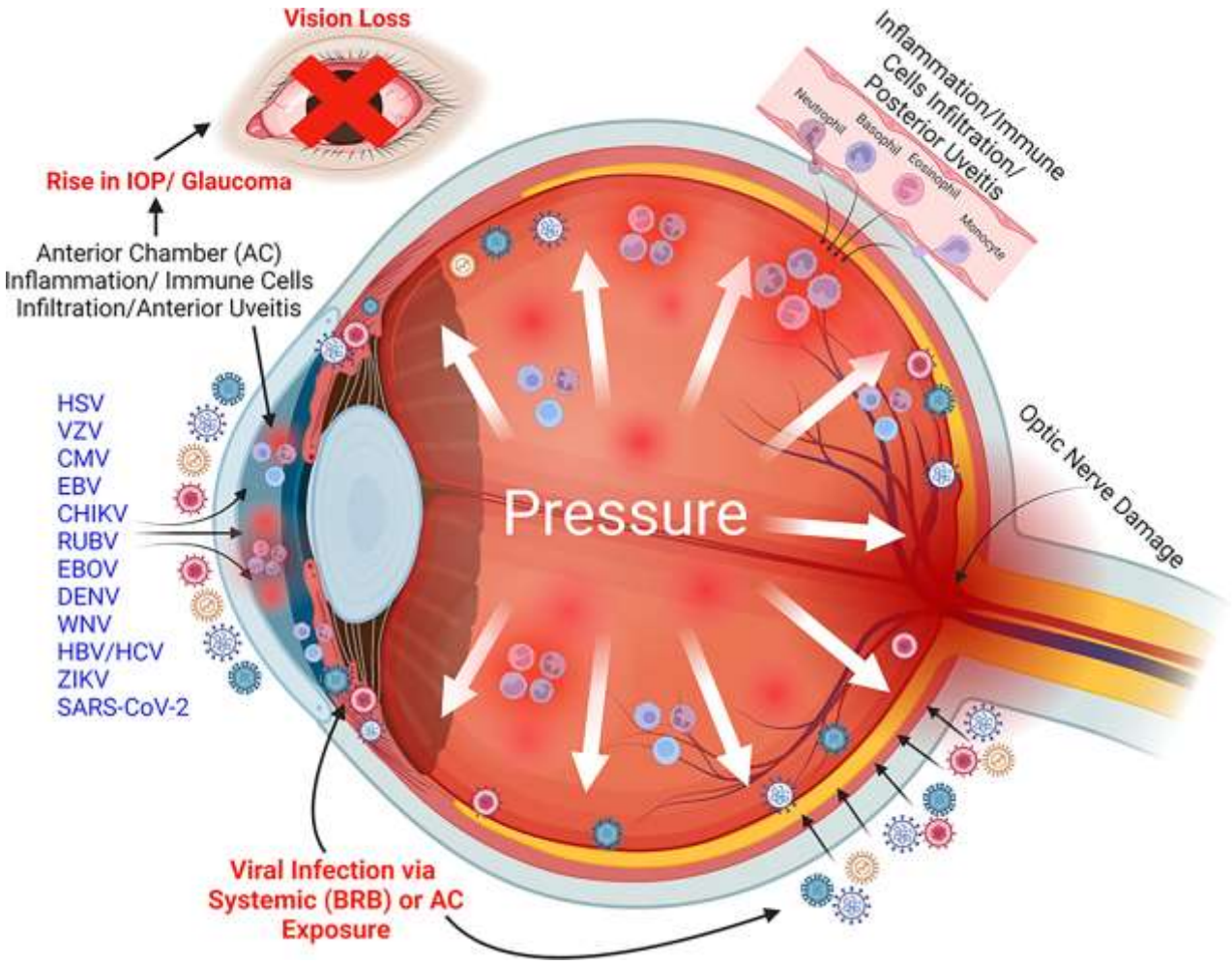
**Moderately elevated**

a. 26–32
b. 21–26

**High**

a. > 32
b. > 26

stabilized - the patient is observed for 6 months or more, during which time the vision and condition of the optic nerve do not deteriorate;




Unstable - the doctor detects deterioration during subsequent examinations.

**Stages of glaucoma depending on the condition of the optic nerve** :

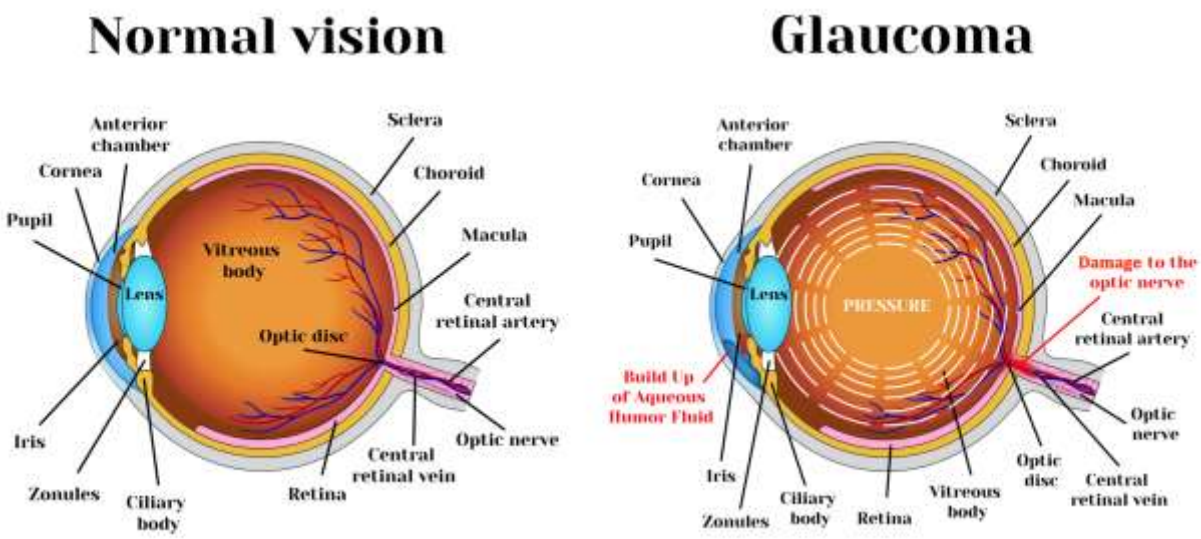
a. elementary;
b. developed;
c. advanced stage;
d. terminal.

The condition of the optic nerve can only be assessed through an ophthalmological examination. The division of the development of glaucoma into stages is arbitrary.

**By time of onset of glaucoma** :

a. congenital: in children under 3 years of age;
b. infantile: from 3 to 10 years old;
c. Minors: from 11 to 35 years old;
d. Glaucoma in adults over 35 years of age.

When making a diagnosis, doctors take into account the type of glaucoma, its stage, the level of intraocular pressure, and changes in vision. An example of a diagnosis: primary open-angle, advanced stage with moderately elevated pressure, stabilized glaucoma.




**Results**: In a healthy eye, aqueous humor circulates easily and unhindered. If the production or outflow of this fluid is impaired, glaucoma develops: when its large amount accumulates, intraocular pressure (IOP) increases.

Normal IOP is 18-24 mm Hg. Art. If intraocular pressure is high, it is called ocular hypertension. Doctors may suspect glaucoma in a person who has no symptoms of the disease but has elevated IOP.

Increased intraocular pressure leads to irreversible damage to the optic nerve and blindness.

The causes of ocular hypertension and glaucoma are very diverse, many of which are still controversial in scientific circles.

**Primary glaucoma**

The main cause of primary glaucoma is tissue dystrophy due to a metabolic defect at the cellular level, which leads to impaired or loss of vision. Translated from Greek, dystrophy means "disorder of tissue nutrition."

There are two main forms of primary glaucoma: open-angle and closed-angle. The disease is most often diagnosed in people over the age of 40.

Approximately 90 percent of all glaucoma cases develop primary open-angle glaucoma.

Primary open-angle glaucoma (POAG) occurs when the outflow of aqueous humor from the angle of the anterior chamber of the eye is impaired, meaning that changes affect the drainage system of the eye.

The process develops over a long period of time and is not noticeable to the patient. As a result, excessive pressure damages the optic nerve: first, peripheral vision is lost, and then central vision. Due to the insidious nature of the disease, primary open-angle glaucoma is sometimes called the silent thief of vision.

In primary open-angle glaucoma, there is an open angle in the anterior chamber of the eye and a violation of the outflow of fluid in the drainage system.

**Open-angle glaucoma symptoms**

Primary open-angle glaucoma is the most common type of glaucoma. The damage to the eye occurs slowly and painlessly, so many people are unaware they have the disease. Often, the disease has caused irreversible vision damage long before it is diagnosed.

A typical symptom of primary open-angle glaucoma (POAG) is the gradual loss of peripheral (side) vision. Patients describe this condition as the appearance of "blind spots" in the periphery. This is followed by a loss of central vision - it is difficult for a person to see objects that are directly in front of them. The process can progress to complete blindness.

**Visual impairment in open-angle glaucoma depending on the stage of the disease**

a. Patients with glaucoma have atypical complaints:
b. temporary blurred vision;
c. rainbow circles around light sources;
d. flashing mosquitoes;
e. headache;
f. eye pain;
g. lacrimation.

Examination of patients with POAG reveals periodic or persistent elevations in intraocular pressure. The difference between the two eyes may be more than 2-3 mmHg. Art., and the range of fluctuations during the day may exceed 5 mm Hg. Art.

On average, it can take 1 to 5 years from the onset of the disease to its first symptoms. In some cases, glaucoma can go through all stages from initial to final within 5 years.

**Symptoms of angle-closure glaucoma**

Primary angle-closure glaucoma (PACG) is less common and the "silent thief of sight" - manifests itself much more clearly than the open-angle form of the disease.

Often, angle-closure glaucoma is characterized by attacks that occur due to a sudden blockage of the angle of the anterior chamber and a violation of the outflow of intraocular fluid. Between attacks, patients do not feel any discomfort. And only when the process becomes chronic, do patients with POAG develop complaints similar to those of patients with POAG.

**Symptoms of an attack of closed-angle glaucoma:**

a. sudden and very severe pain in the eyes;
b. severe headache (localized in the area of the superciliary arches);
c. blurred vision;
d. vomiting and/or nausea;
e. halos or colored rings that appear around light sources;
f. redness of the eyes;
g. excessive lacrimation;
h. Pupil dilation - asymmetrical in both eyes.

Although the disease is bilateral, an acute attack rarely develops in both eyes simultaneously.

**External signs of an acute attack of glaucoma:**

a. loss of corneal transparency - due to swelling of the cornea and clouding of the aqueous humor;

b. asymmetry, pupil dilation and lack of response to light;

c. gray-green color of the pupil - when the lens swells, it loses its transparency and becomes clogged in the pupil area;

d. sharp thickening of the eye - a significant increase in tone when pressing on the eye;

e. flattening of the iris pattern due to swelling;

f. narrowing of the space between the iris and the cornea;

g. redness of the eye - as a result of constriction of blood vessels and impaired blood flow.

h. In closed-angle glaucoma, vision loss can occur within hours of the onset of the disease.

i. To prevent damage to the optic nerve, you should seek medical attention immediately - doctors will immediately lower the eye pressure.

j. Acute eye pain can be caused by an acute attack of glaucoma - without urgent help, a person can lose their vision.

In a patient with closed-angle glaucoma, various factors can trigger an acute attack:

a. stress,

b. excessive excitement,

c. working for long periods of time with your head bowed,

d. large amounts of drunk liquid,

e. hypothermia,

f. medical dilation of the pupil to treat or diagnose eye disease.

If the angle of the anterior chamber of the eye does not close completely, the patient develops a subacute attack of glaucoma. Its manifestations are not as pronounced as in an acute attack:

a. mild pain in the eye,

b. rainbow circles facing the light,

c. blurred vision,

d. mild corneal edema,

e. moderate pupil dilation.

Even minor manifestations of glaucoma cannot be ignored - with each attack, the optic nerve becomes more and more damaged, which threatens complete loss of vision.

Long-term glaucomatous processes - impaired fluid outflow and increased intraocular pressure - lead to serious consequences.

Glaucoma optic neuropathy is damage to the optic nerve due to compression and circulatory problems. This initially leads to partial loss of vision and then blindness.

Terminal painful glaucoma is a condition in which the affected eye experiences constant pain that occurs in the final stages of the disease. Painkillers are ineffective, and the only way to alleviate the condition is to lower the intraocular pressure, but even this therapy only provides temporary relief.

Surgical methods for painful glaucoma are also ineffective and often cause postoperative complications. A radical method to relieve a patient with terminal glaucoma from severe pain is to remove the eyeball.

**Conclusion**: It is important to detect glaucoma before vision problems occur. A person who develops this pathology does not experience discomfort for a long time and does not seek medical attention, so an ophthalmologist can detect the insidious disease, for example, during a medical examination.

Laboratory tests are not prescribed to diagnose glaucoma, instrumental diagnostics are performed using various methods;

Tonometry is a measurement of intraocular pressure. When analyzing the data, absolute IOP values, its daily fluctuations, the difference in tone between the two eyes, and the characteristics of fluctuations when changing body position from horizontal to vertical are taken into account. If an excess of the norm is detected, monitoring is carried out for several days.

**List of used literature:**

1. Andryev S. et al. Experience with the use of memantine in the treatment of cognitive disorders //Science and innovation. – 2023. – T. 2. – №. D11. – C. 282-288.
2. Antsiborov S. et al. Association of dopaminergic receptors of peripheral blood lymphocytes with a risk of developing antipsychotic extrapyramidal diseases //Science and innovation. – 2023. – T. 2. – №. D11. – C. 29-35.
3. Asanova R. et al. Features of the treatment of patients with mental disorders and cardiovascular pathology //Science and innovation. – 2023. – T. 2. – №. D12. – C. 545-550.
4. Begbudiyev M. et al. Integration of psychiatric care into primary care //Science and innovation. – 2023. – T. 2. – №. D12. – C. 551-557.
5. Кадирова А, Юсупов А, Бобоев С. Интраокулярная коррекция миопии высокой степени. Каталог монографий. 2023;(1):1-104.
6. Юсупов АА, Хамракулов СБ, Бобоев СА. КОРРЕКЦИЯ ВРОЖДЕННОЙ АНИЗОМЕТРОПИЧЕСКОЙ МИОПИИ ВЫСОКОЙ СТЕПЕНИ С ИСПОЛЬЗОВАНИЕМ ФАКИЧНЫХ ИНТРАОКУЛЯРНЫХ ЛИНЗ. Advanced Ophthalmology. 2023;1(1):187-190. doi:10.57231/j.ao.2023.1.1.044
7. Абдурахманович БС, Абдуазизович ЮА. МИКРОИМПУЛЬСНАЯ ЛАЗЕРНАЯ ЦИКЛОФОТОКОАГУЛЯЦИЯ В ЛЕЧЕНИИ РЕФРАКТЕРНОЙ ГЛАУКОМЫ. Advanced Ophthalmology. Published online April 19, 2023. Accessed February 2, 2025. https://journals.scinnovations.uz/index.php/ao/article/view/536
8. А.т А, Ж.а Р, А.а Ю, И.н Я. ОЦЕНКА ЭПИДЕМИОЛОГИЧЕСКОЙ СИТУАЦИИ С ДИАБЕТИЧЕСКОЙ РЕТИНОПАТИЕЙ В ГОРОДЕ САМАРКАНД. Экономика и социум. 2024;(4-1 (119)):758-761.
9. Бабаев СА, Кадирова АМ, Хамракулов СБ. ПРИМЕНЕНИЕ ЗАДНЕКАМЕРНЫХ ФАКИЧНЫХ ИОЛ ПРИ КОРРЕКЦИИ МИОПИИ ВЫСОКОЙ СТЕПЕНИ. GOLDEN BRAIN. 2024;2(1):329-335.

10. Абдурахманович БС, Муратовна КА. РЕЗУЛЬТАТЫ ЛЕНСЭКТОМИИ В ЛЕЧЕНИИ БОЛЬНЫХ С ПЕРВИЧНОЙ ЗАКРЫТОУГОЛЬНОЙ ГЛАУКОМОЙ. Advanced Ophthalmology. Published online April 19, 2023. Accessed February 2, 2025. https://journals.scinnovations.uz/index.php/ao/article/view/535
11. З ЖД, А БС. РЕЗУЛЬТАТЫ ОЦЕНКИ УРОВНЯ ЭНДОТЕЛИНА-1 И Д-ДИМЕРОВ В СЛЕЗНОЙ ЖИДКОСТИ У ПАЦИЕНТОВ С АРТЕРИАЛЬНОЙ ГИПЕРТЕНЗИЕЙ. SCIENTIFIC JOURNAL OF APPLIED AND MEDICAL SCIENCES. 2024;3(3):300-307.
12. З ЖД, А БС. РЕЗУЛЬТАТЫ ОЦЕНКИ ЭФФЕКТИВНОСТИ КОМПЛЕКСНОГО ЛЕЧЕНИЯ У ПАЦИЕНТОВ С 3-4 СТАДИЯМИ ГИПЕРТОНИЧЕСКОЙ АНГИОРЕТИНОПАТИИ. SCIENTIFIC JOURNAL OF APPLIED AND MEDICAL SCIENCES. 2024;3(3):308-315.
13. Юсупов А, Василенко А, Юсупова Н. Результаты хирургической коррекции высокой анизометропии у больных с косоглазием. Журнал проблемы биологии и медицины. 2018;(4 (104)):135-136.
14. Жалалова Д, Норматова Н, Бобоев С. Фенофибраты в лечении диабетической офтальмопатии. Каталог монографий. 2023;(1):2-120.
15. Косимов РЭ, Бобоев СА, Кадирова АМ. ХИРУРГИЧЕСКОЕ ЛЕЧЕНИЕ ВТОРИЧНОГО РАСХОДЯЩЕГОСЯ КОСОГЛАЗИЯ У ДЕТЕЙ. Advanced Ophthalmology. 2023;1(1):128-131. doi:10.57231/j.ao.2023.1.1.030
16. Abdurakhmanovich BS, Botirovich KS, Sadullaevich RS. CORRECTION OF ANISOMETROPIA BY PHAKIC INTRAOCULAR LENSES IN PATIENTS WITH CONGENITAL MYOPIA. World Bulletin of Public Health. 2024;33:98-100.
17. S.A.Boboev, Boboev SS, Khamrakulov SB, Abdullaeva DA. EFFICACY AND SAFETY OF DIODE-LASER TRANSSCLERAL CYCLOPHOTOCOAGULATION IN THE TREATMENT OF REFRACTORY GLAUCOMA. World Bulletin of Public Health. 2022;10:35-37.
18. S.a B, S.s B, S K, D.r A. Micropulse Transscleral Cyclophotocoagulation in Combination with Anti-Vegf Therapy in Patients with Neovascular Glaucoma. International Journal of Alternative and Contemporary Therapy. 2024;2(5):149-155.
19. Tolibovich AA, Alimjanovich RJ, Abduazizovich YA, Shavkatjonovna XM. OFTALMOLOGIK YORDAM XOLATI VA UNI DIABETIK RETINOPATIYA BILAN KASALLANGAN BEMORLARDA TAKOMILLASHTIRISH (ADABIYOT SHARHI). JOURNAL OF BIOMEDICINE AND PRACTICE. 2023;8(4). Accessed February 2, 2025. https://tadqiqot.uz/index.php/biomedicine/article/view/8258
20. Kadirova A, Boboev S, Khamidova F, Sobirova D, Khamrakulov S. PERIPHERAL PREVENTIVE LASER COAGULATION OF THE RETINA IN PATIENTS WITH HIGH DEGREE MYOPIA. Journal of Survey in Fisheries Sciences. 2023;10(2S):3932-3940. doi:10.17762/sfs.v10i2S.1716
21. Erkinovich KR. SURGICAL TREATMENT OF JOINT HORIZONTAL STRABISMUS. World Bulletin of Public Health. 2022;10:173-178.

22. Bo'Riyev B. et al. Features of clinical and psychopathological examination of young children //Science and innovation. – 2023. – T. 2. – №. D12. – C. 558-563.
23. Borisova Y. et al. Concomitant mental disorders and social functioning of adults with high-functioning autism/asperger syndrome //Science and innovation. – 2023. – T. 2. – №. D11. – C. 36-41.
24. Ivanovich U. A. et al. Efficacy and tolerance of pharmacotherapy with antidepressants in non-psychotic depressions in combination with chronic brain ischemia //Science and Innovation. – 2023. – T. 2. – №. 12. – C. 409-414.
25. Nikolaevich R. A. et al. Comparative effectiveness of treatment of somatoform diseases in psychotherapeutic practice //Science and Innovation. – 2023. – T. 2. – №. 12. – C. 898-903.
26. Novikov A. et al. Alcohol dependence and manifestation of autoagressive behavior in patients of different types //Science and innovation. – 2023. – T. 2. – №. D11. – C. 413-419.
27. Pachulia Y. et al. Assessment of the effect of psychopathic disorders on the dynamics of withdrawal syndrome in synthetic cannabinoid addiction //Science and innovation. – 2023. – T. 2. – №. D12. – C. 240-244.
28. Pachulia Y. et al. Neurobiological indicators of clinical status and prognosis of therapeutic response in patients with paroxysmal schizophrenia //Science and innovation. – 2023. – T. 2. – №. D12. – C. 385-391.
29. Pogosov A. et al. Multidisciplinary approach to the rehabilitation of patients with somatized personality development //Science and innovation. – 2023. – T. 2. – №. D12. – C. 245-251.
30. Pogosov A. et al. Rational choice of pharmacotherapy for senile dementia //Science and innovation. – 2023. – T. 2. – №. D12. – C. 230-235.
31. Pogosov S. et al. Gnostic disorders and their compensation in neuropsychological syndrome of vascular cognitive disorders in old age //Science and innovation. – 2023. – T. 2. – №. D12. – C. 258-264.
32. Pogosov S. et al. Prevention of adolescent drug abuse and prevention of yatrogenia during prophylaxis //Science and innovation. – 2023. – T. 2. – №. D12. – C. 392-397.
33. Pogosov S. et al. Psychogenetic properties of drug patients as risk factors for the formation of addiction //Science and innovation. – 2023. – T. 2. – №. D12. – C. 186-191.
34. Prostyakova N. et al. Changes in the postpsychotic period after acute polymorphic disorder //Science and innovation. – 2023. – T. 2. – №. D12. – C. 356-360.
35. Prostyakova N. et al. Issues of professional ethics in the treatment and management of patients with late dementia //Science and innovation. – 2023. – T. 2. – №. D12. – C. 158-165.
36. Prostyakova N. et al. Sadness and loss reactions as a risk of forming a relationship together //Science and innovation. – 2023. – T. 2. – №. D12. – C. 252-257.
37. Prostyakova N. et al. Strategy for early diagnosis with cardiovascular diseaseisomatized mental disorders //Science and innovation. – 2023. – T. 2. – №. D12. – C. 166-172.

38. Rotanov A. et al. Comparative effectiveness of treatment of somatoform diseases in psychotherapeutic practice //Science and innovation. – 2023. – T. 2. – №. D12. – C. 267-272.
39. Rotanov A. et al. Diagnosis of depressive and suicidal spectrum disorders in students of a secondary special education institution //Science and innovation. – 2023. – T. 2. – №. D11. – C. 309-315.
40. Rotanov A. et al. Elderly epilepsy: neurophysiological aspects of non-psychotic mental disorders //Science and innovation. – 2023. – T. 2. – №. D12. – C. 192-197.
41. Rotanov A. et al. Social, socio-cultural and behavioral risk factors for the spread of hiv infection //Science and innovation. – 2023. – T. 2. – №. D11. – C. 49-55.
42. Rotanov A. et al. Suicide and epidemiology and risk factors in oncological diseases //Science and innovation. – 2023. – T. 2. – №. D12. – C. 398-403.
43. Sedenkov V. et al. Clinical and socio-demographic characteristics of elderly patients with suicide attempts //Science and innovation. – 2023. – T. 2. – №. D12. – C. 273-277.
44. Sedenkov V. et al. Modern methods of diagnosing depressive disorders in neurotic and affective disorders //Science and innovation. – 2023. – T. 2. – №. D12. – C. 361-366.