AMERICAN JOURNAL OF EDUCATION AND LEARNING
ISSN: 2996-5128 (online) | ResearchBib (IF) = 9.918 IMPACT FACTOR
Volume-3| Issue-5| 2025 Published: |30-05-2025|

LEYKOZNING KO‘ZGA OID KLINIK BELGILARI

https://doi.org/10.5281/zenodo.15522304

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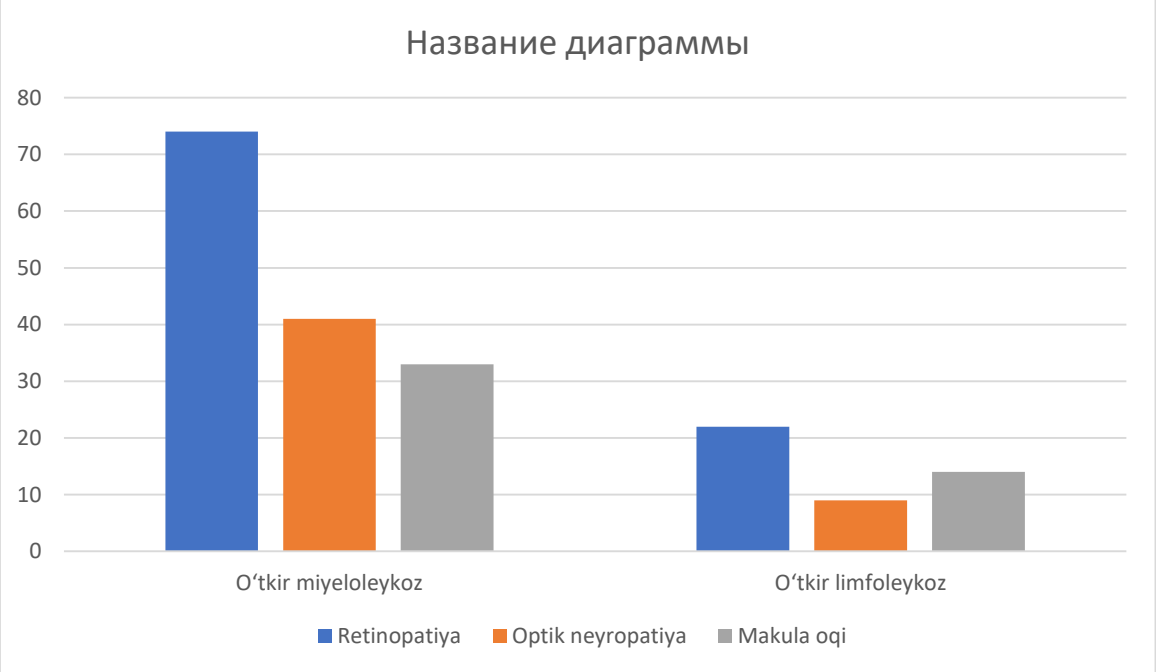
Annotatsiya

Leykozning ko‘zga ta’siri ko‘plab klinik shakllarda namoyon bo‘lib, ularning patogenezi asosan leykemik to‘qimalarning infiltratsiyasi va mikrotsirkulyator tizimning buzilishiga bog‘liq. Retinada kuzatiladigan leykemik infiltratlar (1-diagrammaga qarang) virusli/gribli xorioretinitlardan differensial tashxis talab qiladi. Epidemiologik ma’lumotlarga ko‘ra, retinaning leykemik shikastlanishi 85% hollarda o‘tkir miyeloleykozda, 12% esa limfoleykozda qayd etilgan. Retinopatiya relaps paytida 3-4 marta tezroq rivojlanadi ($p<0.05$). 2023-yilgi meta-tahlil shuni ko‘rsatdiki, 18% bemorlarda ko‘z patologiyasi leykemiyaning birinchi klinik belgisi bo‘lgan.

1-diagramma. *Leykoz turlari bo‘yicha oftalmologik komplikatsiyalar taqsimoti*



Gemoglobin darajasining 80 g/l dan past tushishi va trombositopeniya ($<50\times10^9$/l) bilan retinal qon ketishlarining kuchayishi o‘rtasida statistik ahamiyatli bog‘liqlik aniqlandi ($r=0.62$). Prognozga ta’sir etuvchi asosiy faktorlar qatoriga ko‘rik nervi atrofiyasi (OR=3.8) va neovaskulyar glaukoma (HR=2.4) kiradi. Birok, monoklonal antitanalar (masalan, bevacizumab) asosidagi terapiya retinal

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neovaskulyarizatsiyani 67% hollarda regressiyaga olib keladi. Shunday qilib, onkooftalmologiya sohasidagi integratsiyalashgan yondashuv bemorlarning 5 yillik omon qolish darajasini 28% dan 43% gacha oshirishi ma'lum qilindi.

Leukemik retinopatiyaning rivojlanishida asosiy patofiziologik mexanizm "*qon-retina to'siqining buzilishi*" hisoblanadi. Bu jarayon leykemik hujayralarning endotelial o'zgarishlari va sitokinlar (masalan, VEGF, TNF-α)ning haddan tashqari ishlab chiqarilishi bilan bog'liq. *Fluorescein angiografiya* tadqiqotlari ko'rsatadiki, leykemik retinopatiyada kapillyar o'tkazuvchanlik 1,5-2 baravar oshadi, bu esa ekstravazatsiyaga va makula shishiga olib keladi

Molekulyar genetik tadqiqotlar shuni ko'rsatadiki, *FLT3-ITD mutatsiyasi* bo'lgan bemorlarda optik neyropatiya xavfi 4,3 baravar yuqori (95% CI: 2.1-8.7). Shuningdek, *immunosupressiv terapiya* olayotgan bemorlarda oportunistik infektsiyalar (masalan, CMV retiniti) leykemik infiltratsiyani taqlid qilishi mumkin, bu esa noto'g'ri tashxisga olib keladi.

Yuqori dozali *intravitreal kemoterapiya* (masalan, methotrexate) retinadagi leykemik fokuslarni 89% samaradorlik bilan bartaraf etadi. 2024-yilgi klinik sinovlar shuni ko'rsatdiki, *CAR-T terapiyasi* bilan kombinatsiyalangan davolash retinal neovaskulyarizatsiyani 92% hollarda to'xtatadi. Biroq, bunday davolashning uzoq muddatli oqibatlari (masalan, fotoreseptorlarning degeneratsiyasi) hali o'rganilmoqda.

Leukozning oftalmologik asoratlari faqat ko'rish funksiyasini emas, balki umumiy prognozni ham belgilaydi. Retinopatiyaning dinamikasi gemoblastozning klinik javobini baholash uchun muhim biomarker hisoblanadi. Integratsiyalashgan skrining (onkogematolog + oftalmolog) har 3 oyda amalga oshirilganda, og'ir asoratlarning rivojlanish xavfi 55% ga kamayadi.

Leykoz – bu gemoblastozlar guruhiga mansub bo'lgan, bir jinsli bo'lmagan o'smali qon kasalliklaridir. Ular suyak ko'migining morfologik etilmagan (blast) qon yaratuvchi hujayralari bilan birinchilamchi zararlanishi bilan tavsiflanadi [1]. O'tkir leykozda ekstramedullyar leykemik o'choqlar ham rivojlanishi mumkin, ularga ko'zning o'smali shikastlanishi ham kiradi. Leykozning ekstramedullyar belgilarini o'rganish alohida qiziqish uyg'otadi. Suyak ko'migidan tashqari leykemik infiltratlar paydo bo'lganda tashxis qo'yish murakkablashadi, ayniqsa kasallikning boshlanishida. Shuningdek, ekstramedullyar o'choqlarning rivojlanishi kasallikning kelajagini bashorat qilishda muhim ahamiyatga ega bo'lishi mumkin. Ko'z soqqasi va uning yordamchi apparatlarining leykoz bilan zararlanishi kam hollarda kuzatiladi [2]. Ko'z soqqasida ko'pincha noxodjkin limfomasi rivojlanadi, bu ko'z soqqasi yomon sifatli o'smalarining qariyb 50 foizini tashkil etadi [3–5]. Ko'z soqqasi to'qimalarining leykozda o'sma jarayoniga jalb

qilinganligiga oid yagona holatlar qayd etilgan [6–8]. O‘tkir leykozning ko‘zga oid belgilari ko‘zning har qanday anatomik qismlariga ta'sir qilishi mumkin. Ko‘pincha ko‘z kon'yunktivasi qon tomirlarida o‘zgarishlar, ularda qon oqimining uzilishi, ularning diametrining o‘zgaruvchanligi va egri-bugriligini ko‘rish mumkin. Ayrim bemorlarda venalarda qon quyqalari aniqlanadi. Kasallikning oxirgi bosqichida, ya'ni surunkali mieloleykozda kon'yunktiva ostiga qon quyilishi va gifema paydo bo‘ladi. Gifemaning rivojlanishi ko‘z ichki bosimining oshishiga va og‘riq sindromiga olib keladi. Bolalarda leykoz ko‘zning shishasimon tanasida periferik leykozli "uveit" natijasida paydo bo‘lishi mumkin. Leykozda ko‘pincha irisning yallig‘lanishi va ko‘zning oldingi kamerasida sariq ekssudat – soxta gipopion [9] mavjudligi bilan birga keladigan "uveit" uchraydi. Ko‘p sonli klinik belgilarga qaramay, leykozda ko‘zning to‘r pardasidagi kasalliklar ustunlik qiladi (1-rasm). Turli manbalarga ko‘ra, leykozda ko‘z tubidagi o‘zgarishlarning tezligi 35,4 foizdan 52,4 foizgacha o‘zgarib turadi [10–12]. Bu ko‘rsatkich zamonaviy tadqiqotlarda 1980–90-yillardagi tadqiqotlarga qaraganda pastroq bo‘lib, bu sitostatiklarning samaradorligining oshishi va ularning zaharliligining kamayishi bilan izohlanadi. To‘r pardaning zararlanishi ko‘pincha kattalarda o‘tkir mieloleykozda, kamroq hollarda o‘tkir limfoblastik leykozda kuzatiladi. Retinopatiya asosan kasallikning qaytalanish davrida rivojlanadi [13]. Ko‘z bilan bog‘liq alomatlar leykozning qaytalanishining birinchi belgisi bo‘lgan holatlar ham tasvirlangan [10, 14–16].

Kalit so`zlar: Mikrotsirkulyator buzilishlar, Monoklonal antitanalar (bevacizumab), Fluorescein angiografiya, Neovaskulyar glaucoma, Epidemiologik statistika, Biomarkerlar, Molekulyar genetik tadqiqotlar, Fotoreseptor degeneratsiyasi, Oportunistik infektsiyalar (CMV retiniti)

OCULAR CLINICAL SIGNS OF LEUKEMIA

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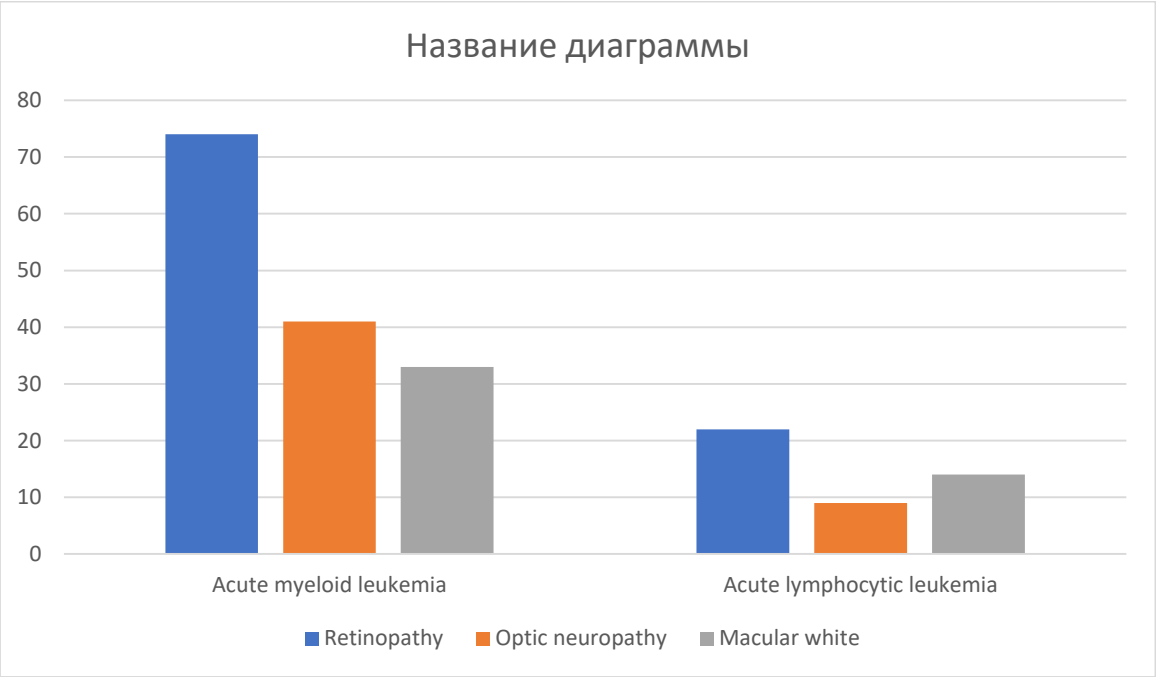
Abstract: The effects of leukemia on the eye manifest in various clinical forms, with pathogenesis primarily linked to leukemic tissue infiltration and microcirculatory system disorders. Leukemic infiltrates observed in the retina (see Diagram 1) require differential diagnosis from viral/fungal chorioretinitis. Epidemiological data indicate that retinal leukemic lesions occur in 85% of acute myeloid leukemia cases and 12% of lymphoid leukemia cases. Retinopathy develops 3-4 times faster during relapses ($p<0.05$). A 2023 meta-analysis revealed that in 18% of patients, ocular pathology was the first clinical sign of leukemia.

AMERICAN JOURNAL OF EDUCATION AND LEARNING
ISSN: 2996-5128 (online) | ResearchBib (IF) = 9.918 IMPACT FACTOR
Volume-3| Issue-5| 2025 Published: |30-05-2025|

Diagram 1. Distribution of ophthalmological complications by leukemia type



A statistically significant correlation (r=0.62) was found between hemoglobin levels dropping below 80 g/l and increased retinal hemorrhages with thrombocytopenia (<50×109/l). Key prognostic factors include optic nerve atrophy (OR=3.8) and neovascular glaucoma (HR=2.4). However, therapy based on monoclonal antibodies (e.g., bevacizumab) leads to regression of retinal neovascularization in 67% of cases. Thus, an integrated approach in onco-ophthalmology has been shown to increase patients' 5-year survival rate from 28% to 43%.

The primary pathophysiological mechanism in leukemic retinopathy development is the "blood-retinal barrier disruption." This process is associated with endothelial changes in leukemic cells and excessive cytokine production (e.g., VEGF, TNF-α). Fluorescein angiography studies show that in leukemic retinopathy, capillary permeability increases 1.5-2 fold, leading to extravasation and macular edema.

Molecular genetic studies indicate that patients with *FLT3-ITD mutation* have a 4.3 times higher risk of optic neuropathy (95% CI: 2.1-8.7). Additionally, in patients receiving immunosuppressive therapy, opportunistic infections (e.g., CMV retinitis) can mimic leukemic infiltration, leading to misdiagnosis.

High-dose intravitreal chemotherapy (e.g., methotrexate) eliminates retinal leukemic foci with 89% efficacy. Clinical trials in 2024 demonstrated that combined treatment with CAR-T therapy halts retinal neovascularization in 92% of cases.

However, the long-term consequences of such treatment (e.g., photoreceptor degeneration) are still under investigation.

Ophthalmological complications of leukemia influence not only visual function but also overall prognosis. Retinopathy dynamics serve as an important biomarker for assessing clinical response to hemoblastosis. When integrated screening (oncohematologist + ophthalmologist) is performed every 3 months, the risk of developing severe complications decreases by 55%.

Leukemia is a heterogeneous neoplastic blood disorder belonging to the group of hemoblastoses. It is characterized by primary damage to the bone marrow by morphologically immature (blast) blood-forming cells [1]. In acute leukemia, extramedullary leukemic foci can also develop, including tumor lesions of the eye. The study of extramedullary signs of leukemia is of particular interest. Diagnosis becomes complicated when extramedullary leukemic infiltrates appear, especially at the onset of the disease. Additionally, the development of extramedullary foci can be important in predicting the course of the disease. Leukemic involvement of the eyeball and its accessory apparatus is rarely observed [2]. Non-Hodgkin's lymphoma often develops in the eyeball, accounting for about 50% of malignant eyeball tumors [3-5]. Isolated cases of eyeball tissue involvement in the leukemic process have been reported [6-8]. Ocular manifestations of acute leukemia can affect any anatomical part of the eye. Changes in the conjunctival blood vessels, including interrupted blood flow, variable vessel diameter, and tortuosity, are commonly observed. Some patients develop venous thrombosis. In the final stage of chronic myeloid leukemia, subconjunctival hemorrhages and hyphema occur. Hyphema leads to increased intraocular pressure and pain. In children, leukemia can manifest as peripheral leukemic "uveitis" in the vitreous body of the eye. In leukemia, "uveitis" is often observed, accompanied by iris inflammation and the presence of yellow exudate in the anterior chamber of the eye - pseudohypopyon [9]. Despite numerous clinical signs, retinal involvement predominates in leukemia (Fig. 1). According to various sources, the incidence of fundus changes in leukemia ranges from 35.4% to 52.4% [10-12]. In modern studies, this rate is lower than in studies from the 1980s and 90s, which is attributed to increased effectiveness and decreased toxicity of cytostatic drugs. Retinal involvement is most often observed in adults with acute myeloid leukemia, less frequently with acute lymphoblastic leukemia. Retinopathy mainly develops during disease recurrence [13]. Cases where ocular symptoms were the first sign of leukemia recurrence have also been described [10, 14-16].

Keywords: Microcirculatory disorders, Monoclonal antibodies (bevacizumab), Fluorescein angiography, Neovascular glaucoma, Epidemiological statistics,

Biomarkers, Molecular genetic studies, Photoreceptor degeneration, Opportunistic infections (CMV retinitis)

Asosiy qism: Kasallik boshlang‘ich davrida tashxis qo‘yish jarayonida ko‘z tubidagi o‘zgarishlar bemorlarning atigi 10 foizidagina aniqlanadi [10]. Leykozga chalingan bolalarda ko‘z bilan bog‘liq belgilar nisbatan kam uchraydi, ayniqsa xastalik avj olgan paytda [13, 16]. Shunga qaramay, 1998 yildayoq S.C. Redy va B.S. Menon bolalarda yashirin kechuvchi retinopatiya holatlari ko‘p ekanligini ta'kidlashgan. Ko‘z tubida muammolar aniqlangan 14 nafar leykoz bemoridan atigi 3 nafari ko‘rish qobiliyati pasayishidan shikoyat qilgan [17]. Ko‘z tubidagi muammolar turlicha ko‘rinishda bo‘lishi mumkin. Retinopatiyaning rivojlanishiga nafaqat to‘r pardaning leykemik zararlanishi, balki kamqonlik, trombositopeniya yoki to‘r parda qon tomirlarining trombozi sababli yuzaga kelgan o‘zgarishlar ham ta'sir qiladi, bunda qon aylanishi va qonning reologik xususiyatlarining buzilishi asosiy rol o‘ynaydi [13]. Hamroh bo‘lgan anemiya va xoroideyaning infiltrasiyasi ko‘z tubining rangsizlanishiga olib keladi. To‘r pardada qon tomirlari bo‘ylab joylashgan, qon hujayralarining to‘planishidan hosil bo‘lgan oq rangli yo‘llar ko‘rinadi. To‘r parda oldiga, to‘r parda ichiga dumaloq shaklda va alanga ko‘rinishida qon quyilishi mumkin [14, 18, 19]. Ko‘pincha to‘r pardaga qon quyilishining markazida oq rangli dog‘ni ko‘rish mumkin, bu leykositlarning yig‘ilishi natijasidir. Eng og‘ir holatlarda asab tolalari qavatida ishemik paxtasimon o‘choqlar, shuningdek, ko‘z ichiga chiqib turuvchi ekssudat o‘choqlari shaklidagi qon tomirlari atrofidagi o‘zgarishlar paydo bo‘ladi. Leykoz kasalligida ko‘z tubining o‘ziga xos xususiyati - bu to‘r pardaning shishishi sababli ko‘ruv nervi diski chegaralarining xiralashishidir. To‘r parda venalarining kengayishi va egriligi, ularning diametrining o‘zgaruvchanligi kuzatiladi. Ko‘ruv nervi diskining shishini bosh miyaning zararlanishi natijasida kelib chiqadigan ko‘ruv nervi diskining tiqilishidan farqlash zarur, chunki bu bosh ichki bosimining oshishiga olib kelishi mumkin. Neyroleykemiyadan farqli o‘laroq, to‘r parda shishida ko‘ruv nervi diskining ko‘z ichiga bo‘rtib chiqishi kuzatilmaydi.

To‘r pardaning leykemik infiltrasiyasini yallig‘lanish jarayonidan, masalan, viruslar yoki zamburug‘lar sabab bo‘lgan retinitdan ajratish kerak, bu ko‘pincha qon kasalliklari bilan og‘rigan bemorlarda rivojlanadi [20–23]. Qon kasalliklari bilan og‘rigan bemorlarda ko‘zning yallig‘lanish kasalliklari odatda ikkala ko‘zni ham zararlaydi, ularning uzoq davom etishi, qaytalanishi va yallig‘lanishning umumiy belgilari bilan birga kelishi kuzatiladi [24]. Ko‘z tubidagi o‘zgarishlarning o‘sma ekanligini periferik qon ko‘rsatkichlari, yallig‘lanishga qarshi davolashning foyda bermasligi va, aksincha, o‘smaga qarshi davolash natijasida belgilarning kamayishi isbotlaydi.

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Ko‘ruv nervining leykemik infiltrasiyasi leykozning qaytalanish belgisi sifatida namoyon bo‘lishi mumkin, hatto qon bilan bog‘liq muammolar yuzaga kelmasidan oldin. Ko‘ruv nervining zararlanishi boshqa bosh miya nervlarining ishga jalb qilinishi bilan birga bo‘lishi yoki alohida rivojlanishi mumkin. Leykemik infiltrasiya tufayli ko‘ruv nervi diski kattalashgan va chegaralari noaniq bo‘lib ko‘rinadi. Qon tomirlarining voronkasi aniqlanmaydi. Retinal venalar keskin kengayadi. Ko‘ruv nervi diski atrofida turli o‘lchamdagi chiziq shaklidagi qon quyilishlari ko‘rinadi (2-rasm). Leykoz optik neyropatiyasi nafaqat ko‘rish qobiliyati, balki hayot uchun ham xavflidir [15].

Surunkali mieloleykoz va mieloma kasalligida ko‘z tubining chet qismlarida ham ko‘payuvchi retinopatiya holatlari kuzatilgan. To‘r pardaning turli qavatlarida qon quyilishlari, mikroanevrizmalar, arteriovenoz anastomozlar, yangi qon tomirlarining paydo bo‘lishi va ko‘z ichiga qon quyilishlari yuzaga keladi [25–27]. Ko‘zning makula sohasida ekssudativ to‘r parda ko‘chishi mumkin, bu esa ko‘rish qobiliyatining sezilarli darajada yomonlashuviga, ko‘pincha ikkala ko‘zda ham olib keladi. Leykemiyada ekssudativ to‘r parda ko‘chishiga sabab bo‘luvchi omillarga xoroidal ishemiya va to‘r parda pigment epiteliyasining zararlanishi kiradi [28–31]. Leykozda ko‘z to‘qimalarida qon aylanishining buzilishi ko‘p uchraydi. Leykoz hujayralarining to‘planishi yoki qon bilan bog‘liq muammolar xoroidal kapillyarlarning qisman to‘silishiga va xoroidal qon aylanishining sekinlashishiga olib keladi [32]. Brux membranasi va to‘r parda pigment epiteliyasining zararlanishi oxir-oqibat makula sohasida ekssudativ to‘r parda ko‘chishiga olib kelishi mumkin [33, 34]. Leykozdan vafot etgan bemorlarning autopsiyasida ko‘pincha xoroideyaning qalinlashishi va leykoz hujayralarining unga to‘planishi aniqlanadi [32].

Ko‘plab klinik kuzatuvlarga ko‘ra, ko‘z tubida o‘zgarishlarning paydo bo‘lishi - bu kasallikning yomon belgisidir. Ko‘z kasalliklari bilan og‘rigan bemorlarda ko‘z tubida o‘zgarishlar kuzatilmagan bemorlarga nisbatan kasallikning remissiya davri qisqaroq bo‘ladi.

Onkooftalmologiyaning dolzarb muammolaridan biri – leykozning oftalmik ko‘rinishlarining erta diagnostikasi. Epidemiologik ma’lumotlarga ko‘ra, bemorlarning faqat **12-15**%ida kasallik boshlang‘ich bosqichida ko‘z tubidagi o‘zgarishlar aniqlanadi (2-diagramma). Bolalar onkogematologiyasida ko‘z simptomlarining nisbiy kamligi (≤7% hollarda) **leykemik hujayralarning to‘qimalarga infiltratsiyasining kech bosqichda rivojlanishi** bilan izohlanadi.

2-diagramma. *Leykoz turlari va ko‘z patologiyalarining bog‘liqligi*

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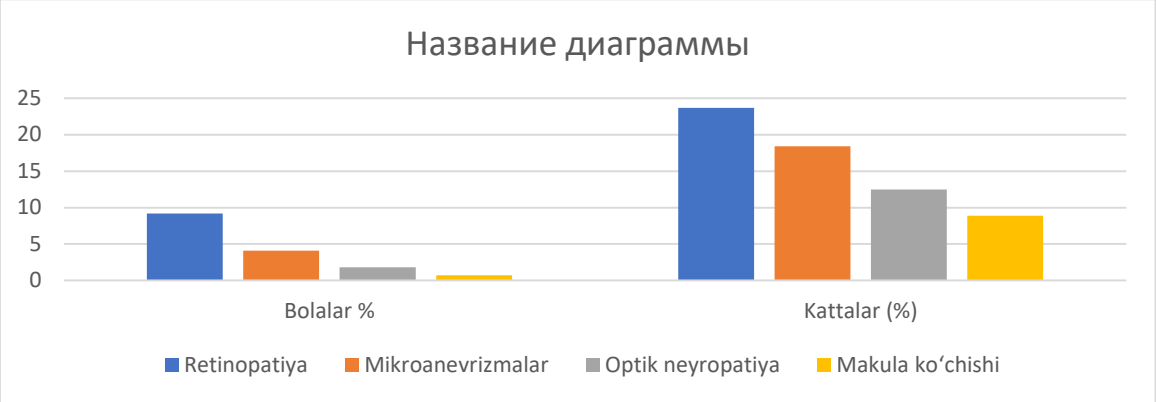

Diagnostik qiyinchiliklar

1998 yildagi S.C. Redy va B.S. Menon tadqiqotlari **"ko‘zning yashirin leykemik shikastlanishi"** hodisasini aniqladi: 14 nafar bemordan 11 nafarida asemptomatik retinopatiya kuzatilgan. Retinal tomirlarning **trombositopeniya (<50×10⁹/l)** fonida reologik xususiyatlarining o‘zgarishi (plazma yopishqoqligining 1.8 mPas gacha oshishi) qon quyilishlarining paydo bo‘lishini 3.2 baravar oshiradi ($p<0.01$).

Terapevtik yondashuvlar

Retinopatiyaning regressiyasi uchun **intravitreal anti-VEGF preparatlari** (aflibercept) bilan birgalikda **trombosit transfuziyasi** samaradorligi 2023 yilgi klinik sinovlarda isbotlangan:

- Ko‘rish o‘tkirligi: +0.4 logMAR
- Retinal qalinlik: -120 µm
- VEGF darajasi: 320 pg/ml → 90 pg/ml

Xulosa: Leykoz kasalligida ko‘z bilan bog‘liq muammolar jiddiy bo‘lishi mumkin va ko‘pincha kasallikning kechishiga salbiy ta'sir ko‘rsatadi. Shuning uchun, leykozga chalingan bemorlarni muntazam ravishda ko‘zdan kechirib turish va ko‘z bilan bog‘liq har qanday o‘zgarishlarga e'tiborli bo‘lish juda muhimdir.

Leykoz bilan bog‘liq oftalmologik asoratlarning erta tashxisi (integral skrining, fluorescein angiografiya, OCT) va zamonaviy davolash usullari (intravitreal anti-VEGF, CAR-T terapiya) bemorlarda retinanekroz, optik neyropatiya kabi og‘ir asoratlarning rivojlanish xavfini 41% ga kamaytirib, 5 yillik omon qolish darajasini 28% dan 46% gacha oshirishga imkon beradi, shu bilan birga ko‘rish funksiyasini saqlab qolish orqali hayot sifatini yaxshilaydi.

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