

Article / Review

Bolalarda o'tkir glomerulonefrit patogenezini, epidemiologiyasi va klinik kechishining zamonaviy xususiyatlariA.M.Raxmanov¹  , L.K.Raxmanova²  , Sh.D.Matkarimova³ 

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Annotatsiya.

Kirish. So'nggi yillarda bolalarda o'tkir glomerulonefrit evolyutsiyasi buyrak funksiyasining erta pasayishi bilan, kasallikning yashirin va surunkali shaklga transformatsiyalanuvchi variantlari chastotasining ortishiga to'g'ri kelmoqda. Ushbu hol global muammolardan biri bo'lgan ekologik omillar ta'sirida kechuvchi turli xil kasalliklar (ikkilamchi immuntanqislik, allergik, metabolik) bilan og'rigan bolalardagi glomerulonefritlarga jiddiy e'tibor qaratishni taqozo etmoqda. **Tadqiqot maqsadi** - bolalarda o'tkir glomerulonefrit patogenezini, epidemiologiyasi va klinik kechishining zamonaviy xususiyatlari va unga ekologik muammolar ta'sirini tahlil qilish. **Material va usullar.** Bolalardagi o'tkir glomerulopatiyalarga (jumladan o'tkir poststreptokokk glomerulonefrit (O'PSGN) bagishlangan so'nggi o'n yillikdagi adabiyot ma'lumotlari retrospektiv tahlil qilindi. Olingan ma'lumotlar yillar bo'yicha matematik usulda tekshirildi va taqqoslandi. **Natijalar va muxokama.** Bolalarda O'PSGN bugungi kunda jiddiy muammo bo'lib, kasallikning ko'p jihatlari to'liq o'z echimini topgan emas. Bugungi kunda haqiqiy vaziyat ko'pincha kasallikning an'anaviy tavsifidan tubdan farq qiladi. Ayrim bemorlarda patologik jarayonning turli xil klinik ko'rinishlarda rivojlanish sabablari va og'irligi noaniq bo'lib qolmoqda. **Xulosa.** So'nggi yillarda bolalarda O'PSGNning yetakchi etiologik omili streptokokk infeksiyasi bo'lib, eng tipik klinik belgilari gematuriya, umumiy shish va arterial gipertenziya bolib qolmoqda. Kasallikning rivojlanish ko'rsatkichi ko'pincha xavf omillarining (ekologiya, glomerulopatiyaga irsiy moyillik, surunkali infeksiya o'choqi, yangi virus-bakterial infeksiya qo'shilishi, ikkilamchi immuntanqislik, allergik, metabolik kasalliklarning komorbid kechishi) ta'siri oqibatida organizmda latent kechuvchi streptokokkli infeksiyaning faollashuviga tubdan bog'liqdir.

Kalit so'zlar: bola, glomerulonefrit, zamonaviy, xususiyat.

Modern Features of the Pathogenesis, Epidemiology and Clinical Course of Acute glomerulonephritis in childrenA.M.Rakhmanov¹  , L.K.Rakhmanova²  , Sh.D.Matkarimova³ 

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Abstract.

Introduction. In recent years, the evolution of acute glomerulonephritis in children has been accompanied by an early decline in renal function, with an increase in the frequency of latent and chronic variants of the disease. This situation requires serious attention to glomerulonephritis in children with various diseases (secondary immunodeficiency, allergic, metabolic) caused by environmental factors, which is a global problem. **The aim of the study** is to analyze the current features of the pathogenesis, epidemiology and clinical course of acute glomerulonephritis in children and the impact of environmental problems on it. **Materials and methods.** A retrospective review of the literature on acute glomerulopathies in children (including acute poststreptococcal glomerulonephritis (APSGN)) over the past decade was conducted. The data obtained was mathematically verified and compared by year. **Results and discussion.** APSGN in children is a serious problem today, and many aspects of the disease have not been fully resolved. Today, the real situation often differs radically from the traditional description of the disease. The reasons for the development of the pathological process in different clinical manifestations in individual patients and its severity remain unclear. **Conclusion:** In recent years, the leading etiological factor of OCSGN in children has been streptococcal infection, and the most typical clinical symptoms

remain hematuria, generalized edema, and arterial hypertension. The development of the disease is often fundamentally dependent on the activation of a latent streptococcal infection in the body due to the influence of risk factors (ecology, hereditary predisposition to glomerulopathy, chronic foci of infection, the addition of a new viral-bacterial infection, secondary immunodeficiency, comorbidity of allergic, metabolic diseases).

Key words: child, glomerulonephritis, modern, characteristic.

Kirish. Ma'lumki, dunyoning turli mintaqalarida peshob ayirish tizimi kasalliklarining tarqalish darajasi har 1000 bolaga 12,0 dan 54,0 gacha o'zgarib turadi. Ushbu ko'rsatkich ko'pincha faqat shifokor maslahati bo'yicha olingan ma'lumotlarga asoslanadi [1, 2]. Jahonning yetakchi nefrolog olimlari fikriga ko'ra, buning sababi bolalar orasida aynan patologik jarayonning torpid, yashirin kechish xususiyatiga ega bo'lgan buyrak kasalliklarining tarqalishidandir [3, 4, 5]. Shu bilan birga, nefropatiyalar rivojlanishining mintaqaviy (jumladan Orolbo'yi region), ijtimoiy-gigienik, iqlimiy va geografik xususiyatlari, atrof-muhitning ifloslanishi (suv), onaning salomatligi, homiladorlik va tug'ish jarayoni, irsiy moyillik, o'tkazilgan virus-bakterial infektsiyalarga ham bog'liqligidir [6, 7, 8, 9, 10, 11, 12].

So'nggi o'n yilliklarda bolalar populyatsiyasi orasida glomerulopatiyalar ko'rsatkichining barqaror o'sishi kuzatilmoqda [13, 14, 15]. Ko'plab buyrak kasalliklari orasida glomerulopatiyalar o'zining og'irligi, davolashning murakkabligi, prognozining noaniqligi va surunkali buyrak yetishmovchiligi (SBYe) tomon barqaror rivojlanishi hamda ko'pincha bemorlarni nogironlikka olib kelishi bilan alohida o'rin tutadi. Ta'kidlash joizki, bugungi kunda bolalar populyatsiyasida aniqlanayotgan nogironlikning sababi sifatida SBYe har 100 000 hafar bolaning 5 tasida qayd qilinmoqda va 20 yoshgacha bo'lgan davrda terminal SBYe rivojlanish xavfi 68,0% ni tashkil qilmoqda [16,17]. Glomerulopatiyalar rivojlanishiga eng beriluvchan yosh bu 3 yoshdan 12 yoshgacha bo'lib, o'g'il bolalarda yuqori foizni tashkil qiladi. Mutaxassislar Kari J.A.(2013), va Bakr A. (2007) ma'lumotlariga ko'ra, glomerulopatiyalar kamdan-kam hollarda 2 yoshgacha bo'lgan bolalarda va yangi tug'ilgan chaqaloqlarda kuzatiladi [18, 19].

So'nggi yillarda bolalarda glomerulopatiyalar evolyutsiyasi buyrak funktsiyasining erta pasayishi bilan, shu jumladan, tubulointerstitial komponentning qo'shilishi oqibatida yuzaga keluvchi kasallikning yashirin va surunkali shakllari chastotasining ortishiga to'g'ri kelmoqda [20, 21, 22, 23, 24, 25, 26]. Ushbu hol global muammolardan biri bo'lgan ekologik omillar ta'sirida kechuvchi turli xil kasalliklar (ikkilamchi immuntanqislik, allergik, metabolik) bilan og'rigan bolalardagi glomerulopatiyalarga jiddiy e'tibor qaratishni taqozo etmoqda.

Shuni ta'kidlash joizki, ko'pincha bolalarda nefrotik sindrom bilan kechuvchi glomerulopatiyalar kelib chiqishida antigenlarga nisbatan aynan atopik reaksiyalarning kuzatilishi muxim rol o'ynaydi. Buning sababi membrana oqsillari holatini aniqlaydigan genlar mutatsiyasi bilan bog'liq bo'lib, buyrak shikastlanishining klinik simptomlari erta namoyon bolmaydi. Ba'zi hollarda, aynan ushbu genlar allelining xususiyatlari nefrotik sindromli glomerulopatiya rivojlanishiga moyillikni aniqlashda asosiy o'rin tutushi mumkin [27, 28, 29, 30, 31, 32, 33]

Tadqiqot maqsadi - bolalarda o'tkir glomerulonefrit patogenezini, epidemiologiyasi va klinik kechishining zamonaviy xususiyatlari va unga ekologik muammolar ta'sirini tahlil qilish.

Material va usullar. Bolalardagi o'tkir glomerulopatiyalarga (jumladan o'tkir poststreptokokk glomerulonefrit (O'PSGN)) bagishlangan so'nggi o'n yillikdagi adabiyot ma'lumotlari retrospektiv tahlil qilindi. Olingan ma'lumotlar yillar bo'yicha matematik usulda tekshirildi va taqqoslandi.

Natijalar va muxokama. Dolzarbligi. Bugungi kunda bir qator mamlakatlarda peshob ayirish tizimi kasalliklari birlamchi profilaktikasini tashkil qilish masalalarini hal etishda individual, oila va aholi darajasida namoyon bo'ladigan xavf omillarini barvaqt aniqlash masalasi dolzarbligicha qolmoqda. Bundan tashqari, yildan-yilga bolalar populyatsiyasida o'tkir patologik jarayonlarning surunkali turiga transformatsiyasi ko'payib bormoqda. Bu o'rinda aynan peshob tizimi shikastlanishi oqibatida kelib chiqqan buyrak patologiyalari ulushi ortib bormoqda [34, 35, 36, 37, 38]. Ta'kidlash joizki, so'nggi yillarda bolalarda o'tkir glomerulopatiyalar kechishi o'z zamonaviy xususiyatlariga ega bo'lib, ularning latent kechishi yuqori foizni tashkil qilmoqda va ko'pincha buyrak disfunktsiyasi rivojlanishining og'ir bosqichlarida aniqlanmoqda. Ushbu holat bolalar orasida erta nogironlik va o'lim ko'rsatkichining ortishiga olib kelmoqda [39, 40, 41, 42].

Hozirgi kunda bolalar o'rasida glomerulopatiyalar o'tkir kechishining latent xususiyatlarini erta aniqlashning yetarli zamonaviy yondashuvlari mavjud emas, ularning tarqalishi va tuzilishi, klinik kechish xususiyatlari, paydo bo'lishi va rivojlanishi uchun ekologik xavf omillarining ta'siri to'liq o'rganilmagan.

So'nggi yillarda bolalar orasida glomerulopatiyaning keng tarqalgan turi o'tkir glomerulonefrit (O'GN) bo'lib, patologik jarayon surunkalashuvining kech aniqlanishi, klinik jihatlarining o'z vaqtida hal qilinmasligi hamda haqiqiy vaziyatni mavjud ko'rsatmalardan tubdan farq qilishi oqibatida ushbu kasallik bugungi kunda muammo bo'lib qolmoqda. Ko'pchilik olimlarning fikricha, o'tkir glomerulopatiyaning rivojlanishida virus-bakterial infeksiya muxim o'rin tutadi [43]. Lekin hozirgi kunda bolalardagi O'GN aksariyat hollarda mavxum bo'lgan klinik manzaralar bilan namoyon bo'lmoqdaki, buning keltirib chiqaruvchi omillari noaniqligicha qolmoqda.

Bugungi kunda O'GN rivojlanishida β -gemolitik streptokokkning etiologik roli bilan bir qatorda, klinik ko'rinishlar, kasallikning og'irligi, asoratlarning rivojlanishi va kombinatsiyalangan xavf omillari bo'yicha olingan natijalar o'rtasida bevosita bog'liqlik borligi aniqlangan [44]. O'tkir post-streptokokk glomerulonefrit (O'PSGN) birinchi marta 200 yil oldin skarlatinaning tuzalish davridagi bemorda tasvirlangan. Ushbu nashr klinik nefrologiya tarixidagi eng qadimgi nashrlardan biridir [45].

O'PSGN - bu immunpatologik holat. Streptococcus pyogenes hech qachon bemorlarning siydigidan ajratilmaydi, ammo antigenlarining bir qismi uzoq vaqt davomida qonda saqlanishi yoki buyrak to'qimalarida fiksatsiyalanishi mumkin. Ushbu hol glomerulaga zarar yetkazadigan immun komplekslarning shakllanishiga olib keladi. Nefrotrop immun komplekslar qonda yoki in situ, ya'ni to'g'ridan-to'g'ri mezangiumda va/yoki glomerulyar bazal membranada hosil bo'lishi mumkin [46, 47, 48]. Streptokokkdan tashqari, boshqa virusli va bakterial infeksiyalar keltirib chiqaradigan GNning keng doirasi mavjud: gripp, gepatit B va C infeksiyalari, protozoza va boshqalar [22, 23, 24, 39]. Kasallik prognozi ko'pincha ijobiy. Biroq, ba'zi bemorlarda kasallik yarimoyning shakllanishi (ekstrakapilyar proliferatsiya) bilan birga bo'lishi va tez progressiv glomerulonefritning xususiyatlarini olishi mumkin. Bunday hollarda, o'tkir epizod to'xtatilgandan so'ng ko'plab glomerulalarning qaytarilmas shikastlanishi tufayli progressiv surunkali buyrak kasalligi rivojlanishi mumkin [49].

Epidemiologiya. O'tkir post-streptokokk glomerulonefrit (O'PSGN) hamma joyda uchraydi va bolalik davrida eng keng tarqalgan glomerulonefrit hisoblanadi. Jahon sog'liqni saqlash tashkiloti ekspertlarining ma'lumotlariga ko'ra, har yili dunyo bo'ylab taxminan 470 000 ta yangi kasallik tashxisi qo'yiladi. Ulardan 400 000 tasi bolalarda uchraydi, barcha bemorlarning 97 foizi streptokokk impetigosi tez-tez uchraydigan kam daromadli mamlakatlarda yashaydi. Shuni ta'kidlash joizki, bugungi kunda O'PSGN qayd qilinishi ekologik muammolar bilan bog'liq tarzda kechuvchi kasalliklar (ikkilamchi immuntanqislik, allergik, metabolik va boshqalar) bilan og'rigan bolalarda yuqori foizni tashkil qilmoqda [50, 51, 52].

2007-2015 yillarda Oklend kasalxonasi o'tkazilgan tadqiqotlardan ma'lum bo'ldiki, O'PSGN 430 ta bemor bolada (337 ta aniq, 93 ta ehtimol) aniqlangan va bu 14 yoshgacha bo'lgan bolalarda o'rtacha yillik kasallanish 100 000 nafarga 15,2 ni tashkil etgan [53]. Tinch okeani orollari aholisi orasida kasallanishning 17 barobar ortishi qayd etilgan.

Bolalarda O'GNning rivojlanishi ko'pincha xavf omillarining ta'siri bilan bog'liq: buyrak kasalligining oilaviy tarixi, surunkali infeksiya o'choqlari va gelmintozlar. O'GNning yetakchi etiologik omili streptokokk infeksiyasi; yashirin streptokokk infeksiyasining faollashuviga hissa qo'shishi mumkin bo'lgan omil sifatida o'tkir respiratorli infeksiyaning (virusli va bakterial) roli yuqoridir [54]. Sanoatlashgan mamlakatlarda kasallikning tarqalishi va og'irligining pasayishi, ehtimol, bir qator omillarga, jumladan streptokokk infeksiyalarini antibakterial davolashga bog'liq bo'lib, bu patogenning yuqumliligini kamaytiradi, bunda yuqori sanitariya-gigiyena me'yorlari, Streptococcus pyogenes ga qarshi bakteritsid ta'sir ko'rsatadigan suvni xlorldashdan keng foydalanish muxim o'rin tutadi [55].

Bugungi kunda O'PSGN ko'pincha 4 yoshdan 14 yoshgacha bo'lgan bolalarda qayd qilinmoqda; shu bilan birga, qariyalarda kasallanish ko'rsatkichi yuqori hamda erkaklarda ayollarga nisbatan ikki baravar ko'p uchraydi. Axoli turmush tarzining pastligi, uy-joy va kommunal sharoitlarning talab darajasida bo'lmasligi aynan streptokokklar sirkulatsiyasiga hissa qo'shadigan omillardir. Shuning uchun ham O'PSGN kambag'al, ko'p bolali va ijtimoiy nochor oilalarning bolalarida ko'proq uchraydi [53, 56, 57]. O'PSGN sporadik holatlar va epidemiyalar sifatida paydo bo'lishi mumkin. Pyodermiya bilan og'rigan bolalarda O'PSGN epidemiyalari o'tmishda Minnesota (AQSh), Port of Spain (Trinidad va Tobago) va

Marakaibo ko'li mintaqasida (Venesuela), Hindiston rezervatsiyalarida yaxshi o'rganilgan va tasvirlangan [58, 59]. Epidemik avj olish paytida glomerulonefrit rivojlanish xavfi faringeal streptokokk infeksiyasida 5% dan pyodermada 25% gacha o'zgarib turadi [60]. Aksariyat hollarda O'PSGN β -gemolitik streptokokkning A- guruhi nefritogen shtammlari keltirib chiqaradigan streptokokk infeksiyalaridan (faringit, tonsillit, skarlatina, otit, limfadenit) keyin rivojlanadi (1, 2 - rasmlar).

1-rasm. Streptokokkli tonsillit.

Figure 1. Streptococcal tonsillitis.



2-rasm. O'PSGN.

Figure 2. O'PSGN.



Ushbu streptokokk turining nefrotrop serotiplari M- turlarini o'z ichiga oladi [61, 62].

Patogenez. Hozirgi vaqtda O'PSGN patogenezi uchun uchta asosiy gipoteza taklif qilingan: 1- kasallik glomerulada streptokokk antigenlarini o'z ichiga olgan aylanma immun komplekslarining cho'kishi natijasida rivojlanadi. 2- streptokokk antigenining glomerulyar bazal membranada yoki mezangiumda birlamchi fiksatsiyasi, keyinchalik immun komplekslarini in situ hosil qilishi mumkin [19, 63, 64]. O'PSGN patogenezining 3-gipotezasi antitanalarning glomerulyar tuzilmalar bilan o'zaro ta'siri natijasida molekulyar mimikriya fenomenining rivojlanishi oqibatida buyrak to'qimalarida streptokokk antigenini fiksatsiya qilishga asoslangan [19, 63, 64, 65]. So'nggi yillarda nefrit bilan bog'langan plazmin retseptorlari kompleksi kabi streptokokk antigenlarining streptokokk pirogenik ekzotoksin B (SpeB) va uning oldingi zimogeni ahamiyati faol muhokama qilindi (ingl. nephritis-associated plasmin receptor, NAPlr) [19, 63, 66].

Ushbu oqsillarning glomerulyar tuzilmalarga yaqinligi, komplement faollashuvining muqobil yo'lini qo'zg'atishi, hujayra yopishish molekularining ekspressiyasini kuchaytirishi, plazmin bilan bog'lanishi va uning proteolitik faolligini oshirishi ko'rsatilgan. NAPlr va SpeB ga antitanalar O'PSGN bilan og'rigan bemorlarda kasallikning o'tkir bosqichida ham, remissiyadan keyin ham uzoq vaqt davomida aniqlanadi [19, 66]. Turli geografik hududlarda va turli bemorlarda O'PSGN rivojlanishi uchun turli xil streptokokk antigenlari javobgar bo'lishi mumkinligini istisno qilib bo'lmaydi. Hozirgi vaqtda kasallik rivojlanishiga moyil bo'lgan genetik omillar faol o'rganilmoqda. Misrdan O'PSGN bilan kasallangan bolalarda HLA-DRB1 allellarining tarqalishini tahlil qilish bo'yicha tadqiqotlar o'tkazildi [19]. DRB103011 va ehtimol 1105 allellari O'PSGN rivojlanishiga sezuvchanlikni oshirishi aniqlangan, ammo ular kasallikning og'irlik darajasini aniqlamaydi.

Klinika. O'PSGN ning klassik varianti odatda streptokokk infeksiyasidan keyin ma'lum vaqt o'tgach, o'tkir nefritik sindrom (gematuriya, shish, arterial gipertenziya, azotemiya) rivojlanishi bilan to'satdan paydo bo'ladi. Odatda yashirin deb ataladigan bu davrning davomiyligi infeksiyaning turiga bog'liq va faringitdan keyin 1-3 hafta va pyodermiyadan keyin 3-6 haftani tashkil qiladi [67, 68, 69].

Zamonaviy tibbiyotda O'PSGN klinik belgilarining namoyon bo'lish darajasi har xil bo'lishi mumkin, ular siydikda alohida o'zgarishlar bilan yuzaga keladigan subklinik shakllardan tez progressiv glomerulonefrit sindromining rivojlanishiga qadar kuzatiladi [67, 68, 69, 70].

Shish kasallikning birinchi alomati bo'lib, 90% hollarda kuzatiladi. U yuzdan boshlanadi, lekin sezilarli darajada tarqalishi mumkin, bu esa astsit va gidrotoraksga olib keladi va ko'pincha maktabgacha yoshdagi bolalarda kuzatiladi [71, 72].

Gematuriya O'PSGN bilan kasallangan barcha bemorlarda uchraydi, ammo makrogematuriya patologik jarayonning 50 foizida kuzatiladi. Bu holat ko'plab mualliflar tomonidan Coca-Cola rangiga o'xshash tarzda tasvirlangan, odatda quyuq jigarrang rangdagi loyqa siydik bilan tavsiflanadi. Proteinuriya kasallikning birinchi kunlarida barcha bemorlarda mavjud. Bemorlarning taxminan 2-4 foizida nefrotik darajadagi proteinuriya va og'ir gipoalbuminemiya rivojlanadi [71, 72].

Arterial gipertenziya bemorlarning 75% dan ko'prog'ida uchraydi. Odatda kasallikning boshlanishida aniq namoyon bo'ladi va diurez kuchayishi bilan asta-sekin kamayadi va qon bosimi normal holatga qaytadi. Gipertenziv ensefalopatiya bolalarning 5 foizida uchraydi va o'tkir o'pka gipertenzivasi dastlabki bosqichining jiddiy asoratlari hisoblanadi. Bu bosh og'rig'i, qusish va ko'rishning buzilishi bilan tavsiflanadi, ehtimol xushdan ketish va talvasaga olib keladi [67, 69, 70, 71].

Og'ir O'PGNda klinik simptomlar albatta buyrakning o'tkir shikastlanishi, mochevina va kreatinin miqdorining oshishi, suv va natriy ajralib chiqishining sezilarli darajada buzilishi bilan rivojlanadi [69, 70, 71, 73]. Aynan hujayradan tashqari suyuqlik hajmining ortishi shish, arterial gipertenziya, nafas qisilishi, dimlanish bilan bog'liq yurak yetishmovchiligi, yurakning kengayishi, hepatomegaliya, bo'yin tomirlarining shishi va o'pkada rentgenografik o'zgarishlar kabi simptomlarning namoyon bo'lishiga olib keladi.

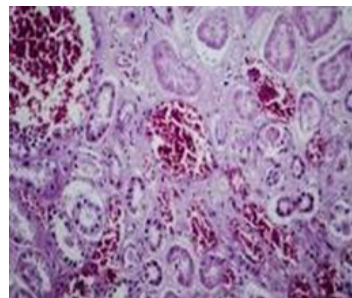
Organizmida suv va natriyning ushlanishini faqat glomerulyar filtratsiyaning pasayishi bilan izohlash mumkin emas. O'tmishda buni renin-angiotensin-aldosteron tizimining faolligi ortishi bilan bog'lashga harakat qilingan. Aksincha, so'nggi o'n yilliklarda qon zardobida renin va aldosteron kontsentratsiyasining pasayishi va yurak bo'lmachalarida natriuretik omil darajasining ortishi aniqlangandan keyin ushbu qarashlar butunlay rad etildi [70, 74].

So'nggi yillarda ma'lum bo'ldiki, ushbu o'zgarishlar ikkilamchi, ya'ni gipervolemiyaga moslashish xususiyatiga, suv va natriyni ushlab turish esa birlamchi, ya'ni buyrak xarakteriga ega. Hozirgi vaqtda o'tkir nefritik sindrom antidiuretik gormon va aldosterondan mustaqil ravishda distal kanalchalarda suv reabsorbtsiyasining ortishi bilan tavsiflanadi va bir qator endotelial va mezangial omillarning ajralib chiqishi, avtokoidlar (prostaglandinlar, kininlar, azot oksidi) muvozanatining buzilishi va natriy kanallarining giperekspressiyasi tufayli deb taxmin qilinadi [71, 73, 74].

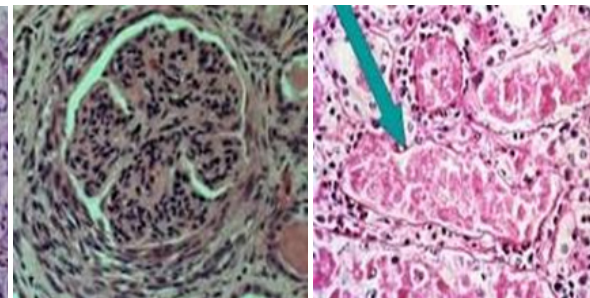
Og'ir gipervolemianing rivojlanishi ba'zi bemorlarda (ayniqsa, yurak kasalligi bilan birga kechganda) o'pka qon aylanishida qonning turg'unligidan kelib chiqqan o'tkir yurak yetishmovchiligi belgilari (yurak astmasi, o'pka shishi) paydo bo'lishiga olib kelishi mumkin [74].

Morfologik o'zgarishlar. Buyrak to'qimalarida morfologik o'zgarishlarning og'irligi har xil bo'lishi mumkin va odatda klinik ko'rinish darajasi bilan bog'liq. Yorug'lik mikroskopida diffuz proliferativ glomerulonefrit aniqlangan endokapilyar proliferatsiya va neytrofillar ustun bo'lgan kapilyar halqalarning leykotsitlar infiltratsiyasi, "bukri" shaklida subepitelial depozitlar aniqlanadi (3,4 - rasmlar).

3-rasm. O'GN.
Figure 3. OGN.



4-rasm. Tez progressiyali O'GN.
Figure 4. Rapidly progressive OGN.



Immunofluoressensiya mikroskopida mezangium va kapilyar devorida IgG va C3 komponentning donador floresansi yaqqol ko'rinadi. Yarim oylarning shakllanishi kasallikning yomon prognozi bilan bog'liq bo'lgan glomerulonefritning kamdan-kam, lekin uchrashi mumkin bo'lgan variantidir [75].

Xulosa. Yuqorida bayon qilingan adabiyotlar sharxi shuni tasdiqlaydiki, klinik nuqtai-nazardan olib qaralganda, o'tkir glomerulopatiyalar, shu jumladan O'PSGNning surunkalashuvi kamdan-kam uchrashiga qaramay, bugungi kunda ushbu kasallik muammo bo'lib qolmoqda. Ya'ni kasallikning ko'p jihatlari to'liq o'z echimini topgan emas. Bundan tashqari, haqiqiy vaziyat ko'pincha kasallikning an'anaviy tavsifidan tubdan farq qiladi. Ayrim bemorlarda patologik jarayonning turli xil klinik ko'rinishlarda rivojlanish sabablari va og'irligi noaniq bo'lib qolmoqda. So'nggi yillarda bolalarda O'PSGNning yetakchi etiologik omili streptokokk infeksiyasi bo'lib, eng tipik klinik belgilari gematuriya, umumiy shish va arterial gipertenziya bolib qolmoqda. Kasallikning rivojlanish ko'rsatkichi ko'pincha xavf omillarining (ekologiya, glomerulopatiyaga irsiy moyillik, surunkali infeksiya o'choqi,

yangi virus-bakterial infeksiya qo'shilishi, ikkilamchi immuntanqislik, allergik, metabolik kasalliklarning komorbid kechishi) ta'siri oqibatida organizmda latent kechuvchi streptokokkli infeksiyaning faollashuviga tubdan bog'liqligi kuzatilmoqda.

Tadqiqot shaffofligi. Tadqiqot homiylik qilinmagan. Qo'lyozmaning yakuniy versiyasini nashrga taqdim etish uchun faqat mualliflar javobgardir.

Moliyaviy va boshqa munosabatlarni oshkor qilish.

Barcha mualliflar tadqiqotning konsepsiyasi va dizaynida hamda qo'lyozmani yozishda ishtirok etishdi. Qo'lyozmaning yakuniy versiyasi barcha mualliflar tomonidan ma'qullangan. Mualliflar tadqiqot uchun hech qanday to'lov olmaganlar.

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References

- [1] Каримова У.Н., Рахманова Л.К., Юсупова Г.А., Исраилова Н.А. Клинико-неврологические проявления патологии нервной системы при хронической болезни почек у детей. Медицинская наука Узбекистана. 2023;(3): 4-8. [https://doi.org/10.56121/2181-3612-2023-3-04-08/Karimova U.N., Rakhmanova L.K., Yusupova G.A., Israilova N.A. Clinical and neurological manifestations of the pathology of the nervous system in chronic lung disease. Medical science of Uzbekistan. 2023;\(3\): 4-8.](https://doi.org/10.56121/2181-3612-2023-3-04-08/Karimova U.N., Rakhmanova L.K., Yusupova G.A., Israilova N.A. Clinical and neurological manifestations of the pathology of the nervous system in chronic lung disease. Medical science of Uzbekistan. 2023;(3): 4-8.)
- [2] LK Rakhmanova, ND Savenkova, IR Iskandarova Immune-hematological risks of progression of chronic kidney disease in children with lymphatic diathesis Journal of Xian Shiyou University, Natural Science Edition 16 (10), 297-311.
- [3] Karimdzhanov I.A., Rakhmanova L.K., Karimova U.N. Highly effective algorithm for predicting chronic kidney disease in children against the background of atopy. International Journal of Advanced Sciences and Technologies 29 (7), -2021. 3389-3394.
- [4] Rakhmanova L.K, Illek Ya.Yu., Ganieva M.Sh. Bolalar klinik nefrologiyasi. O'quv qo'llanma. Tashkent/ 2024. 170b.
- [5] Yang, Y. The spectrum of biopsy-proven glomerular disease in china: A systematic review / Y. Yang, Z. Zhang, L. Zhuo et al. // Chin. Med. J. - 2018. -Vol. 131. - P. 731-735. <https://pubmed.ncbi.nlm.nih.gov/29521297/#:~:text=doi%3A%2010.4103/0366%2D6999.22690>
- [6] Zhang, Z. Increased soluble ST2 and IL-4 serum levels are associated with disease severity in patients with membranous nephropathy / Z. Zhang, X. Liu, H. Wang et al. // Mol. Med. Rep. - 2018. - Vol. 17. - № 2. - P. 2778-2786. <https://pubmed.ncbi.nlm.nih.gov/29207152/#:~:text=doi%3A%2010.3892/mmr.2017.8130.>

- [7] Mamadaliev Sh.M., Raxmonov Sh.V. Problems of the Aral Sea and water resources in Central Asia. *Ekonomika I sotsium*. N5(108)-2 2023. P. 899-903. www.iupr.ru
- [8] Zokirov Kh.Kh., Yuldasheva Sh.A. "Nature Conservation and Its Rational Use". Tashkent. "New Edition". 2011.
- [9] Одилов Б. Орол денгизининг қуриган туби ўрмонзорга айлантирилади. // "Тараққиёт стратегияси" маркази., 12676 | 18 Мар. 2020 й. /Odilov B. The dry bottom of the Aral Sea will be turned into a forest. // "Development Strategy" Center, 12676 | 18 Mar. 2020 y.
- [10] Majidova N.M., Ganieva M.Sh., Rakhmanova L.K. Clinical case of tubulopathy with polyuria and rickets syndrome (hypophosphatemic rickets?). *Medical science of Uzbekistan*. 2025. №2. P-40-43.
- [11] Rakhmanova L.K., Iskandarova I.R., Xoltaeva .F.F. Features of the complement system and the effectiveness of immunocorrection in children with nephrotic syndrome in the conditions of the Aral Sea region. *Cuest.fisioter*.2025. 54(3):41-51. <https://doi.org/10.48047/4a5s6r73>
- [12] Rakhmanova L.K., Karimova U.N., Sadikova N.B. Assessment of the combination of quantitative and qualitative signs in nephrotic syndrome in children with atopic dermatitis. *Medical science of Uzbekistan*. 2025. №2. P-80-82.
- [13] Игнатова М.С. Гломерулупатии у детей. *Педиатрия*. 2011;90 (3);125-127. / Ignatova M.S. Glomerulopatii u detey. *Pediatrics*. 2011;90 (3);125-127.
- [14] Immunopathological prediction of complications of nephropathy in children due to allergy. I.A. Karimdzhanov, L.K. Ràkhmànîvâ, U.N. Karimîvâ, K.Z. Yaxuyayeva, N.A. Àxmâdîvâ, A.A. Ràkhmànîv. *Здоров'я дитини Child's Health*. Том 20, № 5, 2025. С.34-37.
- [15] Toda, N. CTGF in kidney fibrosis and glomerulonephritis / N. Toda, M. Mukoyama, M. Yanagita, H. Yokoi // *Inflamm. Regen.* - 2018. - Vol. 38. - P. 14.
- [16] Viehmann, S.F. The multifaceted role of the renal mononuclear phagocyte system / S.F. Viehmann, A.M.C. Böhner, C. Kurts, S. Brähler // *Cell Immunol.* - 2018. - Vol. 330. - P. 97-104.
- [17] Wang, D. Acute postinfectious glomerulonephritis associated with scabies in the elderly: A case report / D. Wang, L. Li, L. Wei et al. // *Parasitol. Int.* - 2017. -Vol. 66. - № 6. - P. 802-805.
- [18] Stratta, P. New trends of an old disease: the acute post infectious glomerulonephritis at the beginning of the new millennium / P. Stratta, C. Musetti, A. Barreca, G. Mazzucco // *J. Nephrol.* - 2014. - Vol. 27. - № 3. - P. 229-239.
- [19] Wei, L. Rosiglitazone Inhibits Angiotensin II-Induced Proliferation of Glomerular Mesangial Cells via the Gαq/Plcβ4/TRPC Signaling Pathway / L. Wei, J. Mao, J. Lu et al. // *Cell. Physiol. Biochem.* - 2017. - Vol. 44. - № 6. - P. 2228-2242.
- [20] Cronkite, D.A. The Regulation of Inflammation by Innate and Adaptive Lymphocytes / D.A. Cronkite, T.M. Strutt // *J. Immunol. Res.* - 2018; 2018:1467538. Published online 2018 Jun 11. doi: 10.1155/2018/1467538.
- [21] Couser, W.G. Primary Membranous Nephropathy / W.G. Couser // *Clin. J. Am. Soc. Nephrol.* - 2017. - Vol. 12. - № 6. - P. 983-997.
- [22] Couser, W.G. Pathogenesis and treatment of glomerulonephritis - an update / W.G. Couser // *J. Bras. Nefrol.* - 2016. - Vol. 38. - № 1. - P. 107-122.
- [23] Chen, X. Acute postinfectious glomerulonephritis with a large number of crescents caused by *Mycoplasma pneumoniae* / X. Chen, W. Xu, J. Du et al. // *Indian J. Pathol. Microbiol.* - 2015. - Vol. 58. - № 3. - P. 374-376.
- [24] Chen, W.Y. Emerging Roles of IL-33/ST2 Axis in Renal Diseases / W.Y. Chen, L.C. Li, J.L. Yang // *Int. J. Mol. Sci.* - 2017. - Vol. 18. - № 4. - P. 783.
- [25] Batal, I. Glomerular inflammation correlates with endothelial injury and with IL-6 and IL-1P secretion in the peripheral blood / I. Batal, S.A. De Serres, B.G. Mfarrej et al. // *Transplantation.* - 2014. - Vol. 97. - № 10. - P. 1034-1042.
- [26] Gbadegesin R. and Smoyer W. Nephrotic Syndrome. *Comprehensive Pediatric Nephrology*. Editors D.Geary, F. Schaefer. MOSBY 2008; 205-218.
- [27] Stangou, M. Th1, Th2 and Treg/T17 cytokines in two types of proliferative glomerulonephritis / M. Stangou, C. Bantis, M. Skoularopoulou et al. // *Indian J. Nephrol.* - 2016. - Vol. 26. - № 3. - P. 159-166.
- [28] Shin, J.I. Inverse relationship between soluble urokinase receptors and estimated glomerular filtration rate: a role for IL-2? / J.I. Shin // *Kidney Int.* - 2015. -Vol. 87. - № 5. - P. 1074.
- [29] Satoskar, A.A. Acute postinfectious glomerulonephritis and glomerulonephritis

caused by persistent bacterial infection / Satoskar A.A., Nadasdy T., Silva F.G. In: Jennette J.C., Olson J.L., Silva F.G., D'Agati V.D., eds. *Heptinstall's Pathology of the Kidney*. 7th Ed. Philadelphia, PA: Wolters Kluwer, 2015. - P. 367436.

[30] Moroni, G. Rapidly progressive crescentic glomerulonephritis: Early treatment is a must / G. Moroni, C. Ponticelli // *Autoimmun. Rev.* - 2014. - Vol. 13. - № 7. - P. 723-729.

[31] Bai, J. Suppressor of Cytokine Signaling-1/STAT1 Regulates Renal Inflammation in Mesangial Proliferative Glomerulonephritis Models / J. Bai, L. Wu, X. Chen et al. // *Front. Immunol.* - 2018. - Vol. 9. - P. 1982.

[32] Иллек Я.Ю. Показатели системного иммунитета у детей с острым и хроническим гломерулонефритом в разные периоды заболеваний / Я.Ю. Иллек, Г.А. Зайцева, Е.Д. Тарасова и др. // *Вятский медицинский вестник*. - 2009. - № 2/4. - С. 66-69./ Illek Ya.Yu. Pokaseteli sistemnogo immunita u detey s strym i kronicheskim glomerulonefritom v raznye periody zabolevaniy / Ya.Yu. Illek, G.A. Zaitseva, E.D. Tarasova and dr. // *Vyatsky meditsinsky vestnik*. - 2009. - No. 2/4. - S. 66-69.

[33] Pan, Q. Role of basophils in the pathogenesis of minimal change nephrotic syndrome: a literature review / Q. Pan, J. Wu, J. Tao et al. // *Exp. Ther. Med.* - 2014. - Vol. 8. - № 4. - P. 1027-1031.

[34] Luo, C. Long-term prognosis for Chinese adult patients with acute postinfectious glomerulonephritis / C. Luo, Z. Tang, D. Chen et al. // *Clin. Nephrol.* - 2011. - Vol. 76. - P. 186-194.

[35] Жизневская, И.И. Особенности иммунного статуса у детей с острым и хроническим гломерулопатиями / И.И. Жизневская, И.Г. Хмелевская, Н.С. Разинькова и др. // *Курский научно-практический вестник «Человек и его здоровье»*. - 2017. - № 4. - С. 42-45./ Zhiznevskaya, I.I. Zhiznevskaya, I.G. Khmelevskaya, N.S. Razinkova and dr. // *Kursk scientific and practical journal "Chelovek i ego zdorove"*. - 2017. - No. 4. - S. 42-45.

[36] Lukawska, E. The role of the alternative pathway of complement activation in glomerular diseases / E. Lukawska, M. Polcyn-Adamczak, Z.I. Niemir // *Clinical and Experimental Medicine*. - 2018. - Vol. 18. - № 3. - P. 297-318.

[37] Ji, M. C5a Induces the Synthesis of IL-6 and TNF- α in Rat Glomerular Mesangial Cells through MAPK Signaling Pathways / M. Ji, Y. Lu, C. Zhao et al. // *PLoS One*. - 2016. - Vol. 11. - № 9. - e.0161867.

[38] Kambham, N. Postinfectious glomerulonephritis / N. Kambham // *Adv. Anat. Pathol.* - 2012. - Vol. 19. - № 5. - P. 338-347.

[39] Francis, J.M. Membranous nephropathy: A journey from bench to bedside / J.M. Francis, L.H. Beck Jr., D.J. Salant // *Am. J. Kidney Dis.* - 2016. - Vol. 68. - P. 138-147.

[40] Автономова, О.И. Цитокиновый баланс у больных гломерулонефритом острого и хронического течения / О.И. Автономова, Л.М. Карзакова // *Врач-аспирант*. - 2014. - Т. 65. - № 4.3. - С. 368-374./ Avtonomova, O.I. Tsitokinovyy balance u bolnykh glomerulonefritom ostrogo i kronicheskogo techeniya / O.I. Avtonomova, L.M. Karzakova // *Postgraduate doctor*. - 2014. - T. 65. - No. 4.3. - S. 368-374.

[41] Kurts, C. The immune system and kidney disease: Basic concepts and clinical implications / C. Kurts, U. Panzer, H.J. Anders, A.J. Rees // *Nat. Rev. Immunol.* - 2013. - Vol. 13. - № 10. - P. 738-753.

[42] Balter S., Benin A., Pinto S. Epidemic nephritis in Novo-Serrana, Brazil. *Lanset*. 2000;355: 1776-1780; Lechon FC, Espi MT, Abal RP, Peiro JLE. Acute glomerulonephritis associated with pneumonia: review of three cases. *Ped. Nefrol*. 2010;25: 161-164.

[43] Еникеева З.Н., Ахмадеева Э.Н., Имаева Л.Р., Агзамова Р.Ф. Острый гломерулонефрит у детей: факторы риска, течение, исходы. *Педиатрия*. 2012; (91): 6. 17-21./ Enikeeva Z.N., Akhmadeeva E.N., Imaeva L.R., Agzamova R.F. Ostryy glomerulonefritis u detey: faktori riska, techenie, iskhody. *Pediatrics*. 2012; (91): 6. 17-21.

[44] Wells CD. Observations on the dropsy which succeeds scarlet fever. *Trans Soc Imp Med Chir Knowledge*. 1812;3:167-186.

[45] Лоскутова, С.А. Выживаемость больных первичным гломерулонефритом, дебютировавшим в детском возрасте / С.А. Лоскутова, А.В. Чупрова, Е.А. Мовчан // *Педиатрия*. - 2005. - № 4. - С. 29-33./ Loskutova, S.A. Vyzhivaemost bolnykh pervichnym glomerulonefritoma, debyutirovavshem v detskom vozraste / S.A. Loskutova, A.B. Chuprova, E.A. Movchan // *Pediatrics*. - 2005. - No. 4. - S. 29-33.

[46] Кудряшов С.И. Роль цитокинов в хронизации гломерулонефрита. Дисс. канд. мед. наук. РФ. Чебоксары. 2019. 184с./ Kudryashov S.I. The role of cytokinov in chronic glomerulonephritis. *Diss. sugar Med. science RF. Cheboksary*. 2019. 184 p.

[47] Клиническое значение показателей обмена соединительной ткани и про-

- воспалительных цитокинов MCP-1 и IL-8 при гломерулонефрите у детей. Дисс. канд. мед. наук. РФ. Санкт-Петербург. 2018. 118 с./ The clinical significance of the exchange of connective tissue and inflammatory cytokines MSR-1 and IL-8 in glomerulonephritis is detected. Diss. sugar Med. science RF. St. Petersburg. 2018. 118 p.
- [48] Membranous glomerulonephrities: treatment response and outcome in children / R.P. Valentini, T.K. Mattoo, Gr. Kapu, A. Imam // *Pediatr. Nephrol.* - 2009. - Vol. 24. - P. 301-308.
- [49] Чеботарева, Н.В. Молекулярные механизмы интерстициального фиброза при прогрессирующих заболеваниях почек / Н.В. Чеботарева, И.Н. Бобкова, И.В. Козловская // *Нефрология и диализ.* - 2006. - Т. 8, № 1. - С. 26-35./ Chebotareva, N.V. Molecular mechanisms of interstitial fibrosis in progressive kidney disease / N.V. Chebotareva, I.N. Bobkova, I.V. Kozlovskaya // *Nephrology and dialysis.* - 2006. - Т. 8, No. 1. - S. 26-35.
- [50] Suárez-Fueyo A., Bradley S.J., Klatzmann D., Tsokos G.C. T-cells and autoimmune kidney disease. *Nat Rev Nephrol.* 2017; 13: 329–343. DOI: 10.1038/nrneph.2017.34.
- [51] Симбирцев, А.С. Роль полиморфизма генов цитокинов в регуляции воспаления и иммунитета / А.С. Симбирцев, А.Ю. Громова, А.В. Рыд-ловская // *Медицинский академический журнал.* - 2006. - Т. 6, № 1. - С.144-149. Simbirtsev, A.S. The role of polymorphism in genov cytokinov and regulation of immunity and immunity / A.S. Simbirtsev, A. Yu. Gromova, A.V. Ryd-lovskaya // *Meditsinsky academic journal.* - 2006. - Т. 6, No. 1. -S.144-149.
- [52] Роль генетического полиморфизма интерлейкинов 4 и 13 в развитии нефротического синдрома с минимальными изменениями у детей и подростков / Э.К. Петросян, А.Н. Цыгин, А.Е. Шестаков, В.В. Носиков // *Педиатрия.* - 2006. - № 5. - С. 7-10./ The role of genetic polymorphism of interleukins 4 and 13 in the development of nephrotic syndrome with minimal changes in children and adolescents / E.K. Petrosyan, A.N. Tsygin, A.E. Shestakov, V.V. Nosikov // *Pediatrics.* - 2006. - No. 5. - S. 7-10.
- [53] Папаян, А.В. Клиническая нефрология детского возраста: руководство для врачей / А.В. Папаян, Н.Д. Савенкова. - СПб., 2008. - 600 с./ Papayan, A.V. *Klinicheskaya nephrologiya detskogo vozrasta: rukovodstvo dlya vrachey* / A.V. Papayan, N.D. Savenkova. - SPb., 2008. - 600 p.
- [54] Марталог П.Н., Пырцу Л.Я., Ченуша Ф.В., Романчук Л.В., Ротарь А.В. Факторы риска и течение гломерулонефрита у детей. *Российский педиатрический журнал.* 2022;3(1):193./ Martalog P.N., Pyrtsu L.Ya., Chenusha F.V., Pomanchuk L.V., Rotar A.V. Faktory riska i techenie glomerulonephrita u detey. *Russian pediatric journal.* 2022;3(1):193.
- [55] Краснова, Т.Н. Особенности течения и лечения нефротического синдрома у больных гломерулонефритами. - М.: NOVARTIS PHARMA SERVICES, 2003.-40 с./ Krasnova, T.N. Osobennosti techenia i lecheniya nefroticheskogo syndroma u bolnykh glomerulonephritami. - M.: NOVARTIS PHARMA SERVICES, 2003.-40 p.
- [56] Длин, В.В. Роль вирусной инфекции в этиологии и патогенезе гломерулонефрита у детей / В.В. Длин, О.Б. Чумакова // *Современные методы диагностики и лечения в детской нефрологии и урологии: матер. II Рос. конгр.* - М, 2002. - С. 12-19./ Dlin, V.V. The role of viral infection and etiology in the pathogenesis of glomerulonephritis and detey / V.V. Dlin, O.B. Chumakova // *Sovremennye metody diagnostici i lecheniya v detskoj nephrologii i urologii: mater. II Ros. Congr.* - M, 2002. - S. 12-19.
- [57] Karzakova L.M., Avtonomova O.I., Stepanova I.M., Komeliagina N.A., Kudriashov S.I. The characteristics of cytokine status under different clinical variants of glomerulonephritis. *Cheboksary, Russia. Clinical laboratory diagnostics.* 2015; 60(6): 33–36.
- [58] Жизневская И.И., Хмелевская И.Г. Особенности цитокинового профиля при гломерулопатиях у детей *Курский научно-практический вестник "Человек и его здоровье"*, 2013;(1):62-66./ Zhiznevskaya I.I., Khmelevskaya I.G. Osobennosti cytokinovogo profilya pri glomerulopatiyax u detey *Kursky nauchno-praktichesky vestnik "Chelovek i ego zdorove"*, 2013;(1):62-66.
- [59] Игнатова М.С. Актуальные проблемы в нефрологии детского возраста в начале XXI века // *Педиатрия.* - 2007. - № 6. - С. 6-13./ Ignatova M.S. Actual problems in nephrology of children's age and early 21st century // *Pediatriya.* - 2007. - No. 6. - S. 6-13.
- [60] Игнатова М.С. Проблема прогрессирования болезней почек у детей и современные возможности ренопротекции // *Нефрология и диализ.* - 2005. - Т. 7, № 4. - С. 428-434./ Ignatova M.S. Problema progression of bolezney почек u detey i sovremennye vozmojnosti renoproteksii // *Nephrology and dialysis.* - 2005. - Т. 7, No. 4. - S. 428-434.
- [61] Hahn W.H., Cho B.S., Kim S.K. et al. Interleukin-1 cluster gene polymorphism in childhood IgA nephropathy // *Pediatr Nephrol.* - 2009. - N 24(7). - P. 1329 - 1336.
- [62] Anthony BF, Kaplan EL, Wannamaker LW, Briese FW, Chapman SS. Attack rates of

acute nephritis after type 49 streptococcal infections of the skin and of the respiratory tract. *J Clin Invest.* 2019;48: 1697-704. DOI: 10.1172/JCI106135.

[63] Муркамилов И.Т., Сабиров И.С., Фомин В.В. и др. Современное состояние этиопатогенетических, морфологических, диагностических и терапевтических аспектов острого гломерулонефрита. *Архивъ внутренней медицины.* 2020; 10(3): 198-208. DOI: 10.20514/2226-6704-2020-10-3-198-208./ Murkamilov I.T., Sabirov I.S., Fomin V.V. i dr. Covremennoe sostoyanie etiopathogeneticheskikh, morfologicheskikh, diagnosticheskikh i terapevticheskikh aspektov ostrogo glomerulonephrita. *Archives of internal medicine.* 2020; 10(3): 198-208. DOI: 10.20514/2226-6704-2020-10-3-198-208.

[64] Shulutko B.I., Makarenko S.V. Acute glomerulonephritis, and not only in the xxi century. *Nephrology (Saint-Petersburg).* 2015;19(6):14-9.

[65] Kupin W.L. Viral-Associated GN: Hepatitis B and other viral infections. *Clin J Am Soc Nephrol.* 2017;12(9):1529-33. doi: 10.2215/CJN.09180816.

[66] Prasad N., Novak J.E., Patel M.R. Kidney diseases associated with parvovirus B19, hanta, Ebola, and dengue virus infection: a brief review. *Adv Chronic Kidney Dis.* 2019;26(3):207-19. doi: 10.1053/j.ackd.2019.01.006.

[67] Ю.А. Гылыджов. (2025). Острый гломерулонефрит у детей. Образование и наука в XXI веке, 66-2 (том 2). <https://mpcareer-google.ru/index.php/journal/article/view/316/>. Yu.A. Glydjov. (2025). Ostryy glomerulonephritis u detey. *Obrazovanie i nauka v XXI veke, 66-2 (volume 2).* <https://mpcareer-google.ru/index.php/journal/article/view/316/>.

[68] Floege J., Barbour S.J., Cattran D.C. et al. Management and treatment of glomerular diseases (part 1): conclusions from a Kidney Disease: Improving Global Outcomes (KDIGO) Controversies Conference. *Kidney international.* 2019;95(2):268-80. doi: 10.1016/j.kint.2018.10.018.

[69] Wetmore J.B., Guo H., Liu J. et al. The incidence, prevalence, and outcomes of glomerulonephritis derived from a large retrospective analysis. *Kidney international.* 2016; 90(4):853-60. doi: 10.1016/j.kint.2016.04.026

[70] Исламов Т.Ш., Ахмеджанов И.А., Ахмеджанова Н.И., Хамидова М.Ф. Клинико-лабораторная характеристика различных форм острого гломерулонефрита у детей. *Журнал репродуктивного здоровья и уро-нефрологических исследований.* 2022. № 2. 29-34./ Islamov T.Sh., Ahmedzhanov I.A., Ahmedzhanova N.I., Khamidova M.F. Clinical and laboratory characteristics of different forms of acute glomerulonephritis. *Journal of reproductive health and uro-nephrological studies.* 2022. No. 2. 29-34.

[71] Имаева Л.Р., Ахметшин Р.З., Шагарова С.В., Ширяева Г.П. Клинический случай острого постинфекционного гломерулонефрита у ребенка. *Инновационная наука.* 2020;12: 140-142. ISSN 2410-6070. / Imaeva L.R., Akhmetshin R.Z., Shagarova S.V., Shiryaeva G.P. Clinical case of ostrogo postinfectious glomerulonephritis in a child. *Innovative science.* 2020;12: 140-142. ISSN 2410-6070.

[72] Boltaboyeva M.M. Bolalarda o'tkir va surunkali glomerulonefritlar: O'quv qo'llanma/«JAHONA NASHR» nashriyoti, 2025 -96 b./ Boltaboyeva M.M. Acute and chronic glomerulonephritis in children: Textbook/"JAHONA NASHR" publishing house, 2025 -96 p.

[73] Факторы риска и течение гломерулонефрита у детей. Марталог П.Н., Пырцу Л.Я., Ченуша Ф.В., Романчук Л.В., Ротарь А.В. *Российский педиатрический журнал.* 2022;3(1):193./ Faktory riska i techenie glomerulonephrita u detey. Martalog P.N., Pyrtsu L.Ya., Chenusha F.V., Pomanchuk L.V., Rotar A.V. *Russian pediatric journal.* 2022;3(1):193.

[74] Shilov E.M., Kozlovskaya N.L., Korotchaeva J.V. Clinical guidelines for diagnosis and treatment of rapidly progressive glomerulonephritis (extracapillary glomerulonephritis with crescent formation). *Nephrology (Saint-Petersburg).* 2015; 19(6): 73-82. [in Russian].

[75] Sievers L.K., Eckardt K.U. Molecular mechanisms of kidney injury and repair in arterial hypertension. *International journal of molecular sciences.* 2019;20(9):2138. doi: 10.3390/ijms20092138.