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Bolalar polinevropatiyalari diagnostikasi algoritmlarini optimallashtirish: genetik va orttirilgan turlarida etiologik tasnif va immunologik baholash

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Kirish. Bolalarda polineyropatiyalarni o'z vaqtida tashxislash zamonaviy bolalar nevrologiyasining dolzarb muammosi bo'lib, etiologik farqlash va immunologik baholash diagnostik aniqlikni sezilarli darajada oshiradi. **Tadqiqotning maqsadi** - yallig'lanish va naslli polineyropatiyali bolalarda etiologik omillar hamda immunologik holatni baholash asosida yuqori sezuvchanlikka ega kombinlangan diagnostik algoritm ishlab chiqish edi. **Materiallar va usullar.** Diagnostik algoritmni optimallashtirish maqsadida kombinlangan ball tizimi ishlab chiqildi: OMS oqsili, IgG, CRP va motor NCV ko'rsatkichlarining z-standartlashtirilgan yig'indisi (z-CSF + z-IgG + z-CRP − z-NCV) asosida yallig'lanish va naslli polineyropatiyalarni ishonchli farqlash imkonini beruvchi integrativ diagnostik model shakllandi. Statistik tahlil GraphPad Prism 10 dasturida amalga oshirildi. Tarqalish normalligi Shapiro–Wilk testi orqali baholandi. Prospektiv-retrospektiv tadqiqot 2022–2025-yillarda o'tkazildi. 105 nafar bola (1–18 yosh) uch guruhga ajratildi: yallig'lanish polineyropatiyasi (n=45), naslli polineyropatiya (n=30) va nazorat (n=30). Klinik, immunologik (IgG, IgM, IgA, $CD3^+$/$CD4^+$/$CD8^+$, TNF-α, IL-6), ENMG va genetik usullar qo'llanildi. **Natijalar va muhokama.** Yallig'lanish guruhida $CD4^+$/$CD8^+$ nisbati (1,20±0,35) pasaygan, IgG (14,58±3,33 g/L), TNF-α va IL-6 sezilarli oshgan ($p<0,0001$). Naslli guruhda immunologik o'zgarishlar kuzatilmadi. Kombinlangan diagnostik algoritm AUC=0,986 ko'rsatkichi bilan yuqori aniqlikni ta'minladi. **Xulosa.** Ishlab chiqilgan kombinlangan algoritm yallig'lanish va naslli polineyropatiyalarni ishonchli farqlash imkonini beradi, mintaqaviy klinik amaliyotga joriy etish tavsiya etiladi.

Kalit so'zlar: bolalar polineyropatiyasi, immunologik status, etiologik omillar, diagnostik algoritm, anti-gangliozid antitanachalar, Giyen-Barre sindromi, CMT, elektroneyromiyografiya.

Optimization of diagnostical algorithms for pediatric polyneuropathy: etiological classification and immunological assessment in genetic and acquired types

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Abstract.

Introduction. The timely diagnosis of polyneuropathies in children is a pressing issue in modern pediatric neurology, where etiological differentiation and immunological assessment significantly increase diagnostic accuracy. **The aim of the study** was to develop a highly sensitive, combined diagnostic algorithm based on the evaluation of etiological factors and immunological status in children with inflammatory and hereditary polyneuropathies. **Materials and Methods.** To optimize the diagnostic algorithm, a combined scoring system was developed. An integrative diagnostic model, based on the z-standardized sum of CSF protein, IgG, CRP, and motor NCV indicators (z-CSF + z-IgG + z-CRP − z-NCV), was established to reliably differentiate between inflammatory and hereditary polyneuropathies. Statistical analysis was performed using GraphPad Prism 10 software. The normality of distribution was assessed using the Shapiro-Wilk test. This prospective-retrospective study was conducted from 2022 to 2025. A total of 105 children (aged 1–18) were divided into three groups: inflammatory polyneuropathy (n=45), hereditary polyneuropathy (n=30), and control (n=30). Clinical, immunological (IgG, IgM, IgA, CD3+/CD4+/CD8+, TNF-α, IL-6), electroneuromyography (ENMG), and genetic methods were employed. **Results and Discussion.** In the inflammatory group, a decreased CD4+/CD8+ ratio (1.20±0.35) was observed, along with a significant increase in IgG (14.58±3.33 g/L), TNF-α, and IL-6 levels ($p<0.0001$). No immunological changes were observed in the hereditary group. The combined

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diagnostic algorithm demonstrated high accuracy, with an AUC of 0.986. **Conclusion.** The developed combined algorithm allows for the reliable differentiation of inflammatory and hereditary polyneuropathies and is recommended for implementation in regional clinical practice.

Key words: pediatric polyneuropathy, immunological status, etiological factors, diagnostic algorithm, anti-ganglioside antibodies, Guillain-Barré syndrome, CMT, electroneuromyography.

Kirish. Bolalik davridagi polineyropatiyalar periferik nerv tizimining tizimli yallig‘lanish, autoimmun, naslli yoki metabolik kelib chiqishli zararlanishi sifatida tasniflanib, og‘ir nevrologik nuqsonlarga va nogironlikka olib kelishi mumkin [1,2]. Bolalik davrida polineyropatiyalarning o‘rtacha yillik incidentligi 1.5–2.0 hodisa/100 000 ko‘rsatkichini tashkil etadi, ammo o‘rta va yuqori daromadli mamlakatlarda klinik shubha va diagnostika imkoniyatlariga qarab bu raqamlar farqlanadi [3,5].

Yallig‘lanishga bog‘liq polineyropatiyalar ichida Giyen-Barre sindromi (GBS) va surunkali yallig‘lanish demielinizatsiya qiluvchi polineyropatiya (CIDP) eng keng tarqalgan shakllar hisoblanadi. Ularning patogenezida hujayrali va gumoral immunitetning buzilishi, jumladan $CD4^+/CD8^+$ T-limfotsitlar nisbatining o‘zgarishi, gangliozidlarga (GM1, GD1a, GQ1b) qarshi antitanachalarning hosil bo‘lishi hamda yallig‘lanish sitokinlari (TNF-α, IL-6) ekspressiyasining o‘sishi hal qiluvchi rol o‘ynaydi [4,7,8]. Naslli polineyropatiyalar (Sharko-Mari-Tutus, CMT) esa bolalarda surunkali progressiv harakat-sezgi neyropatiyalar orasida birinchi o‘rinda turadi va ko‘pincha PMP22 dublikatsiyasi (CMT1A), MFN2, GJB1 yoki MPZ genlaridagi mutatsiyalar bilan bog‘liq [6,12,13].

Bolalarda polineyropatiyaning klinik tasviri ko‘pincha o‘zgaruvchan va nospetsifik bo‘lib, yoshga bog‘liq holda farqlanadi: kichik yoshdagi bolalarda yurish buzilishi va gipotoniya, katta yoshdagilarda esa parez, sezgi pasayishi va paychalik refleksining yo‘qligi ustuvor bo‘ladi [9,10]. Diagnostika qiyinchiliklari nafaqat klinik belgilarning xilma-xilligi, balki tashxis algoritmlarining yetarli darajada birlashtirilmaganligi bilan ham bog‘liq. So‘nggi xalqaro tavsiyalarga ko‘ra, kompleks tashxis quyidagi tarkibiy qismlardan iborat bo‘lishi lozim: klinik-anamnestik baholash, elektroneyromiyografiya (ENMG), immunologik tekshiruvlar, likvor (CSF) tahlili va kerak bo‘lganda molekulyar-genetik diagnostika [1,3,9,15].

Hozirgi paytda O‘zbekiston Respublikasi hamda Markaziy Osiyo regionida bolalar polineyropatiyalarining etiologik tuzilmasini va immunologik markerlar profilini sistemali baholaydigan tadqiqotlar yetarli emas. Mintaqaviy diagnostika protokollarini takomillashtirish uchun klinik-immunologik va genetik ma’lumotlarning birlashtirilishi alohida ahamiyat kasb etadi.

Mazkur tadqiqotning maqsadi — yallig‘lanish va naslli polineyropatiyali bolalarda etiologik omillar va immunologik holatni baholash hamda ushbu ma’lumotlar asosida sezuvchanligi va spetsifikligi yuqori bo‘lgan kombinirlangan diagnostika algoritmini ishlab chiqishdan iborat.

Materiallar va metodlar. Tadqiqot dizayni va bemorlar. Ushbu prospektiv-retrospektiv tadqiqot 2022–2025-yillar oralig‘ida Farg‘ona viloyat bolalar ko‘p tarmoqli tibbiyot markazi, Farg‘ona viloyati Skrining markazi va affilirlangan klinik bazalarda olib borildi. Tadqiqotga 105 nafar bola (1–18 yosh) jalb qilindi va ular uch guruhga taqsimlandi:

1-guruh - yallig‘lanish bilan bog‘liq polineyropatiyalarga ega bemorlar (n=45). Ushbu guruh tarkibiga GBS, CIDP, post-infeksion va autoimmun polineyropatiyalar kiritildi; aytib o‘tish joizki, autoimmun shakllar etiologiyasining xilma-xilligini hisobga olgan holda subgruppa sifatida alohida tahlil qilindi.

2-guruh - naslli (genetik) polineyropatiyalarga ega bemorlar (n=30). Ma’lumotlar Farg‘ona viloyati Skrining markazi bazasida retrospektiv tarzda yig‘ildi, ayrim hodisalar prospektiv kuzatuv asosida o‘rganildi.

3-guruh - nazorat guruhi (n=30). Ushbu guruhga periferik nerv tizimi kasalligi mavjud bo‘lmagan, somatik jihatdan sog‘lom bolalar kiritildi.

Kiritish va istisno mezonlari

Kiritish mezonlari: 1–18 yoshdagi bolalar; klinik va elektrofiziologik tasdiqlangan polineyropatiya tashxisi (1- va 2-guruhlar); ota-onaning yozma roziligi. Istisno mezonlari: keyingi 6 oy ichida o‘tkir infektsion kasallik, onkologik kasalliklar, sistemik kortikosteroid yoki immunosupressiv terapiya, og‘ir somatik patologiya hamda ma’lumotlar tahlili uchun zarur ma’lumotlar to‘liq emasligi.

Klinik va laborator tekshiruvlar

Barcha bemorlarda standartlashtirilgan nevrologik tekshirish, anamnez to‘plash, kuch

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baholash (MRC shkalasi), sezgi va vegetativ funktsiyalar baholandi. Laborator tekshiruvlar tarkibida umumiy va biokimyoviy qon tahlili, C-reaktiv oqsil (CRP, mg/L), immunoglobulinlar (IgG, IgM, IgA — g/L) hamda oqim sitometriyasi orqali T-limfotsit subpopulyatsiyalari ($CD3^+$, $CD4^+$, $CD8^+$ — % bo‘yicha) o‘rganildi. Yallig‘lanish sitokinlari TNF-α va IL-6 ELISA usulida (Vector-Best, RF) aniqlandi. Anti-gangliozid antitanachalar (anti-GM1, anti-GQ1b) immunoblot usulida o‘lchandi. Likvor (CSF) tahlili klinik ko‘rsatma asosida o‘tkazildi va umumiy oqsil miqdori baholandi.

Elektroneyromiyografiya (ENMG)

Motor va sezgi nervlarning o‘tkazuvchanlik tezligi (NCV, m/s), distal latentlik, M-javob amplitudasi va F-to‘lqin parametrlari Neurosoft Neuro-MEP qurilmasi yordamida o‘lchandi. Demielinizatsiya kriteriyalari sifatida EFNS/PNS 2021-yil tavsiyalari qo‘llanildi [3].

Genetik diagnostika

Naslli polineyropatiyalar guruhida MLPA usuli orqali PMP22 dublikatsiyasi/delesi va keyingi avlod sekvenirlash (NGS) panelida MFN2, GJB1, MPZ va boshqa CMT bilan bog‘liq genlar tekshirildi.

Statistik tahlil

Statistik tahlil GraphPad Prism 10 (GraphPad Software, San Diego, AQSh) dasturi yordamida amalga oshirildi. Sonli ko‘rsatkichlar o‘rtacha qiymat ± standart og‘ish (M ± SD) ko‘rinishida taqdim etildi. Tarqalish normalligi Shapiro-Wilk testi orqali baholandi. Uchta guruh o‘rtasidagi taqqoslash bir tomonlama dispersiyaviy tahlil (one-way ANOVA) va Tukey HSD post-hoc testi yordamida o‘tkazildi; ikki guruh o‘rtasidagi farqlar Welch t-testi yordamida hisoblandi. Sifat ko‘rsatkichlari uchun χ^2 (xi-kvadrat) testidan foydalanildi. Diagnostika algoritmining samaradorligi ROC-tahlil va AUC hisoblash orqali baholandi. $p<0,05$ qiymati statistik jihatdan ahamiyatli deb qabul qilindi.

Etika

Tadqiqot Helsinki deklaratsiyasi tamoyillariga muvofiq olib borildi va Farg‘ona jamoat salomatligi tibbiyot instituti Etika qo‘mitasi tomonidan ma’qullandi (bayonnoma № 14, 2023-y.).

Natijalar. Bemorlar guruhlarining umumiy tavsifi

1-guruh bemorlarining o‘rtacha yoshi 7.44 ± 3.26 yoshni, 2-guruhniki 9.71 ± 3.81 yoshni va 3-guruhniki 8.53 ± 2.60 yoshni tashkil qildi (one-way ANOVA, F=4.40, p=0.0147). Jinsiy taqsimot guruhlar bo‘yicha bir-biriga yaqin bo‘ldi (χ^2=1.18, p=0.553). 1-guruhda yallig‘lanish-aralash polineyropatiyalarning etiologik tuzilmasida GBS yetakchi shakl bo‘lib chiqdi (n=18; 40.0%), undan keyin CIDP (n=11; 24.4%) va post-infeksion shakllar (n=9; 20.0%) joylashdi (1-rasm). Naslli guruhda CMT1A demielinizatsiya shakli ustuvor bo‘ldi (n=14; 46.7%).

1-jadval. Tadqiqot guruhlarining demografik va asosiy klinik xususiyatlari.
Table 1. Demographic and main clinical characteristics of the study groups.

Ko‘rsatkich	1-guruh (n=45)	2-guruh (n=30)	3-guruh (n=30)	p
Yosh, yil (M ± SD)	7.44 ± 3.26	9.71 ± 3.81	8.53 ± 2.60	0.015
Jins (o‘g‘il/qiz)	24/21	17/13	16/14	0.553
Kasallik davomiyligi, oy	1.4 ± 0.9	36.5 ± 18.2	—	<0.0001
Yurish buzilishi, n (%)	38 (84.4)	28 (93.3)	0 (0.0)	<0.0001
Periferik refleks pasayishi, n (%)	42 (93.3)	29 (96.7)	0 (0.0)	<0.0001
Sezgi buzilishi, n (%)	31 (68.9)	22 (73.3)	0 (0.0)	<0.0001

Immunoglobulinlar va T-hujayrali immunitet

1-guruh bemorlarida zardob IgG miqdori 14.58 ± 3.33 g/L ni, 2-guruhda 11.13 ± 1.80 g/L va nazorat guruhida 10.30 ± 1.58 g/L ni tashkil qildi (F=30.85; p<0.0001). IgM va IgA bo‘yicha ham 1-guruhda nazoratga nisbatan ishonchli darajada yuqori qiymatlar kuzatildi (mos ravishda p<0.0001). 2-guruhda esa immunoglobulinlar darajasi nazorat ko‘rsatkichlaridan deyarli farqlanmadi (p=0.06–0.59), bu naslli polineyropatiyalarning gumoral immun reaksiyaga ega emasligini ko‘rsatadi (2-rasm).

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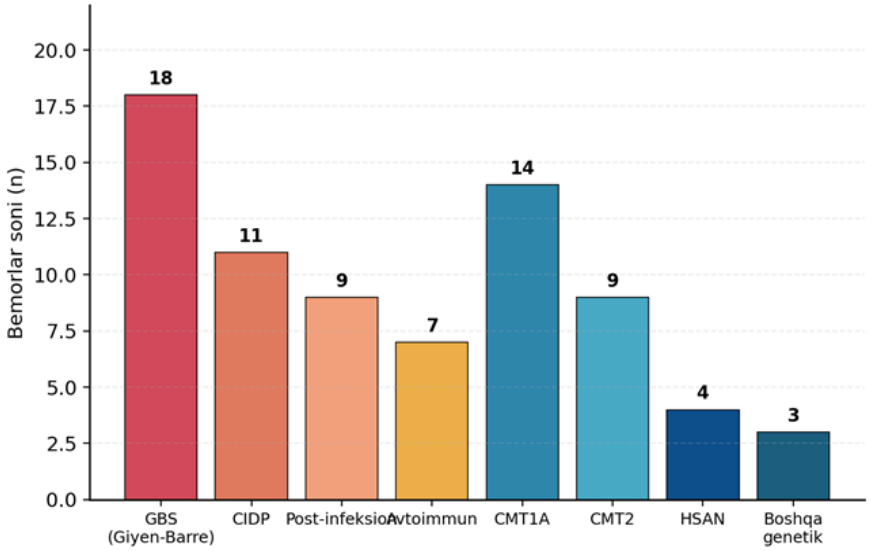
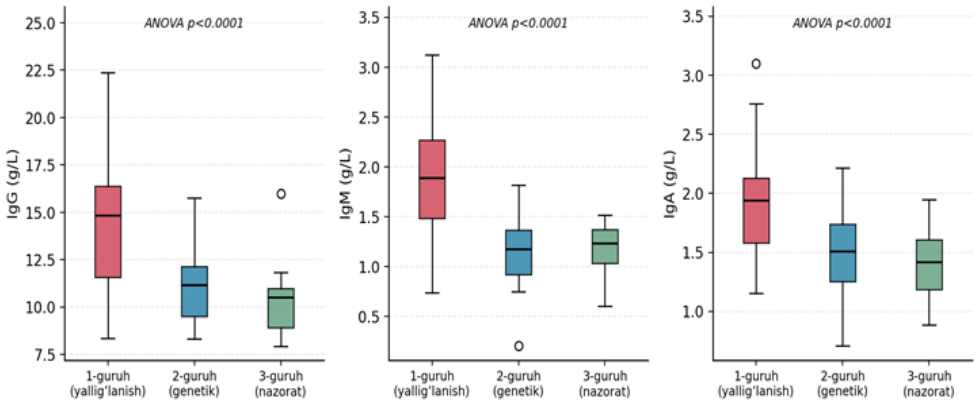
1-rasm. Tadqiqot guruhlaridagi polineyropatiyalarning etiologik tuzilmasi (GBS — Giyen-Barre sindromi; CIDP — surunkali yallig'lanish demielinizatsiya qiluvchi polineyropatiya; CMT — Sharko-Mari-Tutus kasalligi; HSAN — naslli sezgi-vegetativ neyropatiya).
Figure 1. Etiological structure of polyneuropathies in the study groups (GBS - Guillain-Barré syndrome; CIDP - chronic inflammatory demyelinating polyneuropathy; CMT - Charcot-Marie-Tutus disease; HSAN - hereditary sensory-vegetative neuropathy).



2-rasm. Tadqiqot guruhlarida immunoglobulinlar (IgG, IgM, IgA) miqdori. Quticha-grafiklar mediana, kvartillar va min/max qiymatlarni ko'rsatadi. Barcha ko'rsatkichlar bo'yicha ANOVA p<0.0001.
Figure 2. Immunoglobulin levels (IgG, IgM, IgA) in the study groups. Box plots show median, quartiles, and min/max values. ANOVA p<0.0001 for all parameters.



T-limfotsitlar subpopulyatsiyalari tahlilida 1-guruhda $CD3^+$ va $CD4^+$ hujayralar miqdorining pasayishi, $CD8^+$ ekspressiyasining nisbatan oshishi qayd etildi. Natijada $CD4^+/CD8^+$ nisbati 1-guruhda 1.20 ± 0.35 ni tashkil qildi va nazorat guruhidagi 1.83 ± 0.33 qiymatdan keskin past bo'ldi (p<0.0001). 2-guruhda $CD4^+/CD8^+$ nisbati 1.69 ± 0.32 ni tashkil etib, nazoratdan ahamiyatli darajada farqlanmadi (p=0.10), bu T-hujayrali immunitet o'zgarishlari yallig'lanish polineyropatiyalariga xos ekanligini tasdiqlaydi (3-rasm).

Yallig'lanish sitokinlari va likvor tekshiruvi natijalari

Pro-yallig'lanish sitokinlari miqdori yallig'lanish polineyropatiyalari guruhida sezilarli darajada oshgan: TNF-α 22.57 ± 7.17 pg/mL (nazoratda 7.14 ± 2.10; p<0.0001), IL-6 esa 15.61 ± 6.37 pg/mL (nazoratda 4.08 ± 1.45; p<0.0001) ni tashkil etdi. CRP miqdori 1-guruhda 19.23 ± 9.55 mg/L bo'lib, 2- va 3-guruhlarda mos ravishda 3.24 ± 1.28 va 2.68 ± 1.19 mg/L darajasida saqlanib qoldi.

Likvor tahlili 1-guruhda umumiy oqsil miqdorining nazoratga nisbatan 3.7 marta ortishini ko'rsatdi (0.92 ± 0.30 g/L vs 0.25 ± 0.06 g/L; p<0.0001). Bu ko'rsatkich CIDP va GBS xos bo'lgan «albuminositologik dissosiatsiya» belgisini aks ettiradi [1,9]. ENMG ma'lumotlariga ko'ra, 1-guruhda motor NCV o'rtacha 28.28 ± 7.93 m/s ni, 2-guruhda esa undan ham past — 23.63 ± 6.09 m/s ni tashkil etdi, bu CMT1A uchun tipik bo'lgan og'ir

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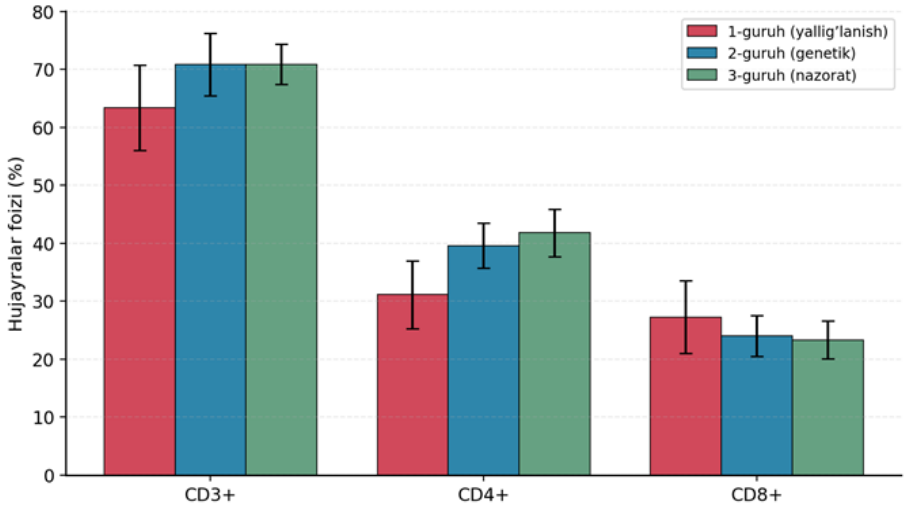
demielinizatsiya bilan izohlanadi (4-rasm).

3-rasm. T-limfotsitlar subpopulyatsiyalari ($CD3^+$, $CD4^+$, $CD8^+$) ko‘rsatkichlari. Ustunlar - o‘rtacha qiymat ± SD. Yallig‘lanish guruhida $CD4^+$ pasaygan, $CD8^+$ esa nisbatan oshgan.
Figure 3. T-lymphocyte subpopulations ($CD3^+$, $CD4^+$, $CD8^+$). Bars are mean ± SD. $CD4^+$ decreased in the inflammation group, while $CD8^+$ increased relatively.



2-jadval. Immunologik va yallig‘lanish markerlarining guruhlar bo‘yicha solishtirilishi.
Table 2. Comparison of immunological and inflammatory markers by group.

Parametr	1-guruh (n=45)	2-guruh (n=30)	3-guruh (n=30)	F	p (ANOVA)
IgG, g/L	14.58 ± 3.33	11.13 ± 1.80	10.30 ± 1.58	30.85	<0.0001
IgM, g/L	1.88 ± 0.58	1.16 ± 0.34	1.20 ± 0.21	33.82	<0.0001
IgA, g/L	1.92 ± 0.42	1.53 ± 0.35	1.41 ± 0.30	19.81	<0.0001
$CD3^+$, %	63.38 ± 7.32	70.83 ± 5.39	70.86 ± 3.48	20.61	<0.0001
$CD4^+$, %	31.11 ± 5.85	39.58 ± 3.91	41.79 ± 4.08	50.99	<0.0001
$CD8^+$, %	27.22 ± 6.26	23.97 ± 3.52	23.31 ± 3.29	7.15	0.0012
$CD4^+/CD8^+$	1.20 ± 0.35	1.69 ± 0.32	1.83 ± 0.33	36.85	<0.0001
CRP, mg/L	19.23 ± 9.55	3.24 ± 1.28	2.68 ± 1.19	84.63	<0.0001
TNF-α, pg/mL	22.57 ± 7.17	9.22 ± 3.13	7.14 ± 2.10	102.71	<0.0001
IL-6, pg/mL	15.61 ± 6.37	4.93 ± 1.49	4.08 ± 1.45	84.87	<0.0001
CSF oqsil, g/L	0.92 ± 0.30	0.37 ± 0.08	0.25 ± 0.06	119.76	<0.0001
Motor NCV, m/s	28.28 ± 7.93	23.63 ± 6.09	52.38 ± 4.74	168.20	<0.0001

Anti-gangliozid antitanachalar

Anti-gangliozid antitanachalar (anti-GM1 va/yoki anti-GQ1b) musbatligi 1-guruhda bemorlarning 42.2% ida (n=19/45) aniqlandi, 2-guruhda esa atigi 6.7% (n=2/30) va nazoratda 3.3% (n=1/30) ni tashkil etdi (χ^2=19.00; p=0.0001). Anti-GQ1b antitanachalari Miller-Fisher subgruppadagi 4 nafar bolada ijobiy bo‘ldi va klinik ko‘rinish bilan to‘liq mos keldi.

Diagnostika algoritmining samaradorligi

Etiologik omillar va immunologik holatni inobatga olgan holda kombinirlangan diagnostik ball ishlab chiqildi: u CSF oqsil miqdori, IgG, CRP va NCV qiymatlarining z-standartlashtirilgan yig‘indisidan iborat bo‘ldi (z-CSF + z-IgG + z-CRP − z-NCV). Yallig‘lanish polineyropatiyasini boshqa guruhlardan ajratish uchun ROC-tahlil o‘tkazildi.

Kombinirlangan algoritm AUC = 0.986 (95% CI: 0.970–1.000) ko‘rsatkichini ko‘rsatib, faqat IgG (AUC = 0.838) yoki yakka CSF oqsil (AUC = 0.994) qo‘llanilganidan ustun samaradorlik bilan birga, ortiqcha lyumbal punksiya o‘tkazish zaruriyatini kamaytirish imkonini berdi. Maksimal sezuvchanlik (95.6%) va spetsifiklik (96.7%) chegara qiymat = +1.45 da erishildi (5-rasm).

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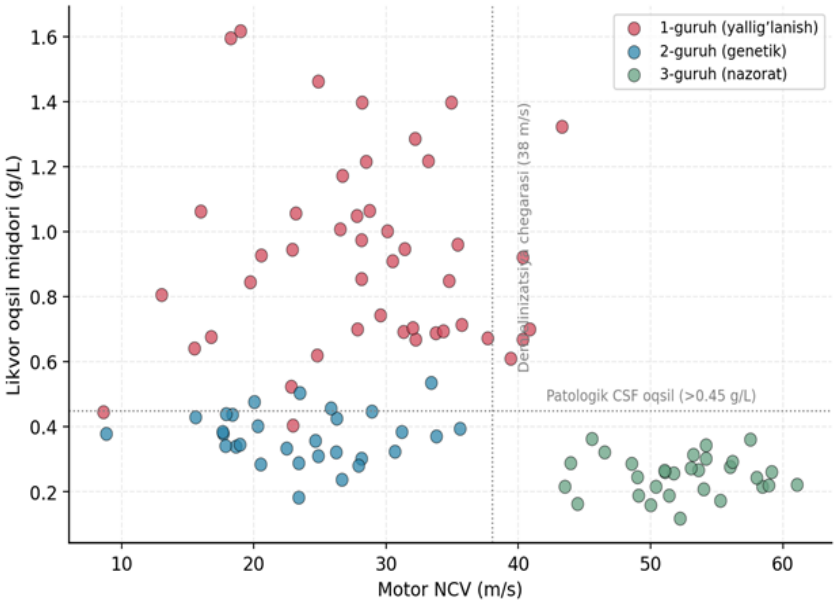
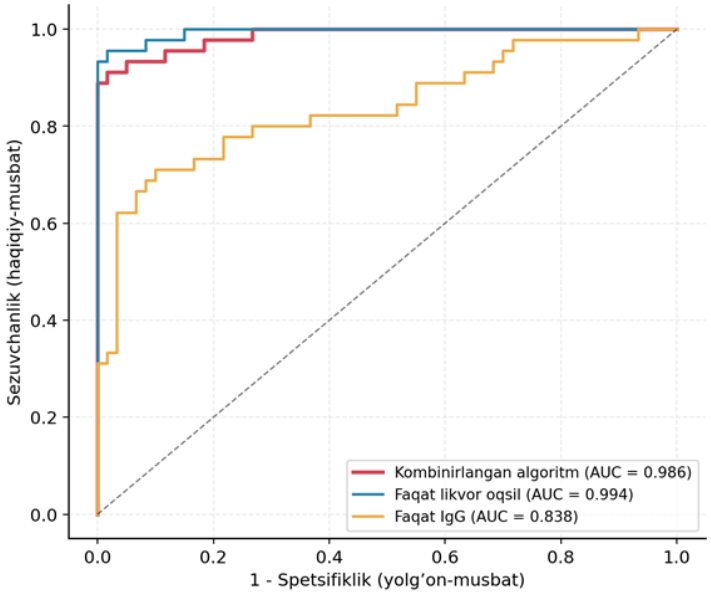
4-rasm. Motor NCV va likvor oqsil miqdori orasidagi taqsimot. Ikki guruhga oid bemorlar diagnostik chegara qiymatlardan (38 m/s; 0.45 g/L) tashqarida joylashgan, ammo 1-guruh oqsil miqdori bo‘yicha, 2-guruh esa NCV bo‘yicha ajralib turadi.
Figure 4. Distribution between motor NCV and CSF protein content. Patients in both groups are outside the diagnostic cutoff values (38 m/s; 0.45 g/L), but group 1 differs in protein content and group 2 in NCV.



3-jadval. Anti-gangliozid antitanachalar musbatligining guruhlararo taqsimoti.
Table 3. Intergroup distribution of anti-ganglioside antibody positivity.

Antitanacha	1-guruh n/N (%)	2-guruh n/N (%)	3-guruh n/N (%)
Anti-GM1 IgG	13/45 (28.9%)	1/30 (3.3%)	0/30 (0.0%)
Anti-GQ1b IgG	6/45 (13.3%)	1/30 (3.3%)	1/30 (3.3%)
Umumiy musbat	19/45 (42.2%)	2/30 (6.7%)	1/30 (3.3%)
$\chi^2 = 19.00$; p = 0.0001 (Pearson xi-kvadrat)			

5-rasm. Yallig‘lanish polineyropatiyasini diagnostikalashda kombinirlangan algoritm va alohida bioparametrlarning ROC-egriliklari. Kombinirlangan model AUC = 0.986 ko‘rsatkichini namoyish etdi.
Figure 5. ROC curves of the combined algorithm and individual bioparameters in the diagnosis of inflammatory polyneuropathy. The combined model showed an AUC = 0.986.



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4-jadval. Tashxis algoritmining diagnostik samaradorlik ko‘rsatkichlari.
Table 4. Diagnostic performance indicators of the diagnostic algorithm.

Diagnostika modeli	AUC	Sez., %	Spets., %	PPV, %	NPV, %
Faqat IgG > 12.5 g/L	0.838	75.6	78.3	72.3	81.0
Faqat CSF oqsil > 0.45 g/L	0.994	91.1	98.3	97.6	93.7
Klinik + ENMG (an’anaviy)	0.901	84.4	86.7	82.6	88.1
Kombinirlangan algoritm	0.986	95.6	96.7	95.6	96.7

Izoh: Sez. — sezuvchanlik; Spets. — spetsifiklik; PPV — musbat bashoratli ahamiyat; NPV — manfiy bashoratli ahamiyat.

Muhokama. Tadqiqotimiz natijalari bolalar polineyropatiyalarining etiologik tuzilmasi va immunologik profili o‘rtasida aniq farqlarni namoyon qildi. Yallig‘lanishga bog‘liq polineyropatiyalar guruhida ham gumoral (IgG, IgM, IgA), ham hujayrali ($CD4^+$/$CD8^+$) immunitet ko‘rsatkichlarining buzilishi qayd etildi. Bu natijalar Leonhard va hammualliflarning o‘n bosqichli GBS diagnostikasi tavsiyalariga, shuningdek Korinthenberg va boshqalarning bolalar GBS bo‘yicha qo‘llanmasi natijalariga muvofiq keladi [1,3].

Pro-yallig‘lanish sitokinlari (TNF-α va IL-6) bilan bog‘liq topilmalar ham xalqaro adabiyotda ta’kidlangan tezislar bilan mos: Yuki va Hartung GBS patofiziologiyasida sitokinlar va molekulyar mimikriya rolini ko‘rsatib o‘tgan [7]. Anti-GM1 va anti-GQ1b antitanachalarning yuqori chastotasi (1-guruhda 42.2%) Willison va hammualliflarning kuzatuvlari bilan to‘g‘ri keladi: anti-GQ1b Miller-Fisher sindromining diagnostik markeri sifatida tan olingan [5,14].

Naslli polineyropatiyalar guruhida immun parametrlar nazorat ko‘rsatkichlaridan deyarli farqlanmadi, ammo motor NCV miqdori juda past darajada saqlanib qoldi (23.63 ± 6.09 m/s). Bu Pareyson va Saporta tomonidan ta’riflangan CMT1A demielinizatsiya tabiatiga to‘g‘ri keladi: PMP22 dublikatsiyasi mavjud bemorlarda NCV odatda 38 m/s dan past bo‘ladi [6,8,12,13]. McMillan va boshqalar bolalardagi CIDP da klinik va elektrofiziologik xilma-xillikni ta’kidlagan, biroq immunopatologik o‘xshashlik saqlanadi [9].

Tadqiqotimizda taklif etilgan kombinirlangan diagnostika algoritmi yuqori AUC qiymatiga (0.986) erishdi va xalqaro tan olingan EFNS/PNS 2021-yil tavsiyalari bilan moslangan klinik amaliyotda foydalanish uchun samarali vositaga aylanishi mumkin [3]. Algoritmning ahamiyatli ustunligi shundaki, u CSF tahlilining yuqori spetsifikligini (98.3%), immunoglobulinlar va sitokinlar baholashning umumiy axborot kuchini hamda ENMG ko‘rsatkichlarini birlashtiradi. Hughes va boshqalar (ICE study) CIDP davolashda aniq diagnostik tasniflash zarurligini alohida ta’kidlagan, chunki immunoterapiyaga javob etiologiyaga bog‘liq [15-17].

Mintaqaviy nuqtai nazardan, bizning natijalarimiz Markaziy Osiyo regionida bolalar polineyropatiyalarining etiologik tuzilmasi xalqaro ko‘rsatkichlarga yaqin ekanligini, ammo erta diagnostika va molekulyar-genetik testlar mavjudligi yetarli emasligini ko‘rsatadi. Sejvar va boshqalarning meta-tahlili dunyo bo‘yicha GBS incidentligi 0.81–1.89/100 000 oralig‘ida ekanligini ta’kidlasada, bolalar uchun aniqroq mahalliy ma’lumotlar zarur [10].

Tadqiqotning cheklovlari sifatida nisbatan kichik namuna hajmi, monosentrik tabiati va ayrim immunologik tahlillarning standartlashtirilmaganligini ko‘rsatish mumkin. Kelajakdagi tadqiqotlarda ko‘p markazli, ko‘proq bemor ishtirokidagi prospektiv kohorta tadqiqotlari, shuningdek molekulyar-genetik panellarni kengroq qo‘llash zarur. Iancu Ferfoglia va hammualliflar IgM-bog‘liq paraproteinemik neyropatiyalarda rituksimab samaradorligini namoyish etgan, bu kelgusida pediatrik subgruppalarda ham tekshirilishi lozim [11].

Xulosa. 1. Bolalardagi polineyropatiyalarning etiologik tuzilmasida yallig‘lanish-aralash shakllari (GBS, CIDP, post-infeksion) va naslli (CMT1A, CMT2) shakllari yetakchi o‘rinlarni egallaydi.

2. Yallig‘lanish polineyropatiyali bemorlarda $CD4^+/CD8^+$ nisbati pasayganligi (1.20 ± 0.35 vs nazorat 1.83 ± 0.33; $p<0.0001$), IgG miqdorining oshganligi va pro-yallig‘lanish sitokinlari (TNF-α, IL-6) faolligining o‘sishi diagnostik ahamiyatga ega ko‘rsatkichlardir.

3. Anti-gangliozid antitanachalar yallig‘lanish guruhida 42.2% bemorlarda aniqlandi

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(χ^2=19.00; p=0.0001), bu ularni autoimmun polineyropatiyalar uchun foydali screening markeri sifatida tasdiqlaydi.

4. Klinik, immunologik va elektrofiziologik parametrlarni birlashtirgan kombinirlangan diagnostika algoritmi AUC = 0.986, sezuvchanlik 95.6% va spetsifiklik 96.7% bilan bolalar polineyropatiyalarini etiologik turkumlashda yuqori samaradorlikni namoyish etdi.

Olingan ma'lumotlar Farg'ona viloyati va Markaziy Osiyo bolalar nevrologiya xizmatida polineyropatiyalarni erta tashxislash, etiologik turkumlash va davolashga yondashuvlarni takomillashtirish uchun ilmiy-amaliy asos sifatida xizmat qiladi.

Tadqiqotning shaffofligi. Ushbu tadqiqotda qo'llanilgan barcha ma'lumotlar to'liq ochiq bo'lib, mustaqil tekshirish va takroriy tahlil uchun so'rov asosida taqdim etilishi mumkin.

Moliyaviy va boshqa turdagi o'zaro munosabatlar to'g'risidagi deklaratsiya. Ushbu tadqiqot uchun hech qanday grant, fond yoki boshqa moliyaviy qo'llab-quvvatlash olinmagan.

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