Impact Factor: 5.723

ISSN: 2181-0982
DOI: 10.26739/2181-0982
www.tadqiqot.uz

JNNR

JOURNAL OF NEUROLOGY AND NEUROSURGERY RESEARCH



Volume 7, Issue 6
2026

ISSN 2181-0982

Doi Journal 10.26739/2181-0982

ЖУРНАЛ НЕВРОЛОГИИ И НЕЙРОХИРУРГИЧЕСКИХ ИССЛЕДОВАНИЙ

ТОМ 7 НОМЕР 6

JOURNAL OF NEUROLOGY AND NEUROSURGERY RESEARCH

VOLUME 7, ISSUE 6

МАҚОЛАДА КЕЛТИРИЛГАН ДАЛИЛЛАРНИНГ ТЎҒРИЛИГИ УЧУН МУАЛЛИФ МАСЪУЛДИР | АВТОР НЕСЕТ ОТВЕТСТВЕННОСТЬ ЗА ДОСТОВЕРНОСТЬ ФАКТОВ ИЗЛОЖЕННЫХ В СТАТЬЕ

ТОШКЕНТ-2026

МИНИСТЕРСТВО ЗДРАВООХРАНЕНИЯ РЕСПУБЛИКИ УЗБЕКИСТАН ОБЩЕСТВЕННОЕ ОБЪЕДИНЕНИЕ НЕВРОЛОГОВ УЗБЕКИСТАНА

ЖУРНАЛ НЕВРОЛОГИИ И НЕЙРОХИРУРГИЧЕСКИХ ИССЛЕДОВАНИЙ

Бухарский государственный медицинский институт и tadqiqot.uz

Главный редактор:

Ходжиева Дилбар Таджиевна
доктор медицинских наук, профессор Бухарского государственного медицинского института. (Узбекистан).
ORCID ID: 0000-0002-5883-9533

Зам. главного редактора:

Хайдарова Дилдора Кадировна
доктор медицинских наук, профессор Ташкентский государственный медицинский университет. (Узбекистан).
ORCID ID: 0000-0002-4980-6158

Рецензируемый научно-практический журнал "Журнал неврологии и нейрохирургических исследований" Публикуется 6 раза в год
№6 (07), 2026
ISSN 2181-0982

Адрес редакции:
ООО Tadqiqot город Ташкент, улица Амира Темура пр.1, дом-2.
web: http://www.tadqiqot.uz/;
Email: info@tadqiqot.uz
Тел: (+998-94) 404-0000

Макет и подготовка к печати проводились в редакции журнала.

Дизайн - оформления:
Хуршид Мирзахмедов

Журнал зарегистрирован в Управлении печати и информации г. Ташкента Рег. № от 01.07.2020 г.

Электронная версия журнала на сайтах:
https://tadqiqot.uz, www.bsmi.uz

- - -

Журнал включен в перечень научных изданий, рекомендованных к публикации основных научных результатов диссертаций по медицинским наукам с 27 сентября 2024 года Высшей аттестационной комиссией Республики Узбекистан (письмо № 361/6 от 2024 года).

РЕДАКЦИОННАЯ КОЛЛЕГИЯ:

Хайдаров Нодиржон Кадирович – доктор медицинских наук, профессор, ректор Ташкентский государственный медицинский университет. (Узбекистан).
Нуралиев Неккадам Абдуллаевич - доктор медицинских наук, профессор, иммунолог, микробиолог, проректор по научной работе и инновациям Бухарского государственного медицинского института. (Узбекистан).
Кариев Гайрат Маратович – доктор медицинских наук, профессор, директор Республиканского научного центра нейрохирургии Узбекистана. (Узбекистан).
Федин Анатолий Иванович - доктор медицинских наук, профессор, заслуженный врач РФ. Российский национальный исследовательский медицинский университет имени Н.И. Пирогова. (Россия).
Маджидова Екутхон Набиевна - доктор медицинских наук, профессор, Ташкентский государственный медицинский университет. (Узбекистан).
Рахимбаева Гулнора Саттаровна - доктор медицинских наук, профессор, Ташкентский государственный медицинский университет. (Узбекистан).
Джурабекова Азиза Тахировна – доктор медицинских наук, профессор Самаркандского государственного медицинского института. (Узбекистан).
Мамадалиев Абдурахмон Маматкулович - доктор медицинских наук, профессор Самаркандского государственного медицинского института. (Узбекистан).
Чутко Леонид Семенович - доктор медицинских наук, профессор, руководитель Центра поведенческой неврологии Института мозга человека им. Н.П. Бехтеревой. (Россия).
Муратов Фахмитдин Хайритдинович - доктор медицинских наук, профессор Ташкентский государственный медицинский университет. (Узбекистан).
Дьяконова Елена Николаевна - доктор медицинских наук, профессор, Ивановская государственная медицинская академия. (Россия).
Труфанов Евгений Александрович – доктор медицинских наук, профессор Национальный университет охраны здоровья Украины имени П.Л. Шупика и указать его расположение (Украина)
Норов Абдурахмон Убайдуллаевич – доктор медицинских наук, профессор, главный врач Бухарского областного многопрофильного медицинского центра. (Узбекистан)
Абдуллаева Наргиза Нурмаматовна – доктор медицинских наук, профессор Самаркандского государственного медицинского института. (Узбекистан).
Азизова Раъно Баходировна - доктор медицинских наук, доцент Ташкентский государственный медицинский университет. (Узбекистан).
Давлатов Салим Сулаймонович - Начальник отдела надзора качества образования, доцент Бухарского государственного медицинского института. (Узбекистан).
Артыкова Мавлюда Абдурахмановна - доктор медицинских наук, профессор Бухарского государственного медицинского института. (Узбекистан).
Уринов Мусо Болтаевич - доктор медицинских наук, доцент Бухарского государственного медицинского института. (Узбекистан).
Киличев Ибодулла Абдуллаевич – доктор медицинских наук, профессор Ургенчского филиала Ташкентской медицинской академии. (Узбекистан).
Рашидова Нилуфар Сафоевна - доктор медицинских наук, доцент Ташкентский государственный медицинский университет. (Узбекистан).
Ганиева Манижа Тимуровна - кандидат медицинских наук, доцент Таджикского государственного медицинского университета (Таджикистан).
Хазраткулов Рустам Бафоевич - доктор медицинских наук, руководитель научного отдела сосудистой патологии центральной нервной системы Республиканского специализированного научно – практического медицинского центра нейрохирургии, профессор кафедры нейрохирургии Центра развития профессиональной квалификации медицинских работников (Узбекистан).
Нуралиева Хафиза Отаевна - кандидат медицинских наук, доцент Тошкентского фармацевтического института. (Узбекистан).
Исмаилова Раъно Олимджановна – DSc, руководитель научного отдела патологии позвоночника и спинного мозга Республиканского специализированного научно – практического медицинского центра нейрохирургии (Узбекистан).
Югай Игорь Александрович – старший научный сотрудник отделения нейрохирургии детского возраста Республиканского специализированного научно – практического медицинского центра нейрохирургии. Доцент кафедры нейрохирургии Центра развития профессиональной квалификации медицинских работников (Узбекистан).
Иноятова Ситора Ойбековна - DSC, доцент кафедры Неврологии и народной медицины, Ташкентского государственного медицинского университета.
Абдукодиров Элдор Исроилович - DSC, доцент кафедры Неврологии и народной медицины, Ташкентского государственного медицинского университета.
Ахророва Шахло Ботировна - доцент кафедры неврологии Бухарского государственного медицинского института (DSc)

MINISTRY OF HEALTH OF THE REPUBLIC OF UZBEKISTAN PUBLIC ASSOCIATION OF NEUROLOGISTS OF UZBEKISTAN

JOURNAL OF NEUROLOGY AND NEUROSURGICAL RESEARCH

Bukhara State Medical Institute and tadqiqot.uz

Chief Editor:

Khodjieva Dilbar Tadjiyevna
Doctor of medical Sciences, Professor, Bukhara state medical Institute. (Uzbekistan).
ORCID ID: 0000-0002-5883-9533

Deputy editor-in-chief:

Khaydarova Dildora Kadirovna
Doctor of Medical Sciences, Professor of the Tashkent State Medical University. (Uzbekistan).
ORCID ID: 0000-0002-4980-6158

Peer-reviewed scientific and practical journal "Journal of Neurology and Neurosurgical Research"
Published 6 times a year
#6 (07), 2026
ISSN 2181-0982

Editorial address:
Tadqiqot LLC the city of Tashkent, Amir Temur Street pr.1, House 2.
Web: http://www.tadqiqot.uz/;
Email: info@tadqiqot.uz
Phone: (+998-94) 404-0000

Layout and preparation for printing held in the editorial office of the journal.

Design – pagemaker:
Khurshid Mirzakhmedov

Journal is registered at the Office of Press and Information Tashkent city, Reg. No. July 1, 2020

Electronic version of the Journal on sites:
www.tadqiqot.uz, www.bsmi.uz

- - -

The journal is included in the list of scientific publications recommended for publication of the main scientific results of dissertations in medical sciences since September 27, 2024 by the Higher Attestation Commission of the Republic of Uzbekistan (letter No. 361/6 dated 2024).

EDITORIAL TEAM:

Khaydarov Nodirjon Kadirovich - Doctor of Medicine, Professor, Rector of Tashkent State Medical University. (Uzbekistan).
Nuraliev Nekkadam Abdullaevich - Doctor of Medical Sciences, Professor, Immunologist, Microbiologist, Vice-Rector for Research and Innovation of the Bukhara State Medical Institute. (Uzbekistan).
Kariev Gayrat Maratovich - Doctor of Medicine, Professor, Director of the Republican Scientific Center for Neurosurgery of Uzbekistan. (Uzbekistan).
Anatoly Ivanovich Fedin - Doctor of Medical Sciences, professor, Honored Doctor of the Russian Federation. Russian National Research Medical University named after N.I. Pirogova. (Russia).
Madjidova Yokutxon Nabievna - Doctor of Medicine, Professor, Tashkent State Medical University. (Uzbekistan).
Rakhimbaeva Gulnora Sattarovna - Doctor of Medical Sciences, Professor, the Tashkent State Medical University. (Uzbekistan).
Djurabekova Aziza Taxirovna - Doctor of Medicine, Professor, the Samarkand State Medical Institute. (Uzbekistan).
Mamadaliev Abdurakhmon Mamatkulovich - Doctor of Medical Sciences, Professor of the Samarkand State Medical Institute. (Uzbekistan).
Chutko Leonid Semenovich - Doctor of Medicine, Head of the Center for Behavioral Neurology of the Institute of Human Brain named after N.P. Bekhtereva. (Russia).
Muratov Fakhmitdin Khayritdinovich - Doctor of Medical Sciences, Professor, the Tashkent State Medical University. (Uzbekistan).
Dyakonova Elena Nikolaevna - Doctor of Medicine, professor of the Ivanovo State Medical Academy. (Russia).
Trufanov Evgeniy Aleksandrovich - Doctor of Medical Sciences, Professor, P.L. Shupyk National University of Health Protection of Ukraine and indicate its location (Ukraine).
Norov Abdurakhmon Ubaydullaevich - Doctor of Medicine, professor, Chief Physician of the Bukhara Regional Multidisciplinary Medical Center. (Uzbekistan).
Abdullaeva Nargiza Nurmamatovna - Doctor of Medicine, professor of the Samarkand State Medical Institute. (Uzbekistan).
Azizova Rano Baxodirovna - doctor of medical Sciences, associate Professor of the Tashkent State Medical University. (Uzbekistan).
Davlatov Salim Sulaimonovich - Head of the Department of education quality supervision, associate Professor of the Bukhara state medical Institute. (Uzbekistan).
Artykova Mavlyuda Abdurakhmanovna - Doctor of Medical Sciences, Professor of the Bukhara State Medical Institute. (Uzbekistan).
Urinov Muso Boltaevich - Doctor of Medicine, Associate Professor, Bukhara State Medical Institute. (Uzbekistan).
Kilichev Ibodulla Abdullaevich - Doctor of Medicine, professor of the Urgench branch of the Tashkent Medical Academy. (Uzbekistan).
Rashidova Nilufar Safoevna - doctor of medical Sciences, associate Professor of the Tashkent State Medical University. (Uzbekistan).
Ganieva Manizha Timurovna - Candidate of Medical Sciences, Associate Professor, Tajik State Medical University. (Tajikistan).
Hazratkulov Rustam Bafoevich - Doctor of Medicine, head of the scientific department of vascular pathology of the central nervous system of the Republican specialized scientific and practical medical center for neurosurgery, professor of the department of neurosurgery at the Center for the development of professional qualifications of medical workers (Uzbekistan).
Nuralieva Hafiza Otayevna - Candidate of medical Sciences, associate Professor, Toshkent pharmaceutical Institute. (Uzbekistan).
Ismailova Rano Olimdjanovna - Doctor of Medicine, head of the spine department of the Republican specialized scientific and practical medical center of neurosurgery (Uzbekistan).
Yugay Igor Aleksandrovich - senior research of the scientific department of pediatric of the Republican specialized scientific and practical medical center for neurosurgery. Associate professor of the department of neurosurgery at the Center for the development of professional qualifications of medical workers (Uzbekistan).
Inoyatova Sitora Oybekovna – DSc Associate Professor, Department of Neurology and Traditional Medicine, Tashkent State Medical University
Abdukodirov Eldor Isroilovich – DSc Associate Professor, Department of Neurology and Traditional Medicine, Tashkent State Medical University
Akhrorova Shakhlo Botirovna - Associate Professor of the Department of Neurology, Bukhara State Medical Institute, Doctor of Science (DSc).

СОДЕРЖАНИЕ | CONTENT

1. Толибов Дилшод Сирожович, Абдурасулова Нозимахон Абдувохид қизи
БОШ МИЯ СУРУНКАЛИ ИШЕМИЯСИ ФОНИДА СОМАТОВЕГЕТАТИВ СИНДРОМ РИВОЖЛАНИШИДА КОРТИЗОЛ ВА β-ЭНДОРФИННИНГ ДИАГНОСТИК АҲАМИЯТИ ҲАМДА ИНДИВИДУАЛЛАШТИРИЛГАН ТЕРАПИЯ САМАРАДОРЛИГИ……7

2. Tulyaganov Davron Baxtiyarovich, Maksudova Laylo Baxtiyarovna, Berdiyev Nodirbek Fayziyevich, Yavkachov Avazbek Azizbekovich, Bokiyeva Fayziniso Adxam qizi, Jabborova Mamura Ismatullo qizi
O‘TKIR ISHEMIK INSULTDA INTENSIV TERAPIYA VA KLINIK NATIJALARNI PROGNOZLASHNING ZAMONAVIY YONDASHUVLARI……12

3. Davlatbayev Alisher Faxraddin o‘g‘li, Majidova Yoqutxon Nabiyevna
ISHEMIK INSULTDAN KEYINGI KOGNITIV BUZILISHLARDA TRANSKUTAN VAGUS NERV STIMULYATSIYASI: NEYROPLASTIKLIK, BDNF, EEG BIOMARKERLARI VA PRECIZION REABILITATSIYA MODELI……..18

4. Xodjiyeva Dilbar Tadjiyevna, Farmonov Hayotbek Fakhriddin o‘g‘li
SERVIKOGEN BOSH OG‘RIG‘IDA MIOFASSIAL TRIGGER NUQTALARNING KLINIK AHAMIYATI: FENOTIPLASHDAN MAQSADLI BOTULIN TOKSINI-A TERAPIYASIGACHA……..25

5. Fayziyeva Ra’nogul Hoji qizi, Azizova Ra’no Bahodirovna
GENERALIZATSIYALASHGAN VA STRUKTUR FOKAL EPILEPSIYADA INTERIKTAL ELEKTROENSEFALOGRAFIK KO‘RSATKICHLARNING QIYOSIY TAHLILI……...32

6. Yavkachov Avazbek Azizbekovich, Alimov Doniyor Anvarovich, Berdiyev Nodirbek Fayziyevich, Bokiyeva Fayziniso Adxam qizi, Jabborova Mamura Ismatullo qizi
INSULTDA D-DIMER, GFAP VA FAST SHKALASINING DIAGNOSTIK VA PROGNOSTIK AHAMIYATI (ADABIYOTLAR SHARHI)……...37

7. Mamajonov Farrux Erkin o`gli, Musayeva Yulduz Alpisovna
YUZ NERVI NEVROPATIYASIDA KECHKI ASORATLARNI ERTA BASHORATLASHDA ELEKTRONEYROMIOGRAFIYA VA PROYALLIG'LANISH SITOKINLARI KO'RSATKICHLARINING PROGNOSTIK AHAMIYATI……...44

8. Раҳимбаева Гулнора Саттаровна, Нажмутдинова Дилором Камардиновна, Абдураҳмонова Қутлибика Бахтиёр қизи
ИНСУЛИНРЕЗИСТЕНТЛИКНИНГ ИШЕМИК ИНСУЛЬТ ПАТОГЕНЕЗИ ВА ПРОГНОЗИДАГИ АҲАМИЯТИ……...50

9. Абдукадирова Дилфуза Тахировна, Шохобиддинова Гулираъно Каримжон кизи, Солижонов Мухаммадрузи Ихтиёржон угли
НАРУШЕНИЯ СНА ПРИ СИНДРОМЕ БЕСПОКОЙНЫХ НОГ: КЛИНИЧЕСКИЕ И ГЕНДЕРНЫЕ ОСОБЕННОСТИ…..56

10. Maftuna Baxtiyor qizi Ibodulloyeva, Zarifboy Rajabovich Ibodullayev
ISHEMIK INSULTDAN KEYIN QO‘SHIMCHA PSIXOMOTOR TERAPIYANING SAKKIZ HAFTALIK KURSI DAVOMIDA QO‘L VA OYOQ HARAKAT FAOLIYATINING TIKLANISHI……..63

11. Узакова Мусавара Хисравовна, Джурабекова Азиза Тахировна
КЛИНИКО-НЕЙРОФИЗИОЛОГИЧЕСКИЕ И НЕЙРОПСИХОЛОГИЧЕСКИЕ КОРРЕЛЯТЫ ТЯЖЕСТИ ТИКОЗНЫХ РАССТРОЙСТВ У ДЕТЕЙ……69

12. Каримбердиев Азиз Даврон угли, Рахимбаева Гульнара Саттаровна, Вахабова Наргиза Максудовна
КЛИНИЧЕСКАЯ ЭФФЕКТИВНОСТЬ РИТМИЧЕСКОЙ ТРАНСКРАНИАЛЬНОЙ МАГНИТНОЙ СТИМУЛЯЦИИ В ПЕРИОД РАННЕЙ РЕАБИЛИТАЦИИ ПОСЛЕ ИШЕМИЧЕСКОГО ИНСУЛЬТА……...74

13. Клычева Раушания Ислямовна, Рахимбаева Гулнора Саттаровна
ОСОБЕННОСТИ УРОВНЕЙ МОЗГОВОГО НЕЙРОТРОФИЧЕСКОГО ФАКТОРА BDNF И КОРТИЗОЛА У ПАЦИЕНТОВ С РАЗЛИЧНЫМИ ФОРМАМИ ЭПИЛЕПСИИ……..79

14. Абдуллаева Муборак Бекқуловна, Абдуллаева Муниса Мансуровна, Ҳамидов Отабек Ҳаётбекович, Актамова Мадинабону Ўктам қизи, Маҳмудов Абдуллоҳ Салоҳиддин ўғли
КОМПРЕССИОН ВА ЯЛЛИҒЛАНИШ ГЕНЕЗЛИ ПЛЕКСОПАТИЯЛАРДА ЭЛЕКТРОНЕЙРОМИОГРАФИК НАМУНАЛАРНИНГ КЛИНИК ВА НЕЙРОФИЗИОЛОГИК ХУСУСИЯТЛАРИ……..83

15. Madjidova Yakutxon Nabiyevna, Nizamxo‘jayeva Shahzoda Baxtiyor qizi
SURUNKALI MIYA ISHEMIYASI BO‘LGAN BEMORLARNING PSIXOEMOTSIONAL VA KOGNITIV PROFILI…….89

16. Абдуллаева Муборак Бекқуловна, Ҳамидов Отабек Ҳаётбекович, Халилов Дилшод Равшан ўғли, Актамова Мадинабону Ўктам қизи, Маҳмудов Абдуллоҳ Салоҳиддин ўғли
ТУНГИ ЭНУРЕЗНИНГ НЕЙРОФИЗИОЛОГИК АСОСЛАРИ: ЗАМОНАВИЙ ДИАГНОСТИК ЁНДАШУВЛАР ВА КЛИНИК АҲАМИЯТИ (адабиётлар шарҳи)……...96

17. Садыкова Гулчехра Кобуловна, Хаитбаева Шукуржон Хаитбаевна
РАННЯЯ ДИАГНОСТИКА ДЕТСКОГО ЦЕРЕБРАЛЬНОГО ПАРАЛИЧА: СОВРЕМЕННЫЕ МЕТОДЫ НЕЙРОРАЗВИТИЙНОЙ ОЦЕНКИ И АЛГОРИТМ КЛИНИЧЕСКОГО ВЫЯВЛЕНИЯ…….102

18. Сирожидинова Нилуфар Шарофидиновна, Хайдаров Нодир Кадирович
ПСИХОЭМОЦИОНАЛЬНЫЕ НАРУШЕНИЯ В ПЕРИОД ЛАКТАЦИИ: НЕЙРОЭНДОКРИННЫЕ, ГЕМОДИНАМИЧЕСКИЕ МЕХАНИЗМЫ И ЭФФЕКТИВНОСТЬ КОМПЛЕКСНОЙ АМБУЛАТОРНОЙ РЕАБИЛИТАЦИИ……112

19. Ярашев А.Р., Эшонов О.Ш.
ИММУННЫЕ МЕХАНИЗМЫ РЕПЕРФУЗИОННОГО СИНДРОМА ПРИ ИШЕМИЧЕСКОМ ИНСУЛЬТЕ ПОСЛЕ СИСТЕМНОЙ ВНУТРИВЕННОЙ ТРОМБОЛИТИЧЕСКОЙ ТЕРАПИИ…….116

20. Садикова Адиля Назруллаевна, Маджидова Якутхон Набиевна, Бобоев Кодиржон Тухтабаевич
ГЕНЕТИЧЕСКИЕ МАРКЕРЫ ЭНДОТЕЛИАЛЬНОЙ И ГИПОКСИЧЕСКОЙ ДИСРЕГУЛЯЦИИ ПРИ ХРОНИЧЕСКОЙ ИШЕМИИ МОЗГА РАЗЛИЧНОЙ СТЕПЕНИ ТЯЖЕСТИ……...123

21. Узаков Журабек Бахтиерович, Мавлянова Зилола Фархадовна
ДИАГНОСТИЧЕСКАЯ ЗНАЧИМОСТЬ НЕЙРОФИЗИОЛОГИЧЕСКИХ И СТРУКТУРНО-ГЕМОДИНАМИЧЕСКИХ ИЗМЕНЕНИЙ ПРИ ГЕМИФАЦИАЛЬНОМ СПАЗМЕ……129

22. Нестерова Светлана Ивановна, Ходжаева Дилбар Таджиевна, Каюмов Холмурод Найимович,
МКБ-11: ГЛАВА 26, ФАКУЛЬТАТИВНОЕ ДВОЙНОЕ КОДИРОВАНИЕ И ИНТЕГРАТИВНАЯ РЕАБИЛИТАЦИЯ ДЕТЕЙ С ДЕТСКИМ ЦЕРЕБРАЛЬНЫМ ПАРАЛИЧОМ……134

23. Хайдарова Дилдора Кадировна, Кудратова Шаходат Рамазоновна
КОГНИТИВНЫЕ ФУНКЦИИ ПО ШКАЛАМ SLUMS И MINI-COG У БОЛЬНЫХ ХРОНИЧЕСКОЙ ИШЕМИЕЙ МОЗГА НА РАЗНЫХ СТАДИЯХ ХРОНИЧЕСКОЙ БОЛЕЗНИ ПОЧЕК……..138

24. Рахимова К.Э., Омонова У.Т.
НЕЙРОВИЗУАЛИЗАЦИОННЫЕ И ЭЛЕКТРОЭНЦЕФАЛОГРАФИЧЕСКИЕ ОСОБЕННОСТИ У ДЕТЕЙ С ДЕТСКИМ ЦЕРЕБРАЛЬНЫМ ПАРАЛИЧОМ И СОПУТСТВУЮЩИМ РАССТРОЙСТВОМ АУТИСТИЧЕСКОГО СПЕКТРА……142

25. Ахтамова Шахзода Фозиловна
РЕЧЕВЫЕ НАРУШЕНИЯ У ДЕТЕЙ С ДЕТСКИМ ЦЕРЕБРАЛЬНЫМ ПАРАЛИЧОМ……..147

26. Холиков Шухрат Ашурович
НЕЙРОФИЗИОЛОГИЧЕСКИЕ КРИТЕРИИ ОГРАНИЧЕНИЯ ОБЪЁМА РЕЗЕКЦИИ ИНТРАМЕДУЛЛЯРНЫХ ОПУХОЛЕЙ СПИННОГО МОЗГА……154

ISSN: 2181-0982
www.tadqiqot.uz
JOURNAL OF NEUROLOGY AND NEUROSURGERY RESEARCH
ЖУРНАЛ НЕВРОЛОГИИ И НЕЙРОХИРУРГИЧЕСКИХ ИССЛЕДОВАНИЙ

UDK 617.53-06:616.857-02

Xodjiyeva Dilbar Tadjiyevna
Farmonov Hayotbek Fakhriddin o‘g‘li
Buxoro davlat tibbiyot instituti

SERVIKOGEN BOSH OG‘RIG‘IDA MIOFASSIAL TRIGGER NUQTALARNING KLINIK AHAMIYATI: FENOTIPLASHDAN MAQSADLI BOTULIN TOKSINI-A TERAPIYASIGACHA

http://dx.doi.org/10.5281/zenodo.23945405

ANNOTATSIYA

Servikogen bosh og‘rig‘i (SBO) bo‘yin umurtqa pog‘onasi, yuqori servikal bo‘g‘imlar va yumshoq to‘qimalardan kelib chiqadigan ikkilamchi bosh og‘rig‘i bo‘lib, klinik fenotipi ko‘pincha migren va zo‘riqish tipidagi bosh og‘rig‘i bilan qisman ustma-ust tushadi. Ayniqsa, faol miofassial trigger nuqtalar tipik bosh og‘rig‘ini qayta chaqirishi, mahalliy bosimga sezuvchanlikni oshirishi va bo‘yin harakatlari bilan bog‘liq simptomlarni kuchaytirishi mumkin. Shu sababli SBOni faqat anatomik tashxis sifatida emas, balki mushak-skelet, miofassial va nositseptiv komponentlari turli darajada ifodalangan geterogen klinik sindrom sifatida baholash muhimdir. Ushbu maqolada SBOda miofassial trigger nuqtalarning diagnostik va patofiziologik ahamiyati, bosim og‘riq bo‘sag‘asi, cervical range of motion, Cervical Flexion-Rotation Test, Neck Disability Index va Headache Impact Test-6 kabi ko‘rsatkichlarning klinik qiymati tahlil qilindi. Shuningdek, A tip botulin toksinining mushak relaksatsiyasi bilan bir qatorda periferik nositseptiv mediatorlar ajralishini kamaytirishi mumkin bo‘lgan mexanizmlari va SBOda uni qo‘llashga oid mavjud klinik dalillar tanqidiy ko‘rib chiqildi. Hozirgi dalillar BoNT-A samaradorligi bo‘yicha bir xil xulosaga kelish imkonini bermaydi; bu holat bemorlarni tanlash mezonlari, doza, preparat, in’eksiya nuqtalari va natijalarni baholash usullarining geterogenligi bilan izohlanadi. Maqolada faol trigger nuqtalar soni va lokalizatsiyasi, tipik og‘riqni palpatsiya bilan qayta chaqirish, bosim og‘riq bo‘sag‘asi, bo‘yin harakatchanligi, NDI, HIT-6 va bosh og‘rig‘i kundaligi kabi parametrlarni birlashtiruvchi klinik fenotiplash yondashuvi taklif etiladi. Bu yondashuv BoNT-Aga ehtimoliy javob beruvchi bemorlarni kelgusida prospektiv ravishda aniqlash uchun ilmiy asos yaratishi mumkin.

Kalit so‘zlar: servikogen bosh og‘rig‘i, miofassial trigger nuqta, A tip botulin toksini, botulinoterapiya, bosim og‘riq bo‘sag‘asi, cervical range of motion, Neck Disability Index, Headache Impact Test-6, personalizatsiyalangan davolash.

Ходжиева Дилбар Таджиевна
Фармонов Хаётбек Фахриддин угли
Бухарский государственный медицинский институт

КЛИНИЧЕСКОЕ ЗНАЧЕНИЕ МИОФАСЦИАЛЬНЫХ ТРИГГЕРНЫХ ТОЧЕК ПРИ ЦЕРВИКОГЕННОЙ ГОЛОВНОЙ БОЛИ: ОТ ФЕНОТИПИРОВАНИЯ К ТАРГЕТНОЙ ТЕРАПИИ БОТУЛИНИЧЕСКИМ ТОКСИНОМ ТИПА А

АННОТАЦИЯ

Цервикогенная головная боль (ЦГБ) представляет собой вторичную головную боль, связанную с патологией структур шейного отдела позвоночника и мягких тканей шеи, при этом ее клинический фенотип частично перекрывается с мигренью и головной болью напряжения. Активные миофасциальные триггерные точки могут воспроизводить привычную для пациента головную боль, снижать порог болевой чувствительности при давлении и усиливать симптомы, связанные с движениями шеи. Это позволяет рассматривать ЦГБ не как однородный синдром, а как гетерогенное состояние с различной выраженностью суставного, мышечного, миофасциального и ноцицептивного компонентов. В статье проанализированы клиническое и патофизиологическое значение миофасциальных триггерных точек, диагностическая роль порога болевой чувствительности при давлении, объема движений шеи, Cervical Flexion-Rotation Test, Neck Disability Index и Headache Impact Test-6. Критически рассмотрены механизмы действия ботулинического токсина типа А (BoNT-A), включающие не только снижение мышечной гиперактивности, но и модуляцию периферической ноцицептивной передачи, а также имеющиеся клинические данные о применении BoNT-A при ЦГБ. Результаты исследований остаются неоднородными: положительные эффекты в отдельных работах сосуществуют с качественными плацебо-контролируемыми исследованиями без убедительного преимущества препарата. Вероятными причинами являются различия в диагностических критериях, дозах, препаратах, целевых мышцах, точках введения и фенотипах пациентов. В качестве перспективного направления

предложено объединение количества и локализации активных триггерных точек, воспроизведения привычной боли при пальпации, порога болевой чувствительности, показателей подвижности шеи, NDI, HIT-6 и дневника головной боли в единый клинический профиль. Такой подход требует проспективной проверки, но может стать основой для отбора пациентов с большей вероятностью ответа на таргетную BoNT-A-терапию.

Ключевые слова: цервикогенная головная боль, миофасциальные триггерные точки, ботулинический токсин типа А, ботулинотерапия, порог болевой чувствительности, объем движений шеи, Neck Disability Index, Headache Impact Test-6, персонализированная терапия.

Xodjiyeva Dilbar Tadjiyevna
Farmonov Hayotbek Fakhriddin ugli
Bukhara State Medical Institute

CLINICAL SIGNIFICANCE OF MYOFASCIAL TRIGGER POINTS IN CERVICOGENIC HEADACHE: FROM PHENOTYPING TO TARGETED BOTULINUM TOXIN TYPE A THERAPY

ANNOTATION

Cervicogenic headache (CGH) is a secondary headache attributed to cervical structures and soft tissues, with a clinical phenotype that may partially overlap with migraine and tension-type headache. Active myofascial trigger points can reproduce a patient's familiar headache, increase local pressure sensitivity, and amplify symptoms provoked by cervical movement or sustained posture. These observations support a view of CGH as a heterogeneous clinical syndrome in which articular, muscular, myofascial, and nociceptive mechanisms contribute in different proportions. This narrative review analyzes the clinical and pathophysiological significance of myofascial trigger points in CGH and examines the value of pressure pain threshold assessment, cervical range of motion, the Cervical Flexion-Rotation Test, the Neck Disability Index, and the Headache Impact Test-6 for phenotyping patients. The biological rationale for botulinum toxin type A (BoNT-A) is considered beyond muscle relaxation, including its capacity to interfere with the release of nociceptive mediators from peripheral sensory terminals. Current clinical evidence for BoNT-A in CGH is inconsistent: some controlled studies report reductions in headache intensity and frequency, whereas a methodologically rigorous crossover trial found no significant advantage over placebo. Recent reviews emphasize substantial heterogeneity in diagnostic criteria, toxin formulation, dose, injection sites, and outcome measures. We therefore propose a hypothesis-generating clinical framework that integrates the number and distribution of active trigger points, reproduction of the habitual headache on palpation, pressure pain thresholds, cervical mobility, functional disability, headache burden, and analgesic use. Such multidimensional phenotyping may provide a more rational basis for future prospective studies designed to identify patients who are more likely to respond to trigger point-targeted BoNT-A therapy.

Keywords: cervicogenic headache, myofascial trigger points, botulinum toxin type A, botulinum therapy, pressure pain threshold, cervical range of motion, Neck Disability Index, Headache Impact Test-6, personalized treatment.

I. Introduction

Cervicogenic headache (CGH) is classified by the International Classification of Headache Disorders, third edition (ICHD-3), as a secondary headache caused by a disorder of the cervical spine or the soft tissues of the neck. The diagnosis requires evidence of a cervical lesion or disorder capable of producing headache and evidence supporting a causal relationship, such as temporal association, improvement in parallel with the cervical disorder, reduced cervical range of motion with provocation of headache, or abolition of pain after diagnostic blockade [1]. The same classification also acknowledges a clinically important area of overlap: headache causally associated with cervical myofascial pain may resemble tension-type headache, and an appendix diagnosis of headache attributed to cervical myofascial pain remains available while the evidence base continues to evolve [1]. This diagnostic boundary is not a semantic issue. It directly affects patient selection, study populations, and the interpretation of treatment response.

The epidemiology of CGH is more uncertain than often assumed. A 2025 systematic review and meta-analysis that applied International Classification of Headache Disorders criteria found limited population-level evidence and reported a prevalence of 3.9% in the single eligible population-based study, while pooled relative frequency in tertiary headache clinics was approximately 3.1% [2]. The scarcity and heterogeneity of high-quality epidemiological data reinforce the need for rigorous phenotyping rather than broad labels. In clinical practice, patients diagnosed with CGH frequently present with unilateral or side-dominant pain, posterior-to-anterior radiation, restricted neck movement, provocation by cervical maneuvers, local tenderness, and altered motor control [1,3,4]. Yet no single physical sign is sufficiently specific to establish the diagnosis in isolation [3,4].

Among the most clinically intriguing findings is the presence of active myofascial trigger points in the suboccipital and cervical muscles. Trigger points are not unique to CGH, but increased sensitivity in specific muscles, reproduction of the patient's familiar pain pattern, and asymmetry in pressure pain thresholds may help define a clinically meaningful subgroup [7–9]. This subgroup is especially relevant to interventions directed at skeletal muscle and peripheral nociceptive input. Botulinum toxin type A (BoNT-A) is an established neuromuscular therapeutic agent with additional antinociceptive actions. Although it is approved for chronic migraine, its role in CGH remains uncertain because controlled trials have produced conflicting results and injection protocols vary markedly [13–16].

The central argument of this review is that the apparent inconsistency of BoNT-A studies in CGH may partly reflect a mismatch between a heterogeneous syndrome and relatively uniform injection strategies. If the clinically relevant pain generator differs between patients, fixed-site treatment may dilute benefit in the subgroup whose habitual headache is reproducibly driven by active myofascial trigger points. A phenotypic approach that combines headache characteristics, cervical functional impairment, trigger point mapping, pressure pain thresholds, and disability measures could therefore provide

a more rational platform for future targeted trials and prognostic modeling.

II. Materials and methods

This article was prepared as a focused narrative review and clinical-conceptual synthesis. PubMed/MEDLINE and journal publisher databases were searched through September 2026 using combinations of the terms “cervicogenic headache”, “myofascial trigger point”, “pressure pain threshold”, “cervical flexion-rotation test”, “Neck Disability Index”, “Headache Impact Test-6”, “botulinum toxin type A”, “botulinum therapy”, “central sensitization”, and “manual therapy”. Priority was given to ICHD-3 diagnostic guidance, systematic reviews and meta-analyses, diagnostic accuracy studies, randomized controlled trials, and recent clinical reviews. Older mechanistic and clinical studies were retained when they represented foundational evidence directly relevant to myofascial pain, trigger points, or BoNT-A in CGH. Because this is not a formal systematic review, no pooled effect estimate was calculated. The purpose was to integrate the available evidence into a clinically testable framework aligned with future prospective studies of trigger point-targeted BoNT-A therapy.

III. Cervicogenic headache as a clinical phenotype

The concept of CGH is based on referred pain from cervical structures to the head through convergence of upper cervical and trigeminal nociceptive afferents. Clinically, the syndrome often begins in the upper neck or occipital region and spreads anteriorly toward the temporal, frontal, or orbital areas. Side-locked pain, aggravation by neck movement, sustained posture, or digital pressure over cervical structures, and restricted cervical mobility support a cervical source, but none of these findings is absolutely specific [1,3]. Migrainous features such as nausea or photophobia can occur, generally to a lesser degree than in migraine, further complicating differential diagnosis [1].

A major advance in the clinical understanding of CGH has been the shift from single-test diagnosis to impairment patterns. Jull emphasized that reduced movement, upper cervical joint signs, and impaired deep cervical flexor function are more informative when interpreted together than when considered as isolated abnormalities [3]. Demont and colleagues reached a similar conclusion in a systematic review of diagnostic studies: history, signs, symptoms, and physical examination can support the diagnosis, but the certainty of evidence varies and no single finding should dominate clinical reasoning [4].

The Cervical Flexion-Rotation Test (CFRT) is one of the better studied physical examination tools. In C1–C2-related CGH, early work demonstrated high sensitivity and specificity when performed by trained examiners [6]. A later meta-analysis found pooled sensitivity and specificity around 0.83, although certainty of evidence was limited by the small number of studies and methodological concerns [5]. The test is therefore best regarded as one component of a broader phenotype rather than a definitive standalone test. The same principle applies to cervical range of motion (CROM), which captures functional restriction but cannot establish causality by itself.

Functional impact should also be quantified. The Neck Disability Index (NDI) provides a structured measure of neck-related functional limitation, while the Headache Impact Test-6 (HIT-6) estimates the burden of headache on daily life. A 28-day headache diary adds information that questionnaires cannot provide reliably from recall alone: monthly headache days, duration of individual episodes, peak and average intensity, provocation patterns, and analgesic consumption. When combined, these measures characterize severity along multiple dimensions and create reproducible endpoints for therapeutic studies.

IV. Myofascial trigger points: from association to clinical target

Myofascial trigger points are hyperirritable areas within skeletal muscle that may produce local pain, referred pain, motor dysfunction, or autonomic responses when stimulated. Their diagnostic reproducibility has been debated, but in headache disorders the clinically most relevant feature is not merely tenderness: it is the reproduction of the patient’s familiar pain pattern. This distinction matters because generalized tenderness can reflect sensitization, anxiety, sleep disruption, or chronic pain burden, whereas a site that reliably reproduces the habitual headache may have greater mechanistic relevance.

The relationship between myofascial pain and CGH has been recognized for decades. Jaeger reported that patients with cervicogenic headache had multiple trigger points on the symptomatic side and that palpation reproduced the typical headache in a substantial proportion of cases [9]. More recent work has refined this concept using pressure algometry. Olivier and colleagues found that trigger point sensitivity in the upper trapezius was greater in patients with cervicogenic than non-cervicogenic headache, suggesting that local pressure sensitivity may contribute to clinical differentiation [7]. Systematic review data also indicate that pressure pain thresholds in the cranio-cervical region are often reduced in headache populations, although the strength and location of this effect vary between diagnoses [8].

These observations fit a plausible pathophysiological sequence. Repetitive nociceptive input from cervical joints, muscles, fascia, or entheses may increase excitability within the trigeminocervical complex. Persistent muscle overload, altered motor control, protective co-contraction, and painful movement can then reinforce peripheral nociception. As pain becomes chronic, pressure hypersensitivity may extend beyond the original local generator, reflecting broader changes in pain processing. Importantly, central sensitization should not be inferred from a single questionnaire or tender point. The 2025 modification of the Central Sensitization Inventory for patients with headache provides a useful clinical measure of symptom burden, but mechanistic interpretation still requires caution [17].

This leads to a clinically useful distinction between a trigger point that is merely present and an active trigger point that is concordant with the patient’s complaint. For future BoNT-A trials, the latter is more relevant. A myofascial-dominant CGH phenotype could reasonably be defined by a cluster of features: reproducible habitual headache on trigger point palpation, multiple active points in anatomically plausible cervical muscles, reduced local pressure pain thresholds, pain provoked by cervical movement or posture, and measurable neck-related disability. Such a phenotype is a research hypothesis rather than an established diagnostic subtype, but it can be tested prospectively.

Table 1

Candidate clinical markers for a myofascial-dominant cervicogenic headache phenotype

Clinical domain	Practical measurement	Potential interpretation for targeted BoNT-A research
Habitual headache reproduction	Standardized palpation of suspected active trigger points	High face-validity marker that the examined muscle contributes to the patient's typical pain pattern; should be recorded as present/absent and by muscle.
Trigger point burden	Number and anatomical distribution of active trigger points	May reflect the extent of clinically relevant myofascial involvement; requires standardized examination.
Pressure pain threshold	Digital pressure algometry at predefined cervical muscles	Quantifies mechanical sensitivity and allows side-to-side and longitudinal comparison.
Cervical mobility	CROM: flexion, extension, lateral flexion, rotation	Captures mechanical restriction and provocation; useful as a functional outcome rather than a standalone diagnostic criterion.
Upper cervical function	Cervical Flexion-Rotation Test	Supports identification of upper cervical movement impairment, particularly C1–C2-related dysfunction.
Neck-related disability	Neck Disability Index (NDI)	Measures the functional consequences of cervical pain and dysfunction.
Headache burden	HIT-6 and 28-day headache diary	Measures real-world impact, headache days, duration, intensity, provocation, and analgesic use.
Pain amplification	Central sensitization symptom screening plus widespread PPT when feasible	May identify patients in whom generalized pain amplification could reduce the specificity of local trigger point findings.

V. Why targeted bont-a is biologically plausible

Botulinum toxin type A is best known for cleaving SNAP-25 at presynaptic cholinergic nerve terminals, thereby reducing acetylcholine release and producing temporary chemodenervation. In myofascial pain, however, muscle relaxation is unlikely to be the only relevant mechanism. Experimental and translational studies show that BoNT-A can influence nociceptive signaling by reducing exocytotic release of mediators such as glutamate, substance P, and calcitonin gene-related peptide from sensory terminals, thereby modulating peripheral sensitization [16]. These effects provide a biologically plausible bridge between trigger point-targeted injection and headache reduction, especially when the injected muscle is a reproducible source of the patient's pain.

The therapeutic logic is therefore twofold. First, reducing excessive or painful muscle activity may interrupt a mechanical cycle of overload, protective spasm, and painful movement. Second, decreasing peripheral nociceptive drive may reduce the barrage of input reaching the trigeminocervical complex. The second mechanism is particularly relevant to chronic pain states, because sustained peripheral input can help maintain central amplification even after the initial structural trigger becomes less prominent. Nevertheless, this mechanistic plausibility does not prove clinical efficacy in CGH. The central question is whether the right patients and the right muscles are being treated.

A fixed injection protocol assumes that the same muscles and anatomical sites are relevant across patients. A trigger point-guided strategy assumes the opposite: that the clinically dominant myofascial generators vary between individuals and should be mapped before injection. Muscles that commonly deserve examination include the upper trapezius, splenius capitis, splenius cervicis, semispinalis capitis, sternocleidomastoid, levator scapulae, and suboccipital muscles. The selection of a target should be based on concordant clinical evidence, not tenderness alone. This is precisely where trigger point mapping, pressure pain thresholds, cervical movement testing, and headache reproduction can become a coherent treatment-selection strategy rather than a collection of unrelated measurements.

VI. Current clinical evidence for bont-a and why results conflict

The clinical literature on BoNT-A in CGH is small and inconsistent. A randomized, double-blind, placebo-controlled crossover trial by Linde and colleagues included 28 patients with long-standing treatment-resistant CGH and used fixed injection sites in neck muscles. The study did not demonstrate a significant benefit of onabotulinumtoxinA over placebo for the primary endpoint or secondary outcomes [14]. By contrast, Karadaş and colleagues studied 40 patients and reported reductions in pain severity and frequency, with between-group differences favoring BoNT-A at 12 weeks [13]. These apparently conflicting results have shaped the controversy surrounding botulinum therapy in CGH.

The most recent focused review of BoNT-A in cervicogenic headache, published in 2026, identified only five eligible studies and emphasized substantial heterogeneity in diagnostic criteria, toxin formulations, dosing, target muscles, injection techniques, and outcome measures [15]. Several studies suggested benefit, particularly in chronic or treatment-resistant populations and in protocols that incorporated trigger-point guidance, but the authors concluded that the overall evidence remains inconclusive [15]. This conclusion is clinically important: absence of consistent superiority in a heterogeneous syndrome does not automatically mean that all biologically defined subgroups are equally unlikely to respond.

Broader treatment literature also argues against positioning BoNT-A as a universal first-line intervention. Myofascial release can reduce pain and disability in CGH, although certainty of evidence is limited by study heterogeneity [10]. A 2025 network meta-analysis found that manual therapy approaches can improve pain and function, while a 2026 network meta-analysis comparing multiple CGH treatments highlighted the value of

multimodal rehabilitation strategies [11,12]. These data support a stepped approach: establish the diagnosis, treat modifiable cervical impairments, and reserve invasive or targeted interventions for carefully selected patients with persistent disability or a clear clinical pain generator.

Why might BoNT-A studies disagree? At least six sources of heterogeneity are plausible. First, CGH itself is diagnostically heterogeneous. Second, some studies use fixed-site protocols while others individualize targets. Third, different toxin preparations and dose-equivalence assumptions may not be interchangeable. Fourth, outcome timing varies and may not align with the pharmacodynamic window of BoNT-A. Fifth, clinically meaningful response may be obscured if headache frequency, intensity, disability, and analgesic use are analyzed separately without a predefined responder definition. Sixth, central pain amplification and psychosocial comorbidity may reduce the specificity of a purely peripheral intervention. These factors make patient selection and baseline phenotyping central scientific questions rather than secondary details.

Table 2

Selected evidence on botulinum toxin type A in cervicogenic headache

Study	Design / population	Injection strategy	Main finding	Interpretive point
Linde et al., 2011 [14]	Randomized, double-blind, placebo-controlled crossover; 28 adults with treatment-resistant CGH	Fixed sites in neck muscles on the pain side	No significant advantage over placebo for the primary endpoint or secondary efficacy outcomes	High-quality negative trial suggests that fixed-site treatment is not universally effective.
Karadaş et al., 2012 [13]	Placebo-controlled clinical trial; 40 patients	Multiple cranial and cervical muscles; total 150 U	Pain severity and frequency improved, with significant between-group differences at 12 weeks	Positive signal, but protocol differed substantially from the negative crossover study.
Vasconcelos Fonseca & Camões-Barbosa, 2026 [15]	Focused review; 5 studies (4 RCTs, 1 prospective study)	Heterogeneous formulations, doses, muscles, and techniques	Overall evidence inconclusive; several studies reported benefit, especially in trigger point-guided or refractory populations	Supports standardized phenotyping and injection protocols before efficacy can be judged definitively.

VII. Toward a predictive clinical model

A clinically useful predictive model should begin with a clear definition of response. For a future prospective study, response could be defined using a composite anchored in clinically meaningful change: reduction in headache days, reduction in average or peak pain intensity, improvement in NDI or HIT-6, and reduction in acute analgesic use. A composite approach would better reflect the multidimensional burden of CGH than a single pain score. Importantly, the response threshold must be prespecified before analysis to avoid data-driven definitions.

Baseline candidate predictors should be measurable, reproducible, and biologically plausible. These include disease duration, monthly headache days, side-locked versus alternating pain, pain provocation by cervical movement, number and distribution of active myofascial trigger points, reproduction of the habitual headache during palpation, pressure pain threshold at predefined muscles, cervical range of motion, CFRT results, NDI, HIT-6, and analgesic use. Age, sex, prior treatment exposure, affective symptoms, sleep quality, and indicators of pain amplification may act as modifiers or confounders and should be collected systematically when feasible.

The statistical pathway should also be planned prospectively. Univariate comparisons can identify candidate variables associated with response, but multivariable logistic regression is needed to determine independent predictors. Receiver operating characteristic (ROC) analysis may then be used to identify clinically useful cut-off values for continuous measures such as pressure pain threshold, NDI, HIT-6, or headache frequency. Internal validation, penalization when the number of predictors is large relative to the sample size, and transparent reporting of calibration and discrimination are necessary to prevent an apparently strong model from collapsing in a new cohort. A prediction model is clinically useful only if it performs beyond the development dataset.

An especially important hypothesis is that the predictive value may reside not in one variable but in concordance between domains. For example, a patient with multiple active trigger points, reproducible habitual headache on palpation, reduced local pressure pain threshold, restricted cervical rotation, and high neck-related disability may represent a more coherent myofascial phenotype than a patient with diffuse tenderness, normal cervical mobility, and widespread pressure hypersensitivity. Testing such interactions could reveal why two patients carrying the same diagnostic label respond differently to the same intervention.

This framework also has practical value outside research. It encourages the clinician to answer three questions before considering targeted BoNT-A: Is the headache diagnosis secure? Is there evidence that a particular cervical muscle or myofascial region reproduces and sustains the patient's typical pain? Is the overall pain pattern sufficiently localized that reducing peripheral input is biologically likely to matter? These questions do not replace guidelines or clinical judgment, but they reduce the risk of treating an anatomical map rather than a patient-specific pain generator.

VIII. Discussion

The available literature supports a nuanced interpretation of myofascial involvement in CGH. Trigger points are neither

pathognomonic nor clinically irrelevant. Their value depends on how they are defined and integrated with the rest of the examination. Simple tenderness has low specificity; reproduction of the habitual headache, asymmetric sensitivity, restricted movement, and functional impairment together provide a stronger clinical signal. This is consistent with contemporary CGH research emphasizing impairment patterns rather than isolated tests [3,4].

The same principle helps explain the uncertain literature on BoNT-A. The treatment has a plausible mechanism and some positive clinical signals, but it is not supported by uniformly positive randomized evidence [13–16]. A binary conclusion that BoNT-A either "works" or "does not work" for all CGH is therefore premature. A more useful question is whether a definable phenotype exists in which peripheral myofascial input is sufficiently important that targeted chemodenervation and sensory modulation produce a clinically meaningful effect. The current evidence cannot answer that question because most studies were not designed around validated phenotype-response relationships.

Recent systematic reviews of manual therapy, myofascial release, and multimodal treatment also reinforce the importance of conservative care [10–12]. This should not be interpreted as an argument against interventional treatment. Rather, it defines the clinical context in which targeted BoNT-A should be studied: patients with a confirmed diagnosis, persistent symptoms despite appropriate conservative management, and demonstrable active myofascial generators. Such enrichment may increase biological coherence and reduce the noise that undermines small clinical trials.

Several limitations of the proposed framework should be acknowledged. First, trigger point examination is examiner-dependent, and standardization of palpation pressure, anatomical mapping, and the definition of concordant pain is essential. Second, pressure pain threshold values are influenced by local tissue characteristics and broader pain sensitivity. Third, CROM and CFRT assess function but do not identify a specific nociceptive generator. Fourth, the overlap between CGH, migraine, tension-type headache, and occipital neuralgia can lead to diagnostic contamination [1,18]. Finally, the prognostic role of NDI, HIT-6, trigger point count, and algometry for BoNT-A response remains hypothetical until tested prospectively.

Despite these limitations, the proposed approach offers a practical research pathway. It converts a broad dissertation question into measurable clinical variables and a testable therapeutic hypothesis. A well-designed prospective study could randomize or compare patients receiving individualized trigger point-targeted BoNT-A with a standardized treatment strategy, follow outcomes at 4, 8, 12, and ideally 24 weeks, and use multivariable modeling to distinguish responders from non-responders. Such a design would address the central gap identified by recent reviews: not merely whether BoNT-A has an average effect, but which patient profile is most likely to benefit and why.

IX. Conclusion

Cervicogenic headache is a clinically heterogeneous secondary headache in which cervical joint dysfunction, muscle impairment, myofascial pain, and altered nociceptive processing may contribute in different proportions. Active myofascial trigger points are clinically relevant when they reproduce the patient's habitual headache and are supported by concordant findings such as reduced pressure pain thresholds, restricted cervical mobility, positive upper cervical functional testing, and measurable disability. Botulinum toxin type A has a biologically plausible role through both neuromuscular and antinociceptive mechanisms, but current evidence in CGH remains inconsistent because the available studies are few and heterogeneous. Future research should therefore move beyond fixed injection maps and diagnosis-only enrollment toward standardized clinical phenotyping. Integrating trigger point mapping, pressure algometry, CROM, CFRT, NDI, HIT-6, headache diaries, and analgesic use may allow the development and validation of prognostic criteria for targeted BoNT-A therapy. The key scientific objective is not to assume efficacy in all patients, but to identify the subgroup in whom a reproducible myofascial pain generator is sufficiently dominant to make individualized BoNT-A treatment rational and clinically meaningful.

REFERENCES

1. Headache Classification Committee of the International Headache Society (IHS). The International Classification of Headache Disorders, 3rd edition. Cephalalgia. 2018;38(1):1–211. doi:10.1177/0333102417738202.

2. Robinson CL, Christensen RH, Al-Khazali HM, Amin FM, Yang A, Lipton RB, Ashina S. Prevalence and relative frequency of cervicogenic headache in population- and clinic-based studies: a systematic review and meta-analysis. Cephalalgia. 2025;45(3):3331024251322446. doi:10.1177/03331024251322446.

3. Jull G. Cervicogenic headache. Musculoskelet Sci Pract. 2023;66:102787. doi:10.1016/j.msksp.2023.102787.

4. Demont A, Lafrance S, Benaissa L, Mawet J. Cervicogenic headache, an easy diagnosis? A systematic review and meta-analysis of diagnostic studies. Musculoskelet Sci Pract. 2022;62:102640. doi:10.1016/j.msksp.2022.102640.

5. van der Meer HA, Visscher CM, Vredeveld T, Nijhuis van der Sanden MWG, Engelbert RHH, Speksnijder CM. The diagnostic accuracy of headache measurement instruments: a systematic review and meta-analysis focusing on headaches associated with musculoskeletal symptoms. Cephalalgia. 2019;39(10):1313–1332. doi:10.1177/0333102419840777.

6. Ogince M, Hall T, Robinson K, Blackmore AM. The diagnostic validity of the cervical flexion-rotation test in C1/2-related cervicogenic headache. Man Ther. 2007;12(3):256–262. doi:10.1016/j.math.2006.06.016.

7. Olivier B, Pramod A, Maleka D. Trigger Point Sensitivity Is a Differentiating Factor between Cervicogenic and Non-Cervicogenic Headaches: A Cross-Sectional, Descriptive Study. Physiother Can. 2018;70(4):323–329. doi:10.3138/ptc.2017-38.

8. Castien RF, van der Wouden JC, De Hertogh W. Pressure pain thresholds over the cranio-cervical region in headache: a systematic review and meta-analysis. J Headache Pain. 2018;19(1):9. doi:10.1186/s10194-018-0833-7.

9. Jaeger B. Are "cervicogenic" headaches due to myofascial pain and cervical spine dysfunction? Cephalalgia. 1989;9(3):157–164. doi:10.1046/j.1468-2982.1989.0903157.x.

10. Lu Z, Zou H, Zhao P, Wang J, Wang R. Myofascial Release for the Treatment of Tension-Type, Cervicogenic Headache or Migraine: A Systematic Review and Meta-Analysis. Pain Res Manag. 2024;2024:2042069. doi:10.1155/2024/2042069.

11. Xu X, Ling Y. Comparative safety and efficacy of manual therapy interventions for cervicogenic headache: a systematic review and network meta-analysis. Front Neurol. 2025;16:1566764. doi:10.3389/fneur.2025.1566764.

12. Koonalinthip N, Koonalintip P, Stonsaovapak C. The Comparative Efficacy of Treatments for Cervicogenic Headache: A Systematic Review and Network Meta-Analysis of Randomised Controlled Trials. Eur J Pain. 2026;30(2):e70219. doi:10.1002/ejp.70219.

13. Karadaş Ö, Öztürk B, Ulaş ÜH, Kütükçü Y, Odabaşı Z. The efficacy of botulinum toxin in patients with cervicogenic headache: a placebo-controlled clinical trial. Balkan Med J. 2012;29(2):184–187. doi:10.5152/balkanmedj.2012.003.

14. Linde M, Hagen K, Salvesen Ø, Gravdahl GB, Helde G, Stovner LJ. Onabotulinum toxin A treatment of cervicogenic headache: a randomised, double-blind, placebo-controlled crossover study. Cephalalgia. 2011;31(7):797–807. doi:10.1177/0333102411398402.

15. Vasconcelos Fonseca C, Camões-Barbosa A. Botulinum Toxin Type A for Cervicogenic Headache: A Review of the Current Evidence. Cureus. 2026;18(7):e113464. doi:10.7759/cureus.113464.

16. Matak I, Bölcskei K, Bach-Rojecky L, Helyes Z. Mechanisms of Botulinum Toxin Type A Action on Pain. Toxins (Basel). 2019;11(8):459. doi:10.3390/toxins11080459.

17. Bakhtadze MA, Berdnikova AV, Kukushkin ML. The Central Sensitization Inventory: modification for tension-type headache, migraine and cervicogenic headache. Russian Journal of Pain. 2025;23(3):22–32. doi:10.17116/pain20252303122.

18. Lefel N, van Suijlekom H, Cohen SPC, Kallewaard JW, Van Zundert J. Cervicogenic headache and occipital neuralgia. Pain Pract. 2025;25(1):e13405. doi:10.1111/papr.13405.

ISSN 2181-0982

Doi Journal 10.26739/2181-0982

ЖУРНАЛ НЕВРОЛОГИИ И НЕЙРОХИРУРГИЧЕСКИХ ИССЛЕДОВАНИЙ

JOURNAL OF NEUROLOGY AND NEUROSURGERY RESEARCH

Editorial staff of the journals of www.tadqiqot.uz
Tadqiqot LLC the city of Tashkent,
Amir Temur Street pr.1, House 2.
Web: http://www.tadqiqot.uz/; Email: info@tadqiqot.uz
Phone: (+998-94) 404-0000

Контакт редакций журналов. www.tadqiqot.uz
ООО Tadqiqot город Ташкент,
улица Амира Темура пр.1, дом-2.
Web: http://www.tadqiqot.uz/; Email: info@tadqiqot.uz
Тел: (+998-94) 404-0000