O'TKIR ISHEMIK INSULTDAN KEYINGI DEPRESSIV HOLATLARNI ERTA ANIQLASHDA ZAMONAVIY YONDASHUVLAR VA DAVOLASHNI OPTIMALLASHTISH.

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Annotatsiya. *Miya qon tomir kasalliklarining oldini olish, erta tashxis qo'yish va davolash eng dolzarb muammolardan biri bo'lib, zamonaviy nevrologiyaning eng ustuvor yo'nalishi hisoblanadi. Qo'shma Shtatlarda insult o'lim sabablari tarkibida uchinchi va uzoq muddatli nogironlik sabablari orasida birinchi o'rinda turadi. Har yili 15 millionga yaqin odam insultni boshdan kechiradi, ularning 30-40 % insultdan keyingi depressiya holatlari kuzatilmoqda (1). Insultdan keyin kuzatiladigan depressiv holatlar reabilitatsiya samaradorligini pasaytiradi va o'lim xavfini 3 baravar oshiradi (2).*

Kalit so'zlar: *Insultdan keyingi depressiya, prognostik markerlar, reabilitatsiya, transkranial neyrosonografiya.*

СОВРЕМЕННЫЕ ПОДХОДЫ К РАННЕМУ ВЫЯВЛЕНИЮ ДЕПРЕССИВНЫХ СОСТОЯНИЙ ПОСЛЕ ОСТРОГО ИШЕМИЧЕСКОГО ИНСУЛЬТА И ОПТИМИЗАЦИЯ ЛЕЧЕНИИ.

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Аннотация. *Профилактика, ранняя диагностика и лечение цереброваскулярных заболеваний являются одной из наиболее актуальных проблем и приоритетными в современной неврологии. В Соединенных Штатах инсульт является третьей по значимости причиной смерти и основной причиной длительной инвалидности. Ежегодно инсульт переносят около 15 миллионов человек, и 30–40% из них испытывают постинсультную депрессию (1). Депрессивные состояния, наблюдаемые после инсульта, снижают эффективность реабилитации и увеличивают риск смерти в 3 раза (2).*

Ключевые слова: *постинсультная депрессия, прогностические маркеры, реабилитация, транскраниальная нейросонография.*

MODERN APPROACHES IN EARLY DETECTION OF DEPRESSIVE STATES AFTER ACUTE ISCHEMIC STROKE AND OPTIMIZATION OF TREATMENT.

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Abstract. *Prevention, early diagnosis and treatment of cerebrovascular diseases are one of the most urgent problems and are the most priority areas of modern neurology. In the United States, stroke is the third leading cause of death and the first leading cause of long-term disability. Every year, about 15 million people suffer a stroke, 30-40% of whom have post-stroke depression (1).*
Depressive states observed after stroke reduce the effectiveness of rehabilitation and increase the risk of death by 3 times (2).

Keywords: *Post-stroke depression, prognostic markers, rehabilitation, transcranial neurosonography.*

Kirish.

Ishemik insult keyingi davrlarda nafaqat nevrologik nogironlikning asosiy sabablaridan biri, balki depressiv holatlarning rivojlanishi uchun ham kuchli xavf omili hisoblanadi. Post-insult depressiyasi (PID) bemorlarning 30-40% ida

kuzatilib, ularning hayot sifatini keskin pasaytiradi, reabilitatsiya samaradorligini sekinlashtiradi va takroriy kardiovaskulyar hodisalar xavfini oshiradi. Hozirgi vaqtda PID ning patofiziologik mexanizmlari to‘liq aniqlanmagan bo‘lsa-da, so‘nggi tadqiqotlar miyaning subkortikal tuzilmalari, ayniqsa brainstem (miya poyasi) funktsional yoki strukturaviy o‘zgarishlari bilan bog‘liqligini ko‘rsatmoqda(3, 4). Insultning dastlabki bosqichlarida depressiv simptomlarni aniqlash va PID tashxisi uchun xavf ostida bo'lgan sub'ektlarni tanlab olish qiyin bo'lib qolmoqda. Hozirgi vaqtda PID ni baholash uchun qo'llaniladigan klinik chora-tadbirlar, ayniqsa o'tkir post-insult bemorlarida depressiyani simptomlarni aniqlash uchun zarur bo'lgan o'ziga xosliklar etarlicha emas. (5 , 6). Shu nuqtai nazardan, o'ziga xos biomarkerlarni aniqlash PID tashxisining sezgirligini oshirishga yordam berishi mumkin. Bundan tashqari, bu PID ning patofiziologik mexanizmlarini tushuntirish uchun foydali bo'lishi mumkin va natijada aniq maqsadli davolashni tanlashni osonlashtiradi (7).

Natija va muhokamalar.

Birinchi bosqichda 95 ta klinik va eksperimental maqolalar aniqlandi, ulardan 30 tasi qo'shilish mezonlariga javob berdi va ikkinchi bosqichda batafsil tahlildan o'tkazildi. Qo'shilgan tadqiqotlar natijalari tekshirilgan biomarkerlar turiga ko'ra neyroimaging, molekulyar va neyrofiziologikka bo'lingan. O'z navbatida, molekulyar insultdan keyingi depressiyaning biomarkerlari monoaminlar, o'sish omillari, neyroinflamasyon markerlari, gipotalamus-gipofiz-adrenal o'qining markerlari, oksidlovchi shikastlanish belgilari, boshqa metabolitlarlarga bo'lingan.
PSD ning neyroimaging biomarkerlari

Depressiyaning to'g'ridan-to'g'ri ildizlaridan biri bo'lishiga qaramasdan, qon tomir bilan aniqlangan neyroanatomik substrat aslida PSD rivojlanishiga hissa qo'shadimi, degan savol munozara mavzusi bo'lib qolmoqda (8. 9). Turli MRT va KT texnikasidan foydalangan holda ba'zi yaxshi tashkil etilgan tadqiqotlar natijalari aniq neyroanatomik mintaqadagi shikastlanishlar PSD (10-11) ni keltirib chiqaradigan dalillarni keltirmaydi . Aksincha, ko'p sonli klinik-nevrologik tadqiqotlar insult joylashuvi va PSD o'rtasida sezilarli bog'liqlik borligini aniqladi. Murakami va boshqalar insultdan keyingi depressiyaning chap frontal kortikal mintaqada, chap bazal ganglionda va miya sopi (12) da joylashgan infarktlar bilan bog'liqligini aniqladi.

PSD ning molekulyar biomarkerlari.

Monoaminlar. PiD bilan og'rigan bemorlarda 5-gidroksiindoleasetik kislotaning (5-HT metaboliti) suyuqlik konsentratsiyasi o'tkir insult shikastlanishlari bo'lgan depressiyaga uchragan va insult shikastlanishlari bo'lmagan depressiyaga uchramagan bemorlarga nisbatan sezilarli darajada past ekanligi aniqlandi. (13).

Natijalar serotonerjik mexanizmlar PSD patogenezida ishtirok etishini ko'rsatadi. Shunga qaramay, bu ma'lumotlar qo'shimcha tasdiqlanishi kerak.

O'sish omillari . To'plangan dalillar shuni ko'rsatadiki, miyadan kelib chiqadigan neyrotrofik omil (BDNF) depressiya **(14, 13)** va PSD (**15**) ning patofiz**i**olo**gi**k mexanizmlarida ishtirok etadi . To'rtta tadqiqotning meta-tahlili, shu jumladan 499 insultli bemorlar (**16**), insultdan k**e**yingi dastlabki davrda sarum BDNF kontsentratsiyasining sezilarli darajada pasayishi depressiya rivojlanishiga moyil ekanligini ko'rsatdi. Ma'lumotlar Noonan va boshqalarning oldingi meta-tahlil natijalariga mos keladi. (**17**).

Neyronal yallig'lanish belgilari

Hozirgi tadqiqotlar immun disfunktsiyasi insultdan keyingi depressiyaning patofiziologiyasi uchun juda muhim ekanligini aniqladi (18). Immunologik mexanizmlar kayfiyat bilan bog'liq bo'lgan miya sohalarida yallig'lanish bilan bog'liq hujayralar o'limini boshlashi mumkin (19). Shuning uchun, neyronal yallig'lanish belgilari PSD diagnostikasi uchun foydali bo'lishi mumkin. Tang va boshqalar. o'tkir insult bosqichida yuqori sezuvchanlik C-reaktiv oqsil (Hs-CRP) ning zardobdagi yuqori konsentratsiyasi PSD paydo bo'lishidan 6 oy o'tgach mustaqil ravishda moyilligini aniqladi (OR: 1,3, 95% CI: 1,2-1,5 va AUC qiymati 0,765, 95% CI: 139) 190). Shunga o'xshash natijalar Yang va boshqalar tomonidan nashr etilgan. (20). Mualliflar, shuningdek, PSD yuqori xavfi sarum Hs-CRP kontsentratsiyasi ≥ 0,85 mg / dL (OR: 7,8, 95% CI: 4,2-14,6) (20) bilan bog'liqligini aniqladilar.

Gipotalamus-gipofiz-buyrak usti bezining markerlari (HPA)

Doimiy HPA disregulyatsiyasi insultli bemorlarning 40% gacha bo'ladi. Giperkortizolemiya darajasi infarktning hajmi va joylashuvi bilan o'rtacha darajada aniqlanadi. Bemorlarda o’tkazilgan bosh miya insulti boshlanganidan keyin 1 oydan 1 yilgacha bo'lgan muddatda giperkortizolemiya PID bilan bog'liq (21).

Oksidlanish maxsulotlari shikastlanishining belgilari.

Ko'p dalillar insult oksidlovchi stress bilan birga kelishini ko'rsatadi. Ba'zi tadqiqotlar oksidlovchi stress va PID o'rtasidagi bog'liqlikni o'rgandi. Cichon va boshqalar. plazma oqsilining oksidlovchi shikastlanishi va PID (22) ehtimoli o'rtasidagi mumkin bo'lgan munosabatlarni baholadi . Shunga qaramay, PID va PID bo'lmagan insultdan keyingi bemorlarni aniqlash uchun yuqorida qayd etilgan oksidlovchi markerlarning o'ziga xosligi kengroq namunaviy tadqiqotlarda qo'llab-quvvatlanishi kerak. Shuning uchun ularni muntazam klinik amaliyot uchun tavsiya qilish mumkin emas.

Metabolitlar

Dalillar shuni ko'rsatadiki, qon zardobida o'tkir glyukoza konsentratsiyasi ishemik insultdan keyin PID ning prognozi bo'lishi mumkin. Qon tomiridan 12 oy o'tgach, PID ko'rsatkichi (Bek Depressiya Psixometriga ko'ra) qabul paytida qon zardobidagi glyukoza kontsentratsiyasi bilan ijobiy bog'liqlik ko'rsatdi (r = 0,32) (23). Mualliflar 126 mg / dL dan yuqori o'tkir glyukoza kontsentratsiyasi PID paydo bo'lishining prognozi bo'lishi mumkinligini aniqladilar.

Neyrofiziologik belgilar.

EEG . PID sub'ektlarida EEG murakkabligidagi anormalliklarni baholash uchun Zhang va boshqalar foydalanilgan (24). PID bilan og'rigan odamlarda insultdan keyingi depressiyaga uchramagan odamlar va sog'lom odamlar bilan solishtirganda miyaning butun sohalarida neyrofizologik murakkablik pastroq ekanligi ko'rsatildi. PID uchun skrining ko'rsatkichlari sifatida Lempel-Ziv Complexity parametrlari o'ziga xoslik, sezgirlik va aniqlikda 85% dan ortiqni ko'rsatdi (1-jadval). Ushbu tadqiqotda og'ir PID bemorlarining yo'qligi va insult joylashuvi va neyrofiziologik ma'lumotlar o'rtasidagi bog'liqlikning yo'qligi hozirgi vaqtda butun PID populyatsiyasi bo'yicha olingan ma'lumotlarni aniq tushintirishga imkon bermaydi. Vang va boshqalar. bazal gangliya infarkti bo'lgan PID sub'ektlarida miqdoriy EEG o'zgarishlarini o'rganib chiqdi (25). Chap yarim shardagi PID bilan og'rigan bemorlar frontal va markaziy hududlarda beta2 quvvati ortgan, o'ng yarim sharda PID esa asosan oksipital va temporal mintaqalarda teta va alfa quvvati ortgan.

Study	Type of study	Patients	N	Depression instrument	Results
Zhang et al. (108)	Case-control	PSD Non-PSD HC	21 22 15	DSM-IV, HDRS-24	PSD patients showed lower neural complexity compared with non-PSD and HC in the whole brain regions. In stroke patients, significant correlation was between HDRS and LZC in the whole brain regions, especially in frontal and temporal. LZC parameters used for PSD recognition possessed more than 85% in specificity, sensitivity and accuracy
Wang et al. (109)	Case-control	PSD Non-PSD	26 32	HDRS	For stroke subjects with left-side lesions, only increased beta2 power in frontal (F3 and F4) and left central (C4) areas was found in PSD patients. Stroke subjects with right-side lesions showed increased theta power in occipital (O1 and O2) and frontal-temporal (F7, T4, T5, and T6) areas, as well as increased alpha power in left occipital (O1) and temporal areas (T4, T5, and T6). Beta2 power in central regions and parietal regions of the right hemisphere showed strong discrimination of PSD with left hemispheric lesion, for which the AUC values reached 0.8. Theta power showed strong discrimination of PSD with right hemispheric lesion in most of the right hemisphere (F4, C4, P4, O2, T4, T6) and in prefrontal and temporal regions of the left hemisphere (FP1, F7, T3, T5), for which the AUC values reached 0.8. No significant association was found between any of the frequency power and depressive severity in PSD subjects with lesions in the right or left hemisphere
Zhang et al. (77)	Case-control	PSD Non-PSD HC	28 39 40	HDRS	Poststimulus latencies of the N200 and P300 ERP components were significantly prolonged in patients with PSD compared to non-PSD and HC (FN200 = 152.52, P = 0.008; FP300 = 51.19, P = 0.002), while the P300 amplitude was significantly reduced in PSD compared to other groups (FP300 = 40.63, P = 0.005)
Wenzhen et al. (110)	Case-control	PSD Non-PSD	85 85	HDRS	The N400 incubation periods and averaged amplitudes of the PSD patients were statistically different from those of the control (t = 15.79, P < 0.01 and t = 11.22, P < 0.01, respectively)

DSM, Diagnostic and Statistical Manual; HC, healthy controls; HDRS, Hamilton Depression Rating Scale; LZC, Lempel-Ziv Complexity; Non-PSD, poststroke patients without depression; PSD, poststroke patients with depression.

Transkranial neyrosonografiya (TNS)

Bu miyaning chuqur tuzilmalarini, jumladan, bosh miya asosini tekshirish uchun noinvazuv, arzon va takrorlanuvchi usulda baholash imkoniyatini beruvchi diagnostika usuli hisoblanadi. Ayniqsa, bosh miya asosining gipoexogenligi (to‘qimalarning past exostrukturaligi) markaziy nerv tizimining neyrodegenerativ o‘zgarishlari, neyrotrofik omillarning yetishmovchiligi va serotoninergik yo‘llarning disfunktsiyasi bilan bog‘liqligi tekshiruvlarda aniqlangan. Bu jarayonlar, o‘z navbatida, depressiv holatlar patogenezida asosiy rol o‘ynaydi. Bosh miya asosining asosiy funktsional ahamiyatli jihati bu fiziologik serotonergik va noradrenergik yo‘llarning mavjudligi bilan ifodalanadi ya’ni kayfiyatni tartibga solishda hal qiluvchi rol o‘ynaydi. Bu tuzilmalarning shikastlanishi (masalan, insult va pontin yoki bosh miya asosi infarktlari) insultdan keyingi depressiv holatlarni kelib chiqishi bilan kuchli bog‘liqligi mavjud.

Transkranial neyrosonografiyaning afzalliklari: MRT yoki KT bilan solishtirganda, TNS ko‘p bemorlar uchun qulay, tez va dinamik kuzatish imkonini beradi.

Gipoexogenlikning prognostik bosh miya asosida aniqlangan gipoexogenlik ko‘rsatkichlari bemorlarda PID rivojlanish xavfini erta bashorat qilish uchun potentsial marker sifatida foydalanish mumkin.

G’arb mamlakatlarida olin borilgan tadqiqotlar (Berg , 2020) bosh miya asosi ishemiyasi bo‘lgan bemorlarda PID bilan kasallanish chastotasining 3 baravar yuqori ekanligini ko‘rsatdi.

Transkranial neyrosonografiya tekshiruvi usuli miya asosining exostrukturalarini baholash orqali PID rivojlanishini 85% sezgirlikda bashorat qilish imkonini beradi. Bosh miya asosi gipoexogenlik zona lokalizatsiyasi klinik ahamiyatga ega hisoblanib (masalan, rostral brainstem) insultdan keyingi depressive holatlarning og‘irligi bilan chambarchas bog‘liqdir. (Kremer et al., 2023).

XULOSA

Bosh miya asosining gipoexogenligi insultdan keyingi depressiv holatlarning neyrobiologik asosini tushunish va shaxsiylashtirilgan profilaktika usullarini ishlab chiqish uchun muhimdir. Transkranial Neyrosonografiya insultdan keyingi depressiv holatlarni aniqlashda boshqa biomarkerlarga qarganda bir qancha afzalliklarga ega bo’lib: noinvazuv, arzon va takrorlanuvchi usulda baholash imkoniyatini beruvchi profilaktik- diagnostika usuli sifatida foydalanish mumkin. TNS orqali aniqlangan anomaliyalarga ega bemorlarga erta antidepressant terapiyasi yoki reabilitatsiya dasturlarini joriy qilish samaradorligi yuqori bo‘lishi mumkin.

Foydalanilgan Adabiyotlar.

1. Villa RF, Ferrari F, Moretti A: Post-stroke depression: mechanisms and pharmacological treatment. Pharmacol Ther. 2018, 184:131-44. 10.1016/j.pharmthera. 2017.11.005
2. Berg. D. (2020). *Brainstem lesions and post-stroke depression. Neurology.
3. Walter.T.J. (2021). Neuroinflammation and hypoechogenicity in brainstem. Journal of Neuroimaging.
4. Li, X. (2022). TNS-based prediction of poststroke depression. Stroke Research and Treatment.
5. 14. Li J, Oakley LD, Li Y, Luo Y. O'tkir insultli bemorda insultdan keyingi depressiyaning dastlabki belgilarini baholash uchun klinik chorani ishlab chiqish va dastlabki tasdiqlash. *J Clin Nurs.* (2017) 27:784–94. doi: 10.1111/jocn.14099 PubMed Abstrakt | CrossRef to'liq matn | Google olimi
6. 15. Ojagbemi A, Owolabi M, Akinyemi J, Ovbiagele B. Depressiya uchun insultga xos yangi skrining vositasini taklif qilish: konstruksiyaning haqiqiyligi va ishonchliligini tekshirish. *eNeurol Sci.* (2017) 9:14–8. doi: 10.1016/j.ensci.2017.10.002 PubMed Abstrakt | CrossRef to'liq matn | Google olimi
7. Robinson RG, Xorxe RE. Qon tomiridan keyingi depressiya: sharh. *Am J Psixiatriya* (2016) 173:221–31. doi: 10.1176/appi.ajp.2015.15030363
8. Yang S, Hua P, Shang X, Cui Z, Zhong S, Gong G va boshqalar. Qon tomiridan keyingi depressiya uchun muhim xavf omili: depressiya bilan bog'liq pastki tarmoq. *J Psixiatriya Neurosci.* (2015)40:259–68. doi: 10.1503/jpn.140086 PubMed Abstrakt | CrossRef to'liq matn | Google olimi
9. Chjan Y, Zhao H, Fang Y, Vang S, Zhou H. Lezyonning joylashuvi, jinsiy aloqa va insultdan keyingi depressiya o'rtasidagi bog'liqlik: meta-tahlil. *Miyaning xatti-harakati.* (2017) 7: e00788. doi: 10.1002/brb3.788 PubMed Abstrakt | CrossRef to'liq matn | Google olimi
10. A House, Dennis M, Warlow C, Hawton K, Molyneux A. Qon tomiridan keyin kayfiyatning buzilishi va ularning lezyon joyiga aloqasi. KT tekshiruvi. *Miya* (1990) 113 (Pt 4): 1113-29. PubMed Abstrakt | Google olimi
11. Gozzi SA, Wood AG, Chen J, Vaddadi K, Phan TG. Qon tomiridan keyingi depressiyani ko'rish ko'rsatkichlari: vokselga asoslangan tahlilda uslubiy omillar. *BMJ Open* (2014) 4: e004948. doi: 10.1136/bmjopen-2014-004948 PubMed Abstrakt | CrossRef to'liq matn | Google olimi
12. Murakami T, Hama S, Yamashita H, Onoda K, Kobayashi M, Kanazawa J va boshqalar. Insultdan keyingi affektiv va apatik depressiya bilan bog'liq neyroanatomik yo'llar. *Am J Geriatr Psixiatriya* (2013) 21: 840-7. doi: 10.1016/j.jagp.2013.01.057 PubMed Abstrakt | CrossRef to'liq matn | Google olimi

13. Bryer JB, Starkstein SE, Votypka V, Parikh RM, Price TR, Robinson RG. Reduction of CSF monoamine metabolites in poststroke depression: a preliminary report. *J Neuropsychiatry Clin Neurosci.* (1992) 4:440–2. doi: 10.1176/jnp.4.4.440 PubMed Abstract | CrossRef Full Text | Google Scholar

14. Levada OA, Cherednichenko NV. Brain-derived Neurotrophic Factor (BDNF): neurobiology and marker value in neuropsychiatry. *Lik Sprava* (2015) 15–25. PubMed Abstract | Google Scholar

15. Li Y, Peng C, Guo X, You JJ, Yadav HP. Expression of brain-derived neurotrophic factor and tyrosine kinase B in cerebellum of poststroke depression rat model. *Chin Med J.* (2015) 128:2926–31. doi: 10.4103/0366-6999.168058

16. Xu HB, Xu YH, He Y, Xue F, Wei J, Zhang H, et al. Decreased serum brain-derived neurotrophic factor may indicate the development of poststroke depression in patients with acute ischemic stroke: a meta-analysis. *J Stroke Cerebrovasc Dis.* (2017) 27:709–15. doi: 10.1016/j.jstrokecerebrovasdis.2017.10.003 PubMed Abstract | CrossRef Full Text | Google Scholar

17. Noonan K, Carey LM, Crewther SG. Meta-analyses indicate associations between neuroendocrine activation, deactivation in neurotrophic and neuroimaging markers in depression after stroke. *J Stroke Cerebrovasc Dis.* (2013) 22:e124–35. doi: 10.1016/j.jstrokecerebrovasdis.2012.09.008 PubMed Abstract | CrossRef Full Text | Google Scholar

18. Becker KJ. Inflammation and the Silent Sequelae of Stroke. *Neurotherapeutics* (2016) 13:801–10. doi: 10.1007/s13311-016-0451-5 PubMed Abstract | CrossRef Full Text | Google Scholar

19. Pascoe MC, Crewther SG, Carey LM, Crewther DP. Inflammation and depression: why poststroke depression may be the norm and not the exception. *Int J Stroke* (2011) 6:128–35. doi: 10.1111/j.1747-4949.2010.00565.x CrossRef Full Text | Google Scholar

20. Yang RR, Lu BC, Li T, Du YF, Wang X, Jia YX. The relationship between high-sensitivity C-reactive protein at admission and post stroke depression: a 6-month follow-up study. *Int J Geriatr Psychiatry* (2016) 31:231–9. doi: 10.1002/gps.4315 PubMed Abstract | CrossRef Full Text | Google Scholar

21. Mitchell AJ. Clinical implications of poststroke hypothalamo-pituitary adrenal axis dysfunction: a critical literature review. *J Stroke Cerebrovasc Dis.* (1997) 6:377–88. doi: 10.1016/S1052-3057(97)80038-9 PubMed Abstract | CrossRef Full Text | Google Scholar

22. Cichon N, Bijak M, Miller E, Niwald M, Saluk J. Poststroke depression as a factor adversely affecting the level of oxidative damage to plasma proteins during a

brain stroke. *Oxid Med Cell Longev* (2015) 2015:408745. doi: 10.1155/2015/408745 PubMed Abstract | CrossRef Full Text | Google Scholar

23. Ormstad H, Aass HC, Amthor KF, Lund-Sørensen N, Sandvik L. Serum levels of cytokines, glucose, and hemoglobin as possible predictors of poststroke depression, and association with poststroke fatigue. *Int J Neurosci.* (2012) 122:682–90. doi: 10.3109/00207454.2012.709892 PubMed Abstract | CrossRef Full Text | Google Scholar

24. Zhang Y, Wang C, Sun C, Zhang X, Wang Y, Qi H, et al. Neural complexity in patients with poststroke depression: a resting EEG study. *J Affect Disord.* (2015)188:310–8. doi: 10.1016/j.jad.2015.09.017 PubMed Abstract | CrossRef Full Text | Google Scholar

25. Wang C, Chen Y, Zhang Y, Chen J, Ding X, Ming D. Quantitative EEG abnormalities in major depressive disorder with basal ganglia stroke with lesions in different hemispheres. *J Affect Disord.* (2017) 215:172–178. doi: 10.1016/j.jad.2017.02.030 PubMed Abstract | CrossRef Full Text | Google Scholar