

ISSN: 3030-3877

AJCD
2024

<https://tadqiqot.uz/index.php/spjacd>



Annals of clinical disciplines



VOLUME 2, ISSUE 3/1

2025

ISSN 3030-3877

DOI Journal 10.26739/3030-3877

ANNALS OF CLINICAL DISCIPLINE

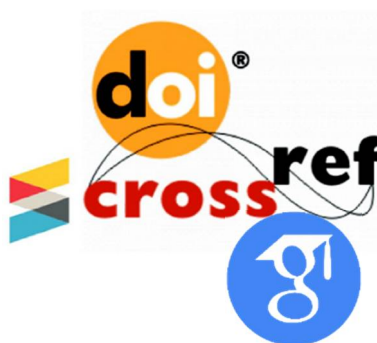
2 ЖИЛД, 3/1 СОН

АННАЛЫ КЛИНИЧЕСКИХ ДИСЦИПЛИН

ТОМ 2, НОМЕР 3/1

КЛИНИК ФАНЛАР ЙИЛНОМАСИ

VOLUME 2, ISSUE 3/1



ТОШКЕНТ-2025

ANNALS OF CLINICAL DISCIPLINE

АННАЛЫ КЛИНИЧЕСКИХ ДИСЦИПЛИН | КЛИНИК ФАНЛАР ЙИЛНОМАСИ

№3/1 (2025) DOI <http://dx.doi.org/10.26739/0000-0000-2025-3/1>

BOSH MUHARRIR: | ГЛАВНЫЙ РЕДАКТОР: | CHIEF EDITOR:

Sh. J. Teshayev

“Klinik fanlar yilnomasi” jurnali bosh muharriri, Buxoro davlat tibbiyot instituti rektori, t.f.d., professor

BOSH MUHARRIR O'RINBOSARI: | ЗАМЕСТИТЕЛЬ
ГЛАВНОГО РЕДАКТОРА: | DEPUTY CHIEF EDITOR:

D. A. Xasanova

“Klinik fanlar yilnomasi” jurnali bosh muharrir o'rinbosari, Buxoro davlat tibbiyot instituti anatomiya va klinik anatomiya kafedrası professori, DSc

РЕДАКЦИОННАЯ КОЛЛЕГИЯ:

- **U.K. Abdullayeva** - “Klinik fanlar yilnomasi” jurnali mas'ul kotibi, Buxoro davlat tibbiyot instituti fakultet va gospital terapiya, nefrologiya va gemodializ kafedrası dotsenti, DSc;
- **M.J. Sanoyeva** - Buxoro davlat tibbiyot instituti nevrologiya kafedrası dotsenti, DSc
- **A.G. Gadayev** - Toshkent tibbiyot akademiyasi 3-son ichki kasalliklar kafedrası professori, t.f.d.
- **A.R. Obloqulov** - Buxoro davlat tibbiyot instituti, yuqumli kasalliklar va bolalar yuqumli kasalliklari kafedrası mudiri, t.f.d., professor
- **D.A. Nabiyeva** - Toshkent tibbiyot akademiyasi, 1-son fakultet va gospital terapiya, kasb kasalliklari kafedrası mudiri, t.f.d., professor
- **Sh.T. O'roqov** - Buxoro davlat tibbiyot instituti xirurgik kasalliklar kafedrası mudiri, DSc, dotsent
- **M.M. Karimov** - Respublika ixtisoslashtirilgan terapiya va reabilitatsiya ilmiy-amaliy tibbiyot markazi “Gastroenterologiya” ilmiy laboratoriyasi boshlig'i, t.f.d., professor
- **N.U. Narzullayev** - Buxoro davlat tibbiyot instituti otorinilaringologiya kafedrası professori, DSc
- **G.N. Sobirova** - Toshkent tibbiyot akademiyasi reabilitatsiya va jismoniy tarbiya kafedrası professori, t.f.d.
- **F.S. Raupov** - Buxoro davlat tibbiyot instituti bolalar xirurgik kasalliklari kafedrası mudiri, DSc, dotsent
- **Sh.B. Axrorova** - Buxoro davlat tibbiyot instituti, nevrologiya kafedrası dotsenti, DSc.
- **V.R. Akramov** - Buxoro davlat tibbiyot instituti travmatologiya va neyroxirurgiya kafedrası mudiri, DSc, dotsent
- **I.K. Sadullojeva** - Buxoro davlat tibbiyot instituti bolalar kasalliklari propedevtikasi va bolalar nevrologiyasi kafedrası mudiri, DSc, dotsent

РЕДАКЦИОННЫЙ СОВЕТ:

- **G.J. Jarilkasimova** - Buxoro davlat tibbiyot instituti oilaviy shifokorlarni qayta tayyorlash kafedrası professori, DSc
- **U.S. Mamedov** - Buxoro davlat tibbiyot instituti onkologiya kafedrası mudiri, DSc, dotsent
- **A.A. Saidov** - Buxoro davlat tibbiyot instituti ortopedik stomatologiya va ortodontiya kafedrası professori DSc
- **N.N. Karimova** - Buxoro davlat tibbiyot instituti 3-son akusherlik va ginekologiya kafedrası mudiri, DSc, dotsent
- **U.K. Qayumov** - tibbiyot xodimlarini kasbiy malakasini oshirish markazi ichki kasalliklar kafedrası mudiri, t.f.d., professor
- **M.E. Raximova** - Toshkent tibbiyot akademiyasi, 3-son ichki kasalliklar kafedrası dotsenti, t.f.d.
- **R.I. To'raqulov** - Toshkent tibbiyot akademiyasi, 3-son ichki kasalliklar kafedrası professori, t.f.d.
- **Ch.S. Pavlov** - I.M. Sechenov nomidagi birinchi Moskva davlat tibbiyot universiteti terapiya kafedrası mudiri, t.f.d., professor
- **L.B. Novikova** - Rossiya Federatsiyasi Sog'liqni saqlash vazirligining “Janubiy Ural davlat tibbiyot universiteti” federal davlat byudjet oliy ta'lim muassasasi dermatovenerologiya kafedrası professori, t.f.d.
- **O.I. Letyayeva** - Rossiya Federatsiyasi Sog'liqni saqlash vazirligining “Janubiy Ural davlat tibbiyot universiteti” federal davlat byudjet oliy ta'lim muassasasi dermatovenerologiya kafedrası professori, t.f.d.
- **I.V. Reverchuk** - I.Kant nomidagi Boltiq federal universiteti psixonevrologiya va psixosomatika kafedrası mudiri, t.f.d., professor
- **Edip Gonullu** - Izmir Bakirchay universiteti anesteziya va reanimatsiya kafedrası dotsenti, t.f.d.
- **Eva Lietto** - Italiya Campania universiteti “Luigi Vanvitelli”ning tarjima tibbiyot fanlari kafedrası mudiri, t.f.d., professor

Журнал включен в перечень ВАК национальных научных изданий, рекомендуемых для публикации основных научных результатов диссертаций по медицинским наукам постановлением № 369/6 от 5 апреля 2025 г.

© Page Maker | Верстка | Саҳифаловчи: Хуршид Мирзахмедов

О журнале

Журнал зарегистрирован в Агентство информации и массовых коммуникаций при Администрации Президента Республики Узбекистан
№ С-239963 от 14 марта 2024 года

Адрес редакции: Республика Узбекистан, 200114,
г. Бухара, ул. Гиждуван, 23
Телефон: +998(65)2230050
Сайт: <https://tadqiqot.uz/index.php/spjacd>
e-mail: abumkur14@gmail.com

1. Abdullayeva U.K., Rakhimova M.B.	
Pomegranate seed oil for ulcerative colitis.....	7
2. Abdurakhmonova Q., Rakhimbaeva G.	
Inflammatory biomarkers and insulin resistance in acute ischemic stroke of different etiologies...	12
3. Boymuradov Sh.A., Khatamov U.A.	
Biomechanical and clinical analysis of atypical counterforces in maxillofacial trauma from electromechanical impacts.....	19
4. Baymuratova A.Ch.	
Tizza bo'g'imi ichki strukturasi jarohatlanishlarini utt orqali klinik baholash va fizioterapevtik reabilitatsiyani optimallashtirish	26
5. Boboyev Sh.R., Saidov G.N., Keldiyorova Z.D.	
Tajribaviy oshqozon-ichak traktining sirka kislotasi bilan kimyoviy kuyishida yo'g'on ichak devoridagi morfologik o'zgarishlarning qiyosiy tavsifi.....	32
6. Jumaeva G.A.	
Klinik farmakologiyada innovatsion o'quv modeli: talaba markazli yondashuv.....	38
7. Keldiyorova Z.D., Zaripova S.O.	
O'tkir respirator virusli infeksiyalar va torch- infeksiyasi bilan kasallangan bolalarda klinik-laborator o'zgarishlar.....	43
8. Khalikova F.Sh.	
Morphological structure of the thyroid gland of white rats in chemotherapy of breast cancer.....	51
9. Mirzayeva M.R., Yodgorova M.Sh.	
Latent course of liver fibrosis in patients with chronic hepatitis B: comparative evaluation of noninvasive diagnostic methods (APRI, FIB-4 and fibroscan).....	58
10. Muradimova A.R., Madjidova Y.N., Khidoyatova D.N., Sharipov F.R.	
Bioacoustic correction of cognitive impairment in stroke patients.....	69
11. Najmutdinova D.Q., Ismoilova Sh.S.	
2-tur natriy-glyukoza kotransportyori ingibitorlarini qo'llashda jigar fibroz ko'rsatkichlari va insulinga rezistentlik dinamikasi.....	74
12. Nazirxujayev F.A.	
Farg'ona viloyati aholisi orasida "O'rta yer dengizi parxezi"ni qo'llash orqali surunkali pankreatit xurujlari profilaktikasini optimallashtirish.....	79
13. Nurmatova O.A.	
Bolalarda nefrotik sindromning profilaktikasini takomillashtirish yo'llari (Farg'ona viloyati misolida).....	84
14. Ortiqboev J.O.	
Molecular genetic analysis of the renin–angiotensin system in chronic kidney disease.....	89

15. Oxunova M.J.	
Farg'ona viloyatida 3 yoshgacha bolalarda pnevmoniya rivojlanishiga ta'sir etuvchi asosiy xavf omillari.....	95
16. Rasulova N.R. Temirova N.R.	
Tajribaviy kimyoterapiya natijasida ayrisimon bezdagi morfologik o'zgarishlarning qiyosiy tahlili.....	99
17. Raxmatullayeva G.K., Axmedova N.A., Kasimova M.B., Xudayberganova N.X.	
Oshqozon-ichak kasalliklarida so'rovnomas o'tkazish va uni o'rganish.....	108
18. Subkhanova Z.S., Raimkulova N.R.	
Interrelationship between gastroesophageal reflux disease and respiratory diseases: general pathogenetic mechanisms and modern research findings.....	112
19. Subxanova Z.S., Raimkulova N.R.	
Gastroezofageal reflyuks kasalligi, surunkali obstruktiv o'pka kasalligi va bronxial astma: komorbidlikning dolzarb masalalari.....	115
20. Temirova M.B., Kasimova M.B., Axmedova N.A.	
Metabolik assotsirlangan jigar yog' kasalligida bemorlarning tana tuzilishini bioimpedance spektroskopiya yordamida baholash.....	118
21. Tukhtaeva N.Kh., Musayev H.N.	
Development of gastropathy against the background of nonsteroidal anti-inflammatory drugs in patients with rheumatoid arthritis against the background of virulent genes of helicobacter pylori.....	123
22. Абдуллаева У.К., Рахимова М.Б.	
Ярали колит хавф омиллари: замонавий қарашлар.....	128
23. Абдулхаков И.У., Абдулхаков М.И.	
Общеврачебная практика как эффективная модель профилактики неинфекционных заболеваний.....	132
24. Адизова С.Р., Носирова З.Ж.	
Преэклампсия оғирлашувида гемостаз тизимининг таъсири.....	143
25. Акрамов О.З., Аблязов О.В., Кадыров Ш.У.	
Инновационные подходы нейровизуализации при опухолях функционально важных участков мозга у детей.....	149
26. Аликулова Н.А., Ўринова Г.М.	
Мия инфаркти ва СМИ ўтказган беморларда клиник-функционал кўрсаткичлар динамикаси.....	156
27. Алимов А.А., Алимов А.В., Шарипов А.М., Шоикрамов Ш.Ш.	
Профиль инотропной поддержки у детей после коррекции врождённых пороков сердца: частота, дозы и динамика применения.....	166
28. Аллокулов Р.Р., Акрамов В.Р., Нуралиев Ф.Н.	
Кекса ва қари ёшдаги шахсларда гонартрознинг клиник-иммунологик жихатлари.....	172

29. Асадов Х.Ф.

Хирургия мигрени: сравнительный анализ двух хирургических методик лечения височной мигрени: нейроэктомия и декомпрессия ветви скуловисочного нерва.....182

30. Аскарров Ш.Ш., Салахитдинов Ш.Н.

Перспективные интервенционные технологии при тромботических осложнениях острого коронарного синдрома.....187

31. Гадаев А., Халимова Х., Гадаева Н.

Ўпканинг сурункали обструктив касаллиги ва артериал гипертензияда буйраклар дисфункцияси (адабиётлар шархи).....193

32. Гаффорова В.Ф., Ходжиева Д.Т.

Клинико-нейрофизиологические характеристики фебрильных судорог у детей.....200

33. Давлетова Ш.Б.

Роль микробиоты кишечника в развитии неврологических заболеваний у детей.....205

34. Даминов Б.Т., Ортикбоев Ж.О.

Уровень витаминов группы В и витамина Д у больных с хронической болезнью почек в зависимости от прогрессирования заболевания.....216

35. Джураев И.Б., Курбанов О.М., Бабаназаров У.Т.

Клинические проявления патологий желудка у больных в критическом состоянии в условиях искусственного дыхания.....221

36. Жалалова В.З.

Оптимизация восстановительных процессов при физических нагрузках с помощью спирулины: морфофункциональные и клинические аспекты.....229

37. Ибодуллаева Н.М.

Бачадон саратон олди касалликлари бўлган аёлларда неврологик ва психоэмоционал ўзгаришларнинг психосоматик жиҳатлари.....237

38. Иноятов А.Ш., Ражаматов Т.Р., Умаров Б.Я.

Функциональная и иммунологическая обоснованность применения щечного жирового лоскута при палатопластике в детском возрасте.....241

UDC 616.831-005.1-036.11:577.1

¹Abdurakhmonova Qutlibika, ²Rakhimbaeva Gulnora

Tashkent State Medical University, Tashkent, Uzbekistan

¹<https://orcid.org/0009-0006-2415-8218>²<https://orcid.org/0000-0002-5547-7610>**INFLAMMATORY BIOMARKERS AND INSULIN RESISTANCE IN ACUTE ISCHEMIC STROKE OF DIFFERENT ETIOLOGIES** <http://dx.doi.org/10.5281/zenodo.17352420>**ABSTRACT**

This study explores differences in inflammatory biomarkers in the acute period of ischemic stroke according to different etiologies and emphasizes the role of insulin resistance (IR) as a contributing metabolic factor. Serum levels of ICAM-1, hs-CRP, SII, and NLR were compared between stroke subtypes, and associations with IR were analyzed. Findings suggest that cardioembolic strokes exhibit higher inflammatory activity, whereas lacunar subtypes show lower biomarker levels. Moreover, IR was frequent among patients and correlated with elevated inflammatory markers, suggesting a synergistic role in worsening vascular damage.

Key words: acute ischemic stroke, insulin resistance, inflammation biomarkers, TOAST, cardioembolic stroke.

¹Абдурахмонова Кутлибика, ²Рахимбаева Гульнора

Ташкентский государственный медицинский университет, Ташкент, Узбекистан

¹<https://orcid.org/0009-0006-2415-8218>²<https://orcid.org/0000-0002-5547-7610>**ВОСПАЛИТЕЛЬНЫЕ БИОМАРКЕРЫ И ИНСУЛИНОРЕЗИСТЕНТНОСТЬ ПРИ ОСТРОМ ИШЕМИЧЕСКОМ ИНСУЛЬТЕ РАЗНОЙ ЭТИОЛОГИИ****АННОТАЦИЯ**

Это исследование посвящено изучению различий в уровнях воспалительных биомаркеров в остром периоде ишемического инсульта в зависимости от его различных этиологий и подчёркивает роль инсулинорезистентности (ИР) как значимого метаболического фактора. В работе сравнивались сывороточные уровни ICAM-1, hs-CRP, SII и NLR между подтипами инсульта, а также анализировались их ассоциации с ИР. Результаты показали, что при кардиоэмболическом инсульте наблюдается более выраженная воспалительная активность, тогда как при лакунарном подтипе уровни биомаркеров были ниже. Кроме того, у пациентов часто выявлялась ИР, которая коррелировала с повышенными воспалительными маркерами, что указывает на её синергическую роль в усугублении сосудистого повреждения.

Ключевые слова: острый ишемический инсульт, инсулинорезистентность, воспалительные биомаркеры, TOAST, кардиоэмболический инсульт.

¹Абдурахмонова Қутлибика, ²Рахимбаева Гулнора
Тошкент давлат тиббиёт университети, Тошкент, Ўзбекистон

¹<https://orcid.org/0009-0006-2415-8218>

²<https://orcid.org/0000-0002-5547-7610>

ТУРЛИ ЭТИОЛОГИЯЛИ ИШЕМИК ИНСУЛЬТДА ЯЛЛИҒЛАНИШ БИОМАРКЕРЛАРИ ВА ИНСУЛИН РЕЗИСТЕНТЛИГИ

АННОТАЦИЯ

Ушбу тадқиқот турли этиологияли ишемик инсультнинг ўткир даврида яллиғланиш биомаркерлари даражасидаги фарқларни ўрганади ҳамда инсулин резистентлигини (IR) муҳим метаболик омил сифатидаги ролини баҳолайди. Тадқиқотда ICAM-1, hs-CRP, SII ва NLR кўрсаткичлари инсультнинг турли типлари ўртасида таққосланди ва уларнинг IR билан боғлиқлиги таҳлил қилинди. Натижалар шуни кўрсатдики, кардиоэмболик инсультларда яллиғланиш фаоллиги юқорироқ бўлиб, лакунар инсультларда биомаркерлар даражаси пастроқ кузатилди. Бундан ташқари, беморларда IR тез-тез учраб, яллиғланиш маркерларининг ошиши билан боғлиқ экани аниқланди, бу эса унинг томир шикастланишини кучайтиришда синергетик роль ўйнашини кўрсатади.

Калит сўзлар: ўткир ишемик инсульт, инсулинга резистентлик, яллиғланиш биомаркерлари, TOAST, кардиоэмболик инсульт.

Background. In developed countries, stroke ranks as the third most common cause of death and is the leading contributor to long-term disability. Ischemic stroke, which constitutes about 87% of all strokes [5], is the predominant type. Acute stroke treatment, where feasible, involves reopening the blocked artery, typically through intravenous administration of recombinant tissue plasminogen activator (tPA). In cases of large intracranial vessel occlusion, this approach is supplemented with endovascular mechanical thrombectomy [17].

The TOAST (Trial of Org 10172 in Acute Stroke Treatment) classification system [1] is commonly used to categorize ischemic stroke causes. This system identifies large artery atherosclerosis (20%), cardioembolism (20%), small vessel occlusion (20%), other determined causes (5%, such as dissections or genetic disorders), and a cryptogenic category (30%) for strokes of unknown origin [3]. Identifying the cause of the stroke is crucial for initiating targeted secondary prevention strategies that effectively reduce the risk of recurrence. However, the presence of multiple potential causes in a single patient can create diagnostic uncertainty and complicate treatment prioritization. Biomarkers hold promise for assisting in determining the underlying cause.

Since the causes of stroke subtypes vary (e.g., atherosclerosis versus cardiac embolism), it is hypothesized that specific serum protein expression profiles could be associated with different stroke etiologies. While no biological marker has yet been identified that can definitively determine stroke cause [16], blood-based biomarkers may help improve classification of ischemic stroke mechanisms, enabling more precise therapeutic interventions. A better understanding of ischemic stroke pathophysiology could lead to enhanced prevention strategies as well as advances in diagnosis and treatment. Therefore, this study aims to explore potential links between different etiological stroke subtypes.

Growing evidence indicates that systemic inflammation and metabolic dysfunction, particularly insulin resistance (IR), are central to the pathophysiology of ischemic stroke. IR contributes to endothelial dysfunction, accelerates atherosclerosis, promotes platelet activation, and enhances pro-inflammatory cytokine release. Clinical studies demonstrate that IR not only increases the risk of first-ever ischemic stroke but also worsens outcomes and elevates the likelihood of

recurrence. Despite this, IR is rarely considered in biomarker-based stratification of ischemic stroke patients.

Purpose of the study. To compare the concentration of inflammatory biomarkers (ICAM-1, hs-CRP, SII, NLR) between different ischemic stroke subtypes and to evaluate the contribution of insulin resistance to biomarker changes and stroke outcomes.

Materials and methods. This observational study included 68 consecutive patients admitted with acute ischemic stroke (AIS) to the Department of Neurology, Tashkent Medical Academy, between January and June 2022. In addition, 32 age- and sex-matched healthy volunteers served as the control group. Eligible patients were adults (≥ 18 years) presenting within 24 hours of the first-ever ischemic stroke. AIS was confirmed by clinical evaluation and neuroimaging (CT or MRI). At admission, demographic and clinical data, vascular risk factors, and lifestyle habits were collected through structured questionnaires and chart reviews. Stroke severity was assessed using the National Institutes of Health Stroke Scale (NIHSS), and functional outcome during hospitalization was determined using the modified Rankin Scale (mRS). Stroke subtype classification followed the TOAST criteria, which divide ischemic strokes into large artery atherosclerosis (LAA), cardioembolism, small vessel occlusion, other determined etiology, and cryptogenic stroke. Peripheral venous blood was obtained within 24 hours of admission. Serum was separated by centrifugation within 30 minutes and stored at -20°C until analysis. High-sensitivity C-reactive protein (hs-CRP) and intercellular adhesion molecule-1 (ICAM-1) concentrations were determined using commercial ELISA kits (AO “Vector-Best,” Russia; BioVendor R&D, Czech Republic). The systemic immune-inflammation index (SII) and neutrophil-to-lymphocyte ratio (NLR) were calculated from routine complete blood counts. Fasting blood glucose and insulin were measured in all AIS patients. Homeostasis Model Assessment of Insulin Resistance (HOMA-IR) was calculated. HOMA-IR value >2.5 was considered evidence of insulin resistance.

Results. Overall, 68 patients suffering from acute ischemic stroke (AIS) with a mean age of 63.8 years were included in the study. The mean systolic blood pressure (SBP) was 154.4 ± 28.6 mmHg, and the mean diastolic pressure was 91.8 ± 7.9 mmHg. Hypertension was present in all patients (100%), while diabetes mellitus was observed in 8 (11.8%) and atrial fibrillation in 6 (8.8%). Coronary heart disease was found in 14 patients (20.6%).

Insulin resistance (IR), assessed by the HOMA-IR index, was detected in **28 patients (41.2%)**. Importantly, a considerable proportion of patients with IR had no prior diagnosis of diabetes, confirming the presence of subclinical metabolic dysfunction. The mean HOMA-IR across the entire cohort was 2.8 ± 1.1 , compared to 1.6 ± 0.5 in the control group ($p < 0.01$).

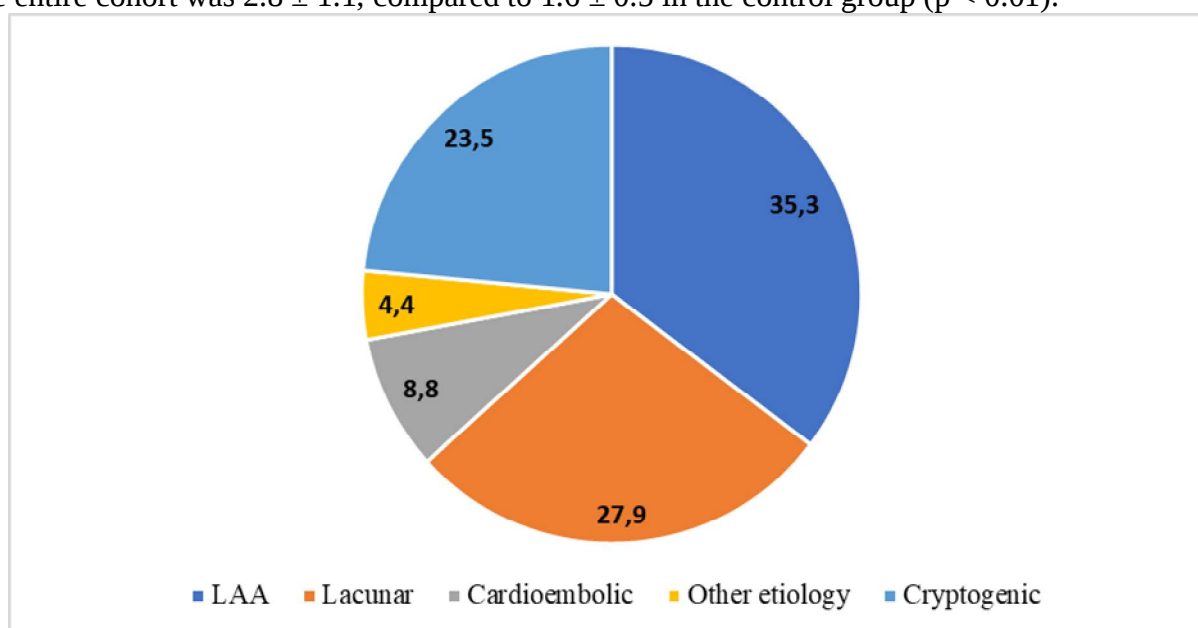


Figure 1. Frequency of stroke subtypes

When analyzed according to TOAST subtypes, **cardioembolic strokes** demonstrated the highest inflammatory activity, with elevated ICAM-1 (328.47 ± 196.83 ng/ml), hs-CRP (4.75 ± 2.26 mg/L), SII (2006.33 ± 1769.41), and NLR (9.03 ± 8.27). **Lacunar strokes** showed the lowest biomarker concentrations across all parameters. Large artery atherosclerosis (LAA) and cryptogenic strokes presented intermediate values (Table 1).

Insulin resistance was most frequent in **LAA (50%)** and **cardioembolic (50%)** subtypes, while only 23.7% of lacunar stroke patients demonstrated IR. The presence of IR was consistently associated with higher biomarker levels: patients with IR had significantly higher ICAM-1 ($p < 0.01$), hs-CRP ($p < 0.05$), and NLR ($p < 0.05$) compared to non-IR patients within the same subtype.

Table 1

Concentration of biomarkers and insulin resistance according to ischemic stroke subtypes

Biomarkers / HOMA-IR	LAA	Cardioembolic	Lacunar	Other etiology	Cryptogenic	Control group
ICAM-1 (ng/ml)	$210.13 \pm 167.8^*$	$328.47 \pm 196.83^*$	$151.64 \pm 47.94^*$	$194.07 \pm 100.5^*$	$200.92 \pm 94.75^*$	76.5 ± 17.55
hs-CRP (mg/L)	$3.05 \pm 1.68^*$	$4.75 \pm 2.26^*$	$2.3 \pm 1.17^*$	$2.98 \pm 1.37^*$	$3.47 \pm 2.04^*$	0.98 ± 0.23
SII	$1134.99 \pm 1333.89^*$	$2006.33 \pm 1769.41^*$	$554.6 \pm 470.6^*$	$732.57 \pm 695.3^*$	$911.37 \pm 815.9^*$	257.7 ± 45.66
NLR	$4.98 \pm 5.88^*$	$9.03 \pm 8.27^*$	$3.03 \pm 2.58^*$	$3.55 \pm 2.57^*$	$4.03 \pm 3.35^*$	1.72 ± 0.43
HOMA-IR (mean)	$3.1 \pm 1.2^*$	$3.3 \pm 1.1^*$	2.1 ± 0.8	2.5 ± 0.9	2.6 ± 1.0	1.6 ± 0.5
IR prevalence (%)	50%	50%	23.7%	33.3%	37.5%	9.3%

* $p < 0.01$ vs. control

Functionally, IR was linked to worse short-term outcomes: 67.9% of patients with IR had an mRS ≥ 3 at discharge compared to 38.1% of those without IR ($p < 0.05$). This highlights the synergistic role of metabolic and inflammatory mechanisms in determining stroke severity.

Parameter	IR (n=28)	Non-IR (n=40)	p-value
Mean ICAM-1 (ng/ml)	298.6 ± 172.4	184.7 ± 95.2	<0.01
Mean hs-CRP (mg/L)	4.12 ± 1.87	2.71 ± 1.39	<0.05
Mean NLR	6.82 ± 4.44	3.95 ± 2.73	<0.05
HOMA-IR	3.3 ± 1.0	1.9 ± 0.6	<0.001
Poor outcome (mRS ≥ 3 at discharge)	67.9%	38.1%	<0.05

Explanation *- $p < 0.001$

Point estimates presented in the following can be interpreted as the mean difference between a specific etiology. Inflammatory biomarker's levels were significantly higher in the cardioembolic stroke subjects compared with the large vessel atherosclerotic subtype. Furthermore, inflammatory biomarkers levels were significantly lower in the lacunar subtype.

Discussion. The present study demonstrated significant variations in inflammatory biomarker levels among ischemic stroke subtypes. Cardioembolic strokes were characterized by markedly elevated concentrations of ICAM-1, hs-CRP, SII, and NLR, whereas lacunar strokes displayed the lowest values. These findings support previous reports that immune activation and systemic inflammation differ according to stroke mechanisms, reflecting distinct underlying pathophysiological processes [6,10,12,18].

A key addition in this study was the evaluation of insulin resistance (IR). We observed IR in more than 40% of patients, including those without a history of diabetes mellitus. Importantly, IR was associated with significantly higher levels of ICAM-1, hs-CRP, and NLR, irrespective of the stroke subtype, and was linked to unfavorable functional outcomes at discharge. These observations are in agreement with earlier research indicating that IR accelerates atherosclerosis, promotes systemic inflammation, and contributes to plaque instability [7,21,23].

The biological plausibility of these findings is well supported. IR impairs endothelial insulin signaling, leading to increased expression of adhesion molecules such as ICAM-1 and VCAM-1 [19]. In addition, IR enhances the release of pro-inflammatory cytokines (IL-6, TNF- α), which elevate circulating CRP and promote leukocyte activation [8,22]. These mechanisms explain the synergistic relationship between IR and elevated inflammatory biomarkers in acute ischemic stroke.

Our findings are consistent with studies showing that biomarkers like hs-CRP, NLR, and ICAM-1 may differentiate between stroke etiologies [11,14,15]. Moreover, they extend prior work by demonstrating that metabolic dysfunction, specifically IR, amplifies these inflammatory processes and worsens short-term prognosis. Taken together, these results underscore the importance of integrating metabolic and inflammatory markers into stroke risk assessment and management.

Several limitations should be acknowledged. The sample size was relatively modest, and IR was assessed only once in the acute phase, which may not capture temporal fluctuations. Moreover, residual confounding by obesity, lipid abnormalities, or glycemic variability cannot be excluded. Further large-scale, prospective studies are needed to confirm these findings and to evaluate the role of therapeutic interventions targeting IR in ischemic stroke patients.

Conclusion. This study highlights that inflammatory biomarker profiles differ significantly between ischemic stroke subtypes and that the coexistence of insulin resistance amplifies systemic inflammation and worsens functional outcomes. Cardioembolic strokes exhibited the highest biomarker levels, while lacunar strokes had the lowest, and the presence of IR was consistently associated with increased inflammatory activity across all subtypes.

Our results suggest that routine assessment of IR (e.g., via HOMA-IR) in acute ischemic stroke could serve as a simple and cost-effective tool to identify high-risk patients. Combining inflammatory biomarkers with metabolic indicators may improve etiological classification, prognostic accuracy, and guide more personalized prevention and treatment strategies.

In conclusion, insulin resistance represents an important metabolic factor in the pathophysiology of ischemic stroke. Incorporating IR into biomarker-based models could enhance patient stratification, optimize secondary prevention, and open new therapeutic opportunities [7,19,21,23].

References

1. Adams HP Jr, Bendixen BH, Kappelle LJ, et al. Classification of subtype of acute ischemic stroke. Definitions for use in a multicenter clinical trial. TOAST. Trial of Org 10172 in acute stroke treatment. *Stroke*. 1993;24(1):35-41.
2. Ageno W, Finazzi S, Steidl L, et al. Plasma measurement of D-dimer levels for the early diagnosis of ischemic stroke subtypes. *Arch Intern Med*. 2002;162(22):2589-2593.
3. Amarenco P, Bogousslavsky J, Caplan LR, Donnan GA, Hennerici MG. Classification of stroke subtypes. *Cerebrovasc Dis*. 2009;27(5):493-501.

4. Banks JL, Marotta. CA. Outcomes validity and reliability of the modified Rankin scale: implications for stroke clinical trials: a literature review and synthesis. *Stroke*. 2007;38(3):1091-1096.
5. Benjamin EJ, Muntner P, Alonso A, et al. Heart disease and stroke statistics–2019 update: a report from the American Heart Association. *Circulation*. 2019;139(10):e56-e528.
6. Boos CJ, Anderson RA, Lip. GY. Is atrial fibrillation an inflammatory disorder? *Eur Heart J*. 2006;27(2):136-149.
7. Cai Z, He W, Zhuang F- J, et al. The role of high high- sensitivity C- reactive protein levels at admission on poor prognosis after acute ischemic stroke. *Int J Neurosci* 2019;129:423–9.
8. Eikelboom JW, Hankey GJ, Baker RI, et al. C- reactive protein in ischemic stroke and its etiologic subtypes. *J Stroke Cerebrovasc Dis* 2003;12:74–81.
9. Ertl M, Meisinger C, Linseisen J, Baumeister SE, Zickler P, Naumann M. Long-term outcomes in patients with stroke after in-hospital treatment-study protocol of the prospective stroke cohort Augsburg (SCHANA study). *Medicina (Kaunas)*. 2020;56(6):280.
10. Guo Y, Lip GY, Apostolakis. S. Inflammation in atrial fibrillation. *J Am Coll Cardiol*. 2012;60(22):2263-2270.
11. Harpaz D, Seet RCS, Marks RS, Tok AIY. Blood-based biomarkers are associated with different ischemic stroke mechanisms and enable rapid classification between cardioembolic and atherosclerosis etiologies. *Diagnostics (Basel)*. 2020;10(10):804.
12. Jickling GC, Sharp. FR. Biomarker panels in ischemic stroke. *Stroke*. 2015;46(3):915-920.
13. Kasner SE, Chalela JA, Luciano JM, et al. Reliability and validity of estimating the NIH stroke scale score from medical records. *Stroke*. 1999;30(8):1534-1537.
14. Laloux P, Galanti L, Jamart. J. Lipids in ischemic stroke subtypes. *Acta Neurol Belg*. 2004;104(1):13-19.
15. Liu LB, Li M, Zhuo WY, Zhang YS, Xu AD. The role of hs-CRP, Ddimer and fibrinogen in differentiating etiological subtypes of ischemic stroke. *PloS One*. 2015;10(2):e0118301.
16. Montaner J, Ramiro L, Simats A, et al. Multilevel omics for the discovery of biomarkers and therapeutic targets for stroke. *Nat Rev Neurol*. 2020;16(5):247-264.
17. Powers WJ, Rabinstein AA, Ackerson T, et al. Guidelines for the early management of patients with acute ischemic stroke: 2019 update to the 2018 guidelines for the early management of acute ischemic stroke: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke*. 2019;50(12):e344-e418.
18. Sonderer J, Katan Kahles. M. Aetiological blood biomarkers of ischaemic stroke. *Swiss Med Wkly*. 2015;145:w14138.
19. Stanimirovic DB, Wong J, Shapiro A, et al. Increase in surface expression of ICAM-1, VCAM- 1 and E- selectin in human erebromicrovascular endothelial cells subjected to ischemia-like insults. *Acta Neurochir Suppl* 1997;70:12–6.
20. von Elm E, Altman DG, Egger M, et al. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement: guidelines for reporting observational studies. *PLoS Med*. 2007;4(10):e296.
21. Wei P, Han B, Zhang W- J, et al. Effect of Ticagrelor on the serum level of Hs-CRP, ESM- 1 and short- term prognosis of patients with acute STEMI. *Exp Ther Med* 2017;13:604–8.
22. Winbeck K, Poppert H, Etgen T, et al. Prognostic relevance of early serial C- reactive protein measurements after first ischemic stroke. *Stroke* 2002;33:2459–64.
23. Yu B, Yang P, Xu X, et al. C- reactive protein for predicting all- cause mortality in patients with acute ischemic stroke: a meta- analysis. *Biosci Rep* 2019;39:BSR20181135.
24. Yuan BB, Luo GG, Gao JX, et al. Variance of serum lipid levels in stroke subtypes. *Clin Lab*. 2015;61(10):1509-1514. doi:10.7754/ clin.lab.2015.150118

25. Zambrelli E, Emanuele E, Marcheselli S, Montagna L, Geroldi D, Micieli G. Apo(a) size in ischemic stroke: relation with subtype and severity on hospital admission. *Neurology*. 2005;64(8):1366-1370.

ANNALS OF CLINICAL DISCIPLINE

АННАЛЫ КЛИНИЧЕСКИХ ДИСЦИПЛИН КЛИНИК ФАНЛАР ЙИЛНОМАСИ

Научно-практический журнал по всем
направлениям медицины
основан в 2024 году

Бухарским государственным
медицинским институтом

Выходит один раз в 3 месяца

Учредитель Бухарский государственный
медицинский институт