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Самарканд, Узбекистан**ДИАГНОСТИЧЕСКИЕ АСПЕКТЫ ЭНДОТЕЛИАЛЬНОЙ И КОРОНАРНОЙ МИКРОВАСКУЛЯРНОЙ ДИСФУНКЦИИ ПРИ ИШЕМИЧЕСКОЙ БОЛЕЗНИ СЕРДЦА (ОБЗОР ЛИТЕРАТУРЫ)****For citation:** Abdullayeva Z.A., Tashkenbaeva E.N., Rasulova D.O., Alimov F.O. Diagnostic aspects of endothelial and coronary microvascular dysfunction in ischemic heart disease (literature review). Journal of cardiorespiratory research. 2026, vol 7, issue 3.<http://dx.doi.org/10.26739/2181-0974/2026/7/3/1>**АННОТАЦИЯ**

Коронарная микроваскулярная дисфункция (КМД) и эндотелиальная дисфункция — ключевые механизмы ишемии миокарда у больных хроническим коронарным синдромом (ХКС) без обструктивного поражения коронарных артерий (ANOCA/INOCA), выявляемые у 30–50% таких пациентов и повышающие риск сердечно-сосудистых событий в 2–2,5 раза. Цель обзора — систематизировать клинические, биомаркерные и генетические предикторы КМД. Проанализированы публикации PubMed, Scopus, eLibrary, КиберЛенинка за 2015–2026 гг. Систематизированы клинические предикторы (женский пол, СД, метаболический синдром, эпикардиальное ожирение), биомаркеры эндотелиальной дисфункции и воспаления (ADMA, эндотелин-1, IL-6, hs-CRP), новые маркеры (sTREM2, FT4/FT3, GDF-15), генетические полиморфизмы (NOS3, ACE, EDN1/PHACTR1) и методы диагностики (CFR, IMR, ПЭТ). Приведена статистика ВОЗ и Республики Узбекистан. Комплексная оценка данных предикторов перспективна для персонализации диагностики ХКС, в том числе в узбекской популяции.

Ключевые слова: коронарная микроваскулярная дисфункция; эндотелиальная дисфункция; хронический коронарный синдром; ANOCA; INOCA; биомаркеры; генетические полиморфизмы.

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SamSMU, Samarkand, Uzbekistan**Alimov Fozilbek Odilovich**Resident of Master's program of the Department of Internal Medicine and Cardiology №2,
SamSMU, Samarkand, Uzbekistan**DIAGNOSTIC ASPECTS OF ENDOTHELIAL AND CORONARY MICROVASCULAR DYSFUNCTION IN ISCHEMIC HEART DISEASE (LITERATURE REVIEW)****ANNOTATION**

Coronary microvascular dysfunction (CMD) and endothelial dysfunction are key mechanisms of myocardial ischemia in chronic coronary syndrome (CCS) patients without obstructive coronary disease (ANOCA/INOCA), found in 30–50% of cases and doubling cardiovascular risk. The aim was to systematize clinical, biomarker and genetic predictors of CMD based on PubMed, Scopus, eLibrary sources (2015–2026). Clinical

predictors, endothelial and inflammatory biomarkers (ADMA, endothelin-1, IL-6, hs-CRP), emerging markers (sTREM2, FT4/FT3, GDF-15), genetic polymorphisms (NOS3, ACE, EDN1/PHACTR1) and diagnostic modalities (CFR, IMR, PET) were systematized. Global and Uzbekistan statistics are presented. Integrated assessment of these predictors is promising for personalizing CCS diagnosis, including in the Uzbekistan population.

Keywords: coronary microvascular dysfunction; endothelial dysfunction; chronic coronary syndrome; ANOCA; INOCA; biomarkers; genetic polymorphisms.

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YURAK ISHEMIK KASALLIGIDA ENDOTELIAL VA KORONAR MIKROVASKULAR DISFUNKSIYANI TASHXISLASH JIHATLARI (ADABIYOTLAR SHARHI)

ANNOTATSIYA

Koronar mikrovaskulyar disfunksiya (KMD) va endotelial disfunksiya obstruktiv koronar kasalliksiz surunkali koronar sindromli (SKS) bemorlarda (ANOCA/INOCA) miokard ishemiyasining asosiy mexanizmlari bo'lib, bemorlarning 30–50% ida aniqlanadi va yurak-qon tomir xavfini 2–2,5 baravar oshiradi. Maqsad — KMD ning klinik, biomarker va genetik prediktorlarini tizimlashtirish (PubMed, Scopus, eLibrary, 2015–2026). Klinik prediktorlar, endotelial va yallig'lanish biomarkerlari, yangi markerlar (sTREM2, FT4/FT3, GDF-15), genetik polimorfizmlar (NOS3, ACE, EDN1/PHACTR1) va diagnostika usullari (CFR, IMR, PET) tizimlashtirildi. Bu ma'lumotlarni kompleks baholash SKS diagnostikasini individuallashtirish uchun istiqbolli hisoblanadi.

Kalit so'zlar: koronar mikrovaskulyar disfunksiya; endotelial disfunksiya; surunkali koronar sindrom; ANOCA; INOCA; biomarkerlar; genetik polimorfizmlar.

Kirish: Yurak-qon tomir kasalliklari (YUQTK) dunyodagi o'limning yetakchi sababi bo'lib qolmoqda: har yili ulardan 17,9–19,8 million kishi vafot etadi, global YUQTK holatlari soni esa 30-yil ichida deyarli ikki baravar ko'paydi (271 dan 523 milliongacha); 2050-yilga borib kasallik tarqalishi 90 foizga, o'lim esa 73 foizga oshishi bashorat qilinmoqda [1, 2]. O'zbekiston Respublikasida yurak-qon tomir kasalliklari o'lim holatlarining 56 foizini va aholining nogironlik holatlarining 25 foizini keltirib chiqaradi, mamlakat esa yurak-qon tomir kasalliklaridan o'lim bo'yicha Markaziy Osiyo mintaqasidagi yetakchilar qatoriga kiradi [3, 4]; shu bilan birga, milliy skrining dasturlari 2024-yilda o'lim strukturasidagi yurak-qon tomir kasalliklari ulushining 61 foizdan 57 foizgacha pasayishini ta'minladi [5].

Stenokardiya va miokard ishemiyasi belgilari bo'lgan bemorlarning sezilarli qismida epikardial arteriyalarning obstruktiv shikastlanishi (ANOCA/INOCA) kuzatilmaydi; surunkali koronar sindromning (SKS) ushbu fenotipining yetakchi mexanizmlari sifatida koronar mikrovaskulyar disfunksiya (KMD) va endotelial disfunksiya e'tirof etilgan bo'lib, ular bunday bemorlarning 30-50 foizida aniqlanadi va salbiy yurak-qon tomir hodisalari (YUTXO) xavfini 2,08-2,45 baravar oshiradi [6, 7, 37]. Yangilangan ESC tavsiyalari (2024) koronar disfunksiyaning patofiziologik endotiplari tushunchasini kiritadi [8], biroq tashxis aniqligining yaxshilanishi (CorCTA tadqiqoti) hali alomatlarining yaxshilanishiga - "diagnostik va terapevtik uzilish"ga o'tkazilmagan [7]. Bu xavfning erta tabaqalanishi uchun, shu jumladan O'zbekiston sog'liqni saqlash sharoitida qo'llanilishi mumkin bo'lgan

KMDning mavjud klinik-biomarker va genetik prediktorlarini tizimlashtirish zarurligini asoslaydi.

Sharhning maqsadi: YUIK bilan og'rigan bemorlarda endotelial va koronar mikrovaskulyar disfunksiyaning klinik, biomarker, genetik va instrumental prediktorlarini tizimlashtirishdan iborat.

Material va usullar: 2015-2026-yillarda "coronary microvascular dysfunction," "endothelial dysfunction," "ANOCA/INOCA," "biomarkers," "genetic polymorphism" va ularning rus tilidagi muqobillari bo'yicha PubMed/MEDLINE, Scopus, Web of Science, eLibrary.ru, CyberLeninka, inLibrary.uz nashrlari tahlil qilindi.

PATOFIZIOLOGIK MEXANIZMLAR

KMDning umumiy patogenetik bo'g'ini koronar qon oqimiga 95% gacha qarshilikni ta'minlaydigan koronar mikrotomirlar (prearteriolalar, 40-400 mkm li arteriolalar, <10 mkm li kapillyarlar) endoteliysining shikastlanishi bo'lib, bunda vazodilatatsiyalovchi (NO, prostatsiklin) va vazokonstriktor (endoelin-1, tromboksan A2) omillar muvozanati buziladi [6, 9, 36]. Asosiy mexanizmlar: NO biosinguvchanligining pasayishi, surunkali subklinik yallig'lanish, oksidativ stress, mikrotomirlarning tarkibiy qayta tuzilishi (silliq mushak hujayralari gipertrofiyasi, perivaskulyar fibroz, kapillyarlarning siyraklashishi) [7, 10]. Tizimli endotelial disfunksiya bilan patogenetik o'xshashliklar kuzatiladi, bu esa umumiy prediktorlarni izlashni asoslaydi [7].

KLINIK BASHORATLAR

1-jadval. KMD va endotelial disfunksiyaning klinik bashoratchilari

Bashoratchi	Mexanizm	Ma'lumotlar
Ayol jinsi / postmenopauza	Estrogenlar yetishmovchiligi → eNOS faolligining pasayishi, vazokonstriksiyaning ustunligi	KMD ayollarda mikrotomir genezli YUQTK holatlarining 70% dan ortig'iga sabab bo'ladi; ayollarda CFR pasayishi ko'proq uchraydi (20% va 6%, p=0,033) [11-13].
2-tur qandli diabet	ADMA↑, ekzosomal mikroRNKlarning disregulyatsiyasi, endoteliotsitlarning yog' kislotalari almashinuvining buzilishi	QD bilan og'rigan INOCA bemorlarida mikroRNKning sezilarli disregulyatsiyasi [14, 15]

Bashoratchi	Mexanizm	Ma'lumotlar
Metabolik sindrom / epikardial semizlik	Mikrotsirkulyatsiyaga yog' to'qimasining yallig'lanishga qarshi parakrin ta'siri	Epikardial yog' - KMDning mustaqil prediktori, sezgirligi 85%, o'ziga xosligi 75% [16]
Arterial gipertenziya	Tomir devorini qayta qurish, NO miqdorini kamaytirish, sVCAM-1/vWF darajasini oshirish	AG bilan og'rigan bemorlarda ICAM-1, vWF va ET-1 ko'rsatkichlarining oshishi [17]
Migren, chekish, CBK	Oksidlovchi stress, ADMA/sVCAM-1 to'planishi	Migren ko'pincha ANOCA bilan og'rigan ayollarda uchraydi (45% va 14%) [13, 18]
FT4/FT3 subklinik o'zgarishlari	Qalqonsimon bezning qon tomir tonusini boshqarishi	Eutireoidlarda KMDning mustaqil prediktori INOCA (OSH 7,62; AUC 0,78) [1]

Erkaklarda ko'pincha epikardial spazm, ayollarda esa mikrotomirlar spazmi va CFR pasayishi aniqlanadi, bunda vazomotor disfunktsiyaning umumiy chastotasi taqqoslanadi (95% va 88%) [13, 20]. **ENDOTELIAL DISFUNKSIYA, YALLIG'LANISH BIOMARKERLARI VA YANGI ISTIQBOLLI MARKERLAR**

Eng ko'p o'rganilgan markerlar ADMA va endotelin-1 hisoblanadi; yallig'lanish markerlari (IL-6, hs-CRP) mustaqil prognostik qiymatga ega, bir qator yangi ko'rsatkichlar (sTREM2, FT4/FT3, GDF-15) esa istiqbolli deb hisoblanadi (2-jadval). 2).

2-jadval. Koronar mikrovaskulyar va endotelial disfunktsiyaning biomarkerlari

Guruh	Biomarker	Ahamiyat
Endotelial	ADMA	eNOS ingibitori; MASE bilan bog'liq (OR 0,19; p=0,013), AT1 bilan bog'liq oksidlanish stressi orqali KMDni kuchaytiradi [7, 21]
Endotelial	Endotelin-1 (ET-1)	Kuchli vazokonstriktor; darajasi KMDda koronar qon oqimi bilan teskari bog'liq [7, 21, 22]
Endotelial	vWF, sVCAM-1, sICAM-1, E-selektin	Endoteliy faollashuvi/shikastlanishi markerlari KMD, AG, CBKda yuqori bo'ladi [17, 18, 23]
Endotelial	Endotelial CD144+ mikroarrachalar (EMPs)	Noinvaziv surrogat marker: IMR bilan bog'liqlik, AUC 0,76, NPV 85,7% [24]
Yallig'lanishli	hs-CRP	"Quyi oqim" - yallig'lanish belgisi, IL-6 ga qaraganda kamroq o'ziga xos [25, 26]
Yallig'lanishli	IL-6	Aterogeneza sababchi rol; ANOCA bilan og'rigan ayollarda - MASEning mustaqil bashoratchisi, kardiometabolik yuqori xavf belgisi [25, 27]
Yallig'lanishli	TNF- α , miR-34a/145/222, SIRI	Endoteliyning faollashuvi va qon tomirlarining qayta shakllanishi; koronar qon oqimining sekinlashishi fenomeni bilan bog'liqligi [28]
Yangi/istiqbolli	sTREM2	CFR, IMR, MRR, RRR bilan bog'liq; KMDning mustaqil bashoratchisi [29]
Yangi/istiqbolli	FT4/FT3 nisbati	Eutireoidlarda KMD prediktori INOCA (AUC 0,78) [19]
Yangi/istiqbolli	GDF-15	Kardiometabolik/buyrak biomarkerlari; SKSda KMD bo'yicha to'g'ridan-to'g'ri ma'lumotlar cheklangan [30, 31]
Yangi/istiqbolli	Ekzosomal miR-16-2-3p	QDda KMD bilan bog'liq bo'lib, endoteliotsitlarda yog' kislotalarining parchalanishini boshqaradi [15]
Yangi/istiqbolli	Kopeptin, sST2, galektin-3, endokan, MMP-9	O'KSda o'rganilgan; XKSda KMDga ekstrapolyatsiya qilish validatsiyani talab qiladi [32]

GENETIK MARKERLAR

KMD va endotelial disfunktsiyaga irsiy moyillik asosan NO, endotelin sintezi va renin-angiotenzin tizimi genlarining

polimorfizmlari orqali amalga oshiriladi (1-jadval). KMD genetik determinatsiyasining asosiy bevosita isboti PHACTR1/EDN1 lokusini o'rganishdir [33].

3-jadval. Endotelial va koronar mikrovaskulyar disfunktsiyaning genetik belgilari

Gen / lokus	Ko'p shakllilik	Ta'sir	ED/KMD bilan bog'liqlik
NOS3 (eNOS)	G894T (Glu298Asp); T-786C; VNTR 4b/4a	eNOS faolligi/transkripsiyasining pasayishi	YUIK, MI, ishemik insult, koronar vazospazm xavfi (T/T genotipi) [34-36]
ACE	I/D (rs1799752)	AAF faolligining o'zgarishi	Arteriya devorining subklinik o'zgarishlari, endotelial disfunktsiya [37]
EDN1 (endotelin-1)	Lys198Asn (rs5370); tagSNP rs6458155	ET-1 ekspressiyasi/sekretsiasining kuchayishi	Diffuz epikardial spazm, YUIK xavfi (OSH 1,53-1,55), insult [33, 34]

Gen / lokus	Ko'p shakllilik	Ta'sir	ED/KMD bilan bog'liqlik
PHACTR1/EDN1	rs9349379-G (6p24 lokusi)	↑ endotelin ekspressiyasi $\geq 20\%$	Tasdiqlangan KMD bilan og'rigan bemorlarda allel chastotasi yuqoriroq (46% va 39%, $p=0,013$), bu birinchi bevosita dalil hisoblanadi [33].
ALDH2 / RNF213	rs671; p.R4810K	Aldegidlar detoksikatsiyasining buzilishi; endotelial/GMK stress	Koronar vazospazm assotsiatsiyasi (Sharqiy Osiyo kogortalari) [34]
AGT, ESR1	T704C (rs699); -351A>G, -397T>C;	Angiotenzinogen darajasining o'zgarishi; estrogena sezuvchanlik	Qon tomir tonusi va menopauzadan keyingi mikrovaskulyar disfunktsiya bilan bog'liqlik [16, 22]

Ko'pchilik assotsiatsiyalar vazospastik stenokardiya kogortalarida va umumiy populyatsiyada olingan; SKSda bevosita KMDni o'rganish kam uchraydi, bu esa genetik profilashni, shu

jumladan o'zbek populyatsiyasi uchun ham istiqbolli yo'nalishga aylantiradi.

INSTRUMENTAL DIAGNOSTIKA

4-jadval. KMDni tashxislash usullari

Usul	Tur	Mezonlar
CFR (koronar qon oqimi zaxirasi)	Invaziv/noinvaziv (ExoKG, PET, MRT)	CFR $< 2,0-2,5$ bo'lganda KMD [15, 20]
IMR / MRR	Invaziv, termodilyutsion	IMR ≥ 25 birlikda KMD; MRR - epikardial patologiyaga bog'liq emas [28, 30]
Angio-IMR	Invaziv bo'lmagan, angiografiyaga asoslangan	PET va invaziv indekslarga qarshi validatsiyalanadi [45]
Yurak PET / MRT tekshiruvi	Invaziv bo'lmagan, referens	MFR $< 2,0$; MPRI - yarim miqdoriy baholash [3,37]
Atsetilxolin bilan sinama	Invaziv funksional	Endoteliyaga bog'liq mikrovaskulyar/epikardial vazospastik disfunktsiyani aniqlash [9]

KMD mexanizmlarini tushunishdagi yutuqlarga qaramay, xavfning kompleks klinik-biomarker va genetik stratifikatsiyasining yagona algoritmi mavjud emas. O'zbek populyatsiyasida muhim polimorfizmlarning (NOS3, EDN1/PHACTR1) tarqalishi haqidagi ma'lumotlar deyarli mavjud emas. Diagnostik va terapevtik uzilish endotipga yo'naltirilgan davolash zarurligini ko'rsatadi. KMDni noinvaziv tashxislash aniqligini oshirish uchun multi-markerli panellarni (ADMA/ET-1, IL-6, sTREM2, asosiy SNP bo'yicha genotiplash) yaratish istiqbolli hisoblanadi.

XULOSA

Endotelial va koronar mikrovaskulyar disfunktsiya YUIK bilan og'rigan bemorlarda miokard ishemiyasining muhim, ammo yetarlicha baholanmagan mexanizmlaridir. Endotelial disfunktsiya va yallig'lanishning klinik bashoratchilari, biomarkerlari, yangi markerlar va genetik polimorfizmlarni tizimlashtirish yurak-qon tomir hodisalarini shaxsiylashtirilgan erta tashxislash va oldini olish uchun asos yaratadi. O'zbekistonda YUQTKning ko'pligi va KMD prediktorlari to'g'risida milliy ma'lumotlarning yo'qligini hisobga olgan holda, mahalliy populyatsiyada istiqbolli tadqiqotlarni davom ettirish dolzarb hisoblanadi.

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