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ЖУРНАЛ КАРДИОРЕСПИРАТОРНЫХ ИССЛЕДОВАНИЙ

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ИНСУЛИНОРЕЗИСТЕНТНОСТЬ КАК ПРЕДИКТОР РЕСТЕНОЗА И НЕСТАБИЛЬНОСТИ АРТЕРИАЛЬНОГО ДАВЛЕНИЯ ПОСЛЕ ЧРЕСКОЖНОГО КОРОНАРНОГО ВМЕШАТЕЛЬСТВА

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АННОТАЦИЯ

Инсулинорезистентность (ИР) — ключевое звено между метаболическими нарушениями и неблагоприятными сердечно-сосудистыми исходами. Данное ретроспективное исследование (2020–2026 гг.) оценивает прогностическую роль индекса HOMA-IR в развитии рестеноза и нестабильности артериального давления (АД) у пациентов после чрескожного коронарного вмешательства (ЧКВ). Установлено, что высокий уровень HOMA-IR является независимым предиктором рестеноза в стенте, повышенной воспалительной активности и долгосрочных осложнений, независимо от наличия диабета. ИР также связана с симпатической гиперактивностью, что провоцирует вариабельность АД в послеоперационном периоде. Учет ИР позволяет персонализировать риски и оптимизировать метаболическую терапию после ЧКВ.

Ключевые слова: Инсулинорезистентность; ЧКВ; рестеноз рестеноз коронарных артерий; нестабильность АД; HOMA-IR; ишемическая болезнь сердца; сахарный диабет.

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INSULIN RESISTANCE AS A PREDICTOR OF RESTENOSIS AND BLOOD PRESSURE INSTABILITY AFTER PERCUTANEOUS CORONARY INTERVENTION

SUMMARY

Insulin resistance (IR) is a critical link between metabolic dysfunction and adverse cardiovascular outcomes. This retrospective study (2020–2026) evaluates the prognostic value of HOMA-IR in predicting in-stent restenosis and blood pressure (BP) instability following percutaneous coronary intervention (PCI). Results indicate that elevated HOMA-IR is an independent predictor of restenosis, increased inflammatory activity, and poor long-term outcomes, regardless of diabetes status. Additionally, IR-related sympathetic hyperactivity contributes to post-PCI hemodynamic instability. Identifying IR as a predictor enhances risk stratification and supports a personalized cardiometabolic approach for PCI patients.

Key words: Insulin resistance; Percutaneous coronary intervention; In-stent restenosis; Blood pressure instability; HOMA-IR; Coronary artery disease; Diabetes mellitus; Endothelial dysfunction; Cardiometabolic risk.

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PERKUTAN KORONAR ARALASHUVDAN SO‘NG RESTENOZ VA ARTERIAL QON BOSIMI BEQARORLIGI PREDIKTORI SIFATIDA INSULINREZISTENTLIK

ANNOTATSIYA

Insulinrezistentlik (IR) metabolik buzilishlar va noxush yurak-qon tomir natijalari o‘rtasidagi asosiy bog‘lovchi bo‘g‘indir. Ushbu retrospektiv tadqiqot (2020–2026) perkutan koronar aralashuvdan (PKA) keyingi restenoz va qon bosimi beqarorligini bashorat qilishda HOMA-IR ko‘rsatkichining rolini baholaydi. Aniqlanishicha, yuqori HOMA-IR darajasi diabet mavjudligidan qat’i nazar, stent ichi restenozi, yallig‘lanish faolligi va uzoq muddatli asoratlar uchun mustaqil prediktor hisoblanadi. Shuningdek, IR simpatik giperaktivlikni keltirib chiqarib, operatsiyadan keyingi qon bosimi o‘zgaruvchanligiga sabab bo‘ladi. IR holatini inobatga olish PKA’dan keyingi xavflarni baholash va davolashni individuallashtirish imkonini beradi.

Kalit so‘zlar: Insulinrezistentlik; PKA; restenoz; qon bosimi beqarorligi; HOMA-IR; yurak ishemik kasalligi; qandli diabet.

By restoring coronary blood flow and lowering ischemic consequences, percutaneous coronary intervention (PCI) has significantly improved the prognosis of patients with obstructive coronary artery disease. In-stent restenosis and post-procedural hemodynamic instability continue to be clinically significant issues that have a detrimental impact on long-term outcomes, quality of life, and healthcare expenditures despite advancements in stent technology and adjunctive medication. The recurrence of these issues highlights the significance of systemic metabolic and inflammatory impacts and implies that mechanisms other than procedural and anatomical variables contribute to adverse outcomes following PCI.

A key element of cardiometabolic dysfunction, insulin resistance is essential to the onset and advancement of atherosclerotic cardiovascular disease. It is typified by decreased peripheral tissue biological reactivity to insulin, which results in endothelial dysfunction, oxidative stress, compensatory hyperinsulinemia, and a persistent low-grade inflammatory state. These pathophysiological mechanisms contribute to the development of coronary restenosis after PCI by promoting the proliferation of vascular smooth muscle cells, poor endothelial repair, and excessive neointimal hyperplasia. Crucially, insulin resistance is a significant risk factor for a wide range of patients receiving coronary procedures because it has detrimental cardiovascular effects even when there is no obvious sign of diabetes mellitus.

Insulin resistance has been linked to disruption of vascular tone and autonomic balance in addition to its structural vascular consequences. Blood pressure variability and instability are caused by sympathetic nervous system activity, salt retention, and arterial stiffness, all of which are influenced by hyperinsulinemia and poor insulin signaling. Because it may impair coronary perfusion, raise myocardial oxygen demand, and increase the risk of recurrent ischemic events, blood pressure instability following PCI, which manifests as labile hypertension or hypotensive episodes, is becoming more widely acknowledged as a hallmark of a poor prognosis. However, there is still a lack of clarity regarding the metabolic factors that contribute to blood pressure instability during PCI.

Data assessing insulin resistance's concurrent effects on both structural (restenosis) and functional (blood pressure instability) outcomes after PCI are still scarce, despite earlier research linking it separately to poor cardiovascular outcomes and restenosis following coronary stenting. Additionally, a lot of research has only looked at

diabetic populations, which may have understated the wider prognostic implications of insulin resistance in mixed cohorts that include individuals with and without diabetes. Clarifying insulin resistance's role in post-PCI outcomes is clinically significant given the condition's increasing incidence globally.

In light of this, the current retrospective study was created to examine insulin resistance in patients treated between 2020 and 2026 as a predictor of coronary restenosis and blood pressure instability after PCI. This study is to improve knowledge of the cardiometabolic parameters impacting post-interventional outcomes and to provide more precise risk assessment and customized management strategies in patients having PCI by combining metabolic, clinical, and procedural data.

Materials and methods: A tertiary-care cardiology center with an active percutaneous coronary intervention (PCI) program hosted this retrospective observational investigation. We examined the medical records of consecutive individuals who had PCI between January 2020 and December 2026. The study was conducted in compliance with the Declaration of Helsinki's tenets, and because it was retrospective in nature, all data were anonymized for analysis. The study protocol was approved by the Institutional Ethics Committee of Samarkand State Medical University. Due to the retrospective design, informed consent was waived.

Study population: Participants had to be adults (≥18 years old) with either acute coronary syndrome or stable coronary artery disease who had undergone successful PCI with drug-eluting stent placement. Patients who had comprehensive clinical records, angiographic results, fasting insulin and glucose readings, and follow-up data were included. To reduce confounding, the analysis excluded patients having a history of coronary artery bypass grafting, documented autoimmune or inflammatory illnesses, active cancer, severe hepatic or renal failure, or incomplete blood pressure or metabolic data.

Assessment of insulin resistance: The homeostasis model assessment of insulin resistance (HOMA-IR), which is computed using the usual formula, was used to assess insulin resistance:

HOMA-IR is calculated as follows: (fasting plasma glucose [mmol/L] × fasting plasma insulin [μIU/mL]) / 22.5.

Patients were divided into two groups based on population-based criteria and established literature: insulin resistance was defined as HOMA-IR >2.5. Instead of being an exclusion criterion, diabetes mellitus was noted and used as an analytical variable to evaluate how it interacted with insulin resistance.

Clinical and procedural data collection: Medical records were used to extract demographic information, cardiovascular risk factors (such as body mass index, smoking status, diabetes mellitus, hypertension, and dyslipidemia), and past cardiovascular history. The indication for PCI, the quantity and kind of stents inserted, the target vessel, the features of the lesion, and the success of the procedure were all included in the procedural data. Antiplatelet medications, statins, and antihypertensive medications as clinically required were all administered to all patients during and after PCI in accordance with conventional guidelines.

Outcome definitions: The main outcome was coronary restenosis, which was defined as either clinically motivated repeat revascularization due to restenosis or angiographically confirmed luminal narrowing of ≥50% at the stented segment or its margins during follow-up. Clinically significant variability in systolic and/or diastolic blood pressure during the post-PCI follow-up period was defined as blood pressure instability. This included hospitalization due to blood pressure dysregulation, documented excessive blood pressure variability on serial measurements, or recurrent episodes of hypertension or hypotension requiring treatment adjustment.

Follow-up and measurements: Outpatient visits, hospital records, and repeat coronary angiography when clinically indicated were the sources of follow-up data. Standardized methods were used to take blood pressure readings during hospital stays and follow-up appointments. In accordance with institutional standards, laboratory measures such as insulin, lipid profile, fasting glucose, and inflammatory markers were gathered both at baseline and during normal follow-up.

Statistical analysis: Standard statistical software was used to conduct the statistical analysis. While categorical variables were shown as frequencies and percentages, continuous variables were given as mean ± standard deviation or median with interquartile range, as applicable. The chi-square test for categorical variables and the student's t-test or Mann-Whitney U test for continuous variables were used to compare groups. After controlling for age, sex, diabetes mellitus, and other pertinent clinical variables, multivariable regression analysis was used to find independent predictors of restenosis and blood pressure instability.

RESULTS:

STUDY POPULATION AND PATIENT DISTRIBUTION: 328 of the 412 patients who had PCI between 2020 and 2026 satisfied the inclusion requirements and were included in the final analysis. Patients with incomplete metabolic data (n = 46), previous coronary artery bypass grafting (n = 21), active inflammatory or malignant illness (n = 11), or inadequate follow-up (n = 6) were excluded.

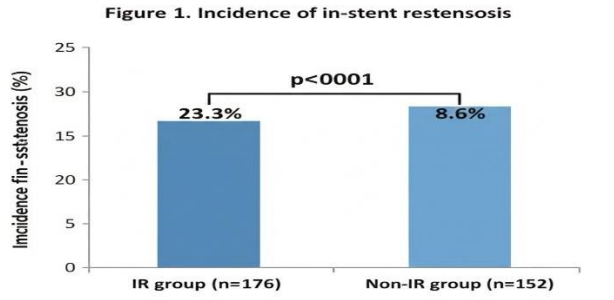
Patients were split into two groups according to their HOMA-IR values:

1. Insulin resistance (IR) group: 176 individuals
2. non-IR group (non-insulin resistance group): n = 152

112 patients (34.1%) had diabetes mellitus, and the prevalence was higher in the IR group (72 patients, 40.9%) than in the non-IR group (40 patients, 26.3%, p<0.05).

Baseline demographic, clinical, and metabolic characteristics are summarized in Table 1. There were no statistically significant differences between groups in age, sex distribution, smoking status, body mass index, or PCI indication (p>0.05). However, patients in the IR group demonstrated significantly higher fasting insulin levels, fasting plasma glucose, HbA1c, and C-reactive protein compared with the non-IR group (p<0.05).

Figure 1. Incidence of in-stent restenosis after PCI according to insulin resistance status:



During a median follow-up period of 18 months (IQR 12—30), angiorchanbly confirmed in-stent restensis ocured in 54 patients (16.5%.The incience of restenosis was significaly higher the IR group compared with the non-IR garoup (23.3% vs 8 group (23.3% vs 8.6%), p<0001), nf1.001) even Muliviaciae logistic regrcssion analysis anansiidedinsullin preditene as an predictor of restenosis (OR 2.41; 1.48— even after diabets for diabetes for hytehstus, lipid profile, steid profile, stent number, and lesion characisistics.

During a median follow-up period of 18 months (IQR 12–30), angiographically confirmed in-stent restenosis occurred in 54 patients (16.5%). The incidence of restenosis was significantly higher in the IR group compared with the non-IR group (23.3% vs 8.6%, p<0.001).

Multivariate logistic regression analysis identified insulin resistance as an independent predictor of restenosis (OR 2.41; 95% CI 1.48–3.92; p<0.001), even after adjustment for diabetes mellitus, hypertension, lipid profile, stent number, and lesion characteristics.

Table 2. Distribution of post-PCI outcomes according to insulin resistance status:

Table 2. Distribution of post-PCI outcomes according insulin resistance status

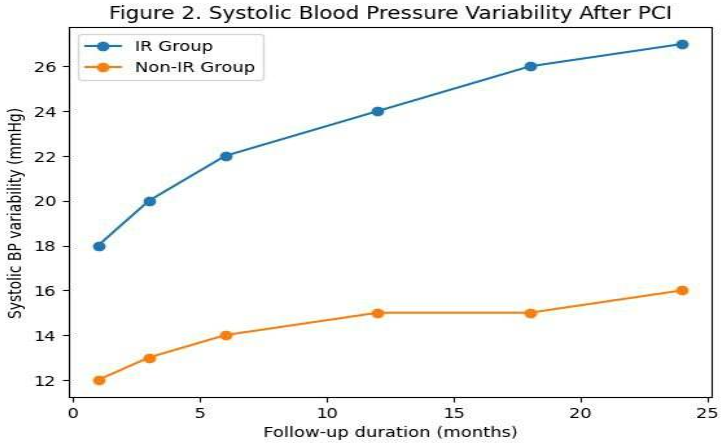
Outcome	IR group (n = 176)	Non-IR group (n = 152)	p-value
In-stent restenosis, n (%)	54 (30.7%)	13 (8.6%)	<0.001
Blood pressure instability, n (%)	68 (38.6%)	28 (18.4%)	<0.001
No adverse outcome, n (%)	54 (30.7%)	111 (73.0%)	<0.001

Restenosis and blood pressure instability were substantially more common in insulin-resistant patients than in non-IR patients.

BLOOD PRESSURE INSTABILITY AFTER PCI: 96 patients (29.3%) had blood pressure instability during the follow-up period. Blood pressure instability was more common in the IR group than in the non-IR group (38.6% vs. 18.4%, p<0.001).

Insulin-resistant patients had higher rates of antihypertensive medication modifications, higher systolic and diastolic blood pressure variability, and more frequent hypertensive crises. Systolic blood pressure fluctuation was significantly positively correlated with HOMA-IR findings (r=0.42). p<0.001).

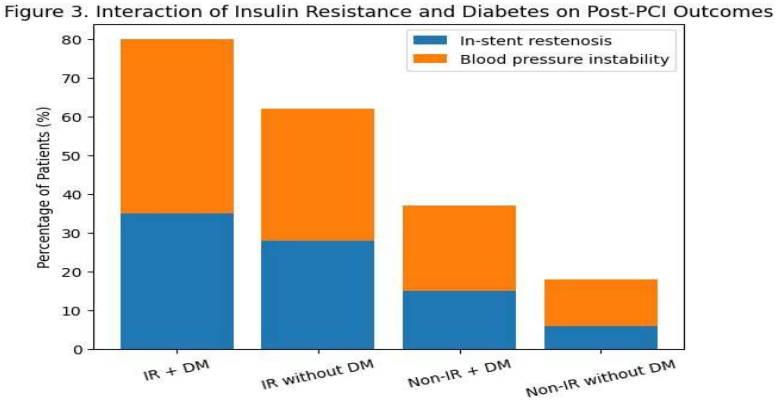
Figure 2. Systolic blood pressure variability during follow -up in insulin-resistant and non-insulin-resistance patients:



As shown in Figure 2, patients with insulin resistance demonstrated significantly greater systolic blood pressure variability throughout follow-up compared with non-IR patients."

Interaction between insulin resistance and diabetes mellitus: Both diabetic and non-diabetic individuals with insulin resistance exhibited considerably greater rates of restenosis and blood pressure instability than non-IR patients, according to subgroup analysis. Significantly, insulin-resistant non-diabetic patients fared worse than insulin-resistant diabetic patients (p<0.05).

Figure 3. Interaction between insulin resistance and diabetes mellitus on post-PCI outcomes:



As shown in Figure 3, insulin resistance was associated with higher rates of restenosis and blood pressure instability regardless of diabetic status."

DISCUSSION: The current retrospective analysis shows that blood pressure instability after percutaneous coronary intervention and in-stent restenosis are both strongly and independently predicted by insulin resistance. The main conclusion of this investigation is that, even after controlling for conventional cardiovascular risk variables and the existence of diabetes mellitus, individuals with insulin resistance had noticeably worse post-PCI outcomes than patients without insulin

resistance. This finding emphasizes insulin resistance's clinical significance as a separate pathophysiological entity rather than just a stand-in for hyperglycemia.

Endothelial dysfunction is one of the main processes that connect insulin resistance to restenosis. Vascular smooth muscle cell proliferation and neointimal hyperplasia are encouraged by insulin resistance, which is linked to decreased nitric oxide bioavailability, elevated oxidative stress, and enhanced inflammatory activity. The development of in-stent restenosis following PCI is mostly influenced by these processes. Further evidence that chronic low-grade inflammation plays a role in poor vasculature remodeling after cardiac intervention comes from the higher levels of inflammatory markers seen in insulin-resistant individuals in this study.

Patients with insulin resistance also had much higher rates of blood pressure instability following PCI. The combined effects of arterial stiffness, poor vascular responsiveness, and autonomic nervous system imbalance—all of which are frequently seen in insulin-resistant states—may account for this observation. It has been demonstrated that insulin resistance increases the activity of the sympathetic nervous system and decreases baroreflex sensitivity, which increases blood pressure fluctuation. There may be a reciprocal association between blood pressure fluctuation and unfavorable vascular outcomes since persistent hemodynamic instability may worsen endothelial damage and hasten restenosis.

Crucially, subgroup analysis showed that regardless of diabetes status, insulin resistance increased risk. Compared to diabetic patients without insulin resistance, non-diabetic patients with insulin resistance showed greater incidences of restenosis and blood pressure instability. A crucial therapeutic implication of this discovery is that relying just on diabetes mellitus as a measure of metabolic risk may cause non-diabetic but insulin-resistant patients' cardiovascular susceptibility to be underestimated. Therefore, early detection of insulin resistance may enhance risk assessment and direct more stringent post-PCI surveillance.

Our findings suggest the support and expand upon earlier studies showing the predictive significance of insulin resistance in cardiovascular disease. The current research highlights the wider metabolic milieu influencing vascular healing during PCI, whereas previous investigations have mostly focused on glycemic management and diabetic status. Clinicians may more accurately identify patients at increased risk for restenosis and blood pressure instability by incorporating insulin resistance into post-PCI risk assessment.

This study has several limitations that should be noted. Insulin resistance was measured using HOMA-IR rather than direct tests of insulin sensitivity, and the retrospective methodology raises the likelihood of selection bias. Furthermore, the study was only carried out at one location, which might have limited how broadly the results can be applied. However, the validity of the findings is strengthened by the comparatively large sample size, clearly defined patient groups, and thorough follow-up.

In conclusion, our findings offer compelling evidence that insulin resistance, independent of diabetes mellitus, is a clinically significant predictor of worse outcomes following PCI. These results imply that tailored metabolic therapies may enhance the prognosis following PCI and justify the inclusion of insulin resistance testing in routine cardiovascular risk appraisal.

Conclusion: Both in-stent restenosis and blood pressure instability after percutaneous coronary intervention are significantly and independently predicted by insulin resistance. The current study highlights the significance of metabolic dysfunction beyond overt hyperglycemia by showing that poor post-PCI outcomes are more strongly linked to insulin resistance than to diabetic mellitus alone. Regardless of whether they had diabetes or not, patients with insulin resistance showed increased hemodynamic instability and restenosis rates throughout follow-up.

These results highlight the clinical significance of detecting insulin resistance in PCI patients, including those without a diabetes diagnosis. It may be possible to enhance post-procedural risk categorization and enable earlier, more focused therapies by integrating insulin resistance testing into routine cardiovascular screening. To find out if therapeutic approaches targeted at increasing insulin sensitivity can lower restenosis rates and improve blood pressure stability following PCI, future prospective studies are necessary.

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