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Article

Hemostatic and Inflammatory Biomarkers Associated with Suspected Restenosis after Percutaneous Coronary Intervention in Type 2 Diabetes

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Abstract: Background: Diabetes-associated metabolic and inflammatory disturbances may contribute to recurrent coronary narrowing after percutaneous coronary intervention (PCI). Objective: To describe the distribution of hemostatic, metabolic, and inflammatory biomarkers in a previously reported clinical series stratified by suspected post-PCI restenosis. Methods: This descriptive manuscript is based on aggregate results reported from a 2022–2024 single-center cohort comprising 110 patients undergoing PCI and 30 apparently healthy comparators. Reported biomarkers included glycated hemoglobin (HbA1c), D-dimer, fibrinogen, high-sensitivity C-reactive protein (hs-CRP), interleukin-6 (IL-6), and HOMA-IR. Results: Suspected restenosis was reported in 42/110 patients (38.2%). Compared with the group without suspected restenosis, the suspected-restenosis group showed higher HbA1c (8.9 ± 1.1% versus 7.4 ± 0.8%), D-dimer (1.62 ± 0.41 versus 0.72 ± 0.19 μg/mL), and hs-CRP (6.9 ± 2.0 versus 3.8 ± 1.2 mg/L). Reported apparent ROC areas were 0.89 for D-dimer, 0.86 for fibrinogen, and 0.83 for hs-CRP; these estimates could not be independently verified. Conclusion: The reported biomarker patterns support further evaluation of integrated risk stratification, but diagnostic adjudication, patient-level analysis, and external validation are prerequisites to clinical deployment.

Keywords: type 2 diabetes mellitus; percutaneous coronary intervention; suspected in-stent restenosis; D-dimer; fibrinogen; hs-CRP; IL-6; risk stratification

Introduction

Patients with type 2 diabetes mellitus (T2DM) frequently exhibit diffuse coronary atherosclerosis, endothelial dysfunction, insulin resistance, and persistent low-grade inflammation. Percutaneous coronary intervention improves myocardial perfusion in appropriately selected patients, but recurrent luminal narrowing remains a challenge despite modern drug-eluting stents. The mechanisms of in-stent restenosis include neointimal hyperplasia, neoatherosclerosis, and mechanical factors such as stent underexpansion [1].

Hemostatic abnormalities may coexist with these processes. Fibrinogen reflects coagulation substrate availability and an acute-phase response, whereas D-dimer indicates fibrin formation and degradation but is nonspecific. These biomarkers should not be conflated with direct angiographic evidence of restenosis or with stent thrombosis. This study describes previously reported biomarker patterns in a local PCI cohort and evaluates the interpretive limits of their proposed prognostic use.[2]

Materials and Methods

Study setting and participants. The source article describes a single-center investigation performed during 2022–2024 at Bukhara Regional Multidisciplinary Medical Center. It reports 110 patients aged 18–74 years with ischemic heart disease undergoing PCI and 30 healthy volunteers as a comparison group. The patient cohort was stratified into 68 individuals without and 42 with clinically or biomarker-suspected restenosis during 12 months of follow-up. The original source reports 61 men and 59 women, totaling 120 rather than 110; therefore, sex distribution is deliberately omitted pending source-data reconciliation.[3]

Measurements. According to the source report, routine assessment included clinical history, HbA1c, glucose, HOMA-IR, lipid markers, hs-CRP, D-dimer, fibrinogen, platelet count, activated partial thromboplastin time, prothrombin time, and international normalized ratio. IL-6, IL-1β, and tumor necrosis factor-α were also reported. The original publication lists electrocardiography, echocardiography, and vascular ultrasonography among the instrumental examinations. However, the proportion of restenosis cases confirmed by coronary angiography, coronary computed tomography, or intravascular imaging is not specified.[4]

Analysis and reporting. This paper reproduces reported group-level mean ± dispersion values without recalculation because anonymized patient-level measurements, missingness patterns, biomarker collection times, and the original analysis code are unavailable. The source refers to ROC analysis and logistic regression, although model coefficients, confidence intervals, thresholds, and validation procedures are absent. Its classification outcome is therefore described here as “suspected restenosis” rather than adjudicated angiographic in-stent restenosis.[5]

Results

During the reported 12-month follow-up, 42 of 110 patients (38.2%; calculated from the published numerator and denominator) were classified as having suspected restenosis; 68 were classified as not having suspected restenosis. The observed proportions cannot be compared directly with modern angiographic ISR rates because the endpoint criteria, device types, lesion characteristics, and screening intensity were not described.[6]

Across all six tabulated measures, the suspected-restenosis group had higher reported values than the group without suspected restenosis (Table 1). In particular, HbA1c differed by 1.5 percentage points, D-dimer by 0.90 μg/mL, and hs-CRP by 3.1 mg/L. These are descriptive between-group differences and do not demonstrate independent predictive effects. The source states that the differences were statistically significant ($p < 0.05$) but does not provide test statistics, individual p values, or adjustment for multiple comparisons.[7]

The source additionally reports apparent areas under the ROC curve (AUCs) of 0.89 for D-dimer, 0.86 for fibrinogen, and 0.83 for hs-CRP, as well as 88% sensitivity and 81% specificity for a combined HbA1c–D-dimer–hs-CRP–IL-6 model. These figure 1. are presented as unverified source-reported estimates, not independently reproduced performance measures.[8]

Table 1. Reported metabolic, hemostatic, and inflammatory biomarkers by clinical grouping[9]

Marker	Healthy comparators (n=30)	No suspected restenosis (n=68)	Suspected restenosis (n=42)
HbA1c (%)	5.6 ± 0.4	7.4 ± 0.8	8.9 ± 1.1
D-dimer (μg/mL)	0.28 ± 0.12	0.72 ± 0.19	1.62 ± 0.41

Marker	Healthy comparators (n=30)	No suspected restenosis (n=68)	Suspected restenosis (n=42)
Fibrinogen (mg/dL)	295 ± 45	365 ± 55	472 ± 70
hs-CRP (mg/L)	1.3 ± 0.6	3.8 ± 1.2	6.9 ± 2.0
IL-6 (pg/mL)	2.8 ± 1.1	6.4 ± 2.3	11.6 ± 3.7
HOMA-IR	1.8 ± 0.7	3.6 ± 1.2	5.2 ± 1.6

Data transcribed from the supplied sample article; dispersion measure was not explicitly defined in the source.

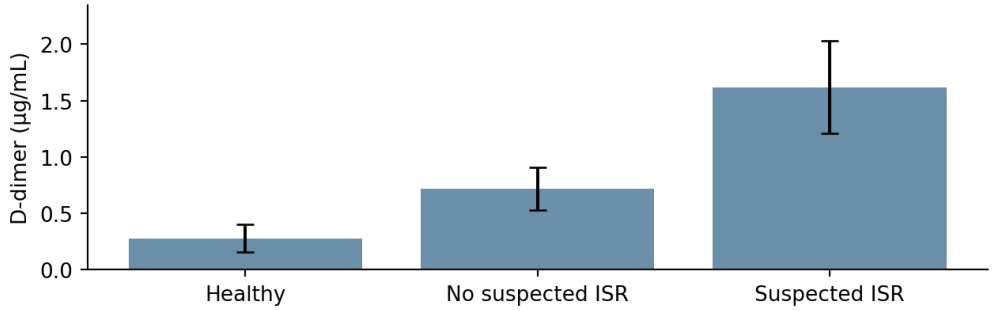


Figure 1. Source-reported group-level D-dimer values. Error bars reproduce the published ± figures; their statistical definition was not provided.[10]

Interpretation of biomarker differences

The markedly higher D-dimer and fibrinogen levels in the suspected-restenosis group are consistent with a procoagulant and inflammatory phenotype. Nevertheless, D-dimer is increased in multiple clinical states, including thrombosis, infection, malignancy, renal dysfunction, and recent procedures. Fibrinogen is likewise influenced by inflammation and other systemic factors. As a result, neither marker alone can establish the occurrence, site, severity, or mechanism of coronary restenosis.[11]

A parallel rise in HbA1c, HOMA-IR, hs-CRP, and IL-6 suggests clustering of dysglycemia, insulin resistance, and inflammation. Because these measures were reported at group level without precise timing relative to PCI and outcome identification, temporal sequence and causality cannot be inferred. Prospective baseline measurement is necessary to test predictive value rather than association with an already suspected event.[12]

Discussion

The present summary highlights a potentially informative conjunction of metabolic, inflammatory, and coagulation-related markers among people with diabetes undergoing coronary intervention. These results align with the biological expectation that diabetes is associated with enhanced platelet reactivity, endothelial dysfunction, and vascular inflammation. Yet most of the biomarkers are nonspecific and may reflect the systemic burden of disease rather than causal drivers of neointimal growth.[13]

The most important methodological limitation is the uncertain definition of the outcome. Coronary in-stent restenosis is fundamentally an anatomical diagnosis; symptoms and circulating markers may prompt assessment but are insufficient to confirm it. In selected cases with recurrent ischemic symptoms, evidence-based evaluation should determine whether repeat coronary imaging is warranted. Intravascular ultrasound or optical coherence tomography may clarify whether a recurrent lesion reflects stent underexpansion, neointimal tissue, or neoatherosclerosis [14].

The apparent discriminative ability reported for D-dimer, fibrinogen, and hs-CRP is hypothesis generating. With only 42 reported events, a four-biomarker model may be susceptible to optimism, especially when its specification and internal validation are not documented. Assessment of calibration, optimism-corrected discrimination, threshold stability, decision-curve utility, and external reproducibility would be needed before selecting a clinically actionable cutoff. The

appropriate comparison is not only a single biomarker but also a clinical baseline model incorporating age, diabetes duration, prior PCI, kidney function, lesion complexity, stent properties, and medication adherence.

Risk-factor modification after PCI should remain grounded in established cardiovascular prevention recommendations. Intensive lipid lowering, individualized glycemic treatment, smoking cessation, blood-pressure management, and secondary prevention therapies reduce overall cardiovascular risk. SGLT2 inhibitors and GLP-1 receptor agonists with demonstrated benefit have an established role in appropriate patients with T2DM and atherosclerotic cardiovascular disease [1]. However, an improvement in global cardiovascular outcomes should not be represented as proof of a specific reduction in in-stent restenosis.

Antiplatelet treatment is determined by the clinical presentation, procedural complexity, ischemic risk, bleeding risk, and need for oral anticoagulation. D-dimer or fibrinogen elevations alone are not accepted indications for intensifying antiplatelet treatment or initiating anticoagulation. In particular, prevention of stent thrombosis should be distinguished from prevention of tissue-mediated ISR; a biomarker-driven antithrombotic strategy would require a dedicated prospective trial [1,2].

Directions for prospective validation

A future study should predefine angiographically confirmed ISR or clinically driven target-lesion revascularization, document stent generation and lesion characteristics, obtain biomarkers before or shortly after PCI at standardized time points, and distinguish baseline predictors from time-updated monitoring variables. Multivariable models should address missing data and report odds ratios or hazard ratios with 95% confidence intervals. Bootstrap internal validation and an external temporal or multicenter cohort would permit meaningful assessment of transportability. Publication should also provide ethics approval, consent procedures, the complete flow of eligible patients, and adherence to STROBE reporting principles.

This manuscript was prepared from a previously supplied article and institutional methodological recommendation; it is not a new prospective or retrospective analysis of original patient records. The source contains a participant-count inconsistency (61 men plus 59 women versus total n=110), does not specify the criteria used to diagnose restenosis, and does not document the statistical pipeline sufficiently to reproduce its AUCs, sensitivity, or specificity. No effect-size confidence intervals or model coefficients were available. Consequently, the results should be interpreted as descriptive and exploratory, not as validated diagnostic or prognostic estimates.[15]

Conclusion

In the supplied clinical series, suspected post-PCI restenosis in patients with type 2 diabetes was accompanied by higher reported HbA1c, D-dimer, fibrinogen, hs-CRP, IL-6, and HOMA-IR values. A combined metabolic–hemostatic–inflammatory assessment is a reasonable research hypothesis, but these biomarkers cannot independently diagnose coronary restenosis or justify alteration of antithrombotic therapy. Standardized endpoint adjudication, correction of the underlying dataset, and adequately validated multivariable analysis are essential before clinical implementation.

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