World Journal of Biology Pharmacy and Health Sciences
eISSN: 2582-5542
Cross Ref DOI: 10.30574/wjbphs
Journal homepage: https://wjbphs.com/

(REVIEW ARTICLE)

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Managing ischemic risk after PCI in diabetic patients: advances in antiplatelet strategies

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World Journal of Biology Pharmacy and Health Sciences, 2025, 22(02), 164-170

Publication history: Received on 25 March 2025; revised on 30 April 2025; accepted on 02 May 2025

Article DOI: https://doi.org/10.30574/wjbphs.2025.22.2.0459

Abstract

Background: Type 2 diabetes mellitus (T2DM) significantly increases the risk of adverse cardiovascular outcomes, particularly following percutaneous coronary intervention (PCI). Diabetic patients experience accelerated atherosclerosis, heightened platelet reactivity, and endothelial dysfunction, contributing to an increased risk of recurrent ischemic events, including myocardial infarction and stent thrombosis. Optimal antiplatelet therapy in this high-risk group remains a clinical challenge due to the need to balance thrombotic protection with bleeding risk.

Objective: This review aims to summarize current antiplatelet strategies and highlight recent advances in managing ischemic risk in diabetic patients after PCI. Emphasis is placed on drug selection, therapy duration, individualized approaches, and emerging evidence from clinical trials and precision medicine.

Methods: A narrative review of key randomized controlled trials, meta-analyses, guideline documents (ESC, ACC/AHA), and observational studies published between 2009 and 2024 was conducted using PubMed and clinical trial registries. Studies focusing on antiplatelet therapy in patients with T2DM undergoing PCI were prioritized.

Key Findings: Ticagrelor and prasugrel offer superior platelet inhibition and ischemic protection compared to clopidogrel in diabetic patients, though at the cost of increased bleeding. Risk stratification tools such as the DAPT and PRECISE-DAPT scores assist in tailoring therapy duration and intensity. Recent trials (e.g., THEMIS-PCI, PEGASUS-TIMI 54, TWILIGHT) support individualized strategies including de-escalation and monotherapy in select patients. Innovations such as platelet function testing, CYP2C19 genotyping, dual pathway inhibition, and artificial intelligence-driven risk prediction are paving the way for precision-guided antiplatelet therapy.

Conclusion: Management of ischemic risk in diabetic patients post-PCI should be individualized and evidence-based. Future research should focus on validating precision tools, refining risk models, and identifying optimal long-term strategies that balance ischemic protection with safety.

Keywords: Diabetes Mellitus; Dual Antiplatelet Therapy; PCI; Ischemic Risk; Ticagrelor; Precision Medicine

1. Introduction

Cardiovascular disease remains the leading cause of morbidity and mortality worldwide, and type 2 diabetes mellitus (T2DM) is a major contributor to this burden. Coronary artery disease (CAD) is present in more than 50% of individuals with diabetes and accounts for a substantial proportion of adverse clinical outcomes in this population [1]. Globally, the number of adults with diabetes has risen dramatically, exceeding 537 million in 2021 and projected to reach 783 million

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by 2045, with over 75% of cases occurring in low- and middle-income countries [2]. The presence of diabetes confers a two- to four-fold increase in the risk of CAD, and diabetic patients who undergo percutaneous coronary intervention (PCI) are particularly vulnerable to recurrent ischemic complications, including myocardial infarction, target lesion revascularization, and stent thrombosis [3].

The pathophysiology of atherothrombosis in T2DM is complex and multifactorial. Central to this is platelet hyperreactivity, a well-recognized feature in diabetic patients that contributes to an increased propensity for thrombotic events. Hyperglycemia, insulin resistance, and oxidative stress promote enhanced platelet adhesion, aggregation, and secretion through multiple molecular pathways, including increased intracellular calcium mobilization and overexpression of surface glycoprotein IIb/IIIa and P-selectin [4]. Additionally, diabetic platelets are less responsive to the endogenous inhibitory effects of nitric oxide and prostacyclin, further predisposing to thrombus formation [5].

Alongside platelet dysfunction, endothelial dysfunction plays a critical role in accelerating atherosclerosis and thrombosis in diabetes. Hyperglycemia-induced formation of advanced glycation end-products (AGEs), activation of protein kinase C, and increased reactive oxygen species contribute to reduced nitric oxide bioavailability and impaired vasodilation [6]. The diabetic endothelium exhibits increased permeability, inflammation, and expression of adhesion molecules, which facilitate monocyte recruitment and foam cell formation—key events in plaque development and instability [7]. These changes collectively render the atherosclerotic plaques in T2DM more prone to rupture and thrombosis, which are the principal mechanisms underlying acute coronary syndromes and stent-related complications.

Despite advancements in PCI techniques and stent technologies, diabetic patients remain at elevated risk of adverse outcomes post-revascularization. Several studies have demonstrated that even with drug-eluting stents (DES), the risk of in-stent restenosis, late stent thrombosis, and long-term mortality is significantly higher in diabetic individuals compared to their non-diabetic counterparts [8]. The pathobiology of restenosis in diabetes includes increased neointimal hyperplasia and vascular remodeling, driven by persistent inflammation and smooth muscle proliferation [9]. Furthermore, diabetic patients undergoing PCI often present with more diffuse and multivessel disease, small-caliber vessels, and longer lesion lengths, which complicate both the procedural strategy and long-term management [10]. These anatomical complexities, combined with the prothrombotic and proinflammatory milieu of diabetes, demand a more aggressive and tailored antithrombotic approach to reduce the risk of recurrent ischemic events.

Dual antiplatelet therapy (DAPT), consisting of aspirin and a P2Y12 receptor inhibitor, is the foundation of secondary prevention following PCI. However, diabetic patients demonstrate attenuated response to certain agents—most notably clopidogrel—due to impaired hepatic conversion to the active metabolite and upregulated platelet P2Y12 receptor expression [11]. Consequently, high on-treatment platelet reactivity (HPR) is more prevalent in this subgroup and has been linked to poor cardiovascular outcomes, including stent thrombosis and myocardial infarction [12]. In response to these findings, newer-generation P2Y12 inhibitors such as ticagrelor and prasugrel have been evaluated in randomized controlled trials and shown to provide more potent and consistent platelet inhibition in diabetic patients. However, this enhanced efficacy is accompanied by an increased risk of bleeding, particularly in older patients or those with renal dysfunction common comorbidities in T2DM [13]. Therefore, balancing ischemic and bleeding risks remains a key challenge in this population, highlighting the need for individualized DAPT strategies.

The importance of tailoring antiplatelet therapy in diabetic patients cannot be overstated. Risk stratification tools such as the DAPT score and the PRECISE-DAPT score aid clinicians in determining optimal therapy duration and intensity, but they may not fully capture the unique thrombotic and hemorrhagic risk profiles in diabetes. Moreover, recent advances in pharmacogenomics, platelet function testing, and artificial intelligence (AI)-driven prediction models are opening new avenues for precision-guided antiplatelet therapy [14]. In addition to optimizing agent selection and duration, emerging strategies such as dual pathway inhibition (e.g., aspirin plus low-dose rivaroxaban), de-escalation protocols, and early monotherapy in selected patients are under active investigation, with promising early results [15]. Furthermore, trials specific to the diabetic population, including THEMIS-PCI, TWILIGHT-DM, and HOST-EXAM-DM, are contributing critical evidence to guide clinical decision-making in this high-risk group.

In summary, patients with T2DM represent a distinct clinical entity in the context of PCI, characterized by heightened thrombotic risk, suboptimal response to traditional antiplatelet agents, and a more complex vascular substrate. The growing body of evidence underscores the need for an individualized and dynamic approach to antiplatelet therapy, guided by patient-specific clinical, procedural, and biologic factors. This review aims to critically evaluate current antiplatelet strategies, assess the comparative effectiveness of available agents, and highlight future directions in managing ischemic risk in diabetic patients following PCI.

2. Current antithrombotic strategies post-PCI

Effective secondary prevention after percutaneous coronary intervention (PCI) in patients with type 2 diabetes mellitus (T2DM) hinges on the optimization of antithrombotic therapy. Owing to their heightened thrombotic risk and variable response to standard agents, diabetic patients require tailored antiplatelet strategies that balance ischemic protection against bleeding complications. Dual antiplatelet therapy (DAPT), comprising aspirin and a P2Y12 receptor inhibitor, remains the foundational treatment in this setting, but the choice of agent and duration of therapy must be adapted to individual risk profiles.

2.1. Dual Antiplatelet Therapy (DAPT)

The use of DAPT is the standard of care following PCI to reduce the incidence of stent thrombosis and major adverse cardiovascular events (MACE). Aspirin acts through irreversible inhibition of cyclooxygenase-1 (COX-1), reducing thromboxane A2-mediated platelet activation, while P2Y12 inhibitors block ADP-induced platelet aggregation via the P2Y12 receptor pathway. This synergistic mechanism effectively suppresses platelet reactivity and thrombus formation on stent surfaces [15]. DAPT is recommended for all patients undergoing PCI with drug-eluting stents, and its importance is particularly magnified in diabetic patients, who exhibit higher baseline platelet reactivity and impaired fibrinolysis [16]. The addition of a potent P2Y12 inhibitor such as ticagrelor or prasugrel has been shown to provide superior ischemic protection in this population compared to clopidogrel [17].

2.2. Duration: Short, Standard, and Extended DAPT

Determining the optimal duration of DAPT post-PCI in diabetic patients involves a careful assessment of both ischemic and bleeding risks. Guidelines from the European Society of Cardiology (ESC) and American College of Cardiology (ACC) generally recommend a standard DAPT duration of 6 to 12 months following PCI, depending on the clinical setting (acute coronary syndrome vs. stable CAD) and procedural complexity [18]. For diabetic patients with high ischemic risk (e.g., prior MI, multivessel disease, complex stenting), extended DAPT beyond 12 months may be beneficial. This is supported by trials such as PEGASUS-TIMI 54, which demonstrated that prolonged DAPT with ticagrelor reduced MACE in patients with prior MI, including a substantial proportion with T2DM [19]. Conversely, shorter DAPT durations (3–6 months) may be appropriate in patients with high bleeding risk, especially the elderly or those with chronic kidney disease, although caution is warranted in diabetics due to their elevated ischemic burden [20]. The DAPT and PRECISE-DAPT scores are widely used to individualize therapy duration. Diabetic patients often score favorably for prolonged DAPT given their higher thrombotic risk, but concurrent bleeding risks must also be considered [21].

2.3. Agent Selection

2.3.1. Clopidogrel: Limitations in T2DM

Clopidogrel has historically been the most widely used P2Y12 inhibitor, but its limitations in diabetic patients are well recognized. It is a prodrug requiring biotransformation via hepatic cytochrome P450 enzymes, particularly CYP2C19, to generate its active metabolite. Diabetic patients often exhibit reduced enzymatic activity, increased platelet turnover, and enhanced expression of platelet ADP receptors—all of which contribute to high on-treatment platelet reactivity (HPR) and reduced clinical efficacy [22]. The PLATO and TRITON-TIMI 38 trials, as well as real-world registries, have consistently demonstrated that diabetic patients receiving clopidogrel have higher rates of recurrent ischemic events and stent thrombosis compared to those treated with more potent P2Y12 inhibitors [23]. As a result, clopidogrel is now considered suboptimal for high-risk diabetic patients, particularly in the setting of acute coronary syndrome or complex PCI.

2.3.2. Ticagrelor and Prasugrel: Preferred in High-Risk Patients

Ticagrelor and prasugrel are newer-generation P2Y12 inhibitors that offer more potent and consistent platelet inhibition, independent of hepatic activation pathways. These agents have shown clear advantages in T2DM populations. Ticagrelor, a reversible direct-acting P2Y12 inhibitor, demonstrated superiority over clopidogrel in the diabetic subgroup of the PLATO trial, significantly reducing rates of cardiovascular death, MI, and stroke [24]. Similarly, prasugrel, an irreversible inhibitor, was associated with lower ischemic events compared to clopidogrel in the diabetic cohort of TRITON-TIMI 38, albeit with a modest increase in bleeding [25]. Both agents are now recommended for use in T2DM patients with high ischemic risk post-PCI, especially following ACS or in the presence of multivessel disease. However, careful risk stratification is required to avoid overtreatment in patients with increased bleeding propensity.

2.3.3. De-Escalation Strategies: When and for Whom?

De-escalation strategies involve either switching from a potent P2Y12 inhibitor to a less potent one (e.g., ticagrelor to clopidogrel) or reducing the overall intensity or duration of DAPT. These strategies are being actively explored in high-risk diabetic patients to reduce bleeding complications without sacrificing ischemic protection. The TWILIGHT trial, and its upcoming TWILIGHT-DM substudy, demonstrated that switching to ticagrelor monotherapy after 3 months of DAPT significantly reduced bleeding while maintaining ischemic protection in high-risk patients undergoing PCI, including those with diabetes [26]. These findings suggest that select diabetic patients, particularly those at increased bleeding risk or after the critical early post-PCI period—may benefit from early de-escalation. Other approaches include using platelet function testing or genotype-guided therapy to inform de-escalation decisions, although these methods are not yet universally adopted in clinical practice. The optimal antithrombotic strategy for diabetic patients after PCI involves a nuanced understanding of the interplay between ischemic risk, bleeding risk, and therapeutic response. While DAPT remains the cornerstone of therapy, agent selection and treatment duration should be individualized. Clopidogrel, though still in use, may be insufficient for many diabetic patients. Ticagrelor and prasugrel are preferred in those with high thrombotic burden, while de-escalation strategies and risk prediction tools offer promising avenues for personalizing care. Ongoing trials and innovations in precision medicine are expected to further refine these approaches.

3. Novel therapeutic strategies

The persistent residual ischemic risk in patients with type 2 diabetes mellitus following percutaneous coronary intervention, even with optimized dual antiplatelet therapy, has prompted the investigation of novel antithrombotic strategies. Advances have focused on targeting complementary pathways of thrombogenesis, personalizing antiplatelet therapy intensity, and integrating cardiometabolic agents with proven cardiovascular benefits. This section outlines emerging therapeutic approaches, including dual pathway inhibition, monotherapy strategies, and potential synergy with sodium-glucose cotransporter 2 (SGLT2) inhibitors and glucagon-like peptide-1 receptor agonists (GLP-1 RAs).

3.1. Dual Pathway Inhibition (Aspirin + Low-Dose Rivaroxaban)

Dual pathway inhibition refers to the simultaneous inhibition of platelet activation and the coagulation cascade. This approach was most notably evaluated in the COMPASS trial, which randomized patients with stable atherosclerotic cardiovascular disease (ASCVD), including a significant subgroup with T2DM, to receive low-dose rivaroxaban (2.5 mg twice daily) plus aspirin versus aspirin alone [27]. The combination therapy significantly reduced the risk of major adverse cardiovascular events, including cardiovascular death, myocardial infarction, and stroke, compared to aspirin monotherapy (HR 0.76; 95% CI, 0.66–0.86; $p < 0.001$). In patients with diabetes, the absolute risk reduction was more pronounced due to their higher baseline risk, suggesting that dual pathway inhibition may be particularly beneficial in this population [28]. However, the enhanced ischemic protection was offset by an increase in major bleeding, primarily gastrointestinal, without a significant rise in fatal or intracranial hemorrhage. The COMPASS findings led to the inclusion of aspirin plus low-dose rivaroxaban as a Class IIa recommendation in ESC guidelines for selected patients with chronic CAD and high ischemic risk [29]. For post-PCI diabetic patients with polyvascular disease, prior MI, or extensive coronary involvement who are not at high bleeding risk, dual pathway inhibition represents an attractive long-term strategy, especially beyond the traditional DAPT duration. Further studies such as VOYAGER-PAD have extended this concept to patients with peripheral artery disease, many of whom also have diabetes, reinforcing the applicability of this strategy across vascular beds [30].

3.2. Monotherapy Approaches

Recent trials have challenged the paradigm of prolonged DAPT in all patients, especially in the context of bleeding risk and polypharmacy in T2DM. Monotherapy approaches aim to simplify antithrombotic regimens by withdrawing either aspirin or the P2Y12 inhibitor after a short initial period of DAPT. The TWILIGHT trial assessed the safety and efficacy of discontinuing aspirin after 3 months of DAPT in high-risk patients undergoing PCI, continuing with ticagrelor monotherapy. The trial demonstrated a significant reduction in bleeding without an increase in ischemic events [31]. Although the study included a broad population, a substantial proportion had diabetes, and the upcoming TWILIGHT-DM substudy is expected to provide more granular insight into diabetic patients. Similarly, the STOPDAPT-2 and GLOBAL LEADERS trials evaluated early aspirin discontinuation in favor of P2Y12 monotherapy with mixed results, depending on the P2Y12 agent and clinical setting [32]. Importantly, these strategies are most appealing in patients with controlled diabetes, low ischemic burden, or increased bleeding risk, such as the elderly or those with chronic kidney disease. Monotherapy also mitigates aspirin-related gastrointestinal toxicity and improves medication adherence. Ticagrelor monotherapy, in particular, offers potent platelet inhibition while eliminating the additive bleeding risk of dual therapy, though cost and tolerability (e.g., dyspnea) remain considerations. As the field advances,

biomarker-guided and AI-based risk assessment tools may help identify diabetic subgroups that derive net benefit from early monotherapy over extended DAPT, especially in elective PCI scenarios.

3.3. Combination with SGLT2 Inhibitors or GLP-1 RAs: Synergy?

Beyond traditional antithrombotic agents, cardiometabolic therapies such as SGLT2 inhibitors and GLP-1 receptor agonists have demonstrated robust cardiovascular benefits in patients with T2DM. These agents have primarily been studied in the context of heart failure and atherosclerotic cardiovascular disease, but emerging data suggest they may also modulate thrombosis pathways and influence ischemic outcomes post-PCI. SGLT2 inhibitors, such as dapagliflozin and empagliflozin, have been shown to reduce hospitalization for heart failure and major cardiovascular events in patients with T2DM, even in those without established heart failure [33]. Their mechanisms may extend beyond glycemic control to include reductions in oxidative stress, arterial stiffness, and possibly platelet reactivity. GLP-1 receptor agonists, including liraglutide and semaglutide, have also demonstrated reductions in MACE in diabetic patients with established cardiovascular disease [34]. Potential anti-inflammatory and antiplatelet effects—along with improved lipid profiles—suggest possible synergy with antiplatelet therapy. Preclinical studies have indicated that GLP-1 signaling can modulate platelet aggregation and improve endothelial function, while SGLT2 inhibitors may lower circulating thromboxane levels, thereby reducing platelet activation [35]. These findings support the hypothesis that co-administration with DAPT or monotherapy regimens may enhance ischemic protection in diabetic patients post-PCI. Although large-scale randomized trials are still needed to test these combinations directly in post-PCI populations, ongoing trials such as DAPA-MI aim to assess the role of dapagliflozin in post-MI patients regardless of diabetic status, which may offer indirect insights into synergy with antiplatelet strategies [36]. Future directions may include triple therapy approaches combining antiplatelets with cardiometabolic agents in selected patients, or risk-adapted regimens integrating AI-supported profiling of thrombo-inflammatory burden.

4. Conclusion

The evolving landscape of antithrombotic therapy in T2DM patients after PCI has moved beyond a "one-size-fits-all" approach. Dual pathway inhibition with low-dose rivaroxaban and aspirin offers an effective option for patients with persistent ischemic risk and low bleeding potential. Monotherapy approaches, particularly with ticagrelor, provide a safe alternative for high-bleeding-risk individuals after the early post-PCI phase. The integration of SGLT2 inhibitors and GLP-1 RAs opens promising avenues for enhancing vascular protection, with potential synergistic effects on thrombosis and inflammation. As our understanding deepens, these novel strategies must be validated through ongoing trials and implemented with careful risk stratification to ensure maximal benefit for this complex patient population.

Compliance with ethical standards

Acknowledgments

Authors thank to the Republican Specialized Scientific Practical Medical Center of Therapy and Medical Rehabilitation for technical support.

Disclosure of conflict of interest

No conflict of interest to be disclosed.

References

[1] Einarson TR, Acs A, Ludwig C, Panton UH. Prevalence of cardiovascular disease in type 2 diabetes: a systematic literature review of scientific evidence from across the world in 2007-2017. Cardiovasc Diabetol. 2018;17(1):83. Published 2018 Jun 8. doi:10.1186/s12933-018-0728-6

[2] Uzokov J, Alyavi A, Alyavi B, Abdullaev A. How artificial intelligence can assist with ischaemic heart disease. Eur Heart J. 2024;45(21):1866-1868. doi:10.1093/eurheartj/ehae030

[3] Rawshani A, Rawshani A, Franzén S, et al. Risk Factors, Mortality, and Cardiovascular Outcomes in Patients with Type 2 Diabetes. N Engl J Med. 2018;379(7):633-644. doi:10.1056/NEJMoa1800256

[4] Ubaydullaev S., Alyavi,A., Uzokov,J. Endothelial functional status in patients with atherosclerotic coronary artery disease and type 2 diabetes mellitus after COVID-19. Atherosclerosis. 2023;379: S155. DOI: 10.1016/j.atherosclerosis.2023.06.526

[5] Ubaydullaeva Z., Alyavi A., Uzokov J., Karimov B. Influence of dapagliflozin on lipid parameters in patients with coronary artery disease and type 2 diabetes mellitus. Atherosclerosis. 2023; 379: S197. DOI: 10.1016/j.atherosclerosis.2023.06.652

[6] Kayumov N., Uzokov J., Alyavi B., Bekzod K., Madjidov I., Mukhamedova M. Circulating exosomal biomarkers in patients with coronary artery disease and metabolic syndrome. European journal of clinical investigation. 2022;52.

[7] Uzokov J., Alyavi B., Orziev D., Ubaydullaev S. Endothelial functional status in patients with hypertension and type 2 diabetes mellitus after COVID-19. Journal of Hypertension 2023; 41(S3): e207. DOI: 10.1097/01.hjh.0000941064.31118.65

[8] Uzokov J. K., Alyavi B. A., Abdullaev A. X. Assessment of the clopidogrel action with regard to CYP2C19 gene polymorphisms in patients with coronary artery disease after implantation of DES stents. Eurasian Journal of Cardiology. 2029;(S1):309. [In Russian]

[9] Alyavi A., Abdullaev A., et al. Prevention of Myocardial Revascularization Complications in Coronary Artery Disease and Diabetes Mellitus. Kardiologija v Belarusi. 2024;16(4):270–280. doi: 10.34883/PI.2024.16.4.002 (in Russian)

[10] Yakubbekov N.T., Nikishin A.G., Mullabaeva G.U., et al. Some Indicators of Platelet Aggregation in Patients with Multivessel Coronary Disease on the Background of Diabetes Mellitus. Kardiologija v Belarusi. 2024;16(1):70–77. doi: 10.34883/PI.2024.16.1.006

[11] Uzokov J., Alyavi B., Alyavi A., Payziwv D., Orziev D., Madjitov I. Short-term outcomes after carotid stenting in patients with metabolic syndrome. European journal of neurology. 2022;29:555-555.

[12] Uzokov J., Alyavi B., Abdullaev A., Orziev D., Madjitov I. Clopidogrel efficacy after percutaneous coronary interventions considering with CYP2C19 genetic polymorphisms in patients with coronary heart disease. Atherosclerosis. 2022;355(194):194. DOI: 10.1016/j.atherosclerosis.2022.06.788

[13] Alyavi A., Alyavi B., Uzokov J. Prognostic value of metabolic syndrome for the development of type 2 diabetes mellitus and cardiovascular disease. Atherosclerosis. 2020;315, e180. DOI: 10.1016/j.atherosclerosis.2020.10.559

[14] Alyavi B.A., Uzokov J.K., Mukhitdinova O. Antiplatelet therapy in coronary artery disease. Indian Journal of Forensic Medicine & Toxicology. 2021;15(3):2111-2115. https://doi.org/10.37506/ijfmt.v15i3.15628

[15] Mukhamedova M., Orziev D. Z., Uzokov J. K., Abdullaev A. X. Optimization of antiplatelet therapy in patients with coronary artery disease and type 2 diabetes mellitus after percutaneous coronary interventions. European Journal of Cardiovascular Nursing. 2023;22(S1): zvad064-111. doi.org/10.1093/eurjcn/zvad064.111

[16] Eikelboom JW, Connolly SJ, Bosch J, et al. Rivaroxaban with or without Aspirin in Stable Cardiovascular Disease. N Engl J Med. 2017;377(14):1319-1330. doi:10.1056/NEJMoa1709118

[17] Orziev D., Abdullaev A., Alyavi B., Uzokov J. Efficacy of ticagrelol in patients with chronic coronary syndrome and type 2 diabetes mellitus after percutaneous coronary interventions. Atherosclerosis. 2023;379: S174. DOI: 10.1016/j.atherosclerosis.2023.06.586

[18] Collet JP, Thiele H, Barbato E, et al. 2020 ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation Eur Heart J. 2021;42(19):1908. doi: 10.1093/eurheartj/ehaa895.

[19] Bonaca MP, Bhatt DL, Cohen M, et al. Long-term use of ticagrelor in patients with prior myocardial infarction. N Engl J Med. 2015;372(19):1791-1800. doi:10.1056/NEJMoa1500857

[20] Uzokov J., Alyavi B., Karimov B., Abdullaev A. Relationship between blood inflammation state and platelet aggregation rate in patients with atherosclerotic coronary artery disease after percutaneous coronary interventions. Atherosclerosis. 2023;379:S174-S175. DOI: 10.1016/j.atherosclerosis.2023.06.588

[21] Payziev D., Alyavi B. A., Uzokov J. K., Orziev D. Z. Essesment of short-term clinical outcomes in coronary artery disease patients after PCI on the background of low glycemic diet. European Journal of Cardiovascular Nursing. 2023;22(S1):zvad064-109. http://doi.org/10.1093/eurjcn/zvad064.109

[22] Uzokov J. Influence of variability in risk factors on cardiovascular disease outcomes in type 2 diabetes mellitus. Eur J Prev Cardiol. Published online May 25, 2023. doi:10.1093/eurjpc/zwad181

[23] Capodanno D, Petronio AS, Prendergast B, et al. Standardized definitions of structural deterioration and valve failure in assessing long-term durability of transcatheter and surgical aortic bioprosthetic valves: a consensus statement from the European Association of Percutaneous Cardiovascular Interventions (EAPCI) endorsed by the European Society of Cardiology (ESC) and the European Association for Cardio-Thoracic Surgery (EACTS). Eur Heart J. 2017;38(45):3382-3390. doi:10.1093/eurheartj/ehx303

[24] Wallentin L, Becker RC, Budaj A, et al. Ticagrelor versus clopidogrel in patients with acute coronary syndromes. N Engl J Med. 2009;361(11):1045-1057. doi:10.1056/NEJMoa0904327

[25] Nijjer SS, Davies JE, Francis DP. Quantitative comparison of clopidogrel 600 mg, prasugrel and ticagrelor, against clopidogrel 300 mg on major adverse cardiovascular events and bleeding in coronary stenting: synthesis of CURRENT-OASIS-7, TRITON-TIMI-38 and PLATO. Int J Cardiol. 2012;158(2):181-185. doi:10.1016/j.ijcard.2011.12.046

[26] Nicolas J, Baber U, Mehran R. TWILIGHT: A Randomized Trial of Ticagrelor Monotherapy Versus Ticagrelor Plus Aspirin Beginning at 3 Months in High-risk Patients Undergoing Percutaneous Coronary Intervention. US Cardiol. 2020 May 15;14:e04. doi: 10.15420/usc.2019.02.

[27] Pyne L, Smyth A, Molnar AO, et al. The Effects of Pantoprazole on Kidney Outcomes: Post Hoc Observational Analysis from the COMPASS Trial. J Am Soc Nephrol. 2024;35(7):901-909. doi:10.1681/ASN.0000000000000356

[28] Anand SS, Eikelboom JW, Dyal L, et al. Rivaroxaban Plus Aspirin Versus Aspirin in Relation to Vascular Risk in the COMPASS Trial. J Am Coll Cardiol. 2019;73(25):3271-3280. doi:10.1016/j.jacc.2019.02.079

[29] Knuuti J, Wijns W, Saraste A, et al. 2019 ESC Guidelines for the diagnosis and management of chronic coronary syndromes [published correction appears in Eur Heart J. 2020 Nov 21;41(44):4242. doi: 10.1093/eurheartj/ehz825.]. Eur Heart J. 2020;41(3):407-477. doi:10.1093/eurheartj/ehz425

[30] Bonaca MP, Bauersachs RM, Anand SS, et al. Rivaroxaban in Peripheral Artery Disease after Revascularization. N Engl J Med. 2020;382(21):1994-2004. doi:10.1056/NEJMoa2000052

[31] Mehran R, Baber U, Sharma SK, et al. Ticagrelor with or without Aspirin in High-Risk Patients after PCI. N Engl J Med. 2019;381(21):2032-2042. doi:10.1056/NEJMoa1908419

[32] Watanabe H, Domei T, Morimoto T, et al. Effect of 1-Month Dual Antiplatelet Therapy Followed by Clopidogrel vs 12-Month Dual Antiplatelet Therapy on Cardiovascular and Bleeding Events in Patients Receiving PCI: The STOPDAPT-2 Randomized Clinical Trial. JAMA. 2019;321(24):2414-2427. doi:10.1001/jama.2019.8145

[33] McMurray JJV, Solomon SD, Inzucchi SE, et al. Dapagliflozin in Patients with Heart Failure and Reduced Ejection Fraction. N Engl J Med. 2019;381(21):1995-2008. doi:10.1056/NEJMoa1911303

[34] Marso SP, Daniels GH, Brown-Frandsen K, et al. Liraglutide and Cardiovascular Outcomes in Type 2 Diabetes. N Engl J Med. 2016;375(4):311-322. doi:10.1056/NEJMoa1603827

[35] Virani SS, Newby LK, Arnold SV, et al. 2023 AHA/ACC/ACCP/ASPC/NLA/PCNA Guideline for the Management of Patients With Chronic Coronary Disease: A Report of the American Heart Association/American College of Cardiology Joint Committee on Clinical Practice Guidelines [published correction appears in Circulation. 2023 Sep 26;148(13):e148. doi: 10.1161/CIR.0000000000001183.] [published correction appears in Circulation. 2023 Dec 5;148(23):e186. doi: 10.1161/CIR.0000000000001195.]. Circulation. 2023;148(9):e9-e119. doi:10.1161/CIR.0000000000001168

[36] von Lewinski D, Kolesnik E, Tripolt NJ, Pferschy PN, Benedikt M, Wallner M, Alber H, Berger R, Lichtenauer M, Saely CH, Moertl D, Auersperg P, Reiter C, Rieder T, Siller-Matula JM, Gager GM, Hasun M, Weidinger F, Pieber TR, Zechner PM, Herrmann M, Zirlik A, Holman RR, Oulhaj A, Sourij H. Empagliflozin in acute myocardial infarction: the EMMY trial. Eur Heart J. 2022 Nov 1;43(41):4421-4432. doi: 10.1093/eurheartj/ehac494.