E-ISSN: 3062-3227

INTERVENTIONAL CARDIOLOGY PERSPECTIVES

OFFICIAL JOURNAL OF THE SOCIETY OF CARDIOVASCULAR INTERVENTIONS

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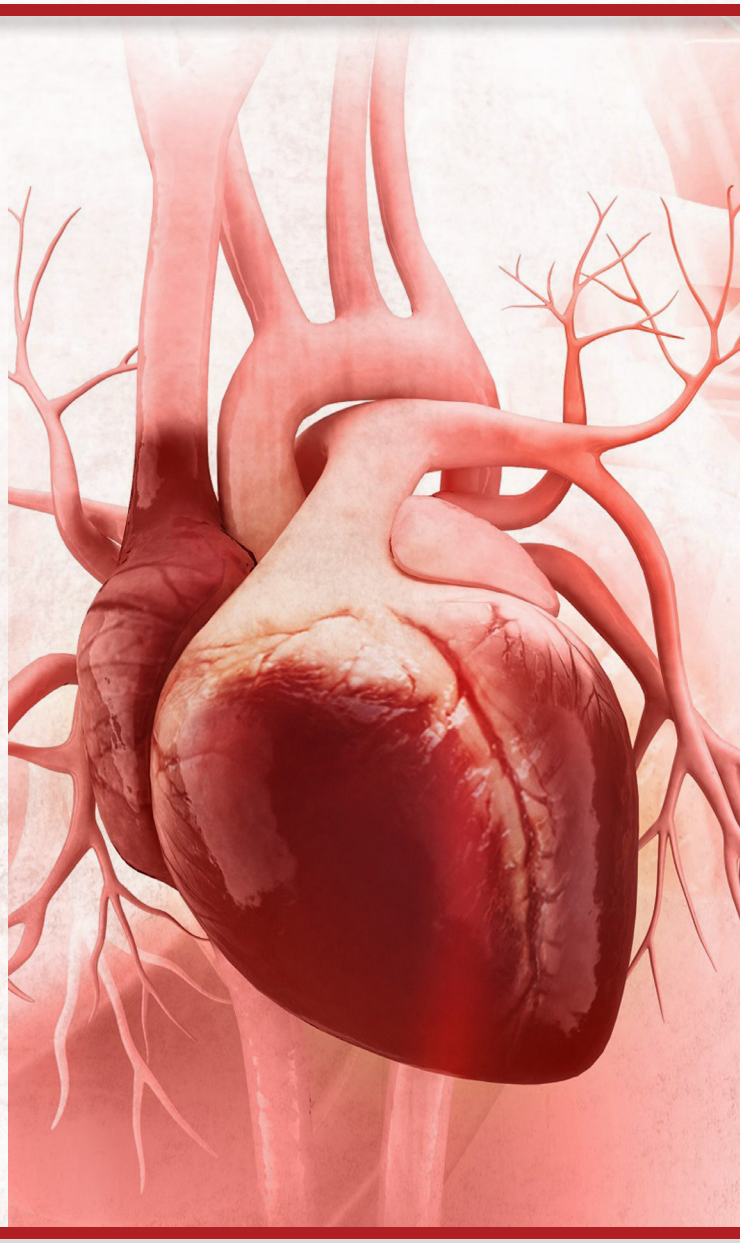
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VOLUME: 2 ISSUE: 1 APRIL 2026

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VOLUME: 2 | ISSUE: 1 | APRIL 2026

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The editorial and publication processes of Interventional Cardiology Perspectives are conducted in accordance with the guidelines of the ICMJE, WAME, CSE, COPE, EASE, and NISO, as well as the Principles of Transparency and Best Practice in Scholarly Publishing.

The journal is published online and is indexed in **DOAJ, GALE, EBSCO, Sudoc, İdealOnline, J-Gate, Turkiye Citation Index**, and **Türk Medline.**

Owner: Society of Cardiovascular Interventions

Responsible Manager: Harun Kundi

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Web: www.galenos.com.tr
Publisher Certificate Number: 14521

Publication Date: April 2026
E-ISSN: 3062-3227
International scientific journal published quarterly.

INTERVENTIONAL CARDIOLOGY PERSPECTIVES

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INTERVENTIONAL CARDIOLOGY PERSPECTIVES

Inter Cardio Pers 2026;2(1):1-2

DOI: 10.4274/intercardiopers.2026.2026.250326

EDITORIAL

Mid-Term Reassurance, Long-Term Questions: Reintervention After TAVR

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The rapid evolution of transcatheter aortic valve replacement (TAVR) has fundamentally transformed the management of severe aortic stenosis. Initially reserved for inoperable or high-risk patients, TAVR has steadily expanded across the full surgical risk spectrum, including younger individuals with longer life expectancy. As this transition progresses, focus has shifted from procedural success toward a more fundamental question: how durable are transcatheter valves over the long term, and what role will reintervention play in the lifetime management of aortic valve disease?

Long-term follow-up from landmark randomized trials recently updated in *The New England Journal of Medicine* and *JACC* is beginning to provide crucial insights as the field approaches the 7-year horizon. In the PARTNER 3 trial evaluating balloon-expandable valves in low-risk patients, extended follow-up shows sustained clinical outcomes with low rates of structural valve failure and reintervention, broadly comparable to surgical bioprosthetic replacement.[1] Similarly, follow-up from the Evolut Low Risk trial assessing self-expanding valves confirms excellent hemodynamic performance through 6–7 years, while highlighting a gradual accumulation of valve-related events over time.[2]

These findings provide reassuring evidence that contemporary transcatheter valves perform well through the mid-term period. Yet they also mark a pivotal transition. For the first time, durability curves are extending into the period when structural valve deterioration historically emerges in surgical bioprostheses typically beyond the first decade after implantation. Consequently, interpreting reintervention rates requires careful clinical context.

Randomized trials offer the most rigorous framework for evaluating device durability. Patients enrolled in studies such as PARTNER 3 and Evolut Low Risk follow structured protocols that include regular clinical assessment and scheduled echocardiographic surveillance. Valve hemodynamics are closely monitored, and potential valve dysfunction is assessed using standardized definitions and independent adjudication committees. Within this framework, reintervention reflects a carefully considered clinical decision guided by systematic surveillance.

In contrast, real-world practice is far less uniform. Outside the trial environment, follow-up imaging varies substantially, and thresholds for repeat intervention differ across institutions and operators. In elderly or highly comorbid patients, clinicians may adopt conservative management even when valve dysfunction is detected. Conversely, specialized structural heart programs may perform earlier redo procedures in selected patients with symptomatic valve failure. As a result, observed reintervention rates in routine practice reflect not only valve durability but also differences in clinical decision-making, institutional expertise, and patient selection.

This distinction becomes increasingly relevant as TAVR moves into younger populations. In the earliest TAVR cohorts—largely high-risk, older patients overall mortality limited the number of individuals surviving long enough to develop structural valve deterioration. In contemporary low-risk populations, life expectancy is substantially longer, making valve durability a key determinant of long-term procedural success. Thus, longer-term follow-up from randomized trials is critical to understanding how transcatheter valves may perform over decades rather than years.

Another significant development is the growing feasibility of redo TAVR procedures and the emergence of a "lifetime management" approach for aortic valve disease. Valve-in-valve TAVR has shown high procedural success and favorable early outcomes in observational studies.[3,4] As experience with repeat transcatheter interventions increases, reintervention may be seen not merely as a therapy failure but as a component of longitudinal disease management. Nevertheless, the mechanism and timing of initial valve dysfunction—whether due to structural valve degeneration, progressive regurgitation, or prosthesis–patient mismatch may strongly influence the feasibility and success of subsequent procedures.

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Cite as: Kundi H. Mid-term reassurance, long-term questions: reintervention after TAVR. *Inter Cardio Pers.* 2026;2(1):1-2

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These considerations underscore the need for ongoing data generation and careful interpretation of durability trends. Extended follow-up from contemporary randomized trials will be essential. As the PARTNER 3 and Evolut Low Risk cohorts approach the 10-year mark, they will provide crucial insights into whether durability trends stabilize or accelerate. Greater emphasis should also be placed on mechanistic characterization of bioprosthetic valve dysfunction rather than relying solely on reintervention as an endpoint. The VARC-3 consensus definitions provide a framework to distinguish structural valve deterioration, non-structural valve dysfunction, and clinical valve failure.[5] Finally, these emerging durability data must inform heart-team discussions when selecting the optimal initial treatment strategy for younger patients likely to require multiple valve procedures over their lifetime.

In summary, seven-year data from randomized trials suggest that contemporary TAVR platforms remain highly effective through the mid-term period, with relatively low rates of valve failure and reintervention. At the same time, extending follow-up into longer time horizons reminds us that midterm reassurance should not be mistaken for definitive long-term durability. As the field moves toward a lifetime management paradigm for aortic valve disease, careful interpretation of reintervention trends—and continued surveillance of valve performance will remain essential for guiding patient selection and ensuring long-term outcomes in younger TAVR recipients.

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INTERVENTIONAL CARDIOLOGY PERSPECTIVES

Inter Cardio Pers 2026;2(1):3-8

DOI: 10.4274/intercardiopers.2026.2026-1-2

ORIGINAL ARTICLE

Design and Rationale of the DCB-DCS Study: Procedural Success and Short- and Long-Term Outcomes of Drug-Coated Coronary Balloons Used in Different Clinical Scenarios

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ABSTRACT

Background: Drug-coated balloons (DCBs) are now an established therapeutic option for the treatment of in-stent restenosis (ISR) and small-vessel coronary artery disease. They enable effective local delivery of antiproliferative agents without leaving a permanent metallic scaffold. Nevertheless, real-world evidence regarding the performance of DCBs in broader and more complex coronary lesion subsets remains limited.

Aim: The [DCB in different clinical scenarios (DCS)] study (ClinicalTrials.gov identifier: NCT06915597) is designed to evaluate procedural strategies, safety, and clinical outcomes associated with DCB use across a wide range of coronary lesion types in routine clinical practice.

Study Design: This is a multicenter, observational, prospective and retrospective study.

Methods: The DCB-DCS study is a national, multicenter, observational registry that integrates both prospective and retrospective cohorts from 17 centers across Türkiye. Consecutive patients undergoing DCB angioplasty for *de novo* lesions, ISR, small-vessel disease, bifurcation lesions, or chronic total occlusions will be enrolled. The selection of the DCB device, lesion preparation strategy, and use of intravascular imaging will be left entirely to the operator's discretion. All procedures will be performed in accordance with contemporary DCB recommendations. Routine angiographic follow-up will not be mandated. The primary endpoint is procedural success. Secondary endpoints include target lesion revascularization, target vessel revascularization, acute vessel occlusion, all-cause mortality, and bleeding events.

Results: Data collection is currently ongoing. Baseline demographic and clinical characteristics, procedural details, and clinical outcomes will be analyzed after completion of patient enrollment and follow-up.

Conclusion: As the largest and most comprehensive DCB registry conducted in Türkiye, the DCB-DCS study will provide real-world evidence on the expanding application of DCBs across diverse coronary scenarios. The findings are expected to identify optimal procedural approaches and inform the design of future randomized controlled trials comparing DCBs with contemporary-generation stent technologies.

Keywords: Design and methodology, drug-coated balloon, multicenter trial

INTRODUCTION

Coronary drug-coated balloons (DCBs) have emerged over the past decade as an important device-based strategy for the treatment of coronary artery disease (CAD). Initially established as an effective therapy for in-stent restenosis (ISR), DCBs combine transient local delivery of an antiproliferative drug with the advantage of leaving no permanent implant. This approach avoids additional layers of metal and preserves future coronary options. The therapeutic rationale, together with accumulating clinical evidence, has driven interest in expanding DCB use to other clinical scenarios, including de novo small-vessel disease and selected complex lesions.[1,2] High-quality randomized trials support the use of DCBs in specific settings. The BASKET-SMALL 2 trial demonstrated non-inferiority of paclitaxel-coated balloon angioplasty compared with drug-eluting stents (DES) for *de novo* small-vessel coronary lesions, establishing DCB as a viable alternative in vessels where stent implantation is undesirable. Similarly, randomized and large observational studies in ISR have consistently reported favorable outcomes with DCB treatment compared with plain balloon angioplasty and a competitive clinical profile relative to repeat stenting.[2,3] Despite growing evidence, several knowledge gaps persist. Most published data originate from selected patient populations, single-device cohorts, or trials focused on a single clinical scenario

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Cite as: Kahraman F, Kılınç M, Göktekin Ö. Design and rationale of the DCB-DCS study: procedural success and short- and long-term outcomes of drug-coated coronary balloons used in different clinical scenarios. *Inter Cardio Pers.* 2026;2(1):3-8

Received: 22.01.2026
Accepted: 02.03.2026
Epub: 06.03.2026
Publication Date: 10.04.2026

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(e.g., ISR or small-vessel disease). Comparative effectiveness, safety, and long-term outcomes of different DCB platforms across a broad spectrum of real-world clinical scenarios—including acute coronary syndromes, *de novo* large-vessel lesions, bifurcations, calcified lesions after adequate preparation, and mixed prospective/retrospective cohorts—remain incompletely characterized. Moreover, heterogeneity in lesion preparation, imaging guidance, device selection, and endpoint definitions across studies complicates literature synthesis, prompting calls for standardized definitions and endpoints in DCB research.[4,5] Concurrently, regulatory approvals and the introduction of newer devices have increased access to coronary DCB technology, highlighting the need for real-world data to inform best practices and patient selection. Contemporary registry and consensus literature emphasize the importance of large, multicenter, pragmatic datasets that capture the heterogeneity of routine clinical practice and enable subgroup analyses to identify patients and lesion types most likely to benefit from a DCB-first or DCB-only approach.[6]

Against this background, the DCB-DCS study—a national, multicenter (17 centers in Türkiye), observational registry combining prospective and retrospective cohorts—aims to systematically describe contemporary patterns of DCB use, procedural strategies (including lesion preparation and adjunctive imaging), and clinical and angiographic outcomes across diverse clinical scenarios. The study is designed to address pragmatic evidence gaps by providing real-world effectiveness and safety data, harmonized using standardized endpoints, and to generate hypotheses for future randomized evaluations.

METHODS

Study Design

This study is a multicenter, observational registry of DCB interventions, integrating both retrospective and prospective data. Data will be collected from 17 centers across Türkiye, including all consecutive patients undergoing DCB treatment for diverse clinical scenarios—*de novo* lesions, small vessels, ISR, bifurcation lesions, and chronic total occlusions (CTOs). The study was approved by Pamukkale University Ethics Committee (approval no: 07, dated 08.04.2025) and aims to comprehensively evaluate procedural characteristics, lesion preparation strategies, and short- to mid-term outcomes associated with DCB use in routine clinical practice. A summary of the study design is provided in Table 1.

Rationale for Mixed Prospective and Retrospective Design

A hybrid prospective-retrospective design was adopted to maximize sample size, capture temporal trends in DCB utilization, and enhance external validity. Retrospective data facilitate rapid accrual of real-world cases and hypothesis generation, while the prospective arm allows standardized data collection, predefined endpoint assessment, and bias monitoring. This approach balances feasibility with methodological rigor and supports robust subgroup analyses. All diagnostic and therapeutic procedures, including DCB use, will be performed according to standard clinical indications and physician discretion, without any protocol-mandated interventions or experimental procedures. Data will be collected solely for observational and analytical purposes, and patient management will not be influenced by study participation.

Study Population

Eligible participants are adults (≥18 years) who underwent percutaneous coronary intervention with a DCB for various clinical scenarios, including *de novo* coronary lesions, small-vessel disease, ISR, bifurcation lesions, and CTO. Inclusion and exclusion criteria are detailed in Table 2. All participants must be capable of providing informed consent. The study flow—including patient enrollment, eligibility assessment, and follow-up—is illustrated in Figure 1.

Sample Size and Enrollment Strategy

This registry is designed as a large, nationwide, multicenter study intended to reflect contemporary real-world practice across a broad spectrum of clinical scenarios. Based on the procedural volume of participating centers, approximately 2,000–3,000 patients are

Table 1. Study overview and design summary

Feature	Description
Study name	DCB-DCS
Design	Multicentre, observational (retrospective + prospective registry)
Centers	17 tertiary cardiology centers across Türkiye
Population	Consecutive patients undergoing DCB angioplasty
Clinical scenarios	*de novo* lesions, small vessels, ISR, bifurcation, CTO
Primary endpoint	Procedural success (residual stenosis <30%, TIMI 3 flow, no major dissection)
Secondary endpoints	TLR, TVR, death, acute occlusion, bleeding, MACE
Follow-up	Clinical follow-up at 1, 6, and 12 months; angiographic follow-up per operator discretion
Statistical plan	Descriptive statistics, Kaplan–Meier survival analyses, subgroup comparison by scenario

CTO: Chronic total occlusion, DCB: Drug-coated coronary balloon, ISR: In-stent restenosis, MACE: Major adverse cardiac events, TLR: Target lesion revascularization, TVR: Target vessel revascularization, TIMI 3: Thrombolysis in myocardial infarction grade 3, DCS: Different clinical scenarios

Table 2. Inclusion and exclusion criteria

Inclusion criteria	Exclusion criteria
Age ≥18 years	Cardiogenic shock at presentation
Undergoing DCB angioplasty for eligible lesion	Unsuitable anatomy for DCB treatment
Any clinical presentation (stable CAD, ACS)	Contraindication to antiplatelet therapy
Availability of complete procedural data	Life expectancy <1 year

DCB: Drug-coated coronary balloon, CAD: Coronary artery disease, ACS: Acute coronary syndrome

expected to be enrolled over an 18–30 month recruitment period. Consecutive patient inclusion will be encouraged at each site to minimize selection bias.

Given the observational nature of the registry, a formal power calculation was not considered mandatory. Nevertheless, the planned sample size is expected to provide adequate statistical precision for evaluating clinical outcomes and to support meaningful subgroup analyses. Enrollment distribution across centers will be monitored to ensure balanced representation and data quality.

Procedural Characteristics

No specific intervention is mandated by the study protocol; the decision to use a DCB is entirely at the discretion of the treating physician, based on clinical judgment and individual patient characteristics. All procedures will be performed in accordance with current DCB recommendations and best-practice guidelines.

Lesion preparation is considered a key determinant of procedural success. Operators will aim for residual stenosis <30%, thrombolysis in myocardial infarction grade 3 (TIMI 3) flow, and absence of major dissections (type A–C dissections are acceptable). Predilatation may be performed using standard, cutting, or scoring balloons, according to lesion characteristics. Intravascular imaging modalities, such as intravascular ultrasound (IVUS) or optical coherence tomography (OCT), are encouraged to optimize vessel sizing, guide lesion preparation, and assess adequacy of DCB delivery. DCBs will generally be sized at a 1:1 ratio relative to the reference vessel diameter, with inflation maintained for 30–60 seconds to ensure optimal drug transfer. Bailout stenting is permitted in cases of significant recoil or flow-limiting dissection following DCB deployment. A summary of recommended lesion preparation strategies and procedural steps is provided in Table 3.

Patients with cardiogenic shock, contraindications to antiplatelet therapy, active bleeding, uncontrolled coagulopathy, known allergy to the drug coating or balloon material, life expectancy <1 year, or pregnancy will be excluded from the study.

Clinical Endpoints

The primary endpoint of the registry is target lesion failure at 12 months, defined as a composite of cardiac death, target-vessel myocardial infarction, and clinically driven target lesion revascularization (TLR).

Econdary endpoints include major adverse cardiac events, defined as a composite of all-cause death, myocardial infarction, and clinically driven target vessel revascularization (TVR) as well as the individual components of these composites. Myocardial infarction will be defined according to the Fourth Universal Definition of Myocardial Infarction.

Procedural success is a key secondary procedural endpoint and is defined as successful treatment of the target lesion with a DCB resulting in final TIMI 3 flow, residual stenosis <30% by visual estimation, and absence of flow-limiting dissection requiring bailout stent implantation.

Clinically driven TLR/TVR is defined as repeat revascularization performed in the presence of ischemic symptoms and/or objective evidence of myocardial ischemia, including positive non-invasive testing or angiographic findings consistent with acute coronary syndrome.

Bleeding events will be classified according to the Bleeding Academic Research Consortium criteria. Acute vessel closure is defined as abrupt occlusion of the treated segment occurring during the procedure or within 24 hours.

Angiographic endpoints, including late lumen loss, will be evaluated in patients undergoing clinically indicated follow-up angiography. As routine angiographic follow-up is not mandated, these analyses will be considered exploratory.

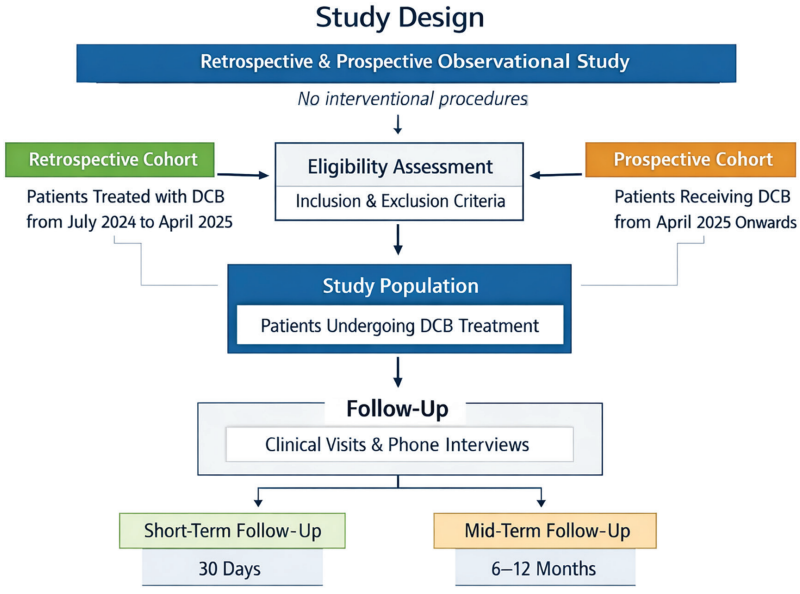


Figure 1. Study flow diagram
DCB: Drug-coated coronary balloon

Table 3. Lesion preparation and procedural recommendations

Procedural step	Recommendation/target
Lesion preparation	Predilatation with standard, cutting, or scoring balloon
Optimal result before DCB	<30% residual stenosis, TIMI 3 flow, no ≥ type C dissection
Balloon sizing	1:1 ratio with reference vessel diameter
Inflation duration	At least 30–60 seconds
Bailout stenting	Permitted if flow-limiting dissection or recoil
Imaging use	IVUS or OCT encouraged for vessel sizing and optimization

DCB: Drug-coated coronary balloon, IVUS: Intravascular ultrasound, OCT: Optical coherence tomography, TIMI 3: Thrombolysis in myocardial infarction grade 3

Follow-up and Outcome Assessment

Clinical follow-up will be conducted at 1, 6, and 12 months after the index procedure, with additional evaluations performed as clinically indicated. Follow-up data will be collected through outpatient clinic visits, structured telephone interviews, and review of hospital records. When available, national health system databases may also be used to enhance event detection and minimize loss to follow-up.

To ensure data quality and consistency across participating centers, standardized data collection forms will be employed. Clinical events will be reported by each site and systematically recorded in the registry database. Where necessary, source document verification may be performed to confirm reported outcomes.

Every effort will be made to achieve complete follow-up. Patients for whom follow-up information cannot be obtained will be censored at the time of the last confirmed contact.

Statistical Analysis

All statistical analyses will be performed using SPSS (IBM Corp., Armonk, NY, USA) or R software (R Foundation for Statistical Computing, Vienna, Austria). Continuous variables will be presented as mean ± standard deviation or median with interquartile range, depending on data distribution. Categorical variables will be expressed as counts and percentages. Normality will be assessed using the Kolmogorov–Smirnov test. Comparisons between groups will be performed using the Student's t-test or Mann–Whitney U test for continuous variables, and the chi-square or Fisher's exact test for categorical variables, as appropriate.

Time-to-event outcomes will be analyzed using Kaplan–Meier survival estimates and compared using the log-rank test. To identify independent predictors of clinical outcomes and adjust for potential confounders related to patient characteristics, lesion complexity, device type, and procedural strategy, multivariable regression models will be constructed. Cox proportional hazards models will be used for time-to-event analyses, with results reported as hazard ratios and 95% CIs. Covariates will be selected based on clinical relevance, prior literature, and variables demonstrating significance in univariate analyses.

Given the multicenter design, clustering by participating centers will be addressed using mixed-effects models or robust standard errors, as appropriate, to account for potential inter-center variability and operator-dependent effects. Subgroup analyses may be conducted across predefined clinical scenarios when clinically meaningful.

Considering the hybrid retrospective–prospective design, the two cohorts will initially be analyzed separately to identify potential differences in baseline characteristics, treatment patterns, and outcomes. Cohort type will then be incorporated as a predefined covariate in multivariable models. Sensitivity analyses restricted to the prospective cohort will be performed to evaluate the robustness of the findings. Additionally, stratified analyses by enrollment period may be conducted to explore potential temporal changes in clinical practice.

Efforts will be made to minimize missing data through standardized collection procedures across centers. When appropriate, multiple imputation techniques may be applied, and sensitivity analyses will be considered to assess the impact of missing data on study outcomes. All tests will be two-sided, and a p value <0.05 will be considered statistically significant.

RESULTS

Data collection is ongoing, and the complete analysis will be reported in a future publication. Key procedural characteristics, clinical outcomes, and follow-up data will be presented once the study is finalized.

DISCUSSION

The DCB-DCS study will provide one of the largest and most comprehensive datasets on the use of DCBs across diverse real-world clinical settings in Türkiye. While the efficacy of DCBs is well established in ISR and small-vessel disease, their expanding role in more complex lesions such as bifurcations, CTO, and large-vessel *de novo* disease—remains an area of growing clinical interest. This registry aims to address this knowledge gap by systematically documenting procedural strategies, device selection, and outcomes in routine interventional practice. Over the last decade, evidence supporting the "leave nothing behind" strategy has gained momentum. DCB angioplasty enables effective antiproliferative drug delivery without permanent scaffolding, representing a paradigm shift in coronary intervention. Randomized and observational studies have demonstrated comparable efficacy and superior preservation of vessel physiology compared with new-generation DES in appropriately selected lesions.[7-9] Additionally, the avoidance of polymer hypersensitivity, late stent thrombosis, and impaired vasomotion makes DCBs particularly advantageous in patients with diffuse disease, high bleeding risk, or contraindications to prolonged dual antiplatelet therapy.[6,10] Despite these advances,

real-world data remain heterogeneous, reflecting variability in lesion preparation, balloon technology, and operator experience. The DCB-DCS registry is designed to address these limitations by combining retrospective and prospective cohorts from 17 centers, thereby capturing temporal trends and practice evolution within a standardized framework. This pragmatic design aligns with recommendations from the Drug-Coated Balloon Academic Research Consortium for harmonized data collection and endpoint definitions.[5,11] Moreover, the inclusion of multiple DCB platforms—paclitaxel- and sirolimus-based—will allow indirect comparison of performance across technologies under real-world conditions.

A key strength of the DCB-DCS study is its inclusivity. By enrolling all-comer patients across a broad spectrum of clinical scenarios and permitting procedural discretion by treating physicians, the registry reflects genuine clinical decision-making rather than protocol-driven treatment algorithms. This approach complements efforts to expand the evidence base beyond tightly controlled randomized trials, acknowledging that registry data provide critical insights into external validity and long-term safety.[12,13] Furthermore, the study will examine how lesion preparation strategies, imaging support (IVUS or OCT), and bailout stenting decisions influence outcomes across lesion subsets. Previous studies have highlighted the importance of meticulous vessel preparation and optimal balloon sizing in achieving durable results with DCBs.[14,15] Incorporating imaging guidance is expected to further refine procedural endpoints and identify predictors of restenosis or late vessel remodeling. The results of the DCB-DCS registry are anticipated to inform clinical practice by identifying patient and lesion subsets that derive the greatest benefit from a DCB-first or DCB-only approach, clarifying procedural best practices, and generating hypotheses for future randomized evaluations. Beyond its national scope, the study may also contribute to international efforts to standardize DCB methodology and integrate this technology into global treatment algorithms for CAD.

Study Limitations

This registry has several limitations inherent to observational research. Despite planned multivariable adjustments, residual confounding cannot be entirely excluded. Additionally, treatment strategies and device selection are at the discretion of the operator, which may introduce variability reflective of real-world practice.

The hybrid retrospective–prospective design may result in heterogeneity in data quality and follow-up completeness; however, predefined statistical strategies are planned to mitigate this effect. Routine angiographic follow-up is not mandated; therefore, angiographic outcomes should be interpreted with caution and considered exploratory.

Although every effort will be made to ensure comprehensive follow-up, missing data or loss to follow-up cannot be completely eliminated. Nonetheless, standardized data collection procedures and structured follow-up methods are expected to support the overall reliability of the registry.

CONCLUSION

The DCB-DCS study is designed to provide comprehensive real-world evidence on the contemporary use of DCBs across a broad spectrum of coronary lesion subsets and clinical scenarios. By including all-comer patients and reflecting routine clinical decision-making, this registry complements existing randomized data and enhances understanding of procedural strategies, imaging guidance, and bailout stenting practices associated with DCB therapy.

The findings are expected to provide valuable insights into external validity, long-term safety, and outcome optimization, thereby informing daily clinical practice and guiding future recommendations and guideline development.

Ethics Committee Approval: The study was approved by Pamukkale University Ethics Committee (approval no: 07, dated 08.04.2025)

Informed Consent: Retrospective and prospective observational study.

Authorship Contributions: Surgical and Medical Practices: F.K., M.K., Ö.G., Concept: F.K., M.K., Ö.G., Design: F.K., M.K., Ö.G., Data Collection or Processing: F.K., M.K., Ö.G., Analysis or Interpretation: F.K., M.K., Ö.G., Literature Search: F.K., Writing: F.K., Ö.G.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study received no financial support.

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INTERVENTIONAL CARDIOLOGY PERSPECTIVES

Inter Cardio Pers 2026;2(1):9-15

DOI: 10.4274/intercardiopers.2026.2025-12-1

ORIGINAL ARTICLE

Association Between Admission Serum Magnesium Level and Clinical End Points in Patients with ST-Elevation Myocardial Infarction that Treated with Primary Angioplasty

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ABSTRACT

Background: Although serum magnesium (Mg) levels have been linked to atherogenesis and cardiovascular disease in the general population, evidence regarding their association with clinical outcomes in patients with acute myocardial infarction (MI) is inconsistent.

Aim: This study investigated the relationship between admission serum Mg levels and both in-hospital and long-term clinical outcomes in patients undergoing primary percutaneous coronary intervention.

Study Design: Retrospective cohort study.

Methods: This study included patients with ST-segment elevation MI (STEMI) who presented within 12 hours of symptom onset and underwent primary percutaneous coronary intervention between February 2011 and April 2015. After predefined exclusions, 1,119 of 1,270 patients were included. STEMI was diagnosed according to European Society of Cardiology/American College of Cardiology Foundation/American Heart Association criteria, and ethics approval was obtained with waiver of written informed consent. Admission clinical data and serum Mg levels were collected from medical records, with Mg measured before coronary angiography and grouped into five categories. All patients underwent femoral coronary angiography/stenting and received guideline-based medical therapy, with echocardiographic and follow-up data obtained from hospital records and telephone interviews.

Results: We included 1,119 patients with STEMI. The median follow-up duration was 25±16 months. An admission serum Mg cut-off of 1.83 mg/dL predicted acute stent thrombosis with 76% sensitivity and 65% specificity (area under the curve: 0.781; 95% confidence interval: 0.543–0.920; p=0.024). Apart from acute stent thrombosis, all other clinical endpoints were comparable across different serum Mg level groups.

Conclusion: Low admission serum Mg was significantly associated with acute stent thrombosis. However, no significant association was observed between serum Mg levels and in-hospital or long-term major adverse cardiovascular events, including cardiovascular mortality, target vessel revascularization, and stroke.

Keywords: Magnesium, myocardial infarction, percutaneous coronary intervention

INTRODUCTION

Since the advent of primary percutaneous coronary intervention (PCI), the treatment of ST-segment elevation myocardial infarction (STEMI) has become substantially more effective.[1,2] Primary PCI is now the default treatment strategy for eligible STEMI patients.[1,2] Despite this advancement, STEMI patients remain at risk for both in-hospital and long-term adverse events.[3,4] Considerable efforts have been made to identify potential contributors to adverse outcomes in patients with acute myocardial infarction (MI). Clinical and laboratory parameters associated with unfavorable outcomes have been investigated, though many show inconsistent or controversial results.[5] Magnesium (Mg^{2+}) is the second most abundant intracellular cation and participates in over 325 enzymatic reactions, including lipid peroxidation, blood pressure regulation, and glucose metabolism.[5] Meta-analyses of prospective cohort studies indicate that Mg plays a critical role in cardiovascular, electrical, and metabolic homeostasis, with an inverse relationship observed between serum Mg levels and cardiovascular disease risk.[6,7] Mg also influences atherogenesis by modulating inflammation and oxidative processes.[6-8]

During the acute phase of MI, serum Mg levels have been reported to decrease transiently.[9] Some studies suggest that low serum Mg is associated with major adverse cardiac events (MACEs), in-hospital stent thrombosis, electrocardiographic no-reflow, and long-term mortality in acute MI patients.[10-12] Conversely, large-scale studies have found no association between low serum Mg levels and either in-hospital

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Cite as: Alioğlu C, Şişman Uzunoğlan BH, Uzunoğlan S, Danışman E, Khanmohammadi M, Uluganyan M. Association between admission serum magnesium level and clinical end points in patients with ST-elevation myocardial Infarction that treated with primary angioplasty. *Inter Cardio Pers.* 2026;2(1):9-15

Received: 30.12.2025
Accepted: 14.03.2026
Publication Date: 10.04.2026

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or long-term adverse events in MI patients.[13,14] Additionally, Naksuk et al.[14] demonstrated a potential adverse effect of higher serum Mg levels ≥2.4 mg/dL in a large cohort of acute MI patients. Given these conflicting findings, we conducted this study to evaluate the potential role of admission serum Mg levels in predicting in-hospital and long-term adverse events in STEMI patients undergoing primary PCI.

METHODS

Study Population

This retrospective study included 1,270 patients recruited between February 2011 and April 2015. All consecutive patients meeting the inclusion criteria were considered. The exclusion criteria were: missing admission Mg levels (n=28), history of end-stage renal disease (n=20), use of Mg supplements (n=5), active cancer (n=12), infection (n=7), and cirrhosis (n=5). Additionally, patients with STEMI who were treated medically (n=29) or underwent coronary artery bypass grafting [(CABG), n=45] were excluded. After applying these criteria, a total of 1,119 patients were included in the analysis. Eligible patients presented within 12 hours of symptom onset and were diagnosed with STEMI, for which primary PCI was performed. STEMI diagnosis was based on the criteria proposed by the European Society of Cardiology/American College of Cardiology Foundation/American Heart Association (ESC/ACCF/AHA) committee.[15] The study was approved by Bezmialem Vakıf University Ethics Committee (approval no: 15, date: 08.10.2025). As this was a retrospective study, written informed consent was not obtained from patients.

Data Collection

Patients' demographic data, medical history, and in-hospital information were obtained from digital medical records. After STEMI diagnosis in the emergency department, venous blood samples were collected from the antecubital vein prior to coronary angiography. Mg levels were categorized as <1.8, 1.8 to <2.0, 2.0 to <2.2, 2.2 to <2.4, and ≥2.4 mg/dL. Laboratory analysis of Mg was performed using a Roche/Hitachi Modular Analyzer (Roche Diagnostics International Ltd., Rotkreuz, Switzerland) with a xylidyl blue reaction. Mg levels are expressed in milligrams per deciliter (mg/dL). Following primary PCI, patients were admitted to the coronary care unit (CCU). Bedside transthoracic echocardiography was performed on the day of PCI. Left ventricular ejection fraction (LVEF) was determined using a modified Simpson's method. Patients' follow-up data were obtained through telephone interviews and/or review of hospital records.

Coronary Angiography and Medication

Coronary angiography and stenting were performed via the femoral approach using a 7-F guiding catheter. At the emergency department, all patients received a loading dose of 300 mg of acetylsalicylic acid and 600 mg of clopidogrel, regardless of prior medication. The procedure employed non-ionic, low-osmolality contrast media. The use of tirofiban was at the discretion of the operator in the catheterization laboratory. After the procedure, patients were transferred to the CCU, where angiotensin-converting enzyme inhibitors, beta-blockers, and statins were administered according to the ESC PCI guidelines.[16]

Definitions

STEMI was defined according to the criteria proposed by the ESC/ACCF/AHA committee.[15] Diabetes mellitus (DM) was defined as a prior diagnosis of DM or a fasting blood glucose level ≥126 mg/dL during hospitalization. Hypertension and hyperlipidemia were defined by a previous diagnosis and/or use of relevant medications. MACEs included cardiovascular death, stroke, and reinfarction. Cardiovascular death encompassed death from any cardiovascular cause, stroke, or sudden unexplained death. Target vessel revascularization (TVR) was defined as the need for coronary stenting or bypass surgery after the initial procedure. Acute stent thrombosis was diagnosed according to the definitions of the Academic Research Consortium.[16,17]

Statistical Analysis

Categorical data are expressed as numbers, and continuous data as mean±standard deviation. One-way ANOVA and chi-square tests were used for univariate comparisons of baseline characteristics among Mg groups. Variables that were statistically significant in bivariate correlations with Mg levels ($p<0.05$) or considered clinically important were included in the final multivariable linear regression model, which was conducted using a forward stepwise approach. A p value <0.05 was considered statistically significant. Data were analyzed using IBM SPSS Statistics version 20 software (IBM, Armonk, NY, USA). The predictive ability of variables was evaluated using receiver operating characteristic (ROC) curves, with the area under the curve (AUC) calculated for each variable.

RESULTS

The study included 1,119 patients with STEMI who were treated with primary PCI. Most patients were male (90%), and the mean age was 54 years. The overall mean follow-up period was 25±16 months. Baseline clinical characteristics of the patients are presented in Table 1. Groups were comparable in terms of age, creatinine, glomerular filtration rate, hypertension, hyperlipidemia, smoking, and family history of coronary artery disease. LVEFs following primary PCI were similar between groups (p=0.424). Admission serum glucose levels and the prevalence of type 2 diabetes mellitus (T2DM) differed significantly between groups (both $p<0.001$). Stent length and diameter were comparable across all groups (p=0.891 and p=0.691, respectively).

Table 2 summarizes the in-hospital and follow-up events. Clinical endpoints, including cardiogenic shock, cardiac death, reinfarction, TVR, MACE, stroke, cardiopulmonary resuscitation, ventricular arrhythmias, heart failure, inotropic usage, and atrial fibrillation (AF), were similar among groups, except for acute stent thrombosis (p=0.046). In-hospital analysis revealed a nominally significant difference in acute stent thrombosis across serum Mg groups (p=0.046); however, this association lost significance after correction for multiple comparisons. ROC curve analysis for predicting acute stent thrombosis is shown in Figure 1. The optimal admission serum Mg cut-off value was 1.83 mg/dL, with 76% sensitivity and 65% specificity [AUC: 0.781; 95% confidence interval (CI): 0.543–0.920; p=0.024]. Long-term follow-up clinical endpoints are presented in Table 3. Groups were similar in terms of cardiac death, late stent thrombosis,

Table 1. Baseline demographic and clinical characteristics

Variables	<1.8 (n=268)	1.8–2.0 (n=381)	2.0–2.2 (n=319)	>2.2 (n=151)	p value
Gender (female/male)	44 (16.4%)/224 (83.6%)	29 (7.6%)/352 (92.4%)	14 (4.4%)/305 (95.6%)	17 (11.3%)/134 (88.7%)	<0.001
Age (years)	53±9	53±9	53±9	55±10	0.387
Magnesium (mg/dL)	1.7±0.1	2±0.1	2.1±0.1	2.5±0.3	<0.001
Potassium (mmol/L)	4.1±0.52	4.06±0.48	4.06±0.47	4.16±0.65	0.223
Glucose (mg/dL)	168±80	157±77	144±61	155±74	0.001
Creatinine (mg/dL)	1±0.3	1.1±0.5	1.1±0.2	1.2±0.8	<0.001
Cholesterol (mg/dL)	187±40	193±44	191±40	193±44	0.381
Triglyceride (mg/dL)	142±101	156±145	162±101	154±84	0.278
LDL (mg/dL)	117±36	119±34	120±35	118±39	0.874
HDL (mg/dL)	41±8	42±10	40±9	41±9	0.181
CK-MB (µg/L)	224±166	241±174	218±179	220±176	0.334
Hematocrit (%)	40.9±3.9	41.6±4.2	41.7±3.6	42.1±4.8	0.055
Leukocyte (10^3/µL)	12.9±3.7	13.2±3.9	12.7±3.8	12.9±3.2	0.402
Platelet (10^3/µL)	262±65.1	256±58	263±72	271±76.2	0.142
Stent length (mm)	19±7	19±6	19±7	20±7	0.891
Stent diameter (mm)	3.1±0.4	3.1±0.3	3.1±0.3	3.1±0.4	0.691
Ejection fraction (%)	48±9	47±11	49±10	48±13	0.424
Glomerular filtration rate	93.9±22.8	90.9±22	91.4±21.9	88.9±24.3	0.066
Follow-up period (months)	25±15	24±16	22±16	21±14	0.081
Diabetes mellitus	90 (34.0%)/175 (66.0%)	75 (19.8%)/303 (80.2%)	61 (19.2%)/256 (80.8%)	31 (20.8%)/118 (79.2%)	<0.001
Hypertension	95 (38.3%)/153 (61.7%)	137 (38.7%)/217 (61.3%)	117 (38.5%)/187 (61.5%)	60 (42.0%)/83 (58.0%)	0.891
Family CAD history	54 (22.4%)/187 (77.6%)	61 (17.8%)/282 (82.2%)	58 (20.0%)/232 (80.0%)	29 (21.0%)/109 (79.0%)	0.569
Hyperlipidemia	113 (43.6%)/146 (56.4%)	133 (37.7%)/220 (62.3%)	123 (40.7%)/179 (59.3%)	55 (40.4%)/81 (59.6%)	0.528
Smoking	157 (65.4%)/83 (34.6%)	234 (68.0%)/110 (32.0%)	195 (66.1%)/100 (33.9%)	80 (60.6%)/52 (39.4%)	0.511

All data were expressed as mean±standard deviation
LDL: Low-density lipoproteins, HDL: High-density lipoproteins, CK-MB: Creatine kinase-MB, CAD: Coronary artery disease

Table 2. Angiographic/procedural and in-hospital cardiac findings

Findings	<1.8 (n=268)	1.8–2.0 (n=381)	2.0–2.2 (n=319)	>2.2 (n=151)	p value
In hospital					
Shock	7 (2.6%)	6 (1.6%)	11 (3.4%)	2 (1.3%)	0.324
Pre-TIMI flow 1–3	228 (85.4%)/23 (8.6%)/16 (6.0%)	339 (89.0%)/27 (7.1%)/15 (3.9%)	267 (84.2%)/35 (11.0%)/15 (4.7%)	128 (85.3%)/16 (10.7%)/6 (4.0%)	0.469
Post-TIMI flow 1–3	29 (11.2%)/13 (5.0%)/217 (83.8%)	37 (9.9%)/24 (6.5%)/311 (83.6%)	23 (7.3%)/16 (5.1%)/275 (87.6%)	11 (7.4%)/12 (8.1%)/126 (84.6%)	0.481
Tirofiban usage	113 (42.2%)	189 (49.6%)	153 (48.0%)	82 (54.3%)	0.121
Sudden death	8 (3.0%)	8 (2.1%)	3 (0.9%)	4 (2.6%)	0.336
Reinfarction	8 (3.0%)	5 (1.3%)	6 (1.9%)	2 (1.3%)	0.441
TVR	13 (4.9%)	13 (3.4%)	11 (3.4%)	8 (5.3%)	0.625
MACE	20 (7.5%)	20 (5.2%)	14 (4.4%)	11 (7.3%)	0.343
Stroke	1 (0.4%)	5 (1.3%)	1 (0.3%)	1 (0.7%)	0.378
CPR	9 (3.4%)	9 (2.4%)	8 (2.5%)	4 (2.6%)	0.883
VT/VF	16 (6.0%)	15 (3.9%)	11 (3.4%)	5 (3.3%)	0.408
Congestive heart failure	43 (16.0%)	46 (12.1%)	36 (11.3%)	16 (10.6%)	0.258
Inotrope agent	23 (8.6%)	24 (6.3%)	26 (8.2%)	8 (5.3%)	0.482

Table 2. Continued

Findings	<1.8 (n=268)	1.8–2.0 (n=381)	2.0–2.2 (n=319)	>2.2 (n=151)	p value
Atrial fibrillation	5 (1.9%)	6 (1.6%)	6 (1.9%)	4 (2.6%)	0.878
A-V blockage	6 (2.2%)	11 (2.9%)	7 (2.2%)	4 (2.6%)	0.931
Late pacemaker	6 (2.2%)	9 (2.4%)	11 (3.4%)	4 (2.6%)	0.784
Acute thrombosis (0–1 days)	5 (1.9%)	1 (0.3%)	2 (0.6%)	0 (0.0%)	0.046
Transfusion	5 (1.9%)	13 (3.4%)	8 (2.5%)	1 (0.7%)	0.269
In follow-up period					
Cardiac death	11 (4.1%)	18 (4.7%)	8 (2.5%)	2 (1.3%)	0.153
Late thrombosis (>30 days)	4 (1.5%)	6 (1.6%)	6 (1.9%)	2 (1.3%)	0.969
Re-infarction	21 (7.8%)	31 (8.1%)	21 (6.6%)	15 (9.9%)	0.578
TVR	51 (19.0%)	78 (20.5%)	49 (15.4%)	25 (16.6%)	0.165
MACE	63 (23.5%)	95 (24.9%)	58 (18.2%)	31 (20.5%)	0.056

All categorical data except pre (0.1.2) and post-TIMI (0.1.2) were expressed as X/Y that X means not exist while Y means exist
TVR: Target vessel revascularization, MACE: Major adverse cardiac events, VT-VF: Ventricular fibrillation ventricular tachycardia, CPR: Cardiopulmonary resuscitation
TIMI: Thrombolysis in myocardial infarction

Table 3. Long-term clinical outcomes

Variables	Unstandardized coefficients		Standardized coefficients	p value
	B	Std. error	Beta	
(Constant)	2.210	0.041	-	<0.001
Follow-up (months)	-0.003	0.001	-0.164	<0.001
GFR	-0.001	0.0001	-0.121	<0.001
DM	-0.066	0.023	-0.105	<0.001
Cardiac death	-0.143	0.056	-0.095	0.011
Acute (0-1 days) thrombosis	-0.235	0.109	-0.078	0.032

The regression model has 11.2 R square value with a significance at p<0.0001
DM: Diabetes mellitus, GFR: Glomerular filtration rate, Std: Standard

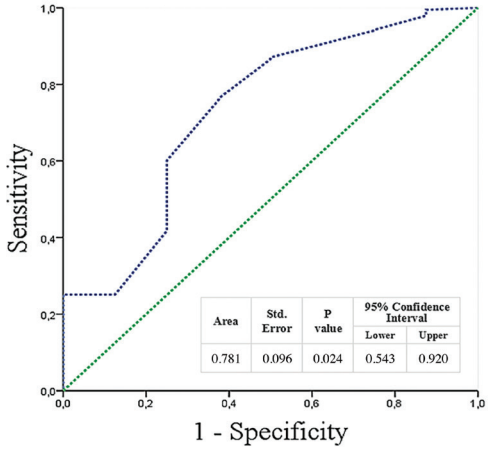


Figure 1. ROC curve of Mg^{+2} levels for acute stent thrombosis
ROC: Receiver operating characteristic, Std: Standard, Mg: Magnesium

reinfarction, TVR, and MACE. Stepwise linear regression analysis of admission serum Mg levels showed a significant relationship with acute stent thrombosis (p=0.032), as detailed in Table 3.

DISCUSSION

The present study demonstrated that low admission serum Mg levels were significantly associated with acute stent thrombosis. However, no significant association was found between admission serum Mg levels and in-hospital or long-term major adverse cardiovascular events, cardiovascular mortality, TVR, or stroke. It has been shown that during the acute phase of MI, transient hypomagnesemia occurs due to a shift from the extracellular to the intracellular space.[9] Therefore, serum Mg levels at the acute phase may not accurately reflect intracellular Mg concentrations. Mg functions correlate better with intracellular levels than with serum levels.[18] The amount of Mg within erythrocytes and lymphocytes may provide a more accurate reflection of intramyocardial Mg.[19] Mg, as an intracellular cation, plays various important roles in atherogenesis and cardiovascular diseases.[7] Under normal circumstances, Mg prolongs the refractory period of the atria and ventricles and stabilizes proarrhythmic substrates.[20] Mg also acts as a physiological calcium antagonist by binding to calcium sites and modifying membrane potential. Guo et al.[21] demonstrated that low Mg

levels are associated with variant angina. As a result, Mg could limit coronary vasospasm and oxidative damage during MI and ischemia, which in turn may reduce infarct size.[22] Following cardiac surgery, $MgSO_4$ administration reduces the incidence of AF.[23,24] The Framingham Heart Study revealed that low Mg levels are associated with an increased risk of AF development in people without cardiovascular disease.[25] In the present study, we did not find any relationship between low serum Mg levels and the development of AF or ventricular arrhythmias. This could be due to a transient drop in serum Mg rather than an absolute decrease. The patient population in this study was different from those in the aforementioned studies.[23-25] We suggest that serum Mg levels should be evaluated differently in acute MI patients compared with the general population and patients undergoing CABG.

Mg improves glucose and insulin metabolism and reduces the risk of developing T2DM and metabolic syndrome.[26,27] Hypomagnesemia, through lipoprotein peroxidation, causes dyslipidemia, which decreases high-density lipoprotein levels and plasma apolipoprotein B, while increasing triglyceride-rich lipoproteins.[28] As a result of impaired glucose and lipid metabolism, hypomagnesemia plays an important role in cardiovascular health.[28] In the present study, we did not find any relationship between serum lipid levels and admission serum Mg levels. This may be due to a transient decrease in serum Mg rather than an absolute deficiency. On the other hand, the presence of DM was less frequent in patients with low serum Mg levels, consistent with previous reports. We suggest that because the low serum Mg levels were transient, no correlation was observed between DM and serum lipid levels. This finding may have occurred by chance.

Mg reduces platelet activation by inhibiting thromboxane A2 and increasing prostacyclin.[29,30] Thus, Mg plays an important role in platelet aggregation and adhesion. Additionally, Mg, acting as a calcium antagonist, exerts a platelet-inhibitory effect.[31] In a large-scale retrospective study, Çiçek et al.[10] showed that a serum Mg level <1.91 mg/dL significantly predicts acute stent thrombosis in STEMI patients treated with primary PCI. In a more recent, relatively small-scale study, patients with STEMI who underwent primary PCI were evaluated by Yuksel et al.,[11] who showed that a serum Mg level ≤1.87 mg/dL predicts electrocardiographic no-reflow and long-term mortality independently. In accordance with these studies, An et al.,[12] in a prospective study, revealed that a low level of serum Mg could be a predictor of MACEs in patients with acute MI treated with drug-eluting stents. In the present study, we detected that a serum Mg level <1.83 mg/dL was significantly associated with acute stent thrombosis. These results are consistent with the findings of Çiçek et al.[10] as both studies were performed in similar patient groups—namely, STEMI patients undergoing primary PCI. Because serum Mg levels may transiently decrease during the acute phase of STEMI, this reduction may alter platelet activity and increase platelet aggregation, thereby contributing to stent thrombosis. We also did not find any correlation between chronic stent thrombosis and admission serum Mg level. These findings suggest that, in the acute phase of STEMI, low serum Mg levels may be associated with increased platelet activity and stent thrombosis. However, although acute thrombosis appeared to differ between groups in the unadjusted analysis, this finding should be interpreted with caution because multiple outcome comparisons were performed. After adjustment for multiple testing, the association was attenuated, suggesting that the result may represent a chance finding rather than a robust signal. Coronary no-reflow is a complex condition with several proposed causes. One of the most important proposed causes of no-reflow is increased platelet reactivity. The results of Yuksel et al.[11] support this theory. The results of the aforementioned study showed that even though angiographic no-reflow (post-TIMI <3) was similar between groups, a low serum Mg level was associated with electrocardiographic no-reflow. In the present study, similarly, we did not find any significant correlation between serum Mg level and post-TIMI flow. We did not evaluate electrocardiographic no-reflow.

On the other hand, studies have demonstrated contrary results. In a moderate-scale study, Vassalle et al.[13] did not find any significant relationship between low serum Mg level and adverse cardiac events (non-fatal MI and all-cause mortality) in acute MI patients. In concordance with this study, a large retrospective study conducted at Mayo Clinic Hospital in patients admitted to the intensive cardiac care unit—mostly composed of acute MI patients—did not reveal any association between serum Mg levels and sudden cardiac death or corrected QT interval.[14] They also found that both admission and post-admission serum Mg level ≥2.4 mg/dL were associated with increased hospital mortality.[14] In a similar context, the Mg on Coronaries Trial Investigators did not find any beneficial effect of early Mg administration on 30-day mortality in high-risk STEMI patients.[32] In our study, No significant association was observed between admission serum Mg level and in-hospital or long-term mortality, reinfarction, TVR, or MACEs. These results are consistent with the aforementioned studies.[13,14,32] These endpoints could be affected by absolute Mg levels rather than a transient decrease in serum Mg.

These findings suggest that the serum Mg level during the acute phase of MI and the Mg level in the general population without cardiovascular disease should be evaluated separately. In this perspective, serum Mg levels in the acute phase of MI are probably transient and may be associated with a temporary increase in platelet reactivity and acute stent thrombosis. Therefore, after the acute phase of MI, serum Mg levels should be re-checked and re-evaluated, with special attention paid to the acute phase of STEMI.

Study Limitations

The present study had several limitations. First, this was a retrospective study and therefore had the inherent limitations of such a design. The causative relationship of all confounders cannot be confirmed. Second, we did not measure serial serum Mg levels; therefore, we could not determine subsequent Mg levels or their effects on outcomes. Third, the study was conducted at a single center, which limits the generalizability of the results. Stent thrombosis may result from multiple factors, and in the present study, we did not evaluate all potential confounders. Finally, electrocardiographic no-reflow was not assessed. Although ROC analysis was performed, the wide CI (AUC 0.781; 95% CI: 0.543–0.920) indicates limited precision of the findings. In addition to the retrospective design and the lack of serial Mg measurements, several other methodological limitations should be acknowledged. No a priori power analysis was performed, which may limit the study's ability to reliably detect clinically meaningful

differences. Furthermore, the statistical modeling strategy was not fully aligned with the nature of the outcomes, as binary endpoints were not evaluated using multivariable logistic regression analysis. This may have limited adequate adjustment for potential confounding factors. Finally, no correction for multiple comparisons was applied despite the evaluation of numerous endpoints, increasing the risk of type I error.

CONCLUSION

In conclusion, this large-scale retrospective study demonstrated that low admission serum Mg levels were significantly associated with stent thrombosis. However, no significant association was observed between serum Mg levels and in-hospital or long-term cardiovascular mortality, stroke, reinfarction, TVR, or major adverse cardiovascular events. When the findings of the present study are considered alongside those of previous reports, it is evident that randomized clinical trials are needed to draw more robust and definitive conclusions.

Ethics Committee Approval: The study was approved by Bezmialem Vakıf University Ethics Committee (approval no: 15, date: 08.10.2025).

Informed Consent: As this was a retrospective study, written informed consent was not obtained from patients.

Authorship Contributions: Concept: C.A., M.K., Design: C.A., M.K., Data Collection or Processing: B.H.Ş.U., Analysis or Interpretation: C.A., S.U., Literature Search: M.U., Writing: C.A., E.D.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study received no financial support.

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INTERVENTIONAL CARDIOLOGY PERSPECTIVES

Inter Cardio Pers 2026;2(1):16-22

DOI: 10.4274/intercardiopers.2026.2026-1-1

ORIGINAL ARTICLE

Clinical and Echocardiographic Factors Associated with 12-Month Mortality After Living-Donor Kidney Transplantation: A Single-Center Cohort

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ABSTRACT

Background: Cardiovascular abnormalities are highly prevalent among candidates for kidney transplantation and significantly affect post-transplant outcomes. Pre-transplant echocardiographic findings and circulating biochemical markers may assist in identifying recipients at increased risk prior to transplantation.

Aim: To determine whether pre-transplant echocardiographic parameters and biochemical markers are predictive of 1-year survival among recipients of living-donor kidney transplants.

Study Design: This study was designed as a single-center retrospective cohort analysis.

Methods: We analyzed data from 178 adult patients who underwent living-donor kidney transplantation between 2021 and 2025. Echocardiographic variables included left ventricular ejection fraction [(LVEF); categorized as reduced <55% vs. preserved ≥55%], hypertrophic cardiomyopathy (HCM), moderate-to-severe valvular regurgitation, and ascending aortic dilatation (defined as ≥40 mm). Biochemical variables comprised N-terminal pro–B-type natriuretic peptide [(NT-proBNP); ≥130 pg/mL) and low-density lipoprotein cholesterol categories. One-year survival was evaluated using Kaplan–Meier survival analysis with log-rank testing. Exploratory univariable Cox proportional hazards regression analysis was conducted to assess associations between clinical variables and mortality.

Results: During the 1-year follow-up period, 13 deaths occurred, corresponding to a mortality rate of 7.3%. In univariable Cox regression analysis, older age, longer duration of dialysis, HCM, and reduced LVEF (<55%) were significantly associated with 12-month mortality. Specifically, reduced LVEF was associated with a higher risk of death (hazard ratio: 4.18, 95% confidence interval: 1.29–13.58; p=0.017). NT-proBNP levels were not significantly associated with mortality.

Conclusion: Older age, prolonged dialysis duration, HCM, and reduced LVEF were associated with increased 12-month mortality in univariable analyses. NT-proBNP was not significantly associated with mortality. Given the limited number of events, these findings should be interpreted with caution and require validation in larger, adequately powered cohorts.

Keywords: Echocardiography, ejection fraction, hypertrophic cardiomyopathy, kidney transplantation, survival

INTRODUCTION

Cardiovascular disease remains the leading cause of both early and late mortality following kidney transplantation, highlighting the critical importance of comprehensive pre-transplant cardiovascular assessment in transplant candidates.[1,2] Although kidney transplantation is considered the gold standard treatment for end-stage renal disease, this high-risk population continues to experience substantial rates of perioperative cardiovascular complications, including myocardial infarction, stroke, and pulmonary embolism.[3] Echocardiographic abnormalities and elevated cardiac biomarkers are highly prevalent among transplant candidates and have consistently been associated with unfavorable postoperative outcomes.[3-5]

Previous studies have demonstrated that reduced left ventricular ejection fraction (LVEF) and elevated natriuretic peptide (NT) levels are strong predictors of mortality among patients receiving dialysis and in deceased-donor transplant cohorts.[6] However, evidence specifically focusing on living-donor kidney transplant recipients in middle-income countries remains scarce. This gap in knowledge is clinically relevant because patient characteristics, perioperative care, and long-term cardiovascular risk profiles may differ substantially from those reported in high-income settings.[7]

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Cite as: Öz MT, Öztürk NN, Barlas İS, Kaya A, Apaydın S. Clinical and echocardiographic factors associated with 12-month mortality after living-donor kidney transplantation: a single-center cohort. *Inter Cardio Pers.* 2026;2(1):16-22

Received: 02.01.2026
Accepted: 02.03.2026
Epub: 11.03.2026
Publication Date: 10.04.2026

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To our knowledge, this study represents the first single-center Turkish cohort to systematically evaluate both LVEF and N-terminal pro–B-type natriuretic peptide (NT-proBNP) levels as prognostic markers in living-donor kidney transplant recipients. By concentrating on this relatively homogeneous population, the present analysis offers region-specific data that complement the existing international literature, in which deceased-donor transplant series predominate.[8]

The present study aimed to determine whether pre-transplant echocardiographic and biochemical markers—including LVEF, hypertrophic cardiomyopathy (HCM), moderate-to-severe valvular regurgitation, ascending aortic dilatation, serum NT-proBNP, low-density lipoprotein (LDL) cholesterol, and dialysis duration (hemodialysis or peritoneal dialysis)—are predictive of 1-year survival in Turkish kidney transplant recipients.[9]

METHODS

Study Design and Population

We performed a single-center retrospective cohort study that included adult patients who underwent living-donor kidney transplantation between June 2021 and August 2025. All recipients were required to have a minimum of 12 months of post-transplant follow-up.

Among the 301 consecutive patients screened for eligibility, 22 were excluded due to incomplete baseline clinical or echocardiographic data, and 3 were excluded because of acute rejection occurring within the first 3 months after transplantation. An additional 98 patients were excluded because 12-month vital status could not be verified, owing to insufficient follow-up duration and/or incomplete medical records. Consequently, the final analytical cohort comprised 178 patients (Figure 1).

Due to the retrospective design using fully anonymized data, this study did not require ethics committee approval or informed consent in accordance with institutional and national research regulations.

Exposure Variables

Pre-transplant echocardiographic variables included LVEF, the presence of HCM (defined as maximum wall thickness ≥15 mm), moderate-to-severe valvular regurgitation, and ascending aortic dilatation (≥40 mm). LVEF was dichotomized as preserved (≥55%) or reduced (<55%) according to institutional reference values and guideline-based definitions of the lower limit of normal systolic function. HCM was defined in accordance with contemporary guideline criteria as a maximal left ventricular wall thickness ≥15 mm in one or more myocardial segments, measured at end-diastole using parasternal long- and short-axis views. These measurements were derived from routine pre-transplant transthoracic echocardiography reports and reflected the maximum recorded wall thickness.

The LVEF threshold of <55% was selected to represent the lower limit of normal systolic function as defined by echocardiographic guidelines and to identify even mild or subclinical systolic dysfunction in this high-risk transplant population. In patients with end-stage renal disease, subtle reductions in LVEF within the traditionally "normal" range may have prognostic significance because of underlying uremic cardiomyopathy and chronic volume overload.

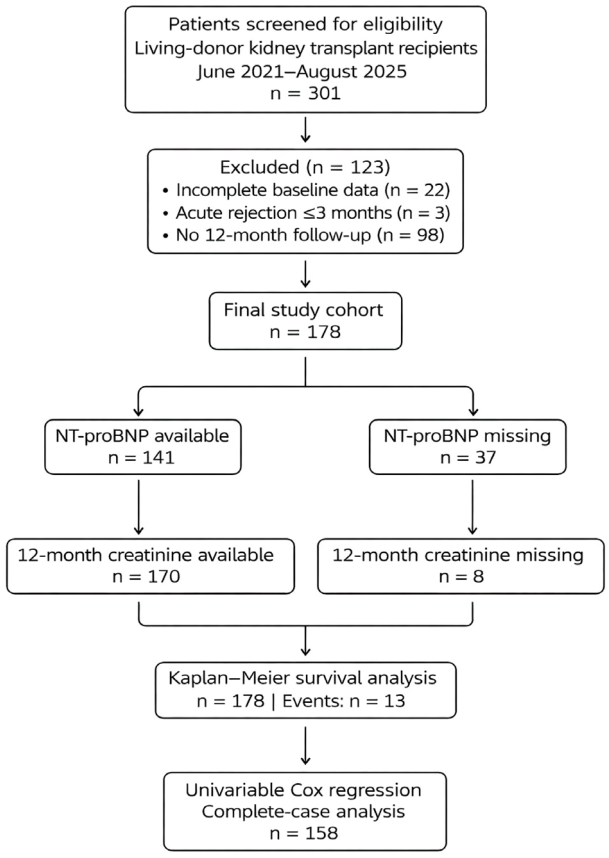


Figure 1. Flow diagram of patient selection and study cohort. A total of 301 consecutive living-donor kidney transplant recipients were screened for eligibility between June 2021 and August 2025. Patients were excluded due to incomplete baseline clinical or echocardiographic data (n=22), acute rejection within the first 3 months after transplantation (n=3), or unavailable 12-month vital status (n=98). The final analytical cohort consisted of 178 recipients, all of whom had complete follow-up for the primary endpoint of 12-month all-cause mortality. NT-proBNP measurements were available in 141 patients, and ascending aortic diameter measurements in 177 patients. All 178 recipients were included in the Kaplan–Meier survival analyses, during which 13 deaths occurred within 12 months

NT-proBNP: N-terminal pro B-type natriuretic peptide

To reduce the risk of misclassification, secondary causes of left ventricular hypertrophy were systematically reviewed using available clinical and echocardiographic data. Patients with concentric hypertrophy attributable to long-standing uncontrolled hypertension, advanced uremic cardiomyopathy, or significant valvular disease (including moderate-to-severe aortic stenosis) were not categorized as having HCM. The presence of systolic anterior motion of the mitral valve and left ventricular outflow tract (LVOT) gradients was documented when observed, and provocative maneuvers were performed when clinically indicated.

Biochemical variables included pre-transplant NT-proBNP levels, dichotomized at ≥130 pg/mL, and LDL cholesterol categorized as <130, 130–189, and ≥190 mg/dL. The ≥130 pg/mL threshold was selected based on prior chronic kidney disease and transplant literature as well as local laboratory reporting standards. Dialysis exposure was

quantified as the total duration of renal replacement therapy before transplantation.

Dialysis duration was defined as the cumulative time on renal replacement therapy prior to transplantation, calculated from the date of dialysis initiation (hemodialysis or peritoneal dialysis) to the date of transplantation and expressed in months. Pre-emptive transplant recipients were assigned a dialysis duration of 0 months.

Pre-transplant transthoracic echocardiography was performed as part of the routine evaluation within 3 months prior to transplantation. LVEF was measured using the biplane Simpson method in accordance with current guideline recommendations. All measurements were obtained by experienced cardiologists and extracted from standardized echocardiographic reports. Systolic anterior motion and LVOT gradients were assessed at rest, with provocation performed when clinically indicated.

Outcome

The primary endpoint of the study was all-cause mortality within 12 months following kidney transplantation.

Statistical Analysis

Survival probabilities were estimated using the Kaplan–Meier method, and between-group comparisons were conducted using the log-rank test. Follow-up time was administratively censored at 12 months for all patients.

Associations between prespecified candidate variables and 12-month mortality were examined using separate univariable Cox proportional hazards (PHs) regression models. Owing to the limited number of events (n=13), multivariable regression analyses and formal interaction testing were not performed to minimize the risk of model overfitting.

The PH assumption was not formally assessed because of the small number of events; therefore, the Cox regression results should be considered exploratory.

Missing data were addressed using a complete-case analysis approach. Continuous variables are reported as mean±standard deviation, whereas categorical variables are presented as counts and percentages. All statistical analyses were conducted using IBM SPSS Statistics version 26 (IBM Corp., Armonk, NY, USA). A two-sided p value <0.05 was considered statistically significant. For predictors with sparse event counts in one category, effect estimates may be unstable and associated with wide confidence intervals (CIs); therefore, these analyses should be interpreted cautiously given the limited number of deaths.

This study was conducted and reported in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology guidelines for observational cohort studies.

RESULTS

Study Population and Follow-up

A total of 178 living-donor kidney transplant recipients were included in the final analysis. The mean age of the cohort was 45.4±13.8 years, and 65.7% were male. Although patients were followed longitudinally for up to 4 years post-transplantation, this analysis focused on outcomes within the first 12 months, which constituted the predefined primary endpoint.

During the first year post-transplantation, 13 deaths occurred, corresponding to a 12-month mortality rate of 7.3%. Vital status at 12 months was available for all 178 recipients, ensuring no loss to follow-up for the primary endpoint. NT-proBNP measurements were available for 141 of 178 recipients, and ascending aortic diameter was recorded in 177 of 178. Each univariable Cox model was analyzed using complete-case data; consequently, the effective sample size varied by predictor.

Baseline demographic, clinical, biochemical, and echocardiographic characteristics are summarized in Table 1.

Kaplan–Meier Survival Analysis

Kaplan–Meier survival curves were generated using time-to-event data, with administrative censoring at 12 months, to evaluate early post-transplant outcomes. The estimated overall 12-month survival rate was 92.7% (95% CI, 87.8–95.8). Survival probabilities over time were compared between groups using the log-rank test. The primary summary measure was the 12-month survival probability.

Recipients with reduced LVEF (<55%) had significantly lower 12-month survival compared with those with preserved systolic function (log-rank χ^2=5.589, p=0.018) (Figure 2). In univariable Cox regression, reduced LVEF was also significantly associated with higher 12-month mortality [hazard ratio (HR): 4.18, 95% CI, 1.29–13.58, p=0.017].

Recipients with elevated NT-proBNP levels (≥130 pg/mL) showed a trend toward lower 12-month survival; however, this difference did not reach statistical significance (log-rank p=0.066). In univariable Cox regression, elevated NT-proBNP was not

Table 1. Baseline demographic, clinical, and echocardiographic characteristics

Variable	Value
N (total)	178
Age, years (mean±SD)	45.39±13.80 (n=178)
Male, n (%)	117 (65.7%)
Diabetes mellitus, n (%)	30 (16.9%)
Hypertension (controlled), n (%)	134 (75.3%)
Hypertension (uncontrolled), n (%)	29 (16.3%)
Hemodialysis history, n (%)	88 (49.4%)
NODAT, n (%)	9 (5.1%)
Reduced EF (<55%), n (%)	18 (10.1%)
Ascending aorta ≥40 mm, n (%)	25 (14.1%) (n=177)
Hypertrophic cardiomyopathy, n (%)	19 (10.7%)
Moderate valvular regurgitation, n (%)	34 (19.1%)
NT-proBNP ≥130 pg/mL, n (%)	66 (46.8%) (n=141)
LDL (coded 0/1/2), n	0: 119; 1: 44; 2: 15
All-cause mortality within 12 months, n (%)	13 (7.3%)

SD: Standard deviation, EF: Ejection fraction, NT-proBNP: N-terminal pro B-type natriuretic peptide, LDL: Low-density lipoprotein, NODAT: New-onset diabetes after transplantation

significantly associated with 12-month mortality (HR: 2.16, 95% CI, 0.72–6.45, p=0.167).

No significant differences in 12-month survival were observed according to HCM, moderate-to-severe valvular regurgitation, ascending aortic dilatation (≥40 mm), or LDL cholesterol categories (all log-rank p>0.05). Kaplan–Meier–derived 12-month survival estimates for echocardiographic and biochemical subgroups are summarized in Table 2.

Exploratory univariable Cox Regression Analysis

Exploratory univariable Cox PH regression analyses were performed to evaluate associations between individual echocardiographic and biochemical parameters and 12-month mortality. Candidate variables included age, sex, diabetes mellitus, hypertension status, dialysis duration, LVEF category, NT-proBNP category, HCM, valvular regurgitation severity, ascending aortic dilatation, and LDL cholesterol category.

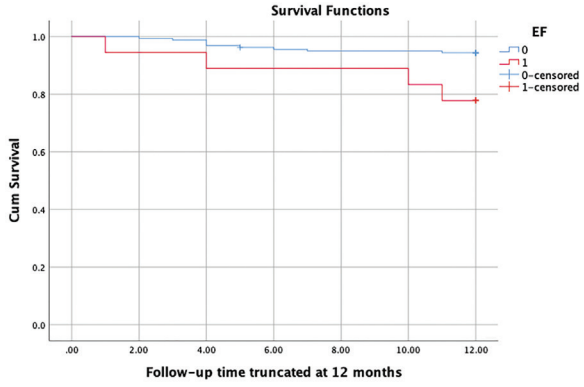


Figure 2. Kaplan–Meier survival curves stratified by ejection fraction (EF ≥55% vs. EF <55%). Patients with reduced EF showed significantly worse survival. Log-rank p=0.018; univariable Cox HR: 4.18 (95% CI: 1.29-13.58), p=0.017
EF: Ejection fraction, HR: Hazard ratio, CI: Confidence interval

In univariable Cox regression, older age (HR: 1.06 per year, 95% CI, 1.01–1.11, p=0.014), longer dialysis duration (HR: 1.16 per month, 95% CI, 1.04–1.29, p=0.006), HCM (HR: 3.43, 95% CI, 1.08–10.94, p=0.037), and reduced LVEF (<55%) (HR: 4.18, 95% CI, 1.29–13.58, p=0.017) were significantly associated with 12-month mortality. The NT-proBNP category was not significantly associated (HR: 2.16, 95% CI, 0.72–6.45, p=0.167) (Table 3).

Subgroup and Sensitivity Considerations

In exploratory subgroup analyses stratified by LVEF and NT-proBNP, recipients with both reduced LVEF and elevated NT-proBNP exhibited numerically higher 12-month mortality, whereas those with preserved LVEF and low NT-proBNP had the most favorable outcomes.

Although follow-up extended beyond 12 months for many patients, restricting analyses to the first post-transplant year enabled a focused assessment of early risk while minimizing heterogeneity from late non-cardiovascular events. Results were directionally consistent across both Kaplan–Meier and Cox regression analyses, supporting the robustness of these findings.

DISCUSSION

This single-center retrospective study evaluated echocardiographic and biochemical predictors of 1-year survival in living-donor kidney transplant recipients from Türkiye. Age, dialysis duration, HCM, and reduced LVEF were associated with 12-month mortality in univariable analyses.[10] NT-proBNP demonstrated a non-significant trend and should be interpreted as exploratory.

Importantly, the combined analysis of LVEF and NT-proBNP was exploratory and underpowered; no causal or synergistic conclusions should be drawn from these subgroup observations.[6,11,12] The absence of statistical significance in unadjusted analyses is likely due to limited statistical power, particularly given the directionally consistent trends observed across Kaplan–Meier and Cox regression analyses.

Table 2. Kaplan–Meier survival analysis of laboratory and echocardiographic parameters

Parameter	Group	12-month survival (%)	Log-rank χ^2	p value
Ejection fraction	≥55%	93.5	5.589	0.018
	<55%	77.8		
NT-proBNP	<130 pg/mL	94.3	3.389	0.066
	≥130 pg/mL	84.8		
HCM	Absent	92.9	1.893	0.169
	Present	83.3		
Ascending aorta	<40 mm	91.9	0.001	0.981
	≥40 mm	91.7		
Valve insufficiency	None/mild–moderate	92.1	0.059	0.808
	Severe	90.9		
LDL	0	91.4	1.257	0.533
	1	95.2		
	2	86.7		

NT-proBNP: N-terminal pro B-type natriuretic peptide, LDL: Low-density lipoprotein, HCM: Hypertrophic cardiomyopathy

Table 3. Exploratory univariable Cox proportional hazards analysis for 12-month mortality

Variable	n	HR	95% CI	p value	Model
Age (per year)	178	1.06	1.01-1.11	0.014	Univariable Cox
Male sex	178	0.88	0.50-1.58	0.577	Univariable Cox
Diabetes mellitus	178	0.82	0.19-3.68	0.801	Univariable Cox
Hypertension (controlled)	178	1.21	0.34-4.33	0.772	Univariable Cox
Dialysis duration (per month)	178	1.16	1.04-1.29	0.006	Univariable Cox
Reduced EF (<55%)	177	4.18	1.29-13.58	0.017	Univariable Cox
NT-proBNP ≥130 pg/mL	141	2.16	0.72-6.45	0.167	Univariable Cox
Hypertrophic cardiomyopathy	178	3.43	1.08-10.94	0.037	Univariable Cox
Severe valvular insufficiency	178	1.18	0.33-4.22	0.802	Univariable Cox
Ascending aorta ≥40 mm	177	1.01	0.23-4.52	0.988	Univariable Cox
LDL 130-189 vs. <130	178	0.61	0.13-2.79	0.524	Univariable Cox
LDL ≥190 vs. <130	178	0.32	0.05-2.28	0.256	Univariable Cox

EF: Ejection fraction, NT-proBNP: N-terminal pro B-type natriuretic peptide, LDL: Low-density lipoprotein, CI: Confidence interval, HR: Hazard ratio

Left Ventricular Ejection Fraction

Reduced LVEF (<55%) was associated with inferior post-transplant survival on unadjusted analysis, reaffirming its prognostic importance in kidney transplant candidates. Although impaired EF was present in a relatively small proportion of recipients (10.1%), these patients experienced substantially worse early outcomes. This finding aligns with previous literature showing that even mild systolic dysfunction increases cardiovascular vulnerability in transplant populations. These results emphasize the importance of careful pre-transplant cardiac assessment and optimization of heart failure management.

Consistent with Kaplan–Meier results, reduced LVEF remained significantly associated with 12-month mortality in univariable Cox regression (HR: 4.18, p=0.017), although CIs were wide due to the limited number of events.

NT-proBNP

Elevated NT-proBNP was not significantly associated with 12-month mortality in univariable Cox regression (p=0.167), although Kaplan–Meier analysis suggested a non-significant trend (log-rank p=0.066). Thus, NT-proBNP findings should be considered exploratory and hypothesis-generating (Figure 3).

Severe Valvular Insufficiency

Severe valvular insufficiency was not associated with 1-year mortality in this cohort. This finding contrasts with reports from chronic kidney disease and dialysis populations, in which advanced valvular disease has been linked to adverse outcomes. The absence of an observed association in our study likely reflects the small number of patients with severe valvular lesions as well as careful pre-transplant selection, whereby individuals with symptomatic or advanced disease are typically excluded from transplantation. Nevertheless, given the recognized progression of valvular abnormalities after transplantation, continued echocardiographic surveillance remains warranted (Figure 4).

Hypertrophic Cardiomyopathy

Although Kaplan–Meier analysis did not demonstrate a statistically significant difference according to HCM status (log-rank p=0.169), HCM was significantly associated with 12-month mortality in univariable Cox regression (HR 3.43, p=0.037). However, CIs were wide because of the limited number of deaths. Accordingly, this finding should be

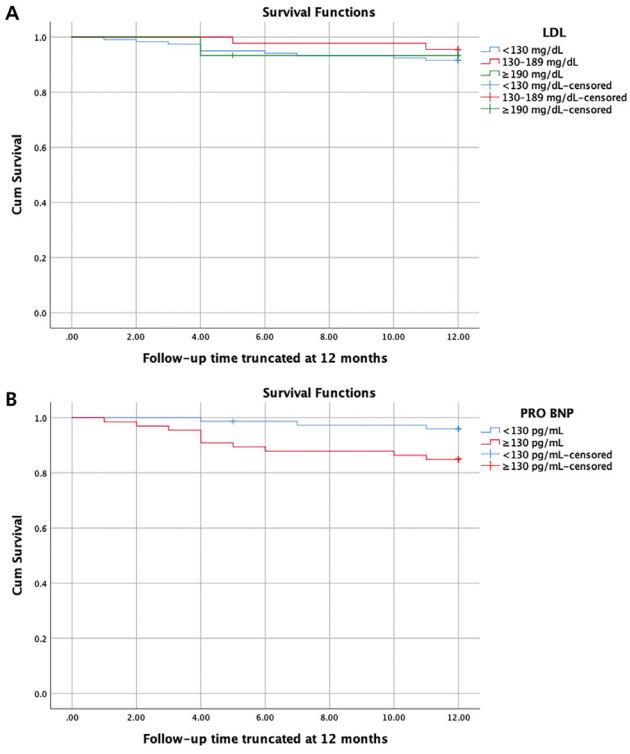


Figure 3. (A) Forest plot showing the association between elevated NT-proBNP (≥130 pg/mL) and 12-month mortality (HR: 2.16, p=0.167). (B) Forest plot of LDL cholesterol categories (<130 mg/dL, 130-189 mg/dL, ≥190 mg/dL) and survival outcomes. No significant associations were observed. p=0.953

NT-proBNP: N-terminal pro B-type natriuretic peptide, LDL, Low-density lipoprotein, HR: Hazard ratio, BNP: B-type natriuretic peptide

interpreted with caution and requires confirmation in larger cohorts (Figure 5).

Ascending Aortic Dilatation

Ascending aortic dilatation (≥40 mm) was not associated with 1-year mortality in this cohort. Although aortic dilatation has been linked to long-term cardiovascular risk in the general population, its prognostic impact may be attenuated in living-donor kidney transplant recipients, who are generally younger and undergo routine imaging surveillance. These findings suggest that baseline aortic diameter alone has limited predictive value for early post-transplant outcomes, underscoring the importance of longitudinal follow-up rather than reliance on isolated pre-transplant measurements (Figure 6).

Clinical Context and Implications

Compared with deceased-donor cohorts from the United States and Europe, living-donor recipients in Türkiye are typically younger, have fewer comorbidities, and benefit from closer perioperative monitoring. These characteristics may partly explain why LDL cholesterol, valvular abnormalities, and aortic dilatation were not associated with early mortality in this cohort, whereas such factors have been linked to long-term outcomes in more heterogeneous and comorbid populations.[13]

From a clinical standpoint, assessment of LVEF and measurement of NT-proBNP may assist in unadjusted risk stratification and in identifying patients who may benefit from closer perioperative cardiovascular monitoring; however, these findings should be considered exploratory. In summary, age, dialysis duration, HCM, and reduced LVEF were associated with 12-month mortality in univariable analyses.

NT-proBNP demonstrated a non-significant trend and should be interpreted as hypothesis-generating.[14]

Study Limitations

A major strength of this study is the inclusion of a homogeneous cohort of living-donor kidney transplant recipients managed under standardized follow-up protocols, thereby minimizing variability in clinical care. The availability of comprehensive echocardiographic parameters and biomarker measurements further enhances the analytical robustness.

Several limitations merit consideration. First, the single-center retrospective design restricts the generalizability of the findings. Second, the limited number of deaths reduced statistical power, potentially obscuring associations with less prevalent variables, such as severe valvular disease or pulmonary hypertension. Third, unmeasured confounders—including variations in immunosuppressive regimens, rejection episodes, and metabolic factors—may have influenced outcomes. Finally, although LDL cholesterol and valvular disease were not significantly associated with mortality in this cohort, the small subgroup sizes necessitate cautious interpretation.

Because this study involved a fixed single-center cohort with only 13 events, formal power calculation was not feasible. Univariable Cox regression was therefore restricted to clinically relevant variables to minimize overfitting, and effect estimates were interpreted with careful consideration of CIs. Larger multicenter cohorts are needed to confirm these associations with greater statistical precision.

Future Directions

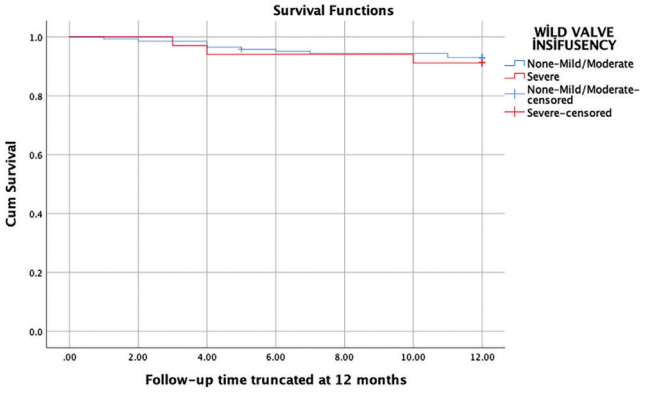


Figure 4. Event-free survival curves according to presence of severe valvular insufficiency. No significant survival difference was detected between groups. Log-rank p=0.808

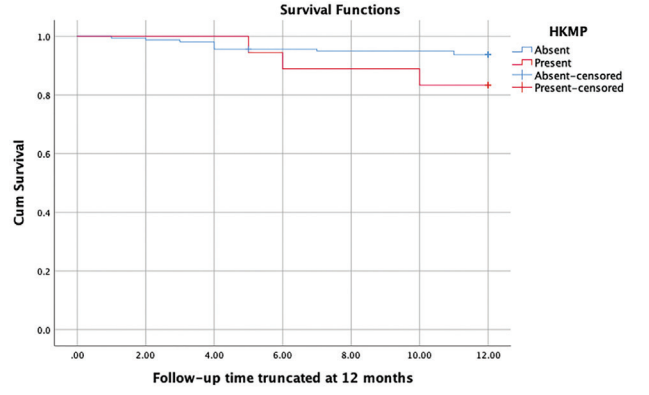


Figure 5. Kaplan–Meier survival curves stratified by hypertrophic cardiomyopathy (HCM). Patients with HCM showed numerically lower 12-month survival, not statistically significant, though the difference was not statistically significant. Log-rank p=0.169

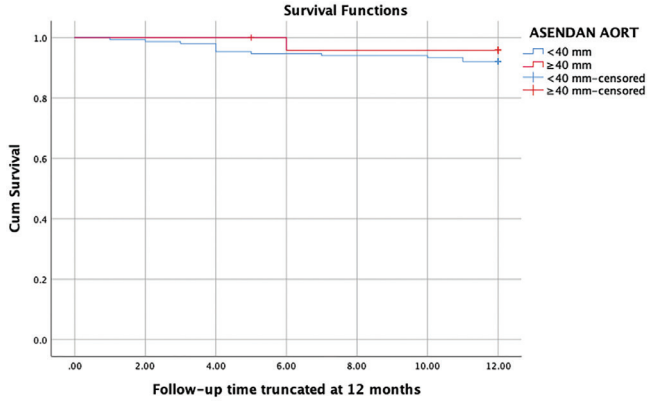


Figure 6. Kaplan–Meier survival curves stratified by ascending aortic dilatation (≥40 mm vs. <40 mm). No survival difference was observed between groups. Log-rank p=0.981

Prospective, multicenter studies with larger sample sizes are required to validate these findings and to further elucidate the interaction between established cardiovascular risk factors and emerging echocardiographic markers, including diastolic dysfunction and right ventricular strain as well as biomarkers such as NT-proBNP and cardiac troponins. Particular attention should be directed toward the prognostic significance of pulmonary hypertension and severe valvular insufficiency, which remain understudied yet clinically important in the transplant population.

CONCLUSION

In this single-center cohort of living-donor kidney transplant recipients, older age, prolonged dialysis duration, HCM, and reduced LVEF (<55%) were associated with 12-month mortality in univariable Cox regression analysis. NT-proBNP was not significantly associated with mortality. Given the limited number of events, these findings should be regarded as exploratory and require confirmation in larger, multicenter studies.

Ethics Committee Approval: This study did not require ethics committee approval due to its retrospective design and the use of fully anonymized data, in accordance with institutional and national research regulations.

Informed Consent: Informed consent was not required because the study was based on fully anonymized retrospective data.

Authorship Contributions: Surgical and Medical Practices: N.N.Ö., İ.S.B., Concept: M.T.Ö., Design: A.K., Data Collection or Processing: A.K., Analysis or Interpretation: S.A., Literature Search: M.T.Ö., S.A., Writing: M.T.Ö.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study received no financial support.

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INTERVENTIONAL CARDIOLOGY PERSPECTIVES

Inter Cardio Pers 2026;2(1):23-25

DOI: 10.4274/intercardiopers.2025.2025-3-5

CASE REPORT

Closing Road and Opening Trap: ProGlide Paradox in TAVI: A Case Report and Management Strategies Review

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ABSTRACT

Transcatheter aortic valve implantation offers a minimally invasive solution for patients with severe aortic stenosis who are at elevated surgical risk. Despite advances in access techniques and closure devices, vascular complications remain a significant procedural hazard. Suture-mediated systems, such as ProGlide, are widely adopted for achieving hemostasis but can cause rare, serious complications, particularly in patients with calcified or complex iliofemoral vascular anatomy. We report a unique case in which a ProGlide suture inadvertently entrapped a 7F sheath, requiring urgent surgical intervention. This underscores the need for meticulous preprocedural imaging, operator vigilance, and adaptable access planning to minimize such complications.

Keywords: Transcatheter aortic valve implantation, transfemoral access, peripheric complications

INTRODUCTION

The prevalence of degenerative aortic stenosis has risen in parallel with increased life expectancy. Although surgical aortic valve replacement remains the definitive treatment, transcatheter aortic valve implantation (TAVI) has emerged as a less invasive alternative, particularly for high-risk patients. Despite ongoing advancements in device technology and procedural techniques, vascular complications—reported in 4.5-15% of cases—continue to challenge interventional cardiologists. These complications are frequently linked to large-bore arterial access and the deployment of closure devices. Vascular complication rates range from 2% to 9% with closure devices commonly used in TAVI procedures, including ProGlide, MANTA, and ProStar.[1,2] We describe a rare complication involving the inadvertent entrapment of a 7F sheath by a ProGlide closure device, necessitating prompt surgical intervention. The report emphasizes the significance of preprocedural assessment, alternative access strategies, and intraprocedural tactics to mitigate vascular complications.

CASE REPORT

A 77-year-old male with a history of coronary artery bypass grafting, atrial fibrillation, diabetes mellitus, and treated diffuse large B-cell lymphoma presented with NYHA class 3 dyspnea. Transthoracic echocardiography demonstrated severe aortic stenosis (mean gradient: 40 mmHg, peak velocity: 4.3 m/s, valve area: 0.7 cm²) with preserved ejection fraction (59%) and moderate aortic regurgitation. A calcified trileaflet aortic valve with a left coronary ostial height of 7 mm was detected on computed tomography (CT) scan. Although the left common iliac artery appeared suitable for access apart from a focal calcific plaque at the target site, the right common iliac artery exhibited significant calcification and stenosis.

Given these findings, vascular access was established via the left groin. After administering 5,000 IU of heparin, a 7-F sheath was positioned in the right femoral artery for angiographic assessment of the left common iliac artery, and a temporary pacemaker lead was inserted via the right femoral vein. However, fluoroscopy identified critical stenosis at the left common iliac junction, which had not been detected on CT, prompting a switch of access to the right femoral artery (Figure 1A). A puncture was subsequently performed at a more favorable site on the same vessel, and two ProGlide devices were pre-deployed using the "pre-close" technique before inserting a 14F sheath for valve delivery.

Due to the proximity of the aortic valve to the left coronary ostium, a protective strategy (Chimney technique) was employed, involving placement of a balloon in the left coronary system via the right radial artery. The 27-mm self-expandable Navitor valve was successfully implanted, and post-dilation with a 25-mm balloon resulted in only trace paravalvular leakage (Figure 1B). Hemostasis was verified following removal of the 14F sheath. However, during attempts to retrieve the 7F sheath, persistent resistance was encountered despite multiple traction maneuvers. Closer inspection revealed that one of

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Cite as: Bulburu İB, Vurucu U, Özkan U. Closing road and opening trap: ProGlide Paradox in TAVI: a case report and management strategies review. *Inter Cardio Pers.* 2026;2(1):23-25

Received: 29.03.2025
Accepted: 11.06.2025
Epub: 11.07.2025
Publication Date: 10.04.2026

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the ProGlide sutures inadvertently engaged the 7F sheath, effectively securing it to the vessel wall (Figure 1C, 1D). To avoid the risk of vascular injury, the patient was promptly transferred to the operating room, and the entrapped sheath was surgically removed. The patient experienced an uneventful recovery and was discharged without further complications. Written informed consent was obtained from the patient and his relatives for the publication of this case report.

DISCUSSION

Vascular complications are among the most prevalent adverse events during TAVI, with major events occurring in approximately 4.5% of cases and being closely linked to both operator experience and patient-specific vascular anatomy.[3] While minor complications, including hematomas or pseudoaneurysms, are frequent and usually manageable, the present case demonstrates an uncommon and potentially hazardous complication related to the use of suture-mediated closure devices.

Several Key Factors Contributed to This Complication

Potential challenges encountered during the TAVI procedure, along with corresponding recommendations, are summarized in Table 1.

Pre-procedural Imaging and Access Selection

Although preprocedural CT imaging is crucial for planning TAVI access, it may fail to detect focal calcific stenoses that become apparent under fluoroscopy due to the number of slices. Ancillary modalities, such as Doppler ultrasound guidance, can enhance the precision of femoral puncture by delineating vascular landmarks and identifying calcific deposits.

Device Selection and Technique

Although sheath sizes have decreased from 18F to 14F due to advancements in TAVI, the sheath-to-femoral artery ratio remains a critical determinant of vascular complications.[4] In this case, the retention of a 7F sheath as a precautionary measure for rapid intervention in the event of femoral rupture was deemed justified. However, the concomitant use of the ProGlide device led to unanticipated suture entrapment. Alternative strategies—including protamine administration before attempting sheath removal—could potentially mitigate vascular tension and facilitate secure extraction of adjunctive sheaths. Protamine may be administered when adequate hemostasis cannot be achieved using closure devices or manual compression.

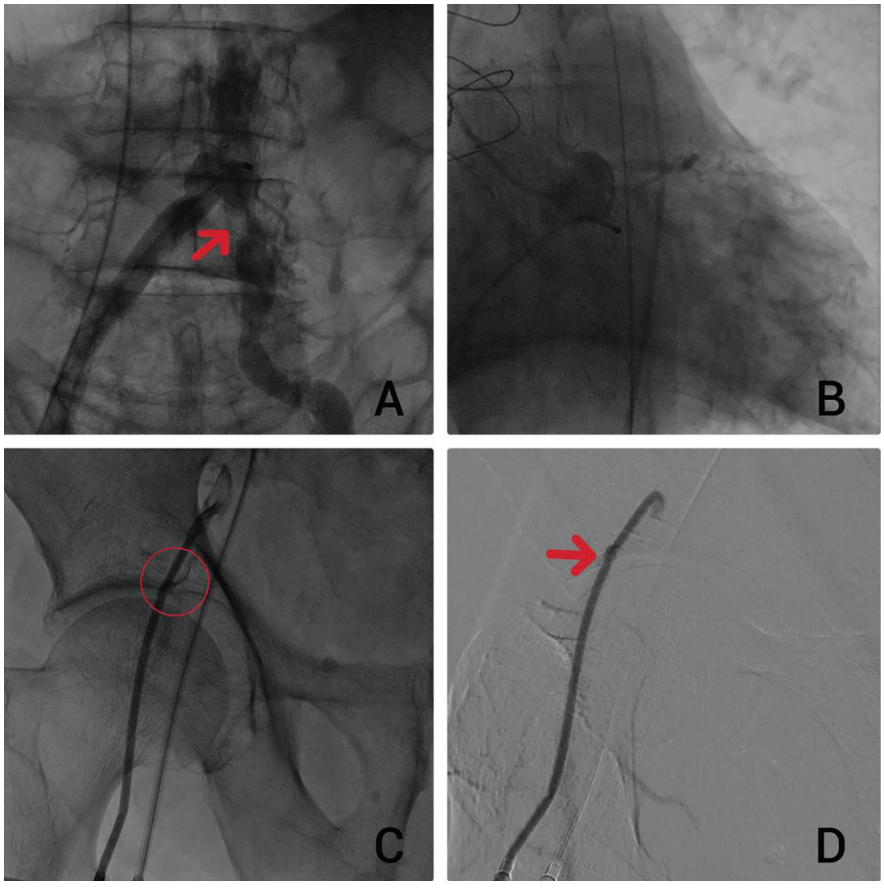


Figure 1. (A) A critical left common iliac junction stenosis indicated by a red arrow. B) Successfully implanted TAVI valve. C) Fluoroscopic image of the ProGlide mark on the 7F sheath and the opaque substance flowing through the holes made by the ProGlide on the sheath, in the red circle. D) DSA image showing the ProGlide suture mark on the 7F sheath, indicated with a red arrow

DSA: Digital subtraction angiography, TAVI: Transcatheter aortic valve implantation

Table 1. TAVI procedural pitfalls and recommendations

Procedural pitfall	Recommendation
Insufficient preprocedural vascular imaging	CT angiography should always be performed to evaluate access vessel size, tortuosity, and extent of calcification
Improper selection of vascular closure devices	Device selection hould be based on access vessel diameter, extent of calcification, and operator experience
Suboptimal puncture site	Use fluoroscopy and ultrasound guidance for precise common femoral artery access and evaluate contralateral femoral and iliac arteries via angiography for crossover feasibility.
Delayed hemostasis or bleeding	Ensure correct closure device deployment; closely monitor access site

CT: Computed tomography

Management of Closure-related Complications

In case of suspected suture entrapment, immediate reevaluation using imaging modalities such as fluoroscopy or ultrasound is recommended. Some authors have suggested that in the presence of calcified vessels, pre-closure may be optimized by balloon angioplasty or even intravascular lithotripsy to improve vessel diameter and reduce the risk of closure device misdeployment—particularly when aiming to preserve the access site, as in our case.[5] Furthermore, awareness of this potential complication should prompt operators to apply controlled traction and, if resistance is encountered, to consider pharmacological reversal of heparin with protamine before proceeding with further manipulation.

Literature Context

Similar complications have been documented in isolated case reports. For example, Hu et al.[5] (2015) reported a case of ProGlide-related vascular injury necessitating surgical repair, highlighting the significance of early recognition and intervention. Other studies have underscored the significance of advanced imaging and alternative access strategies to minimize vascular complications during TAVI.

Overall, this case illustrates the vital role of combining thorough preprocedural planning with intraprocedural vigilance to promptly identify and manage unexpected complications.

CONCLUSION

For patients with severe aortic stenosis, TAVI remains a less-invasive treatment option; however, the risk of vascular complications limits its effectiveness. This rare complication demonstrates the significance of procedural planning, operator training, and the use of alternative adjunctive strategies for calcified vessels. Pre-procedural imaging may be inadequate for accurately assessing vessel anatomy and calcifications, potentially leading to complications during guidewire insertion and sheath entry. In our patient, critical stenosis was not evident on preprocedural CT but was identified on fluoroscopy and necessitated a change of access site. In addition, the need for operators experienced in recognizing complications early and intervening is once again emphasized. The correct use of closure devices is critical, particularly in patients with complex vascular anatomy. Alternative approaches, including peripheral balloon angioplasty or intravascular lithotripsy, for calcified vessels can facilitate correct placement of closure devices and minimize the risk of complications by appropriately dilating the vessels. Incorporating such strategies into routine practice may aid in reducing the incidence of vascular complications, especially in TAVI procedures involving complex anatomy.

Informed Consent: Consent form was filled out by all participants.

Authorship Contributions: Surgical and Medical Practices: İ.B.B., U.V., U.Ö., Concept: İ.B.B., U.V., U.Ö., Design: İ.B.B., U.V., Data Collection or Processing: İ.B.B., Analysis or Interpretation: İ.B.B., U.Ö., Literature Search: İ.B.B., U.V., Writing: İ.B.B., U.V., U.Ö.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study received no financial support.

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INTERVENTIONAL CARDIOLOGY PERSPECTIVES

Inter Cardio Pers 2026;2(1):26-27

DOI: 10.4274/intercardiopers.2025.2025-6-1

CASE REPORT

Below-the-Knee Chronic Total Occlusion: The Power of a Multi-Technique Usage

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ABSTRACT

Chronic limb-threatening ischemia represents the end stage of peripheral arterial disease. In patients presenting with chronic total occlusions (CTOs) of below-the-knee (BTK) arteries, revascularization can be technically challenging. We report a case of a diabetic patient admitted to our diabetic foot care clinic with a non-healing ulcer and focal gangrene on the dorsal surface of the right foot. Revascularization was successfully achieved using a combination of antegrade hydrodynamic contrast recanalization (HDR) and a retrograde "just marker" technique. This case highlights that reliance on a single approach may be insufficient in complex BTK CTOs. The transition between techniques can be crucial for procedural success. In our case, we believe that the HDR technique at the proximal cap facilitated partial channel formation, allowing the retrograde wire to subsequently cross the lesion. Therefore, an initial technique's apparent failure may, in fact, contribute to overall success when used in combination. We propose that HDR is a valuable option in heavily calcified, long-segment CTOs. A collaborative, hybrid approach that incorporates multiple CTO techniques should be considered essential, as the limitations of one method may directly contribute to the success of another.

Keywords: Below the knee chronic total occlusion, diabetic foot, hydrodynamic contrast recanalization, peripheral artery disease, retrograde approach

INTRODUCTION

Chronic limb-threatening ischemia is the end stage of peripheral artery disease.[1] In diabetic patients with chronic total occlusions (CTOs) of below-the-knee (BTK) vessels, revascularization is often technically challenging and, in many cases, cannot be achieved using an antegrade approach alone. Several studies have demonstrated the efficacy and safety of retrograde recanalization, particularly in the femoropopliteal and infrageniculate arterial segments, in patients with advanced atherosclerotic disease.[2]

CASE REPORT

A 70-year-old male with diabetes mellitus (managed with insulin and oral agents for 8 years), hypertension, and stage 3 chronic kidney disease presented to our diabetic foot care clinic with a non-healing ulcer and focal gangrene on the dorsum of his right foot.

Doppler ultrasonography revealed absent flow beginning at the proximal segment of the anterior tibial artery (ATA). Diagnostic peripheral angiography confirmed a CTO of the ATA and multiple high-grade stenoses in the peroneal artery, with no significant distal ATA or pedal arch flow. (Supplementary Video 1).

Because no suitable distal landing zone was available in the ATA, we initially attempted an antegrade approach using the HydroDynamic contrast Recanalization (HDR) technique. (Supplementary Video 2). After engaging the proximal ATA segment with a 0.035" microcatheter, the system was exchanged for a 0.018" microcatheter. The proximal cap was engaged with a 0.014" Hornet™ peripheral wire, followed by the gentle injection of 0.5 cc of contrast to visualize the antegrade course.

Lesion crossing was first attempted with a 0.018" Blackeel™ hydrophilic peripheral guidewire and, after failure, with a 0.018" V-18™ Boston Scientific guidewire. Both attempts were unsuccessful.

Given the failure of the antegrade strategy, we proceeded with a retrograde approach. A 0.018" V-18™ Boston Scientific guidewire was introduced via the lateral plantar artery to access the distal ATA. (Supplementary Video 3). The guidewire passed smoothly through the occlusion into the proximal cap and was successfully advanced into the popliteal artery.

Using the retrograde wire as a fluoroscopic marker, we aligned and advanced the antegrade wire along the same path, entering the true lumen. (Supplementary Video 4). Alignment was confirmed with a tip injection. (Supplementary Video 5).

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Cite as: Süleymanoğlu C, Yurt R. Below-the-knee chronic total occlusion: the power of a multi-technique usage. *Inter Cardio Pers.* 2026;2(1):26-27

Received: 02.06.2025
Accepted: 11.08.2025
Epub: 11.09.2025
Publication Date: 10.04.2026

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Following successful wire passage, multiple balloon inflations were performed, achieving complete revascularization of the ATA. (Supplementary Video 6).

CONCLUSION

Retrograde revascularization is a safe and effective option for treating BTK vessel occlusions in diabetic patients, particularly when the antegrade approach fails. This case underscores the importance of flexibility and the synergistic application of multiple CTO techniques in complex interventions. The failure of one technique does not necessarily indicate ineffectiveness; rather, it may facilitate the success of another. In this instance, we believe the HDR technique may have created microchannels or weakened the proximal cap, enabling easier passage of the retrograde wire.

Collaboration between different CTO strategies is therefore essential. In the setting of heavily calcified, long-segment CTOs, HDR can serve as a valuable adjunct to retrograde approaches.

Retrograde revascularization has emerged as a safe and effective method for treating BTK vessel occlusions in diabetic patients, particularly when conventional antegrade approaches are unsuccessful. This case underscores the value of procedural flexibility and the strategic combination of CTO crossing techniques in managing complex infrainguinal disease.

In this patient, initial recanalization attempts using the HDR technique did not achieve full lumen passage. However, this was not a procedural failure. We believe HDR played a crucial preparatory role by altering the lesion morphology, potentially creating microchannels, weakening the proximal cap, or disrupting heavily calcified plaque. These modifications likely reduced resistance during the subsequent retrograde approach and indirectly contributed to procedural success.

This outcome highlights an important principle in complex CTO interventions: techniques that do not yield immediate technical success may still enhance the effectiveness of subsequent strategies. In this case, synergy between HDR and retrograde wire crossing was critical. Adapting the procedural plan and integrating multiple techniques underscores the importance of operator experience and familiarity with the full spectrum of CTO strategies.

HDR may be particularly valuable as an adjunct in long-segment, heavily calcified BTK occlusions, where direct lumen crossing is often challenging. Its use before retrograde intervention may facilitate wire entry or improve re-entry outcomes by modifying lesion architecture. Rather than viewing individual techniques in isolation, interventionalists should consider their complementary roles in achieving revascularization-especially in high-risk, multi-comorbid patients such as those with diabetes and critical limb ischemia.

Informed Consent: Informed consent was obtained from the patient.

Authorship Contributions: Concept: C.S., Design: C.S., Data Collection or Processing C.S., Literature Search: R.Y., Writing: R.Y.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study received no financial support.

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Supplementary Video 1.
https://youtube.com/shorts/sZQvjhqpZik

Supplementary Video 2.
https://youtube.com/shorts/GPnvs4Q40E8

Supplementary Video 3.
https://youtube.com/shorts/jXb0YfciT1Y

Supplementary Video 4.
https://youtube.com/shorts/o2082JyzCkk

Supplementary Video 5.
https://youtube.com/shorts/AgX-XTNOgcM

Supplementary Video 6.
https://youtube.com/shorts/vAHFkGtAvhs

INTERVENTIONAL CARDIOLOGY PERSPECTIVES

Inter Cardio Pers 2026;2(1):28-29

DOI: 10.4274/intercardiopers.2026.2025-10-3

CLINICAL IMAGE

Congenital Aortopulmonary Fistula Presenting with Chest Pain in an Adult: Diagnostic and Interventional Approach

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Keywords: Aortopulmonary fistula, cardiovascular, coil closure, congenital anomaly, interventional, percutaneous intervention

Congenital aortopulmonary fistula (APF) is a rare abnormal communication between the ascending aorta and the pulmonary artery.[1,2] Its presentation in adulthood is uncommon and may mimic ischemic syndromes, although true myocardial ischemia is typically absent.[1,3] We present the case of an adult patient with recurrent chest pain due to congenital APF that was successfully treated with percutaneous coil embolization.

A 53-year-old man presented with a 1-year history of exertional dyspnea and several weeks of typical angina at rest. His medical history included type 2 diabetes mellitus, hyperlipidemia, and transcatheter coil embolization performed in 2020 for a coronary–pulmonary artery fistula originating from a diagonal branch of the left anterior descending artery.

On admission, electrocardiography (ECG) revealed sinus rhythm without ischemic changes, and echocardiography showed preserved left ventricular function with an ejection fraction of 60% and no valvular abnormalities. The estimated pulmonary artery systolic pressure was within normal limits, with no echocardiographic evidence of pulmonary hypertension. Coronary angiography demonstrated no obstructive stenosis but revealed opacification of the main pulmonary artery from the ascending aorta, which was diagnostic of an APF (Figure 1A). Modified computed tomography (CT) angiography demonstrated the previously implanted coil and the apparent communication between the aorta and the pulmonary artery (Figures 1B and C).

Given the presence of typical angina, coronary fractional flow reserve and myocardial perfusion scintigraphy were performed, both of which were negative for ischemia. After a multidisciplinary heart team discussion, percutaneous coil closure of the fistula was undertaken. Complete angiographic occlusion was achieved without complications (Figure 1D). The patient experienced prompt and sustained relief of chest pain and exertional dyspnea at follow-up.

Congenital APFs are exceptionally rare and often remain undetected until adulthood.[2,3] Most cases are asymptomatic or present with dyspnea or cardiac murmurs; presentation with typical angina is unusual. Given the recent onset of angina at rest, alternative causes of chest pain were systematically investigated. Coronary angiography demonstrated normal epicardial coronary arteries without obstructive disease. Acute coronary syndrome, myocarditis, and valvular pathology were excluded using ECG, cardiac biomarkers, echocardiography, and functional ischemia testing. Therefore, the patient's symptoms were attributed to the hemodynamic and perfusion-related consequences of the APF.

In this patient, the mechanism of pain was likely multifactorial and may have included:

1. Preload-driven myocardial oxygen supply–demand imbalance resulting from a chronic left-to-right shunt that increased ventricular wall stress;
2. Diastolic run-off from the aorta into the pulmonary artery, transiently reducing coronary perfusion pressure;
3. Perivascular nociceptor stimulation secondary to vessel wall stretch;
4. Microvascular ischemia not detectable by conventional fractional flow reserve or perfusion studies.

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Cite as: Batak CN, Kahraman F, Astarcıoğlu MA, Korkmaz M. Congenital aortopulmonary fistula presenting with chest pain in an adult: diagnostic and interventional approach. *Inter Cardio Pers.* 2026;2(1):28-29

Received: 03.11.2025
Accepted: 27.01.2026
Epub: 05.02.2026
Publication Date: 10.04.2026

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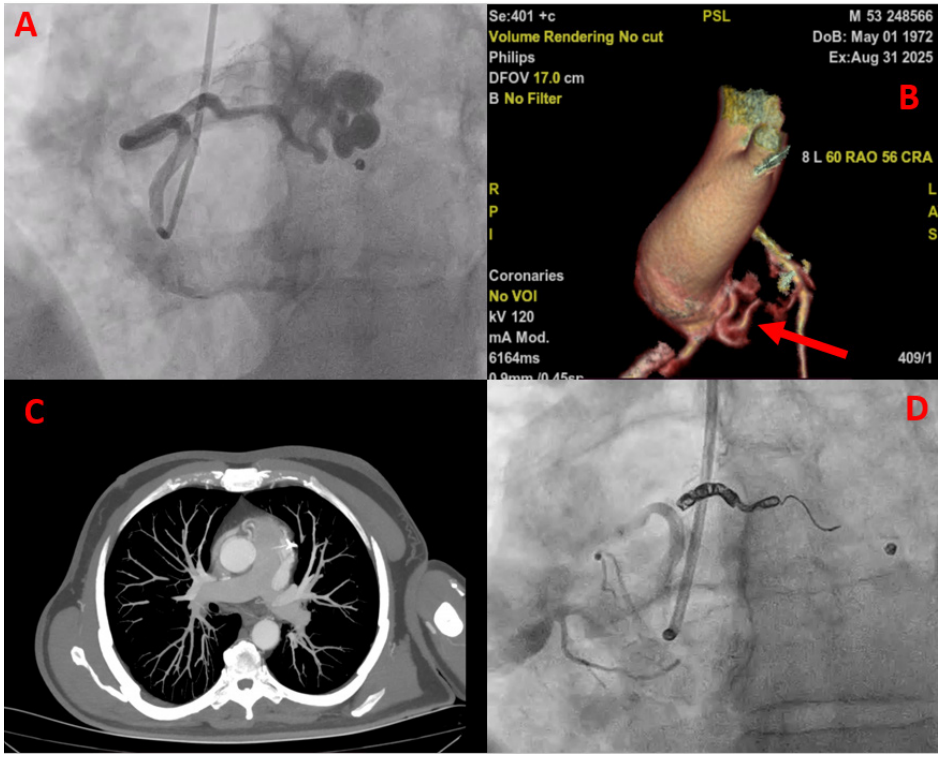


Figure 1. A) Aortopulmonary fistula detected by coronary angiography. B) Three-dimensional computed tomography (CT) angiography demonstrating the origin and course of the aortopulmonary fistula (arrows). C) Two-dimensional CT angiography showing the connection of the fistula to the main pulmonary artery (arrow). D) Angiographic image after percutaneous coil embolization demonstrating complete occlusion of the fistula

These mechanisms may explain the paradox of ischemic-type chest pain despite normal coronary arteries and negative functional ischemia testing.[1,2] Non-invasive assessment suggested the absence of a hemodynamically significant left-to-right shunt; therefore, Qp/Qs measurement by right heart catheterization was not considered necessary.

Multimodality imaging is essential for diagnosis and procedural planning. CT angiography accurately delineates the aortopulmonary connection, defines the fistulous tract, and guides device sizing. Invasive angiography confirms the anatomical features, allows shunt quantification, and enables closure during the same procedure. Functional testing (fractional flow reserve and perfusion imaging) remains useful for excluding concomitant coronary artery disease.[2]

For anatomically suitable fistulas, transcatheter closure is the preferred treatment strategy, offering high procedural success rates, rapid recovery, and durable symptom relief.[2,4,5] Surgical correction is reserved for complex, large, or anatomically unsuitable fistulas. In our case, coil embolization resulted in complete angiographic closure and full symptomatic improvement, highlighting the safety and efficacy of this approach in carefully selected patients.

APF is a rare but important cause of angina in adults with normal coronary arteries. A systematic diagnostic approach integrating multimodality imaging and selective functional testing is essential for identifying the underlying mechanism. Percutaneous transcatheter coil occlusion provides effective and minimally invasive treatment with excellent outcomes in appropriately selected patients.[2,4]

Informed Consent: Written informed consent was obtained from the patient for the publication of this case report and the accompanying images.

Authorship Contributions: Surgical and Medical Practices: C.N.B., F.K., Concept: C.N.B., F.K., M.A.A., M.K., Design: C.N.B., F.K., Data Collection or Processing: C.N.B., F.K., Analysis or Interpretation: C.N.B., F.K., M.A.A., M.K., Literature Search: C.N.B., F.K., Writing: C.N.B., F.K., M.A.A., M.K.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study received no financial support.

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