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# Periprocedural myocardial injury after elective percutaneous coronary intervention in stable angina pectoris: a tertiary center experience

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SOUHRN

**Kontext:** Periprocedurální poškození myokardu a jeho význam pro klinickou praxi u stabilizovaných pacientů je stále předmětem diskuse. Tímto tématem se za celé roky zabývala řada studií s různými definicemi, prahovými hodnotami a biomarkery. Cílem naší studie bylo popsat výsledný klinický stav pacientů s periprocedurálním poškozením myokardu pomocí definic nejnovějších, 4. doporučených postupů UDMI.
**Metody:** Do monocentrické studie bylo retrospektivně zařazeno 238 pacientů po elektivní perkutánní koronární intervenci. Pacienti s periprocedurálním poškozením myokardu byli po výkonu srovnáni s pacienty s normálními hodnotami troponinu z hlediska výsledného klinického stavu. Mezi primární sledované parametry patřily úmrtí, infarkt myokardu, cévní mozková příhoda, refrakterní angina pectoris, revaskularizace cílové léze a hospitalizace pro akutní koronární syndrom do jednoho roku.
**Výsledky:** Periprocedurální poškození myokardu bylo zjištěno u 67,2 % (n = 160) pacientů. Počty lézí a stentů, celková délka stentů, celkový průměr stentů, stav po dilataci, počty překrývajících se stentů, stenty na bifurkacích a skóre SYNTAX byly statisticky významně vyšší ve skupině s poškozením myokardu. Uvedené sledované parametry se vyskytly u 16 pacientů (11 ve skupině s poškozením myokardu a 5 v kontrolní skupině). Během sledování nedošlo k žádnému úmrtí. Poprocedurální zvýšení hodnot troponinu nebylo nijak spojeno se sledovanými parametry (11 vs. 5; $p = 0,56$). V Kaplanově–Meierově analýze se křivky sledovaných parametrů v obou skupinách od sebe nevzdalovaly (log rank test; 95% interval spolehlivosti [CI], $p = 0,71$).
**Závěry:** Velikost a délka stenu, dilatace po výkonu, překrývání stentů a implantace stentů do lézí v bifurkacích zvyšují riziko poškození myokardu. Periprocedurální poškození myokardu u stabilní anginy pectoris nelze použít k predikci výsledného stavu pacienta po jednom roce.



*Keywords:*
Elective percutaneous coronary intervention
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ABSTRACT

**Background:** Periprocedural myocardial injury and its clinical significance in stable patients are still under discussion. This subject has been assessed in many studies with different definitions, thresholds, and biomarkers for years. This study aimed to determine the clinical outcomes of periprocedural myocardial injury based on latest 4th UDMI guideline definitions.
**Methods:** 238 patients who underwent elective percutaneous coronary intervention at single center were retrospectively enrolled. Patients who developed periprocedural myocardial injury were compared with patients with normal troponin values after the procedure for clinical outcomes. Primary clinical endpoints were death, MI, stroke, refractory angina, target vessel revascularization and hospitalization due to acute coronary syndrome at one year.
**Results:** Periprocedural myocardial injury was observed in 67.2% (n = 160) of patients. Number of lesions and stents, total stent length, total stent diameter, post-dilatation, overlapping stents, bifurcation stenting and SYNTAX score were significantly higher in myocardial injury group. Clinical outcomes occurred in 16 patients, 11 of 16 had myocardial injury group, 5 of 16 had control group. No mortality was seen during the follow-up. Postprocedural troponin elevation was not associated with clinical outcomes (11 vs. 5, $p = 0,56$). Kaplan–Meier curve of clinical end points did not show any separation between the curves (Log rank test, 95% CI, $p = 0,71$).
**Conclusion:** Stent size and length, post-dilatation, overlapping stents and stenting of bifurcation lesions lead to increase in myocardial injury. Periprocedural myocardial injury in stable angina does not predict clinical outcomes at one year.

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## Introduction

Percutaneous coronary intervention (PCI) is an effective treatment of both stable and unstable angina pectoris. Despite its efficacy, fatal complications may occur rarely during PCI, such as acute stent thrombosis, acute plaque rupture, stroke and death.[1] However, periprocedural myocardial injury and infarction occur more frequently after PCI.[2] Cardiac troponins (T or I) are specific and sensitive biomarkers for detection of myocardial injury.[3] Periprocedural myocardial injury and infarction with their prognostic value have been assessed for past two decades. In 2000, periprocedural myocardial infarction (Type 4a MI) was defined at first in Universal Definition of Myocardial Infarction (UDMI) guideline which was published by the joint committee of the European Society of Cardiology (ESC) and the American College of Cardiology (ACC). Periprocedural myocardial infarction was termed as an increase in cardiac troponin of 99th upper reference limit (URL) after PCI.[3] UDMI guidelines have been revised in 2007, 2012, and 2018. In 2012 with the third UDMI, periprocedural myocardial injury and periprocedural myocardial infarction (Type 4a MI) were distinguished from each other.[4] Myocardial injury was defined only by elevation of cardiac troponins above five-fold URL.[4] Type 4a MI was above the five-fold cardiac troponin elevation and included angiographic flow limiting situations, such as dissection, slow flow or no-reflow, major epicardial artery or side branch occlusion, distal embolization.[4] Finally, in 2018, fourth UDMI has been published and these definitions have been revised again. According to this guideline, myocardial injury defined by increases of cardiac troponin values above the 99th URL in patients with normal baseline values.[5] Beyond these definitions and revisions, periprocedural myocardial injury and its clinical outcomes are still under discussion.

Stable angina pectoris is a clinical entity that includes typical chest pain with stable duration and frequency lasting for two months. Elective PCI can be performed for treatment, which has a relatively lower risk for ischemic events than the other clinical conditions. In this study, we aimed to investigate periprocedural myocardial injury after elective PCI in stable angina pectoris with new definitions based on the fourth UDMI guideline and its impact of clinical outcomes at 1-year period.

## Materials and methods

### Study population

Based on the definitions in the 'Fourth Universal Definition of Myocardial Infarction' guideline published by the European Society of Cardiology in 2018, 238 patients with stable coronary artery disease who underwent percutaneous coronary intervention between 1st June 2017 and 1st June 2019 in our clinic with baseline troponin T levels were within normal reference range were retrospectively included. The demographic characteristics of the patients were included. Past medical history and comorbidities such as diabetes, hypertension, hyperlipidemia, prior MI, previous PCI and coronary artery bypass graft (CABG) and smoking habits were evaluated via electronic registry database.

Ethics statement was obtained in our hospital's Ethics Committee (date 04/10/2019, No: B08.06, YOK 2.I.U-.E.50.0.05.00/8).

### Biochemical analysis

Troponin T, complete blood count (CBC), blood urea nitrogen (BUN), serum creatinine, electrolytes (sodium and potassium) were measured in all patients before and after 12 hours (12 h) of procedure. Elecsys Troponin T STAT 3rd Generation (Roche Diagnostics, Germany) was used for troponin assay. Assay's 99th percentile upper reference limit (URL) was 0.014 picogram per milliliter (pg/ml). The detection of the troponin T value above the upper reference limit (>0.014 pg/mL) was accepted as myocardial injury.

### Angiographic analysis

All patients' coronary angiograms and angioplasties were evaluated by expert consultant via hospital's electronic registry database. Flow limiting situations such as dissection, coronary slow-flow, no-reflow, side branch occlusion-subocclusion during the procedure had been investigated and noted. Stent types, stent diameters and stent numbers were also noted. SYNTAX (Synergy between Percutaneous Coronary Intervention with TAXUS and Cardiac Surgery) score for lesion complexity was also calculated. In patients who required multiple interventions only a single procedure was included.

### Clinical outcomes

Patients who developed myocardial injury after the procedure were compared with patients whose troponin values remained within the normal reference range after the procedure for clinical outcomes including death, MI, stroke, refractory angina, target vessel revascularization, and hospitalization due to acute coronary syndrome at one year. Clinical outcomes were obtained by telephone interviews with patients and hospital's electronic registry system.

### Statistical analysis

SPSS Windows version 23.0 package program was used for statistical analysis. The conformity of the data to normal distribution was tested with the Kolmogorov–Smirnov test. Continuous variables were indicated as median (25–75 percentiles) and number and % values for categorical variables are given. In comparison of quantitative data in two independent groups, Mann–Whitney U test was used for non-normally distributed properties. Relationships of two independent variables at categorical measurement level with each other were tested with Chi-square test. Logistic regression was used to examine the relationship of clinical events in patients with demographic, biochemical, and angiographic variables. The log-rank test and Kaplan–Meier curve were used to examine the prognostic impact of time-to-event data. *P*-value <0.05 was considered statistically significant.

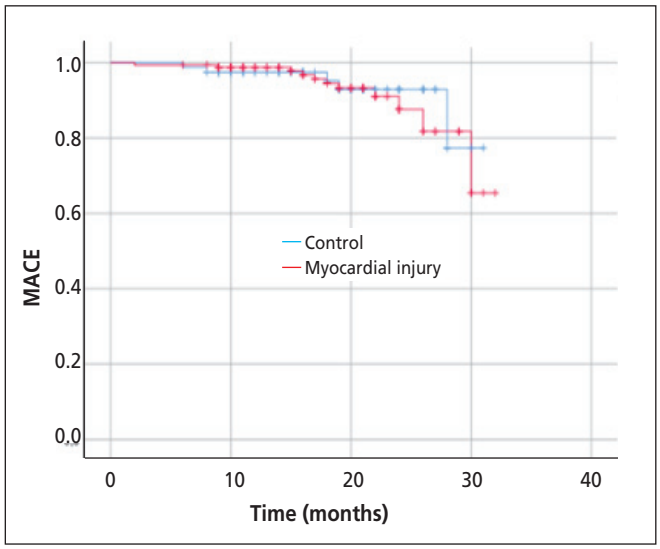


**Fig. 1 – Kaplan–Meier clinical outcome curves for myocardial injury and control groups (Log rank test, 95% confidence interval, $p = 0.71$).**

## Results

Of 259 patients with stable angina pectoris who underwent elective PCI in our clinic, 12 patients with postprocedural troponin elevation and 9 patients with normal postprocedural troponin levels were lost for clinical outcomes and excluded. Study was completed with 238 patients. Mean follow up time for clinical outcomes were 17 months.

In this study, 160 (67.2%) patients had myocardial injury. 40 (16.8%) of them had >5× URL postprocedural troponin elevation. Based on 4th UDMI guideline, only 5 (2.1%) patients met criteria for periprocedural myocardial infarction (Type 4a MI). 78 (31.8%) patients with normal postprocedural troponin levels were included in the control group. Table 1 showed the baseline demographic characteristics for both groups. There were no differences between two groups mostly for demographic characteristics. Biochemical and angiographic characteristics were shown in Table 2 and Table 3. Biochemical parameters did not show any differences between two groups. However, angiographic parameters number of lesions, SYNTAX score, number of stents, total stent length, total stent diameter were significantly higher in myocardial injury group. Performing post-dilatation was also significantly higher in myocardial injury group. Moreover, patients with overlapping stents and PCI for bifurcation lesions had higher myocardial injury. Yet, PCI for chronic total occlusion (CTO) had no difference between groups.

Angiographic flow-limiting conditions occurred in 16 patients. One of them occurred in troponin negative group which had coronary dissection. Six had coronary dissection, six had side-branch occlusion, and five had side-branch subocclusion, which occurred in the myocardial injury group. Only 5 patients met the periprocedural MI criteria based on 4th UDMI guideline. There were no association between the flow-limiting conditions and postprocedural myocardial injury (15 vs. 1, $p = 0.211$).

Clinical outcomes had occurred in 16 patients during follow-up. Eleven of 16 occurred in myocardial injury group, 5 of 16 occurred in control group. Eight patients had target vessel revascularization, 2 had cerebrovascular event, 1 had refractory angina, 5 had hospitalization due to acute coronary syndrome. No mortality was seen during follow-ups. Postprocedural troponin elevation was not associated with clinical outcomes (11 vs. 5, $p = 0.56$). The logistic regression analysis which included post-dilatation, serum hemoglobin level after 12 h and age as independent variables, showed significant correlation between serum hemoglobin levels and clinical outcomes ($p = 0.033$, respectively) (Table 4).

Kaplan–Meier curve of major adverse clinical outcomes by postprocedural troponin levels (Fig. 1) did not show any separation between the curves (Log rank test, 95% CI, $p = 0.71$).

**Table 1 – Baseline features of the study group**

| | Myocardial injury (n = 160) | Control (n = 78) | Overall *p*-value* |
|---|---|---|---|
| Age | 64 (55–70) | 60 (55–66) | **0.025** |
| Male sex, n (%) | 121 (75.6%) | 58 (74.4%) | >0.05 |
| Diabetes mellitus, n (%) | 61 (38.1%) | 32 (41%) | >0.05 |
| Hypertension, n (%) | 99 (61.9%) | 56 (71.8%) | >0.05 |
| Dyslipidemia, n (%) | 101 (63.1%) | 51 (65.3%) | >0.05 |
| Family history of premature CAD, n (%) | 40 (25%) | 16 (20.5%) | >0.05 |
| Smoking, n (%) | 46 (28.8%) | 21 (26.9%) | >0.05 |
| Previous CAD, n (%) | 92 (57.5%) | 53 (67.9%) | >0.05 |
| Prior CABG, n (%) | 20 (12.5%) | 8 (10.3%) | >0.05 |
| Prior PCI, n (%) | 64 (40%) | 35 (44.9%) | >0.05 |
| Heart failure, n (%) | 4 (2.5%) | 1 (1.3%) | >0.05 |
| Chronic kidney disease, n (%) | 8 (5%) | 0 | **0.045** |

**Table 2 – Comparison of study groups according to biochemical characteristics**

| | Myocardial injury (n = 160) | Control (n = 78) | Overall *p*-value* |
|---|---|---|---|
| Hemoglobin (g/dL) | 13.95 (12.7–15) | 14.2 (12.7–15) | >0.05 |
| Hemoglobin – 12 h (g/dL) | 13.1 (11.8–14.3) | 13.1 (11.87–14) | >0.05 |
| Platelet count (×1000) | 232 (193–269.5) | 229.5 (198–264.5) | >0.05 |
| Platelet count – 12 h (×100) | 211.5 (175–237.75) | 214.5 (180.5–240.75) | >0.05 |
| Serum creatinine (mg/dL) | 0.87 (0.78–1.01) | 0.85 (0.75–0.98) | >0.05 |
| Serum creatinine – 12 h (mg/dL) | 0.89 (0.75–1) | 0.89 (0.74–0.98) | >0.05 |

**Table 3 – Comparison of study groups according to angiographic characteristics**

| | Myocardial injury (n = 160) | Control (n = 78) | Overall *p*-value |
|---|---|---|---|
| SYNTAX score | 9 (5.25–16) | 7 (4–12) | **<0.001** |
| Stent length (mm) | 26 (18–44) | 20 (18–30) | **0.001** |
| Maximal stent diameter (mm) | 3.4 (3.1–3.58) | 3.175 (2.87–3.5) | **0.003** |
| Overlapping | 43 (26.9%) | 10 (12.8%) | **0.014** |
| Predilatation | 114 (71.7%) | 56 (71.8%) | >0.05 |
| Postdilatation | 64 (40%) | 19 (24.4%) | **0.017** |
| Bifurcation lesion | 15 (9.3%) | 2 (2.56%) | **<0.05** |
| CTO | 3 (1.6%) | 1 (1.28%) | >0.05 |

**Table 4 – Logistic regression analysis indicating independent predictors of clinical endpoints**

| Variable | Beta | Standard error | Significance |
|---|---|---|---|
| Age | –0.055 | 0.028 | 0.053 |
| Hemoglobin 12 h | –0.347 | 0.163 | **0.033** |
| Troponin T 12 h | –0.272 | 3.259 | 0.933 |
| Post-dilatation | 1.027 | 0.555 | 0.064 |

## Discussion

This study aimed to achieve prognostic significance of myocardial injury which had been developed after percutaneous coronary intervention in stable CAD patients based on 4th UDMI guideline's definition and thresholds. It demonstrated no difference between clinical outcomes in terms of myocardial injury and study groups. 67.2% of patients had postprocedural myocardial injury in this study. If the 3rd Universal Definition of Myocardial Infarction guidelines' definitions could be used as myocardial injury, which included >5× URL troponin elevation after the procedure, this ratio would be 16.8%. Myocardial injury was detected 5–40% after successful percutaneous coronary intervention in previous studies.[6,7] These differences depend on varies among study population, lesion characteristics, and definitions.

Cardiac biomarkers had been used to detect myonecrosis in past decades. CK-MB was the first biomarker to diagnose myocardial injury for elective PCI. Cardiac troponins (troponin T and I) which detect myocardial injury at lower concentrations and have high sensitivity and specificity are used to diagnose myocardial injury and infarction. We preferred cardiac troponin T for our study because of its high sensitivity and had diagnostic criteria for 4th UDMI definition. Significant CK-MB elevation after PCI >3–8× URL had associated with increased in hospital and long-term outcomes.[6,8,9] Mild troponin elevation after PCI has occurred more often than CK-MB. In patients with acute coronary syndrome, mild troponin elevation after PCI was associated with poor clinical outcomes.[10] In patients who have stable angina pectoris, myocardial injury after PCI and its clinical impact are controversial.

In a consecutive series of 1 532 patients who underwent elective PCI for stable angina, Auguadro et al.[11] found a significant difference for major adverse cardiac events for patients with troponin I elevation above than 1 ng/mL after PCI. Troponin I assay's 99th URL was 1 ng/mL and as myocardial injury was accepted troponin I elevation above the 99th URL after PCI. Similarly, Yang et al.[12] analyzed 516 patients who underwent elective PCI. In this study, troponin assay was troponin I and assay's 99th URL was 0,01 ng/mL. Definitions of Type 4a myocardial infarction and injury were based on 3rd UDMI criteria. Type 4a MI was not associated with increased mortality, however, each type 4a MI and myocardial injury were associated with recurrent MI and death (3%, $p$ = 0.02 and 0.03, respectively).[12] In another study, Zeitouni et al.[13] evaluated 1666 patients who underwent elective PCI for stable angina. Criteria for Type 4a MI and myocardial injury were based on the 3rd UDMI guidelines. For detecting myonecrosis, high sensitive troponin T was used. Assay's 99th URL was 14 ng/L. Major adverse cardiac outcomes were associated with Type 4a MI and myocardial injury at 30 days (5.5% vs. 1.2%, adjusted hazard ratio 3.8; 95% confidence interval (CI) 1.9–6.9; $p$ <0,001) and at one year (adjusted hazard ratio 1.7; 95% CI 1.1–2.6; $p$ = 0,004).[13]

Although these studies found a significant association between postprocedural troponin elevation and clinical outcomes, there were also contrary studies which found no association between myocardial injury and clinical outcomes in the literature. Christensen et al.[2] evaluated 2760 patients for prognostic impact of myocardial infarction and injury after elective PCI. Based on the 3rd UDMI definitions for myocardial infarction and injury, troponin T was chosen for biomarker. Assay's 99th URL was 14 ng/L. There was no significant association between Type 4a MI and all-cause mortality (HR 0.92; 95% CI 0.78–1.09; $p$ = 0.35).[2] Garcia-Garcia et al.[14] analyzed 13,452 patients who underwent elective PCI from 5 stent studies and 1 large registry. Troponin and CK-MB were used in this study for myonecrosis, however, assay's 99th URL and cut-off value were not defined. Type 4a myocardial infarction criteria based on the Society for Cardiovascular Angiography and Interventions (SCAI) definitions, which have defined cardiac troponin elevation >70× URL after PCI. There was no significant association between mortality and troponin elevation >70× URL after PCI (HR 1.62; 95% CI 0.88–3; $p$ = 0.11).[14] Recently, Zhou et al.[15] published a study with 1 572 stable patients. Troponin T was used to detect injury and troponin value above than 99th URL after PCI was defined procedural myocardial infarction. Myocardial infarction after elective PCI was not associated with all-cause mortality (10 vs. 8, HR 0.97; 95% CI 0.38–2.46; $p$ = 0.948).[15] In favor of aforementioned studies, no relationship with clinical outcomes and postprocedural myocardial injury were also found in our study. Although adverse clinical events occurred mostly in myocardial injury group in our study, it did not reach statistical significance. This could be associated with low lesion complexity, less complex PCI procedures, and infrequent flow-limiting conditions during PCI, and a small study population.

Myocardial injury has occurred due to silent myocardial cell necrosis, thrombus, micro-embolization, or slow-flow that could not be captured.[16,17] Angiography could explain only 60% to underlying mechanisms of myocardial injury.[18] Side branch occlusion and distal embolization were major causes of myocardial injury.[19] Bifurcation stenting, stent length, number of implanted stents were also associated with myocardial injury.[13,20,21] We found that myocardial injury arm had more bifurcation PCI, number of stents and stent length than control group like aforementioned studies. Stent length longer than 30 millimeters have predicted major adverse cardiovascular outcomes independently.[13] Beyond these mechanisms myocardial injury could occur after diagnostic coronary angiography, advanced age (>65 years) and fluoroscopy time had associated with troponin elevation.[22] Thus, clinical decision still relies on cardiac biomarkers. On the other hand, various definitions and thresholds, different assays to diagnose myocardial injury could affect clinical outcomes among studies. Myocardial injury and its prognosis are still controversial. Further studies are needed to explain this subject.

## Study limitations

There were some limitations about these studies. It had a retrospective design and patients were included at single center. Therefore it was liable to bias. Data and clinical outcomes had gathered from hospital's electronic registry system. Finally this study had small sample size which underpowered to detect meaningful differences.

## Conclusion

Number of lesions and stents, total stent length and diameter, post-dilatation, overlapping stents and PCI for bifurcation lesions were significantly higher in myocardial injury group. Myocardial injury based on the 4th UDMI after percutaneous coronary intervention with stable angina was not associated with death, MI, stroke, target vessel revascularization, refractory angina and hospitalization due to acute coronary syndrome at one year.

### References


1. Montalescot G, Sechtem U, Achenbach S, et al. 2013 ESC guidelines on the management of stable coronary artery disease: the Task Force on the management of stable coronary artery disease of the European Society of Cardiology. Eur Heart J 2013;34:2949–3003.
2. Christensen MK, Huang H, Torp-Pedersen C, et al. Incidence and impact on prognosis of peri-procedural myocardial infarction in 2760 elective patients with stable angina pectoris in a historical prospective follow-up study. BMC Cardiovasc Disord 2016;16:16–140.
3. Myocardial infarction redefined: a consensus document of the Joint European Society of Cardiology/American College of Cardiology Committee for the redefinition of myocardial infarction. J Am Coll Cardiol 2000;36:959–969.
4. Thygesen K, Alpert JS, White HD, et al. Third universal definition of myocardial infarction. Eur Heart J 2012;33:2551–2567.
5. Thygesen K, Alpert JS, Jaffe AS, et al. Executive Group on behalf of the Joint European Society of Cardiology (ESC)/American College of Cardiology (ACC)/American Heart Association (AHA)/World Heart Federation (WHF) Task Force for the Universal Definition of Myocardial Infarction. Fourth universal definition of myocardial infarction. J Am Coll Cardiol 2018;72:2231–2264.
6. Califf RM, Abdelmeguid AE, Kuntz RE, et al. Myonecrosis after revascularization procedures. J Am Coll Cardiol 1998;31:241–251.
7. Fuchs S, Kornowski R, Mehran R, et al. Prognostic value of cardiac troponin-I levels following catheter-based coronary interventions. Am J Cardiol 2000;85:1077–1082.
8. Akkerhuis KM, Alexander JH, Tardiff BE, et al. Minor myocardial damage and prognosis: are spontaneous and percutaneous coronary intervention related events different? Circulation 2002;105:554–556.
9. Abdelmeguid AE, Topol EJ, Whitlow PL, et al. Significance of mild transient release of creatine kinase-MB fraction after percutaneous coronary interventions. Circulation 1996;94:1528–1536.
10. Cannon CP, Weintraub WS, Demopoulos LA, et al. Comparison of early invasive and conservative strategies in patients with unstable coronary syndromes treated with the glycoprotein IIb/IIIa inhibitor tirofiban. N Engl J Med 2001;344:1879–1887.
11. Auguadro C, Scalise F, Manfredi M, et al. The prognostic role of troponin I elevation after elective percutaneous coronary intervention. J Cardiovasc Med (Hagerstown) 2015;16:149–155.
12. Yang X, Tamez H, Lai C, et al. Type 4a myocardial infarction: Incidence, risk factors, and long-term outcomes. Catheter Cardiovasc Interv 2017;89:849–856.
13. Zeitouni M, Silvain J, Guedeney P, et al.; ACTION Study Group. Periprocedural myocardial infarction and injury in elective coronary stenting. Eur Heart J 2018;39:1100–1109.

14. Garcia-Garcia HM, McFadden EP, von Birgelen C, et al. Impact of Periprocedural Myocardial Biomarker Elevation on Mortality Following Elective Percutaneous Coronary Intervention. JACC Cardiovasc Interv 2019;12:1954–1962.
15. Zhou Y, Chen Z, Ma J, et al. Incidence, predictors and clinical significance of periprocedural myocardial injury in patients undergoing elective percutaneous coronary intervention. J Cardiol 2020;76:309–316.
16. Prasad A, Herrmann J. Myocardial infarction due to percutaneous coronary intervention. N Engl J Med 2011;364:453–464.
17. Herrmann J. Peri-procedural myocardial injury: 2005 update. Eur Heart J 2005;26:2493–2519.
18. Muschart X, Slimani A, Jamart J, et al. The different mechanisms of periprocedural myocardial infarction and their impact on in-hospital outcome. J Invasive Cardiol 2012;24:655–660.
19. Bulluck H, Paradies V, Barbato E, et al. Prognostically relevant periprocedural myocardial injury and infarction associated with percutaneous coronary interventions: a Consensus Document of the ESC Working Group on Cellular Biology of the Heart and European Association of Percutaneous Cardiovascular Interventions (EAPCI). Eur Heart J 2021;42:2630–2642.
20. Loeb HS, Liu JC. Frequency, risk factors, and effect on long-term survival of increased troponin I following uncomplicated elective percutaneous coronary intervention. Clin Cardiol 2010;33:E40–E44.
21. Koskinas KC, Ndrepepa G, Raber L, et al. Prognostic impact of periprocedural myocardial infarction in patients undergoing elective percutaneous coronary interventions. Circ Cardiovasc Interv 2018;11:e006752.
22. Abu Sharar H, Wohlleben D, Vafaie M, et al. Coronary angiography-related myocardial injury as detected by high-sensitivity cardiac troponin T assay. EuroIntervention 2016;12:337–344.