The relationship between long-term statin usage and myocardial injury related to percutaneous coronary interventions evaluated by high-sensitivity troponins

Yüksek duyarlılıkli troponin ile değerlendirilen perkütan koroner girişime bağlı miyokart hasarının statin kullanımıyla ilişkisi

Ajar Koçak, M.D.1, Serkan Ünlü, M.D.1, Gökhân Gökalp, M.D.1, Niyazi Samet Yılmaz, M.D.2, Özlem Gülbahar, M.D.2, Adnan Abacı, M.D.1

1Department of Cardiology, Gazi University School of Medicine, Ankara, Turkey
2Department of Medical Biochemistry, Gazi University School of Medicine, Ankara, Turkey

ABSTRACT

Objective: Myocardial injury related to percutaneous coronary interventions (PCI) might adversely affect the prognosis of patients with coronary artery disease. Our study aimed to investigate the effects of long-term statin usage on myocardial injury related to elective PCI.

Methods: In our study, total 102 patients were included and evaluated in 3 groups based on the statin usage before PCI, “potent statin” group (n=26), “weak statin” group (n=23), and “statin free” group (n=53). The occurrence of the procedural complications was identified (n=31). The myocardial injury was determined by serial high-sensitivity troponin T (hsTnT) testing at 0th, 2nd, 4th, and 12th hour of the procedure.

Results: The increase in hsTnT values in the 2nd and 4th hour was significantly lower in the potent statin group than in the other 2 groups (p=0.008 and p=0.009, respectively). In patients with procedural complications, the increase in hsTnT levels at the 2nd, 4th, and 12th hour were also lower in the potent statin group (p=0.032, p=0.019, and p=0.006, respectively). Also, in patients with procedural complications, hsTnT levels exceeding the myocardial infarction limit at the 4th and 12th hour were lower in the potent statin group (p=0.039 and p=0.006, respectively).

Conclusion: These results show that elective PCI related myocardial injury was less frequent in patients who were using high-dose statins. This result was more pronounced in patients who developed complications during the procedure.

Percutaneous coronary intervention (PCI) is a frequently used procedure in the treatment of coronary artery disease (CAD). However, even elective PCI procedures may cause myocardial injury.1,2 Prolonged balloon inflation time, side-branch occlusion, obstructive coronary dissection, or distal embolization of thrombotic and atherosclerotic materials can lead to myocardial ischemia and result in ischemia-related myocardial injury.3,4 Regardless of the mechanism, myocardial injury may adversely affect the morbidity and mortality of the patients.5 The elevated cardiac troponin level is a specific marker for myocardial injury.6 With the introduction of newly developed high-sensitive troponins (hsTnT), it has

ÖZET

Amaç: Perkütan koroner girişimler (PKG) ilişkili miyokardiyal hasar, koroner arter hastalığı olan hastaların prognozu olumsuz etkileyebilir. Çalışmamızın amacı, uzun süreli statin kullanımının elektif PKG ile ilişkili miyokardiyal hasar üzerine etkilerini araştırmaktır.

Yöntemler: Çalışmamıza toplam 102 hasta dahil edildi ve PKG öncesinde statin kullanımına göre üç gruba değerlendirildi, “potent statin” grubu (n=26), “zayıf statin” grubu (n=23) ve “statin içermeyen” grubu (n=53). PKG sırasında kompleksiyon gelişen hastalar belirlendi (n=31). Miyokardiyal hasar, işlemin 0, 2, 4 ve 12. saatlerinde seri yüksek duyarlılıkli troponin T (hsTnT) testi ile belirlendi.

Bulgular: Potent statin grubunda 2. ve 4. saatlerde hsTnT değerlerinde artış diğer iki gruba göre anlamlı olarak daha düşüktü (sirasiyla p=0.008 ve 0.009). Prosedürel kompleksiyon yaşanan hastalarla 2., 4. ve 12. saatlerde hsTnT düzeylerindeki artış potent statin grubunda diğer iki grubu göre daha düşük (sirasiyla p=0.032, 0.019 ve 0.006). Ayrıca işlem sırasında kompleksiyon yaşanan hastalarda, potent statin grubunda 4. ve 12. saatlerde hsTnT düzeyleri miyokart enfarktusunun sınırını aşan hastalar daha azdı. (p=0.039 ve 0.006, sırasıyla).

Sonuç: Bu sonuçlar, yüksek doz statin kullanlan hastalarda elektif PKG ile ilişkili miyokardiyal hasarın daha az sıklıkta olduğunu göstermektedir. İşlem sırasında kompleksiyon gelişen hastalarda bu sonuç daha belirgindir.
become possible to detect patients with minor myocardial injury earlier by providing prognostic information in various cardiac pathologies.\[7\]

Statins are the most used lipid-lowering agents in the treatment and prevention of CAD.\[8\] Beside its lipid-lowering effects, the pleiotropic effects carry importance as well.\[9\] Previous studies have suggested that statins may be useful in preventing myocardial injury related to PCI.\[10,11\] Recently, evidence have shown that statins have beneficial effects on reducing endothelial dysfunction, preventing the proliferation of vascular smooth muscle cells, reducing vascular inflammation, and vasoconstriction.\[12\] The bioavailability of nitric oxide is also increased by administering statins, which prevents coronary endothelial cell and cardiomyocyte injury, and play a role in inducing collateral blood flow reducing ischemia.\[9,13\] The lipid-lowering effect of statins also leads to reduced activity of matrix metalloproteinases in the activated macrophages and reduced macrophage accumulation in atherosclerotic lesions. This causes changes in the biochemical characteristics of atherosclerotic plaques, and as a result, it has a plaque-stabilizing effect; thus, plaque rupture and related distal embolization events during PCI may be less frequent in patients with chronic statin use.\[9\] In a very recent study, statins have also proven to play a role in reducing myocardial injury after percutaneous coronary intervention on mRNA levels.\[14\] Although there is no clear evidence that statins have a direct cardioprotective effect, by the above mentioned mechanisms, statins play an important role in decreasing the incidence and degree of myocardial injury related to PCI.\[13,15\]

In this study, we investigated the impact of long-term statin usage on myocardial injury related to PCI detected by hsTnT in patients with stable CAD undergoing elective PCI.

### METHODS

**Study population**

In this cross-sectional, prospective observational study, 102 patients who underwent elective PCI between May 2015 and July 2017 were enrolled. Patients were assigned to 3 groups based on the statin usage for at least 8 weeks before the procedure. Patients who were receiving atorvastatin 40 mg, rosuvastatin 20 mg or higher doses of these statins were included in the “potent statin” group (26 patients); the rest of statin users were included in the “weak statin” group (23 patients), and patients who were not receiving statins were assigned to “statin free” group (53 patients). Any events that may lead to unwanted changes in the normal physiological coronary hemodynamics are defined as procedural complications; these were distal embolization of plaque debris or thrombus, dissection or spasm of coronary arteries, slow coronary flow or no reflow phenomenon after intervention, atherogenic plaque shift, and new branch occlusion. The occurrence of such events was identified by 2 blinded readers, and detailed information about these types of complications are mentioned in the results section of this manuscript. According to the occurrences of procedural complications, a subgroup analysis was done in terms of 2 groups, patients with and without procedural complications.

Patients of age above 18 and below 80 who planned for elective PCI in our hospital and accepted to be part of the study were included in our research, consecutively. Patients who met the European Society of Cardiology criteria of acute coronary syndromes\[16\] and those using antihyperlipidemic drugs other than statins or having no clear information about the usage of statin therapy before PCI were excluded from the study. All patients who met the inclusion criteria were informed about the research and written consents were obtained before participation. The study was approved by the ethical committee of the Gazi University School of Medicine on April 13, 2015 (document number 162). The type of the intervention was decided by the treating physician according to the coronary anatomy, and the antithrombotic treatment protocol of the patients followed our clinic’s routine of 300 mg acetylsalicylic acid and 300 mg clopidogrel as loading dose, and 100 mg acetylsalicylic acid and 75 mg clopidogrel as maintenance therapy. No changes out of the standard treatment plan were implemented among study participants.

The demographic and clinical characteristics of the patients such as age, gender, height, weight,
body mass index, medical history, medication, and results of blood tests were recorded. History of hypertension, hypercholesterolemia, diabetes mellitus, smoking status, and family was carefully noted. Hypertension is defined as a persistent elevation in systolic blood pressure (BP) ≥140 and/or diastolic BP ≥90 mmHg, which is equivalent to a 24-h ambulatory BP monitoring average of ≥130/80 mmHg or home BP monitoring average of ≥135/85 mmHg. Hypercholesterolemia is defined as low-density lipoprotein (LDL)-cholesterol >190 mg/dL, >160 mg/dL with one major risk factor, or >130 mg/dL with 2 cardiovascular risk factors. Participants who smoke any tobacco product, either daily or occasionally, were considered smokers. Participants who were using anti-diabetic treatment or have a fasting blood glucose level ≥126 mg/dL, or by having a non-fasting blood glucose level ≥200 mg/dL was considered diabetic. Patients were considered to have a positive family history of CAD if an immediate family member had fatal/nonfatal myocardial infarction, coronary artery bypass surgery (CABG), or coronary angioplasty before 55 years of age for males and 65 years of age for females.

Blood samples

Blood samples were collected into serum separator tubes (BD Vacutainer VR SSTTM II Advance, Ref: 367953, BD, Plymouth, United Kingdom) at 0th, 2nd, 4th, and 12th hour of PCI. The levels of hsTnT were measured with a hsTnT assay (Elecsys Troponin T high sensitive, Roche Diagnostics, Penzberg, Germany) with lower detection and upper reference (99th percentile) limits of 5 ng/L and 14 ng/L, respectively.

Clinical evaluation of myocardial injury

Myocardial injury was determined by comparing serial hsTnT measurements at 0th, 2nd, 4th, and 12th hour of PCI. Patients were compared in terms of hsTnT elevation rate, exceedance of the 99th percentile upper reference limit and PCI-associated myocardial infarction limit according to the recent guideline of the European Society of Cardiology.\(^\text{[16]}\)

Statistical analysis

Continuous variables were examined by Shapiro-Wilk test to check for the normality of distribution. Continuous variables are presented as a mean±standard deviation or a median and interquartile range. Categorical data are presented as percentages or frequencies. Student t-test and Mann-Whitney U tests were used to compare parametric and nonparametric continuous variables, respectively. Categorical variables were compared by chi-square (\(\chi^2\)) test. One-way analysis of variance was used to investigate differences among more than 2 groups. Bonferroni analysis was used as post-hoc test. A 2-tailed \(p\) value of <.05 was considered statistically significant. All data were analyzed using SPSS version 23.0 (IBM Corp., Armonk, NY, USA).

RESULTS

The basal demographic and clinical characteristics of the study subjects are presented in Table 1, and the drug history of the patients is presented in Table 2. There was no difference in age, sex, and cardiovascular risk factors among the groups. The groups showed similar laboratory findings and similar drug history other than statins.

The hsTnT values before and after the PCI are shown in Figure 1 and Table 3. The increase in hsTnT values on the 2nd and 4th hour after the procedure were lower in the potent statin group than in other groups (Table 3). The difference among groups continued at the 12th hour but showed no statistical significance (Table 3). The number of patients exceeding the 99th percentile reference limit for 2nd, 4th, and 12th hour hsTnT measurements were also lower in the potent statin group than in the other groups (\(p=0.014, p=0.002,\) and \(p=0.297,\) respectively) (Table 3).

PCI-related complications were identified in 31 patients. These were distal embolization (n=1), coronary dissection (n=1), coronary spasm (n=2), slow coronary flow (n=5), no reflow phenomenon (n=3), side-branch occlusion (n=3) and plaque shift (n=16). There was no significant increase in hsTnT values after the procedure in patients without complications, and no significant difference was found between the statin subgroups in this category (Table 3). In patients with complications, the increase in hsTnT levels at the 2nd, 4th, and 12th hours were significantly lower in the potent statin subgroup than in the other groups (Table 3).

In patients with complications, there were no significant differences between the statin and control groups in terms of the proportion of the patients exceeding the 99th percentile hsTnT reference limit.
DISCUSSION

In this study, we investigated the effects of long-term statin usage on myocardial injury related to PCI assessed by hsTnT. The results of the study showed that hsTnT elevation in the initial hours after elective PCI was less frequent in patients using potent statins. This result was more obvious in patients with complicated procedures.

During PCI procedures, the rate of occurrence of myocardial injury, its possible related causes, effects on patient prognosis, and methods of prevention have been investigated in several studies. Regardless of the causes, adverse effects of myocardial injury on patients’
Figure 1. Levels of hsTnT at different hours after PCI are demonstrated for the potent statin, weak statin, and statin free groups. The hsTnT values on the 2nd and 4th hours after PCI were lower in the potent statin group than in the other groups. In patients with procedural complications, hsTnT levels at the 2nd, 4th, and 12th hour after PCI were more significantly lower in the potent statin group. No significant increase was found in hsTnT levels in patients without procedural complications.

Table 2. Drug history of the participants

| Parameters | Patients without procedural complications | Patients with procedural complications |
|------------|------------------------------------------|-------------------------------------|
|            | Statin free (n=36) | Weak statin (n=16) | Potent statin (n=19) | Total (n=71) | p | Statin free (n=17) | Weak statin (n=7) | Potent statin (n=7) | Total (n=31) | p | Total (n=102) | p |
| Antiaggregants | 14 (38) | 10 (62) | 16 (84) | 40 (56) | 0.013 | 8 (47) | 4 (57) | 4 (57) | 16 (51) | 0.846 | 56 (54) | 0.549 |
| Aspirin | 13 (36) | 9 (56) | 11 (57) | 33 (46) | 0.205 | 7 (41) | 4 (57) | 4 (57) | 15 (48) | 0.676 | 48 (47) | 0.514 |
| Clopidogrel | 1 (3) | 1 (6) | 3 (15) | 5 (7) | 0.198 | 0 (0) | 0 (0) | 0 (0) | 0 (0) | - | 5 (5) | 0.156 |
| Dual antiaggregants | 0 (0) | 0 (0) | 2 (11) | 2 (3) | 0.060 | 1 (5) | 0 (0) | 0 (0) | 1 (3) | 0.658 | 3 (2.5) | 0.667 |
| ACEI or ARB | 21 (58) | 9 (56) | 12 (63) | 42 (59) | 0.908 | 10 (58) | 5 (71) | 3 (42) | 18 (58) | 0.554 | 60 (58) | 0.544 |
| Beta-blockers | 7 (19) | 6 (37) | 11 (58) | 24 (33) | 0.015 | 5 (29) | 2 (28) | 4 (57) | 11 (35) | 0.396 | 35 (34) | 0.521 |
| Calcium channel blockers | 4 (11) | 3 (18) | 2 (12) | 9 (12) | 0.707 | 1 (5) | 0 (0) | 0 (0) | 1 (3) | 0.653 | 10 (9.5) | 0.130 |
| Statins | 0 (0) | 16 (100) | 19 (100) | 35 (49) | <0.001 | 0 (0) | 7 (100) | 7 (100) | 14 (45) | <0.001 | 49 (48) | 0.433 |
| Atorvastatin | 0 (0) | 12 (75) | 13 (68) | 25 (35) | - | 0 (0) | 6 (85) | 3 (42) | 9 (29) | - | 34 (33) | - |
| Rosuvastatin | 0 (%0) | 4 (25) | 6 (32) | 10 (14) | - | 0 (0) | 1 (14) | 4 (57) | 5 (16) | - | 15 (14) | - |

ACEI: angiotensin converting enzyme inhibitor; ARB: angiotensin receptor blocker.
Table 3. Comparison of hsTnT (ng/L) values. The hsTnT 99th percentile upper reference limit exceedance rates (%) and hsTnT myocardial infarction limit exceedance rates (%) of patients with different statin usage protocols at different hours after the procedure

| Hours | Groups                  | hsTnT     | Statin free (n=53) | Weak statin (n=23) | Potent statin (n=26) | p     |
|-------|-------------------------|-----------|--------------------|--------------------|----------------------|-------|
|       |                         |          | Level (ng/L)       | Level (ng/L)       | Level (ng/L)         |       |
|       |                         |          | >99% URL           | >99% URL           | >99% URL             |       |
|       |                         |          | > MI limit          | > MI limit          | > MI limit           |       |
|       | No complications        |          | 0 (0%)             | 0 (0%)             | 0 (0%)               | 0.114 |
|       | Complications           |          | 0 (0%)             | 0 (0%)             | 0 (0%)               | 0.157 |
|       | Overall                 |          | 0 (0%)             | 0 (0%)             | 0 (0%)               | 0.096 |
| 0 hour|                         |          | 9.1 (5.6-13.2)     | 11.1 (8.1-13.6)    | 6.6 (5-11.9)         | 0.003 |
| 2nd hour| No complications     |          | 13.4 (8.3-19.8)    | 13.3 (7.7-26.7)    | 6.9 (5-13.4)         | 0.018 |
|       | Complications           |          | 12 (70.5)          | 6 (85.7)           | 3 (42.8)             | 0.214 |
|       | Overall                 |          | 17 (9-72)          | 15 (10-57)         | 9 (5-14)             | 0.008 |
| 4th hour| No complications      |          | 16.1 (11.4-27.3)   | 14.5 (8.3-35.2)    | 9.3 (5-14)           | 0.011 |
|       | Complications           |          | 22 (61.1%)         | 14 (41.1%)         | 4 (21%)              | 0.018 |
|       | Overall                 |          | 28.8 (11.6-114.9)  | 25.3 (9-9-191.8)   | 10.7 (5-2-22)        | 0.009 |
| 12th hour| No complications    |          | 15.1 (10.1-22.8)   | 16.3 (10.8-25.2)   | 13.9 (5.5-35.1)      | 0.042 |
|       | Complications           |          | 20 (55.5%)         | 10 (62.5%)         | 9 (47.3%)            | 0.665 |
|       | Overall                 |          | 31 (12-142)        | 23 (120-67)        | 19 (6-34)            | 0.016 |

hsTnT: high-sensitive troponin T; MI: myocardial infarction; URL: upper reference limit.
morbidity and mortality were demonstrated in clinical trials.\textsuperscript{[5]} Statins are the most used lipid-lowering agents in the treatment and prevention of CAD.\textsuperscript{[8]} Besides its lipid-lowering effects, the pleiotropic effects carry importance as well.\textsuperscript{[9]} There is no clear evidence that statins have a direct cardioprotective effect, but with its lipid-lowering and pleiotropic effects it can be useful in preventing myocardial injury related to PCI.\textsuperscript{[13,15]} Many retrospective studies showed that starting statins before PCI can be effective in reducing the rate and severity of myocardial injury related to PCI.\textsuperscript{[13,15]} The placebo controlled Atorvastatin for Reduction of Myocardial Damage during Angioplasty (ARMYDA) study showed that the circulating levels of myocardial injury markers were significantly lower in patients randomized to 40 mg atorvastatin for 7 days before PCI than in the placebo group.\textsuperscript{[19]} In a recent study, the effects of preoperative statin load in off-pump CABG were investigated. In the study, the patients had similar baseline levels of various cardiac biomarkers (cardiac troponin-I, creatine kinase, and B-type natriuretic peptide); all these biomarkers at 8th, 24th, and 48th hour after CABG were significantly lower in the statin-loaded group supporting the idea of the role of statins in reducing myocardial damage during hemodynamic disturbances and ischemia.\textsuperscript{[20]} As in the results of the previous studies, our study also showed that hsTnT elevation was less frequent after elective PCI in patients using statins. However, previous studies have investigated the effect of initiating statin therapy as a loading dose before the procedure, and limited data are available to assess the effects of long-term statin use on these patients.

Auguadro et al.\textsuperscript{[21]} studied the effects of 3-week statin usage before elective PCI, and their results showed that the patients using statins have lower levels of troponin I and creatine kinase-MB (CK-MB) in blood samples taken after PCI than those who were not using statins. In our study, we included patients who were on statins for a longer period (at least 8 weeks before PCI) to examine the chronic effects of statin use. In the study, high-sensitivity troponin assay was used so that it was possible to show relatively small differences in hsTnT levels between statin users in different doses and statin-free patients. Similar results were observed supporting the idea that long-term statin usage also plays a role in decreasing the rates of myocardial injury related to PCI.

The most significant aspect of our study was evaluating the relationship between the intensity of the statin used and the rates of hsTnT increase during PCI. Previous studies have shown that using more potent statins (in terms of the molecule or dose used) leads to further risk reductions of acute coronary events, revascularization, and ischemic stroke among patients with stable CAD.\textsuperscript{[22]} In our study, we aimed to evaluate the effects of using different doses and molecules of statins on myocardial injury related to PCI, during and after the procedure. In the study, the patients were evaluated in different groups according to the statin used. The increase in hsTnT values after the procedure were lower in the potent statin group than in the other 2 groups (weak statin and statin free), and the rate of myocardial injury in the weak statin group was similar to that of the statin free group. These results suggest that relatively weak statins are not as effective as potent statins in terms of reducing rates and significance of myocardial injury related to PCI.

Another aspect of our study is the effects of statins on myocardial injury in relation to the complications that occurred during PCI. In this subgroup analysis, there were 2 main patient groups (according to the presence or absence of procedural complications) and each group was divided into 3 subgroups according to the statin used, as in the main analysis criteria. Even though the group without procedural complications were slightly older and had a higher percentage of diabetic and hyperlipidemic patients, which was the opposite of the normal expectation, these differences were statistically insignificant. It is noteworthy that the protective effect of statin usage in terms of the increase in hsTnT levels was more prominent in patients with procedural complications, especially in the group receiving potent statins.

Within the group without procedural complications, there were no statistically significant differences in the increase in the rates of hsTnT levels between the subgroups, and there were higher rates of beta-blockers and antiaggregant agents’ use in the potent statin subgroup, which may affected the study results. Despite all the points mentioned, it is worth to notice that the rates of patients exceeding the 99th percentile reference limit of hsTnT at 2nd and 4th hour were lower in the potent statin subgroup than in the other 2 subgroups of patients with no procedural complications.
Limitations

The most important limitation of our study was the small number of patients involved. Another important limitation was the short follow-up period. By monitoring hsTnT levels beyond the 12th hour after the procedure, the impact of statins on decreasing the rates of late myocardial injury could be examined. Finally, there was a lack of clinical follow-up of patients in terms of major cardiovascular events; thus, the relationship between different doses of long-term statin usage and major cardiovascular events in long term was not investigated.

Conclusion

The results of our study showed that myocardial injury related to elective PCI was less frequent among patients using long-term potent statins. This result was more pronounced in the group of patients who developed complications during the procedure. In addition, in these patients low-dose statin usages was not as effective as high-dose usage.

The visual summary of the article can be seen in the Appendix 1.

Ethics Committee Approval: Ethics committee approval was received for this study from the Ethics Committee of Gazi University School of Medicine on (Approval Date: April 13, 2015; Approval Number: 162).

Informed Consent: Written informed consent was obtained from the patients who participated in this study.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept - A.A.; Design - A.K., S.Ü., A.A.; Supervision - A.A.; Resources - A.K., S.Ü., A.A.; Materials - A.K., S.Ü., G.G., N.S.Y., Ö.G., A.A.; Data Collection and/or Processing - A.K., S.Ü., G.G., N.S.Y., Ö.G., A.A.; Analysis and/or Interpretation - A.K., S.Ü., G.G., N.S.Y., Ö.G., A.A.; Literature Search - A.K., S.Ü., A.A.; Writing - A.K., S.Ü., A.A.; Critical Revision - A.K., S.Ü., G.G., N.S.Y., Ö.G.

Funding: This work was supported by the Turkish Society of Cardiology (Grant no.: 2015/2).

Conflict of Interest: None.

REFERENCES

1. Ravkilde J, Nissen H, Mickley H, Andersen PE, Thayssen P, Hørder M. Cardiac troponin T and CK-MB mass release after visually successful percutaneous transluminal coronary angioplasty in stable angina pectoris. Am Heart J 1994;127:13-20. [Crossref]

2. Dash D. Complications of coronary intervention: device embolisation, no-reflow, air embolism. Heart Asia 2013;5:54-8. [Crossref]

3. Klein LW, Kramer BL, Howard E, Lesch M. Incidence and clinical significance of transient creatine kinase elevations and the diagnosis of non-Q wave myocardial infarction associated with coronary angioplasty. J Am Coll Cardiol 1991;17:621-6. [Crossref]

4. Abbas SA, Glazier JJ, Wu AH, Dupont C, Green SF, Pearsall LA, et al. Factors associated with the release of cardiac troponin T following percutaneous transluminal coronary angioplasty. Clin Cardiol 1996;19:782-6. [Crossref]

5. Prasad A, Singh M, Lerman A, Lennon RJ, Holmes DR Jr, Rihal CS. Isolated elevation in troponin T after percutaneous coronary intervention is associated with higher long-term mortality. J Am Coll Cardiol 2006;48:1765-70. [Crossref]

6. Steg G, James SK, Atar D, Badano LP, Lundqvist CB, Borger MA, et al. ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation: The Task Force on the management of ST-segment elevation acute myocardial infarction of the European Society of Cardiology (ESC). Eur Heart J 2012;33:2569-619. [Crossref]

7. Wu AH, Jaffe AS. The clinical need for high-sensitivity cardiac troponin assays for acute coronary syndromes and the role for serial testing. Am Heart J 2008;155:208-14. [Crossref]

8. Stancu C, Sima A. Statins: mechanism of action and effects. J Cell Mol Med 2001;5:378-87. [Crossref]

9. McFarlane SI, Muniyappa R, Francisco R, Sowers JR. Pleiotropic effects of statins: lipid reduction and beyond. J Clin Endocrinol Metab 2002;87:1451-8. [Crossref]

10. Chan AW, Bhatt DL, Chew DP, Quinn MJ, Moliterno DJ, Topol EJ, et al. Early and sustained survival benefit associated with statin therapy at the time of percutaneous coronary intervention. Circulation 2002;105:691-6. [Crossref]

11. Walter DH, Fichtlscherer S, Britten MB, Rosin P, Auck-Schwelk W, Schachinger V, et al. Statin therapy, inflammation and recurrent coronary events in patients following coronary stent implantation. J Am Coll Cardiol 2001;38:2006-12. [Crossref]

12. Gelosa P, Cimino M, Pignieri A, Tremoli E, Guerrini U, Sironi L. The role of HMG-CoA reductase inhibition in endothelial dysfunction and inflammation. Vasc Health Risk Manag 2007;3:567-77. [Crossref]

13. Oesterle A, Laufs U, Liao JK. Pleiotropic effects of statins: A boulevard to cardioprotection. Arab J Chemistr 2016;9(S1):S21-7. [Crossref]
17. Miller WL, Garratt KN, Burritt MF, Lennon RJ, Reeder GS, Jaffe AS. Baseline troponin level: key to understanding the importance of post-PCI troponin elevations. Eur Heart J 2006;27:1061-9. [Crossref]
18. Califf RM, Abdelmeguid AE, Kuntz RE, Popma JJ, Davidson CJ, Cohen EA, et al. Myonecrosis after revascularization procedures. J Am Coll Cardiol 1998;31:241-51. [Crossref]
19. Pasceri V, Patti G, Nusca A, Pristipino C, Richichi G, Di Sciascio G, et al. Randomized trial of atorvastatin for reduction of myocardial damage during coronary intervention: results from the ARMYDA (Atorvastatin for Reduction of Myocardial Damage during Angioplasty) study. Circulation 2004;110:674-8. [Crossref]
20. Kaushik A, Kapoor A, Agarwal SK, Pande S, Tewari P, Majumdar G, et al. Statin reload before off-pump coronary artery bypass graft: effect on biomarker release kinetics. Ann Card Anaesth 2020;23:27-33. [Crossref]
21. Auguadro C, Manfredi M, Scalise F, Mortara A, Specchia G. Protective role of chronic statin therapy in reducing myocardial damage during percutaneous coronary intervention. J Cardiovasc Med 2006;7:416-21. [Crossref]
22. Cholesterol Treatment Trialists’ (CTT) Collaboration; Baigent C, Blackwell L, Emberson J, Holland LE, Reith C, et al. Efficacy and safety of more intensive lowering of LDL cholesterol: a meta-analysis of data from 170 000 participants in 26 randomised trials. Lancet 2010;376:1670-81. [Crossref]

Keywords: Percutaneous coronary intervention; troponin; myocardial injury; statins

Anahtar Kelimeler: Perkütan koroner girişim; troponin; miyokardyal hasar; statinler
Elective PCI related myocardial injury was less frequent in patients who were using high dose statins. This result was more pronounced in patients who developed complications during the procedure.

Koçak A et al. Turk Kardiol Dern Ars. doi: 10.5543/tkda.2021.11292

Appendix 1. Visual summary of the article.