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Effect Of Long-term Treatment By Statin In Near-infrared Spectroscopy

Romain Didier, Smita Negi, Ota Hideaki, Ron Waksman
Medstar, District of Columbia, DC

BACKGROUND Rupture of lipid core plaque (LCP) is central to the pathology of an acute coronary syndrome (ACS). Intracoronary near-infrared spectroscopy (NIRS) has been shown to identify lipid core plaque (LCP) in patients undergoing coronary angiography (CA) in ACS. Studies using NIRS have shown that acute, intensive statin therapy reduces lipid core burden index (LCBI), a surrogate for lipid content in LCP. We aimed to determine if chronic long term statin use affects lipid content of LCP as measured by NIRS.

METHODS Forty-seven consecutive patients undergoing CA and NIRS with intravascular ultrasonography for at least one obstructive lesion were included. Lipid core burden index, LCBI divided by the length of scanned artery LCBI/L and the MaxLCBI 4mm (maximum value of LCBI for any of the 4-mm segment) were obtain from the scan of the three vessel except the left main and stented segment. Baseline patient characteristics and medication use was recorded. The patients receiving long-term treatment by statin before hospitalization (n=28) were compared with those without (n=19).

RESULTS Patients had a mean age of 64.4 years with a prevalence of men (70.2%), diabetic (40.4%), hypercholesterolemia (89%) and smoking history (10%). In the group receiving statin, 10 patients had Atorvastatin (10 to 80 mg), 10 Rosuvastatin (5 to 40 mg) and 8 Simvastatin (10 to 40 mg). No significant difference was found for all NIRS's parameters in the both groups.

	Group statin (n=28)	Group without statin (n=19)	P
LCBI (average)	116.1	113.63	0.46
LCBI/L(average)	3.4	2.31	0.19
MaxLCBI 4mm (average)	397.46	443.63	0.31

CONCLUSION Although the intensive statin therapy decreases the LCBI, the presence of statin with usual doses before performing NIRS does not appear to predict the result of LCBI. The control by spectroscopy in the same patient on the occasion of new angiography after statin dose optimization should be a good way for assesses its risk of ACS.

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Comparisons of Diltiazem alone versus Diltiazem with Nitrate Treatment on 5-year Clinical Outcomes in Patients with Significant Coronary Artery Spasm; A Propensity Score Matching Study

Byoung Geol Choi,¹ Seung-Woon Rha,² Se Yeon Choi,² Ji Young Park,³ Sang-Ho Park,⁴ Woong Gil Choi,⁵ Soo Hyun Kim,⁶ Eun-Gyu Lee,⁷ Jihun Ahn,⁸ Sang Yeub Lee,⁹ Sang Min Kim,⁹ Min Woong Kim,¹⁰ Seong Gyu Yoon,¹ Tae Hoon Ahn,¹¹ Dong Joo Oh²
¹Department of Medicine, Korea University Graduate School, Seoul, Korea, Republic of; ²Cardiovascular Center, Korea University Guro Hospital, Seoul, Korea, Republic of; ³Cardiology Department, Eulji General Hospital, Seoul, Korea, Republic of; ⁴Cardiology Department, Soonchunhyang University Cheonan Hospital, Cheonan, Korea, Republic of; ⁵Division of Cardiology, Konkuk University Chungju Hospital, Chungju, Korea, Republic of; ⁶Division of Cardiology, Konkuk University Chungju Hospital, chungju, Korea, Republic of; ⁷Cardiovascular Center, Andong Sungso Hospital, Andong, Korea, Republic of; ⁸Department of Internal Medicine, Soonchunhyang University Gumi Hospital, Gumi, Korea, Republic of; ⁹Cardiovascular Center, Chungbuk National University Hospital, Chungbuk, Korea, Republic of; ¹⁰Department of Cardiology, Hanyang University Medical Center, Hanmaeum Hospital, Changwon, Korea, Republic of; ¹¹Gachon University of Medicine and Science, Gil Hospital, Incheon, Korea, Republic of

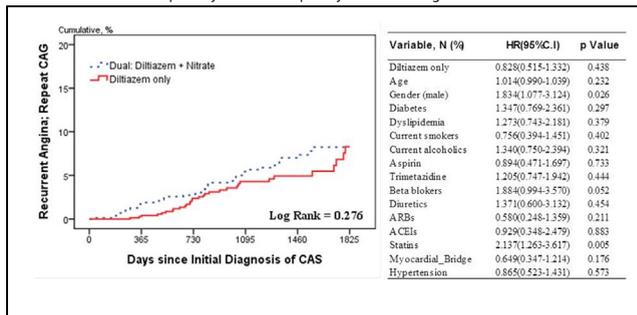
BACKGROUND It has been well known that a major cause of vasospastic angina is endothelial dysfunction and subsequent significant coronary vasoconstriction. Thus, diltiazem and nitrate can be beneficial as selective coronary vasodilators for pts with significant vasoconstriction. However, there is no study comparing the efficacy of diltiazem alone versus diltiazem with nitrate treatment for long-term clinical outcomes in vasospastic angina pts.

METHODS A total of 3,360 consecutive pts without significant coronary artery disease (CAD) underwent acetylcholine (Ach) provocation test between Nov. 2004 and May. 2014 and positive coronary artery spasm (CAS) pts were enrolled. Significant CAS was defined as > 70% of narrowing by incremental intracoronary injection of 20, 50 and 100 µg into left coronary artery. Pts were divided into two groups: the diltiazem group (n=842), the dual group (diltiazem+nitrate, n=1,899). To adjust potential confounders, a propensity score matched (PSM) analysis was performed using the logistic regression model.

RESULTS After PSM analysis, 2 propensity-matched groups (811 pairs, n = 1,622, C-statistic=0.708) were generated and the baseline characteristics of the two groups were balanced. At 5 years, there were similar incidence of individual hard endpoints including mortality, myocardial infarction, revascularization and recurrent angina requiring repeat coronary angiography between the two groups (Table and Figure).

CONCLUSIONS Despite the expected improvement of endothelial function and relieving vasoconstriction, combination of diltiazem and nitrate treatment were not superior to diltiazem alone in reducing clinical events and ischemic symptoms up to 5-year.

Table. Clinical Outcomes up to 5-years after Propensity Score Matching



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Incidence and Predictors Of Bleeding And Stent Thrombosis With Bivalirudin During Percutaneous Coronary Intervention

Tilak Pasala, Rama Dilip Gajulapalli
Case Western Reserve University/MetroHealth, Cleveland, OH

BACKGROUND Bivalirudin is a synthetic reversible inhibitor of thrombin and is popularly used in the United States in patients undergoing percutaneous coronary intervention (PCI). While the evidence shows a bleeding benefit with bivalirudin compared to heparin, there are concerns raised regarding the risk of peri-procedural stent thrombosis. We aimed to quantify and analyze the incidence and predictors of the risk of bleeding and stent thrombosis with bivalirudin.

METHODS We searched PubMed, the Cochrane library, and major meeting and journal abstracts for studies that reported events with bivalirudin use during PCI. Pooled event rate (ER) and 95% confidence intervals (CI) were calculated with random-effects for up to 30-day bleeding events and stent thrombosis.

RESULTS We identified 70 studies (n = 71,299) that used bivalirudin as the anticoagulant of choice for PCI. Major bleeding events were noted in 1053 of 55636 (1.9%) and minor bleeding were noted in 1104 of 24969 (4.4%) patients. Majority of bleeding events were due to use of transfusion (1.6%) and access site bleeding (1.1%). Intracranial and retroperitoneal bleeding were rare (<0.1%). Definite stent thrombosis occurred in 266 of 29875 (0.9%) patients, of which majority were acute stent thrombosis. Pooled event rates for bleeding and stent thrombosis are reported in the figure. Majority of the studies did not report the acuity of stent thrombosis limiting the analysis. On meta-regression analysis, the use of drug eluting stents (DES) was associated with a lower risk of stent thrombosis (t = -2.44, p = 0.022).

CONCLUSIONS Bivalirudin is associated with a 2% risk of major and a 4.4% risk of minor bleeding, majority of which are transfusion and access-site related. The risk of stent thrombosis is around 0.9% and the use of DES decreases this risk.

Outcome	Event Rate (95% CI)
Bleeding	
Major	0.018 (0.015, 0.021)
Minor	0.044 (0.035, 0.053)
Access site	0.011 (0.008, 0.014)
Intracranial	0.000 (0.000, 0.001)
Retroperitoneal	0.001 (0.001, 0.002)
Transfusion	0.017 (0.013, 0.021)
Definite stent thrombosis	
Overall	0.008 (0.006, 0.010)
Acute	0.010 (0.005, 0.015)
Subacute	0.006 (0.003, 0.009)