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SIDE BRANCH PLAQUE SHIFT IN BIFURCATION LESIONS IN CORONARY ARTERIES AND LOW DENSITY LIPOPROTEIN AS ITS MARKER

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Contribution
BO conceived the idea, designed the study, collected data, and manuscript writing was done by BO.

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ABSTRACT

Objective: In this study, the effect of lipid profile on plaque shift was investigated considering the known effect of atherosclerosis.

Methodology: A total of 660 patients, 457 males and 203 females, participated in the study. Average age is 60.3 ± 12 years. Two study groups included this study one was no plaque shift, the other one was plaque shift group.

Results: In laboratory parameters of all groups triglyceride mean 164.7± 132.3 mg/dl, HDL mean 40± 11.1 mg/dl and LDL mean 129.1± 43.3 mg/dl were calculated. In lipid profile TG and HDL has no differences in two groups. Mean of HDL 40.6±11.2 mg/dl in no plaque shift group and 39.4±11.0 mg/dl in plaque shift group. And also mean of TG 167.8±145.5 mg/dl and 161.1±115.6 mg/dl consecutively. LDL mean was 123.9±43 mg/dl in no plaque shift group, 135.1±42.9 mg/dl in plaque shift group. Only high LDL levels correlated with plaque shift degree, plaque shift length and shift lesion percentage difference before and after the procedure.

Conclusion: There is a correlation between LDL levels and plaque shift in coronary bifurcation lesions due to the volume of lipid core in plaque formation. The benefit is to predict the plaque shift that may occur in coronary bifurcation interventions with an easily measured parameter before the procedure.

Keywords: LDL, plaque shift, bifurcation lesions, lipid profile, side branch occlusion

INTRODUCTION

Coronary heart diseases are the most common group of diseases worldwide and atherosclerosis is usually seen as cause. Atherosclerosis is an inflammatory disease, starts in the early stages of life, appears in the artery wall with lipid, fibrin and cell accumulation. The disease causes narrowing in the artery lumen with the accumulation of plaques. Many risk factors have been identified for atherosclerosis. Just as dyslipidemia is important for the disease, under certain conditions, despite lower levels of low-density lipoprotein (LDL) levels, the disease may appear due to other factors.[1,2] Prevention of atherosclerosis resulting from many factors can be foreseen by treating risk factors; listed as antihypertensive treatments, antilipidemic treatments, smoking cessation, lifestyle change. Lipoprotein, inflammatory cell, foam cell accumulation, smooth muscle cell proliferation, calcification play a role in the pathophysiology of the disease.

One of the most important factors for atherosclerosis is high LDL levels. LDL accumulates oxidized to the subendothelial region through the artery wall. Monocytes infiltrate the artery wall from the plasma and turn into macrophages. Macrophages phagocyte oxidized LDL and turn into foam cells. It contributes to the inflammatory process in the vascular wall.[3] Plaque is generally composed of necrotic nucleus, foam cells, smooth muscle cells and fibrous cap. One of the first studies showing the relationship between cholesterol and atherosclerosis was done by Ignatowski.[4] LDL is oxidized and aggregates to the arterial wall.[5] Oxidized LDL stimulates chemoattractant synthesis for immune system cells. Thus, monocyte and macrophage migration is stimulated.[6,7]

Pathogenesis of atherosclerosis was investigated by microscopic evaluation of lesions of different ages. One of the most important research on this subject was done by Stary et al.[8-10] With this study, American Heart Association classification, types I–VIII of atherosclerosis was defined.[11] Virmani et al. made a simple classification. In this classification, lesion morphology and disease were correlated.[12] Atherosclerosis is common in bifurcation areas due to shear stress. 15% -20% of all coronary diseases are bifurcation lesions.[13]

Percutaneous coronary intervention is more difficult and complex in bifurcation lesions. As a result, the process success rate decreases and the restenosis rate increases.[14] In studies performed, long-term success of single stenting technique was better than two-stent strategy in bifurcation lesions.[15] Since procedural stenting is a less complex procedure, the processing time is shorter, the contrast agent used is less, the amount of radiation is less.

The major disadvantage of the provisional stenting technique is the loss of the side branch, its incidence is 15% to 41%. Plaque and carina shift results side branch loss.[16,17] Bifurcation lesions, stenting techniques and plaque shift have been investigated. Plaque shift has many defined determinants listed as major branch stent type, major branch distal lesion, initial TFC (TIMI frame count) value, alpha angle, over-dilatation of stent and side branch lesion before treatment.

Estimating the loss of side branches results in the use of side branch protection methods (balloon protection or elective two-stent strategy) and the success of the procedure. All these studies have only one purpose, to determine plaque shift to side branches before the procedure and to choose a suitable technique accordingly. In this study, the effect of lipid profile on plaque shift was investigated considering the known effect of atherosclerosis.

METHODOLOGY

In this study patient were selected retrospectively from bifurcation lessions treated with provisional technique between 2014 and 2015 in Adana Numune Training and Research Hospital. A total of 660 patients, 457 males and 203 females, participated in the study. Average age is 60.3 ± 12 years. Patients with a two-stent strategy with a side branch diameter below 2mm and a history of CABG (coronary artery bypass grafting) were excluded. None of our patients had a history of antilipidemic drug before the procedure, all of them were new diagnosis. Approved by the local ethics committee. Patients undergoing provisional stenting to the bifurcation lesion were selected. Coronary angiography was performed with the standard

protocol. Those with more than 70% lesions were considered critical.

Angiographic procedures were performed by experienced invasive cardiologists. Coronary angiography CDs and operation reports were examined and two groups were determined. One is plaque shift group (308) and the other one is no plaque shift (352). Side branch before and after intervention lession amount, lesion percentage difference before and after the procedure, degree of shift, length of the shift plaque and laboratory parameters LDL, HDL (high density lipoprotein) and TG (triglycerides) recorded. Degree of shift 0 to 3; 0 means no plaque shift, 1 minimal, 2 middle and 3 serious plaque shift. Degree of shift was determined with TIMI in side branch after procedure, before-after procedure lesions percentage and length of plaque shift (Table 1).

Table 1: Plaque shift degree

Plaque shift degree	TIMI	Before-after procedure lesions percentage (%)	Length of plaque shift (mm)
0	3	0	0
1	3	0-70	3mm>
2	3	70-90	3-5mm
3	0-1-2-3	90-100	5mm<

SPSS 22.0 (SPSS, Inc., Chicago, IL, USA) was used for statistical analysis and P <0.05 values were considered statistically significant. Independent sample t-test was used.

RESULTS

A total of 660 patients, 457 males and 203 females, participated in the study. Average age is 60.3 ± 12 years. In laboratory parameters of all groups triglyceride mean 164.7 ± 132.3 mg/dl, HDL mean 40 ± 11.1 mg/dl and LDL mean 129.1 ± 43.3 mg/dl were calculated. Diagnosis distribution of the patients is as follows; 146 (22.1%) chronic coronary diseases, 120 (18.2%) unstable angina pectoris (USAP), 206 (31%) acute anterior myocardial infarction, 59 (8.9%) acute inferior myocardial infarction.

The initial SYNTAX score average was calculated as 13.9± 7.5. When categorized according to Medina classification 0.1.0. 221 (33.5%), 0.11. 11 (1.7%), 1.1.1. 27 (4.1%), 1.01. 5 (0.8%), 1.1.0. 317 (48%), 1.0.0. 79(12%). 352 (53.3%) not occured plaque shift (no plaque shift group), 308(46.7%) were occured plaque shift (plaque shift group). In plaque shift group 90 (13.6%) minimal, 58 (8.8%) medium, 160 (24.2%) serious plaque shift were recorded. In plaque shift group age mean 59.8±11.6 and in no plaque shift group age mean was 60.8±12.3. There were no differences. And mean of syntax score before intervention consecutively 14.1±7.9, 13.8±7, no differences seen, shown in Table 2.

Table 2: Plaque shift group and no plaque shift group comparison

	Plaque shift	N	Mean±Std. Deviation	P-value
Age (years)	-	352	59.8±11.6	0.294
	+	308	60.8±12.3	
SYNTAX score before intervention	-	352	14.1±7.9	0.552
	+	308	13.8±7.0	
LDL (mg/dl)	-	352	123.9±43	0.001
	+	308	135.1±42.9	
HDL (mg/dl)	-	352	40.6±11.2	0.21
	+	308	39.4±11.0	
TG (mg/dl)	-	352	167.8±145.5	0.53
	+	308	161.1±115.6	

Lipid profile TG and HDL had no differences in two groups. Mean of HDL 40.6±11.2 mg/dl in no plaque shift group and 39.4±11.0 mg/dl in plaque shift group. And also mean of TG 167.8±145.5 mg/dl and 161.1±115.6 mg/dl consecutively. And there no differences in both groups, shown in Table 2. LDL mean was 123.9±43 mg/dl in no plaque shift group, 135.1±42.9 mg/dl in plaque shift group. LDL levels correlated with plaque shift in groups. Increasing in LDL levels resulted to plaque shift in bifurcation lessions shown in Table 2. High LDL levels correlate with plaque shift degree (r=0.200), plaque length (r=0.195) and lesion percentage difference before and after the procedure (r=0.268).

DISCUSSION

In our study no plaque shift and plaque shift groups, we observe that LDL levels effects plaque shift occurence. Plaque shift levels, plaque shift degree, lession percentage difference before and after the procedure with LDL levels correlated. As known, the LDL level is important for plaque formation in atherosclerosis.[4] In our study, we determined the

level of LDL as an undefined side-branch loss determinant.

The main determinants of plaque shift were investigated and no studies were found with LDL levels. In our study, we found a correlation between LDL levels and plaque shift. In the light of our findings, in patients undergoing bifurcation, the prediction of plaque shift may be increased according to LDL levels. In the bifurcation lesion stenting, the provisional technique is recommended as the first choice by the European Bifurcation Club. In the European Society of Cardiology (ESC) 2014 myocardial revascularization guideline, the provisional stent technique for bifurcation lesions and a side branch lesion> 3 mm in length was proposed as a class IIa. Plaque shift parameters have been determined in studies about bifurcation lesions in coronary artery, listed as plaque shift, carina shift, spasm, and dissection.[18-21]

Studies have been carried out to determine the causes of side branch loss. In the COBIS II (COronary BIfurcation Stenting) study, carina angle (alpha) was an important factor with a loss of side branch. In another study, the angle of the carina is a valuable factor for the loss of side branches.[22] Ghayemian et al. found that the main branch proximal lesion rate was found to be related to the loss of the side branch.[23] In a study conducted by Aliabadi and colleagues,> 50% lesion presence increased side branch loss in side branch.[24] Kralev and colleagues have shown that side branch osteal lesion presence in patients with ST-elevation patients increases the need for side branch intervention.[21]

Another consequence of the IVUS study of Jianqiang Xu et al. was found to be related to the need for atmospheric side branch intervention from the distal main stent.[25] In fact, all these studies have only one purpose, to predict the loss of side branches that cause mortality and morbidity before the procedure and to choose the most appropriate procedure technique accordingly. In our study, LDL level, which is a risk for atherosclerosis and should be screened routinely in patients, was determined as a predictive marker for side branch loss.

Limitations of our study plaque formation characteristics and atherosclerosis pathology had not be defined by an intravascular technique. Maybe in future studies intravascular ultrasound, and multidetector computed tomography angiography can be use to identify plaque characteristic such as lipid core and calcification.

CONCLUSION

There is a correlation between LDL levels and plaque shift in coronary bifurcation lesions due to the volume of lipid core in plaque formation. The benefit is to predict the plaque shift that may occur in coronary bifurcation interventions with an easily measured parameter before the procedure. Thus, we can anticipate the complications that may occur during the procedure.

REFERENCES

1. Yusuf S, Hawken S, Ounpuu S, Dans T, Avezum A, Lanas F, et al. INTERHEART Study Investigators. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. Lancet. 2004;364(9438):937-52.
2. Lim SS, Vos T, Flaxman AD, Danaei G, Shibuya K, Adair-Rohani H, et al. A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990-2010: a systematic analysis for the Global Burden of Disease Study 2010. Lancet. 2012;380(9859):2224-60.
3. Sakakura K, Nakano M, Otsuka F, Ladich E, Kolodgie FD, Virmani R. Pathophysiology of atherosclerosis plaque progression. Heart Lung Circ. 2013;22(6):399-411.
4. Konstantinov IE, Jankovic GM. Alexander I. Ignatowski: a pioneer in the study of atherosclerosis. Tex Heart Inst J. 2013;40(3):246-49.
5. Steinberg D, Witztum JL. Oxidized low-density lipoprotein and atherosclerosis. Arterioscler Thromb Vasc Biol. 2010;30(12):2311-16.
6. Libby P, Ridker PM, Hansson GK. Progress and challenges in translating the biology of atherosclerosis. Nature. 2011;473(7347):317-25.
7. Subramanian M, Tabas I. Dendritic cells in atherosclerosis. Semin Immunopathol. 2014;36(1):93-102.
8. Stary HC, Blankenhorn DH, Chandler AB, Glagov S, Insull W Jr, Richardson M, et al. A definition of the intima of human arteries and of its atherosclerosis-prone regions. A report from the Committee on Vascular Lesions of the Council on Arteriosclerosis, American Heart Association. Arterioscler Thromb. 1992;12(1):120-34.
9. Stary HC, Chandler AB, Glagov S, Guyton JR, Insull W Jr, Rosenfeld ME, et al. A definition of initial, fatty streak, and intermediate lesions of atherosclerosis. A report from the Committee on

Vascular Lesions of the Council on Arteriosclerosis, American Heart Association. Circulation. 1994;89(5):2462-78.
10. Stary HC, Chandler AB, Dinsmore RE, Fuster V, Glagov S, Insull W Jr, et al. A definition of advanced types of atherosclerotic lesions and a histological classification of atherosclerosis. A report from the Committee on Vascular Lesions of the Council on Arteriosclerosis, American Heart Association. Arterioscler Thromb Vasc Biol. 1995;15(9):1512-31.
11. Stary HC. Natural history and histological classification of atherosclerotic lesions: an update. Arterioscler Thromb Vasc Biol. 2000;20(5):1177-78.
12. Virmani R, Kolodgie FD, Burke AP, Farb A, Schwartz SM. Lessons from sudden coronary death: a comprehensive morphological classification scheme for atherosclerotic lesions. Arterioscler Thromb Vasc Biol. 2000;20(5):1262-75.
13. Takebayashi H, Haruta S, Kohno H, Ichinose H, Taniguchi M, Shimakura T, Ohe T, et al. Immediate and 3-month follow-up outcome after cutting balloon angioplasty for bifurcation lesions. J Interv Cardiol. 2004;17(1):1-7.
14. Yamashita T, Nishida T, Adamian MG, Briguori C, Vaghetti M, Corvaja N, et al. Bifurcation lesions: two-stents versus one stent--immediate and follow-up results. J Am Coll Cardiol. 2000;35(5):1145-51.
15. Steigen TK, Maeng M, Wiseth R, Erglis A, Kumsars I, Narbute I, et al. Randomized study on simple versus complex stenting of coronary artery bifurcation lesions: the Nordic bifurcation study. Circulation. 2006;114(18):1955-61.
16. Cervinka P, Foley DP, Sabaté M, Costa MA, Serrano P, Ligthart JM, et al. Coronary bifurcation stenting using dedicated bifurcation stents. Catheter Cardiovasc Interv. 2000;49(1):105-11.
17. Timurkaynak T, Ciftci H, Ozdemir M, Cengel A, Tavil Y, Kaya M, et al. Sidebranch occlusion after coronary stenting with or without balloon predilation: direct versus conventional stenting. J Invasive Cardiol. 2002;14(9):497-501.
18. Assali AR, Vaknin-Assa H, Lev E, Teplitsky I, Dvir D, Brosh D, et al. Drug eluting stenting in bifurcation coronary lesions long-term results applying a systematic treatment strategy. Catheter Cardiovasc Interv. 2012;79(4):615-22.
19. Louvard Y, Lefèvre T, Morice MC. Percutaneous coronary intervention for bifurcation coronary disease. Heart. 2004;90(6):713-22.
20. Poerner TC, Kralev S, Voelker W, Sueselbeck T, Latsch A, Pfleger S, et al. Natural history of small and medium-sized side branches after coronary stent implantation. Am Heart J. 2002;143(4):627-35.
21. Kralev S, Poerner TC, Basorth D, Lang S, Wolpert C, Haghi D, et al. Side branch occlusion after coronary stent implantation in patients presenting with ST-elevation myocardial infarction: clinical impact and angiographic predictors. Am Heart J. 2006;151(1):153-57.
22. Vassilev D, Gil R. Clinical verification of a theory for predicting side branch stenosis after main vessel stenting in coronary bifurcation lesions. J Interv Cardiol. 2008;21(6):493-503.
23. Ghayemian A, Nozari Y, Safir M, Mohammadpour RA. Predictors of side branch occlusion and its early complications after angioplasty in bifurcation lesions. Pak J Med Sci. 2008; 24: 430-35.
24. Aliabadi D, Tilli FV, Bowers TR, Benzuly KH, Safian RD, Goldstein JA, et al. Incidence and angiographic predictors of side branch occlusion following high-pressure intracoronary stenting. Am J Cardiol. 1997;80(8):994-97.
25. Xu J, Hahn JY, Song YB, Choi SH, Choi JH, Lu C, et al. Carina shift versus plaque shift for aggravation of side branch ostial stenosis in bifurcation lesions: volumetric intravascular ultrasound analysis of both branches. Circ Cardiovasc Interv. 2012;5(5):657-62.