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Original Article

Statin Therapy is not Associated with Improved Overall Survival after Transcatheter Aortic Valve Replacement (TAVR)

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ABSTRACT

Background: Previous studies have indicated that there may be a survival advantage for patients on statins after surgical or transcatheter aortic valve replacement (TAVR). However, the role of adjunct statin following TAVR remains ill-defined. In this study, we aim define the effect that statin have on outcomes after TAVR, particularly in regard to statin dose intensity. **Methods and Results:** A retrospective chart review case-control study of 342 consecutive patients who underwent a TAVR for severe aortic stenosis was performed to determine the effect statin use, stratified by intensity of lipid-lowering effect, on outcomes after TAVR. In a well match patient cohort, the use of statin of any intensity was not associated with any significant difference in overall survival at 1 month (p=.635), 6 months (p=.814), 1 year (p = .845) or 2 years (p = .926). However, the use of statin did decrease in the incidence of stroke or TIA in the in-hospital and discharge to 30 days post-TAVR time periods (p = .001 and .004, respectively). **Conclusion:** This study gives evidence to suggest that there is no association between statins of any lipid lower intensity and improved overall survival. However, a statistical significant trend towards a reduction in the incidence of perioperative stroke or TIA was observed.

Key words: HMG Co-A reductase inhibitor, TAVR, Stroke, TIA, Statin.

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INTRODUCTION

HMG-CoA reductase inhibitors have been showed to be effective for primary and secondary prevention of cardiovascular and cerebrovascular events in a wide range of patients.[1-3] High intensity statins appear to be of particular benefit in patients with established cardiovascular disease.[4] However, whether or not the benefits of statin can be realized in patients with symptomatic aortic stenosis remains unclear. Previous studies have not established any a mortality benefit of statin in patients with non-corrected aortic stenosis.[5] Conversely, patients undergoing surgical aortic valve replacement, there is evidence to suggest a mortality benefit after bioprosthetic valve replacement, which may be as high as a 42% reduction in mortality.[6]

More recently, advances in transcatheter aortic valve replacement (TAVR) has made surgical aortic valve replacement unnecessary for many non-low risk surgical candidates.[7] This new era in the treatment of aortic stenosis has raised questions about the possible benefit of statin in this traditionally higher risk surgical group with aortic stenosis treated with the minimally invasive TAVR. Thus far, only one study on the effect on HMG-CoA reductase inhibitors on outcomes in patients after TAVR has been published. In 2017, Hudred *et al.* published a retrospective study of TAVR patients, which showed a high-intensity statin therapy was associated with a 64% reduction in all-cause mortality at 2 years.[8] In this study, we attempt to reaffirm the findings of Hudred *et al.* and further define the mechanism by which statin therapy may confer a survival advantage post-TAVR.

MATERIALS AND METHODS

A single institution case-control study was conducted using data from a large tertiary care referral center. We performed a retrospective chart review of 342 consecutive patients who underwent a transcatheter aortic valve replacement (TAVR) at Sanford Health in Fargo, ND from 8/10/2012 to 11/15/2016 for severe aortic stenosis, defined as an aortic valve area less than 1 cm^2. The last date of data acquisition was 1/4/2017. The study population was divided in cohorts based on statin usage and intensity of statin therapy at the time of TAVR. Patients not on a HMG-CoA reductase inhibitors were designated as controls. Statin intensity cohorts were defined using the ACC/AHA guidelines.[9] A cohort of statin users on any dose of statin was also compared. Primary outcomes were overall survival at one month, six months, one year, and two years post-TAVR. Secondary outcomes were procedural complications, major adverse cardiovascular and cerebrovascular events (MACCE) defined as death from any cause, myocardial infarction, rehospitalization, or stroke, cardiovascular mortality, myocardial infarction, stroke/transistent ischemia attack (TIA), heart failure (HF) exacerbation, or rehospitalization for any reason in defined time periods. Pre and postprocedural echocardiographic data was also compared. The clinical outcomes were assessed in accordance with the standardized endpoint definitions for TAVR of the Valve Academic Research Consortium-2.[10] Heart failure exacerbation was defined as a gradual or rapid change in heart failure signs and symptoms resulting in a need for a change in therapy or hospitalization.

Informed consent was not required for inclusion in our retrospective study due to the nature of the study, and the absence of any direct interventions. This study protocol received dual IRB approval from the University of North Dakota IRB and from the Sanford Health IRB. The Fisher's exact test was performed to determine statistical significance of categorical data and ANOVA was used to determine the statistical significance of continuous variables. All p-values were two-sided, and p-values < 0.05 were considered significant.

RESULTS

A total of 342 patients were included in the analysis. The baseline characterizes of all cohorts are given in Table 1. Statistically significant differences were noted between the cohorts in terms of sex, preprocedural coronary artery disease (CAD), preprocedural carotid artery disease, history of coronary artery bypass graft (CABG) prior to TAVR, and aspirin use. The lowest proportion of male subjects was seen in the non-statin cohort. The cohort size of the moderate and low intensity statin groups were similar. The baseline and one year post-TAVR echocardiographic data for all cohorts is given in Table 2. There were no signi-

Table 1: Baseline Characteristics.

	Any Statin (n = 243)	High Intensity (n = 50)	Moderate Intensity (n = 106)	Low Intensity (n = 87)	No Statin (n = 99)	P-value
Age	79.4 (8.4)	77.3 (8.8)	78.8 (8.5)	81.3 (7.6)	79.3 (10.2)	.066
Male sex	145 (59)	36 (72)	65 (61)	45 (52)	46 (46)	.019
BMI	30.3 (6.1)	30.9 (5.9)	30.2 (6.1)	30.2 (6.4)	30.6 (6.2)	.952
Caucasian race	241 (99)	50 (100)	105 (99)	86 (99)	98 (99)	.918
EuroSCORE (%)	9.2 (6.5)	8.9 (6.3)	8.7 (5.5)	10.0 (7.6)	7.3 (5.9)	.055
STS Risk Score (%)	7.0 (4.0)	6.2 (3.1)	7.0 (4.1)	7.5 (4.2)	6.2 (4.4)	.265
Preprocedural Hypertension	220 (91)	47 (94)	95 (90)	78 (90)	81 (82)	.132
Preprocedural Coronary Artery Disease	194 (80)	44 (88)	92 (87)	58 (67)	57 (58)	<.001
Baseline Ejection Fraction <40%	36 (15)	8 (16)	15 (14)	13 (15)	13 (13)	.991
Preprocedural New York Heart Association (NYHA) Class III OR IV Symptoms	102 (42)	19 (38)	44 (42)	39 (45)	50 (51)	.549
Preprocedural Diabetes Mellitus	96 (40)	25 (50)	41 (39)	30 (34)	26 (26)	.050
Prior Stroke/Transient Ischemia Attack (TIA)	32 (13)	10 (20)	12 (11)	10 (11)	7 (7)	.182
Preprocedural Atrial Fibrillation	77 (32)	15 (30)	35 (33)	27 (31)	27 (27)	.921
Preprocedural Chronic Obstructive Lung Disease	47 (19)	13 (26)	21 (20)	13 (15)	26 (26)	.306
Preprocedural Serum Creatinine (mg/dL)	1.2 (0.8)	1.3 (1.0)	1.2 (0.4)	1.3 (1.0)	1.3 (1.0)	.892
Preprocedural Estimated Glomerular Filtration Rate < 60 mL/min	115 (47)	21 (42)	50 (47)	44 (51)	49 (49)	.895
Preprocedural Abdominal Aortic Aneurysm	31 (13)	6 (12)	15 (14)	10 (11)	7 (7)	.572
Preprocedural Carotid Artery Stenosis >50% or Prior Carotid Endarterectomy	77 (32)	18 (36)	36 (34)	23 (26)	16 (16)	.022
Preprocedural Peripheral Arterial Disease	70 (29)	17 (34)	35 (33)	18 (21)	23 (23)	.220
Prior Coronary Artery Bypass Graft	87 (36)	23 (46)	43 (41)	21 (24)	10 (10)	<.001
Prior Percutaneous Coronary Intervention	98 (40)	19 (38)	49 (46)	30 (34)	27 (27)	.064
Prior Permanent Pacemaker	32 (13)	5 (10)	12 (11)	15 (17)	8 (8)	.391
Prior Aortic Valvuloplasty	43 (18)	7 (14)	16 (15)	20 (23)	16 (16)	.592
Warfarin	53 (22)	10 (20)	25 (24)	18 (21)	22 (22)	.985
DOAC	6 (2)	3 (6)	1 (1)	2 (2)	5 (5)	.284
ASA	198 (81)	41 (82)	88 (83)	69 (79)	61 (62)	.001
ADP receptor inhibitor	80 (33)	19 (38)	35 (33)	26 (30)	27 (27)	.705
ACE inhibitor	83 (34)	19 (38)	34 (32)	30 (34)	27 (27)	.679
Angiotensin II receptor blocker	50 (21)	14 (28)	18 (17)	19 (22)	11 (11)	.103
Beta blocker	187 (77)	38 (76)	86 (81)	63 (72)	65 (66)	.137
Calcium channel blocker	74 (30)	18 (36)	32 (30)	24 (28)	24 (24)	.624
Thiazide diuretic	53 (22)	9 (18)	23 (22)	21 (24)	18 (18)	.850
Loop diuretic	121 (50)	27 (54)	54 (51)	40 (46)	48 (48)	.914
Spironolactone	8 (3)	1 (2)	5 (5)	2 (2)	4 94)	.861

Values are mean (standard deviation) or n (%).

ficant differences in any variable of interest at both baseline and at one year follow-up. The TAVR procedural characteristics are given in Table 3. No differences were observed in regard to the TAVR approach, length of stay, valve size, or specific type of valve deployed. Finally, the primary and secondary outcomes of this study are presented in Table 4. No statistically significant differences in overall survival at any of the predefined time periods were observed, nor was there any dissimilarities in procedural complications. A significant difference in the incidence of stroke or TIA both in hospital and within 30 days of discharge was noted, with a higher incidence of these events in the non-statin cohort. This difference was not sustained in the time periods beyond 30 days post-TAVR.

DISCUSSION

This study significant increases the understanding of the possible events that HMG Co-A reductase inhibiters have on post-TAVR outcomes and calls into question the external validity of previously published research in this area. Unlike the work published by Hudred *et al.* this study did not find any differences in overall survival between patients taking statin of any intensity and overall survival. We also did not observe any meaningful differences in outcomes of many of this studies other important outcomes.

However, in contrast to the work Hudred *et al.* this study does present evidence to suggest benefit from statin usage in reducing the incidence of

Table 2: Echocardiographic Data

	Any Statin	High Intensity	Moderate Intensity	Low Intensity	No Statin	P-value
Preprocedural						
Aortic valve area (VTI) (cm^2)	0.89 (0.32)	0.84 (0.22)	0.90 (0.35)	0.90 (0.34)	0.86 (0.46)	.790
Peak aortic velocity (cm/s)	412.4 (66.6)	414.2 (58.4)	412.6 (74.2)	410.9 (61.9)	420.7 (65.1)	.856
Peak aortic gradient (mmHg)	69.7 (21.1)	69.8 (19.1)	70.5 (23.5)	68.7 (19.4)	73.9 (19.3)	.487
Mean aortic gradient (mmHg)	43.9 (13.0)	44.0 (11.6)	44.6 (14.2)	43.0 (12.4)	46.8 (12.6)	.324
Ejection fraction (%)	57.4 (12.7)	55.9 (12.5)	57.4 (12.7)	58.2 (12.8)	57.7 (13.0)	.891
Stroke volume (mL)	86.5 (20.5)	83.6 (19.2)	86.8 (19.3)	88.2 (22.7)	84.6 (22.9)	.768
Moderate aortic regurgitation (%)	21	20	19	24	15	.624
Severe aortic regurgitation (%)	5	4	3	7	4	.746
Moderate mitral regurgitation (%)	23	28	18	25	21	.622
Severe mitral regurgitation (%)	4	2	6	3	2	.659
1 year post-TAVR						
Aortic valve area (VTI) (cm^2)	2.00 (0.59)	1.81 (0.51)	2.07 (0.65)	2.01 (0.53)	1.97 (0.65)	.631
Peak aortic velocity (cm/s)	224.2 (49.5)	231.7 (50.2)	226.4 (59.5)	218.3 (35.2)	213.9 (49.8)	.637
Peak aortic gradient (mmHg)	21.3 (10.5)	22.4 (9.7)	22.0 (12.4)	20.1 (8.5)	19.3 (9.1)	.672
Mean aortic gradient (mmHg)	12.2 (6.2)	12.0 (5.1)	12.8 (7.1)	11.7 (5.9)	10.6 (4.9)	.519
Ejection fraction (%)	57.9 (13.1)	56.7 (14.4)	58.0 (13.4)	58.4 (12.4)	58.8 (12.8)	.985
Stroke volume (mL)	94.8 (27.8)	84.4 (20.9)	101.1 (32.1)	92.1 (23.7)	90.0 (30.4)	.256
Moderate aortic regurgitation (%)	11	6	7	17	19	.301
Moderate mitral regurgitation (%)	13	17	5	19	12	.340
Severe mitral regurgitation (%)	6	0	7	7	5	.990

Values are mean (standard deviation) or %.

Table 3: Procedural Characteristics.

	Any Statin (n = 243)	High Intensity (n = 50)	Moderate Intensity (n = 106)	Low Intensity (n = 87)	No Statin (n = 99)	P-value
Approach						
Transfermoral	194 (80)	40 (80)	87 (82)	67 (77)	84 (85)	.715
Transapical	39 (16)	7 (14)	15 (14)	17 (20)	11 (11)	.053
Transaortic	5 (2)	1 (2)	2 (2)	2 (2)	2 (2)	.958
Trans-subclavian	4 (2)	2 (4)	1 (1)	1 (1)	2 (2)	.707
Transcaval	1 (0)	0 (0)	1 (1)	0 (0)	0 (0)	.840
Mean LOS after TAVR (days)	4.3 (4.4)	4.0 (3.6)	4.1 (4.0)	4.7 (5.3)	5.8 (11.3)	.210
Valve type						
First generation Sapien	64 (26)	10 (20)	24 (23)	30 (34)	28 (28)	.300
Sapien XT	39 (16)	7 (14)	15 (14)	17 (20)	12 (12)	.691
Sapien S3	81 (33)	22 (44)	38 (36)	21 (24)	33 (33)	.425
First Generation CoreValve	53 (22)	8 (16)	28 (26)	17 (20)	21 (21)	.627
CoreValve Evolute	6 (2)	3 (6)	1 (1)	2 (2)	5 (5)	.284
Mean valve size (mm)	26.0 (2.6)	26.1 (2.4)	26.2 (2.7)	25.7 (2.7)	25.9 (2.9)	.757

Values are mean (standard deviation) or n (%).

Table 4: Primary and Secondary Outcomes.

	Any Statin	High Intensity	Moderate Intensity	Low Intensity	No statin	P-value
% Survival > 1 month	95 (232/243)	96 (48/50)	96 (102/106)	94 (82/87)	92 (91/99)	.635
% Survival > 6 month	89 (169/190)	92 (34/37)	90 (70/78)	87 (65/75)	88 (74/84)	.814
% Survival > 1 year	79 (127/160)	82 (23/28)	75 (51/68)	83 (53/64)	80 (51/64)	.845
% Survival > 2 year	61 (67/109)	71 (12/17)	59 (29/49)	60 (26/43)	58 (22/38)	.926
Periprocedural Major Vascular	8 (20)	12 (50)	6 (6)	9 (8)	9 (9)	.732
Periprocedural Minor Vascular	8 (20)	6 (3)	8 (9)	9 (8)	10 (10)	.799
Periprocedural Increase in Serum Creatinine >1.5x baseline	7 (16)	10 (5)	5 (5)	7 (6)	3 (3)	.469
In Hospital						
Cardiovascular (CV) Mortality	5 (12)	4 (2)	4 (4)	7 (6)	7 (7)	.775
Myocardial Infarction	1 (2)	0 (0)	2 (2)	0 (0)	0 (0)	.412
Stroke/TIA	1 (2)	0 (0)	1 (1)	1 (1)	7 (7)	.001
Heart Failure (HF) exacerbation	18 (44)	14 (7)	18 (19)	21 (18)	29 (29)	.120
Discharge to 30 days						
MACCE	17 (39)	19 (9)	16 (16)	18 (14)	20 (18)	.963
CV Mortality	0 (0)	0 (0)	0 (0)	0 (0)	1 (1)	.676
Myocardial Infraction	2 (4)	2 (1)	1 (1)	3 (2)	0 (0)	.663
Stroke/TIA	0 (0)	0 (0)	0 (0)	0 (0)	4 (4)	.004
HF exacerbation	16 (36)	10 (5)	18 (18)	16 (13)	16 (15)	.854
Rehospitalization For Any Reason	17 (39)	19 (9)	16 (16)	18 (14)	18 (17)	.985
30 days- 6 months						
MACCE	27 (47)	26 (9)	27 (20)	27 (18)	29 (22)	.994
CV Mortality	3 (5)	0 (0)	4 (3)	3 (2)	0 (0)	.430
Myocardial Infraction	2 (3)	0 (0)	3 (2)	1 (1)	0 (0)	.639
Stroke/TIA	3 (6)	6 (2)	1 (1)	4 (3)	0 (0)	.302
HF exacerbation	18 (32)	18 (6)	16 (12)	21 (14)	8 (6)	.258
Rehospitalization For Any Reason	24 (42)	26 (9)	24 (18)	22 (15)	27 (20)	.976
6 months-1 year						
MACCE	31 (41)	29 (7)	38 (21)	25 (13)	28 (14)	.695
CV Mortality	3 (4)	8 (2)	4 (2)	0 (0)	0 (0)	.180
Myocardial Infraction	2 (2)	8 (2)	0 (0)	0 (0)	4 (2)	.070
Stroke/TIA	2 (3)	0 (0)	2 (1)	4 (2)	0 (0)	.631
HF exacerbation	20 (26)	8 (2)	20 (11)	25 (13)	18 (9)	.559
Rehospitalization For Any Reason	27 (36)	25 (6)	34 (19)	21 (11)	28 (14)	.681

Values are % (n).

MACCE = major adverse cardiovascular and cerebrovascular events, defined as death from any cause, myocardial infarction, rehospitalization, and stroke.

immediate and short-term stroke or TIA. This finding is may be clinical significant and a compelling indication for use of these drugs in the preprocedural time period. Additionally, it would appear that this observed benefit is consistent with the documented effects of preprocedural statins in percutaneous coronary intervention (PCI), carotid endarterectomy (CEA), and coronary artery bypass grafting (CABG).[11-13] Based upon the findings of our study and in the absence of other indications, we believe it is reasonable to consider giving a statin loading dose near the time of TAVR or starting a statin pre-TAVR pre-operative and continuing it short-term after TAVR.

There are some important differences between our study and the work by Hudred *et al*. Most importantly, our study is more consistent with the current ACC/AHA guidelines in that, we chose to make a distinction between low and moderate intensity statin. Although we did not observe any significant differences among these two groups, they should not be consider equal given the recommendations for appropriate statin use by the ACC/AHA.[9] Yet, another difference between our studies lies in each study's baseline characteristics. The Hudred *et al*. study did include significant baseline differences in regard to hypertension, diabetes mellitus, dyslipidemia, and peripheral artery disease, which were not apparent in our study. It is likely that some of the perceived benefit of statin on overall survival seen the Hudred *et al*. study is the result of medical optimization of comorbid conditions, and not a true benefit of the interaction between statins and TAVR. Also, the study we presents here, address several other

important TAVR such as preprocedural complications, rehospitalization, and heart failure exacerbations which did not differ in our study.

This study also attempted to define a possible mechanism for potential benefit of statin following TAVR. We include an analysis of preprocedural and 1 year post-TAVR echocardiographic data for each cohort. Since it would appear that the survival benefit of TAVR over medical therapy is directly related to relieving the obstruction created by a stenotic aortic valve, it is reasonable to assume that if there was additional benefit from statin therapy it would manifest as improvement in echocardiographic parameters. However, no significant changes were observed between the statin and non-statin groups.

On the other hand, the pleiotropic effect of statins may indeed be of benefit to TAVR patients. Statin effects outside of LDL lowering, such as improvement in endothelial function, anti-inflammatory, and anti-thrombotic effects could be the mechanism by which statins reduce the incidence of stroke and TIA in our study. It may be assumed that the implantation of a large vascular foreign body, such as a percutaneous TAVR valve, could disrupt endothelial homeostasis and have downstream inflammatory effects. We invite more study into this area to further define the role endothelial function, inflammation and the role of adjunctive pharmacotherapy in the peri-operative period of TAVR.

Finally, our study does have some limitations including its retrospective design, single center experiences, and variability in the length of post-TAVR follow-up. We did have a few statistically significant differences in baseline characteristics. Previous studies have shown that male sex may be a negative prognostic marker post-TAVR, however the effect size of this appears to be small.[14] A history of CABG and CAD regardless of severity prior to TAVR has not been associated with worse outcomes after TAVR.[15] Additionally, we are not aware any data that would suggest a difference in outcomes in patients not on aspirin prior to TAVR.

CONCLUSION

In this study, no association between HMG Co-A reductase inhibitors and improvement in overall survival as found regardless of lipid lowering intensity. However, statin usage was associated with a reduction in the incidence of stroke/TIA in the immediate to short-term post-TAVR period. This study calls into question the external validity of previous published data which suggested mortality benefit from statin after TAVR. More study into the role of statins in reducing periprocedural complications of TAVR is warranted.

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