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Original Article

Association between statins and deep vein thrombosis

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Abstract

Aim: The aim of this study is to investigate the relationship between statin use and deep vein thrombosis (DVT).
Material and Methods: Between 2022 and 2024, 1833 patients (926 males; mean age 50.2±0.37 years) who applied to our clinic were included in the study. Following exclusion criteria, the patients were divided into two groups. Group A (n=226): patients diagnosed with DVT, and Group B (n=1607): Patients without DVT. Then, patients diagnosed with DVT (n=226) were divided into two separate subgroups. Group C (n=16): Statin users, and Group D (n=210): statin non-users. Comparisons were made between these groups in terms of demographic data, comorbidities, statin usage, laboratory parameters, and DVT localizations.
Results: Statin usage was significantly lower in the DVT group than in the non-DVT group (16 (7.1%) and 184 (11.4%), respectively, p=0.04). In Group C, the incidence of DVT in the superficial femoral vein was higher than in Group D (p=0.03). Although no statistically significant difference was found, the incidence of DVT in the iliac veins was higher in Group D, while the incidence of DVT in the common femoral vein was higher in Group C. Similarly, rare localizations of DVT are more frequent in Group D than in Group C. DVTs in statin non-users tended to be more widespread than in statin users. A negative significant association was found between statin usage and DVT (p=0.001, OR= -0.084 [95% CI, -0.123–0.036].
Conclusion: We determined that statin usage was lower in patients with DVT than in those without DVT. Statin usage may be protective against DVT. Although no statistically significant difference was found, DVTs in statin non-users tended to be more widespread than in statin users. We found that DVTs in statin non-users affected more proximal segments, whereas in statin users, more distal venous segments were affected.

Keywords: Statin, deep vein thrombosis, venous thromboembolism, pulmonary embolism, dyslipidemia

INTRODUCTION

Deep vein thrombosis (DVT), is usually characterized by the formation of a clot in the lower extremity venous system and, it is the third most common cause of death from cardiovascular disease after heart attack and stroke [1]. To treat DVT and prevent recurrent thrombosis attacks, anticoagulant treatments are used for 3-6 months, taking into account the patients' bleeding risk factors, and if necessary, lifelong extended treatment is given. Other drugs can be used to support the treatment in addition to anticoagulants.

The use of 3-hydroxy-3-methyl coenzyme A (HMG-CoA) reductase inhibitors (statins) is important in the primary and secondary prevention of cardiovascular disease by reducing the rates of coronary events in individuals with both normal and high cholesterol values [2]. They stabilize atherosclerotic plaque and reduce the incidence of thrombotic events such

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as myocardial infarction (MI) and stroke. In recent years, many studies have shown that statins have pleiotropic and antithrombotic effects in addition to their effects on cholesterol metabolism [3]. In a systematic meta-analysis by Kunutsor et al., statin use, particularly rosuvastatin, was shown to have a beneficial effect on venous thromboembolism (VTE) [4]. Nguyen et al reported that statin use was associated with a reduced risk of recurrent VTE [5]. Another meta-analysis did not support the findings that statins have a significant protective effect on VTE [6]. Gaddam et al. reported that atorvastatin delays thrombus formation in arterial channels exposed to antioxidant stress [7].

Although there are many studies in the literature on the risk of venous thrombosis with statin use, there is not enough evidence for routine use in the treatment or prophylaxis of DVT. For this reason, we designed this study. The aim of this study is to investigate the relationship between statin use and deep vein thrombosis.

MATERIAL AND METHODS

This study was designed as a single-center retrospective cohort study including a total of 1833 patients. All patients over the age of 18 who applied to the Cardiovascular Surgery outpatient clinic of İstanbul Başakşehir Çam and Sakura City Hospital between January 2022 and May 2024 were included in the study. Patients with a history of cardiac surgery/cardiac interventional procedure, those who underwent interventional procedures due to chronic venous insufficiency (CVI) or peripheral artery disease, thrombophlebitis, genetic predisposition to thrombosis, use of anticoagulants due to another diagnosis, and pregnant women were excluded from the study. Without patient selection, all patients who met these criteria and whose data were available between these dates were included in the study.

All patients' basic demographic characteristics, medical history, family history, DVT localization, laboratory parameters, and all medications used were recorded by reviewing the medical records. DVT was diagnosed by Doppler ultrasonography (DUS) or computed tomography (CT) venography. The initial diagnosis of DVT was based on direct visualization of thrombus, absence of compression on gray scale, and identification of a filling defect or absence of thrombus or flow in the colored column of the vascular lumen on DUS. Patients with suspected pulmonary embolism (PE) were diagnosed by computed tomography pulmonary angiography. Low-molecular-weight heparin (LMWH) and warfarin or direct oral anticoagulant (DOAC) were used as initial therapy in newly diagnosed patients. In patients whose international normalization ratio (INR) value could not be kept at an effective level after using warfarin for at least 2 months, DOAC treatment was started.

The patients were first divided into two groups. Group A (n=226): Patients diagnosed with DVT, and Group B (n=1607): Patients without DVT. Comparisons were made between the groups in terms of demographic data, comorbidities, PE rates, medication data, and laboratory parameters. Then, patients diagnosed with DVT (n=226) were divided into two separate subgroups. Group C (n=16): Statin users and Group D (n=210): statin non-users. A comparison was made between these subgroups in terms of DVT localizations and vascular involvement segments.

This study was approved by the İstanbul Başakşehir Çam and Sakura City Hospital Ethics Committee (Decision no: 2024-71, Date: 26.06.2024).

Statistics

Data were analyzed by using SPSS software version 20.0 (IBM, USA). Continuous variables in the study were presented as minimum, maximum, median, and interquartile range. Categorical variables were expressed as numbers and percentages. The normality of distribution was assessed by the Kolmogorov–Smirnov test. For numerical variables, differences between patients and controls were tested using t test for parametric data or the Mann–Whitney U test for non-parametric data. Categorical variables were analyzed using the Pearson $\chi 2$ test and Fisher's exact test for parametric and nonparametric data, respectively. Multivariate logistic regression analysis was conducted to identify the factors affecting DVT. The level of statistical significance was set at $p<0.05$.

RESULTS

Following exclusion criteria, data from a total of 1833 patients were analyzed. The mean age was 50.2±0.37 years and 926 (50.5%) of the patients were male. A comparison of demographic data and comorbidities between the groups is shown in Table 1. No difference was found between the groups in terms of gender and weight. However, age and height were higher in Group A (DVT group) than in Group B ($p<0.001$ and $p<0.001$, respectively). No difference was found in the rates of coronary artery disease (CAD), hypertension, and rheumatic disease between Group A and Group B. However, diabetes mellitus (DM), chronic obstructive pulmonary disease, cerebrovascular diseases (CVA), PE, and malignancy rates were significantly higher in Group A than in Group B ($p=0.02$, $p=0.01$, $p<0.001$, $p<0.001$, $p<0.001$, respectively). Statin usage was significantly lower in the DVT group than in the non-DVT group (16 (7.1%) and 184 (11.4%), respectively, $p=0.04$). The CVI rate was also lower in Group A than in Group B ($p<0.001$).

Table 1. Patient's demographic data, and comorbid diseases

		Group A DVT (+) (n=226)		Group B DVT (-) (n=1607)		
		Min-max or n (%)	Median (IQR)	Min-max or n (%)	Median (IQR)	p
Demographic data	Gender male	105 (46.5)		821 (51.1)		0.19
	Age (years)	18-99	58 (26)	18-92	50 (24)	**<0.001***
	Height (cm)	144-200	171 (12)	140-195	167 (13)	**<0.001***
	Weight (kg)	40-131	78 (16)	37-160	78 (21)	0.22
Comorbid diseases	Coronary artery disease	16 (7.1)		112 (7)		0.95
	Hypertension	58 (25.7)		353 (22)		0.21
	Diabetes Mellitus	47 (20.8)		242 (15.1)		**0.02***
	COPD	16 (7.1)		53 (3.5)		**0.01***
	Cerebrovascular accident	19 (8.4)		18 (1.1)		**<0.001***
	Statin usage	16 (7.1)		184 (11.4)		**0.04***
	Pulmonary embolism	13 (5.8)		3 (0.2)		**<0.001***
	Rheumatic disease	3 (1.3)		14 (0.9)		0.5
	Malignancy	19 (8.4)		15 (0.9)		<0.001*
	CVI	62 (27.4)		769 (47.8)		**<0.001***

IQR: interquartile range, DVT: deep vein thrombosis COPD: chronic obstructive pulmonary disease, CVI: chronic venous insufficiency, CEAP: clinical-etiology-anatomy-pathophysiology

The patients' laboratory parameters are shown in Table 2. No difference was found between the groups in terms of leukocyte, platelet, urea, LDL, and total cholesterol, sodium, potassium, alanine aminotransferase (ALT), and aspartate aminotransferase (AST) values. Hemoglobin value was higher in Group B than in Group A (mean 13.2 g/dl and 12.4 g/dl, respectively, p<0.001). As expected, C-reactive protein (CRP) and ferritin values were higher in Group A than in Group B (p<0.001 and p<0.001, respectively).

Table 2. Comparison of laboratory parameters between groups

	Group A DVT (+) (n=226)		Group B DVT (-) (n=1607)		
	Min-max	Median (IQR)	Min-max	Median (IQR)	p
Leukocyte (109/L)	1.5-26	8.1 (3.5)	0.7-24.9	7.5 (2.7)	0.14
Hemoglobin (g/dl)	6.3-17.6	13 (3)	3.9-18	13 (2)	**<0.001***
Platelet (109/L)	13-615	252 (100)	37-641	258 (86)	0.97
Urea (mg/dL)	12-175	32 (18)	4-180	27 (12)	0.82
Creatinine (mg/dL)	0.2-8	0.8 (0.41)	0.04-12	0.7 (0.22)	**<0.001***
Ldl cholesterol (mg/dL)	32-212	108 (34.3)	21-259	112 (43)	0.14
Total cholesterol (mg/dL)	56-288	184 (51.3)	71-380	184 (55)	0.82
Sodium (mEq/L)	124-154	139 (4)	121-151	139 (3)	0.10
Potassium (mEq/L)	3-6.5	4.2 (0.6)	3-6	4.2 (0.5)	0.99
ALT (IU/L)	5-276	17 (12)	2-275	16 (10)	0.050
AST (IU/L)	3-1335	19 (11.3)	6-159	18 (8)	0.20
CRP (mg/dL)	0.02-250	2.8 (13.6)	0.01-150	0.6 (2.1)	**<0.001***
Ferritin (ml/ng)	3-1133	62 (85.3)	1-2000	38 (51)	**<0.001***

IQR: interquartile range, ALT: alanine aminotransferase, AST: aspartate aminotransferase, CRP: C-reactive protein

Medications used in DVT patients are shown in Table 3. For initial treatment, warfarin was used in 79 (35%) patients, LMWH in 98 (43.4%) patients, and DOAC in 49 (21.7%) patients. Of the patients, 111 (49.1%) had acute DVT, 35 (15.5%) had subacute DVT, and 101 (44.7%) had chronic DVT. 115 (50.9%) of the patients had right-sided DVT, 132 (58.4%) had left-sided DVT, and 22 (9.7%) had bilateral DVT. DVTs were frequently detected in the common femoral vein, superficial femoral vein, and popliteal vein. Recanalization was observed in 43 (19%) patients.

Table 3. Medication and disease data of deep vein thrombosis patients

		n=226 (%)
Initial therapy	**Warfarin**	79 (35)
	Low molecular weight heparin	98 (43.4)
	Direct oral anticoagulant	49 (21.7)
Maintenance therapy	**Acetylsalicylic acid**	13 (5.8)
	Warfarin	109 (48.2)
	Low molecular weight heparin	17 (7.5)
	Direct oral anticoagulant	87 (38.4)
Deep vein thrombosis time	**Acute**	111 (49.1)
	Subacute	35 (15.5)
	Chronic	101 (44.7)
Deep vein thrombosis localization	**Right leg**	115 (50.9)
	Left leg	132 (58.4)
	Bilateral	22 (9.7)
	Common iliac vein	5 (2.2)
	External iliac vein	39 (17.3)
	Common femoral vein	124 (54.9)
	Superficial femoral vein	162 (71.7)
	Popliteal vein	163 (72.1)
	Crural vein	55 (24.3)
	Jugular vein	1 (0.4)
	Superior vena cava	1 (0.4)
	Inferior vena cava	3 (1.3)
	Axillary vein	4 (1.8)
	Subclavian vein	5 (2.2)
	Brachial vein	4 (1.8)
	Recanalization	43 (19)

The comparison after dividing DVT patients into two separate subgroups as statin users and statin non-users is shown in Table 4. In Group C (statin users), the incidence of DVT in the superficial femoral vein was higher than in Group D (p=0.03). Although no statistically significant difference was found, the incidence of DVT in the iliac veins was higher in Group D, while the incidence of DVT in the common femoral vein was higher in Group C. Similarly, although no statistical difference was found, rare types of DVT such as jugular vein, vena cava, axillary vein, and brachial vein were also seen more frequently in Group D than in Group C. In addition, in the comparison of the prevalence of concurrent vascular involvement in patients with DVT, although again no statistical difference was found, only femoral + popliteal vein involvement was higher in Group C than in Group D. In contrast, involvement of more proximal segments, such as iliac + femoral + popliteal + crural vein association, iliac + femoral + popliteal vein association, and iliac + femoral vein association, was higher in Group D than in Group C. This is also shown graphically in Figure 1.

Table 4. Comparison of deep vein thrombosis locations between statin users and non-statin users

	Group C Statin (+) (n=16)	Group D Statin (-) (n=210)	p
Common iliac vein	0	5 (2.4)	0.69
External iliac vein	1 (6.3)	38 (18.1)	0.19
Common femoral vein	10 (62.5)	114 (54.3)	0.52
Superficial femoral vein	15 (93.8)	147 (70)	**0.03***
Popliteal vein	11 (68.8)	152 (72.4)	0.47
Crural vein	1 (6.3)	54 (25.7)	0.06
Jugular vein	0	1 (0.5)	0.92
Superior vena cava	0	1 (0.5)	0.92
Inferior vena cava	0	3 (1.4)	0.80
Axillary vein	0	4 (1.9)	0.74
Subclavian vein	0	5 (2.4)	0.69
Brachial vein	0	4 (1.9)	0.74
Recanalization	5 (31.3)	40 (19)	0.19
Iliac + femoral + popliteal + crural vein	0	3 (1.4)	0.80
Iliac + femoral + popliteal vein	1 (6.3)	22 (10.5)	0.49
Iliac + femoral vein	0	11 (5.2)	0.43
Femoral + popliteal vein	10 (62.5)	85 (40.5)	0.08

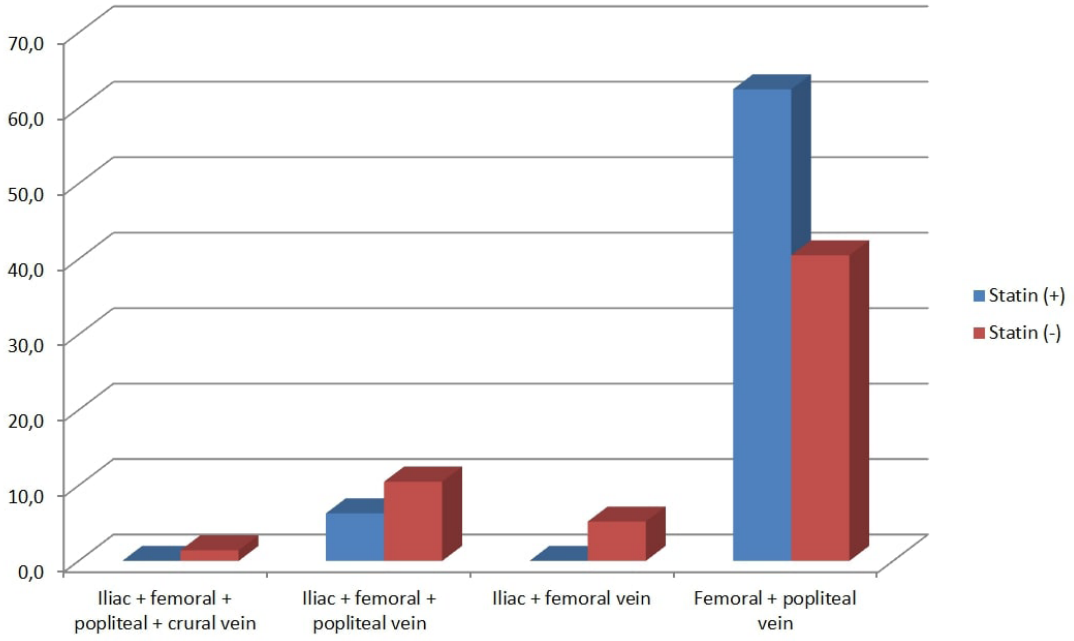

Figure 1. Comparison of affected vascular segments between statin user and non-user subgroups in patients with deep vein thrombosis

Multivariate logistic regression analysis results are shown in Table 5. Accordingly, no significant association was found between gender, CAD, hypertension, DM, COPD, and DVT. However, a significant association was found between advanced age, CVA, PE, malignancy history, and DVT ($p<0.001$, $p<0.001$, $p<0.001$ and $p<0.001$, respectively). A negative significant association was found between statin usage and DVT ($p=0.002$, OR= 0.400 [95% CI, 0.224–0.714].

Table 5. Multivariate logistic regression analysis

	Odds ratio	CI %95	p
Gender male	0.801	0.593-1.080	0.14
Age	**1.034**	1.023-1.045	**<0.001***
Coronary artery disease	0.519	0.267-1.009	0.053
Hypertension	0.720	0.487-1.065	0.10
Diabetes Mellitus	1.264	0.838-1.908	0.26
COPD	1.336	0.712-2.623	0.34
Cerebrovascular accident	**7.305**	3.503-15.726	**<0.001***
Statin usage	**0.400**	0.224-0.714	**0.002***
Pulmonary embolism	**35.474**	9.499-132.482	**<0.001***
Malignancy	**6.330**	2.966-13.512	**<0.001***

COPD: chronic obstructive pulmonary disease, CI: confidence interval

DISCUSSION

Dyslipidemia is one of the most important risk factors for cardiovascular system diseases. Genetic factors and abnormal irregularities in lipid levels can lead to atherosclerosis and cardiovascular system (CVS) diseases. Dyslipidemia has prothrombotic, vascular endothelium-modifying, and platelet activation effects [8]. Additionally, an association has been shown between increased lipoprotein (a) levels and the occurrence of venous thromboembolism [9].

Venous and arterial thrombosis are diseases that can be seen together and they have similar risk factors. Due to the nature of the common pathways, many drugs such as antiplatelet and anticoagulant treatments can be used for primary and secondary prevention in both diseases. Statins used in the treatment of dyslipidemia are also used especially in arterial pathologies. There is still debate about whether they are beneficial in VTE. There are studies in the literature with opposing results on this subject. Many studies show that statin treatment has beneficial effects in terms of VTE [2,4,5,10-13]. On the other hand, there are also studies reporting that statin treatment has no protective effect on VTE [6,14]. Therefore, we planned this cohort study. As a result of this study, we found that the risk of DVT can be reduced with statin use. In addition, we think that the venous segments affected by DVT can be reduced by statin usage.

Statins, in addition to lowering cholesterol levels, also have pleiotropic and antithrombotic effects [3]. Statin treatment reduces the expression of monocyte chemotactic protein 1 (MCP-1), CRP, and IL-6, which are elevated in VTE [15]. In addition, it affects many factors such as nitric oxide, thromboxane A2, NADPH oxidase, oxLDL-C, CD36, tissue factor, endothelial protein C, antithrombin III, Factor V, and von Willebrand [16-18]. Statins also have antithrombotic effects, such as inhibition of isoprenylation of signaling proteins, reduction of tissue factor expression and thrombin production, attenuation of fibrinogen dissociation, and activation of factors V and VII [19,20]. It also increases Kruppel-like factor 2 (KLF-2), activates thrombomodulin expression and protein C pathway in the endothelium [21].

Due to all these mechanisms of action, we think that statins may also be beneficial in the treatment of VTE. Therefore, in this study, we first divided the patients into those with and without DVT. Statin usage was significantly lower in those with DVT than in those without DVT. However, we found that DVT patients were older and had more comorbid diseases. Therefore, we performed multivariate logistic regression analysis to better demonstrate the relationship between statin usage and DVT. As a result, we found that statin usage may be protective against DVT. We found that factors such as PE, malignancy, and advanced age also increased the risk of DVT, as expected.

Another important finding in our study is the area affected by DVT in the venous system. Although many studies have been conducted on the risk of DVT with statins, there is insufficient data on the prevalence of DVT localizations. For this purpose, we conducted a new analysis on the subgroup that only included DVT patients. As can be seen in Figure 1, DVTs seen in statin non-users tended to show more widespread involvement compared to statin users. In addition, DVTs seen in statin non-users were more likely to involve more proximal segments such as the iliac vein, while relatively more distal venous segments such as the femoral vein were more commonly involved in statin users. However,

these findings did not show a statistical difference. This may be related to the decrease in the number of patients in Groups C and D, especially since subgroup analysis was performed. Nevertheless, we believe that these results, which we found differently from many previous studies, will shed light on future studies that include larger numbers of patients.

In our study, the results could not be analyzed for different forms of statins. In addition, the analysis could not be done for different doses of the same drug form. We have stated these in the limitations. It has been reported that statins may be more beneficial in this regard at higher doses [10]. In addition, different drug forms affect different mechanisms. For example, simvastatin impairs prothrombin, factor (F) V, and FXIII activation and increases FVa inactivation by activated protein C, but the effect of pravastatin on these factors is unknown [22]. Similarly, simvastatin suppresses tissue factor activity in macrophages, but pravastatin does not have this effect [23]. Therefore, future studies investigating each drug separately may provide clearer information.

Limitations

The first limitation of this study is that it is a retrospective and single-center study. Second, since the study was conducted with patients who applied to the cardiovascular surgery clinic, CVS diseases were also more common than in the normal population. It would be useful to conduct future studies to ensure that these factors do not affect the current results. Third, although the total number of patients was high, the number of patients in Groups C and D decreased relatively, especially after dividing into subgroups. To reach better results regarding DVT localizations, studies with more patients in this group are needed. Fourth, since all patients admitted to our hospital were included in the study except for the exclusion criteria, the number of patients diagnosed with DVT was lower than in the other group. This caused an imbalance in the numbers between the groups. Finally, the analysis could not be performed by separating the types of statin drugs and doses. Considering that different results may be obtained with different drugs and doses, future studies regarding these subtypes will allow us to reach more definitive results.

CONCLUSION

In this study, we determined that statin usage was lower in patients with DVT than in those without DVT. We also found that statin usage may be protective against DVT. In addition, although no statistically significant difference was found, DVTs in statin non-users tended to be more widespread than in statin users. We found that DVTs in statin non-users affected more proximal segments, whereas in statin users, more distal venous segments were affected. However, these findings need to be investigated by future studies.

Ethics Committee Approval: This study was approved by the İstanbul Başakşehir Çam and Sakura City Hospital Ethics Committee (Decision no: 2024-71, Date: 26.06.2024).

Patient Consent for Publication: Since our study is retrospective, patient consent was not obtained. However, an ethics committee decision was made.

Data Sharing Statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

Author Contributions: All authors contributed equally to the article.

Conflict of Interest: The authors declared no conflicts of interest with respect to the authorship and/or publication of this article.

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