

Atorvastatin and growth, rupture of small unruptured intracranial aneurysm: results of a prospective cohort study

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On behalf of the Small Unruptured Aneurysms Study Group

Abstract

Background and aims: The role of statins in unruptured intracranial aneurysm (UIA) growth and rupture remains ambiguous. This study sought to determine whether atorvastatin is associated with aneurysm growth and rupture in patients harboring UIA <7 mm.

Methods: This prospective, multicenter cohort study consecutively enrolled patients with concurrent UIA <7 mm and ischemic cerebrovascular disease from four hospitals between 2016 and 2019. Baseline and follow-up patient information was recorded. Because of the strong anti-inflammatory effect of aspirin, patients using aspirin were excluded. Patients taking atorvastatin 20 mg daily were atorvastatin users. The primary and exploratory endpoints were aneurysm rupture and growth, respectively.

Results: Among the 1087 enrolled patients, 489 (45.0%) took atorvastatin, and 598 (55%) took no atorvastatin. After a mean follow-up duration of 33.0 ± 12.5 months, six (1.2%) and five (0.8%) aneurysms ruptured in atorvastatin and non-atorvastatin groups, respectively. In the adjusted multivariate Cox analysis, UIA sized 5 to <7 mm, current smoker, and uncontrolled hypertension were associated with aneurysm rupture, whereas atorvastatin [adjusted hazard ratio (HR) 1.495, 95% confidence interval (CI) 0.417–5.356, $p=0.537$] was not. Of 159 patients who had follow-up imaging, 34 (21.4%) took atorvastatin and 125 (78.6%) took no atorvastatin. Aneurysm growth occurred in five (14.7%) and 21 (16.8%) patients in atorvastatin and non-atorvastatin groups (mean follow-up: 20.2 ± 12.9 months), respectively. In the adjusted multivariate Cox analysis, UIAs sized 5 to <7 mm and uncontrolled hypertension were associated with a high growth rate; atorvastatin (adjusted HR 0.151, 95% CI 0.031–0.729, $p=0.019$) was associated with a reduced growth rate.

Conclusions: We conclude atorvastatin use is associated with a reduced risk of UIA growth, whereas atorvastatin is not associated with UIA rupture.

The trial registry name: The Clinic Benefit and Risk of Oral Aspirin for Unruptured Intracranial Aneurysm Combined With Cerebral Ischemia

Clinical Trial Registration-URL: <http://www.clinicaltrials.gov>

Unique identifier: NCT02846259

Keywords: atorvastatin, growth, risk factors, rupture, unruptured intracranial aneurysm

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Introduction

Unruptured intracranial aneurysm (UIA) is a common cerebrovascular disease. The prevalence of UIA is approximately 3%–7% in the general population. The risk of rupture of UIA is approximately 1%–2% per year. The growth of UIA is a major risk factor for rupture. The role of statins in UIA growth and rupture remains ambiguous. This study sought to determine whether atorvastatin is associated with UIA growth and rupture in patients harboring UIA <7 mm.

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$$\left(\frac{1}{n} \sum_{i=1}^n Y_i \right) - \left(\frac{1}{n} \sum_{i=1}^n Y_i \right) = 0$$

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Data collection

The data collection process involves several steps, including data entry, cleaning, and validation. The researchers ensure that the data is accurate and complete by conducting thorough checks and corrections. This process is crucial for the reliability of the study's findings.

Outcome and radiological assessment

The outcome and radiological assessment are performed using advanced imaging techniques and statistical analysis. The researchers evaluate the radiological findings against established criteria to determine the severity and extent of the condition being studied. This assessment provides valuable insights into the progression and management of the disease.

Follow-up

The follow-up phase involves monitoring the subjects over a period of time to assess the long-term effects of the intervention or treatment. This helps in understanding the durability of the results and identifying any potential complications or relapses.

Statistical analysis

The primary outcome was the proportion of patients who were able to walk without assistance at 6 weeks. Secondary outcomes included the proportion of patients who were able to walk with assistance, the proportion of patients who were able to walk with a cane, the proportion of patients who were able to walk with a walker, and the proportion of patients who were able to walk with a wheelchair. All outcomes were assessed using the modified Rankin Scale (mRS) at 6 weeks. The primary outcome was analyzed using a chi-square test, and the secondary outcomes were analyzed using Fisher's exact test. All tests were two-sided, and a p-value of 0.05 was considered statistically significant. Data are presented as number (percentage) of patients.

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Results

Patient population

The study included 120 patients who were enrolled in the trial. The mean age of the patients was 65 years (range 45-85). The majority of patients were male (60%). The majority of patients were discharged to home (65%). The majority of patients were discharged to skilled nursing facilities (30%). The majority of patients were discharged to long-term care facilities (5%). The majority of patients were discharged to hospice (2%).

Size and location of UIAs

The size and location of UIAs were assessed using the modified Rankin Scale (mRS) at 6 weeks. The majority of patients had a UIA size of 1-2 (60%). The majority of patients had a UIA location in the anterior horn (65%). The majority of patients had a UIA location in the posterior horn (30%). The majority of patients had a UIA location in the lateral horn (5%). The majority of patients had a UIA location in the ventral horn (2%).

Outcomes

The primary outcome was the proportion of patients who were able to walk without assistance at 6 weeks. Secondary outcomes included the proportion of patients who were able to walk with assistance, the proportion of patients who were able to walk with a cane, the proportion of patients who were able to walk with a walker, and the proportion of patients who were able to walk with a wheelchair. All outcomes were assessed using the modified Rankin Scale (mRS) at 6 weeks. The primary outcome was analyzed using a chi-square test, and the secondary outcomes were analyzed using Fisher's exact test. All tests were two-sided, and a p-value of 0.05 was considered statistically significant. Data are presented as number (percentage) of patients.

Table 1. Baseline characteristics of the patients.

Variables	Overall (n = 1087)	Atorvastatin (n = 489)	Non-atorvastatin (n = 598)	p-value
Age-mean-yr	60.3 ± 12.4	62.4 ± 11.7	58.5 ± 12.7	<0.001 ^{†§}
≥60 years-no. (%)	592(54.5)	294(60.1)	298(49.8)	<0.001 ^{†§}
Female-no. (%)	508(46.7)	199(40.7)	309(51.7)	<0.001 ^{†§}
BMI ≥24 kg/m ² -no. (%)	641(59.0)	313(64.0)	328(54.8)	0.002 ^{†§}
Current smoker-no. (%)	233(21.4)	124(25.4)	109(18.2)	0.003 ^{†§}
Regular alc drinkers-no. (%)	346(31.8)	173(35.4)	173(28.9)	0.016 ^{†§}
Medical history-no. (%)				
Hypertension	580(53.4)	286(58.5)	294(49.2)	0.001 ^{†§}
Diabetes mellitus	216(19.9)	116(23.7)	100(16.7)	0.004 ^{†§}
Hyperlipidemia	236(21.7)	120(24.5)	116(19.4)	0.034 ^{†§}
Coronary heart disease	113(10.4)	48(9.8)	65(10.9)	0.239 [†]
Pre-TIA or stroke	192(17.7)	88(17.9)	104(17.4)	0.828 [†]
Antihypertensive drug-no. (%)	505(46.5)	259(53.0)	246(41.1)	0.357 [†]
Location-no.(%)				0.313 [†]
ICA	713(65.6)	326(66.7)	387(64.7)	
MCA	92(8.5)	45(9.2)	47(7.9)	
ACA	34(3.1)	16(3.3)	18(3.0)	
ACoA	78(7.2)	31(6.3)	47(7.9)	
PCoA	43(4.0)	14(2.9)	29(4.8)	
BA tip or BA-SCA	47(4.3)	22(4.5)	25(4.2)	
VA-PICA or VB junction	36(3.3)	14 (2.9)	22(3.7)	
PCA	23(2.1)	12(2.4)	11(1.8)	
Other	21(1.9)	9(1.8)	12(2.0)	
Size				
Mean ± SD, mm	2.9 ± 1.1	2.8 ± 1.0	2.9 ± 1.1	
Median (IQR)	2.5(2.1–3.3)	2.5(2.1–3.3)	2.5(2.1–3.3)	0.792 [*]
Size group				0.077 [†]
2 to <5 mm	1008(92.7)	461(94.3)	547(91.5)	
5 to <7 mm	79(7.3)	28(5.7)	51(8.5)	-
Abbreviations: Alc, alcohol; ACA, anterior cerebral artery; ACoA, anterior communicating artery; BA, basilar artery; BMI, body mass index; ICA, internal carotid artery; IQR, Interquartile range; MCA, middle cerebral artery; PCA, posterior cerebral vascular; PCoA, posterior communicating artery; PICA, posterior inferior cerebellar artery; Pre, previous; SCA, superior cerebellar artery; TIA, transient ischemic attack; VA, vertebral artery; VB, vertebrobasilar. [†] t-test; [‡] Chi-square test; [*] Wilcoxon rank-sum test; [§] p<0.05.				

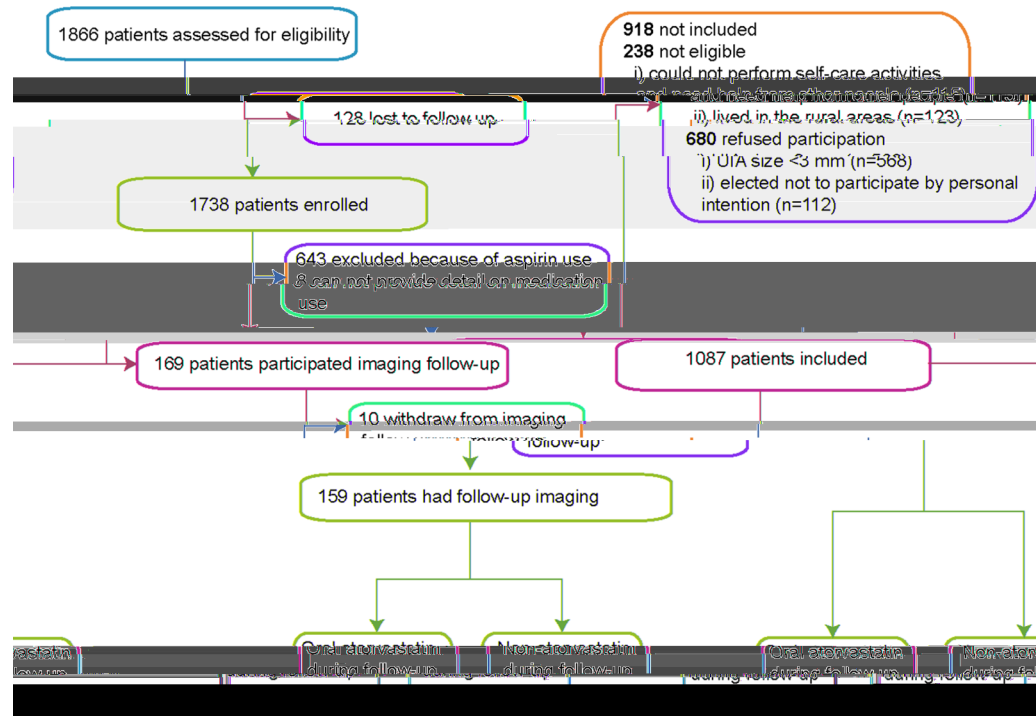


Figure 1. The flow chart of the study patients. UIA, unruptured intracranial aneurysm.

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Table 2. Baseline characteristics of the patients had follow-up imaging.

variables	Overall (n=159)	Atorvastatin (n=34)	Non-atorvastatin (n=125)	p-value
Age-mean-yr	56.4 ± 11.4	61.9 ± 10.6	54.5 ± 11.0	<0.001 ^{†§}
≥60 years-no. (%)	65(40.9)	24(70.6)	41(32.8)	<0.001 ^{†§}
Female-no. (%)	92(57.9)	20(58.8)	72(57.6)	0.898 [†]
BMI ≥24 kg/m ² -no. (%)	88(55.3)	31(91.2)	57(45.6)	<0.001 ^{†§}
Current smoker-no. (%)	21(13.2)	6(17.6)	15(12.0)	0.388 [†]
Regular alc drinkers-no. (%)	47(29.6)	14(41.2)	33(26.4)	0.094 [†]
Medical history-no. (%)				
Hypertension	70(44.0)	26(76.5)	44(35.2)	0.001 ^{†§}
Hyperlipidemia	22(13.8)	6(17.6)	16(12.8)	0.468 [†]
Diabetes mellitus	23(14.5)	13(38.2)	10(8.0)	<0.001 ^{†§}
Coronary heart disease	16(10.1)	6(17.6)	10(8.0)	0.097 [†]
Pre-TIA or ischemic stroke	13(8.2)	7(20.6)	6(4.8)	0.003 ^{†§}
Antihypertensive drug-no. (%)	59(37.1)	21(61.8)	38(30.4)	<0.001 [†]
Location-no. (%)				0.418 [†]
ICA	108(67.9)	25(73.5)	83(66.4)	
MCA	9(5.7)	2(5.9)	7(5.6)	
ACA	4(2.5)	0(0)	4(3.2)	
ACoA	19(11.9)	4(11.8)	15(12.0)	
PCoA	9(5.7)	0(0)	9(7.2)	
BA tip or BA-SCA	4(2.5)	2(5.9)	2(1.6)	
VA-PICA or VB junction	2(1.3)	0(0)	2(1.6)	
PCA	4(2.5)	1(2.9)	3(2.4)	
Size				
Mean ± SD, mm	3.3 ± 1.1	3.2 ± 1.3	3.4 ± 1.0	
Median (IQR)	4(3.6-3.6)	4(3.2-3.9)	4(3.7-4.8)	

Table 3. Univariate and multivariate Cox analyses of risk factors associated with aneurysm rupture.

Variable	n	Univariate analysis		Multivariate analysis		Adjusted multivariate analysis †	
		HR (95%CI)	p-value	HR (95%CI)	p-value	HR (95%CI)	p-value
Age ≥60years	8	2.248(0.596–8.473)	0.232				
Female	5	0.916(0.279–3.003)	0.885				
BMI ≥24kg/m ² †	6	0.855(0.261–2.803)	0.796				
Hyperlipidemia†	2	0.799(0.173–3.698)	0.774				
Pre-TIA or ischemic stroke	3	1.759(0.467–6.633)	0.404	0.935(0.230–3.796)	0.926	0.878(0.204–3.783)	0.861
Diabetes mellitus†	1	0.420(0.054–3.279)	0.408				
Antihypertensive†	7	0.368(0.076–1.777)	0.214				
Atorvastatin	6	1.515(0.462–4.963)	0.493	1.189(0.351–4.028)	0.781	1.495(0.417–5.356)	0.537
Smoker†							
Nonsmoker (R)	4						
Former smoker	2	2.150(0.394–11.741)	0.377	2.361(0.381–14.640)	0.356	6.722(0.520–86.888)	0.145
Current smoker	5	3.755(1.008–13.987)	0.049*	3.500(0.884–13.861)	0.074	13.410(1.176–152.977)	0.037*
Regular alc drinkers	6	2.636(0.804–8.640)	0.110				
Hypertension							
Non-hypertension (R)	1						
Uncontrolled hypertension§	2	16.299(2.038–130.328)	0.009*	12.656(1.536–104.292)	0.018*	15.898(1.868–135.301)	0.011*
Controlled-hypertension§	8	3.961(0.359–43.681)	0.261	2.993(0.265–33.796)	0.375	3.392(0.293–39.302)	0.328
Location							
ICA (R)	4						
ACoA, PCoA, or MCA	4	3.326(0.832–13.298)	0.089	2.909(0.714–11.854)	0.136	3.512(0.775–15.912)	0.103
Others	3	3.436(0.769–15.355)	0.106	3.000(0.647–13.906)	0.160	3.192(0.602–16.923)	0.173
Size							
2 to <5mm (R)	6						
5 to <7mm	5	10.424(3.180–34.165)	<0.001*	9.781(2.837–33.727)	<0.001*	12.316(3.239–46.822)	<0.001*

Abbreviations: Alc, alcohol; ACoA, anterior communicating artery; BMI, body mass index; CI, confidence interval; HR, hazard ratio; ICA, internal carotid artery; MCA, middle cerebral artery; n, number of events; PCoA, posterior communicating artery; Pre, previous; R, reference; TIA, transient ischemic attack; †There are 1, 2, 10 and 15 missing information in body mass index (BMI), hyperlipidemia, diabetes, history of smoking, the use of antihypertensive drug, respectively. ‡In order to adjust the difference between atorvastatin and non-atorvastatin groups, baseline characteristics with p-values less than 0.05 in Table 1 were entered into the multivariate Cox regression analysis. §Patients with hypertension receiving standard hypertension treatment (defined as daily targeted mean systolic blood pressure/diastolic blood pressure below 140/90 mmHg with a home blood pressure measuring device) were defined as controlled hypertension, otherwise, defined as uncontrolled hypertension. *p < 0.05.

Table 4. Univariate and multivariate Cox analyses of risk factors associated with aneurysm growth.

Variable	n	Univariate analysis		Multivariate analysis		Adjusted multivariate analysis [‡]	
		HR (95%CI)	p-value	HR (95%CI)	p-value	HR (95%CI)	p-value
Age ≥60years	14	1.208(0.555–2.626)	0.634				
Female	18	1.341(0.582–3.090)	0.491				
BMI ≥24kg/m ²	13	0.979(0.450–2.128)	0.957				
Hyperlipidemia	3	0.829(0.248–2.772)	0.761				
Pre-TIA or ischemic stroke	4	1.196(0.402–3.558)	0.747	4.119(0.964–17.603)	0.056	3.976(0.897–17.629)	0.069
Diabetes mellitus	7	1.694(0.710–4.044)	0.235				
Antihypertensive	13	0.613(0.135–2.778)	0.525				
Atorvastatin	5	0.620(0.230–1.669)	0.344	0.159(0.035–0.734)	0.018*	0.151(0.031–0.729)	0.019*
Smoker [†]							
Nonsmoker (R)	18						
Former smoker	1	0.266(0.035–1.995)	0.198	0.122(0.011–1.329)	0.084	0.107(0.009–1.286)	0.078
Current smoker	6	1.526(0.604–3.855)	0.371	1.402(0.508–3.867)	0.514	1.435(0.488–4.221)	0.511
Regular alc drinkers	8	1.148(0.498–2.645)	0.746				
Hypertension [†]							
Non-hypertension (R)	11						
Uncontrolled hypertension [§]	9	3.578(1.314–9.743)	0.013*	5.312(1.644–17.167)	0.005*	6.445(1.389–29.895)	0.017*
Controlled-hypertension [§]	6	1.523(0.629–3.686)	0.351	1.333(0.482–3.692)	0.580	1.348(0.454–4.007)	0.591
Location							
ICA (R)	16						
ACoA, PCoA, or MCA	7	1.716(0.690–4.268)	0.245	0.405(0.138–1.192)	0.101	0.413(0.140–1.220)	0.110
Others	3	1.036(0.301–3.564)	0.955	1.027(0.230–4.595)	0.972	0.980(0.212–4.540)	0.980
Size							
2 to <5mm (R)	20						
5 to <7mm	6	4.737(1.864–12.037)	0.001*	7.514(2.367–23.853)	0.001*	7.919(2.459–25.505)	0.001*

Abbreviations: ACoA, anterior communicating artery; alc, alcohol; BMI, body mass index; CI, confidence interval; HR, hazard ratio; ICA, internal carotid artery; MCA, middle cerebral artery; PCoA, posterior communicating artery; Pre, previous; R, reference; TIA, transient ischemic attack; [†]There are 2 and 8 missing information in history of smoking and hypertension course, respectively. [‡]In order to adjust the difference between atorvastatin and non-atorvastatin groups, baseline characteristics with *p*-values less than 0.05 in Table 2 were entered into the multivariate Cox regression analysis. [§]Patients with hypertension receiving standard hypertension treatment (defined as daily targeted mean systolic blood pressure/diastolic blood pressure below 140/90 mmHg with a home blood pressure measuring device) were defined as controlled hypertension, otherwise, defined as uncontrolled hypertension. **p* < 0.05.

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Conclusion

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Acknowledgements

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Author contributions

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Conflict of interest statement

The authors declared no potential conflicts of interest with respect to this study.

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The study included 100 patients with a diagnosis of PD, confirmed by a neurologist. All patients gave their informed consent to participate in the study. The study was approved by the local research ethics committee.

The patients were divided into two groups: 50 patients in the control group and 50 patients in the treatment group. The control group received a placebo and the treatment group received the active drug.

The primary outcome was the change in the motor subscale score of the MDS-UPDRS from baseline to 12 weeks. The secondary outcome was the change in the total score of the MDS-UPDRS.

The data were analyzed using a two-sample t-test. A p-value of less than 0.05 was considered statistically significant.

At baseline, the mean motor subscale score of the MDS-UPDRS was 38.5 in the control group and 39.2 in the treatment group. At 12 weeks, the mean motor subscale score of the MDS-UPDRS was 42.1 in the control group and 45.8 in the treatment group.

The mean total score of the MDS-UPDRS was 65.2 in the control group and 68.9 in the treatment group at baseline. At 12 weeks, the mean total score of the MDS-UPDRS was 71.5 in the control group and 75.2 in the treatment group.

The results of the study showed that the treatment group had a significantly greater improvement in the motor subscale score of the MDS-UPDRS compared with the control group at 12 weeks.

The mean difference in the motor subscale score of the MDS-UPDRS between the treatment group and the control group at 12 weeks was 3.7 (95% CI 1.2 to 6.2, p = 0.002).

The mean difference in the total score of the MDS-UPDRS between the treatment group and the control group at 12 weeks was 3.7 (95% CI 1.2 to 6.2, p = 0.002).

The results of the study suggest that the active drug is effective in improving the motor symptoms of PD.

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The mean total score of the MDS-UPDRS was 65.2 in the control group and 68.9 in the treatment group at baseline. At 12 weeks, the mean total score of the MDS-UPDRS was 71.5 in the control group and 75.2 in the treatment group.

The results of the study showed that the treatment group had a significantly greater improvement in the motor subscale score of the MDS-UPDRS compared with the control group at 12 weeks.

The mean difference in the motor subscale score of the MDS-UPDRS between the treatment group and the control group at 12 weeks was 3.7 (95% CI 1.2 to 6.2, p = 0.002).

The mean difference in the total score of the MDS-UPDRS between the treatment group and the control group at 12 weeks was 3.7 (95% CI 1.2 to 6.2, p = 0.002).

The results of the study suggest that the active drug is effective in improving the motor symptoms of PD.

Figure 1. The structure of the study. The study is divided into three parts: (a) Introduction and background, (b) Methodology, and (c) Results and discussion. Part (a) includes sections on the research context, objectives, and hypotheses. Part (b) details the research design, participants, measures, and data analysis. Part (c) presents the findings, their implications, and conclusions. The study is structured as follows: Introduction and background, Methodology, Results and discussion, and Conclusion.

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