Comparison of apolipoprotein A1 and carotid artery ultrasonography findings among stroke subtypes and controls

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Background and Aims

Treatment with statins reduces the ratio of cardiovascular events in high-risk patients, but residual risk persist.

Apolipoprotein A1 (ApoA-1), which is a structural protein of high-density lipoprotein cholesterol, may be associated with the generative pathology of ischemic stroke.

The aim of this study was to investigate ApoA-1 and carotid artery ultrasonography findings among ischemic stroke subtypes and control.

Methods

We enrolled a total of 86 patients with stroke and age matched controls（CONT, n=20）.

The stroke patients were divided into 3 subtypes :

- cardio-embolic infarction (CE, n=13)
- atherothrombotic infarction (ATBI, n=36)
- lacunar infarction (LI, n=17)

We measured the total number of plaque, and plaque thickness, and the carotid intima-media thickness (IMT), which is a surrogate marker of subclinical atherosclerosis, at the common carotid artery (CCA), carotid bifurcation (BIF), and internal carotid artery (ICA) using B-mode ultrasound.

Carotid plaques were classified into 4 types :

low intensity, intermediate intensity, high intensity, and mixed type.

Serum lipids (TC, LDL-C, HDL-C, TG), LP(a), apolipoproteins (A1, A2, B, C2, C3, and E), RLP-C, MDA-LDL, HbA1c were analyzed using commercially available kit. Furthermore, HDL-C/ApoA-1, LDL-C/ApoB were calculated.

Results

	ATBI	CE	LI	CONT
n	40	13	16	20
% female	58.3	69.2	58.8	35.0
age	71.0 ± 9.6	64.7 ± 13.5	71.8 ± 12.0	67.6 ± 8.2
BMI	23.9 ± 3.4	22.4 ± 3.1	22.7 ± 2.6	23.8 ± 3.9
SBP	129.0 ± 17.4$^{\#}$	125.5 ± 17.2$^{\#}$	144.1 ± 20.0	132.8 ± 14.7
DBP	70.7 ± 10.6	74.9 ± 10.2	78.8 ± 15.8	74.9 ± 12.5
% smoking	32.4	23.1	18.8	15.8
% alcohol drinking	27.0	38.5	37.5	15.8
% dyslipidemia	97.5$^{※}$	92.3	81.3	75.0
% diabetes	42.5	15.4	31.2	20.0
% hypertension	87.5$^{※}$	53.8	87.5	50.0
% AF	10.0§	100$^{\#※}$	0	0
% statin drug	87.5	61.5	56.3	70.0

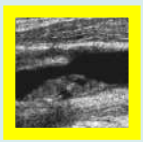
$^{§}P < 0.05$, *vs.* CE, $^{\#}P < 0.05$, *vs.* LI; $^{※}P < 0.05$, *vs.* CONT.

	ATBI	CE	LI	CONT
TC	180.3 ± 36.0	191.5 ± 40.5	197.9 ± 34.4	206.2 ± 30.4
TG	152.2 ± 90.6	161.3 ± 116.6	120.5 ± 75.4	144.7 ± 63.8
HDL-C	54.3 ± 13.0	55.9 ± 17.9	65.3 ± 20.1	66.7 ± 24.2
LDL-C	105.0 ± 29.6	111.2 ± 29.4	116.2 ± 37.3	119.1 ± 33.0
Lp (a)	17.5 ± 22.9	23.8 ± 34.8	27.7 ± 41.5	15.8 ± 11.9
ApoA-1	127.5 ± 21.0$^{※}$	133.4 ± 25.5	133.8 ± 19.0	148.7 ± 22.1
ApoA-2	27.2 ± 4.9	29.9 ± 5.9	26.1 ± 4.0	30.2 ± 4.8
ApoB	77.1 ± 18.4	87.6 ± 19.1	76.4 ± 30.8	76.4 ± 9.6
ApoC-2	4.3 ± 1.7	5.9 ± 2.5	4.4 ± 2.6	5.0 ± 1.7
ApoC-3	9.6 ± 4.5	11.6 ± 3.4	8.7 ± 3.0	10.9 ± 3.1
ApoE	3.2 ± 1.3	3.8 ± 1.1	3.2 ± 0.8	3.4 ± 0.8
HDL-C/ApoA-1	0.42 ± 0.06	0.40 ± 0.08	0.44 ± 0.09	0.46 ± 0.10
LDL-C/ApoB	1.3 ± 0.2$^{\#}$	1.3 ± 0.2	1.6 ± 0.6	1.5 ± 0.3
RLP-Cho	7.3 ± 8.1	10.1 ± 7.8	6.5 ± 4.3	6.9 ± 3.2
MDA-LDL	103.8 ± 34.9	129.8 ± 53.3	108.2 ± 53.2	93.2 ± 19.0
HbA1c	6.6 ± 1.4	6.1 ± 0.9	6.3 ± 1.4	6.2 ± 0.7

$^{\#}P < 0.05$, *vs.* LI, $^{※}P < 0.05$, *vs.* CONT.

	ATBI	CE	LI	CONT
CCA, number	1.6 ± 1.5$^{§,\#,※}$	0.3 ± 1.2	0.0 ± 0.0	0.2 ± 0.8
CCA, mm	2.2 ± 1.2	2.4	-	1.7 ± 0.4
BIF, number	2.4 ± 1.3$^{§,\#,※}$	0.3 ± 1.2	0.3 ± 1.1	0.4 ± 1.0
BIF, mm	2.1 ± 1.2	2.1	1.6	2.1 ± 0.2
ICA, number	1.9 ± 1.3$^{§,\#,※}$	0.3± 1.2	0.0 ± 0.0	0.1 ± 0.5
ICA, mm	2.7 ± 1.1	1.9	-	1.2 ± 1.6
Stenosis,%	10.8	8.3	0	0
PSV	62.1 ± 17.6	60.2 ± 15.4	68.2 ± 16.7	65.3 ± 19.6
EDV	13.5 ± 4.7	13.0 ± 3.3	14.9 ± 3.0	16.5 ± 5.9
meanV	25.5 ± 7.0	24.8 ± 6.1	29.0 ± 5.5	30.4 ± 8.5
PI	2.5 ± 2.3	1.9 ± 0.4	1.9 ± 0.4	1.7 ± 0.7
IMT CCA	0.9 ± 0.3	0.8 ± 1.0	0.7 ± 0.2	0.8 ± 0.2
No plaque ,%	8.1$^{§,\#,※}$	84.6	86.6	78.9
Soft, %	5.6$^{§,\#,※}$	0	0	0
Inter,%	35.1$^{§,\#,※}$	7.7	6.7	0
Mixed,%	35.1$^{§,\#,※}$	0	0	10.5
Hard,%	16.2$^{§,\#,※}$	0	0	0

One-way analysis of variance followed by a Scheffe test for post hoc analysis, and the chi-square test, $^{§}P < 0.05$, *vs.* CE ; $^{\#}P < 0.05$, *vs.* LI ; $^{※}P < 0.05$, *vs.* CONT.



Soft & Mixed

Hard

Analyzed plaques of ATBI using B-mode ultrasound

	Soft + Mixed	Intermediate	Hard
n	16	14	6
% female	37.5	33.3	80.0
age	72.6 ± 9.6	70.3 ± 7.5	75.8 ± 10.0
BMI	23.2 ± 3.2	24.5 ± 3.1	22.9 ± 5.1
SBP	130.8 ± 21.8	132.2 ± 15.0	118.6 ± 10.8
DBP	70.9 ± 10.4	71.7 ± 9.6	61.0 ± 10.3
% smoking	33.3	26.7	0
% alcohol drinking	33.3	20.0	0
% dyslipidemia	93.8	100	100
% diabetes	31.2	60.0	40.0
% hypertension	93.8	86.7	100
% AF	12.5	6.7	20.0
% statin drug	87.5	80.0	100

	Soft + Mixed	Intermediate	Hard
TC	175.0 ± 41.2	182.1 ± 33.4	177.2 ± 16.8
TG	131.4 ± 99.0	150.1 ± 59.1	161.0 ± 91.7
HDL-C	54.2 ± 11.5	54.1 ± 13.5	49.8 ± 14.3
LDL-C	103.2 ± 29.0	107.9 ± 32.3	101.8 ± 21.4
Lp (a)	24.2 ± 31.5	14.8 ± 17.1	13.8 ± 10.5
ApoA-1	123.5 ± 20.1	130.5 ± 26.6	123.3 ± 9.1
ApoA-2	26.3 ± 5.3	28.1 ± 5.7	26.2 ± 3.4
ApoB	73.9 ± 18.2	78.3 ± 19.4	81.5 ± 22.0
ApoC-2	3.6 ± 1.2	5.0 ± 1.9	4.3 ± 1.8
ApoC-3	7.7 ± 2.7	10.7 ± 4.6	9.3 ± 4.8
ApoE	3.0 ± 1.4	3.1 ± 0.9	3.6 ± 1.7
HDL-C/ApoA-1	0.44 ± 0.04	0.41 ± 0.05	0.36 ± 0.05$^{※}$
LDL-C/ApoB	1.4 ± 0.2	1.3 ± 0.2	1.4 ± 0.4
RLP-Cho	6.8 ± 10.5	6.6 ± 4.0	8.4 ± 6.6
MDA-LDL	91.4 ± 31.7	115.1 ± 38.4	100.8 ± 17.3
HbA1c	6.3 ± 1.0	6.9 ± 1.9	6.7 ± 1.1

$^{※}P < 0.05$, *vs.* Soft + Mixed

Conclusion

These findings suggest that patients with ATBI had a lot of and thick plaques at carotid artery and serum ApoA-1 and LDL-C/ApoB levels might be associated with the development of ATBI. Additionally, HDL-C/ApoA-1 levels might be associated with the development of the hard plaque formations of carotid arteries.

11th World Stroke Congress, Montreal CANADA
October 17-20, 2018