Previous treatment with NOAC is associated with slower ultra-early intracranial hematoma growth compared with vitamin K antagonists

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BACKGROUND

Ultra-early hematoma growth (UHG) estimates the initial hematoma expansion rate in acute non-traumatic intracranial hemorrhage (ICH), representing a marker of its hemostatic properties.

Patients with ICH and oral anticoagulation are at particular risk of hematoma expansion, however the impact of different anticoagulants has not yet been fully ascertained.

AIMS

To compare UHG in patients on NOAC and vitamin K antagonists (VKA).

METHODS

Multicentre and retrospective cohort study;

A total of 131 patients were included with acute non-traumatic ICH on oral anticoagulation admitted during 30-months period, with baseline ICH volume and time from symptom-onset to first CT-scan available for analysis;

Medical records were analysed for demographic, clinical, laboratory and imaging data;

UHG was defined as the relation between baseline ICH volume (mL)/time of symptom-onset to first CT-scan (hours), with volume obtained by planimetry (ITK-SNAP);

Univariate analysis (Pearson’s chi-square/Fisher’s exact test for categorical variables and Student’s t-test/Mann-Whitney U test for continuous variables) and propensity score matching (PSM) analysis (adjusted for age, elevated admission systolic BP and anticoagulation status) were performed;

Statistical significance p<0.05.

RESULTS

Of the 131 patients, 18 (13.7%) were anticoagulated with a NOAC (11 on dabigatran and 7 on rivaroxaban) and 113 (86.3%) with a VKA.

Characteristics of the cohort according to type of anticoagulant were demonstrated in the table. Values represent n (%) or median (interquartile range).

	VKA (n=113)	NOAC (n=18)	P Value
Demographic characteristics			
Age	75.0 (68.0-81.0)	82.5 (75.6-85.0)	0.003
Female sex	52 (46%)	9 (50%)	0.753
Previous medical history			
Arterial hypertension	99 (87.6%)	18 (100%)	0.215
Diabetes mellitus	27 (23.9%)	3 (16.7%)	0.763
Dyslipidemia	50 (44.2%)	7 (38.9%)	0.670
Coronary heart disease	8 (7.1%)	3 (16.7%)	0.177
Cardiac failure	21 (18.8%)	1 (5.6%)	0.307
Mechanic heart valve	9 (8.0%)	0 (0.0%)	0.610
Peripheral artery disease	1 (0.9%)	0 (0.0%)	0.863
Excessive weight	12 (11.7%)	2 (11.1%)	0.654
Smoking	7 (6.7%)	0 (0.0%)	0.592
Previous ischemic stroke	20 (17.7%)	5 (27.8%)	0.337
Previous hemorrhagic stroke	3 (2.7%)	1 (5.6%)	0.450

RESULTS

	VKA (n=113)	NOAC (n=18)	P Value
Clinical Score			
CHA ₂ DS ₂ Vasc	4.0 (3.0-5.0)	4.0 (3.5-6.0)	0.033
HASBLED	3.0 (2.0-3.0)	2.5 (2.0-3.3)	0.866
Admission evaluation			
NIHSS	10.5 (5.0-18.0)	10.2 (5.0-15.5)	0.668
Glasgow Coma Scale	13.0 (11.0-15.0)	13.5 (11.0-14.25)	0.913
Systolic BP (mmHg)	159.5 (±28.7)	180.1 (±17.7)	0.005
Reason for anticoagulation treatment			
Non-valvular AF	86 (76.1%)	16 (88.9%)	0.360
Baseline CT characteristics			
Deep localization	73 (64.6%)	11 (61.1%)	0.774
Intraventricular rupture	66 (58.4%)	9 (50.0%)	0.503
Posterior fossa	23 (20.4%)	5 (27.8%)	0.537
Hemorrhage volume (mL)	18.2 (4.3-46.8)	16.6 (4.3-35.4)	0.756
Time from symptom to CT scan (hours)	7.0 (2.5-15.1)	7.5 (3.4-23.0)	0.420

On univariate analysis:

Median UHG did not show significant differences (VKA 2.42 mL/h vs. NOAC 1.93 mL/h, p=0.470).

On PSM analysis (with a match in 18 NOAC- and 18 VKA-treated patients):

Median UHG* was higher in VKA-treated patients (VKA 5.73 mL/h vs. NOAC 1.93 mL/h, *p=0.026);

Hemorrhage volume was higher in VKA-treated patients (VKA 27.05 mL vs. NOAC 16.6 mL, p=0.195)

CONCLUSION

UHG in VKA- was higher when compared to NOAC-treated patients using matching statistics (when considering groups with similar age and admission systolic BP).

Our study suggests that NOAC-treated patients may have an initial slower rate of hematoma expansion comparing to VKA-treated patients.

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