

Anticoagulant Related Intracerebral Hemorrhage Outcome

Randomized trials in patients with [atrial fibrillation](#) (AF) show that direct [non vitamin K oral anticoagulants](#) (NOACs) have about half the incidence of [intracerebral hemorrhage](#) (ICH) compared to VKA but with a similar efficacy in preventing ischemic stroke ¹⁾.

In a international collaborative multicenter pooled analysis, baseline ICH volume, hematoma expansion, 90-day mortality, and functional outcome were similar following NOAC-ICH and VKA-ICH ²⁾.

The aim of a study was to prospectively validate prior findings of smaller hematoma volume and lesser neurological deficit in [Non vitamin K oral anticoagulant](#) (NOAC) compared with [Vitamin K antagonist](#) (VKA)-related intracerebral hemorrhage (ICH).

It was a prospective 12-month observational study in 15 tertiary [stroke centers](#) in the [United States](#), [Europe](#), and [Asia](#). Consecutive patients with premorbid [modified Rankin Scale](#) score of <2 with acute nontraumatic anticoagulant-related ICH divided into 2 groups according to the type of anticoagulant: NOAC versus VKA.

They recorded baseline ICH volume, significant hematoma expansion (absolute [12.5 mL] or relative [>33%] increase), neurological severity measured by National Institutes of Health Stroke Scale score, 90-day mortality, and functional status (modified Rankin Scale score).

The cohort comprised 196 patients, 62 NOAC related (mean age, 75.0 ± 11.4 years; 54.8% men) and 134 VKA related (mean age, 72.3 ± 10.5 ; 73.1% men). There were no differences in vascular comorbidities, antiplatelet, and statin use; NOAC-related ICH patients had lower median baseline hematoma volume (13.8 [2.5-37.6] versus 19.5 [6.6-52.0] mL; $P=0.026$) and were less likely to have severe neurological deficits (National Institutes of Health Stroke Scale score of >10 points) on admission (37% versus 55.3%, $P=0.025$). VKA-ICH were more likely to have significant hematoma expansion (37.4% versus 17%, $P=0.008$). NOAC pretreatment was independently associated with smaller baseline hematoma volume (standardized linear regression coefficient: -0.415 [95% CI, -0.780 to -0.051]) resulting in lower likelihood of severe neurological deficit (odds ratio, 0.44; 95% CI, 0.22-0.85) in multivariable-adjusted models.

Patients with NOAC-related ICH have smaller baseline hematoma volumes and lower odds of severe neurological deficit compared with VKA-related ICH. These findings are important for practicing clinicians making anticoagulation choices ³⁾.

Serotonin-modulating antidepressants (selective serotonin reuptake inhibitors [SSRIs], serotonin-norepinephrine reuptake inhibitors [SNRIs]) are frequently used in combination with warfarin, but it is unclear whether this combination of drugs influences outcome after primary intracerebral hemorrhage (PICH).

Concurrent use of warfarin and a serotonin-modulating antidepressant, relative to warfarin alone, seemed to increase the case fatality rate for PICH. This finding should be taken into account if

hematoma evacuation is planned ⁴⁾.

Whether **intracerebral hemorrhage** (ICH) survivors should restart antithrombotic drugs is unknown.

1)

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