# Apixaban

- APOE epsilon4 and Risk of Intracranial Hemorrhage in Patients With Atrial Fibrillation Taking Apixaban
- Intrinsic Activation of the Coagulation Pathway Induces Mouse Sciatic Nerve Hypo-Excitability
- Non-vitamin K antagonist oral anticoagulants (NOACs) and risk of spontaneous intracranial hemorrhage in patients with ischemic stroke: An analysis using Taiwan's National Health Insurance Research Database
- Management and Outcomes of Intracranial Hemorrhage in Atrial Fibrillation Patients: Highlighting Practices in Saudi Arabia
- Perioperative management of direct oral anticoagulants in patients having a high-bleed-risk surgery or neuraxial procedure: the Perioperative Anticoagulant Use for Surgery Evaluation (PAUSE)-2 pilot randomized trial
- A Case of Extended Duration Triple Antithrombotic Therapy in a Patient With an Intracranial Stent and Atrial Fibrillation
- Anticoagulation Reversal in Intracerebral Hemorrhage: A Case Report on the Efficacy of Andexanet Alfa in an Apixaban-Treated Patient
- The rate of postoperative hematoma following risk-adapted cessation of oral anticoagulation in patients undergoing endoscopic endonasal surgery for pituitary adenomas

Apixaban is a direct oral anticoagulant (DOAC) that belongs to the class of medications known as factor Xa inhibitors. It is used for anticoagulation therapy to reduce the risk of blood clots and strokes in certain medical conditions.

It reversibly blocks the enzymatic function of factor Xa in converting prothrombin to thrombin.

## Apixaban in Neurosurgery

If the patient is on apixaban at a prophylactic dose, it should be discontinued 26 to 30 hours before a neuraxial puncture or catheter placement. May be restarted 4 to 6 hours after the puncture or catheter removal/manipulation.

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The benefit of apixaban over aspirin for the prevention of recurrent cerebral ischemia is under current investigation [1].

Currently, conventional heparin and warfarin remain first choice anticoagulants. If newer anticoagulants are considered, although study numbers are small, at this stage Apixaban appears to be associated with lesser bleeding risk than Rivaroxaban [2].

## Discontinuation

Apixaban Discontinuation.

Compared with warfarin, the direct oral anticoagulant apixaban reduces the risk of stroke or systemic embolism, intracranial haemorrhage, and case fatality in patients with atrial fibrillation. Compared with aspirin, apixaban reduces the risk of stroke or systemic embolism in patients with atrial fibrillation, and has a similar risk of intracerebral haemorrhage. Novel oral anticoagulants have not been evaluated in patients with atrial fibrillation and a recent intracerebral haemorrhage. To inform a phase III trial, the phase II Apixaban versus Antiplatelet drugs or no antithrombotic drugs after anticoagulation-associated intraCerebral HaEmorrhage in patients with Atrial Fibrillation (APACHE-AF) trial aims to obtain estimates of the rates of vascular death or non-fatal stroke in patients with atrial fibrillation and a recent anticoagulation-associated intracerebral haemorrhage treated with apixaban and in those in whom oral anticoagulation is avoided.

APACHE-AF is a phase II, multicentre, open-label, parallel-group, randomised clinical trial with masked outcome assessment. One hundred adults with a history of atrial fibrillation and a recent intracerebral haemorrhage during treatment with anticoagulation in whom clinical equipoise exists on the optimal stroke prevention strategy will be enrolled in 14 hospitals in The Netherlands. These patients will be randomly assigned in a 1:1 ratio to either apixaban or to avoiding oral anticoagulation. Patients in the control group may be treated with antiplatelet drugs at the discretion of the treating physician. The primary outcome is the composite of vascular death or non-fatal stroke during follow-up. We aim to include 100 patients in 2.5 years. All patients will be followed-up for the duration of the study, but at least for 1 year. Recruitment commenced in September 2014 and is ongoing. This trial is funded by the Dutch Heart Foundation (2012 T077) and ZonMW (015008048) [3].

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Prior antiplatelet therapy for large vessel occlusion (LVO) in patients with non-valvular atrial fibrillation (NVAF) newly initiated on apixaban was associated with major bleeding, which was more frequent in the antiplatelet group without intravenous thrombolysis (IVT). [4].

## Hematoma expansion with Apixaban

- Association of Biomarkers With Intracerebral Hematoma Expansion and Arterial Thromboembolic Events in Patients With Acute Intracranial Hemorrhage: The ANNEXA-I Biomarker Substudy
- Efficacy of pro-haemostatic agents in the management of factor Xa inhibitor-associated intracranial haemorrhages
- Andexanet alfa in patients with factor Xa inhibitor-associated intracranial hemorrhage: The prospective observational multicenter ASTRO-DE study
- Acute internal carotid artery occlusion following administration of Andexanet alfa for the reversal of direct factor Xa inhibitors in patients with cerebral hemorrhage
- Co-administration of Four-Factor Prothrombin Complex Concentrate With Andexanet alfa for Reversal of Nontraumatic Intracranial Hemorrhage
- Activated Prothrombin Complex Concentrates for the Treatment of Factor Xa Inhibitor-Associated Spontaneous Intracerebral Hemorrhage
- Administration of andexanet alfa for traumatic intracranial hemorrhage in the setting of massive apixaban overdose: A case report
- A Combination of Ex Vivo and In Vivo Strategies for Evaluating How Much New Oral Anticoagulants Exacerbate Experimental Intracerebral Bleeding

A case report describes a 69-year-old female who initially presented to an emergency department at a community hospital due to a ground-level fall with traumatic intracranial hemorrhage. The patient reportedly ingested apixaban 275 mg, carvedilol 250 mg, atorvastatin 1,200 mg, and unknown amounts of amlodipine and ethanol. Anti-inhibitor coagulant complex, an aPCC, was administered approximately 3 hours after presentation. Initial thromboelastography performed approximately 4 hours after presentation showed a prolonged reaction time of 16.8 minutes. Ongoing imaging and evidence of coagulopathy prompted repeated aPCC administration to a cumulative dose of approximately 100 U/kg. The patient underwent craniotomy with hematoma evacuation. Postoperative imaging showed expansion of the existing intracranial hemorrhage and new areas of hemorrhage. Andexanet alfa was administered approximately 18 hours after presentation, followed by repeat craniotomy with evacuation of the hematoma. No further expansion of the intracranial hemorrhage was observed, and the reaction time on thromboelastography was normalized at 6.3 minutes.

Conclusion: This case suggests that andexanet alfa may have a role in the management of traumatic hemorrhage in the setting of an acute massive apixaban overdose. Use of andexanet alfa, PCC, and aPCC in this context requires further research [5)]

1)
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4)
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5)
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