

# Subdural hematoma and anticoagulant therapy

The impact of antithrombotic therapy on the outcome of subdural hematoma is not well characterized to date.

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In clinical practice, there is a prevailing impression that, especially subdural hematomas of chronic and sub-chronic duration with or without acutization (evidence of “fresh” bleeding) are in many cases related directly to the use of anticoagulant therapy.

Statistical analysis showed a significantly greater incidence of spontaneous SDH (subdural hematoma) in patients taking oral anticoagulation therapy (Fisher exact test,  $p < 0.01$ ). A survey confirmed the existence of a relationship between oral anticoagulant therapy and SDH, in particular the subgroup of [spontaneous subdural hematoma](#).

## Epidemiology

Subdural hematomas are less frequently reported than intracerebral hemorrhage (ICH) in patients receiving [oral anticoagulation](#) OACs, but they are crucial to recognize because they are life-threatening and amenable to surgical therapy. OACs increase the risk of subdural hematoma 4- to 15-fold.

The rate of OAC-associated subdural hematoma is related to the intensity of anticoagulation, advanced patient age, and perhaps cerebral atrophy. In contrast to OAC-related ICH, hypertension does not appear to be associated with OAC-associated subdural hematoma, and precipitation by minor head trauma is notable. The absolute rate of subdural hematoma can be estimated as approximately 0.2%/y in elderly patients given OACs (INR $\approx$ 3).

The absolute rate of subdural hematoma during dual antiplatelet therapy is low, averaging 1.1 per 1000 patient-years. Chronic treatment with clopidogrel plus aspirin significantly increases the risk of subdural hematoma compared with aspirin alone <sup>1)</sup>

## Clinical features

Headache and mental status changes are prominent early symptoms, which can evolve for days to weeks. Approximately 20% are bilateral.

## Outcome

The mortality of OAC-associated subdural hematoma in series was 13% to 20%.

Reversal of anticoagulation and surgical drainage are usually undertaken; it is unclear whether small subdural hematomas causing minimal symptoms in patients receiving OACs can be safely managed nonsurgically, with reversal of anticoagulation <sup>2)</sup>

Results clearly indicate that prehospital antithrombotic therapy was independently associated with

inferior outcome of patients with [acute subdural hematoma](#), while no association for patients with [chronic subdural hematoma](#) (CSDH) was observed <sup>3)</sup>.

see [Chronic subdural hematoma and anticoagulant therapy](#)

## Case series

### 1976

In 1976 a review of the literature finds 150 cases. Subdural hematomas occurs in about one third of the patients presenting hemorrhage of central nervous system related to anticoagulant therapy. Among subdural hematomas of any cause, the possible role of a previous anticoagulant therapy is stressed in 4,8 to 14% of cases. In most cases, long term anticoagulants were indicated for arterial or heart (ischemic) lesions (16/22 cases). In 6 cases, anticoagulants were indicated for prevention or treatment of pulmonary embolies. In 18 cases, anticoagulant drug is from the group of dicoumarol or phenylindanedione. In 3 cases, the only anticoagulant given to patient was heparin. Pathogenic study suggests that hypocoagulability might not always be the only factor of bleeding: high blood pressure, other drugs and head trauma (10 cases) are often associated. Clinical features, in our series, are similar to those encountered in cases of subdural hematoma of any cause. According to the existence of a cranial injury and to the chronology of anticoagulant therapy, the authors divide their 22 patients into 3 groups. The use of protamin sulfate or human plasma fraction PPSB provides in few minutes a normal coagulability. Neurosurgical treatment in all author cases evacuated in 11 patients a chronic subdural hematoma, in 2 cases an acute, and in 9 cases a subacute hematoma. Results were fair in 19 patients with no sequelae. 3 patients died during the immediate post-operative period <sup>4)</sup>.

1)

Bakheet MF, Pearce LA, Hart RG. Effect of addition of clopidogrel to aspirin on subdural hematoma: meta-analysis of randomized clinical trials. Int J Stroke. 2014 Dec 3. doi: 10.1111/ijss.12419. [Epub ahead of print] PubMed PMID: 25472690.

2)

Hart RG, Boop BS, Anderson DC. Oral anticoagulants and intracranial hemorrhage. Facts and hypotheses. Stroke. 1995 Aug;26(8):1471-7. Review. PubMed PMID: 7631356.

3)

Baraniskin A, Steffens C, Harders A, Schmiegel W, Schroers R, Spangenberg P. Impact of pre-hospital antithrombotic medication on the outcome of chronic and acute subdural hematoma. J Neurol Surg A Cent Eur Neurosurg. 2014 Jan;75(1):31-6. doi: 10.1055/s-0032-1325629. Epub 2013 Feb 20. PubMed PMID: 23427037.

4)

Bret P, Lecuire J, Lapras C, Deruty R, Desgeorges M, Prudhon JL. [Subdural hematoma and anticoagulant therapy]. Neurochirurgie. 1976;22(6):603-20. French. PubMed PMID: 67569.

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