

# Chronic subdural hematoma and anticoagulant therapy

*J.Sales-Llopis*

*Neurosurgery Department, University General Hospital of Alicante, Foundation for the Promotion of Health and Biomedical Research in the Valencian Region (FISABIO), Alicante, Spain*

**Chronic subdural hematoma** (CSDH) in the elderly population, especially in men, is frequently associated with falls and **anticoagulation** or **antithrombotic** therapy. The indication for these medications, especially in elderly patients at risk for falls, should be carefully evaluated and controlled <sup>1)</sup>.

This association, for patients under anticoagulant therapy, appears even stronger in those patients who develop a CSDH in the absence of a recent trauma <sup>2)</sup>.

The risk of developing a CSDH was at least 42.5 times higher in **warfarinised** patients and also increased for patients on aspirin, although this risk could not be quantified <sup>3)</sup>.

A patient undergoing **aspirin** treatment must be considered at risk for development of chronic subdural hematoma. Aspirin should not be prescribed to patients with post-traumatic headaches <sup>4)</sup>.

## Bilateral hematoma

The bilateral hematomas tended to happen in hemodialysis cases, and in patients on warfarin or anti-platelet drugs. Bilateral hematomas were found in 41.7%, 37.5% and 33.3% respectively. The ratio of PT and aPTT coagulation time was as follows: PT ratio was 1.040 in hemodialysis cases and 1.082 in warfarin-applied cases. aPTT ratio was 1.022 in hemodialysis cases and 1.055 in warfarin-applied cases. These results suggest that the suppressed coagulation ability and platelet function are involved in the genesis of bilateral chronic subdural hematomas <sup>5)</sup>.

## Restarting

Clinicians regularly confront the dilemma of whether or not to restart anticoagulant and **antiplatelet** medication after CSDH, yet there is little evidence to support the decision-making process.

Databases including MEDLINE, Cochrane, ISI Web of Knowledge, Embase and Google Scholar were searched for retrospective and prospective studies looking specifically at patients presenting with CSDH whilst on anticoagulant or **antiplatelet** medication which had data on subsequent recurrence and thromboembolic events.

Three relevant studies were found, totalling to 64 patients. In those restarted on anticoagulation, 11.1% experienced recurrences and 2.2% experienced thromboembolic events. In the control group that was not restarted on anticoagulation, 22.2% experienced recurrences and no patient experienced thromboembolic events. All recurrences and thromboembolic events occurred within the first 4 weeks of the initial surgical evacuation. Conclusions. The review seems to paradoxically suggest a lower bleeding risk and a higher thromboembolism risk when anticoagulation is restarted, although few concrete conclusions can be taken from a pool of 64 patients. The decision on whether or not to restart anticoagulation in patients who present with CSDH whilst on anticoagulation has little

empirical evidence to support a decision either way; more data are required to allow clinicians to make informed decisions about whether or not to restart anticoagulation, and if so, which drug, at what time-point and at what dose/therapeutic target <sup>6)</sup>.

Gonugunta et al. suggest recommencing warfarin 3 weeks after surgical evacuation of CSDH in anticoagulated patients <sup>7)</sup>.

Early resumption of anticoagulant therapy (within 3 days) did not cause intracranial rebleeding in any operative patient. All the chronic SDH patients and some of the subcortical hematoma patients had a good outcome <sup>8)</sup>.

For Yeon et al. restarting warfarin therapy does not need to be withheld for more than 3 days after burr hole drainage, particularly in patients with a high thromboembolic risk <sup>9)</sup>.

## Outcome

A study has not demonstrated an adverse effect of the warfarin on the outcome of treatment for CSDH <sup>10)</sup>.

## Recurrence

It does not seem to influence the rate of CSDH recurrence <sup>11) 12)</sup>.

## Case series

### 2016

Amano et al., retrospectively analyzed 150 consecutive patients with CSDH who underwent neurosurgical interventions at Kyushu Rosai Hospital from 2011 to 2015 and followed them for more than 3 months.

Of the 150 study patients, 44 received antithrombotic therapy. All anticoagulants and 76% of the antiplatelet agents were discontinued before surgical treatment of CSDH and resumed within 1 week except in 4 patients whose treatment was terminated and 7 patients who developed postoperative complications or underwent reoperations before resumption of these agents. Postoperative hemorrhagic complications associated with surgical treatment of CSDH occurred in 8 patients (5.3%), and there was no significant difference in the incidence of these complications between patients with and without antithrombotic therapy (6.8% vs. 4.7%, respectively;  $p=0.90$ ). Postoperative thromboembolic complications occurred in 5 patients (5.4%), including 4 patients with antithrombotic therapy; these complications developed before resumption of antithrombotic agents in 2 patients. There was a significant difference in the incidence of postoperative thromboembolic complications between patients with and without antithrombotic therapy (9.1% vs. 0.9%, respectively;  $p=0.04$ ). There were no significant differences in the incidence of radiographic deterioration or reoperation of ipsilateral or contralateral hematomas between patients with and without antithrombotic therapy after surgical treatment of unilateral CSDH.

A history of antithrombotic therapy was significantly correlated with the incidence of postoperative thromboembolic complications in patients with CSDH. Antithrombotic agents should be resumed as soon as possible when no hemorrhagic complication is confirmed after neurosurgical intervention for CSDH <sup>13)</sup>.

## 2013

A retrospective review of 239 patients undergoing surgery for CSDH over a period of six years (2006-2011). The majority of patients (63%) in the non-trauma group were receiving anticoagulants and/or antiplatelet agent therapy prior to CSDH presentation, compared to 42% in the trauma group. Twenty-four percent experienced recurrence of the CSDH. There was no association between recurrence and anticoagulant and/or antiplatelet agent therapy.

Anticoagulant and/or antiplatelet aggregation agent therapy is more prevalent among non-traumatic CSDH patients but does not seem to influence the rate of CSDH recurrence <sup>14)</sup>.

## 2009

In the non-trauma group 71% of patients were treated with anticoagulants or antiplatelet aggregation agents (AAA) compared to 18% in the trauma group. Considering only AAA, 59% of the non-trauma patients were treated with these drugs versus 17% of patients in the trauma group. The recurrence rate for all patients was 17%. These findings confirm that the use of anticoagulants and AAA is over-represented in patients with non-traumatic CSDH. In this study, recurrence was not associated with previous use of anticoagulants or AAA <sup>15)</sup>.

## 2006

Eighty-one cases of chronic subdural haematomas (CSDH) admitted to the neurosurgical unit of the Royal Hobart Hospital, Tasmania, Australia, over a 5-year period were reviewed. The use of anticoagulant therapy as a causative agent in the development of CSDH was investigated. We suspected a high incidence of anticoagulant or anti-thrombotic therapy. We found that anticoagulant therapy was used by a significant percentage of CSDH patients. In the patient group presenting to our unit the risk of developing a CSDH was at least 42.5 times higher in warfarinised patients and also increased for patients on aspirin, although this risk could not be quantified <sup>16)</sup>.

## 2001

There is a perception that patients who develop a chronic subdural haematoma (CSDH), whilst taking warfarin, do less well than those not taking warfarin. This study looks at such patients to determine the truth of this perception. A retrospective analysis of two time periods (1990-1992 and 1995-1997) looking at all patients with CSDH admitted to this neurosurgical unit for treatment, to determine the incidence and to look more closely at those on warfarin. The influence of warfarin on the incidence, severity and outcome has been studied. Between 1990 and 1992, 11.8% of those patients with CSDH were taking warfarin, whilst in 1995-1997 20% were on warfarin. The overall number of referrals of CSDH increased from 34 to 150 patients during these time periods. There were no differences in age,

sex or other medical disorders between the two groups. No adverse events occurred when the warfarin was stopped temporarily for treatment of the CSDH. There was no increase in recurrence rate in those on warfarin, compared with those not on warfarin. This study, whilst demonstrating an increase in the number of referrals of CSDH and patients with CSDH taking warfarin, has not demonstrated an adverse effect of the warfarin on the outcome of treatment for CSDH. The authors suggest recommencing warfarin 3 weeks after surgical evacuation of CSDH in anti coagulated patients <sup>17)</sup>.

## 1999

The records of seven patients with mean GCS = 14.2 and mean clinical grade = 1.85 affected by chronic subdural hematoma and in treatment with anticoagulants were examined retrospectively. All the patients underwent subtemporal craniectomy plus closed drainage or burrhole(s) plus closed drainage after immediate correction of hypocoagulability by administration of vitamin K and fresh frozen plasma and normalization of PA by calcium heparin.

Outcome was good for all the patients except one who died because of cerebral herniation due to massive solid subdural hematoma during extracorporeal dialysis. Complications included: intracerebral hemorrhage, solid subdural hematoma, slow brain reexpansion, subdural collection reaccumulation and cerebral embolism. Three patients required re-operation. Mean duration of hospital stay was 18 days with range from 7 to 24 days.

Basing on this retrospective study and the proposed pathophysiology, the guidelines for optimal management of this subgroup of patients are proposed. Recommendations include the immediate correction of hypocoagulability, the appropriate surgical technique and the cautious conversion to oral anticoagulation as well as the appropriate timing of such conversion <sup>18)</sup>.

## 1995

In 2 patients, warfarin was discontinued and its effect neutralized by vitamin K, then surgery was performed after the thrombotest value exceeded 50%. No uncontrollable bleeding occurred at surgery. Warfarin was discontinued until 3-7 days postoperatively. Intravenous heparin administration was used to prevent embolic complications and the dose was modified based on the activated clotting time measured at the bedside. One patient, who could not receive heparin administration because of massive bleeding, developed myocardial infarction due to coronary artery thromboembolism 2 days after operation and died 4 days later. The other patients received heparin administration and were alive and well at the most recent follow-up examinations. Heparin administration monitored by activated clotting time is a useful method to prevent embolic and bleeding complications in the surgical treatment of intracranial hemorrhage in patients with prosthetic heart valves receiving long-term anticoagulant therapy <sup>19)</sup>.

## 1991

3 cases with good outcome They recommend conservative treatment is to be the first choice, if conditions allow it. Surgery can be performed by burr hole irrigation when indicated. Precautions should be taken not to injure the inner membrane of the hematoma or the brain proper, and the need

for slow decompression should be kept in mind <sup>20)</sup>.

## Case reports

### 2014

A 78-year-old man who had a history of myocardial and cerebral infarction and who was treated with aspirin and warfarin, presented with left chronic subdural hematoma. Cerebral computed tomography showed severe brain compression of hematoma with midline shift, indicating the need for emergent surgery. The hematology and clotting tests upon admission revealed severe thrombocytopenia (platelet count,  $1.3 \times 10(4)/\mu\text{L}$ ) with normal clotting activity. Because platelet aggregation was evident in the smear, we re-examined the patient for hematology using tubes that contained heparin, showing also low platelet count ( $2.3 \times 10(4)/\mu\text{L}$ ). The day on admission, we performed irrigation and drainage of the chronic subdural hematoma through single burr-hole craniostomy. During surgery, we used 10 units of platelet concentrates (PCs) for the reason that the patient was taking aspirin and coagulopathy derived from low platelet count could not be excluded. After surgery, we re-evaluated the hematology of the blood stored in tubes that contained ethylenediaminetetraacetic acid (EDTA) with or without kanamycin (KM). Treatment with KM dissociated EDTA-induced platelet aggregation and revealed platelet counts with highest accuracy (no KM treatment,  $1.3 \times 10(4)/\mu\text{L}$ ; KM treatment,  $15.2 \times 10(4)/\mu\text{L}$ ). This phenomenon is called EDTA-Dependent Pseudothrombocytopenia (PTCP) defined as falsely low platelet counts reported by automated hematology analyzers due to platelet aggregation. Awareness of the phenomenon will enable neurosurgeons to manage patients with PTCP appropriately and clinical laboratory especially in emergency hospital is recommended to prepare for the hematological tubes being added KM in routine analysis, resulting in preventing mistaken diagnosis <sup>21)</sup>.

### 2010

A 64-year-old female receiving clopidogrel and aspirin antiaggregation therapy after percutaneous coronary intervention for non-STEMI myocardial infarction developed nontraumatic bilateral subdural hematoma with dizziness, vertigo and headache. Craniotomy had to be postponed because of reduced ADP platelet aggregability. Four days after clopidogrel withdrawal and transfusion of 12 platelet concentrate units, ADP aggregation transiently normalized and bilateral trepanation with hematoma evacuation was performed. The procedure was followed by excellent neurologic and clinical recovery; however, decreased platelet aggregability was recorded by postoperative day 12 despite strict clopidogrel and other platelet inhibitor withdrawal. Suspicion of Glanzmann thrombasthenia was excluded by flow cytometry. Two weeks after neurosurgery, the right femoral vein thrombosis was detected by color doppler ultrasonography and therapy with fractionated heparin was initiated, followed by warfarin. The risk and incidence of hemorrhagic complications of antiaggregation and anticoagulation therapy are discussed. Caution is warranted on prescribing this potentially harmful therapy to older patients, generally burdened with other chronic comorbidities <sup>22)</sup>.

### 2003

We present a patient on warfarin in whom a drainage port system was attached to the skull, successfully draining a subacute subdural hematoma.

An elderly male presented to our institution with right hemiparesis a week following a motor vehicle accident. He was on warfarin for recurrent pulmonary emboli and suffered from severe coronary artery disease. Physical examination demonstrated a grade 3/5 hemiparesis and a computerized tomography (CT) scan confirmed the diagnosis of subacute subdural hematoma. He underwent twist drill craniostomy and attachment of the subdural evacuating port system. Recovery in this patient was dramatic.

The subdural evacuating port system (SEPS) permits the neurosurgeon to drain subacute or chronic hematomas by a method that is minimally invasive, simple, and safe. The SEPS appears to promote brain expansion without the potential biohazards of other standard techniques <sup>23)</sup>.

## 1984

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