Original Article

# Acute Subdural Hematoma Evacuation: Predictive Factors of Outcome


## Abstract

**Background:** Acute subdural hematoma (aSDH) is a major cause of admission at Neurosurgical Emergency Department. Nevertheless, concerns regarding surgical indication in patients with multiple comorbidities, poor neurological status, antithrombotic therapy, and older age still persist. Therefore, a correct recognition of predictive outcome factors at hospital discharge is crucial to an appropriate neurosurgical treatment. **Methods:** Eighty-nine medical records of consecutive patients with age ≥18 years old who were submitted to aSDH evacuation between January 2008 and May 2012 were reviewed. Demographic characteristics, neurological status on admission, anticoagulant or antiplatelet therapy, and outcome on discharge were collected. Patients with insufficient data concerning these variables were excluded from the study. **Results:** Sixty-nine patients were included; 52% were male; 74% were older than 65 years; 41% were under oral antithrombotic therapy (OAT); at admission, 54% presented with Glasgow coma scale (GCS) ≤8; 23% were submitted to a craniectomy instead of a craniotomy; 26% of the patients died, 32% were dependent, and 42% were independent on discharge. Crude analysis revealed craniectomy, A/A pupils, GCS ≤8 at admission statistically significant related with the worst outcome ($P < 0.05$). In the adjusted evaluation only A/A pupils ($P = 0.04$) was associated to poor outcome (spontaneous etiology $P = 0.052$). Considering daily living independency at hospital discharge, either male gender ($P = 0.044$) and A/A pupils ($P = 0.030$) were related to the worst outcome. No effect of age in outcome was observed. **Conclusions:** Male gender and A/A pupils are associated with lower probability of achieving independency living at hospital discharge. A/A pupils, low GCS at admission, spontaneous etiology, and craniectomy were associated with the worst outcome. Age and OAT were not predictive factors in this series. Caution should be taken when considering these factors in the surgical decision.


**Keywords:** *Acute subdural hematoma, age, anticoagulant/antiplatelet therapy, craniectomy, craniotomy, Glasgow outcome score*

**José Pedro Lavrador, Joaquim Cruz Teixeira, Edson Oliveira, Diogo Simão, Maria Manuel Santos, Nuno Simas**

*Department of Neurosurgery, Hospital Santa Maria, Lisbon, Portugal*

## Introduction

Acute subdural hematomas (aSDH) account for 50%–60% of all subdural hematomas. In the majority of cases, they are related with a traumatic event. Rarely, they may occur spontaneously or after a minor trauma in patients receiving anticoagulant or antiplatelet therapy or after aneurysmatic rupture, mainly posterior communicant artery.[1] Considering the posttraumatic aSDH, two main types should be considered: (1) the aSDH related to contusion, laceration and intracerebral hematoma (ICH) – burst lobe syndrome- and (2) the aSDH related to rupture of bridging vessel. The first one is usually related with significant primary brain injury, often with no lucid interval and may show delayed neurological deterioration. The second one is related to brain acceleration-deceleration during violent head motion, with less severe brain injury and the presence of lucid interval.

In Western countries, road traffic accidents are one of the leading causes of death and disability among individuals aged <45 years old. In Portugal, the TBI incidence was 137/100,000 habitants between 1996 and 1997 with a male: female ratio of 3:1.[2] These numbers have been decreasing in the last years – 2000 to 2010 - due to the restrict safety measures implemented on traffic policy. The incidence is moving toward the older individuals due to a number of risk factors: normal cerebral atrophy related to aging, stretching the bridging veins from the dura, anticoagulant, or antiplatelet intake due to cardiovascular risk factors or primary/secondary prevention and increased risk of minor trauma.[3,4]

The problem of oral antithrombotic therapy (OAT) is rising and is a matter of concern in these patients. Some studies



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***Address for correspondence:***
*José Pedro Lavrador, MD, Hospital Santa Maria, Av. Prof. Egas Moniz, 1649-035 Lisboa, Portugal.*
*E-mail: jose.pedro.lavrador@gmail.com*

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suggest that patients undergoing oral anticoagulation therapy (OAT) at the time of injury have worst prognosis compared with those who are not.[5] Others authors argue that patients under OAT are frequently older than those who are not and the difference in outcome may be due to an age-related factor.[6] When considering the antiplatelet therapy, there are no evidence that single antiplatelet therapy increases the risk of major bleeding (placebo 2.4% vs. clopidogrel 2.7% in the CURE study). However, hemorrhagic complication seems to increased when we consider double antiplatelet drugs.[7] Postoperatively, patients on OAT or antiplatelet therapy show no increased risk of rebleeding if they receive reversal agents or platelets.[8] However, OAT is still not extensively studied in aSDH.

The neurosurgical treatment approach to the patient with aSDH is wide and ranges from palliative care to surgical evacuation. Considering the increased incidence in older ages frequently associated with significant medical comorbidities and low physiological reserve, the identification of prognostic factors is of paramount importance to rationalize its therapeutic approach. Age and anticoagulant/antiplatelet therapy are among important factors that may influence the prognostic on discharge. However, the improvement in intensive care units and the ability to complete or partially reverse the effect of these drugs may attenuate the influence of these variables in the outcome. Other factors may also play a role in prognosis such as neurological status at admission, gender surgical approach, and mechanism of injury. It is important to establish its influence on outcome to reasonably decide each clinical approach. In this article, the authors revised a single-institution series of aSDH to evaluate the impact of these factors in outcome at discharge.

## Methods

### Patient selection and data recording

This is a retrospective cohort study in two phases: a cross-sectional phase where the patients included in the sample were evaluated for the following described variables, and a follow-up phase until hospital discharge or a death event occurred. From January 2008 to May 2012, 89 patients were admitted to Hospital Santa Maria Emergency Room and submitted to aSDH evacuation in the Neurosurgery Department. The following variables were analyzed gender (male/female), age (<65 years old/≥65 years old), mechanism of injury (spontaneous/traumatic), OAT (none/antiplatelet therapy – AA/anticoagulant therapy – AC/both – AA/AC), Glasgow coma scale (GCS) at admission, acute brain injury (ABI) severity (severe - ≤8/moderate - 9–12/mild - 13–15), pupil reactivity (isochoric and reactive [IR]/anisocoric and arreactive [AA]), surgical technique (craniotomy/craniectomy), and Glasgow outcome scale (GOS) at discharge. The outcome was also evaluated according to the following categories: death (GOS 1), poor outcome (GOS 2–3), and good outcome (GOS 4–5); and also dichotomized in death/dependent (1–3) versus independent (4–5).

The inclusion criteria were adult patients (age ≥18 years old) admitted to Hospital Santa Maria Emergency Room and submitted to aSDH evacuation in the Neurosurgery Department between January 2008 and May 2012. The exclusion criterion was unknown data concerning the study variables. Sixty-nine patients were included in the study.

### Surgical technique

The patient is placed in dorsal decubitus with the head fixed in a Mayfield head holder and laterally rotated toward the opposite side of the hematoma. A trauma flap and the craniotomy are performed after a variable number of burr holes concerning the surgeon preference and the patient characteristics. The dura mater is widely opened and the hematoma is evacuated. The decision of replace the bone is taken by the surgeon concerning the brain parenchyma appearance. The dura is not watertight sutured although artificial dural substitute is used. In this way, a dural expansion is obtained for potential brain parenchyma swelling. The trauma flap is closed with absorbable sutures, in the deeper layers, and with skin stitches. In patients under anticoagulation therapy, fresh frozen plasma or Octaplex® was used to correct the INR, and in those under antiplatelet therapy, pools of platelets were used according to surgeon preference.

### Statistical evaluation

The statistical evaluation was performed using STATA version 13.0 (Texas, USA). For dichotomous evaluation, the Pearson and Fisher Chi-square test were used; for categorical outcomes, an ordered logistic regression was performed; and for dichotomous outcomes with continuous exposure variables an unpaired *t*-test was used. We report $P$ values and standard deviations for continuous variables. For outcome assessment, performed using GOS and the rearranged categories, we adjust for the following confounders based on the literature review: age, gender, surgical technique, ICP monitoring, OAT, mechanism of lesion, pupils' size, and reactivity and GCS at admission. $P < 0.05$ was deemed statistically significant.

## Results

The studied population involved 69 patients, 52% were male, 74% were older than 65 years, and 41% were under OAT. At admission, 54% presented with GCS ≤8 and 23% were submitted to a craniectomy instead of a craniotomy. Regarding the outcome at discharge, 42% were independent, 32% were dependent, and 26% of the patients died.

### Demographics

The sample was divided into two groups according to age (<65 vs. ≥65 years old). In the <65 years group, lower

Glasgow coma scale (GCS) (6.3 ± 2.0), AA pupils (82%), craniectomy (47%), male gender (76%), severe ABI (88%), intracranial hypertension (29%), and no OAT intake (12%) were found statistically significant ($P < 0.05$). The traumatic etiology was the most frequent in both groups (88% - <65 vs. 75% - ≥65) [Table 1].

According to gender, male subgroup was associated with younger age (65 ± 16.8), traumatic lesion mechanism (89%) and low GCS (7.8 ± 3.7) ($P < 0.05$). Even though pupil size and reactivity ($P = 0.096$) and ABI severity ($P = 0.104$) did not reach statistically significance, AA (50% vs. 30%) and severe ABI (64% vs. 42%) were more frequent in men [Table 2].

## Outcome

### *Age and gender*

Either considered as a continuous or a dichotomous variable (<65 vs. ≥65 years old), age was not associated with the outcome according to GOS.

In general, gender was not also associated with outcome. Nevertheless, when considering a binary outcome (death/dependent vs. independent), male gender was associated with a poor outcome ($P = 0.044$) [Table 3].

### *Clinical factors*

The influence of the clinical factors in outcome was analyzed using GOS scale and the previous mentioned rearranged groups.

In patients with GOS 1, craniectomy (44%), A/A pupils (36%), low GCS at admission (7.1 ± 0.8), and severe ABI (38%) were more frequent ($P < 0.05$). Spontaneous aSDH had also higher frequency in GOS 1 group (53%) but with did not reach statistical significance ($P = 0.082$) [Table 4].

When considering the follow-up GOS groups (death/poor outcome/good outcome), all the previous clinical factors remain significantly associated with death with the exception of craniectomy ($P = 0.117$) [Table 5].

Considering the evaluated clinical factors, while the crude analysis showed craniectomy, A/A pupils, low GCS at admission and severe ABI statistically significant related to the worst outcome ($P < 0.05$), the adjusted evaluation revealed only A/A pupils ($P = 0.04$). When considering the follow-up GOS groups (death/poor outcome/good outcome), only the spontaneous etiology reaches a statistically meaningful value for poor outcome ($P = 0.027$). Finally, in the dichotomous outcome analysis, either male gender ($P = 0.044$) and A/A pupils ($P = 0.030$) are related with death/dependent [Table 6].

**Table 1: Baseline characteristics of the study population - age**

| Characteristic | <65 *n*=17 (25%) | >65 *n*=52 (75%) | *P* |
|---|---|---|---|
| Age | 47.1 (13.0) | 77.5 (7.0) | <0.0001 |
| Craniectomy (%) | 8 (47) | 8 (15) | 0.007 |
| ICP monitoring (%) | 5 (29) | 4 (8) | 0.035 |
| OAT (%) | 2 (12) | 26 (50) | 0.009 |
| None (%) | 15 (88) | 26 (50) | 0.0067 |
| AA (%) | 1 (6) | 11 (21) | |
| AC (%) | 1 (6) | 14 (27) | |
| AA + AC (%) | 0 | 1 (2) | |
| Mechanism of lesion (%) | | | |
| Traumatic | 15 (88) | 39 (75) | 0.326 |
| Spontaneous | 2 (12) | 13 (25) | |
| A/A pupils (%) | 14 (82) | 14 (30) | <0.0001 |
| GCS at admission | 6.3 (2.0) | 9.4 (3.8) | <0.0001 |
| ABI severity (%) | | | |
| Mild ABI | 0 | 15 (42) | 0.0008 |
| Moderate ABI | 2 (12) | 15 (29) | |
| Severe ABI | 15 (88) | 22 (29) | |

ICP – Intracranial pressure; OAT – Oral antithrombotic therapy; AA – Antiaggregation; AC – Anticoagulation; A/A – Anisochoric areactive; GCS – Glasgow Coma Scale; ABI – Acute brain injury

**Table 2: Baseline characteristics of the study population - gender**

| Characteristic | Male *n*=36 (52%) | Female *n*=33 (48%) | *P* |
|---|---|---|---|
| Age (%) | 65 (16.8) | 75 (13.0) | 0.0071 |
| <65 | 13 (36) | 4 (12) | 0.021 |
| >65 | 23 (64) | 29 (88) | |
| Craniectomy (%) | 10 (28) | 6 (18) | 0.345 |
| ICP monitoring (%) | 6 (17) | 3 (9) | 0.351 |
| OAT (%) | 15 (42) | 13 (39) | 0.848 |
| None (%) | 21 (58) | 20 (61) | 0.87 |
| AA | 8 (22) | 4 (12) | |
| AC (%) | 7 (19) | 8 (24) | |
| AA + AC (%) | 0 | 1 (3) | |
| Mechanism of lesion (%) | | | |
| Traumatic | 32 (89) | 22 (67) | 0.025 |
| Spontaneous | 4 (11) | 11 (33) | |
| A/A pupils (%) | 18 (50) | 10 (30) | 0.096 |
| GCS at admission | 7.8 (3.7) | 9.6 (3.4) | 0.0331 |
| ABI severity (%) | | | |
| Mild ABI | 6 (17) | 9 (27) | 0.0872 |
| Moderate ABI | 7 (19) | 10 (30) | |
| Severe ABI | 23 (64) | 14 (42) | |

ICP – Intracranial pressure; OAT – Oral antithrombotic therapy; AA – Antiaggregation; AC – Anticoagulation; A/A – Anisochoric areactive; GCS – Glasgow Coma Scale; ABI – Acute brain injury

## Discussion

The outcome of adult patients submitted to aSDH evacuation in this series is similar to recent neurosurgical series concerning this issue. Although initial studies reported higher mortality rates ranging from 40% to 90%, in the recent years, neuroanesthesia improvement, dedicated intensive care units, and surgical advances changed this scenario.[3,9] However, aSDH still caries has a high morbidity and mortality. Therefore, a correct recognition of predictive outcome factors is crucial to an appropriate neurosurgical treatment.

**Table 3: Baseline characteristics of the study population - Glasgow Outcome Scale categories (1-3 vs. 4-5)**

| Characteristic | 1-3 (*n*=40) | 4-5 (*n*=29) | *P* |
|---|---|---|---|
| Age | 68.4 (16.0) | 72.2 (15.6) | 0.321 |
| Age category (>65 years) | 29 (92.5) | 23 (79.3) | 0.517 |
| Gender (male) | 25 (62.5) | 11 (38.0) | 0.044 |
| Craniectomy (%) | 11 (27.5) | 5 (17.2) | 0.319 |
| ICP monitoring (%) | 6 (15) | 3 (10.3) | 0.571 |
| OAT (%) | 15 (37.5) | 13 (44.8) | 0.541 |
| None (%) | 25 (62.5) | 16 (55.2) | |
| AA (%) | 7 (17.50) | 5 (17.24) | |
| AC (%) | 7 (17.50) | 8 (27.59) | |
| AA + AC (%) | 1 (2.50) | 0 | |
| Mechanism of lesion (%) | | | |
| Traumatic | 30 (75.0) | 24 (82.75) | 0.441 |
| Spontaneous | 10 (25.0) | 5 (12.5) | |
| A/A pupils (%) | 22 (55.0) | 6 (20.7) | 0.004 |
| GCS at admission | 7.8 (3.6) | 9.8 (3.38) | 0.023 |
| ABI severity (%) | | | |
| Mild ABI | 6 (15) | 9 (31.03) | 0.016 |
| Moderate ABI | 7 (17.50) | 10 (34.48) | |
| Severe ABI | 27 (67.50) | 10 (34.48) | |

ICP – Intracranial pressure; OAT – Oral antithrombotic therapy; AA – Antiaggregation; AC – Anticoagulation; A/A – Anisochoric areactive; GCS – Glasgow Coma Scale; ABI – Acute brain injury

There is data reporting gender influence in prognosis in traumatic brain injury studies.[10] In a meta-analysis, Farace and Alves found poorer outcome in females surviving severe traumatic brain injury than males.[11] However, there is also studies suggesting good outcome in clinical use of progesterone after severe TBI.[12-14] Indeed, progesterone seems to decrease cerebral edema and promote neuroprotection.[13,14] In this study, male gender is associated with poor outcome in the dichotomized analysis. This finding may be related with a higher probability of high-energy injuries in the male population[10] which is supported by a significant lower GCS in this group.

Age has been considered an important prognostic factor concerning the outcome, particularly in traumatic brain injury patients.[10,15] However, other authors alerted for discordant results in their series with no influence detected for age as an isolated poor prognostic factor.[16] aSDH is frequent in older individuals due to age-related cerebral atrophy, increased subdural space, stretching of bridging veins, and higher risk for trauma due to gait or orthopedic-related problems.[3,17] The surgical and anesthetic risk is also higher considering common comorbidities and decreased physiological reserve.[18] No meaningful differences were found between both considered age groups (<65 vs. ≥65 years old) in the present series. Some reasons may explain these results. Indeed, the older individuals are not only more prone to develop aSDH but also they usually present it after low-energy impact. On the other hand, their age-related cerebral atrophy increases the compliance to the developing hematoma. These factors

**Table 4: Baseline characteristics of the study population - Glasgow Outcome Scale**

| Characteristic | 1 (*n*=18) | 2 (*n*=7) | 3 (*n*=15) | 4 (*n*=17) | 5 (*n*=12) | *P* |
|---|---|---|---|---|---|---|
| Age | 70.2 (2.6) | 61 (8.6) | 69.7 (4.5) | 72.4 (3.2) | 72 (5.4) | 0.448 |
| Gender (%) | | | | | | |
| Male | 9 (25) | 5 (14) | 11 (31) | 7 (19) | 4 (11) | 0.190 |
| Female | 9 (27) | 2 (6) | 4 (12) | 10 (30) | 8 (24) | |
| Craniectomy (%) | 7 (44) | 3 (19) | 1 (6) | 4 (25) | 1 (6) | 0.045 |
| ICP monitoring (%) | 2 (22) | 1 (11) | 3 (33) | 1 (11) | 2 (22) | 0.985 |
| OAT (%) | 8 (29) | 2 (7) | 5 (18) | 10 (36) | 3 (11) | 0.855 |
| None (%) | 10 (56) | 5 (71) | 10 (67) | 7 (41) | 9 (75) | 0.829 |
| AA (%) | 3 (17) | 1 (14) | 3 (20) | 5 (29) | 0 | |
| AC (%) | 4 (22) | 1 (14) | 2 (13) | 5 (29) | 3 (25) | |
| AA + AC (%) | 1 (6) | 0 | 0 | 0 | 0 | |
| Mechanism of lesion (%) | | | | | | |
| Traumatic | 10 (19) | 6 (11) | 14 (26) | 15 (28) | 9 (17) | 0.082 |
| Spontaneous | 8 (53) | 1 (7) | 1 (7) | 2 (13) | 3 (20) | |
| A/A pupils (%) | 10 (36) | 7 (25) | 5 (18) | 4 (14) | 2 (7) | 0.002 |
| GCS at admission | 7.1 (0.8) | 6.8 (1.4) | 9.1 (0.96) | 9.2 (0.89) | 10.8 (0.81) | 0.003 |
| ABI severity (%) | | | | | | |
| Mild ABI | 2 (13) | 1 (7) | 3 (20) | 5 (33) | 4 (27) | 0.004 |
| Moderate ABI | 2 (12) | 0 | 5 (29) | 5 (29) | 5 (29) | |
| Severe ABI | 14 (38) | 6 (16) | 7 (19) | 7 (19) | 3 (8) | |

ICP – Intracranial pressure; OAT – Oral antithrombotic therapy; AA – Antiaggregation; AC – Anticoagulation; A/A – Anisochoric areactive; GCS – Glasgow Coma Scale; ABI – Acute brain injury

**Table 5: Baseline characteristics of the study population - Glasgow Outcome Scale categories (1 vs. 2-3 vs. 4-5)**

| Characteristic | 1 (*n*=18) | 2-3 (*n*=22) | 4-5 (*n*=29) | *P* |
|---|---|---|---|---|
| Age | 70.2 (2.6) | 66.9 (4.1) | 72.2 (2.9) | 0.543 |
| Age category (>65 years) | 14 (77.8) | 15 (68.18) | 23 (79.31) | 0.764 |
| Gender (male) | 9 (50) | 16 (72.7) | 11 (37.9) | 0.205 |
| Craniectomy (%) | 7 (44) | 4 (25) | 5 (31) | 0.117 |
| ICP monitoring (%) | 2 (22) | 4 (44) | 3 (33) | 0.810 |
| OAT (%) | 8 (29) | 7 (25) | 13 (46) | 0.816 |
| None (%) | 10 (24) | 15 (37) | 16 (39) | 0.951 |
| AA (%) | 3 (25) | 4 (33) | 5 (42) | |
| AC (%) | 4 (27) | 3 (20) | 8 (53) | |
| AA + AC (%) | 1 (100) | 0 | 0 | |
| Mechanism of lesion (%) | | | | |
| Traumatic | 10 (19) | 20 (37) | 24 (44) | 0.058 |
| Spontaneous | 8 (53) | 2 (13) | 5 (33) | |
| A/A pupils (%) | 10 (36) | 12 (43) | 6 (21) | 0.009 |
| GCS at admission | 7.06 (0.81) | 8.4 (0.80) | 9.9 (0.63) | 0.010 |
| ABI severity (%) | | | | |
| Mild ABI | 2 (13) | 4 (27) | 9 (60) | 0.008 |
| Moderate ABI | 2 (12) | 5 (29) | 10 (59) | |
| Severe ABI | 14 (38) | 13 (35) | 10 (27) | |

ICP – Intracranial pressure; OAT – Oral antithrombotic therapy; AA – Antiaggregation; AC – Anticoagulation; A/A – Anisochoric/Areactive; GCS – Glasgow Coma Scale; ABI – Acute brain injury

**Table 6: Glasgow Outcome Scale in the adjusted analysis of studied population**

| | GOS (*P*) | GOS categories | |
|---|---|---|---|
| | | 1-3 versus 4-5 (*P*) | 1 versus 2-3 versus 4-5 (*P*) |
| Age | 0.111 | 0.085 | 0.115 |
| Gender | 0.155 | 0.116 | 0.044 |
| Craniectomy | 0.059 | 0.195 | 0.706 |
| ICP monitoring | 0.823 | 0.623 | 0.424 |
| OAT | 0.972 | 0.566 | 0.341 |
| Mechanism of lesion | 0.052 | 0.027 | 0.156 |
| A/A pupils | 0.040 | 0.105 | 0.030 |
| GCS at admission | 0.169 | 0.153 | 0.306 |

ICP – Intracranial pressure; OAT – Oral antithrombotic therapy; A/A – Anisochoric areactive; GCS – Glasgow Coma Scale; GOS – Glasgow Outcome Scale

decrease the primary parenchymal injury related to the initial event and may increase the salvageability of the brain with a correct and time-adjusted surgical intervention. Younger patients were submitted to surgical evacuation even in severe lesions in the present series (lower GCS, AA pupils, severe ABI, and intracranial hypertension statistically significant in <65 years old group). These differences introduced a bias in outcome analysis concerning age. Even though the younger patients (<65 years) had more severe injuries, the outcome was the same. Paradoxically, this finding probably supports a role for age in TBI outcome.

The vast majority of patients included have suffered a traumatic mechanism of injury (78%) and this found to be a significant prognostic toward good outcome at discharge. On the other hand, spontaneous mechanism of injury was a significant prognostic factor among patients who have died during hospitalization. OAT, myeloproliferative disorders, and inherited coagulation disorders are among the most common ones related to spontaneous aSDH.[19-22]

The clinical severity is an important prognostic factor. GCS scale and pupillary size, symmetry and reactivity are broad used elements to evaluate the level of consciousness and brain stem lesions in trauma patients.[23] Nevertheless, a reliable GCS score main be hard to get in acute situations according to medical sedation, paralysis and/or intoxication.[24] Motor response is the most reproductive element in trauma patients as the others responses may be blunt.[25] Considering these limitations, the GCS included was the one recorded at hospital admission. Meaningful differences were found favoring better outcomes in patients with higher admission GCS evaluations.

Abnormalities in pupillary reactivity have been widely related with neurological prognosis as they reflect brainstem compression or intrinsic lesion and are associated with poorer outcome.[26] This is also supported by the findings of significant good outcome in patients with IR pupils and poorer outcome in patients with AA pupils. It is argued pupillary reactive is a more reliable parameter in the early phase after TBI due to less influence by sedation and paralysis compared to GCS.[26] No patient with bilateral fixed and dilated pupils was submitted to surgery in the present series.

OAT has been empirically regarded as a prognostic factor of poor outcome in aSDH. Nevertheless, different authors report concordant results supporting neither rebleeding nor mortality among this group of patients and alert for the fact that these results may be affected by the concomitant presence of other potential comorbidities as age and poor medical condition.[5,6,8,27] Considering that 75% of patients were ≥65 years old, it is not surprising that 41% of patients were under OAT. When comparing patients under OAT with those who were not, no significant difference was found among the GOS subgroups, and no statistically meaningful trend was observed either in OAT or in no-OAT group. Furthermore, 46% of OAT patients were independent on discharge. Even though it is known that previously OAT increases the risk and the extent of aSDH,[28] the accurate acute treatment through the reversal of iatrogenic hemorrhagic condition minimizes its influence on outcome after surgical evacuation. These results contrast with those of ICH.[29]

The surgical approach is difficult to analyze concerning the personal issues involved in the chosen procedure. The reason most frequently reported to perform a craniectomy was the existence of intracerebral lesions and brain swelling. Among the literature, discordant results are found some authors report no differences between the two methods[30] (craniotomy and craniectomy) while others report a tendency toward the poorer outcome in craniectomy patients.[31,32] Atmospheric pressure, cerebral blood flow (CBF), cerebrospinal fluid, and the impediment of venous return have all been implicated in the cognitive and neurological changes that occur after large craniectomies. Indeed, the CBF is reduced after the acute phase due to the atmospheric pressure and a decrease in cerebral metabolic glucose rate and cerebral metabolic oxygen rate are observed.[33] Even though this may certainly contribute to the outcome, one can assume that the patients who underwent craniectomy were the most extent lesions who intrinsically had a poor prognosis, producing a bias in the presented results.[33,34]

## Conclusion

aSDH is a frequent neurosurgical condition responsible for significant morbidity and mortality. The core of the decision-making process should be built not only by intrinsic hematoma features but also by prognostic factors that may help predicting the outcome and establishing a reasonable approach considering the invasiveness and aggressiveness of the therapeutic measures. Age and OAT were not related with a poor outcome. Male gender has a lower probability of independency living at hospital discharge. AA pupils, low GCS at admission, spontaneous etiology, and craniectomy were associated with the worst outcome.

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### Conflicts of interest

There are no conflicts of interest.

## References

1. Marbacher S, Fandino J, Lukes A. Acute subdural hematoma from ruptured cerebral aneurysm. Acta Neurochir (Wien) 2010;152:501-7.
2. Santos ME, De Sousa L, Castro-Caldas A. Epidemiology of craniocerebral trauma in Portugal. Acta Med Port 2003;16:71-6.
3. Hanif S, Abodunde O, Ali Z, Pidgeon C. Age related outcome in acute subdural haematoma following traumatic head injury. Ir Med J 2009;102:255-7.
4. Taussky P, Hidalgo ET, Landolt H, Fandino J. Age and salvageability: Analysis of outcome of patients older than 65 years undergoing craniotomy for acute traumatic subdural hematoma. World Neurosurg 2012;78:306-11.
5. Senft C, Schuster T, Forster MT, Seifert V, Gerlach R. Management and outcome of patients with acute traumatic subdural hematomas and pre-injury oral anticoagulation therapy. Neurol Res 2009;31:1012-8.
6. Oyama H, Kito A, Maki H, Hattori K, Noda T, Wada K. Acute subdural hematoma in patients with medication associated with risk of hemorrhage. Neurol Med Chir (Tokyo) 2011;51:825-8.
7. Mehta SR, Yusuf S, Peters RJ, Bertrand ME, Lewis BS, Natarajan MK, *et al.* Effects of pretreatment with clopidogrel and aspirin followed by long-term therapy in patients undergoing percutaneous coronary intervention: The PCI-CURE study. Lancet 2001;358:527-33.
8. Panczykowski DM, Okonkwo DO. Premorbid oral antithrombotic therapy and risk for reaccumulation, reoperation, and mortality in acute subdural hematomas. J Neurosurg 2011;114:47-52.
9. Antoniadis G, Richter HP. Treatment measures in traumatic extracerebral hemorrhage. Management of epi-/subdural hemorrhage and prognosis. Unfallchirurg 1993;96:582-6.
10. Maas AI, Murray GD, Roozenbeek B, Lingsma HF, Butcher I, McHugh GS, *et al.* Advancing care for traumatic brain injury: Findings from the IMPACT studies and perspectives on future research. Lancet Neurol 2013;12:1200-10.
11. Farace E, Alves WM. Do women fare worse: A metaanalysis of gender differences in traumatic brain injury outcome. J Neurosurg 2000;93:539-45.
12. Vandromme M, Melton SM, Kerby JD. Progesterone in traumatic brain injury: Time to move on to phase III trials. Crit Care 2008;12:153.
13. Sayeed I, Stein DG. Progesterone as a neuroprotective factor in traumatic and ischemic brain injury. Prog Brain Res 2009;175:219-37.
14. Stein DG, Wright DW. Progesterone in the clinical treatment of acute traumatic brain injury. Expert Opin Investig Drugs 2010;19:847-57.
15. Servadei F. Prognostic factors in severely head injured adult patients with acute subdural haematoma's. Acta Neurochir (Wien) 1997;139:279-85.
16. Pospiech J, Kalff R, Herwegen H. Prognostic factors in acute traumatic epi- and subdural hematoma. Aktuelle Traumatol 1993;23:1-6.
17. Petridis AK, Dörner L, Doukas A, Eifrig S, Barth H, Mehdorn M. Acute subdural hematoma in the elderly; clinical and CT factors influencing the surgical treatment decision. Cent Eur Neurosurg 2009;70:73-8.
18. Pasternak JJ, Lanier WL. Neuroanesthesiology update. J Neurosurg Anesthesiol 2010;22:86-109.
19. Cermelj M, Negro F, Schijman E, Ferro AM, Acerenza M, Pollola J. Neurosurgical intervention in a haemophilic child with a subdural and intracerebral haematoma. Haemophilia 2004;10:405-7.
20. Jeffree RL, Gordon DH, Sivasubramaniam R, Chapman A. Warfarin related intracranial haemorrhage: A case-controlled study of anticoagulation monitoring prior to spontaneous subdural or intracerebral haemorrhage. J Clin Neurosci 2009;16:882-5.
21. Basmaci M, Hasturk AE. Chronic subdural hematoma in a child with acute myeloid leukemia after leukocytosis. Indian J Crit Care Med 2012;16:222-4.
22. Fryburg K, Nguyen HS, Cohen-Gadol AA. Spontaneous acute subdural hematoma due to fondaparinux: Report of two cases. Surg Neurol Int 2011;2:44.
23. Teasdale G, Jennett B. Assessment of coma and impaired consciousness. A practical scale. Lancet 1974;2:81-4.
24. Stocchetti N, Pagan F, Calappi E, Canavesi K, Beretta L, Citerio G, *et al.* Inaccurate early assessment of neurological severity in head injury. J Neurotrauma 2004;21:1131-40.
25. Marmarou A, Lu J, Butcher I, McHugh GS, Murray GD, Steyerberg EW, *et al.* Prognostic value of the Glasgow Coma Scale and pupil reactivity in traumatic brain injury assessed pre-hospital and on enrollment: An IMPACT analysis. J Neurotrauma 2007;24:270-80.
26. Marmarou A, Lu J, Butcher I, McHugh GS, Mushkudiani NA,

Murray GD, *et al.* IMPACT database of traumatic brain injury: Design and description. J Neurotrauma 2007;24:239-50.
27. Rozzelle CJ, Wofford JL, Branch CL. Predictors of hospital mortality in older patients with subdural hematoma. J Am Geriatr Soc 1995;43:240-4.
28. Mattle H, Kohler S, Huber P, Rohner M, Steinsiepe KF. Anticoagulation-related intracranial extracerebral haemorrhage. J Neurol Neurosurg Psychiatry 1989;52:829-37.
29. Curtze S, Strbian D, Meretoja A, Putaala J, Eriksson H, Haapaniemi E, *et al.* Higher baseline international normalized ratio value correlates with higher mortality in intracerebral hemorrhage during warfarin use. Eur J Neurol 2014;21:616-22.
30. Chen SH, Chen Y, Fang WK, Huang DW, Huang KC, Tseng SH. Comparison of craniotomy and decompressive craniectomy in severely head-injured patients with acute subdural hematoma. J Trauma 2011;71:1632-6.
31. Li LM, Kolias AG, Guilfoyle MR, Timofeev I, Corteen EA, Pickard JD, *et al.* Outcome following evacuation of acute subdural haematomas: A comparison of craniotomy with decompressive craniectomy. Acta Neurochir (Wien) 2012;154:1555-61.
32. Kalayci M, Aktunç E, Gül S, Hanci V, Edebali N, Cagavi F, *et al.* Decompressive craniectomy for acute subdural haematoma: An overview of current prognostic factors and a discussion about some novel prognostic parametres. J Pak Med Assoc 2013;63:38-49.
33. Oh CH, Park CO, Hyun DK, Park HC, Yoon SH. Comparative study of outcomes between shunting after cranioplasty and in cranioplasty after shunting in large concave flaccid cranial defect with hydrocephalus. J Korean Neurosurg Soc 2008;44:211-6.
34. Schaller B, Graf R, Sanada Y, Rosner G, Wienhard K, Heiss WD. Hemodynamic and metabolic effects of decompressive hemicraniectomy in normal brain. An experimental PET-study in cats. Brain Res 2003;982:31-7.