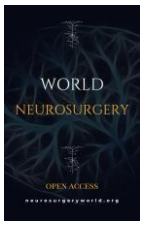
Contents lists available at: WNEURO

World Neurosurgery

Journal homepage: neurosurgeryworld.org



Research Article

Predictive Factors of Poor Prognosis in Acute Subdural Hematoma: Experience of the University Hospital of Bouaké

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ARTICLE INFO

Keywords:
Mortality
traumatic brain injury
acute subdural hematoma

ABSTRACT

Objective: To identify, within our hospital setting, the predictive factors associated with poor prognosis in patients presenting with acute subdural hematoma (ASDH), with the aim of optimizing management and improving survival through the implementation of targeted therapeutic strategies.

Materials and Methods: We conducted a retrospective, observational, and analytical study over a four-year period (January 1, 2020 - December 31, 2023), including all patients admitted to the neurosurgery and intensive care units for the management of ASDH. Initial univariate analysis compared clinical, radiological, and outcome characteristics between survivors and non-survivors. Independent predictors of mortality were subsequently identified using multivariate logistic regression.

Results: A total of 124 patients were included, of whom 88 survived (70.97%) and 36 died (29.03%). The mean age was 33.51 years (range: 9-70 years), with a marked male predominance (115 men, 92.74% vs. 9 women, 7.26%). The overall mortality rate was 40.91%. Multivariate analysis identified the following independent predictors of death: initial Glasgow Coma Scale ≤ 13 (OR = 5.3; $p < 0.0001$), pupillary diameter abnormality (OR = 28.281; $p < 0.0001$), hematoma thickness ≥ 15.8 mm (OR = 11.051; $p < 0.0001$), midline shift ≥ 13 mm (OR = 6.02; $p < 0.0001$), presence of temporal or tonsillar herniation (OR = 8.034; $p = 0.000$), Rotterdam CT score ≥ 4.19 (OR = 14; $p = 0.000$), and non-admission to intensive care (OR = 5.84; $p = 0.01$).

Conclusion: Initial neurological status, CT markers of severe cerebral compression, and timely access to intensive care are major determinants of survival in ASDH. Early recognition of these factors should prompt rapid and aggressive management to improve patient outcomes.

1. Introduction

A traumatic brain injury (TBI) refers to trauma to the skull that results in either transient or permanent brain dysfunction, with or without loss of consciousness. It can lead to death or various dysfunctions, such as motor, cognitive, and/or psychological impairments [1]. TBIs significantly impact not only the patient's life but also that of their family and close circle. [1] When severe (Glasgow Coma Scale score of 8 or less) [2-4]. TBIs represent a major cause of morbidity and mortality in developing countries [5, 6]. In France, there are approximately 8,000 deaths and 150,000 cases of TBI annually. In 2005, 30,000 TBI patients were reported to be living with severe disabilities [6, 7]. Traumatic brain injury is the leading cause of death among young adults in both developed and developing countries [2, 4]. However, in developing countries, mortality is partly attributed to the very limited resources available for patient care [4]. The high morbidity and mortality associated with severe TBIs make it a significant public health issue [6]. The most common causes include road traffic accidents (RTAs) and falls. TBIs show a strong male predominance, with a sex ratio consistently ranging between 1.4 and 4. The incidence of TBIs varies between 0.9 and 7 per thousand, depending on the study location, study years, and study types [1-4, 8].

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10.31487/j.WNEURO.2025.04.02
Received 24 November, 2025; Accepted 16 December, 2025
Available online 13 May, 2026

Among severe TBIs (sTBI), acute subdural hematoma (ASDH) (accounting for 40-50% of sTBIs) is a devastating clinical entity. It involves intracranial lesions characterized by blood accumulation between the dura mater and the brain. The prognosis, which is difficult to predict, ranges from complete recovery to dependency on caregivers or death [6]. Mortality rates as high as 60% have been reported [5, 6]. The clinical presentation of ASDH typically manifests within the first three days following high velocity cranioencephalic trauma (common in younger patients) or minor trauma (in elderly patients or those on anticoagulants). In the most severe cases, neurological symptoms appear immediately. The causes include the rupture of a vein or artery or bleeding from a cerebral contusion. Paraclinical diagnosis relies on non-contrast cranial computed tomography (CT), where ASDH appears as a spontaneously hyperdense, crescent-shaped, hemispheric collection adjacent to the skull with poorly defined borders [5, 6]. It is differentiated on CT from chronic subdural hematoma (CSDH), which appears as an isodense or hypodense collection. When heterogeneous hyperdensity is observed within the collection, it indicates recent bleeding within the CSDH [3, 5, 6].

Surgical treatment involves the evacuation of ASDH via a large craniotomy, with or without replacement of the bone flap, and wide opening of the dura mater, also known as decompressive craniectomy [3, 6, 9-11]. Any subdural hematoma with a thickness exceeding 10 mm or causing a midline shift (MLS) greater than 5 mm must be urgently evacuated, regardless of the Glasgow Coma Scale (GCS) score [8, 12]. Surgery is also recommended for any subdural hematoma in cases of severe TBI with a decrease in GCS by 2 or more points between the time of trauma and hospitalization and/or the presence of pupillary abnormalities (bilateral mydriasis or anisocoria) [12].

The surgical indications are as follows: an ASDH thickness (EH) greater than 10 mm and/or a midline shift (MLS) of 5 mm or more, regardless of the Glasgow coma scale (GCS) score. For ASDHs with a thickness less than 10 mm and an MLS less than 5 mm, surgery is indicated only if the GCS decreases by 2 points compared to the initial GCS, or if the pupils are asymmetric or fixed and dilated, or if the intracranial pressure (ICP) exceeds 20 mm Hg [6]. Several prognostic studies have identified age, initial GCS, and pupillary abnormalities as prognostic factors [3-17]. However, very few of these prognostic studies are based on African data [1, 4, 7]. The objective of this study is to determine the factors associated with poor prognosis in ASDH within our context. This study aims to improve patient survival by implementing appropriate management strategies.

2. Materials and Methods

2.1. Type of Study

This is a retrospective, observational, and analytical study conducted on patients admitted to neurosurgery and intensive care units for the management of acute subdural hematoma (ASDH) over a 4-year period, from January 1, 2020, to December 31, 2023.

2.2. Study Population

Using the hospitalization registry of the department, we identified all patients admitted with a primary diagnosis of ASDH, with or without traumatic edema-hemorrhagic contusion. All patients, regardless of age or gender, were included. Given the retrospective nature of the study, no consent was obtained from the patients. However, they were informed during follow-up telephone interviews about the anonymized use of their data.

2.3. Data Collection

Clinical and paraclinical data were extracted from patient medical records. The Glasgow Outcome Scale (GOS) at 6 months post-trauma was determined from rehabilitation reports, postoperative follow-up consultations, communication with attending physicians, or intensive care unit records.

For each patient, the following variables were collected: Sociodemographic data included age, sex, and admission delay. Clinical parameters comprised history of chronic alcoholism, initial Glasgow coma scale (GCS) score, pupil status, neurological deficits, and higher cognitive functions. Paraclinical data included CT scan findings (hematoma location, presence or absence of edema-hemorrhagic contusion, hematoma thickness), indirect signs of intracranial hypertension (midline shift [MLS], temporal or brainstem herniation), and radiological indicators of cerebral edema (visualization of basal cisterns, lateral ventricles, cortical sulci, and comparison between hematoma thickness and MLS). Preoperative laboratory assessments (anemia, prothrombin time) were also recorded, and the Rotterdam score was calculated for each patient. Therapeutic variables comprised operative delay, type of surgical procedure, and perioperative transfusions (pre-, intra-, and postoperative). Evolutionary data included mortality, postoperative rehabilitation, infection, and admission to intensive care.

2.4. Statistical Analysis

Univariate and bivariate descriptive analyses were performed. Qualitative variables were compared using the Chi-square test or Fisher's exact test when appropriate. Quantitative variables were compared using the Student's t-test or, in cases of non-normal distribution or heteroscedasticity, the non-parametric Mann-Whitney test. Variables associated with poor prognosis at a significance level of $p < 0.05$ and deemed clinically relevant were introduced into a multivariate logistic regression model. Only variables that met the assumptions of the multivariate model were retained. Prognostic factors were considered significant at $p < 0.05$. A 95% confidence interval was applied for all estimates. Statistical analysis and data coding were conducted using SPSS IBM version 22 (Statistical Package for the Social Sciences).

2.5. Definition of Study Criteria

ASDH was defined paraclinically by the presence of a hyperdense collection, irrespective of the duration of installation. Outcome was assessed using the modified Jennett and Bond (1975) GOS classification, validated in French (Annex 3). A GOS score of 1 or 2 was considered a poor prognosis. For GCS, the lowest score recorded before sedation—usually reported by first responders—was used.

2.6. Radiological Imaging Definitions

All admission brain CT scans and their reports were reviewed by a single evaluator. The primary elements analyzed were those described in the neurosurgeon's written report, interpreted in relation to the patient's clinical course. Specific analyses included the following: cortical sulci were classified as clearly visible on both hemispheres, erased on one hemisphere, or erased on both hemispheres, based on axial slices passing through the lateral ventricles and Sylvian fissures. Infra-tentorial basal cisterns, particularly peri-mesencephalic cisterns, were assessed according to the Rotterdam score classification and described as clearly visible, partially visible, or absent. Contusions were characterized by both localization and volume, with volume estimated using the standard formula (ABC/2), where A, B, and C represent the largest diameters (length, width, and height).

3. Results

During the study period, a total of 124 patients were admitted for the management of acute subdural hematoma (ASDH) in the Neurosurgery Department of Bouaké University Hospital, out of 2180 cases of traumatic brain injury, representing a prevalence of 17.42%. Among the 124 cases, 88 patients survived, while 36 patients died, resulting in a mortality rate of 40.91%. The mean age of the study population was 33.51 years, ranging from 9 to 70 years. The gender distribution included 115 males (92.74%) and 9 females (7.26%). At 6 months post-trauma, 36 patients (29%) had a poor prognosis. Chronic alcoholism was reported in 22.58% of patients, and 60.7% had experienced high-kinetic trauma. The average hematoma thickness in the cohort was 13.5 mm, with values ranging from 3 to 25 mm. The difference between hematoma thickness and midline shift (MLS) varied between 3 and 15 mm, while the median MLS was 10 mm (interquartile range: 2.25-16 mm). The mean Rotterdam score was 3.96.

Multivariate analysis identified several factors as significant predictors of mortality. An initial Glasgow coma scale (GCS) score of 13 or lower was associated with a markedly increased risk (OR = 5.3, $p < 0.0001$). Abnormalities in pupil diameter were among the strongest predictors (OR = 28.281, $p < 0.0001$). Radiological markers, including hematoma thickness ≥ 15.8 mm (OR = 11.051, $p < 0.0001$) and MLS ≥ 13 mm (OR = 6.02, $p < 0.0001$), also strongly correlated with poor outcomes. The presence of temporal or tonsillar herniation further increased mortality risk (OR = 8.034, $p = 0.000$). Similarly, a Rotterdam score of 4.19 or higher was highly predictive (OR = 14, $p = 0.000$). Finally, non-admission to intensive care significantly worsened prognosis (OR = 5.84, $p = 0.01$), underscoring the critical importance of intensive management in ASDH.

The characteristics of the study population, clinical examination findings, initial imaging results, and therapeutic management are presented in (Tables 1-3). The analysis of mortality risk factors is detailed in (Table 4), while the results of logistic regression analysis are summarized in (Table 5). In this study, an association was observed between initial Glasgow Coma Scale score, pupil status, average hematoma thickness, median MLS, presence of temporal herniation, mean Rotterdam score, admission to intensive care, and the occurrence of death.

Table. 1. Population description.

Sociodemographics	Alive	Deceased	Total
Age			
< 40 years	64 (70,33%)	27 (29,67%)	91
> 40 years	24 (72,73%)	9 (27,27%)	33
Sexe			
Male	80 (69,57%)	35 (30,43%)	115
Female	8 (88,89%)	1 (11,11%)	9
Chronic Alcoholism			
Yes	21 (75%)	7 (25%)	28
No	67 (69,79%)	29 (30,21%)	96
Clinical			
Admission delay			
< 24h	6 (100%)	0	6
> 24h	82 (71,93%)	36 (31,57%)	114
Initial GCS			
<8	0	4 (100%)	4
8-13	71 (69,61%)	31 (30,39%)	102
14-15	17 (94,44%)	1 (5,56%)	18
Pupillary Status			
Normal	73 (78,49%)	20 (21,51%)	93
Anisocoria	9 (47,37%)	10 (52,63%)	19
Myosis	5 (50 %)	5 (50 %)	11
Mydriasis	0	1 (100%)	1
Neurological Deficit			
Yes	50 (68,49%)	23 (31,51%)	73

No	38 (74,51%)	13 (25,49%)	51
Cognitive function			
Yes	25 (78,12%)	7 (21,88%)	32
No	28 (54,90%)	13 (25,49%)	51
Untested	35 (68,63%)	16 (31,37%)	51

Table. 2. CT findings and biology.

Average Hematoma Thickness, mm	*11,2*	*15,8*	27,0
Average Midline Shift, mm	*7*	*13*	20
Average (ET - MS), mm	0,6	*1,5*	2,1
Presence of brain contusion			
Yes	*37 (54,41%)*	*31 (45,59%)*	68
No	*51 (91,07%)*	*5 (8,93%)*	56
Temporal/Uncal Herniation			
Yes	*63 (75,90%)*	*20 (24,10%)*	83
No	*25 (60,97%)*	*16 (39,03%)*	41
Rotterdam Score	3,73	4,19	7,92
Cortical Sulcus effacement			
Visible bilaterally	*13 (72,22%)*	*5 (27,78%)*	18
Not visible unilaterally	*62 (70,45%)*	*26 (29,55%)*	88
Not visible bilaterally	*13 (72,22%)*	*5 (27,78%)*	18
Biology			
Coagulation impairment			
Yes	*65 (71,43%)*	*26 (28,57%)*	91
No	*23 (69,7%)*	*10 (30,3%)*	33
Anemia			
Yes	*32 (57,14%)*	*24 (42,86%)*	56
No	*56 (82,35%)*	*12 (17,65%)*	68

Table. 3. Therapeutic characteristics.

Therapeutic characteristics	Alive	Deceased	Total
Surgical Timing (n=97)			
< 24h	*4 (80%)*	*1 (20%)*	5
> 24hs	*59 (64,13%)*	*33 (35,87%)*	92
Treatment Type			
Surgical	*62 (63,92%)*	*35 (36,08%)*	97
Non surgical	*26 (96,3%)*	*1 (3,7%)*	27
Surgical Technique			
Decompressive hemicraniectomy	*21 (55,26%)*	*17 (44,74%)*	38
Decompressive hemicraniectomy + hematoma evacuation	*41 (69,49%)*	*18 (30,51%)*	59
Intraoperative blood loss			
< 500ml	*32 (72,73%)*	*12 (27,27%)*	44
> 500ml	*56 (70%)*	*24 (30%)*	80
Outcomes			
ICU admission			
Yes	*60 (68,18%)*	*16 (44,44%)*	76
No	*28 (31,82%)*	*20 (55,56%)*	48
Post operative infection			
Yes	*1 (9,09%)*	*10 (90,91%)*	11
No	*87 (76,99%)*	*26 (23,01%)*	113
Rehabilitation			
Yes	*49 (89.09%)*	*6 (10,91%)*	55
No	*39 (56,52%)*	*30 (43,47%)*	69
Glasgow Outcome Score			

[1-2]	*2 (5,55%)*	34 (94,45%)	36
[3-5]	86 (97,73%)	2 (2,27%)	88

Table. 4. Analysis of mortality risk factors.

Variables	Deceased	Alive	Total	OR	IC	P value
Sociodemographics						
Age						
< 40 years	27	64	91		*-120,36-68,3639*	*0,357*
> 40 years	9	24	33			
Sexe					0,4371-159,564	
Male	35	80	115	3.473		0,445
Female	1	8	9			
Chronic Alcoholism						
Yes	7	21	28		*0,405 -2,727*	0,593
No	29	67	96			
Clinical						
Admission delay						
< 24h	0	6	6		*0,589-3,639*	0,179
> 24h	36	82	118			
Initial GCS						
<8	4	0	4			
8-13	31	71	102		*1,049- 7,342*	**0,0004**
14-15	1	17	18			
Pupillary Status						
Normal	20	74	94			
Anisocoria	10	9	19		*1,774- 11,843*	**0,0017**
Myosis	5	5	10			
Mydriasis	1	0	1			
Neurological Deficit						0,599
Yes	23	50	73		-	
No	13	38	51			
Cognitive function						
Yes	7	25	32			
No	13	28	41		-	0,584
Untested	16	35	51			
CT Findings						
Average Hematoma Thickness, mm	**15,8**	11,2			*0,025- 0,261*	**<0,0001**
Average Midline Shift, mm	**13**	7				**<0,0001**
Average (ET - MS), mm	1,5	0,6				
Presence of brain contusion					*1,207- 5,399*	
Yes	31	37	68			1,89
No	5	51	56			
Temporal/Uncal Herniation						
Yes	20	63	83			**<0,0001**
No	16	25	41			
Rotterdam Score	**4,19**	3,73	7,92			**<0,0001**
Cortical Sulcus effacement						
Visible bilaterally	5	13	18			
Not visible unilaterally	26	62	88	-	-	0,98
Not visible bilaterally	5	13	18			
Biology						
Coagulation impairment						
Yes	26	65	91			0,851

No	10	23	33			
Anemia						
Yes	24	32	56			0,19
No	12	56	68			
Therapeutic characteristics						
Surgical Timing (n=97)						
< 24h	1	4	5	0,45	0,008-4,7999	0,654
> 24hs	33	59	92			
Treatment Type					2,180-616,87	
Surgical	35	62	97	144,791		**0,0005**
Non surgical	1	26	27			
Surgical Technique						
Decompressive hemicraniectomy	17	21	38			0,21
Decompressive hemicraniectomy + hematoma evacuation	18	41	59			
Intraoperative blood loss					-109,02-57,02	
< 500ml	12	32	44			0,23
> 500ml	24	56	80			
Outcomes						
ICU admission						
Yes	**5**	**39**	44			**0,01**
No	**31**	**49**	80			
Post operative infection						
Yes	10	1	11			0,55
No	26	87	113			
Rehabilitation					0,049-0,446	
Yes	6	49	55			72,079
No	30	39	69			
Glasgow Outcome Score				576,36	82,029-8129	
[1-2]	34	2	36			1,1
[3-5]	2	86	88			

Table. 5. Analysis regression logistic multivariate.

Variables	ORa	IC	P value
Sociodémographic			
Age	3,475	0,589-3,639	0,742
Clinical			
Admission delay	1,7	0,222-1,892	0,32
Initial GCS ≤ 13	5,3	1,589-2,639	**< 0,0001**
Pupil diameter abnormalities	28,2	-	**< 0,0001**
Neurological deficit	1,34	0,565-3,282	0,54
CT Finding			
Average Hematoma Thickness, mm ≥15,8 mm	11,051	*0,222-1,894*	**<0,0001**
Average Midline Shift ≥13 mn	6,02	-	**<0,0001**
Average (ET - MS), mm	3,08	-	0,428
Temporal/Uncal Herniation	8,034	*0,405-2,727*	**0,000**
Rotterdam Score ≥ 4,19	14	-	**0,000**
Cortical Sulcus effacement	0,667	-	0,919
Biology			
Coagulation impairment	1,42	-	0,74
Therapeutic characteristics			
Surgical timing (n=97)	0,865	-	0,345
Intraoperative blood loss	2,08	-	0,08
Outcomes			

ICU admission	5,84	-	**0,01**
Rehabilitation			0,79
Glasgow Outcome Score	2,435	*0,453-2,476*	0,213

4. Discussion

This study aimed to identify the predictive factors of poor prognosis in patients with acute subdural hematoma (ASDH). The significant predictors of mortality identified were: an initial Glasgow Coma Scale (GCS) score ≤ 13 (OR = 5.3, p < 0.0001), pupil diameter abnormalities (OR = 28.281, p < 0.0001), hematoma thickness ≥ 15.8 mm (OR = 11.051, p < 0.0001), midline shift (MLS) ≥ 13 mm (OR = 6.02, p < 0.0001), presence of temporal or tonsillar herniation (OR = 8.034, p = 0.000), a Rotterdam score ≥ 4.19 (OR = 14, p = 0.000), and non-admission to intensive care (OR = 5.84, p = 0.01). The strength of this study lies in its sample size of 124 patients, which reduces selection bias and enhances the reliability of the findings.

4.1. Mortality Rate

The study revealed a mortality rate of 40.91% among patients treated for ASDH. Although slightly lower than rates reported in other studies [6, 7, 14], this remains significantly high. This elevated mortality rate may be attributed to delays in transferring patients to specialized centers due to transportation challenges and long distances, which exacerbate the harmful effects of secondary brain injury mechanisms (ACSOS).

4.2. Predictive Factors of Poor Prognosis

4.2.1. Initial Alteration in Consciousness

Clinical evaluation is essential for assessing the severity of traumatic brain injury and estimating the risk associated with intracranial lesions. The Glasgow coma scale (GCS) is the most commonly used tool for this purpose, analyzing eye opening, motor responses, and verbal responses to stimuli. In this study, altered consciousness was observed in 106 cases, with nearly all deceased patients (97.22%) having a GCS score ≤ 13. This demonstrates a significant association between mortality and impaired consciousness at initial clinical examination (OR = 5.3, p < 0.0001). Similar findings have been reported in other studies [6-18]. This association may be explained by the fact that altered consciousness often reflects hemodynamic and ventilatory instability, as well as worsening secondary brain injuries due to ACSOS.

4.2.2. Pupil Diameter Abnormalities

In this study, 44% of deceased patients presented with pupil abnormalities. Analysis revealed that pupil diameter abnormalities increased the risk of mortality by more than 28 times (OR = 28.2, p < 0.0001), consistent with findings from other studies [7, 10, 19]. Pupil abnormalities are indicative of specific brain injuries, particularly temporal herniation or brainstem compression, making them reliable markers of severe traumatic brain injury and predictors of mortality in post-traumatic ASDH.

4.2.3. Hematoma Thickness

All deceased patients in this study had hematoma thickness ≥ 15.8 mm (OR = 11.051, p < 0.0001). The detrimental impact of hematoma thickness exceeding 15 mm has also been highlighted in other studies, such as Baucher [3] (p = 0.002), who reported a slightly higher threshold (> 20 mm) associated with poor prognosis. Hematoma thickness is an independent marker of severity, integrated into prognostic scores such as CRASH and IMPACT [19, 20]. Its utility in identifying patients at risk of post-operative complications (e.g., recurrence, cerebral edema) has also been noted [20, 21].

4.3. Midline Shift (MLS)

Midline shift observed on CT scans is a direct indicator of mass effect and intracranial pressure, reflecting critical brain compression and major risks of cerebral herniation, which explains its association with high mortality in ASDH. In this study, the risk of mortality was 6 times higher for patients with MLS ≥ 13 mm (OR = 6.02, p < 0.0001). This observation aligns with findings by Attal [6], who noted that MLS was more pronounced in deceased patients (p < 0.0001), even though no specific prognostic threshold was reported.

4.4. Presence of Temporal or Tonsillar Herniation

Temporal or tonsillar herniation in ASDH indicates compression of vital structures, leading rapidly to irreversible neurological and cardiopulmonary failure, which explains its association with mortality. In this study, the risk of mortality was 8 times higher in cases with temporal or tonsillar herniation compared to cases without (OR = 8.034, p = 0.000). Other studies [22, 23] have similarly reported a 5-fold increase in mortality risk (OR = 5.09, p = 0.0004) in cases of cerebral herniation in general.

4.5. Rotterdam Score

A Rotterdam score ≥ 4 was significantly associated with poor prognosis (OR = 14, p = 0.000). In this study, all patients with a Rotterdam score > 4 died. This finding is consistent with the literature [24], which emphasizes the importance of this score as a reliable prognostic marker in severe traumatic brain injuries, including ASDH. The Rotterdam score incorporates essential radiological criteria such as midline shift, lateral ventricle status, and effacement of subarachnoid spaces, reflecting the severity of mass effect and intracranial hypertension. Severe mass effect predisposes patients to transtentorial or subfalcine herniation, leading to vital center failure and increased mortality.

4.6. Non-Admission to Intensive Care

In this study, non-admission to intensive care was significantly associated with mortality in patients with ASDH (OR = 5.84, p = 0.01). The mortality rate was 38% for patients not admitted to intensive care, compared to 11.38% for those who were. This observation aligns with

the literature, which highlights the critical importance of intensive care management and rigorous neurological and hemodynamic monitoring in this context [25, 26]. Non-admission often reflects resource limitations, such as the availability of intensive care beds, which can hinder optimal management and increase the risk of secondary cerebral ischemia and death. Thus, the absence of intensive care admission in patients with ASDH emerges as a poor prognostic factor, underscoring the need for systematic referral to intensive care units for appropriate monitoring and intervention to improve survival and functional outcomes.

4.7. Limitations

While this study provides important insights into the predictors of mortality in ASDH, several limitations must be acknowledged. First, although the cohort included 124 patients, the sample size is relatively modest for a robust multivariate analysis, and a larger population would strengthen the statistical validity of the results. Second, the retrospective design introduces inherent biases in data collection, which could be better addressed through a prospective study. Finally, the single-center nature of the study may restrict the generalizability of the findings to other clinical contexts; a multicenter approach would improve the applicability of the results, particularly in resource-limited settings such as ours.

5. Conclusion

This retrospective study identified several key predictive factors of poor prognosis in patients with acute subdural hematoma (ASDH). High mortality was strongly associated with initial impaired consciousness (GCS ≤ 13), the presence of pupil abnormalities indicating severe brain injury, hematoma thickness of at least 15.8 mm, and midline shift of 13 mm or greater, both reflecting a critical mass effect. Additional predictors included the presence of temporal or tonsillar herniation, a Rotterdam score of 4.19 or higher, and lack of admission to intensive care, underscoring the essential role of intensive management. Together, these findings highlight the decisive influence of both initial clinical presentation and radiological parameters on ASDH prognosis. They also draw attention to the significant challenges imposed by resource limitations, particularly restricted access to intensive care, which has a direct impact on patient survival.

Conflicts of Interest

None.

Annex

SCORE DE ROTTERDAM

Classification	*Interpretation*
• **Basal cisterns** ○ 0: normal ○ 1: compressed ○ 2: absent • **Midline shift** ○ 0: no shift or ≤ 5 mm ○ 1: shift > 5 mm	In adults the mortality at six months increases with the score: • **Score 1** : 0 % • **Score 2 :** 7 % • **Score 3** : 16 % • **Score 4 :** 26 % • **Score 5** : 53 % • **Score 6** : 61% Children have lower mortality in lower Rotterdam scores (scores 2 and 3), and higher mortality at higher scores (scores 4 to 6)
• **Epidural mass lesion** ○ 0 : present ○ 1 : absent	
• **Intraventricular blood or traumatic SAH** ○ 0 : absent ○ 1 : present	

GOS

"**1**. Dead: As a direct result of brain trauma, or … due to secondary complications or other complications**"**

"**2**. Vegetative State: Patients who remain unresponsive and speechless…."

"**3**. Severe Disability: The patient is conscious but needs the assistance of another person for some activities of daily living every day….."

"**4**. Moderate Disability: Such a patient is able to look after himself at home, to get out and about to the shops and to travel by public transport. However, some previous activities, either at work or in social life, are now no longer possible by reason of either physical or mental deficit…."

"**5**. Good Recovery: This indicates the capacity to resume normal occupational and social activities, although there may be minor physical or mental deficits…social outcome should be included in the assessment here, such as leisure activities and family relationships."

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