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TRANSIENT LOSS OF CONSCIOUSNESS IN THE MANIFESTATION OF CEREBRAL ARTERIAL ANEURYSM RUPTURE AS A PREDICTOR OF THE PROBABILITY OF FATAL OUTCOME. A UNIVARIATE ANALYSIS

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Introduction. Transient loss of consciousness (TLOC) is a common manifestation of aneurysmal subarachnoid hemorrhage (aSAH), observed in 26–53% of patients, and may serve as a prognostic indicator. However, limited research in Ukrainian literature addresses TLOC as a predictor of aSAH severity and outcomes.

Objective. The study aimed to assess the impact of TLOC during the manifestation of ICAA rupture on the time to fatal outcome in patients with WFNS grades I-III.

Materials and methods. A retrospective analysis was conducted on the records of 60 deceased patients diagnosed with aSAH. Patients were divided into two groups based on the presence (n=22) or absence (n=38) of TLOC during the manifestation of the aSAH. Survival was analyzed using Kaplan-Meier curves and log-rank tests to determine independent predictors of mortality.

Results. Median survival time was identical in both groups at 13 days, indicating similar overall dynamics of aSAH progression regardless of TLOC presence. However, the mean survival was shorter in Group 1 (14±3 days) compared to Group 2 (19±2 days), suggesting a trend toward reduced survival in patients with TLOC (p=0.186). Early survival (days 1–12) was similar across groups, with survival declining to 50% by day 12. After day 13, survival in Group 1 declined below 30% by day 15, while Group 2 maintained 30% survival until day 18. Late-stage outcomes showed accelerated mortality in Group 1, with all patients deceased by day 62, compared to day 82 in Group 2.

Conclusions. Patients who experienced TLOC tend to have shorter survival times, faster clinical deterioration, and a higher risk of fatal outcomes within the first 30 days after ICAA rupture, necessitating closer monitoring and more intensive therapy for these patients.

Keywords: subarachnoid hemorrhage; ruptured cerebral aneurysm; loss of consciousness; survival; mortality.

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ТРАНЗИТОРНА ВТРАТА СВІДОМОСТІ В МАНІФЕСТАЦІЇ РОЗРИВУ ЦЕРЕБРАЛЬНИХ АРТЕРІАЛЬНИХ АНЕВРИЗМ ЯК ПРЕДИКТОР ВІРОГІДНОСТІ НАСТАННЯ ФАТАЛЬНОГО РЕЗУЛЬТАТУ. ОДНОФАКТОРНИЙ АНАЛІЗ

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Тимчасова втрата свідомості (ТВС) є поширеним проявом субарахноїдального крововиливу з потенційним прогностичним значенням, проте в українській літературі бракує досліджень щодо її ролі як предиктора. У цьому дослідженні проаналізовано вплив ТВС під час розриву церебральної артеріальної аневризми на летальність пацієнтів з оцінкою тяжкості стану за шкалою WFNS I-III. Ретроспективний аналіз 60 історій хвороб померлих пацієнтів показав, що пацієнти з ТВС мають вищий ризик летального наслідку, швидше погіршення стану та скорочений час виживання. ТВС рекомендовано враховувати як предиктор прогнозу у пацієнтів з розривом церебральної артеріальної аневризми.

Ключові слова: субарахноїдальний крововилив, розрив мозкової аневризми, втрата свідомості, виживаність, летальність.

Introduction

Transient loss of consciousness (TLOC) is a common presentation of aneurysmal subarachnoid hemorrhage (aSAH), occurring in 26–53% of patients [1; 2]. TLOC in aSAH is defined as a sudden, involuntary loss of alertness, awareness, or responsiveness to external stimuli occurring in the prehospital setting [2].

Several pathophysiological processes can explain the occurrence of TLOC in patients with aSAH. Rupture of intracranial cerebral arterial aneurysms (ICAAs) causes a sudden increase in intracranial pressure and a decrease in cerebral perfusion, leading to transient global ischemia and hypoxia of critical brain structures, including the reticular formation [3]. It is known that an increase in intracranial

pressure with a rapid decrease in cerebral perfusion pressure can occur in less than 1 minute after ICAA rupture. The transient reduction in perfusion pressure likely contributes to the full recovery of consciousness in patients with TLOC [1].

At the same time, considering the mechanisms of impaired consciousness in aSAH, it should be noted that it is hemorrhagic products that activate neuroinflammation, disrupting the blood-brain barrier and contributing to brain edema. Oxidation of hemoglobin leads to the formation of free radicals that damage cells, while activation of Toll-like receptor 4 stimulates the release of cytokines, causing neuronal death [4]. Thus, TLOC in aSAH is mainly caused by a sudden increase in intracranial pressure. In contrast, prolonged impaired consciousness is caused by a cascade of delayed pathogenetic mechanisms: neuroinflammation, disruption of the blood-brain barrier integrity, oxidative stress, and brain edema [2–4].

According to the American Heart Association guidelines for the management of patients with aSAH, validated clinical scales, such as the Hunt and Hess (H&H) or the World Federation of Neurosurgical Societies (WFNS) scales, should be used promptly for initial assessment of clinical severity and prognosis (Class I; Level of Evidence B) [5]. However, a prospective study has shown that clinical assessment of patients' severity using the H&H and WFNS scales can vary in the first days after ICAA rupture and usually stabilizes within three days of hospitalization [6]. It is important to note that syncope refers to a transient decrease in the level of consciousness but not to states such as muffled consciousness, sopor, or coma, which are associated with different mechanisms of development of depression of consciousness and syncope in aSAH.

The clinical severity of WFNS patients is one of the most important and objective predictors of adverse outcomes in aSAH. In particular, numerous studies have shown that WFNS IV-V is an independent predictor of gross neurological deficit and mortality [7–11].

Previous studies have shown that TLOC at the time of presentation of ICAA rupture is associated with worse functional outcomes three months after aSAH, according to the Glasgow Outcome Scale, and there is a trend towards increased mortality among patients with TLOC [12]. TLOC is considered a single or combined predictor that can significantly affect the incidence of complications, treatment outcomes, and prognosis of patients with aSAH [13]. TLOC at ICAA rupture, together with WFNS IV–V, hydrocephalus, vasospasm, hypernatremia, and delayed cerebral ischemia (DCI), have been established as major predictors of mortality in SAH [14]. In addition, the combined effect of TLOC and WFNS scores above III at admission is a powerful predictor of adverse outcomes (Glasgow outcome scale 1–3), exceeding the prognostic significance of each factor alone [12]. While another study identified TLOC, along with age, systolic blood pressure at admission, H&H grade IV–V, seizures, Fisher grade III–IV, size of ruptured ICAA, microsurgical clipping, and CSF replacement, as an independent risk factor for aSAH outcomes [15]. However, other published data suggest an ambiguous relationship between TLOC and DCI. For example, TLOC during ictus in patients with H&H grades I and II was independently associated with cardiac complications and nosocomial infections but not with DCI [3]. Similarly, TLOC during ictus was associated with greater bleeding, cerebral edema, seizures, and cardiopulmonary arrest, which predicted death or severe disability at 12 months. However, in these cases, no association with DCI or rebleeding has been found [2].

The duration of TLOC is known to be prognostic. TLOC lasting more than 1 hour is associated with a 6-fold increased risk of developing a DCI compared with patients who had a TLOC lasting less than 1 hour or who remained conscious [15]. Furthermore, prolonged TLOC (more than 10 minutes, which doubles the risk, and more than 1 hour, which increases the risk 6-fold) has been identified as an independent risk factor for cognitive impairment after treatment of ruptured anterior communicating artery aneurysms [16].

The impact of TLOC on the clinical course of aSAH remains a relevant topic for the medical community since the available medical literature presents only a limited number of studies analyzing the features of TLOC in ICAA rupture. At the same time, there are currently no clinical markers that could predict mortality with sufficient accuracy in the acute period of aSAH at the prehospital stage. In the available Ukrainian scientific literature, TLOC during the manifestation of aSAH is not considered a separate predictor of the course of the disease or its consequences. Studies focus on the influence of the severity of patients according to the validated WFNS and H&H scales, focal neurological deficit, localization of ICAA, other prognostic factors, such as the severity of the WFNS and H&H scales, features of the aneurysm localization, and other clinical indicators, on the outcomes of treatment and recovery [17; 18]. For instance, endovascular embolization of high-grade cerebral arteriovenous malformations, as reported in recent studies, demonstrates manageable consequences and a positive impact on quality of life [19]. Additionally, the role of therapeutic hypothermia in neuroprotection, particularly in traumatic brain injuries and ischemic stroke, is highlighted as a significant factor in enhancing patient recovery and reducing neurological damage [20]. Thus, all of the above points to a potential additional negative impact of TLOC in patients with a severe stage (WFNS IV–V), while the effect of TLOC on the development of fatal outcomes in patients with a potentially favorable prognosis (WFNS I–III) has not been studied sufficiently. In this context, the novelty of this study lies in the focus on the role of TLOC in patients with WFNS I–III as a prognostic factor, which allows us to propose new approaches to predicting the course of aSAH.

Objective. The study aimed to assess the impact of TLOC during the manifestation of IICAA rupture on the time to fatal outcome in patients with WFNS grades I–III.

Materials and methods

A retrospective single-center analysis was conducted, which included 60 case histories of deceased patients with aSAH from 2000 to 2023. The diagnosis of aSAH was confirmed by CT/CT angiography. Inclusion criteria were deceased patients aged 18 years and older with aSAH, WFNS I-III severity, and available anamnestic data on the presence or absence of TLOC in the manifestation of

ICAA rupture. Exclusion criteria were patients younger than 18 years, incomplete medical documentation, lack of confirmation of the aneurysmal nature of SAH according to imaging studies, traumatic SAH, other causes of intracranial hemorrhage (arteriovenous malformations, neoplasms, coagulopathy, etc.), and scores on the modified WFNS scale IV or V. Patients with a WFNS III score, for whom history taking regarding TLOC might be unreliable, were included only if TLOC was confirmed by eyewitnesses of the event. Cases in which witnesses reported ictus accompanied by tonic-clonic seizures were also excluded.

The study was conducted in accordance with the principles of the Code of Ethics of the World Medical Association (Declaration of Helsinki). This study received ethical approval in accordance with Protocol No. 7 of the meeting of the Bioethics Commission of Odesa National Medical University dated September 30, 2019.

The gender distribution among the studied patients included 33 men (55%) and 27 women (45%). The mean age of the studied patients was 51±15 years, median 49 years, minimum age 18 years, and maximum age 78 years. The patients were divided into two groups depending on the presence or absence of TLOC in the manifestation of ICAA rupture. Group 1 included 22 patients with TLOC (37%), and Group 2 – 38 patients without TLOC. The frequency of TLOC during the manifestation of ICAA rupture is consistent with known literature data [1; 2].

The study groups were compared taking into account age, gender, clinical severity according to the modified WFNS scale, the presence of intracranial and infectious complications, the extent of treatment, as well as the time points of the fatal course in both study groups.

The obtained data were analyzed using descriptive statistics. Parametric methods were used for variables with a normal distribution, and nonparametric methods were used for others (Mann-Whitney test for continuous and ordinal variables, χ^2-test for categorical variables). Kaplan-Meier analysis and log-rank test were used to compare survival. A P-value < 0.05 was considered statistically significant. Calculations were performed in JASP 0.19.2.

Results

Group 1 consisted of 22 patients with TLOC in the manifestation of ICAA rupture, equally distributed by gender: 11 men (50%) and 11 women (50%). The average age of the studied patients was 49±15 years, median – 47 years, minimum age – 23 years, maximum age – 77 years. Group 2 consisted of 38 patients without TLOC in the manifestation of ICAA rupture. Among them, 22 (58%) were men, and 16 (42%) were women. In Group 2, age characteristics similar to Group 1 were observed: mean age – (52±15) years, median – 50 years, minimum age 18 years, maximum age – 78 years. Comparison of the studied groups by gender did not reveal statistically significant differences (χ^2=0.351, p=0.554). Analysis of patient age characteristics also confirmed comparability (U=461.500, p=0.509).

When analyzing the location of ICAA among patients in Group 1, the vast majority had anterior circulation aneurysms (91%). The distribution of localizations was as follows: bifurcation of the internal carotid artery (ICA) was observed in 2 (9%) patients, the middle cerebral artery (MCA) was affected in the M1 segment in 2 (9%) patients, and in the M2 segment in 1 (5%) patient. ICAA of the anterior cerebral artery was found in the A1 segment in 1 (5%) patient. The most common localization was the anterior communicating artery in 11 (50%) patients. In Group 2, similar to Group 1, most patients (35 individuals, 92%) had anterior circulation ICAA. The most common location of ICAA was the anterior communicating artery, observed in 21 patients (55%). The MCA bifurcation was affected in 4 patients (10%), while the M1 and M2 MCA segments accounted for 2 (5%) and 1 (3%) cases, respectively. ICAAs of the ICA bifurcation and the subclinoid part of the ICA were found in 1 patient each (3%). The anterior cerebral artery was affected in the A1 segment in 1 (3%) patient and the A2–A3 segments in 1 (3%) patient. ICAAs of the posterior communicating artery were observed in 3 (7%) patients. ICAAs of the posterior circulation were significantly less common in both groups, aligning with the general trend in the localization of ICAAs. In Group 1, ICAAs of the vertebral artery were observed in 1 patient (5%), at the junction of the superior cerebellar artery with the posterior cerebral artery in another patient (5%). In Group 2, basilar artery bifurcation aneurysms were identified in 2 (5%) patients, and aneurysms of the posterior inferior cerebellar artery were identified in 1 (3%) patient. However, analysis of the location of the ICAA did not reveal significant differences between the groups (χ^2=10.972, p=0.531).

The distribution of modified WFNS scores on admission differed between groups. Group 1 was dominated by patients with WFNS III (11 patients, 50%), indicating a more severe condition of patients with TLOC (Figure 1). In Group 2, however, a more even distribution of patients was observed: WFNS I on admission was recorded in 14 (36%) patients, WFNS II and III – in 12 (32%) patients each (Figure 2).

At the same time, despite a higher proportion of patients with worse clinical conditions in Group 1, statistical analysis did not reveal a significant difference in severity between groups (U=342.000, p=0.218).

Regarding the distribution of intracranial complications, in Group 1, DCI was observed in 13 (59%) patients and hydrocephalus in 7 (32%) patients. In Group 2, DCI was recorded in 12 (32%) patients and hydrocephalus in 7 (18%) patients. Infectious complications in Group 1 were exclusively represented by nosocomial pneumonia (19 patients, 86%), while in Group 2, in addition to nosocomial pneumonia (34 patients, 89%), urinary tract infections (6 patients, 16%) and meningitis (1 patient, 3%) were detected. Although the proportion of DCI was higher in Group 1 compared to Group 2, statistical analysis did not reveal a significant difference (χ^2=5.369, p=0.147).

Thus, the groups were comparable in all analyzed parameters without statistically significant differences. However, analyzing the rate of fatal outcome, it was found that the time to fatal outcome in Group 1 was (14±12) days versus (19±15) days in Group 2, but the median time to death remained the same in both groups and was 13 days. The shortest time to death in Group 1 was 1 day, while in Group 2 it was 3 days. The longest time to death was also

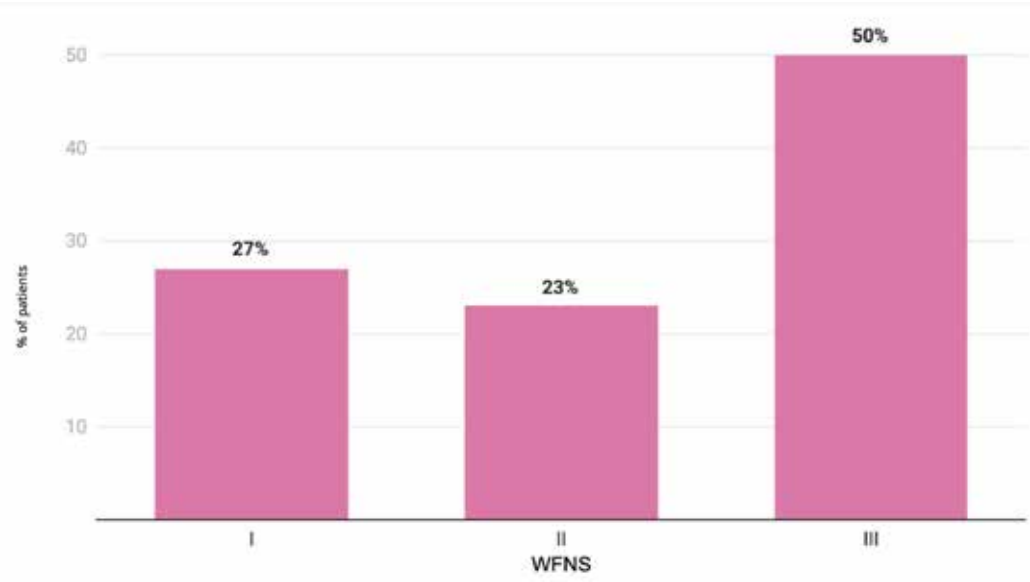

Fig. 1. Severity distribution of patients in Group 1 based on the modified WFNS scale

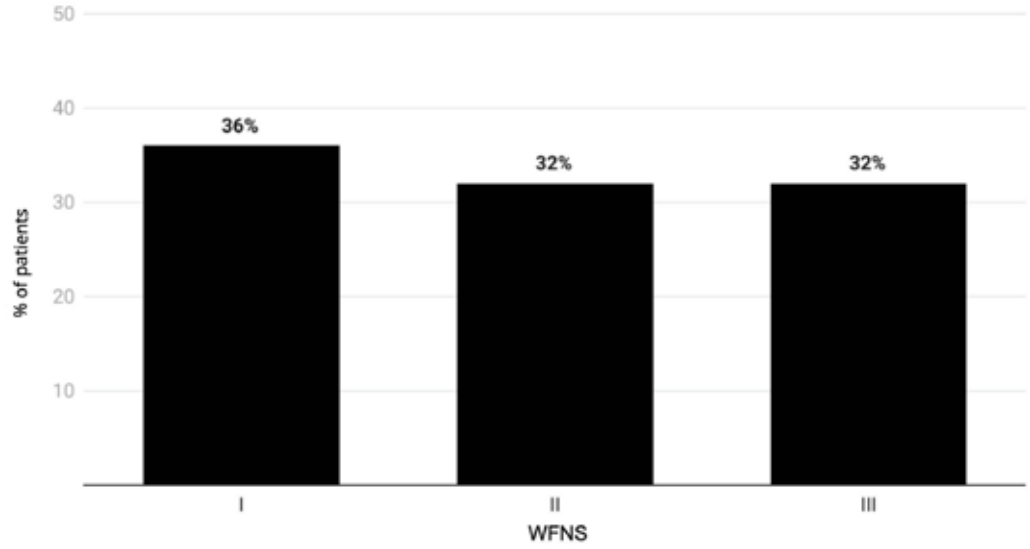

Fig. 2. Severity distribution of patients in Group 2 based on the modified WFNS scale

longer in Group 1 (82 days versus 62 days in Group 2), but statistical analysis confirmed the absence of significant differences between the groups (U=482.500, p=0.335).

Among patients in Group 1, 5 individuals (23%) received conservative treatment. Surgical intervention was performed in 17 patients: ICAA clipping was performed in 13 cases (59%), and ICAA trapping in 4 cases (18%). In Group 2, 14 (37%) patients were treated conservatively. Surgical intervention was performed in 24 patients: ICAA clipping in 19 (50%) patients, ICAA trapping in 3 (8%) patients, and exploratory craniotomy in 2 (5%) patients. Unlike Group 2, where the distribution between conservative treatment and surgical intervention was more balanced, surgical intervention prevailed in Group 1. At the same time, TLOC did not show a statistically significant effect on the choice of treatment tactics (χ^2=3.514, p=0.319).

In both groups, the median survival time remained the same and equaled 13 days, indicating a similarity in the overall dynamics of the course of aSAH regardless of the presence of TLOC. At the same time, the limited median survival was longer in Group 2 (19±2 days) compared to Group 1 (14±3 days), indicating a trend towards shorter survival time in patients with TLOC. The log-rank test results of (χ^2=1.751, p=0.186) did not reveal statistically significant differences between the groups.

For a detailed analysis of survival, the following key time points were selected: survival rates of 90%, 50%, 30%, and 0%. These time points made it possible to assess the survival of patients at different stages after ICAA rupture, starting from the early critical days of hospitalization to the later periods. Thus, in the first days after rupture (days 1–4), the survival rate was 90%. In the first two weeks after ICAA rupture (median time), when half of the patients are still alive, this corresponds to a survival rate of 50%. The 30% survival rate in the context of this study is a critical marker for identifying differences in the rate of fatal outcomes in

the study groups. Achieving complete lethality in the group determines the overall survival time interval.

In the initial stages (up to day 12), the rate of survival decline in both groups is similar. In Group 1, the survival rate decreases to 86.4% on day 6, and in Group 2 – to 89.5% on day 5. This indicates that in the early period, hemorrhage is the main factor affecting survival, regardless of the TLOC. By day 12, both study groups demonstrate the same survival rate, which decreases to approximately 50%. The median survival time for both groups is 13 days. However, after day 13, a significant difference in the rate of survival decline between the groups begins to appear. In Group 1, survival decreases to 36.4% on day 14 and falls below 30% already on day 15. In Group 2, the survival rate (31.6%) is reached only on day 18. Thus, Group 1 shows a faster deterioration, as the case fatality rate reaches 70% approximately three days earlier than in Group 2.

In the later stages, the differences in survival become even more pronounced. In Group 1, all patients die by day 62, while in Group 2 this process continues until day 82. After reaching the critical survival level of 30%, in Group 1, a more rapid deterioration of the patient's condition is observed, while in Group 2, the course of the disease remains more gradual. Therefore, the presence of TLOC contributes to the earlier death of patients after ICAA rupture, particularly in the later stages of the disease. A graphical presentation of the results of the survival analysis for the studied groups is shown in Figure 3.

Discussion

It is important to separate the impact of TLOC from the severity of impaired consciousness. H&H and WFNS scores IV and V are strong predictors of poor prognosis, which can significantly influence the results of the analysis of the role of TLOC as an independent prognostic factor. In addition, patients with such scores, by definition, have a depressed level of consciousness, and the mechanisms of loss of consciousness in these cases may differ from those characteristics of TLOC in aSAH [12]. Given that patients with high H&H and WFNS scores have TLOC by definition, it is advisable to focus on the TLOC assessment on patients with lower severity. This provides a clearer understanding of the impact of TLOC on the prognosis of aSAH.

It is also worth noting that for statistical evaluation, scientific studies consider the severity of the condition of patients with aSAH precisely as a static indicator, determined once at the time of hospitalization. In contrast, TLOC is a dynamic parameter that can appear and disappear depending on the clinical course of the disease. This emphasizes the need to consider TLOC in a broader clinical context, as it can affect prognosis even before the initial diagnosis is made [12–15].

This study has several limitations. First, due to its retrospective nature and single-center design, the results may not apply to all patients, as they relate to only one medical institution and a limited number of cases. The analysis includes only 60 cases, which reduces the precision of the conclusions and does not allow for the inclusion of patients with other causes of SAH or survivors. The assessment of TLOC was based on medical history, which may be inaccurate due to the lack of witnesses at the time of the ICAA rupture. In addition, the duration of TLOC was not documented, which makes it difficult to classify it into short, medium, and long duration. Taking into account the duration of TLOC could allow for a more accurate analysis. Also, there may be recall errors in the prehospital phase, which may affect the assessment of TLOC. To obtain accurate results, multicenter studies with a prospective design are needed in the future.

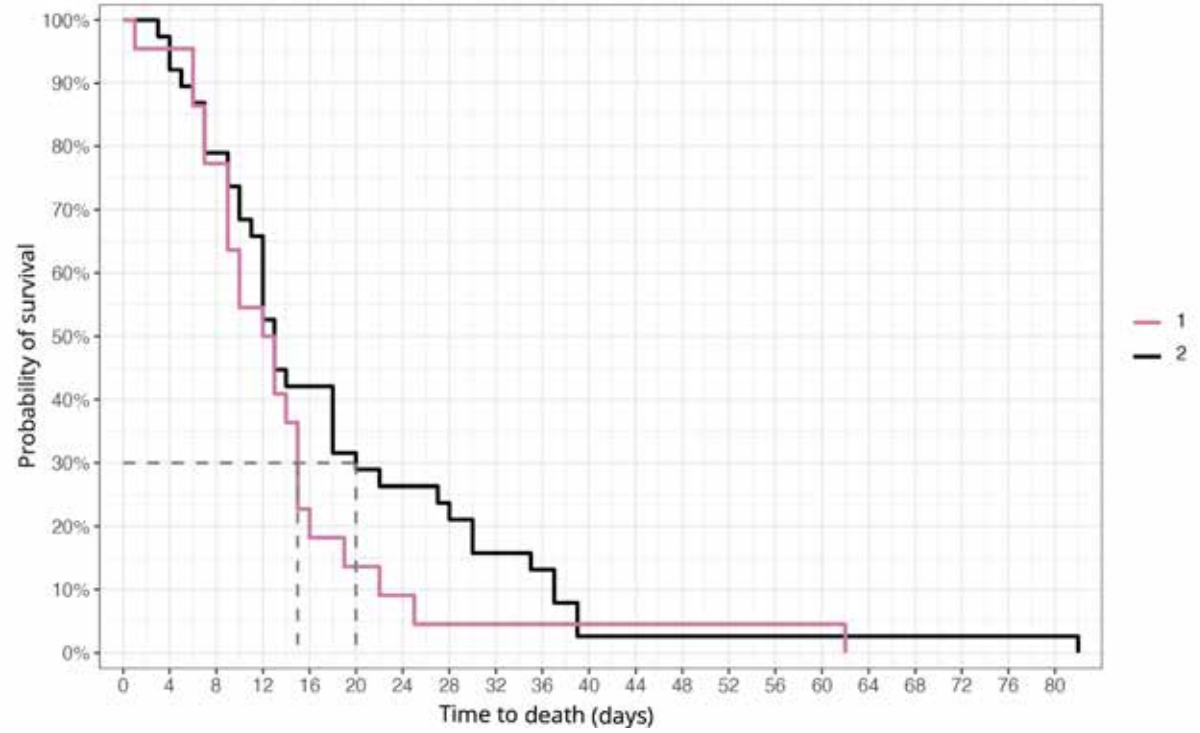

Fig. 3. Kaplan-Meier survival curves for patients in Groups 1 and 2

Conclusions

Patients who lost consciousness tended to have shorter survival times, more rapid deterioration, and a higher risk of death in the first 30 days after ICAA rupture, necessitating more careful monitoring and more intensive therapy in these patients. Patients with TLOC also reached total mortality 20 days earlier than patients who remained conscious at the time of ICAA rupture. These data highlight the importance of considering loss of consciousness as a predictor in assessing the prognosis of patients with ICAA rupture.

Future research in the context of TLOC in aSAH should focus on better understanding the mechanisms of fluctuations in consciousness in the acute period. In particular, it is necessary to investigate the factors leading to TLOC, as well as the impact of different degrees of severity of the condition on the dynamics of consciousness in patients with different degrees of severity. This will allow the development of more accurate methods for assessing the severity of the condition and reduce the risk of errors in determining the necessary treatment.

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