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ORIGINAL RESEARCH / *ÖZGÜN ARAŞTIRMA*

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Short-term Results of Patients with Spontaneous Subarachnoid Hemorrhage in Intensive Care Unit: Single-center Experience

Yoğun Bakımda Spontan Subaraknoid Kanamalı Hastaların Kısa Dönem Sonuçları: Tek Merkez Tecrübeleri

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ABSTRACT *Objective:* Few studies have evaluated patients with spontaneous subarachnoid haemorrhage (sSAH) from an intensivist perspective. This study aimed to report the results of patients with sSAH in a high-volume centre monitored by a team experienced in the fields of brain surgery, interventional radiology and intensive care.
Materials and Methods: Data of patients with sSAH followed up between January 2014 and July 2018 in the intensive care unit (ICU) were retrieved from ICU patient observation charts, file records and hospital automated information system.
Results: This study enrolled 150 patients, of which 61 (40.7%) patients died despite receiving intensive care. Mortality rates between patients with (42.8%) and without (40%) vasospasm were comparable (p=0.917). Vasospasm developed in 37.8% of the 45 patients who underwent endovascular coiling and in 19.2% of those who underwent neurosurgical clipping (p=0.044). The median times that elapsed before endovascular or surgical procedures were 2.5 [interquartile range (IQR): 2-5] days in the surviving group and 2 (IQR: 1-5) days in the deceased group (p=0.164). Blood sodium and blood chloride levels were significantly higher in the deceased group from the third day onward. The median blood sodium level exceeded 142 mEq/L in the deceased group, but was lower than 142 mEq/L on the same day in the surviving group.
Conclusion: The results of this study suggest that Glasgow coma scale (GCS) at admission to the ICU is one of the important factors that affect treatment success. GCS is an important independent factor in selecting the timing or type of treatment (surgical clipping/endovascular coiling) and medical treatments such as nimodipine in patients with sSAH requiring intensive care. In addition, the incidence of vasospasm was higher in patients who underwent endovascular coiling. Increased sodium and chloride values during follow-up are the only parameters significantly associated with mortality.
Keywords: Endovascular procedures, intensive care unit, mortality, spontaneous subarachnoid haemorrhages, subarachnoid haemorrhage therapy, subarachnoid haemorrhage surgery

ÖZ *Amaç:* Spontan subaraknoid kanamalı (sSAK) hastaların yoğun bakımcılar gözüyle değerlendirildiği az sayıda çalışma mevcuttur. Çalışmamızda; mortalitesi ciddi oranda yüksek böyle bir hastalığın beyin cerrahisi, girişimsel radyoloji, yoğun bakım alanında deneyimli bir ekiple takip edildiği bir high volume center'daki takip sonuçlarını paylaşmayı amaçladık.
Gereç ve Yöntem: Yoğun bakım ünitemizde (YBÜ) Ocak 2014-Temmuz 2018 tarihleri arasındaki yaklaşık 5 yıl boyunca izlenen sSAK hastalarının verileri, YBÜ hasta izlem çizelgeleri, dosya kayıtları ve hastane otomasyon sistemi kullanılarak toplanmıştır.
Bulgular: Çalışmaya 150 hasta dahil edildi ve bunların 61'i (%40,7) yoğun bakıma kabul edilmesine rağmen öldü. Vazospazmı olan (%42,8) ve olmayan (%40) hastalar arasındaki ölüm oranları benzerdi (p=0,917). Endovasküler koil uygulanan 45 hastanın %37,8'inde ve beyin cerrahisi klipsleme işlemi yapılan hastaların %19,2'sinde vazospazm gelişti (p=0,044). Endovasküler veya cerrahi prosedürlerden önce geçen medyan süre, hayatta kalan grupta 2,5 [çeyrekler arası aralık (IQR): 2-5] gün ve kaybedilen 2 (IQR: 1-5) gündü (p=0,164). Üçüncü günden itibaren kaybedilen grupta kan sodyum ve kan klorür seviyeleri anlamlı ölçüde yüksekti. Medyan kan sodyum düzeyi kaybedilen grupta 142 mEq/L'yi aştı, ancak hayatta kalan grupta aynı gün 142 mEq/L'den düşüktü.
Sonuç: Bu çalışma ile YBÜ'ye kabulde Glasgow koma skalasının (GKS) tedavi başarısını etkileyen önemli etkilerden biri olduğunu söyleyebiliriz. GKS, yoğun bakım gerektiren sSAK hastalarının tedavisinde zamanlama veya tedavi türü (cerrahi/endovasküler klips) ve nimodipin gibi tıbbi tedavilere ek olarak önemli bir bağımsız faktörüdür. Takipteki sodyum ve klor değerlerinin artışı ise mortalite üzerinde anlamlı bulunan tek parametrelerdir.
Anahtar Kelimeler: Endovasküler prosedürler, yoğun bakım ünitesi, ölüm, spontan subaraknaoid kanama, subaraknoid kanama tedavisi, subaraknoid kanama cerrahisi

Introduction

Subarachnoid hemorrhage (SAH) is a destructive event involving significant mortality and morbidity, frequently as high as 45%. Most SAH derives from ruptured intracranial saccular aneurysms. The presence of aneurysm is generally unexpected until the development of SAH. Following acute bleeding, rebleeding incidence is 3-4% in the first 24 h, and a 1-2% risk every day in the first month (1). One study reported an overall annual adjusted incidence rate of 10.3 per 100,000 person-years for spontaneous SAH (sSAH) [95% confidence interval (CI); 10.2-10.3] (2). Surgical or endovascular aneurysm repair is the only effective method of treatment (3). Crucial causes of mortality and morbidity are arterial vasospasm following acute treatment, delayed ischemic neurological deficits and cerebral infarction. This high-risk patient group must therefore be closely followed-up, particularly in the first 14 days after acute treatment. SAH management guidelines recommend prompt referral to high-volume centers. Decisions concerning aneurysm treatment should be taken by experienced surgeons, interventionalists, and neurological intensive care specialists (3). Although parameters of monitorization have been established in sSAH patients requiring intensive care follow-up, there is still no definite indication regarding which characteristics suggest that patients should be observed in intensive care. Although SAH is a disease with high mortality, the limited availability of intensive care beds makes it difficult for all patients to receive this care. This study aimed to examine the outcomes and characteristics of isolated sSAH patients followed up in intensive care.

Materials and Methods

Study Design

This study involved a retrospective examination of clinical data for patients admitted to the intensive care unit (ICU) of a high-volume university hospital due to sSAH. Ethical approval was granted by Ondokuz Mayıs University Clinical Research Ethics Committee (decision no: 2018/440, date: 28.09.2018). Data for sSAH patients enrolled between January 2014 and July 2018 were retrieved from ICU patient observation charts, records of file, and the hospital system. The study group consisted of 150 patients with the diagnosis of SAH whose data were available and accessible from patient record system during these years. Patients with insufficient data for analysis, traumatic SAH, or aged under 18 or stayed in brain surgery ward were excluded (Figure 1).

Intensive Care Monitoring

Our unit is an 18-bed general ICU within a university hospital. All SAH patients are evaluated at the bedside at daily visits by the relevant member of the neurosurgery teaching staff. Decisions to perform surgical clipping or endovascular coiling are made by consultants from the interventional radiology and neurosurgery departments. This treatment is arranged as early as possible for all patients.

Admission criteria to the ICU for SAH patients are; who could not be extubated after postoperative period following emergency surgery, uncontrollable seizure, mechanical ventilation requirement due to neurological or respiratory instability without surgical or endovascular procedures having yet been performed, close monitoring requirements, hemodynamic instability, or a Glasgow coma score (GCS) <8.

Patient Data

Patients' demographic data, diagnoses responsible for sSAH (aneurysm, arterial malformation, etc.), site of aneurysm, computed tomography (CT) findings other than SAH, treatments administered, timing of the surgical/endovascular procedure performed, vasospasm development, whether or not nimodipine was used, receipt

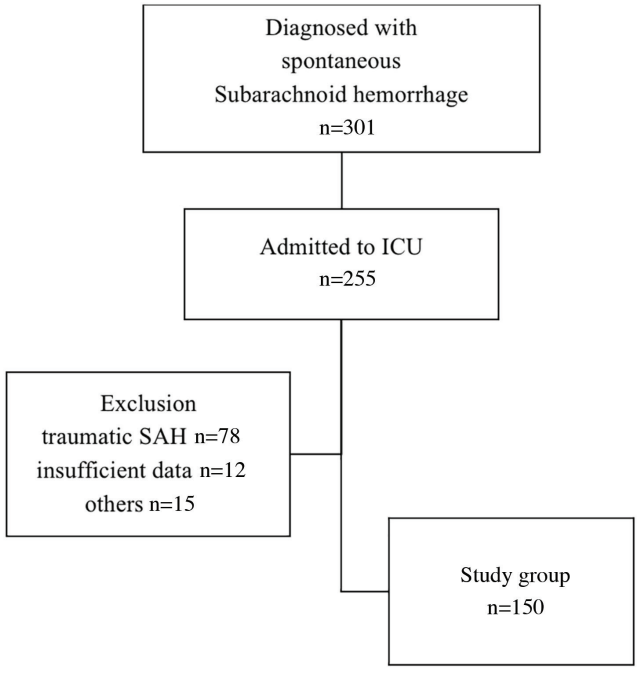

Figure 1. Study flow chart
ICU: Intensive care unit, SAH: subarachnoid hemorrhage

of inotrope/vasopressor therapy, mechanical ventilation requirement, GCS scores at presentation based on presence of neurological deficits according to the World Federation of Neurosurgeons scale (lowest value for each day) (4), length of stay in the ICU and mechanical ventilation, brain death and donation status, important laboratory tests during admission or for a maximum 28 days (Na, Cl, C-reactive protein, white blood cell values, haemoglobin/haematocrit, creatinine, and blood urea nitrogen), and outcomes were recorded.

Clinical Follow-up

In our clinic, CT angiography (CTA) or digital subtraction angiography are performed on patients diagnosed with SAH using cerebral CT before surgical clipping or endovascular coiling procedures. Magnetic resonance imaging is not routinely performed at the preoperative period. After initial treatment all patients underwent early CT scanning, together with recurrent CT scans in the event of neurological instability. External ventricular drainage was applied for brain relaxation in case of patients with suspected intracranial hypertension.

Regulation of blood pressure was applied to ensure systolic blood pressure (SBP) <160 mmHg or mean arterial pressure <110 mmHg, and avoiding hypotension, in all cases. Intracranial pressure was not measured in all patients, only in case of clinical necessity or suspicion. Patients' hourly GCS values were recorded by nurses. Deep vein thrombosis prophylaxis was applied to all patients with pneumatic compression prior to aneurysm treatment. Prophylaxis continued after aneurysm treatment with low molecular weight heparin. Antiseizure drug therapy was not given routinely to all patients. Pain control and ulcer prophylaxis were applied in all cases.

The presence of vasospasm was determined on the basis of clinical and symptomatic criteria. Development of new focal or global neurological disorders that could not be explained in terms of states as hydrocephaly, bleeding, metabolic abnormalities, infection and surgical or endovascular complications was the defining of vasospasm (5,6).

CTA was generally performed to confirm vasospasm in suspected cases. Hypovolemia was avoided in the treatment of vasospasm, and fluid and vasopressor support was applied to establish SBP levels between 160 and 180 mmHg. Blood glucose was regulated in the limits of between 100-180 mg/dL. No patients followed-up due to SAH received nimodipine as a prophylaxis against vasospasm. Only patients with risk factors such as severe bleeding and close proximity to major intracerebral blood vessels, age <50, and hyperglycemia are started on nimodipine for vasospasm. The recommended therapeutic nimodipine dose is 60 mg every 4 h (7,8). No balloon angioplasty or intra-arterial vasodilators were employed for vasospasm treatment in any cases.

Statistical Analysis

IBM SPSS V23 software (Chicago, USA) was used for data analyses. Normality of distribution was examined using the Shapiro-Wilks and Kolmogorov-Smirnov tests. The Kruskal-Wallis, Mann-Whitney U, Student's t, and chi-square tests were used for comparisons between the groups. General linear modeling, and the Wilcoxon and Friedman tests were used for serially measured data. Percentage, mean (± standard deviation), and median (25-75th quartile) values were used for data expressing. The chi-square test was applied to compare qualitative data. Categorical data were expressed as frequency and percentages.

Results

One hundred fifty patients requiring intensive care and diagnosed with sSAH were enrolled in the study. Eighty-nine (59.3%) of the 150 patients were discharged from intensive care, while mortality in intensive care occurred in 61 (40.7%). Ninety (60%) of the 150 patients enrolled were female and 60 (40%) were male. Mean ages in the two groups were similar, at 56.7±13.4 years in the survived group and 56.8±16.79 in the non-survived group (p=0.966).

Aneurysm was the most common diagnosis responsible for SAH in both groups. Fifty-five (44.1%) of the aneurysmal SAH patients died, compared to 50% of patients with arteriovenous malformation related sSAH. No statistically significant difference was determined in mortality rates according to causes of SAH (p=0.361).

Localizations of aneurysm were predominantly in the anterior communicating artery (ACA). Twenty-one (32.8%) of the 64 patients with aneurysm in the ACA were lost, while the mortality rate in middle cerebral artery aneurysms was 13 (34.2%), and 3 (60%) in posterior communicating artery (PCA) aneurysms. Although the mortality rate was higher in PCA aneurysms, no statistically significant difference was determined in mortality rates (p=0.067). The most common accompanying non-SAH findings at CT were intracerebral hematoma and intraventricular hemorrhage, observed in 50 patients each.

Vasospasm developed in 35 (23.3%) of the 150 patients. The mortality rate in patients developing vasospasm (42.8%) was similar to that of the patients without vasospasm (40%) (p=0.917). There was no difference in mortality between the groups. Nimodipine was used in the treatment of 33 of the patients diagnosed of SAH, with mortality occurring in 14 (42.4%) of these. This rate was similar to that in the patients not using nimodipine during treatment (40.1%) (p=0.974).

Thirty-five (23.3%) patients were started on vasopressor/inotrope therapy, with mortality occurring in 30 (85.7%) of these. The mortality rate in patients not started on was 26.9%, significantly lower than in the group receiving vasopressor/inotrope therapy (p<0.001) (Table 1).

Calculation of times elapsed from first presentation to hospital with SAH to first endovascular or surgical procedures revealed a median value of 2 days (1-5 days). Median times were 2.5 days (2-5) and 2 days (1-5) in the survived and non-survived group respectively. The difference between the groups in terms of procedure times was not statistically different (p=0.164). There was no effect on the day of vasospasm development and mortality (p=0.114). These values were 3 days (2.25-5.75) in the survived group and 2 days (1-4) in the non-survived group. Vasospasm developed in 17 (37.8%) of the 45 patients undergoing endovascular coiling and in 14 (19.2%) of the 73 receiving neurosurgical clipping (p=0.044). No statistically significant relation was observed between mortality and duration spent in the emergency department before admission to the ICU. Median waiting duration were 10 h (5-22) in the survived group and 10 h (5-24) in the non-survived group (p=0.780). 81.3% of patients was treated with mechanical ventilation. Median mechanical ventilation duration was 5 days (3.25-8) in the non-survived group and 1 day (1-2.25) in the survived group (p<0.001). Lengths of ICU stay were similar in the two

Table 1. Categorical comparison of the survived and non-survived groups

		Mortality			
		No	Yes		
Parameter		**n (%)**	**n (%)**	**Total**	**p**
Gender	Female	54 (60)	36 (40)	90	
	Male	35 (58.33)	25 (41.67)	60	0.839
Diagnosis	Aneurysm	82 (59.9)	55 (40.1)	137	
	AVM	5 (50)	5 (50)	10	
	Other	2 (66.7)	1 (33.3)	3	0.361
Site of aneurysm	ACA	43 (67.19)	21 (32.81)	64	
	MCA	25 (65.79)	13 (34.21)	38	
	PCA	2 (40)	3 (60)	5	
	Other	19 (44.19)	24 (55.81)	43	0.067
SAH CT findings	Intracerebral hematoma	30 (60)	20 (40)	50	
	Intraventricular hemorrhage	25 (50)	25 (50)	50	
	Subdural hematoma	7 (58.33)	5 (41.67)	12	
	Other	27 (71.05)	11 (28.95)	38	0.263
Treatment applied	Surgical	55 (75.3)	18 (24.7)	73	
	Endovascular	31 (68.9)	14 (27.1)	45	0.58
Vasospasm	No	69 (60)	46 (40)	115	
	Yes	20 (57.14)	15 (42.86)	35	0.917
Nimodipine use	No	70 (59.83)	47 (40.17)	117	
	Yes	19 (57.58)	14 (42.42)	33	0.974
Inotrope/vasopressor support	No	84 (73.04)	31 (26.96)	115	<0.001*
	Yes	5 (14.29)	30 (85.71)	35	

SAH: Subarachnoid hemorrhage, CT: computed tomography, ACA: anterior communicating artery, AVM: arteriovenous malformation, MCA: middle cerebral artery, PCA: posterior communicating artery, *indicates statistical significance

groups (p=0.070) (Table 2). Six (17.6%) of the 34 patients with brain death became organ donors.

The median GCSs at presentation was 9. GCS scores were significantly lower in the first eight days of monitoring in the non-survived group compared to the survived group. At admission median GCS was 12 [interquartile range (IQR): 9-14] in the survived and 5 (IQR: 5-7) in the non-survived groups (p<0.001). In the follow-up days statistically significant different keep going (Figure 2). Both blood sodium and blood chloride levels were similar in the two groups in the first two days, but were significantly higher in the mortality group as of the third day. Median blood sodium levels exceeded 142 mEq/L in the non-survived group, but were lower than 142 mEq/L on the same days in the survived group. Median blood chloride levels were above 105 mEq/L in the non-survived group, but below 105 mEq/L in the survived group. Although statistically significant differences were determined on some days in other laboratory parameters, no significant trend was observed (Figure 2).

Discussion

The present study analyzed data for patients with sSAH followed up in the ICU. While several studies have evaluated the pathophysiology and clinical characteristics of sSAH, uncertainties still exist regarding intensive care management (9-11). In terms of short-term outcome, one meta-analysis involving 33 studies reported mortality rates of 8.3-66.7% in patients with SAH (12). High mortality rate of 45% in cases of aneurysmal SAH was repoeted on the another study (1). Only sSAH patients with neurological, hemodynamic, or respiratory instability were admitted to the study. These patients' median GCS score at presentation was 9, and 81.3% required mechanical ventilation support. Our mortality rate of 40.7% in sSAH patients requiring intensive care management is therefore not surprising. This rate is compatible with general adult intensive care mortality, at between 30% and 65% (13-15). This result may be can explain with arterial vasospasm and delayed ischemic neurological deficits in addition to the severe condition of patients whose were admitted to the ICU. Besides of that SAH guidelines recommend that patients be referred to high-volume centers in the early period, but make no specific reference to the type of ICU (1,16). In the present study, length of stay in the emergency department for critical sSAH patients before admission to the ICU, the timing of the endovascular or surgical treatment applied, and the development of vasospasm had no effect on intensive care mortality. This may be related to all patients being in poor clinical condition and irrespective of rapid application of standardized protocols to all patients. Lott et al. (17) compared specialty ICUs with general ICUs and evaluated critical disease outcomes of various diagnoses. That study involved 124 ICUs and 11,984 patients. No difference was observed in mortality rates between general ICUs and specialty ICUs including neurological intensive care. Egawa et al. (18) reported improved neurological outcomes in SAH patients receiving intensive care including neurointensive care, but longer intensive care stays [median (IQR), 12 (9-14.3) days]. The median intensive care stay in the survived group in the present study was 5 (3-10) days, and 6 (4-12) days in the non-survived group. These short lengths of stay may be explained with correct circulation of intensive care. Our center is the best equipped neurosurgery center in the region, and has considerable experience with rapid diagnosis and treatment of unstable SAH patients being

Table 2. Demographic comparison of the survived and non-survived groups

	Survived	Non-survived	Survived	Non-survived	p**
Parameter	**n (%)**	**n (%)**			
Age (year)	89 (59.3)	61 (40.7)	56.7±13.4	56.8±16.79	0.966
Endovascular/surgical procedure time (days)	86 (72.9)	32 (27.1)	2.5 (2-5)	2 (1-5)	0.164
Days to vasospasm development	20 (57.14)	15 (42.86)	3 (2.25-5.75)	2 (1-4)	0.114
Length of stay in emergency department (hours)	89 (59.3)	61 (40.7)	10 (5-22)	10 (5-24)	0.780
Duration of mechanical ventilation (days)	62 (50.8)	60 (49.2)	1 (1-2.25)	5 (3.25-8)	<0.001*
Length of intensive care stay (days)	89 (59.3)	61 (40.7)	5 (3-10)	6 (4-12)	0.070

Number of patients are not same in all parameters, first two columns show number of patients. Normally distributed data expressed as mean ± standard deviation, non-normally distributed data expressed as median (25-75th quartile).
*Indicates statistical significance, **p value refers to comparison of durations (year, day, hour)

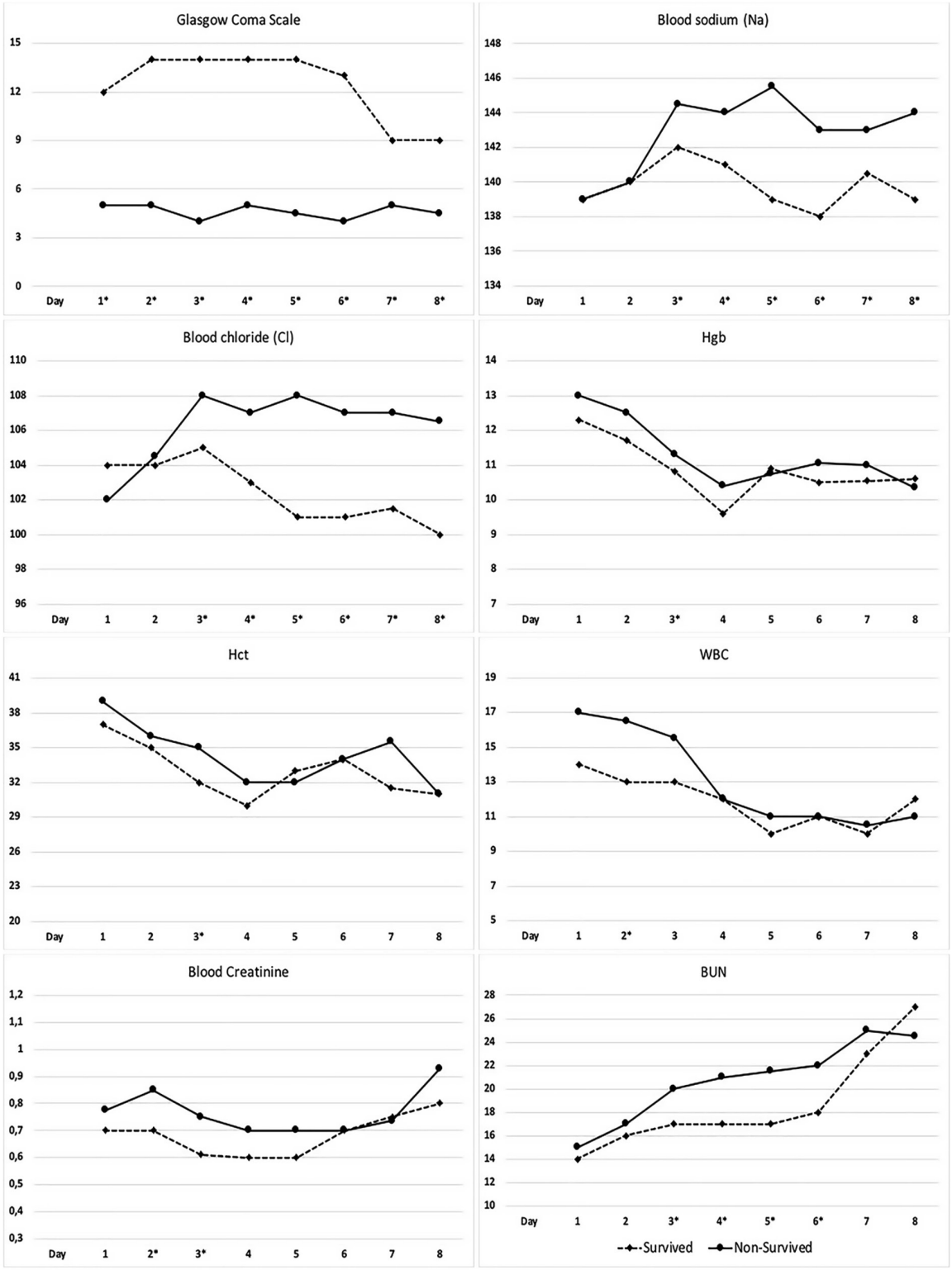

Figure 2. Plot graphs comparing laboratory parameters in the non-survived and survived groups
*Above the days indicates a statistically significant difference between the two groups.

Hgb: Haemoglobin, Hct: haematocrit, BUN: blood urea nitrogen, WBC: white blood cell
Units: Na: mEq/L, Cl: mEq/L, Hgb: g/dL, Htc: %, Leukocyte: 10^9/L, Creatinine: mg/dL, BUN: mmol/L

referred to our ICU. All patients are therefore transferred to our ICU if indicated following rapid triage and stabilization. The great majority of SAH patients in our unit are admitted either postoperatively or in case of mechanical ventilation or requirement of close monitoring, hemodynamic instability, or GCS<8, while other patients are monitored on the ward or under emergency conditions. Patients responding to treatment are rapidly discharged from intensive care after extubation, while patients requiring palliative care are referred to surrounding hospitals and palliative units.

SAH represents 1-7% of all strokes (19). Aneurysm rupture is the cause in 85% of patients (20). The location of the aneurysm is frequently in the ACA (40%). Aneurysm was the most common cause of SAH at a rate of 91.3%, and 46.7% of aneurysms were located in the ACA in the present study. Surgical clipping or endovascular coiling are employed in treatment. Two randomized studies compared endovascular treatment with open surgery for intracranial aneurysms, the (21-24). Although both reported significantly greater obliteration rates and improved durability with open-surgery compared to endovascular procedures, better functional outcomes were achieved at 1 year with endovascular treatment. Surgery was performed on 48.6% of the patients in the present study, and endovascular treatment on 30%, depending on clinical indication. There was no significant difference in terms of short-term intensive care mortality between the two. Similarly, Koivisto et al. (25) were not find significant difference in terms of long-term mortality and morbidity between the two techniques. Whether aneurysm should be repaired using neurosurgical clipping or endovascular coiling depends on the patient's age, the presence of large intracranial hematomas requiring emergency extraction, clinical status, associated illnesses, the size, shape and location of the ruptured aneurysm, the available equipment and individual skills (26). One meta-analysis revealed that the risk of poor outcomes decreased to 23% at 1 year with coiling, compared to 34% following clipping (odds ratio: 1.48, 95% CI: 1.24-1.76), while no difference in mortality was observed (27). In the hands of an experienced surgeon surgical methods of cerebral aneurysms with the evolution of microsurgical techniques is an effective and safe procedure. Since our surgical team is highly experienced in critical cases, surgical rates may have been found to be high at the decision stage. On the other hand treatment at specialized neurosurgical centers is associated with better outcome compared with treatment at lower-volume centers. Aneurysm must be repaired as early as possible, and preferably within 24 h (3). No difference was determined in this study between non-survived patients within this period and surviving patients, and mean time to procedure was 48 h (1-5 days).

Delayed ischemic neurological deficit associated with arterial vasospasm and development of cerebral infarction affect patient outcomes following successful surgical or endovascular ruptured aneurysm repair. Vasospasm is believed to result from spasmogenic substances produced during the breakdown of subarachnoid blood. The cerebral arteries thus contract, and blood flow to the brain is reduced. Although not all patients are symptomatic, vasospasm develops in roughly 70% of SAH patients, with delayed cerebral injury (DCI) occurring in 40% of these (28). Angiographic vasospasm is seen in between 30% and 70% of angiograms performed on the seventh day following SAH, while in 20-30% of patients clinical or subclinical vasospasm is observed. Symptomatic vasospasm has been linked to clinical decline and worse prognosis (29,30). The incidence of development of symptomatic vasospasm was 23.3% and was similar with others. Vasospasm generally appears in 4-14 days, peaks in 7-10 days, and resolves by day 21 (31). Vasospasm was observed in the first seven days in all patients in our study. The current gold standard oral nimodipine, has been shown to decrease the risk of DCI and to be associated with better neurological outcomes (32). Patients are recommended to take nimodipine for 21 days in case of increased risk of DCI and vasospasm (33). However, it is not clear yet the evidence that nimodipine reduceses the incidence of vasospasm (7,8,34,35). In the present study, a low number of patients at risk of vasospasm used nimodipine, but no positive effect was observed on mortality.

SBP is recommended to be maintained at below 160 mmHg in all sSAH patients before aneurysm obliteration. On the other hand, triple-H therapy consists of hemodilution, hypervolemia, and hypertension (36). However, recent studies have shown that hypervolemia and hemodilution are associated with poorer outcomes. New evidence recommends euvolemic hypertension in order to increase cerebral blood flow (32). Other non-medical recommendations include balloon angioplasty and intra-arterial vasodilators. Although there is evidence that these treatments applied are not significantly effective in the prevention of vasospasm (7,8), a difference has been shown between methods in some studies. Mielke et al. (9) reported

that aneurysm clipping was associated with a greater incidence of vasospasm. Similarly in the present study, the incidence of vasospasm was higher in patients undergoing endovascular coiling (p=0.044). However, we could not find difference in mortality between the two groups of developing and non-developing vasospasm (p=0.974). This result can be explained by the prognosis of critical patients with sSAH who need intensive care is already worse than other SAH patients and perhaps there is an asymptomatic group in the group without vasospasm. Isolated asymptomatic angiographic vasospasm is traditionally not usually treated, unless the vasospasm is particularly severe.

Both hypo- and hypernatremia may be seen in the critical care management of SAH patients. Hyponatremia is associated with a longer hospital stay and cerebral infarction, although whether or not it affects neurological outcomes is still controversial (37). It frequently develops due to inappropriate anti-diuretic hormone secretion, cerebral salt loss, and glucocorticoid deficiency (38). We observed a significant increase in blood sodium and chloride levels in the non-survived group from day 3 onward. Median blood sodium levels in the non-survived group were above 142 mEq/L, while median chloride values in the same group were above 105 mEq/L. Similarly, studies have associated high sodium levels with poor neurological outcomes (39,40). In agreement with the present study, Okazaki et al. (41) showed that a cut-off point of 145 mEq/L was associated with poor outcomes. Our results are compatible with those findings. This has been attributed to SAH-related hypothalamic dysfunction triggering central diabetes insipidus (39). On the other hand, the resuscitation fluids selected in patients also affect biological changes.

There are a number of limitations to this study. First, since complete data could not be obtained due to its retrospective nature, pathologies at control CT performed on patients with suspected vasospasm could not be evaluated, and only the first CT findings were recorded. Second, only short-term results were evaluated, and long-term neurological status and mortality are unknown. Third, patients' neurological status was assessed using GCS only, other SAH evaluation scales were not employed. Age-related-co-morbidities capable off affecting patient outcomes were not evaluated. Finally, intracranial pressure was not measured in all cases, and data for all the cases in which it was performed were unavailable, and these were therefore not included in the analysis.

Conclusion

Ours is one of the few studies to evaluate the intensive care outcomes of sSAH patients. While algorithms for the clinical monitoring and treatment of sSAH patients have been produced, interventions capable of reducing mortality in critical patients requiring intensive care are unknown. With this study we could say GCS at admission to ICU is one of the important effects which affects treatment success. GCS is an important independent factor as the timing or type of treatment (surgical clipping/endovascular coiling) and medical treatments such as nimodipine in the treatment of sSAH patients requiring intensive care. Besides in the present study, the incidence of vasospasm was higher in patients undergoing endovascular coiling. Early increased sodium and chloride values were associated with intensive care mortality. This finding shows the need for focus on other parameters in addition to the normal care standard in order to improve mortality in patients with clinically severe SAH, and for the planning of further intensive care prospective studies.

Ethics

Ethics Committee Approval: Approval for the study was granted by the Ondokuz Mayıs University Clinical Research Ethics Committee (decision no: 2018/440, date: 28.09.2018).

Informed Consent: Retrospective study.

Peer-review: Externally peer-reviewed.

Authorship Contributions

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