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ORIGINAL ARTICLE

Medical Complications Drive Length of Stay After Brain Hemorrhage: A Cohort Study

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Abstract
Introduction Longer length of stay (LOS) is associated with higher complications and costs in ICU patients, while hospital protocols may decrease complications and LOS. We hypothesized that medical complications would increase LOS after spontaneous subarachnoid (SAH) and intracerebral (ICH) hemorrhage after accounting for severity of neurologic injury in a cohort of consecutively admitted patients.

Methods We prospectively recorded admission characteristics, hospital complications, and LOS for 122 patients with SAH and 56 patients with ICH from February 2006 through March 2008. A multidisciplinary Neuro-ICU team included a dedicated pharmacist and intensivist on daily rounds. Hospital protocols set glucose control with intravenous insulin, ventilator bundles, pharmacist involvement, and hand hygiene. Associations were explored with univariate statistics (*t*-tests, ANOVA, or non-parametric statistics as appropriate) and linear regression (repeated after log transformation of ICU and hospital LOS).
Results Factors associated with longer LOS after SAH and ICH were similar. In both SAH and ICH the strongest drivers of LOS were infection, fever, and acute lung injury. For SAH, vasospasm and Glasgow Coma Scale were also significant in some models, while in patients with ICH the volume of the initial bleed was significant in some models.
Conclusion LOS after spontaneous brain hemorrhage is driven by medical complications even after the adoption of dedicated intensive care medical staff, pharmacist involvement, and evidence-based protocols for ICU care. Further alterations in care will be necessary to eliminate "preventable" complications and minimize LOS after brain hemorrhage.

Keywords Length of stay · Subarachnoid hemorrhage · Intracerebral hemorrhage · Outcomes · Pneumonia · Bacteremia · Acute lung injury

Abbreviations

ICH	Intracerebral hemorrhage
ICU	Intensive care unit
GCS	Glasgow Coma Scale
LOS	Length of stay
SAH	Subarachnoid hemorrhage
VAP	Ventilator associated pneumonia

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Introduction

Length of stay (LOS) is an important outcome measure because longer LOS is associated with worse outcomes, more nosocomial infection, and increased costs. LOS may be a cause of complications and worse outcome. Pulmonary complications [1] and infection [2] after subarachnoid hemorrhage (SAH) are associated with an increased LOS. In a prospective trial, early tracheostomy for patients with an anticipated long LOS reduced mortality chiefly by reducing the incidence of pneumonia [3]. Medical complications may also be the effect of longer LOS. The longer a patient is ventilated, the greater the risk of ventilator associated pneumonia [4].

A variety of ICU interventions improve morbidity and (in some cases) mortality in the ICU. Intravenous insulin therapy [5, 6], central venous catheter placement protocols [7], ventilator bundles (acid suppression, elevation of the head of the bed, oral hygiene) [8, 9], ventilator weaning protocols [10], high intensity intensivist staffing [11], pharmacist staffing [12], and multidisciplinary collaboration [13] all have prospective data to support them. Specifically in Neuro-ICUs, the involvement of neurointensivists may improve outcomes and reduce LOS [14, 15]. There are few data regarding the incidence and impact of medical complications when all these interventions are present.

Most clinical reports in neurologic critical care present data on the drivers of vital status (mortality), and some on functional status (Rankin or Glasgow Outcome scale). These data usually show that complications increase LOS, but there are few data on what drives LOS as the outcome of interest. In brain hemorrhage the predominant drivers of vital status and functional outcome are neurologic scales (Glasgow Coma Scale), complications (vasospasm), and resuscitation status. We wondered if medical complications would drive LOS after taking neurologic severity into account.

Materials and Methods

Study Population

We prospectively enrolled consecutive patients. SAH was diagnosed by the admission CT scan or by xanthochromia of the cerebrospinal fluid if the CT was non-diagnostic. ICH was diagnosed by CT and acute neurologic symptoms. Patients admitted within 14 days of spontaneous hemorrhage were included; patients with trauma, arteriovenous malformation rupture, vasculitis, and other structural lesions were excluded. All data were prospectively recorded. The study was approved by the Institutional Review Board (IRB). Written informed consent was obtained from the patient or a legally authorized representative in all cases, except when the patient died in hospital or no representative could be located for an incapacitated patient, in which case the IRB approved collection of data in a registry without consent.

Clinical Management

For patients with SAH, diagnostic catheter or CT angiography and aneurysm obliteration with surgical clipping or endovascular coiling were performed as soon as possible. Enteral nimodipine was given unless the systolic blood pressure was <120 mmHg. All patients received pravastatin 40 mg daily. Phenytoin use was minimized [16], generally restricted to patients with a witnessed seizure, epileptiform EEG, or poor-grade patients being transported to us for evaluation and aneurysm obliteration. We maintained central venous pressure ≥ 5 mmHg [17]. An external ventricular drain (EVD) was placed in all patients with symptomatic hydrocephalus or IVH with an abnormal Glasgow Coma Scale (GCS) [18], or later decrease in GCS attributable to hydrocephalus or intraventricular hemorrhage (IVH). Transcranial Doppler (TCD) sonography was performed daily. Vasospasm was defined as any mean transcranial Doppler velocity >120 cm/s or clinical vasospasm (an increase in NIH Stroke Scale ≥ 2 points or decrease in GCS of ≥ 1 point without better clinical explanation).

For patients with ICH, blood pressure was lowered to systolic BP of ≤ 140 mmHg [19]; intravenous nicardipine or labetalol was used if needed. Craniotomy was considered on a case by case basis. An EVD was placed for GCS <15 or significant I.

ICU Management

Critically ill patients in the Neuro-ICU were cared for by attending medical staff and house officers from the Department of Neurological Surgery (BRB, HB) or Neurology with mandatory consultation from a multidisciplinary Neuro-ICU team. The Neuro-ICU team is composed of an attending board-certified in anesthesia critical care (PT) or neurocritical care (AMN, SLB), house staff from neurology, neurological surgery and anesthesiology, and a critical care pharmacist. ICU nurses were expected to join bedside rounds for patients under their care. Ventilator management was at the discretion of the critical care team and implemented by certified respiratory therapists. Tidal volumes and plateau pressures were minimized [20]. Patients with acute lung injury (ALI) were managed with reduced tidal volumes and minimized plateau pressures [21]. Patients were screened daily for extubation and ventilator weaning. All patients in this report were treated with

hospital-wide protocols for intensive glucose control (goal 80–140 mg/dl with insulin infusions), prevention of ventilator associated pneumonia with a "bundle" (elevated head of the bed, gastric acid suppression, oral hygiene), full barrier precautions and "time out" procedures for central venous catheter insertion, and hand hygiene with soap and water or alcohol-based hand rub upon entering and exiting patient rooms. Sequential compression devices were applied to the lower limbs, but anticoagulants were avoided. Deep venous thrombosis (DVT) was confirmed with ultrasound when prompted by typical clinical findings or weekly screenings for non-ambulatory patients.

Clinical data were prospectively collected. We recorded baseline demographic and past medical history data onto standardized forms. A study neurologist performed a neurological and general medical evaluation on admission. Neurological status on admission was assessed with the GCS, classified as in the World Federation of Neurologic Surgeons score (15, 13–14, 7–12, and 3–6). We calculated physiologic derangement with the SAH-Physiologic Derangement Score [22], which is derived from APACHE 2 and accounts for admission acidemia, elevated alveolar–arterial oxygen gradient, abnormal blood pressure, and hyperglycemia. We prospectively recorded the occurrence of bacteremia (any positive blood cultures, with the exception of one bottle of coagulase-negative *Staphylococcus* that was clinically judged to be a contaminant). Pneumonia was diagnosed by US Centers for Disease Control criteria [23] and the date of appearance was recorded. ICH volume was categorized into $\leq$15 ml, 16–30 ml, 31–60 ml, and $>$60 ml because it was not normally distributed. ALI or the acute respiratory distress syndrome (ARDS) was diagnosed by consensus criteria [24]. Fever was confirmed with a core temperature of $\geq$38°C (100.4F) and treated with acetaminophen 650–1000 mg every 6 h and surface cooling blankets (MediTherm III, Gaymar Inc., Orchard Park, NY, USA). The number of days febrile was highly correlated with fever burden (maximum daily temperature minus 38°C summed from days 0 through 13); we present data on days febrile throughout for consistency. These were categorized into quartiles (0, 1–4, 5–8, and 9–14 days febrile) because of non-normality.

Statistical Analysis

Groups were compared with *t*-tests, Mann-Whitney *U*, or ANOVA as appropriate. Univariate associations with LOS were explored first. Variables that were associated with ICU or hospital LOS in univariate analysis ($P \leq 0.05$) were eligible for a multivariate model. We used stepwise linear regression ($P \leq 0.05$ to enter, $P > 0.1$ to exit) to choose the variables most significantly associated with LOS in descending order of importance. ICU and hospital LOS were skewed with a tail to the right (some patients had a very long LOS) so we repeated the stepwise linear regression model with natural logarithm-transformed LOS to see if results were similar. Statistical calculations were made with standard commercial software (SPSS version 16, Chicago, IL).

Results

Patient Characteristics

Of the patients with SAH, 81 (66%) were women, and the mean age was 54.7 $\pm$ 13.7 years. Of the patients with ICH, 27 (48%) were women, and the mean age was 63.9 $\pm$ 14.6 years.

Lower GCS was associated with fewer ventilator-free days, more fever and higher ICH volume in patients with both SAH (Table 1a) and ICH (Table 1b), but not ALI, bacteremia, pneumonia, DVT, or medical history. Clinical variables that had significant associations with ICU LOS are shown in Table 2a, and for hospital LOS in Table 2b. Both neurologic and medical variables were associated with longer LOS in patients with SAH and ICH. Variables that were not associated with a different LOS included NIH Stroke Scale on admission, age, pulmonary embolism, pack-years of cigarette use, correct diagnosis on first physician contact, IVH, history of coronary artery disease or myocardial infarction, sex, or SAH-Physiologic Derangement Score ($P > 0.1$ for all).

Medical complications, rather than neurologic severity of illness, were more strongly associated with LOS for patients with both SAH and ICH. These are described in detail.

SAH and LOS

The results of the stepwise linear regression model for SAH and ICU LOS are shown in Table 3. Both models for ICU LOS and natural logarithm-transformed LOS are shown. Note that both models contain pneumonia, vasospasm, and ALI. Models for hospital LOS and natural logarithm-transformed LOS are shown in Table 4. Note that both models contain bacteremia, days febrile, pneumonia, and ALI.

ICH and ICU LOS

When ICU LOS after ICH was the outcome, days febrile, ventricular drain, pneumonia, and GCS entered the model in that order. When the regression was repeated after natural logarithm transformation of ICU LOS, days febrile, ICH volume, and history of hypertension entered the model.

Table 1 Clinical characteristics of patients with (a) SAH stratified (b) ICH stratified by Glasgow Coma Scale (GCS). GCS was related to ventricular drain ($P = 0.01$), ventilator-free days ($P < 0.001$), days with Tmax $\geq$ 100.4F ($P = 0.002$), and ICH volume ($P = 0.001$) only. Data are N(%) or median [IQR] as appropriate

(a) SAH stratified				
GCS	3–6	7–12	13–14	15
No.	15	14	25	68
ALI/ARDS	3 (20)	3 (21)	4 (16)	3 (4)
DVT	2 (13)	2 (14)	4 (16)	10 (15)
Pneumonia	2 (13)	4 (28)	2 (8)	4 (6)
Ventilator-free days	0 [0–0]	1 [0–8.25]	14 [10–14]	14 [13–14]
Days Tmax $\geq$ 100.4F	5 [1–10]	9 [5–10.25]	5 [1.5–8.5]	2 [0–5.75]
Bacteremia	1 (7)	2 (14)	1 (4)	1 (1)
ICH volume	0 [0–20]	0 [0–0]	0 [0–0.75]	0 [0–0]
Ventricular drain	12 (80)	9 (64)	14 (56)	26 (38)
History of HTN	6 (40)	6 (42)	13 (52)	28 (41)
History of diabetes	1 (7)	2 (14)	1 (4)	3 (4)
Vasospasm	6 (40)	10 (71)	17 (68)	38 (56)
(b) ICH stratified				
GCS	3–6	7–12	13–14	15
No.	10	18	15	13
ALI/ARDS	0	2 (11)	0	1 (8)
DVT	1 (10)	1 (6)	0	0
Pneumonia	2 (2)	5 (28)	2 (13)	0
Ventilator-free days	0 [0–0.5]	5 [0.75–11.25]	14 [4.75–14]	14 [14–14]
Days Tmax $\geq$ 100.4F	1.5 [0–10]	8.5 [1–11.25]	2 [0–6.25]	0 [0–1.25]
Bacteremia	0	2 (11)	0	0
ICH volume	55 [34.5–85]	28 [9.5–52.5]	16 [5–25]	4 [1.5–18]
Ventricular drain	3 (30)	6 (33)	4 (26)	1 (8)
History of HTN	7 (70)	13 (72)	11 (73)	11 (84)
History of diabetes	2 (20)	4 (22)	6 (40)	3 (20)

ICH and Hospital LOS

Days febrile, pneumonia, and ventilator-free days entered the model predicting hospital LOS after ICH. When the outcome was natural logarithm-transformed hospital LOS, days febrile, ventilator-free days, pneumonia, and ICH volume (in that order) entered the model.

Medical Complications as Cause or Effect of Long LOS After SAH

Patients met criteria for pneumonia 6.5 [3.25–10.5] days after hemorrhage, and for ALI or ARDS 5.5 [2–10] days after hemorrhage. In contrast, bacteremia (21 [7.5–27] days) and discovery of DVT (9.5 [5.5–13] days) occurred later. Fever was common throughout the first 14 days after hemorrhage.

Discussion

Medical complications were the primary drivers of LOS after SAH and ICH. Neurologic grade entered multivariate models after medical complications, if at all. These results likely indicate that neurologic grade determines functional outcomes but medical complications drive LOS after SAH and ICH. ICU management and the prevention of early medical complications are probably critical for minimizing LOS after spontaneous brain hemorrhage.

All the models for patients with SAH included pneumonia and ALI. Fever, vasospasm, and bacteremia were also commonly seen. The results for patients with ICH were similar, although a reduced number of patients may have reduced our power to see some associations. These data reinforce the message for intensivists caring for patients with neurologic disease that infection, fever, and lung injury will affect LOS most. Results were similar whether using raw LOS or natural logarithm-transformed LOS, so the models are robust.

ALI, DVT, pneumonia, or history of hypertension or diabetes were not related to GCS for either SAH or ICH on admission. The development of these complications may depend on other factors that may not be captured by the GCS, such as hemiparesis, dysphagia, or ventilatory support. Lower GCS was related to fewer ventilator-free days, higher ICH volume, and more fever.

Table 2 Variables associated with (a) ICU LOS, (b) hospital LOS

	SAH ($N = 122$)			ICH ($N = 56$)		
	N (%)	LOS	P	N (%)	LOS	P
(a) ICU LOS						
Glasgow coma scale			0.03			NS
3–6	15	12.4 ± 11.4		10	5.9 ± 7.5	
7–12	14	20.2 ± 13.1		17	11.1 ± 8.2	
13–14	25	14.3 ± 7.5		15	7.6 ± 12.3	
15	68	12.4 ± 8.6		13	8.8 ± 9.4	
ALI or ARDS	13	23.4 ± 9.9 vs. 12.3 ± 8.9	<0.001	3	20.8 ± 8.8 vs. 8.7 ± 10.0	0.05
Deep venous thrombosis	18	22.5 ± 13.9 vs. 12.3 ± 7.8	0.006	2	14.4 ± 9.7 vs. 9.4 ± 1.3	NS
Pneumonia	12	27.1 ± 12.9 vs. 12.3 ± 7.9	<0.001	8	23.5 ± 8.6 vs. 12.7 ± 11.6	0.05
Ventilator-free days (0–13)			<0.001			0.004
0	19	14.4 ± 13.0		14	6.9 ± 9.8	
1–5	12	18.3 ± 6.6		14	13.8 ± 5.3	
6–13	25	20.1 ± 12.7		5	17.3 ± 13.1	
14	66	10.2 ± 4.9		22	8.9 ± 9.4	
Days with core temperature ≥ 100.4F (days 0–13)			<0.001			<0.001
0	31	8.5 ± 7.9		19	4.9 ± 9.4	
1–3	33	13.1 ± 9.9		19	5.2 ± 7.8	
4–8	33	15.5 ± 9.0		6	19.1 ± 8.9	
9–14	25	18.6 ± 8.6		13	14.9 ± 4.1	
History of hypertension	53	16.3 ± 10.9 vs. 11.8 ± 7.9	0.02	41	9.1 ± 10.2 vs. 8.0 ± 7.0	NS
Bacteremia (except one bottle of *S. epidermidis*)	5	30.9 ± 20.8 vs. 13.1 ± 8.2	NS	2	12.2 ± 1.1 vs. 8.7 ± 9.6	0.04
History of diabetes	7	18.0 ± 14.6 vs. 13.5 ± 9.2	NS	15	14.0 ± 12.3 vs. 7.7 ± 8.9	0.04
Columbia CT Score			0.05	N/A		
0. No blood	2	6.1 ± 1.6				
1. Thin SAH, no bilateral IVH	31	10.1 ± 6.9				
2. Thin SAH, bilateral IVH	2	6.4 ± 4.3				
3. Thick SAH, no bilateral IVH	72	15.4 ± 10.2				
4. Thick SAH, bilateral IVH	15	15.6 ± 10.0				
TCD > 120 cm/sec or clinical vasospasm	71	17.3 ± 10.1 vs. 8.9 ± 6.2	<0.001	N/A		
Volume of ICH			NS			0.03
15 ml or less	117	13.7 ± 9.5		25	8.0 ± 8.1	
16–30 ml	3	12.2 ± 7.1		11	16.2 ± 12.7	
31–60 ml	2	10.4 ± 8.2		12	10.1 ± 11.2	
>60 ml	0			8	3.5 ± 5.0	
Ventricular drain	61	16.9 ± 11.2 vs. 10.6 ± 6.3	<0.001	14	16.7 ± 12.9 vs. 7.0 ± 1.2	0.002
(b) Hospital LOS						
Glasgow coma scale			0.01			0.04
3–6	15	15.5 ± 13.9		10	7.3 ± 9.2	
7–12	14	26.4 ± 13.4		17	19.9 ± 11.9	
13–14	25	20.4 ± 8.6		15	15.1 ± 10.6	
15	68	17.2 ± 8.6		13	14.2 ± 11.7	
ALI or ARDS	13	28.8 ± 8.6 vs. 17.5 ± 9.9	<0.001	3	22.5 ± 7.4 vs. 14.2 ± 12.3	NS
Deep venous thrombosis	18	27.4 ± 12.2 vs. 17.1 ± 9.2	0.003	2	18.9 ± 3.3 vs. 14.1 ± 11.9	NS
Pneumonia	12	32.3 ± 12.1 vs. 17.3 ± 9.1	<0.001	8	23.5 ± 8.6 vs. 12.7 ± 11.6	0.02
Ventilator-free days (0–13)			<0.001			0.001
0	19	16.5 ± 14.7		14	7.5 ± 10.4	

Table 2 continued

	SAH ($N = 122$)			ICH ($N = 56$)		
	N (%)	LOS	*P*	*N* (%)	LOS	*P*
1–5	12	22.7 ± 8.1		14	20.7 ± 8.9	
6–13	25	24.2 ± 11.9		5	26.5 ± 13.7	
14	66	15.5 ± 6.3		22	14.2 ± 11.8	
Days with core temperature ≥ 100.4F (days 0–13)			<0.001			0.001
0	31	12.3 ± 9.5		19	9.5 ± 10.7	
1–3	33	16.9 ± 10.7		16	10.8 ± 11.1	
4–8	33	20.9 ± 8.9		6	25.4 ± 7.6	
9–14	25	25.1 ± 8.4		13	21.2 ± 9.7	
History of hypertension	53	21.4 ± 10.8 vs. 16.5 ± 9.5	0.008	41	14.5 ± 12.3 vs. 13.5 ± 10.3	NS
Bacteremia (except one bottle of *S. epidermidis*)	5	41.1 ± 13.6 vs. 17.7 ± 9.1	<0.001	2	32.3 ± 15.2 vs. 13.6 ± 11.3	0.03
History of diabetes	7	22.5 ± 12.3 vs. 18.4 ± 10.2	NS	15	20.4 ± 13.4 vs. 12.6 ± 11.1	0.03
TCD > 120 cm/s or clinical vasospasm	71	21.8 ± 10.5 vs. 14.4 ± 8.5	<0.001	N/A		
Volume of ICH			NS			0.04
15 ml or less	117	18.7 ± 10.2		25	13.6 ± 8.6	
16–30 ml	3	21.7 ± 16.4		11	23.6 ± 12.5	
31–60 ml	2	14.9 ± 14.6		12	13.1 ± 13.9	
>60 ml	0	18.7 ± 10.3		8	8.4 ± 14.3	
Ventricular drain	61	21.8 ± 11.6 vs. 15.6 ± 7.8	0.001	14	20.2 ± 13.9 vs. 12.8 ± 11.1	NS

Medical complications were seen despite the mandatory involvement of intensivists and pharmacists and evidence-based protocols to optimize patient care. These interventions likely reduced the incidence of infection, minimized antibiotic use, and accelerated ventilator weaning, but were not enough to negate their impact on LOS. Even in hospitals with established protocols, the acceptance and use of every protocol is not universal, and continued innovations are necessary to ensure adherence [25]. Hospital-wide interventions intended to reduce complications (insulin infusions, ventilator bundles, etc.) were in place before we started our prospective databases, so we do not have a comparison group.

Infection might be either a cause (pneumonia early in the hospital course prolongs LOS) or effect (more days on the ventilator leads to pneumonia) of longer LOS in a given patient population. We were careful to prospectively document the timing of each complication, and this is helpful for interpretation. Complications that occurred early (pneumonia and ALI) are more likely to be causes of longer LOS, so successful efforts to combat these complications are most likely to reduce LOS. The impact of pneumonia on ICU and hospital length of stay is similar to that of other large databases [26]. Bacteremia, however, was a late complication (median 21 days from hemorrhage) and reflects patients who already had a long LOS. This is not to say bacteremia is not important, but that preventing it may not reduce LOS. Hospital protocols to improve the sterility of central venous catheter insertion probably contributed to the low risk of early bacteremia. Antibiotic-coated catheters may be helpful for minimizing bacteremia from the central venous catheters that have been in place longer than a week [27].

The optimal treatment of fever and its possible causes are not clear from these data. Pharmacist involvement in narrowing antibiotic coverage and minimizing the use of phenytoin likely reduced fever, but fever cannot generally be avoided in brain hemorrhage because most fever often not due to infection [28]. Highest recorded fever has been associated with longer LOS in a general sample of Neuro-ICU patients [29]. Technologies to control fever, such as endovascular devices [30], blankets [31], and iced saline [32] are effective for fever reduction. We are unaware of high-quality data that show a standing protocol on the treatment of fever reduces LOS or improves outcome. We collected data on fever only from the day of hemorrhage (day 0) through day 13; further temperature elevations were not recorded. Central fever is seen within a few days of SAH, peaks about a week after SAH, and subsides after 14 days [33]. Thus, fever likely drives LOS rather than being a cause of it. We did not record fever 14 or more days after hemorrhage, so we cannot say if fever after day 14 is associated with an increased LOS.

Table 3 Linear regression models for ICU LOS in patients with SAH, in order of entry

SAH—ICU length of stay			SAH—ln ICU length of stay*		
Variable	Est. (95% CI)	*P*	Variable	Est. (95% CI)	*P*
Pneumonia	7.7 (3.3–12.2)	0.001	Days febrile/quartile	1.4 (1.2–1.6)	<0.001
Vasospasm	5.7 (3.3–8.3)	<0.001	Vasospasm	1.7 (1.4–2.2)	<0.001
DVT	6.4 (2.9–9.9)	<0.001	Pneumonia	2.0 (1.3–3.1)	0.002
ALI	6.9 (3.0–10.9)	0.001	GCS/category	1.2 (1.1–1.4)	0.004
Bacteremia	9.6 (3.2–16.1)	0.004	ALI	1.8 (1.1–2.8)	0.009
Ventricular drain	3.4 (0.95–5.9)	0.007			

* Values have been reverse transformed

Table 4 Variables associated with hospital LOS after SAH, in order of variable entry

SAH—hospital length of stay			SAH—ln hospital length of stay*		
Hospital LOS	Est. (95% CI)	*P*	ln hospital LOS	Est. (95% CI)	*P*
Bacteremia	14.5 (7.5–21.8)	<0.001	Days febrile/quartile	1.4 (1.3–1.6)	<0.001
Days febrile/quartile	2.7 (0.9–3.6)	0.001	GCS/category	1.4 (1.2–1.5)	<0.001
Pneumonia	7.5 (2.6–12.2)	0.003	Pneumonia	1.7 (1.2–2.6)	0.01
ALI	7.0 (2.6–11.4)	0.002	ALI	1.8 (1.3–2.7)	0.002
Vasospasm	3.9 (1.1–6.7)	0.006	Bacteremia	1.9 (1.0–3.5)	0.05
DVT	5.3 (1.5–9.2)	0.007			

* Values have been reverse transformed

ALI was common in one large retrospective series of SAH patients and associated with worse outcomes [34]. Strategies to minimize the later development of ALI, such as the minimization of tidal volume and plateau pressures [35], may be helpful for patients with SAH to reduce LOS. Further studies will be needed to accurately predict which patients are most likely to develop ALI in the Neuro-ICU.

We found the presence of a ventricular drain was associated with increased ICU LOS in some models. Hydrocephalus may prolong LOS while a ventricular drain is evaluated for removal. This is typically not done while vasospasm is a clinical concern after SAH, and weaning of the drain may be delayed until the patient is otherwise stable. Clinical studies to assess which patients will require ventricular drainage or a permanent shunt have not yielded clear answers, and protocols for removal of ventricular drains are variable [36]. While most clinicians agree that a ventricular drain should be placed in a patient with brain hemorrhage and a depressed level of consciousness, hydrocephalus remains a predominantly clinical diagnosis, and the lack of standardized diagnostic criteria and protocols for drain removal make it difficult to determine a consistent effect on LOS. Intrathecal tPA hastened clot resolution but did not decrease LOS in a pilot study [37].

Conclusion

LOS after spontaneous brain hemorrhage is primarily driven by medical complications. Specific attention to the prevention of pneumonia, acute lung injury, and fever will probably be the most effective avenues for future research to minimize ICU LOS, hospital LOS, and complications.

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