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Original Article

The Impact of Elevated Mean Arterial Pressure on Mortality in Spontaneous Subarachnoid Hemorrhage

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Introduction: Subarachnoid hemorrhage (SAH) remains a critical neurological emergency with high mortality and morbidity. Mean arterial pressure (MAP) plays an importance role in cerebral perfusion and hemodynamic stability in SAH patients. However, excessive MAP elevation potentially worsening clinical outcomes. **Objective:**This study investigates the impact of elevated MAP on in-hospital mortality among SAH patients. **Method:** A retrospective cross-sectional study was conducted using medical records of SAH patients admitted to Dr. Soetomo Academic Medical Center Hospital from 2013 to 2021. A total of 360 patients met the inclusion criteria. MAP was calculated upon admission and categorized as ≥125 mmHg or <125 mmHg. The primary outcome was in-hospital mortality. Logistic regression analysis was performed to assess the association between MAP and mortality while adjusting for confounders. **Result:** Among 360 SAH patients, 44.8% did not survive hospitalization. The mean age was 54 years, with an initial mean MAP of 117.45±21.6 mmHg. Bivariate analysis showed that MAP ≥125 mmHg significantly increased mortality risk (OR = 1.93; 95% CI: 1.24–2.98; p = 0.002). Multivariate logistic regression identified MAP ≥125 mmHg as an independent predictor of mortality (Adjusted OR = 1.795; p = 0.012), alongside age (Adjusted OR = 2.043; p = 0.004), infection (Adjusted OR = 2.442; p = 0.001), and hydrocephalus (Adjusted OR = 2.174; p = 0.003). **Conclusion:** Elevated MAP (≥125 mmHg) is significantly associated with increased in-hospital mortality in SAH patients. These findings highlight the importance of early hemodynamic

Keywords: Hemodynamic management, Mean arterial pressure, Mortality, Subarachnoid hemorrhage

Highlights
- Elevated MAP is associated with increased in-hospital mortality in SAH patients
- High MAP increases the risk of rebleeding, cerebral edema, and vasospasm
- MAP, age, infection, and hydrocephalus are independent predictors of mortality

Introduction

Mean arterial pressure (MAP) plays an integral role in the management of subarachnoid hemorrhage (SAH), serving as a crucial determinant of patient prognosis.[1] SAH, particularly in its aneurysmal form, remains a life-threatening neurological event with high rates of morbidity and mortality. Its prevalence encompasses approximately 5% of all cases of stroke, with a global incidence estimated at 6.1 per 100,000 person-years.[2] The case fatality rate of SAH ranges from 30% to 50% within the first month following the hemorrhage

and nearly half of the survivors suffer from long-term neurological deficits.[1] These impairments severely affect quality of life, making the management of SAH particularly complex and critical.

MAP is defined as the average arterial pressure throughout a single cardiac cycle. It is a key factor in ensuring adequate cerebral perfusion, which is essential for maintaining neurological function.[3] Maintaining an optimal MAP is critical to preventing secondary brain injury after SAH. The brain relies on a constant and adequate blood supply to function properly, and fluctuations in blood pressure can lead to significant complications. In SAH patients, cerebral autoregulation is often compromised, rendering them particularly vulnerable to changes in blood pressure.[4] As a result, both hypotension and hypertension pose significant risks. Hypotension can lead to cerebral ischemia and delayed cerebral infarction, while elevated MAP can exacerbate cerebral edema, increase the risk of rebleeding, and trigger vasospasm.[5] Vasospasm, a major contributor to delayed cerebral ischemia, is strongly associated with poor neurological outcomes[5].

Given the pivotal role of MAP in patient outcomes, its management in SAH is a complex and nuanced. The primary goal is to maintain cerebral perfusion while minimizing the risks associated with abnormal blood pressure.[6] Several hemodynamic management strategies have been proposed to achieve this, including fluid resuscitation, vasopressor therapy, and the judicious use of antihypertensive medications.[7] Intravenous fluid administration is commonly used as the initial approach to increase blood volume and improve MAP.[8] In contrast, for patients with elevated MAP, antihypertensive medications such as labetalol or nicardipine may be necessary.[9] These medications help lower blood pressure and reduce associated risks. The goal of antihypertensive therapy is to strike a balance—ensuring adequate cerebral perfusion while minimizing the risk of rebleeding or vasospasm.[10]

Despite the clear risks associated with abnormal MAP levels, determining the optimal MAP target for SAH patients remains an area of ongoing debate.[11] Various studies have proposed different MAP targets, often influenced by specific clinical contexts and patient characteristics. Some guidelines suggest maintaining MAP between 70-90 mmHg during the acute phase of SAH management, while others advocate for a more individualized approach based on the response to treatment and comorbidities.[12] This variability underscores the need for further research to develop robust, evidence-based guidelines for MAP management in SAH patients. An individualized approach is crucial, as patients respond differently to treatment.[12] Factors such as age, severity of hemorrhage, and underlying conditions must be considered when determining the optimal MAP target.[13] Continuous monitoring of both neurological status and hemodynamic parameters is essential in making informed treatment decisions and achieving the best possible outcomes.[14]

Understanding the impact of MAP on the prognosis of SAH is fundamental to optimizing patient care. A refined hemodynamic strategy that carefully modulates MAP could not only reduce mortality but also minimize long-term neurological deficits.[15] Although many long-term SAH survivors maintain physical independence, few are entirely free from disability, with cognitive impairment and anxiety remaining common even years after the event.[16] Proper MAP management may help mitigate some of these long-term effects, ultimately improving survivors' quality of life. Future studies should aim to determine the most effective MAP targets and management strategies tailored to different patient populations.[11] Additionally, further research into the relationship between MAP fluctuations and neurological outcomes could provide invaluable insights for optimizing SAH patient care.[17] By advancing our understanding of MAP's role in patient outcomes, we can refine treatment protocols, improve survival rates, reduce long-term disabilities, and contributes to improving the overall quality of care.

Objective

To evaluate the impact of elevated MAP on mortality in patients with spontaneous SAH and its implications for blood pressure management.

Methods

This study employs a retrospective analytical observational design using a cross-sectional approach. The target population consists of all patients admitted to the Neurology Inpatient Unit at Dr. Soetomo Academic Medical Center Hospital between January 1, 2013, and December 31, 2021. The accessible population includes patients diagnosed with non-traumatic SAH who meet the inclusion and exclusion criteria.

Inclusion criteria include patients with non-traumatic SAH within the first 48 hours of onset. Exclusion criteria comprise incomplete medical records, non-aneurysmal causes, recurrent SAH, a history of brain surgery, and SAH accompanied by hyponatremia. A total of 360 patients meeting the inclusion and exclusion criteria were included in the study, using a total sampling technique.

Study variables are categorized into independent, dependent, and confounding variables. The independent variable is MAP, calculated using the formula: MAP = (Systolic Blood Pressure + (2 × Diastolic Blood Pressure)) / 3, recorded upon patient admission to the Emergency Unit.[3] MAP is categorized as ≥125 mmHg and <125 mmHg, determined based on the cut-off value.

The dependent variable is patient mortality, categorized into survival and non-survival based on the patient's outcome during hospitalization. Confounding variables include age (≥60 years and <60 years),[18] hyperglycemia (random blood glucose >150 mg/dL or ≤150 mg/dL),[19] hypertension (systolic blood pressure ≥140 mmHg or diastolic blood pressure ≥90 mmHg), and infection (diagnosed according to the Systemic Inflammatory Response Syndrome [SIRS] criteria).[20]

Data were obtained from medical records, including head CT scans, laboratory results, and patient histories. The study was conducted at the Neurology Department and Medical Records Unit of Dr. Soetomo Academic Medical Center Hospital, Surabaya, between November 2021 and May 2022. The process included selecting eligible subjects, recording clinical data, and calculating MAP.

Statistical analysis was performed using SPSS software. Descriptive statistics summarized patient characteristics, while chi-square tests assessed associations between variables. Simple logistic regression analysis was used to evaluate the effect of MAP on mortality outcomes while controlling for confounding factors. A p-value of <0.05 was considered statistically significant. Results are reported as odds ratios (OR) with 95% confidence intervals (CI).

Ethical approval was obtained from the Research Ethics Committee of Dr. Soetomo Academic Medical Center Hospital (Approval Number: 0901/LOE/301.4.2/V/2022), with all procedures ensuring patient confidentiality and data protection.

Results

A total of 360 subarachnoid hemorrhage (SAH) patients were included in this study, with 198 patients (55.2%) surviving and 162 patients (44.8%) not surviving during hospitalization. The mean age of non-survivors (57.40 ± 12.10 years) was significantly higher than that of survivors (50.83 ± 14.55 years) ($p < 0.001$, independent t-test).

Bivariate analysis indicated that several variables were significantly associated with mortality in SAH patients (Table 1). Patients aged 60 years or older had a higher risk of mortality compared to those younger than 60 years. (OR = 2.29; 95% CI: 1.44-3.62; $p < 0.001$). MAP ≥ 125 mmHg was associated with an increased risk of mortality compared to MAP < 125 mmHg (OR = 1.93; 95% CI: 1.24-2.98; $p = 0.002$). The presence of infection (OR = 2.45; 95% CI: 1.51-4.31; $p < 0.01$), and a Glasgow Coma Scale (GCS) score < 15 (OR = 2.17; 95% CI: 1.36-24.3; $p = 0.001$) were also associated with a higher likelihood of mortality. Additionally, hydrocephalus was significantly associated with increased mortality risk (OR = 2.55; 95% CI: 1.51-4.00; $p < 0.001$).

In contrast, factors such as sex ($p = 0.181$), history of diabetes ($p = 0.31$), history of hypertension ($p = 0.18$), smoking habits ($p = 0.75$), and hyperglycemia ($p = 0.075$) were not significantly associated with mortality in SAH patient.

Hemodynamic parameters showed significant differences between groups (Table 2). The MAP was significantly higher in the non-survivor group (121.82 ± 23.32 mmHg) compared to the survivor group (113.88 ± 19.60 mmHg) ($p = 0.001$, independent t-test), indicating a strong association with in-hospital mortality. Since the normality test indicated that SBP and DBP were not normally distributed, the Mann-Whitney U test was used for these comparisons.

The results showed that the non-survivor group had significantly higher SBP (167.49 ± 32.64 mmHg vs. 156.32 ± 29.72 mmHg, $p = 0.001$) and DBP (98.98 ± 20.86 mmHg vs. 92.66 ± 16.17 mmHg, $p = 0.002$) compared to the survived group. These findings suggest that elevated blood pressure parameters, particularly MAP, are significantly associated with increased in-hospital mortality in SAH patients.

Multivariate logistic regression analysis identified several independent predictors of mortality in SAH patients (Table 3). Age (Adjusted OR = 2.043; $p = 0.004$), MAP (Adjusted OR = 1.795; $p = 0.012$), infection (Adjusted OR = 2.442; $p = 0.001$), and hydrocephalus (Adjusted OR = 2.174; $p = 0.003$) remained significant independent factors contributing to increased mortality risk. These findings underscore the importance of these factors in clinical decision-making and highlight their relevance in guiding the management of SAH patients to reduce mortality risk.

Discussion

SAH is a critical neurological condition with significant mortality and morbidity rates.[21] Effective management requires a comprehensive understanding of key physiological factors, such as blood pressure—particularly MAP.[15] This study identifies a significant association between elevated MAP (≥125 mmHg) and increased mortality in SAH patients, underscoring the importance of careful MAP regulation to improve outcomes.

Elevated MAP plays a crucial role in cerebral perfusion and secondary brain injury following SAH. While adequate MAP is essential for maintaining cerebral blood flow, excessive MAP may exacerbate complications such as vasospasm, rebleeding, and cerebral edema—factors that worsen neurological outcomes and increase mortality risk.[8] Our findings indicate that patients with MAP ≥125 mmHg experienced significantly higher mortality rates, aligning with prior research highlighting the detrimental imparct of elevated blood pressure in SAH. The disruption of cerebral autoregulation due to high

MAP can result in hyperperfusion or hypoperfusion, both of which can cause secondary brain injury.[22]

Beyond MAP, several other factors significantly influence mortality in SAH patients. Multivariate logistic regression analysis identified age, infection, and hydrocephalus as independent predictors of mortality. Advanced age (≥60 years) is well-established as a poor prognostic factor due to diminished physiological reserves and heightened susceptibility to brain injury.[18] Systemic infections, may trigger inflammatory responses and contribute to multi-organ dysfunction.[20] Hydrocephalus, characterized by cerebrospinal fluid accumulation raises intracranial pressure and compromises cerebral perfusion, thereby worsening outcomes.[23]

Table 1. Baseline characteristics of SAH patients stratified by survival outcome

Characteristics		Non-survivors, n(%)	Survivors, n(%)	OR (95%CI)	p-value
Age	≥ 60	64 (39.5)	44 (22.2)	2.29 (1.44-3.62)	<0.001*
	< 60	98 (60.5)	154 (77.8)		
MAP	≥ 125	72 (44.4)	58 (29.3)	1.93 (1.24-2.98)	0.002*
	<125	90 (55.6)	140 (70.7)		
Sex	Male	76 (46.9)	79 (39.9)	1.33 (0.87-2.02)	0.181
	Female	86 (53.1)	119 (60.1)		
History of Diabetes	Yes	20 (12.3)	18 (9.1)	1.40 (0.71-2.76)	0.310
	No	142 (87.7)	180 (90.9)		
History of Hypertension	Yes	100 (61.7)	112 (56.6)	1.24 (0.81-1.89)	0.180
	No	62 (38.3)	86 (43.4)		
Smoking	Yes	63 (38.9)	74 (37.4)	1.07 (0.69-1.63)	0.750
	No	99 (61.1)	124 (62.8)		
Infection	Yes	46 (29.6)	28 (14.1)	2.45 (1.51-4.31)	<0.001*
	No	114 (70.4)	170 (85.9)		
Hyperglycemia	Yes	53 (32.7)	50 (25.3)	1.43 (0.91-2.27)	0.075
	No	109 (67.3)	148 (74.7)		
GCS	<15	124 (76.5)	119 (60.1)	2.17 (1.36-24.3)	0.01*
	15	38 (23.5)	79 (39.9)		
Hydrocephalus	Yes	58 (34.6)	35 (17.7)	2.55 (1.51-4.00)	<0.001*
	No	106 (65.4)	163 (82.3)		

Using Chi-square test; *p<0.05
CI: Confidence Interval, GCS: Glasgow Coma Scale, MAP: Mean Arterial Pressure, OR: Odds Ratio

Table 2. Comparison of hemodynamic parameters between survivors and non-survivors of SAH

Variable	Non-Survivors (n=162)	Survivors (n=198)	p-value
SBP (mmHg)	167.49 ± 32	156.32 ± 29	0.001[a]*
DBP (mmHg)	98.98 ± 20	92.66 ± 16	0.002[a]*
MAP (mmHg)	121.82 ± 23	113.88 ± 19	0.001[b]*

[a]Using Mann-Whitney U-test, [b]Using independent t-test; *p<0.05
DBP: Diastolic Blood Pressure, MAP: Mean Arterial Pressure, SBP: Systolic Blood Pressure

Table 3. Logistic regression analysis of factors associated with mortality in SAH Patients

Variable	Adjusted OR	p-value
Age	2,043	0.004*
MAP	1,795	0.012*
Infection	2,442	0,001*
Hydrocephalus	2,174	0,003*

*p<0.05
OR: Odds Ratio

The relationship between MAP and mortality is further demonstrated by hemodynamic differences between survivors and non-survivors. As shown in Table 2, non-survivors had significantly higher systolic blood pressure (167.49 ± 32.64 mmHg), diastolic blood pressure (98.98 ± 20.86 mmHg), and MAP (121.82 ± 23.32 mmHg) compared to survivors (systolic BP: 156.32 ± 29.72 mmHg, diastolic BP: 92.66 ± 16.17 mmHg, MAP: 113.88 ± 19.60 mmHg), with all values reaching statistical significance. These findings reinforce the role of elevated MAP as a contributor to mortality in SAH.[24]

Logistic regression analysis (Table 3) confirms that elevated MAP (≥125 mmHg) is an independent predictor of mortality (Adjusted OR = 1.795, p = 0.012), alongside age (≥60 years; Adjusted OR = 2.043, p = 0.004), infection (Adjusted OR = 2.442, p = 0.001), and hydrocephalus (Adjusted OR = 2.174, p = 0.003). These results underscore the necessity of a multifaceted approach to SAH management, incorporating optimal MAP control alongside interventions targeting infection prevention and hydrocephalus treatment.[14]

Clinically, these findings suggest that strict blood pressure control—particularly maintaining MAP within an optimal therapeutic range—is crucial in reducing adverse outcomes. Given the strong association between elevated MAP and mortality, further studies are required to determine the ideal MAP threshold for SAH patients and to develop evidence-based guidelines for blood pressure management protocols.[11] Additionally, investigating the pathophysiological

mechanisms linking elevated MAP to poor outcomes may aid in the development of targeted therapeutic strategies.[11]

Consistent with prior studies, our analysis—based on a large cohort and extended observation period—demonstrated a significant association between elevated MAP and mortality in patients with hemorrhagic stroke. Even modest elevations in MAP were predictive of adverse outcomes. Future research should also explore the impact of hypotension, specifically the relationship between reduced MAP and mortality, in this high-risk group.[25]

The mechanisms behind the negative impact of high MAP in SAH patients involve several factors. First, increased MAP can result in cerebral hyperperfusion, potentially damaging already vulnerable brain tissue. Hyperperfusion may compromise vascular integrity and elevate the risk of vasospasm—a narrowing of cerebral arteries that can reduce blood flow.[11] Elevated MAP may also aggravate cerebral edema, thereby increasing intracranial pressure and worsening neurological injury.[26]

Appropriate clinical interventions are essential for effective MAP management in SAH. Antihypertensive agents such as labetalol or nicardipine have been shown to safely lower MAP without significantly reducing cerebral perfusion.[27] However, overly aggressive blood pressure reduction can cause cerebral hypoperfusion, which may worsen clinical outcomes.[5,24] Therefore, individualized MAP targets should be tailored based on patient-specific factors such as aneurysm stability, presence of vasospasm, and cardiovascular status. [28,29] During endovascular procedures such as coiling, maintaining MAP below 110 mmHg and avoiding abrupt fluctuations in blood pressure are key to minimizing the risk of aneurysm rupture; this typically translates to a systolic blood pressure goal below 160 mmHg. [30] complications.

High blood pressure may increase risk of rebleeding and delayed cerebral ischemia (DCI).[5] An observational study by Gathier *et al.* In 2022, involving 1,167 SAH patients further supports this notion. It showed that maintaining MAP below 100 mmHg reduced the risk of rebleeding, whereas MAP below 60 mmHg was associated with an increased risk of DCI.[31]

Despite advancements in management, further research is needed to develop evidence-based guidelines for blood pressure control in SAH patients. This study has several limitations. Its cross-sectional design limits causal inference between MAP and clinical outcomes. As a single-center study, its findings may be generalizable to other populations. Additionally, blood pressure was measured only at baseline in the emergency unit, without accounting for fluctuations during hospitalization, which may influence outcomes.

Future multi-center longitudinal studies with continuous MAP monitoring are crucial to refine MAP thresholds and optimize blood pressure targets to reduce complications.[11] These studies should also examine how demographic and clinical factors such as age, gender, and comorbidities influence the response to MAP regulation. Broad-based investigations can help develop more precise, effective treatment strategies for managing MAP and improving outcomes in SAH patients.

Conclusion

Elevated MAP is a contributing factor to in-hospital mortality in spontaneous SAH patients, though it is the least significant among the independent predictors identified, which include infection, hydrocephalus, and age. This study demonstrates that an elevated MAP (≥125 mmHg) is significantly associated with increased in-hospital mortality. These findings underscore the importance of comprehensive clinical management, with particular focus on infection control, hydrocephalus treatment, and age-related risk, alongside careful hemodynamic monitoring. Further prospective studies are warranted to establish the optimal MAP target for effective SAH management.

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Conflict of interest

The authors declare no conflict of interest related to this study.

Ethic consideration

This study was conducted in accordance with the ethical standards of the Research Ethics Committee of Dr. Soetomo Academic Medical Center Hospital Surabaya. Ethical approval was obtained from the Research Ethics Committee (Ethical Approval Number: 0901/LOE/301.4.2/V/2022). Informed consent was waived due to the retrospective of the study, and patient confidentiality was strictly maintained.

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Author contribution

Achmad Firdaus Sani: Conceptualization, Methodology, Writing–Original Draft, Supervision. **Taurus Laisari:** Data

Curation, Investigation, Formal Analysis. **Muh. Wildan Yahya:** Data Curation, Formal Analysis, Writing–Review and Editing. **Vita Kusuma Rahmawati:** Data Curation, Formal Analysis, Writing–Review and Editing. **Faishol Hamdani:** Data Curation, Formal Analysis, Writing–Review and Editing. **Dedy Kurniawan:** Conceptualization, Methodology, Writing–Original Draft, Supervision. **Sita Setyowatie:** Data Curation, Investigation, Formal Analysis.

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