The THAI *Journal of* SURGERY

Official Publication of The Royal College of Surgeons of Thailand

Vol. 39 October - December 2018 No. 4

Original Article

Traumatic Subarachnoid Hemorrhage Lesion Progression on Computed Tomography and Neurological Outcomes

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Abstract

Objective: **The study aimed to investigate the neurological outcomes among traumatic brain injury patients with traumatic subarachnoid hemorrhage (tSAH) and the prognostic factors of intracranial lesion progression on admission- and subsequent-CT scans.**

Materials and Methods: **The study included moderate to severe traumatic brain injury patients who, had tSAH on admission and a CT scan between: January 2009 to December 2011. All patients, included in the study, had a subsequent CT scan within 48 hours after their initial scan. The outcomes were evaluated with Extended Glasgow Outcome Scale (GOSE) at 6 months after injury.**

Results: **468 patients with moderate to severe traumatic brain injuries were reviewed. 147 patients (31.4%) had tSAH on admission along with a CT scan. Among this group, 38 patients (27.3%) had CT lesion progression. The 6-month mortality rate was 16.5%. Multivariate analysis, factors independently related to unfavorable outcome were age (adjusted OR 1.05, 95% CI 1.02-1.07), the Glasgow Coma Scale score (GCS) (adjusted OR 3.47, 95% CI 1.42-9.99) and lesion progression (adjusted OR 9.15, 95% CI 3.78-27.08)**

Conclusion: **The outcome of patients with tSAH at admission is related to age, admission GCS and significant CT progression.**

Keywords: **Traumatic subarachnoid hemorrhage, Lesion progression, Outcome**

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Introduction

Traumatic brain injury (TBI) is the leading cause of death in young adults and has high magnitude to economic loss especially in low to middle income countries[1,2]. Traumatic subarachnoid hemorrhage (tSAH) is a common lesion that found in moderate to severe traumatic brain injured patients up to 60% in some literatures[3]. This lesion had been clarified as the poor prognostic indicator for a long time[4,5]. Unfortunately, few studies have focused on the patterns or amount of tSAH, the associated lesions on CT scan, and progression of lesion as the independent factors for poor neurological outcome[3,6-9]. But previous reports classified the same characteristics between tSAH and aneurysmal subarachnoid hemorrhage for outcome studies[10]. The patterns of traumatic and aneurysmal subarachnoid hemorrhage are different such as distributions, clearance of blood and time to developed vasospasm[11,12]. In our knowledge, the outcome study of tSAH thickness and subsequent CT scan finding are not found

The aim of this study was to find out relating factors with neurological outcome at 6 months post injury in tSAH patientsand the prognostic factors of intracranial lesion progression.

Material and Medthod

Patients

The present study retrieved the data of 468 moderate to severe TBI patients who admitted to Songklanagarind hospital after injury between January 1, 2009, and December 31, 2011. The study protocol was approved by the ethics committee at faculty of medicine, Prince of Songkla university (EC number: 56-011-10-4-3). From the hospital database, one hundred and forty-seven patients (31.4%) had a diagnosis of tSAH on an admission CT scan. The exclusion criteria were penetrating head injury, patients needing cardiopulmonary resuscitation and brain death at the emergency department. Patients who did not perform subsequent CT scan within 48 hours after admission were not included. Our study excluded data for outcome study from 8 patients that loss follows up at 6 months.

CT evaluation

All patients underwent an initial CT scan within 8 hours after injury and a subsequent CT scan within 48 hours after the initial CT scan. All CT scans were reviewed retrospectively in all moderates to severe traumatic brain injured patients by the first author (AK) which questionable CT findings would be confirmed by certified neuroradiologists.

The amount of tSAH on the CT scan was measured in axial view and recorded the maximum thickness in millimeter. Topographical location of subarachnoid blood on the CT scan was determined and recorded. The associated lesion (or pathology) that related to traumatic brain injury was evaluated such as cerebral contusion, subdural hematoma, epidural hematoma, intraventricular hemorrhage and skull fracture.

Time interval between injury and the initial CT scan was estimated. All subsequent CT scan that performed within 48 hours was studied and used its data from the worse CT scan in case of multiple for subsequent CT scans.

Lesion progression was defined as subsequent CT scan finding change to worsening Marshall's classification[13] or need any surgical intervention depended on CT scan finding.

Outcome

The authors measured global neurological outcome with the Extended Glasgow Outcome Scale at 6 months after injury by chart review and telephone[14]. The outcome was reported in dichotomous fashion that patients in good recovery and moderate disability (upper and lower level) were grouped in "favorable outcome" and others were "unfavorable outcome".

Statistical analysis

Data were analyzed by program R for Mac Os X, version 2.15.1. Results are presented as percentages for categorical data and means with standard deviations for continuous data. Univariate statistical analyses were performed using Pearson chi-square test and Fisher exact test for categorical data and the Mann-Whitney U-test for continuous data. Multiple logistic regressions were performed for Multivariate statistical analyses and reported as adjusted odds ratios and 95% confidence intervals. Statistical significance was p-value < 0.05.

Results

Patient's characteristics

The present resultsrecruited 139 patients with

Table 1 Patients characteristic associated 6-months neurological outcome

Characteristics	All patients (N=139)	GOSE at 6 months		Adjusted OR	95% CI.
		Favorable (N=84)	Unfavorable (N=55)		
Sex					
• Male	104 (74.8)	65 (77.4)	39 (70.9)	1	
• Female	35 (25.2)	19 (22.6)	16 (29.1)	1.40	0.64-3.05
Age, years	139	35.8 ± 13.4	43.3 ± 19.6	1.03	1.01-1.05
GCS					
3-8	94 (67.6)	49 (58.3)	45 (81.8)	3.11	1.44-7.54
9-12	45 (32.4)	35 (41.7)	10 (18.2)	1	
ISS					
1-15	33 (23.7)	28 (33.3)	5 (9.1)	1	
> 15	106 (76.3)	56 (66.7)	50 (91.0)	2.09	1.05-4.27
Pupil unreactive On admission	13 (9.4)	2 (2.4)	11 (20.8)	8.82	2.45-79.60
Hypoxemia, PaO2 < 80 mmHg	11 (7.9)	6 (7.1)	5 (9.1)	1.32	0.36-4.54
Hypotension SBP < 100 mmHg	24 (17.3)	8 (9.5)	16 (29.1)	3.76	1.54-10.37
Hypothermia Temp < 36 °C	11 (7.9)	2 (2.38)	9 (16.4)	6.74	1.81-54.09

GOSE, extended-Glasgow outcome scale; GCS, Glasgow coma score; ISS, injury severity score.

tSAH who completed follow up at 6 months after injury. The mean age of the patients was 38 years (range, 15 - 88 years) and 104 patients (74.8%) were male. The most common injury mechanism was motorcycle accident. Ninety-four patients (67.6%) had severe traumatic brain injuries (GCS ≤ 8) and 76.3% had Injuries Severity Score (ISS) more than 15. The ISS was evaluated after resuscitation. (Table 1)

Admission CT scans, lesion progression and operative management

The mean time from injury to initial CT scan was 95.2 ± 82.8 minutes and mean time from initial CT scan to worse subsequent CT scan was 656.3 ± 428 minutes. Only 14 patients (10.1%) had isolated subarachnoid hemorrhage. Cerebral contusion is a most associated lesion that found in 90 patients (61.2%).

Distribution of tSAH was diffused and usually found in one convexity (68 patients, 48.9%) and both convexity (55 patients, 39.6%).

Subsequent CT scan revealed cerebral ischemia in 27 patients (19.4%). Progressive brain lesion caused to change Marshall's classification or operate were 38 patients (27.3%).

Sixty patients (43.2%) had operated immediately after admission and 22 patients (15.8%) after subsequent CT scan. The majority of operative procedures were decompressive craniectomy in 45 patients.

Clinical outcome and prognostic factors

After injury for 6 months, we used GOSE for evaluated patient's outcome. Result were 23 (16.5%) died and 84 patients (60.5%) had favorable outcomes that all of isolated tSAH patients has included. Prognostic factors are presented in Table 1-3.

On univariate analysis, the 6-month neurological outcome was independently associated with patients who had older age, severe traumatic brain injury, severe ISS, unreactive pupils, present of hypoxemia (PaO2 < 80 mmHg), hypothermia (body temperature < 36.0 ˚C), associated with CT scan shown basal cistern obliteration, maximum thickness of tSAH > 7 mm., intraventricular hemorrhage, cerebral infarction and significant lesion progression on subsequent CT scan.

After controlling for other factors and used multinomial logistic regression analysis, neurological outcome was independently with age (adjusted OR 1.05, 95% CI 1.02-1.07), severe traumatic brain injury (adjusted OR 3.47, 95% CI 1.42 - 9.99) and lesion progression (adjusted OR 9.15, 95% CI 3.78 - 27.08) although tSAH distribution and maximum thickness were not associated.

Table 2 Admission CT scan finding associated 6-months neurological outcome

Admission CT finding	All patients (N=139)	GOSE at 6 months		Adjusted OR	95% CI.
		Favorable (N=84)	Unfavorable (N=55)		
Marshall classification					
• DI II	61 (43.9)	46 (27.3)	15 (27.3)	1	
• DI III	29 (20.9)	14 (16.7)	15 (27.3)	3.21	1.29-8.50
• DI IV	10 (7.2)	6 (7.1)	4 (7.3)	2.08	0.47-8.17
• EML	32 (23.0)	15 (17.9)	17 (31.0)	3.39	1.40-8.77
• NEML	7 (5.0)	3 (3.57)	4 (7.3)	3.86	0.82-22.8
Basal cistern obliteration	72 (51.8)	36 (42.9)	36 (65.5)	2.89	1.25-5.18
Midline deviation ≥ 5 mm	29 (20.9)	15 (17.9)	14 (25.5)	1.57	0.68-3.60
Location of tSAH					
Right convexity	38 (27.3)	23 (27.4)	15 (27.3)	2.54	0.72-13.9
Lt convexity	30 (21.6)	21 (25.0)	9 (16.4)	1.70	0.44-9.53
Both convexity	55 (39.6)	27 (32.1)	28 (50.9)	4.00	1.20-21.2
Right Sylvian	14 (10.1)	10 (11.9)	4 (7.3)	0.71	0.17-2.21
Left Sylvian	16 (11.5)	10 (11.9)	6 (10.9)	1.02	0.31-2.94
Both Sylvian	24 (17.3)	11 (13.1)	13 (23.6)	1.93	0.78-4.97
Interhemispheric	38 (27.3)	21 (25.0)	17 (30.9)	1.34	0.63-2.86
Basal cistern	32 (23.0)	15 (17.9)	17 (30.9)	2.04	0.93-4.62
Maximum thickness of tSAH, mm.					
0.1-7	12 (8.6)	6 (7.1)	6 (10.9)	1	
> 7	25 (18.0)	7 (8.4)	18 (32.7)	2.47	2.07-14.8
IVH	22 (15.8)	9 (10.7)	13 (23.6)	2.52	1.02-6.74
Maximum thickness of EDH > 5 mm	33 (23.7)	24 (28.6)	9 (16.4)	0.50	0.19-1.14
Maximum thickness of SDH > 5 mm	50 (36.0)	25 (29.8)	25 (45.5)	1.95	0.97-4.01
Contusion volume, mL.					
< 20					
≥ 20	128 (92.1)	78 (92.9)	50 (90.9)	1	
	11 (7.9)	6 (7.1)	5 (9.1)	1.32	0.36-4.54

GOSE, extended-Glasgow outcome scale; DI II, diffuse injury grade II; DI III, diffuse injury grade III; DI IV, diffuse injury grade IV; EML, evacuated mass lesion; NEML, non-evacuated mass lesion; tSAH, traumatic subarachnoid hemorrhage; IVH, intraventricular hemorrhage; EDH, epidural hematoma; SDH, subdural hematoma.

Table 3 Subsequent CT scan finding associated 6-months neurological outcome

Subsequent CT finding	All patients (N=139)	GOSE at 6 months		Adjusted OR	95% CI.
		Favorable (N=84)	Unfavorable (N=55)		
Cerebral infarction	27 (19.4)	7 (8.3)	20 (36.4)	5.97	2.45-17.3
Marshall classification					
• DI II	50 (36.0)	41 (48.8)	9 (16.4)	1	
• DI III	27 (19.4)	15 (17.9)	12 (21.8)	3.52	1.28-10.7
• DI IV	8 (5.8)	5 (6.0)	3 (5.5)	2.78	0.49-13.4
• EML	36 (25.9)	18 (21.4)	18 (32.7)	4.37	1.72-12.5
• NEML	18 (12.9)	5 (6.0)	13 (23.6)	10.7	3.34-45.5
Significant lesion progression	38 (27.3)	10 (11.9)	28 (50.9)	7.35	3.29-18.6

GOSE, extended-Glasgow outcome scale; DI II, diffuse injury grade II; DI III, diffuse injury grade III; DI IV, diffuse injury grade IV; EML, evacuated mass lesion; NEML, non-evacuated mass lesion.

Discussion

The elderly and severe traumatic brain injury were found associated with unfavorable neurological outcome in almost traumatic brain injury studies[11,15,16], including patients with traumatic subarachnoid hemorrhage patients in our study. When severe traumatic brain injuries are selected, the rate of unfavorable outcomes was 81.8% and up to 85% in a multicenter study[11]. However, the present study found that all patients with isolated tSAH has favorable outcome. Previous studies have shown that isolated tSAH in all Glasgow Coma Scale score is a relatively less severe finding and not likely to require neurosurgical intervention[17,18].

Despite the consistent evidence of an association between tSAH and worse prognosis, the underlying mechanism is not known. Proposed explanations include the possibilities that tSAH itself initiates or is associated with damaging mechanisms[19] or that SAH is an indicator of greater severity of parenchymal brain damage[20] the same as with other hemorrhagic lesion such as contusion or subdural hematoma. The authors have shown that maximum thickness of tSAH and obliterated basal cistern were independent predictor of unfavorable neurological outcome in univariate analysis. After applied multivariate logistic regression model, both characteristics were not associated with outcomes. In contrast to several previous studies, the prognostic value of the characteristics of tSAH such as maximum thickness or distribution essentially lesion at basal cistern were associated with outcomes[3,9,21,22]. Thus, the prognostic values of thickness of subarachnoid blood and anatomical distribution of blood are still debated.

The relationship between CT progression that change in Marshall classification and/or operative management and unfavorable neurological outcomes was detected in stronger degree than other CT findings. This result was relevance to previous studies[6,11,23].

Thus, evolution of intracranial lesions might be considering in the better prognostic parameter for evaluate TBI patients than admission characteristic alone.

Study Limitation

This study has many limitations. The management has been varied due to lack of protocol driven that decisions about therapeutic interventions were depended on many neurosurgeons at our institution. Furthermore, the study was limited to a single center where management may vary from other institutes. The authors cannot definitively conclude that there were not any non-traumatic SAH in our study due to did not routinely perform a vascular study such as CT angiography or cerebral angiography on all trauma patients with SAH to rule out an antecedent aneurysmal rupture as an etiology for the traumatic event.

Evaluation of SAH has been mentioned in the literatures, including motion artifacts, related to anemia, imaging qualities and interpreter's experience. In terms of the reliability of SAH measurements have reported a moderate agreement for detected tSAH among study centers ($K = 0.57$)[24] and grading system of traumatic subarachnoid hemorrhage were not well studied[25]. In this study, the authors did not have radiographic assessment of cerebral vasospasm with Doppler ultrasound examinations or xenon computed tomography, therefore, present study are not able to relate our data to arterial blood flow but the cerebral ischemia could be detected in small samples and not related to outcomes.

Conclusion

Incidence of tSAH was 31.41% in moderate to severe traumatic brain injured patients but isolated tSAH is rare. About 40% of patients had unfavorable outcome that independent to age, admission GCS and significant CT progression.

Acknowledgment

The authors would like to thank Mr. Andrew Jonathan Tait, International affairs unit, Faculty of medicine, Prince of Songkla university for manuscript review and language advice.

Potential Conflicts of Interest

None

REFERENCES

1. Ministry of Public Health. Bureau of Health Policy and Strategy. Public health statistic 2010. Bangkok: The Ministry; 2010.
2. Ministry of Public Health. National Injury Surveillance. Annual epidermiological surveillance report 2010. Bangkok: The

Ministry; 2010.
3. Wong GK, Yeung JH, Graham CA, Zhu XL, Rainer TH, Poon WS. Neurological outcome in patients with traumatic brain injury and its relationship with computed tomography patterns of traumatic subarachnoid hemorrhage. J Neurosurg 2011;114:1510-5.
4. Maas AI, Marmarou A, Murray GD, Teasdale SG, Steyerberg EW. Prognosis and clinical trial design in traumatic brain injury: The IMPACT study. J Neurotrauma 2007;24:232-8.
5. Perel P, Arango M, Clayton T, Edwards P, Komolafe E, Popcock S, et al. Predicting outcome after traumatic brain injury: Practical prognostic models based on large cohort of international patients. BMJ 2008;336:425-9.
6. Chieregato A, Fainardi E, Morselli-Labate AM, Antonelli V, Compagnone C, Targa L, et al. Factors associated with neurological outcome and lesion progression in traumatic subarachnoid hemorrhage patients. Neurosurgery 2005; 56:671-80.
7. Greene KA, Marciano FF, Johnson BA, Jacobowitz R, Spetzler RF, Harrington TR. Impact of traumatic subarachnoid hemorrhage on outcome in nonpenetrating head injury. Part I: A proposed computerized tomography grading scale. J Neurosurg1995;83:445-52.
8. Ishibashi A, Yokokura Y. Clinical analysis of traumatic subarachnoid hemorrhage. Kurume Med J 1991;38:167-71.
9. Tu CJ, Liu JS, Song DG, Zhen G, Luo HM, Liu WG, et al. Maximum thickness of subarachnoid blood is associated with mortality in patients with traumatic subarachnoid haemorrhage. J Int Med Res 2011;39:1757-65.
10. Husain N, Husain MA, Ahmad T. Outcome of traumatic subarachnoid hemorrhage. APMC 2010;4:166-71.
11. Servadei F, Murray GD, Teasdale GM, Dearden M, Lannotti F, Mass AJ, et al. Traumatic subarachnoid hemorrhage: Demographic and clinical study of 750 patients from the European brain injury consortium survey of head injuries. Neurosurgery 2002;50:261-7.
12. Martin NA, Doberstein C, Zane C, Caron MJ, Thomas K, Becker DP. Posttraumatic cerebral arterial spasm: Transcranial Doppler ultrasound, cerebral blood flow, and angiographic findings. J Neurosurg 1992;77:575-83.
13. Marshall LF, Klauber MR, van Berkum Clark M. A new classification of head injury based on computerized tomography. J Neurosurg 1991;75[Suppl]:S14-20.
14. Shukla D, Devi Bi Fauagrawal A, Agrawal A. Outcome measures for traumatic brain injury. Clin Neurol Neurosurg 2011; 113:435-41.
15. Freytag E. Autopsy findings in head injuries from blunt forces: Statistical evaluation of 1367 cases. Arch Pathol 1963; 75:402-13.
16. Kakarieka A, Braakman R, Schakel EH. Clinical significance of the finding of subarachnoid blood on CT scan after head injury. Acta Neurochir(Wien) 1994; 129:1-5.
17. Lee JJ, Segar DJ, Asaad WF. Comprehensive assessment of isolated traumatic subarachnoid hemorrhage. J Neurotrauma 2014;31:595-609.
18. Phelan HA, Richter AA, Scott WW, Pruitt JH, Madden CJ, Rickert KL, et al. Does isolated traumatic subarachnoid hemorrhage merit a lower intensity level of observation than other traumatic brain injury?. J Neurotrauma 2014;31:1733-6.
19. Harders A, Kakarieka A, Braakman R. Traumatic subarachnoid haemorrhage and its treatment with nimodipine: The German tSAH Study Group. J Neurosurg1996; 85:82-9.
20. Servadei F, Nasi MT, Giuliani G, Cremonini AM, Cenni P, Zappi D, et al. CT prognostic factors in acute subdural hematomas: The value of the "worst" CT scan. Br J Neurosurg2000; 14:110-6.
21. Miki T, Ikeda Y, Utsugi O, Ito H. Evaluation of traumatic subarachnoid haemorrhage on computed tomography. J Clin Neurosci 1998;5:49-57.
22. Morris GF, Marshall LF. A new practical classification of traumatic subarachnoid hemorrhage. Clin Neurol Neurosurg1997;99(Suppl 1):S16.
23. Stein SC, Spettel C, Young G, Ross SE. Delayed and progressive brain injury in closed-head trauma: radiological demonstration. Neurosurgery 1993;32:25-30.
24. Mattioli C, Beretta L, Gerevini S, Veglia F, Ceterio G, Cormio M, et al. Traumatic subarachnoid hemorrhage on the computerized tomography scan obtained at admission: a multicenter assessment of the accuracy of diagnosis and the_potential impact on patient outcome. J Neurosurg2003; 98:37-42.
25. Greene KA, Jacobowitz R, Marciano FF, Johnson BA, Spetzler RF, Harrington TR. Impact of traumatic subarachnoid hemorrhage on outcome in nonpenetrating head injury. Part II: Relationship to clinical course and outcome variables during acute hospitalization. J Trauma 1996;41:964-71.

บทคัดย่อ **เลือดออกใต้ชั้นอะแรคนอยด์ภายหลังสมองบาดเจ็บ: ลักษณะรอยโรคที่เลวลงจากภาพถ่ายรังสีคอมพิวเตอร์สมองและผลลัพธ์การรักษาทางระบบประสาท**

อนุกูล แก้วบริสุทธิ์สกุล, หัชชา ศรีปลั่ง, นครชัย เผื่อนปฐม

วัตถุประสงค์: เพื่อศึกษาผลการรักษาทางระบบประสาทของผู้ได้รับบาดเจ็บสมองที่พบเลือดออกใต้ชั้นอะแรคนอยด์ และศึกษาปัจจัยที่ใช้พยากรณ์การเปลี่ยนแปลงที่เลวลงของรอยโรคจากการบาดเจ็บสมองเมื่อติดตามด้วยภาพถ่ายรังสีคอมพิวเตอร์สมอง

วัสดุและวิธีการ: ศึกษาข้อมูลย้อนหลังในผู้ได้รับบาดเจ็บสมองระดับปานกลางถึงรุนแรงที่พบเลือดออกใต้ชั้นอะแรคนอยด์ตั้งแต่แรกรับจากภาพถ่ายรังสีคอมพิวเตอร์สมองระหว่างเดือนมกราคม ปี ค.ศ. 2009 ถึงเดือนธันวาคม ปี ค.ศ. 2011 โดยผู้ป่วยทุกรายจะต้องได้รับการติดตามภาพถ่ายรังสีคอมพิวเตอร์สมองภายใน 48 ชั่วโมงหลังเข้ารับการรักษา และประเมินผลการรักษาด้วย Extended Glasgow outcome scale (GOSE) ที่ 6 เดือนหลังได้รับบาดเจ็บ

ผลการศึกษา: ผู้ได้รับบาดเจ็บที่ศีรษะระดับปานกลางถึงรุนแรง 468 ราย ผลการตรวจภาพถ่ายรังสีคอมพิวเตอร์สมองตั้งแต่แรกรับในโรงพยาบาล พบเลือดออกใต้ชั้นอะแรคนอยด์ 147 ราย คิดเป็นร้อยละ 31.4 โดยในกลุ่มนี้มีผู้ป่วย 38 รายที่มีรอยโรคที่เลวลงเมื่อติดตามภาพถ่ายรังสีคอมพิวเตอร์สมอง มีอัตราการเสียชีวิตที่ 6 เดือนหลังการบาดเจ็บร้อยละ 16.5 และปัจจัยที่สัมพันธ์กับผลการรักษาที่ไม่ดี คือ อายุมาก (adjusted odds ratio (OR) = 1.05, 95% confidence interval (CI) 1.02-1.07) ระดับความรู้สึกตัวแรกรับต่ำ (adjusted OR 3.47, 95% CI 1.42-9.99) และการตรวจพบรอยโรคที่เลวลง (adjusted OR 9.15, 95% CI 3.78-27.08)

สรุปผลการศึกษา: ปัจจัยที่ส่งผลต่อการรักษาในผู้ป่วยเลือดออกใต้ชั้นอะแรคนอยด์จากอุบัติเหตุคือ อายุ ระดับความรู้สึกตัว และการเปลี่ยนแปลงของรอยโรคในสมอง

คำสำคัญ: เลือดออกใต้ชั้นอะแรคนอยด์จากอุบัติเหตุ, รอยโรคที่เลวลง, ผลการรักษา