Asian Biomedicine Vol. 8 No. 5 October 2014; 609 - 613
DOI: 10.5372/1905-7415.0805.334

Brief communication (Original)

Prognostic value of the Marshall computed tomography classification for traumatic subarachnoid hemorrhage

Fang Wang, Xin Huang, Liang Wen, Jiang-biao Gong, Hao Wang, Gu Li, Ren-ya Zhan, Xiao-feng Yang
Department of Neurosurgery, First Affiliated Hospital, Zhejiang University School of Medicine, Hangzhou 310003, China

Background: The Marshall computed tomography (CT) system for classification of traumatic brain injury (TBI) includes the most important independent prognostic variables except for traumatic subarachnoid hemorrhage (tSAH).
Objectives: To evaluate the prognostic effect of tSAH on different injury types based on the Marshall CT system.
Methods: We performed a retrospective study. All patients with severe closed head injury admitted from February 2011 to July 2012 were included. Their scans were classified into two groups: localized injury and diffuse injury using the Marshall classification. Outcomes were compared between patients with tSAH and those without tSAH among the two groups.
Results: Ninety-six patients were included in this study. Seventy-two (75%) were found to have tSAH, and outcomes significantly negatively correlated with tSAH in both localized injury and diffused injury groups.
Conclusions: tSAH had an important effect on the patients' outcome. Although the Marshall classification includes important independent prognostic variables, tSAH should also be added.

Keywords: Marshall CT classification, severe traumatic brain injury (sTBI), traumatic subarachnoid hemorrhage (tSAH)

The Marshall computed tomography (CT) system is the most widely used neuroimaging classification system for traumatic brain injury (TBI). It uses important independent prognostic variables based on CT scan findings; including the state of the cisterna ambiens, midline shift, and the presence of local lesions, to categorize patients into six different groups (**Table 1**) [1]. However, traumatic subarachnoid hemorrhage (tSAH), which is believed to be another important independent prognostic variable, is not included in this system.

tSAH is common in patients with severe traumatic brain injury (TBI). The incidence of tSAH based on CT findings is 23%–63% [2]. Moreover, many studies have demonstrated that it is also a strong independent prognostic variable in TBI, which should be added to the Marshall system. We performed this study to evaluate the effect of tSAH on patients' outcomes, and to determine whether the Marshall CT classification should include this variable.

Correspondence to: Xiao-feng Yang, Department of Neurosurgery, First Affiliated Hospital, Zhejiang University School of Medicine, Hangzhou 310003, China.
E-mail: zjdxwf@163.com

Materials and methods

Design

A retrospective study was performed at the First Affiliated Hospital, College of Medicine, Zhejiang University. This study was reviewed and approved by the Ethics in Biomedical Research Committee of Zhejiang University. Patients with severe closed head injury, admitted between February 2011 and July 2012, were included. All patients or their families gave informed consent. Severe head injury was defined as a Glasgow Coma Scale (GCS) of ≤8 requiring emergency resuscitation [3]. The Marshall CT system classification was based on a CT scan performed within the first 24 hours after trauma. If there were two or more CT images during this time, the assessment was based on the image that was believed to be more ominous. Patients with associated severe trauma of other organs or who did not have a CT scan taken within the first 24 hours after trauma were excluded.

Data collected from medical records included patients' demographics, cause and nature of injury and in-hospital treatment. Outcome was assessed using the Glasgow Outcome Scale (GOS) at 6 months after injury [4]. To compare the outcome between different

Table 1. The Marshall CT system for classification of TBI

Category	Definition
Diffuse injury I	No visible intracranial pathology seen on CT scan
Diffuse injury II	Cisterns are present with midline shift of 0–5 mm and/or lesions densities present; no high or mixed density lesion >25 ml; may include bone fragments and foreign bodies
Diffuse injury III (swelling)	Cisterns compressed or absent with midline shift of 0–5mm; no high or mixed density lesion >25 mL
Diffuse injury IV (shift)	Midline shift >5 mm; no high or mixed density lesion >25 mL
Evacuated mass lesion	Any lesion surgically evacuated
Non-evacuated mass lesion	High or mixed density lesion >25 ml; not surgically evacuated

patient groups, we defined GOS scores 1, 2, and 3 as poor outcome, and GOS scores 4 and 5 as good outcome.

All initial CT scans were independently evaluated by four observers using the Marshall system and assessed for tSAH based on CT image. Labels on all CT images were covered with a cardboard to blind observers to the date, name, age, and sex. Patients were grouped according to the analysis of CT images and placed in two groups: localized injury and diffuse injury based on the Marshall system.

The research team developed a data sheet, listing relevant descriptors and treatment variables, and used this for review of charts for information regarding TBI.

Statistical analysis

We compared patients with tSAH and those without tSAH to determine the impact of tSAH on outcomes between these patients. Differences between the two groups were compared using independent Student *t* tests for continuous variables, and a Pearson chi-square test or Fisher exact test for ranked variables. Data are presented as mean ± standard deviation and were analyzed using SPSS version 13.0 for Windows (SPSS Inc, Chicago, IL, USA). $P < 0.05$ were considered statistically significant.

Results

Ninety-six patients were included in this study. Seventy-two patients (75%) had tSAH based on CT images. Among them, eight (8%) patients had traumatic intraventricular hemorrhages (tIVH) (**Table 2**).

According to their GOS score 6 months after head trauma, 18 patients (19%) had good recoveries. Among them, eight patients had tSAH. Thirteen patients (14%) had an outcome with moderate disability and, among them, eight patients had tSAH. Nineteen patients (20%) had severe disability and among them 16 patients had tSAH. Twelve patients (13%) were in a vegetative state and among them nine patients had tSAH. Thirty-four patients died and among them, 31 patients had tSAH. Among the eight patients with tIVH, there were three patients who died, three patients in a vegetative state and another two patients with severe disability.

Furthermore, among the patients with tSAH only 16 patients had good outcomes and the number of patients having good outcomes was 22% (16/72). This is in contrast to 63% (15/24) among the patients without tSAH. There was significantly negative correlation with tSAH ($P < 0.05$, **Table 3**).

We also analyzed the influence of tSAH in patients with different injury types based on the Marshall CT system. There were 64 patients with localized injury and 32 with diffuse injury. In the group with localized injury, 29 patients had died, and 21 patients lived in vegetative state or with severe disability. The remaining 14 patients had good outcomes and the patients without tSAH had better outcomes (53%, 8/15) than those with tSAH (12%, 6/49) with statistically significant differences. Among the group with diffuse injury, 5 patients had died and 10 patients lived in a vegetative state or with severe disability. The remaining 17 patients had good outcomes. In this group, patients without tSAH also had better outcomes (78%, 7/9) than those with tSAH (44%, 10/23), but without statistical differences (**Table 4**). There were no differences in the GCS scores of the patients at admission, age, or sex, between the two groups (**Table 5**).

Table 2. Clinical characteristics of 96 patients with severe TBI

Characteristics	No. (%)
Age (years)	
<15	5 (5%)
15–45	44 (45%)
46–65	33 (34%)
>65	14 (15%)
Sex	
Male	71 (74%)
Female	25(26%)
Cause of injury	
Traffic accident	69 (72%)
Fall	15 (16%)
Assault	12 (12%)
GCS score within 12 h postinjury	
3	27 (28%)
4	14 (15%)
5	20 (21%)
6	10 (10%)
7	10 (10%)
8	15 (16%)
Marshall computed tomography classification of TBI	
Class I	0 (0)
Class II	21 (22%)
Class III	6 (6%)
Class IV	5 (5%)
Class V	51 (53%)
Class VI	13 (14%)
tSAH	
With tSAH	72 (75%)
Without tSAH	24 (25%)

GCS, Glasgow Coma Scale; tSAH, traumatic subarachnoid hemorrhage; TBI, traumatic brain injury

Table 3. The outcome of patients

	Good outcome	Poor outcome
With tSAH	16 (22%)	56 (78%)
Without tSAH	15 (63%)	9 (38%)

tSAH, traumatic subarachnoid hemorrhage, *$P < 0.05$

Table 4. The comparison of patients' outcome between the patients with tSAH and those without tSAH

	Diffuse injury group			Localized injury group		
	With tSAH	Without tSAH	*P*	With tSAH	Without tSAH	*P*
Good outcome	10	7	0.08	6	8	0.001*
Poor outcome	13	2		43	7	

tSAH, traumatic subarachnoid hemorrhage, Chi-Square test *$P < 0.05$

Table 5. Differences of GOS score and other factors between suffusion and location group with or without tSAH

	Diffuse injury group			Localized injury group		
	With tSAH	**Without tSAH**	***P***	**With tSAH**	**Without tSAH**	***P***
Patients (n)	23	9		49	15	
GCS	5.78	5.78	0.949	4.59	5.31	0.295
Age (y)	41.48	35.33	0.413	50.39	41.27	0.130
M/F(n)	20/3	5/4	0.057	36/13	10/5	0.611

CT grading, the average grade of the Marshall CT classification; GCS, Glasgow Coma Scale; GOS, Glasgow Outcome Scale; M/F (n), the number of male and female; Operation (n), the number of patients with operations; tSAH, traumatic subarachnoid hemorrhage.

Discussion

Severe TBI carries high mortality and disability. In previous studies, the mortality was between 28.1% and 44%, and in our series it was 35%.

It was reported that 23%–63% of patients with severe TBI had tSAH discovered on CT [2]. In our series, the incidence of tSAH was 75%, which is higher than in previous reports. Patients with subdural hematoma or cerebral contusion are likely to have tSAH that is associated with a worse outcome [2, 5, 6]. Our patients with tSAH also had worse outcomes compared with those without tSAH. Traumatic intraventricular hemorrhage (tIVH), a serious type of tSAH, was more common in our series. With an incidence of 8% it is higher than what was previously reported [7]. This is also associated with higher morbidity and mortality. tIVH was considered to be an independent predictor in previous studies [8] and not only caused by the association to other predictors [9-11].

However, the mechanisms by which tSAH has an additional negative effect on patient outcomes are poorly understood. Whether the relationship of tSAH with poor outcome in TBI is merely an epiphenomenon or the result of some direct cause is unclear. Some investigators believe that tSAH is merely a part of an otherwise severe TBI [12], while others argue that it directly causes additional independent adverse reactions such as vasospasm and ischemia [13]. Chieregato et al. [14] evaluated 141 patients with a CT diagnosis of tSAH to determine whether the amount of subarachnoid blood and the presence of associated parenchymal damage are powerful independent factors associated with poor outcomes. At the present time, no proven treatment regimen aimed specifically at decreasing the detrimental effects of tSAH is known. Calcium channel blockers were thought to control vasospasm, but two large randomized controlled trials showed only a modest increase in the proportion of favorable outcomes in patients with severe head injury treated with nimodipine [15, 16]. After re-analysis, it was concluded that the overall benefit of treating unselected head injured patients with nimodipine is unlikely to be clinically relevant [17]. Given that Armin et al. [18] had concluded that tSAH might primarily be an early indicator of associated and evolving brain injuries, vigilant diagnostic surveillance including serial head CT and prevention of secondary brain damage owing to hypotension, hypoxia and intracranial hypertension might be more cost-effective than attempting to treat potential adverse sequelae associated with tSAH.

At present, the Marshall CT system for classification of TBI is the most commonly used approach for evaluating head trauma, and it is helpful for neurosurgeons to predict outcomes [19]. Although several other classification systems have been developed, the Marshall CT system is still the most commonly used because of its simplicity. This system has provided important information even without inclusion of tSAH. According to Fisher's grading criteria based on CT scans, which is used as a grading system for patients with tSAH [20], prognosis is poor among patients with large clots or a thick layer of SAH, and with significant ICH or IVH [6]. In this study, we analyzed whether tSAH would influence the outcome of different types patients according to the Marshall CT system. Our findings showed that, in patients with localized injury, tSAH shows a negative effect on outcome. We therefore recommend including tSAH in the Marshall CT system.

Conclusion

Our findings show that patients with tSAH had worse outcomes compared with those without tSAH. When an analysis of the influence of tSAH on the outcomes of patients with different types of head trauma was performed, it was found that patients without tSAH had better outcomes compared with those with tSAH in a group of patients with localized injury. Although the Marshall CT system for classification has included most important independent prognostic variables, we recommend that tSAH be added, at least for those with localized injuries.

References

1. Marshall LF, Marshall SB, Klauber MR, Van Berkum Clark M, Eisenberg HM, Jane JA, et al. A new classification of head injury based on computerized tomography. J Neurosurg. 1991; 75:S14-20.
2. Kakarieka A. Review on traumatic subarachnoid hemorrhage. Neurol Res. 1997; 19:230-2.
3. Teasdale GM, Jennett B. Assessment of coma and impaired consciousness. Lancet. 1974; 2:81-4.
4. Jennett B, Bond M. Assessment of outcome after severe brain damage. Lancet. 1975; 1:480-4.
5. 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 Neurosurg. 1995; 83:445-52.
6. Okten AI, Gezercan Y, Ergun R. Traumatic subarachnoid hemorrhage: a prospective study of 58 cases [in Turkish]. Ulus Travma Acil Cerrahi Derg. 2006; 12: 107-14.
7. Atzema C, Mower WR, Hoffman JR, Holmes JF, Killian AJ, Wolfson AB. Prevalence and prognosis of traumatic intraventricular hemorrhage in patients with blunt head trauma. J Trauma. 2006; 60:1010-7.
8. Maas AI, Hukkelhoven CW, Marshall LF, Steyerberg EW. Prediction of outcome in traumatic brain injury with computed tomographic characteristics: a comparison between the computed tomographic classification and combinations of computed tomographic predictors. Neurosurgery. 2005; 57: 1173-82.
9. Cordobes F, de la Fuente M. Intraventricular hemorrhage in severe head injury. J Neurosurg. 1983; 58:217-22.
10. Eisenberg HM, Gary HE Jr, Aldrich EF, Saydjari C, Turner B, Foulkes MA, et al. Initial CT findings in 753 patients with severe head injury. A report from the NIH traumatic coma data bank. J Neurosurg. 1990; 73:688-98.
11. Lee JP, Lui TN, Chang CN. Acute post-traumatic intraventricular hemorrhage analysis of 25 patients with emphasis on final outcome. Acta Neurol Scand. 1991; 84:85-90.
12. Chang EF, Meeker M, Holland MC. Acute traumatic intraparenchymal hemorrhage: risk factors for progression in the early post-injury period. Neurosurgery. 2006; 58:647-56.
13. Taneda M, Kataoka K, Akai F, Asai T, Sakata I. Traumatic subarachnoid hemorrhage as a predictable indicator of delayed ischemic symptoms. J Neurosurg. 1996; 84:762-8.
14. 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.
15. Bailey I, Bell A, Gray J, Gullan R, Heiskanan O, Marks PV, et al. A trial of the effect of nimodipine on outcome after head injury. Acta Neurochir (Wien). 1991; 110: 97-105.
16. Harders A, Kakarieka A, Braakman R. Traumatic subarachnoid hemorrhage and its treatment with nimodipine. German tSAH Study Group. J Neurosurg. 1996; 85:82-9.
17. Murray GD, Teasdale GM, Schmitz H. Nimodipine in traumatic subarachnoid haemorrhage: a re-analysis of the HIT I and HIT II trials. Acta Neurochir (Wien). 1996; 138:1163-7.
18. Armin SS, Colohan AR, Zhang JH. Traumatic subarachnoid hemorrhage: our current understanding and its evolution over the past half century. Neurol Res. 2006; 28:445-52.
19. Hiler M, Czosnyka M, Hutchinson P, Balestreri M, Smielewski P, Matta B, et al. Predictive value of initial computerized tomography scan, intracranial pressure, and state of autoregulation in patients with traumatic brain injury. J Neurosurg. 2006; 104:731-7.
20. Fisher CM, Kistler JP, Davis JM. Relation of cerebral vasospasm to subarachnoid hemorrhage visualized by computerized tomographic scanning. Neurosurgery. 1980; 6:1-9.