

Clinical Article

Clinical Features of Acute Subdural Hematomas Caused by Ruptured Intracranial Aneurysms

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Objective : Spontaneous acute subdural hematomas (aSDH) secondary to ruptured intracranial aneurysms are rarely reported. This report reviews the clinical features, diagnostic modalities, treatments, and outcomes of this unusual and often fatal condition.

Methods : We performed a database search for all cases of intracranial aneurysms treated at our hospital between 2005 and 2010. Patients with ruptured intracranial aneurysms who presented with aSDH on initial computed tomography (CT) were selected for inclusion. The clinical conditions, radiologic findings, treatments, and outcomes were assessed.

Results : A total of 551 patients were treated for ruptured intracranial aneurysms during the review period. We selected 23 patients (4.2%) who presented with spontaneous aSDH on initial CT. Ruptured aneurysms were detected on initial 3D-CT angiography in all cases. All ruptured aneurysms were located in the anterior portion of the circle of Willis. The World Federation of Neurosurgical Societies grade on admission was V in 17 cases (73.9%). Immediate decompressive craniotomy was performed 22 cases (95.7%). Obliteration of the ruptured aneurysm was achieved in all cases. The Glasgow outcome scales for the cases were good recovery in 5 cases (21.7%), moderate disability to vegetative in 7 cases (30.4%), and death in 11 cases (47.8%).

Conclusion : Spontaneous aSDH caused by a ruptured intracranial aneurysm is rare pattern of aneurysmal subarachnoid hemorrhage. For early detection of aneurysm, 3D-CT angiography is useful. Early decompression with obliteration of the aneurysm is recommended. Outcomes were correlated with the clinical grade and CT findings on admission.

Key Words : Acute subdural hematoma · Subarachnoid hemorrhage · Intracranial aneurysm · CT angiography.

INTRODUCTION

Subarachnoid hemorrhage (SAH) caused by a ruptured intracranial aneurysm is a devastating disease with high mortality and morbidity⁷. SAH associated with acute subdural hematoma (aSDH) originating from ruptured intracranial aneurysms is rare and carries a poor prognosis^{8,15,20,24}. The majority of post-traumatic aSDH result from vascular injury in the brain cortex. Because of rapid clinical deterioration, early decompression is mandatory, but the prognosis remains poor in many cases. Previous investigators have reported small case series of spontaneous aSDH caused by an aneurysmal rupture^{2,12,20,24}. The majority of these series reported poor clinical grades and suggested the use of various detecting modalities and treatment plans^{5,8,11,15,18}. However, these cases were not numerous and consistently col-

lected single-center experiences are extremely rare. The objective of this report was to review and discuss the clinical aspects of our single-center experience with 23 cases of SAH associated with an aSDH originating from a ruptured intracranial aneurysm.

MATERIALS AND METHODS

All patients with ruptured intracranial aneurysms who were admitted to our department between January 2005 and December 2010 were identified. The medical records and films of the patients were retrospectively reviewed. We selected cases of aSDH secondary to a ruptured intracranial aneurysm. The criteria for selection were; 1) no history of head trauma, 2) radiologist-confirmed definitive subdural hematoma on initial computed tomography (CT) imaging, 3) CT angiography or digital subtraction angiography (DSA) demonstrating an intracranial aneurysm, and 4) a visible subdural hematoma with evidence of a ruptured aneurysm by operative report. The following information was collected and assessed : 1) patient demographic data; 2) clinical status on admission according to the Glasgow Coma Scale (GCS) and the World Federation of Neurosurgical

• Received : March 23, 2011 • Revised : May 2, 2011

• Accepted : July 1, 2011

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Societies (WFNS) grade; 3) location of the ruptured aneurysm; 4) existence of the intracerebral hematoma (ICH), intraventricular hematoma (IVH), midline shifting (≥ 5 mm)^{13,25}, and thickness of subdural hematoma, Fisher grade of the SAH; 5) the methods of treatment received; and 6) outcome evaluated by the Glasgow Outcome Scale. To assess statistical significance, frequencies were evaluated with the chi-square test (SPSS for windows version 13.0 : SPSS INC., Chicago, IL). *p*-values <0.05 were considered significant.

RESULTS

A total of 551 patients were treated for ruptured intracranial

aneurysms between January 2005 and December 2010 at our institution. We selected 23 patients (4.2%) who presented with aSDH caused by aneurysmal rupture and who met our inclusion criteria. The clinical characteristics, radiologic findings, treatments, and outcomes of these 23 patients are summarized in Table 1. Eight patients (34.8%) were male and 15 (65.2%) were female. The mean patient age was 55.9±17.6 years (range, 26-85 years). Ten patients (43.5%) had been treated for hypertension. One patient (Case 5) underwent craniotomy and clipping of a ruptured anterior communicating artery (ACoA) aneurysm 3 years prior to admission. Two patients (Cases 15, 17) had a history of cerebral infarction. On admission, the mean GCS score was 7.0 (range, 3-15), the WFNS grade was grade V

Table 1. Summary of clinical and radiological findings of 23 patients presenting with aSDH caused by ruptured intracranial aneurysms

Case	Age/ Sex	GCS	WFNS grade	Location of aneurysm	Fisher grade	Presence of ICH	Presence of IVH	Midline shifting*	Thickness of SDH (cm)	Treatment	GOS	Complication
1	54/F	6	V	Acoma	IV	(+)	(-)	(+)	>1	Decompressive hemi-craniectomy+Clipping	5	
2	36/M	5	V	MCA (Lt.)	IV	(-)	(-)	(+)	>1	Decompressive hemi-craniectomy+Clipping	5	
3	32/M	5	V	MCA (Rt.)	IV	(+)	(-)	(+)	<1	Decompressive hemi-craniectomy+Clipping	1	
4	73/M	6	V	ACoA	IV	(+)	(-)	(+)	>1	Craniotomy+Clipping	5	Hydrocephalus
5	60/F	5	V	ACoA	IV	(+)	(-)	(+)	>1	Craniotomy+Clipping	1	
6	71/F	14	II	PCoA (Rt.)	IV	(-)	(+)	(-)	<1	Craniotomy+Clipping	2	Vasospasm
7	49/M	12	IV	MCA (Lt.)	IV	(-)	(+)	(+)	<1	Craniotomy+Clipping	5	
8	38/M	15	I	MCA (Rt.)	III	(-)	(-)	(-)	<1	Craniotomy+Clipping	5	
9	52/M	3	V	MCA (Rt.)	III	(-)	(-)	(+)	>1	Decompressive hemi-craniectomy+Coiling	2	Hydrocephalus
10	51/F	4	V	MCA (Lt.)	IV	(+)	(+)	(+)	<1	Craniotomy+Clipping	1	Cerebral edema
11	70/F	4	V	distal ACA (Lt.)	IV	(+)	(+)	(+)	<1	Craniotomy+Clipping	1	
12	85/F	13	III	MCA (Lt.)	IV	(+)	(+)	(-)	>1	Coiling	1	Rebleeding
13	31/M	6	V	ACoA	IV	(+)	(+)	(+)	>1	Decompressive hemi-craniectomy+Clipping	3	
14	80/F	6	V	PCoA (Rt.)	IV	(+)	(+)	(+)	>1	Craniotomy+Clipping	3	
15	77/F	9	IV	distal ACA (Rt.)	IV	(+)	(+)	(+)	>1	Decompressive hemi-craniectomy+Clipping	3	
16	73/F	5	V	MCA (Rt.)	IV	(+)	(+)	(+)	>1	Decompressive hemi-craniectomy+Clipping	1	
17	79/F	4	V	PCoA (Rt.)	IV	(-)	(+)	(+)	>1	Decompressive hemi-craniectomy+Clipping	1	
18	42/F	5	V	MCA (Rt.)	IV	(+)	(+)	(+)	>1	Craniotomy+Clipping	1	
19	61/M	3	V	MCA (Rt.)	IV	(+)	(+)	(+)	<1	Decompressive hemi-craniectomy+Clipping	1	
20	54/F	6	V	ACoA	IV	(+)	(-)	(+)	<1	Decompressive hemi-craniectomy+Clipping	3	
21	26/F	5	V	ICA Bifur. (Rt.)	IV	(+)	(-)	(+)	<1	Craniotomy+Clipping	1	Cerebral edema
22	41/F	4	V	MCA (Lt.)	IV	(+)	(+)	(+)	<1	Decompressive hemi-craniectomy+Clipping	1	
23	51/F	15	I	PCoA (Rt.)	III	(-)	(-)	(-)	<1	Craniotomy+Clipping	4	Vasospasm

*Midline shifting was defined the distance of midline structure from midline ≥ 5 mm. GCS : Glasgow Coma Scale, WFNS : World Federation Neurological Scale, ICH : intracerebral hemorrhage, IVH : intraventricular hemorrhage, GOS : glasgow outcome scale, ACoA : anterior communicating artery, MCA : middle cerebral artery, PCoA : posterior communicating artery, ACA : anterior cerebral artery, ICA Bifur. : Internal cerebral artery bifurcation, Lt. : left, Rt. : right, aSDH : acute subdural hematoma

in 17 (73.9%).

In all cases the ruptured aneurysms were detected by initial 3D CT angiography. All ruptured aneurysms were located in the anterior portion of the circle of Willis. Locations of the ruptured aneurysms were : 11 (47.8%) in the middle cerebral artery (MCA; Fig. 1), 5 (21.7%) in the ACoA, 4 (17.4%) in the posterior communicating artery (PCoA). All patients had SAH on initial CT. Sixteen patients (69.6 %) also had ICH, 13 (56.5%) presented with IVH, 19 (82.6%) demonstrated midline shifting (≥ 5 mm), and 12 (52.2%) showed with subdural hematoma greater than 1 cm. Among 16 patients showed ICH, 12 cases of ICH were adjacent with subdural hematoma. Fisher grade IV hemorrhages were present in 20 patients (87.0%) and grade III hemorrhages were 3 patients (13.0%).

Surgical decompression and clipping or endovascular intervention following clinical and radiologic evaluation were immediately performed on the day of admission except in one patient (Case 9). Eleven patients (47.8%) underwent craniotomy, evacuation of a subdural hematoma, and aneurysmal neck clipping in one stage. Ten patients (43.5%) underwent decompressive hemi-craniectomy, evacuation of subdural hematomas, and aneurysmal neck clipping in one stage. Some cases required a second stage operation. WFNS grade V ($p=0.004$), midline shifting ($p=0.028$) correlated with selecting decompressive hemi-craniectomy. Two patients (Cases 10 and 21) underwent decompressive hemi-craniectomy 3 days (Case 10) and 5 days (Case 21) after the first operation due to increased intracranial pressure (IICP) caused by cerebral edema. One patient (Case 9) underwent decompressive hemi-craniectomy followed by a second stage endovascular coiling of an aneurysm. The remaining patient (Case 12) underwent endovascular coiling only.

The outcomes of these patients were good recovery in 5 patients (21.7%), moderate disability in 1 (4.3%), severe disability in 4 (17.4%), vegetative in 2 (8.7%), and death in 11 (47.8%). Presence of ICH ($p=0.025$), and IVH ($p=0.022$) correlated with the outcomes of vegetative or death. Cases 6 and 23 had favorable GCS scores and WFNS grades initially; however, the clinical

courses of these patients were complicated by vasospasms, resulting in Case 6 being classified as vegetative and Case 23 as having moderate disability.

DISCUSSION

The incidence of aSDH caused by rupture of an intracranial aneurysm is reported as varying from 1.3 to 7.9%^{2,5,10,11,14,15,17,20,21}. In our experience the incidence was 4.2% during the six years of the review period. Several mechanisms to explain the occurrence of aSDH after aneurysmal rupture have been proposed : 1) successive small bleeding from the ruptured aneurysm allowing aneurysmal adhesion to the arachnoid membrane with the final rupture occurring into the subdural space; 2) the arachnoid membrane being directly torn by high systolic pressures from the bleeding aneurysm; 3) the arachnoid membrane being ruptured by rapid accumulation of blood under pressure from the leaking aneurysm; 4) erosion of the cavernous sinus wall by acute enlargement of the intracavernous aneurysm following thrombosis causes a hematoma of the subdural space; and 5) a ruptured distal ACA aneurysm adheres directly to either the dura or falx in the adjacent subdural space causing a subdural hematoma^{2,3,6,9,12,16,22}. In our cases, twelve cases showed large volume of ICH were adjacent with subdural hematoma. This finding suggests that the majority of subdural hematomas in the current series occurred by ruptured subarachnoid membrane due to high-pressure of ICH.

On review of the literature, the locations of ruptured aneurysms in cases of SAH with aSDH were ICA in 43% of cases, MCA in 22%, ACoA in 22%, and other locations in 13%. The location of the ruptured aneurysm in the posterior portion of the circle of Willis is quite rare^{8,19,20}. In the current series, it is remarkable that the majority of the aneurysms have occurred in the MCA. The proportion of ACoA aneurysms was similar to prior reports. Aneurysms in posterior portion of the circle of Willis were not observed.

In a prior case report by Meyer, the initial condition on ad-

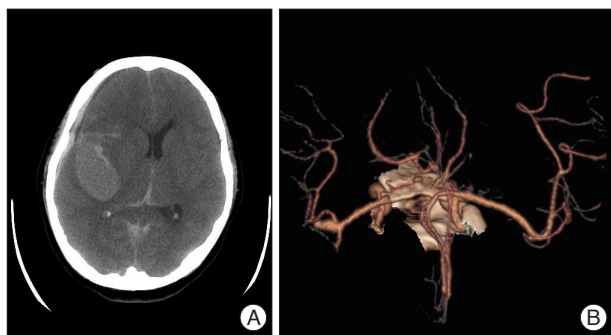


Fig. 1. Case 18. A : Computed tomography (CT) on admission demonstrates an acute subdural hematoma in the right convexity, intracerebral hematoma in the right hemisphere, and right-to-left midline shifting. B : 3D-CT angiography demonstrates aneurysm of the right middle cerebral artery bifurcation.

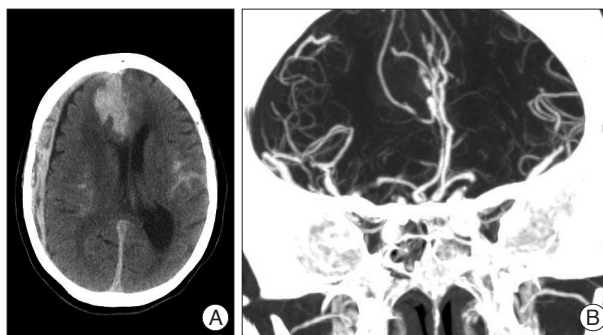


Fig. 2. Case 15. A : Computed tomography (CT) on admission demonstrates an acute subdural hematoma from the right interhemispheric fissure to the right convexity, an intracerebral hematoma in the right frontal lobe, and right-to-left midline shifting. B : 2D selecting image of 3D-CT angiography demonstrates an aneurysm of the right distal anterior cerebral artery.

mission was extremely poor (preoperative Hunt and Hess grade IV to V=96.6%)¹⁵. Gelabert-Gonzalez reported four cases, of which three were WFNS grade V and one was grade IV. WFNS grade V accounted for 17 cases (73.9%) in the current series. Secondary to the poor clinical status of these types of patients, some authors have questioned the need to perform angiography immediately; these authors recommend angiography if the clinical condition of patients is stable and does not require immediate surgical intervention²¹. Some authors have suggested that emergency craniotomy and evacuation of the hematoma should precede angiography to allow for rapid cerebral decompression; later, if the patient is stable, angiography should be performed for definitive aneurysm clipping^{3,12,20,24}. In more recent reports, 3D-CT angiography has been recommended for detecting the presence of an aneurysm as it has the advantages of rapid results, good quality, noninvasiveness compared to DSA, and is easier to perform on patients in poor neurological condition^{14,23}. At our hospital, all patients suspected of suffering a stroke routinely undergo 3D-CT angiography. All ruptured aneurysms in the current series were detected initially by 3D-CT angiography. Additional studies for determining the existence of an aneurysm were unnecessary; all patients underwent surgery or other intervention after 3D-CT angiography without time wasting for additional study.

Various plans for management of an aSDH caused by a ruptured intracranial aneurysm have been put forth in previous reports. In cases with relatively good clinical status, elective clipping of the aneurysm along with evacuation of the hematoma could be performed; however, in the face of rapid clinical deterioration, an emergency craniotomy for subdural evacuation should precede obliteration of the aneurysm or further investigative studies^{20,24}. Patients with midline shifting is a sign of IICP and possibly impending transtentorial herniation. Therefore, early craniotomy should be executed to correct the IICP. In the current series, Fisher grade IV hemorrhages accounted for 20 patients (87.0%); nineteen of these patients demonstrated midline shifting on initial CT imaging. Among the three patients with a Fisher grade III hemorrhage, one patient (Case 9) demonstrated a thick SAH with significant midline shifting of greater than 5 mm. Twenty-two of 23 patients underwent early craniotomy or decompressive hemi-craniectomy. The aneurysms were approached and clipped after removal of the hematoma and ventricular drainage to diminish brain swelling in 21/23 cases. In one patient (Case 9), approaching the aneurysm was impossible although decompressive craniectomy with evacuation of the hematoma were performed. This patient required a second stage intervention. The remaining patient (Case 12) presented with advanced age, good GCS scores, and an absence of midline shifting. Accordingly, this patient underwent endovascular coiling only. Unfortunately, after coiling, severe rebleeding occurred, resulting in death. Among the various treatment strategies that have been suggested, and considering the complications of rebleeding and rapid aggravation of cerebral edema, we

recommend that early decompression and obliteration of the aneurysm undertaken in a single stage, if possible.

Although some authors have reported good outcomes, aSDH due to aneurysmal rupture carries a poor prognosis in the vast majority of cases. In previous reports, if patients had good initial clinical and radiological status, outcomes were fair. However, if patients presented with severe neurological deterioration, findings of IICP (such as midline shifting), and a large hematoma volume, outcomes were poor^{5,8,11,15,20,24}. In the current series, some patients (Cases 1, 2, 4, 7) had good outcome although they presented WFNS grade 4 or 5 on admission. However, poor clinical grade and CT findings were generally associated with unfavorable outcomes. The presence of ICH and IVH were correlated with outcomes but thickness of subdural hematomas did not show statistical significance. Many complications of SAH, such as rebleeding, vasospasm, and hydrocephalus, can affect the clinical course and outcome of patients. In our series, two patients had significant morbidity despite with relatively good clinical grades on admission. Case 6 was vegetative caused by vasospasm. One patient (case 12) died from rebleeding secondary to incomplete packing of coils. Based on these findings, we suggest that outcomes following aSDH caused by ruptured intracranial aneurysms are affected by clinical and neurophysiological status based not only on IICP signs due to aSDH but also the clinical course of SAH itself.

CONCLUSION

Spontaneous aSDH caused by ruptured intracranial aneurysms is rare, but is associated with poor clinical prognosis. We recommend CT angiography for early detection of such aneurysms. Early decompression with obliteration of the aneurysm is our preferred treatment plan. Favorable outcomes are associated with good clinical grades and of the absence of increased intracranial pressure on admission; however, clinical prognosis remains poor even in the presence of these favorable prognostic signs.

References

1. Anderson GB, Steinke DE, Petruk KC, Ashforth R, Findlay JM : Computed tomographic angiography versus digital subtraction angiography for the diagnosis and early treatment of ruptured intracranial aneurysms. *Neurosurgery* 45 : 1315-1320; discussion 1320-1312, 1999
2. Barton E, Tudor J : Subdural haematoma in association with intracranial aneurysm. *Neuroradiology* 23 : 157-160, 1982
3. Clarke E, Walton JN : Subdural haematoma complicating intracranial aneurysm and angioma. *Brain* 76 : 378-404, 1953
4. Dehdashti AR, Rufenacht DA, Delavelle J, Reverdin A, de Tribolet N : Therapeutic decision and management of aneurysmal subarachnoid haemorrhage based on computed tomographic angiography. *Br J Neurosurg* 17 : 46-53, 2003
5. Gelabert-Gonzalez M, Iglesias-Pais M, Fernández-Villa J : Acute subdural haematoma due to ruptured intracranial aneurysms. *Neurosurg Rev* 27 : 259-262, 2004
6. Hatayama T, Shima T, Okada Y, Nishida M, Yamane K, Okita S, et al. : [Ruptured distal anterior cerebral artery aneurysms presenting with

- acute subdural hematoma : report of two cases]. *No Shinkei Geka* 22 : 577-582, 1994
7. Hop JW, Rinkel GJ, Algra A, van Gijn J : Case-fatality rates and functional outcome after subarachnoid hemorrhage : a systematic review. *Stroke* 28 : 660-664, 1997
 8. Inamasu J, Saito R, Nakamura Y, Ichikizaki K, Suga S, Kawase T, et al. : Acute subdural hematoma caused by ruptured cerebral aneurysms : diagnostic and therapeutic pitfalls. *Resuscitation* 52 : 71-76, 2002
 9. Ishibashi A, Yokokura Y, Sakamoto M : Acute subdural hematoma without subarachnoid hemorrhage due to ruptured intracranial aneurysm--case report. *Neurol Med Chir (Tokyo)* 37 : 533-537, 1997
 10. Ishikawa E, Sugimoto K, Yanaka K, Ayuzawa S, Iguchi M, Moritake T, et al. : Interhemispheric subdural hematoma caused by a ruptured internal carotid artery aneurysm : case report. *Surg Neurol* 54 : 82-86, 2000
 11. Kocak A, Ates O, Durak A, Alkan A, Cayli S, Sarac K : Acute subdural hematomas caused by ruptured aneurysms : experience from a single turkish center. *Turk Neurosurg* 19 : 333-337, 2009
 12. Kondziolka D, Bernstein M, ter Brugge K, Schutz H : Acute subdural hematoma from ruptured posterior communicating artery aneurysm. *Neurosurgery* 22 : 151-154, 1988
 13. Marshall LF, Marshall SB, Klauber MR, Van Berkum Clark M, Eisenberg H, Jane JA, et al. : The diagnosis of head injury requires a classification based on computed axial tomography. *J Neurotrauma* 9 Suppl 1 : S287-S292, 1992
 14. McLaughlin MR, Jho HD, Kwon Y : Acute subdural hematoma caused by a ruptured giant intracavernous aneurysm : case report. *Neurosurgery* 38 : 388-392, 1996
 15. Meyer F, Sandvoss G : Acute subdural hematoma associated with non-traumatic aneurysm rupture. *Zentralbl Neurochir* 58 : 66-70, 1997
 16. Missori P, Fenga L, Maraglino C, Rocchi G, Nardacci B, Calderaro G, et al. : Spontaneous acute subdural hematomas. A clinical comparison with traumatic acute subdural hematomas. *Acta Neurochir (Wien)* 142 : 697-701, 2000
 17. Nizzoli V, Brambilla P, Tonnarelli GP : Acute subdural hematoma. Spontaneous forms of arterial origin. *Eur Neurol* 20 : 4-8, 1981
 18. Nonaka Y, Kusumoto M, Mori K, Maeda M : Pure acute subdural haematoma without subarachnoid haemorrhage caused by rupture of internal carotid artery aneurysm. *Acta Neurochir (Wien)* 142 : 941-944, 2000
 19. Nowak G, Schwachenwald S, Kehler U, Müller H, Arnold H : Acute subdural haematoma from ruptured intracranial aneurysms. *Acta Neurochir (Wien)* 136 : 163-167, 1995
 20. O'Sullivan MG, Whyman M, Steers JW, Whittle IR, Miller JD : Acute subdural haematoma secondary to ruptured intracranial aneurysm : diagnosis and management. *Br J Neurosurg* 8 : 439-445, 1994
 21. Rengachary SS, Szymanski DC : Subdural hematomas of arterial origin. *Neurosurgery* 8 : 166-172, 1981
 22. Triantafyllopoulou A, Beaumont A, Ulatowski J, Tamargo RJ, Varelas PN : Acute subdural hematoma caused by an unruptured, thrombosed giant intracavernous aneurysm. *Neurocrit Care* 5 : 39-42, 2006
 23. Villablanca JP, Hooshi P, Martin N, Jahan R, Duckwiler G, Lim S, et al. : Three-dimensional helical computerized tomography angiography in the diagnosis, characterization, and management of middle cerebral artery aneurysms : comparison with conventional angiography and intraoperative findings. *J Neurosurg* 97 : 1322-1332, 2002
 24. Weir B, Myles T, Kahn M, Maroun F, Malloy D, Benoit B, et al. : Management of acute subdural hematomas from aneurysmal rupture. *Can J Neurol Sci* 11 : 371-376, 1984
 25. Yuh EL, Gean AD, Manley GT, Callen AL, Wintermark M : Computer-aided assessment of head computed tomography (CT) studies in patients with suspected traumatic brain injury. *J Neurotrauma* 25 : 1163-1172, 2008