DEGREE OF MIDLINE SHIFT FROM COMPUTED TOMOGRAPHY (CT SCAN) CAN PREDICT CLINICAL OUTCOME IN PATIENTS WITH HEAD IN JURIES

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DEGREE OF MIDLINE SHIFT FROM COMPUTED TOMOGRAPHY (CT SCAN) CAN PREDICT CLINICAL OUTCOME IN PATIENTS WITH HEAD INJURIES

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ABSTRACT

Midline shift after traumatic brain injury (TBI) is widely recognized as an important marker of severe injury. Previous reports have described the association of a large amount of midline shift on computed tomography (CT) scans with poor outcomes or other adverse sequelae of TBI. The purpose of this investigation was to predict outcomes of patients with TBI by examming degree of midline shift in relation to various Glasgow Coma scale score (GCS scores) and to determine the correlation between mortality rates and midline shift by CT scan.

Two hundred and seventeen cases of TBI admitted to the regional trauma center in the Siriraj Hospital from January 1999 to 2004 were analyzed. The cases, which covered a comprehensive rang of degrees of midline shift, were divided into three categories; no shift, shift up to 10mm, shift greater than 10mm. The effect of lesions as showed by CT scans was determined by separating patients into those with and without midline shift and then re-examining the relationship of degree of midline shift and GCS scores to Clinical outcomes by both univariate and multivariate analysis.

It was found that the mean shift in a group of 50 patients with midline shift who died was 7.05mm, whereas the mean shift in those with good outcomes (167 cases) was 3.25mm. The degree of midline shift in TBI was significantly associated with the outcomes by univariate analysis ($P = .001$), but not significantly associated with the outcome by multivariate analysis ($P = .723$). In patient with lower GCS scores, CT scan findings were significantly related to outcomes both in univariate and multivariate analysis (*OR 10.42, 95% CI 2.24-48.43*, $P =< .001$).

The degree of midline shift in CT findings was significant in predicting clinical outcomes. Mortality increased 4 times in patients with head injury with midline shift greater than 10mm, but was not significant in predicting outcomes when combined with GCS scores.

KEY WORDS: MIDLINE SHIFT / HEAD INJURY / CT SCAN / GCS SCORE

67pp.

ระดับของ Midline shift จากภาพถ่ายเอ็กซเรย์คอมพิวเตอร์สมองสามารถทำนายผลการรักษาของผู้ป่วยอุบัติเหตุที่ศีรษะ (DEGREE OF MIDLINE SHIFT FROM COMPUTED TOMOGRAPHY (CT SCAN) CAN PREDICT CLINICAL OUTCOME IN PATIENTS WITH HEAD INJURIES)

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บทคัดย่อ

Midline shift (MS) หลังจากได้รับอุบัติเหตุที่ศีรษะ ถือเป็นตัวบ่งชี้ที่สำคัญในการบอกถึงระดับความรุนแรงของการบาดเจ็บ จากรายงานที่ผ่านมาได้อธิบายถึง MS ขนาดใหญ่จากภาพเอ็กซเรย์คอมพิวเตอร์สมอง กับผลการรักษาของผู้ป่วยที่ไม่ดี ซึ่งจุดประสงค์ของการศึกษาวิจัยครั้งนี้ก็เพื่อทำนายผลการรักษาของผู้ป่วยที่ได้รับอุบัติเหตุทางศีรษะ โดยการหาความสัมพันธ์ระหว่างระดับของ MS ที่ได้จากภาพเอ็กซเรย์คอมพิวเตอร์สมองกับความรู้สึกตัวเบื้องต้น (GCS Score) และทำนายอัตราการตายที่เพิ่มขึ้นเมื่อเทียบกับผู้ป่วยที่ไม่มี MS

ได้ศึกษาวิเคราะห์จากผู้ป่วยทั้งหมด 217 รายที่มาด้วยอุบัติเหตุที่ศีรษะและเข้ารับการรักษาที่ศูนย์อุบัติเหตุโรงพยาบาลศิริราช ช่วงปี 2542-2547 โดยแบ่งระดับของ MS ออกเป็น 3 กลุ่ม คือ 1.ไม่มี Midline shift 2. Midline shift ไม่เกิน 10mm, 3. Midline shift มากกว่า 10mm. และศึกษาวิเคราะห์ความสัมพันธ์ระหว่างระดับของ MS กับ GCS score ของผู้ป่วย พร้อมกับวิเคราะห์ความสัมพันธ์ระหว่างระดับของ MS กับอัตราการตายของผู้ป่วยที่มาด้วยอุบัติเหตุที่ศีรษะ

ซึ่งการศึกษาวิจัยครั้งนี้พบว่าผู้ป่วยกลุ่มที่เสียชีวิต ทั้งหมด 50 ราย เฉลี่ย MS เท่ากับ 7.05mm. ในขณะที่ผู้ป่วยที่ผลการรักษาดีทั้งหมด 167 ราย เฉลี่ย MS เท่ากับ 3.25mm. และพบว่าระดับของ MS ไม่มีความสัมพันธ์กับ GCS score แต่มีความสัมพันธ์กับผลการรักษาอย่างมีนัยสำคัญทางสถิติเมื่อทำการวิเคราะห์แบบ univariate analysis และมีอัตราการตายเพิ่มขึ้น 4 เท่าเมื่อระดับของ MS มากกว่า 10mm. แต่ไม่มีความสัมพันธ์กับผลการรักษาเมื่อวิเคราะห์แบบ multivariate analysis อย่างมีนัยสำคัญ

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LIST OF ABBREVIATIONS

Abbreviation	**Term**
TBI	Traumatic Brain Injury
GCS	Glasgow Coma Scale
CT scans	Computed Tomography scans
SDH	Subdural heamatoma
EDH	Epidural heamatoma
ICH	Intracerebral heamatoma
DAI	Diffused Axonal injury
SDH with MS	Subdural heamatoma with Midline Shift
EDH with MS	Epidural heamatoma with Midline Shift
SHI	Severe Head Injury

CHAPTER I
INTRODUCTION

Head injury is a significant medical, social, and economic problem all over the world. Road accidents are the most frequent cause of head injury, subsequent is hit, fall and sport accidents. Head injury in Thailand occurs in highest risk groups such as in male and young age group. In 2002 Royal Thai Police reported (1) statistical of traffic accident compare between in 2001 and 2002 total accident in 2001 were 125,432, in 2002 were 149,600 increase 24,168 were 19.37 %, and total damaged resources in 2001 were 1,240,801,187 baths, in 2002 were 1,494,936,815 increase 254,135,628 baths. The mortality rate in 2001 were 11,652, in 2002 were 13,116 this was injury increase 1,464 equal to 11.16 %. The injured patients in 2001 were 53,960 and 2002 were 69,313, which were injury increase 15,353 equal to 22.15%, almost 77% were head injury, and therefore, if we can early detection or prognosis of the patients with head injury, this will be lead to early evaluation and treatment.

Injury to the head may damage the scalp, skull or brain. The most important consequence of head trauma is traumatic brain injury. Head injury may occur either as a closed head injury, such as the head hitting a car’s windshield, or as a penetrating head injury, as when a bullet pierces the skull. Both may cause damage that rang from mild to profound. Very severe injury can be fatal because of profound brain damage. Brain injury is most likely to occur in males between ages 15 and 24, usually as a result of car and motorcycle accidents. About 70% of all accidental deaths are due to head injuries, as are most of the disabilities that occur after trauma.

The outcome of traumatic brain injury (TBI) is related to the initial level of injury. While the initial GCS score provides a description of the initial neurologic condition, it does not correlate tightly with outcome. Various methods have been used in an attempt to predict the outcome of TBI. However, one simplified model uses 3 factors; age, motor score of the GCS, and papillary responsive to provide a probability of outcome. The Traumatic Coma Data Bank (23) analyzed patients with head injuries and identified 5 factors that correlated with a poor outcome, as follows: (1) age older than 60 years, (2) initial GCS score of less than 5, (3) presence of a fixed dilated pupil, (4) prolonged hypotension or hypoxia early after injury, and (5) presence of a surgical intracranial mass lesion.

The relationship between the type of injury, as determined by CT scan, and patients outcome has been described previously (22, 30, 43,). Mass lesion (Epidural Heamatoma EDH, Subdural Heamatoma SDH, Intracranial Heamatoma ICH) with midline shift is a computed tomography (CT scan) finding often seen in the acute stage of head trauma and found to correlate with poor outcome. Which it seen logical to assume that mass lesion with midline shift has a worse outcome than without midline shift, this has not been systematically studied. There is no simple model to predict poor outcome or mortality rate in traumatic brain injury.

Degree of midline shift after traumatic brain injury is widely recognized as an important marker of severe injury. Numerous reports describe the association of a large amount of midline shift in patients Acute Subdural Heamatoma or Epidural Heamatoma on CT scan with poor outcome or other adverse sequelae of traumatic brain injury (2, 11, 12, 29). For example, the presence of intracranial hemorrhage and midline shift on the admission CT scan has been found to correlate with poor outcome following severe head injury. In contrast, the association between the quantitative degree of midline shift and patients poor outcome or mortality rate has not been as well defined. A study by Jeffrey Englander, MD, et al. (3) in 2003 concluded that the presence of either a midline shift greater than 5mm or a subcortical contusion on acute CT scan is associated with greater needs of assistance with ambulation, Activities of daily living, and global supervision at rehabilitation discharge. Although other variables such as Glasgow Coma Scale score, age, abnormal motor responses, CT lesion finding, pupillary abnormalities, and episodes of hypoxia and hypertension, have been subsequently introduced to build more complex and accurate prognostic model. (4-5), have not defined the relationship between degree of midline shift and mortality rate. In contrast to a study by Narayan (43), the extent of midline shift did not add significantly to the prognostic capability of their model. Selladurai (32) and Jinn- Rung (29) also noticed that the degree of midline shift did not prove to be of a predictive significance, because patients poor outcome from influenced by the location of intracerebral lesion and the presence of bilateral abnormalities.

Although several studies have examined the relationship between the quantitative extent of midline shift and both low admission GCS score and high intracranial pressure, but the extent of midline shift was not compared with patient outcome in these studies. (2, 40, 43)

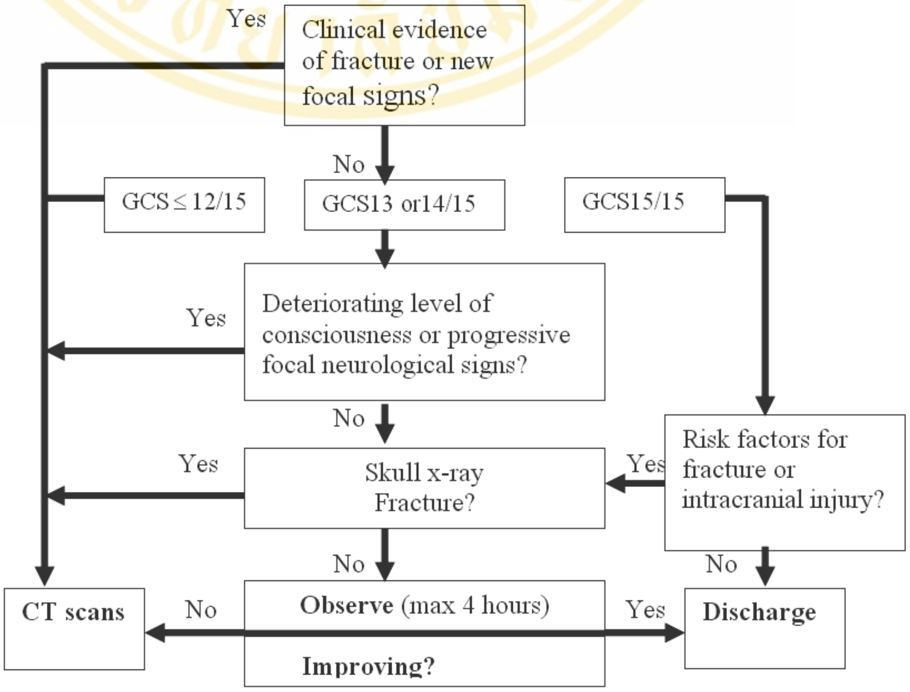
So it is also know that the lower GCS score (less than 5) and large degree of midline shift (> 5mm) were shown to have a value in predicting poor outcome in patients severe brain injury, have not defined the interaction between the quantitative extent of midline shift with GCS score and patient outcome or mortality rate.

Our study had three objectives; were to determine the usefulness of midline shift only, midline shift combine with Glasgow Coma Scale score (GCS) and midline shift combine with CT lesion finding to predict poor outcome or death rate in patients head injury following both univariate and multivariate analysis. Midline shift was considered when the shift of the septum pellucidum from the midline.

In this study, the relationships between clinical data, GCS score and lesion with midline shift in CT scan were evaluated. We attempt to find predictor for outcome evaluation.

CHAPTER II
LITERATURE REVIEW

Computed tomography has become the first step in evaluating patients with acute head injury. The urge to start with plain x-rays (seen especially with the emergency medicine physician) particularly in cases of "minor" head trauma should be resisted. Negative plain x-rays may lead to a false of security. Indeed, any patient with head trauma in whom x-rays are considered for "medical-legal reasons," warrants a CT study. In the past, multidisciplinary groups developed indications for obtaining plain x-rays following head trauma, which have subsequently been transposed to the CT examination. These indications included focal neurologic findings, altered level of consciousness after trauma, palpable depressed skull fracture, clinical signs of basilar fracture ("raccoon "eyes, blood behind the ear drums), evidence of cerebrospinal fluid (CSF) leak from the nose or ear, and penetrating head injury. From Scottish Intercollegiate Guidelines Network (SIGN 2000) recommend to indications for CT scan in patients head trauma:



Admission GCS score

From Scottish Intercollegiate Guidelines Network (SIGN 2000) recommend to use clinical assessment Glasgow Coma Scale score (GCS) to determine how to manage the patients head injury (6):

Severity	**SCORE**
Mild	15
Moderate	13-14
Severe	3-12

Table1. The GCS score is calculated by adding up the sum of the 3 scale components, with a resulting score ranging from 3 (worst) to 15 (best)

Component	**Score**
Eye opening	
Spontaneous	4
To speech	3
To pain	2
None	1
Verbal response	
Oriented	5
Confused conversation	4
Inappropriate words	3
Incomprehensible sounds	2
None	1
Motor response	
Obeys commands	6
Localized pain	5
Normal flexion (withdrawal)	4
Abnormal flexion (decorticate)	3
Extension (decelerate)	2
None	1

In 2004 Bk Gan et al. (7) found that the GCS of ≤ 8 on admission was significant in predicting mortality in the elderly.

In 2001 Jun-ichi Ono, Akira Yamaura et al. concluded that outcome was best assessed by a combination of GCS score, age and multiplicity of CT lesion in the diffuse brain injury group (8).

Shameran Slewa-Younan concluded that men's levels of injury severity were greater than women's despite the same admission criteria being applied to both sexes, which their resulted found that men had significantly greater levels of injury severity as indicated by the Glasgow Coma Scale score and length of posttraumatic amnesia when compared with women (9).

Mechanism of injury

In 1998 J. Tobias Nagurney et al. noted that Falls are second only to Motor vehicle accidents among unintentional traumatic cause of death in all ages especially Fall in the elderly to closed head trauma represent a significant cause of morbidity and mortality (10).

Typology of Traumatic Brain Damage

Skull Fracture

The presence of skull fracture indicates that impact to the head has occurred with force. Interestingly, some patient with a skull fracture may have no evidence of brain damage and make an uneventful recovery, and the presence of skull fracture is a poor predictor for an associated intra cranial injury, likewise the absence of a fracture does not exclude such an abnormality (11). However, patients suffering a skull fracture due to head trauma have a much higher incidence of intra cranial hematoma than those who do not sustain a fracture (12). The type of fracture found following trauma is dictated in port by the shape of the object that makes contact with the head. Flat-shaped objects tend to produce fissure fracture, which can extend into the base of the skull, while angled or pointed objects produce a localized or stellate fracture (13). Fracture at the base of the skull may give rise to infection. Although, all injuries of this nature are at increased risk for infection but these fracture often pass though the petous bone or the anterior cranial fossa (cribiform plate) and cause leakage of cerebrospinal fluid (CSF) though the nose, mouth, or ear.

An important to separate skull fracture into the following categories: Linear fracture, Compound fractures and depressed fractures.

Linear fractures are the most common type of skull fracture, which it heal rapidly in children but, quite slowly in adults, sometimes being evident on examinations done many years after an episode of trauma.

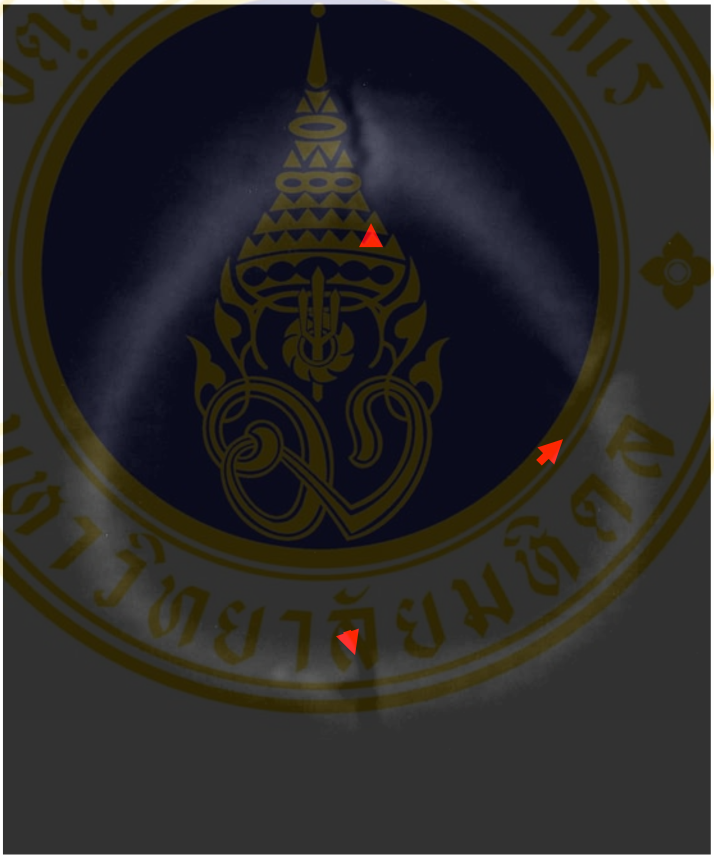


Fig.2- 1 *Linear fractures in children: Skull radiograph (lateral projection)- Linear fracture is demonstrated in temporo-parietal bone (arrow). In pediatric populations, There are still persist of cranial sutures as sagittal suture in anterior and posterior aspect of cranial vault (arrowhead)*

Compound fractures may occur as the result of either injury to the overlying scalp or extension of a fracture into a sinus cavity. The latter variety may be more difficult to detect radio graphically and often remains occult. Which, the majority of cerebrospinal fluid leaks are the results of this fracture.

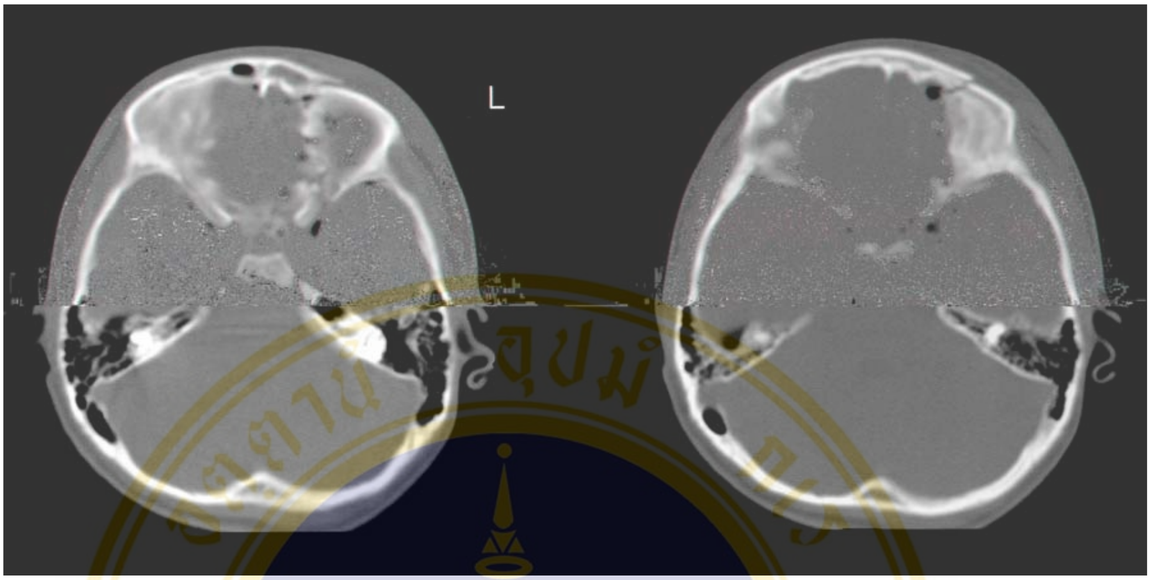


Fig. 2-2 *Compound fracture of frontal bone is demonstrated with line of fracture involve left frontal passing through frontal sinus (arrows). Multiple small bubble of air (hypodensity spots) is noted in base of skull.*

Almost all depressed fractures are associated with some comminuting and therefore in general are the result of rather severe trauma. Assessment of the need for surgical correction of a depressed fracture is best made on the basis of the clinical findings and with CT scan examination.

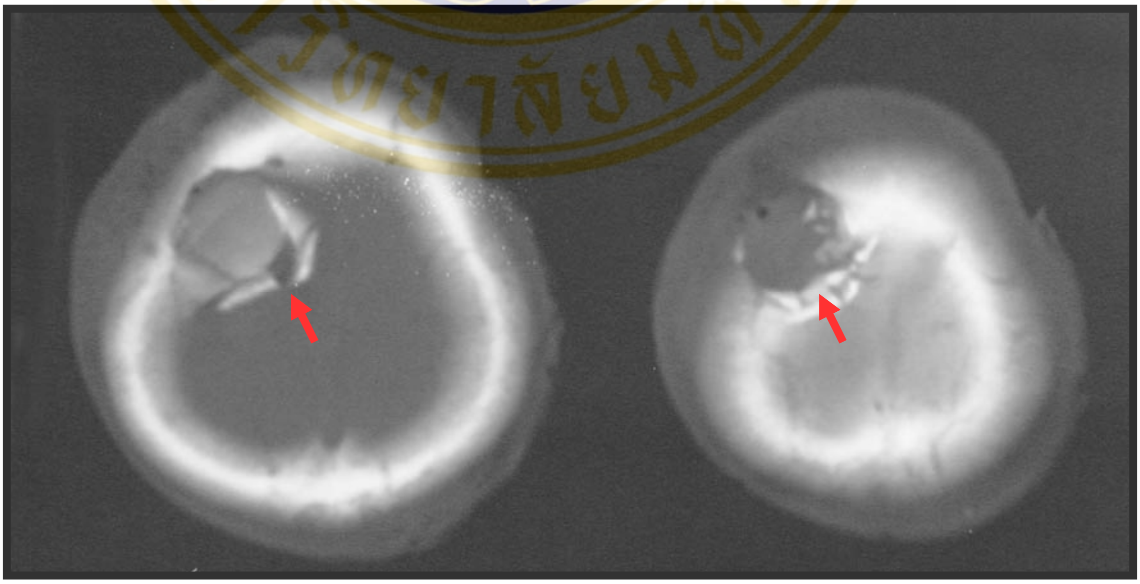


Fig. 2-3 *Depressed skull fracture: communited fracture of right frontal bone with compression to underlying tissue.*

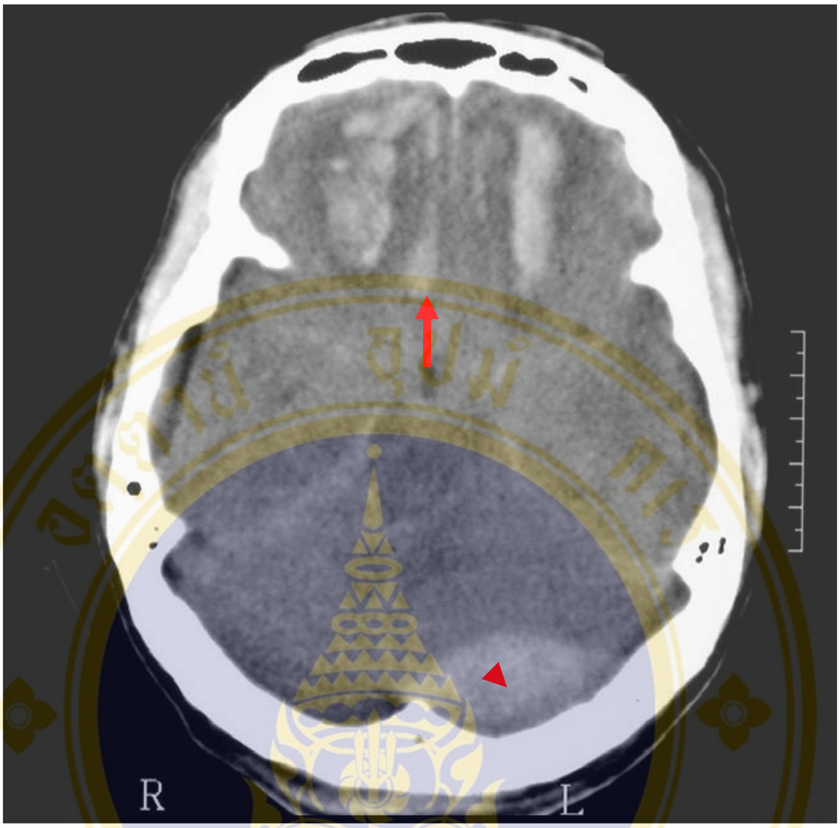


Fig. 2-4 *Cranial CT scans of head injury patient showed areas of hemorrhage in bifrontal lobes caused by gliding of brain tissue over cranial bone (arrow). Another hyperdensity of lentiform hematoma as epidural hematoma is demonstrated in left occipital region (arrow head)*

Focal Brain Damage

Contusion and Laceration

A contusion is a type of focal brain damage caused mainly by contact between the surface of the brain and the bony protuberance of the skull or by rapid acceleration-deceleration (14). The most frequent sites of this type of injury are the inferior portions of the frontal and temporal lobes, areas where irregular bony surfaces are adjacent to brain. Contusions are not usually found in the parietal and occipital lobes unless there is a skull fracture in their areas (13). (Fig 2-4)

Contusions are divided into coup lesion, those occurring immediately beneath the point of impact, and countercoup lesions, those occurring in the portion of the brain that is opposite the point of impact in the direction of the applied force.

By definition, the pia-arachnoids membranes are intact over surface contusions, but they are torn following lacerations. Considered to be the hallmark of brain damage due to head injury, they have a very characteristic distribution generally affecting the frontal poles, the orbital gyri, and the cortex above and below the Sylvain fissure, the inferior surfaces of the cerebella hemispheres are affected (15).

The initial appearance of contusions evolves over time. Shortly injury, a contusion is visible as microscopic regions of per vascular hemorrhage that follows the tracts of small vessels in the cortex, and it usually runs perpendicular to the cortical surface. Over time, blood products seep into the adjacent cortex and neurons structures in the immediate vicinity begin to degenerate. The destruction of neurons subsequently produces a glial scar (16). In some case, the hemorrhage will extend into the white matter, causing demyelination of axons and loss of neuronal tracts and the contusion develops into a shrunken glial scar, which is apparent to the naked eye at autopsy (17).

Old contusions can develop a pyramidal shape with the apex of the pyramid at the depths of the cortex and the base of the pyramid at the crest of a gyrus (13). Neuropathological studies contra intuitively demonstrate that, in patients who receive frontal or occipital brain injury, contusions are almost always more severe in the frontal lobes regardless of the point of injury. The use of contusion indexes has shed doubt on the concept of severity from countercoup contusions.

Hemorrhage and Hematoma

The most common cause of the clinical deterioration is hemorrhage. Hemorrhages following head injury generally occur in their areas of the brain;

1. Within the extradural, subdural, or sub arachnoids spaces
2. Intra parenchyma
3. Into the ventricles.

Extradural, Subdural, and intra cerebral hematomas cause expanding intra cranial lesions. These in turn produce a mass effect promoting increased intra cranial pressure, and these compress the surface of the brain. Sub arachnoids hemorrhage is often associated with the formation of contusions. Intra ventricular hemorrhage is often seen in patients with diffuse axonal injury.

Extradural (Epidural) Hematoma

About 85% of epidural hematoma patients will also demonstrate a concurrent skull fracture. The most common anatomical site for epidural hematomas is the temporal region. Less commonly these occur in other regions. When they with a direct impact, they will often deform inward, develop a fracture line, and transect or rupture an artery, or occasionally a vein, lying on the inner table of the skull. In cause where an artery is ruptured, the arterial pressure quickly forces blood into the potential space between the skull and the dura, producing an enlarging mass. It is this potential for quick enlargement that may produce a life-threatening situation due to pressure transfer throughout the brain, often resulting in downward herniation of the inferior brain and thus compromising brain stem structures. As the hematoma enlarges, it will gradually strip the dura from the skull and form an ovoid mass that progressively indents and flattens the adjacent brain.

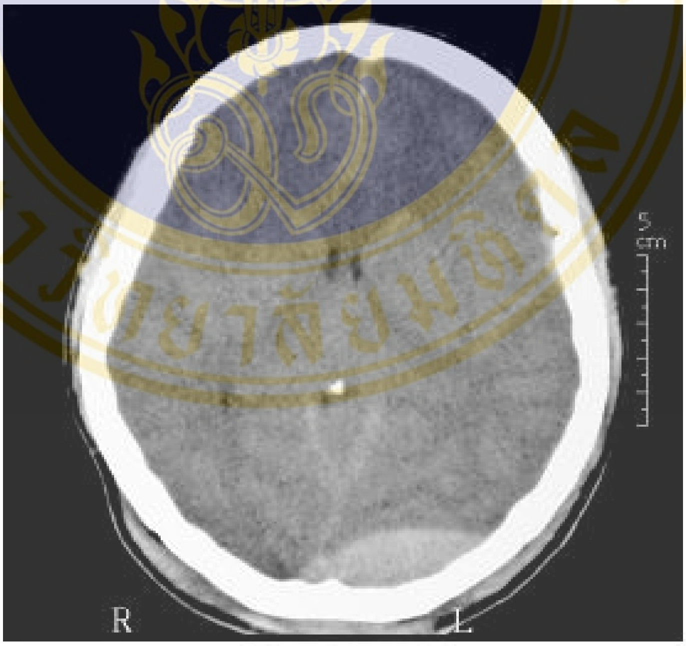


Fig. 2-5 *Epidural hematoma in Cranial CT scan is characterized by biconvex shape of*
hyperdensity and can across midline (arrows)

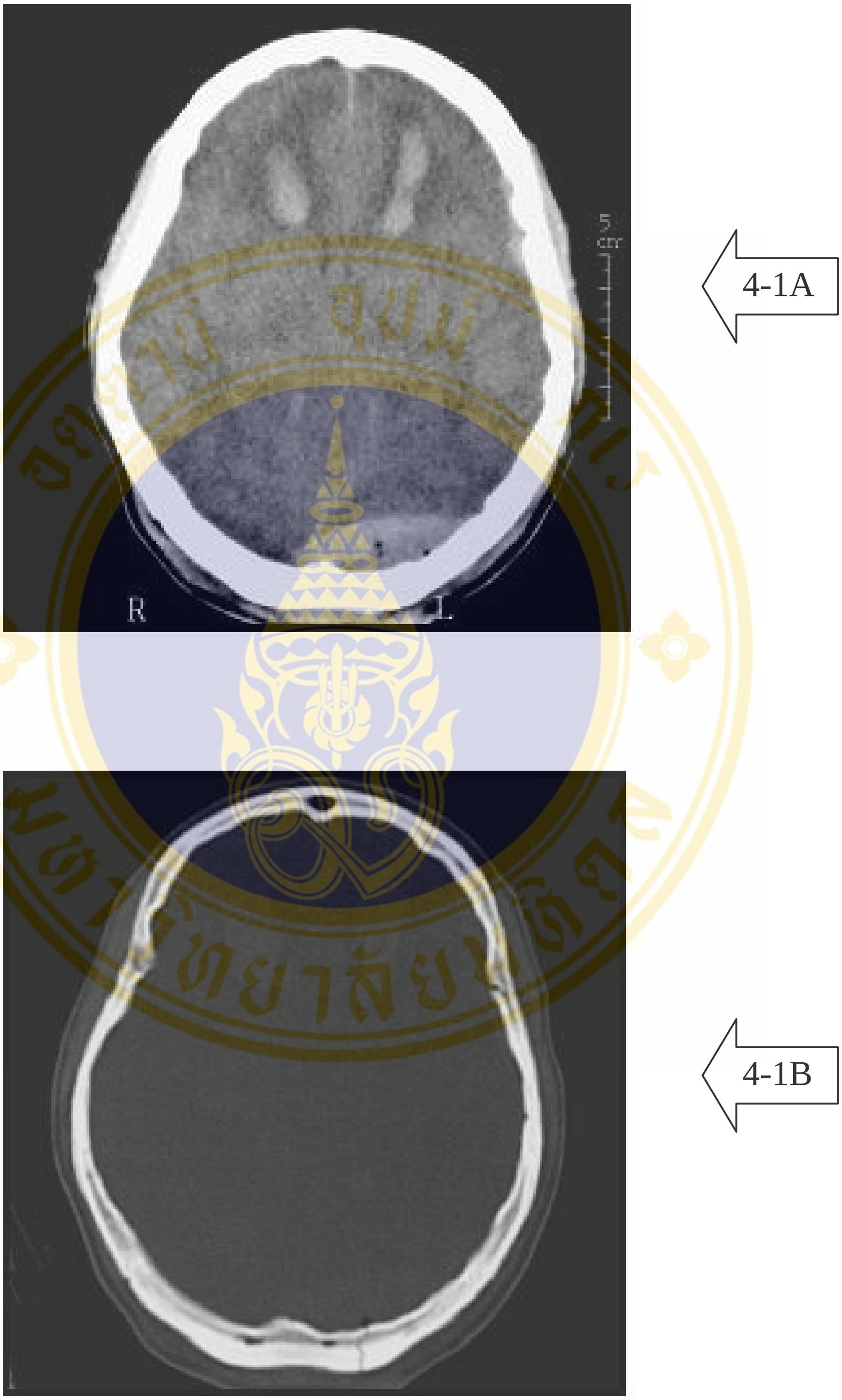


Fig. 2-6, Cranial CT scan in soft tissue window (Fig.4-1A) and bony window (Fig.4-1B) *shows air bubble entering into cranial vault resulting from fracture skull. The line of fracture skull in left occipital bone over epidural hematoma.*

If the hematoma is small and no neurological deficit, surgical evacuation may not be required, and may be observed with careful clinical and CT follow-up but in many case, in order to save the patient's life, open-head evacuation is the treatment of choice. Large hematomas will undergo partial organization and their centers will remain cystic, filled with dark viscous fluid (18). In case where epidural hematomas in the temporo-parietal region are situated so as to cause maximum displacement of the brain stem as they enlarge. Symptomatic hematomas should be evacuated without delay.

Subdural Hematoma

Clinically subdural hematomas are classified as acute SDH (presenting within the first 24 hr. of injury), sub acute SDH (presenting after 1 to 10 day), and chronic SDH (presenting beyond 10 days).

Subdural hematomas usually result from closed head trauma, which causes tearing of a dural sinus rupture of the bridging vein, and there may be little other evidence of brain damage, which the hemorrhage comes from a ruptured cortical artery. Subdural hematoma tend to cover the entire hemisphere if bleeding is extensive, and they are almost always more extensive than Epidural hematoma. However, most cases of Subdural hematoma are associated with considerable brain damage, and the mortality and morbidity is greater in subdural hematomas than in epiduraks, and these hematomas are usually associated with skeletal injury, and they may contain a blood clot consisting of xanthochromic fluid. In these cases, they are referred to as subdural hygromas.

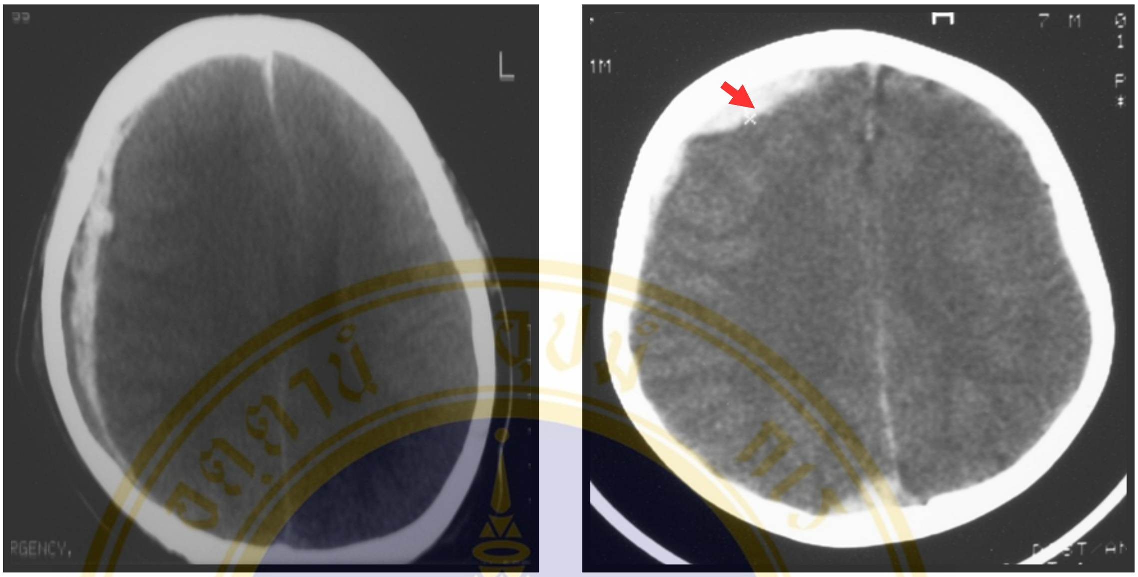


Fig. 2-7 *Acute subdural hematoma: “cresentric shape” of hyperdensity of acute hematoma seen in right frontoparietal region.*

Acute Subdural hematoma is the most common traumatic lesion that requires neurosurgical intervention, occurring in about 5% of all patients with head trauma and in over 50% of those with severe head injury. The mortality rate in patients with acute subdural hematomas accompanying severe head injury ranges from 50 to 88%. Advancing patients age. The increasing size of the hematoma and the delay in the evacuation of the hematoma are all factors associated with poor prognosis (7, 8, 12). In patients who are comatose at the time of initial examination, the mortality rate approaches 100% without surgery. Even with surgical intervention, the mortality rate remains at 60-80%. Mortality incidence does decrease to below 50% if surgical intervention occurs within 3-4 hr. of the injury. In patients with acute subdural hematoma, the residual disability is greater than in patients with epidural hematomas or chronic subdural hematomas because acute subdural hematomas entities generally have a greater degree of associated cerebral damage.

Chronic Subdural Hematoma

Chronic Subdural Hematoma may present weeks or months after what appeared originally to be a trivial head injury. The hematoma occurs when a bridging vain is torn and a slow effusion of venous blood into the subdural space occurs. These bridging veins are most vulnerable to tearing when the subdural space is enlarged, as occurs with atrophy in elderly and alcoholic patients. Eventually, it becomes large enough to distort and even herniated the brain downward, and increase in mass effect may produce more dramatic changes, including unconsciousness. Patients with these symptoms are encountered in the emergency room often after seemingly minor trauma. Patients so well after the drainaged of chronic subdural hematomas, because there is usually no associated cerebral parenchyma injury. Although the subdural space frequently remains enlarged after evacuation of a chronic subdural hematoma. This is owing to the length of time that the chronic subdural hematoma has been present and also to cerebral atrophy.

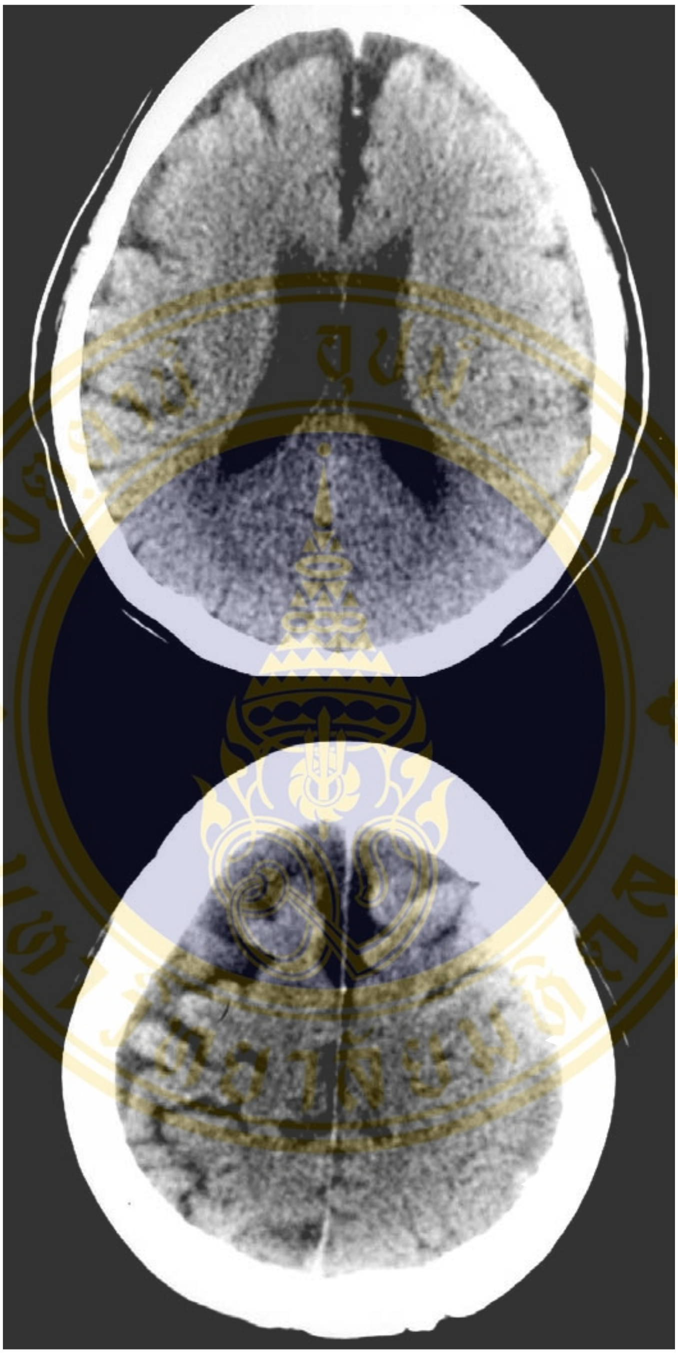


Fig. 2-8 *Subacute subdural hematoma: Isodense subdural hematoma in left parietal region (arrow) with direct cause pressure effect to cortical cerebral gyrus and sulci*

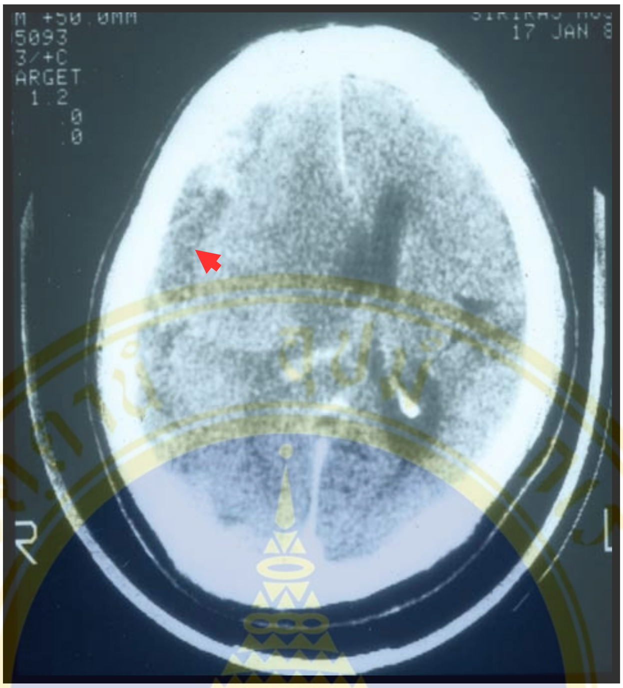


Fig. 2-9 *Chronic Subdural Hematoma shows crescentric shape of hypodensity of old blood clot (arrow) in right frontotemporal region.*

Subarachnoid Hemorrhage

Subarachnoid hemorrhage occurs frequently with head injuries and almost always is associated with other injuries of the brain. In the rare instance when it is the predominant or only abnormality present following an injury, one should consider rupture of a berry aneurysm of a posttraumatic pseudoaneurysm, as its etiology. Like the patient with spontaneous sub arachnoids hemorrhage, the patient with traumatic Subarachnoids hemorrhage is at risk for development of communicating hydrocephalus owing to obstruction of the extra cerebral cerebrospinal fluid pathways.

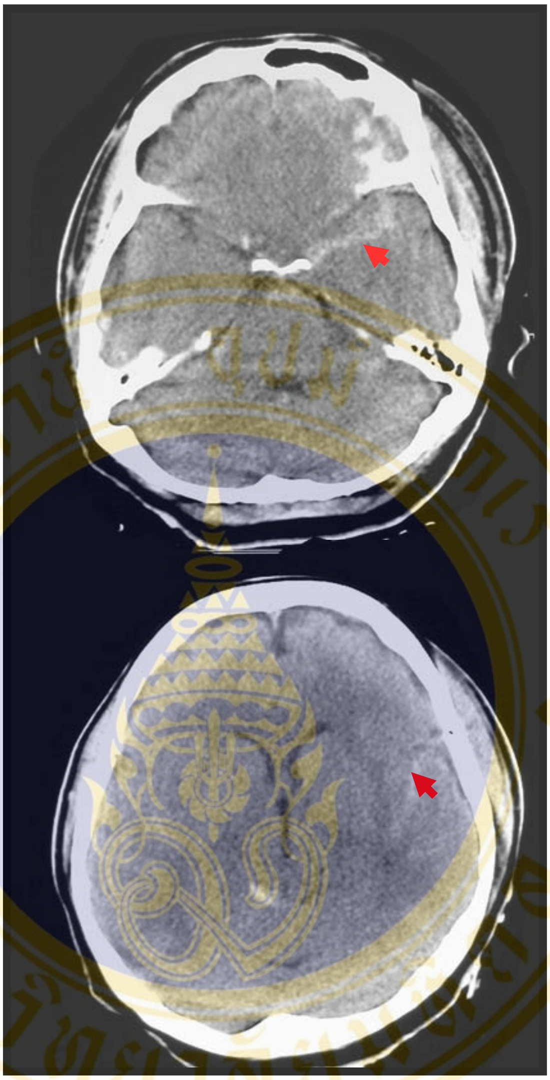


Fig. 2-10 Subarachnoid hemorrhage: *Traumatic subarachnoid hemorrhage is demonstrated in left basal cistern and left sylvian as arrows*

Generally, there is some degree of Subarachnoid hemorrhage associated with contusion or intra ventricular hemorrhage. It is also a frequent occurrence in patients who sustain diffuse axonal injury. Japanese neurosurgical studies have demonstrated that in closed head trauma, those patients who exhibited subarachnoid blood on admission CT scan developed ischemic symptoms between days 4 and 16 after head injury. There has been found a close correlation between the main site of the sub arachnoids blood and the location of focal severe vasospasm in the same anatomic area. Though, in the acute care setting, CT is the preferred method for demonstrating subarachnoid hemorrhage.

Intraparenchyma Hemorrhage

The definitions of intracerebral hematomas are those that generally occur within the brain tissue and are not directly related to the surface of the brain. They are caused by the rupture of internal blood vessels within the brain that are often found deep in the cerebral hemisphere, particularly in the frontal and temporal region following closed head injury. Sequential CT scans have shown that these hemorrhage are often multiple, and their appearance on CT scan is often delayed and may become apparent only several hours or the following day after admission.

In the Glasgow database, intracerebral bleeding or hematomas were found to be present in 16% of the cases. Patients with this type of bleeding have an increased incidence of diffuse axonal injury or what are known as gliding hematomas. Gliding contusions are usually bilateral, but often they are asymmetrical and sometimes restricted to the white matter. Their presence is more related to diffuse than focal brain injury.

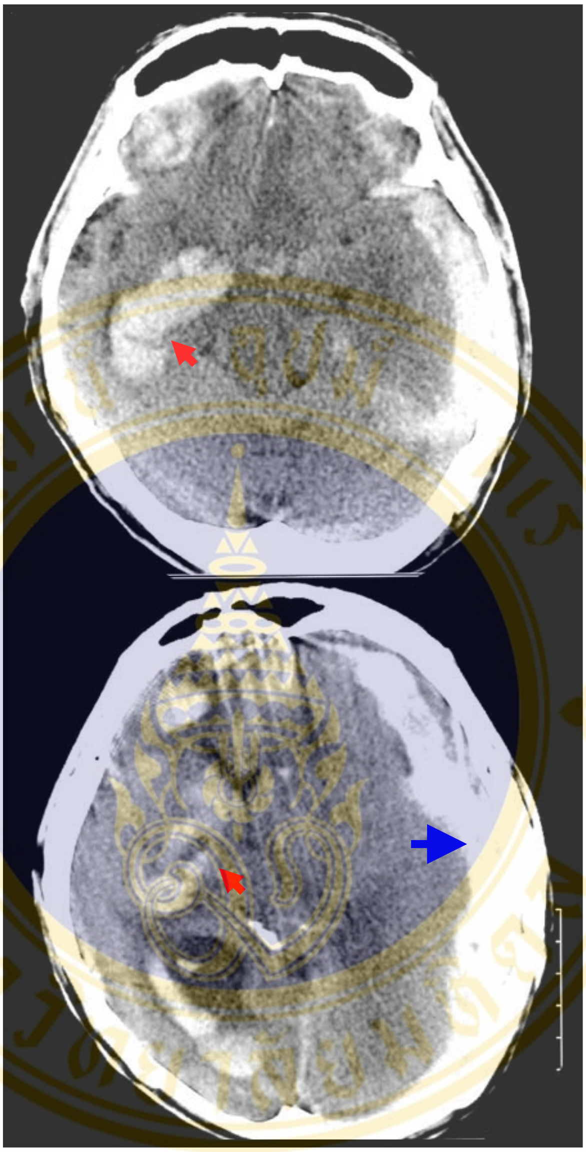


Fig. 2-11 *Intracerebral hematomas occur following head injury in right basal ganglia (arrow) and associated with thick acute subdural hematoma in left frontotemporoparietal region (large arrow)*

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If a solitary hematoma is found deep within the brain of a patient following head injury, the differential diagnosis includes either a hypertensive bleed or the rupture of a saccular aneurysm due to the head injury. However, if the hemorrhage is in the sub frontal or temporal regions, it is more likely to be trauma than of spontaneous vascular origin. Modern brain imaging has revealed that small hematomas or bleeding deeply seated in the brain is often found in the basal ganglia. In these patients, there is a reduced incidence of a lucid interval following injury and an increased incidence of gliding contusion and diffuse axonal injury. If has been suggested that patients found to have basal ganglia bleeding or hematomas shortly after head injury are likely to have sustained diffuse brain damage at the time of the injury. CT observation reveals that a considerable proportion of intra cerebral bleeding and hematomas are not detected until 48 hr. after injury.

Intraventricular Hemorrhage

Often mean tearing of the septum pellucid, and usually seen in patients with diffuse axonal injury.

Diffuse Brain Damage

There are four principal types of diffuse brain damage, and three are seen frequently in patients who survive their injuries long enough to be admitted to the hospital: diffuse axonal injury, ischemic brain injury, and brain edema. The fourth principal type is diffuse vascular injury. Patients who sustain this generally die very soon after their head injuries.

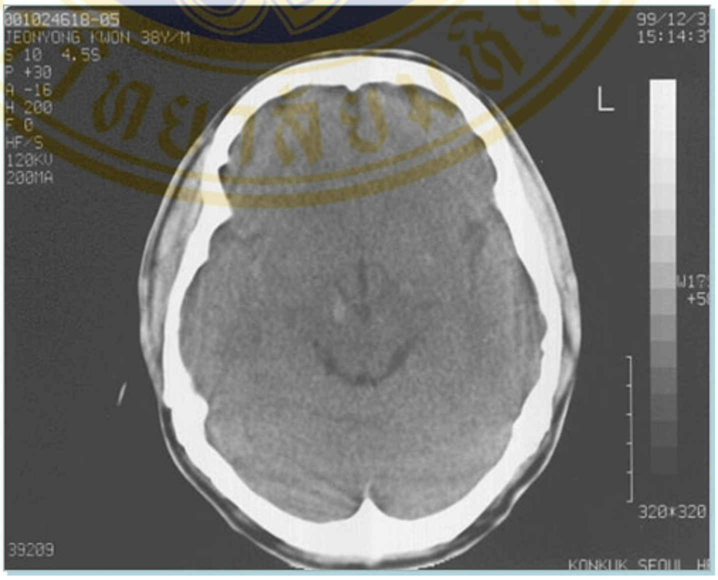

Fig. 2-12 *of patients with DAI, 80% demonstrate multiple hemorrhages areas of injury on CT. Note that the hemorrhages are characteristically located at the gray-white matter interface.*

Diffuse Axonal Injury (white matter shearing)

This severe injury may show virtually no macroscopic change in the affected brain. There is disruption of axons in the sub cortical parasagital white matter and in various other sites, including the internal and external capsules, far nix and cerebellum. It is claimed to result from acute lateral acceleration or deceleration of the brain within the rigid cranium and can occur without anything actually striking the head. It occurs most commonly in automobile accidents.

Small focal hemorrhages have been described in the corpus calsosum and in the posterolateral quadrant of the rostra brainstem and such lesions could theoretically be demonstrated, as can small sub cortical hemorrhages. Susceptibility weighted (T2) MRI is most sensitive at detecting hemorrhage, including old hemorrhage.

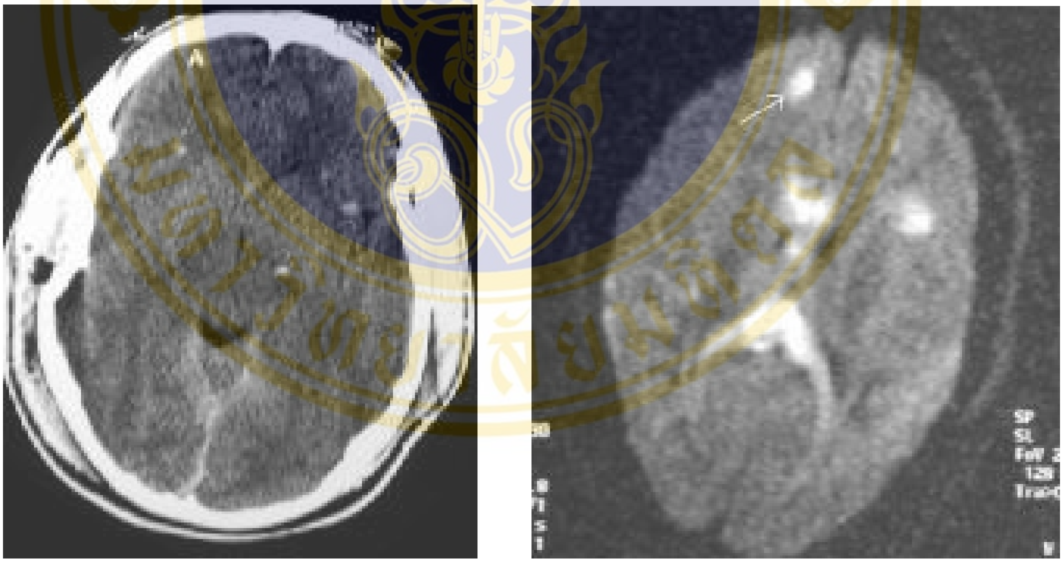

Fig. 2-13 *Non-contrast CT of a trauma patient demonstrates multiple petechial hemorrhages (arrows) consistent with diffuse axonal injury. Note that the hemorrhages are characteristically located at the gray-white matter interface. MRI diffusion sequence demonstrating multiple foci of abnormal increased signal at the gray-white matter junction (arrow) and within the corpus callosum in a patient with diffuse axonal injury.*

Severe diffuse axonal injury not accompanied by an intracranial mass lesion occurs in almost 50% of patients with a severe head injury, cause 55% of all deaths after head injury, and is the most common cause of the chronic vegetative state and severe disability until death (15, 19). Severe causes of diffuse axonal injury have three distinctive features: (1) a focal lesion in the corpus callosum, which usually extends anterior and posterior along the axis and lies to one side of the midline, and is often associated with intra ventricular hemorrhage; (2) focal lesions of the rostra brain stem adjacent to the superior cerebella peduncles; and (3) microscopic evidence of widespread damage to axons. Damage to axons seems to be mostly involved in the corpus callosum and rostra brain stem lesions. Patients who sustain diffuse axonal injury often have associated gliding contusions, and henatomas in the basal ganglia and hippocampi. These injuries are particularly associated with acceleration-deceleration trauma in motor vehicle accidents, but they have also been described after assaults. Some patients who fall from considerable height will also sustain diffuse axonal injuries (20, 21). Pathological histochemistry has demonstrated the presence of axonal swellings appearing 3 to 12 h after an injury (15). Diffuse axonal injury should be suspected strongly if there are focal lesion in the corpus callosum and the appropriate areas of the brain stem noted by CT or MRI. If gliding contusions or hematomas are found in the basal ganglia on appropriate brain imaging, the likelihood is even greater that diffuse axonal injury has occurred.

Brain Swelling

Brain Swelling occurs frequently in association with head injury and may be localized or generalized. It can occur singly or in combination with other focal brain injuries. It may contribute to the elevation of intra cerebral pressure by impeding homodynamic corrections within the brain following trauma. Swelling of sufficient severity may cause morbidity or death by distant trauma to the brain stem. The causes of swelling are not always clear, but in many cases, they are due to an increase in the cerebral blood volume, which causes congestive brain swelling. Swelling may also result from increased water content of the brain tissue, producing cerebral edema. There are three principal types of brain swelling: (1) swelling adjacent to contusions, (2) diffuse swelling of one cerebral hemisphere, and (3) diffuse swelling of both cerebral hemispheres (15).

Skull volume is finite. As mass lesion such as hematomas occur, intra cranial pressure may increase. This increase is often contributed to by brain swelling. Swelling of one hemisphere is seen most often due to an acute subdural hematoma over that hemisphere (22). Diffuse swelling of both hemispheres tends to appear in younger head injury victims. The pathogenesis of this type of brain swelling is not clear but may be related to loss of vasomotor tone and subsequent vasodilatation.

Brain Shift and Herniation

Brain Shift and Herniation occurs frequently from secondary injury after head trauma. If a hematoma continues to enlarge, or focal swelling of adjacent brain tissue increases, the brain is shifted away from the growing mass, and structure that normally lie in the midline may be displaced. The falx is a very tough and adherent tissue and tends to remain in the midline. As a result, the cingulated gurus may herniate under the free edge of the flax and cause compression or distortion of the pericallosal arteries. Because the foramen of Monroe becomes occluded in this process of midline shift, the contra lateral ventricle may become dilated while the ventricle on the side of the mass becomes compressed. This sign on CT scan is a reliable indication that intra cranial pressure is increased

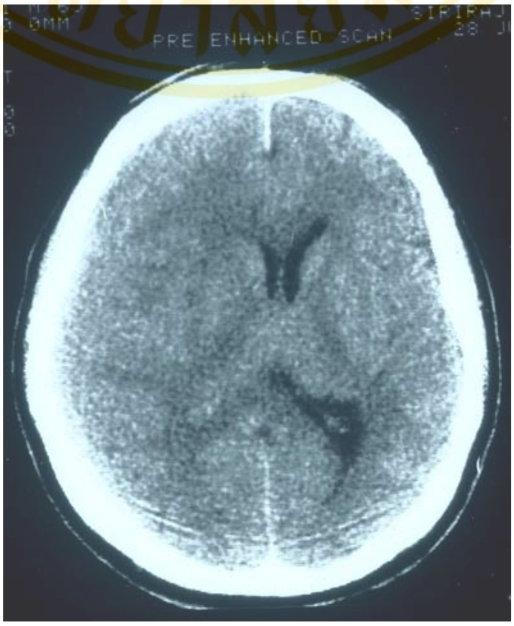

Fig. 2-14 *Brain Shift less than 10mm*

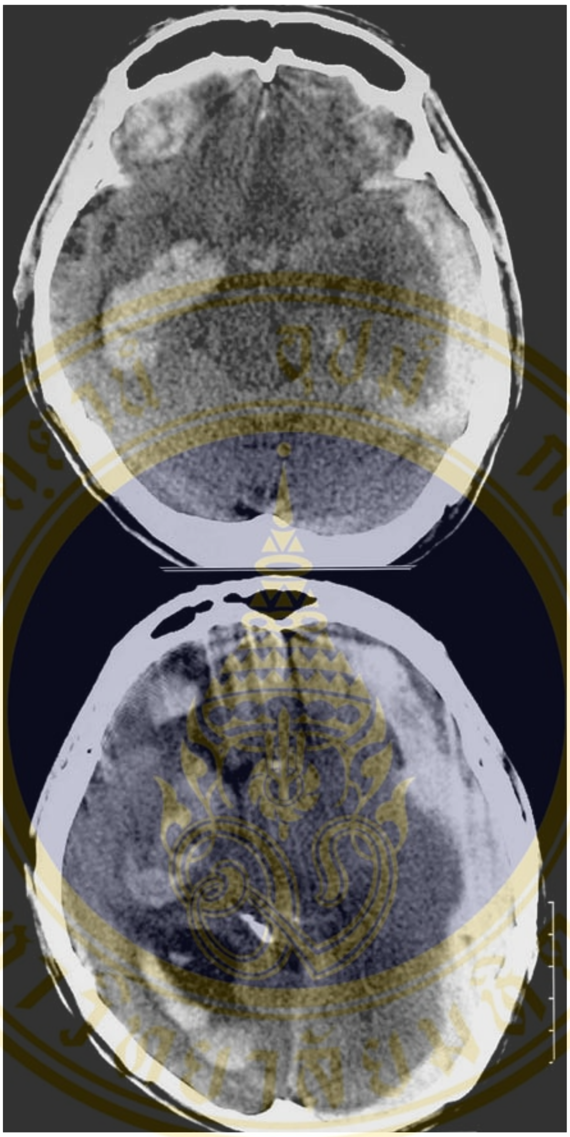

Fig. 2-15 *Brain Shift greater than 10mm*

With a hematoma, compression of the supratentorial compartment may occur. This is usually in lateral and compresses the posterior cerebral artery and the third cranial nerve on the same side as the mass, markedly enlarging the ipsilateral pupil. In bilateral or frontal lesions, the swelling may cause posterior herniation compressing the rectal plate, which results in bilateral pupil abnormalities and inability of the patient to look upward. With infratentorial masses, or a further progression of a supratentorial mass, herniation eventually occurs with downward displacement of the cerebella tonsils through the foramen magnum. This will compress the medulla, causing apnea followed by cardiac arrest and death.

Imaging

X-RAYS

Skull X-rays has essentially been superseded by CT scanning and their routine use in the emergency evaluation of head injured patients has been questioned.

Fig. 2-16 Skull film**:** *Lateral* ***skull*** *radiograph reveals a linear radiolucency in parietal bone (arrow).*

Skull radiographs

- Once an important part of the head injury evaluation, skull radiographs have been replaced by CT scans and are rarely used in patients with closed head injury.
- Skull radiographs are occasionally used in the evaluation of penetrating head trauma, and they can help provide a rapid assessment of the degree

of foreign body penetration in no missile penetrating head injuries (eg, stab wounds).

- Skull radiographs are sometimes used in patients with gunshot wounds to the head to screen for retained intracranial bullet fragments.

Computed Tomography use in the Acute Care setting of brain trauma

Computerized tomography (CT) has been widely accepted as the primary radiographic diagnostic tool for evaluating head injury patients because of its sensitivity and relative expediency. Moreover, CT has various advantages: it is more widespread, low in cost, and safe, and it has rapid imaging time. CT has eventually replaced skull x-ray as the primary imaging tool in head injury because it provides imaging not only of the brain but also of other soft tissues, as well as the bony calvarium. CT will not show every calvarial fracture, but it will show a sufficient number of depressed ones and reveal basilar skull fractures that planar x-ray do not demonstrate. CT is also the method of choice for demonstrating fractures of the facial bones, including the Para nasal sinuses and orbits.

CT's main role in the screening of brain injury is to separate patients into three categories: 1 those with normal intra cranial structures, 2 those with focal intra axial or extra axial hematomas, and 3 those with a more diffuse pattern of brain injury. However, a car dial rule to be followed when evaluating acute head injury is that normal findings by CT scan do not exclude central nervous system injury. In fact, in those patients demonstrating mild cognitive impairment who are triaged in the emergency department, greater age, a Glasgow Coma Scale (GCS) score of 14 or 15, and cranial soft tissue injury are risk factor for CT-detected intra cranial hemorrhage. It is advised that when an admission CT scan demonstrates evidence of diffuse brain injury, follow-up scans should be performed, because approximately one in six such patients will demonstrate significant CT evolution of injury over time. Table1. Categorize the appearance of CT imaging of brain trauma.

Skull Fracture

CT with bone window settings is now the method of choice for determining the presence of skull fracture, rather than standard planar cranial x-ray. However, when the neuropsychiatry examiner reviews medical records and observes shortly after the time of trauma prior evidence of a skull fracture, it must be remembered that bony injury is significant, not only as a sign of potential brain injury, but also as a pathway for the spread of infection. Although skull x-ray have been sub option in demonstrating cranial fractures, but now high-resolution cranial CT with thin sections is the best modality for demonstrating such fractures.

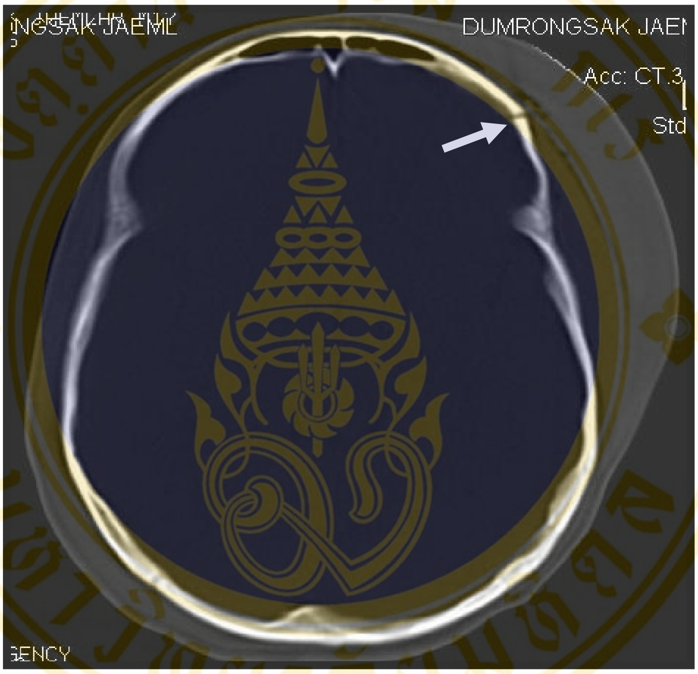

Fig. 2-17 ***CT scans*** *in skull trauma usually using of bone window to identify discontinuity of bony contour of cranial vault. This CT image shows fracture in left frontal bone (arrow)*

Contusions

On CT imaging, contusions may produce high-density (hemorrhagic) or low-density (no hemorrhagic) areas of mass effect. The single most frequent hemorrhagic brain lesion seen on CT is a hemorrhagic contusion. One problem with CT in recognizing small superficial contusions when a thin stripe of high-density cortical blood lies next to high-density bone is that an artifact may obscure the blood in the hemorrhage. Blood on the surface of the brain adjacent to bone may produce a beam-hardening artifact. Contusion of the parietal vertex and inferior temporal lobe may be partially volumes with contiguous bone on the axial CT slice, resulting in an overall bone density that obscures the presence of the contusion. Usually, coronal images are not obtained during CT studies in the emergency department, and as a result, such contusions are frequently missed.

Brain Stem Injury

CT imaging is a very poor modality for detecting the dorsolateral aspect of the upper midbrain. This injury occurs as the brain stem strikes the edge of the tentorium. However, this is the most common location for acute posttraumatic brain stem injury. Only 10% of brain stem injuries are clearly detected on CT, and they are usually associated with diffuse axonal injury. In 1988, L.I.Z. Gentry, J.C. Godersky et al (24) described that, with the advent of CT scan and MRI, it became possible to study the pathological changes in the brain stem while patients are alive. The superior sensitivity of MRI over CT in detecting the lesion and anatomical delineation of the lesion in patients with intra cranial pathology has been well documented, especially in the case of patients with a no hemorrhagic lesion or brain stem lesion.

Extradural (Epidural) Hematoma

Epidural hematomas are occurs frequently in the posterior fossa as a result of tearing of dural veins or sinuses. A skull fracture is found in more than 95% of the case of epidural hematoma. The CT appearance of an epidural hematoma depends on the source of the bleed, the interval between the time of injury and the CT acquisition, the severity of the hemorrhage, and the degree of blood clot organization or breakdown. The majority of epidural hematomas have the appearance of a biconvex lens on the CT scan. Vertex epidural hematomas may not be seen on axial CT images unless they have a significant mass effect as a result of pushing the dura onto the brain. An epidural hematoma is usually homogeneously hyper dense on CT. If focal isodensity or hypo density zones are noted within the hematoma, this usually indicates the presence of active bleeding or a coagulopathy. An irregular hypo dense swirl correlates with active bleeding in the majority of cases.

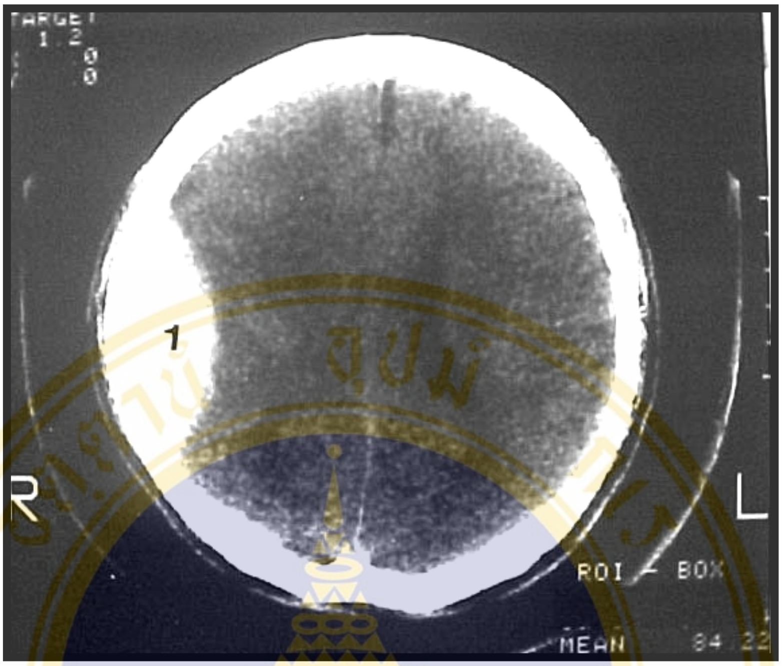

Fig. 2-18 Epidural heamatoma: *CT scan show acute epidural hematoma as lentiform shape of hyperdensity of blood clot (1) in right parietal region. No midline shifted in this patient*

Subdural Hematoma

The subdural hematoma space is a potential space that lies between the dura and the arachnoids membranes. During trauma, the arachnoids may be torn and separated from the dura and associated with tearing of the bridging veins by rapid acceleration or deceleration of the head. Subdural hematomas are classified as either acute or chronic. They do not cross the midline because they fixed by sites of dural attachment at the falx and tentorium. . Subdural hematomas represent 10 to 20% of all craniocerebral trauma cases and occur in up to 30% of fatal brain injuries. An acute subdural hematoma can appear isodense against gray matter if the hemoglobin concentration is below 10 to 11 g/dl. If the subdural hematoma is not isodense, it is easily recognized on CT as an extra axial, crescent, and homogeneously hyper dense collection of blood that conforms to the cerebral surface. If often has a mass effect that can be gauged by the degree of sulcal effacement and inward buckling of the gray matter-white matter interface. Often there is a midline shift of the falx noted. Bilateral isodense subdural hematomas may cause diagnostic difficulty, but they can be detected if one pays attention to identifying the displacement of gray matter with effacement of cortical sulci and compressed ventricles. Contrast enhancement of the CT scan may be needed to assist with diagnosis of bilateral isodense subdural hematoma. On CT scan, parietal acute inter hemispheric subdural hematomas often are found. These subdural are hyper dense when acute. In patients who are brought to medical attention a week or more after injury, the subdural hematomas may be isodense or hyper dense relative to brain tissue, and therefore more difficult to recognize on CT. Sometime, diffuse brain swelling is seen on CT accompanying shaking injury, and at the present time. Its etiology remains not completely understood.

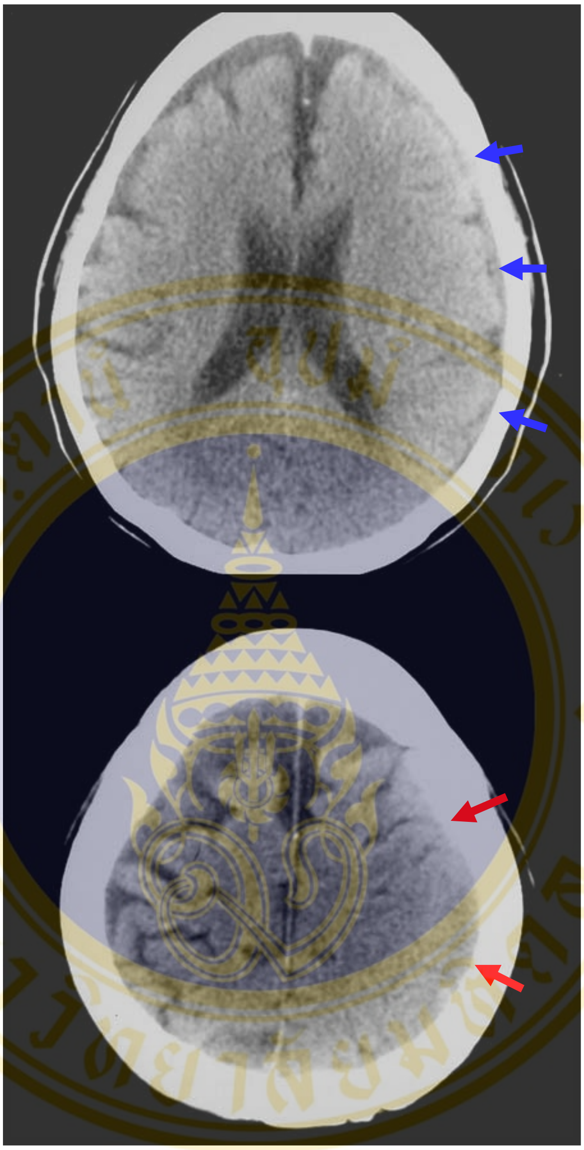

Fig. 2-19 *Isodense of subdural hemorrhage in left frontoparietal region to brain parenchyma, however, by using clinical history as well as pressure effect of hemorrhage to cortical gyrus are helpful in diagnosis.*

Subarachnoid Hemorrhage

CT defines acute sub arachnoids hemorrhage quite effectively. CT is the procedure of choice for identifying the radiographic findings of sub arachnoids hemorrhage because blood that occupies the full thickness of a CT slice reveals the increased density as a distinct area of brightness. When a sub arachnoids hemorrhage is present along the falx, it typically disappears during the ensuing week. In children, the incidence of subarachnoids hemorrhage identified on CT increases with the increasing severity of a head injury. However, sub arachnoids hemorrhage is difficult to appreciate when the CT study is done more than several days after trauma.

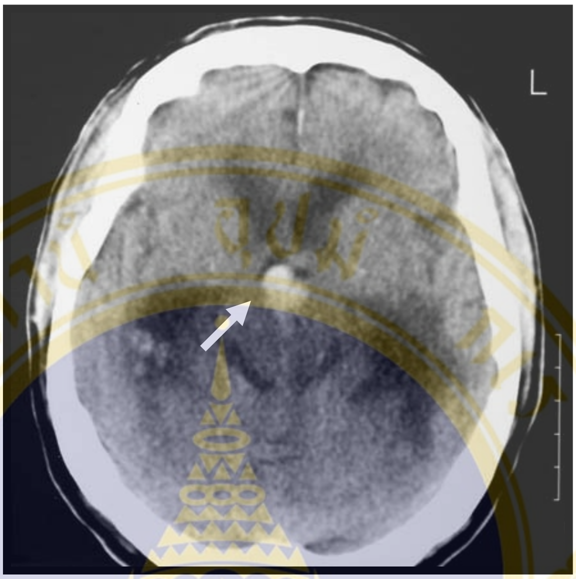

Fig. 2-20 Intraventricular Hemorrhage: *Hematoma in the 3rd ventricle is demonstrated as hyperdense of hematoma*

Intraparenchyma Hemorrhage

The intracerebral bleeding may occur from coup or countercoup mechanisms. Other areas wherein Intraparenchyma Hemorrhage may occur are the anterior and middle cranial fosse, the sphenoid wings, and petrous ridges. On CT, large intracerebral hematomas generally occur in the same distribution of contusion. That is, they are mostly found in the frontal and temporal brain areas. They may be related to a hemorrhagic contusion into which bleeding has occurred with clot formation. The clot may dissect through the white matter, or it may arise from the rupture of a penetrating vessel deep within the white matter. However, even contusions without significant hemorrhage appear as high-density areas.

Intraventricular Hemorrhage

As many as 25 % of patients with severe head injury have Intraventricular Hemorrhages. Focal and diffuse areas of high attenuation are identified within the ventricles following CT imaging in these cases. Blood tends to settle in the dependent portions of the ventricles where a cerebrospinal fluid-blood level forms. If no rebleeding occurs, intra ventricular hemorrhage is rarely seen after about 1 week. Intra ventricular hemorrhage frequently exists with other findings of head trauma. The hemorrhage can be a consequence of tearing of subependymal veins or rupture of the ependymal layer and an extension of a sub arachnoids hemorrhage or a parenchyma hemorrhage. CT reveals this finding effectively.

Diffuse Axonal Hemorrhage

Most injuries will be noted in the lobar white matter, particularly at the corticomedullary junction of the frontal and temporal lobes. Diffuse axonal injury may also occur near or in the corpus callosum, or in the dorsolateral aspect of the brain stem in cases of severe trauma. About 75% of callosal injuries occur in the posterior body and selenium of the corpus callosum because the posterior falx prevents lateral displacement of the hemispheres during rotational acceleration of the head. In summary, Diffuse Axonal Injury (DAI) occur in four sites on CT examination: 1 corpus callosum, 2 corticomedullary junction, 3 upper brain stem and 4 the basal ganglia. However, it often is not possible to see small hemorrhages in the corpus callosum on CT. After edema resolves and hemorrhage is physiologically removed, the CT scan may appear normal even though the patient has significant cognitive and behavioral abnormalities. In other cases, the follow-up CT scan may show only generalized cerebral atrophy.

Brain Swelling

Diffuse cerebral swelling is commonly associated with closed-had injury and is well visualized by CT. On normal CT soft tissue window settings, the cerebellum, the cerebral vasculature, and the dural surfaces (falx and tentorium) appear hyper dense against the background of diffusely swollen, edematous hypo dense brain. A herniation across the tentorium is commonly present, and the mortality rate is high if swelling is not controlled quickly.

Brain Shift and Herniation

Four main types of brain displacement can occur: 1 subfacine, 2 descending and ascending transtentorial, 3 descending and ascending transalar, and 4 cerebellar tonsillar herniation.

Subfacine herniation describes a midline shift that displaces the cingulated gyrus beneath the falx. A midline shift of 5 mm or more is considered significant from a surgical standpoint. A shift of this magnitude is associated with a 50% mortality rate (3). The presence of midline shift > 5 mm is a commonly used indication for emergency evacuation of an acute intra cranial hematoma, whether trauma or non traumatic (9, 10). Therefore, midline shift after traumatic brain injury is widely recognized as an important marker of severe injury. Numerous reports described the association of a large amount of midline shift on computed tomographic (CT) scan (usually described as > 5 mm) with poor outcome or other adverse sequel of traumatic brain injury (24, 25). In transtentorial herniation, the descending type is the result of medial and inferior displacement of the uncus and parahippocampal gyrus of the temporal lobe through the tentorial notch. On CT scan, it will be seen as an encroachment on the lateral aspect of the ipsilateral suprasellar cistern. In severe cases, the brain stem will be displacement and the contra lateral cerebral peduncle will be compressed against the adjacent tentorial incisura. Complete uncal herniation results in obliteration of the suprasellar and perimesencephalic cisterns.

Ascending transtentorial herniation is much less common than the descending variety but may occur in two clinical situations: 1 direct effect of a posterior fossa, or 2 following rapid decompression of a supratentorial space-occupying lesion. CT will reveal flattening of the posterior aspect of the quadrigeminal plate cistern. Eventually, compression of the cerebral aqueduct causes hydrocephalus of the third and lateral ventricles. Transalar herniation refers to brain shifts across the sphenoid wing. These shifts may be caused by swelling or bleeding in the anterior cranial fossa (descending type) or in the midline cranial fossa (ascending type). If transalar herniation is severe enough, it can cause infarctions in the distribution of both the anterior and middle cerebral artery branches. Tonsillar herniation results from an enlarging mass in the posterior fossa or following supratentorial cerebral swelling. The CT scan will demonstrate crowding of the cisterna magna by the downward displacement of the cerebral tonsils. This resulted in obliteration of the cerebrospinal fluid cisterns around the medulla. The ultimate resultof tonsillar herniation is cardiopulmonary arrest due to brain stem compression.

Magnetic Resonance Imaging

Magnetic Resonance Imaging (MRI) provides accurate detail of parenchyma damage, specifically small collections and non-hemorrhagic contusions. However, the information provided is not significantly better than that obtained from CT scanning to warrant routine use of MRI in the acute phase of injury. In addition, the placement and monitoring of an acute traumatized patient in an MRI scanner poses an additional risk that does not justify its use in that situation.

MRI may have an important role in prognostication at a later stage in management, particularly in mild and moderate head injury.

MRI may be used in some patients with TBI to assess for arterial injury or venous sinus occlusion.

MRI is superior to CT scan for helping identify diffuse axonal injury (DAI) and small intraparenchymal contusions. DAI is defined as neuronal injury in the subcortical gray matter or the brainstem as a result of severe rotation or deceleration.

In 1988, Hadley D.M., et al found that MRI was more sensitive to cerebral abnormalities associated with traumatic unconsciousness and detected parenchymal lesions both in patients in coma and in those who had lost consciousness for only a few minutes. Lesion seen with MRI but not with CT included non-haemorrhagic contusions and abnormalities thought to reflect shearing injuries of white mater and intracerebral vessels (50).

Compare CT and MRI in the evaluation of acute head injury

Computed tomography and MRI are the most common techniques in patients who have suffered brain injury. Computed tomography is currently the first imaging technique to be used after head injury, in those settings where CT is available. Using CT, scalp, bone, extra-axial hematomas, and parenchyma injury can be demonstrated. Computed tomography is rapid and easily performed also in monitored patients. It is the most relevant imaging procedure for surgical lesions. Computed tomography is a suitable method to follow the dynamics of lesion development giving an insight into the corresponding pathological development of the brain injury. The advantages of CT include widespread availability, rapid imaging time, relatively low cost and safety.

Its prognostic ability has been widely studied and is generally significant and accepted (26, 27).

Magnetic resonance imaging is more sensitive for all posttraumatic lesions except skull fractures and sub arachnoids hemorrhage, but scanning time is longer, and the problem with the monitoring of patients outside the MRI field is present. If CT does not demonstrate pathology as can adequately be explained to account for clinical state, MRI is warranted. Follow-up is best done with MRI, as it is more sensitive to parenchyma changes. In routine MR protocol gradient-recalled-echo sequences should be included at any other time after a traumatic event since they are very sensitive in detection of hemosiderin as well as former hematoma without hemosiderin. The MR signal intensity varies depending on sequences and time scanning after trauma. Orrison WW, Gentry LR et al. found that the sensitive of MR was significantly higher than that of CT for the detection of contusion, shearing injury, subdural and epidural hematoma, and sinus involvement. The sensitivity of CT was significantly higher than that of MR for the evaluation of fracture. The sensitivities of MR and CT were statistically equivalent for the detection of superficial soft-tissue injury. The overall sensitivity of MR for the detection of abnormalities in acute head trauma was 96.4%, and for CT was 63.4% (28).

Cerebral Angiography

Cerebral angiography should be considered when a vascular injury such as carotid artery dissection is suspected in head injured patients. A large isodense lesion on CT scan may indicate this, or when the patient’s clinical condition is not consistent with the CT findings, for example, a dense hemi paresis in the absence of a mass lesion.

In the absence of a CT scanner, cerebral angiographies may be used in the diagnosis of intracranial mass lesion, although this is now rare. Currently, angiography in used in acute head injury only when a vascular injury may be present. This includes patients with unexplained neurologic deficits, especially in the setting of temporal bone fractures, and patients with clinical evidence of a potential carotid injury.

Midline shift in CT scan occurred frequently in patients with head injury and is one of the most important factors in the prognosis poor outcome, especially patients severe head injury with Acute Subdural Hematoma (ASDH) and Epidural Hematoma (EDH). Many previous result studies (34, 39, 47) revealed that midline shift was a significant predictor of mortality. Mass effect, a factor closely related to haematoma size, which can be measured simply as midline shift has been known to correlate closely with outcome. Although the authors did not measure the degree of midline shift, Fearnside MR, Cook RJ et al. showed that mortality increased with the degree of shift. Kotwica and Brzezinski (52) showed 42% favourable outcomes and a mortality of 39% when the midline shift was below 1.5 cm, 25% favourable outcomes and a mortality of 52% when this shift was from 1.5 cm to 3 cm and 8% favourable outcomes and a mortality of 76% whenever the shift exceeded 3 cm. In contrast to a study by Narayan (43), the extent of midline shift did not add significantly to the prognostic capability of their model. Selladurai (32) also noticed that the degree of midline shift did not prove to be of a predictive significance. In study by A.A. Azian et al. in 2001 found that of all the patients with midline shift (36 cases), 16 cases had GCS score of 9-15 while 20 cases fell in to the GCS score range of 3-8 which is the unfavorable group. The multiple variate analysis found that midline shift was noted to be significant.

In our studied were to midline shift only, midline shift combine with Glasgow Coma Scale score (GCS) and midline shift combine with CT lesion finding to predict poor outcome (death rate) in patients head injury following both univariate and multivariate analysis.

Benefit from this study

In our study can determine correlation between GCS score and midline shift by CT scan finding, it found that the patients lower GCS score after head trauma no significant related with degree of midline shift in patients head injury. But lower GCS score related significant with CT lesion finding. So indications for head CT scan film followed by;

1. GCS less than 13 at any point since the injury
2. GCS equal to 13 or 14 at 2 hours after the injury

3. Suspected open or depressed skull fracture
4. Any sign of basal skull fracture
5. Post- traumatic seizure
6. Focal neurological deficit
7. More than one episode of vomiting
8. Amnesia for greater than 30 minutes of events before impact
9. Patients older 65 years, coagulopathy or dangerous mechanism of injury

Midline shift as measured from the septum pellucidum can predict death or poor outcome in patients head injury, and found that the likelihood of death after head injury is increased 4 times if midline shift is greater than 10mm, and is decreased if midline shift is less than 10mm. by univariate, but the multiple variate analysis found that midline shift was noted to be significant. The reason of not significant may be due to

- patients have multiple contusion and brain oedema without midline shift
- patients diffuse axonal injury without midline shift
- patients have multiple injury and head injury
- patients poor outcome or death is also influence by the location of intracerebral lesion and the present of bilateral abnormalities
- Older patients and lesion with midline shift < 10mm.
- Attempts to improve the accuracy of predictions have considered additional factors such as age, intracranial pressure, nutrition, reflex eye movements, the type of intracranial lesion, and the location of the lesion. The interaction of many effects has been studied using computer multifactorial analyses. It is obvious that outcome from acute brain injury depends on the complex interaction of a multitude of factors, and additional useful criteria are actively being sought.
- Degree of midline shift combined GCS score in traumatic head injury was interaction significantly associated with poor outcome only with severe head injury, no difference in patients mild and moderate injury.

CHAPTER III

MATERIALS AND METHODS

This study was conducted on 217 cases of traumatic head injury admitted to the regional trauma center in the Siriraj Hospital during January 1999 until 2004.

Criteria for inclusions into the study were: (1) All traumatic head injury patients and CT scan film with and without midline shift. Midline shift in this study included all over degree and divided into three categories classification were no shift, shift up to 10 mm, shift greater than 10 mm. (2) Select cases found CT scan film and complete medical record. (3) Patients over 3 years of age. Severity of the head injury was determined clinically on admission to the Accident and emergency Unit using the Glasgow Coma Scale (GCS) score which incorporated measurements of the best motor and verbal responses as well as eye opening. Initial GCS score was considered the first GCS taken in the emergency room within 24 h of injury. On admission, all patients had registered the Glasgow Coma Scale (GCS score), the GCS score was divided into three categories according to SIGN 2000 Guideline (6) classification.

In addition, other associated injures sustained also documented example, extremity fracture, chest, limb or pelvic injury as well as presence or absence of skull fracture. The patients with acute hypertension before trauma were excluded.

CT brain scan was performed using Philip CT scanner. Axial slices of 10 mm. and 5 mm. for posterior fossa were done parallel to the orbito-meatal line from the base of skull up to the vertex. All slices were done contiguously with brain and bone windows without intravenous contrast.

The midline shift in CT scan is defined as the absolute distance (in mm) the septum pellucidum of the brain is displaced with respect to the midline which is determined by averaging the distance between the inner tables of the skull (29). In 1991, Lobato et al. was divided the midline shift into three categories for convenience of data analysis. Classification >= 15 mm, 6-15 mm, <=5mm. Intracranial hemorrhages for recorded into this study were: Intracerebral (ICH), Extradura (EDH), Subdural (SDH), Intraventricular hemorrhage (IVH), Subarachnoid (SAH), Hemorrhagic contusion, Brain edema (30).

Locations were noted for all types of intracranial hemorrhage. The number (single or multiple), sides, site of the hematomas were also estimated for midline shift. Intracerebral, Extradural and Subdural hemorrhages. The outcome of the patients at discharge was categorized according to the Glasgow outcome scale by clinical examination as proposed by Jennett and Bond (31) as: I death; II persistent vegetative state; III severe disability (conscious but disabled); IV moderate disability (disabled but independent) and V good recovery. In this study divided into two groups were good and poor outcome. Good outcome included improved and good recovery; poor outcome included severe disability and death from discharge status.

The purpose of this investigation was to examine the relationship between degrees of midline shift from CT scan finding and Glasgow Coma Scale score (GCS) for predict outcome in patients after head injury. Therefore the prognostic factors in patients with moderate and severe head injury can guide for review included:

Clinical data:

- Admission GCS
- Age
- Gender
- Mechanism of injury
- Pupillary reactions
- Focal neurological signs

Typology of Traumatic Brain Damage:

- Skull fracture
- Epidural hematoma (EDH)
- Subdural hematoma (SDH)
- Intracerebral hematoma (ICH)
- Diffuse Axonal Injury (DAI)
- Brain Shift and Herniation

Imaging Studies:

Skull radiographs

- Once an important part of the head injury evaluation, skull radiographs have been replaced by CT scans and are rarely used in patients with closed head injury.

- Skull radiographs are occasionally used in the evaluation of penetrating head trauma, and they can help provide a rapid assessment of the degree of foreign body penetration in nonmissile penetrating head injuries (eg, stab wounds).
- Skull radiographs are sometimes used in patients with gunshot wounds to the head to screen for retained intracranial bullet fragments.

CT scan

- A CT scan is the diagnostic study of choice in the evaluation of traumatic brain injury (TBI) because it has a rapid acquisition time, is universally available, is easy to interpret, and is reliable.
- The standard CT scan for the evaluation of acute head injury is a noncontrast scan that spans from the base of the occiput to the top of vertex in 5-mm increments.
- Three data sets are obtained from the primary scan, (1) bone windows, (2) tissue windows, and (3) subdural windows.

Statistical Analysis

Univariate analysis was performed to determine statistically significant potential predictor variables to be included in the multivariate analysis.

Description and logistic regression method was applied to select the most appropriate statistical model to determine the predictors of this study. Odds ratio with 95% confidence (by forward stepwise) interval was used to estimate the association and strength of the determinant to the outcome (percent death). P values less than 0.05 were considered as statistically significant.

CT and Traumatic Brain Injury

Lesion	**Image Findings**
Skull fracture:	Calvarial disruption on bone window settings
Contusions	Usually adjacent to anterior and middle cranial fossa, sphenoid wings, And petrous ridges, most frequent in frontal and temporal poles and undersurfaces of frontal lobes; hemorrhagic lesions, high-density; no hemorrhagic lesions, low- density.
Epidural hematoma	Usually presents as a high-density, biconvex lens; does not cross suture Margins; focal iso-or hypo density consistent with active bleeding or coggulopathy
Subdural hematoma	Acute: isodense against gray matter if hemoglobin less than 10-11 g/dl; if not isodense, present as a crescent-shaped hyper dense collection that conforms to the gyral-sulcal pattern; does not cross the falx Chronic: fluid usually appears hypo dense due to blood product breakdown, but density higher than CSF due to protein content; upon complete breakdown of product, fluid may be isodense to brain
Subarachnoid hemorrhage	Linear hyper dense fluid collection within sulci and fluid cisterns
Intraparenchyma hemorrhage	Mostly found in frontal and temporal brain areas; usually hyper dense in appearance; serum from a clot may cause a rim of hypodensity; edema may produce a mass effect; in older lesions, new vessel formation may enhance as a rim with contrast agents; clot resorption may leave a cavity
Intraventricular hemorrhage	Focal and diffuse hyper density within the ventricles; blood tens to settle in the occipital horns

Diffuse axonal injury	Most injuries in lobar white matter at corticomedullary junction of frontal and temporal lobes; also appears at or in the corpus callosum and dorsolateral brain stem; usually appears as small hyperdense bleeds in these areas
Brain swelling	Obliteration of cerebral sulci and basal cisterns; effacement of gray matter-white matter interface; edematous brain usually hypodense
Chronic neuro degeneration	Irregular brain surface with hypodensity within parenchyma; overlying cerebrospinal fluid space may enlarge; cortical gyri size may diminish with increased ventricular size

CHAPTER IV

RESULTS

A total of 453 cases at admitted the Department of Trauma and have been examined CT scan in Siriraj Hospital during January 1999 until October 2004, 46 cases exclude because not available medical record and 190 cases not available CT scan film, 217 cases found CT scan film and complete medical record. Demographic feature and injury characteristics are listed in table 1.In 56.6% of patients trauma was due to traffic accidents, in 42.8% were due to fall or assaults. MCA accidents are the most frequent cause of head injury with highest in group were young men, youngest was 3 years and oldest in this study was 93 years (mean age 35.7 years), including 58 cases brain shift up to 10mm. (mean age 37.88 years), 38 cases shift greater than 10 mm. (mean age 42.02 years), and no shift 121 cases, traumatic brain injury with midline shift in this study.

UNIVARIATE ANALYSISES

The results of univariate analysis showing the associated with outcome are shown in table 2, and 3

39 of 217 patients had 30(76.9%) cases good outcome, 9 (23.1%) died in female patients group. 178 of 217 patients had 137(77.0%) cases good outcome, 41(23.0%) cases died for male group. Mortality in male group was 1.94 time to female group (OR 1.94 95% CI 0.59-6.28, p = 0.26)

Total 189 cases in patients group <60 years, 148 (78.3%) cases good outcome, 41(21.7%) cases died. Of 27 cases in patients group ≥60 years, 19 (67.9%) cases good outcome, 9 (32.1%) cases died. Mortality in patients group ≥60 years was higher than patients group <60 years (OR 1.71, 95 % CI 0.72-4.1, p = 0.224) the univariate analysis sex, ages were no significant prognostic variables to outcome.

Of the 121 cases, 103(85.1%) cases good outcome, 18(14.9%) cases died in patients brain injury without midline shift, 42 of 58 (72.4%) cases good outcome, 16(27.6%) cases died in patients brain injury with midline shift up to 10 mm., and 16 of 38(42.1%) cases died, 22(57.9%) cases good outcome in patients brain injury with midline shift greater than 10mm. Mortality was the highest in patients brain injury with midline shift greater than 10mm (OR 4.45, 95% CI 1.95-10.13) followed by patients brain injury with midline shift up to 10mm (OR 2.33, 95% CI 1.08-5.03, P = 0.001).

Total 49 of 50 (98.0%) cases in normal and other brain injury by CT scan film good outcome, 1(2.0%) case died. In patients brain injury with Subdural Heamatoma (SDH) 23 cases, 19(82.6%) cases good outcome, 4(17.4%) cases died, while patients SDH with midline shift of 60 cases, 23(38.3%) cases died, 37(61.7%) cases good outcome. In patients brain injury with Epidural Heamatoma (EDH) 20 cases, 17(85.0%) cases good outcome, 3(15.0%) cases died, patients brain injury with EDH with midline shift total 23 cases, 19(82.6%) cases good outcome, 4(17.4%) cases died. For patients severe head injury or diffuse axonal injury of 15 cases, 13(86.7%) cases died, 2(13.3%) cases good outcome. Mortality was the highest in patients severe head injury or diffuse axonal injury (OR 318.09, 95% CI 26.74-3783.9) followed by patients SDH with midline shift (OR 30.42, 95% CI 3.93-235.07) and patients EDH with midline shift(OR 10.30, 95% CI 1.08-98.07) (*P value = 0.000*).

Total 49 cases, 47(95.9%) cases good outcome, 2(4.1%) cases died in patients mild injury, 33 cases, 31(93.9%) cases good outcome, 2(6.1%) cases died in patients moderate injury, For patients severe injury of 135 cases, 89(65.9%) cases good outcome, 46(34.1%) cases died. Mortality in patients severe injury was 12.14 time to patients mild injury (OR 12.14, 95% CI 2.82-52.25) and mortality in patients moderate injury was 1.51 time to patients mild injury (OR 1.51, 95% CI 0.20-11.33) (*P value = 0.001*).

By univariate analysis, significant prognostic variables report were degree of midline shift, CT scan finding and Glasgow Coma Scale score (GCS) to Outcome in patients head injury (Table 4-2, 4-3).

Table 4-1 Demographic characteristic of patients brain injury with midline shift

Variables	Details of patients Total (N = 217)
Age (years)	Mean = 35.7 Median = 30.0 Range = 90 Minimum =3 Maximum = 93 < 60 = 189 (87%) ≥60 = 28 (13%)
Sex	M = 178 (82%) F = 39 (18%)
Mechanism	Traffic accidents = 123 (56%) Fall = 52 (24%) Hit or Blunt object = 35 (16%) Other = 6 (3%)
Midline shift	No shift = 121 (58%) Shift up to 10mm = 58 (27%) Shift greater than 10mm = 38 (17%)
CT scan finding	Normal & other = 50 (23%) Subdural Heamatoma (SDH) = 23 (10%) Epidural Heamatoma(EDH) = 20 (9%) Intracerebral Heamatoma (ICH) = 26 (12%) SDH with Midline shift = 60 (28%) EDH with Midline shift = 23 (10%) Severe head injury or DAI = 15 (10%)
GCS score	Mild injury = 49 (22%) Moderate injury =33 (15%) Severe injury = 135 (62%)

** DAI (Diffuse Axonal injury), GCS score (Glasgow Coma Scale score)*

Table 4-2 Association between Sex, Ages, Degree of Midline shift, CT scan finding, GCS score and Outcome in patients head injury

Variables	Results					
	Good		Death		χ 2	P Value
	N (167)	%	N (50)	%		
Age (years)						
< 60	148	78.3	41	21.7	1.502	NS
≥ 60	19	67.9	9	32.1		
Sex						
Male	137	77.0	41	23.0	<0.001	NS
Female	30	76.9	9	23.0		
Midline shift						
No shift	103	85.1	18	14.9	13.01	0.001
Shift up to 10mm	42	72.4	16	27.6		
Shift greater than 10mm	22	57.9	16	42.1		
CT scan finding						
Normal & other	49	98.0	1	2.0	59.65	<0.001
SDH	19	82.6	4	17.4		
EDH	17	85.0	3	15.0		
ICH	24	92.3	2	7.7		
SDH with Midline shift	37	61.7	23	38.3		
EDH with Midline shift	19	82.6	4	17.4		
Severe head injury or DAI	2	13.3	13	86.7		
GCS score						
Mild injury	47	95.9	2	4.1	24.56	<0.001
Moderate injury	31	93.9	2	6.1		
Severe injury	89	65.9	46	34.1		

** NS = non significant (p-Value < 0.05)*

***** *SDH (Subdural heamatoma), EDH (Epidural heamatoma), ICH (Intracerebral heamatoma), SDH with MS (Subdural heamatoma with midline shift), EDH with MS(Epidural heamatoma with midline shift), SHI or DAI (Severe head injury or Diffuse Axonal injury)*

Table 4-3 Univariate analysis showing the relationship between independent variables by logistic regression with p-value for sequential tests, Odds ratios (OR) and 95% confidence intervals (95% CI) for predicting outcome of patients head injury.

Variables	P-Value	Death (%)	Crude Odds ratio (OR)	95% CI
Age (years)	0.224			
< 60		21.7	1	-
≥ 60		32.1	1.71	(072-4.1)
Sex	0.268			
Female		23.0	1	-
Male		23.0	1.94	(0.59-6.28)
Midline shift	0.001	14.9	1	-
No shift		27.6	2.33	(1.08-5.03)
Shift up to 10mm		42.1	4.45	(1.95-10.13)
Shift greater tha10mm				
CT scan finding	<0.001			
Normal & other		2.0	1	-
SDH		17.4	10.30	(1.08-98.07)
EDH		15.0	8.63	(0.84-88.62)
ICH		7.7	4.07	(0.35-47.19)
SDH with Midline shift		38.3	30.42	(3.93-235.07)
EDH with Midline shift		17.4	10.30	(1.08-98.07)
Severe head injury or DAI		86.7	318.09	(26.74-3783)
GCS score	0.001			
Mild injury		4.1%	1	-
Moderate injury		6.1%	1.51	(0.20-11.33)
Severe injury		34.1%	12.14	(2.82-52.25)

Table 4-4 Showed final multivariate analysis for outcome correlation between Ages, Sex, CT scan finding, brain with midline shift, GCS score and outcome in patients head injury. Adjusted Odd ratio by Logistic Regression analysis

Variables		OR	95% C.I.		*P value*
			Lower	Upper	
CT scan finding	N+ other	1			<0.001
	SDH	13.588	1.241	148.806	
	EDH	14.326	1.302	157.681	
	ICH	3.887	0.328	46.011	
	SDH with MS	30.942	3.910	244.862	
	EDH with MS	11.232	1.136	111.014	
	SHI or DAI	201.513	16.77	2421.23	
GCS score	Mild	1			0.001
	Moderate	1.71	0.217	13.587	
	Severe	10.42	2.244	48.435	
Midline shift	No shift	1			0.731
	Shift up to 10mm	2.18	0.299	15.84	
	Shift greater tha10mm	2.34	0.265	20.68	
GCS * Midline shift	Moderate* Shift up to 10mm	0.446	0.008	25.83	0.988
	Moderate* Shift greater than 10mm	0.545	0.000	7.439	
	Severe * Shift up to 10mm	0.564	0.028	11.30	
	Severe * Shift greater than 10mm	140.95	0.000	1.367	

***** *SDH (Subdural heamatoma), EDH (Epidural heamatoma), ICH (Intracerebral heamatoma), SDH with MS (Subdural heamatoma with midline shift), EDH with MS (Epidural heamatoma with midline shift), SHI or DAI (Severe head injury or Diffuse Axonal injury)*

The results of univariate logistic analysis are shown in table 4-3. By univariate analysis, significant prognostic variables reported were degree of midline shift in CT scans, GCS score, CT scans finding lesion.

MULTIVARIATE ANALYSIS

After adjusting effect by multivariate analysis, significant predictors found were CT scan finding lesion and GCS score. Degree of midline shift, sex, ages had no significant variables to outcome (table 4-4). Brain with midline shift in patients head injury was no statistically correlation significant to outcome by multivariate analysis (Table 4-4). While CT scan finding combination with GCS score would to be the most important prognostic variable to significant predict outcome in patients head injury,

Although GCS score from this study was found to be significant when using both univariate and multivariate analysis (p =<0.001), it was not interaction significant variables when combined with degree of midline shift for predict clinical death following the logistic regression analysis (p = 0.988) (Table 4- 4).

Table 4-5 Univariate Analysis showed the relationship between SDH with MS and EDH with MS for predict clinical outcome

Variable	OR	95%CI	P-Value
SDH	1		
SDH with MS	3.42	2.92-18.64	<0.001
EDH	1		
EDH with MS	2.37	2.61-13.59	<0.001

Table 4-6 Multivariate Analysis showed the relationship between SDH with MS and EDH with MS for predict clinical outcome

Variable	OR	95%CI	P-Value
SDH	1		
SDH with MS	1.65	0.07-37.04	
EDH	1		
EDH with MS	0.79	0.08-60.10	
GCS score			**0.002**
Mild	1		
Moderate	1.73	0.22-13.48	0.003
Severe	10.39	2.27-47.61	0.001

The results of univariate logistic analysis are shown in table 4-5. By univariate analysis, the SDH with MS and EDH with MS were significant variables to predict outcome when compare SDH without MS and EDH without MS, but not significant variables to predict outcome by multivariate analysis (table 4-6).

CHAPTER V
DISCUSSION

Midline shift in CT scan occurred frequently in patients with head injury and is one of the most important factors in the prognosis poor outcome, especially patients severe head injury with Acute Subdural Hematoma (ASDH) and Epidural Hematoma (EDH). In our studied were to midline shift only, midline shift combine with Glasgow Coma Scale score (GCS) and midline shift combine with CT lesion finding to predict poor outcome (death rate) in patients head injury following both univariate and multivariate analysis. Which, midline shift is not only a result of heamatoma presence but also of concomitant brain contusion. The presence of an area of contused brain was seen in almost 80% of patients and it was revealed that the larger the contusion the less satisfactory the results of treatments, with large hemispheric contusion mortality was 85% and no satisfactory result were obtained. Brain contusion, especially when associated with subdural heamatoma, leads to a rapid development of brain swelling (22) and decreased cerebral blood flow (2), increased ischaemic damage, caused by the direct effect of blood overlying the cortex. Our finding found that midline shift in patients both with SDH and EDH is very important for mortality rate, in patients head injury with midline shift up to 10mm. increase 2 times mortality, while in patients with midline shift > 10mm. increase 4 times mortality compared no shift, especially patients with ASDH with shift increase 3 times mortality compared ASDH without shift, increase 30 times mortality compared normal patients, increase 10 times in patients EDH with shift compared normal patients.

Similar result were found by Kotwica and Brzezinski (52) showed 42% favourable outcomes and a mortality of 39% when the midline shift was below 1.5cm, 25% favourable outcomes and a mortality of 52% when this shift was from 1.5cm to 3cm and 8% favourable outcomes and a mortality of 76% whenever the shift exceeded 3 cm. Mean midline shift was 2.9mm. In those patients with a favourable recovery and 12.8 mm in those cases with poor outcome (53), but their studied only analyzed a group of patients severe head injury with ASDH. Alex B and

Valadka (2) concluded that midline shift after severe traumatic brain injury is associated with reduced cerebral metabolic rate of oxygen ($CMRo_2$) and shift was associated with higher intracranial pressure (ICP) regardless of whether or not subdural haematoma (SDH) is present in patients severe injury (Glasgow motor score ≤ 5), they study compared averaged cerebral metabolic parameters of patients with midline shift > 5 mm to patients with shift ≤ 5 mm on initial CT scan, the associated between degree of midline shift and patients mortality rate has not been as well defined. In 1991 Keith B, Quattrocchi et al. (56) studied to Quantification of midline shift as a predictor of poor outcome following head injury, they indicates that midline shift out of proportion to the extent of intracranial hemorrhage is a highly useful predictor of poor patient outcome following head injury, and found that patients with Diffuse Axonal Injury (DAI) without midline shift had poor outcome 100%, which was also Hiler M et al.(57) in 2006 concluded that Mean ICP values in patients with DAI cannot be predicted by using degree of midline shift in the Marshall CT scan classification(23).Therefore in recent years, the GCS score also has been the most widely used predictor of outcome(4, 6, 8).Attempts to improve the accuracy of predictions have considered additional factors such as age, intracranial pressure, nutrition, reflex eye movements, the type of intracranial lesion, the location of the lesion. The interaction of many effects has been studied using computer multifactorial analyses. It is obvious that outcome from acute brain injury depends on the complex interaction of a multitude of factors, and additional useful criteria are actively being sought.

Degree of midline shift from CT scan film can predict clinical outcome score (GOS) in patients with head injuries. We studied to traumatic brain injury represents rang of injury from mild head injury that many fully recovery to severe injury associated with outcome (death rate). We wished optimization of clinical data such as age, gender, mechanisms, Glasgow Coma Scale score (GCS) and CT scan finding must to important prognostic variable to predict outcome in patients traumatic brain injury. The result of this study following are showed correlation between clinical factor (GCS score, Age, Sex), CT scan finding, patients with midline shift and outcome (death rate), and showed degree of midline shift combine Glasgow Coma Scale (GCS) score in patients head injury to predict death rate.

Clinical data considerations after head injury

The following information was obtained from the hospital record: Age, gender, mechanism of injury, admission GCS score, GCS during hospitalization, pupillary reaction, eyes movement examination, focal neurological signs and CT scan finding, Which its are directly proportional to outcome in patients traumatic brain injury. Thus clinical data is important predictor variable to predict outcome in patients head injury (4, 8, 47, 49, 50).

In this study revealed that age > 60 years have death rate more than younger 1.71 times, male have death rate more than female 1.9 times, but its were no significance in determining the outcome following head injury after performing multivariate analysis(p= 0.60 and p = 0.27). This result is in also with many previous studies, which indicated that age, sex were a major role in the prognosis of head injury (9, 10, 20, 21). Bkk Gan et al. study showed that the elderly group, the mortality (55.4%) was slightly more than double that of the younger group (20.9%), Thus Bkk Gan (7) concluded that age must be considered an independent factor in outcome prediction in the elderly with moderate and severe traumatic brain injury, And J. Pelaez et al (51) found that the cause of death was due to the head injury in patients younger than 25 years was 83.3% compared in older than 65 years was 100%, Thus J. Pelaez concluded that to group patients according to initial CT scan findings and age allows to identify subsets of patients with bad outcome. But elderly patients do not seem to be a subset with poorer outcome in severe head injury. Shameran Slewa. Yaunan et al. (9) concluded that men’s levels of injury severity were greater than women’s despite the same admission criteria being applied to both sexes.

In this study found that elderly group (> 60years) was no statistic significant correlation with outcome (death rate) by multivariable analysis. (p = 0.61), and also sex in this study was no statistic significant with outcome (p = 0.27) by multiivariable analysis. Our study may data collected don’t enough and difference admission criteria being applied to both sex.

Therefore, The Traumatic Coma Data Bank analyzed patients with head injuries and identified 5 factors that correlated with a poor outcome, as follows (1) age older than 60 years, (2) initial GCS score of less than 5, (3) presence of a fixed dilated pupil, (4) prolonged hypotension or hypoxia early after injury, and (5) presence of a surgical intracranial mass lesion. Daniel L. et al. (50) concluded that factors that are independently predictive of outcome include age, GCS scores, Injuries Severity Score (ISS) and papillary reactivity. Among patients with GCS scores of 3 to 8, the factors that are most predictive of outcome seem to be those related to the severity of the head injury, whereas in patients with GCS scores of 9 to 15, the factors that correlate with outcome are those that relate to the severity of multi system injury.

6.1 Correlation between midline shift in patients brain injury and outcome

Different methods of classification of intracranial injury as seen on CT scan film have been utilized by different research in their study to predict prognosticate outcome following head injury. Some of the classifications were according to the anatomical site of the haematomas (37, 39). In our study used the midline shift, which was also Athiappon (47) and Toutant (40), which this we study found that patients with midline shift in death group of 50 cases mean shift was 7.05mm. And patients with midline shift in good outcome group of 167 cases mean shift was 3.25mm. In this study we found that degree of midline shifts were statistically significant as a determinant of outcome for univariable analysis (*p* value = 0.001). But was not statistically significant when combined with other more significant variables following the logistic regression analysis (*p* value = 0.738) as for the sex and ages.

Pillai SV et al. In 2003 concluded that the absence of the horizontal oculocephalic reflex, a poor motor score of GCS and the presence of midline shift on CT scan are the most important factors predicting poor outcome in patients with severe diffuse brain injury. In both the retrospective and the prospective groups, the treatment intensity was greater for those patients with greater midline shift, and effaced cisterns or ventricles or with a worse GCS score. Ideally, the validation of a prediction model should involve a large number of patients from different places and times. (51)

The importance of midline shift in predicting the outcome following severe diffuse brain injuries was also reported by Fearnside et al (39) found that midline shift was an important predictor of mortality along with intra-ventricular blood and cerebral edema. Wardslaw et al (58) reported that while the presence of SAH and the “overall appearance of the scan” (severe focal or diffuse injury as opposed to normal/ mild/ moderate injury) were very useful in predicting outcome, midline shift on CT scan did not have much significance.

This study found that the presence of midline shift especially with SDH was significant. In patients brain injury out of 121 patients without midline shift, 18(14.9%) cases died, 103 (85.1%)cases good outcome, Out of 58 cases in patients brain injury with midline shift up to 10mm group were 16 (27.6%) cases died, 42(72.4%) cases good outcome and mortality rate was 2.33 times with patients no shift group, While 38 patients brain injury with midline shift greater than 10mm group were 16(42.1%) cases died, 22(57.9%) cases good outcome, which found that mortality rate was 4.45 times with patients no shift group by univariable analysis (Table 4-3), but was not statistically significant when combined with other significant variables following the logistic regression analysis (p = 0.73)(Table 4-4), This results is also with studies by Narayan (43), the extent of midline shift did not add significantly to the prognostic capability of their model. Selladurai also noticed that the degree of midline shift did not prove to be of a predictive significance. (32)

6.2 Correlation between CT scan finding in patients brain injury and outcome

CT undoubtedly allows rapid diagnosis of hematomas and classification of intra cranial pathology. The number and distribution of CT findings was associated with differential outcome. This we study found that ether univariate or multivariate analysis showed statistically significant variables to outcome included ICH, EDH, SDH, IVH, SAH as well as midline shift. This study found that the presence of midline shift especially with SDH was significant. This meant that the outcome would be poorest if the midline shifts with SDH compared to other lesion in patients brain injury (30.42 time), (p = 0.001) except Diffuse brain injury and severe head injury 318.09 times for outcome. (Table 4-3) Reviews by V. Juran et al. showed that SDH with Midline shift treatment was possible in certain cases. It could be successful in smaller haematomas in patients in a good clinical condition. The location and volume of SDH was not included as one of the variable in this study, thus the volume of SDH present no statistical correlation was to predict outcome. Kuday concluded also that no statistical correlation was found between the locations of the haematoma with the outcome (44). The most frequent location of SDH in this study was in the Frontotemporal region. Thus we concluded that CT scan finding was the most important prognostic variable to predict outcome. Also to another study in which CT scan findings of SDH as well as GCS score were found to be the most important prognostic variable.

However in this study the mortality rate was found to be significantly greater in patients with lesion with Midline shift. According to a review by Fearnside (39) this type of hemorrhage was generally associated with poor outcome. Yamaura noted a higher mortality when SDH was associated with the presence of parenchyma lesion (45). Poorer outcome was observed in this study when SDH were associated with Midline shift (38.33%) than without Midline shift (13.04%), while poorer outcome when EDH was associated with Midline shift (17.39%) than without Midline shift (7.69%). The higher morbidity or mortality in SDH compared to EDH is due to the consequence of a mass effect and that SDH are frequently associated with direct brain injury (34). While 24(92.3%) out of 26 patients with hemorrhagic contusion had a good outcome, which is quite similar with analysis by Kunishio where 66.7% of their patients with contusion had a good outcome (46).

6.3 Correlation between GCS score and outcome

GCS score was found to have to predictive factor of outcome following statistical analysis. The GCS score was well correlated with outcome in which higher mortality was associated with a lower GCS score. However, it was observed that the GCS score only became statistically significant by logistic regression when analyzed in combination with other variable, i.e. papillary reflex and types of CT scan findings. (34)

GCS score has been widely adopted and used in classification of severity in head injury patients. Teasdale and Jennett have demonstrated the predictive power of GCS to outcome, which was further demonstrated in the Traumatic Coma Data Bank analysis (27). GCS score has been found to be a good indicator of outcome in many other studies including a local study by Selladurai (32). As well as others showed that over 95% of patients with a score of 4 or less are likely to have a poor outcome compared with those with a score of 8 or a more. In this study found that out of 47 patients with GCS of 15 (mild injury), 2 (4.1%) cases died, 47 (95.9%) cases good outcome. 31 patients with GCS of 13-14 (moderate injury), 2(6.1%) cases died, 31 (93.9%) cases good outcome, while 135 patients with GCS score of ≤12 (severe injury), 46(34.1%)cases died, 89(65.9%) cases good outcome, and found that mortality rate in patients moderate injury was 0.703 time of patients mild injury. (P value = 0.000), while mortality rate in patients severe injury was 12.14 times of patients mild injury. (P value = 0.001) Table 4-4

In this study found that GCS score was statistically to be significant to outcome using both univariate and multivariate analysis.

Lipper (33) in his study showed a significant number of patients had a GCS score between 5 and 7 and that GCS was not very helpful in predicting outcome. Pupillary reflex or response to light was found statistically to be significant to outcome using both univariate and multivariate analysis in A.A. Azian (34). As well as in other publications (35, 36), Kido et al also showed a significant relationship existed between lesion size and GCS an admission. Larger lesion enabled prediction of lower GCS scores as well as in the present this study (37).

6.4 Correlation between GCS score and midline shift

In this study found that GCS score was statistic significant correlation with midline shift in patients head injury (p = 0.047). Although GCS score from this study was found to be significant when using both univariate and multivariate analysis (p = <0.001), but it was not interaction significant variables when combined with degree of midline shift for predict clinical death following the logistic regression analysis (p = 0.988) except patients severe injury (GCS < 12) with brain shift greater than 10 mm were increase death rate 140.95 time compare patients mild injury (GCS = 15) without midline shift.

In multiple analysis for outcome showed correlation between CT scan finding, brain with midline shift, GCS score and outcome in patients head injury adjusted Odd ratio by Logistic Regression analysis found that mortality rate in patients only SDH was 23.0 times compare patients normal injury (p =<.001), while mortality rate increase 27.6 times in patients SDH with midline shift, mortality rate in patients head injury with EDH was 10.5 times, while patients EDH with midline shift was 10.5 times compare patients normal injury. Thus we concluded that brain with midline shift in patients head injury were statistically correlation significant to outcome by univariate analysis (p =<.001) (Table 4-3).But brain with midline shift in patients head injury were no statistically correlation significant to outcome by multivariate analysis (p =0.738) (Table 4-4). While CT scan finding combination with GCS score would to be the most important prognostic variable to significant predict outcome by both univariate and multivariate analysis

In this study it was observed that the likelihood of becoming death after head injury is increase if lesion with midline shift greater than without midline shift, so we conclude that lesion with midline shift in patients head injury could be used as a prognostic factor to predict death or poor outcome. Despite the most of the mortality rate and poor outcome depends not only degree of midline shift with lower GCS score but on application on the combination of clinical factors, CT feature and associated injury. Which E.Gaitur point out that outcome depend not only on the combination of clinical and CT feature, but on application of accurate criteria for conservative or surgical treatment, diagnostic possibilities, intensive therapy methods and surgical management optimization as well as.

CHAPTER VI

CONCLUSION

Computed tomographic scanning in both developed and developing countries is at the moment indispensable in the diagnostic evaluation of patients suffering from significant head injuries in an emergency situation. The advantages of CT include widespread availability, rapid imaging time, relatively low cost and safety. Its prognostic ability has been widely studied and is generally significant and accepted. However Narayan (43) and Lobato (42) reported that a single CT scanning does not allow an absolute prognosis and is inferior to prediction made using clinical parameters. They pointed out that serial CT scanning is needed for management and prognostication. Lee (49), as in other studies (39, 47), found that a correlation exists between the CT scan appearance and the clinical status indicated by the GCS score.

In this study conclusion that the Degree of Midline shift in traumatic head injury was significantly associated with outcome (death rate) by univariate analysis (*P value = .001*), but no significantly associated with outcome by multivariate analysis (*P value = 0.731).* In patient's lower GCS score was significant related with outcome both univariate and multivariate analysis (*P value =<.001*).It is not obvious that increase mortality from the interaction of degree of midline shift and GCS score by multivariate analysis (P value = 0.988) except patients severe injury (GCS score < 12) combination with patients with brain shift greater than 10mm. And CT lesion finding was significant related with poor outcome (death) both univariate and multivariate analysis (*P value=< .001*). The mortality rate was found to be significantly greater in patients with lesion with Midline shift; poorer outcome was observed in this study when lesion was associated with Midline shift than without Midline shift. And lower GCS score combination with CT scan lesion was poorest outcome especially severe head injury and patients SDH with midline shift by multivariate analysis.

Suggestion for further study in prospective study and collect more sample size is suggested because the prospective study can give more patients information.

REFERENCES

1. http://www.police.go.th/traff_main.htm 2004;
2. Alex B, MD, Shankar P, et al. Midline shift after severe head injury: Pathophysiologic Implication. J trauma 2000; 49: 1-10.
3. Jeffrey Englander, MD, et al. The association of early computed tomography scan findings and ambulation, self-care, and supervision needs at rehabilitation discharge and at 1 year after traumatic brain injury. Arch Phys Med Rehabil.2003 Feb; 84.
4. Chesnut RM, Marshall LF, Klauber MR, et al. Division of Neurological Surgery UMC. The role of secondary brain injury in determining outcome from severe head injury. J Trauma 1993; 34(2): 216-22.
5. Choi SC, Muizelaar JP, Barnes TY, et al. Prediction tree for severely head injured patients. J Neurosurg 1991; 75:251-5.
6. Teasdale G.M. and SIGN Guideline Development Group. Early management of patients with a head injury. Scottish Intercollegiate Guidelines Network 2000.
7. Bk Gan, JHG Lim, IHB Ng. Outcome of Moderate and Severe Traumatic Brain Injury Amongst the Elderly in Singapore. Ann Acad Med Singapore 2004; 33: 63-7.
8. Jun-ichi Ono, Akira Yamaura, Motoo Kubota. Outcome prediction in severe head injury: analyses of clinical prognostic factors. Neuroscience (2001); 8 (2): 120-123.
9. Shameran Slewa-Yaunan, PhD, Alisa M, Green, BA, Ian J.Sex Differences in Injury Severity and Outcome Measures After Traumatic Brain Injury.Arch Phys Med Rehabil, March 2004; 85: 376-9.
10. Tobias Nagurney, Pierre Borczuk,Stephen H. Thomas. Elderly patients with closed head trauma after a fall: Mechanisms and outcomes. Emergency Medicine, March 1998; 16: No. 5. p. 709-713.

11. Cooper, P.R., Skull fracture and traumatic cerebral spinal fluid fistulas, in Head injury, Cooper, P.R., Ed., Williams & Wilkins, Baltimore, 1993; p. 115.
12. Mendelow, A.D., Teasdale, G., Jennett, B., et al., Risk of intracranial heamatoma in head injury to adults, BMJ, 287,1173,1983.
13. Roberts, G.W., Leigh, P.N., and Weinberger, D.R., Neuropsychiatric Disorders, Wolfe, London, 1993; p.5.1.
14. Graham, D.I., Gennarelli, T.A., and Melntos, T.K., Trauma, in Greenfield's Neuropathology, 7th ed., Graham, D.I. and Lantos, P.L., Eds., Arnold, London,2002, p. 823.
15. Graham, D.I.,Neuropathology of head injury, in Neurotrauma, Narayn, R.K., Wilberger, J.E., and Povlishock, J.T.,Eds., W.B. Saunders, Philadelphia, 1996; p.43.
16. Robert P. Granacher. Traumatic brain injury: methods for clinical and forensic neuropsychiatric assessment. 2003; p.10.
17. DiMaio, V.J. and DiMaio, D., Forensic Pathology, 2nd ed., CRC Press, Boca Raton, FL, 2001, p. 147.
18. Bullock, R. and Teasdale, G., Surgical management of traumatic intracranial hematomas, in Handbook of Clinical Neurology, Vol. 15, Head Injury, Braackman R., Ed., Elsevier, Amsterdam, 1990, p. 249.
19. Adams, J.H., Mitchell, D.E., Graham, D.I., et al., Diffuse brain damage of immediate impact type, Brain, 1977;100: 489.
20. Adams, J.H., Graham, D.I., Murray, L.S., et al., Diffuse axonal injury due to nonmissile head injury in humans: an analysis of 45 eases, Ann. Neurol., 1982; 12: 557.
21. Adams, J.H., Doyle, D., Graham, D.I., et al., Diffuse axonal injury in head injuries caused by a fall, Lancet.1984; 2:1420.
22. Lobato, R.D., Sarabia, R., Cordobes, F., et al., Posttraumatic cerebral hemispheric Swelling: analysis of 55 cases studied with computerized tomography. J. Neurosurg., 1988; 68: 417.

23. Marshall, L.F., S.B., Klauber, M.R., et al., A new classification of head injury based on computerized tomography, J. Neurosurg. 1991; 75: S14,.

24. L.I.Z. Gentry, J.C. Godersky, B. Thompson and V.D. Dunn, Prospective comparative study of intermediate-field MR and CT in the evaluation of closed head trauma. *AJNR* **9** (1988), pp. 91–100.

25. Kase CS, Williams JP, Wyatt DA, Mohr JP.Lobar intracerebral hematomas: clinical and CT analysis of 22 cases. Neurology.1982; 32:1146-1150.

26. Massaro F, Lanotte M, Faccani G, Triolo C. one hundred and twenty- seven cases of acute subdural haematoma operated on: correlation between CT scan finding and outcome. Acta Neurochir(Wein).1996;138:185-191.

27. Aldrich EF, Eisenberg HM. Saydjari C. et al. Predictors of mortality in severely head-injuried patients with civilian gunshot wound: are port from the NIH Traumatic Coma Data Bank. Surg Neeurol. 1992; 38: 418- 423.

28. Orrison WW, Gentry LR, Stimac GK, Tarrel RM,Espinosa MC, Cobb LC. Blinded comparision of cranial CT and MR in closed head injury evaluation: AJNR Am J Neuroradiol.1994 Feb;15(2):351-6.

29. Jinn-Rung, Kuo MD et al. Coagulopathy as a parameter to predict the outcome in head injury patient analysis of 61 cases. J of clinical Neuroscience 2004.

30. Lobato RD, Rivas JJ, Gomez PA, et al. Head injured patients who talk and deteriorate into coma: analysis of 211 cases studied with computederized tomography. J Neurosurg.1991; 75:256-261.

31. Jennett B, Bond M. Assessment of outcome after severe brain damage. A practical Scale, Lancet 1975; 1:480-484.

32. Selladurai BM, Jayakumar R, Tan YY, Low HC (2002) outcome prediction in early management of severe head injury: an experience in Malaysia. Br J Neurosurg 6: 2002 oct; 16(5): 487-93.

33. Lipper KH, Kishore PRS, Emas GG, Domingues da Silva AA, Choi SC, Becker DP (1985) Computed tomography in the prediction of outcome in head injury. Am J Roent 144: 483-486.

34. A. A. Azian, A. A. Nurulazman,I. L. Shuaib, et al. Computed Tomography of the Brain in Predicting Outcome of Traumatic Intracranial Haemorrhage in Malaysian Patients.Acta Neurochir (Wien)(2001) 143: 711-720.

35. Braakman R, Gelpke GJ, Habbema JDF, Maas AIR, Minderhoud JM Systemic selection of prognostic features in patients with severe head injury. Neurosurgery 6:362-370

36. Jennett B (1976) Assessment of the severity of head injury. J Neurosurg Psychiatry 39: 647-655

37. Kido DK, Cox C, Hamill RW, Rothernberg BM, Woolf PD. Traumatic brain injuries: predictive usefulness of CT Radiology 1992; 82: 777-781

38. Valadka AB, Narayan RK (1996) Emergency room management of the head-injuried patient. In: Narayan RK, Wilberger Jr JE, Povlishock JT (eds) Neurotrauma. McGraw-Hill, New York, pp 119-135

39. Fearnside MR, Cook RJ, McDougall P, McNeil RJ. The westmead head injury project outcome in severe head injury. A comparative analysis of pre-hospital, clinical and CT variables. Br J Neurosurg 1993; 7: 267-279

40. Toutant SM, Klauber MR, Marshall LF et al. Absent or compressed basal cisterns on first CT scan: ominous predictors of outcome in severe head injury. J Neurosurg 1984; 2: 323-331

41. Gennarelli TA, Spielman GM, Langfitt TW, et al. Influence of the type of intracranial lesion on outcome from severe head injury. A multicenter study using new classification system. J Neurosurg 1982; 56: 26-32

42. Lobato RD, Cordobes F, Rivas JJ et al. Outcome from severe head injury related to the type of intracranial lesion. A computed tomography study. J Neurosurg 1983; 12: 161-168

43. Narayan RK, Greenberg RP, Miller JD Improved confidence of outcome prediction in severe head injury. A comparative analysis of the clinical examination, multimodality evoked potentials, CT scanning and intracranial pressure. Neurosurgery 1981; 54: 751-762

44. Kuday C, Uzan M, Hanci M. Statistical analysis of the factors affecting the outcome of extradural heamatomas: 115 cases. Acta Neurochir (Wein) 1994; 131:203-206

45. Yamaura A, Nakamura T, Ono J, Ariga N, Horie T, Shinohara Y, Isobe K, Kokubo Y, Makino H. Prognostic value of CT in head injuries. Neurol Med Chir (Tokyo) 1981; 21:685-696

46. Kunishio K, Shinohara C, Tokunaga K, Matsushisa T, Moriyama E, Norikane Y, Tanaka R. Analysis of long-term social rehabilitation of brain contusion. No Shinkei Geka 1992; 20:959-963

47. Athiappan S, Muthukumar N, Srinivasan US. Influence of basal cisterns, midline shift and pathology on outcome in head injury. Ann Acad Med Singapore 1993; 22(3): 452-455

48. L. Song, T. Xu, Y Zhang, Z.R. He, B.Y. Rong, et al. Bilateral traumatic intracranial haematoma. J Neurosurg 1997.

49. LeeTT, Aldana PR, Kirton OC, Green BA. Follow-up computerized tomography (CT) scan in moderate and severe head injuries: correlation with Glasgow coma scores (GCS), and complication rate. Acta Neurochir (Wein) 1997; 19:1042-1047

50. Dent, Daniel L. Croce, Martin A, et al. Prognostic Factors after Acute Subdural Heamatoma. J Trauma- InjuryInfection & Critical Care.1995; 39 (1): 36-43

51. Pillai SV, Kolluri VR, Praharaj SS. Outcome prediction model for severe diffuse brain injuries: Development and evaluation. J Neurisurg 2003; 51(3): 345-349

52. Kotwica Z, Brzezinski J. Acute subdural haematoma in aduts: an analysis of outcome in comatose patients. Acta Neurochir 1993; 121: 95-99

53. Yanaka K, Kamezaki T, Yamada T. et al. Acute subdural haematoma: prediction of outcome with linear discriminant function. Neurol Med Chir(Tokyo)1993; 33: 552-558

54. Cook RJ, Dorsch NW, Fearnside MR, Chaseling R. Outcome prediction in extradural haematomas. Acta Neurochir (Wien).1988; 95:90-94.

55. Dent DL, Croce MA, Menke PG, et al. Prognostic factors after acute subdural hematoma. J Trauma.1995; 39:36-42.
56. Keith B. Quattocchi, MD, et al. Quantification of midline shift as a predictor of poor outcome following head injury. Surg Neurol. 1991; 35: 183-8
57. Hiler M, Czosnyka M, Hutchinson P, et al. Predictive value of initial computerized tomography scan, intracranial pressure, and state of autoregulation in patients with traumatic brain injury.2006 May; 104(5): 731-7
58. Wardlaw J M, Easton V J, Statham P. Which CT features help predict outcome after head injury. J Neurol . 2002; 72: 188-192

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