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Clinical Article

Post-Traumatic Cerebral Infarction : Outcome after Decompressive Hemicraniectomy for the Treatment of Traumatic Brain Injury

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Objective : Posttraumatic cerebral infarction (PTCI), an infarction in well-defined arterial distributions after head trauma, is a known complication in patients with severe head trauma. The primary aims of this study were to evaluate the clinical and radiographic characteristics of PTCI, and to assess the effect on outcome of decompressive hemicraniectomy (DHC) in patients with PTCI.

Methods : We present a retrospective analysis of 20 patients with PTCI who were treated between January 2003 and August 2005. Twelve patients among them showed malignant PTCI, which is defined as PTCI including the territory of Middle Cerebral Artery (MCA). Medical records and radiologic imaging studies of patients were reviewed.

Results : Infarction of posterior cerebral artery distribution was the most common site of PTCI. Fourteen patients underwent DHC an average of 16 hours after trauma. The overall mortality rate was 75%. Glasgow outcome scale (GOS) of survivors showed that one patient was remained in a persistent vegetative state, two patients were severely disabled and only two patients were moderately disabled at the time of discharge. Despite aggressive treatments, all patients with malignant PTCI had died. Malignant PTCI was the indicator of poor clinical outcome. Furthermore, Glasgow coma scale (GCS) at the admission was the most valuable prognostic factor. Significant correlation was observed between a GCS less than 5 on admission and high mortality ($p<0.05$).

Conclusion : In patients who developed non-malignant PTCI and GCS higher than 5 after head injury, early DHC and duroplasty should be considered, before occurrence of irreversible ischemic brain damage. High mortality rate was observed in patients with malignant PTCI or PTCI with a GCS of 3-5 at the admission. A large prospective randomized controlled study will be required to justify for aggressive treatments including DHC and medical treatment in these patients.

Key Words : Brain trauma · Cerebral infarction · Decompressive craniectomy.

INTRODUTION

Posttraumatic cerebral infarction (PTCI) is a well-known complication of traumatic brain injury, with a frequency ranging from 1.9% to 10.4%[17,24,26,27]. PTCI has been introduced as an indicator of poor clinical outcome, and is associated with a high mortality rate, despite appropriate medical and surgical interventions. Infarction of the occipital lobe after compression of the Posterior Cerebral Artery (PCA) against the rigid edge of the tentorium by the herniating medial temporal lobe is a well-recognized mechanism leading to PTCI[10,17,22]. Various mechanisms have been suspected for this complication, including cerebral vasospasm, vascular injury, embolization, and systemic hypoperfusion[8,10,13,17,19,21,22,24,26,27]. Malignant middle cerebral artery (MCA) infarction in a cerebral infarction in MCA territory was first described by Hacke[4]. The term "malignant" MCA infarction has been used to describe impending cerebral herniation due to fatal brain swelling and high mortality in patients with ischemic stroke. Similarly, we defined a malignant PTCI as a PTCI subtype including the territory of MCA. Until now, there have been few clinical and radiological data defining early predictors of development of PTCI. Early identification of patients who are at particular risk for PTCI would be extremely helpful. For determination of clinical and radiographic parameters that may aid in identification of PTCI patients who are at high risk for fatal brain swelling, we conducted an evaluation of early clinical, laboratory, and radiological characteristics associated with PTCI including malignant PTCI in consecutive PTCI patients admitted to our institution.

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MATERIALS AND METHODS

Patient population

Low attenuated lesions in well-defined arterial distribution on brain computed tomography (CT) after head trauma were defined as PTCI (Fig. 1). In particular, PTCI including MCA territory was defined as malignant PTCI. We conducted a retrospective review of 830 patients with traumatic brain injuries, who underwent treatment between February 2003 and August 2005 at our hospital. Twenty patients (2.4 %), in whom distinctly marginated areas of low attenuation in a typical vascular distribution were detected on unenhanced computed tomography (CT) scans, were selected for this study. The study included 16 men and four women, and the mean age was 44.95 years (range from 2 to 80 years). Twelve patients among them showed PTCI including MCA territory, so-called malignant PTCI.

The following patient data were retrospectively reviewed: age, date of the accident, mechanism of the injury, duration from accident to recognition of cerebral infarction by brain CT, and duration between accident and surgery; neurological status at the time of admission and prior to decompression; occurrence of hypotension (systolic arterial pressure less than 90 mmHg or/and diastolic pressure less than 40 mmHg for more than 10 minutes); laboratory findings from arterial and venous blood, occurrence of an episode of hypoxemia (arterial pressure of oxygen, PaO2 less than 60 mmHg); initial and follow-up CT and CT angiography (CTA) findings. The midline shift on the CT scan was defined as the absolute distance (mm) that the septum pellucidum of the brain was displaced away from the midline, which was determined as an average by calculating the distance between both inner tables inside the skull. Outcome was determined according to the glasgow outcome scale (GOS) at the time of hospital discharge : 1 (death), 2 (persistent vegetative state), 3 (severe disability), 4 (moderate disability), and 5 (good recovery). Patients with favorable outcomes were defined as those with GOS scores of 5 or 4, whereas an unfavorable outcome was defined as a GOS scores of 3, 2, or 1.

Management of intracranial pressure

All patients received the usual protection of the airways, endotracheal tube placement and venous access with infusion of volume for hemodynamic stabilization. Neurological assessment was performed using the Glasgow Coma Scale (GCS) score, pupillary size and reaction, as well as other brain stem reflexes and limb movements. All patients received intravenous treatment with mannitol for reduction of intracranial pressure (ICP). It was administered at a dose of 0.5 g/kg as an intravenous bolus injection, repeated at 4-8 hours, and guided by a target serum osmolality of 310-320 mOsm/L. Fourteen patients underwent decompressive hemicraniectomy (DHC). Patients with an initial and persistent GCS score of 3 and/or bilaterally fixed and dilated pupils did not undergo surgical decompression. Urgent surgery was performed upon recognition of clinical evidence of herniation and radiological signs of raised ICP on CT scan. A large frontotemporoparietal craniectomy was performed on the side ipsilateral to the PTCI, and the temporal squama was rongered out until the floor of the middle cranial fossa was exposed. The dura was incised in star fashion to allow for outward expansion of the brain and dura is closed very loosely with Neuro-Patch Synthetic Dura Substitute (Medtronic) or pericranium. The bone has been stored under sterile conditions at -80C. Postoperative barbiturate coma therapy was applied in five patients. It was administered at a loading dose of 5 mg/kg IV over 10 minutes with continuous infusion of 5 mg/kg/hr for 24 hours. After 24 hours, we reduced infusion to 2.5 mg/kg/hr.

Statistical analysis

The SPSS program for Windows V12.0 (SPSS, Chicago, IL, USA), including Chi square test, Fisher's exact test, and Mann-Whitney U test was used for analysis of the independent contribution of predictive factors to outcome. For all analyses, a p-value of <0.05 was considered statistically significant.

RESULTS

Characteristics of patients

Mechanisms of injury included motor vehicle accidents in nine patients, falls in ten patients, and violence in one patient. Table 1 shows the demographic data, the mechanism of injury, location of infarction, and time interval.

Brain lesions and midline shift

Based on the brain CT, the relationships between infarction and brain lesion for 20 patients with PTCI or malignant PTCI were evaluated. Radiological signs consistent with raised ICP were observed on the brain CT scans, either on the initial scan or on the scan obtained when the patient showed deterioration. Details of CT scan findings are given in Table 1. In nonmalignant PTCI patients, the most common brain lesions were acute subdural hematoma (acute SDH) (Fig. 2A) in three patients

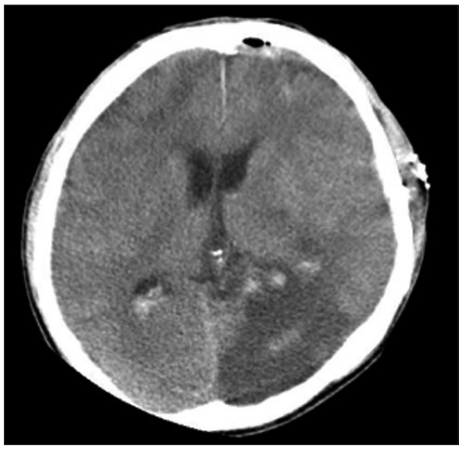

Fig. 1. Computerized tomography showing a post-traumatic cerebral infarction on the left posterior cerebral artery distribution.

Table 1. Demographics and clinical data

Variables	Malignant PTCI (n=12)	Nonmalignant PTCI (n=8)	*p*-value
Sex			0.018
Male	11 (91.7%)	3 (37.5%)	
Female	1 (8.3%)	5 (62.5%)	
Age	37.6±24.4	56±15.9	0.077
Mechanism			
Traffic accidence	6 (50%)	3 (37.5%)	0.670
Fall	5 (41.7%)	5 (62.5%)	0.650
Violence	1 (8.3%)	0	1.000
Brain lesions			
Acute EDH	1 (8.3%)	3 (37.5%)	0.109
Acute SDH	8 (66.7%)	3 (37.5%)	0.642
T-SAH	2 (16.7%)	0 (0%)	0.642
T-ICH	1 (8.3%)	2 (25%)	0.356
Midline shift (mm)	16±6.2	15.5±7.0	0.903
Duration between accident with infarction	28.2±43.7	14.75±6.3	0.316
Initial GCS			
5≤	9 (75%)	4 (50%)	0.356
5>, 10<	3 (25%)	3 (37.5%)	0.642
10≥	0	1 (12.5%)	0.400
Clinical outcome			0.147
Favorable	0	2 (25%)	
Unfavorable	12 (100%)	6 (75%)	
Mortality	12 (100%)	3 (37.5%)	0.001

Acute EDH : acute epidural hematoma, Acute SDH : acute subdural hematoma, T-SAH : traumatic subarachnoid hemorrhage, T-ICH : traumatic intracerebral hematoma, GCS : Glasgow Coma Scale

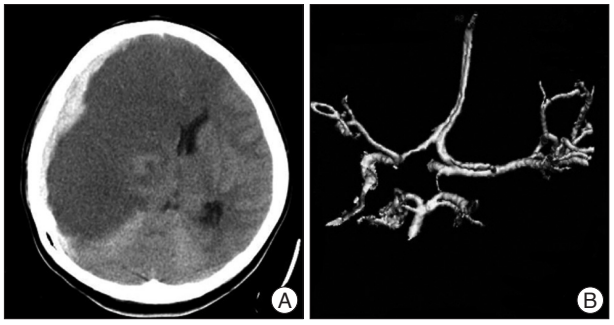

Fig. 2. A : Computerized tomography (CT) showing an acute subdural hematoma with a milti-territory infarction including middle cerebral artery distribution (malignant post-traumatic cerebral infarction), B : CT angiography showing the vascular distortions of both anterior cerebral arteries and compression of the right middle cerebral artery (arrow) caused by a mass effect.

(37.5%) and acute epidural hematoma (acute EDH) (Fig. 4) in three patients (37.5%). Traumatic intracerebral hematoma (T-ICH) (Fig. 3) occurred in two patients (25%) In patients with malignant PTCI, brain lesions were as follows : Acute SDH in eight (66.7%), traumatic subarachnoid hemorrhage (T-SAH) in two (16.7%), T-ICH in one (8.3%), and acute EDH in one patient (8.3%), respectively. This study showed that any brain lesion could result in PTCI or malignant PTCI; however acute SDH was the most common brain lesions.

Midline shifts resulting from space-occupying lesions were evaluated (Table 1). Eight patients were excluded because they had no midline shift on brain CT, due to diffuse brain swelling or bilateral hematoma. Mean midline shifts in 4 patients with nonmalignant PTCI were 15.5 mm (range from 9 to 23 mm), and 16 mm (range from 5 to 20 mm) in patients with malignant PTCI. Preoperative CT scan reveals right sided acute epidural hematoma and a initial GCS score of 5 (Fig. 4A). He had a midline shift of 18 mm on brain CT. Postoperative CT showing a multi-territory infarction including middle cerebral artery distribution (Fig. 4B), he died despite any modality of treatment. Representative cases of acute SDH show a midline shift of 13 mm on brain CT (Fig. 5A). The patient was admitted to our institution with an initial GCS score of 7. The following CT performed after craniectomy and evacuation of hematoma shows PTCI in the posterior cerebral artery territory (Fig. 5B). She was moderately disabled at the time of discharge.

Site of infarction

Sites of infarction territory were evaluated on the basis of brain CT (Table 2). Infarction was most common in the PCA distribution in 16 patients (80%). The most common site of infarction was the whole territory in 8 patients (40%), followed by PCA distribution in 7 patients (35%).

Duration between accident and surgery/between accident and infarction

Emergent surgery was performed in 14 of 20 patients with PTCI (70%). The interval between the time of the accident and surgery varied from three hours to four days, with most occurring within the first 24-hour period. Mean duration between accident and surgery was 15.6 hours (range from 3 to 94 hours). Mean duration between accident and the time of infarction was 22.8 hours (range from 3 to 136 hours). Among them, seven of 12 patients with malignant PTCI underwent DHC. Due to our small sample size and heterogeneous group, we could not demonstrate a correlation between latency onset of symptoms and surgical intervention and outcome.

Laboratory findings

Laboratory findings through arterial/venous blood and body temperature were reviewed (Table 3). Laboratory findings of

venous blood included white blood cell (WBC) count, platelet count, hemoglobin, prothrombin time (PT), and activated partial thromboplastin time (aPTT). Laboratory findings of arterial blood included potential of hydrogen (pH), arterial pressure of oxygen (PaO2), and arterial pressure of carbon dioxide (PaCO2). Low systolic BP and hypoxemia had not been in this study. This study showed increased WBC count and serum glucose level. However, traumatic stress itself could induce an increase in these two values; therefore, these findings could not be used for characterization of PTCI.

Outcomes

The mean GCS on admission was from 3 to 11. Thirteen patients (65%) were admitted to our institution with a GCS score of 3-5: six patients (30%) had a GCS score of 6-9, and one (5%) had GCS scores over 10. GOS results showed that 15 patients died, one patient was remained in a persistent vegetative state, two patients were severely disabled and two patients were moderately disabled at the time of discharge. The overall mortality rate was 75% (15 of 20 patients). All patients with malignant PTCI died despite any modality of treatment. Furthermore, the GCS score on admission was an important factor for prediction of survival in our series. The mortality rate of patients with malignant PTCI or PTCI admitted with a GCS of 3-5 was significantly high; $p<0.05$ between the two GCS groups (Table 4). Of the six patients with a GCS score of more than 5, four patients survived; of the 14 patients with a GCS score of less than 5, 13 patients died.

DISCUSSION

Traumatic brain swelling may lead to refractory ICP and subsequent brain death. Despite the availability of CT scan, advances in monitoring and intensive care management, mortality and morbidity of patients with PTCI remains high. In 1920, Meyer[15] first reported on occipital lobe infarction caused by compression of the posterior cerebral artery in a case of tentorial herniation. Sato et al.[22] reported that the incidence of occipital lobe infarction was 9% among patients with transtentorial herniation on brain CT. Gross mechanical shift of the brain and herniation across the falx and/or tentorium accounted for most cases of infarction, ranging from 81% to 88%[17,24]. In our study, PTCI was a result of a focal mass effect and/or gross mechanical displacement of the brain, producing transfalcine or transtentorial herniation in all patients. In this study, infarction at the territory of PCA was the most common, consistent with the results of Mirvis et al.[17,26]. Subfalcian herniation of the cingulate gyrus results in compression of one or both ACA or the callosomarginal artery and its branches[12,21]. Compression of these arteries may produce infarctions in the region of the paracentral lobule or superior frontal gyrus and adjacent cingulate gyrus. Infarction in the proximal distribution of the ACA was observed in twelve of our series of 20 patients, including ten patients

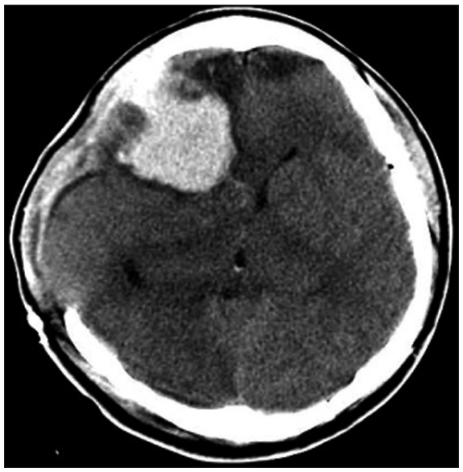

Fig. 3. Computerized tomography showing a traumatic intracerebral hematoma with a post-traumatic cerebral infarction on the right posterior cerebral artery distribution.

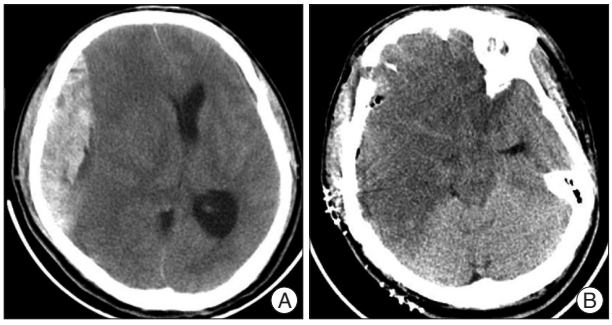

Fig. 4. A : Computerized tomography (CT) showing an acute epidural hematoma with a midline shift, B : postoperative CT showing a multi-territory infarction including middle cerebral artery distribution.

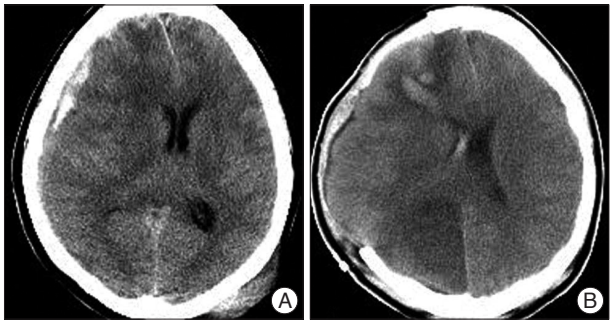

Fig. 5. A : Computerized tomography (CT) reveals right sided acute subdural hematoma with a midline shift, B : The following CT performed after craniectomy and evacuation of blood shows PTCI in the posterior cerebral artery territory.

Table 2. Sites of infarction on brain CT

Vascular territory	No	Mortality
PCA	7 (35%)	3 (42.9%)
MCA+ACA	3 (15%)	3 (100%)
MCA+PCA	1 (5%)	1 (100%)
ACA	1 (5%)	0 (0%)
Whole territory infarction	8 (40%)	8 (100%)

ACA : anterior cerebral artery, PCA : posterior cerebral artery, MCA : middle cerebral artery

Table 3. Laboratory data

Variables	Malignent PTCI (n=12)	Nonmalignant PTCI (n=8)	*p*-value
Venous			
PT (INR)	13.6±2.3	13.85±2.1	0.838
APTT (sec)	37.2±7.6	32.3±4.5	0.119
WBC (/mm^3)	19,091.7±7,974.9	14,650±3,890.3	0.163
Platelet(10^3/mm^3)	203.2±62.7	142.4±56.4	0.040
Hemoglobin (g/dL)	12.2±1.9	11.0±2.3	0.230
Glucose (mg/dL)	244.2±96.6	230.1±45.7	0.708
Arterial			
pH	7.377±0.9	7.4±0.1	0.615
PaO_2 (mmHg)	161.2±66.3	116.5±40.6	0.107
$PaCO_2$ (mmHg)	34.3±10.6	31.2±9.4	0.511
Body temperature (°C)	36.65±0.8	36.29±0.5	0.294

PT : prothrombin time, APTT : activated partial thromboplastin time, pH : potential of hydrogen, PaO_2 : arterial pressure of oxygen, $PaCO_2$: arterial pressure of carbon dioxide, WBC : white blood cell

Table 4. Mortality according to GCS on admission

GCS	>5	≤5	*p*-value
Mortality (+)	3 (20%)	12 (80%)	0.0138
Mortality (-)	4 (80%)	1 (20%)	

GCS : Glasgow Coma Scale

with bilateral ACA infarction. In our study, all cases of bilateral ACA infarction developed in patients with malignant PTCI, resulting in 100 % mortality.

MCA territory infarction occurs due to a gross mass effect and herniation or severe brain swelling/edema[17,24,26]. Potential contributing factors include stretching and attenuation of the MCA, increased ICP, and a direct pressure effect from an extraaxial hematoma[17,24,28]. In our study, all patients of MCA territory infarction were associated with infarct of additional territories, and died. Similarly, a gross mass effect and herniation can produce stretching and attenuation of small perforating arteries, such as lenticulostriate, thalamoperforators, and brain stem perforating arteries, resulting in infarction of their vascular distributions[17,24]. Lenticulostriate/thalamoperforators territory infarctions produced poor clinical outcomes, because their occurrence was associated with MCA territory infarction. Other mechanisms have been reported in the previous literature. Infarction of cortical and subcortical regions can result from a direct mass effect by an overlying hematoma, limiting arterial flow, or disturbance of venous drainage. Intracranial arterial vasospasm in association with T-SAH is another mechanism of PTCI. Posttraumatic angiographic vasospasm occurs with an incidence ranging from 2% to 41%[19]. We did not perform angiography; therefore, we cannot determine the role of vasospasm in our series. Severe brain swelling and raised ICP are major predictors of mortality and morbidity in traumatic brain injury. PTCI, as a complication in severe head trauma, has been known to be an indicator of poor clinical outcome[17,20,27]. However, malignant PTCI has not yet been widely introduced. In this study, we considered the malignant PTCI as PTCI that included the territory of MCA. In this study, there were no statistically important differences between malignant PTCI and nonmalignant PTCI. However, malignant PTCI had poor clinical outcome and high mortality rate compared to nonmalignant PTCI. Regardless of surgery and barbiturate coma therapy, all patients with malignant PTCI died. PTCI caused by raised ICP or brain swelling appears to be one of the secondary injuries. Secondary injuries may be more devastating than primary injuries, and are potentially preventable. Early recognition of causative factors and prompt treatment might prevent PTCI. Unfortunately, factors for prediction of malignant PTCI or PTCI have not been determined in previous studies. Findings from a recent study showed a low GCS (3-8), low systolic BP, blunt vascular injury, and treatment with recombinant factor VIIa (rFVIIa), and there was a significant association of brain shift and herniation with occurrence of cerebral infarction[26,27]. The larger the infracted area, the greater the risk of developing fatal brain edema[18]. Early hypodensity involving more than 50% of the MCA territory on initial CT is a major predictor of severe brain swelling in patients with ischemic stroke[9,11,20]. The distance of midline shift is generally considered to indicate the severity of injury and is a risk factor for poor outcome. Maas et al reported that the degree of midline shift had strong relation with poor clinical outcome[14]. In our study, a severe midline shift of more than 5 mm was detected on brain CT of 12 PTCI patients excluding diffuse brain swelling or bilateral hematoma. This study revealed that mean midline shifts in 4 patients were 15.5 mm (range from 9 to 23 mm) in patients with PTCI, and 16 mm (range from 5 to 20 mm) in patients with malignant PTCI, and there was no significant association of brain shift and malignant PTCI or nonmalignant PTCI. Whether midline shift was observed or not, if PTCI was showed in CT scan, this may indicate the developing of ongoing brain damage and edema formation. Therefore, we thought that the degree of midline shift was not related with poor clinical outcome in patients with PTCI, which may differ from non-PTCI head injury. Other laboratory features in this study included elevated WBC count and serum glucose level. Even though these features may be nonspecific parameters of acute stress without a specific pathophysiological role, the possibility of laboratory predictors of PTCI has been considered and the need remains for prospective randomized studies to clarify these roles as predictable factors.

DHC with duroplasty has been reported to be an effective procedure for treatment of refractory raised ICP in cases of se-

vere head injury or larger hemispheric infarction[2,7,20,25]. Decompressive surgery to reduce ICP has been advocated for more than 50 years[25], and much emphasis has been placed on this life-saving procedure in recent years. Removal of a large portion of the calvarium increases the amount of intracranial volume available for brain swelling[3]. Because DHC leads to the fastest relief by immediate reduction of raised ICP, it is reasonable to perform the procedure early in the post-traumatic period, before occurrence of irreversible injury. DHC decreases direct pressure on the brain and minimizes secondary ischemia due to reduced blood flow by tissue pressure or compression of supplying arteries[1]. DHC for treatment of ischemic stroke could reduce the mortality from 80% in conservatively treated patients to 34% in the group of patients who underwent surgery, or even 16% if initiated very early[4,5,23]. Eberle BM et al.[2] found that a survival rate of 74.4% is promising and 41.9% showed a favorable neurological outcome when DHC was performed in patients with post traumatic intractable IICP. Howard JL et al.[7]. reported a long term favorable outcome in 30% of DHC patients had. In our study, all patients who survived underwent DHC, and mean duration between accident and operation was six hours. The optimal timing for DHC is unclear; however, findings from a recent study demonstrated improved outcome with early DHC[2]. Patients with a malignant form of raised ICP in whom medical therapy is ineffective are most likely to benefit from early DHC. However, there are clearly less aggressive forms of brain swelling, which respond well to conventional medical therapy. Therefore, prediction of which patients are likely to develop raised ICP that is difficult to treat medically early in the post-traumatic course is important. Due to our small sample size and heterogeneous group, we could not demonstrate a correlation between latency between onset of symptoms and surgical intervention and outcome. GCS score on admission was the most important factor for prediction of survival in our series. Early DHC and duroplasty should be considered in patients with a GCS higher than 5, before raised ICP can become established and contribute to an irreversible cascade of events. On the other hand, the question of whether one should operate on a patient with a GCS of less than 5 or not remains debatable. Careful selection of patients is critical in order to avoid unnecessarily aggressive procedures in patients who have little chance of benefit. The subgroup of patients with malignant PTCI or nonmalignant PTCI who were admitted with a GCS of less than 5 on admission showed a high incidence of mortality despite any aggressive therapy. Regardless of pupillary size and reaction, 13 of 14 patients presenting with a GCS of 3-5 in our study died, and the only surviving patient remained in a persistent vegetative state. The cause of poor results in our study is probably erroneous indication or late timing of decompressive surgery. In this instance, irreversible ischemic damage to the brain exists with no chance of recovery. There are limitations to these results because of the small sample size and heterogeneity of the patient population. The subgroup analysis from a large population of different types of hemorrhage patients is needed in future to clarify this issue. Based on these results, we recommend that surgical intervention is probably not indicated in patients with GCS scores of 5 or less.

CONCLUSION

PTCI is a severe complication after head trauma, resulting in poor clinical outcome and high mortality rate. Because of high mortality rate and very poor clinical outcome in patients with malignant PTCI or PTCI with a GCS of 3-5 at the admission, we recommend that aggressive management including early DHC and duroplasty should be considered in patients who develop non-malignant PTCI and GCS higher than 5 after head injury. In the future, large prospective randomized controlled study will be required to justify for aggressive treatments including DHC in patients with malignant PTCI or PTCI with a GCS of 3-5 at the admission.

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