EFFECT OF TRANSVENTRICULAR CEREBROSPINAL FLUID IN PATIENT WITH SEVERE BRAIN DAMAGE WITH THIN ACUTE SUBDURAL BLEEDING. A COMPARATIVE STUDY WITH DECOMPRESSION TREPANATION ACTION

by Muhammad Parenrengi

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ABSTRACT

Severe brain injury is a serious public problem in health service centers and needs continuous improvement of management system. Acute subdural hematomas is the main cause of mortality in severe brain injury with intracranial mass lesion, averaging 42%-90%. There are different considerations until this time about the management of thin acute subdural hematomas less than one centimeter of thickness that have mass lession effect with cerebral midline shift of more than five millimeters in severe brain injury. Observational prospective study, sample number 38 cases of severe brain injury with thin subdural hematomas in August 2005 until July 2006. There are 19 cases treated with surgical decompression craniotomy and 19 cases treated with transventricular CSF drainage. The two groups were followed up concerning mortality and out come until three months after procedure treatment. Statistically analysis with ordinal regression and Fisher's exact and t 2 sample tests. Mortality of decompression craniotomy in severe brain injury group is 57,9%, vegetative and severe disability are 5,3%, moderate disability and full recovery are 15,8%, compare with trans ventricular CSF drainage 36,8% mortality and 5,3% vegetative and severe disability5,3%, moderate disability and 42,1% full recovery.Ordinal regression analysis for Craniotomy decompression and CSF drainage to dependent variabel are significance for mortality with p value 0,041. There are significant factors showing that trans ventricular CSF drainage of thin acute subdural hematomas in severe head injury is better than surgical decompression craniotomy management.

Keywords: *Subdural hematomas, severe brain injury, trans ventricular CSF drainage, surgical decompression craniotomy.*

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INTRODUCTION

As the increase in welfare and development of transportation technology, the total number of vehicles more and more with higher welfare. Ironically, the number of traffic accidents increased from time to time, this is caused by road construction is not comparable with the increase in the number of motor vehicles and low level of road user discipline. Of overall head injuries caused by traffic accidents 65% to 70% classified mild, moderate and the remainder were classified as heavy degrees.

Data collected during the three years starting from 2001 to 2003 cases of head injury in the IRD Dr. Soetomo Consecutive 1685, 1850 and 1735 cases annually. Of this amount Severe Brain Damage 222 (13.2%), 273 (14.76%) and 278 (16.02%). The incidence of complications of acute subdural hemorrhage approximately 13% of the total head injuries with mortality rates ranging from 40% to 70%.

Until now, there are still differences of opinion about the management of patients with brain injury, especially with thin acute subdural bleeding complications (hematoma thickness less than 10 mm), which provides mechanical mass effect of midline shift (MLS) greater than 5 mm. Decompression flow adherents believe that a thin acute subdural hemorrhage (PSDAT) that provides the mechanical effect, will cause effects of global cerebral ischemia, which in turn would reduce or interfere with an extensive cerebral perfusion, so that surgical decompression and hematoma evacuation should be done as quickly as possible to fix disturbed cerebral perfusion (Bullock 2002), while other experts believe that the mechanical effects on PSDAT actually not caused by the hematoma, and reperfusion injury that occurs as a result of too rapid decompression which would result in worse conditions so that surgical decompression and hematoma evacuation will not solve existing problems (Keer 2001, Shore 2004). Subdural bleeding on the brain injury has been known as a cause

of mortality in patients with acute non-missile brain injury, but significant decrease in mortality was found in patients who have performed decompression and evacuation of blood clots compared with the early work done without decompression (Krause 1996, Mass 1997, Kasan 2000). Shore (2004), comparing the drainage of fluid serebrospinalis (CSS) continuously and periodically at the severe brain damage and CSS continuous drainage was better in lowering of ICT (intra-cranial pressure). Ghajar (1993) conducted a drainage severe brain damage patients with CSS in ICTs more than 15 mmHg compared with no drainage CSS, it turns out there are differences in the output. In the group that carried out drainage CSS mortality rate 12%, moderate in the group without drainage CSS 53% mortality rate. Kerr (2001) conducted a study on patients with various bleeding severe brain damage (SDH, EDH, ivh, ICH) that there is no indication of the operation result that drainage CSS as much as 3 cc of ICT can reduce the 2.6 mm Hg to 3.4 mm Hg until the tenth minute .

There are several factors that also determine outcomes of the PSDA. Bullock, 2002 find the number PSDA 40-60% mortality at all ages with a Glasgow Coma Scale (GCS) 3-15 time of admission to hospital, and mortality rate of 57-68% of patients who performed the action evakuasai hematoma. The older group of patients, mortality increased PSDA. In patients over 50 years reached 75% (Bullock 2002, Meagher 2004, Krause 1996).

Initial level of consciousness and post-resuscitation before surgery significantly enough to determine treatment outcome. Patients who are still conscious at the time of preoperative death rate 9%, while the patient unconscious prior to surgery, 40-65% death rate. 74% mortality rate in patients with GCS 4-5, compared to 36% in patients with GCS 6-8. 47% mortality rate found in patients who do not realize hematoma evacuation measures less than two hours after trauma, compared with patients who did not realize who performed the action hematoma evacuation of more than two hours post-trauma death rate increased to 80%. Other reports suggested the death rate 30% when the operation is less than four hours after trauma, compared with 90% mortality rate if surgery is done more than four hours post-trauma (Bullock 2002, Meyer 1996, Meagher 2004, Krause 1996).

Classified as an acute subdural hemorrhage when the bleeding is providing clinical manifestations in the period up to 72 hours of post-traumatic. There are other authors define the PSDA if bleeding is still a component of the composition of the blood clot (Graham 1996, Mc Cormick 1996, Meyer 1996).

In general, experts agree that the acute post-traumatic bleeding ekstraaksial with an inch or more thick, will provide the mechanical effects of cerebral action which gives an indication for surgical intervention, whereas if the thickness is less than one centimeter hematoma and did not cause the mechanical effects of less than five MLS mm do not require surgical intervention (Bullock 2002, Mass 1997, Meyer 1996). Based on the assumption that view, restriction PSDAT made, the criteria included in this study when the thickness is less than one centimeter, but the mechanical effects of cerebral form of the MLS is more than five millimeters.

Another source of bleeding can cause subdural hemorrhage is a laceration or a small artery and vein rupture associated with cerebral contusions. In a small percentage, bleeding occurs from a small cortical artery laceration which sometimes get out of araknoid and embedded in duramater.

Cerebral ischemic damage is important neuropatologis findings in patients who die after the PSDA, although its cause is still poorly known. Edema cerebral subdural hematoma in the region could emerge as a consequence of reperfusion following the evacuation of blood clots, blood vessels, strangulation of bone decompression at the edge of the progression of edema and cerebral swelling had exist (Cooper 2000, Geisler 1996).

The effects of mechanical and ischemic damage to the hypothalamus and brain stem compression caused by cerebral produce vasomotor paralysis that increases the volume of blood flow. This is the main mechanism that mulates the increase in the volume of cerebral swelling and necrosis of neuronal, glial and endothelial cells, enhancing IC and further causes of cerebral ischemia.Damage to the hypothalamus and brain stem is one of the major pathophysiological basis post-decompressive cerebral edema.

Subdural hemorrhage causing an increase of ICT, after three hours of post traumatic to three times higher than normal levels. High ICT and edema hemisperik sometimes become intra operative complications and postoperative treatment of patients with the PSDA that often complicate the neurosurgeon (Crus 1997, Kasan 2000).

Scope of handling emergency is to continue the resuscitation of patients with cerebral severe brain damage, which was started at the scene of trauma, treatment (saving lives) and as soon as systemic traumatic injuries do radiodiagnostic cerebral or spinal injury that accompanies. Emergency handling begins when the patient arrives, and ends after the patient was

transferred to the operating room, living room or intensive care wards, including therein mobilization during diagnostic procedures.

The purpose of handling the emergency room is to prevent the COP into COS triggered either by a systemic disorder. (Bulloc 1996, Meyer 1996, Kasan 2000).

MATERIALS AND METHODS

This research is in the form of analytic observational cohort study prospektive of severe brain damage patients with acute subdural hemorrhage thin which gives the effect of mechanics, started when patients arrived at emergency ward (IRD) until treatment is completed when the patient out of hospital for three months followed by outpatient at outpatient installation (IRJ) Dr. and Dr. Saiful Anwar Malang. Evaluation of outcome measures include how many are dead, vegetative, severe disability, disability being and full recovery for each group.

Started when patients arrived at the IRD severe brain damage Dr. Soetomo and Dr. Saiful Anwar Malang, then worked resuscitation and stabilization of the ABC, the diagnostic process, selection based on inclusion and exclusion criteria following the approval of research studies, and determination of treatment is decided upon by the senior neurosurgeon, implementation and evaluation of therapy outcome start to finish action until three months after the trauma.

Severe Brain Damage patients after resuscitation, head CT scan done, if obtained PSDAT, thickness <10 mm and MLS> 5 mm and meet the inclusion criteria was chosen as a sample. One group performed decompression trepanasi action is another group who performed the action the CSS drainage through the installation of a continuous ventricular drain. Evaluation of both groups of patients died during treatment or patients who live hidup.Untuk evaluation was continued until 3 months post-trauma include vegetative, severe disability, disability is, full recovery for each group

RESULTS

The number of samples entirely counted 40 cases, 20 cases carried trepanasi decompression and 20 cases with continuous drainage transventrikel a case of trepanasi group dropped out because of pneumonia, from the drainage group CSS is also one case of pneumonia dropped out because it arises so that cases can be followed as much 38 cases.

Table 1 Distribution of Sample

Action	Sample Size
Trepanation-Decompression	19
Drainage	19
Total	38

Table 2 Distribution of Age

Age (years old)	Size	Precentation
15-25	9	23.68%
26-35	7	18.42%
36-45	12	31.58%
>45	10	26.32%

Characteristics of patients' age distribution obtained age group 15-25 years and 9 people (23.68%), 26-35 years 7 people (18.42%), 36-45 years 12 people (31.58%) and over age group 45 of 10 people (26.32%).

Table 3 The Results Output by Group Action

GOS	Procedure	
	Trepanation	Dranation CSS
Dead	11 (57.9%)	7 (36.8%)
Vegetatif	1 (5.3%)	1 (5.3%)
Severe Deffect	1 (5.3%)	1 (5.3%)
Average Deffect	3 (15.8%)	2 (10.6%)
Recover	3 (15.8%)	8 (42.1%)
Total	19 (100%)	19 (100%)

The research data from 38 samples that carried as many as 19 samples trepanasi decompression, died 11 cases (57.9%), vegetative one case (5.3%), one case of severe disability (5.3%), and the defect was cured masisng respective three cases (15.8%). A total of 19 cases with drainage CSS, died seven cases (36.8%), severe disability and vegetative each 1 case (5.3%), defects were two cases of (10,6%) and mild disability or recover a total of 8 cases (42.1%).

Ordinal regression analysis of independent variable on the dependent variable trepanasi action research got a significant effect (p 0.041) on the outcomes died (p 0.001), while on the output recovering, disabled are, weight and vegetative defects was not statistically significant.

Table 4 Ordinal regression analysis of action research to GOSin Table 1.

Variable	Estimate	P
Recovery	-0.353	0.428
Average Deffect	0.265	0.549
Severe Deffect	0.486	0.277
Vegetatif	0.705	0.122
Dead	1.702	0.001
Trepanation	1.236	0.041
Drainage		

DISCUSSION

In tables 3 and 4 the results of research and statistical tests with samples of 38 cases dilakuakn trepanasi 19 cases and 19 cases obtained results drainage dead 11 (57.9%) cases in group trepanasi and 7 cases (36.8%) in the drainage group, vegetative and disabilities equal weight of each one case (5.3%), defects were found three cases (15.8%) in group performed trepanasi and 2 cases (10.6%) who carried out drainage and recovered CSS found three cases (15.8%) in group trepanasi decompression and 8 cases (42.1%) in the group that carried out drainage CSS. Ordinal regression analysis of independent variables on the dependent variables the study found that decompression trepanasi actions significantly affecting the output death (p 0.041) while on the dependent variable total cure, handicapped moderate, severe disability, vegetative are not affected significantly. This shows that trepanasi decompression provides a significant effect on output dies larger when compared with drainage in the management of patients with CSS with PSDAT severe brain damage. Patients with cerebral edema, the main problem is the most dominant and this gives the effect. With the drainage of CSS will reduce in stages so that ICT gives the opportunity to adapt, reducing the total intracranial volume and cerebral perfusion fix. ICT related decline with increasing velocity of cerebral blood flow and oxygenation, thus avoiding occur anaerobic metabolism. ATP production further terganggusehingga no cerebral edema can be prevented and cell death does not occur. This is what causes the results of better drainage (Kerr, 2001; Meagher, 2001). Drainage CSS continually on cerebral edema with PSDAT conditions can reduce ICT and improve the perfusion of the brain (Shore 2004, Fortune 1995).

In the group of decompression trepanasi this higher mortality due to cerebral edema condition will occur if done trepanasi strangulation peripheral blood vessels when the defect is less extensive bone decompression inadequate thus hinder the blood circulation in the area so that over time will occur infarction. Another theory is support reperfusion injury either mechanical or chemical so that the more severe edema (Cooper 1996, Samudrala 1996, Kasan 2002). PSDA mortality in patients with traumatic coma high enough had been done despite decompression trepanasi ie 57-68% range (Bullock 2002).

Cerebral edema after thorough evacuation of hematoma is a troublesome complication and is the cause of the high mortality in group trepanasiu PSDA decompression. Inadequate decompression would cause peripheral blood vessels strangulkasi kraniotomi that hinder the flow of drainage. When the last long under it will cause cerebral infarction. This is because blood flow is disrupted causing obstacles causing the local cerebral tissue oxygenation. More information will lead to hypoxia and the outcome is more severe edema (Cooper 2000).

One factor causes edema of the group is the occurrence of decompression trepanasi reperfusion injury, as for the cause, among others, Mechanics, eg decompression in patients with postoperative hematoma evacuation. In the area of negative pressure decompression occurs, so it will be an increase in blood flow occurs, resulting intraveskuler pressure also increases.This will cause the blood vessels rupture causing bleeding again. On the other hand due to the increased intravascular pressure causes the blood vessels so that the reaction of vasoconstriction reducing blood flow resulting ischemia and cerebral edema increased. The cause of both is the biomolecular / biochemical, including inflammatory reactions, free radicals, growth factor, Platelets activating factor (PAF), neurotransmitters, epinephrine and cytokines. The end result of biomolecular or chemical processes causing cell necrosis.

The effects of mechanical and ischemic damage to the hypothalamus and brain stem compression caused by cerebral produce vasomotor paralysis that increases the volume of blood flow. This is the main mechanism that stimulates the increase in the volume of cerebral swelling and necrosis of neuronal, glial and endothelial cells, enhancing ICT and further causes of cerebral ischemia. Damage to the hypothalamus and brain stem is one of the major pathophysiological basis pascadekompresi cerebral edema.

CONCLUSION

In this study we can conclude that in the management of patients with PSDA severe brain damage thin, it was found that trepanasi groups decompression mortality 57.9%, 36.8% CSS drainage groups, the output of vegetative, severe disability and disabled are relatively the same two groups, drainage group cure rate was 42.1%, 15.8% group trepanasi decompression, and drainage CSS gives better results compared with trepanasi decompression in the management of patients with PSDAT severe brain damage because cure rate mortalitsnya bigger and smaller.

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