Endoscopic Management of Intraventricular Extension of Intracerebral Hemorrhage

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Abstract

Background: Although removal of the intracerebral hemorrhage (ICH) following craniotomy and corticectomy performed under direct vision is not technically difficult, the operative procedure carries a high risk in severe cases. **Aim of work:** identify the beneficial of usage of neuroendoscope in cases of intra ventricular hemorrhage (IVH) extended from ICH as regard safety, efficiency, and efficacy in comparison to usage of external ventricular drainage **METHODS:** Twenty patients of spontaneous ICH with intra ventricular extension were divided into two groups, one group operated by endoscopic evacuation and external drainage, while the second group operated by external drainage only. We compared the result of both groups. CT scan of brain was the main diagnostic test preoperatively. **RESULTS:** Mortality rate of Endoscopic Evacuation followed by EVD is 50% while the mortality rate of External ventricular drainage alone is 70%. **CONCLUSION:** Early evacuation as early as 3-4 hours is of great benefit as it allows us to immediately decompress the brain, remove the hematoma before releasing more toxic products to the neural tissue and rapid reversal of ventricle dilatation can prevent hydrocephalus from developing.

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Introduction

Spontaneous intracerebral haemorrhage (SICH) is responsible for 10-15% of the acute stroke. The common causes of SICH are hypertension, aneurysm, arteriovenous malformation (AVM), and vasculopathies. Hypertensive bleeding occurs mainly in putamen, near ruptured aneurysmal site with or without subarachnoid haemorrhage, or at the site of AVM, while bleeding due to angiopathy is usually lobar. Extension of the hemorrhage into the ventricles could occur in 40% of SICH cases, could happen early or late in the sequence of events. The amount of blood in the ventricles relates directly to the degree of injury and likelihood of survival. Secondary tissue injury processes related to intraventricular bleeding can be reversed by removal of clot in animals. Benefits of

clot removal include limitation of inflammation, edema, and cell death, restoration of cerebral spinal fluid flow; intracranial pressure homeostasis, improved consciousness, and shortening of intensive care unit stay (3). Prognosis is determined by the extent of the hemorrhage. Headache, vomiting, confusion, decreased level of consciousness and, hemiparesis, are common clinical findings. The clots tend to disappear within 2 weeks. When clots are symptomatic, intraventricular drainage (possibly bilateral) may be useful, but the blood often occludes catheters used for this purpose (10). Neuroendoscopic management of severe intraventricular hemorrhage (IVH) in patients was safe, efficiently reduced the amount of ventricular blood and ventricular dilatation (20). There is significant relationship between older age and an increased risk of an ICH. Men have a higher incidence of spontaneous ICH than women, especially among those more than 55 years old (4). The lower the level of education, the higher the incidence of an ICH. This was attributed to a lower awareness of the value of primary health care in less educated people (14). The most important non epidemiologic risk factor for spontaneous ICH is hypertension. It was found that hypertension increased the risk of ICH especially in older people. Hypertension after ICH has been reported as a poor prognostic indicator and better outcomes in patients with ICH whose blood pressure was maintained below a mean arterial pressure (24).Some reports indicate that alcohol consumption also increases the risk of spontaneous ICH. Excessive use of alcohol also increases the risk of intracerebral hemorrhage by impairing coagulation and directly affecting the integrity of cerebral vessels. Cerebral amyloid angiopathy, which is characterized by the deposition of β-amyloid protein in the blood vessels of the cerebral cortex and leptomeninges, is another risk factor for intracerebral hemorrhage, particularly in elderly persons.IVH is described in four grades, grade (I) bleeding in small area of the ventricle, grade (II) bleeding occur inside the ventricles, grade (III) ventricles are enlarged by blood, and grade (IV) bleeding into the main brain tissue surrounding the ventricle. IVH hemorrhage is thought to be caused by capillary bleeding due to loss of cerebral autoregulation and abrupt alterations in cerebral blood flow and pressure. This mechanisms account for grades I, II, and III cerebral hemorrhage. Grade IV hemorrhages appear to result from hemorrhagic venous infarctions surrounding the terminal vein and its feeders, probably primarily related to increased venous pressure following or associated with the development of lower-grade hemorrhages. Indeed, the use of the term "periventricular hemorrhagic infarction" has been suggested rather than using the term grade IV hemorrhage. The use of this terminology stresses the current theory that periventricular hemorrhagic infarction is a complication of lower grade hemorrhage (1). Attention has shifted to perilesional brain injury as a potential target for therapeutic intervention in ICH patients. Pathological and experimental studies indicate that a "penumbra" of progressive tissue damage and edema develops in regions immediately surrounding a hematoma. Mechanical injury caused by elevated local tissue pressures, reduction of cerebral blood flow (CBF), infiltration of plasma, and inflammation related to clotting proteins and protease induction have all been implicated as mediators of this form of secondary injury. Clinical neurological deterioration, which occurs in one third of ICH patients, may occur as a direct consequence of this process, or may result indirectly from hyperacute bleeding into the perilesional region or herniation related to brain swelling (18). ICHs commonly occur in the cerebral lobes, basal ganglia, thalamus, brain stem (predominantly the pons), and cerebellum. Extension into the ventricles occurs in association with deep, large hematomas. Edematous parenchyma, often discolored by degradation products of hemoglobin, is visible adjacent to the clot. Histologic sections are characterized by the presence of edema, neuronal damage, macrophages, and

neutrophils in the region surrounding the hematoma. The hemorrhage spreads between planes of white-matter cleavage with minimal destruction, leaving nests of intact neural tissue within and surrounding the hematoma. This pattern of spread accounts for the presence of viable neural tissue in the immediate vicinity of the hematoma (1). Intraparenchymal bleeding results from the rupture of the small penetrating arteries that originate from basilar arteries or the anterior, middle, or posterior cerebral arteries or due to rupture of microaneurysms. Degenerative changes in the vessel wall induced by chronic hypertension reduce compliance and increase the likelihood of spontaneous rupture. Most bleeding occurs at or near the bifurcation of affected arteries, where prominent degeneration of the media and smooth muscles can be seen (23). Computed tomographic (CT) scanning shows that hematomas expand over time in 20 to 36% of cases. This expansion has been attributed to continued bleeding from the primary source and to the mechanical disruption of surrounding vessels. Acute hypertension, a local coagulation deficit, or both may be associated with expansion of the hematoma (19).The clinical presentation of ICH depends on the size, location, and presence of intraventricular extension of the hemorrhage. Headache of variable intensity always occurs and may be accompanied by nausea and vomiting, focal signs, and progressive neurologic deficits (13). Seizures occur in approximately10% of all patients with ICH and in almost one half of patients with lobar hemorrhage. Almost all seizures occur at onset of bleeding or within the first 24 hours after hemorrhage (9). Patients with large hemorrhages present with stupor or coma. This may be secondary to elevated intracranial pressure (ICP) leading to decreased cerebral perfusion or due to direct infiltration or distortion of diencephalic or brainstem structures. Patients with blood extending into the ventricular system often experience a reduced level of alertness because of ventricular ependymal irritation or the development of hydrocephalus (1). Clinically, putaminal hemorrhages present with contralateral motor deficits, gaze paresis, aphasia, thalamic hemorrhages also present with contralateral sensory loss. Pupillary and oculomotor abnormalities may coexist if the thalamic hemorrhage extends into the rostral brainstem. It is important to recognize cerebellar hemorrhages, which present with nausea, vomiting, ataxia, nystagmus, decreased level of consciousness, and ipsilateral gaze palsies or facial paralysis. Pontine hemorrhage present with coma, pinpoint pupils, disturbed respiratory patterns, autonomic instability, quadriplegia, and gaze paralysis. Almost all pontine hemorrhages are fatal. Lobar hemorrhages present according to the location of the hemorrhage (22). Abnormalities indicating higher-level cortical dysfunction, including aphasia, gaze deviation, and hemianopia, may occur as a result of the disruption of connecting fibers in the subcortical white matter and functional suppression of overlying cortex (11). The presence of a large hematoma and ventricular blood increases the risk of subsequent deterioration and death. Expansion of the hematoma is the most common cause of underlying neurologic deterioration within the first three hours after the onset of hemorrhage. Worsening cerebral edema is also implicated in neurologic deterioration that occurs within 24 to 48 hours after the onset of hemorrhage. Infrequently, late deterioration is associated with progression of edema during the second and third weeks after the onset (10). Hemorrhage on CT scan appears as a rounded or elliptical parenchymal mass. It may be mixed iso-/hyperdense if there is rapid bleeding or in cases with coagulopathy. There is peripheral low density, representing the surrounding edema. The acute ICH is usually hyperdense, extend into the lateral ventricle , 3rd, and 4th ventricle, hyrocehpalic changes may early appear. The MRI appearances of intracranial hemorrhage (ICH) vary according to erythrocyte membrane integrity and the molecular state of hemoglobin and its degradation products (7). Recognition of IVH has prognostic value and may help to select patients for novel

interventional therapies. A better understanding of the MRI characteristics of IVH may help to exclude other intraventricular diseases such as neoplasia and ventricular empyema (pyocephalus). Based on the T1-, proton density, and T2-weighted image appearance the IVH/ICH were classified as predominantly containing oxy-or deoxyhemoglobin, intracellular or extracellular methemoglobin, or hemosiderin. Thus, oxyhemoglobin was defined as isointensity on T1- and slightly increased signal on T2-weighted images. Deoxyhemoglobin was defined as slightly low or isointensity on T1- and low signal on T2-weighting. Intracellular methemoglobin was defined as high signal on T1- and low on T2-weighted images, extracellular methemoglobin as high signal on both (5). IVH is subdivided 1) Layered IVH was defined as blood signal that freely flowed in cerebrospinal fluid (CSF) and layered in the dependent portions of the ventricular system resulting in blood-CSF fluid levels often separated by a border-zone, the actual interface between the CSF and the blood products. 2) Clotted IVH was defined as a fixed well-circumscribed intraventricular hematoma that did not layer in the CSF. The signal characteristics of the two forms of IVH reflect different blood degradation rates. The slower degradation in layered IVH than in clotted IVH and IPH relates to various physical factors. The most pertinent variable affecting the degradation rates is probably oxygen and glucose content. Degradation of blood after bleeding proceeds in predictable stages based on the oxidative state of hemoglobin. Hemoglobin in the red blood cell (RBC) fluctuates between an oxygenated and a deoxygenated state, depending on blood oxygen tension (7). When the RBC leaves the vessel, the deoxygenated state begins to predominate, as oxygen content decreases. Further degradation from deoxyhemoglobin to intracellular methemoglobin requires oxygen. In addition, RBC metabolism depends on glucose availability, because the RBC does not have glycogen stores .Glucose deprivation accelerates membrane rupture and promotes the next degradation step and conversion from intracellular to extracellular methemoglobin. The rate of breakdown of hemoglobin in ICH is therefore strongly related to the compartment of the ICH. The ventricular cavity has relatively higher levels of both glucose and oxygen than brain parenchyma. Therefore, layered IVH is probably kept in an earlier degradation stage, while clotted IVH and IPH degrade faster. This slower degradation resulted in persistence of deoxyhemoglobin and intracellular methemoglobin for significantly longer in layered IVH than in clotted IVH or IPH (6).

The aim of the work was to identify the beneficial of usage of neuroendoscope in cases of IVH extended from ICH as regard safety, efficiency, and efficacy in comparison to usage of external ventricular drainage.

Patients and methods

The study was done prospectively in the Neurosurgery Department, Kasr Al Ainy Hospital, Cairo University and El-Sahel teaching hospital. The cases admitted in the period between June 2010 and January 2013, suffering from SICH with intraventricular extension. The study was carried out to identify the beneficial usage of neuroendoscope followed by external ventricular drainage in cases of IVH extended from ICH in comparison to usage of external ventricular drainage alone as a treatment for intraventricular hemorrhage. Patients selected for this study were from 40-70 years old, Glasgow coma scale score of 8 or more, and mild, moderate or severe intraventricular (affecting lateral &third ventricle) hemorrhage. CT scan of the brain was the main diagnostic test used, to verify ICH location, ICH volume, presence of IVH and

associated hydrocephalus. ICH location was determined by the anatomical structure that contained the majority of the hematoma. ICH volume was determined using the (A x B x C)/2, where A is the largest diameter of the hematoma on axial CT scan slice in centimeters; B is the diameter perpendicular to A on the same slice, and C is the thickness of the hematoma on CT in centimeters, also counted as the number of axial cuts on CT multiplied by slice using intraventricular hemorrhage Scoring. Hen Hallevi, et al (18), grading system reported that the third and fourth ventricles contribute much less to the ventricular volume than the lateral ventricles and in the presence of hydrocephalus, the ventricular volume increases through expansion. Authors graded each lateral ventricle with a score of 0 (no blood or small amount of layering), 1 (up to one third filled with blood), 2 (one to two thirds filled with blood), or 3 (mostly or completely filled with blood). The third and fourth ventricles received a score of 0 for no blood or 1 if they were partially or completely filled with blood. Hydrocephalus was coded as present (1) or absent (0).For calculating the IVH score (IVHS)

IVHS = 3 × (RV grade + LV grade) + 3rd ventricle grade + 4th ventricle grade + 3 × Hydrocephalus grade

For calculating the IVH volume there is a quick reference for converting IVHS to IVH volume (Table1)

IVH Score	IVH Volume (mL)	IVH Score	IVH Volume (mL)
1	1.2	13	13.5
2	1.5	14	16.4
3	1.8	15	20.1
4	2.2	16	24.5
5	2.7	17	30.0
6	3.3	18	36.6
7	4.1	19	44.7
8	5.0	20	54.6
9	6.0	21	66.7
10	7.4	22	81.5
11	9.0	23	99.5
12	11.0		

Patients were be randomized in two groups, 1st group was subjected to endoscopic evacuation of the hematoma followed by external ventricular drainage until CSF become clear. Second group was subjected to external ventricular drainage; Odd numbers were entered into 1st group while even numbers were entered in 2nd group according to time of admission. A right frontal burr hole was performed under general anesthesia. This burr hole was placed just anterior to the coronal suture and 2 to 3 cm from midline. Dura was opened after coagulation is done and by use of rigid Karl Storz endoscope (with lens angle 6°) introduced into lateral ventricle, continuous irrigation is done to blood in the lateral ventricle until it becomes clear and one can identify septum pellucidum and foramen of Monro. Septostomy is done in cases where the other ventricle is filled with blood. The foramen of Monro is identified by following the choroid plexus and thalamosriate vein to enter the 3rd ventricle and continuous irrigation is done until the fluid becomes clear. In some cases, 3rd ventriculostomy is done. In two

cases with 4th ventricle filled with blood, mother baby technique was used in which through a rigid endoscope, a flexible endoscope was introduced to reach the 4th ventricle and continuous irrigation done to wash out blood. External ventricular drainage until CSF becomes clear, with drainage bag placed at level 8- 10 cm higher from external auditory canal, was done. All cases were admitted to the intensive care unit. Cases were operated upon within a period of 24 hours of presentation. All patients were evaluated postoperatively by a CT scan of the brain on the second postoperative day. The most common and important associated conditions; hypertension, diabetes mellitus and anticoagulant drugs were evaluated as regards association with the development of the hematoma and the effect on outcome. The outcome of the operated patients was evaluated as regards; residual hematoma, residual neurological deficits, mortality and mortality. Intraoperative difficulties were evaluated. Intraoperative difficulties such as difficult hematoma evacuation, difficult hemostasis, incomplete evacuation and rebleeding were all reported.

Results

This study was conducted on 20 patients. 1st group was subjected to endoscopic evacuation of the hematoma, followed by external ventricular drainage until CSF become clear. 2nd group external ventricular drainage was done alone until CSF become clear.

Table (1): Socio-demographic characteristics of the study groups:

Criteria	Endoscopy (No=10)	EVD (No=10)
Sex		
Male	5 (50.0%)	5 (50.0%)
Female	5 (50.0%)	5 (50.0%)
Age group		
From 40-50years	6 (60.0%)	1 (10.0%)
From 51-60years	2 (20.0%)	3 (30.0%)
From 61-70years	2 (20.0%)	6 (60.0%)
Mean	52.8	55.8

This table shows that the distribution of gender was equal between both groups (50% for each gender). The average age was 52.8 years in endoscopy group and 55.8 years in EVD group.

Table (2): Difference in clinical findings between both groups

Clinical findings	Endoscopy (n=10)	EVD (n=10)	X^2	P
Diabetes:				
Yes	4 (40%)	5 (50%)	0.2	0.6
No	6 (60%)	5 (50%)		
Hypertension:				
Yes	6 (60%)	7 (70%)	1.8	0.1
No	4 (40%)	3 (30%)		

Taking anticoagulant therapy: Yes No	 5 (50%) 5 (50%)	 4 (40%) 6 (60%)	0.2	0.6
Glasgow Coma Scale: 13-15 8-12	 3 (30%) 7 (70%)	 3 (30%) 7 (70%)	0.00	1.0

This table clarifies that 40% of the first group was diabetic compared to 50% of EVD group. 60% in the first group were hypertensive compared to 70% of EVD group. 50% of the first group took anticoagulant therapy compared to 40% of EVD group. 30% in both groups had GCS (13-15), and 70% with a score (8-12). The differences in all variables among both groups were not significant.

Table (3): Relation between neurological status and hypertension.

GCS / **HPT**	**Endoscopy**		**EVD**		**Total**	X^2	**p**
	13-15	**8-12**	**13-15**	**8-12**			
- **Yes**	2	4	2	5	13		
- **No**	1	3	1	2	7	0.3	0.9
Total	3	7	3	7	20		

This table shows that hypertension has no effect on neurological status.

Table (4): Relation between neurological status and diabetes mellitus.

GCS / **DM**	**Endoscopy**		**EVD**		**Total**	X^2	**p**
	13-15	**8-12**	**13-15**	**8-12**			
- **Diabetics**	1	3	2	3	9		
- **Non diabetics**	2	4	1	4	11	0.7	0.8
Total	3	7	3	7	20		

This table shows that DM has no effect on neurological status.

Table (5): Relation between GCS and rate of survival among both groups:

Glasgow Coma Scale (GCS)	**Endoscopic (No=10)**		**EVD (No=10)**		X^2	**p**
	Survived	**Died**	**Survived**	**Died**		
13-15	3 (30.0%)	0 (0.0%)	2 (20.0%)	1(10.0%)	7.03	0.07
8-12	2 (20.0%)	5 (50.0%)	1 (10.0%)	6 (60.0%)		
TOTAL	5 (50.0%)	5 (50.0%)	3 (30.0%)	7 (70.0%)		

This table illustrates that GCS has no effect on the rate of survival among both groups.

Table (6): Relationship between neurological state on admission and length of hospital stay.

Stay / GCS	Endoscopy (N=10)	EVD (N=10)	t-test	p
13-15	8.6 ± 2.3	10 ± 4.2	1.9	0.9
8-12	7 ± 1.4	5.8 ± 1.1	2.1	0.2

This table showed that there is no relation between length of hospital stay and GCS on admission.

Table (7): Relationship between neurological state on admission and IVH volume:

IVH / GCS	Endoscopy (N=10)	EVD (N=10)	t-test	p
13-15	22.1±2.3	15.46±1.5	7.6	0.1
8-12	9.8±1.2	12.05±2.4	2.6	0.9

This table showed that there is no relation between neurological state on admission and IVH volume

Discussion

Intraventricular extension of spontaneous intracerebral hemorrhage represents 20% of primary ICH. Spontaneous intracerebral hemorrhage (SICH) is responsible for 10-15% of the acute stroke. The common causes of SICH are hypertension, aneurysm, arteriovenous malformation (AVM), and vasculopathies. Hypertensive bleeding occurs mainly in putamen, near ruptured aneurysmal site with or without subarachnoid hemorrhage, or at the site of AVM, while bleeding due to angiopathy is usually lobar. An intraventricular hemorrhage is a bleeding into the ventricular system and results from physical trauma or from hemorrhage in stroke (15). The incidence of ICH increases above the age of 55 (14). In this study the age of patients ranged from 40 – 70 years old (mean age 54.3 years old). In group A (endoscopic group) age ranged from 40 - 70 years with mean 52.8 years old, while in group B (EVD group) ranged from 52 - 70 years with mean 55.8 years old. Qureshi et al. (22) found that ICH is more common in males than in females. In this study the male: female ratio was 1:1. Hisatomi et al., (20), reported that less hematoma growth and good outcome are obtained in patients who achieved the lowest BP levels during the first 24 hours after onset of hemorrhage, particularly those who achieve on-treatment levels of systolic BP between 130 and140 mm Hg. However, intensive BP reduction may lower cerebral perfusion pressure and induce ischemia in hypoperfused tissue surrounding the hematoma. In this study control blood pressure was done in ICU either using lasix or tridil in severe hypertension within 1st 12 hours of admission during preparation for surgery aiming for systolic blood pressure of 140-150 mm Hg. Hypertension is the most common risk factor, Other etiologic factors include cerebral vascular anomalies like AVM and aneurysms, coagulopathies, choroid plexus tumors and cysts, moyamoya disease arteritis and dural AVF. Diabetes mellitus (DM) is usually associated with early mortality and blood sugar at time of admission in patients with ischemic stroke has been reported to be associated with poor outcome. In this study, hypertension was the main factor in intraventricular

hemorrhage which represents 70% of patients, DM as another risk factor was present in 45% of cases. The risk of ICH with intraventricular extension in patients presented with oral anticoagulation increases more than 10 folds in patients older than 50 years. The hematoma is larger in size. The management of these patients requires rapid reversal of their coagulopathy. Vitamin K, fresh frozen plasma, or Prothrombin complex concentrate are used (25). In this study, 45% of the patients take oral anticoagulant; and size of intraventricular hematoma ranged from 6.4-30 cm3 in patients taking oral anticoagulant, while the size of intraventricular hematoma ranged from 3.3-16.4 cm3 in patients who had not received oral anticoagulant. Fresh frozen plasma, 2 units every 8 hours for 24 hours was used in those patients' until INR reach 1.5. The usage of oral anticoagulants is associated with not only more size of hematoma, either intracerebral or intraventricular, but also difficulty in surgery due to excessive oozing of blood during surgery which is difficult to control. More morbidity and mortality occur to those patients. In ICH, 60% of the patients experience smooth gradual deterioration. The clinical presentation of ICH hemorrhage with intraventricular extension may vary from relatively minor pure motor hemiparesis to profound weakness and sensory loss (2). In this study, GCS was ranged from 8 – 15 associated with hemiparesis which ranged from grade 0 to grade 4. Most patients presented late with severe affection of the conscious level. The mechanisms by which IVH volume affects outcome likely include increased intracranial pressure with reduced cerebral perfusion, mechanical disruption, ventricular wall distension, and possibly an inflammatory response. Although IVH volume in itself is associated with poor outcome, Hen Hallevi (16) observed the previously reported stronger association with total volume (TV). They identified a poor-outcome threshold of 50 ml above which all patients had a poor outcome and a total volume (TV) >60 ml was identified for mortality. In this study, the present of intraventricular hemorrhage is a predictive value indicates poor prognosis as range of mortality of ICH in many studies ranged from 20-47% while mortality rate in this study is 60%. Another observation found in this study was that not only the volume of intraventricular hemorrhage affects conscious level but also presence of blood in 4^{th} ventricle , rapid appearance of hydrocephalic changes , initial volume of intracerebral hemorrhage and the dynamic process in which rapid increase in the volume of blood either intracerebral or intraventricular. Intraventricular hemorrhage in group A (endoscopic group) varies from 3.3 cm3 to 30cm3 with mean 13.6 cm3, while in group B (EVD group) varies from 5 cm3 to 30cm3 with mean 13.58 cm3. According to GCS, patients with GCS 12 – 15 the size of intraventicular hemorrhage in group A ranged from 16.4 - 30cm3 with mean 22.1cm3, while in group B intraventricular hemorrhage volume ranged from 7.4 - 30cm3 with mean 15.46 . In patients with GCS 8 – 12 intraventricular hemorrhage volume in group A ranged from 3.3 -30 cm3 with mean 9.8cm3, while in group B intraventricular hemorrhage volume ranged from 4 -20.1cm3 with mean 12.05 cm3. In this study ,there is the relationship between neurological state on admission and hospital stay in which the mean hospital stay in group A (endoscopic group) with GCS 13-15 is 8.6day, while in GCS 8-12 is 7 day. In group B (EVD group) the mean hospital stay in GCS 13-15 is 10day, while in GCS 8-12 is 5.8 day. Due to the fact that deep areas of the brain, as basal ganglia and thalamus are less able to accommodate large volumes, the conscious level of the patient on admission was related to the size of the hematoma so the larger the volume of the hematoma, the worst the conscious level (8). In this study, the prognosis is affected by the relation of the initial conscious level patients, in which 83.33% of patients with GCS 13-15 survived, while only 21.4% with GCS 8-12 survived. Patients, who have no demonstrable cause, even after angiography, seem to have better prognosis than those with a documented

mechanism of hemorrhage. They also found that coagulopathy is a poor prognostic factor. They noted diabetes mellitus to be associated with early mortality. Presence of blood in all ventricles was found to be a poor prognostic factor in our series. The recommendations for impatient care necessitate initial monitoring and management of ICH, patients should take place in an intensive care unit with physician and nursing neuroscience intensive care expertise .This is major project that need coordination between neurosurgeon, well trained nursing, available beds in ICU and encouragement from government to archive this major project to save lives of many patients. Glucose should be monitored and normogylcemia is recommended. Clinical seizures should be treated with antiepileptics (21). In this study annticonvulsants as prophylaxis were administered to all our patients from the fear of seizures not due to the locations of the hematoma but rather due to the surgical procedure. Many approaches were utilized to evacuate intraventricular hematoma one of these approaches was reported by Ferro (12). In this study single right frontal burr holes were performed. The sudden rise in ICP associated with initial bleeding may cause a significant reduction in cerebral blood flow, potentially leading to ischemia. The obstruction of normal cerebrospinal fluid flow and the mass effect associated with hematoma and clots within the ventricles produce further damage. Persistence of blood in the fourth ventricle is related to poor outcome in IVH patients (20). As regards relationship between neurological state on admission and outcome in each group, survived patients in endoscopic management are 30 % with GCS 13 -15, while 20% with GCS 12 -8. Survived patients in EVD management 20% with GCS 13 -15, while with GCS 12 -8 are 10%. As regards overall mortality in each approach the mortality rate of endoscopic evacuation followed by EVD is 50 % while the mortality rate of external ventricular drainage alone is 70%.

Conclusion

Evacuation of spontaneous intracerebral hemorrhage with intraventricular extension is not only necessary to decompress the brain and to decrease the intracranial pressure but to remove the hematoma that releases toxic products to the neural tissue causing more destruction Endoscopic evacuation is minimally invasive technique designed to facilitate the evacuation of the hematoma, decrease mortality rate and shunt dependence. Patients with spontaneous ICH need special ICU care. The cause of death in many cases was related to perioperative complications rather than the hematoma or the operation. The ICH patients need properly trained physicians and nursing staff to deal with their condition, mortality results from lost hope in these patients whereas the condition is treatable with proper aggressive measures. In this study we found that early evacuation as early as 3-4 hours is of great benefit as it allows us to immediately decompress the brain, remove the hematoma before releasing more toxic products to the neural tissue and rapid reversal of ventricle dilatation can prevent hydrocephalus from developing. Proper training of the ICU staff and nursing staff on how to deal with cases of ICH, educating them that these are not hopeless cases or mortality cases and good outcome can be achieved with proper ICU management and proper care.

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