www.surgicalneurologyint.com

ScientificScholar® Knowledge is power
Publisher of Scientific Journals

Surgical Neurology International

Editor-in-Chief: Nancy E. Epstein, MD, Clinical Professor of Neurological Surgery, School of Medicine, State U. of NY at Stony Brook.

SNI: Neurovascular

Editor
Kazuhiro Hongo, M.D.,
Shinshu University, Matsumoto, Japan

SNI Open Access

Original Article

Efficacy of translamina terminalis ventriculostomy tube in prevention of chronic hydrocephalus after aneurysmal subarachnoid hemorrhage

Ahmed Abdelaziz Elsharkawy, Essam Ahmed Abdelhameed

Department of Neurosurgery, Tanta University, Tanta, Gharbia, Egypt.

E-mail: *Ahmed Abdelaziz Elsharkawy - ahmed.elsharkawy@med.tanta.edu.eg; Essam Ahmed Abdelhameed - esam.hameed@yahoo.com

***Corresponding author:**
Ahmed Abdelaziz Elsharkawy,
Department of Neurosurgery,
Tanta University, Elbahr st.,
Tanta - 11113, Gharbia, Egypt.

ahmed.elsharkawy@med.tanta.edu.eg

Received : 15 May 2020
Accepted : 03 July 2020
Published : 12 September 2020

DOI
10.25259/SNI_278_2020

Quick Response Code:

ABSTRACT

Background: Chronic shunt-dependent hydrocephalus is still a common complication after aneurysmal SAH (aSAH) and is associated with increased morbidity. Pathology of chronic shunt-dependent hydrocephalus after aSAH is complex and multifactorial which makes its prevention challenging. We thought to evaluate whether external ventricular drainage (EVD) through fenestrated lamina terminalis would decrease the rate of chronic shunt-dependent hydrocephalus after aSAH.

Methods: A retrospective analysis of 68 consecutive patients with aSAH who underwent microsurgical clipping of the ruptured aneurysm. Patients were divided into two groups: Group A included patients with lamina terminalis fenestration without insertion of ventriculostomy tube and Group B included patients with EVD through fenestrated lamina terminalis. Demographic, clinical, radiological, and outcome variables were compared between groups.

Results: Group A comprised 29 patients with mean age of 47.8 years and Group B comprised 39 patients with mean age of 46.6 years. Group B patients had statistically significant ($P < 0.05$) lower incidence of chronic shunt-dependent hydrocephalus than Group A patients (30.8% vs. 55.2%, respectively).

Conclusion: EVD through fenestrated lamina terminalis is safe and may be effective in decreasing the incidence of chronic shunt-dependent hydrocephalus after aSAH.

Keywords: Aneurysmal subarachnoid hemorrhage, External ventricular drainage, Lamina terminalis, Shunt-dependent hydrocephalus

INTRODUCTION

Chronic shunt-dependent hydrocephalus is a common consequence after aneurysmal subarachnoid hemorrhage (aSAH) with reported incidence of 8.9–48%. It contributes largely to patient long-term morbidity, hospital readmission, and health-care economic burden occurring after aSAH. Several factors have been identified to predict the development of chronic hydrocephalus after aSAH including radiological findings as hydrocephalus at presentation, intraventricular blood, intracerebral hematoma and high Fisher grade, clinical data as high Hunt and Hiss score, and laboratory data as sustained systemic inflammatory response. The rate of development of chronic hydrocephalus after aSAH was affected by treatment modality used to secure the ruptured aneurysm, whether microsurgical clipping or endovascular coiling, in some reports and not affected in other ones.[1,2,9,11,13,15-19,23,29,30,32,33,46,47,49]

It has been reported that maneuvers accelerating the clearance of blood in the basal cisterns could halt the development of symptomatic ventriculomegaly and shunt dependence after aSAH.[30] It was also suggested that fenestration of lamina terminalis during microsurgery for anterior circulation aneurysms decreases the incidence of chronic hydrocephalus and shunt dependence after aSAH.[29] The objective of our study was to evaluate whether external ventricular drainage through a catheter inserted into fenestrated lamina terminalis during microsurgery for clipping of ruptured anterior circulation aneurysms was associated with reduced rate of development of chronic hydrocephalus and shunt dependence.

MATERIALS AND METHODS

The present study is conducted with the approval of our university ethical committee. This is a retrospective analysis of prospectively collected data on patients with intracranial aneurysms (IAs) who were managed at our university emergency hospital between July 2013 and July 2019**.** We enrolled in this study patients with ruptured anterior circulation IAs who underwent microsurgical clipping of their ruptured IAs. Our plan was to assess the effect of performing EVD through a translamina terminalis tube inserted during clipping surgery on the development of chronic hydrocephalus after aSAH. We divided our patients into two groups; Group A: patients who underwent aneurysm clipping with lamina terminalis fenestration only without inserting EVD tube and Group B: patients in whom EVD through a translamina terminalis tube was done during clipping surgery. We excluded patients in whom EVD was done by conventional ventriculostomy through Kocher burr hole before surgery or through the Paine's point during surgery and patients who presented by Hunt and Hess score V.

The general surgical steps for clipping of the ruptured IAs were essentially the same in both groups. Either lateral supraorbital or pterional craniotomy was done in all cases. After dural opening, CSF is released from the basal cisterns and from the lamina terminalis to lax the brain. The ruptured aneurysm is then clipped under temporary proximal control. In Group B, a ventriculostomy tube is inserted, under direct microscopic visualization, inside the third ventricle through the fenestrated lamina terminalis to come out beneath the basal brain surface [Figure 1]. The tube is externalized through a craniotomy burr hole and a separate skin puncture and connected to external drainage system for CSF release and daily cytochemical analysis. Weaning of EVD starts on day 7–10 after surgery. The external reservoir is gradually elevated over 2 days to maximum of 20 cm water then clamped for 1–2 days with serial clinical examination and CT scans to monitor any clinical deterioration or ventriculomegaly. Prophylactic intravenous broad-spectrum antibiotics are given to all patients in both groups from day of hospital admission till discharge.

Statistical analysis

We assessed the distribution and frequency of all demographic, radiologic, and clinical variables. Covariates were compared between Group A and Group B using the Chi-squared test for categorical variables, the Student's *t*-test for parametric continuous variables, and Mann–Whitney U-test for nonparametric continuous variables. $P < 0.05$ was considered statistically significant. All statistics were calculated using SPSS software version 25 (SPSS, Inc., Chicago, Illinois).

RESULTS

A total of 83 consecutive patients underwent microsurgical clipping for ruptured anterior circulation IAs at the Neurosurgery Department in Tanta University Hospitals, Egypt, between July 2013 and July 2019. Two of them had Hunt and Hess Grade V. Ten patients required insertion of ventriculostomy tube on admission before the definitive clipping surgery. In three patients, the intracranial pressure was extremely high and CSF release from basal cisterns and lamina terminalis was not possible except after inserting a ventriculostomy tube inside the lateral ventricle from Paine's point. These 15 patients were excluded from our study. The remaining 68 patients were included in our study where lamina terminalis fenestration was done during clipping surgery without other ventriculostomy. We divided our patients into two groups; Group A operated on between July 2013 and June 2016 with lamina terminalis opening but without inserting ventriculostomy tube during clipping surgery and Group B patients operated on between July 2016 and July 2019 with lamina terminalis opening and with inserting a translamina terminalis EVD tube during the clipping surgery.

Demographic information for patients in both groups are summarized in [Table 1]. There was no significant difference between patients in both groups as regard age or gender of patients, associated comorbidities, or locations of the ruptured aneurysm. [Table 2] shows clinical and radiological finding of patients in both groups. There was also no significant difference in the clinical grade, assessed by Hunt and Hess scale, of patients in both groups. There was also no significant difference in the CT finding of hydrocephalus or the amount of SAH, evaluated by Fisher's grading system, of patients of both groups. Furthermore, there was no

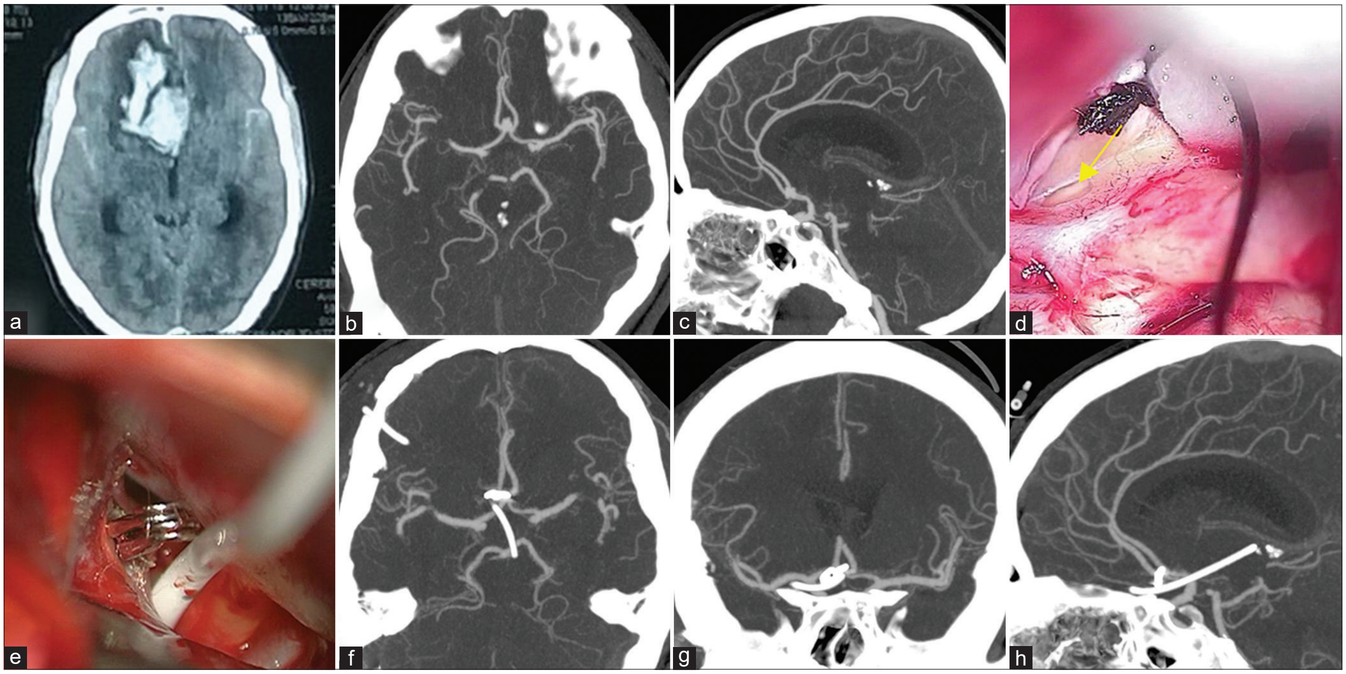

Figure 1: (a) computed tomographic (CT) axial scan, obtained at admission, demonstrating subarachnoid hemorrhage (SAH) and intracerebral hematoma (ICH) in a 54-year-old woman with Hunt and Hess Grade IV. CT angiogram (b) axial view (c) sagittal view demonstrating a small aneurysm at the anterior communicating artery. (d) intraoperative microscopic image showing fenestrated lamina terminalis (arrow). (e) intraoperative microscopic image showing the catheter inserted inside the fenestrated lamina terminalis after clipping of the ruptured aneurysm. Postoperative CT angiogram (f) axial view (g) coronal view (h) sagittal view demonstrating the tip of the catheter in the third ventricle.

significant difference in timing of surgery for patients in both groups.

Group A comprised 29 patients; 18 females and 11males, their age ranged from 30 to 65 years with a mean age of 47.8 years, 8 patients were smokers, 10 were hypertensive, 12 patients had diabetes mellitus, and 1 case had positive family history of aneurysmal subarachnoid hemorrhage [Table 1].

On admission, 7 patients had H&H classification Grade II, 15 patients Grade III, and 7 patients Grade IV. Admission brain CT showed subarachnoid hemorrhage in all patients, hydrocephalus in 5 patients, intracerebral hematoma in 16 patients, and intraventricular hemorrhage in 14 patients. The location of the ruptured IA, in this group, was clearly defined by preoperative cerebral CTA. The ruptured aneurysm was located on the anterior communicating artery in 14 patients, on the MCA in 9 patients, the posterior communicating segment of ICA in 3 patients, on the ophthalmic segment of the ICA in 2 patients, and on the ICA bifurcation in 1 patient [Table 2].

Most patients in this group underwent clipping surgery in the early period after aneurysm rupture. Seventeen patients were operated on within 24 h after aneurysm rupture, 7 patients in the 2nd day after rupture, and 5 patients were operated on late because of delayed referral and/or bad general condition at admission. In all patients, in this group, the lamina terminalis was fenestrated early in the operation with no associated complication [Table 2].

During the early, the first 21 postoperative days, follow-up: nine patients had hydrocephalus and conventional VP shunt was inserted, two patients died (one from vasospasm and one from ventilator associated pneumonia), one patient had hemiparesis, and another patient was aphasic. Twenty-seven patients were scheduled for 6 months clinical and radiological follow-up. By the end of the 6 months follow-up period, two patients were lost, 7 patients developed hydrocephalus, and a ventriculoperitoneal shunt was inserted.

Group B comprised 39 patients; 22 females and 17 males, their age ranged from 28 to 66 years with a mean age of 46.6 years, 12 patients were smokers, 15 were hypertensive, 14 had diabetes mellitus, and 1 case had positive family history of aneurysmal subarachnoid hemorrhage [Table 1].

On admission, 10 patients had H&H classification Grade II, 18 patients Grade III, and 11 patients Grade IV. Admission brain CT showed subarachnoid hemorrhage in all patients, hydrocephalus in 7 patients, intracerebral hematoma in 16 patients, and intraventricular hemorrhage in 22 patients. The location of the ruptured IA, in this group, was clearly defined by preoperative cerebral CTA. The ruptured aneurysm was located on the anterior

Table 1: Demographic information of patients in both groups.

	Group A (29 patients)		Group B (39 patients)		*P* value
Mean age of patients, years (range)	47.8	(30–65)	46.6	(28–66)	0.683
Gender					0.645
Men, *n* (%)	11	(37.9)	17	(43.6)	
Women, *n* (%)	18	(62.1)	22	(56.4)	
Family history and comorbidities					
Hypertension (%)	10	(34.5)	15	(38.5)	0.741
Diabetes mellitus (%)	12	(41.4)	14	(35.9)	0.651
Smoking (%)	8	(27.6)	12	(30.8)	0.728
Family history of SAH (%)	1	(3.4)	1	(2.6)	0.834

Table 2: Clinical and radiological variables on admission in both groups.

	Group A (29 patients) (%)		Group B (39 patients) (%)		*P* value
Hunt and Hess grade, *n* (%)					0.886
II	7	(24.1)	10	(25.6)	
III	15	(51.7)	18	(46.2)	
IV	7	(24.1)	11	(28.2)	
Fisher grade, *n* (%)					0.444
I	1	(3.4)	0		
II	7	(24.1)	8	(20.5)	
III	2	(6.9)	6	(15.4)	
IV	19	(65.5)	25	(64.1)	
Hydrocephalus, *n* (%)					0.852
Present	5	(17.2)	7	(17.9)	
Absent	24	(82.8)	32	(82.1)	
Intracerebral hematoma					0.248
Present	16	(55.2)	16	(41)	
Absent	13	(44.8)	23	(59)	
Intraventricular hemorrhage					0.506
Present	14	(48.3)	22	(56.4)	
Absent	15	(51.7)	17	(43.6)	
Timing of surgery, *n* (%)					0.958
24 h	17	(58.6)	26	(66.7)	
24 h–48 h	7	(24.1)	8	(20.5)	
Late	5	(17.2)	5	(12.8)	
Aneurysm location					0.711
MCA	9	(31)	15	(38.5)	
ACom	14	(48.3)	15	(38.5)	
Carotid bifurcation	1	(3.4)	2	(5.1)	
PCom	3	(10.3)	5	(12.8)	
Ophthalmic	2	(6.9)	2	(5.1)	

ACom: Anterior communicating artery, MCA: Middle cerebral artery, PCom: Posterior communicating artery

communicating artery in 15 patients, on the MCA in 15 patients, the posterior communicating segment of ICA in 5 patients, on the ophthalmic segment of the ICA in 2 patients, and on the ICA bifurcation in 2 patients [Table 2].

Most patients in this group underwent clipping surgery in the early period after aneurysm rupture. Twenty-six patients were operated on within 24 h after aneurysm rupture, 8 patients in the 2nd day after rupture, and 5 patients were operated on late because of delayed referral and/or bad general condition at admission. In all patients, in this group, a translamina terminalis, EVD tube was inserted regardless presence or absence of hydrocephalus. We encountered no complication related to lamina terminalis fenestration or tube insertion.

During the early, the first 21 postoperative days, follow-up: eight patients had positive CSF findings for infection during daily CSF analysis and were treated by systemic and intraventricular antibiotic injection. Infection subsided in all patients. Eight patients developed hydrocephalus; the tube was obstructed by blood clots in two patients and slipped in two patients. Three patients failed weaning with evidence of neurologic decline and ventriculomegaly during tube occlusion. One patient developed hydrocephalus and neurologic decline 2 days after tube removal in the 12th postoperative day. Conventional VP shunt was ultimately inserted for those eight patients. One patient had lower limb monoparesis, another patient had 3rd nerve palsy, and one patient was aphasic. Two patients died (one from cardiopulmonary problems and one from electrolyte disturbances). Thirty-seven patients were scheduled for 6 months clinical and radiological follow-up. By the end of the 6 months follow-up period, two patients were lost, four patients developed hydrocephalus, and a ventriculoperitoneal shunt was inserted.

We used the modified Rankin score to document clinical outcome of our patients; Group A included 10 patients symptom free (Grade 0), seven patients Grade 1, three patients Grade 2, two patients Grade 3, and three patients Grade 5, while Group B included 18 patients Grade 0, 12 patients Grade 1, one patient Grade 2, two patients Grade 3, and two patients Grade 5. Two cases were lost from follow-up in each group and two cases have expired during their hospital stay in each group. Statistically, there was no significant difference in outcome of both groups. The incidence of permanent VP shunt dependence was significantly lower in Group B than in Group A patients (30.8% vs. 55.2%, respectively) [Table 3].

DISCUSSION

Hydrocephalus is a common finding with aSAH and a major contributor to the occurrence of vasospasm with subsequent poor clinical outcomes.[9] The origin of development of early hydrocephalus after aneurysmal subarachnoid hemorrhage may be increased CSF viscosity causing communicating hydrocephalus or obstruction of CSF pathway by thick blood clots. Hydrocephalus can also develop after latent period due to arachnoid scarring decreasing CSF absorption and/or causing its pathway obstruction. All efforts should be directed to treat and prevent hydrocephalus to improve clinical outcome.[18] The incidence of late hydrocephalus and shunt dependence following aSAH in the literature is highly variable.[7,9,16,18,21,36,38,41,43,45]

Table 3: Outcome at the end of 6 months follow-up

	Group A (29 patients) (%)		Group B (39 patients) (%)		*P* value
Outcome					0.253
Modified ranking score					
0	10	(34.5)	18	(46.2)	
1	7	(24.1)	12	(30.8)	
2	3	(10.3)	1	(2.6)	
3	2	(6.9)	2	(5.1)	
5	3	(10.3)	2	(5.1)	
Mortality, *n* (%)	2	(6.9)	2	(5.1)	
Lost from follow-up, *n* (%)	2	(6.9)	2	(5.1)	
Permanent VP shunts insertion	16	(55.2)	12	(30.8)	0.043

The lamina terminalis is a thin sheet of gray matter and pia mater stretches upward between the optic chiasm and rostrum of the corpus callosum. It forms the anterior wall of the third ventricle.[50] Its opening allows entrance to the third ventricle and release of significant amount of CSF, this facilitates aneurysm dissection without excessive brain retraction.[42] Microscopic connection of the third ventricle to the basal cisterns through the lamina terminalis in its anterior wall is similar to connection of the third ventricle to subarachnoid spaces through its floor through endoscopic third ventriculostomy and can be considered a solution for hydrocephalus.[22] The effect of lamina terminalis fenestration on reducing the incidence of development of chronic hydrocephalus after aSAH has been supported by some reports and refused by others.[3,5,12,16,22,29,44]

The cornerstone in operating on ruptured IAs is to have slack brain for minimal brain retraction at the same time of maintaining adequate cerebral perfusion. In some situations of massive subarachnoid hemorrhage (SAH) or presence of a large space-occupying intracerebral hematoma with angry brain and awkward neuroanesthetic measures, CSF release is the way of choice to decrease the intracranial pressure and minimize brain retraction.[37] CSF can be released from the lumbar subarachnoid space through an external lumbar drainage. It can also be released from the basal cisterns around the optic nerve and internal carotid artery. All these maneuvers may still be insufficient in some situations of angry brain.[3]

We used to open the lamina terminalis in all cases of ruptured anterior circulation aneurysms treated through pterional or lateral supraorbital approaches. We believe this allows release of significant amount of CSF from the third ventricle rendering the brain lax and facilitating aneurysm dissection and clipping with minimal brain retraction. Furthermore, it is considered similar to endoscopic third ventriculostomy and helps in management of hydrocephalus in the acute stage. Since July 2016, we started to leave a silicon tube inside the third ventricle through the fenestrated lamina terminalis and connect to EVD system to wash bloody CSF from the ventricles to outside of the body aiming to ultimately decrease arachnoid scarring. We typically open basal cisterns around the carotid artery and optic nerves then fenestrate the lamina terminalis early before starting dissection of the aneurysm for clipping. In one case of ruptured anterior communicating aneurysm, the dome was projecting downward into the lamina terminalis. Hence, lamina terminalis fenestration was performed after securing the aneurysm. The lamina terminalis tube, when used, was always inserted after final clipping of the aneurysm not to obscure the operative field.

The technique of lamina terminalis ventriculostomy during microsurgery for ruptured IAs has provoked some criticism. It was claimed that a conventional ventriculostomy through Kocher burr hole before surgery or intraoperative ventriculostomy through the Paine's point would be more reasonable to avoid retraction of swollen brain, especially when there is no need to approach the midline. The big advantage of lamina terminalis ventriculostomy is that it is done under direct vision which avoids all possible complications of other blind ventriculostomy techniques. We also avoid performing extra burr holes and the tube can be inserted safely and CSF drained regardless the size of the ventricle. Furthermore, frontal lobe is retracted for only short period till lamina terminalis is reached and there was no complication from this brief retraction.[24,28]

Lehto *et al*.[24] reported their experience with insertion of lamina terminalis tube during microsurgery of 78 patients with ruptured anterior circulation aneurysms. They had no procedure-related complications, about 10% of their patients needed revision because of either tube obstruction (7 patients) or tube dislodgement (one patient). They documented positive CSF culture for 7 patients (9%) who needed antibiotic treatment. Twenty-two patients (28%) in their series required a permanent shunt.

The results of Lehto *et al*.[24] are close to the results of lamina terminalis tube insertion in Group B of our patients. Although we used the regular ventricular catheter which is thicker and more rigid than the special thin catheter they used, we did not have any complication related to lamina terminalis tube insertion. We had higher rate of positive CSF culture (21%) despite our routine use of prophylactic antibiotics while they did not use antibiotic prophylaxis. This might be related to higher temperature and humidity in our country. About 18% of our patients required revision and ultimate shunt insertion in the early postoperative period because of tube obstruction in two patients, tube dislodgement in two patients, failed weaning in three patients, and hydrocephalus in one patient 2 days after tube removal. About 31% of our patients ultimately needed permanent CSF shunt which is very close to results of their series (28%).

In the literature, there is broad variation in the incidence of ventriculostomy-related infection ranging from 1% to 27%. There is also wide difference in management strategies. Among these strategies are the catheter exchange which cannot be simply done for the lamina terminalis ventriculostomy tube, while it can be simply done for conventional ventriculostomy tubes. The reported data do not justify this catheter exchange for infection prophylaxis as it does not reduce the risk of CSF infection.[4,6,10,14,20,25-27,34,35,39,40,48,52] We used to give prophylactic systemic antibiotic to all our patients with ventriculostomy and when CSF culture is positive, we start intraventricular antibiotic injection through the catheter. We did not have neurological sequelae or death related to catheter infection.

Cerebral CTA was our imaging modality of choice for diagnosis and surgical planning in this series. We believe that multislice helical CTA is more practical than digital subtraction angiography because it is highly sensitive, easily available at all times, less time consuming and is less invasive. CT images can easily demonstrate hydrocephalus, intracerebral hematoma, intraventricular hemorrhage, and even calcification in the vessel wall or the aneurysm. The 3D reconstruction helps to show the surgical view and aneurysm relation to bony landmarks as the anterior clinoid process.

There have been some features reported to be associated with increased incidence of development of chronic hydrocephalus after aSAH including acute stage hydrocephalus, poor Fisher's grade, and poor Hunt and Hess grade.[3,16,22,36,41-43,45] We had no significant difference ($P > 0.05$) between both groups regarding these factors which might influence the rate of development of chronic hydrocephalus [Table 2]. This strongly suggests that the difference in incidence of chronic hydrocephalus between both groups was related to the effect of lamina terminalis EVD. In Group A, 7 patients (43.8%), of the 16 cases who passed the acute stage without shunt insertion and who were available at the end of 6 months follow-up, developed hydrocephalus and permanent shunt was inserted. In Group B, 4 patients (14.8%), of the 27 cases who passed the acute stage without shunt insertion and who were available at the end of 6 months follow-up, developed hydrocephalus and permanent shunt was inserted. Insertion of lamina terminalis tube was associated with statistically significant ($P < 0.05$) lower incidence of late hydrocephalus. Finally, Group B patients had statistically significant ($P < 0.05$) lower incidence of chronic shunt-dependent hydrocephalus than Group A patients (30.8% vs. 55.2%, respectively) [Table 3].

Reviewing the literature regarding the histopathological basis[8,21,51], clinical and radiological predictors[11,15,18,19,30,33,36,41,47] of chronic hydrocephalus after aSAH suggest that the disease is complex and multifactorial requiring innovations to prevent and treat hydrocephalus without resorting to CSF shunting. Although there have been some reports suggesting that some drugs as cilostazol[31] or some surgical maneuvers as fenestration of lamina terminalis[5,22,29] or tandem fenestration of lamina terminalis and Lilliquist membrane[3,42] can reduce the development chronic shunt-dependent hydrocephalus after aSAH, the situation is still challenging. We believe that some extra few minute consuming steps, during open microsurgical clipping, to reduce the clot burden and to accelerate drainage of subarachnoid blood products, could decrease the rate of chronic shunt-dependent hydrocephalus after aSAH without exposing the patients to more complications.

Limitations

The results of the present work must be interpreted in the light of small number of cases and the retrospective nature of the study. Prospective studies with larger number of cases are recommended to overcome these limitations in forthcoming research.

CONCLUSION

EVD through fenestrated lamina terminalis is safe and may be effective in decreasing the incidence of chronic shunt-dependent hydrocephalus after aSAH.

Declaration of patient consent

Patient's consent not required as patients identity is not disclosed or compromised.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Adams H, Ban VS, Leinonen V, Aoun SG, Huttunen J, Saavalainen T, *et al*. Risk of shunting after aneurysmal subarachnoid hemorrhage: A collaborative study and initiation of a consortium. Stroke 2016;47:2488-96.
2. Adil SM, Liu B, Charalambous LT, Kiyani M, Gramer R, Swisher CB, *et al*. Healthcare economics of hydrocephalus after aneurysmal subarachnoid hemorrhage in the United States. Transl Stroke Res 2019;10:650-63.
3. Akyuz M, Tuncer R. The effects of fenestration of the interpeduncular cistern membrane arousted to the opening of lamina terminalis in patients with ruptured ACoA aneurysms: A prospective, comparative study. Acta Neurochir (Wien) 2006;148:725-3.
4. Alleyne CH Jr., Hassan M, Zabramski JM. The efficacy and cost of prophylactic and perioprocedural antibiotics in patients with external ventricular drains. Neurosurgery 2000;47:1124-7.
5. Andaluz N, Zuccarello M. Fenestration of the lamina terminalis as a valuable adjunct in aneurysm surgery. Neurosurgery 2004;55:1050-9.
6. Arabi Y, Memish ZA, Balkhy HH, Francis C, Ferayan A, Al Shimemeri A, *et al*. Ventriculostomy-associated infections: Incidence and risk factors. Am J Infect Control 2005;33:137-43.
7. Auer LM, Mokry M. Disturbed cerebrospinal fluid circulation after subarachnoid hemorrhage and acute aneurysm surgery. Neurosurgery 1990;26:804-8.
8. Aydin MD, Kanat A, Turkmenoglu ON, Yolas C, Gundogdu C, Aydin N. Changes in number of water-filled vesicles of choroid plexus in early and late phase of experimental rabbit subarachnoid hemorrhage model: The role of petrous ganglion of glossopharyngeal nerve. Acta Neurochir (Wien) 2014;156:1311-7.
9. Black PM. Hydrocephalus and vasospasm after subarachnoid hemorrhage from ruptured intracranial aneurysms. Neurosurgery 1986;18:12-6.
10. Bota DP, Lefranc F, Vilallobos HR, Brimioulle S, Vincent JL. Ventriculostomy-related infections in critically ill patients: A 6-year experience. J Neurosurg 2005;103:468-72.
11. Chang SI, Tsai MD, Yen DH, Hsieh CT. The clinical predictors of shunt-dependent hydrocephalus following aneurysmal subarachnoid hemorrhage. Turk Neurosurg 2018;28:36-42.
12. Chohan MO, Carlson AP, Hart BL, Yonas H. Lack of functional patency of the lamina terminalis after fenestration following clipping of anterior circulation aneurysms. J Neurosurg 2013;119:629-33.
13. Connolly ES Jr., Rabinstein AA, Carhuapoma JR, Derdeyn CP, Dion J, Higashida RT, *et al*. Guidelines for the management of aneurysmal subarachnoid hemorrhage: A guideline for healthcare professionals from the American heart association/American stroke association. Stroke 2012;43:1711-37.
14. Dasic D, Hanna SJ, Bojanic S, Kerr RS. External ventricular drain infection: The effect of a strict protocol on infection rates and a review of the literature. Br J Neurosurg 2006;20:296-300.
15. de Oliveira JG, Beck J, Setzer M, Gerlach R, Vatter H, Seifert V, *et al*. Risk of shunt-dependent hydrocephalus after occlusion of ruptured intracranial aneurysms by surgical clipping or endovascular coiling: A single-institution series and meta-analysis. Neurosurgery 2007;61:924-33.
16. Dehdashti AR, Rilliet B, Rufenacht DA, de Tribolet N. Shunt-dependent hydrocephalus after rupture of intracranial aneurysms: A prospective study of the influence of treatment modality. J Neurosurg 2004;101:402-7.
17. Di Russo P, Di Carlo DT, Lutenberg A, Morganti R, Evins AI, Perrini P. Shunt-dependent hydrocephalus after aneurysmal subarachnoid hemorrhage. A systematic review and meta-analysis. J Neurosurg Sci 2019;64:181-9.
18. Dorai Z, Hynan LS, Kopitnik TA, Samson D. Factors related to hydrocephalus after aneurysmal subarachnoid hemorrhage. Neurosurgery 2003;52:763-9.
19. Erixon HO, Sorteberg A, Sorteberg W, Eide PK. Predictors of shunt dependency after aneurysmal subarachnoid hemorrhage: Results of a single-center clinical trial. Acta Neurochir (Wien) 2014;156:2059-69.
20. Flibotte JJ, Lee KE, Koroshetz WJ, Rosand J, McDonald CT. Continuous antibiotic prophylaxis and cerebral spinal fluid infection in patients with intracranial pressure monitors. Neurocrit Care 2004;1:61-8.

21. Kanat A, Turkmenoglu O, Aydin MD, Yolas C, Aydin N, Gursan N, *et al*. Toward changing of the pathophysiologic basis of acute hydrocephalus after subarachnoid hemorrhage: A preliminary experimental study. World Neurosurg 2013;80:390-5.
22. Komotar RJ, Olivi A, Rigamonti D, Tamargo RJ. Microsurgical fenestration of the lamina terminalis reduces the incidence of shunt-dependent hydrocephalus after aneurysmal subarachnoid hemorrhage. Neurosurgery 2002;51:1403-12.
23. Koyanagi M, Fukuda H, Saiki M, Tsuji Y, Lo B, Kawasaki T, *et al*. Effect of choice of treatment modality on the incidence of shunt-dependent hydrocephalus after aneurysmal subarachnoid hemorrhage. J Neurosurg 2018;130:949-55.
24. Lehto H, Dashti R, Karatas A, Niemela M, Hernesniemi JA. Third ventriculostomy through the fenestrated lamina terminalis during microneurosurgical clipping of intracranial aneurysms: An alternative to conventional ventriculostomy. Neurosurgery 2009;64:430-4.
25. Lo CH, Spelman D, Bailey M, Cooper DJ, Rosenfeld JV, Brecknell JE. External ventricular drain infections are independent of drain duration: An argument against elective revision. J Neurosurg 2007;106:378-83.
26. Lozier AP, Sciacca RR, Romagnoli MF, Connolly ES Jr. Ventriculostomy-related infections: A critical review of the literature. Neurosurgery 2002;51:170-81.
27. Lyke KE, Obasanjo OO, Williams MA, O'Brien M, Chotani R, Perl TM. Ventriculitis complicating use of intraventricular catheters in adult neurosurgical patients. Clin Infect Dis 2001;33:2028-33.
28. Maniker AH, Vaynman AY, Karimi RJ, Sabit AO, Holland B. Hemorrhagic complications of external ventricular drainage. Neurosurgery 2006;59 Suppl 2:ONS419-24.
29. Mao J, Zhu Q, Ma Y, Lan Q, Cheng Y, Liu G. Fenestration of lamina terminalis during anterior circulation aneurysm clipping on occurrence of shunt-dependent hydrocephalus after aneurysmal subarachnoid hemorrhage: Meta-analysis. World Neurosurg 2019;129:e1-5.
30. Mijderwijk HJ, Fischer I, Zhivotovskaya A, Bostelmann R, Steiger HJ, Cornelius JF, *et al*. Prognostic model for chronic shunt-dependent hydrocephalus after aneurysmal subarachnoid hemorrhage. World Neurosurg 2019; 124:e572-e579.
31. Nakatsuka Y, Kawakita F, Yasuda R, Umeda Y, Toma N, Sakaida H, *et al*. Preventive effects of cilostazol against the development of shunt-dependent hydrocephalus after subarachnoid hemorrhage. J Neurosurg 2017;127:319-26.
32. O'Kelly CJ, Kulkarni AV, Austin PC, Urbach D, Wallace MC. Shunt-dependent hydrocephalus after aneurysmal subarachnoid hemorrhage: Incidence, predictors, and revision rates. Clinical article. J Neurosurg 2009;111:1029-35.
33. Paisan GM, Ding D, Starke RM, Crowley RW, Liu KC. Shunt-dependent hydrocephalus after aneurysmal subarachnoid hemorrhage: Predictors and long-term functional outcomes. Neurosurgery 2018;83:393-402.
34. Park P, Garton HJ, Kocan MJ, Thompson BG. Risk of infection with prolonged ventricular catheterization. Neurosurgery 2004;55:594-9.
35. Pfisterer W, Muhlbauer M, Czech T, Reinprecht A. Early diagnosis of external ventricular drainage infection: Results of a prospective study. J Neurol Neurosurg Psychiatry 2003;74:929-32.
36. Pietila TA, Heimberger KC, Palleske H, Brock M. Influence of aneurysm location on the development of chronic hydrocephalus following SAH. Acta Neurochir (Wien) 1995;137:70-3.
37. Randell T, Niemela M, Kytta J, Tanskanen P, Maattanen M, Karatas A, *et al*. Principles of neuroanesthesia in aneurysmal subarachnoid hemorrhage: The Helsinki experience. Surg Neurol 2006;66:382-8.
38. Saliou G, Paradot G, Gondry C, Bouzerar R, Lehmann P, Meyers ME, *et al*. A phase-contrast MRI study of acute and chronic hydrodynamic alterations after hydrocephalus induced by subarachnoid hemorrhage. J Neuroimaging 2012;22:343-50.
39. Sandalcioglu IE, Stolke D. Failure of regular external ventricular drain exchange to reduce CSF infection. J Neurol Neurosurg Psychiatry 2003;74:1598-9.
40. Schade RP, Schinkel J, Visser LG, Van Dijk JM, Voormolen JH, Kuijper EJ. Bacterial meningitis caused by the use of ventricular or lumbar cerebrospinal fluid catheters. J Neurosurg 2005;102:229-34.
41. Sheehan JP, Polin RS, Sheehan JM, Baskaya MK, Kassell NF. Factors associated with hydrocephalus after aneurysmal subarachnoid hemorrhage. Neurosurgery 1999;45:1120-7.
42. Sindou M. Favourable influence of opening the lamina terminalis and Lilliequist's membrane on the outcome of ruptured intracranial aneurysms. A study of 197 consecutive cases. Acta Neurochir (Wien) 1994;127:15-6.
43. Tapaninaho A, Hernesniemi J, Vapalahti M, Niskanen M, Kari A, Luukkonen M, *et al*. Shunt-dependent hydrocephalus after subarachnoid haemorrhage and aneurysm surgery: Timing of surgery is not a risk factor. Acta Neurochir (Wien) 1993;123:118-24.
44. Tomasello F, d'Avella D, de Divitiis O. Does lamina terminalis fenestration reduce the incidence of chronic hydrocephalus after subarachnoid hemorrhage? Neurosurgery 1999;45:827-31.
45. Vale FL, Bradley EL, Fisher WS 3rd. The relationship of subarachnoid hemorrhage and the need for postoperative shunting. J Neurosurg 1997;86:462-6.
46. Wessell AP, Kole MJ, Cannarsa G, Oliver J, Jindal G, Miller T, *et al*. A sustained systemic inflammatory response syndrome is associated with shunt-dependent hydrocephalus after aneurysmal subarachnoid hemorrhage. J Neurosurg 2019;130:1984-91.
47. Wilson CD, Safavi-Abbasi S, Sun H, Kalani MY, Zhao YD, Levitt MR, *et al*. Meta-analysis and systematic review of risk factors for shunt dependency after aneurysmal subarachnoid hemorrhage. J Neurosurg 2017;126:586-95.
48. Wong GK, Poon WS, Wai S, Yu LM, Lyon D, Lam JM. Failure of regular external ventricular drain exchange to reduce cerebrospinal fluid infection: Result of a randomised controlled trial. J Neurol Neurosurg Psychiatry 2002;73:759-61.
49. Yamada S, Ishikawa M, Yamamoto K, Ino T, Kimura T, Kobayashi S, *et al*. Aneurysm location and clipping versus coiling for development of secondary normal-pressure

hydrocephalus after aneurysmal subarachnoid hemorrhage: Japanese stroke data bank. J Neurosurg 2015;123:1555-61.
50. Yasargil MG. Microneurosurgery, Volume I: Microsurgical Anatomy of the Basal Cisterns and Vessels of the Brain, Diagnostic Studies, General Operative Techniques and Pathological Considerations of the Intracranial Aneurysms. Germany: Thieme; 1984.
51. Yolas C, Ozdemir NG, Kanat A, Aydin MD, Keles P, Kepoglu U, *et al*. Uncovering a new cause of obstructive hydrocephalus following subarachnoid hemorrhage: Choroidal artery vasospasm-related ependymal cell degeneration and aqueductal stenosis-first experimental study. World Neurosurg 2016;90:484-91.
52. Zabramski JM, Whiting D, Darouiche RO, Horner TG, Olson J, Robertson C, *et al*. Efficacy of antimicrobial-impregnated external ventricular drain catheters: A prospective, randomized, controlled trial. J Neurosurg 2003;98:725-30.

How to cite this article: Elsharkawy AA, Abdelhameed EA. Efficacy of translamina terminalis ventriculostomy tube in prevention of chronic hydrocephalus after aneurysmal subarachnoid hemorrhage. Surg Neurol Int 2020;11:283.