

Surgical Experience of Anterior Circulation Aneurysms at a Tertiary Care Center.

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Abstract: Internal carotid circulation aneurysms constitutes 85% of all intracranial aneurysms. The close relationship of the internal carotid artery and its branches with the brain and cranial nerves, and the distribution of the flow of internal carotid artery, make these aneurysms difficult, and complicated in view of their surgical treatment. Great attention to detail, and clear knowledge of the microsurgical anatomy of the carotid arteries, helps the safe and successful surgical management of these aneurysms. We present our surgical experience with 50 anterior circulation aneurysms treated from November 2017 to March 2019. The aneurysms arose from the ICA: 13(26%), MCA: 20(40%), and ACA: 17(34%). The mean GCS of patients subjected to clipping was 13. The mean duration between ictus and surgery was 3.8 days. The WFNS grades on admission were I(29), II(07), III(06), IV(08). The Fisher grades were Grade I(01), II(35), III(11), IV(03). Serious neurological morbidity was observed in 6(12%) patients. Mortality occurred in 7(14%). Surgical clipping of internal carotid aneurysms can be conducted safely when the anatomical characteristics of the intracranial vessels are preserved. Poor WFNS and Fisher grades appear to be associated with poor outcome. Early surgery can be carried out without devastating complications.

Keywords: Anterior cerebral artery(ACA), glasgow coma scale(GCS), internal cerebral artery(ICA), middle cerebral artery(MCA), world federation of neurosurgical societies(WFNS) grade

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I. Introduction

The internal carotid circulation aneurysms represent 85% of all intracranial aneurysms [1]. The close relationship of the internal carotid artery and its branches with the brain and cranial nerves, and the distribution of the flow of internal carotid arteries, make these aneurysms difficult, and potentially complicated. Great attention to detail, and clear knowledge of the microsurgical anatomy of the carotid system, facilitates the safe and successful management of these aneurysms. The pertinent surgical anatomy, preoperative preparation, intraoperative procedures, operative approaches, and potential complications will be reviewed and presented.

II. MATERIAL AND METHODS

The data of 50 patients who were diagnosed as anterior circulation aneurysms from November 2017 to March 2019 in GGH Guntur was analyzed on the basis of GCS, WFNS grading, Fisher grading, timing between the ictus and surgery retrospectively. Medical chartings, angiographic studies, magnetic resonance imaging scans, computed tomographic, and CT angiography scans were carefully reviewed. Patients' age, sex, Fisher grades, WFNS grades, and aneurysm locations were recorded. All diagnoses were made on CT angiography which was our main modality of investigation. In case of doubt, patients underwent MRI angiography, and/or digital subtraction angiography(DSA) to confirm the aneurysm. The surgical planning was done using CT angiography in all cases. The aneurysms were seen to be arising from the internal carotid artery: 13(26%), middle cerebral artery: 20(40%), and anterior cerebral artery: 17(34%). Patients who had subarachnoid hemorrhage were admitted to an intensive care unit for evaluation, and in preparation for surgery.¹ A complete preoperative medical evaluation was obtained in all patients to prevent intra-operative cardiac, respiratory, or renal complications that may develop from the use of mannitol, hypertension, hypotension, blood loss, or the use of cardiac suppressants such as barbiturates or lidocaine. All patients were given nimodipine 1 mg per kg orally 4 hourly until surgery. The mean Glasgow Coma Scale of patients subjected to clipping was 13. The mean duration between ictus and surgery was 3.8 days. The WFNS grades on admission were I(29), II(07), III(06), IV(08). The Fisher grades were Grade I(01), II(35), III(11), IV(03).

During induction, the patients were given intravenous phenytoin 15 mg per kg, ceftriaxone 1000 mg, and mannitol 1.5 gm per kg. Drainage of cerebrospinal fluid from the cisterns was effective in achieving cerebral relaxation during the subarachnoid dissection. Arterial blood gases, end-expiratory CO₂ recordings, and pulse oximetry were monitored throughout the operation. Arterial pCO₂ was maintained at about 30-35 mmHg, pO₂ at about 95 mmHg, and oxygen saturation at about 100%. The patients were maintained normothermic and normotensive throughout the operation. Intra-operative hypotension was only used when proximal arterial control could not be accomplished with a temporary clipping.

Surgical clipping was done in all cases as we don't have facility for endovascular procedures. Pterional approach was used in all cases of aneurysms. Intraoperatively temporary clips were used occasionally to prevent the rupture of the aneurysm. Accordingly, the aneurysm was delineated and clipped using Sugita aneurysms clips whose sizes were planned according to neck length measured pre-operatively. Lamina terminalis was fenestrated, especially in all cases of anterior communicating artery aneurysms to prevent hydrocephalus at the end of procedures. Thorough wash was given with normal saline after opening of lamina terminalis to remove any clots in all cases. [2]. After surgery, patients were monitored in the Neuro Intensive Care Unit and triple H therapy, and nimodipine was given for the prevention of cerebral vasospasm. Patients returned to neurosurgical wards and discharged to home or rehabilitation centers according to their neurological status. A follow-up brain CT was done on the 1st postoperative day to see the status of brain and take necessary management measures for prevention of any complications. (Figs. 1, 2).

The outcome of the patients was assessed 6 weeks after the discharge from the hospital using the Glasgow Outcome Scale and was analyzed with various prognostic factors, including the post-operative vasospasm [13].

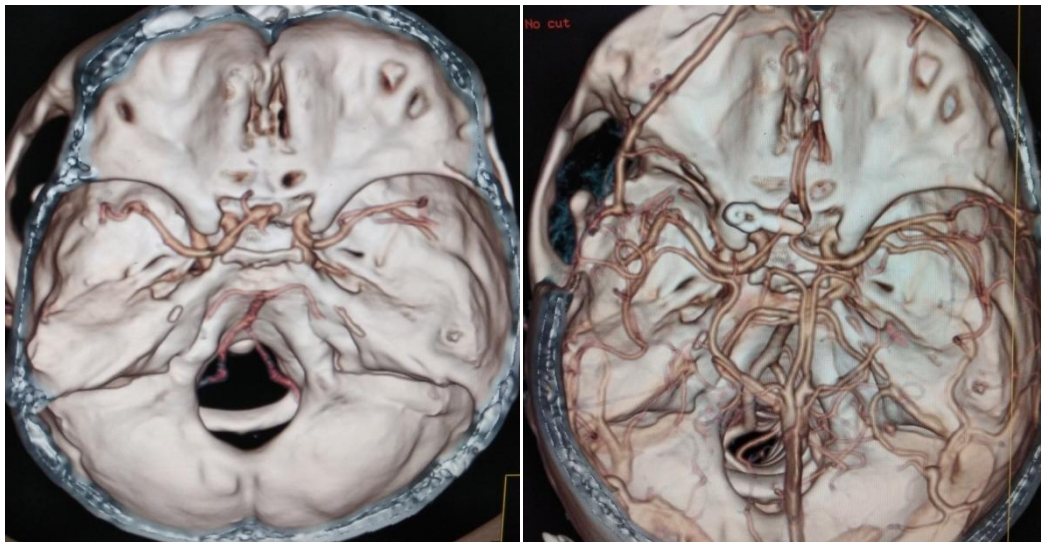


Figure 1: preoperative and postoperative images of Anterior communicating artery aneurysm



Figure 2: preoperative and postoperative images of internal carotid artery aneurysm

III. Results:

Between November 2017 and March 2019, 50 consecutive patients underwent surgical treatment for anterior circulation aneurysms. The mean age of the patients was 55.4 years. 62% of patients were women ($n = 31$) and 38% were men ($n = 19$). The mean WFNS grade at the time of admission was 1.76, with a standard deviation of 1.17, and the mean Fisher score was 2.43, with a standard deviation of 0.64. The aneurysms were seen arising from the internal carotid artery: 13(26%), middle cerebral artery: 20(40%), and anterior cerebral artery: 17(34%). Demographics characteristics and aneurysm locations are shown in Table 1.

In our series, aneurysm clipping was successfully achieved in all the patients. Major and minor neurological deficits related to clipping were 4% and 8%, respectively. Mortality occurred in 7(14%); these patients died from a hemispheric infarction (3), severe vasospasm (2), myocardial infarction (2). Hemorrhage related to surgery was observed in 4%. Cerebral vasospasm was observed in 7(14%) of the cases, out of which five patients recovered from vasospasm. 86% of the patients attained a favorable outcome (moderate, mild, or no disability). Only six patients (12%) had a poor clinical outcome. Overall, WFNS Grades IV and V, and Fisher grade V were associated with high risk for vasospasm and with poor clinical outcome. (Table 2)

In one case, a surgical site infection was seen and was managed by targeted antibiotic therapy. In another case, the postoperative seizure was treated by a combination of antiepileptic drugs.

One patient with anterior communicating artery aneurysm developed hydrocephalus post-operatively, for which VP shunt was done.

One patient had kinking of ICA due to clip (figure 2) which was identified on post-operative DSA and which could have been avoided if we had intra-operative Indocyanine green (ICG) facility at our center. Fortunately patient was asymptomatic and was discharged with no complications.

Table 1: Demographics characteristics and aneurysm location

Demographics characteristics	
Total patients (n)	50
Mean age (year, mean±SD)	55.4±8.1
Male	19
Female	31
Fisher (mean±SD)	2.43±0.64
WFNS (mean±SD)	1.76±1.17
Aneurysm location	
Anterior communicating	17
ICA	13
MCA	20
Total	50
SD: Standard deviation, MCA: Middle cerebral artery	

Table2: Outcome related to surgery

Total aneurysms	50
Favorable outcome (%)	86%
Poor clinical outcome (%)	14%
Hemorrhage related to surgery (%)	04%
Major neurological deficit (%)	04%
Minor neurological deficit (%)	08%
Cerebral vasospasm (%)	14%
Mortality	14%

IV. Discussion

Anatomical considerations

The internal carotid artery enters the skull through the foramen lacerum and becomes intracranial as it passes through the petrous canal into the cavernous sinus. Several small branches arise from the cavernous portion of the internal carotid, including the inferior hypophysial artery, the meningo-hypophysial trunk, and the artery to the clivus. As the internal carotid artery leaves the cavernous sinus, it traverses the subarachnoid space giving rise to the ophthalmic artery, the posterior communicating artery, the anterior choroidal artery, and terminates dividing into the anterior and middle cerebral arteries. For the purpose of this discussion, emphasis will be placed on the most proximal portion of the circle of Willis.

The internal carotid artery from the cavernous sinus to the bifurcation is variable in length, and in the number of branches, it gives rise to. Besides the ophthalmic artery, the posterior communicating and anterior choroidal arteries, there are branches directed to the anterior perforated substance, and in some cases, a proximal branch to the temporal pole or to the inferior frontal area [3,4,5,6]. The perforating branches generally arise from the posterior wall of the internal carotid, either before or after the bifurcation, not from the bifurcation itself. The perforating branches from the anterior cerebral artery may arise from a common trunk, or they may arise as several small branches directed to the medial portion of the anterior perforated substance. The anterior perforating arteries originating from the middle cerebral artery arise from a single common trunk or from several small perforating branches. These middle cerebral perforators may originate from the most medial portion of the artery, close to its origin (37%). or they may originate from the middle one-third of the M-1 segment (47%). The remaining (16%) perforating vessels arise from the most lateral portion of M-1. The pattern of arterial supply to the anterior perforated substance is one of dominance by either the anterior cerebral or the middle cerebral; the branches arising from the other artery will be smaller and fewer in number [5,6,8]. In a few cases, two large vessels may arise from the distal portion of the A-1 segment and go to the anterior perforated substance retrogradely, substituting for the recurrent artery of Huebner. When the territories of the anterior and middle cerebral perforators overlap, permanent occlusion of one perforator may not be of great importance and may go unnoticed clinically. However, when the perforating vessel is dominant, permanent occlusion of this artery may be followed by a severe and lasting neurological deficit.

As the anterior cerebral passes from the carotid bifurcation to the interhemispheric fissure, it gives rise to branches to the anterior perforated substance, the inferior frontal area, the frontopolar artery, the recurrent artery of Heubner, the septal perforating arteries and the anterior communicating artery. The anterior cerebral then continues in the interhemispheric fissure towards the convexity. The recurrent artery of Huebner has a variable origin [2,3,9]. In most cases (65%) it arises from the proximal A-2 segment immediately after the origin of the anterior communicating artery. In 25% of cases, the recurrent artery arises from the A-1 segment, proximal to the anterior communicating artery. In the remaining 10%, the recurrent artery originates at the level of the anterior communicating artery. The recurrent artery of Huebner and the septal perforators are the most important branches of the proximal portion of the anterior cerebral artery, supplying the anterior limb of the internal capsule, the anterior commissure, the septal region, the anterior portion of the fornix and hypothalamus, and the most anterior portion of the thalamus [2,3,9]. The middle cerebral artery continues in the Sylvian fissure and divides in two or three primary trunks, from which will originate the branches to the cerebral cortex.^{7,8} Particularly important are the branches to the rolandic area, the angular gyrus, and the opercular area. Damage to one of these branches is poorly tolerated. Every branch of the middle cerebral artery should be identified prior to permanent clip placement [10].

Veins from the frontal and temporal portions of the cerebral convexity drain into the Sylvian or middle cerebral veins, in the Sylvian fissure. The Sylvian vein is composed of several tributary veins that originate from the anterior frontal lobe, including the frontal pole, the orbital surface, the anterior opercular area, and the anterior and inferior portion of the insular lobe. Venous tributaries from the temporal lobe are variable and include branches from the temporal pole, the most anterior portion of the superior and mid temporal gyri, and the Sylvian portion of the temporal lobe. The frontal and temporal venous afferents join to form one or two primaries, middle cerebral or Sylvian venous trunks, but there can be as many as four or as few as one. The primary venous trunks drain directly into the sphenopetrosal sinus. The Sylvian veins are predominantly located on the temporal side of the Sylvian fissure and can be easily identified and spared. In most patients, the temporal tip veins may be coagulated and transected, but in some cases, damage to these veins may result in venous congestion, cerebral edema, or venous infarction.” Venous drainage from the cerebral convexity returns to the systemic circulation by several convexity veins; the two largest are the posterior temporal or vein of Labbe, which drains into the transverse sinus, and the mid parietal or vein of Trolard, draining into the superior sagittal sinus. Removing more than one vein in the mid and posterior portion of the superior sagittal sinus, or transecting the veins of Trolard or Labbe can result in significant venous stasis and venous infarction.

Yasargil[11] described the subarachnoid cisterns at the base of the brain as small envelopes that encircle all structures contained in the subarachnoid space. In the supraclinoid carotid area, there are cisterns about the internal carotid artery, the carotid bifurcation, the middle cerebral, the anterior cerebral, the optic nerve, the chiasm, the prechiasmatic cistern, the cistern of the lamina terminalis, and the confluence of the Sylvian fissure to the middle cerebral cistern. Wide opening and sharp dissection of these cisterns are required to expose internal carotid artery aneurysms safely[12].

The recent advances in endovascular intervention with the introduction of coils and flow diversion techniques in the last years has led to the treatment of cerebral aneurysms with endovascular intervention first whenever possible [14,15]. However, this may not be applicable in many centers in developing countries like India due to costly supplies that make surgical clipping the standard treatment modality for aneurysms.

The optimum and ideal timing of microsurgical clipping after aneurysmal subarachnoid hemorrhage was a matter of controversy in the last five decades [16]. With the introduction of microsurgical techniques in the 1960s and till the 1970s, the majority of neurosurgeons practiced late surgery 2 or 3 weeks after subarachnoid hemorrhage. They believed that surgery on edematous and vulnerable brain was dangerous and associated with a high rate of complications [16,17,18].

Whitfield and Kirkpatrick, in their Cochrane review in 2001, found only one randomized clinical trial on the timing of surgery after rupture of aneurysms [19]. In this study, patients with early surgery (days 0–3) had a good outcome, and patients with intermediate surgery (days 4–7) had a poor outcome [20]. A systemic review that included the above-mentioned study and another ten observational studies by de Gans and his colleagues [21] demonstrated that early and intermediate surgical clipping of aneurysms resulted in better outcomes than late clipping. Historically, this led to a popular belief that surgical clipping during the period of vasospasm, especially between days 5 and 10, is considered unsafe and associated with more risk of delayed cerebral ischemia [16,22].

However, several recently published studies mention that the outcome was not worse when compared to intermediate with the late clipping [16,19]. Dorhout Mees and colleagues evaluated the timing of aneurysm surgery after rupture of aneurysms in 2016 patients randomized for coiling or clipping within the International Subarachnoid Aneurysmal Trial and supported the current guidelines for early clipping of aneurysm in subarachnoid hemorrhage patients. The authors do not recommend late clipping in patients between days 5 and 10 after subarachnoid hemorrhage [16]. Orakdogan and colleagues evaluated the timing of surgery with several

prognostic factors in patients who underwent surgical clipping for ruptured cerebral aneurysms and observed that mortality rate was significantly higher in the day 0 period and the mortality rate did not differ significantly from the other stages of the disease [23]. The above-mentioned studies supported the practice of aneurysm treatment once diagnosed even in the period of vasospasm, as in our series.

Cerebral vasospasm remains a leading cause of delayed morbidity and mortality in patients with aneurysmal subarachnoid hemorrhage [24]. The severity of vasospasm is proportional to the quantity and duration of contact with subarachnoid hemorrhage [25,26]. In patients who survive the initial bleeding after rupture of the aneurysm, about 40 to 70% developed arterial narrowing with 20–30% manifested neurological deficits [26,27]. Prevention of vasospasm and the delayed cerebral ischemia after ruptured cerebral aneurysms is limited to specific drug treatment and manipulation of blood pressure, intravascular volume, and blood viscosity [28]. Surgical management allowed clot removal with irrigation of the basal cisterns, which theoretically may reduce the incidence of vasospasm [22,27].

In our series, we found that preoperative clinical grading was the most important prognostic factor, and patients with good WFNS and Fisher grades usually had a good outcome. Patients with symptomatic vasospasm after clipping were associated with the poor outcome with two patients who died from aggravated vasospasm. This finding is in agreement with other previously published studies investigating the prognostic factors after microsurgical clipping of cerebral aneurysms [20,23]. Orakdogan and others reported that WFNS grade, age, aneurysm size, and symptomatic vasospasm were the most important prognostic factors in patients undergoing surgical clipping after rupture of cerebral aneurysms [23]. Samaha et al. observed that preoperative neurological state and the occurrence of vasospasm were the main prognostic factors [29].

The limitations of our study are, it is retrospective, includes a small number of patients, evaluates the surgical outcome only and not the whole management for the patients with anterior circulation aneurysms, and evaluates the patients with the delayed cerebral ischemic deficit with brain CT. However, it shows that safe clipping during the period of vasospasm, which is a common practice in developing countries like India due to some logistic difficulties and economic burden. We performed clipping in all cases as we don't have facility for endovascular procedures. Our study demonstrates that patients should not be delayed till the end of the vasospasm period, and the ruptured aneurysm should be secured early whenever applicable to prevent rebleeding and allow treatment of vasospasm with triple H therapy safely. The better surgical outcome appears to be associated with early surgery, good patient's clinical grades, Fisher grades, and aggressive vasospasm management.

V. Conclusion

We found that microsurgical clipping of the anterior circulation aneurysms can be done safely during the period of vasospasm without devastating complications. The clipping of aneurysm in patients with late referral in developing countries should be done to prevent rebleeding and to allow for the triple H therapy application safely. WFNS grades I and II and Fisher grade I and II had a good prognosis after clipping. WFNS grade IV and V and Fisher grade IV patients had a higher tendency for risk of vasospasm. The better surgical outcome appears to be associated with early surgery, good patient's clinical grades, Fisher grades, and aggressive vasospasm management. Though a tertiary health care center, we are equipped with Moller Wedel (VM 500) operating microscope without intra-operative ICG facility, still our surgical outcome stands comparable with other institutes in India.

References:

- [1]. Anterior circulation aneurysms: surgical perspectives Diaz FG, Fessler RD, Velardo B, Kennedy C, Wilner H.J Clin Neurosci. 1994 Oct;1(4):222-30.
- [2]. 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.
- [3]. Varkey GP (ed) . Anesthetic Considerations in the Surgical Repair of Intracranial Aneurysms. International Anesthesiology Clinics. Boston: Little Brown, 1982. Gibo H, Lenkey C, Rhoton AL. Microsurgical anatomy of the supraclinoid portion of the internal carotid artery. J Neurosurg 1981;55:560-74.
- [4]. Gomes F, Dujovny M, Umansky F, AusmanJI, Diaz FG, Ray WJ, Mirchandani GF. Microsurgical anatomy of the recurrent artery of Heubner. J Neurosurg 1984;60:130-39.
- [5]. Comes FB, Dujovny M, Umansky F, Berman SK, Diaz FG, Ausman JI, Mirchandani HG, Ray WJ. Microanatomy of the anterior cerebral artery. Surg Neurol 1986;26:129-41.
- [6]. Grand W. Microsurgical anatomy of the proximal middle cerebral artery and internal carotid bifurcation. Neurosurgery 1980;7:215-18.
- [7]. Umansky F, Montoya Juarez S, Dujovny M, Ausman JI, Diaz FG, Gomes F, Mirchandani HG, Ray WJ. Microsurgical anatomy of the proximal segments of the middle cerebral artery. J Neurosurg 1984;61:458-67.
- [8]. Umansky F, Gomes FB, Dujovny M, Diaz FG, Ausman JI, Mirchandani HG, Berman SK. The perforating branches of the middle cerebral artery: A microanatomical study. J Neurosurg 1985;62:261-68.
- [9]. Yokoh A, Ausman JI, Dujovny M, Diaz FG, Berman SK, Sanders J, Mirchandani HG. Anterior cerebral artery reconstruction. Neurosurg 1986;19:26-35.

- [10]. Suzuki-J. Cerebral Aneurysms, Experience with 1000 Directly Operated Cases. Tokyo: Neuron Publishing Co, 1979.
- [11]. Yasargil MG, Antic J, Laciga R, Jain KK, Hodosh RM, Smith RD. Microsurgical pterional approach to aneurysms of the basilar bifurcation. *Surg NemoI* 1976;6:83-91.
- [12]. Diaz FG, Umansky F, Mehta B, Montoya S, Dujovny M, Ausman JI, Cabezudo J. Cerebral revascularization to a main limb of the middle cerebral artery in the sylvian fissure. An alternative approach to conventional anastomosis. *J Neurosurg* 1985;63:21-29.
- [13]. Jennet B, Bond M. Assessment of outcome after severe brain damage. A practical scale. *Lancet*. 1975;1:480-4.
- [14]. Murayama Y, Malisch T, Guglielmi G, Mawad E, Vinuela F, Duckwiler GR, Gobin YP, Klucznik RP, Martin NA, Frazee J. Incidence of cerebral vasospasm after endovascular treatment of acutely ruptured aneurysm: report of 69 cases. *J Neurosurg*. 1997;87:830-5.
- [15]. Taha MM, Nakahara I, Higashi T, Iwamuro Y, Iwaasa M, Watanabe Y, Tsunetoshi K, Munemitsu T. Endovascular embolization vs surgical clipping in treatment of cerebral aneurysms: morbidity and mortality with short-term outcome. *Surg Neurol*. 2006;66:277-84.
- [16]. Dorhout Mees SM, Molyneux AJ, Kerr RS, Algra A, Rinkel GJ. Timing of aneurysm treatment after subarachnoid hemorrhage. *Stroke*. 2012;43:2126-9.
- [17]. Bohm E, Hugosson R. Experiences of surgical treatment of 400 consecutive ruptured cerebral arterial aneurysms. *Acta Neurochir (Wein)*. 1978;40:33-43.
- [18]. Kassel NF, Drake CG. Timing of aneurysm surgery. *Neurosurgery*. 1982;10: 514-9.
- [19]. Whitfield PC, Kirkpatrick PJ. Timing of surgery for aneurysmal subarachnoid hemorrhage. *Cochrane Database Syst Rev*. 2001;2:CD001697.
- [20]. Ohman J, Heiskanen O. Timing of operation for ruptured supratentorial aneurysm: a prospective randomized study. *J Neurosurg*. 1989;70:55-60.
- [21]. de Gans K, Nieuwkamp DJ, Rinkel GJ, Algra A. Timing of aneurysm surgery in subarachnoid hemorrhage: a systemic review of the literature. *Neurosurgery*. 2002;50:336-40.
- [22]. Kassel NF, Torner JC, Jane JA, Haley EC Jr, Adams HP. The international cooperative study on the timing of aneurysm surgery. Part 2: surgical results. *J Neurosurg*. 1990;73:37-47.
- [23]. Orakdogan M, Emon ST, Somay H, Engin T, Ates O, Berkman MZ. Prognostic factors in patients who underwent aneurysmal clipping due to spontaneous subarachnoid hemorrhage. *Turk Neurosurg*. 2016;26(6):840-8.
- [24]. Murayama Y, Malisch T, Guglielmi G, Mawad E, Vinuela F, Duckwiler GR, Gobin YP, Klucznik RP, Martin NA, Frazee J. Incidence of cerebral vasospasm after endovascular treatment of acutely ruptured aneurysm: report of 69 cases. *J Neurosurg*. 1997;87:830-5.
- [25]. Fisher CM, Kistler JP, Davis JM. Relation of cerebral vasospasm to subarachnoid hemorrhage visualized by computerized tomographic scanning. *Neurosurgery*. 1980;6:1-9.
- [26]. Yao PS, Chen GR, Zheng SF, Kang DZ. Predictors of postoperative cerebral ischemia in patients with ruptured anterior communicating artery aneurysms. *World Neurosurg*. 2017;103:241-7.
- [27]. Mahaney KB, Todd MM, Torner JC for the IHAST investigators. Variation of patient characteristics, management, and outcome with timing of surgery for aneurysmal subarachnoid hemorrhage. *J Neurosurg*. 2011;114:1045-53.
- [28]. Grasso G, Alafaci C, Loch Macdonald R. Management of aneurysmal subarachnoid hemorrhage : state of the art and future prospective. *Surg Neurol Int*. 2017;8:1-15.
- [29]. Samaha E, Rizk T, Nohra G, Mohasseb G, Okais N. Intracranial arterial aneurysm: from diagnosis to treatment. A retrospective study of 46 surgically treated cases. *J Med Liban*. 1998;46:122-5.

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