Postoperative Imprint of Surgical Treatment for A1 Segment Aneurysms of the Anterior Cerebral Artery

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Abstract

Objective: To retrospectively analyze the clinical data of 23 patients with proximal anterior cerebral artery (A1 segment) aneurysms who underwent surgical treatment, and to explore the techniques and methods of surgical management.

Methods: From January 2004 to December 2014, a total of 1,437 intracranial aneurysms were treated surgically, among which 23 cases (1.6%) were A1 segment aneurysms. All patients received a confirmed diagnosis through digital subtraction angiography (DSA) or CT angiography (CTA) preoperatively, and underwent surgical clipping for intracranial A1 segment aneurysms. Postoperatively, "3H" therapy was administered to prevent vasospasm. Outpatient follow-up was conducted at 6, 12, 48, and 60 months after surgery, with CT angiography (CTA) re-examination, and the Glasgow Outcome Scale (GOS) was used to evaluate postoperative outcomes.

Results: All A1 aneurysms were completely clipped via pterional craniotomy approach; 7 patients (30.43%) had multiple aneurysms. The mean follow-up time was 38.52 months (range 6-60 months), with a mean Glasgow Outcome Scale score of 4.83 (range III-V).

Conclusion: Careful preoperative analysis of imaging data is crucial for diagnosis and treatment. The key to surgical treatment is protecting perforating arteries from injury; adequate opening of the Sylvian fissure and temporary occlusion of the parent artery during surgery can make the procedure safe and effective.

Full Text

Surgical Management of Proximal Anterior Cerebral Artery (A1) Aneurysms

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Abstract

Objective: To retrospectively analyze the clinical data of 23 patients with proximal anterior cerebral artery (A1 segment) aneurysms treated surgically, and to explore surgical techniques and methods. **Methods:** Between January 2004 and December 2014, 1,437 patients with intracranial aneurysms underwent surgical treatment, among which 23 cases (1.6%) of A1 segment aneurysms were selected. All patients were diagnosed preoperatively by digital subtraction angiography (DSA) or CT angiography (CTA), and underwent surgical clipping of the intracranial A1 segment aneurysms. Postoperatively, "3H" therapy was administered to prevent vasospasm. Outpatient follow-up was conducted at 6, 12, 48, and 60 months after surgery with CTA re-examination, and postoperative outcomes were evaluated using the Glasgow Outcome Scale (GOS). **Results:** All A1 aneurysms were completely clipped via a pterional craniotomy approach. Seven patients (30.43%) had multiple aneurysms. The mean postoperative follow-up duration was 38.52 months (range 6–60 months), with a mean GOS score of 4.83. **Conclusion:** Careful preoperative analysis of imaging data is crucial for diagnosis and treatment. The key to successful surgery is protecting perforating arteries from injury; adequate opening of the Sylvian fissure and temporary occlusion of the parent artery can make the procedure safe and effective.

Keywords: anterior cerebral artery; aneurysm; microneurosurgery

Introduction

Proximal anterior cerebral artery (A1) aneurysms are relatively rare in clinical practice, with reported prevalence rates below 2% of all intracranial aneurysms [1-6]. To date, only a limited number of studies have reported on their surgical management [1, 2, 6-8]. However, A1 aneurysms possess unique clinical characteristics, such as frequent association with cerebrovascular malformations or multiple aneurysms, and they tend to rupture at smaller diameters compared to aneurysms at other locations [1, 2, 6-8]. Additionally, because A1 aneurysms are often adjacent to perforating arteries, surgical clipping is particularly challenging [1, 2, 5, 9-10]. Over the past decade, the Department of Neurosurgery at the General Hospital of PLA has diagnosed and surgically treated 23 cases of A1 aneurysms. We now summarize their clinical and imaging features, surgical techniques, and treatment outcomes to provide guidance for clinical practice.

Methods

Study Subjects

This study collected data from 1,437 patients who underwent surgical treatment for intracranial aneurysms at our hospital between January 2004 and December 2014. Among these, 23 patients with A1 aneurysms confirmed by digital subtraction angiography (DSA) or CT angiography (CTA) were selected. Informed consent was obtained from the patients for the use of their case data.

General Data

Among all 23 patients who met the inclusion criteria, 15 were male (65.22%) and 8 were female (34.78%), with ages ranging from 16 to 72 years (mean 50.26 years). Twenty-one patients (91.30%) were admitted due to subarachnoid hemorrhage (SAH), of which 16 cases (16/21) were caused by ruptured A1 aneurysms, and 2 cases (2/21) had concurrent intracranial hematoma. According to the Hunt-Hess (H-H) grading system, there were 2 cases of Grade 0 (unruptured). DSA revealed that 14 aneurysms were located on the right anterior cerebral artery and 9 on the left. The aneurysms were saccular in 22 cases or fusiform in 1 case (Case 1,). The mean diameter was 5.80 mm (range 3.20–9.70 mm), with ruptured aneurysms averaging 6.00 mm (range 2.00–9.70 mm, n=16) and unruptured aneurysms averaging 5.40 mm (range 1.80–7.80 mm, n=7). Twenty-one aneurysms originated from the proximal A1 segment and 2 from the distal segment. Three cases were associated with cerebrovascular anomalies (2 cases of contralateral A1 segment hypoplasia and 1 case of contralateral A1 segment aplasia,).

Treatment Methods

All aneurysms were surgically clipped via a pterional approach. The key surgical steps were as follows: after dural incision, the anterior part of the Sylvian fissure was dissected under the microscope. The lateral frontal lobe was retracted to expose the supraclinoid segment of the internal carotid artery, its bifurcation, and the A1 segment of the anterior cerebral artery. The A1 artery was dissected distally to expose the aneurysm, and the aneurysm neck was completely freed before final clipping with an aneurysm clip. Intraoperative Doppler ultrasound was used to measure and record blood flow velocities in the ipsilateral A1 and A2 segments. Somatosensory and motor evoked potentials were monitored using intraoperative electrophysiological monitoring. For multiple aneurysms, we attempted to clip all lesions in a single session; if clipping was difficult, a second-stage procedure was performed. To prevent postoperative cerebral vasospasm, the "3H" therapy (hypervolemia, hemodilution, and hypertension) was administered, along with continuous intravenous infusion of nimodipine at 7.5 g/(kg · min).

Follow-up

All patients were followed up at outpatient clinics at 6, 12, 48, and 60 months postoperatively, during which cerebrovascular CTA was performed to assess for recurrence. The Glasgow Outcome Scale (GOS) score was used to evaluate prognosis and was recorded at each follow-up visit. The GOS score from the final follow-up was used as the evaluation criterion in this study.

Results

All 23 aneurysms in this series were completely clipped. Among them, 7 patients (30.43%) had multiple aneurysms, totaling 17 lesions. One patient (Case 3) had two pairs of mirror aneurysms, comprising 5 aneurysms in total ([Figure 1: see original paper]), with 3 ipsilateral A1 segment aneurysms clipped in the same session. The remaining 6 patients with multiple aneurysms each had 2 lesions, which were all completely clipped during surgery. Postoperatively, only one patient ([Figure 2: see original paper]) developed transient upper extremity weakness (Grade 4 muscle strength) with mild aphasia, but normal muscle tone. This patient was treated with nimodipine for vasodilation and intensive functional rehabilitation, and had essentially recovered at the 6-month follow-up. All other patients had uneventful postoperative courses without complications. The mean follow-up duration for this cohort was 38.52 months (range 6–60 months). The GOS grade was [text incomplete] with a mean score of 4.83 ().

Discussion

A1 segment aneurysms exhibit several characteristic features: (1) Low incidence, accounting for less than 2% of all intracranial aneurysms [1-6]. In our series, they represented only 1.60% (23/1,437) of intracranial aneurysms treated surgically at our institution, consistent with literature reports. (2) Higher prevalence in females has been reported [1, 6, 11]; however, in our 23 cases, males predominated (15/23), which slightly differs from previous reports. (3) Right-sided predominance has been observed [3, 5, 7, 10-11], and our series similarly showed a right-side predominance (14/23). (4) They can be associated with aneurysms at other intracranial locations (i.e., multiple aneurysms) [1-2, 4-8, 12]; we found 7 cases of multiple aneurysms (34.43%). Therefore, preoperative DSA is essential to determine whether additional aneurysms are present, particularly when the responsible lesion cannot be identified, as DSA provides crucial information for surgical planning. (5) The reported incidence of associated cerebrovascular malformations is as high as 40% [2-4, 11-13], but we identified only 3 cases with cerebrovascular anomalies, possibly related to regional or ethnic differences.

Compared with aneurysms at other intracranial locations, smaller A1 aneurysms are more prone to rupture, with reported average diameters of less than 7.00 mm at presentation [2-3, 5-7, 12, 14]. In our series, the mean aneurysm diameter was 5.80 mm, while ruptured aneurysms averaged 6.00 mm. However, ruptured A1 aneurysms less frequently form intracranial hematomas, with an incidence of only 10% [4-6]. This is presumably because the A1 segment is often adherent or embedded in the frontal lobe base, preventing hematoma formation [2, 5, 12]. Since most A1 aneurysms project posteriorly [1-6, 12] (confirmed intraoperatively in our series), two-dimensional DSA studies, especially in cases with multiple aneurysms, are prone to misdiagnosis. Therefore, multi-angle DSA examinations should be performed in patients with suspected A1 aneurysms.

The fundamental principle of A1 aneurysm surgery is preservation of adjacent perforating arteries. Anatomical studies have shown that 8–12 perforating arteries arise from the A1 segment, penetrate the anterior perforated substance, and supply blood to the septum pellucidum, anterior commissure, globus pallidus, anterior limb of the internal capsule, anteroinferior striatum, and anterior hypothalamus [9-10, 15]. These perforators are end arteries, and their injury can lead to limb paralysis. Due to the fragile nature of A1 aneurysms, dissection of the aneurysm, parent artery, and perforating arteries is extremely difficult. Therefore, proximal vascular occlusion to reduce intra-aneurysmal blood flow and decrease aneurysmal tension is crucial for facilitating intraoperative dissection and clipping. Meanwhile, placement of temporary clips should avoid damaging the recurrent artery of Heubner, and occlusion time should not be prolonged. Intraoperative somatosensory evoked potential (SEP) monitoring is particularly important in patients with contralateral A1 segment hypoplasia. To prevent the aneurysm clip from compressing perforating arteries during brain retractor removal, a smaller clip (slightly wider than the aneurysm neck) should be selected after dissecting the aneurysm neck, and clipping should be performed parallel to the skull base angle; the clip can even be trimmed to an appropriate length under the operating microscope [12]. To confirm patency of the parent artery after clipping, all cases in our series underwent intraoperative color Doppler ultrasound or indocyanine green angiography (ICGA) after papaverine application to the parent artery, preventing parent artery stenosis or inadvertent clipping of perforating arteries, while SEP monitoring was used to assess limb function. Postoperative "3H" therapy was administered to prevent cerebral vasospastic ischemia, and continuous intravenous nimodipine infusion was used for vasodilation and improved cerebral perfusion, contributing to better outcomes. One patient in our series developed right upper extremity weakness (Grade 4) and mild aphasia on postoperative day 3. CT confirmed basal ganglia infarction, but intraoperative assessment had shown patent parent and perforating arteries with no abnormal SEP changes. We hypothesize that this ischemic event was related to postoperative cerebral vasospasm. After treatment with nimodipine and intensive rehabilitation, the patient' s upper extremity strength returned to normal at discharge, and aphasia had completely resolved at the 6-month follow-up.

In the majority of our 23 patients (19 cases), the aneurysm dome projected posteriorly, consistent with literature reports [1, 2, 4-8, 12]. This orientation also explains why A1 aneurysms tend to be embedded in or adherent to the frontal lobe base, and improper retraction of the frontal lobe during surgery can cause rupture of these high-risk lesions. When using the pterional approach for aneurysm clipping, posteriorly projecting A1 aneurysms are often obscured by the parent artery in the surgical field, making dissection of the aneurysm neck and perforating arteries difficult, particularly when subarachnoid hemorrhage has soiled the operative field. Based on our experience, wide opening of the Sylvian fissure, carotid cistern, and chiasmatic cistern significantly expands the surgical view, facilitating manipulation while reducing frontal lobe retraction. This is particularly crucial in patients with SAH and severe cerebral edema. If the aneurysm remains poorly visualized after opening these structures, partial resection of the gyrus rectus may be performed to improve exposure. A contralateral pterional approach may be considered in two situations: (1) when contralateral aneurysms coexist, especially when the contralateral lesion is the responsible aneurysm; and (2) when the A1 aneurysm projects posteroinferiorly and is obscured by the parent artery via the ipsilateral pterional approach. However, if preoperative assessment cannot clearly determine the morphology and projection of the A1 aneurysm, especially when it is the responsible lesion, a contralateral pterional approach should not be selected blindly to avoid adverse outcomes. In our experience, the responsible A1 aneurysm should be prioritized in such cases. If simultaneous clipping of the contralateral non-responsible aneurysm is difficult, staged clipping or postoperative endovascular treatment may be considered. Only one patient in our series (Case 4) underwent a two-stage procedure, while the remaining 22 cases underwent ipsilateral pterional aneurysm clipping. Clinical observations show that saccular aneurysms are common in the A1 segment, while fusiform aneurysms are rare [1-2, 4-8, 12]; we had only one fusiform A1 aneurysm (Case 1). Because the wall of this type of aneurysm incorporates several perforating arteries, direct clipping can easily cause infarction in their vascular territory, making management extremely challenging. To date, only a few successful surgical clipping cases have been reported [5, 16-17]. Therefore, for fusiform A1 aneurysms, if the contralateral A1 segment has good blood flow that can compensate for ipsilateral A1 flow, aneurysm trapping should be selected. This method preserves the recurrent artery of Heubner and thus results in fewer postoperative ischemic strokes [18]. When the contralateral A1 artery cannot compensate for ipsilateral A1 supply, clipping or trapping the aneurysm may cause severe consequences, and aneurysm wrapping should be considered, with reinforcement of the aneurysm wall to reduce rupture risk.

Currently, endovascular treatment of intracranial aneurysms has achieved satisfactory results [19]. For A1 aneurysms, the unique anatomical location makes endovascular therapy more challenging than for aneurysms at other sites. However, endovascular embolization may be prioritized for A1 aneurysms that are small, have a relatively narrow neck, project medially, arise from a wide parent artery, are Hunt-Hess grade [text incomplete], and are not associated with intracranial hematoma. Surgical treatment should be the first choice for A1 aneurysms with concurrent intracranial hematoma (>30 mL), severe parent artery vasospasm, Hunt-Hess grade [text incomplete], or failed endovascular treatment.

Although the prevalence of proximal anterior cerebral artery A1 segment aneurysms is extremely low, their surgical management is particularly challenging. Meticulous preoperative imaging evaluation and formulation of a comprehensive treatment plan are crucial for improving surgical efficacy and prognosis. Wide opening of the Sylvian fissure and temporary parent artery occlusion are key to safe surgical execution, protecting perforating arteries from injury and preventing intraoperative aneurysm rupture. Additionally, intraoperative adjuncts such as somatosensory evoked potential monitoring, color Doppler ultrasound, or indocyanine green angiography can reduce surgical risks and minimize complications.

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Note: Figure translations are in progress. See original paper for figures.