Intracranial aneurysm clipping complications

- Statin versus no statin after treatment with pipeline embolization device for intracranial aneurysms: a meta-analysis
- Unilateral approach to bilateral middle cerebral artery aneurysms: a large series and a proposed grading system to predict technical difficulties
- Endovascular treatment of intracranial aneurysms with the Woven EndoBridge- safety and efficacy
- Rapid ventricular pacing in cerebral aneurysm clipping: institutional workflow, systematic review, and single-arm meta-analysis
- Multidimensional outcome after endovascular or microsurgical occlusion of ruptured intracranial aneurysms - Comparative analysis of a prospective Swiss multicenter study
- Microsurgical treatment of 723 cerebral aneurysms: a single-center prospective study
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- Microsurgical management of 883 previously coiled intracranial aneurysms: a systematic review, meta-analysis, and meta-regression of its effectiveness and safety

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Clipping of an intracranial aneurysm, while often effective, can lead to several neurological and surgical complications, depending on factors like aneurysm location, size, patient comorbidities, and timing of the intervention (ruptured vs. unruptured).

 Neurological Complications Ischemic Stroke

Due to vasospasm, vessel manipulation, or temporary clip occlusion.

May result in hemiparesis, aphasia, or visual field deficits.

Cranial Nerve Palsies

Particularly involving CN III (oculomotor), CN IV (trochlear), or CN VI (abducens), depending on aneurysm location (e.g., posterior communicating artery).

CN VII (facial) can be affected in certain posterior circulation aneurysms.

Seizures

Can occur postoperatively, especially in cortical exposure or in cases with intraoperative bleeding.

Cerebral Edema

Related to surgical manipulation or ischemia.

Hydrocephalus

Can be communicating (due to subarachnoid hemorrhage from a ruptured aneurysm) or obstructive (due to surgical scarring or mass effect).

Cognitive and Behavioral Changes

Frontal lobe or temporal lobe manipulations can result in personality or memory changes.

Rebleeding

Rare after clipping if clip is properly placed, but can occur if the clip migrates or is incomplete.

 Surgical/Technical Complications Incomplete Aneurysm Occlusion

Leads to risk of regrowth or rebleeding.

Detected on postoperative angiography.

Parent Vessel or Perforator Injury

Particularly dangerous in anterior communicating, MCA bifurcation, or basilar apex aneurysms.

Clip Slippage or Migration

Can result in aneurysm remnant or bleeding.

Aneurysm Rupture Intraoperatively

Requires rapid control, can result in massive brain swelling or infarction.

CSF Leak or Wound Complications

More common in posterior fossa approaches.

 Incidence Neurological deficit (temporary or permanent): 5–15%.

Stroke: 5–10%.

Mortality: 1–3% (lower in unruptured aneurysms, higher in ruptured).

A patient’s condition, the type, location, and size of the aneurysm, as well as other factors determine the potential risks and complications associated with these surgical procedures. The current combined morbidity and mortality rate related to the neurosurgical clipping of an incidental intracranial aneurysm is between 5% and 10%. The risk may be somewhat higher with large aneurysms, particularly the deepest ones. Surgery poses the lowest risk when it is performed before an aneurysm ruptures. However, there are certain risks and complications associated with these treatment options. They include:

Blood Clots

Swelling in the Brain

Bleeding in the Brain

Weakness

Paralysis

Loss of Sensation

Loss of Vision

Confusion

Loss of Speech & Other Cognitive Functions

Short-term Memory Problems

Infections

Vasospasm

Seizures

Hydrocephalus

Stroke

Death

Rebleeding

Of the 18,000 persons who survive the initial rupture of an aneurysm annually, 3,000 either die or are disabled from rebleeding.

Some believe the incidence of rebleeding is as high as 30%.

The highest incidence occurs in the first 2 weeks after initial hemorrhage.

Peaks in the incidence of rebleeding occur in the first 24 to 28 hours and at 7 to 10 days.

Rebleeding within the first 24 to 48 hours is the leading cause of death in persons surviving the initial bleed.

Approximately 70% of patients who rebleed will die.

The onset of rebleeding is usually accompanied by sudden severe headache, often associated with severe nausea and vomiting; a decrease in or loss of consciousness; and new neurological deficits. Death may occur. Rebleeding can be confirmed by a CT scan or a sudden spike in ICP with new blood seen in the bag if a ventricular drain is in place. Early treatment, with either surgical or endovascular methods, of the aneurysm is the most effective means of preventing rebleeding.

Cerebral Vasospasm

Of the 18,000 persons annually who survive initial aneurysmal rupture, 3,000 either die or are disabled from cerebral vasospasm.

Vasospasm occurs in approximately 30% of patients.

By definition, cerebral vasospasm is narrowing of a cerebral blood vessel and causes reduced blood flow distally, which may lead to delayed ischemic deficit and cerebral infarction if left untreated.

Besides the damage done by the initial SAH, brain damage produced by vasospasm is an important

cause of morbidity and mortality after hemorrhage, with 14% to 36% of patients suffering disability and death.

Since improved treatment of aneurysmal subarachnoid hemorrhage has occurred with early and improved microsurgery, new endovascular techniques and better post operative care and monitoring, vasospasm has significantly decreased as the cause of death over the last ten years (from 35% in the seventies to less than 10% at this time).

The present rescue therapies, which include 'triple H therapy' HHH, (hypertension/hypervolemia/ hemodilution), interventional procedures such as balloon angioplasty, intra-arterial nicardipine and other vasodilators, are associated with significant morbidity, and are labor intensive and expensive.120 A drug that would prevent delayed ischemic effects and minimize the amount of rescue therapy and optimize late outcome is desirable. When the patient's condition deteriorates 3 to 14 days after SAH, vasospasm should be considered as the possible cause. A CT scan should be performed immediately to rule out hydrocephalus, infarction, or rebleeding.

Vasospasm can decrease cerebral perfusion to an area, causing ischemia and perhaps infarction, and can lead to further deterioration of neurological function.

Vasospasm may be differentiated as either angiographic or symptomatic.

Angiographic vasospasm refers to narrowing of a cerebral arterial territory, as noted on angiography, without clinical symptoms.

Symptomatic vasospasm is the clinical syndrome of delayed cerebral ischemia associated with angiographically documented narrowing of a major cerebral arterial territory and TCD elevation of a specific arterial territory.

Vasospasm develops 3 to 14 days after SAH (peaking at 7 to 10 days), although the onset may be delayed up to 21 days.

Most of the studies have been retrospective analyses of single institution experiences, demonstrating results with limited generalization given their inherent selection bias [1].

The interpretation of other multicenter studies is equally limited given their focus on specific subgroup data [2]

or their consideration of all morbidity as one variable [3].

Bekelis et al., from Portsmouth performed a retrospective cohort study of patients who had undergone cerebral aneurysm clipping (CAC) in the period from 2005 to 2009 and were registered in the Nationwide Inpatient Sample (NIS) database. A model for outcome prediction based on preoperative individual patient characteristics was developed.

Of the 7651 patients in the NIS who underwent CAC, 3682 (48.1%) had presented with unruptured aneurysms and 3969 (51.9%) with subarachnoid hemorrhage. The respective inpatient postoperative risks for death, unfavorable discharge, stroke, treated hydrocephalus, cardiac complications, Deep-Vein Thrombosis, pulmonary embolism, and acute renal failure were 0.7%, 15.3%, 5.3%, 1.5%, 1.3%, 0.6%, 2.0%, and 0.1% for those with unruptured aneurysms and 11.5%, 52.8%, 5.5%, 39.2%, 1.7%,

2.8%, 2.7%, and 0.8% for those with ruptured aneurysms. Multivariate analysis identified risk factors independently associated with the above outcomes. A validated model for outcome prediction based on individual patient characteristics was developed. The accuracy of the model was estimated using the area under the receiver operating characteristic curve, and it was found to have good discrimination.

The featured model can provide individualized estimates of the risks of postoperative complications based on preoperative conditions and can potentially be used as an adjunct in decision making in cerebrovascular neurosurgery [4].

Of 818 patients undergoing Microsurgical Clipping of Intracranial Aneurysms who underwent cranial operations, 28 (3.4%) had a ventriculoperitoneal shunt. Four of these 28 (14.3%, 95% confidence interval [CI] 4.0%-32.7%) developed postoperative complications, compared to 42 of 790 (5.3%, 95% CI 4.0%-7.1%) without a history of VP shunt (P = .07). In addition, patients with a shunt were more likely to have longer cranial procedures (P = .04), longer hospital stays (P = .05), and more computed tomography scans during their craniotomy-associated admission (P = .002). Multivariate analysis, though not significant, demonstrated that the presence of a shunt contributed to the development of complications (odds ratio [OR] 2.24, 95% CI .70-7.13, P = .17). Length of surgery (OR 1.17, 95% CI 1.04-1.31, P = .01) and length of stay (OR 1.04, 95% CI 1.01-1.07, P = .01) were significantly longer in those with a postoperative complication.

Linzey et al. from Ann Arbor, found a nonsignificant trend toward increased postoperative complications in patients with a VP shunt who underwent a subsequent cranial operation [5].

Clipping is an independent risk factor for the development of early cerebral infarcts, whereas delayed cerebral infarcts are associated with angiographic vasospasm. Early cerebral infarcts are stronger predictors of worse outcome than delayed infarction [6].

Incomplete clipping

Incomplete aneurysm clipping.

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