Updates on Neuroendovascular Treatment of Cerebral Aneurysms

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Neuroendovascular treatment is a minimally invasive technique in treating vascular diseases of the central nervous system. Conditions in the past that would have required surgical intervention can be considered for treatment by using an endovascular approach. With the advances in technology, many devices have been invented over time.

The author reviewed recent articles related to neuroendovascular treatment and discusses current treatment options, and future trends, especially in cerebral aneurysms and cerebral vasospasm after the aneurysms ruptured.

Endovascular coiling of cerebral aneurysms

The primary goal of treatment of cerebral aneurysms is to prevent future rupture. Both surgical clipping and endovascular coiling can achieve the same goal.

The advent of microneurosurgical techniques and advancements in cerebrovascular surgery (temporary clipping, neuroprotection, etc.) have extended the applicability of aneurysm surgery and improved surgical outcomes. In spite of these, there remain aneurysms which are difficult to clip even in the best of hands.

For ruptured cerebral aneurysms, a large randomized trial (the International Subarachnoid Aneurysm Trial study) reported that for patients with aneurysms suitable for both treatments, endovascular coiling is more likely to result in independent survival at 1 year than neurosurgical clipping, and the survival benefit continues for at least 7 years. The risk of late rebleeding is low, but is more common after endovascular coiling than after neurosurgical clipping.[1]

For unruptured cerebral aneurysms, a recent report from the International Study of Unruptured Intracranial Aneurysms II (ISUIA II) showed that overall surgical morbidity and mortality rates at 1 year were 12.6% for patients who had no history of prior subarachnoid hemorrhage (SAH) and 10.1% for patients who had history of prior SAH, with substantial increasing risk at $\geq$ 50 years of age. On the other hand, overall morbidity and mortality rates associated with endovascular coiling were 9.1% at 30 days and 9.5% at 1 year.[2]

Overall, looking at the results of ISUIA and ISAT when the anatomy is favorable, an endovascular approach seems to be the treatment of choice in patients over 50 years of age and in those with posterior circulation aneurysms.

Technological advances of coils have improved this method of treatment. Over the last several years, the number of coil sizes has been increased, three dimensional softer coils allowing safer coil placement have become available. To decrease recanalization rates of embolized aneurysms, various modifications to standard coils such as biologically active surfaces (Matrix; Boston Scientific, Nexus; Microtheraputics-EV3) or coated coils with a swelling hydrogel (HydroCoil) have been developed and brought to clinical use. The objective of these developments is to promote clot organization and fibrosis or to increase packing density thereby improving the long term results of aneurysm embolization.

Another significant technical refinement has been in the area of microcatheters. Braided, hydrophilically coated microcatheters have been invented, which allow improved access to many aneurysms thereby increasing chances of obtaining a complete aneurysm obliteration.

Wide-necked aneurysms are difficult to treat with detachable coils because of coil herniation into the parent artery. To achieve feasibility in treating wide-necked aneurysms by endovascular treatment, a balloon-assisted coiling technique had been introduced. Many balloons are available on the market, with different sizes, shapes, compliance and trackability.

The recent development of self-expandable intracranial stents with better trackability and flexibility has been used as an alternative to the balloon remodeling technique. The ideal aneurysm to stent is one that has not ruptured and is treated in an elective fashion after appropriate antiplatelet therapy has been initiated. Premedication with antiplatelet therapy may be associated with an increased risk of hemorrhage in those patients with acutely ruptured aneurysms who might benefit from stent-assisted coiling. Stent design modifications such as asymmetric stents, covered stents, and semicovered stents will soon become available.

A new endovascular method is to fill the aneurysms with the liquid embolic agent Onyx HD 500 (Micro Therapeutics, Irvine, CA). Experimental application of this technique has been described.[3] A prospective observational study was conducted in 20 European centers enrolling a consecutive series of 119 patients with 123 aneurysms

judged suitable for Onyx treatment. At 1 year, procedure- or device-related permanent neurologic morbidity was present in eight of 97 patients (8.2%). Seven patients died (7.2%). Delayed occlusion of the parent vessel occurred in nine patients (9.2%).[4] Long term follow-up of these patients needs to be followed.

Not only have the new devices been invented for curing cerebral aneurysms, but new imaging modalities are also important. Three-dimensional angiography is becoming the gold standard for decision making regarding optimal aneurysm treatment.

Endovascular treatment of cerebral vasospasm

Subarachnoid hemorrhage (SAH) caused by intracranial aneurysm rupture is one of the most serious health-care problems. The main complications after a SAH event are rebleeding and cerebral vasospasm. The policy of early aneurysm occlusion has led to progressively lower rates of rebleeding. However, despite the improvements in management of aneurysmal SAH, vasospasm remains an important cause of morbidity and mortality. It is present angiographically in up to 70% of patients and is clinically symptomatic in 30% of patients.[5] Around 50% of patients with symptomatic vasospasm will develop ischemic infarct.

New diagnostic imaging modalities

Transcranial ultrasonography (TCD) is useful in patients who have SAH for detecting and monitoring angiographic vasospasm in basal segments of the intracranial arteries, especially the MCA and basilar artery. The sensitivity may be increased by studying trends in flow velocities over time. Increases in velocity of more than 50 cm/s in 24 h are worrisome for the onset of clinical vasospasm. TCD can reliably predict the absence or presence of significant angiographic MCA vasospasm when the flow velocities are less than 120 or greater than 200 cm/s. By using color-coded TCD, the sensitivity and specificity for detection of vasospasm can be improved when compared with conventional TCD.[6]

The INVOS transcutaneous cerebral oximeter (Somanetics Corporation, Troy, MI), a cutaneous sensor capable of measuring cortical oxygen saturation, is a new method of monitoring for vasospasm. The cutaneous sensor pads are applied on both sides of the forehead. Although the numerical value displayed by the monitor is not always an accurate indicator of cerebral oxygenation, the minute-to-minute saturation trend can be helpful in identifying those patients developing cortical ischemia from vasospasm.[7]

Recent developments in phase contrast magnetic resonance angiography technology have made it possible to obtain quantitative flow values for all major vessels in the cerebral circulation. Besides vessel diameter, flow rate and wall shear stress values can be measured. The error in this noninvasive technique has been calculated to be less than 7%.[8]

What's new in triple H therapy?

In a randomized controlled trial, there was no difference in mean global or regional CBF and rates of symptomatic vasospasm during the treatment period between hypervolemic and normovolemic patients.[9] Analysis of trials also found that hypervolemia did not improve the outcome or reduce the incidence of delayed ischemia.[10]

Endovascular treatment of cerebral vasospasm

Marked advances in instrumentation over the last decade have led to the development of several endovascular strategies to treat vasospasm.

Endovascular therapy for cerebral vasospasm consists of two forms: the infusion of vasodilating agents such as inhibitors of phosphodiesterase (papaverine, amrinone, milrinone), calcium channel antagonists and balloon angioplasty.

Intra-arterial infusion of vasodilating agents has no long-term benefit in the treatment of vasospasm, therefore requiring additional infusion or balloon angioplasty.[11] Their use is only for treating distal vessel vasospasm because of the risk of vessel rupture with balloon angioplasty for distal small vessels.[12] A recent study found that the treatment with intra-arterial papaverine has a neuronal toxic effect,[13] therefore it could be replaced by using other drugs, such as verapamil, nimodipine and nicardipine.

Angioplasty for cerebral vasospasm can be performed with compliant balloons that are composed of variations of silicone. Fortunately, vasospasm responds to low dilation pressures from these compliant balloons, which decreases the risks of rupture. Compliant angioplasty balloons are available in two basic designs: flow-directed and over-the-wire.

For flow-directed balloons, they are used for large, proximal spastic intracranial arteries. More recently, promising over-the-wire silicone balloon microcatheter systems have been introduced to allow a wider application of angioplasty techniques in small vessels, particularly to the A1 and proximal M2.

Overall the angiographic efficacy of combined endovascular treatment for cerebral vasospasm has been shown to be extremely high: almost 100% in most series.[14,15] A good recovery has been noted in 22-50% of cases.[14,15] Clinical improvement with intra-arterial infusion of vasodilating agents varies between 29-76%.[16,17]

Good results have been found to correlate with early and aggressive intervention after the failure of medical ("triple-H") therapy. Both animal and human data suggest that early intervention produces far better results. Rosenwasser et al found that 70% of patients treated within 2 hours of symptom onset improved clinically, compared to 40% who were treated after 2 hours.[18]

The disadvantages of angioplasty arise in those cases in which CT scans already demonstrate hypodense areas that may represent ischemia. Such territories are at risk of reperfusion injury and hemorrhage. Reported complications resulting from balloon angioplasty for cerebral vasospasm following subarachnoid hemorrhage include perforation, aneurysm re-rupture, branch occlusion and hemorrhagic infarct.[19]

Advanced age and poor clinical status at the time of SAH onset are poor clinical outcome predictors despite endovascular treatment in patients with symptomatic vasospasm.[20]

CONCLUSION

Modern technology and minimally invasive techniques make it possible for physicians to treat the patients with no harm and favorable outcome, especially for those with cerebral aneurysms and cerebral vasoapasm, by the endovascular approach.

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