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PERSPECTIVE

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A Perspective on Cerebral Vasospasm Post Aneurysmal Subarachnoid Hemorrhage: Synopsis, Guidelines, Treatment, & Advances

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ABSTRACT

Cerebral vasospasm is a serious complication that occurs after Aneurysmal Subarachnoid Hemorrhage (ASAH), affecting approximately one-third of patients with ASAH. It can lead to Delayed Cerebral Infarction (DCI), resulting in high mortality and severe disability. Current guidelines recommend a combination of proximal cerebral vessel balloon angioplasty and intra-arterial vasodilators or solely the use of intra-arterial vasodilators for treating cerebral vasospasm. However, distal balloon angioplasty is not recommended due to its associated risks, such as arterial rupture. DCI, often caused by cerebral vasospasm, can be diagnosed clinically or radiologically. Treatment aims to prevent DCI and includes the administration of Nimodipine, maintaining euvolemia, and stabilizing blood pressure. In cases where standard management fails, endovascular rescue interventions can be considered, such as endovascular vasodilators and spasmolytics, as well as mechanical angioplasty. Some previous treatments, including statin therapy and magnesium sulfate, have shown limited benefit in treating cerebral vasospasm. Prophylactic treatments for cerebral vasospasm have been explored but are not yet supported by guidelines, as they have shown increased risks without clear benefits. Recent developments have focused on technological advances in balloon and stent technology. Newer balloons, such as the Scepter XC balloon, have demonstrated efficacy in treating vasospasm in both proximal and distal vessels. Improved navigation, lower rates of rupture, and increased success rates have been observed. Stent-retrievers have also shown promise in dilating cerebral vessels without occlusion, resulting in improved vessel diameter, blood flow, and neurological outcomes. The emerging theme in post ASAH cerebral vasospasm treatment is the advancement of balloons and stents, enabling safer and more effective treatment of distal vessel vasospasm. These advancements offer potential solutions for cases that are refractory to standard therapies and show promise in improving patient outcomes.

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Description

Cerebral vasospasm is a formidable complication after Aneurysmal Subarachnoid Hemorrhage (ASAH), affecting nearly one-third of all ASAH patients. Cerebral vasospasm refers to narrowing of the lumen of one or multiple cerebral arteries due to contractions in arterial smooth muscle, due to ischemia, injury, and endothelial dysfunction [1]. These patients are at a significant risk for mortality and severe disability from delayed cerebral infarction following cerebral vasospasm [1]. Currently, AHA guidelines for subarachnoid hemorrhage suggest the most efficacious treatment strategy for treatment of cerebral vasospasm is either a combination of proximal cerebral vessel balloon angioplasty and intra arterial vasodilators, or solely the use of intra arterial vasodilators [2]. The use of distal balloon angioplasty is not recommended, due to the significant risks associated with this procedure. Distal balloon angioplasty refers to Transluminal Balloon Angioplasty (TBA) of the portions of the circle of willis which are distal to the most proximal segments [3]. For example, the second and third aspects of the middle cerebral artery (M2, M3) would be considered distal cerebral arteries, while the first segment (M1) would be considered proximal. Arterial rupture is the most serious complication of TBA, and historically the distal cerebral arteries are at highest risk given their smaller size and decreased wall strength.

The following perspective will discuss the most updated guidelines in treating cerebral vasospasm post ASAH and recent advances in treatment illustrated in the literature.

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Delayed cerebral infarction

Delayed Cerebral Infarction (DCI) refers to ischemia shortly after ASAH occurrence and treatment. Most common cause of DCI in the context of ASAH is cerebral vasospasm, which typically occurs 3-14 days following ASAH [1]. Studies have shown that risk of death for patients with DCI compared to those without DCI is nearly 5 times greater [2]. DCI can be diagnosed clinically and radiologically. Clinically, patients will present with symptoms of cerebral infarction: paresthesias, paresis, bulbar deficits, and hyperreflexia [3]. Radiologically, cerebral infarct or cerebral vessel vasospasm can be visualized. However, the diagnosis of DCI is more reliant on clinical findings than radiographic. Studies have shown radiographic evidence of cerebral vasospasm without clinical symptoms of DCI, illustrating that radiographic vasospasm can be observed even if they are minor or asymptomatic [4].

Treatment of post-ASAH cerebral vasospasm

The treatment of cerebral vasospasm post ASAH aims to prevent the onset of DCI. In doing so, intrathecal or intra-arterial vasodilators are primary options. According to the ‘‘2023 Guideline for the Management of Patients With Aneurysmal subarachnoid hemorrhage: A Guideline From the American Heart Association/ American Stroke Association” patients with diagnosed or suspected cerebral vasospasm should be given Nimodipine, a dihydropyridine calcium channel blocker (COR 1 LOE A) [5]. Emphasis is also placed on the maintenance of euvolemia, which involves monitoring for volume depletion and hypervolemia (COR 2a LOE B-NR) [5]. Also important is blood pressure stability, while blood pressure instability is associated with poorer outcomes, especially hypotension. Most recent data suggest that induced hypertension with usage of presors fluids is favorable in patients with DCI (COR 2b and LOE B-NR) [5]. In patients with vasospasm refractory to the preceding management, endovascular rescue interventions are viable options. These include endovascular vasodilators and spasmolytics in both proximal and distal cerebral vessels. Examples of well-studied vasodilators include papaverine or intra-arterial nimodipine (COR 2b LOR B-NR) [6,7]. Intrathecal vasodilators come with risks of systemic hypotension, which require careful monitoring to avoid DCI and systemic pathology. Further endovascular rescue interventions include mechanical angioplasty to dilate cerebral vessels (COR 2b LOR B-NR) [5]. This involves the use of either compliant or noncompliant balloons, and carries risks of vessel occlusion, rupture, and hemorrhage. Current guidelines state angioplasty to be limited to only proximal aspects of the cerebral vessels, as distal segments have thinner arterial walls, more tortuous, and increased risk for rupture [8]. Guidelines also include dual endovascular therapy with intrathecal vasodilators and mechanical proximal balloon angioplasty. Specifically, distal segments are more suited for intrathecal vasodilators with proximal segments obtaining mechanical angioplasty [9]. Previous treatments that are not currently supported in the literature include statin therapy (COR 3 LOR A) and recent meta analysis consensus is that statin therapy does not have benefit for patients experiencing cerebral vasospasm [10]. Similarly, while magnesium sulfate has been shown to improve vasospasm, a recent analysis demonstrated that this improvement was not clinically relevant in terms of affecting outcomes after vasospasm [11].

Prophylactically treating for cerebral vasospasm post ASAH has been explored by several studies [12,13]. Interventions include prophylactic balloon angioplasty or hemodynamic augmentation, through the use of pressors and local vasodilators. However, no prophylactic treatments at this time have been approved by AHA guidelines. In fact, the phase II clinical trial of prophylactic balloon angioplasty in cerebral vasospasm was terminated early due to increased rates of vessel rupture and harm to patients [14].

Recent developments and technological advances

Advances in balloon technology have allowed interventionalists to angioplasty distal cerebral vessels, especially newer, smaller, and more navigable balloons the Scepter XC balloon was recently tested in two separate case series in which it proved efficacy in treating vasospasm in both proximal and distal vessels [15,16]. The Scepter XC balloon is the lowest profile compliant balloon currently on market, and allows for navigation of tortuous vessel diameters with lower rates of rupture. In a single center study, Hyper Glide/Hyper Foam balloons were tested on both proximal and distal vessel segments, resulting in a 88.9% angioplasty success rate [16]. Using a dual lumen balloon, first of its kind, Tsogakas et al., assessed the efficacy of balloon angioplasty for proximal and distal cerebral segments, finding a statistical significant angiographic improvement in vasospasm [17]. New advances have also been made in stent technologies, useful in the dilation of cerebral vessels. While angioplasty has been effective, they are limited by transient vessel occlusion while balloon is being expanded to mechanically dilate vessels [18]. In contrast, stent-retrievers avoid this problem by dilating vessels without occluding them. In a recent small case series of 6 patients and 14 vasospastic vessels, the Solitaire stent-retriever allowed easy access to targeted vasculature, with improved vessel diameter and blood flow, in addition to improvement in neurological outcomes [19].

Conclusion

A salient theme within the new advances in post ASAH cerebral vasospasm treatment is that balloons are becoming better at traversing tortuous vessel segments and less likely to rupture vessels, due to smaller size, greater flexibility, and refinement in design. The result of this is increasing evidence of the efficacy of distal vessel angioplasty in case series and single center cohort studies, which historically has been too risky and even currently is not recommended by the AHA. This is especially important given that other endovascular rescue therapies for distal vessel vasospasm, such as intrathecal vasodilators, are often limited by transient effects, resulting in recurrent vasospasms which are increasingly refractory to therapy. In these situations, recent advances show promise for providing successful treatment and improved outcomes.

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