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REVIEW

Critical Care Guidelines on the Endovascular Management of Cerebral Vasospasm

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Abstract Cerebral vasospasm and delayed cerebral ischemia account for significant morbidity and mortality after aneurysmal subarachnoid hemorrhage. While most patients are managed with triple-H therapy, endovascular treatments have been used when triple-H treatment cannot be used or is ineffective. An electronic literature search was conducted to identify English language articles published through October 2010 that addressed endovascular management of vasospasm. A total of 49 articles were identified, addressing endovascular treatment timing, intra-arterial treatments, and balloon angioplasty. Most of the available studies investigated intra-arterial papaverine or balloon angioplasty. Both have generally been shown to successfully treat vasospasm and improve neurological condition, with no clear benefit from one treatment compared with another. There are reports of complications with both therapies including vessel rupture during angioplasty, intracranial hypertension, and possible neurotoxicity associated with papaverine. Limited data are available evaluating nicardipine or verapamil, with positive benefits reported with nicardipine and inconsistent benefits with verapamil.

Keywords Balloon angioplasty · Intra-arterial · Nicardipine · Papaverine · Verapamil

Introduction

Cerebral vasospasm and delayed cerebral ischemia (DCI) account for the majority of morbidity and mortality for patients who survive to undergo treatment following aneurysmal subarachnoid hemorrhage (SAH). Angiographic vasospasm is observed in 30–70% of patients between days 5 and 14 following the initial aneurysmal bleed [1, 2]. Approximately 50% of patients with angiographic vasospasm will develop DCI, with 15–20% of these patients suffering stroke or death despite maximal therapy [3, 4].

Medical management of vasospasm primarily consists of hemodynamic augmentation that is associated with significant risks of complications, such as heart failure and pulmonary edema [5]. Endovascular therapies, such as intra-arterial vasodilator administration or transluminal balloon angioplasty, might benefit patients with cerebral vasospasm when hemodynamic therapy has failed or when there is concern for complications of hemodynamic therapy.

Despite the potential benefit from endovascular therapy, clear guidelines directing use of these treatments for vasospasm after SAH are not available. The decision of when to intervene endovascularly is not clear and certain across all patients. A review of the medical literature was conducted to determine the role of endovascular treatment in the management of cerebral vasospasm.

Methods

A search was performed of the English language literature published through October 2010 using MEDLINE, the

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Cochrane Controlled Trials Registry, and the National Institutes of Health/National Library of Medicine clinical trials registry. Candidate articles were identified by using the following search terms: (1) "vasospasm," or "delayed cerebral ischemia," or "delayed ischemic neurological deficit" with (2) "subarachnoid hemorrhage," and (3) "endovascular," or "intra-arterial," or "angioplasty." Articles were selected by reviewing titles and abstracts. Included articles were those that addressed endovascular treatment of vasospasm in clinical populations of at least 10 patients. Case reports were excluded. Among selected articles, quality of evidence was evaluated using the GRADE classification system [6].

Summary of the Literature

A total of 49 articles were included in this review. Among selected articles, 3 addressed endovascular treatment timing [7–9], 12 papaverine [11–25], 4 verapamil [26–29], 3 nicardipine [30–32], and 27 balloon angioplasty [7–10, 33–55]. The quality of evidence was low or very low for intra-arterial treatments and moderate for balloon angioplasty.

Timing of Endovascular Intervention

There have been three studies analyzing the timing of endovascular intervention for cerebral vasospasm: one multicenter randomized clinical trial studying prophylactic intervention [7] and two retrospective case series analyzing early versus delayed intervention after the onset of cerebral vasospasm [8, 9].

In a multicenter randomized clinical trial studying prophylactic balloon angioplasty for aneurysmal SAH patients [7], 175 patients with Fisher Grade III SAH were randomized to either prophylactic balloon angioplasty within 96 h after rupture ($n = 85$) or no prophylactic balloon angioplasty ($n = 90$). Target vessels were the bilateral A1 segment of the anterior cerebral artery, M1 segment of the middle cerebral artery, P1 segment of the posterior cerebral artery, basilar artery, and intradural segment of the dominant vertebral artery. Patients undergoing prophylactic balloon angioplasty had a non-significant lower incidence of DCI ($P = 0.30$). A statistically significant reduction in the number of prophylactic balloon angioplasty–treated patients requiring therapeutic rescue angioplasty was observed relative to controls ($P = 0.03$). There was a non-significant difference in 3-month clinical outcomes ($P = 0.54$). Four patients had vessel perforations during prophylactic balloon angioplasty resulting in 3 deaths. Before completion of the study, the treatment protocol was revised to exclude angioplasty of the bilateral A1 and P1 segments due to complications related to balloon angioplasty in these vessels.

Rosenwasser et al. retrospectively reviewed 84 patients who underwent balloon angioplasty with or without intra-arterial papaverine [8]. Treatment occurred within 2 h of neurological decline in 51 patients and >2 h after neurological decline in 33 patients. Patients treated within 2 h had significantly better neurological improvement than the delayed treatment patients. Bejjani et al. retrospectively analyzed 31 patients treated with balloon angioplasty, with treatment within 24 h of neurological decline in 21 patients and >24 h in 10 patients [9]. There was likewise more significant improvement in the patients treated early rather than those for whom treatment had been delayed.

Endovascular Treatments

The indications for endovascular intervention for cerebral vasospasm are not well elucidated. Endovascular intervention may be a beneficial adjunct or a replacement for medical management when medical management has failed or when there is concern for complications, such as heart failure or pulmonary edema. A number of intra-arterial agents have been described for the endovascular treatment of cerebral vasospasm [10]; however, most data come from relatively small, retrospective case series. In many cases, studies did not clarify whether data were collected prospectively or retrospectively (Table 1). Intra-arterial papaverine was studied in a small, prospective pilot study ($N = 11$) [17] and a dose-escalation study [24]. Nicardipine was evaluated in a small, prospective study ($N = 18$) [30]. Furthermore, there has been no standard dosing regimen utilized for administering intra-arterial agents, making utilization in clinical practice more challenging. Balloon angioplasty has also been evaluated with a prospective, phase II, randomized, clinical trial [7].

Intra-Arterial Vasodilators

Papaverine is an alkaloid substance that induces vasodilation of cerebral and coronary arteries through direct interactions on smooth muscle cells. Mechanistically, papaverine acts by inhibiting cyclic adenosine monophosphate and cyclic guanosine 3,5 monophosphate phosphodiesterase activity. Kassell et al. in 1992 described intra-arterial injection of papaverine for the treatment of cerebral vasospasm [11]. Two-thirds of their patients showed marked angiographic improvement following treatment and 4 in 12 patients showed clinical improvement (33.3%). Multiple case series have reported successful treatment of cerebral vasospasm using intra-arterial papaverine, with good angiographic and clinical results [11–22] (See Table 1). Two studies, however, did not find clinical benefit [23, 24], and one study reported neurological decline with possible neurotoxicity with intra-arterial injection of papaverine [25].

Table 1 Studies evaluating intra-arterial vasodilators after SAH

Reference	Design	Outcome
Papaverine		
Kassell et al. [11]	Anecdotal series of 12 patients treated with 300 mg of papaverine over 1 h	Marked angiographic reversal of narrowing in 8 patients; dramatic reversal of neurological deficits in 4; clinical deterioration in 2
Kaku et al. [12]	Series of 10 patients treated with superselective infusion of 0.2% papaverine in areas not accessible to balloon	Clinical improvement in 8 of 10 patients
Clouston et al. [13]	Nineteen treatments for 14 patients infused 6 h to 2 days after spasm became apparent clinically	Angiographic improvement in 18 of 19 sessions; best results with superselective infusion. Dramatic clinical improvement in 7 of 14 patients
Dalbasti et al. [14]	Patients at high risk for vasospasm were assigned to controlled-release papaverine pellets placed during surgery ($N = 44$) or control ($N = 73$)	Vasospasm developed in 1 of 44 patients with papaverine versus 34 of 73 controls
Fandino et al. [15]	Series of 10 patients. Balloon angioplasty preceded papaverine for 3 patients	Jugular bulb oxygen saturation improved in all patients
Firlik et al. [16]	Fifteen consecutive patients with symptomatic vasospasm received 23 treatments	Major clinical improvement in 6 of 23 treatments
Little et al. [17]	Prospective, pilot study in 11 patients with clinically significant vasospasm	Good overall clinical outcome in 7 of 11 patients
Liu et al. [18]	Retrospective review of 17 patients treated with multiple papaverine infusions	Mean cerebral circulation time improved from 6.54 to 4.19 s with papaverine. Mean circulation time in comparator control group was 5.21 s
Morgan et al. [19]	Ninety-two patients treated with papaverine as part of an intensive management program	Among patients experiencing vasospasm, 74% were independent compared with 84% independent with no vasospasm
Numaguchi and Zoarski [20]	Twenty-eight patients treated with papaverine infusion for vasospasm	Angiographic improvement occurred after 98% of treatments. Only 50% of patients with acute symptoms showed remarkable clinical improvement after papaverine
Segawa et al. [21]	Fifteen patients with vasospasm and neurological deterioration	Improved paresis or level of consciousness in 7 patients, no effect in 6. Two cases of intracerebral hematoma
Yoshimura et al. [22]	Retrospective review of 19 patients with symptomatic vasospasm treated with papaverine infusion	Symptoms reversed quickly in 15 of 19 patients
Polin et al. [23]	Thirty-one patients treated with papaverine for symptomatic vasospasm in North American trial of tirilazad for aneurysmal subarachnoid hemorrhage matched to untreated patients	No difference in 3-month Glasgow Outcome Scale between treated and untreated patients
Sawada et al. [24]	Dose-escalation study in 46 patients with vasospasm	Most beneficial dose resulted in successful dilation in 24 of 30 infused territories, with 7 patients showing marked reversal of neurological deficits (44%)
Smith et al. [25]	Retrospective review of 5 patients with vasospasm treated with intra-arterial papaverine preserved with chlorobutanol	Marked neurological decline in all patients immediately after infusion
Verapamil		
Albanese et al. [26]	Retrospective analysis of 12 patients with medically refractory vasospasm treated with intra-arterial verapamil	No new infarcts on imaging for 9 of 12 patients At final follow-up after 6–12 months, modified Rankin score $\leq$2 in 8 of 12 patients

Table 1 continued

Reference	Design	Outcome
Feng et al. [27]	Retrospective review of 29 patients treated for vasospasm with intra-arterial verapamil	Neurological improvement in 5 of 17 procedures when intra-arterial verapamil was the only treatment
Keuskamp et al. [28]	Retrospective review of 10 patients with vasospasm treated with intra-arterial verapamil	Neurological improvement after 8 of 12 procedures, with no change in 4 patients
Mazumdar et al. [29]	Retrospective assessment of change in vessel diameter measured in 15 patients undergoing intra-arterial verapamil for vasospasm	No significant improvement in vessel diameter
Nicardipine		
Badjatia et al. [30]	Prospective evaluation of 18 patients with vasospasm treated with intra-arterial nicardipine alone	Neurological improvement in 8 of 18 patients
Linfante et al. [31]	Retrospective review of 22 consecutive patients with vasospasm treated with intra-arterial nicardipine	Modified Rankin score ≤ 2 in 11 of 22 patients at discharge
Tejada et al. [32]	Retrospective review of 11 consecutive patients with vasospasm treated with intra-arterial nicardipine	Clinical improvement in 10 of 11 patients

Verapamil is an L-type calcium channel blocker. Four retrospective case series [26–29] have reported intra-arterial injection of verapamil for cerebral vasospasm (Table 1). Two studies observed improvements in arterial diameter without significant side effects [27, 28], while one trial failed to show arterial diameter improvement following intra-arterial verapamil treatment [29]. Continuous high-dose verapamil was administered to 12 patients with medically refractory vasospasm through indwelling microcatheters [26]. The treatment proved to be effective, and only 4 vessels required balloon angioplasty. No adverse complications occurred.

Nicardipine is a dihydropyridine calcium antagonist that possesses virtually equivalent pharmacologic activity to nimodipine. Three retrospective case series have reported that intra-arterial nicardipine dilates vessels in vasospasm and transiently improves neurological deficits [30–32] (See Table 1).

Balloon Angioplasty

Transluminal balloon angioplasty for vasospasm has been reported in 27 publications identified from 1984 to 2008 with 1,028 patients [7–10, 33–55]. Most studies were retrospective case series, with one prospective, randomized controlled trial that investigated prophylactic balloon angioplasty discussed earlier [7]. Improvements in vessel diameters as well as neurological deficits were observed in most studies following balloon angioplasty [7–10, 33–52]. Successfully treated vessels using balloon angioplasty translated into a reduced incidence of delayed cerebral ischemia on radiographic imaging in several studies [35, 45, 47, 48, 51]. Several studies have compared balloon angioplasty with intra-arterial papaverine or combination therapy. Lewis et al. observed significant improvement in transcranial Doppler velocity, and cerebral perfusion was analyzed using single-photon emission computed tomography (SPECT) imaging for vessels treated with balloon angioplasty compared with intra-arterial papaverine [53]. Three other studies failed to show any significant clinical benefit for patients managed with balloon angioplasty relative to intra-arterial papaverine or combination therapy [46, 54, 55]. Complications of balloon angioplasty including vessel perforation [7, 44, 47, 48], hemorrhage [9], and death [7, 47, 48] were reported.

Retreatment

Vasospasm may persist and thus necessitate multiple endovascular treatments. It is not known why vasospasm resolves completely after one endovascular procedure in some patients, while others require multiple procedures. When prophylactic balloon angioplasty was performed in a

multicenter prospective randomized controlled trial [7], the number of therapeutic rescue angioplasties performed was significantly reduced compared with controls ($P = 0.03$). One very small series of 12 patients showed that using indwelling microcatheters with continuous intra-arterial infusion of high-dose verapamil reduced the need for multiple trips to the angiography suite [26].

Conclusions

Most of the available studies included in this review investigated intra-arterial papaverine or balloon angioplasty, although interpretations are limited by a predominance of retrospective analyses and relatively small sample sizes in most studies. Intra-arterial papaverine and balloon angioplasty have generally each been shown to successfully reduce vasospasm and neurological deficits. Limited data have failed to establish superiority of intra-arterial papaverine or balloon angioplasty. Furthermore, limited data are available for evaluating nicardipine or verapamil, with positive benefits reported with nicardipine and inconsistent benefits with verapamil.

Prophylactic balloon angioplasty has been linked to a reduction in the need for therapeutic rescue balloon angioplasty in aneurysmal SAH patients. Prophylactic treatment, however, has been associated with potential risks, and the data have not shown an improvement in clinical outcome after prophylactic treatment.

In summary, endovascular intervention for clinically identified vasospasm may be indicated as when medical management has failed or when there is a concern for complications from medical management. The most complete data are available for intra-arterial papaverine or balloon angioplasty.

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