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Clinical Article

Safety and Efficacy of Transluminal Balloon Angioplasty Using a Compliant Balloon for Severe Cerebral Vasospasm after an Aneurysmal Subarachnoid Hemorrhage

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Objective : Vasospasm of cerebral vessels remains a major source of morbidity and mortality after an aneurysmal subarachnoid hemorrhage (SAH). The purpose of this study was to evaluate the safety and efficacy of transluminal balloon angioplasty (TBA) for SAH-induced vasospasm.

Methods : Eleven patients with an angiographically confirmed significant vasospasm (>50% vessel narrowing and clinical deterioration) were studied. A total of 54 vessel segments with significant vasospasm were treated by TBA. Digital subtraction angiography was used to confirm the presence of vasospasm, and TBA was performed to dilate vasospastic arteries. Medical and angiographic reports were reviewed to determine technical efficacy and for procedural complications.

Results : TBA using Hyper-Glide or Hyper-Form balloons (MicroTherapeutics, Irvine, CA) was successfully accomplished in 88.9% vasospastic segments (48 of 54), namely, in the distal internal carotid artery (100%, n=7), the middle cerebral artery (100%), including the M1 (n=10), M2 (n=10), and M3 segments (n=4), in the vertebral artery (100%, n=2), basilar artery (100%, n=1), and in the anterior cerebral artery (ACA), including the A1 (66%), A2 (66%), and A3 segments (100%). Vessel diameters significantly increased after TBA. There were no cases of vessel rupture or thromboembolic complications. GCS at one day after TBA showed an improvement in all patients except one.

Conclusion : This study suggests that TBA using Hyper-Glide or Hyper-Form balloons is a safe and effective treatment for subarachnoid hemorrhage-induced cerebral vasospasm.

Key Words : Transluminal balloon angioplasty · Vasospasm · Subarachnoid hemorrhage · Endovascular procedure.

INTRODUCTION

Cerebral vasospasm continues to be one of the leading causes of death and disability following a subarachnoid hemorrhage (SAH)[12]. Blood products that collect after SAH and remain in prolonged contact with the cerebral vessel walls induce vasospasm, which results in a narrowing of vessel lumens and compromises cerebral blood flow and oxygenation.

Clinical vasospasm can result in ischemic sequelae manifested by confusion, a decreased level of consciousness, speech and motor impairments, increased blood pressure, and a worsening headache[19].

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The primary goal of endovascular treatment for symptomatic vasospasm is to increase cerebral blood flow (CBF), and thus, to prevent infarction. In 1984, Zubkov and colleagues[27] first reported treating 33 SAH patients using balloon catheters to dilate 105 vasospastic arteries, and demonstrated a technical capacity to treat these stenotic vessels using endovascular techniques. Hoh and Ogilvy[10] estimated that 62% of patients improved clinically after TBA. As compared with the intraarterial injection of pharmacological agents, the limitation of TBA was the difficulty experienced accessing distal vasospastic segments. TBA has not been used in the distal segments of the cerebral vasculature, primarily because of safety concerns related to thinner arterial walls and the technical challenge of more distal navigation in the cerebrovasculature[6,9].

In the present study, we retrospectively studied the procedural safety and clinical efficacy of TBA for vasospasm. Furthermore, we analyzed the technical feasibility of using compliant (Hyper-Glide) or super-compliant (Hyper-Form) balloons to

navigate into distal vasospastic vessels.

MATERIALS AND METHODS

Patients with a subarachnoid hemorrhage (SAH) of all clinical grades were admitted to our department of neurosurgery. Whenever possible, surgery or coil embolization was performed within 24 hours of hemorrhage. All patients received nimodipine intravenously (2 mg/hour) about 2 weeks after diagnosis of SAH. After surgery or coil embolization, computed tomography (CT) was performed in cases that showed clinical deterioration. In the absence of structural causes, impending cerebral ischemia attributable to vasospasm was defined as an increase in transcranial Doppler (TCD) flow velocity to 150 cm/second. When TCD flow velocity was >150 cm/second, magnetic resonance imaging (MRI), including diffusion weighted (DWI) imaging and MR angiography (MRA), of the brain was performed. If an acute infarction by DWI or vessel narrowing by MR angiography was found, diagnostic cerebral angiography was recommended. The presence of a vasospasm was first verified by angiography, which was compared with the initial angiograms obtained after SAH.

Indications for TBA in specific vessels were based on clinical symptoms or the presence of significant vasospasm, the latter of which was defined as a decrease of >50% in the diameter of a vessel segment versus that in the baseline angiogram obtained at admission.

All angiographic procedures were performed by an experienced neurointerventionist through the transfemoral approach under local anesthesia. Electrocardiogram, arterial oxygen saturation, and blood pressure were appropriately monitored. After performing diagnostic angiography to establish a diagnosis of vasospasm, baseline activated clotting times (ACT) were obtained. The patient concerned received systemic heparinization and a bolus injection of heparin 3,000 IU just prior to the therapeutic procedure. An additional 1,000-IU bolus of heparin was administered every hour to maintain an ACT of >200 seconds throughout the procedure. A 6F guiding catheter (Envoy; Cordis Endovascular Corporation, Miami, FL) was positioned in the distal cervical ICA, and preprocedural angiograms were then obtained in orthogonal planes. In all patients, a 4×15 or 4×20 mm-sized compliant balloon (HyperGlide, MicroTherapeutics, Irvine, CA) for intracranial ICA or a 4×7 mm-sized super-compliant balloon (HyperForm, MicroTherapeutics, Irvine, CA) for intracranial arteries (except the ICA) was placed in the narrowed vessel segment. The balloon was tested and prepared with a 30% diluted contrast solution prior to placement to ensure rapid balloon deflation was achieved. After proper placement through the stenotic segment, the balloon was carefully inflated until the narrowed vessel segment almost reached its normal diameter on the initial angiogram obtained after SAH. The balloon was immediately deflated as soon as the target diameter was reached, and then the procedure was repeated. TBA was attempted in all stenotic vessel segments. Post-procedural angiography was performed to assess angiographic results, and post-procedural CT to identify the presence of intracerebral hemorrhage or of thromboembolic complications.

The primary safety points were the occurrence of vessel rupture due to balloon inflation or wire perforation and thromboembolic complications. Other complications were also recorded, including any evidence of arterial injury or groin hematoma requiring transfusion or surgical repair.

The efficacy of TBA was assessed by monitoring neurologic condition and CT images. We used the Glasgow Coma Scale (GCS) after the procedure and the Glasgow outcome Scale (GOS) at discharge to access clinical outcomes. New ischemic lesions on CT scans obtained after initiating TBA were considered to be related to persistent vasospasm after treatment.

RESULTS

The characteristics of the 11 SAH patients that underwent TBA using a Hyper-Glide (MicroTherapeutics, Irvine, CA, USA) or a Hyper-Form balloon (MicroTherapeutics, Irvine, CA) in stenotic vessel segments between January 2008 and December 2008 are summarized in Table 1.

TBA was successfully accomplished in 48 of the 54 vasospastic segments (88.9%), that is, in distal internal carotid arteries (100%, n=7), middle cerebral arteries (100%; M1 n=10, M2 n=10, and M3 segments n=4), anterior cerebral artery [70%; A1 (66%, 6 of 9), A2 (66%, 6 of 9), and A3 segments (100%, n=2)], vertebral arteries (100%, n=2), and in a single basilar artery (100%, n=1) (Table 2). TBA was attempted in all ACA segments and was successfully accomplished in 66% of A1 and A2 segments. In patients 1, 6, and 11, we were unable to navigate a balloon microwire into the A1 segment because of an unfavorable angle of the anterior cerebral artery origin, and therefore, the untreated A1 and A2 segments were treated by intraarterial papaverine injection.

Immediate post-procedural angiographies demonstrated significant increases in vessel diameters in all eleven patients. Vasospasm did not recur in any patients. No procedure-related complications, including vessel dissection, rupture, or thromboembolic events were identified.

Of the eleven patients, nine patients were discharged with GOS '5'. Patient 1 had a small amount of subdural hematoma (SDH) at the craniotomy site and suffered from increased SDH after TBA. He finally died of aspiration pneumonia. Patient 6 showed acute infarction of low-density on pre-procedural CT, and an intracranial hemorrhage (ICH) occurred within a low density lesion on pre-procedural CT one week later. This was considered a hemorrhagic transformation of a subacute infarction. He was discharged with GOS '3'.

DISCUSSION

Severe narrowing or vasospasm of the large arteries is a com-

Table 1. Summary of characteristics of patients with cerebral vasospasm

Patients No	Sex/Age	Location of Aneurysms	Aneurysm Treatment	Days between SAH and TBA	GCS		Recurrent Vasospasm	GOS at Discharge
					Pre-TBA	1st Post-TBA day		
1	M/32	Left ACOM	Clip	7	14	7	No	1
2	M/54	Right ACOM	Coil	8	14	15	No	5
3	F/42	Left PCOM	Coil	12	13	15	No	5
4	F/44	Left PICA	Coil	0*	11	12	No	5
5	F/46	Rt. PCOM	Coil	0*	14	14	No	5
6	M/26	Left MCA	Coil	7	15	15	No	3
7	M/54	Left ACOM	Clip	9	13	15	No	5
8	F/53	Left distal ICA	Clip	13	11	13	No	5
9	M/62	Left MCA bifurcation	Clip	13	12	15	No	5
10	M/35	Right MCA bifurcation	Coil	13	15	15	No	5
11	F/60	Left ACOM and PCOM	Coil	14	14	14	No	5

*Coil embolization and TBA at the same procedure. SAH : subarachnoid hemorrhage, TBA : transluminal balloon angioplasty, GOS : Glasgow outcome score, GCS: Glasgow coma scale, ACOM : anterior communicating artery, PCOM : posterior communicating artery, PICA : posterior inferior cerebellar artery, ICA : internal cerebral artery, MCA : middle cerebral artery

Table 2. Location of vessels treated with transluminal balloon angioplasty

Patients No.	Affected vessel segment	Failed TBA segments	Causes of Failed TBA
1	Right distal ICA, M1, A1, A2	A1, A2	Fail to navigate balloon catheter into A1 segment
2	Right A1, A2		
3	Left M1, M2, M3, A1, A2, A3		
4	Both V4, BA		
5	Right distal ICA, M1, M2		
6	Left distal ICA, M1, A1, A2	A1, A2	Fail to navigate balloon catheter into A1 segment
7	Right M1, M2, A2, A3		
8	Left distal ICA, M1,M2,M3, A1		
9	Left distal ICA, M1,M2		
10	Both distal ICA, A1, A2 Right M1, M2, M3, Lt M2		
11	Both M1, M2, A1, A2	A1, A2	Fail to navigate balloon catheter into A1 segment

TBA : transluminal balloon angioplasty, ICA : internal cerebral artery, M1, M2, M3 : 1st, 2nd, 3rd segment of middle cerebral artery, A1, A2, A3 : 1st, 2nd, 3rd segment of anterior cerebral artery, V4 : 4th segment of vertebral artery, BA : Basilar artery

Table 3. Efficacy of Transluminal balloon angioplasty

Patients No.	Symptoms and Signs		Clinical Results at Discharge
	Pre-TBA	Post-TBA	
1	Drowsy mentality and disorientation. Right motor weakness (4/5) Small amount of SDH at craniotomy site	Stupor Increased amount of SDH	Dead
2	Drowsy mentality, headache, disorientation	Alert, mild headache.	Alert
3	Drowsy mentality, disorientation. Confusion, Right motor weakness (4/5)	Alert Full recovery of motor weakness	Alert
4	Deep drowsy	Drowsy	Alert
5	Drowsy	Drowsy	Alert
6	Acute infarction with small hemorrhagic transformation at left middle cerebral artery territory	No increased hemorrhagic transformation after 1 day Increased hemorrhage after 1 week	Stupor
7	Right motor weakness (3/5) and dysarthria.	Full recovery of motor weakness and dysarthria	Alert
8	Right motor weakness (4/5) and motor aphasia	Full recovery of motor weakness and motor aphasia	Alert
9	Confusion and disorientation	Alert	Alert
10	Left motor weakness (4/5)	Full recovery of motor weakness	Alert
11	Drowsy	Drowsy	Alert

TBA : transluminal balloon angioplasty, SDH : subdural hematoma

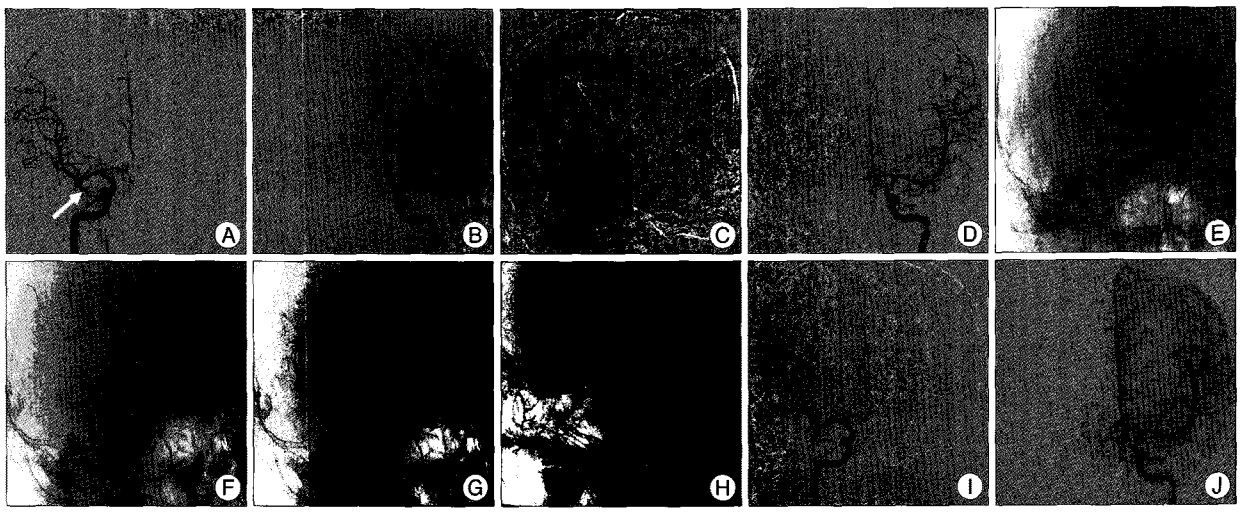

Fig. 1. Both ICA angiograms demonstrated severe vasospasm in a patient in whom worsening left side motor power developed 13 days after coil embolization of right MCA bifurcation aneurysm. A and B : Anteroposterior views of both ICA angiograms showed a ruptured right MCA bifurcation aneurysm (arrow) without vasospasm. C and D : Anteroposterior views of both ICA angiograms 13 days after the coil embolization demonstrated mild to severe vasospasm of bilateral distal ICA, A1, and A2, right M1, M2, and M3, and left M2 segments. E-H : Balloon angioplasty was done using Hyperform balloon for vasospastic vessels including ICA, MCA and ACA. I and J : Anteroposterior views of both ICA angiograms after balloon angioplasty showed improvement of cerebral blood flow with restoration of the vasospastic vessels. ICA : internal cerebral artery, MCA : middle cerebral artery.

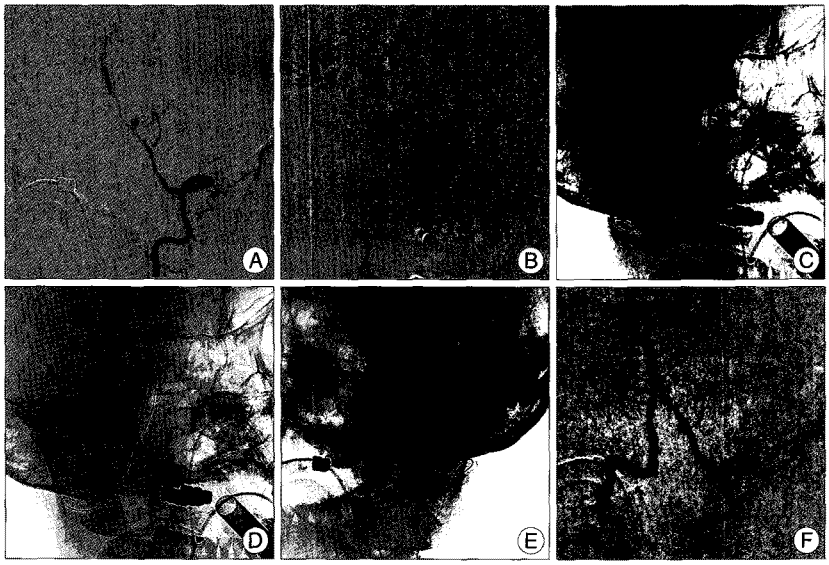

Fig. 2. Both VA angiograms demonstrated severe vertebral in both VA and basilar arteries. A : Oblique anteroposterior view of left vertebrobasilar angiogram shows a ruptured aneurysm arising from left posterior inferior cerebellar artery and diffuse vasospasm of left vertebral and basilar arteries. B : Anteroposterior view of right vertebrobasilar angiogram shows diffuse vasospasm of vertebral and basilar arteries. C and D : Oblique anteroposterior angiogram demonstrate balloon angioplasty for vasospasm of right vertebral and basilar arteries. E : Oblique anteroposterior angiogram demonstrates coil embolization of the posterior inferior cerebellar artery aneurysm and balloon angioplasty for vasospasm of left vertebral artery. F : Immediate post-porcedural left vertebrobasilar angiogram reveals complete embolization of the aneurysm and restoration of the vasospastic vertebral and basilar arteries.

mon delayed phenomenon after SAH[24]. Vasospasm may result in reduced cerebral flow and tissue ischemia due to interrelated physiological processes, which include a reduction in distal perfusion pressure, and often, distal autoregulatory dysfunction[26].

Progress in the treatment of vasospasm has not evolved in parallel with the treatment of ruptured aneurysms. Current standard medical treatment options include the administration of nimodipine and controlled hypervolemia, hypertension, and hemodilution therapy[18]. For symptomatic vasospasm refractory to hemodynamic therapy, endovascular strategies, such as balloon angioplasty[6] and intra-arterial spasmolysis with papaverine[12] or nimodipine[2] have been recommended.

Intraarterial papaverine (IAP) has been shown to reverse angiographic vasospasm and to reduce velocities detected by TCD[7,12]. However, clinical trials have failed to demonstrate the effectiveness of IAP on outcome[17]. The most likely reason for this failure is its short effect duration, which is limited to an only a few hours[23]. A rapid increase in intracranial pressure (ICP) is another serious complication of IAP[15]. This may attributed to a post-infusion increase in CBF and cerebral blood volume due to cerebral vasodilatation. Other reported complications include thrombocytopenia, hypotension, seizures, transient neurological deficits, monocular blindness, precipitation of papaverine crystal emboli during infusion, and paradoxical worsening of vasospasm leading to cerebral infarction[14,19].

Intra-arterial nimodipine (IAN) in patients with severe vasospasm has been reported by several authors[3,4,8]. Data produced in a larger series by Biondi et al.[2] suggests that IAN is effective

and safe for the treatment of vasospasm after SAH. However, no information is available regarding its influence on cerebral perfusion. Some of the main systemic complications of nimodipine are hypotension, bradycardia, rash, and diarrhea. Neither Biondi et al.[2] nor Hui and Lau[11] reported any significant changes in blood pressure, heart rate, or intracranial pressure after nimodipine infusion, though one patient experienced a run of ventricular ectopic beats over 10 to 15 seconds. Furthermore, there is a lack of information about the effect duration of nimodipine infusion.

The use of TBA in narrowed cerebral vessel segments resulting from vasospasm was first described in 1984 by Zubkov[27]. In a recent report, Zwienenberg-Lee et al.[28] described the probable superior effectiveness of TBA as compared with a pharmacological treatment, and its relatively low risk.

The precise mechanism by which TBA reverses cerebral vasospasm remains only incompletely understood. Furthermore, the durability of the procedure and the reasons for the low rate of recurrent spasm of treated vessel segments remain to be elucidated[5,13]. Using the scanning electron microscope, Yamamoto et al.[25] examined changes in the three-dimensional structure of connective tissues in vessel walls after balloon angioplasty. It was found that the normal structure of collagen fibers in vessel walls was significantly affected by balloon dilation. Stretched and torn fibers were observed when 3 atm of pressure was applied, but endothelium was not damaged. These investigators concluded that balloon dilation mechanically disrupts proliferating connective tissues in the vasospastic vessel wall, and that this prolongs the effect of the procedure.

Beck et al.[1] reported that TBA leads to hemodynamic effects that can be quantified using perfusion-weighted (PW)/DW MR imaging. Furthermore, in cases with a severe PW/DW imaging mismatch, successful TBA improved tissue perfusion and prevented cerebral infarction. Terry et al.[22] reported upon the safety and technical efficacy of TBA. Seventy-five patients underwent 85 TBAs for the treatment of SAH-induced vasospasm. No vessel rupture or perforation occurred, and TBA was successfully accomplished in the distal ICA (100%), proximal middle cerebral (94%), vertebral (73%), and basilar arteries (88%). However, TBA was successful in only 34% of ACAs, because of severe vasospasm or an unfavorable angle.

The present study demonstrates that balloon angioplasty is safe and has a high rate of technical efficacy in most stenotic segments, with a success rate of 100% for the distal ICA, MCA, and vertebrobasilar artery and of 70% for the ACA. In patients 1, 6, and 11, we were unable to navigate a balloon microwire into the A1 segment because of an unfavorable angle of the anterior cerebral artery origin. As was found by Terry[22], the success rate for the ACA was lower than found for other intracranial arteries, and this too was found to be the result of an angle that prevented navigation of the balloon microwire. Accordingly, the untreated A1 and A2 segments were treated by intraarterial papaverine injection.

In the present study, complications, including, vessel dissection, rupture, or thromboembolic events, did not occur that could be directly attributed to the angioplasty procedure. Unlike coronary balloons[21], compliant (Hyper-Glide) and super-compliant (Hyper-Form) balloons are suitable for SAH-induced intracranial spastic vessels. To prevent vessel injuries, we carefully inserted balloons and slowly inflated them in spastic vessel segments to 80-100% of normal vessel diameter, and they were then immediately deflated. We adopted this procedure for all spastic vessels.

Although Sedat[20] reported restenosis after balloon angioplasty for a cerebral vasospasm, almost all clinical studies[9,16,22,28] have concluded that angioplasty achieves long-lasting dilatation of spastic cerebral arteries. Furthermore, in our cases, no vasospasm recurred after TBA.

Our study has a number of limitations that should be considered. First, it is limited by its retrospective nature, second, the small number of patients recruited, and third, no objective analysis was performed on the effect of balloon angioplasty in terms of pre- and post-operative perfusion or angiography.

However, this study demonstrates that TBA provides a technically safe and effective treatment method for vasospasm that improves cerebral blood flow and prevents tissue infarction.

CONCLUSION

Endovascular therapies should be considered when symptomatic cerebral vasospasm becomes refractory to maximal medical management. Our research demonstrates that TBA for vasospasm is technically safe and effective treatment method to improve cerebral blood flow and prevent tissue infarction. However, the safety and efficacy of this technique compared with medical therapy and intra-arterial injection of vasodilators remains to be determined. Greater experience should allow better definition of the role of TBA in the treatment of SAH-induced vasospasm.

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