Surgical Neurology International

OPEN ACCESS

For entire Editorial Board visit : http://www.surgicalneurologyint.com

Editor-in-Chief: **James I. Ausman**, MD, PhD University of California, Los Angeles, CA, USA

Original Article

Cerebral artery restenosis following transluminal balloon angioplasty for vasospasm after subarachnoid hemorrhage

Katsuya Umeoka, Shushi Kominami, Takayuki Mizunari, Yasuo Murai[1], Shiro Kobayashi , Akira Teramoto[1]

Department of Neurosurgery, Nippon Medical School, Chiba-Hokusou Hospital, 1715 Kamakari, Inzaishi, Chiba, 270-1694, [1]Department of Neurosurgery, Nippon Medical School, 1-1-5 Sendagi, Bunkyo-ku, Tokyo, 113-8603, Japan

E-mail: *Katsuya Umeoka - katsuya@nms.ac.jp; Shushi Kominami - shushi@nms.ac.jp; Takayuki Mizunari - mizunari@nms.ac.jp; Yasuo Murai - ymurai@nms.ac.jp; Shiro Kobayashi - sirok@nms.ac.jp; Akira Teramoto - a-tera@nms.ac.jp
*Corresponding author

Received: 3 September 10 Accepted: 14 March 11 Published: 19 April 11

This article may be cited as:
Umeoka K, Kominami S, Mizunari T, Murai Y, Kobayashi S, Teramoto A. Cerebral artery restenosis following transluminal balloon angioplasty for vasospasm after subarachnoid hemorrhage. Surg Neurol Int 2011;2:43.

Available FREE in open access from: http://www.surgicalneurologyint.com/text.asp?2011/2/1/43/79758

Abstract

Background: Although percutaneous transluminal angioplasty (PTA) is a widely used less invasive method to treat coronary artery stenosis, 10% of treated patients experience restenosis. Restenosis also occurs in approximately 5% of patients subjected to carotid artery stenting. Animal and human data suggested that re-stenosis is a response to injury incurred during PTA. As PTA has come into wide use to manage symptomatic cerebral vasospasm after subarachnoid hemorrhage (SAH) we studied the incidence of restenosis after PTA for cerebral vasospasm.

Methods: Our study population consisted of 32 patients who had undergone PTA. They were followed by cerebral or 3DCT angiography or MRA for 6 126 months post-PTA (mean 48.65 months) to diagnose restenosis of the cerebral artery. We compared the size of the cerebral artery on the PTA and the contralateral side.

Results: All 32 patients underwent successful PTA of 38 vascular territories and all manifested angiographic improvement of vasospasm. None suffered restenosis during the follow up period.

Conclusion: PTA resulted in a significant improvement in the vessel diameter in patients with vasospasm after SAH and they did not suffer restenosis in the course of prolonged follow-up.

Key Words: Percutaneous transluminal angioplasty, restenosis, subarachnoid hemorrhage, vasospasm

Access this article online

Website: www.surgicalneurologyint.com

DOI: 10.4103/2152-7806.79758

Quick Response Code:

INTRODUCTION

Symptomatic cerebral vasospasm is the most common contributor to acute focal ischemia after aneurysmal subarachnoid hemorrhage (SAH) and at least two-thirds of SAH patients present with angiographic evidence of some degree of vessel narrowing between days 4 and 14.[19] As a 1.5 to 3 fold increase in morbidity and mortality during the first 2 weeks after aneurysmal SAH is attributable to cerebral vasospasm,[8,15,20,26] its prompt recognition and treatment may improve treatment outcomes. The first line treatment for cerebral vasospasm is blood pressure elevation, volume expansion, and hemodilution (triple H therapy),[18,19,21] however, up to 40% of patients fail to respond.[2,13] Zubkov *et al.*[29] first reported performing PTA in patients with cerebral vasospasm. Subsequently,

its safety and efficacy for reversing cerebral vasospasm have been documented.[3,7,14,23,29] Although PTA is now widely used as a less invasive method to treat coronary artery stenosis, 10% of patients suffer restenosis.[12] Restenosis also occurs after carotid artery stenting (CAS) in no less than 5% of patients[16,17,24] and in 36% of patients who had undergone stenting to treat intracranial atherosclerosis.[25] The rate of restenosis after PTA for cerebral vasospasm remains to be investigated. In animal models, atherosclerotic vessels manifested a more severe degree of restenosis[6,11] and Tsuruta *et al.*[27] developed a model of post angioplasty stenosis using rats fed a normal diet. Normal arteries may develop cerebral vasospasm, and PTA is the technique used to expand these vessels. We investigated the incidence of restenosis after PTA for cerebral vasospasm.

MATERIALS AND METHODS

We reviewed 32 patients with aneurysmal SAH who suffered symptomatic cerebral vasospasm and were treated with PTA between 1995 and 2009. All underwent aneurysmal clipping within 2 days of rupture. Clinically suspected symptomatic vasospasm was considered an indication for diagnostic four vessel cerebral angiography. Diagnostic angiograms were obtained to confirm the adequacy of aneurysmal clipping and the presence of vasospasm. Vasospasm was considered severe when there was angiographic evidence of narrowing of the arterial lumen by more than 50% of the normal caliber. Patients with angiographically confirmed vasospasm and patients in whom the distribution explained the deficit underwent PTA. Arterial access was gained via a 90 cm 6Fr sheath (Envoy, MPC, Cordis, Johnson and Johnson, Tokyo, Japan) placed percutaneously in the femoral artery. A 2.5 to 5.0 mm 0.08 mL PTA catheter (model TRANVAS, MAVI balloon, Kaneka Medix, Osaka, Japan) was advanced coaxially through a guide catheter. Subsequently, mechanical dilation by PTA was performed to treat proximal vasospasm. Angioplasty balloons were inflated for 2 s to the vessel diameter; the procedure was usually performed segmentally from distal to proximal points. All angioplasty balloon manipulations were under systemic heparinization (5000 U) performed immediately before balloon introduction and not reversed after the procedure. A postangioplasty angiogram was obtained in all cases to evaluate changes in the vessel caliber and to identify any vessel damage. No arteries were expanded beyond their normal diameter during angioplasty, and no balloon ruptures occurred. After their return to the neurosurgical intensive care unit the patients received standard medical management for vasospasm. After intensive care we performed 3DCTA, angiography, or MRA to diagnose aneurysmal recurrence and arterial restenosis.

RESULTS

Our study included 32 patients (12 men and 20 women, mean age 51.7 years, range 27 80 years) with aneurysms in the anterior circulation. PTA was used successfully to treat 38 vascular territories in the 32 patients with post SAH vasospasm. The patient characteristics are shown in Table 1. All patients manifested angiographic improvement of vasospasm. Follow up was by cerebral or 3DCT angiography or MRA and ranged from 6 to 126 months (mean 12 months) to diagnose restenosis of the cerebral artery. Comparison of the size of the cerebral artery on the PTA and the contralateral side indicated that none of the 32 patients suffered restenosis.

Table 1: Characteristics of our 32 patients with percutaneous transluminal angioplasty for cerebral vasospasm

Case	Age/Sex	Site of PTA	Duration of follow-up (months)
1	80/F	bil M1	50
2	59/M	Lt M1	22
3	36/F	bil M1	50
4	36/F	Lt M1	16
5	55/F	Rt M1	9
6	40/F	bil M1	126
7	42/F	bil M1	120
8	59/F	Rt M1	116
9	51/F	Lt A1 M1	107
10	44/M	bil M1	37
11	58/M	Lt A1	90
12	51/F	bil IC M1	26
13	51/M	Lt M1 A1	32
14	51/F	Rt IC M1 A1 Lt M1	91
15	51/M	Lt M1 A1	91
16	55/M	bil A1 M1	93
17	45/F	bil IC M1	84
18	38/M	Lt M1	51
19	76/F	Lt M1	6
20	54/F	Rt M1	8
21	36/M	Lt M1	15
22	44/F	bil M1	9
23	65/F	Lt A1 M1	20
24	54/M	Rt M1	33
25	27/M	Lt M1	10
26	67/M	Rt M1	110
27	73/F	Rt M1	31
28	55/F	bil M1	37
29	63/F	Rt M1	18
30	43/F	bil A1 M1	22
31	62/F	bil M1	20
32	36/M	Lt IC	7

M1: M1 portion of the MCA, A1: A1 portion of the ACA, ICA: Internal carotid artery, PTA: Percutaneous transluminal angioplasty

ILLUSTRATIVE CASE

A 36 year old woman presented with massive SAH due to a dorsal aneurysm at the left internal carotid artery (Hunt and Hess grade III, Fisher group 3). She underwent trapping of the left internal carotid artery and a radial artery external carotid artery bypass involving the M2 portion of the MCA. Although she responded well to verbal commands after the operation, one week later she developed consciousness disturbance; the mean TCD velocity was increased to 140 cm/s. Emergent angiography demonstrated severe spasms of the M1 portion of the MCA and the A1 portion of the ACA [Figure 1a]. Immediate PTA to the M1 portion of the MCA resulted in normalization of the vessel size [Figure 1b]; her consciousness disturbance improved and the mean TCD velocity was 80 cm/sec. She was discharged 1 month later without neurological deficit. She was followed at regular intervals and 3DCTA obtained 50 months after PTA yielded no evidence of MCA restenosis [Figure 1c].

DISCUSSION

Our assessment of the incidence of restenosis after PTA for post SAH vasospasm documented no restenosis in the course of prolonged follow up.

Suggested mechanisms underlying the occurrence of restenosis include intimal hyperplasia[4,5,9,28] attributable to the migration and proliferation of vascular smooth muscle cells and the migration and myofibroblastic transformation of adventitia cells.[9,28] Platelets and inflammatory cells such as macrophages and lymphocytes accumulate after endothelial and medial injury.[4]

Wound healing after PTA is dominated by smooth muscle cells.[28] Wall lacerations that occur during PTA expose smooth muscle cells to blood, an event that initiates an excessive fibrocellular tissue response.[9]

PTA restenosis is usually related to atherosclerotic lesions in extracranial and intracranial vessels and not normal endothelialization. We posit that in our patients, the absence of restenosis after intracranial angioplasty for vasospasm was attributable to the absence of atherosclerosis in the vessel wall.

Transmission electron micrographs of arterial walls after PTA showed flattening of endothelial cells, straightening and thinning of the internal elastic lamina, and straightening and crowding of the smooth muscle cells in the tunica media without evidence of frank rupture. These observations suggest that cellular and subcellular mechanical disruption may result in the post PTA impairment of smooth muscle function.[1,23] Mechanical injury to the arterial wall during PTA may produce hyperplasia and result in restenosis of the treated arterial segment. Andersen *et al.*[1] who developed a stenotic model using an oversized chain encircled angioplasty balloon produced a circumferential deep vessel wall injury in pigs by inflating and withdrawing the balloon in the coronary artery. They hypothesized that in their model, deep vessel wall injury induced an exuberant circumferential healing response thereby increasing the risk for stenosis.

In a canine model of cerebral vasospasms, histological evaluation of the PTA segments immediately after PTA showed denuding of endothelial cells and stretching of the internal elastic lamina without disruption of the muscle layer.[22] These histological changes are different from the vascular changes usually observed after PTA for atherosclerotic arteries such as destruction of the vascular structure, desquamation, and splitting of the media or internal elastic layer.[10]

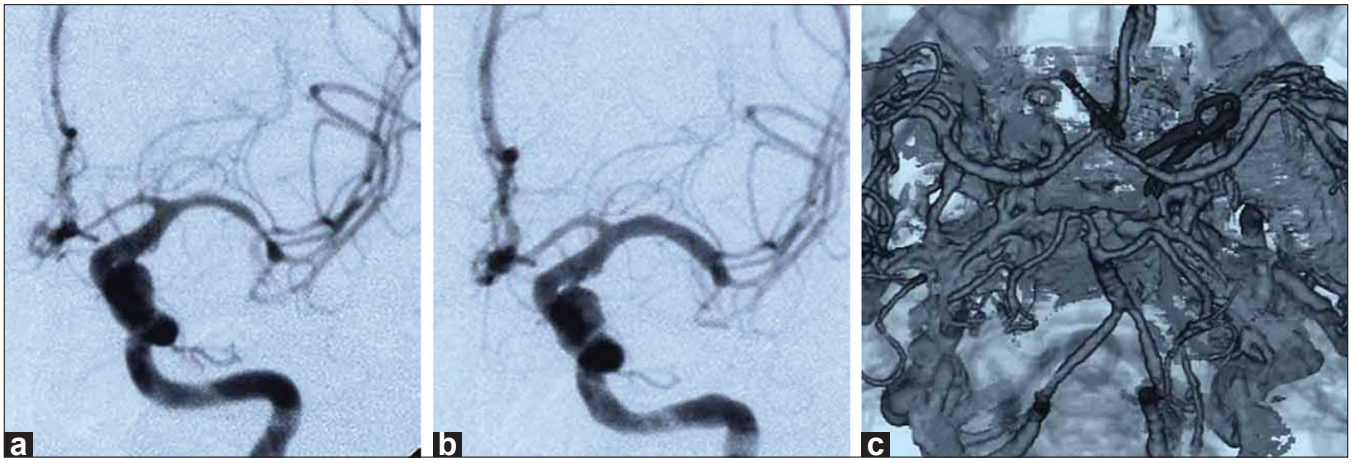

Figure 1: (a) Left internal carotid arteriogram (frontal view) showing severe vasospasm of the M1 portion of the MCA and the A1 portion of the ACA. (b) Angiogram obtained immediately after angioplasty showing the MCA to be normal in size. (c) 3D-CTA obtained 50 months after percutaneous transluminal angioplasty. There is no evidence of MCA restenosis

In our patients the angioplasty balloons were inflated to the vessel diameter and no arteries were expanded beyond their normal diameter during the procedure. Consequently, there was no mechanical injury to the arterial wall and there were no restenotic sequelae in any of the 32 patients. PTA should only be performed by expert operators, taking care that there is no damage to the vascular endothelium.

CONCLUSION

PTA is a less invasive method to treat cerebral vasospasm after aneurysmal SAH. Mechanical injury to the arterial wall during PTA may result in post PTA restenosis. Only expert operators should perform PTA and damage to the vascular endothelium must be avoided.

REFERENCES

1. Andersen HR, Maeng M, Thorwest M, Falk E. Remodeling rather than neointimal formation explains luminal narrowing after deep vessel wall injury: Insights from a porcine coronary (re)stenosis model. Circulation 1996;93:1716-24.
2. Biller J, Godersky JC, Adams HP Jr. Management of aneurysmal subarachnoid hemorrhage. Stroke 1988;19:1300–5.
3. Brothers MF, Holgate RC. Intracranial angioplasty for treatment of vasospasm after subarachnoid hemorrhage: Technique and modifications to improve branch access. AJNR Am J Neuroradiol 1990;11:239–47.
4. Bruneval P, Guermonprez JL, Perrier P, Carpentier A, Camilleri JP. Coronaryartery restenosis following transluminal coronary angioplasty. Arch Pathol Lab Med 1986;110:1186-7.
5. Carson SN, Esquivel CO, French SW. Experimental carotid stenosis due to fibrous intimal hyperplasia. Surg Gynecol Obstet 1981;153:883-8.
6. Clowes AW, Ryan GB, Breslow JL, Karnovsky MJ. Absence of enhanced intimal thickening in the response of the carotid arterial wall to endothelial injury in hypercholesterolemic rats. Lab Invest 1976;35:6-17.
7. Clyde BL, Firlik AD, Kaufmann AM, Spearman MP, Yonas H. Paradoxical aggravation of vasospasm with papaverine infusion following aneurysmal subarachnoid hemorrhage. Case report. J Neurosurg 1996;84:690–5.
8. Dorsch NW, King MT. A review of cerebral vasospasm in aneurysmal subarachnoid hemorrhage. Part I: Incidence and effects. J Clin Neurosci 1994;1:19-26.
9. Essed CE, van den Brand M, Becker AE. Transluminal coronary angioplasty and early restenosis. Fibrocellular occlusion after wall laceration. Br Heart J 1983;49:393-6.
10. Fujii Y, Takahashi A, Yoshimoto T. Percutaneous transluminal angioplasty in a canine model of cerebral vasospasm: Angiographic, histologic, and pharmacologic evaluation. Surg Neurol. 1995;44:163-70.
11. Fukuyama J, Ichikawa K, Hamano S, Shibata N. Tranilast suppresses the vascular intimal hyperplasia after balloon injury in rabbits fed on a high-cholesterol diet. Eur J Pharmacol 1996;318:327-32.
12. Garg S, Serruys PW: Coronary stents. Looking forward. J Am Coll Cardiol 2010;56:S43-78.
13. Haley EC Jr, Kassell NF, Torner JC, Truskowski LL, Germanson TP. A randomized trial of two doses of nicardipine in aneurysmal subarachnoid hemorrhage: A report of the Cooperative Aneurysm Study. J Neurosurg 1994; 80:788–96.
14. Higashida RT, Halbach VV, Dowd CF, Dormandy B, Bell J, Hieshima GB. Intravascular balloon dilatation therapy for intracranial arterial vasospasm: Patient selection, technique, and clinical results. Neurosurg Rev 1992;15: 89–95.
15. Kassell NF, Sasaki T, Colohan AR, Nazar G. Cerebral vasospasm following aneurysmal subarachnoid hemorrhage. Stroke 1985;16:562–72.
16. Koebbe CJ, Liebman K, Veznedaroglu E, Rosenwasser R. The role of carotid angioplasty and stenting in carotid revascularization. Neurol Res 2005;27: S53-8.
17. Levy EI, Hanel RA, Lau T, Koebbe CJ, Levy N, Padalino DJ. Frequency and management of recurrent stenosis after carotid artery stent implantation. J Neurosurg 2005;102:29-37.
18. Loch Macdonald R. Management of cerebral vasospasm. Neurosurg Rev 2006;29:179–93.
19. Macdonald LR, Weir B. Epidemiology. Cerebral vasospasms. San Diego (CA): Academic Press; 2001.
20. Macdonald RL, Pluta RM, Zhang JH. Cerebral vasospasm after subarachnoid hemorrhage: The emerging revolution. Nat Clin Pract Neurol 2007;3:256–63.
21. Mathis JM, Jensen ME, Dion JE. Technical considerations on intra-arterial papaverine hydrochloride for cerebral vasospasm. Neuroradiology 1997; 39:90–8.
22. Megyesi JF, Findlay JM, Vollrath B, Cook DA, Chen MH. *In vivo* angioplasty prevents the development of vasospasm in canine carotid arteries. Pharmacological and morphological analyses. Stroke 1997;28:1216-24.
23. Rabinstein AA, Friedman JA, Nichols DA, Pichelmann MA, McClelland RL, Manno EM, *et al*. Predictors of outcome after endovascular treatment of cerebral vasospasm. AJNR Am J Neuroradiol 2004; 25:1778–82.
24. Setacci C, Pula G, Baldi I, de Donato G, Setacci F, Cappelli A. Determinants of in-stent restenosis after carotid angioplasty: A case-control study. J Endovasc Ther 2003;10:1031-8.
25. Siddiq F, Vazquez G, Memon MZ, Suri MF, Taylor RA, Wojak JC, *et al*. Comparison of primary angioplasty with stent placement for treating symptomatic intracranial atherosclerotic diseases: A multicenter study. Stroke 2008;39:2505-10.
26. Treggiari-Venzi MM, Suter PM, Romand JA. Review of medical prevention of vasospasm after aneurysmal subarachnoid hemorrhage: A problem of neurointensive care. Neurosurgery 2001;48:249–61.
27. Tsuruta W, Yamamoto T, Suzuki K, Yoshida F, Matsumura A. Simple new method for making a rat carotid artery post-angioplasty stenosis model. Neurol Med Chir (Tokyo) 2007;47:525-9.
28. Ueda M, Becker AE, Naruko T, Kojima A. Smooth muscle cell de-differentiation is a fundamental change preceding wound healing after percutaneous transluminal coronary angioplasty in humans. Coron Artery Dis 1995;6:71-81.
29. Zubkov YN, Nikiforov BM, Shustin VA. Balloon catheter technique for dilatation of constricted cerebral arteries after aneurysmal SAH. Acta Neurochir (Wien) 1984;70:65-79.