

Safety and Efficacy of Percutaneous Transluminal Angioplasty for Intracranial Atherosclerotic Stenosis

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Background and Purpose Percutaneous transluminal angioplasty (PCTA) is increasingly used to treat extracerebral arterial stenosis. The present study evaluates the safety and efficacy of PCTA treatment of symptomatic intracranial atherosclerotic stenosis.

Methods A series of 22 vessels in 17 patients were treated with PCTA. All patients had recurrent neurological symptoms referable to the stenotic vessel despite optimal medical therapy. Critical (>70%) arterial stenosis was confirmed by angiogram, and angioplasty was performed with a 3.0- to 3.5-mm Stealth balloon.

Results The average preangioplasty stenosis (North American Symptomatic Carotid Endarterectomy Trial criteria) was $72 \pm 8\%$ (mean \pm SD), with a significant improvement seen after angioplasty; the best angiographic stenosis (after healing of

intimal injury, if any) was $43 \pm 24\%$ ($P < .001$). Overall PCTA was successful in 82% of the vessels. There were two strokes during angioplasty for a 30-day morbidity rate of 9.1% per treated vessel and 11.7% per case. The other 15 patients were clinically evaluated at 3 and 6 months; all cases were without further events. Restenosis was evaluated in 8 patients (12 vessels) with an angiogram at 6 months showing further improvement compared with the initial post-PCTA stenosis ($51 \pm 10\%$ versus $37 \pm 21\%$ [$P = .05$]).

Conclusions PCTA may be a beneficial therapy in selected cases of symptomatic intracranial atherosclerotic stenosis. Further study using a randomized trial is needed. (*Stroke*. 1995;26:1200-1204.)

Key Words • angioplasty, transluminal • cerebral arteries • endovascular therapy

Percutaneous transluminal angioplasty (PCTA) is an established method of treating coronary, femoral, renal, and other arterial stenosis. PCTA has recently been used to treat extracranial stenosis secondary to atherosclerosis,¹⁻⁷ fibromuscular dysplasia,^{8,9} and vasculitis.¹⁰ The overall efficacy rates (less than 50% residual stenosis) for these studies range from 50% to 70% with a reported 5% to 10% complication rate.¹¹ However, treatment of intracranial vessels has been avoided because of concerns about inducing ischemia through thromboemboli and risk of vessel rupture.

Recent advances in microcatheter and balloon technology have led to a renewed interest in treating intracranial lesions. Several studies have found PCTA to be very beneficial in cases of intracranial vasospasm in subarachnoid hemorrhage.¹²⁻¹⁵ There have been several case reports of successful treatment of intracranial atherosclerosis,^{1,2} but these have not been evaluated in a larger series. This study was undertaken to evaluate the

safety and efficacy of PCTA in our group of selected patients with high-grade intracranial stenosis who were symptomatic despite optimal medical therapy.

Subjects and Methods

Between July 1992 and July 1994 transluminal balloon angioplasty was performed in 22 intracranial vessels in 17 patients. All patients gave written informed consent to participate, with treatment being offered on a compassionate-use basis. Selection criteria for therapy included (1) at least 70% stenosis of one intracranial vessel; (2) recurrent transient ischemic attack or stroke referable to this vessel (see Table) despite maximal medical therapy, which included therapeutic anticoagulation or in some cases combined aspirin and anticoagulation; and (3) stenosis presumed secondary to atherosclerosis.

All patients had a complete neurological examination, routine laboratory tests, and head CT before angiogram. Baseline four-vessel angiography was performed to evaluate the collateral supply distal to the stenotic vessel, to look for tandem stenotic lesions, and to evaluate the size of the normal vessel adjacent to the stenosis. The size of the normal vessel was determined by placing 10-mm round markers on both sides and the front and back of the head. Anteroposterior and lateral radiographs were obtained. On the assumption that the normal vessel was midway between the two markers, a 10-mm standard was made by averaging the largest diameter of the two circles (which appear oblong if they are not exactly perpendicular to the x-ray beam). The normal vessel was then measured with this standard.

A 6F sheath was placed in the femoral artery. Heparin (5000 U IV) was administered at the start of the procedure, and an activated clotting time was measured to ensure that it was greater than 200 seconds. A 6F guiding catheter (Cordis Envoy, Cordis Endovascular System) was navigated over a 0.035-in guide wire into the cervical portion of the vertebral or internal

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From the Oregon Stroke Center, Departments of Neurology (W.M.C., M.L.W., B.M.C.), Neurosurgery, Radiology, and the Dotter Interventional Institute (W.M.C., S.L.B., G.N.), Oregon Health Sciences University, Portland; and the Neurology (B.M.C.) and Neurosurgery (O.R.O'B.) Services, Portland Veterans Administration Medical Center, Oregon.

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Correspondence to Dr Wayne M. Clark, Department of Neurology L104, Oregon Health Sciences University, 3181 SW Sam Jackson Park Rd, Portland, OR 97201. E-mail clark w@ohsu.edu.

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Results of Percutaneous Transluminal Angioplasty for Intracranial Stenosis

Case	Vessel	Pre Symptoms	Pre Angio, %	Acute Post, %	Best Post, %	6 Mo Post, %	Complications	6 Mo Symptoms	Long-term, mo
1	LVA: V3	2, 3	78	42	42	...		N	54
2	RVA: V4	2, 4	73	72	32	32	Dissection	N	16
	BA: ↓SCA		72	46	46	50			
3	LVA: V3	3, 4	83	27	27	...	Diplopia	N	27
4	LVA: V4	3, 4	73	65	65	70		N	32
5	LVA: V4	2, 5	64	38	0	0	Stroke	NA	9
	BA: ↑AICA		65	62	0	0			
6	LICA: Cav	1, 2	70	50	50	53		N	12
	LVA: V3		86	46	44	44			
	RVA: V3		70	47	30	30			
7	RVA: V4	3, 5	60	31	31	...		N	12
8	RICA: Pet	2, 5	66	51	51	52	Dissection	N	27
9	BA: ↑AICA	3, 5	74	50	43	43		N	20
10	LVA: V3	3, 6	79	48	48	50	Headache	N	19; died MI
11	LICA: SC	1	67	25	25	...		N	17
12	RVA: ↓AICA	3, 4	65	46	46	...		N	8
13	BA: ↑AICA	2, 4	81	95	95	...		N	9
14	LICA: SC	1, 2	73	39	25	25	Dissection	N	8
15	LMCA: M1	1, 2	68	100	100	...	Stroke	NA	50
16	BA: ↑AICA	3, 6	82	59	59	...		N	51; TIA, 7
	LVA: V3		55	45	45	...			
17	LMCA: M1	1, 2	81	44	44	...		N	8

Pre Symptoms indicates predominant symptoms patient was having prior to angioplasty (1, aphasia; 2, hemiparesis; 3, ataxia; 4, diplopia; 5, dysarthria; and 6, cortical blindness); Pre Angio, stenosis on baseline angiogram (represents the highest stenosis seen on either anteroposterior or lateral view); Acute Post, residual stenosis immediately after percutaneous transluminal angiography (identical view as baseline); Best Post, best angiographic stenosis (least amount of residual stenosis on any posttreatment angiogram, same view as baseline); Complications, events within 24 hours of procedure; 6 Mo Symptoms, symptom (stroke or transient ischemic attacks [TIA]) reoccurrence within 6 months; Long-term, total duration of follow-up per case (patient is asymptomatic unless indicated); LVA, left vertebral artery; RVA, right vertebral artery; V3, just proximal to posterior inferior cerebellar artery; V4, just distal to posterior inferior cerebellar artery; BA, basilar artery; ↓SCA, just proximal to superior cerebellar artery; ↑AICA, just distal to anterior inferior cerebellar artery; ↓AICA, just proximal to anterior inferior cerebellar artery; LICA, left internal carotid artery; RICA, right internal carotid artery; Cav, cavernous; Pet, petrous; SC, supraclinoid; LMCA, left middle cerebral artery; M1, M1 segment; N, none; NA, not applicable; and MI, myocardial infarction.

carotid artery. A balloon dilation catheter (Stealth, Target Therapeutics Inc) was navigated over a 0.014-in guide wire (Dasher-14, Target Therapeutics Inc) across the lesion. The choice of inflated diameter of the balloon angioplasty catheter was determined by the diameter of the normal adjacent vessel. The inflated balloon diameter did not exceed the diameter of the normal vessel. The length of the balloon was determined by the length of the stenotic lesion. The length of the balloon exceeded the length of the stenosis by at least 5 mm. The guide wire was removed and replaced with a Stealth valve wire with a 2-cm tip. The balloon was inflated to 7 atm for 10 seconds using a 75% mixture of Isovue-300 and saline. With the balloon deflated but still in position, a contrast injection was made through the guiding catheter to assess response to the angioplasty. If the vessel was still stenosed and if there was slow blood flow around the balloon, the angioplasty was repeated once or twice. If tandem lesions were present, the more severely stenotic lesion was treated first. A repeat angiogram was then performed to assess vessel stenosis and to look for evidence of distal embolization.

Heparin was continued after the procedure, with the dose adjusted to maintain the partial thromboplastin time at 45 to 60 seconds. The femoral artery sheath was removed with the patient still receiving heparin. Manual femoral artery compression for 15 minutes generally was sufficient to obtain hemostasis.

All patients were monitored in the neurological critical care unit for at least 24 hours. A detailed neurological examination was performed immediately after angioplasty and then daily during hospitalization. In most cases, the patients were discharged within 48 hours. Patients were treated with Coumadin (international normalized ratio, 2.0 to 2.5) for at least 3 months. All cases were clinically evaluated at 3 and 6 months. A repeated angiogram was performed at approximately 6

months in twelve cases to evaluate restenosis and assess the need for continued anticoagulation.

All angiograms were later read by an independent neuro-radiologist not involved in the angioplasty procedure who was blinded to the angioplasty status of each film. Stenosis was measured by the criteria of the North American Symptomatic Carotid Endarterectomy Trial (NASCET) (comparing diameter at the site of greatest narrowing to diameter of normal artery distal to lesion).¹⁶ Overall treatment efficacy was determined by corrected paired *t* tests.

Results

The clinical and angiographic features of all patients entered into this study are given in the Table. There were 2 women and 15 men aged 57 to 79 years. All patients had recurrent neurological symptoms referable to the stenotic vessel before treatment. The initial head CT was negative in patients with transient ischemic attacks and showed one or more ischemic strokes in patients with fixed deficits. Four patients had multiple vessels treated. Angioplasty was performed in these additional vessels if it was felt that they were likely contributing to the patients' recurrent symptoms. PCTA was performed on 22 vessels, including 16 vertebral or basilar and 6 internal carotid or middle cerebral arteries (MCA). The baseline angiogram showed a preangioplasty stenosis of $72 \pm 8\%$ (mean \pm SD). Although at least one vessel in every case was initially graded as at least 70%, the measured stenosis was later reduced in some cases by the independent neuro-radiologist (see Table).

There was a significant ($P < .0001$) reduction in stenosis to $51 \pm 18\%$ seen on the immediate post-PCTA

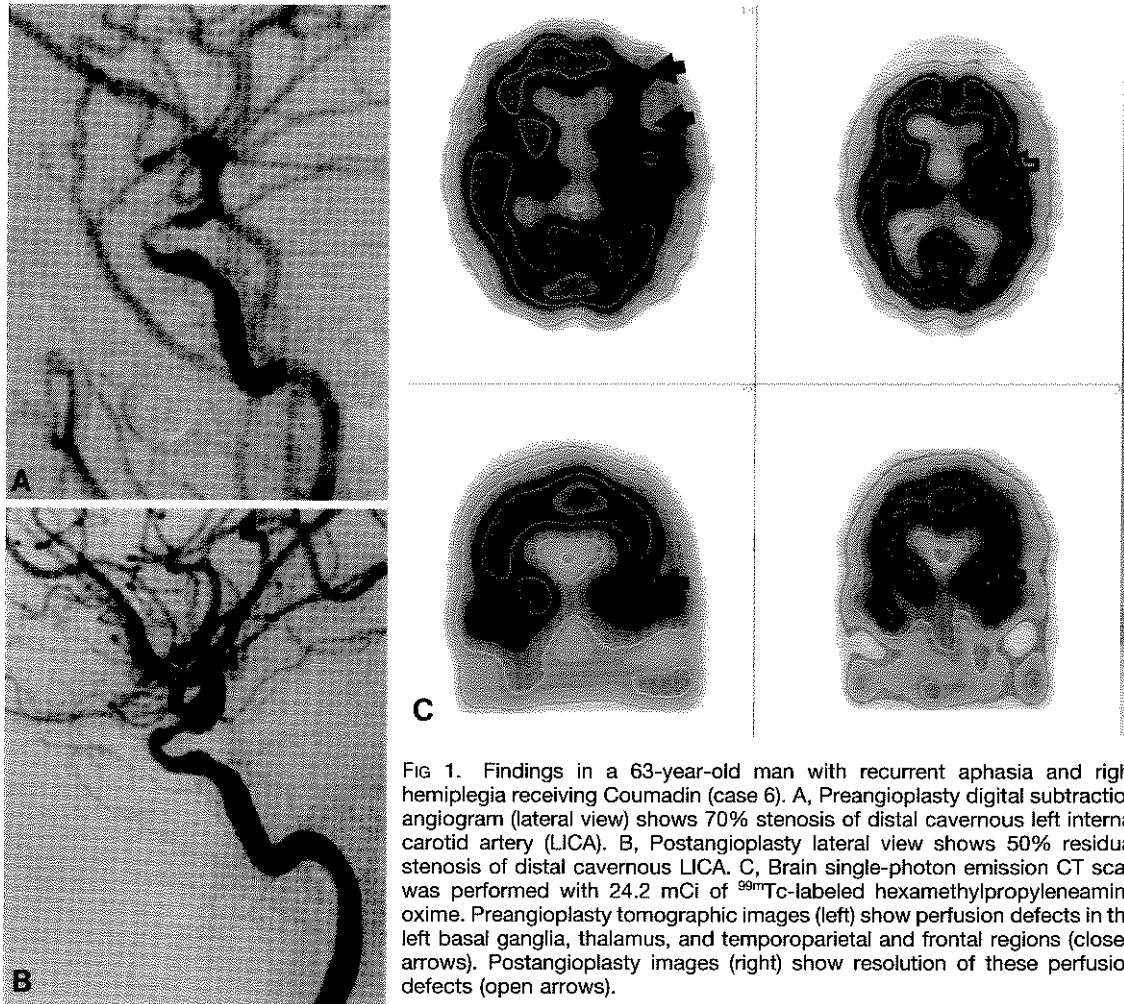


FIG 1. Findings in a 63-year-old man with recurrent aphasia and right hemiplegia receiving Coumadin (case 6). A, Preangioplasty digital subtraction angiogram (lateral view) shows 70% stenosis of distal cavernous left internal carotid artery (LICA). B, Postangioplasty lateral view shows 50% residual stenosis of distal cavernous LICA. C, Brain single-photon emission CT scan was performed with 24.2 mCi of ^{99m}Tc -labeled hexamethylpropyleneamine oxime. Preangioplasty tomographic images (left) show perfusion defects in the left basal ganglia, thalamus, and temporoparietal and frontal regions (closed arrows). Postangioplasty images (right) show resolution of these perfusion defects (open arrows).

angiogram. However, three of the vessels showed evidence of intimal injury (small intimal dissections or thrombosis) on the acute-stage films, making it difficult to accurately estimate the residual stenosis. These cases all showed resolution of these processes on subsequent angiograms. The subsequent angiogram was substituted for the acute-stage angiogram in these cases to determine an overall "best angiographic stenosis" after angioplasty of $43\pm 23\%$. Eight of the patients (12 vessels) underwent a repeated angiogram at 6 months to evaluate restenosis. In these cases, the degree of stenosis actually improved from an acute post-PCTA residual stenosis of $51\pm 10\%$ to $37\pm 21\%$ at approximately 6 months ($P=.05$). We did not obtain repeated angiograms for the other 9 patients because of lack of insurance authorization or patient reluctance. In two cases (nos. 2 and 17), we initially attempted opening the stenosis with intra-arterial urokinase. No improvement in stenosis was seen, and PCTA was performed the following day. Illustrative cases are shown in Figs 1 and 2.

Pre- and post-PCTA brain single-photon emission CT (SPECT) scans were performed with 24.2 mCi of ^{99m}Tc -labeled hexamethylpropyleneamine oxime in three patients (nos. 8, 12, and 18; Table). Scans obtained after PCTA showed at least a 20% improvement in the pre-PCTA perfusion defects in all three patients. A representative example is shown in Fig 1C.

There were no cases of vessel rupture or death. There were two strokes associated with PCTA for a major complication rate of 9.1% per vessel and a complication rate of 12% per case. One patient had a large MCA stroke during PCTA due to an intimal dissection, causing a complete occlusion of the MCA. This patient did not respond to attempts of repeat angioplasty or thrombolysis. The second patient had a pontine stroke presumably due to a penetrating branch artery occlusion during PCTA of the basilar artery. Additional mild complications included headache in one patient, transient diplopia in one patient, and asymptomatic intimal dissection in three patients (Table).

Results of follow-up are shown in the Table. Excluding the two patients with PCTA-related strokes, all patients had resolution of symptoms with no further neurological events at 3 and 6 months. In addition, during 22 ± 16 months of long-term follow-up (range, 8 to 54 months), no strokes occurred in the territory of the PCTA-treated artery.

Discussion

In this study, we found that PCTA could successfully treat intracranial atherosclerotic stenosis with a relatively low complication rate. Overall, we observed a 29% absolute improvement in stenosis. In the coronary circulation, a "successful angioplasty" is defined as at least

