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BACKGROUND AND PURPOSE: **Acute vertebrobasilar ischemic stroke is often associated with high morbidity and mortality with limited therapeutic options. Endovascular treatment with thrombolysis has offered some hope for affected patients; however, overall outcomes have been less than satisfactory. In this report, we present the results of our approach in six consecutive cases of acute vertebrobasilar ischemic stroke by combined proximal vessel stent placement and thrombolysis.**

METHODS: **Six consecutive cases were retrospectively reviewed for the clinical outcome of patients presenting to our institution with acute posterior circulation stroke who underwent cerebral revascularization including proximal arterial stent placement by using balloon-expandable coronary stents and intraarterial thrombolysis. All of these patients were initially evaluated by stroke team neurologists and imaged with MR, including diffusion-weighted imaging documenting acute posterior circulation stroke. MR angiography of the circle of Willis was also obtained. Short-term follow-up was conducted to assess National Institutes of Health stroke scores (NIHSS) and modified Rankin scores.**

RESULTS: **In these six cases, a combined approach of proximal arterial stent placement (five cases of vertebral artery origin and one case of carotid and subclavian stent placement plus vertebral artery revascularization) and thrombolysis was performed at variable times after stroke onset (range, 30 hours to 5 days). Four of the six patients had good basilar artery recanalization (Thrombolysis in Myocardial Infarction [TIMI] grade 0–1 before tissue plasminogen activator thrombolysis and TIMI grade 2 after procedure). Four of six patients had excellent immediate recovery and were discharged to an acute rehabilitation unit or their homes with improved neurologic symptoms and functional status. Two patients died: one patient presented with coma at outset with an NIHSS of 38, and the other patient probably had reocclusion of the basilar artery within 24 hours despite initial postprocedural improvement.**

CONCLUSION: **We demonstrate that, in the setting of acute stroke, stent placement in combination with revascularization and thrombolysis is practical and allows quick access to a clot and simultaneously increases perfusion through collaterals during the thrombolytic process. In particular, basilar thrombolysis may be facilitated by proximal vertebral stent placement as concomitant atheromatous vertebrobasilar stenosis is common.**

Patients with vertebrobasilar occlusive disease are at risk for stroke that often leads to fatality or disabling neurologic symptoms (1). These patients with symptomatic vertebrobasilar stenosis are usually initially treated medically with antiplatelet agents and systemic anticoagulation. The risk of stroke, however, remains high. When medical therapy fails, there are limited therapeutic options. Without treatment, the prognosis of vertebrobasilar ischemic stroke is grim. Acute occlusion of the basilar artery carries an estimated 80–90% mortality rate (2–5). Endovascular treatment with thrombolysis, however, offers a viable therapeutic option and is now a widely accepted practice in this setting (4–8). The morbidity and mortality of treatment is related to the location and extent of the clot and presence of collateral flow. The presence of atheromatous stenosis or occlusion, complete basi-

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lar thrombosis, and absence of collaterals are predictive of a poor outcome (9, 10).

In patients presenting with acute basilar artery thrombosis, there is often concomitant generalized atherosclerotic disease most commonly affecting the vertebral artery origin (11, 12). In the acute setting, these lesions can be aggressively treated by revascularization and thrombolysis via an endovascular approach. Theoretically, primary stent placement with or without balloon angioplasty in the stenotic proximal artery (most frequently vertebral artery origin) allows collateral flow to the basilar system, thereby buying time through enhanced cerebral circulation during thrombolysis and providing improved access for intraarterial administration of tissue plasminogen activator (tPA). This approach is facilitated by the availability of flexible coronary stents, which have been demonstrated to be effective and safe in several cases involving endovascular treatment of stenotic proximal (1, 13–18) and intracranial (19, 20) vertebral arteries as well as the basilar artery (21).

We report our initial and short-term follow-up findings in six consecutive cases of vertebrobasilar thrombolysis with acute proximal arterial stent placement.

Methods

Between May 2000 and April 2001, six consecutive male patients (average age, 64.3 years; age range, 50–80 years) with acute posterior circulation ischemic stroke underwent cerebral revascularization with a combination of proximal arterial stent placement and tPA thrombolysis. Some of these patients presented directly to our institution shortly after their ictus, and a number of the patients were transferred from other hospitals after failing anticoagulation with a stuttering course. Their clinical presentation and imaging findings are summarized in Table 1. The time between the onset of neurologic symptoms (defined as the time preceding which the patient was in his or her usual state of health) and endovascular intervention varies widely, ranging from 30 hours to 5 days (Table 2). These patients frequently presented with initial ischemic symptoms followed by acute deterioration.

All of these patients were examined with MR imaging, including diffusion-weighted imaging documenting acute posterior circulation stroke. All had diffusion abnormality, and three (patients 2, 5, and 6) had large diffusion-perfusion mismatches. Perfusion-weighted imaging findings were not available for patient 4 at the time of ictus. MR angiography of the circle of Willis was also obtained in all patients. All of these patients demonstrating clinical and radiologic evidence of acute posterior circulation ischemia, with or without MR angiography (MRA) confirmation of basilar occlusion, further underwent cerebral angiography with the intention to treat. In all of these patients, diagnostic angiography showed severe concomitant atherosclerotic disease affecting the vertebral artery origin—and at times also subclavian arteries—significantly limiting the access to the basilar artery for endovascular treatment as well as decreasing collateral flow.

Interventions

A transfemoral approach was used in each case. All patients were intubated for airway protection and monitored by our neuroscience critical care unit (NCCU) stroke team. After arterial puncture and insertion of a 6F vascular sheath, systemic anticoagulation was maintained with a 5000-U bolus followed by 1000 U/h intravenous heparin. In five patients the vertebral origin was treated, and in one patient the subclavian artery was treated by primary stent placement. The origin of the vertebral artery (or site of diseased vessel) was approached with a 6F guiding catheter (Envoy MPC; Cordis, Miami, FL). A balloon-expandable "coronary" stent (typically 3–4 mm in diameter by 1.5–3.0 mm in length) was advanced through the stenosis over a 0.014′ × 300-cm-long, soft-tipped guidewire (Luge; Boston Scientific Corporation, Natick, MA). A variety of stents was used, including the Tetra/Duet/Penta/Tristar monorail stent system (Guidant, Temecula, CA), BX Velocity (Cordis), and Corinthian (Cordis). Several stents used in this series reflected the rapid development of commercially available stents as well as the evolution of our techniques.

The use of stents and tPA in this series was not approved by the Food and Drug Administration, but the procedure was reviewed and approved by the Johns Hopkins Medicine Institutional Review Boards and performed with informed consent.

A different strategy was used in patient 3, who had a right common carotid string sign (99% stenosis), a left common carotid occlusion, a right vertebral occlusion, and a 90% left vertebral stenosis, in addition to the basilar artery thrombosis. Stents were first placed in the right common carotid artery and left subclavian artery. A 6F Envoy guiding catheter was placed in the right common carotid artery, and the stenosis was predilated with a 2.4 mm × 2 cm long balloon (Ranger, Boston Scientific). A 2.8-cm-long × 4-mm-diameter stent (Tristar, Guidant) was then passed through the stenosis and maximally inflated to 4.4 mm. A 6 × 20-mm balloon-expandable stent (Corinthian, Cordis) was deployed in the 80% left subclavian stenosis. Angioplasty was performed in the left vertebral artery origin by using a 3.5-mm balloon (Ranger, Boston Scientific) passed over a 300-cm-long, 0.014-inch guidewire (Luge, Boston Scientific), but marked proximal sinuosity did not allow the placement of a stent. A microcatheter (Turbo Tracker 18, Target Therapeutics, Fremont, CA) was finally advanced coaxially over the 300-cm wire, and thrombolysis was performed from the C4–C5 level.

In all the other cases, after vertebral artery stent placement, a microcatheter was passed over a 300-cm, 0.014-inch guidewire kept across the stent for tPA infusion. The basilar artery thrombus was reached in all these cases, and the microwire was used to disrupt the clot mechanically and thus reduce the tPA dose required. The total dose of tPA was kept at 20 mg or less, with the exception of patient 3, who received 24 mg.

Postprocedure Care

The patients were maintained on heparin for 24 hours and then began to receive coumadin, at times in combination with clopidogrel, for 6 weeks, depending on the residual clot burden. All patients were monitored in the NCCU during the immediate 24–48 hours following the procedure. They were subsequently transferred to a regular ward and then to an acute rehabilitation unit. Patients returned at 2 weeks after discharge from the rehabilitation unit for a follow-up visit with the neurologist at the stroke clinic, and subsequently every 6–12 weeks. If the patient resided in another state, follow-up interviews were conducted by phone with the patient and local physician. The patient's function in daily living and residual neurologic deficits were specifically assessed.

Results

In each of these patients, we deployed a balloon-expandable stent at the stenotic segment that provided access for subsequent thrombolytic therapy. This maneuver required between 10 and 15 minutes of additional procedure time.

Seven lesions were treated with primary stent placement, and one by angioplasty alone. In five pa-

TABLE 1: Summary of Patients with Acute Vertebrobasilar Ischemic Stroke: Presentation, MR Imaging and Angiographic Findings, and Neurointervention

Patient no./ Age (y)/Sex	Presenting Symptoms	DWI and/or PWI Findings	DW-PW Mismatch	MRA Findings	Angiographic Findings	Treatment	Pre-TIMI	Post-TIMI
1/50/M	Wallenberg syndrome followed 6 days later by right hemiparesis, slurred speech, and diplopia	Right PICA infarct, new left pontine infarct 6 days later	7.1:22	No right VA or BA flow, possible left VA stenosis	Occluded mid-basilar artery, nearly occluded right VA, pre-occlusive left VA origin	Tristar coronary stent at left VA origin, 14 mg tPA thrombolysis of BA	1	2
2/64/M	Transient diplopia followed by acute onset of slurred speech and transient right leg weakness	Small left pontine lesion, bright on DWI	0.8:131.4	Right VA and mid-basilar stenosis	Severely stenotic non-dominant right VA perfusing the right PICA, pre-occlusive stenosis of left VA origin, BA occlusion	Tristar coronary stent at the left VA origin, 9 mg tPA thrombolysis of basilar at the level of PICA	0	2
3/80/M	Dysarthria, weakness, dizziness; next a.m., inability to move out of bed, vomiting and unresponsive	Acute CVA: right pons, bilateral occipital lobes, rights thalamus	12.1:49.1	Stenosis of left cavernous ICA, occluded left ICA, decreased flow in left PCA	90 95% right ICA stenosis, left ICA occlusion, severe stenosis in mid left SCA and left VA origin, basilar artery apex thrombosis, occluded right VA origin	4 mm × 3.8 cm Guidant Tristar stent in right CCA and 6 mm × 2 cm wall stent in left SCA, 24 mg tPA thrombolysis of BA	0	2
4/60/M	Acute stroke with left hemiparesis followed by coma 3 days later	Right thalamo-capsular and right pons, upper midbrain infarct	PWI not performed	Basilar thrombosis	>90% stenosis, right VA origin; occluded left VA; occlusion of BA at junction of proximal and mid-third; left subclavian steal; 70–80% stenosis, proximal left SCA; >90% stenosis, diffuse and severe atheromatous disease	Tetra coronary stent at right VA origin, 19.5 mg tPA, thrombolysis of BA with minimal dissolution of clot	0	0
5/72/M	Diplopia and ataxia, 36-h duration, followed by coma	Small left pontine lesion	0.4:174.1	Distal basilar artery thrombosis	80% stenosis, right PICA origin; 90%, right AICA; distal BA occlusion; PCA not seen; 40–50% stenosis, distal left CCA	AVE stent, right VA origin; 9 mg tPA and angioplasty of BA	0	1
6/60/M	Headache, diplopia, vertigo, ataxia, acutely lethargic	Bilateral AICA distribution, small pons and midbrain, left occipital and corpus callosum, and left thalamus infarcts	5.6:166.5	No flow in BA	Left VA occlusion, high-grade stenosis with slow flow in right VA, no perfusion of BA	Vertebral origin stent, 9 mg tPA, proximal BA thrombolysis	0	2

Note.—DWI indicates diffusion-weighted imaging; PWI, perfusion-weighted imaging; DW-PW mismatch, diffusion-perfusion mismatch; MRA, MR angiography; TIMI, thrombolysis in myocardial infarction; Pre-TIMI, TIMI grade before treatment; post-TIMI, TIMI grade after treatment; PICA, posterior inferior cerebellar artery; VA, vertebral artery; BA, basilar artery; tPA, tissue plasminogen activator; CVA, cerebrovascular accident; ICA, internal carotid artery; PCA, posterior cerebral artery; SCA, superior cerebellar artery; CCA, common carotid artery; AICA, anterior inferior cerebellar artery.

TABLE 2: Clinical Outcome

Patient	Time Elapsed, Symptom Onset to Treatment (h)	Clinical Outcome	NIHSS (Admission)	Modified Rankin Score (Discharge)	NIHSS (F/U)	Rankin (F/U)	Time Elapsed, Treatment to Follow-up (d)	Follow-up Imaging Findings
1	134	Gradual and good recovery, d/c to acute rehabilitation	25	4	4	2	189	MR and MRA (at 32 mo): patent BA
2	30	Improved extremity strength, dysarthria, and hypophonia	6	4	4	3	66	Angiography (at 19 mo): BA stenosis with collaterals, patent left VA origin stent
3	57	Gradual and steady recovery, transferred to acute rehabilitation after 1 week	8	4	5	4	21	MR and MRA (at 1 wk): patent VB system, occluded left ICA
4	60	Brain stem and subarachnoid hemorrhage, condition deteriorated, patient expired	38	N/A	N/A	N/A	N/A	N/A
5	86	Immediate improvement followed by rapid deterioration, minimal brain stem reflexes the next morning, care withdrawn, patient died	3	N/A	N/A	N/A	N/A	N/A
6	99	Bilateral cerebellar infarct with hemorrhagic conversion, fair and gradual recovery	8	3	3	2	84	MR and MRA (2 wk): atherosclerotic irregularity with stenosis at junction of right VA and BA

Note:—NIHSS indicates National Institutes of Health Stroke Score; F/U, follow-up; MRA, MR angiography; BA, basilar artery; VA, vertebral artery; VB, vertebrobasilar; ICA, internal carotid artery.

tients, the lesion treated was located at the origin of the vertebral artery (Fig 1). One patient (patient 3) with basilar artery thrombosis underwent primary stent placement of the proximal right common carotid and left subclavian arteries, in addition to left vertebral angioplasty and basilar artery thrombolysis. All six of these patients had basilar artery thrombosis and underwent recombinant tPA thrombolysis following angioplasty and stent placement.

In four of six patients, immediate improvement of neurologic symptoms occurred after endovascular treatment. They continued to improve slowly but steadily throughout their hospitalization and were able to proceed to physical therapy, occupational therapy, and speech therapy in the acute rehabilitation unit with excellent functional recovery (Table 2). Of note, in patients 1 and 6 the recovery course was marked by hemorrhagic transformation of preprocedural cerebellar infarct, which did not alter the favorable clinical progress.

Patient 3 had excellent improvement of neurologic symptoms after extensive neurointervention, including multiple stent placement, angioplasty, and thrombolysis. He was transferred to an acute rehabilitation unit after a month of hospitalization. He continued to have steady improvement of disability, was able to ambulate with moderate assistance in the beginning, and progressed to requiring minimal assistance at the end of 3 weeks of rehabilitation. Six months from his initial stroke, however, he was readmitted after being found collapsed and had evolving massive right middle cerebral artery infarct. He died within a few days in the hospital.

In patient 4, who was comatose at presentation and had a preprocedural NIHSS of 38, a critical proximal vertebral artery stenosis was treated by stent placement without difficulty, yielding an excellent angiographic result and allowing easy passage of a microcatheter for basilar artery thrombolysis at the junction of the proximal and middle thirds. Basilar artery thrombolysis was difficult, however, most probably because of the age and extent of the clot and lack of collaterals. Little progress was made with the size of the clot even after sequential delivery of a total of 19.5 mg tPA (Thrombolysis in Myocardial Infarction [TIMI] scale 0, unchanged). After 19.5 mg of tPA infusion, a control angiogram demonstrated contrast material in the fourth ventricle—interpreted as a potential extravasation even though several subsequent control angiograms showed no evidence of vessel rup-

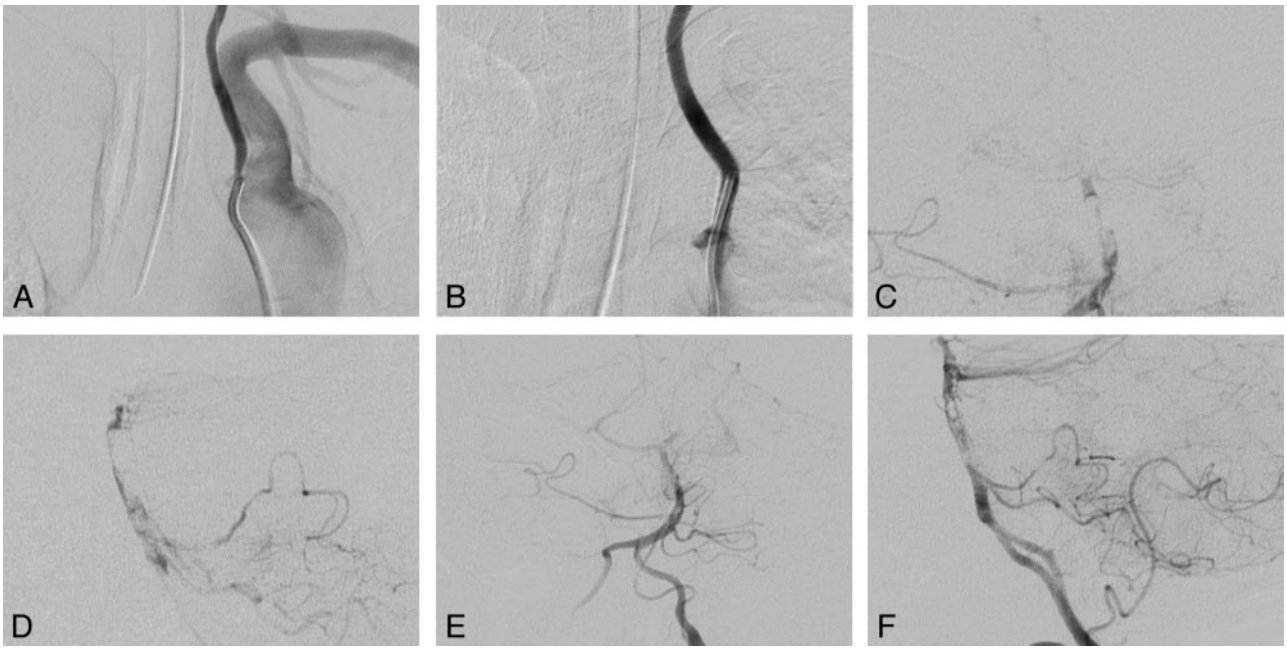

FIG 1. A 50-year-old man who presented with right medullary syndrome, followed by right hemiparesis, slurred speech, and diplopia 6 days later.

A, Left vertebral angiogram, anteroposterior view, showing a high-grade stenosis of the left vertebral artery origin from the aortic arch.

B, Left vertebral angiogram after placement of an ACS multilink Tristar coronary stent (3 mm in diameter x 30 mm length).

Anteroposterior (*C*) and lateral (*D*) views of selective basilar angiogram, showing multiple clots and distal basilar occlusion.

Anteroposterior (*E*) and lateral (*F*) views of left vertebral angiogram, showing reperfusion of the posterior circulation after infusion of 14 mg of tPA.

ture or leak. The procedure was terminated at this time. The patient's clinical condition deteriorated rapidly, and he died 2 days later.

Patient 5 had very dramatic improvement of neurologic symptoms after the procedure, and the postprocedural angiography showed patency of the proximal two-thirds of the basilar artery, but the basilar tip demonstrated only partial recanalization (TIMI 1 flow). The next morning, his condition deteriorated rapidly, with worsening neurologic symptoms and minimal brain stem reflexes that the NCCU physician attributed to reocclusion of his basilar artery. He died within 24 hours.

Overall, three of the six patients improved by two TIMI grades (prethrombolysis TIMI scale of 0 and postprocedural TIMI scale of 2), two patients improved by one grade (from TIMI 1 to 2 and from TIMI 0 to 1), and one had no change in TIMI scale (0 before and after thrombolysis). Follow-up imaging demonstrated basilar artery patency up to 32 months in patient 1, although frequently atherosclerotic involvement persisted and progressed in some patients (Table 2).

Discussion

Thrombolysis is gaining acceptance as a therapeutic practice in selected patients with acute basilar artery occlusion, as there is increased awareness of patients presenting with vertebrobasilar ischemia, aided by advanced MR imaging for prompt and accurate diagnosis and successful thrombolytic therapy in anterior-circulation stroke. On the basis of several uncontrolled series of intraarterial thrombolysis in basilar artery occlusion during the past 10 years, recanalization of the basilar artery occurs in 40–83% of cases after thrombolysis. Patients reported to have good outcomes after thrombolysis have been estimated to range from 19–56% with a mean of 29.7% (22). Such a wide range of outcomes may be partly related to technical expertise and partly related to variable patient condition at presentation. Nevertheless, the overall rate of good clinical outcome is far from satisfactory, although it is slightly better compared with no treatment because of the grim prognosis related to the natural history of the disease.

Several retrospective studies have identified the best predictor of good outcome in basilar artery thrombolysis to be distal clot location (9, 10). This is most probably related to the perception that distal clot location is more likely the result of an embolic event in an otherwise healthy basilar artery, whereas mid or proximal basilar artery occlusion is most frequently associated with underlying arterial disease. The occluded basilar artery may be difficult to recanalize; there is usually residual stenosis even after aggressive thrombolysis, and the reocclusion rate following successful thrombolysis remains as high as 13–30% (5, 22). All of our patients had basilar artery occlusion in a background of diffuse atherosclerotic disease and therefore were in the population with poorest prognosis. Several groups have attempted a combination of thrombolysis with angioplasty or stent placement at the site of vertebrobasilar occlusion with

some reported success (19, 20); however, the reported risk associated with these maneuvers remains high.

To improve the outcome of basilar thrombolytic therapy in this patient population with diffuse atherosclerosis, we believe that it is important to address the associated atheromatous lesions for at least three reasons. First, even with successful recanalization of basilar occlusion, the result is far less permanent if there is severe atherosclerosis in the remaining vessel that serves as the nidus for additional or recurrent sites of thrombotic stenosis and occlusion. Second, treating a proximal stenotic lesion allows better perfusion to the brain stem through collateral flows during and after the procedure. Third, opening the proximal stenosis directly provides a ready access to the site of basilar occlusion for tPA thrombolysis.

In a series of seven patients with acute or chronic vertebrobasilar insufficiency due to severe stenoses at the origin of the vertebral artery, Piotin et al (17) reported safe and effective endovascular treatment by combining percutaneous transluminal angioplasty and primary stent placement. Several other groups (1, 13–16, 18) have also successfully stented the proximal vertebral artery and vertebral origins in patients symptomatic of posterior circulation ischemia or insufficiency. There was no procedure-related complication. At long-term follow-up, Chastain et al (16) reported that 4% of their patients had recurrent symptoms at 25 months ± 10, and Malek et al (1) reported that 57% of patients were symptom-free at 21 months ± 4.

Primary stent placement reduces the risk of complications such as arterial spasm, intimal dissection, and distal embolism that can be associated with angioplasty without stent placement (17). Coronary stents are well suited in the proximal vertebral artery, because the vertebral caliber is similar to that of coronary arteries (ie, 3–5 mm). In addition, the trackability of a coronary stent allows for the ease of navigation through tortuous vessels. It is also noted that the stent mesh probably prevents elastic recoil and early restenosis after percutaneous angioplasty, allowing for a more prolonged patency (17). Finally, the procedure requires no more than approximately 10–15 minutes of additional procedure time but offers tremendous advantages of improved perfusion and open access, which, we believe permit a longer therapeutic window and more efficient delivery of thrombolytic agents. This is particularly valuable in the setting of acute vertebrobasilar thrombosis treatment.

Herein, we report primary stent placement in the proximal vessel combined with basilar artery thrombolysis in six patients who presented acutely with posterior circulation ischemia as a result of vertebrobasilar thrombosis. Basilar artery reperfusion was achieved in all but one of the patients following tPA thrombolytic therapies. In most of these patients, basilar recanalization was purposely left incomplete at the end of the procedure (TIMI grade 2), and the remaining clot was cleared by the residual administered tPA, intravenous heparin, and antiplatelet agent. This strategy was chosen to minimize hemorrhagic complications. Despite partial basilar recanalization and imperfect angiographic results, there was excellent clinical recovery in our patients with progressive improvement of neurologic symptoms at immediate- and short-term follow-up, as confirmed by MRA. This is consistent with von Kummer and Hacke's (23) experience with fibrinolytic therapy in the anterior circulation; a good clinical outcome correlated with not only early, but also delayed, recanalization. At times our decision was for the patient to receive intravenous heparin following angioplasty and stent placement without tPA. If total vessel clearance was achieved, however, no heparin was administered following the procedure. Cross et al (24) recently reported their experience with basilar artery thrombolysis with tPA and indicated a high (three of four cases, or 75%) rate of hemorrhagic complications. Their approach, however, used intraarterial doses of tPA ranging from 20 to 50 mg, significantly higher compared with those administered in our institution (usually up to 20 mg). Our result indicated adequate vertebrobasilar reperfusion with either complete or partial recanalization even out to 5 days in five of six patients. We attribute this success to combined stent placement and thrombolysis. The major advantage of this simple technique is likely an augmented perfusion through improved collateral flows, thus rendering the thrombolysis procedure safer and more effective while requiring a smaller dose of tPA.

Stent placement can occasionally be difficult. An example is demonstrated in patient 3, in whom the proximal vertebral artery at the origin followed such a tortuous course that even a flexible coronary stent could not be deployed. We performed angioplasty alone in the proximal vertebral artery instead. Because the patient also had severe, diffuse atheromatous disease with complete occlusion of the right vertebral origin and left common carotid artery as well as lesions affecting the left subclavian artery (70% occlusion) and right common carotid artery (95% occlusion), these lesions were treated with stent placement before lysis of the basilar apex thrombus. The rationale for this strategy is the same as that for stent placement in the vertebral artery origin: improved perfusion via collateral flows through both anterior and posterior circulations after stent placement. The proximal diseased vessels may provide augmented cerebral perfusion during the basilar thrombolytic process and consequently a better clinical outcome. This patient's sudden demise after a massive anterior circulation infarct 6 months following the initial stroke was most likely attributed to his extensive underlying progressive atherosclerotic vasculopathy. His initial response to therapy and absence of recurrent posterior circulation infarct in the interval were nevertheless an encouraging outcome.

Patient selection remains important. To date, there is no specific guideline as to who should receive aggressive revascularization in the setting of acute posterior circulation ischemic stroke. In one series, thrombolysis has been successfully delivered in treat-

ing posterior circulation ischemia between 6 hours and 79 hours of stroke onset (25), and Becker et al (5) found no correlation between the ability to recanalize and the time to therapy from last neurologic deterioration. In our series, time since stroke onset was therefore not used as an exclusion criterion, although the long average lapse of 78 hours (30 hours to 5 days) in part reflected referral pattern. For example, some patients are transferred to our institution after they have failed anticoagulation. Often the decision to treat these patients by means of aggressive interventions was not made until after the patient had had rapid clinical deterioration some time following the initial presentation of neurologic symptoms characteristic of vertebrobasilar ischemia. As for the use of imaging as a guide for disease management, diffusion abnormality in the posterior circulation distribution revealed by MR imaging was an important finding to confirm and define the clinically suspected acute infarction. Perfusion-weighted imaging was performed in all but one patient, as was follow-up serially obtained diffusion-perfusion data. A diffusion-perfusion mismatch was not used to determine patient selection in this study but is currently under active investigation for its usefulness as a prognostic indicator and perhaps a guideline for treatment. Finally, MRA of the circle of Willis was obtained in all patients and was helpful for identifying vertebrobasilar abnormality (thrombosis or stenosis) but was not used as the primary guide for therapy.

Conclusion

Our strategy is effective in partially restoring and at minimum preventing further deterioration of the devastating neurologic sequelae from vertebrobasilar stroke under the worst circumstances, with improved safety margin. Many of these patients were able to assume a premorbid independent functional existence after rehabilitation. Considering the overall favorable outcome of the cases presented here, we believe that treatment of underlying atherostenotic lesions by stent placement in the proximal vessel(s) can be performed in conjunction with and before vertebrobasilar thrombolysis. Further study with formal outcome assessment at 90 and 180 days would be important to evaluate intermediate and long-term efficacy of this therapeutic intervention.

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