# Relationship between clot location and outcome after basilar artery thrombolysis.

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# Relationship between Clot Location and Outcome after Basilar Artery Thrombolysis

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**PURPOSE:** To identify factors that predict survival and good neurologic outcome in patients undergoing basilar artery thrombolysis. **METHODS:** Over a 42-month period, 20 of 22 consecutive patients with angiographic proof of basilar artery thrombosis were treated with local intraarterial urokinase. Brain CT scans, neurologic examinations, symptom duration, clot location, and degree of recanalization were analyzed retrospectively. **RESULTS:** Overall survival was 35% at 3 months. Survival in patients with only distal basilar clot was 71%, while survival in patients with proximal or midbasilar clot was only 15%. At 3 months, 29% of patients with distal basilar clot and 15% of patients with proximal or midbasilar clot had good neurologic outcomes (modified Rankin score of 0 to 2 and Barthel index of 95 to 100). Complete recanalization was achieved in 50% of patients; 60% of those survived and 30% had good neurologic outcomes. Of patients with less than complete recanalization, only 10% survived. Neither duration of symptoms before treatment (range, 1 to 79 hours), age (range, 12 to 83 years), nor neurologic status at the initiation of treatment (Glasgow Coma Scale score range, 3 to 15) predicted outcome. Pretreatment CT findings (positive or negative for related ischemic changes) did not predict outcome or hemorrhagic transformation. **CONCLUSION:** The single best predictor of survival after basilar thrombosis and intraarterial thrombolysis was distal clot location. Complete recanalization favored survival. Radiologically evident related infarctions, advanced age, delayed diagnosis, and poor pretreatment neurologic status did not predict poor outcome and therefore should not be considered absolute contraindications for intraarterial thrombolysis in patients with basilar artery thrombosis.

**Index terms:** Arteries, basilar; Thrombolysis; Thrombosis, arterial

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Basilar artery thrombosis is a catastrophic event. Mortality of patients treated with traditional, nonthrombolytic methods is reported to be 80% to 100% when there is angiographic or pathologic confirmation of the diagnosis (1–8). Return to good neurologic status has been exceptional in survivors (9). Early reports of direct intraarterial thrombolysis for basilar thrombosis were promising (10–12), but even with advances in thrombolytic techniques, death remains the most likely outcome (13, 14).

Over a 42-month period, our group used intraarterial thrombolysis to treat 20 patients with basilar thrombosis. Contrary to our expectations, we observed that some patients had poor outcomes (modified Rankin scores of 3 to 6) despite early and complete arterial recanalization, whereas other patients had good outcomes (modified Rankin scores of 0 to 2 and Barthel indexes of 95 to 100) when treated after long delays in diagnosis and when arterial recanalization was incomplete. We retrospectively reviewed our clinical and radiologic data to identify factors that might determine outcome after basilar artery thrombosis and thrombolysis.

## Materials and Methods

From December 1992 through May 1996, 20 of 22 patients with angiographically confirmed basilar artery thrombosis were treated with intraarterial urokinase. One patient had preangiographic computed tomographic (CT)

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findings of extensive pontine infarction, was comatose on arrival from an outside hospital, and died shortly after transfer. Another patient had had a craniotomy 2 days before angiography, which contraindicated treatment.

The timing of referral for treatment varied widely in this group of patients. Some had delays in diagnosis, some were referred from outside hospitals, and some were observed for variable periods on anticoagulant therapy before endovascular intervention was requested.

Each patient underwent noncontrast brain CT immediately before angiography to exclude hemorrhage, mass, or extensive brain stem infarction. We considered recent surgery, recent trauma, or known hemorrhagic disorders to be contraindications for intraarterial thrombolysis. Informed consent for the angiographic and thrombolytic procedures was obtained from each patient or nearest relative. Neurologic examinations were performed at admission or transfer and again immediately before thrombolytic therapy. Any patient with respiratory compromise was intubated. The neurology-neurosurgery intensive care unit (ICU) team or the pediatric ICU team assisted with patient treatment during the procedure, and each patient had continuous electrocardiographic, blood pressure, and oxygen saturation monitoring.

A femoral artery sheath was inserted, connected to a pressurized line, and sutured in place. A diagnostic angiogram was obtained, including an initial arch aortogram and bilateral selective vertebral angiograms. Angiography was performed on one of two digital angiographic units (NeuroStars, Siemens, Erlangen, Germany), except in one early case (patient 2) in which cut film and digital subtraction were used on an older angiographic unit (Philips, Amsterdam, Holland). A nonionic contrast agent (Optiray 320, Mallinckrodt Medical, St Louis, Mo) was used in all cases. Once the diagnosis of basilar thrombosis was confirmed and the plan for thrombolysis agreed upon among the interventional neuroradiologist, neurologist, and family, systemic anticoagulation was begun (if not already done so) with intravenous heparin. Activated clotting times were monitored to maintain one and one-half to two times that of baseline during the procedure. Heparin anticoagulation was maintained after the procedure in the neurology-neurosurgery ICU with partial thromboplastin time monitoring for a variable length of time, except for patients with hemorrhage.

For thrombolysis, a guiding catheter was placed in the vertebral artery with the clearest route of access to the basilar artery. A microcatheter was advanced through the guiding catheter to the site of the thrombus. If possible, the microcatheter was embedded in the surface of the clot. If not, the microcatheter was guided as close to the clot surface as possible. In all cases, urokinase was infused through a single end-hole microcatheter (Tracker or Fastracker 18, Target Therapeutics, Fremont, Calif; Transit or Rapidtransit, Cordis, Miami, Fla). In two patients (cases 9 and 14), a multi–side-hole microcatheter (Microsoftstream, Target Therapeutics) was introduced initially and replaced because that catheter's stiffness prevented advancement to the basilar artery.

Urokinase (Abbott Labs, Abbott Park, Ill), mixed as 5000 U/mL normal saline, was administered at the site of the thrombus by intermittent boluses injected through the microcatheter. The delivery rate was generally 1 mL, or 5000 U, every 20 to 30 seconds. Progress of thrombolysis was monitored by intermittent superselective angiography, usually after each 100 000 to 200 000 U infused. Urokinase infusion was stopped whenever recanalization of the basilar artery was complete, when 250 000 to 500 000 U failed to change the degree of obstruction, or when hemorrhage was suspected clinically. The dose of urokinase was limited to 1 000 000 U/h and the total dose never exceeded 2 000 000 U. Heparin was discontinued and reversed with intravenous protamine sulfate if hemorrhage was strongly suspected or confirmed. Angioplasty of an underlying stenosis at the site of thrombosis was not performed, except in one case of midbasilar stenosis (patient 19) with a microballoon catheter (Fasstealth 3.0 mm, Target Therapeutics).

Bilateral common carotid angiography was performed in most cases, but the procedure usually followed completion of the basilar thrombolysis procedure to avoid delaying treatment. Postprocedural noncontrast brain CT scans were obtained in all cases.

Each patient was transferred to the neurology-neurosurgery ICU or pediatric ICU. Neurologic examinations were performed when the patient arrived in the ICU and serially thereafter during hospitalization. The femoral artery sheath was removed after 1 to 2 days when heparin therapy could be suspended for hemostasis. An additional postprocedural CT scan was obtained if there was clinical deterioration later in the patient's course. If basilar rethrombosis was suspected, a repeat angiogram was obtained. If rethrombosis was confirmed, retreatment with intraarterial urokinase was performed in the manner described above. Five patients had follow-up angiography for suspected rethrombosis. Of those, two patients (cases 7 and 10) had recurrent basilar artery thrombus and were retreated with urokinase and three patients (cases 3, 4, and 8) had no recurrent clot.

A review of patients' records and telephone interviews was approved by the Human Studies Committee. Each patient's chart was examined to determine age, risk factors for stroke, baseline functional level, time of symptom onset, presenting symptoms, neurologic status on admission, and neurologic status immediately before the procedure, immediately after the procedure, and at last evaluation. Glasgow Coma Scale (GCS) scores were computed on the basis of the findings at neurologic examination. A 3-month or longer follow-up modified Rankin score and Barthel index were determined at telephone follow-up by a neurologist. Long-term follow-up neurologic status from any subsequent outpatient or inpatient visit was recorded when available. The time delay between onset of symptoms and start of urokinase infusion was determined, as was the duration of the procedure. The duration of symptoms was defined as the number of hours from the onset of symptoms until the infusion of urokinase was begun. Onset of symptoms was defined as the last time the patient

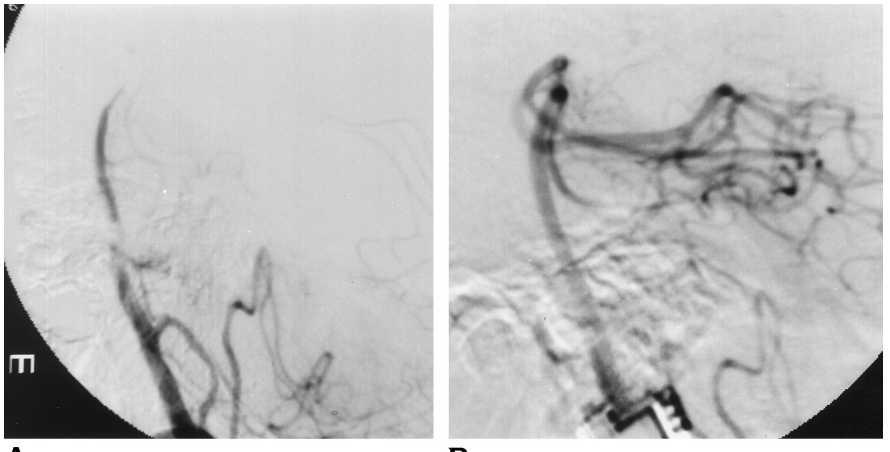


Fig 1. Case 4: Distal basilar thrombosis.

*A*, Lateral projection of the initial vertebral angiogram shows an occlusion of the distal basilar artery (TIMI score of 0). There are collaterals from the posterior inferior cerebellar arteries to the superior cerebellar arteries.

*B*, After 750 000 U intraarterial urokinase, the lateral projection of the final vertebral angiogram shows complete recanalization of the basilar artery (TIMI score of 3) and no underlying arterial disease.

felt normal or was at neurologic baseline. The dose of urokinase, the details of heparin therapy, and any noted clinical deterioration were recorded.

Every patient's angiograms and CT scans were reviewed along with the original radiologic reports and radiologic nursing notes. Angiograms were examined to document the actual site of thrombosis, the presence of any underlying arterial disease, the position of the microcatheter during thrombolysis, and the degree of basilar artery perfusion before and after thrombolysis according to perfusion criteria established by the Thrombolysis in Myocardial Infarction Study Group (TIMI) (15). Distal basilar clots were defined as those that obstructed the basilar tip, the posterior cerebral origins, or the superior cerebellar origins, but did not extend inferiorly to the level of the anterior inferior cerebellar artery (Fig 1). Midbasilar clots were defined as those that obstructed the basilar artery at or about the level of the anterior inferior cerebellar arteries, and proximal clots were defined as those at or below the vertebrobasilar junction. CT scans were examined for the presence of any related ischemic changes or hemorrhage and correlated with clinical findings during the hospital course. All films were available for review with the exception of one pretreatment CT scan; for this patient (case 15), the report alone was used to enter radiologic findings.

Tables were constructed for data analysis. Fisher's Exact Test was used for comparisons of patient subgroups listed in each table.

## Results

Table 1 lists the patients, giving their age, time from initial symptoms to treatment, premorbid functional level, neurologic status at the time of treatment, pretreatment CT findings, location of the basilar artery clot and initial degree of perfusion, dose of urokinase, degree of recanalization achieved, survival, and functional status at 3 months according to a modified Rankin score and Barthel index for each. A modified Rankin score of 0 is normal, a score of 2 reflects slight disability, and a score of 6 indicates death. A Barthel index of 100 indicates complete functional independence.

Table 2 lists the relationship between survival and clot location. Overall survival at 3 months was 35%. When clot was confined to the basilar tip region, survival was 71%. Survival was only 15% ($P = .02$) if clot involved the lower or midbasilar artery.

A "good" neurologic outcome in this study was defined as a modified Rankin score of 0 to 2 and a Barthel index of 95 to 100. Nine patients (45%) had preexisting disability resulting from associated medical conditions, such as heart disease. Only 20% of patients had a good neurologic outcome. There was a trend for better neurologic outcome in patients with distal basilar thrombosis. With distal basilar clot, 29% (two of seven) had a good outcome. With middle or proximal basilar clot, 15% (two of 13) had a good outcome ($P = .59$).

Pretreatment noncontrast brain CT findings did not predict survival or neurologic outcome. Of the 20% of patients with good neurologic outcome, half had negative and half had positive CT findings ($P = 1.00$). Pretreatment CT findings also failed to predict nontechnical posttreatment hemorrhagic complications or events. Contrary to expectations, all hemorrhagic transformations occurred in patients in whom the pretreatment CT scans were negative (three of 11), and no hemorrhage occurred in the nine patients in whom pretreatment CT scans were positive ($P = .22$).

Time between symptom onset and treatment varied considerably, from 1 to 79 hours. All

**TABLE 1: Findings in 20 patients with basilar artery thrombosis**

| Case | Age, y | Rankin (Baseline) Score | Glasgow Coma Scale (Pretreatment) Score | Time from Symptom Onset to Treatment, h | CT Findings (before Treatment) | Site of Clot in Basilar Artery | Initial Perfusion TIMI Score | Urokinase Dose, in million units | Final Perfusion TIMI Score | Modified Rankin Score at 3 Mo | Barthel Index at 3 Mo |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 67 | 0 | 9 | 7 | Negative | Distal | 2 | 1.00 | 2 | 6 | |
| 2 | 30 | 2 | 11 T | 14 | Positive | Proximal | 0 | 1.50 | 0 | 6 | |
| 3 | 68 | 1 | 6 T | 10 | Negative | Proximal | 0 | 0.75 | 3 | 6 | |
| 4 | 83 | 2 | 14 | 4 | Negative | Distal | 0 | 0.75 | 3 | 3 | 40 |
| 5 | 56 | 0 | 7 T | 27 | Positive | Proximal | 0 | 0.25 | 0 | 6 | |
| 6 | 57 | 3 | 9 T | 11 | Negative | Middle | 1 | 0.50 | 3 | 6 | |
| 7 | 40 | 0 | 3 T | 27 | Positive | Distal | 0 | 0.50 | 3 | 4 | 60 |
| 8 | 32 | 0 | 10 | 22 | Negative | Distal | 1 | 1.00 | 3 | 0 | 100 |
| 9 | 50 | 0 | 11 | 14 | Positive | Proximal | 0 | 1.00 | 0 | 6 | |
| 10 | 55 | 0 | 7 | 23 | Positive | Proximal | 0 | 0.875 | 0 | 6 | |
| 11 | 73 | 0 | 11 T | 25.5 | Negative | Proximal | 0 | 0.25 | 0 | 6 | |
| 12 | 66 | 3 | 7 | 1 | Negative | Distal | 0 | 1.75 | 3 | 6 | |
| 13 | 69 | 2 | 11 T | 8 | Negative | Middle | 0 | 1.00 | 0 | 6 | |
| 14 | 56 | 2 | 15 | 79 | Positive | Distal | 0 | 1.50 | 3 | 2 | 100 |
| 15 | 71 | 1 | 4 | 5.5 | Negative | Middle | 0 | 0.50 | 3 | 6 | |
| 16 | 47 | 0 | 6 | 9.5 | Negative | Middle | 0 | 1.00 | 3 | 6 | |
| 17 | 78 | 1 | 15 | 72 | Negative | Middle | 2 | 0.75 | 3 | 2 | 100 |
| 18 | 12 | 0 | 6 | 27 | Positive | Distal | 0 | 1.75 | 2 | 2 | 100 |
| 19 | 50 | 0 | 5 | 12 | Positive | Middle | 0 | 0.50 | 0 | 6 | |
| 20 | 78 | 0 | 6 | 24 | Positive | Distal | 2 | 1.25 | 3 | 3 | 45 |

**TABLE 2: Survival by clot location**

| Clot Location | Alive at 3 Mo | Dead at 3 Mo |
|---|---|---|
| Distal | 5 | 2 |
| Middle, proximal | 2 | 11 |

Note.—$P = .02$.

hemorrhagic transformations occurred in patients treated within 10 hours of symptom onset (three of seven). A greater number of patients were treated after 10 hours, and no hemorrhages occurred in that group of 13 patients ($P = .03$).

Two patients (cases 5 and 19) had hemorrhagic events related to technical complications. One patient had an arterial perforation at the distal vertebral artery caused by a microcatheter as it was being advanced across an acute angle in the vessel. The other patient had an arterial rupture during angioplasty of a tight basilar artery stenosis. No patient who had a hemorrhage survived, whether the hemorrhage resulted from a technical complication or as a transformation of a related infarction.

Early treatment did not guarantee good outcome. None of the seven patients treated within 10 hours of symptom duration had a good outcome; all patients with good outcomes were in the group whose symptom duration was longer than 10 hours of (four of 13) ($P = .25$). Initial neurologic status was not a reliable predictor of outcome. One of 11 patients with GCS scores under 10 and three of eight patients with GCS scores of 10 or above at the time of treatment had good outcomes ($P = .26$). Age, in general, did not correlate with outcome. The average age of patients was 57 years (range, 12 to 83 years). Three of 11 patients under age 60 had good outcomes, and one of nine patients over age 60 had a good outcome ($P = .59$). There was a trend toward increased survival with complete recanalization. Six of 11 patients with a TIMI score of 3 survived, whereas only one of nine patients with a TIMI score of 0 to 2 survived ($P = .07$). The degree of recanalization did not correlate as well with neurologic outcome. Three of 11 patients with a TIMI score of 3 had good neurologic outcomes and one of nine patients with a TIMI score of 0 to 2 had a good neurologic outcome ($P = .59$).

## Discussion

The clinical diagnosis of basilar artery thrombosis can be difficult and the definitive diagnosis is often delayed, in part because basilar thrombosis is relatively rare, and the initial evaluating physicians (who are usually not neurologists) may not make the correct diagnosis from the sometimes vague presenting symptoms. For

example, in a woman (case 6) hospitalized with congestive heart failure, the lack of mental alertness was initially attributed by her internists to poor oxygenation. In a child (case 18), headache and lethargy were initially attributed by her pediatricians to a viral syndrome. In such cases, it is not until focal neurologic signs become obvious or until the patient markedly deteriorates that neurologic consultation is sought and the diagnosis suspected. Even a neurologist may be misled by initial neurologic signs and conclude that deficits are the result of a carotid event (16). Should such delays in diagnosis contraindicate thrombolysis for basilar thrombosis?

To limit hemorrhagic transformation of infarctions, it has become fairly standard practice in the anterior circulation to restrict intraarterial thrombolysis to the first 6 hours of symptoms and intravenous thrombolysis to the first 3 hours. Clinical evidence supports this rationale (17–19); however, as the present study indicates, in cases of basilar thrombosis, intraarterial thrombolytic treatment should still be considered when symptom duration exceeds 6 hours. The time window for intraarterial thrombolytic therapy in the present series was extended considerably in cases of basilar thrombosis because of the poor prognosis associated with conservatively treated cases. Extending this time limit may have proved beneficial to some patients. Furthermore, extending the time limit did not increase the risk of hemorrhagic transformation, at least in this small series. It is possible that the brain stem is tolerant to longer periods of ischemia than is the cerebral cortex.

In a previous study of outcome after basilar thrombolysis (14), patients were excluded from thrombolysis if brain CT scans showed any evidence of ischemia. In the present study, the pretreatment brain CT scans were used to exclude a single patient who had extensive, well-defined infarction in the brain stem, and would have been used to exclude any patient with hemorrhage (none in our series). Subtle brain stem ischemic changes on CT, however, were not contraindications for treatment. We allowed wide latitude with cerebellar ischemic changes. In one patient (case 18), unilateral cerebellar hemispheric edema was so extensive by the time of angiography that a decompressive suboccipital craniectomy was performed the day after thrombolysis. Nonetheless, that patient returned to a very productive and active life (modified Rankin score 2). Our study indicates that if patients had been excluded from thrombolysis simply on the basis of any related ischemic changes on CT scans, a subgroup of patients who ultimately did well would have been excluded. Furthermore, if all patients with related ischemic CT changes had been excluded, no patient who had a subsequent hemorrhagic transformation would have been excluded.

Neurologic status just before thrombolysis did not predict ultimate outcome, but the duration of severe neurologic deficits might be an important prognostic indicator. Some patients had stuttering clinical courses and were treated conservatively during the early, less severe stages. After sudden clinical deterioration, they were referred for thrombolysis. Other patients were treated after shorter durations consisting of more severe deficits. These differences might have implications for eventual recovery. Collateral circulation could also be an important factor in predicting outcome, and we intend to investigate the value of collaterals in basilar thrombosis in a subsequent effort.

Mortality and functional status determinations are time dependent, and the selection of end points can influence reported outcomes. The patients who did well after basilar thrombolysis tended to improve further after hospital discharge, while those who were severely impaired tended to deteriorate. Neurologic outcome was better in survivors at the 3-month evaluation point than at the time of hospital discharge. The survival rate, however, would have been higher had it been reported at hospital discharge, since two patients (cases 3 and 13) died after transfer to nursing homes.

Complications can be divided into those occurring during the actual procedure and those occurring subsequent to the procedure. There were two technical complications. In one (case 5), the microcatheter could not be passed around a tortuous, diseased segment of the distal vertebral artery near the vertebrobasilar junction, where there was a coincidental aneurysm. Urokinase was therefore delivered proximal to the posterior inferior cerebellar artery, remote from the clot surface in the basilar artery, but this had no visible effect after 250 000 U. A second attempt was made to advance the microcatheter to the clot surface, but the microwire or microcatheter perforated the vertebral artery, resulting in a subarachnoid hemor-

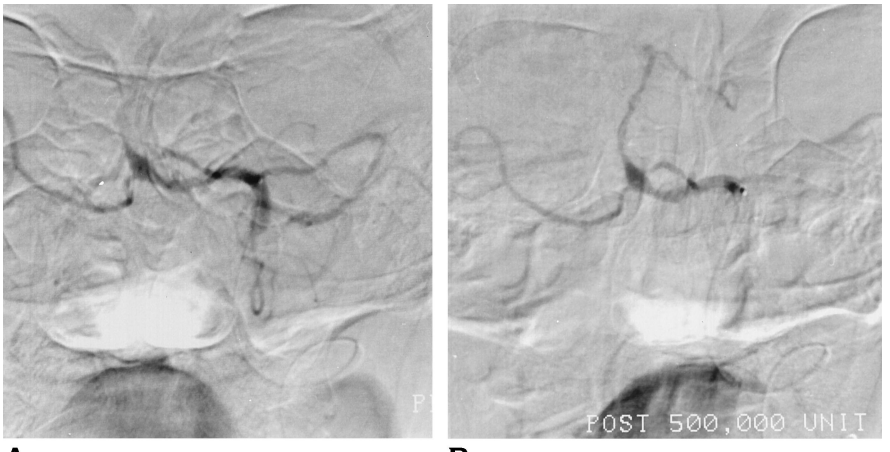
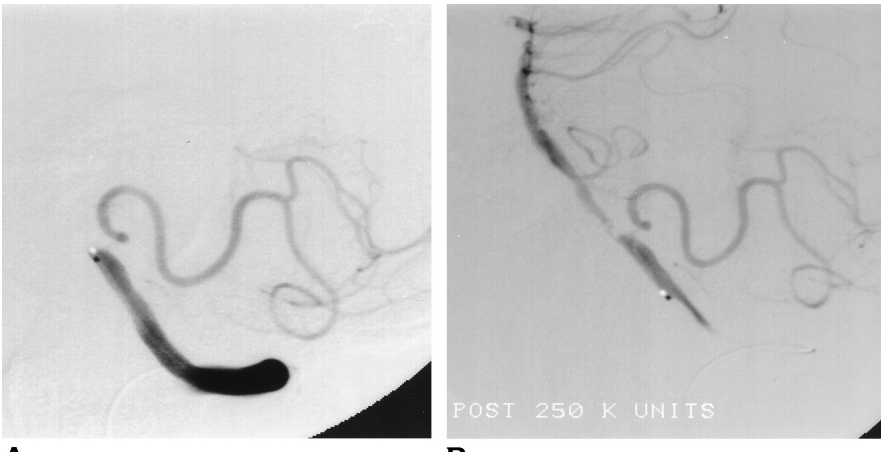



Fig 2. Case 13: Midbasilar thrombosis.
*A*, Initial frontal projection shows an occlusion of the midbasilar artery distal to the right anterior inferior cerebellar artery–posterior inferior cerebellar artery.
*B*, After 500 000 U urokinase, there is recanalization of an atherosclerotic and stenotic basilar artery, but the artery reoccluded, despite additional urokinase infusion.

rhage. In the second case, in a patient with midbasilar clot and midbasilar stenosis (case 19), clot repeatedly reformed and occluded the vessel between urokinase infusions. Angioplasty was performed in an attempt to reduce the degree of stenosis and encourage patency, but the vessel perforated, resulting in subarachnoid hemorrhage. In both instances, heparin was immediately reversed with protamine and further thrombolysis was not attempted. While these procedural steps were believed justified in patients with an extremely high likelihood of death otherwise, interventionalists should recognize that basilar angioplasty and extensive catheter manipulation in diseased arteries carry significant risk.

There were three cases of hemorrhagic transformations of infarctions after thrombolysis. In one patient (case 16), a 5-mm right midbrain hemorrhage was evident immediately after thrombolysis, but there were no signs of further clinical deterioration. The patient was maintained on heparin until warfarin levels were therapeutic, and his neurologic status improved. One week after thrombolysis, the hemorrhage enlarged to involve most of the midbrain, a finding that was associated with clinical deterioration. This decline was attributed to a late hemorrhagic transformation while the patient was on an anticoagulant rather than to hemorrhagic transformation related to the thrombolytic procedure. In a second patient (case 13), a dorsal pontine hemorrhage occurred within 24 hours of thrombolysis while the patient was receiving heparin. This event was associated with clinical deterioration (Fig 2). In a third patient (case 15), a brain stem and bithalamic hemorrhage with ventricular extension occurred within 24 hours while the patient was on heparin infusion and was associated with clinical deterioration. None of these patients had ischemic changes evident on preprocedural brain CT scans. This hemorrhagic transformation rate of 15% is identical to that found in one study of control subjects and patients with occlusion of the middle cerebral artery treated with intraarterial thrombolytics within 6 hours of the event (H. A. Rowly, W. P. Dillon, R. T. Higashida, et al, "Acute Middle Cerebral Artery Thrombolysis: Neuroradiologic Findings in the PROACT Study," presented at the annual meeting of the American Society of Neuroradiology, Seattle, Wash, June 1996) and similar to that (14%) found in a study of patients with basilar thrombosis treated with intraarterial thrombolytics within 48 hours (14). Higher rates, up to 34%, have been quoted for intravenous thrombolysis in patients treated within 6 hours of symptom onset (19). The longer interval from symptom onset to thrombolytic treatment in this small series did not appear to increase the rate of hemorrhagic transformation beyond that reported in series with shorter time frames in other circulations, and no factors were identified that would have accurately eliminated the patients who hemorrhaged from treatment.

All hemorrhagic transformations occurred in the group of patients treated within 10 hours of symptom onset. We cannot explain why the opposite was not true. Perhaps there are two distinct subpopulations of patients, one in whom there is an acute and precipitous decline and one in which there is a slower, more steplike deterioration (20). The patients with longer ischemic tolerance and without early severe deterioration may have had better collateral circu-




A B

Fig 3. Case 9: Proximal thrombosis.

*A*, Lateral projection of the initial superselective left vertebral angiogram shows an occlusion just distal to the posterior inferior cerebellar artery. There is a tight stenosis of the posterior inferior cerebellar artery origin. The right vertebral artery (not shown) was also occluded.

*B*, After 250 000 U urokinase, there is recanalization, but a severe stenosis of the distal left vertebral artery is present. The artery reoccluded at the site of stenosis, despite additional heparin and urokinase.

lation, but we do not have the data at present to confirm it.

Our data help to identify some favorable prognostic signs in a population of essentially unselected patients referred for thrombolytic therapy with angiographic confirmation of the diagnosis of basilar thrombosis. The group of patients who did best were those who had clot confined to the distal basilar artery and who had complete recanalization. This observation confirms findings of another recent study (14). Distal basilar thrombosis is often of embolic origin (21, 22); perhaps lysis of an embolus lodged in a normal artery is more favorable than lysis of thrombus formed at a site of atherosclerosis. In adults with midbasilar and proximal basilar thrombosis, there is often underlying basilar artery atherosclerosis. Here, clot may be more prone to recur. In support of this notion, in two patients in the present series (cases 9 and 19), thrombus repeatedly reformed on atherosclerotic stenoses (Fig 3).

It is noteworthy that no adult had a good neurologic outcome without complete recanalization, but the single child in our series did. Basilar thrombosis is rarely seen in childhood (23). We do not yet have sufficient experience to state whether recovery in children is better than in adults with similar occlusions.

These data alone do not prove a benefit for thrombolysis in cases of basilar thrombosis, but there are some historical facts to consider in addition to the high mortality rates associated with conservatively treated basilar thrombosis. In contrast to our observation that lysis of a distal basilar artery clot is often associated with a good outcome, persistent occlusion of the distal basilar artery has often been associated with a poor outcome. A report of 10 patients who survived basilar thrombosis without thrombolysis listed no survivors with a distal basilar occlusion (9). A study of seven surgical occlusions of the basilar artery for aneurysm treatment (24) found that those who had clips placed on the proximal basilar artery or distal vertebral arteries had better neurologic outcomes than those who had clips placed on the distal basilar artery. Standardization of outcome end points has been lacking in most series of basilar thrombosis, but a 71% survival rate, as we report after thrombolysis, has never been suggested as the natural history of distal basilar thrombosis. Only a randomized study would prove or disprove benefit, but defining treatment for the control subjects could prove problematic.

## Conclusions

Among the factors evaluated in this series of patients with basilar artery thrombosis treated with intraarterial urokinase, the best determinant of survival was distal basilar artery clot location. Complete recanalization favored survival and should be the goal of thrombolytic treatment. Delays in diagnosis, advanced age, severe neurologic deficits, and ischemic changes on CT scans should not be considered absolute contraindications for intraarterial thrombolysis in cases of basilar thrombosis.

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