

**Endovascular recanalization of acute intracranial vertebrobasilar artery occlusion  
using local fibrinolysis and additional balloon angioplasty**

Junji Kashiwagi, M.D.<sup>1</sup>, Hiro Kiyosue, M.D.<sup>2</sup>, Yuzo Hori, M.D.<sup>3</sup>, Mika Okahara, M.D.<sup>1</sup>,  
Shuichi Tanoue, M.D.<sup>2</sup>, Yoshiko Sagara, M.D.<sup>2</sup>, Toshi Abe, M.D.<sup>4</sup>, Hiromu Mori, M.D.<sup>2</sup>

<sup>1</sup>Department of Radiology, Shinbeppu Hospital, 874-0833 Tsurumi 3898, Beppu, Oita,  
Japan

<sup>2</sup>Department of Radiology, Oita University Faculty of Medicine, 879-5593 Idaigaoka  
1-1, Hasamacho, Yufu, Oita, Japan

<sup>3</sup>Department of Radiology, Nagatomi Neurosurgical Hospital, 870-0820, Nishiomichi  
2-1-20, Oita, Oita, Japan

<sup>4</sup>Department of Radiology, Kurume University School of Medicine, 830-0011,  
Asahimachi 67, Kurume, Fukuoka, Japan

Address correspondence to Junji Kashiwagi, Department of Radiology, Shinbeppu  
Hospital, Tsurumi 3898, Beppu, Oita, Japan

Postcode 874-0833

FAX: 81-97726-4170, TEL: 81-97722-0391

email: [jaykay@med.oita-u.ac.jp](mailto:jaykay@med.oita-u.ac.jp)

Purpose: Vertebrobasilar artery occlusion (VBO) produces high mortality and morbidity due to low recanalization rate utilization in endovascular therapy. The use of percutaneous transluminal angioplasty (PTA) to improve recanalization rate additional to local intra-arterial fibrinolysis (LIF) was investigated in this study. Results obtained following recanalization therapy in acute intracranial VBO are reported.

Methods: Eighteen consecutive patients with acute VBO underwent LIF with or without PTA, from August 2000 to May 2006. Eight patients were treated using LIF alone, and 10 required additional PTA. Rate of recanalization, neurological status before treatment, and clinical outcomes were evaluated.

Results: Seventeen of 18 patients achieved recanalization. One procedure-related complication of subarachnoid hemorrhage occurred. Overall survival rate was 94.4% at discharge. Seven patients achieved good outcomes [modified Rankin scale (mRS) 0–2], and the other 11 had poor outcomes (mRS 3–6). Five of six patients who scored 9–14 on the Glasgow Coma Scale (GCS) before treatment displayed good outcomes, whereas 10 of 12 patients who scored 3–8 on the GCS showed poor outcomes. GCS prior to treatment showed a statistically significant correlation to outcomes ( $p < 0.05$ ). Moreover, the National Institutes of Health Stroke Scale (NIHSS) before treatment correlated well with mRS (correlation coefficient: 0.487). No statistical difference between the good

and poor outcome groups was observed for the duration of symptoms, age, etiology and occlusion site.

Conclusions: Endovascular recanalization can reduce mortality and morbidity of acute VBO. Good GCS and NIHSS scores prior to treatment can predict the efficacy of endovascular recanalization.

Keywords: basilar artery occlusion; interventional radiology; thrombolysis; percutaneous transluminal angioplasty

## **Introduction**

Acute vertebrobasilar artery occlusion leads to poor morbidity and mortality, even following anticoagulation and platelet inhibitor therapy, with the mortality rate with anticoagulation therapy reported to be approximately 90% [1]. Several reports have shown that local intra-arterial fibrinolysis (LIF) of vertebrobasilar artery occlusion can improve mortality and morbidity. However, recanalization rates are relatively low and mortality has remained high, at ~60%, even with the use of LIF [1-5]. In patients with embolic vertebrobasilar artery occlusion, the embolus is often so large as to be resistant to LIF, and requires high doses of fibrinolytic agents, which can cause hemorrhagic complications. In patients with atherothrombotic occlusion, recanalization can rarely be obtained by LIF alone. Furthermore, residual high grade stenosis usually remains after recanalization, which often causes reocclusion. The reocclusion rate after LIF has been reported to be 10–30% [2,6]. Percutaneous transluminal angioplasty (PTA) has been developed for the treatment of intracranial atherosclerotic stenosis. Some investigators have applied PTA for acute occlusion, which can lead to higher recanalization rates and decrease morbidity. However, few reports have described the efficacy and safety of PTA for the treatment of acute intracranial vertebrobasilar artery occlusion in a large number of cases. In this study, retrospective analysis of patients with acute

vertebrobasilar artery occlusion treated using LIF with or without PTA, in our institutions, was performed. This study, therefore, aimed to establish the factors increasing survival rate and good neurological outcomes.

## **Methods**

### **Patients**

Endovascular treatment of vertebrobasilar artery occlusion included patients admitted with neurological symptoms who were subsequently found to have vertebrobasilar artery occlusion using magnetic resonance angiography (MRA) and/or computed tomography (CT). Exclusion criteria for endovascular recanalization were a large brain stem infarction [more than half the area of the brain stem on at least one axial slice of CT and/or magnetic resonance imaging (MRI)], recent history (within 4 weeks) or presence of intracranial hemorrhage or hemorrhagic infarction, or long duration after onset (>12 hours). From August 2000 to May 2006, 18 consecutive patients [14 males and 4 females, aged 50–84 years (mean age 69.9 years)] with acute intracranial vertebrobasilar artery occlusion underwent endovascular recanalization therapy at our institutions, including LIF using urokinase, and PTA. Informed consent for endovascular treatment was obtained from each patient and/or their nearest relatives.

Their clinical findings, angiograms and outcomes are summarized in Table 1. The duration between the onset of neurological symptoms and endovascular recanalization therapy ranged from 1 to 8.5 hours. The median Glasgow Coma Scale (GCS) score on admission was 7 (range 3–14). The median National Institutes of Health Stroke Scale (NIHSS) were 20.5 (range 3-35). Among the 18 patients, etiology of occlusion was cardiogenic embolism in 11 patients and atherothrombotic occlusion in 7 patients. All patients underwent non-contrast brain CT before treatment to exclude hemorrhagic transformation of brain parenchyma and diffuse brain stem infarction. MRI and MRA were performed in all but three patients. Neurosurgeons and/or neurologists estimated neurological status before treatment and scored with the GCS and NIHSS. Clinical outcomes at 3 months were classified using the modified Rankin scale (mRS). Mean hospitalization days and follow-up days were 61.4 and 697, respectively.

### **Angiography and interventions**

In most cases, a transfemoral arterial or a brachial arterial approach was taken for transarterial digital subtraction angiography of the aortic arch, bilateral vertebral arteries, and bilateral common carotid arteries. The site of vertebrobasilar artery occlusion was categorized as: distal vertebral artery (VA), intracranial portion of the VA; proximal basilar artery (BA), from the vertebral arterial confluence to the origin of the anterior

inferior cerebellar artery; mid BA, from the origin of the anterior inferior cerebellar artery to the origin of the superior cerebellar artery; distal BA, distal to the superior cerebellar artery. Collateral flow that opacified the BA from the tip inferiorly to the level of the superior cerebellar arteries was termed distal, while collateral flow that opacified the BA from the tip to below the level of the anterior inferior cerebellar arteries was termed distal and middle. The number of collateral opacification of the P1 segment of the posterior cerebral artery (from the basilar tip to the confluence of the posterior communicating artery: PcoA) was also recorded. At least one P1 segment could be obtained from nine patients via carotid injections. One P1 segment with leptomeningeal collateral flow via vertebral injections was also obtained from one patient.

All procedures were performed under local anesthesia with intravenous injection of heparin (1500–3000 U), patients with poor GCS required intubation, but no patients underwent intravenous anesthetics or inhalation anesthetics. A microcatheter was advanced distally beyond the occlusion site through a guiding catheter which was placed at the cervical portion of the VA. Urokinase was infused at a rate of 60 000 U every 5 minutes. During urokinase infusion, the tip of the microcatheter was slowly pulled back to the proximal portion of the occlusion site, and angiography was



performed after injection of every 60 000 U to estimate recanalization. When no or minimal thrombus regression was observed on angiography after injection of 120 000 U of urokinase, PTA was performed using a small balloon catheter with a balloon diameter 0.5–1 mm less than the estimated arterial diameter, and a low inflation pressure (<4 atmospheric pressure). Selection of PTA balloon catheter depended on the operator, i.e. 5 Gateway (Boston scientific, Natick, MA, USA), 4 fasSTEALTH (Boston Scientific) and 1 Sasuga (Boston Scientific). When recanalization was obtained after PTA, additional infusion of urokinase was performed until sufficient lysis of the residual thrombus in the vertebral BA was obtained, or 360 000 U of urokinase were infused. Total dosage of urokinase used ranged from 120 000 to 360 000 (mean 228 000) U. Eight patients were treated with LIF alone and 10 patients were treated with LIF and additional PTA. Six of the seven patients with atherothrombotic occlusion were treated using LIF and additional PTA. Seven out of 11 patients with cardiogenic thromboembolism were treated by LIF alone. Follow-up CT scanning was performed immediately, 12 hours, 24 hours and 3 days after the procedure. Continuous intravenous infusion of heparin (10 000 to 15 000 U/day) was performed for 3 days after the procedure. Antiplatelets were administrated after endovascular procedure for all cases of atherothrombotic occlusion. In contrast, no cases of cardiogenic thromboembolism

were treated with antiplatelets.

Recanalization rate, complications and clinical outcomes were evaluated. Recanalization was classified with arterial occlusive lesion score (AOL score) [7] which focused on recanalization of the primary arterial occlusive lesion. The AOL score was defined as: 0 = no recanalization of the primary occlusion, I = incomplete or partial recanalization of the primary occlusion with no distal flow, II = incomplete or partial recanalization of the primary occlusion with distal flow, or III = complete recanalization of the primary occlusion with distal flow. We also analyzed the relationship between neurological outcomes and factors potentially affecting these outcomes, including age, duration before treatment, sites of occlusion, etiology of occlusion, use of additional PTA, GCS and NIHSS before treatment. All statistical analyses were performed using SPSS version 10.0.7J software. Student's *t* test, Fisher's exact test,  $\chi^2$  test and Pearson's product moment correlation coefficient were used.

## **Results**

Vertebrobasilar artery occlusion as observed with vertebral angiography was found in the distal VA in 2 patients, mid BA in 6 patients and distal BA in 10 patients. In both patients with distal VA occlusion, contralateral vertebral angiography showed

hypoplasty of the contralateral VA. Two of the 10 patients with distal BA occlusion showed concurrent occlusion of the VA at the origin and hypoplasty of the contralateral VA.

Collateral opacification of the distal BA beyond the occlusion site and the P1 segment of the posterior cerebral artery are shown in Table 1. Distal BA opacification from collaterals could be estimated only when the occlusion site was below the mid BA. Six patients with mid BA occlusion were observed. Three patients displayed no collateral distal basilar flow, whilst three were observed with retrograde collateral flow via PcoA or leptomeningeal anastomosis. There was no correlation between the opacification of the distal BA and mRS (Table 2). Two patients with distal VA occlusion revealed retrograde distal to middle BA opacification in one patient and no retrograde BA opacification in the other. The outcomes were the same as mRS 1. There was no significant correlation between the opacification number of P1s and mRS (Table 2). However, a trend showed that only 20.0% of patients could achieve good outcomes when no P1 opacification was observed. By contrast, 50.0% of patients showed good outcomes when at least one P1 opacification site was present.

#### *Technical results and complications*

Recanalization was achieved in 17 of the 18 patients (94.4%). After LIF using 120 000 U urokinase, complete recanalization (AOL III) was obtained in 6 patients, partial recanalization (AOL II) was found in 5, and no recanalization (AOL 0) was found in 7 patients. No patients had minimal recanalization without distal flow (AOL I).

Additional PTA was performed in all 7 patients of AOL 0, and in 3 of the 5 patients classified as AOL II. Finally, recanalization of the vertebrobasilar artery was obtained in 17 out of 18 patients, which included 9 complete recanalizations without residual stenosis or thrombi at the occlusion site (AOL III) and 8 incomplete recanalizations with residual filling defects or stenosis (AOL II). All 9 cases which showed complete recanalization had cardiogenic embolism due to atrial fibrillation. Among the 8 patients who showed incomplete recanalization, all except 2 had atherothrombotic occlusion. The remaining patient suffered subarachnoid hemorrhage caused by penetration of the microguidewire during its passage through the occlusion site of the BA. The procedure was, therefore, interrupted in this patient, and resulted in no recanalization (AOL 0).

Acute or subacute reocclusion was not observed. Two patients who had been recanalized with PTA showed restenosis 1 month after the procedures and were retreated by stenting. Hemorrhagic infarction after the procedure was observed in 4 patients on follow-up CT. In 3 patients, petechial hemorrhagic changes that did not

affect the outcomes were found in the occipital lobe. Massive symptomatic hematoma was observed in one patient, which caused severe neurological aggravation. No other complications relating to the procedures were observed.

*Clinical outcomes and related factors (Table 3)*

Outcomes at discharge or 3 months after treatment were favorable (mRS 0–2) in seven patients (38.9%) and poor (mRS 3–6) in 11 patients (61.1%). During a follow-up period of 0–62 months (mean 22.9 months), one patient died 23 days after the procedure due to pneumonia. Good outcomes were more frequently observed in patients with atherothrombotic occlusion (43%) than in those with cardiogenic thromboembolism (36%); however, there was no statistically significant difference between both etiologic groups (Table 3).

No statistical relationship was shown between AOL score after treatment and mRS.

Both patients whose clots were located at the distal VA showed good neurological outcomes, as did five of the 16 patients (31.3%) with mid or distal BA occlusion. Although patients with mid/distal BA occlusion tended to show poorer outcomes than those with proximal occlusion, statistical analysis showed no correlation between

outcomes and site of occlusion.

The mean duration between onset of symptoms and start of endovascular procedure was similar in both the good (260 min, 60–445 min) and poor outcome groups (250 min, 110–510 min).

Five out of 6 patients (83.3%) who scored 9–14 on the GCS before treatment showed good outcomes. By contrast, only two of 12 patients who scored 3–8 on the GCS recovered well. There was a statistically significant correlation between GCS score before treatment and neurological outcomes ( $P=0.013$ ).

Pearson's correlation coefficient was 0.487 between NIHSS and mRS which demonstrated a significant correlation. As NIHSS score increased, so did mRS observed. All the patients who demonstrated a NIHSS greater than 29 returned a mRS of 5 or 6.

No significant correlation between outcomes and age was observed.

#### *Etiology and related factors (Table 4)*

No differences were found in ages and mean number of occlusion of PCA and SCA after procedure in both etiologies. Patients with cardiogenic thromboembolism tended to be poor GCS score and NIHSS than the patients with atherothrombotic occlusion. Atherothrombotic occlusion required additional PTA (6 of 7) more

frequently, and therefore, it required longer procedure time (199 minutes) than cardiogenic thromboembolism (requirement of PTA, 4 of 11; procedure time, 122 minutes). However, there were no statistically significant differences between both etiologies in those factors. A significant difference was found in AOL score which shows lower in atherothrombotic occlusion (1.71) than cardiogenic thromboembolism (2.73) ( $p=0.01$ ).

#### *Use of additional PTA (Table 5)*

There were 8 patients treated by LIF alone (LIF group) and 10 patients treated by LIF and additional PTA (PTA group). PTA group included larger number of atherothrombotic occlusion (6 of 10 patients) than LIF group (1 of 8 patients). The mean ages, initial GCS and NIHSS were similar in both groups. PTA group showed slightly longer procedure time (164 minutes vs. 143 minutes), slightly lower AOL score (2.10 vs. 2.63), and better outcome (good outcome: 50% vs. 25%). However, there were no statistically significant differences.

## **Discussion**

Acute intracranial vertebrobasilar artery occlusion is often associated with high

morbidity and mortality, even when treated with anticoagulation and platelet inhibitors [1]. During the last two decades, several authors have reported the efficacy of LIF for the treatment of occluded vertebrobasilar artery. However, reports of recanalization rates were low (44-65%), and mortality rates were still high (>50%) [1-5]. A higher recanalization rate was considered to lead to a lower mortality rate. Hacke et al. [1] reported that the recanalization rate by LIF was 44% and the mortality rate was 69%. Recently, Arnold et al. [8] reported that the recanalization rate by LIF and PTA was 80% and the mortality rate was 42%. Ezaki et al. [9] reported similar results, with a recanalization rate for LIF and PTA of 92% and a mortality rate of 27%. Approximately 10% of patients were treated with PTA in these studies [8,9]. More recently, Eckert et al. [10] reported the results of a multicenter study of aggressive therapy with intravenous abciximab and intra-arterial rtPA, and additional PTA/stenting, in 47 patients with acute vertebrobasilar occlusion. Balloon PTA or stenting was performed in 31.9% of patients, resulting in a 72% recanalization rate and a 38% mortality rate. These results suggest that higher recanalization rates may be related to lower mortality. Lindsberg et al. [11] reported the systematic analysis of the treatment for vertebrobasilar artery occlusion. From their data, successful recanalization was a decisive factor in promoting good outcomes, as the likelihood of good outcomes in the non-recanalized cases was only 2%.



In the present study, we achieved 94% recanalization and 94% survival. PTA was performed in 55.5% of patients who had residual stenosis/thrombus after LIF with a small dosage of urokinase (120 000 U). Prolonged infusion of thrombolytic agents in LIF-resistant patients, such as those with firm clots or atherothrombotic occlusion, may lead to lower recanalization and increased risk of hemorrhagic complications. PTA can immediately restore blood flow in the stenotic or occlusive artery by crushing the clots or dilation of the atherosclerotic stenosis. Additional PTA in the procedure would be a major contributor to the high recanalization rates and good outcomes in our study. The dose of urokinase used (mean 228 000 U) was less than that in other reports (400 000–1 800 000 U) [1-6,8,9]. Massive hemorrhagic transformation is one of the most important complications associated with endovascular recanalization. This complication occurred after treatment of acute vertebrobasilar artery occlusion in 8–25% of previously reported cases. Only one case (5.6%) in our study showed symptomatic hemorrhage after treatment. The lower incidence of hemorrhagic complications may be related to good outcomes, and may have been caused by the low dosage of urokinase. Therefore, early judgment of LIF-resistant occlusion and additional PTA could be associated with good outcome. However, there are potential risks of balloon PTA for acute occlusion of intracranial arteries, which include arterial dissection, subarachnoid hemorrhage and

acute occlusion, which can be fatal. Nahser *et al.* [12] have discussed their experiences of 20 patients with vertebrobasilar artery stenosis treated by balloon angioplasty, and presented a review of 126 published cases. From their data, angiographic complications occurred in seven patients (8.2%; five dissections, one acute occlusion of the BA, and one perforating occlusion), of which four (4.7%) were fatal. No dissection was encountered in our study. We performed balloon angioplasty using a small caliber balloon (0.5–1 mm less than the ideal size) inflated gently with low pressure (<4 atm). Use of these techniques may reduce the risk of angioplasty-related vessel injury. We experienced one complication directly related to the procedure, in which a microguidewire penetrated the BA during navigation through the occluded portion of the artery. The penetrated portion of the BA was occluded, and CT following the procedure showed only a small subarachnoid hemorrhage. The complication seemed not to worsen patient outcomes. However, careful attention should be practiced when manipulating the catheter and guidewire through the occluded portion of the artery. Another potential disadvantage of balloon PTA is clot disruption and dislocation especially in cases of cardiogenic embolism, which can cause distal embolisms and occlusion of perforators. Recent developing technique of clot retrieve such as suction catheters technique and mechanical clot retriever might have an

advantage in order to remove large clots without clot disruption in cases of cardiogenic thromboembolism [13-18]. These reports were consisted with small number of cases and further investigations would be required. The utility of these extraction and thrombectomy devices for acute vertebrobasilar artery occlusion is limited to patients with embolic occlusion, who constitute less than half of patients. It would be easier to perform LIF with additional PTA than to use extraction/thrombectomy devices for the treatment of acute vertebrobasilar occlusion.

Other factors may affect patient outcomes. Brandt *et al.* [3] reported that distal BA occlusion and atherothrombotic occlusion tended to show high mortality.

Atherothrombotic occlusion also showed significant resistance to recanalization. By contrast, Cross *et al.* [19] showed better neurological outcomes in patients with distal basilar thrombosis, where the survival rate in patients with distal BA occlusion was significantly higher than in those with proximal and middle BA occlusion (71 versus 15%). Furthermore, this study mentioned that distal BA occlusion is frequently of embolic origin, and can often be recanalized by LIF. In our study, good outcomes were more frequently observed in patients with atherothrombotic occlusion than in those with cardiogenic thromboembolism (43 versus 36%). Regarding the site of occlusion, mid/distal BA occlusion tended to show poorer outcomes than proximal occlusion.

These differences in outcomes may have been caused by the different techniques used and the recanalization rates. Our technique using additional balloon PTA achieved a recanalization rate of 94% and 94% survival. Therefore, duration of ischemia and collateral blood flow are more important than the nature of the occlusion resistant to LIF. Cross *et al.* [4] demonstrated that most patients (83%) who had good collateral flow also had good neurological prognosis. These results suggest that recanalization of atherothrombotic occlusion of the vertebrobasilar artery may give good neurological outcomes when patients have good collateral blood flow. We investigated collateral flows in relation to distal basilar trunk opacification, PcoA visualization and P1 segment visualization. There was no significant correlation between the above items and mRS. However, there was a slight tendency, when a patient had at least one P1 opacification, 5 of 10 patients (50.0%) showed good outcomes, indicating that better outcomes occur with atherothrombotic occlusion. Cardiogenic thromboembolisms can occlude the distal portion of the BA, so simultaneous occlusion of the P1 segment of the posterior cerebral artery can easily occur.

Pretreatment GCS can reflect severity of brain stem ischemia, and was significantly correlated to outcomes in our study and in previous studies. We found that patients who scored >8 on the GCS had significantly better outcomes than patients who scored 3–8.

Arnold *et al.* [8] reported that low NIHSS scores (<20) are predictive of favorable outcomes. Our results also showed NIHSS had a significant correlation coefficient with mRS. Ezaki *et al.* [9] reported that patients with a GCS score of <10 upon admission had a worse prognosis. The present study demonstrated that the initial condition of a patient is a predictor of outcomes.

The duration of ischemia before treatment may influence patient outcomes. Eckert *et al.* [5] reported that early treatment within 6 hours of onset leads to significantly better neurological outcomes than delayed treatment. Arnold *et al.* [8] also reported that recanalization could be performed more frequently in cases treated within 6 hours, and that recanalization itself is an independent predictor of favorable outcomes. However, our results showed no difference in the mean duration of ischemia between the good outcome and poor outcome groups. Further investigations are necessary to determine the therapeutic time window that will achieve the best results.

Stenting for acute occlusion was not performed in our study, although two patients were treated using stenting for vertebrobasilar artery restenosis. Spreer *et al.* [20] reported three cases of primary stenting for residual stenosis immediately after LIF. There have been a few reports [10,20-22] of intracranial stenting for vertebrobasilar artery occlusion after LIF. Eckert *et al.* [10] reported that additional PTA/stenting was

performed after LIF in 31.9% of patients. However, they did not describe these cases in detail, such as the number of cases, efficacy and complications. Levy *et al.* [23,24] compared the safety of intracranial stenting for severe vertebrobasilar artery stenosis using three different methods: direct stenting without predilatation, stenting with predilatation, and staged stenting following balloon PTA. Direct stenting for acute vertebrobasilar artery occlusion was shown to possibly have a higher risk of complications than LIF with additional balloon PTA using a low inflation pressure.

There are possible factors affecting the results of this study. First, we use MR images for exclusion of patients. Evaluation by MRI, especially when using diffusion weighted image before treatment, might increase patients who met excursion criteria compared with CT evaluation alone. Second, we perform all procedure under local anesthesia because it can be performed immediately after diagnostic angiography without duration of preparing general anesthesia. Shorter duration after onset (within 12 hours) than other articles (within 18-79 hours) [1-5] might influence our good survival rate.

## Conclusion

PTA can feasibly be performed after LIF in patients with acute vertebrobasilar artery occlusion. Although our study was limited by the small number of cases, we achieved a

high recanalization rate and low mortality. Furthermore, a notable strong correlation of pretreatment GCS score with outcomes was observed.

## References

1. Hacke W, Zeumer H, Ferbert A, Bruckmann H, del Zoppo GJ (1988) Intra-arterial thrombolytic therapy improves outcome in patients with acute vertebrobasilar occlusive disease. *Stroke* 19:1216-1222
2. Becker KJ, Monsein LH, Ulatowski J, Mirski M, Williams M, Hanley DF (1996) Intraarterial thrombolysis in vertebrobasilar occlusion. *AJNR Am J Neuroradiol* 17:255-262
3. Brandt T, von Kummer R, Muller-Kuppers M, Hacke W (1996) Thrombolytic therapy of acute basilar artery occlusion. Variables affecting recanalization and outcome. *Stroke* 27:875-881
4. Cross DT 3rd, Moran CJ, Akins PT, Angtuaco EE, Diringer MN (1997) Relationship between clot location and outcome after basilar artery thrombolysis. *AJNR Am J Neuroradiol* 18:1221-1228
5. Eckert B, Kucinski T, Pfeiffer G, Groden C, Zeumer H (2002) Endovascular therapy of acute vertebrobasilar occlusion: early treatment onset as the most important factor. *Cerebrovasc Dis* 14:42-50
6. Zeumer H, Freitag HJ, Zanella F, Thie A, Arning C (1993) Local intra-arterial fibrinolytic therapy in patients with stroke: urokinase versus recombinant tissue



plasminogen activator (r-TPA). *Neuroradiology* 35:159-162

8. Arnold M, Nedeltchev K, Schroth G, Baumgartner RW, Remonda L, Loher TJ, Stepper F, Sturzenegger M, Schuknecht B, Mattle HP (2004) Clinical and radiological predictors of recanalisation and outcome of 40 patients with acute basilar artery occlusion treated with intra-arterial thrombolysis. *J Neurol Neurosurg Psychiatry* 75:857-862

7. Khatri P, Neff J, Broderick JP, Khoury JC, Carrozzella J, Tomsick T (2005) Revascularization end points in stroke interventional trials: recanalization versus reperfusion in IMS-I. *Stroke* 36:2400-2403

9. Ezaki Y, Tsutsumi K, Onizuka M, Kawakubo J, Yagi N, Shibayama A, Toba T, Koga H, Miyazaki H (2003) Retrospective analysis of neurological outcome after intra-arterial thrombolysis in basilar artery occlusion. *Surg Neurol* 60:423-430

10. Eckert B, Koch C, Thomalla G, Kucinski T, Grzyska U, Roether J, Alfke K, Jansen O, Zeumer H (2005) Aggressive therapy with intravenous abciximab and intra-arterial rtPA and additional PTA/stenting improves clinical outcome in acute vertebrobasilar occlusion: combined local fibrinolysis and intravenous abciximab in acute vertebrobasilar stroke treatment (FAST): results of a multicenter study. *Stroke* 36:1160-1165

11. Lindsberg PJ, Mattle HP (2006) Therapy of basilar artery occlusion: a systematic analysis comparing intra-arterial and intravenous thrombolysis. Stroke 37:922-928

12. Nahser HC, Henkes H, Weber W, Berg-Dammer E, Yousry TA, Kuhne D (2000) Intracranial vertebrobasilar stenosis: angioplasty and follow-up. AJNR Am J Neuroradiol 21:1293-1301

13. Bose A, Henkes H, Alfke K, Reith W, Mayer TE, Berlis A, Branca V, Sit SP (2008) The Penumbra System: a mechanical device for the treatment of acute stroke due to thromboembolism. AJNR Am J Neuroradiol 29:1409-1413

14. Mayer TE, Hamann GF, Brueckmann H (2002) Mechanical extraction of a basilar-artery embolus with the use of flow reversal and a microbasket. N Engl J Med 347:769-770

15. Mayer TE, Hamann GF, Schulte-Altendorneburg G, Bruckmann H (2005) Treatment of vertebrobasilar occlusion by using a coronary waterjet thrombectomy device: a pilot study. AJNR Am J Neuroradiol 26:1389-1394

16. Mangiafico S, Cellerini M, Nencini P, Gensini G, Inzitari D (2005) Intravenous glycoprotein IIb/IIIa inhibitor (tirofiban) followed by intra-arterial urokinase and mechanical thrombolysis in stroke. AJNR Am J Neuroradiol 26:2595-2601

17. Versnick EJ, Do HM, Albers GW, Tong DC, Marks MP (2005) Mechanical

thrombectomy for acute stroke. AJNR Am J Neuroradiol 26:875-879

18. Chapot R, Houdart E, Rogopoulos A, Mounayer C, Saint-Maurice JP, Merland JJ (2002) Thromboaspiration in the basilar artery: report of two cases. AJNR Am J Neuroradiol 23:282-284

19. Cross DT 3rd, Moran CJ, Akins PT, Angtuaco EE, Derdeyn CP, Diringer MN (1998) Collateral circulation and outcome after basilar artery thrombolysis. AJNR Am J Neuroradiol 19:1557-1563

20. Spreer J, Els T, Hetzel A, Arnold S, Klisch J, Huppertz HJ, Oehm E, Schumacher M (2002) Primary stenting as emergency therapy in acute basilar artery occlusion. Neuroradiology 44:791-795

21. Phatouros CC, Higashida RT, Malek AM, Smith WS, Mully TW, DeArmond SJ, Dowd CF, Halbach VV (1999) Endovascular stenting of an acutely thrombosed basilar artery: technical case report and review of the literature. Neurosurgery 44:667-673

22. Callahan AS 3rd, Berger BL (2000) Basilar artery endoprosthesis placement: rescue therapy for recurrent thrombosis. J Neuroimaging 10:47-48

23. Levy EI, Hanel RA, Bendok BR, Boulos AS, Hartney ML, Guterman LR, Qureshi AI, Hopkins LN (2002) Staged stent-assisted angioplasty for symptomatic intracranial vertebrobasilar artery stenosis. J Neurosurg 97:1294-1301

24. Levy EI, Hanel RA, Boulos AS, Bendok BR, Kim SH, Gibbons KJ, Qureshi AI, Guterman LR, Hopkins LN (2003) Comparison of periprocedure complications resulting from direct stent placement compared with those due to conventional and staged stent placement in the basilar artery. J Neurosurg 99:653-660

## Figure legends

Fig. 1. A 77-year-old man (case 12) with sudden onset loss of consciousness and quadriparesis at the time of presentation. Pretreatment GCS score was 5. (A) Initial non-contrast CT shows the high density of the basilar tip. (B) Diffusion-weighted image showing the absence of high signal intensity area in the brain stem. (C) MRA shows occlusion of the distal portion of the basilar artery. The right posterior cerebral artery shown was most likely supplied via the right posterior communicating artery. (D) Cerebral angiography, started 3.5 hours after onset, shows that the tip of the basilar artery was occluded with a thrombus. Both posterior cerebral arteries and the right superior cerebellar artery cannot be seen. (E) A microcatheter was introduced beyond the thrombus. 120 000 U urokinase were injected slowly. The right posterior cerebral artery and the superior cerebellar artery could not be seen after this procedure; however, the residual thrombus occluded the left posterior cerebral artery. PTA was performed from the distal basilar artery to the left posterior cerebral artery. Recanalization was achieved immediately. Another 60 000 U urokinase were injected to resolve the distal embolism. (F) Final angiography shows complete recanalization of the basilar artery; however, the slow blood flow caused by the distal embolism can be seen in the right superior cerebellar artery and left posterior cerebral artery. (G,H) Diffusion-weighted

images performed 8 days after the procedure show no brain stem infarction and minimal left thalamic infarction. The cortical infarction of the left posterior lobe is an old infarction which existed before onset. The patient had mild right hemiparesis and an mRS of 3.