PAPER

# Clinical and radiological predictors of recanalisation and outcome of 40 patients with acute basilar artery occlusion treated with intra-arterial thrombolysis

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**Objective:** To define predictors of recanalisation and clinical outcome of patients with acute basilar artery occlusions treated with local intra-arterial thrombolysis (IAT).
**Methods:** Vascular risk factors, severity of the neurological deficit graded by the National Institutes of Health stroke scale (NIHSS), and radiological findings were recorded at presentation. Outcome was measured using the modified Rankin scale (mRS) three months later and categorised as favourable (mRS 0–2), poor (mRS 3–5), or death (mRS 6).
**Results:** 40 patients were studied. Median NIHSS on admission was 18. Mean time from symptom onset to treatment was 5.5 hours (range 2.3 to 11). Outcome was favourable in 14 patients (35%) and poor in nine (23%); 17 (42%) died. There were two symptomatic cerebral haemorrhages (5%). Recanalisation of the basilar artery was achieved in 32 patients (80%); it was complete (TIMI grade 3) in 20% and partial (TIMI grade 2) in 60%. In multivariate logistic regression analysis, low NIHSS score on admission ($p = 0.002$) and vessel recanalisation ($p = 0.005$) were independent predictors of favourable outcome. Recanalisation occurred more often with treatment within six hours of symptom onset ($p = 0.003$) and when admission computed tomography showed a hyperdense basilar artery sign ($p = 0.007$). In a univariate model, quadriplegia ($p = 0.002$) and coma ($p = 0.004$) were associated with a poor outcome or death.
**Conclusions:** Low baseline NIHSS on admission and recanalisation of basilar artery occlusions predict a favourable outcome after intra-arterial thrombolysis. Early initiation of IAT and the presence of a hyperdense basilar artery sign on CT were associated with a higher likelihood of recanalisation.

Acute basilar artery occlusion is a serious condition which often results in severe disability or death.[1 2] Several series have suggested that local intra-arterial thrombolysis (IAT) improves basilar artery recanalisation and clinical outcome.[3–5] In these studies various thrombolytic agents and time windows to treatment were used. However, even with thrombolysis the prognosis remains poor in a large proportion of the patients, and whether to treat or withhold thrombolysis in a given patient is a crucial clinical question. To make such a decision some predictive variables may guide the clinician. To date, the variables reported to have influenced clinical outcome in previous case series have been heterogeneous. We therefore analysed the clinical findings and radiological data of our patients with acute stroke caused by basilar artery occlusion who had been treated with IAT within 12 hours of symptom onset, to identify factors that predict recanalisation and clinical outcome.

## METHODS

We studied 40 consecutive patients with basilar artery occlusion treated with IAT using urokinase. Thirty two patients were treated at the university hospital of Berne from December 1992 to February 2002 and eight patients at the university hospital of Zurich from September 1997 to February 2002. Ten of the Bernese patients were described in an earlier report.[6]

Inclusion criteria for IAT in patients with suspected basilar artery occlusion were as follows:

- A clinical diagnosis of acute stroke established by a staff neurologist;
- computed tomography (CT) ($n = 37$) or magnetic resonance imaging (MRI) ($n = 3$), including T2 weighted, fast echo gradient, and diffusion weighted images, ruled out an intracranial haemorrhage;
- occlusion of the basilar artery documented by cerebral four vessel arteriography;
- expected initiation of treatment was within the first 12 hours of symptom onset;
- no clinical or laboratory contraindications for IAT;
- verbal informed consent of the patient or their relatives was obtained.

From January 1998 to February 2002, 27 patients with basilar artery occlusion were treated in the two hospitals (22 in Berne and five in Zurich) and nine were not (five in Berne and four in Zurich). The reasons why nine patients were not treated with IAT were as follows: time delay from symptom onset to admission >12 hours ($n = 7$); vertebral artery occlusion and contralateral vertebral artery hypoplasia preventing access to the occluded basilar artery ($n = 2$); and acute basilar artery occlusion after open heart surgery ($n = 1$). Six patients in whom clinical findings suggested basilar artery occlusion had a normal angiogram. Before 1998 we did not record the data on all stroke patients and

**Abbreviations:** mRS, modified Rankin scale; NIHSS, National Institutes of Health stroke scale; TOAST, trial of Org 10172 in acute stroke treatment

angiographies routinely; only those with acute stroke and intra-arterial thrombolysis were noted in a separate registry.

The National Institutes of Health stroke scale (NIHSS) scores on admission were assessed by a staff neurologist.[7] Early CT signs of ischaemia were defined as parenchymal hypodensities in the vascular territory of the basilar artery and were evaluated by a staff neuroradiologist. A hyperdense basilar artery was defined as a hyperdense vessel on non-contrast CT compared visually with the density of other unaffected intracranial vessels.[8] Selective intra-arterial digital subtraction angiography was undertaken from a transfemoral approach using a biplane, high resolution angiography system (Toshiba CAS 500, Tokyo, Japan) with a matrix of 1024×1024 pixels. All patients underwent complete four vessel cerebral arteriography to assess collateral flow and the length of the basilar artery occlusion. In four patients with unilateral vertebral artery hypoplasia and contralateral extracranial high grade vertebral artery stenosis, the stenosis was dilated at angioplasty, in two cases combined with deployment of a stent, in order to gain access to the occluded basilar artery.

The site of basilar artery occlusion was categorised according to the criteria described by Archer *et al*: caudal, from the confluence of the vertebral arteries to the anterior inferior cerebellar artery; mid-basilar, from the anterior inferior cerebellar artery to the superior cerebellar artery; and distal, distal to the superior cerebellar artery.[2] The length of the basilar artery occlusion was defined as ''short'' if only one, and ''long'' if two or more segments of the basilar artery were not contrasted on the arteriogram.[3]

Collaterals were classified according to the criteria of Brandt *et al*: 0, no collaterals; 1 (minimal), antegrade or reversed partial or faint filling of collaterals; 2 (moderate), antegrade or reversed filling of the superior cerebellar arteries; and 3 (maximal), collaterals with antegrade and reversed filling or maximal bilateral filling of the superior cerebellar arteries.[9] For thrombolyis we used a Fast Tracker (Target Therapeutics) microcatheter, which was advanced into the occluded vessel. Urokinase (HS Medac) in a mean dose of 872 000 IU (range 20 000 to 1 250 000 IU) was given directly into the clot or as close as possible to its proximal end over a period of 60 to 90 minutes. Recanalisation was documented by a control arteriogram done immediately after IAT and classified by a neuroradiologist according to thrombolysis in myocardial infarction (TIMI) grades[10] as follows: TIMI grade 0, no recanalisation; TIMI grade 1, minimal recanalisation; TIMI grade 2, partial recanalisation; TIMI grade 3, complete recanalisation.

After IAT, 21 patients received aspirin in a dose of 250 to 500 mg a day. Nineteen patients, who had either been treated before publication of the international stroke trial results[11] or who had a residual high grade basilar artery stenosis on control arteriography after IAT, were given heparin in a therapeutic dose adjusted according to thrombin time. Control CT or MRI was undertaken routinely within 24 hours after IAT. In addition, CT was done in all patients where there was clinical deterioration. A symptomatic haemorrhage was defined as a homogeneous volume of blood associated with clinical deterioration. In two patients with massive cerebellar oedema a craniotomy was carried out. The aetiology of the ischaemic stroke was classified according to the TOAST criteria (trial of Org 10172 in acute stroke treatment), using additional investigations as necessary.[12] Functional outcome at three months was evaluated by different neurologists and classified using the modified Rankin scale (mRS).[13] The investigators were not blinded to the baseline scores and angiographic results.

### Statistics

Statistical analysis was carried out using SPSXX 10 for Macintosh statistical software (SPSS Inc, Chicago, Illinois, USA). Predictors of outcome were analysed by comparing patients with a functional outcome of mRS 0 to 2 (favourable outcome) with those with a functional outcome of 3 to 6 (3 to 5, poor outcome; 6, death). For analysis of the time variable, patients were grouped into those treated within $\leqslant 6$ and $>6$ hours of symptom onset; and to examine collateral circulation patients were dichotomised according to whether they had sufficient collaterals (grade 2 or 3) or insufficient collaterals (grade 0 and 1). To determine predictors of recanalisation, TIMI recanalisation grades 0 and 1 were defined as inadequate recanalisation and TIMI grades 2 and 3 as adequate recanalisation. The $\chi^2$ test was used for cross tabulation. Forward stepwise logistic regression analyses were carried out on the predictive factors identified by univariate analysis.

## RESULTS

### Demographic, clinical, and radiological data

Forty patients (22 men, 18 women) with a mean (SD) age of 58 (15) years (median 60, range 28 to 78) were treated. The median NIHSS score on admission was 18 (range 5 to 38). Thrombolysis was initiated on average 5.5 hours after symptom onset (range 2.3 to 11 hours). Sixteen patients had quadriplegia and 15 were comatose before IAT. There were early CT signs of ischaemia in the basilar artery territory in 10 of 37 patients (27%). A hyperdense basilar artery sign was seen on 26 of the 37 admission CT investigations (70%). Twenty patients (50%) had caudal, 13 (32%) midbasilar, and seven (18%) distal basilar artery occlusions. Seventeen patients had a short and 23 a long occlusion. The demographic, clinical, and radiological findings are summarised in tables 1 and 2.

### Clinical outcome and its predictors

At three months, outcome was favourable (mRS $\leqslant 2$) in 14 patients (35%) and poor (mRS 3 to 5) in nine (23%). Seventeen patients (42%) died (fig 1). The causes of death were failure of brain stem functions in eight, withdrawal of care in seven, and pneumonia in two.

In a univariate model, a baseline NIHSS score of $>20$ ($p<0.0001$), quadriplegia ($p = 0.002$), and coma on admission ($p = 0.004$) were clinical predictors of poor outcome or death. There was no significant association of age, sex, time to treatment, vascular risk factors, or stroke aetiology with clinical outcome (table 1). However, a trend was observed favouring patients treated within six hours of symptom onset, compared with those with later initiation of treatment (41% *v* 18% favourable outcome).

The only radiological predictor of favourable outcome was partial or complete recanalisation as seen on arteriography after IAT. All eight patients without or with minimal recanalisation had a poor outcome or died, whereas the outcome of 14 of 32 patients (44%) with partial or complete recanalisation was favourable ($p = 0.02$). Patients with sufficient collaterals showed a non-significant trend towards a better outcome (table 2). A hyperdense basilar artery sign on the admission CT scan, early CT signs of ischaemia, and localisation and length of basilar artery occlusions were not predictive of the clinical outcome.

After multivariate analysis, NIHSS score on admission ($p = 0.002$) and vessel recanalisation ($p = 0.005$) remained independent variables predicting outcome.

### Recanalisation and predictors of recanalisation

Thrombolysis recanalised eight (20%) of the occluded basilar arteries completely, and 24 (60%) partially. Complete or

**Table 1** Clinical predictors of functional outcome at three months

| Characteristic | Outcome | | p Value univariate | p Value multivariate | Total (n) |
|---|---|---|---|---|---|
| | Favourable (mRS 0–2) | Poor or death (mRS 3–6) | | | |
| No of patients | 14 (35%) | 26 (65%) | | | 40 |
| Age ⩽60 | 9 (45%) | 11 (55%) | | | 20 |
| Age >60 | 5 (25%) | 15 (75%) | NS | | 20 |
| Sex | | | | | |
| Male | 7 (32%) | 15 (68%) | | | 22 |
| Female | 7 (39%) | 11 (61%) | NS | | 18 |
| Diabetes | | | | | |
| Yes | 0 (0%) | 1 (100%) | | | 1 |
| No | 14 (37%) | 24 (63%) | NS | | 38 |
| Smoking | | | | | |
| Yes | 4 (31%) | 9 (69%) | | | 13 |
| No | 10 (38%) | 16 (62%) | NS | | 26 |
| Hypercholesterolaemia | | | | | |
| Yes | 4 (31%) | 9 (69%) | | | 13 |
| No | 10 (38%) | 16 (62%) | NS | | 26 |
| Hypertension | | | | | |
| Yes | 5 (26%) | 14 (74%) | | | 19 |
| No | 9 (45%) | 11 (55%) | NS | | 20 |
| NIHSSS on admission | | | | | |
| ⩽220 | 13 (59%) | 9 (41%) | | | 22 |
| >20 | 1 (6%) | 17 (94%) | <0.0001 | 0.002 | 18 |
| Coma | | | | | |
| Yes | 1 (7%) | 14 (93%) | | | 15 |
| No | 13 (52%) | 12 (48%) | 0.004 | NS | 25 |
| Quadriplegia | | | | | |
| Yes | 1 (6%) | 15 (94%) | | | 16 |
| No | 13 (54%) | 11 (46%) | 0.002 | NS | 24 |
| Time to treatment | | | | | |
| ⩽6 hours | 12 (41%) | 17 (59%) | | | 29 |
| >6 hours | 2 (18%) | 9 (82%) | NS | NS | 11 |
| Stroke aetiology | | | | | |
| Cardioembolic | 6 (50%) | 6 (50%) | | | 12 |
| Unknown | 2 (20%) | 8 (80%) | NS | NS | 10 |
| Large artery disease | 4 (29%) | 10 (71%) | | | 14 |
| VA dissection | 2 (50%) | 2 (50%) | | | 4 |

mRS, modified Rankin scale score; NIHSSS, National Institutes of Heath stroke scale score; VA, vertebral artery.

partial recanalisation was more frequent in patients treated within six hours than in those treated between six and 12 hours after symptom onset (90% *v* 55%, p = 0.011). On univariate analysis the presence of a hyperdense basilar artery sign on CT was associated with a greater likelihood of recanalisation (p = 0.022). Age, sex, stroke aetiology, NIHSS score on admission, quadriplegia, coma, early CT signs of ischaemia, localisation and length of basilar artery occlusion, and collaterals did not predict recanalisation. The demographic, clinical, and radiological findings and their

**Table 2** Radiological predictors of functional outcome at three months

| Characteristic | Outcome | | p Value univariate | p Value multivariate | Total (n) |
|---|---|---|---|---|---|
| | Favourable (mRS 0–2) | Poor or death (mRS 3–6) | | | |
| No of patients | 14 (35%) | 26 (65%) | | | 40 |
| CT dense artery | | | | | |
| Yes | 8 (31%) | 18 (69%) | NS | | 26 |
| No | 3 (27%) | 8 (73%) | | | 11 |
| CT early signs of ischaemia | | | | | |
| Yes | 4 (40%) | 6 (60%) | NS | | 10 |
| No | 7 (26%) | 20 (74%) | | | 27 |
| Length of occlusion | | | | | |
| Short | 7 (41%) | 10 (59%) | NS | | 17 |
| Long | 7 (30%) | 16 (70%) | | | 23 |
| Localisation of occlusion | | | | | |
| Caudal | 8 (40%) | 12 (60%) | NS | | 20 |
| Mid-basilar | 3 (23%) | 10 (77%) | | | 13 |
| Distal | 3 (43%) | 4 (57%) | | | 7 |
| Recanalisation | | | | | |
| TIMI 0 or 1 | 0 (0%) | 8 (100%) | <0.0001 | 0.005 | 8 |
| TIMI 2 or 3 | 14 (44%) | 18 (56%) | | | 32 |
| Collaterals | | | | | |
| Sufficient | 10 (48%) | 11 (52%) | NS | | 21 |
| Insufficient | 4 (21%) | 15 (79%) | | | 19 |

CT, computed tomography; mRS, modified Rankin scale score; TIMI, thrombolysis in myocardial infarction grade.

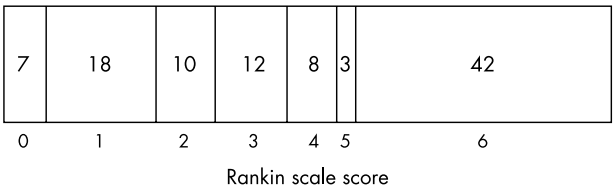


**Figure 1** Outcome according to the modified Rankin scale (RS) at three months. Values are percentages.

correlations with recanalisation are summarised in tables 3 and 4.

On multivariate analysis recanalisation was more likely with treatment within six hours of symptom onset ($p = 0.003$) and when admission CT showed a dense basilar artery sign ($p = 0.007$).

### Late recanalisation

In eight patients (20%) recanalisation of the basilar artery could not be achieved while they were in the angiography suite. Four patients died soon afterwards and did not have any vascular follow up investigations. Three patients were investigated by follow up transcranial Doppler within the first week. In one, the basilar artery was still occluded, and in two it was partially recanalised. A fourth patient underwent MR angiography on day 7. His basilar artery was partially recanalised.

### Complications

Two patients (5%) suffered a symptomatic intracerebral haemorrhage within 24 hours after IAT and died. Two additional patients (5%) developed a local haematoma at the catheter insertion site in the femoral artery. They did not require transfusions or any other intervention.

## DISCUSSION

### Outcome

The course of basilar artery occlusion, with or without the use of antithrombotic agents, is usually poor.[2] Only a few cases with spontaneous favourable outcomes have been reported.[9] Intra-arterial thrombolysis improved survival and recanalisation compared with historical controls.[3] It may also improve the clinical outcome.[5] In this series 58% of the patients with basilar artery occlusion treated with IAT survived. In the series as a whole, 35% had a favourable clinical outcome. Most of previously published series using different thrombolytic agents, selection criteria, and treatment windows up to more than 48 hours after symptom onset reported a higher mortality and less favourable outcomes (table 5).[3–5 14–16] One potential reason for the moderately better results of this series could have been the earlier treatment after symptom onset and the higher dose of thrombolytic agents (mean urokinase dose 872 000 IU), which recanalised 80% of the occluded basilar arteries partially or completely.

### Predictors of favourable outcome

A low NIHSS score on admission in this series predicted a favourable outcome, consistent with observations of thrombolysis in the carotid territory.[17–19] In a univariate analysis, patients with quadriplegia ($p = 0.002$) or coma before thrombolysis ($p = 0.004$ ) were more likely to have a poor outcome than those without these signs. Decreased consciousness has been a predictor of unfavourable outcome in other series as well.[14] On multivariate analysis, however, coma and quadriplegia no longer had any predictive value because of the interaction with the NIHSS score. All patients with coma and quadriplegia have high NIHSS scores, and mostly such patients die or remain severely disabled. Exceptionally, comatose and quadriplegic patients recover, as did one patient in our study who had been comatose and quadriplegic for one hour before IAT and recovered to an mRS score of 2. In a series of Wijdicks *et al*, two patients with locked in syndrome lasting for several hours recovered fully after successful thrombolysis.[20] Thus in our opinion, patients with coma and quadriplegia should not be excluded from IAT, although a poor outcome is more likely.

In this study IAT was undertaken on average five and a half hours after symptom onset. The earlier the treatment was initiated, the more likely was the occluded artery to recanalise. However, this did not result in clinical benefit. There was only a non-significant trend towards better clinical

**Table 3** Clinical predictors of recanalisation

| Characteristic | Recanalisation TIMI 0 or 1 | TIMI 2 or 3 | p Value univariate | p Value multivariate | Total (n) |
|---|---|---|---|---|---|
| No of patients | 8 (20%) | 32 (80%) | | | 40 |
| Age ⩽60 | 5 (25%) | 15 (75%) | NS | | 20 |
| Age >60 | 3 (15%) | 17 (85%) | | | 20 |
| Sex | | | | | |
| Male | 2 (10%) | 20 (90%) | NS | | 22 |
| Female | 6 (33%) | 12 (67%) | | | 18 |
| NIHSSS on admission | | | | | |
| ⩽20 | 4 (22%) | 18 (78%) | NS | | 22 |
| >20 | 4 (29%) | 14 (71%) | | | 18 |
| Coma | | | | | |
| Yes | 5 (33%) | 10 (66%) | NS | | 15 |
| No | 3 (12%) | 22 (88%) | | | 25 |
| Quadriplegia | | | | | |
| Yes | 5 (31%) | 11 (69%) | NS | | 16 |
| No | 3 (12%) | 21 (88%) | | | 24 |
| Time to treatment | | | | | |
| ⩽6 hours | 3 (10%) | 26 (90%) | 0.011 | 0.003 | 29 |
| >6 hours | 5 (45%) | 6 (55%) | | | 11 |
| Stroke aetiology | | | | | |
| Cardioembolic | 1 (9%) | 11 (91%) | | | 12 |
| Unknown | 2 (20%) | 8 (80%) | NS | | 10 |
| Large artery disease | 3 (21%) | 11 (79%) | | | 14 |
| VA dissection | 2 (50%) | 2 (50%) | | | 4 |

NIHSSS, National Institutes of Heath stroke scale score; TIMI, thrombolysis in myocardial infarction grade; VA, vertebral artery.

**Table 4** Radiological predictors of recanalisation

| Characteristic | Recanalisation | | p Value univariate | p Value multivariate | Total (n) |
|---|---|---|---|---|---|
| | TIMI 0 or 1 | TIMI 2 or 3 | | | |
| Dense artery (n) | 8 (20%) | 32 (80%) | | | 40 |
| CT dense artery | | | | | |
| Yes | 3 (12%) | 23 (88%) | 0.022 | 0.007 | 26 |
| No | 5 (45%) | 6 (55%) | | | 11 |
| CT early signs of ischaemia | | | | | |
| Yes | 1 (10%) | 9 (90%) | NS | | 10 |
| No | 7 (26%) | 20 (74%) | | | 27 |
| Length of occlusion | | | | | |
| Short | 2 (12%) | 15 (88%) | NS | | 17 |
| Long | 6 (26%) | 17 (74%) | | | 23 |
| Localisation of occlusion | | | | | |
| Caudal | 5 (25%) | 15 (75%) | NS | | 20 |
| Mid-basilar | 3 (23%) | 10 (77%) | | | 13 |
| Distal | 0 (0%) | 7 (100%) | | | 7 |
| Collaterals | | | | | |
| Sufficient | 4 (19%) | 17 (81%) | NS | | 21 |
| Insufficient | 4 (27%) | 15 (73%) | | | 19 |

CT, computed tomography; TIMI, thrombolysis in myocardial infarction grade.

outcome in patients treated earlier. On the other hand, there are reports of patients with favourable outcomes who had been treated quite late after symptom onset.[4 14] These observations are most probably explained by collateral vessels which preserve the ischaemic tissue at risk of irreversible cell death for a prolonged period. In some series there was a significant association between collaterals and survival.[5 15] In this series, a non-significant trend supports this hypothesis as well. Forty eight per cent of patients with adequate collaterals versus 21% with inadequate collaterals had a favourable outcome. Possibly collaterals that are adequate to preserve tissue at risk for a prolonged period are not always visualised on arteriography, and other methods will be needed to identify patients with salvageable tissue. Pretreatment diffusion and perfusion MRI might help in deciding more accurately than arteriography which patients will benefit from recanalising treatment even after longer time windows.[21]

There was no correlation between age, sex, or vascular risk factors and outcome. A hyperdense basilar artery sign on the admission CT, early CT signs of ischaemia, localisation and length of the basilar artery occlusion, and the presence of collaterals failed to predict the clinical outcome.

## Recanalisation and predictors of recanalisation

Partial or complete recanalisation was achieved in 80% of the patients in this series and in 44% to 75% in other studies (table 5). In contrast to other series, a shorter delay to treatment rendered recanalisation more probable in our patients.[5] In addition, a hyperdense basilar artery sign on the initial CT increased the chances of recanalisation. Embolic clots in a previously unaffected artery may be more likely to appear as a hyperdense basilar artery sign on CT than a clot formed in a pre-existing high grade atherothrombotic basilar artery stenosis, and embolic occlusions might also be more easily recanalised than a thrombosis on an arteriosclerotic vessel wall. Such an interpretation is supported by the finding of other studies that cardioembolic occlusions had higher recanalisation rates than occlusions of other aetiology.[5 22] However, in our series there was no association between a hyperdense basilar artery sign on CT and a cardioembolic origin of the clot. This could partly reflect the large proportion of stroke of undetermined origin, because many patients died before they could undergo ancillary investigations such as echocardiography.

## Complications

Two symptomatic intracerebral haemorrhages occurred. Other studies have also reported low rates of symptomatic haemorrhagic complications in the vertebrobasilar territory. These findings and our results from retrospective analysis of patient series indicate that IAT for basilar artery occlusion is fairly safe when undertaken by an experienced stroke team.[3 5]

## Conclusions

The results of this study and previous series suggest that IAT may be beneficial for selected patients with basilar artery occlusion. A favourable clinical outcome in this study was more common in patients with low NIHSS score before treatment and when recanalisation was achieved. Initiation of IAT within six hours of symptom onset and hyperdense basilar artery sign on CT indicated a greater chance of

**Table 5** Review of published reports on recanalisation and outcome of patients with basilar artery occlusion treated with thrombolysis

| Source | No of patients (mode of treatment) | Maximum time window | Partial or complete recanalisation (n (%)) | Favourable outcome (n (%)), measured by: | Survival (n (%)) |
|---|---|---|---|---|---|
| Hacke[3] | 43 (ia) | >72 hours | 19 (44%) | 10 (23%) (no, minimal, or moderate deficit) | 14(33%) |
| Zeumer[4] | 28 (ia) | Not indicated (mean 8 hours) | 21 (75%) | 10 (35%) (able to work) | 18(64%) |
| Becker[14] | 12 (ia) | 48 hours | 9 (75%) | 3 (25%) (minimal deficits) | 3(25%) |
| Brandt[5] | 42 (ia)/9 (iv) | 48 hours | 26 (51%) | 10 (20%) (Barthel ⩾95) | 16(31%) |
| Cross[15] | 24 (ia) | 82 hours | Not indicated | 6 (25%) (mRS ⩽2) | 9(38%) |
| Huemer[16] | 16 (iv) | 7 hours | 10 (62%) | 3 (19%) (independence from constant support) | 5(31%) |
| Present study | 40 (ia) | 12 hours | 32 (80%) | 14 (35%) (mRS ⩽2) | 23(58%) |

Barthel, Barthel index of activities of daily living; ia, intra-arterial; iv, intravenous; MRS, modified Rankin scale score.

recanalisation. When patients are in coma, quadriplegic, or have already had prolonged symptoms, the decision about whether to attempt IAT or to withhold treatment can be difficult. In such patients, MRI with diffusion and perfusion imaging might help to identify those who are most likely to benefit from IAT.[21] In addition, other techniques and agents to treat basilar artery occlusion might result in better outcomes and need to be studied. These include combined intravenous and intra-arterial thrombolysis, mechanical thrombaspiration and fragmentation, and other antithrombotic agents such as abciximab or tirofiban (alone or in combination with thrombolytics), mechanical recanalisation including thrombus aspiration, primary angioplasty, and stenting of the basilar artery.[23–31]

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