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# Impact of diffusion-weighted MRI-measured initial cerebral infarction volume on clinical outcome in acute stroke patients with middle cerebral artery occlusion treated by thrombolysis

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**Abstract** *Introduction:* Magnetic resonance imaging (MRI) may help identify acute stroke patients with a higher potential benefit from thrombolytic therapy. The aim of our study was to assess the correlation between initial cerebral infarct (CI) volume (quantified on diffusion-weighted MRI) and the resulting clinical outcome in acute stroke patients with middle cerebral artery (MCA) ($M_{1–2}$ segment) occlusion detected on MRI angiography treated by intravenous/intraarterial thrombolysis. *Methods:* Initial infarct volume ($V_{DWI-I}$) was retrospectively compared with neurological deficit evaluated using the NIH stroke scale on admission and 24 h later, and with the 90-day clinical outcome assessed using the modified Rankin scale in a series of 25 consecutive CI patients. The relationship between infarct volume and neurological deficit severity was assessed and, following the establishment of the maximum $V_{DWI-I}$ still associated with a good clinical outcome, the patients were divided into two groups ($V_{DWI-I}$ ≤70 ml and >70 ml). *Results:* $V_{DWI-I}$ ranged from 0.7 to 321 ml. The 24-h clinical outcome improved significantly ($P$=0.0001) in 87% of patients with a $V_{DWI-I}$ ≤70 ml (group 1) and deteriorated significantly ($P$=0.0018) in all patients with a $V_{DWI-I}$ >70 ml (group 2). The 90-day mortality was 0% in group 1 and 71.5% in group 2. The 90-day clinical outcome was significantly better in group 1 than in group 2 ($P$=0.026). *Conclusion:* Clinical outcome could be predicted from initial infarct volume quantified by MRI-DWI in acute CI patients with MCA occlusion treated by intravenous/intraarterial thrombolysis. Patients with a $V_{DWI-I}$ ≤70 ml had a significantly better outcome.

**Keywords** Acute ischemic stroke · Middle cerebral artery occlusion · Diffusion-weighted sequences · Initial infarct volume · Thrombolysis

## Introduction

Even though computerized tomography (CT) is still considered to be the standard brain imaging modality in acute ischemic stroke [1], recent studies have suggested that magnetic resonance imaging (MRI) may help to identify patients with the most potential to benefit from thrombolytic therapy [2–8]. MRI using diffusion-weighted images (DWI) improves the ability to diagnose acute cerebral infarction (CI) in terms of both quantification of actual CI volume and accurate CI localization [9–18]. MRI can also detect significant cerebral arterial occlusion or severe stenosis [19, 20]. MRI allows the evaluation of cerebral collateral flow and also imaging of reversible ischemic changes [21–24]. Nevertheless, to date, few studies have evaluated the impact of these pretreatment MRI parameters on clinical outcome [6–8, 15, 25–27].

The aim of our study was to assess the influence of initial CI volume measured on MRI-DWI sequences on clinical outcome in acute stroke patients with middle cerebral artery (MCA) ($M_{1-2}$ segment) occlusion detected on MR angiography, who subsequently underwent intravenous/intraarterial thrombolysis (IVT/IAT). Initial CI volume ($V_{DWI-I}$) was retrospectively compared with neurological deficit evaluated using the NIH stroke scale (NIHSS) on admission and 24 h later, and with the 90-day clinical outcome using the modified Rankin scale (mRS) in a series of 25 consecutive patients.

The MRI protocol used was based on actual data in the literature and the published results of several recent studies [1, 3–5, 7, 14, 15, 28–33].

## Subjects and methods

A group of 25 consecutive acute ischemic stroke patients with MCA occlusion ($M_{1-2}$ segment) detected on MR angiography, who underwent IVT or IAT according to recent guidelines [34, 35] between January and September 2005, were retrospectively analyzed. The demographic and clinical characteristics of the patients are shown in Table 1.

On admission, blood pressure was measured, electrocardiogram was recorded, and blood samples were taken. Clinical status was evaluated using the NIHSS by a certified neurologist. An MRI examination followed immediately. After admission, patients were treated according to published guidelines [34, 35].

**Table 1** Patient demographic and clinical characteristics

| | |
|---|---|
| Males | 16 |
| Females | 9 |
| Age (mean±SD, years) | 59.1±13.9 |
| Baseline NIHSS (mean±SD) | 14.2±5.8 |
| Time from stroke onset to MRI examination (mean±SD, min) | 149±37 |
| Number of patients treated by IVT | 21 |
| Number of patients treated by IAT | 4 |

MRI was performed on a Magnetom Symphony 1.5-T Maestro Class (Siemens, Erlangen, Germany) with quantum gradients (syngo2004A) and a standard head coil (CP head array coil).

The MRI protocol included the following sequences: (1) localizer, (2) T2-turbo spin echo (TSE), (3) fluid-attenuated inversion recovery (FLAIR), (4) DWI, (5) 3D time of flight magnetic resonance angiography (TOF MRA). The total acquisition time (AT) was 11 min 28 s. Sequences 2–4 were applied to acquire data from the same set of slices (standard number of slices 19, slice thickness 5 mm, distance factor 30%). The standard slice orientation was oblique axial, approximately parallel to skull base in order to minimize susceptibility artifacts in echo-planar imaging (EPI) sequences.

The sequence parameters were as follows: *T2-TSE* TR/TE/ETL 4,000/99/9 ms, FOV 230×173 mm, matrix 256×256, AT 1 min 34 s; *FLAIR* 8,050/112/ETL 21/2 concatenation, FOV 230 mm, FOV phase 76.6%, matrix 256×151, AT 2 min 26 s. These sequences were used to assess hemorrhage and detect local demyelination changes including sites of ischemic demyelination.

The EPI-DWI sequence parameters were as follows: 3,200/94/EPI factor 128/3 averages, FOV 230×230 mm, matrix 128×128 with interpolation, TA 1 min 20 s. MRI data were acquired with three diffusion weightings: b=0, DWI b=500, and DWI b=1,000. The fourth type of image was an automatically created apparent diffusion coefficient (ADC) map. DWI traces show average local diffusivity in the brain tissue examined when b is 500 and 1,000. This sequence was applied to assess hemorrhage (b=0: T2*-EPI, susceptibility-sensitive sequence) and detect sites of reduced diffusion (DWI, b=500 and 1,000). The 3D-TOF MRA sequence parameters were as follows: 43/7.15, 3 slabs, 32 partitions/slab, slice thickness 1 mm, FOV 200×150 mm, matrix 512×192, AT 5:59 min. The images obtained—maximum intensity projection (MIP) and sublayers—would illustrate closure of the main arterial trunk of the circle of Willis or its branches.

Infarct volumes were measured on DWI trace images (b=1,000) and calculated as total hyperintense area in single slices multiplied by effective slice thickness [(actual slice thickness + distance factor)/interslice gap]. Follow-up MRI was performed in all patients after 24 h. Clinical outcome was evaluated 3 months after CI using the mRS. The relationship between the initial infarct volume and neurological deficit severity was assessed, defining a cut-off point for maximum $V_{DWI-I}$ still associated with a good clinical outcome. Several cut-off points of $V_{DWI-I}$ were subsequently tested with the aim of maximizing both sensitivity and specificity for a good clinical outcome (mRS 0–2).

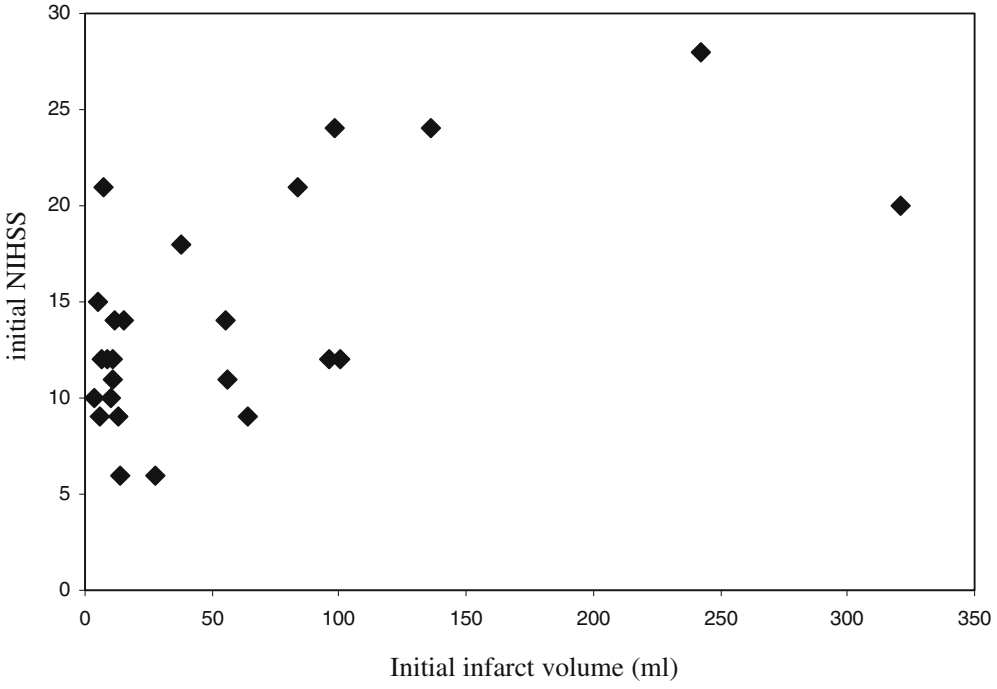



**Fig. 1** Relationship between initial infarct volume and neurological deficit on admission

Pearson's correlation analysis, an independent sample test, and the non-parametric Mann-Whitney test were used to assess the significance of differences.

## Results

The analysis included 25 patients with acute stroke. The mean delay between symptom onset and MRI was 149 min. The demographic and clinical baseline data are shown in Table 1. $V_{DWI-I}$ ranged from 1.3 ml to 321 ml, quantified from hyperintense lesions on DWI sequences.

The relationship between $V_{DWI-I}$ and neurological deficit on admission is demonstrated in Fig. 1, and clinical evolution after 24 h and 90 days is shown in Figs. 2 and 3, respectively. The correlation analysis demonstrated a strong positive correlation between $V_{DWI-I}$ and 24-h clinical outcome ($r$=0.74) and a moderate positive corre-

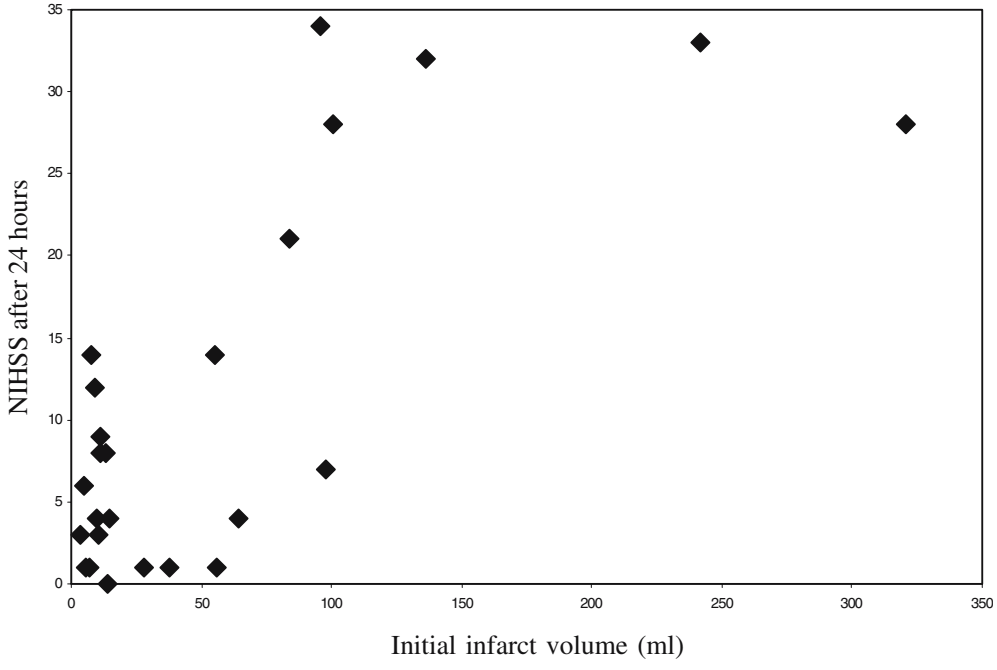



**Fig. 2** Relationship between initial infarct volume and 24-h clinical outcome

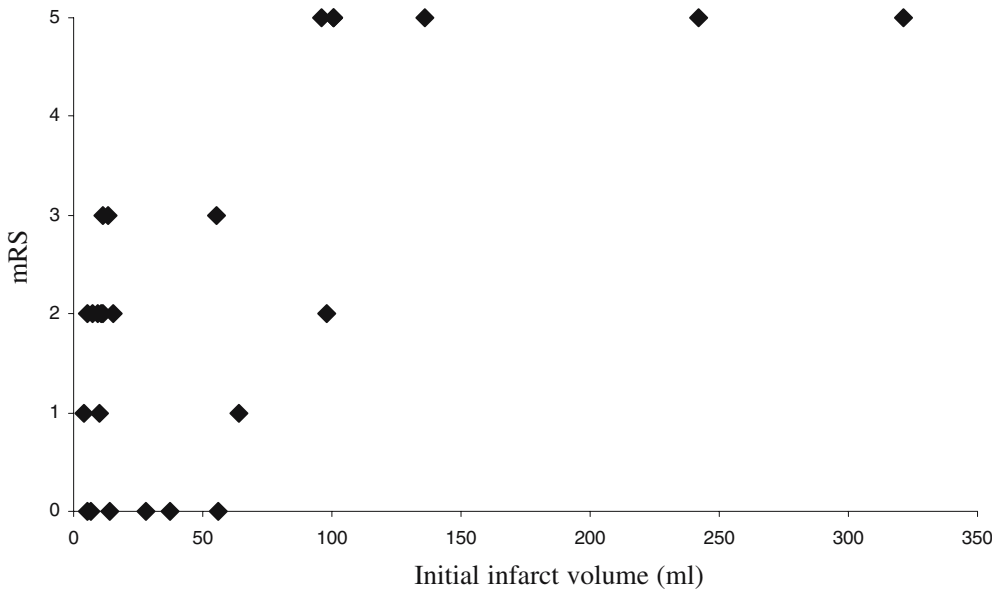


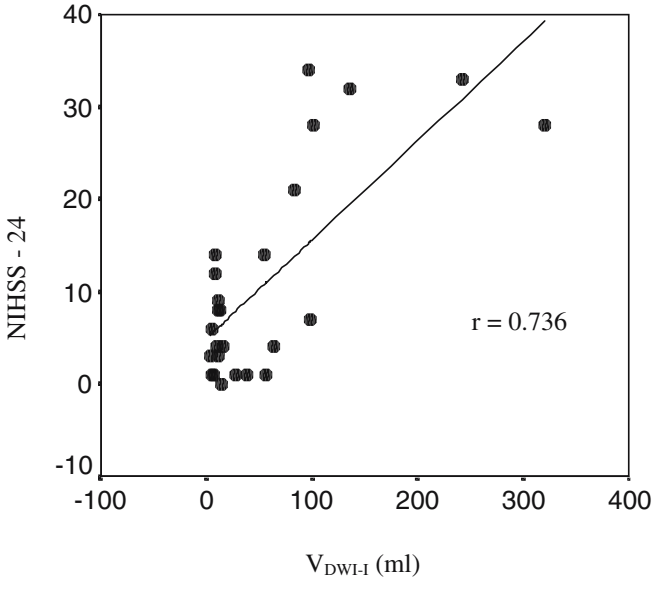

**Fig. 3** Relationship between initial infarct volume and 90-day clinical outcome

lation between $V_{DWI-I}$ and 90-day clinical outcome ($r$=0.67; Figs. 4 and 5).

The cut-off point of 70 ml was found to be the maximum $V_{DWI-I}$ still associated with a good clinical outcome and corresponding to the maximum sensitivity and specificity (80%) achieved. Figure 6 demonstrates this resulting cut-off point, on the basis of which the patients were divided into two groups ($V_{DWI-I}$ ≤70 ml and >70 ml; Table 2).

At 24 h (Fig. 7), patients with $V_{DWI-I}$ >70 ml had a significantly higher NIHSS score ($P$=0.0018).

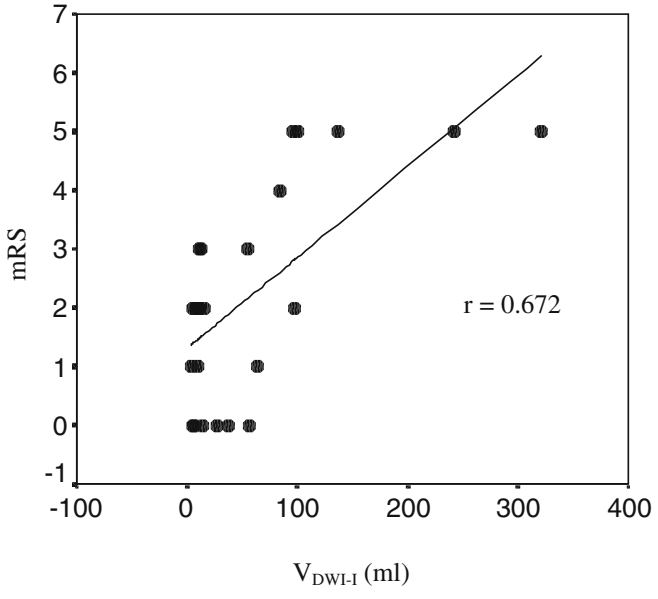
Intracranial hemorrhage was present at 24 h in one patient with $V_{DWI-I}$ ≤70 ml and in three patients with $V_{DWI-I}$ >70 ml (Table 2). There was a statistically significant difference in mRS score between patients with $V_{DWI-I}$ ≤70 ml and those with $V_{DWI-I}$ >70 ml ($P$=0.003, Fisher's exact test). The median mRS scores in the two groups are shown in




NIHSS-24 = NIHSS after 24 hours from stroke onset

$V_{DWI-I}$ = initial infarct volume (ml)

**Fig. 4** Pearson's correlation analysis of the correlation between initial infarct volume ($V_{DWI-I}$) and 24-h clinical outcome




mRS = modified Rankin Scale

$V_{DWI-I}$ = initial infarct volume (ml)

**Fig. 5** Pearson's correlation analysis of the correlation between initial infarct volume ($V_{DWI-I}$) and 90-day clinical outcome

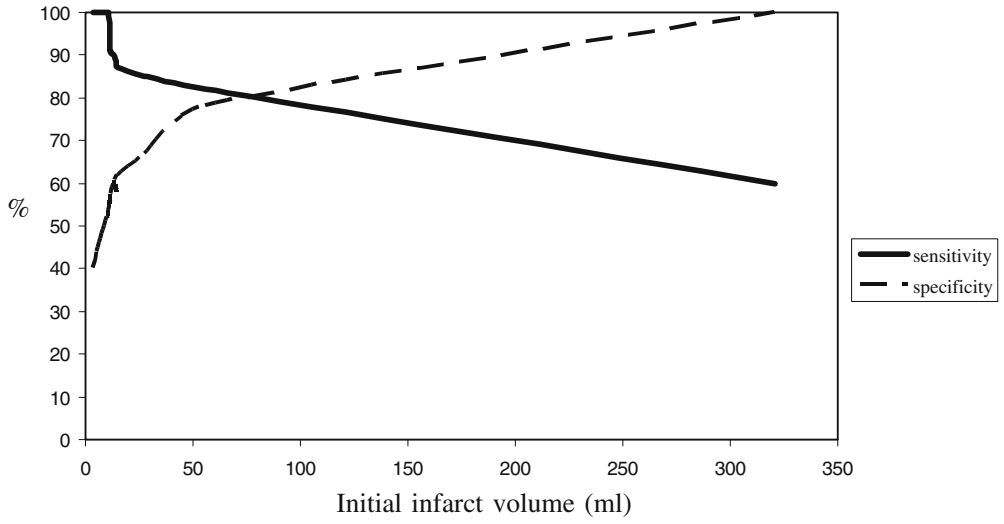



**Fig. 6** Sensitivity and specificity for good clinical outcome (mRS 0–2) in relation to initial infarct volume

Table 2. The independent samples test showed significantly higher mRS (3–5) in those with $V_{DWI-I}$ >70 ml than in those with $V_{DWI-I}$ ≤70 ml ($P$=0.026; Fig. 8). The significance of this difference was confirmed by the non-parametric Mann-Whitney test ($P$=0.003).

Mortality at 90 days was 71.5% in those with $V_{DWI-I}$ >70 ml and 0% in those with $V_{DWI-I}$ ≤70 ml. All these patients died during the first 7 days after thrombolysis; brain edema was the cause of death in all cases. Intracranial hemorrhage also occurred in three of these patients.

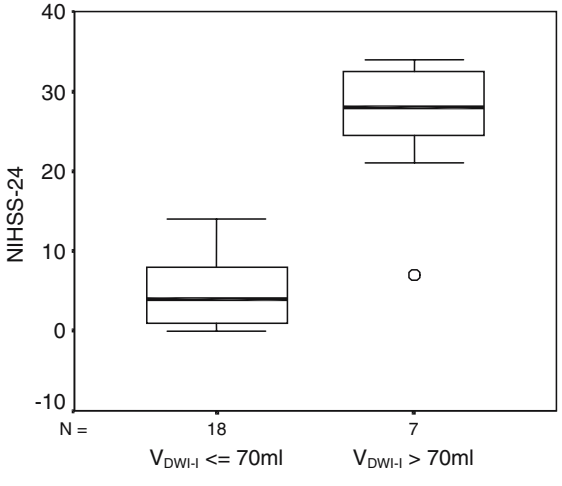
## Discussion

No direct relationship was found between the initial infarct volume measured on DWI and the neurological deficit on admission (Fig. 1). Two patients with the same severity of deficit and the same type of arterial occlusion (MCA, $M_{1-2}$ segment) could have $V_{DWI-I}$ differing by as much as tens of milliliters (Fig. 9). This difference was mainly caused by the actual individual state of cerebral collateral flow.

The actual infarct volume is considered to be an important factor in the decision to perform thrombolysis. Initial infarct volume is considered to be an independent predictor of subsequent spontaneous CI hemorrhagic transformation and also for intracerebral hemorrhage growth after thrombolysis [36]. Based on the results of this study, one may also predict eventual CI progression and the resulting clinical outcome from the initial infarct volume.

**Table 2** Demographic and clinical parameters of patient subgroups

| | Patient group | |
|---|---|---|
| | $V_{DWI-I}$ ≤70 ml | $V_{DWI-I}$ >70 ml |
| Number of patients | 18 | 7 |
| Age (mean±SD, years) | 56±11 | 61±9 |
| Baseline NIHSS | | |
| Mean±SD | 12.6±4.6 | 19.5±6.2 |
| Median | 16 | 21 |
| NIHSS after 24 h | | |
| Mean±SD | 5.2±4.6 | 26.2±9.6 |
| Median | 10 | 28 |
| mRS (day 90) | | |
| Mean±SD | 1.2±1.1 | 4.4±1.1 |
| Median | 2 | 5 |
| Intracranial hemorrhage | | |
| Incidence | 1 | 3 |
| Symptomatic | 0 | 3 |




$V_{DWI-I}$ = initial infarct volume (ml)

NIHSS-24 = clinical outcome after 24 hours from stroke onset

**Fig. 7** Correlation between initial infarct volume and 24-h clinical outcome

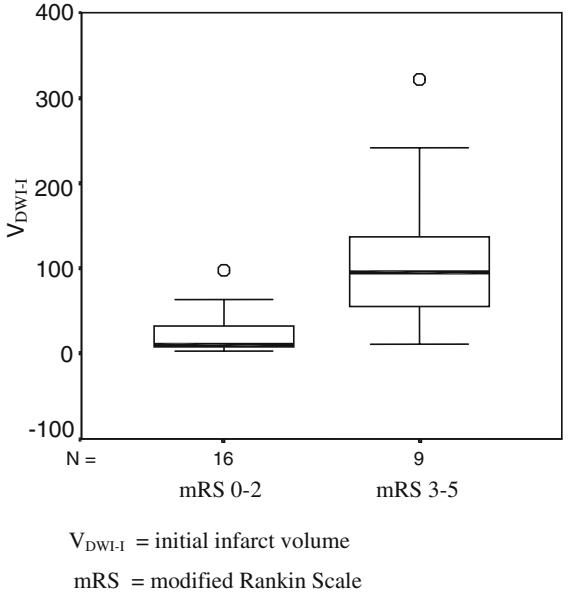



**Fig. 8** Correlation between initial infarct volume ($V_{DWI-I}$) and 90-day clinical outcome

Davalos et al. retrospectively found infarct volume progression in patients with $V_{DWI-I}$ ≤25 ml and initial NIHSS score >8, on average about 68 ml in a group of 166 acute CI patients, examined within 12 h of stroke onset. This progression occurred during the first 6 h in one of three of these patients—mainly in those who did not receive thrombolysis [8].

In the group of patients with $V_{DWI-I}$ ≤70 ml and initial NIHSS >8, infarct volume progression was found in nine patients with a mean volume of progression of 27.5 ml.

Derex et al. observed a significantly worse clinical outcome in association with a larger pretreatment DWI infarct volume in 49 stroke patients treated by IVT. They consider the initial DWI lesion volume and the recanalization to be independent factors in determining the final infarct size [27].

Oppenheim et al. consider quantification of the initial CI volume to be an accurate method for assessing the risk of malignant infarct progression in patients with MCA occlusion. They identified a threshold volume of 145 ml in their group of 28 patients. No patient with a CI volume below this value had a malignant MCA infarction in spite of a high NIHSS score on admission (16.5±4) [15]. In our study, 71.5% of the patients with MCA occlusion and with $V_{DWI-I}$ >70 ml died within 7 days from CI onset (Fig. 4).

Although perfusion-weighted imaging (PWI) is performed as a standard part of the MRI protocol in many

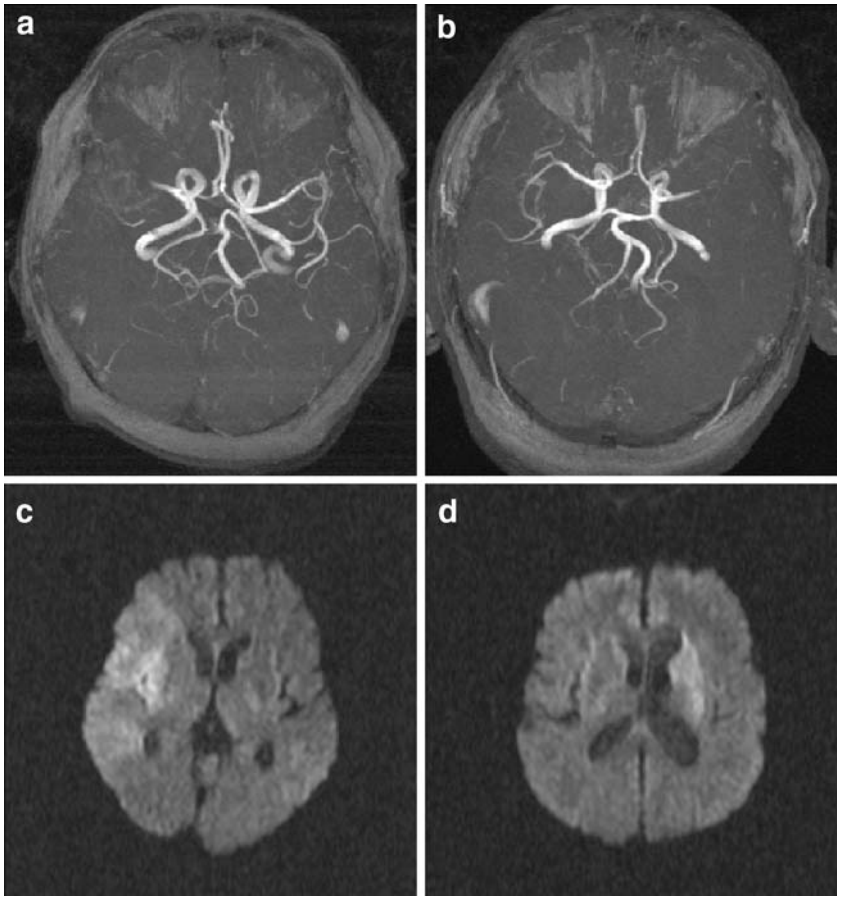



**Fig. 9** Different initial infarct volumes in two patients with MCA occlusion on admission. **a** Right MCA occlusion (3D TOF MRA); **b** left MCA occlusion (3D TOF MRA); **c** right MCA infarct on DWI (volume 136 ml); **d** left MCA infarct on DWI (volume 11.3 ml)

stroke centers and has been used in previously reported studies, we did not use it in our MRI stroke protocol and therefore also did not subsequently interpret the ischemic penumbra according to the PWI/DWI mismatch for several reasons. We believe that PWI/DWI mismatch is an inaccurate approximation of the real ischemic penumbra [37–39]. Furthermore, quantification of PWI lesion volume generally uses only qualitative indices and quantitative calculations are very time-consuming and use controversial mathematical models [40]. Definitive infarct volume is usually far smaller than the initial PWI area [37–39]. For example, Heiss et al. compared the size of the PWI/DWI mismatch area with the area of reduced cerebral blood flow and increased oxygen extraction (over 150%) using positron emission tomography (PET) in acute stroke patients. Despite a good correlation between PWI and PET in the detection of perfusion defect, mismatch volume did not correspond to the volume of the area with increased oxygen extraction on PET scans. Thus PWI/DWI mismatch may not be a reliable correlate of ischemic penumbra [41].

Several methodological limitations of the presented study should be mentioned. The first one is the relatively small number of patients examined. This was a result of the generally low number of patients fulfilling the strict time criteria for thrombolytic therapy, event with MCA occlusion. Secondly, quantification of infarct volume was performed manually, because no semiautomatic quantification software was available. Therefore the quantification could have been affected by subjective operator error. Finally, interpretation of the results may have been limited by nonhomogeneity of the group: the patients had received two types of recanalization therapy, but both with the same effect on the occluded artery.

## Conclusion

Quantification of initial CI volume can be considered important for an accurate and safe evaluation of the indications for thrombolytic therapy in acute stroke patients.

The MRI-DWI quantification of initial CI volume was able to predict clinical outcome in patients with acute CI with MCA occlusion treated by IVT or IAT.

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Study protocol was in compliance with the Declaration of Helsinki (1964) and was approved by the Ethical Committee of the University Hospital, Olomouc, Czech Republic.

**Conflict of interest statement** We declare that we have no conflict of interest.

## References

1. Jansen O, Knauth M, Sartor K (1999) Advances in clinical neuroradiology. Akta Neurol 26:1–7
2. Rotta J (1997) Should thrombolytic therapy be the first-line treatment for acute ischemic stroke? N Engl J Med 337:1309–1310
3. Schellinger PD, Jansen O, Fiebach JB, Pohlers O, Ryssel H, Heiland S, Steiner T, Hacke W, Sartor K (2000) Feasibility and practicality of MR imaging of stroke in the management of hyperacute cerebral ischemia. AJNR Am J Neuroradiol 21:1184–1189
4. Fiebach JB, Jansen O, Schellinger PD, Knauth M, Hartmann M, Heiland S, Ryssel H, Pohlers O, Hacke W, Sartor K (2001) Comparison of CT with diffusion-weighted MRI in patients with hyperacute stroke. Neuroradiology 43:628–632
5. Schellinger PD, Fiebach JB, Jansen O, Ringleb PA, Mohr A, Steiner T, Heiland S, Schwab S, Pohlers O, Ryssel H, Orakcioglu B, Sartor K, Hacke W (2001) Stroke magnetic resonance imaging within 6 hours after onset of hyperacute cerebral ischemia. Ann Neurol 49:460–469
6. Parsons MW, Barber PA, Chalk J, Darby DG, Rose S, Desmond PM, Gerraty RP, Tress BM, Wright PM, Donnan GA, Davis SM (2002) Diffusion and perfusion-weighted MRI response on thrombolysis in stroke. Ann Neurol 51:28–37
7. Röther J, Schellinger PD, Gass A, Sieber M, Villringer A, Fiebach JB, Fiehler J, Jansen O, Kucinski T, Schoder V, Szabo K, Junge-Hülsing GJ, Hennerici M, Zeumer H, Sartor K, Weiller C, Hacke W (2002) Effect of intravenous thrombolysis on MRI parameters and functional outcome in acute stroke <6 h. Stroke 33: 2438–2445
8. Davalos A, Blanco M, Pedraza S, Leira B, Castellanos M, Pumar JM, Silva Y, Serena J, Castillo J (2004) The Clinical-DWI mismatch: a new diagnostic approach to the brain tissue risk of infarction. Neurology 62:2187–2192
9. Warach S, Chien D, Li W, Ronthal M, Edelman RR (1992) Fast magnetic resonance diffusion-weighted imaging of acute human stroke. Neurology 42:1717–1723
10. Baird AE, Warach S (1998) Magnetic resonance imaging of acute stroke. J Cereb Blood Flow Metab 18:583–609
11. Lovblad KO, Laubach HJ, Baird AE, Curtin F, Schlaug G, Edelman RR, Warach S (1998) Clinical experience with diffusion-weighted MR in patients with acute stroke. AJNR Am J Neuroradiol 19:1061–1066

12. Ay H, Buonanno FS, Rodorf G, Schaefer PW, Schwamm LH, Wu O, Gonzalez RG, Yamada K, Sorensen GA, Koroshetz WJ (1999) Normal diffusion-weighted MRI during stroke-like deficits. Neurology 52:1784–1792
13. Neumann-Haefelin T, Moseley ME, Albers GW (2000) New magnetic resonance imaging methods for cerebrovascular disease: emerging clinical applications. Ann Neurol 47:559–570
14. Oppenheim C, Logak M, Dormont D, Lehericy S, Manai R, Samson Y, Marsault C, Rancurel G (2000) Diagnosis of acute ischemic stroke with fluid attenuated inversion recovery and diffusion-weighted MR sequences. Neuroradiology 42:602–607
15. Oppenheim C, Samson Y, Manai R, Laam T, Vandamme X, Crozier S, Srour A, Cornu P, Dormont D, Rancurel G, Marsault C (2000) Prediction of malignant middle cerebral artery infarction by diffusion-weighted imaging. Stroke 31:2175–2181
16. Jansen O, Brückmann H (2002) Ischemic brain diseases. In: Sartor K (ed) Diagnostic and interventional neuroradiology. Thieme, Stuttgart New York, p 148
17. Mullins ME, Lev MH, Schellingerhout D, Koroshetz WJ, Gonzales RG (2002) Influence of availability of clinical history on detection of early stroke using unenhanced CT and diffusion-weighted MR imaging. AJR Am J Roentgenol 179:223–228
18. Mullins ME, Schaefer PW, Sorensen AG, Halpern EF, Ay H, He J, Koroshetz WJ, Gonzalez RG (2002) CT and conventional and diffusion weighted MR Imaging in acute stroke: study in 961 patients at presentation to the emergency department. Radiology 224:353–360
19. Warach S, Li W, Ronthal M, Edelman RR (1992) Acute cerebral ischemia: evaluation with dynamic contrast-enhanced MR imaging and MR angiography. Radiology 182:41–47
20. Evans AJ, Richardson DB, Tien R, Mac Fall JR, Hedlund LW, Heinz ER, Boyko O, Sostman HD (1993) Post-stenotic signal loss in MR angiography; effects of echo time, flow compensation and fractional echo. AJNR Am J Neuroradiol 14:721–729
21. Schellinger PD, Jansen O, Fiebach JB, Heiland S, Steiner T, Schwab S, Pohlers O, Ryssel H, Sartor K, Hacke W (2000) Monitoring intravenous recombinant tissue plasminogen activator thrombolysis for acute ischemic stroke with diffusion and perfusion MRI. Stroke 31:1318–1328
22. Grant PE, He HJ, Halpern E, Wu O, Schaefer PW, Schwamm LH, Budzik RF, Sorensen AG, Koroshetz WJ, Gonzalez RG (2001) Frequency and clinical context of decreased apparent diffusion coefficient reversal in the human brain. Radiology 221:43–50
23. Crisostomo RA, Garcia MM, Tong DC (2003) Detection of diffusion-weighted MRI abnormalities in patients with transient ischemic attack. Correlation with clinical characteristics. Stroke 34:932–937
24. Schaefer PW, Hassankhani A, Pulman C, Sorensen G, Schwamm L, Koroshetz W, Gonzales GR (2004) Characterization and evolution of diffusion MR imaging abnormalities in stroke patients undergoing intra-arterial thrombolysis. AJNR Am J Neuroradiol 25:951–957
25. Kidwell CS, Saver J, Mattiello J, Starkman S, Vinuela F, Duckwiler G, Gobin YP, Jahan R, Vespa P, Kalafut M, Alger JR (2000) Thrombolytic reversal of acute human cerebral ischemic injury shown by diffusion/perfusion magnetic resonance imaging. Ann Neurol 47:462–469
26. Nighoghossian N, Hermier M, Adeleine P, Derex L, Durgor JF, Philippeau F, Ylmaz H, Honnorat J, Dardel P, Berthezene Y, Froment JC, Trouillas P (2003) Baseline magnetic resonance imaging parameters and stroke outcome in patients treated by intravenous tissue plasminogen activator. Stroke 34:458–463
27. Derex L, Nighoghossian N, Hermier M, Adeleine P, Berthezène, Philippeau F, Honnorat J, Froment J-C, Trouillas P (2004) Influence of pretreatment MRI parameters on clinical outcome, recanalization and infarct size in 49 stroke patients treated by intravenous tissue plasminogen activator. J Neurol Sci 225:3–9
28. Albers GW (1999) Expanding the window for thrombolytic therapy in acute stroke. The potential role of acute MRI for patient selection. Stroke 30:2230–2237
29. Barber PA, Davis SM, Darby DG, Desmond PM, Gerraty RP, Yang Q, Jolley D, Donnan GA, Tress BM (1999) Absent middle cerebral artery flow predict the presence and evolution of the ischemic penumbra. Neurology 52:1125–1132
30. Dugar N, Hoggard N, Wilkinson ID, Griffiths PD (2001) MR Imaging in acute stroke. Imaging Decis 5:2–19
31. Samson Y, Crozier S, Deltour S, Obadia M, Manai R, Oppenheim C, Marro B, Dormont D, Marsault C, Rancurel G (2002) L'IRM en urgence avant la thrombolyse (abstract). Rev Neurol Paris 158:1S20
32. Fiebach JB, Schellinger PD, Sartor K, Heiland S, Warach S, Hacke W (2003) Stroke MRI. Steinkopff Verlag, Darmstadt, pp 54–61
33. Schellinger PD, Fiebach JB, Hacke W (2003) Imaging-based decision making in thrombolytic therapy for ischemic stroke. Stroke 34:575–583
34. Hacke W, Kaste M, Bogousslavsky J, Brainin M, Chamorro A, Lees K, Leys D, Kwiecinski H, Toni P, Langhorne P, Diener C, Hennerici M, Ferro J, Sivenius J, Gunnar N, Bath P, Olsen TS, Gugging M, European Stroke Initiative Executive Committee and the EUSI Writing Committee (2003) European Stroke Initiative Recommendations for Stroke Management – update 2003. Cerebrovasc Dis 16:311–337
35. Highasida RT, Furlan AJ (2003) Trial design and reporting standards for intra-arterial cerebral thrombolysis for acute ischemic stroke. Stroke 34: 109–137
36. Selim M, Fink JN, Kumar S, Caplan LR, Horkan C, Chen Y, Linfante I, Schlaug G (2002) Predictors of hemorrhagic transformation after intravenous recombinant tissue plasminogen activator. Stroke 33:2047–2052
37. Barber PA, Darby DG, Desmond PM, Yang Q, Gerraty RP, Jolley D, Donnan GA, Tress BM, Davis SM (1998) Prediction of stroke outcome with echoplanar perfusion- and diffusion-weighted MRI. Neurology 51:418–426
38. Neumann-Haefelin T, Witsack HJ, Wenserski F, Siebler M, Seitz RJ, Modder U, Freund HJ (1999) Diffusion and perfusion-weighted MRI: the DWI/PWI mismatch region in acute stroke. Stroke 30:1591–1597
39. Grandin CB, Duprez TP, Smith AM, Mataigne F, Peeters A, Oppenheim C, Cosnard G (2001) Usefulness magnetic resonance-derived quantitative measurements of cerebral blood flow and volume in prediction of infarct growth in hyperacute stroke. Stroke 32: 1147–1153
40. Sorensen AG, Copen WA, Ostergaard L, Buomanno FS, Gonzales RG, Rordorf G, Rosen BR, Schwamm LH, Weisskoff RM, Koroshetz WJ (1999) Hyperacute stroke: simultaneous measurement of relative cerebral blood volume, relative cerebral blood flow, and mean tissue transit time. Radiology 210:519–527
41. Heiss WD, Sobesky J, Hesselmann V (2004) Identifying thresholds for penumbra and irreversible tissue damage. Stroke 35:2671–2674