Thalamic Diaschisis in Acute Ischemic Stroke
Occurrence, Perfusion Characteristics, and Impact on Outcome

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Background and Purpose—Ipsilateral thalamic diaschisis (ITD) describes the reduction of thalamic function, metabolism, and perfusion resulting from a distant lesion of the ipsilateral hemisphere. Our aim was to evaluate the perfusion characteristics and clinical impact of ITD in acute middle cerebral artery stroke, which does not directly affect the thalamus.

Methods—One hundred twenty-four patients with middle cerebral artery infarction were selected from a prospectively acquired cohort of 1644 patients who underwent multiparametric computed tomography (CT), including CT perfusion for suspected stroke. Two blinded readers evaluated the occurrence of ITD, defined as ipsilateral thalamic hypoperfusion present on ≥2 CT perfusion maps. Perfusion alterations were defined according to the Alberta Stroke Program Early CT Score regions. Final infarction volume and subacute complications were assessed on follow-up imaging. Clinical outcome was quantified using the modified Rankin Scale. Multivariable linear and ordinal logistic regression analysis were applied to identify independent associations.

Results—ITD was present in 25/124 subjects (20.2%, ITD+). In ITD+ subjects, perfusion of the caudate nucleus, internal capsule, and lentiform nucleus was more frequently affected than in ITD− patients (each with $P<0.001$). In the ITD+ group, larger cerebral blood flow ($P=0.002$) and cerebral blood volume ($P<0.001$) deficit volumes, as well as smaller cerebral blood flow–cerebral blood volume mismatch ($P=0.021$) were observed. There was no independent association of ITD with final infarction volume or clinical outcome at discharge in treatment subgroups (each with $P>0.05$). ITD had no influence on the development of subacute stroke complications.

Conclusions—ITD in the form of thalamic hypoperfusion is a frequent CT perfusion finding in the acute phase in middle cerebral artery stroke patients with marked involvement of subcortical areas. ITD does not result in thalamic infarction and had no independent impact on patient outcome. Notably, ITD was misclassified as part of the ischemic core by automated software, which might affect patient selection in CT perfusion–based trials. **(*Stroke*. 2018;49:931-937. DOI: 10.1161/STROKEAHA.118.020698.)**

Key Words: acute stroke ■ brain ischemia ■ cerebral blood flow ■ perfusion ■ thalamus

The thalamus comprises a crucial hub for connections throughout the brain via thalamocortical and corticothalamic pathways.[1] As the thalamus' blood supply originates mainly from perforating arteries of the posterior cerebral artery, relevant involvement in stroke because of middle cerebral artery (MCA) occlusion is not expected.[2,3] However, decreased metabolism and perfusion of the thalamus has been described in subacute[4,5] and chronic[6] stages of ipsilateral MCA stroke with preserved vertebrobasilar blood supply.

This peculiar finding can be referred to as a Diaschisis phenomenon (from Greek διάσχισις, shocked throughout), which generally describes the reduction of function, metabolism, and perfusion in brain areas distant to a cerebral lesion. The term was first used by von Monakow in 1914 and introduced the idea of remote influence between spatially separated cerebral structures.[7] In vivo occurrence of diaschisis was first shown using positron emission tomography imaging in a population of patients with chronic supratentorial stroke, featuring hypometabolism of the contralateral cerebellar hemisphere (crossed cerebellar diaschisis).[8] In the following years, related phenomena like thalamocortical,[9] transhemispheric,[10] and the already mentioned ipsilateral thalamic diaschisis (ITD)[4] were described in different diseases and animal models.

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In subacute and chronic stages after MCA stroke, different studies reported a considerable incidence of ITD from 43% to 86%.[4–6] Regarding the course of ITD, there is a lack of longitudinal studies. Given the prior longitudinal studies on the course of crossed cerebellar diaschisis, it could be assumed that ITD after stroke is also potentially reversible.[4–6,11] Furthermore, it is unclear if there are long-term neurological effects of ITD. In the chronic phase of stroke, some studies report a similar clinical course of patients with or without signs of ITD, whereas others report reduced motor recovery associated with metabolic deficit of the ipsilesional thalamus.[6,12]

In contrast to the formerly used positron emission tomography and single photon emission tomography imaging, the technique of computed tomography (CT) perfusion (CTP) enables to study diaschisis phenomena in the pretherapeutic emergency setting of acute stroke. For acute MCA infarction, a recent study reported thalamic perfusion alterations.[13] As hypoperfusion of the thalamus might also indicate ischemic involvement of the posterior circulation, it poses a potential source of error in the assessment of CTP in acute stroke. As new trials tailor their inclusion criteria closely based on ischemic core volume assessment,[14,15] ITD could also distort the results as a form of nonischemic hypoperfusion.

Therefore, our study aimed to determine occurrence and perfusion characteristics of ITD in acute MCA stroke patients by analyzing CTP examinations. Further, we addressed the influence of acute ITD on morphological and clinical outcome.

Material and Methods

The data that support the findings of this study are available from the corresponding author on reasonable request.

Study Design and Population

This study was approved by the institutional review board of the LMU Munich according to the Declaration of Helsinki of 2013; requirement for informed consent was waived. Subjects were selected from a prospectively acquired cohort of 1644 patients who underwent multiparametric CT, including CTP for suspected stroke.

Inclusion criteria were as follows:

1. Acute MCA ischemia caused by internal carotid artery, M1, or M2 MCA occlusion;
2. Follow-up-confirmed infarction;
3. No vascular pathology of the vertebrobasilar system;
4. Absence of prior ischemic infarcts; and
5. Initial stroke assessment including adequate quality of CTP.

Out of the initial 1644 patients, 303 patients had an internal carotid artery, M1, or M2 MCA occlusion with follow-up-confirmed infarction. Out of these 303 patients, we excluded 89 with vascular abnormalities of the posterior circulation, 10 with acute posterior circulation infarction on follow-up imaging, 53 patients with nondiagnostic or incomplete CTP datasets, and 27 patients with signs of prior ischemic infarctions. This resulted in a total of 124 valid data sets for further analysis. A flow chart of patient selection is illustrated in Figure I in the online-only Data Supplement.

Image Analysis

Detailed methods on CT protocols are provided in the online-only Data Supplement. Spatial characteristics of the ischemic changes were evaluated according to the Alberta Stroke Program Early CT Score regions on cerebral blood flow, cerebral blood volume (CBV), mean transit time, and time to drain maps. Alberta Stroke Program Early CT Score regions include caudate nucleus (C), lentiform nucleus (L), internal capsule (IC), insula (I), anterior MCA cortex (M1), MCA cortex lateral to insular ribbon (M2), posterior MCA cortex (M3), and M4, M5, and M6 cortices which are located superior to M1–M3.[16] ITD was defined as thalamic hypoperfusion as compared with the contralateral side on ≥2 parametric CTP maps, which were assessed by 2 blinded readers (radiology attending with 10 years and radiology resident with 4 years of experience in stroke imaging). Inter-reader agreement was determined by Cohen's κ. In case of disagreement, consensus was reached in a separate session.

To address anomalies of thalamic blood supply, we analyzed CT angiography data for the presence of a persisting fetal posterior cerebral artery.[17] Initial CTP-deficit volume and final infarction volume were assessed using a segmentation algorithm as described before.[13,18] Relative infarction growth was defined as [posttreatment final infarction volume−pretreatment ischemic core volume based on CBV]/[pretreatment total ischemic volume based on cerebral blood flow−pretreatment ischemic core volume based on CBV] in accordance with previous studies.[19,20] Subacute stroke complications were assessed according to the ECASS (European Cooperative Acute Stroke Study). Space-occupying edema was considered present in case of a developing midline shift ≥5 mm on follow-up imaging.[21]

Clinical Outcome Data

Patients of the study population were treated using intravenous thrombolysis or endovascular therapy where applicable. Clinical outcome measures included National Institutes of Health Stroke Scale on admission as well as modified Rankin Scale (mRS) on admission, at discharge, and 90 days after stroke. Neurological deficits prior to the stroke were assessed as premorbid mRS. Patients with insufficient records or premorbid mRS >1, second stroke event, or death to other cause within 90 days were excluded from the clinical outcome analysis (see Figure I in the online-only Data Supplement).

Statistical Analysis

SPSS Statistics 24 (IBM, Armonk/NY) was used for statistical analysis. Normal distribution was evaluated by Kolmogorow–Smirnow test. For non-normal distributed data, Chi-squared test or Fisher exact test was applied for categorical and Mann–Whitney *U* test for continuous values. Independent samples median test was applied for ordinal data. Multiple testing was corrected using Bonferroni's method. Analysis of predictive factors included multivariable linear regression for morphological outcome. Analyses for subacute stroke complications and clinical outcome as dependent variables were performed using binary logistic and ordinal logistic regression. Multicollinearity of independent variables was tested using the variance inflation factor to avoid overfitting of the regression models. Statistical significance was defined for *P* values lower than 0.05.

Results

Patient Characteristics and Inter-Reader Agreement

A total of 124 patients fulfilled the inclusion criteria and were included in our retrospective analysis. Classification for ITD resulted in 99 (79.8%) ITD negative (ITD−) and 25 (20.2%) ITD positive (ITD+) patients. Representative patient examples are shown in Figure 1. Cohen's κ of 0.720 showed substantial inter-reader agreement. ITD+ patients had larger cerebral blood flow (P=0.002) and CBV (P<0.001) deficit volumes and smaller relative cerebral blood flow–CBV mismatch compared with those in the ITD− group (P=0.021). Intravenous thrombolysis treatment was more frequent in the ITD+ group (84.0% versus 60.6%; P=0.028). Age, sex, time from symptom onset, National Institutes of Health Stroke Scale on admission, Alberta Stroke Program Early CT Score, final infarction volume, stroke pathogenesis, occlusion location, cardiovascular risk factors, and the presence of fetal

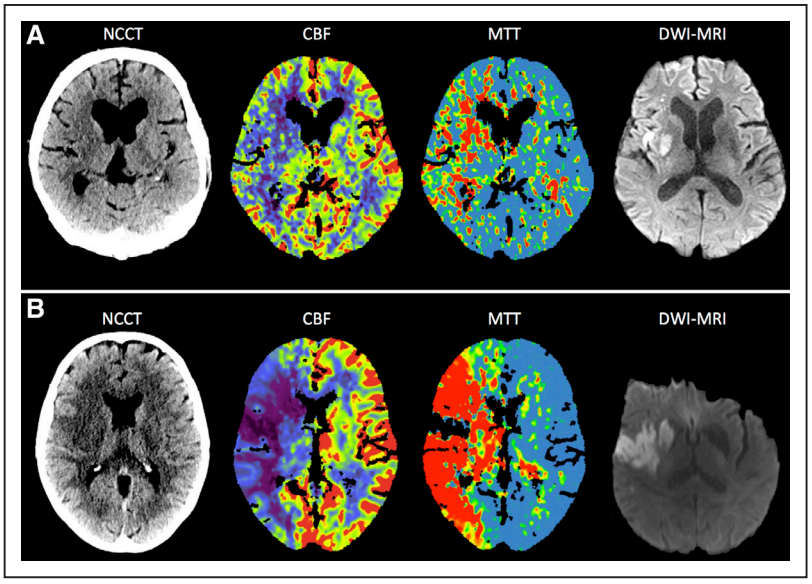

Figure 1. Examples of ITD− (**A**) and ITD+ (**B**) stroke patients. **A**, Seventy-nine-year-old male patient with an acute right-sided ICA occlusion. No thalamic hypoperfusion on CBF and MTT maps or thalamic infarction on follow-up DWI-MRI was detected (admission mRS, 5; discharge mRS, 0 after intravenous thrombolysis; mRS after 90 days, 0). **B**, Seventy-three-year-old female patient with an acute right-sided ICA and M1 occlusion. Detectable hypoperfusion of the thalamus on CBF and MTT maps without thalamic infarction on follow-up DWI-MRI, consistent with ITD (admission mRS, 5; discharge mRS, 4 after endovascular treatment; mRS after 90 days, 1). CBF indicates cerebral blood flow; DWI-MRI, diffusion-weighted imaging–magnetic resonance imaging; ICA, internal carotid artery; ITD, ipsilateral thalamic diaschisis; mRS, modified Rankin Scale; MTT, mean transit time; and NCCT, noncontrast computed tomography.

posterior cerebral artery did not differ significantly between ITD+ and ITD− patients. Also, we found no variants with missing P1 segment in ITD+ patients. In the ITD+ group, higher discharge mRS values were observed compared with the ITD− group (median: 5 versus 4; P=0.049). Detailed characteristics of ITD+ and ITD− patients are shown in Table 1 (additional patient data are given in Table I in the online-only Data Supplement).

Association of ITD With Affected MCA Regions According to ASPECTS

Chi-square test considering all included patients revealed higher frequencies of CTP deficit for M1, M6, C, IC, and L, as well as higher occurrence of crossed cerebellar diaschisis (each with P<0.05) in the ITD+ group. After correction for multiple comparisons according to Bonferroni's method, C (30.6% for ITD− versus 68.0% for ITD+ cases), IC (26.5% for ITD− versus 70.8% for ITD+ cases), and L (34.3% for ITD− versus 76.0% for ITD+ cases) remained significant (each with P<0.001). Details on the spatial characteristics are presented in Table 2.

Association of ITD With Morphologic and Clinical Outcome

Regarding morphological outcome, linear multivariable regression analysis in the subgroups of patients treated with intravenous thrombolysis and endovascular therapy or with intravenous thrombolysis alone did not show significant associations of ITD with final infarction volume or relative infarction growth (each with P>0.05). Regarding clinical outcome, ordinal logistic regression analysis in these treatment subgroups yielded no significant association of ITD with

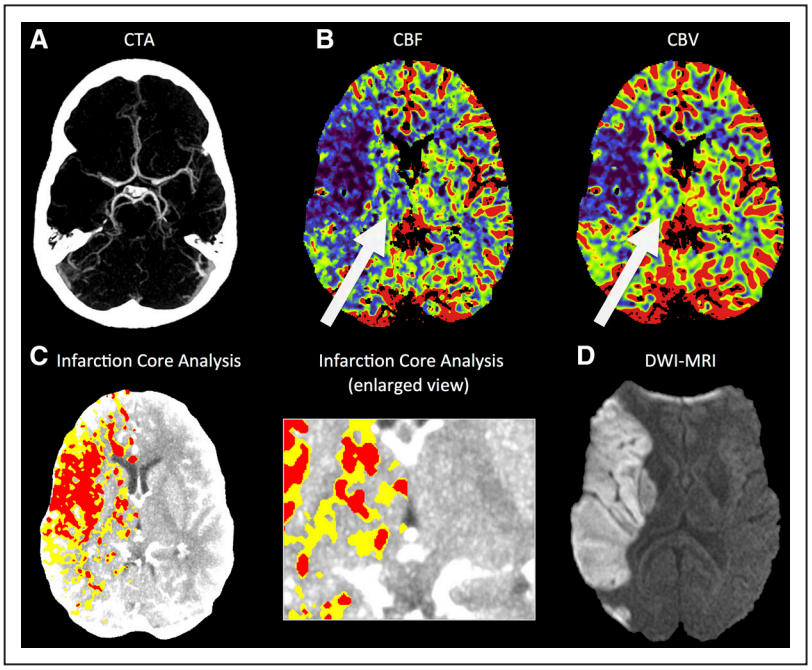

Figure 2. Misinterpretation of ITD by automated infarction core analysis in a 38-year-old male patient with acute right MCA occlusion. **A**, Complete occlusion of the right M1 segment as seen on CTA imaging. No occlusion of the posterior circulation was evident. **B**, Hypoperfusion of the MCA territory on CBF and CBV maps as well as hypoperfusion of the ipsilateral thalamus consistent with ITD (white arrows). **C**, Automated CTP analysis of infarction core was performed. Yellow areas indicate reduced perfusion. Red areas indicate infarction core with reduced CBF and CBV. Note that red areas in the right thalamus resemble misinterpretation of ITD as infarction core. **D**, Follow-up DWI-MRI indicates final infarction after intravenous thrombolysis (IVT) and successful EVT. No damage to the thalamus is seen. CBF, cerebral blood flow; CBV, cerebral blood volume; CTA, computed tomography angiography; CTP, computed tomography perfusion; DWI, diffusion-weighted imaging; EVT, endovascular therapy; MRI, magnetic resonance imaging; and MCA, middle cerebral artery.

Table 1. Characteristics of ITD− and ITD+ Acute Ischemic Stroke Patients

	Overall (N=124)	ITD− (N=99)	ITD+ (N=25)	*P* Value
Patient data				
Age	73 (58–81)	72 (57–80)	74 (71–81)	0.180
Male sex	55 (44.4%)	45 (45.5%)	10 (40.0%)	0.624*
Time from symptom onset	166 (109–303)	171 (113–311)	158 (83–243)	0.345
NIHSS on admission	14 (9–16)	13 (9–16)	15 (13–16)	0.293
Treatment data				
IV thrombolysis	81 (65.3%)	60 (60.6%)	21 (84.0%)	0.028*‡
Endovascular therapy	49 (40.2%)	38 (39.2%)	11 (44.0%)	0.661*
Imaging data				
NCCT-ASPECTS	9 (7–10)	9 (7–10)	8 (7–9)	0.654
Occluded vessels				
ICA	44 (35.5%)	34 (34.3%)	10 (40.0%)	0.597*
Carotid T	12 (9.7%)	11 (11.1%)	1 (4.0%)	0.456†
M1 segment of MCA	69 (55.6%)	53 (53.5%)	16 (64.0%)	0.347*
M2 segment of MCA	27 (21.8%)	23 (23.2%)	4 (16.0%)	0.434*
M3 segment of MCA	6 (4.8%)	5 (5.1%)	1 (4.0%)	1.000†
CBF deficit volume	129 (85–191)	117 (78–178)	180 (134–232)	0.002‡
CBV deficit volume	35 (12–84)	27 (10–71)	85 (59–114)	<0.001‡
CBF-CBV mismatch, %	63 (42–86)	69 (42–88)	56 (36–65)	0.021‡
Final infarction volume	31 (10–87)	23 (9–73)	62 (10–169)	0.105
Fetal PCA	19 (16.5%)	14 (15.2%)	5 (21.7%)	0.530†
Clinical data				
Premorbid mRS	0 (0–0)	0 (0–0)	0 (0–0)	0.964
Admission mRS	5 (4–5)	5 (4–5)	5 (5–5)	0.460
Discharge mRS	4 (2–5)	4 (2–5)	5 (4–5)	0.049‡
90-day mRS	4 (1–5)	4 (1–5)	4 (0–6)	0.777

Values presented are count (percentage) for categorical and median (interquartile range) for ordinal or continuous variables. Time values are presented in minutes, volume values as mL. Nonparametric tests for non-normally distributed continuous variables were performed using the Mann–Whitney *U* test and for ordinal variables using the independent samples median test. ASPECTS indicates Alberta Stroke Program Early CT Score; CBF/CBV, cerebral blood flow/volume; ICA, internal carotid artery; ITD, ipsilateral thalamic diaschisis; IV, intravenous; MCA, middle cerebral artery; mRS, modified Rankin Scale; NCCT, noncontrast computed tomography; NIHSS, National Institutes of Health Stroke Scale; and PCA, posterior cerebral artery.

*Proportion analysis tests for categorical variables were performed using the χ^2 test.

†Proportion analysis tests for categorical variables were performed using Fisher exact test.

‡*P* values <0.05.

mRS at discharge (each with *P*>0.05). The morphological and clinical outcome analysis are presented in Tables 3 and 4 (additional results are provided in Table II through V in the online-only Data Supplement).

Association of ITD With Subacute Stroke Complications

In our binary logistic regression analysis including all subjects, ITD was not associated with hemorrhagic infarction, parenchymal hematoma, extraischemic intracranial hemorrhage, or space-occupying edema (each with *P*>0.05). The analysis of subacute stroke complications is shown in Table VI in the online-only Data Supplement.

Discussion

In our study on the assessment of acute ITD using perfusion-based imaging, ITD occurred in about one fifth of patients with MCA stroke. ITD was more frequent in cases of MCA stroke with subcortical involvement. ITD occurred more frequently in patients with larger ischemic volumes but did not have an independent influence on final infarction volume, relative infarction growth, or clinical outcome.

Table 2. Spatial Characteristics of CT Perfusion Deficit for ITD− and ITD+ Acute Ischemic Stroke Patients

	Overall (N=124)	ITD− (n=99)	ITD+ (n=25)	*P* Value
ASPECTS region				
M1	97 (78.2%)	73 (73.7%)	24 (96.0%)	0.016*‡
M2	106 (85.5%)	82 (82.8%)	24 (96.0%)	0.119†
M3	80 (64.5%)	61 (61.6%)	19 (76.0%)	0.179*
M4	96 (77.4%)	74 (74.7%)	22 (88.0%)	0.157*
M5	108 (87.1%)	84 (84.8%)	24 (96.0%)	0.190†
M6	86 (69.4%)	64 (64.6%)	22 (88.0%)	0.024*‡
C	47 (38.2%)	30 (30.6%)	17 (68.0%)	<0.001*‡§
I	100 (80.6%)	76 (76.8%)	24 (96.0%)	0.044†‡
IC	43 (35.2%)	26 (26.5%)	17 (70.8%)	<0.001*‡§
L	53 (42.7%)	34 (34.3%)	19 (76.0%)	<0.001*‡§
CCD	43 (34.7%)	29 (29.3%)	14 (56.0%)	0.012*

Values are presented as count (percentage). ASPECTS indicates Alberta Stroke Program Early CT Score; C, caudate; CCD, crossed cerebellar diaschisis; I, insular ribbon; IC, internal capsule; ITD, ipsilateral thalamic diaschisis; and L, lentiform nucleus.

*Test for statistical significance was performed using χ^2 test.

†Test for statistical significance was performed using Fisher exact test.

‡*P* values <0.05.

§Significant *P* values after correction for multiple comparisons using Bonferroni's method.

As described before, CTP was able to detect diaschisis phenomena in acute stroke.[13] To avoid the possibility of detecting chronic ITD, we excluded all patients with prior infarction. Moreover, we only included patients with internal carotid artery or MCA occlusions considering the possibility for impaired thalamic blood supply by fetal variants of the posterior cerebral artery. However, examination of CT angiography data for the presence of fetal variants did not result in significant differences between ITD− and ITD+ subjects, which further supports the notion that the observed thalamic hypoperfusion is caused by diaschisis.

Table 3. Analysis of Morphological Outcome Stratified by Treatment

	Final Infarction Volume		Relative Infarction Growth	
Independent Variable	β	*P* Value	β	*P* Value
Patients treated with IVT and EVT (n=38)				
ITD*	−0.158	0.386	0.032	0.863
Patients treated with IVT alone (n=43)				
ITD*	0.093	0.412	−0.099	0.601

A multivariable linear regression analysis was performed for the indicated morphological outcome parameters. ASPECTS indicates Alberta Stroke Program Early CT Score; CBF/CBV, cerebral blood flow/volume; EVT, endovascular thrombectomy; ITD, ipsilateral thalamic diaschisis; IVT, intravenous thrombolysis; and NIHSS, National Institutes of Health Stroke Scale.

*ITD was adjusted for admission NIHSS, ASPECTS, CBF deficit volume, and CBV deficit volume.

Table 4. Analysis of Clinical Outcome Stratified by Treatment

	Discharge mRS	
Independent Variable	OR (95% CI)	*P* Value
Patients treated with IVT and EVT (n=37)		
ITD*	1.211 (0.237–6.196)	0.818
Patients treated with IVT alone (n=36)		
ITD*	1.002 (0.189–5.323)	0.998

A multivariable, ordinal logistic regression analysis was performed for discharge mRS. ASPECTS indicates Alberta Stroke Program Early CT Score; CBF/CBV, cerebral blood flow/volume; CI, confidence interval; EVT, endovascular thrombectomy; ITD, ipsilateral thalamic diaschisis; IVT, intravenous thrombolysis; mRS, modified Rankin Scale; NIHSS, National Institutes of Health Stroke Scale; and OR, odds ratio.

*ITD was adjusted for admission NIHSS, ASPECTS, CBF deficit volume, and CBV deficit volume.

In our study on acute stroke, ITD frequency was lower compared with that in other studies conducted in the setting of chronic or subacute phase after stroke, which ranged from 43% to 86%.[4–6] As CTP is only clinically indicated for acute stroke assessment,[22] we cannot provide data on the course or reversibility of ITD. Yet, we assume no persisting critical thalamic hypoperfusion as thalamic damage was not detected on all follow-up imaging. The discrepant frequencies in the acute and chronic stages might indicate a delayed development of ITD in the course after stroke, conceivably as a consequence of secondary thalamic neurodegeneration.[23]

In accordance with the study from De Reuck et al[6] on patients in the chronic phase of stroke, we also found an increased ischemic involvement of subcortical areas in ITD+ acute stroke patients, including lentiform nucleus, internal capsule, and caudate nucleus. This suggests a pivotal role for the disruption of subcortical input to the thalamus in the development of ITD. Lentiform nucleus, internal capsule, and caudate nucleus all contain neurons and axons of the cortico-basal ganglia-thalamo-cortical loop comprising crucial circuitry of motor function with strong phylogenetic preservation.[24–26] Additionally, the internal capsule contains most of the thalamic radiation, including projections to the cortex and vice versa, depicting a lever for interrupting most of the thalamic output and cortico-thalamic feedback.[27]

On a structural level without consideration of infarct size or distribution in the MCA territory, ipsilesional thalamic atrophy has been reported as a frequent finding after stroke in CT and MRI studies.[28,29] The association between damage of subcortical areas and damage to the thalamus after MCA stroke was stressed by Hervé et al[30] who examined diffusion changes using MRI. In a small sample of 9 patients, they found a significant increase of diffusion variables 6 months after stroke, indicating cellular thalamic damage, especially in cases with lesions of the basal ganglia.

Possible mechanisms for secondary thalamic damage studied in animal models include a combination of retrograde degeneration and neurotoxicity because of disinhibition from loss of GABA-ergic neurons if subcortical areas are involved.[31] This notion is supported by another study that reported a high frequency of diaschisis, as well as development of thalamic

lesions in the course after MCA stroke, with marked involvement of the basal ganglia.[32] As subcortical defects are not only associated with thalamic deficits in function and perfusion—as it is the case in our study—but also structural damage, the findings might reflect different stages of secondary thalamic damage because of MCA territory stroke. Further, a recent study connected increased iron deposition in the thalamus after stroke to worse clinical outcome, suggesting other long-lasting metabolic alterations contributing to thalamic damage.[23]

For our clinical outcome analysis, we found no independent effect of ITD on the outcome of stroke patients at discharge in treatment subgroups. Interestingly, we observed no difference in the clinical parameters on admission based on ITD status. Regarding long-term clinical outcome, however, we cannot draw definitive conclusions based on the limited sample sizes in the treatment subgroups. For morphological outcome, no independent association of ITD with final infarction volume or relative infarction growth was detected.

Recent successful trials on thrombectomy in extended time windows[14,15] applied the technique of CTP to select patients for treatment. The selection was based on cutoff values for the ischemic core volume as detected by automated software. The hypoperfusion of the thalamus, located right next to the subcortical MCA territory, and the frequent co-occurrence with basal ganglia hypoperfusion might falsely overestimate the true size of the ischemic lesion. Our observation based on automated infarction core analysis is illustrated in Figure 2. This important fact should, therefore, be considered in the technical development of automated CTP analysis to increase the applicability of future trials.

One limitation of our study was a noncomplete follow-up mRS after 90 days. Still, our study represents the largest cohort for subacute outcome after ITD in ischemic stroke to date. In addition, our study was conducted in a retrospective design, only including acute stroke patients who had follow-up-confirmed infarctions. Thus, our study could not assess possibly false-positive cases of ITD based on healthy controls. Further, we did not classify ITD based on quantitative parameters, as such measurements are not applied for stroke assessment in the clinical routine. Based on the technical features of CTP, hypometabolism or function as part of the diaschisis complex could not be studied.

Summary/Conclusions

ITD is a frequent phenomenon that occurs in the CTP evaluation of acute MCA stroke, particularly in cases with involvement of the basal ganglia. ITD did not impact the subsequent stroke outcome. Stroke physicians as well as developers of automated CTP assessment as used in current trials should be aware of ITD to avoid its misinterpretation as part of the acute ischemic lesion.

Disclosures

None.

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