Turkish Journal of Cerebrovascular Diseases 2021; 27(1): 42-49 Türk Beyin Damar Hastalıkları Dergisi 2021; 27(1): 42-49
Turk J Cereb Vasc Dis
doi: 10.5505/tbdhd.2021.83788

ORIGINAL ARTICLE **ÖZGÜN ARAŞTIRMA**

ACUTE ISCHEMIC LESIONS IN PATIENTS WITH TRANSIENT ISCHEMIC ATTACK AND ASSOCIATION WITH CLINICAL CHARACTERISTICS

Bülent GÜVEN, Müjdat Deniz BENLİ, Beyza Nur ÇETİN, Hayat GÜVEN

University of Health Sciences Dışkapı Yıldırım Beyazıt Training and Research Hospital, Department of Neurology, Ankara, TURKEY

ABSTRACT

INTRODUCTION: Although transient ischemic attack (TIA) is defined as "a brief episodes of temporary neurological dysfunction due to focal brain, spinal cord, or retinal ischemia without acute infarction"; acute ischemic lesion is observed in approximately 1/3 of the patients and it is accepted as a predictive finding for the risk of developing stroke. In this study, it was aimed to investigate acute ischemic lesions in patients diagnosed with TIA and the relationship between these lesions and clinical features.

METHODS: Patients whose neurological deficits resolved completely within the first 24 hours and clinically diagnosed with TIA were included in the study. Brain diffusion weighted imaging (DWI) was performed in all patients within the first 24 hours after the onset of symptoms; lesions compatible with acute ischemia were determined. Neurological examination findings, recovery time of neurological deficit, vascular risk factors, TIA etiology and $ABCD^2$ scores were recorded.

RESULTS: Ninety-four patients (37 females, 57 males; mean age 68.4±13.8 years) were included in the study. Acute ischemic lesion restricting diffusion was detected on DWI in 40 patients (42.6%). The time to recovery of neurological symptoms was 117.9±105.1 minutes in patients with DWI positive and 58±69.9 minutes in patients with negative DWI, and the difference was statistically significant (p=0.003). The number of patients with symptom duration ≥60 minutes was higher in the DWI positive group than in the negative group (p=0.025); the number of patients with <10 minutes was less (p=0.040).

DISCUSSION AND CONCLUSION: The results of our study indicated that acute ischemic lesions may be encountered quite frequently in patients whose symptoms recover within 24 hours, and that acute ischemic lesions are more likely to occur especially in patients whose symptoms persist for more than one hour; it suggested that new classifications and TIA definitions including subgroups related to the presence of time-dependent and viewable ischemic lesions may be required.

Keywords: Transient ischemic attack, diffusion-weighted imaging, cerebral ischemia.

Address for Correspondence: Bülent Güven, MD. University of Health Sciences Dışkapı Yıldırım Beyazıt Training and Research Hospital, Department of Neurology, Ankara, Turkey.
Phone: +90312 596 20 00 **E-mail:** bulentcanguven@gmail.com
Received: 26.03.2021 **Accepted:** 13.04.2021
ORCID IDs: Bülent Güven 0000-0002-4816-9257, Müjdat Deniz Benli 0000-0003-3660-0158, Beyza Nur Çetin 0000-0002-8230-0597, Hayat Güven 0000-0002-9135-639X.
Please cite this article as following: Güven B, Benli MD, Çetin BN, Güven H. Acute ischemic lesions in patients with transient ischemic attack and association with clinical characteristics. Turkish Journal of Cerebrovascular Diseases 2021; 27(1): 42-49. doi: 10.5505/tbdhd.2021.83788

GEÇİCİ İSKEMİK ATAKLI HASTALARDA AKUT İSKEMİK LEZYONLAR VE KLİNİK ÖZELLİKLER İLE İLİŞKİSİ

ÖZ

GİRİŞ ve AMAÇ: Günümüzde geçici iskemik atak (GİA) "akut infarkt olmaksızın fokal beyin, spinal kord, veya retinal iskemi nedeniyle gelişen kısa süreli ve geçici nörolojik işlev bozukluğu atağı" olarak tanımlansa da; hastalarının yaklaşık 1/3'ünde akut iskemik lezyon görüntülenmekte ve inme gelişme riski için öngörücü bir bulgu olarak kabul edilmektedir. Bu çalışmada GİA tanısı konulan hastalarda akut iskemik lezyonların ve bu lezyonların klinik özelliklerle ilişkisinin araştırılması amaçlanmıştır.

YÖNTEM ve GEREÇLER: Nörolojik defisiti ilk 24 saat içinde tam olarak düzelen ve klinik olarak GİA tanısı konulan hastalar çalışmaya alındı. Tüm hastalara semptom başlangıcından sonraki ilk 24 saat içerisinde beyin difüzyon ağırlıklı görüntüleme (DAG) yapılarak; akut iskemi ile uyumlu lezyonlar belirlendi. Nörolojik muayene bulguları, nörolojik defisitin düzelme süresi, vasküler risk faktörleri, GİA etyolojisi ve $ABCD^2$ puanları kaydedildi.

BULGULAR: Doksan dört hasta (37 kadın, 57 erkek; yaş ortalaması 68.4±13.8) çalışmaya alındı. Kırk hastada DAG'da difüzyon kısıtlayan akut iskemik lezyon saptandı (%42.6). Nörolojik semptomların düzelmesine kadar geçen süre DAG pozitif hastalarda 117.9±105.1 dakika, DAG negatiflerde ise 58±69.9 dakika idi ve aradaki fark istatistiksel olarak anlamlı bulundu (p=0.003). DAG pozitif grupta negatif gruba göre semptom süresi ≥60 dakika olan hasta sayısının daha fazla (p=0.025); <10 dakika olan hasta sayısının ise daha az olduğu (p=0.040) saptandı.

TARTIŞMA ve SONUÇ: Çalışmamızın sonuçları semptomları 24 saat içerisinde düzelen hastalarda akut iskemik lezyonlara oldukça sık olarak rastlanılabileceğine, özellikle semptomları bir saatten daha uzun süren hastalarda akut iskemik lezyon görülme olasılığının daha yüksek olduğuna işaret etmiş; zaman-bağımlı ve görüntülenebilir iskemik lezyon varlığı ile ilişkili alt grupları içeren yeni sınıflandırmalara ve GİA tanımlamalarına gereksinim olabileceğini düşündürmüştür.

Anahtar Sözcükler: Geçici iskemik atak, difüzyon ağırlıklı görüntüleme, serebral iskemi.

INTRODUCTION

The scope of the definition of transient ischemic attack (TIA) has been a controversial issue for many years. Time-based definitions based on the resolution of symptoms within 24 hours were recognized first, and later tissue-based definitions based on the absence of ischemic lesions were accepted (1). While TIA is defined as "a a brief episodes of temporary neurological dysfunction due to focal brain, spinal cord, or retinol ischemia without acute infarction" (1); it is recognized that an acute ischemic lesion is observed in 1/3 of patients whose symptoms last less than 24 hours (2-4). Moreover, it has been demonstrated that diffusion-weighted imaging (DWI) lesions can result in permanent damage in subsequent magnetic resonance imaging (MRI) in patients with TIA (5). As a result, time- and tissue-based definitions have unclear and contradictory aspects. As much as the answer questions; "How are the neurological findings, even if severe, transient despite the development of imageable ischemia? How do pathophysiological mechanisms work?", the one for the question "What should be the terminology that should be used when a neurological deficit lasting less than 24 hours is accompanied by a lesion showing diffusion restriction?" are not yet clear. Approximately half of the patients with stroke can be classified as minor / mild stroke with symptoms that are not crippling or rapidly resolving, although there is no consensus on its definition (6,7). Perhaps it will be better to classify patients with DWI lesions whose symptoms improve within 24 hours as having suffered a mild stroke (7). On the other hand, it has been suggested that the distinction between TIA and minor ischemic stroke, which are the lightest examples of a stroke spectrum with common pathophysiology and requiring similar urgency in terms of diagnosis and treatment, is losing its clinical significance and may not be necessary for practice (8-10).

While TIA is commonly thought to be a benign condition, patients who have TIA are at a high risk of having an early stroke in the following period (1). About 20% of ischemic strokes are preceded by one or more TIAs, and 10-15% of TIA patients have a stroke within the first 3 months, with half of them occurring within the first 48 hours (2,11-13). Therefore, TIA provides an important opportunity for rapid diagnosis of the underlying cause and initiation of treatment before a permanent disability develops (2). The increase in stroke risk after TIA can be predicted by clinical scales, diffusion MRI, and vascular imaging (1). The accompanying acute ischemic lesion on DWI to clinical findings of TIA is

associated with the risk of developing stroke, and it has been suggested to be a strong predictor of stroke (14-19). The presence of DWI lesion in patients with transient neurological symptoms can be considered as a stimulus to initiate more effective prophylactic treatments, as well as supporting the vascular origin of the deficit and contributing to the determination of the mechanism of stroke with its localization and distribution (2).

This study aimed to look into DWI acute ischemic lesions and the relationship between these lesions and clinical features in patients whose symptoms improved within 24 hours and were clinically diagnosed with TIA.

METHODS

The file records of the patients who were hospitalized in the neurology clinic with the diagnosis of TIA between 01.08.2019-01.02.2020 were retrospectively analyzed. The study involved consecutive patients whose symptoms and findings were consistent with stroke, whose neurological deficits healed fully within the first 24 hours, and who underwent brain DWI within the first 24 hours. Patients whose symptoms were consistent with transient global amnesia, who had a history of epilepsy or had epileptic seizures before or during admission, who had a history of migraine, or who had a headache consistent with migraine attack before admission, and who had hypoglycemia at the time of admission were not included in the study. Also, patients with a known neurological disease, severe head trauma, history of intracranial mass, and in whom brain MRI could not be performed were excluded from the study.

Neurological examination findings (motor/hemiparesis, sensory, aphasia/dysphasia, brainstem/cerebellar), recovery time of neurological symptoms and deficits during TIA were recorded. Blood tests of patients, electrocardiography, transthoracic echocardiography, carotid, and vertebral color Doppler Ultrasonography, and 24-hour rhythm Holter, transesophageal echocardiography, and other examinations for stroke etiology were evaluated. Vascular risk factors (hypertension, diabetes mellitus, dyslipidemia, smoking, coronary artery disease, valvular heart disease, atrial fibrillation, history of stroke, and/or TIA) and antiaggregants, anticoagulant or lipid-lowering medications of the patients were recorded. The etiology of TIA/stroke was determined according to the TOAST classification (large artery atherosclerosis, cardioembolism, small-vessel occlusion, other determined etiology and undetermined etiology) (20). $ABCD^2$ (age, blood pressure, clinical weakness, duration, diabetes mellitus) scores were recorded (21).

In DWI performed within the first 24 hours after the symptom onset; the presence of lesions consistent with acute ischemia showing diffusion restriction; the location, number (single, multiple), and size (<1.5 cm,> 1.5 cm) of the lesions (anterior cerebral artery, middle cerebral artery, posterior cerebral artery hemispheric territories, brainstem, cerebellum, and watershed areas), and the time between the onset of symptoms and DWI were determined.

The study was conducted in accordance with the Helsinki Declaration and with the approval of the Clinical Research Ethics Committee of the University of Health Sciences Dışkapı Yıldırım Beyazıt Training and Research Hospital (Date: 16.03.2020, Number: 84/15). Written informed consent was obtained from all patients participating in the study.

Statistical Analysis: SPSS 23.0 program was used for analyzes (Statistical Package for the Social Sciences, version 23.0 for Windows, SPSS Inc., Chicago, IL). Compliance of continuous variables to normal distribution was investigated by the Kolmogorov-Smirnov test. Descriptive statistics for continuous variables were expressed as mean±SD, for categorical variables as number (%). Normally distributed continuous variables were compared using the Student-t-test, and those not showing normal distribution were compared with the Mann-Whitney U test. Pearson's chi-square test was used for categorical variables, and Fisher's exact test was used when chi-square test conditions were not met. The statistical significance level in the analyzes was accepted as $p<0.05$.

RESULTS

Ninety-four patients (37 females, 57 males; mean age 68.4±13.8 years) were included in the study. While acute ischemic lesions restricting diffusion (DWI positive) were detected in 40 patients on DWI (42.6%), no acute ischemic lesion restricting diffusion was detected on DWI in 54 patients (DWI

negative). There was no statistical difference between the DWI positive and negative groups in terms of age and gender. The recovery time of neurological deficit was 117.9 ± 105.1 minutes in patients with positive DWI and 58 ± 69.9 minutes in patients with negative DWI, and the difference was statistically significant (p= 0.003). The number of patients with neurological deficits lasting <10 minutes is lower in the DWI positive group than in the negative group (p= 0.040); the number of patients with neurological deficits of ≥60 minutes is higher (p= 0.025) was detected. There was no difference between DWI positive and negative patients in terms of neurological deficits, vascular risk factors, and ABCD² scores during TIA. The time between symptom onset and DWI was not statistically different between DWI positive and negative patients. While the etiology was determined as small vessel occlusion in 4 patients in the DWI positive group, there was no patient whose stroke was evaluated as small vessel occlusion in the DWI negative group (p= 0.030) (Table 1). In 24 patients with positive DWI, the lesion was single (60%); <1.5 cm (52.5%) in 21 patients; and in the middle cerebral artery territory in 25 patients (62.5%) (Table 2).

DISCUSSION AND CONCLUSION

In our study, an acute ischemic lesion was detected in DWI performed in the first 24 hours in 42.6% of TIA patients. Although it is accepted that approximately 1/3 of TIA patients have acute ischemic lesions (2,4,19), this rate varies within a wide range and it has been reported that 30-50% of patients have acute ischemic lesions on DWI (1,8,14,22-25).

Why can acute ischemic lesions be demonstrated in some patients with TIA, but not in others? The answer to this question is not very clear. This may be partially related to the imaging technique. The time between TIA and neuroimaging likely plays a role in MRI positivity. Hyperacute DWI findings may vary; Positive images may initially be falsely negative or normalize later (4). Serial MRI studies have shown that some ischemic lesions disappear rapidly and may lose visibility after more than 48 hours from the onset of TIA, and it has been suggested that the diagnostic value of MRI performed after TIA is delayed (4,26). Conversely, it has been reported that lesion viewability may be delayed (4,27-29), in which case DWI positivity may be found lower if the first MRI was performed immediately after symptom onset. The sensitivity of DWI in patients with TIA was higher than in the first 3 hours in patients performed in the first 24 hours (28). If the time between the onset of TIA symptoms and the first DWI is less than 2 hours, repeat DWI is recommended to minimize false-negative findings (27). In our study, DWI was performed in all patients in the first 24 hours after the onset of symptoms, and no statistical difference was found between the onset of symptoms and the duration of DWI between patients with and without acute ischemic lesions within the first 24 hours. Apart from the time-dependent sensitivityof DWIin the imaging of acute ischemia; frequently small DWI lesions associated with TIA and possible errors in ADC measurements of small DWI lesions may cause ischemic lesions not to be visualized (30). Also, the fact that the mean ADC values of DWI lesions are lower in patients with transient ischemic deficits than those with permanent deficits may be effective in negative results (2,23).

In our study, the recovery time of neurological deficit was found to be associated with DWI positivity, and acute ischemic lesions were observed more frequently, especially in patients whose symptoms lasted longer than 60 minutes. DWI positivity, on the other hand, was less frequent in patients whose symptoms lasted less than 10 minutes. In studies, long symptom duration was found to be associated with DWI positivity, and the risk of DWI positivity was found to be high, especially when symptoms last longer than 60 minutes (18,23,31,32). The duration of symptoms may reflect the time at which ischemia resolves, and a longer duration of ischemia may increase the likelihood of resulting in infarction. This also suggests that when an ischemic lesion cannot be demonstrated on DWI in prolonged deficits, attention should be paid to the possibility that the symptoms may be due to non-vascular causes.

TIA-related lesions on DWI are often small and multiple (16,23,30,32). The term "footprints of transient ischemic attacks" has been suggested to identify such punctate lesions that persist on DWI after the TIA symptoms have healed completely (33). TIA-associated DWI lesions can be found anywhere in the brain; they can be located in functional brain regions such as the brainstem,

Tablo 1. Characteristics of patients with and without acute ischemic lesions in diffusion-weighted imaging in patients with transient ischemic attacks.

	Patients with ischemic lesions on DWI n=40	Patients without ischemic lesions on DWI n=54	*p*
Age (years)	68.4±15.1	68.4±13	0.988[1]
Gender Male/Female	13/27 (32.5/67.5)	24/30 (44.4/55.6)	0.338
Symptom duration (min)	117.9±105.1	58±69.9	0.003[2]
Symptom duration <10 min	1(2.5)	9 (16.7)	0.040
Symptom duration 10-59 min	14 (35)	26 (48.1)	0.287
Symptom duration ≥ 60 min	25 (62.5)	20 (37)	0.025
TIA symptoms			
Motor	8 (20)	19 (35.2)	0.168
Sensory	17 (42.5)	24 (44.4)	1.000
Aphasia/dysphasia	22 (55)	24 (44.4)	0.422
Brainstem/cerebellar	2 (5)	2 (3.7)	1.000
Vascular risk factors			
Hypertension	23 (57.5)	30 (55.6)	1.000
Diabetes mellitus	11 (27.5)	20 (37)	0.453
Smoking	11 (27.5)	11 (20.4)	0.575
Hypercholesterolemia	9 (22.5)	17 (31.5)	0.466
Hypertriglyceridaemia	12 (30)	11 (20.4)	0.406
Coronary artery disease	13 (32.5)	15 (27.8)	0.790
Heart valve disease	7 (17.5)	8 (14.8)	0.947
Atrial fibrillation	6 (15)	5 (9.3)	0.519
History of previous stroke (number of patients)	4 (10)	7 (13)	0.754
Number of past strokes	0.1±0.3	0.1±0.3	0.660[2]
Previous TIA history (number of patients)	2 (5)	3 (5.6)	1.000
Number of past TIA	0.1±0.3	0.1±0.5	0.891[2]
Drugs used			
Antiaggregants	17 (42.5)	22 (40.7)	1.000
Anticoagulants	2 (5)	5 (9.3)	0.695
Statins	5 (12.5)	4 (7.4)	0.488
Total cholesterol (mg/dl)	200.1±195.9	209±193.9	0.156[2]
LDL (mg/dl)	211.2±317.8	168.8±190.8	0.218[2]
HDL (mg/dl)	48.8±60.7	52.9±59.5	0.221[2]
Triglyceride (mg/dl)	193±188.5	179.2±168.3	0.994[2]
Glucose (mg/dl)	112.7±58.2	115.9±55.5	0.646[2]
HbA1c* (%)	6.7±2.1	6.3±1.1	0.932[2]
Etiology			
Large artery athesclerosis	11 (27.5)	12 (22.2)	0.729
Cardioembolism	7 (17.5)	6 (11.1)	0.559
Small vessel occlusion	4 (10)	0 (0)	0.030
Other causes	0 (0)	0 (0)	
Undetermined	18 (45)	36 (66.7)	0.059
ABCD² score	4.1±1.4	3.9±1.6	0.413[2]
ABCD² score <4	12 (30)	18 (33.3)	0.905
ABCD² score = 4-5	22 (55)	28 (51.9)	0.926
ABCD² score = 6-7	6 (15)	9 (16.7)	1.000
Seymptom onset-DWI time (hours)	6.4±4.9	6.4±5.2	0.764[2]

Data are given as a number (%) or mean±standard deviation.
1Student-t test, 2Mann-Whitney U test
* HbA1c was evaluated in 85 patients.
DWI: Diffusion-weighted imaging, TIA: Transient ischemic attack, LDL: Low-density lipoprotein, HDL: High-density lipoprotein, HbA1c: Glycolyzed hemoglobin

internal capsule, and motor cortex and arterial territories compatible with clinical symptoms, as well as in silent regions (2). In our study, it was found that approximately half of the DWI-positive patients had lesions <1.5 cm and 40% had multiple. It was observed that ischemic lesions were frequently located in the middle cerebral artery territory.

In our research, there was no correlation between the symptoms and signs of TIA and the occurrence of acute ischemic lesions. Motor deficits, unilateral weakness, and aphasia have

Table 2. Infarct number, size, and location in diffusion-weighted imaging.

	n (%)
DWI number of ischemic lesions	
Single	24 (60)
Multiple	16 (40)
DWI ischemic lesion size	
≥1.5 cm	19 (47.5)
< 1.5 cm	21 (52.5)
DWI ischemic lesion location	
Middle cerebral artery territory	25 (62.5)
Posterior cerebral artery territory	4 (10)
Brainstem	5 (15)
Mesencephalon	1 (2.5)
Pons	2 (5)
Bulbus	3 (7.5)
Cerebellum	2 (5)
Border area settled	5 (12.5)
Anterior	1 (2.5)
Posterior	3 (7.5)
Internal	1 (2.5)

Data are given as a number (%).
DWI: Diffusion-weighted imaging.

been found to be correlated with DWI positivity (14,22,24,31,32,34).

In our study, no difference was found between DWI-positive and negative patients in terms of age, gender, and accompanying vascular risk factors. While the etiology was determined as small vessel occlusion in 4 patients in the DWI positive group, there was no patient whose stroke was evaluated as small vessel occlusion in the DWI negative group. Although the difference between the two groups was statistically significant, it was not reliable due to the small number of patients. The number of patients for whom the cause of stroke could not be determined was higher in the DWI negative group, and the difference between the DWI positive group was quite close to the statistical significance limit. It has been suggested that large vessel occlusion is associated with DWI positivity in patients with TIA (2). Stroke or TIA history, diabetes mellitus (24), and atrial fibrillation (14,24) were found to be more common in patients with positive DWI than in negative patients; in another study, age, diabetes mellitus, hypertension, and smoking were not found to be associated with the presence of acute ischemic lesions (22). In our study, no difference was found between the DWI positive and negative groups in terms of the number of patients who used antiaggregant, anticoagulant, and lipid-lowering drugs before TIA.

The existence of DWI lesions in patients with TIA has been associated with a high risk for early recurrence of ischemic events (8,14,17-19,31). In a multi-center study, the rate of recurrence of stroke in TIA patients who underwent DWI was 7.1% in 7 days in positive DWI (tissue-positive), 0.4% in negative patients; at 90 days, DWI was found as 7.7% in positives and 1% in negatives. It has been suggested that a tissue-based definition of TIA may help provide prognostic information (19). In another study, it was reported that the risk of developing stroke was higher in patients with TIA or minor stroke with DWI lesions compared to those without lesions (10.8% at 90 days), and if there is intracranial vessel occlusion in MR angiography, the risk is further increased (32.6%) (35). However, in one study, a DWI lesion accompanying TIA was detected in 11% of patients and did not correlate with the risk of early stroke after TIA (34). The relationship of DWI lesions with the long-term (1-5 years) stroke risk is not clear (8).

In our study, it was found that $ABCD^2$ total scores, which is a prognostic scoring developed to predict the risk of early stroke after TIA, and the distribution of patients according to the score categories indicating low, moderate, and high risk did not differ between the DWI positive, and negative groups. However, since our study is not a long-term follow-up study, it is not possible to comment on the prognostic significance of the presence of acute ischemic lesions in TIA patients only in accordance with $ABCD^2$ scoring. In a study in which DWI-MRI was used, no correlation was found between the presence of acute ischemic lesions in TIA patients and $ABCD^2$ score and an increase in stroke risk (90 days, 1 year, 5 years) (22). However, it has been suggested that adding the presence of brain ischemic lesions to $ABCD^2$ scoring in TIA patients may improve the prediction of stroke in the acute phase after TIA (36,37).

The fact that patients who underwent DWI in the first 24 hours after the onset of TIA were included in the study and no imaging repetitions were performed, may constitute a limitation for our study. Lesions that become positive after 24 hours may be ignored, resulting in errors in the number of DWI-positive patients. It has been recommended that neuroimaging, preferably MRI including diffusion sequences, be performed within 24 hours after the onset of symptoms in patients with TIA (1). However, there is no consensus on the duration of imaging of ischemic

lesions on DWI, it has been reported that lesions that can be imaged in the early period may disappear later or lesions that were not detected at the beginning can be imaged in the following days (4,26-29). In this case, it will be possible to accurately determine the frequency of acute ischemic lesions in patients with TIA with serial DWIs from the onset of symptoms.

Evaluation of acute ischemic lesions is important in patients with transient ischemic attacks, as it is a prognostic indicator for recurrence of stroke, and may guide the determination of treatment approaches. The results of our study suggest that acute ischemic lesions can be encountered quite frequently in patients whose symptoms improve within 24 hours, it pointed out that especially in patients whose symptoms last longer than an hour, the possibility of acute ischemic lesions is higher, suggesting that there may be a need for new classifications and TIA definitions that include subgroups related to time-dependent and visualizable ischemic lesions.

KAYNAKLAR

1. Easton JD, Saver JL, Albers GW, et al; American Heart Association; American Stroke Association Stroke Council; Council on Cardiovascular Surgery and Anesthesia; Council on Cardiovascular Radiology and Intervention; Council on Cardiovascular Nursing; Interdisciplinary Council on Peripheral Vascular Disease. Definition and evaluation of transient ischemic attack: a scientific statement for healthcare professionals from the American Heart Association/American Stroke Association Stroke Council; Council on Cardiovascular Surgery and Anesthesia; Council on Cardiovascular Radiology and Intervention; Council on Cardiovascular Nursing; and the Interdisciplinary Council on Peripheral Vascular Disease. The American Academy of Neurology affirms the value of this statement as an educational tool for neurologists. Stroke 2009; 40(6): 2276-2293.
2. Souillard-Scemama R, Tisserand M, Calvet D, et al. An update on brain imaging in transient ischemic attack. J Neuroradiol 2015; 42(1): 3-11.
3. Ovbiagele B, Kidwell CS, Saver JL. Epidemiological impact in the United States of a tissue-based definition of transient ischemic attack. Stroke 2003; 34(4): 919-924.
4. Brazzelli M, Chappell FM, Miranda H, et al. Diffusion-weighted imaging and diagnosis of transient ischemic attack. Ann Neurol 2014; 75(1): 67-76.
5. Oppenheim C, Lamy C, Touzé E, et al. Do transient ischemic attacks with diffusion-weighted imaging abnormalities correspond to brain infarctions? AJNR Am J Neuroradiol 2006; 27(8): 1782-1787.
6. Roberts PS, Krishnan S, Burns SP, et al. Inconsistent Classification of Mild Stroke and Implications on Health Services Delivery. Arch Phys Med Rehabil 2020; 101(7): 1243-1259.
7. Tse D, Hill MD, Coutts SB. Early secondary prevention in transient ischemic attack (TIA) and minor stroke. Curr Neurol Neurosci Rep 2019; 19(6): 34.
8. Coutts SB. Diagnosis and management of transient ischemic attack. Continuum (Minneap Minn) 2017; 23(1, Cerebrovascular Disease): 82-92.
9. Yu AYX, Coutts SB. Role of brain and vessel imaging for the evaluation of transient ischemic attack and minor stroke. Stroke 2018; 49(7): 1791-1795.
10. Kernan WN, Ovbiagele B, Black HR, et al. American Heart Association Stroke Council, Council on Cardiovascular and Stroke Nursing, Council on Clinical Cardiology, and Council on Peripheral Vascular Disease. Guidelines for the prevention of stroke in patients with stroke and transient ischemic attack: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. Stroke 2014; 45(7): 2160-2236. Erratum in: Stroke 2015; 46(2): e54.
11. Giles MF, Rothwell PM. Risk of stroke early after transient ischaemic attack: a systematic review and meta-analysis. Lancet Neurol 2007 ; 6(12): 1063-1072.
12. Johnston SC, Gress DR, Browner WS, et al. Short-term prognosis after emergency department diagnosis of TIA. JAMA 2000; 284(22): 2901-2906.
13. Wu CM, McLaughlin K, Lorenzetti DL, et al. Early risk of stroke after transient ischemic attack: a systematic review and meta-analysis. Arch Intern Med 2007; 167(22): 2417-2422.
14. Calvet D, Touzé E, Oppenheim C, et al. DWI lesions and TIA etiology improve the prediction of stroke after TIA. Stroke 2009; 40(1): 187-192.
15. Prabhakaran S, Chong JY, Sacco RL. Impact of abnormal diffusion-weighted imaging results on short-term outcome following transient ischemic attack. Arch Neurol 2007; 64(8): 1105-1109.
16. Ay H, Koroshetz WJ, Benner T, et al. Transient ischemic attack with infarction: a unique syndrome? Ann Neurol 2005; 57(5): 679-686.
17. Purroy F, Begué R, Gil MI et al. Patterns of diffusion-weighted magnetic resonance imaging associated with etiology improve the accuracy of prognosis after transient ischaemic attack. Eur J Neurol 2011; 18(1): 121-128.
18. Purroy F, Montaner J, Rovira A, et al. Higher risk of further vascular events among transient ischemic attack patients with diffusion-weighted imaging acute ischemic lesions. Stroke 2004; 35(10): 2313-2319.
19. Giles MF, Albers GW, Amarenco P, et al. Early stroke risk and $ABCD^2$ score performance in tissue- vs time-defined TIA: a multicenter study. Neurology 2011; 77(13): 1222-1228.
20. Adams HP Jr, Bendixen BH, Kappelle LJ, et al. Classification of subtype of acute ischemic stroke. Definitions for use in a multicenter clinical trial. TOAST. Trial of Org 10172 in Acute Stroke Treatment. Stroke 1993; 24(1): 35-41.
21. Johnston SC, Rothwell PM, Nguyen-Huynh MN, et al. Validation and refinement of scores to predict very early stroke risk after transient ischaemic attack. Lancet 2007; 369(9558): 283-292.
22. Anticoli S, Pezzella FR, Pozzessere C, et al. Transient ischemic attack fast-track and long-term stroke risk: Role of diffusion-weighted magnetic resonance imaging. J Stroke Cerebrovasc Dis 2015; 24(9): 2110-2116.
23. Kidwell CS, Alger JR, Di Salle F, et al. Diffusion MRI in patients with transient ischemic attacks. Stroke 1999; 30(6): 1174-1180.

24. Inatomi Y, Kimura K, Yonehara T, et al. DWI abnormalities and clinical characteristics in TIA patients. Neurology 2004; 62(3): 376-380.
25. Prabhakaran S. Triaging patients with transient ischemic attack--what can we learn from diffusion-weighted imaging? Nat Clin Pract Neurol 2008; 4(1): 14-15.
26. Moreau F, Modi J, Almekhlafi M, et al. Early magnetic resonance imaging in transient ischemic attack and minör stroke: do it or lose it. Stroke 2013; 44(3): 671-674.
27. Shono K, Satomi J, Tada Y, et al. Optimal timing of diffusion-weighted imaging to avoid false-negative findings in patients with transient ischemic attack. Stroke 2017; 48(7): 1990-1992.
28. Morita N, Harada M, Satomi J, et al. Frequency of emerging positive diffusion-weighted imaging in early repeat examinations at least 24 h after transient ischemic attacks. Neuroradiology 2013; 55(4): 399-403.
29. Carpentier N, Edjlali M, Bouhafs F, et al. Serial brain MRI in TIA patients. J Neuroradiol 2012; 39(3): 137-141.
30. Bertrand A, Oppenheim C, Lamy C, et al. Comparison of optimized and standard diffusion-weighted imaging at 1.5T for the detection of acute lesions in patients with transient ischemic attack. AJNR Am J Neuroradiol 2008; 29(2): 363-365.
31. Redgrave JN, Coutts SB, Schulz UG, et al. Systematic review of associations between the presence of acute ischemic lesions on diffusion-weighted imaging and clinical predictors of early stroke risk after transient ischemic attack. Stroke 2007; 38(5): 1482-1488.
32. Lamy C, Oppenheim C, Calvet D, et al. Diffusion-weighted MR imaging in transient ischaemic attacks. Eur Radiol 2006; 16(5): 1090-1095.
33. Ay H, Oliveira-Filho J, Buonanno FS, et al. 'Footprints' of transient ischemic attacks: a diffusion-weighted MRI study. Cerebrovasc Dis 2002; 14(3-4): 177-186.
34. Al-Khaled M, Matthis C, Münte TF, et al; QugSS2-Study. The incidence and clinical predictors of acute infarction in patients with transient ischemic attack using MRI including DWI. Neuroradiology 2013; 55(2): 157-163.
35. Coutts SB, Simon JE, Eliasziw M, et al. Triaging transient ischemic attack and minor stroke patients using acute magnetic resonance imaging. Ann Neurol 2005; 57(6): 848-854.
36. Giles MF, Albers GW, Amarenco P, et al. Addition of brain infarction to the $ABCD^2$ Score ($ABCD^2I$): a collaborative analysis of unpublished data on 4574 patients. Stroke 2010; 41(9): 1907-1913.
37. Merwick A, Albers GW, Amarenco P, et al. Addition of brain and carotid imaging to the $ABCD^2$ score to identify patients at early risk of stroke after transient ischaemic attack: a multicentre observational study. Lancet Neurol 2010; 9(11): 1060-1069.

Ethics

Ethics Committee Approval: The study was approved by University of Health Sciences Dışkapı Traning and Research Hospital Clinical Research Ethics Committee (Number: 84/115, Date: 16.03.2020).

Informed Consent: The authors declared that informed consent was signed by the patients.

Copyright Transfer Form: Copyright Transfer Form was signed by all authors.

Peer-review: Internally peer-reviewed.

Authorship Contributions: Surgical and Medical Practices: BG, HG. Concept: BG, HG. Design: BG, HG. Data Collection or Processing: MDÇ, BNÇ. Analysis or Interpretation: BG, HG. Literature Search: BG, MDB, BNÇ, HG. Writing: BG, HG.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study received no financial support.

*This study was presented as an e-poster at the 4th Stroke Academy of Turkey held by the Turkish Cerebrovascular Diseases Society on 18-20 December 2020

Copyright © 2021 by Turkish Cerebrovascular Diseases Society