The impact of neuro-imaging in TIA[1]

Matthew F. Giles
Stroke Prevention Research Unit, NIHR Biomedical Research Centre, John Radcliffe Hospital, Oxford, United Kingdom

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Introduction

There have been considerable advances in the management of transient ischaemic attack (TIA) in recent years. It has been proposed that the distinction between TIA and stroke be changed from a time-based to a tissue-based definition [1]. In addition, prospective studies have shown a high risk of stroke within the immediate hours and days after TIA [2]; prognostic scores have been developed to identify patients at particularly high risk to facilitate triage to hospital care [3] and urgent specialist assessment and treatment with secondary preventive medication has been shown to reduce stroke risk in the acute phase. Neuroimaging in TIA has underpinned these developments.

Defining and distinguishing between TIA and stroke

TIA was originally defined in the 1970's by the World Health Organization (WHO) and National Institute of Neurological and Disorders as "an acute loss of focal brain or monocular function with symptoms lasting less than 24 hours, of presumed vascular cause". The arbitrary cut-off of 24 hours to distinguish TIA from stroke is believed to have been set because brain infarction was considered unlikely when symptoms were short-lived. This "traditional" or time-based definition has the advantage of an informative differential diagnosis and patients presenting with transient focal or global neurological symptoms ("suspected TIA") can be worked through a well recognised diagnostic algorithm to exclude common TIA mimics such as migraine, seizure, syncope and anxiety.

However, in 2002, a new "tissue-based" definition was proposed and has since been adopted by the American Stroke Association [1]. It was argued that the traditional definition was inconsistent with other definitions in clinical medicine, was illogical because symptom duration is a poor marker for the presence of brain infarction, that 24 hours is an unrepresentative cut-point for the continuum of symptom duration and that the concept of time-based definition may hinder the delivery of acute treatment. TIA was redefined as an episode of neurological dysfunction caused by focal brain or retinal ischaemia without evidence of acute infarction on brain imaging. Events which had evidence of infarction (regardless of the duration of symptoms) were classified as stroke. The imaging modality of choice for identifying infarction is diffusion-weighted magnetic resonance imaging (DWI).

This new definition shifts TIA from being a condition diagnosed at the bedside, on clinical grounds to one diagnosed by neuroimaging. The definition has been widely adopted in routine practice in North America, but adoption in Europe has been slower and in the UK, the time-based definition is still in use.

DWI findings in TIA

DWI is an advanced magnetic resonance imaging (MRI) technique which relies on changes in the Brownian motion of water molecules to generate signal contrast. During early ischaemia causing cytotoxic oedema, when water molecules shift from the less restricted extracellular environment into the more restricted intracellular environment, there is decreased water proton movement. Reduced proton diffusion leads to a bright, high-signal DWI lesion. In conjunction with the apparent diffusion coefficient (ADC), DWI is therefore more sensitive for early infarction, when compared to conventional MRI techniques.

Many studies of the association between DWI abnormalities and TIA features (defined by time-based criteria) have been performed, with rates of DWI positivity varying from approximately 10 to 50%, depending on study methods and patient characteristics. If scanned within 24 hours of symptoms, approximately 50% of patients with TIA have a focal abnormality on DWI. Of these, 25% do not have a lesion correlate on T2-weighted MRI. Rates of DWI positivity fall with increasing delay to scanning. Presence of DWI abnormalities is associated with some clinical characteristics of the presenting event. For example in a systematic review of all studies reporting DWI findings and clinical characteristics of presenting TIA, symptom duration over one hour,

Correspondence:
Matthew F. Giles, DPhil
Stroke Prevention Research Unit
NIHR Biomedical Research Centre
John Radcliffe Hospital
UK-Oxford OX2 6HE
United Kingdom
matthew.giles@clneuro.ox.ac.uk

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Table 1 Elements of the ABCD3-I score [6].

Clinical features available at bedside evaluation		Investigation findings available at specialist assessment	
Age ≥60	1		
Blood pressure SBP ≥140 mm Hg or DBP >80	1		
Clinical features Limb weakness or Speech disturbance	2 or 1		
Duration of symptoms >1 hour or 10–60 minutes	2 or 1		
Diabetes	1		
"**D**ual" TIA*	2		
		>50% carotid stenosis	2
		Infarction on DWI	2
Total	9		13

* Dual TIA defined as preceding TIA within 7 days of presenting TIA.

dysarthria, dysphasia and weakness were all significantly associated with abnormalities on DWI as were atrial fibrillation and carotid stenosis >50%, while hypertension, diabetes and patient age were not [3].

Prognostication

Stroke risk following TIA (defined by time-based criteria) has been found to be in the region of 3% at two days and 5% at seven days [2]. Risk scores (ABCD2 score) based on clinical features available at the time of initial assessment in the emergency department or primary care have been developed to identify those at highest early risk [3]. In addition, other factors based on investigation findings have been found to be associated with high early risk, including infarction on DWI and large artery disease (LAA) as the mechanism of TIA.

Until recently, the interaction between these different prognostic factors has been uncertain. For example, motor weakness and symptom duration of over one hour are elements of the ABCD2 score, but are also associated with abnormalities on DWI while patients with TIA caused by LAA are at a high risk of early stroke and are also more likely to have DWI abnormalities. Small study size and inconsistent methodology has hindered the development of a composite prognostic score.

However, in a recent large, multi-centre study, the ABCD2 score was found to be a weak predictor of brain infarction [5]. Despite this association, the addition of brain infarction and carotid stenosis to the ABCD2 score (ABCD3-I score, see table 1) was found to perform better in terms of prognostication of early risk than the ABCD2 score alone. [6].

The management of patients to define prognosis following TIA should therefore follow a staged approach. Firstly clinical features available at initial bedside evaluation should be used and the risk estimate should then be refined using the results of investigations identifying brain infarction and TIA mechanism.

Key words: diagnosis: TIA; MRI; DWI

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