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Transient ischemic attack with deceptive presentation

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Abstract

A patient with an initial diagnosis of TIA presented with a deceptive course of events and unfavorable outcome. Only a non-enhanced brain CT was performed initially. Hemodynamic studies were done later on and revealed internal carotid artery (ICA) dissection on CT-angiography and evidence of irreversible ischemic changes on perfusion studies indicating that the ischemic process presumably was ongoing since the initial ischemic episode. Efforts to define patients with TIA with risk of developing major stroke are ongoing. We hereby exhort for more effort to include hemodynamic studies as early as possible in the radiological work-up of TIA.

Key words: TIA; hemodynamic studies; radiological work-up; perfusion CT; CT-angiography.

Introduction

Transient ischemic attack (TIA) is classically defined as a sudden, focal neurological deficit that lasts for less than 24 hours. Albers et al proposed a new definition omitting the arbitrary 24-hours time limit, defining TIA as brief episode of neurological dysfunction caused by focal brain or retinal ischemia, with clinical symptoms typically lasting less than one hour without objective evidence of acute infarction (Albers *et al.*, 2002). Computed tomography (CT) is the method of choice in the initial radiological work-up of patients with suspected acute ischemic episode. CT-angiography (CTA) or MR-angiography help to define and assess the degree of arterial stenosis and is an essential step in selecting suitable patients for urgent reconstructive carotid surgery. Perfusion CT (CTP) or MR-perfusion (PWI) may help to delineate early ischemic changes e.g. regions of tissue at risk (penumbra).

We report here on a patient who initially had a clinical TIA presentation, but a deceptive course and an unfavourable outcome which were the consequence of the delayed use of multimodal CT.

Case report

A 61-year-old healthy non smoker man with slight untreated hypertension was admitted to the emergency unit (ER) with a history of sudden onset of speech disturbance and left sided hemiparesis. The patient recovered within 2 hours. On admission no neurological abnormality was found. Plain brain CT performed three hours after the ischemic episode was normal. As the patient had an ABCD score of 6 (Rothwell *et al.*, 2005) with an approximate 35% calculated risk of impending major stroke, he was admitted to the ER-ward and scheduled for a thorough clinical and radiological work-up the next morning. Neurological examination performed on the next morning (13 hours after the first TIA) revealed no neurological deficit. Thirty minutes later the patient developed left sided hemiparesis and left-sided hemianopia. Urgent CT showed small cortical and subcortical infarcts in the right frontal and the parietal lobes (Fig. 1A). CTP showed ischemic changes consistent with irreversible ischemic injury [interpretation according to that proposed by (Tomandl *et al.*, 2003)] in the right middle cerebral artery (MCA)-territory (Fig. 1B-D). The CTA findings were consistent with ICA dissection with secondary thrombosis at and above the level of dissection (Fig. 1E-F). Based on CTP- and CTA-findings a decision was made to treat the patient with intra-arterial thrombolysis (started 2 hours after stroke onset) to try to establish a revascularization, primarily of MCA. Unfortunately revascularization of MCA could not be achieved and the patient developed a malignant MCA-infarction that necessitated craniotomy. The final diagnosis was extensive thromboembolic stroke secondary to ICA dissection.

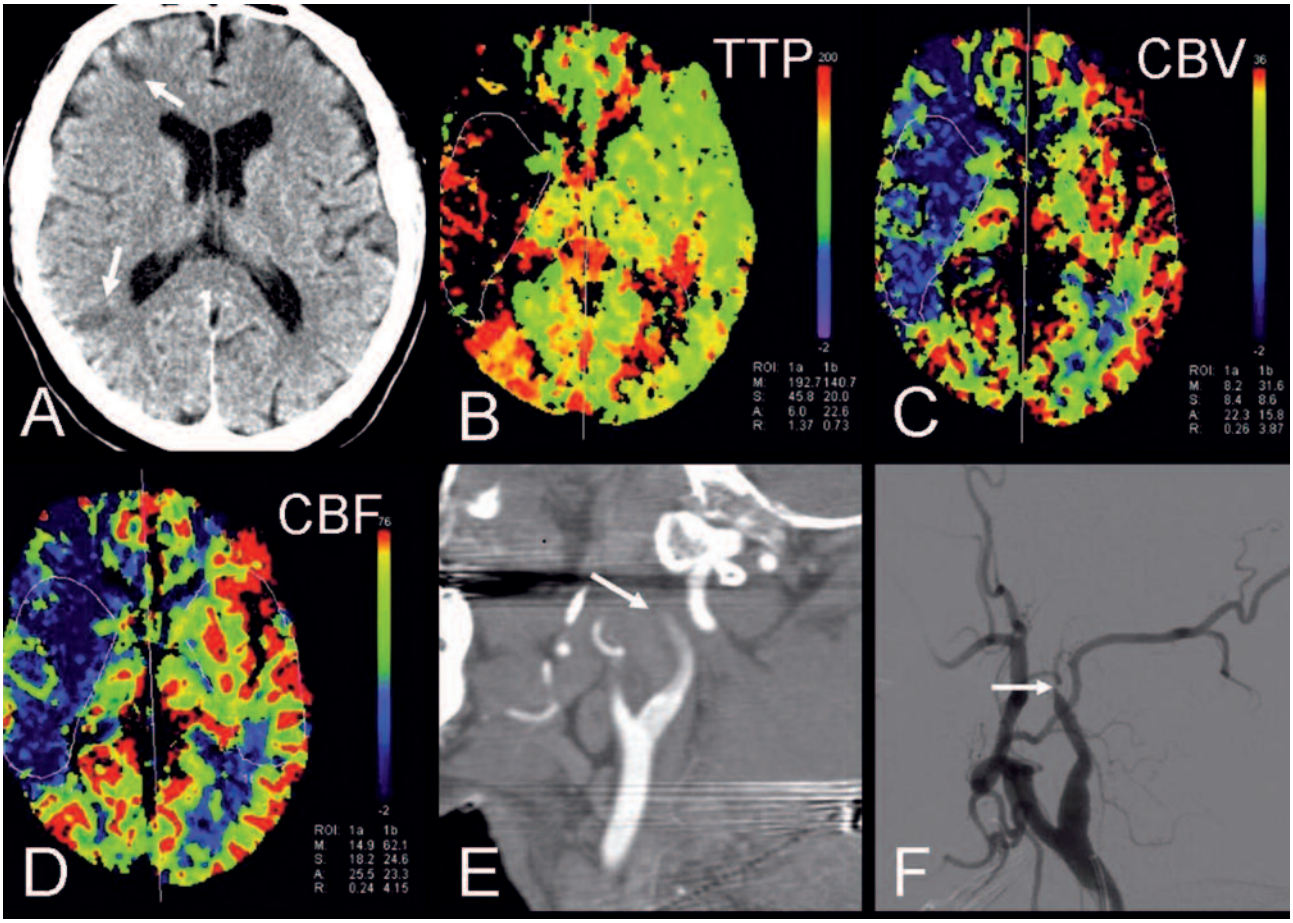

FIG. 1. — (A) Plain CT at time of the second TIA shows small infarcts (arrows). (B-D) perfusion maps showing prolonged TTP and markedly reduced CBV and CBF in the MCA-territory on the right side. CBV and CBF of the affected right side were 26% and 24%, respectively of the corresponding values of the healthy left hemisphere. These findings represent irreversible ischemic changes according to the interpretation proposed by (Tomandl *et al.*, 2003). (E-F) CTA sagittal MIP reconstruction and digital subtraction angiography showing a string-shaped occlusion (arrow) of ICA suggestive of dissection.

At 3-month control the patient had a modified Rankin Scale of 5.

Discussion

This is a case report of one patient with TIA with deceptive course and unfavourable outcome. The clinical outcome might have been improved if further imaging modalities were performed in conjunction with the first TIA. At the moment the patient underwent multimodal CT the clinical diagnosis was a second episode of TIA. The finding of an extensive ICA thrombosis on CTA and of new infarctions on the second plain CT and CTP-changes corresponding to irreversible ischemic lesions suggest that the ischemic process had been in progress presumably since the first TIA despite the fact that stroke symptoms began only few minutes before the perfusion study (as the patient was admitted to the ER ward). An important lesson one could learn from this case report is that patients with TIA might exhibit perfusion abnormalities despite obvious clinical recovery. Although there are different scores to predict which patients with TIA might develop cerebral infarctions (Johnston *et al.*, 2000; Rothwell *et al.*, 2005; Johnston *et al.*, 2007), and despite previous reports on impending stroke following TIA (Itoh *et al.*, 1988; Newman, 2008) as well as reports showing occurrence of perfusion abnormalities in TIA (Lu *et al.*, 2005; Abul-Kasim *et al.*, 2009), there is still no definite benchmark either to guide the admission and management or to define an algorithm for the radiological work-up of TIA. As suggested in literature (Giles *et al.*, 2005), our case report supports the

importance of performing risk stratification at the initial presentation using different available scores and subsequent use of different diagnostic modalities. The use of these modalities varies widely depending on the policy in different institutions, awareness of usefulness of these modalities as well as availability of equipment, competent radiographers and radiologists. Other modalities widely used in the work-up of TIA are SPECT and MR-perfusion. The latter has the advantage of providing a whole brain perfusion map and in combination with diffusion-weighted MRI it can document the presence of ischemic lesion in approximately 50% of patients with TIA (Mlynash *et al.*, 2009). However, the main drawbacks of MRI are its availability, higher cost, and longer examination time. Despite its greater sensitivity compared with CT, SPECT performed without the acetazolamide challenge was reported to provide no additional information with regard to the vascular risk factors and etiology in TIA (Laloux *et al.*, 1996). The distinction between TIA and cerebral infarction has to be compared to angina pectoris and myocardial infarction and a repeated TIA need to be considered as serious entity as an instable angina. As our case report showed TIA is a serious neurological event that warrants immediate diagnostic and probably therapeutic measures. From the diagnostic point of view we believe that inclusion of CTA and CTP in the initial work-up of TIA is a reasonable algorithm. However, this might need to be validated by controlled trials that assess the added utility of these modalities in the management of TIA.

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