Cerebral Infarction With Transient Signs (CITS): Do TIAs Correspond to Small Deep Infarcts in Internal Carotid Artery Occlusion?

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SUMMARY Among 75 patients in whom internal carotid artery (ICA) occlusion was discovered on angiography, 5 presented with transient ischemic attacks (TIAs) without suffering a stroke. Although neurological examination was normal, all had evidence for one (in one instance two) hypodense lesion suggesting infarction contralateral to the neurological dysfunction on computed tomography (CT). These infarcts were small and deeply located, being indistinguishable from lacunes in most cases. We suggest that cerebral infarction with transient signs (CITS) may be a usual finding in patients with ICA occlusion who suffer isolated TIAs. In these cases, CITS may correspond to incomplete cerebral necrosis related to a well-developed collateral supply, or to recurrent ischemia in the region of an old “silent” infarct. CITS should be differentiated from TIAs, which may be diagnosed only in absence of visible structural lesion.

Patients and Case Reports

During the last six years 75 patients (62 3:13 5, mean age 58 years) with angiographically proved occlusion of one or both ICAs have been studied in our department. Among these, 38 were admitted after a stroke, 24 after TIAs and/or amaurosis fugax followed by a stroke, 5 after isolated TIAs (with or without alternating amaurosis fugax), 6 after isolated amaurosis fugax, and 2 were asymptomatic. All these ischemic events were ipsilateral to the occluded ICA, except one brainstem stroke.

Among the 2 women and 4 men who suffered isolated amaurosis fugax, five underwent a CT scan (as a routine procedure before angiography) within one month after admission, without any demonstrable infarct. On the other hand, the 5 patients who experienced isolated TIAs (with or without associated amaurosis fugax) showed one (in 4 cases) or two (in 1 case) deep infarcts contralateral to the symptoms (pure motor hemiparesis in 3 cases, aphasia and hemiparesis in 1 case, ataxic hemiparesis in 1 case). None of these patients had suffered an anterior stroke.

Case 1

This 60-year-old man suffered chronic hypertension and smoked 20 cigarettes/day. He had been taking atenolol and prazosine for many years. He suddenly experienced speech disturbances with word-naming difficulties, associated with weakness of the right limbs and inferior facial paresis on the right. These symptoms cleared up in three hours and the next day a neurologist reported a completely normal examination, except a right carotid bruit. Blood pressure was 180/110 mmHg, without cardiac dysrhythmia. CT (fig. 1), performed after three weeks, showed infarction of the lenticulostriate arteries area on the left, clearly sparing the area of the anterior choroidal artery. Arch angiography showed bilateral occlusion of ICA. Repeated neurological and neuropsychological examinations showed no abnormality.

Case 2

This 52-year-old hypertensive and smoking (20 cigarettes/day) man suffered recurrent episodes of amaurosis fugax in the right eye alternating with transient weakness of the left limbs (duration: 15 min.). After three weeks, he was referred to our hospital, where neurological examination was normal. Blood pressure was 165/80 mmHg, with a regular pulse. CT (fig. 2) showed a small deep infarct in the lower part of the right corona radiata. A four-vessel angiography showed occlusion of the right ICA and irregularities of the vertebral arteries, with good collateral supply through external carotid artery (ECA) pathways and posterior communicating artery.

Case 3

This 72-year-old man was in good health until he experienced 3 episodes of right hemiparesis involving the face over one month, of 5 minutes duration. Neurological examination was normal. Blood pressure was 160/80 mmHg. CT showed a small deep infarct in the lower anterior part of the left corona radiata. Arch
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FIGURE 1. Case 1. CT scan showing a deep infarct in the left lenticulostriate artery area.

carotid angiography showed occlusion of the left ICA with good collateral supply through ECA pathways (Fig. 3).

Case 4
This 66-year-old man suffered hypertension and smoked 15 cigarettes/day. He experienced one episode of amaurosis fugax in the right eye (duration: 10 minutes), followed after 2 days by a transient (1 hour) weakness of the left hand. He was admitted the same day, and neurological examination was normal. Blood pressure was 160/100 mmHg. CT, performed after 2 days, showed two small infarcts in the white matter of the anterior part of the right hemisphere. Arch angiography showed occlusion of the right ICA (fig. 4).

Case 5
This 71-year-old man was in good health until he experienced weakness and clumsiness of the left upper limb, and, to a lesser extent, of the lower limb. The patient was admitted three hours after the beginning of the symptoms and left ataxic hemiparesis was diagnosed. The following day neurological examination was normal, without any more ataxia or weakness of the left limbs. CT showed a small infarct in the posterior limb of the right internal capsule. Arch angiography showed occlusion of the right ICA (fig. 5).

Discussion
Five patients showed TIAS without suffering a stroke among a series of 75 patients in whom ICA occlusion was discovered on angiography. All had a positive CT scan with a hypodense lesion (in one case two lesions) contralateral to the symptoms. Atrophic changes were not conspicuous. On the other hand, CT scan was normal in the five patients who suffered isolated amaurosis fugax and in whom this examination was performed.

CT has already been studied in cases with TIAS of varied origin. Some authors described normal CT scans or incidental findings: Kinkel et al\(^7\) reported 30 normal CT scans and 2 showing diffuse atrophy in 32 patients with TIAS, Constant et al\(^8\) reported 16 patients with TIAS who had a normal CT scan, Laporte et al\(^9\) reported 22 patients with TIAS who had a normal CT scan, and Biller et al\(^10\) reported 32 normal CT scans, 9 showing cortical atrophy, 3 incidental findings, and 1 a hypodense lesion corresponding to the site of neurological dysfunction, among 45 patients with TIAS. On the other hand, other studies more frequently described focal hypodense lesions in patients with isolated TIAS: Perrone et al\(^2\) reported hypodense lesions in 12 patients of a series of 35 with TIAS, Ladurner et al\(^3\) studied 44 patients with TIAS, with hypodense lesions in 8 and atrophy (most often diffuse) in 19, Allen et al\(^4\) reported an abnormal CT scan in 6 of 52 patients with TIAS, and Donnan et al\(^5\) reported hypodense lesions in 13 patients of a series of 25 with TIAS. In this latter study the localization of hypodense lesions was not discussed, but the authors stated that a deep lesion was most likely to be found in patients with repeated bursts of hemiparesis, which they called the “capsular warning syn-
drome." Goldenberg et al also reported 3 cases with a small hypodense lesion among 15 cases suffering TIAs.

These studies suggest that transient neurological dysfunction may in fact not uncommonly occur in the setting of cerebral infarction. This phenomenon was recently discussed by Waxman and Toole who called it "cerebral infarction with transient signs (CITS)." They suggested that some acute infarcts may in fact give rapidly reversible neurological signs, or that old "silent" infarcts may produce a transient dysfunction related to a new episode of borderzone ischemia. However, these authors stated that this phenomenon remained poorly explained, and may correspond to specific vascular lesions with a specific prognosis.

CITS has not been studied in patients with ICA occlusion, except in patient 1 of Waxman and Toole in whom a small infarct in the white matter of the hemisphere contralateral to the hemiparesis was present. Isolated and presenting TIAs have been reported in ICA occlusion series, but without CT data. In our study, all the patients with ICA occlusion who were admitted for investigation of TIAs without stroke had one (or two) small hypodense lesion in an area corresponding to the neurological disturbances. This may suggest that among patients presenting with "TIAs", CITS may be particularly frequent in those with ICA occlusion. It must also be emphasized that all the visible hypodense lesions were small and deep, and in fact could have been diagnosed as lacunes, also because the patients showed "lacunar" syndromes as motor/ataxic hemiparesis. Lacunes may occasionally produce a "TIA," with normal angiography, but small deep infarcts may not always be lacunes. Actually, Mohr stated that deep infarcts due to microembolization from atheroma of the extracranial arteries are not true lacunes, but are often not distinguishable from true lacunes without histological studies. Also, Pullicino et al, who studied 42 patients with small deep infarcts diagnosed on CT found that "in a large number, a source of emboli from either a cardiac or a carotid source was highly probable." In one study, isolated small deep infarcts in the carotid area have been reported in five of 26 patients (19%) with proved ICA occlusion, and in 13 of 60 patients (22%) in another study. It is highly improbable that most of these infarcts are lacunes, but they probably correspond to embolic or hemodynamic ischemia directly related to occlusion of the corresponding ICA.

Our study firstly suggests that CITS is most likely to be found in small deep infarcts resembling lacunes on CT scan. Why do some of these infarcts give rise to transient disturbances, or even remain "silent", in certain cases, while others produce a clear clinical stroke, is a question difficult to answer. One possibility is that the visible hypodense lesion does not correspond to complete neuronal necrosis, and that viable cells remain among the ischemic zone in sufficient number to restore a normal function after the acute phase has resolved. Histological studies would thus be of great interest in these cases. Another possibility is that other parts of the brain may take over the function of the infarcted area, but this seems rather unconvincing in lesions of the internal capsule, and such a functional replacement would probably take place over a much longer period. The fact that CITS may be par-
particularly frequent in ICA occlusion patients who present with 'TIAs' may also be worth considering, because of progressive adaptation of collateral circulation during the process of progressive stenosis that develops into occlusion. In our 2 patients in whom intracranial circulation was studied on angiography, collateral supply to the area distal to the occluded ICA was excellent. The presence of a good collateral system could be one of the factors that would allow some cells to survive in an infarcted area.

Further studies should focus on angiographic data, in order to evaluate what spectrum of vascular lesions underlies CITS and to determine in what conditions without ICA occlusion CITS may occur. Presently, the term TIA should still be reserved to describe clinical episodes of acute ischemic neurological dysfunction which resolve within 24 hours and in which CT shows no evidence for infarction.

References

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