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## Transient global amnesia and cerebral infarct: a case report

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Transient global amnesia refers to a sudden and isolated dysfunction of memory for recent events, lasting a few hours. The pathogenesis of this neurological disorder is still uncertain. The most accepted hypotheses concern ischaemic, epileptic and migraine causes. We now report a case of transient global amnesia associated with computed tomography evidence for a hypodense area in the left thalamus 10 days after the transient memory dysfunctions.

### Introduction

The term transient global amnesia (TGA) refers to a sudden and isolated dysfunction of memory for recent events, lasting a few hours [1]. The following criteria were recently set by Caplan for the correct diagnosis of TGA: (1) the attack onset should be witnessed; (2) dysfunction during the attack should be limited to repetitive queries and amnesia; (3) there should be no other major neurological signs or symptoms; (4) the memory loss should be transient, lasting no more than 48 hours [2]. Attacks generally last from 20 minutes to a few hours, and only occasionally have repetitive attacks been reported [3].

The aetiology of this disease is unclear at present, and vascular [4, 5], epileptic [6] and migraine [7] mechanisms have been proposed. Moreover, TGA has also been described in association with brain tumours [8, 9] and head trauma [10]. We now describe a patient with TGA in whom computed tomography (CT) showed a cerebral infarct in the left thalamus 10 days after the transient memory dysfunctions.

### Case report

A 58-year-old right-handed, diabetic and dyslipidaemic housewife with no history of neurological diseases suddenly started asking her daughter repetitive questions about a 'family argument' although she had already discussed this matter on various occasions. She could also not remember what she had been doing since breakfast the same day. The woman did not complain of headache or any other neurological symptom.

The housewife was admitted to the Institute of Neurosciences of Catania 2 days after this acute episode. Neurological and general examinations were normal. Clinical history revealed that the patient had suffered no prior traumatic injury, and had never suffered psychiatric diseases or other acute neurological dysfunctions, including disturbances of conscious level or cognitive dysfunction. The clinical evidence for the diagnosis of TGA

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comprised reversibility of the syndrome, inability to lay down new memories during the attack, variable retrograde amnesia, amnesic lacuna persisting after the episode and resolution within 24 hours.

*Table 1. Neuropsychological studies*

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Wechsler Adult Intelligence Scale, revised
Temporal Orientation Test
Rey Auditory Verbal Learning Test
Wechsler Memory Scale
Boston Famous Faces Test
Rey's 15 Words
Digit
Corsi's Cube Test
Barrage Attention Test
Benton Visual; Retention Test (Adm. A, Form C)
Orientation on a Geographic Map
Stroop Test

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*Figure 1. Patient with focal lesion consisting of a left thalamic lacuna.*

Various neuropsychological tests (listed in Table 1) were administered to the patient on the following day, and showed no peculiar findings. ECG was normal and Doppler ultrasounds of the extracranial vessels showed no evidence of stenosis.

The EEG showed theta and delta waves over the left hemisphere. A brain CT scan performed 10 days after the admission showed a focal lesion consisting of a left thalamic lacuna (Figure 1). A follow-up CT was executed 3 months after the event and showed no visible lesions.

The patient was discharged on ticlopidine (250 mg/day) and followed as an outpatient for 6 months. During this follow-up period the patient had no recurrence.

### Discussion

There are various hypotheses concerning the pathogenesis of TGA. The most valuable suggestions are migraineous, epileptogenic and ischaemic. The migraineous hypothesis, that states that TGA is a manifestation of an abnormal redistribution of cerebral flow during the vasodilatory phase of migraine, has been suggested by various authors [7, 11] but Kushner and Hauser found no association between TGA and migraine in their case-control analysis [12]. Miller *et al.*, in a retrospective study on 277 patients, found that 14.1% had a history of migraine [13]. The abruptness, shortness and reversibility of the disturbance, and the finding of some EEG anomalies, supports the epileptic origin of TGA [6]. Miller *et al.* found that only 2.9% of patients with TGA had seizure disorders [13]. The ischaemic theory suggests that TGA is due to a transient vascular deficit, mainly of the posterior cerebral arteries, in the brain regions serving memory. In the above-cited paper by Miller *et al.* the patients with cerebrovascular diseases comprised 11.7% [13].

The case we now report meets the requirements set by Caplan for the diagnosis of TGA, since the attack onset was witnessed, the dysfunction was transient and limited to amnesia and repetitive queries, with no other neurological signs or symptoms. The CT scan showed a cerebrovascular lesion consisting of a left thalamic lacuna, and suggests that, of the above hypotheses, the ischaemic one is most consistent with our case report.

The thalamic lesion is consistent with the amnesia since diencephalic structures, and the dorsomedial nucleus in particular, appear to play an important role in memory dysfunctions. Since not all the lesions of the medial thalamus are associated with amnesia it is argued that, to produce a memory deficit, either the amount of the dorsomedial nucleus damaged must be above a certain threshold or a second region, such as the mammillothalamic tract or the anterior nucleus, must also be damaged [14].

In conclusion, TGA can be the isolated manifestation of a cerebrovascular event involving diencephalic structures.

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