

LETTERS TO THE EDITOR

Abnormal cerebral blood flow following transient global amnesia

Transient global amnesia^{1,2} (TGA) is an episodic dysfunction of declarative memory (acquisition of facts, knowledge, and events which are directly accessible to consciousness awareness) for recent events, without neurological signs or symptoms. The patient with TGA hardly recalls either verbal or non-verbal materials presented minutes before, and often repeats the same question many times. We report the changes of cerebral blood flow in two patients with pure TGA studied with technetium 99m (Tc-99m) hexamethylpropyleneamine oxime (HMPAO) single photon emission CT (SPECT).

Patient 1 was a 45-year-old, right-handed office worker with no significant medical history. On 27 August 1991, he came home from his office at 7 pm and asked his family members why he was there and what he was doing there. The witnesses answered, but he repeatedly asked the same questions. His wife took him to our hospital at 8 pm. He was very anxious and repeatedly said that there was something wrong with him. His blood pressure was 134/88 mm Hg and his pulse was regular, at 68 beats per minute. The neurological examination was normal except for severe memory impairment. He could not recall what he had eaten for breakfast that day. His ability to retain new information was severely impaired. Verbal expression, comprehension, and immediate repetition of sentences and words were normal. EEG and MRI of the brain were normal.

He slept well and got up at 7 am the next day. Over the 12 hours from the onset of amnesia, he regained his ability to retain new information, and recovered completely. Cerebral blood flow measurement using 740 MBq of Tc-99m HMPAO³ was done 15 hours after the onset of the attack. The SPECT system was a rotating gamma camera (Toshiba, GCA-901A), combined with an image processing minicomputer (Toshiba, GMS-550U). Acquisition of projection data began five minutes after injection. Data was accumulated for 60 angles (6° step, 360°) with 30 s per angle. Projection data were processed with Butterworth filters. The results of cerebral blood flow measurement showed abnormally low blood flow in the left inferior temporal lobe cortex (fig). A follow-up study done 15 days after onset showed that blood flow in the left inferior temporal cortex had returned to normal (fig).

Patient 2 was a 54-year-old, right-handed housewife with a six-year history of hypertension who suddenly became confused. She had no history of convulsion or migraine. On 12 August 1991, she went swimming in the morning and then returned home. At 2 pm, she asked a family member what she had done that morning. The witness told her what she had done but she repeated the same question. She was told to go to sleep and she went to the bedroom, but she soon came out and asked why she was in a bedroom. Her son took her to our hospital.

We examined her two hours after the onset of memory impairment. Her blood pressure was 164/96 mmHg and her pulse was regular, at 68 beats per minute. The neurological examination was entirely normal except for memory impairment. She could not recall what she had done the previous day. She was unable to retain new information. Verbal expression, comprehension, and immediate repetition of sentences and words were normal. Over the 19 hours from the onset of amnesia, she completely recovered. She could not recall the events of the previous two days or during the attack. EEG during the amnesia was normal. MRI of the brain was normal. Cerebral blood flow measurement was done 20 hours after the onset of the amnesia with Tc-99m HMPAO SPECT as described above. The results showed abnormally low blood flow in the right hippocampus and bilateral thalamus (fig). A follow-up study nine days after onset showed low cerebral blood flow in both hippocampi and thalamus (fig).

The two patients presented here had cerebral blood flow abnormalities soon after the onset of TGA. It is widely accepted that amnesia in TGA is transient and recovery is complete. There have been a few reports of pure TGA cases, however, in which cerebral blood flow change was clearly shown by early and follow-up SPECT studies; Goldenberg *et al*⁴ showed transient ischaemia in the thalamus, and Matsuda *et al*⁵ showed transiently increased perfusion in the left medial temporal lobe including the hippocampus. Our results suggest that both early and follow-up examinations are necessary for studies of the mechanism underlying TGA.

The underlying mechanism of TGA remains unclear. Several aetiologies have been proposed: epileptic seizure, ischaemic cerebrovascular disease, migrainous vasospasm of posterior circulation, and increased neurotransmitter levels. None of these is entirely satisfactory. Caplan¹ proposed the concept of acute arterial dyscontrol. He postulated that acute arterial dyscontrol causes TGA by a transient self-limited alteration of vascular tone in the

posterior circulation. In our report, patient 2 had abnormal cerebral blood flow in the hippocampus and in the thalamus. This is consistent with Caplan's theory. Patient 1 had transient abnormal flow in the temporal cortex. Caplan¹ hypothesised that blood flow changed only in the posterior circulation, so the findings in patient 1 seem inconsistent with his theory because the temporal lobe receives blood from the middle cerebral artery. Regional cerebral blood flow, however, has been found to be frequently abnormal in the temporal lobe.^{5,6}

We believe that transient cerebral blood flow reduction in the left temporal lobe caused the transient amnesia in patient 1. Cerebral blood flow measurement with SPECT soon after the onset of TGA, and again after recovery will give useful clues for further investigations of TGA.

YASUO SAKASHITA

Department of Neurology

TATSUHO SUGIMOTO

Department of Internal Medicine

SUZUKA TAKI

Department of Radiology, Tonami General Hospital,

1-61 Shintomi-cho, Tonami, Toyama 939-13, Japan

HIROSHI MATSUDA

Department of Nuclear Medicine, Kanazawa

University School of Medicine, Kanazawa, Japan

Correspondence to: Dr Sakashita

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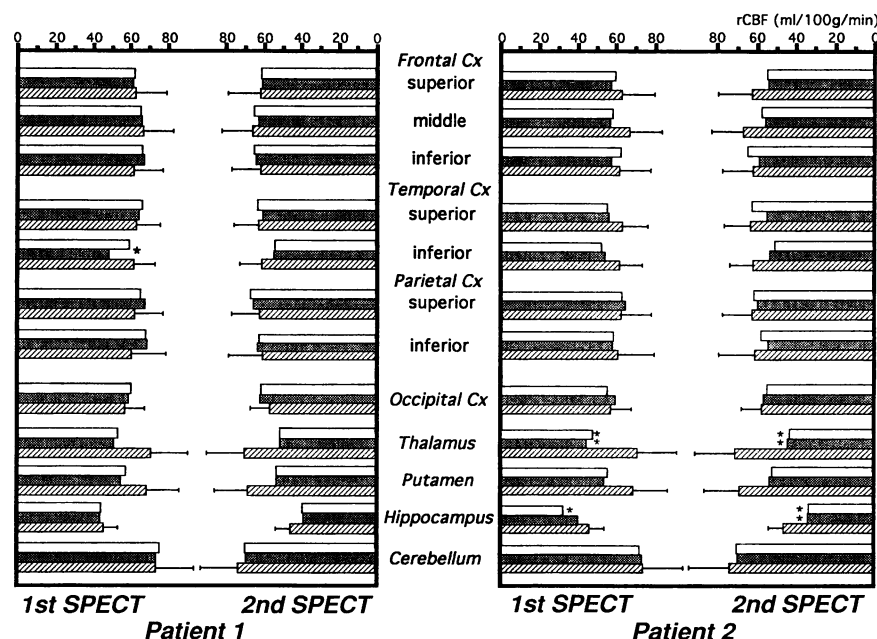


Figure Results of cerebral blood flow (CBF) measurements. Normal values were obtained from 10 age-matched normal volunteers. *Cerebral blood flow lower than mean ($-3SD$) of normal value. (□) right brain; (■) left brain; (▨) normal [mean ($3SD$)] value.