Transient Global Amnesia

Report of Two Cases with Special Reference to Associative Symptoms

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INTRODUCTION

Transient global amnesia was first reported by Bender[1] in 1956 as isolated confusion with amnesia. This syndrome was later named "transient global amnesia" (TGA) by Fisher and Adams[2]. It is characterized by a transient loss of ability to register recent memory with a variable period of retrograde amnesia. It usually affects previously healthy individuals in the sixth to eighth decades of life. About two thirds of the patients are women and most patients have hypertension. Therefore, many authors have suggested that this syndrome is caused by a temporary vascular insufficiency in the memory center of the central nervous system.

We have recently encountered patients with characteristic symptoms of TGA. The purpose of this paper is to present these cases and to discuss pre- and postepisodic features.

CASE REPORT

Case 1: S.S., a 45-year-old employee of a chemical company was seen by us on Nov. 25, 1975. She had no family history of epilepsy or other neurological diseases. She had been hypertensive since she was 28, due to pregnancy kidney. She used to have common headaches for long periods of time. Occasionally they became so severe that she could not sleep at all during the night. On Oct. 14, 1975, she had experienced a severe frontal headache. On Oct. 15, she was called into court in order to accompany her son, who was then a defendant in a case involving a traffic accident. On Oct. 17, she had an amnestic episode while at

work. One week prior to the episode, she had had heavy sleepiness during the day. She went to Hiroshima to receive a medical examination on Oct. 20, which she could remember to some extent, although it was vague. She could not recall precisely with whom she went to Hiroshima or by what kind of transportation. On Oct. 24, she was admitted to the section of internal medicine, Syuto Hospital to receive further medical care. After the admission, she could state exactly the events which occured in the hospital thereafter. Clear memory returned on Oct. 26. Total amnesia took place on Oct. 17 and lasted about 12 hours. During this period, she could not retain recent memory and seemed to be confused. On that day, in a cosmetic shop, she repeated the same questions over and over, and also said repeatedly to the saleswoman that she had bought so much that she must have a right to receive some additional service.

Physical examination revealed: High blood pressure, especially of the diastole (164/108). Her pupils reacted to light normally. Deep tendon reflexes were slightly exaggerated on both sides. No pathological reflexes or clonus were elicited.

Laboratory examinations revealed: Normal serum lipid level; total cholesterol: 184 mg/100 ml; beta lipoprotein: 308 mg/100 ml; neutral fat:113 mg/100 ml; phospholipids:199 mg/100 ml; free fatty acid:181 mEq/1; blood sugar:88 mg/100 ml; ASLO:100 Todd's unit; C-reactive protein:negative; serum serological test for syphilis:negative. Electroencephalogram: Predominant basic activity was low voltage fast alpha rhythm with low voltage beta activity possibly due to the influence of tranquillizers. No slow activity or spike discharges were observed. Blood pressure was still hypertensive (172/100 mm Hg) on Dec. 2.

Case 2: F.M., a 59-year-old female office-worker. At 41 years of age, she had muscle weakness in her lower extremities and was admitted to a hospital for one month. The pathogenesis remained unclarified but the symptoms had vanished spontaneously. Since 54 years of age, she had had hypertension (170/100 mm Hg) and left-sided migraine headaches on occasion. Otherwise, she was entirely healthy and well. Family history was negative for any hereditary, neurological or psychological disease.

On Dec. 13, 1975, while visiting her at work, her husband noted her abnormal behavior. She seemed to be as usual at first sight, however, she asked the same questions at intervals of about five to ten minutes. She asked repeatedly: "Why is this letter here?"; "What is the content of this envelope?" She also asked "Who drank it?", looking at an empty bottle of cocacola. Conversation, writing, and calculational

ability seemed as usual, according to her husband. She was ordered to be confined to bed. On Dec. 18, while having dinner, she suddenly began to weep. She said that she could not understand why she was weeping. Unclear memory registration seemed to last from the 13th to 17 the of December. Within this period, total amnesia took place in the afternoon of the 13th, lasting about 6 hours. She was referred to our clinic on Dec. 19, 1975, when she complained of an uneasy sensation in her head. She had anxiety about the memory loss which she experienced. Except for the period of total amnesia, hazy memories about December 13-17 were occasionally recalled. She became completely clear after Dec. 18th.

Physical examination revealed: No remarkable abnormalities, except for minimal exaggeration of deep tendon reflexes. Electroencephalogram revealed normal alpha activity. Slow waves or spike discharges were not elicited by overbreathing and photic stimulation. Twenty days later, she came to our clinic again. Physical examination at that time showed also no remarkable change. She complained of vague discomfort in the head and feelings of anxiety for the period of amnesia. She stated that she lost her self-confidence concering her physical strength. The patient had showed considerable recovery from retrograde and anterograde amnesia, however, she did not recover from the total amnestic period at all.

COMMENT

Since the first introduction of the term "transient global amnesia (TGA)" by Fisher and Adams in 1958[2], considerably many reports have been published dealing with this syndrome. Typical TGA is characterized by a sudden loss of ability to register recent memory, in association with retrograde amnesia before the amnestic episode. It is said that during the episode, the patient repeats the same questions over and over again. Our two patients had shown these characteristic symptoms. However some different features were also noted.

Common charcteristic features of our cases were: Middle aged females, having occupations, suffered from an attack of amnesia while doing their job in their working place. Both patients had had hypertension for a long time. Case 1 had been hypertensive for about 20 years and case 2 for 5 years. Neurological examinations revealed no remarkable findings. Only the deep tendon reflexes were slightly exaggerated in both cases. No pathological reflexes were elicited at all. Electroencephalogram, recorded one week after the amnestic episode, were not pathological. They recovered within a few days and showed total amnesia

lasting 6 to 12 hours with variable periods of imcomplete amnesia in retrograde and anterograde directions. During the episode, they asked the same questions repeatedly.

However, considerably different features were also recorded between these two cases. In Case 1, the patient had psychic stress before the episode. Her son had caused a traffic accident and injured someone. The court issued a summons on him. Since he was not yet an adult, his mother had to accompany him to court. Her son was ordered to pay a lot of money in compensation for the damage. For one week prior to the onset of the amnestic episode, she had had heavy sleepiness, which seemed to be caused by thalamic-hypothalamic or autonomic dysregulation.

One of the specific symptoms of Case 2 was uncontrollable weeping after the period of amnesia. She herself wondered and felt strange about why trivial matteres made her cry. The other characteristic of Case 2 was a depressive or hypochondriacal state following recovery from the amnestic episode. She complained of anxiety and wanted to keep secret the fact that she had suffered from such an episode.

Case 1 is to be differentiated from psychological amnesia. Concerning this problem, Patten (1971)[3] stated that patients with psychological amnesia were mostly less than 50 years old with a clear psychological situation. Usually they do not ask the same questions repeatedly and do not show a loss of ability to retain recent memories. From this opinion, our case 1 was more plausible as TGA than psychological amnesia. Case 2 had no remarkable psychological stress.

On the pathogenesis, Heathfield et al. (1973)[4] stated that TGA is a syndrome for which there are generally several different causes. In their experience of 31 cases presenting transient amnestic attacks, such causes as TGA, epilepsy, migraine, temporal lobe encephalitis, and psychogenic reaction were accounted for. However, the most frequent cause was, according to them, vascular-hypertension. In Case 2, there was migraine headache, but the symptoms were not of classical migraine.

There is the consideration that if the attacks are due to cerebral ishemia, it is strange that more patients suffering from this condition do not eventually develop cerebral infarction. Moreover, it is strange that recurrent attacks are rarely reported. (Halsey[5]) These considerations are not yet resolved at the present time.

Heathfield et al.[4] suggests the bilateral temporal lobe or thalamic lesion as responsible sites of pathogenesis. Shuttleworth[6] reported cases of TGA with arterial embolism which occured during cardio-angiogra-

phy, and showed the possibility that TGA is induced by arterial embolism in the temporal area of the brain. In our cases, lethargy and emotional lability with uncontrollable crying suggests that the responsible sites of affection would be the thalamus or hypothalamic area in association with amnesia caused by limbic system dysfunction.

Exertion and fatigue are thought to be major precipitating factors according to Heathfield et al.[4] They suggest that the cause of memory impairment is the result of bilateral anterior temporal lesion formation. Transient ischemic attack is the most plausible cause according to them. In our patients, slight elevation of the deep tendon reflexes suggested some impairment of the cerebrovascular system.

Tharp[7] commented on EEG peculiarities, in which he reported paroxysmal sharp waves in the temporal areas. Green and Bennett[8] also reported on EEG abnormality in TGA patients. They found biphasic 50 microvolt spikes or polyspikes in the left mid temporal area. Steinmetz and Vroom[9] reported 4 cases with pathological EEG changes. They considered the most important to be in the hippocampal-fornical-mamillary-diencephalic-cingulate system. As a pathogenetical factor, they also considered embolism. Evans[10] reported change in mood, which consisted of bewilderment or episodes of weeping, as seen in our Case 2, suggesting a lesion in the medial part of thalamus or hypothalamus. Shuttleworth et al.[11] briefly reported on anterograde amnesia. However, this kind of amnesia was rarely reported. Our two cases had had these symptoms for a few days after the end of the true amnesia.

Except for the cardinal symptoms concerning failure in memory, more associative symptoms should be carefully reported, because with them may lie the keys to understanding the pathogenesis of TGA and even the mechanism of memory itself.

SUMMARY

Two cases of transient global amnesia (TGA) were reported. The cases were characterized by middle-aged females with occupations, having onset of TGA in their working place while performing their job. They had a history of hypertension and headaches. There were no physical abnormalities or electroencephalographical changes, except for a slight exaggeration of deep tendon reflexes.

They had experienced total amnesia lasting for a few hours. During these periods, they repeated the same questions over and over again. Before and after the true amnestic period, they showed some different

features. Case 1 had had psychological stress and sleepiness for one week prior to the amnestic episode. Case 2 showed episodes of uncontrollable weeping and depression after the amnestic episode was over.

Past research on the subject, and the problems of associative symptoms were discussed.

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