Unexplained Sudden Amnesia

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Background: In almost all cases of acute, sudden, persistent amnesia, the cause can be determined. Ischemic stroke, hypoglycemia, syncope, and seizure are the most likely causes.

Patients and Methods: In a clinical study, 2 elderly men are described in which sudden, permanent amnesia developed in the absence of a satisfactory explanation. In 1 case, a neuropathologic study disclosed Alzheimer disease; in the other, magnetic resonance imaging showed a temporal lobe abnormality bilaterally.

Results: A man aged 80 years suddenly lost his memory of the previous 60 years. Neuropathologic study 5 years later showed the changes of Alzheimer disease. In the second case a disabling amnesia developed overnight in a man aged 70 years. There was no progression of the disabling amnesia in the next 15 years. Magnetic resonance imaging 10 years from the onset showed abnormality of the medial temporal lobe bilaterally.

Conclusions: In neither case was the amnesia satisfactorily explained. It is likely that rare cases of amnesia occur as the result of an unrecognized pathophysiologic process.

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ily farm in Minnesota and his love of horses. Rarely did he mention his college days. He never spoke of his wartime service or his years as a teacher and political activist. He did not recognize his home where he had lived for 38 years. Occasionally he might recognize his wife as a familiar figure and ask her where his wife was. Almost from the time of onset, he began to say, “I think we’d better go home”; this became the most prominent of several stereotyped utterances ever since. In a few seconds, 60 years of his life were eliminated from his memory.

Prior to the episode he had been remarkably well. Friends were amazed at his physical stamina and mental keenness. He read widely and was interested in everything. He could briskly ascend 5 flights of stairs. He did not smoke, drank very occasionally, and took no medication.

After the onset of the amnesia he was able to carry on a rambling, superficially plausible conversation. He could go out to dinner and go visiting without attracting attention. He had not changed a great deal in the 4 years since the initial episode. His wife thought that if she took him to a wedding at that point, he would not act much differently than he had 4 years ago. At first he continued to tend to his flowers but gave that up after 2 years as he no longer could plan the planting or arrange the rows. Regarding the change in speech that was to develop later, his wife did not notice any word-finding difficulty at the time of the attack or since.

The patient seemed not to understand what his wife said to him, at least he paid no attention except for simple commands. When asked to go to another room to see what it was, he forgot after 10 steps. If he repeated the act several times, he might finally remember the time long enough to tell his wife. He could tell time. He would eat breakfast and 2 minutes later ask where it was. He might finally remember the time long after. If he repeated the act more than once, he might finally remember the time long

The results of a neurological examination apart from cognition were essentially normal. The pupils were equal and reacted to light. The eye movements were full. The face was symmetrical. There was no trace of dysarthria. The arms and legs were used normally. Pinprick was felt everywhere. The tendon reflexes were 2/4 in the arms and absent in the legs. The plantar responses were flexor.

At the request of his wife, he slowly wrote his name with 2 spelling errors, his street address and city with 1 error, and his wife’s name and address with 1 error. He wrote a jargon word for his sister-in-law’s name. The script was nonfluent.

He read aloud from books in English, French, German, and Spanish. He appeared to understand nothing of what he read. According to his wife, for the first year after the initial attack, he could translate a foreign tongue into English, but not thereafter. According to his family he knew German and French well at an early age, his father being German and his mother French.

While being observed over his shoulder, the patient read 2 pages from a book in English with virtually no errors and with proper inflection for questions and proper expression when he read the sentence “He became gloomier at every step.” When a German-language book was placed before him, he pronounced many words correctly but made gross errors on many others. He pronounced the German word war as “was.” On repeated urging, he gave the correct English translation of the German word erzähle as “tells.” When a French-language book was placed before him he read with good
pronunciation (in French) and gave the English translation of several words (for example, assez rapidement as “sufficiently rapidly”). When a book in any of the 4 languages was placed before him, he began at once to read it aloud “at a relentless pace.” When his reading was interrupted by questions or comments from the examiner, he continued to read, occasionally making incoherent, irrelevant replies, that is, the reading tendency dominated. If he lost his place in reading, he could not find it. He did not carry out any written commands. One observer reported that when the patient was still drowsy while recovering from thiopental sodium (Pentothal) anesthesia, he spoke “normally.” This interesting phenomenon was noted again at the second admission to the hospital.

Results of laboratory studies showed no abnormalities. A left-sided internal carotid artery angiogram was normal. An electrocardiogram was within normal limits.

The patient was readmitted 5 months after being discharged from the hospital. His wife reported little or no change in his behavior. He was usually amenable to her guidance and became obstreperous only when his wishes were overridden. He still managed his own toilet care. He balked at eating lunch and supper. He watched television. He prayed in Spanish, French, German, or English.

In the hospital he was agitated, belligerent, and uncontrollable. He fought wildly when tended to and required the constant administration of tranquilizers and sedatives. When quieter by medication, he was asked, “How are you?” to which he replied, “I’m feeling alright.” On another occasion when awakened from a sedative-induced sleep, he spoke intelligibly in French mentioning “the big lane to the house” and some other matters. He did not follow commands given in French, Spanish, or English. He read the label on his coat correctly. After 6 tumultuous days, he was transferred to a state institution for the care of long-term psychiatric cases.

Soon after admission there, he became inactive, spoke very little, no longer read, and was incontinent of bladder and bowel. He became bedridden and died with a high fever 11 months later, 6 years from the onset of the illness.

Autopsy showed that bronchopneumonia was the cause of death. General postmortem examination showed no significant disease elsewhere. The heart weighed 310 g. Neuropathologic examination of the brain revealed diffuse convolutional atrophy with a clear impression of more marked involvement of both temporal lobes. The hippocampal gyri were especially small. The brain weighed 1540 g. In the left inferior temporal gyrus there was an old, small cortical scar 1 cm in diameter and 1 to 2 mm deep, possibly vascular, possibly due to trauma. This was the only focal lesion.

Microscopic examination of frozen sections of the left hippocampus stained using the Bielschowsky silver technique showed widespread neurofibrillary changes and abundant senile plaques indicative of AD. There was no selective loss of neurons in the Sommer sector. Microscopic examination of sections of embedded blocks of tissue stained with the Bodian silver method also showed widespread changes of AD. The substantia nigra was well preserved. Purkinje cells were normal.

Comment. The main feature of this patient’s illness was the unexplained sudden amnesia of a most severe degree involving a retrograde amnesia for some 60 years and a virtually total anterograde amnesia. In view of the later neuropathologic finding of AD, it can be speculated that the unusual dysphasic manifestations that were present 4 years after the onset reflected that process producing a combination of amnesia and aphasia. The picture may have been colored by the patient’s unusual native talents.

The diagnosis of symptomatic AD is definite. No abnormalities were disclosed that might account for the sudden, permanent amnesia. There was no evidence of ischemic thalamic damage or healed encephalitis. The gross and microscopic neuropathologic examinations were extensive. However, the state of the entire hippocampus and fornix on each side may not have been fully scrutinized. A brain weight of 1540 g in an 86-year-old man with AD is remarkable.

No medical or psychological assessment of the patient’s cognitive function was made in the 4 years after the onset of the illness. There is no reason to doubt the wife’s account of the acute amnesia. Whether AD was present and progressing during the initial years is an important question. According to the medical history he could translate a foreign tongue into English in the first year but not thereafter. If this is true, it can be assumed that deterioration occurred. Also his wife did not notice word-finding difficulty early on. There is the history that the patient discontinued tending his garden after 2 years. In the final year of the illness, the course was rapidly progressive. It is possible or even likely that the patient’s wife did not recognize a slow, gradual, incremental decline in a mental state already complex and devastating. In the absence of such an explanation, it would be necessary to explore a possible direct relationship between loss of memory and the aphasic disturbance. In an elderly person could an extremely severe amnesia give rise to elements of an unusual dysphasia? There is no report of such in the literature. For now the aphasic element must be attributed to AD. The acute amnesia is then assigned to another mechanism, one of unknown nature.

CASE 2

Report of Case. A bank president, aged 70 years, came down to breakfast on the morning of November 24, saying that he felt dizzy. His wife suggested that he drink a little brandy and the patient opened a bottle. Twenty minutes later he asked who opened the bottle and denied doing it himself. He was unable to find his keys and stamps. It was evident to his family that his memory was severely impaired. He did not recall visiting his daughter the day before. Prior to the episode he had been extremely active and effective. There was no headache at the time or in the past. He had not fallen. He was taking no medication. He had received an influenza vaccination 10 days before.

When examined 10 days later, the family reported that there had been no change in the interim. His day-to-day memory remained absent. Results of a detailed neurological examination were normal except for memory. He was affable, animated, and cooperative. There was no abulia de-
lay in his responses. He did not recall the national holiday of September 5th or the presidential election day of November 2nd. He did not recall the last bank meeting 6 weeks before. He knew that he had had a heart attack but did not realize it was 18 years ago. His memory for events of the past 20 years was impaired. Recall of his high school and college years appeared to be adequate. There was no aphasia. Reading and writing were performed normally. Proverbs were correctly interpreted. There was no confabulation. Serial subtraction of 7 from 100 was done rapidly with 1 error (55). Given the sentence “Tom and Bill went fishing, they caught 3 striped bass,” he repeated it accurately after 12 minutes. After 27 minutes, he recalled none of it even with prompting and when the sentence was repeated, he disclaimed having heard it before.

The following laboratory tests and procedures were normal: skull x-ray film, computed tomographic scan, electroencephalogram (awake and asleep), bilateral carotid and left vertebral angiograms, cerebrospinal fluid examination, and 20 blood chemistry studies (thyroxine level, 5.3 µg/dL [68 nmol/L]). An electrocardiogram showed no conduction abnormality. A pneumoencephalogram showed enlargement of the left temporal horn.

During the hospital stay of 12 days, memory function did not change. An explanatory diagnosis was not achieved. A temporary cardiac arrest during sleep was considered but against that interpretation is the fact that cerebral low blood flow states do not cause ischemia of the hippocampi but rather of the cerebral border-zones. The acute onset of the deficit and the finding of normal spinal fluid exclude acute encephalitis.

The patient was examined each year thereafter. Ten years later his memory loss was essentially unchanged. He could remember nothing from hour to hour. He spent his time walking, reading, and playing solitaire. He kept a daily diary. He did family chores under his wife’s direction. His general health was excellent. There had been no seizures or cardiac symptoms. Findings from neurological examination were unremarkable. The deficit remained about the same for the next 10 years at which point magnetic resonance imaging showed extensive, bilateral medial temporal lobe abnormalities, resembling the picture reported in transient global amnesia on diffusion-weighted imaging in the 2 to 28 hours after onset, and clearing in the follow-up period. No report of a similar case was found in the literature. As a possible mechanism one could consider the process that underlies transient global amnesia in which the insult was not transient but permanent. No such event has been reported.

The possible cause and explanation of acute permanent amnesia have been considered in each case report without reaching a plausible explanation. The literature contains no reference to similar cases. There was no evidence in either case of an acute strokelike process. It may be relevant that both patients were elderly. The study of further cases using up-to-date technology is awaited with the prospect that an answer will emerge.

Comment. A severe impairment of memory developed overnight in a 70-year-old, healthy man. The deficit remained about the same for the next 10 years at which point magnetic resonance imaging showed extensive, bilateral medial temporal lobe abnormalities, resembling the picture reported in transient global amnesia on diffusion-weighted imaging in the 2 to 28 hours after onset, and clearing in the follow-up period. No report of a similar case was found in the literature. As a possible mechanism one could consider the process that underlies transient global amnesia in which the insult was not transient but permanent. No such event has been reported.

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REFERENCE