Case Reports

Loss of Recent Memory as a Sign of Focal Temporal Lobe Disorder

Report of a Case

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Loss of recent memory is well known as a symptom of diffuse cerebral disease such as senile and presenile dementia, toxic and deficiency states, trauma, and encephalitis. In these cases, memory loss may be only a part of a general disturbance of the intellect. Recently it has become recognized that this symptom may also be a more specific sign of a focal disorder involving the vicinity of either the 3rd ventricle of the hippocampus, or of the hippocampus. The patient in the following case had a striking loss of recent memory as the predominant symptom, with the intellect otherwise quite well preserved. This deficit proved to be due to a tumor largely confined to the deep left temporal lobe and hippocampus. We are unaware of similar examples of memory loss associated with such a discrete neoplasm in this location, and wish to add this case of medial temporal lobe tumor to the other reported cases of focal lesions producing this syndrome.

Case Report

A 62-year-old nurse was admitted to the Piedmont Hospital on March 6, 1963, complaining of loss of memory of about 2 weeks duration. She said that she frequently forgot what she had done or read in the preceding few minutes, and had difficulty finding things at home and at work. Friends had told her that she repeated statements and questions over and over. For the past week she had been aware of lethargy and loss of initiative. She found herself “daydreaming,” and had once just watched the food she was cooking burn. She felt “too bored” to eat, and neglected writing letters to her son and other duties. She developed a tendency to cry easily, and wondered if she were in a state of depression. There had been one brief episode of disturbed vision in which objects seemed distorted in size and shape, but no other illusions, hallucinations, or seizures.

Examination. The general physical and neurological findings were normal. There was no papilledema or visual field defect. Motor, sensory, and reflex testing were all normal. The outstanding abnormality was a severe disturbance in memory for recent events, without any corresponding loss for events prior to her illness. She could not recall what she had been told or what she had read even a few minutes earlier, and would quickly forget instructions. She was especially embarrassed by her inability to recall names of close friends who visited her. There was a mild nominal aphasia and some dyslexia. Some disorientation in space was apparent, causing confusion as to which door of her room led to the bathroom, and failure fully to recognize her own hospital room after a short absence. She could perform calculations readily, could distinguish her right side from her left, and had no finger agnosia. She was able to draw objects and copy figures without evidence of constructive apraxia.

Two weeks after admission she had 2 generalized convulsions, beginning with deviation of the head and eyes to the right. Her memory loss then became even more striking. She could not recall her age, and was confused as to time. She recognized her physicians, but would forget their visits a short while later. If procedures such as arteriography were explained to her in detail, she would soon have no memory of the conversation. Her son, a physician in the Navy, had returned from the Philippines to see her. She knew this when he was with her, and would talk to him in an intelligent, appropriate, seemingly normal manner. However, if asked 15 minutes later if she had seen him, she might say, “Oh no, he is in the Philippines.” She was fully aware of her memory disorder and said she felt as if “half my brain is gone.”

Finally, she developed a complete homonymous hemianopsia. She became somewhat lethargic and indifferent. There was still no papilledema or other evidence of increased intracranial pressure, and no abnormality on sensory, motor, or reflex examination.

Roentgenograms of the skull, electroencephalogram, and the usual laboratory tests of blood and urine were all normal. The cerebrospinal fluid contained no cells and was under a pressure of 75 mm. of water; it had a protein content of 10 mg. per cent. Bilateral carotid arteriography revealed no abnormality of the intracranial vessels. A radioactive mercury brain scan demonstrated a 4.5 cm. area of hyperconcentration of the isotope deep in the left mid-temporoparietal area, almost adjacent to the midline (Fig. 1).

Operation. A left temporoparietal craniotomy was carried out on April 8, 1963, 14 months after the onset of symptoms. The brain was not under increased pressure, and no surface abnormalities were seen. A core of brain 2 cm. in diameter was resected from the low mid-temporal region, corresponding to the area of abnormality seen on the brain scan. Tumor tissue was encountered, medial to the temporal horn of the ventricle. A biopsy was obtained. This was interpreted by the pathologist, Dr. R. B. Vincenzi, as a gemiatocytic astrocytoma. There were areas of hemorrhage and necrosis, with perivascular and endothelial proliferation of blood vessels, indicating malignancy.
Her condition showed little change immediately following operation. Gradually she became less responsive, lapsed into a state of akinetic mutism, and died on April 27, 1963.

Autopsy. Postmortem examination was limited to the brain. There was no evidence of cerebral swelling. There was a surgical wound in the midportion of the left middle and inferior temporal gyri, 2 cm. in diameter, but no other surface distortion. Vessels of the circle of Willis showed only minimal atherosclerosis at their bifurcations. The lateral and 3rd ventricles were not enlarged or displaced in coronal sections. The mammillary bodies, thalamus, and body of the corpus callosum were normal. Beginning at the level of the splenium of the corpus callosum (Fig. 2), a firm, greyish-red tumor was found, which extended across the splenium and included the hippocampal commissure. There was complete infiltration of the crus of the left fornix, but the right was not invaded. The bulk of the tumor lay medial to the collateral trigone of the left lateral ventricle and posterior temporal horn, occupying the posterior portion of the left hippocampus and hippocampal gyrus. It extended posteriorly, medial to the occipital horn, to within 5 cm. of the occipital pole. The amygdala and approximately the anterior half of the left hippocampus were not involved. Except for invasion of the psalterium and splenium of the corpus callosum, the tumor was unilateral. The brain stem and cerebellum were normal. Microscopic examination of the tumor was similar to that of the biopsy.

Discussion

Memory is a complex, integrated function. Besides the initial perception of events, it involves the process which ultimately results in retention, and permits subsequent recall. Only a start has been made in understanding these processes. Much of the knowledge has necessarily come from clinicopathological studies of memory impairment in man, which cannot be exactly duplicated in experimental animals.

In 1887, Korsakow described a peculiar mental syndrome associated with polyneuritis, occurring mostly in alcoholic patients. This syndrome of Korsakow’s psychosis is characterized particularly by the inability to record new memories, old memories remaining relatively intact. Immediate repetition of words and numbers may not be affected as long as attention is not distracted, but memorizing for retention and recall beyond a few minutes is poor or does not occur. There is often disorientation in time, so that supposed dates are erroneous and time intervals are conceived as much shortened, and in space, with difficulty in finding one’s way even in familiar surroundings. There may also be a tendency, known as confabulation, to fabricate false accounts as if to conceal these defects of memory. For some time it has been thought that the mammillary bodies are specifically related to the process of memorizing, because of their consistent involvement in cases of Korsakow’s alcoholic psychosis. More recently it has been indicated that lesions in the medial dorsal nucleus of the thalamus may also be of special significance with respect to memory loss in this disease.

In many diseases, such as encephalitis and senility, the diffuse distribution of the lesion prevents meaningful correlation with the symptoms of memory loss. Intracranial tumors may cause impairment of memory, secondary to the indifference or inattention accompanying frontal lobe destruction, the aphasic disorders with involvement of the dominant cerebral

Fig. 1. Radioisotope brain scan utilizing Hg 203 labeled neohydrin, showing hyperconcentration in astrocytoma of medial left temporo-occipital lobes, including hippocampus. A. (Left) AP view. B. (Right) Lateral view.