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R. Rahme, R. Moussa, A. Awada, I. Ibrahim, Y. Ali, J. Maarrawi, T. Rizk, G. Nohra, N. Okais and E. Samaha

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CASE REPORT

R. Rahme
R. Moussa
A. Awada
I. Ibrahim
Y. Ali
J. Maarrawi
T. Rizk
G. Nohra
N. Okais
E. Samaha

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SUMMARY: Korsakoff-like amnestic syndromes have been rarely described following structural lesions of the central nervous system. In this report, we describe a case of acute Korsakoff-like syndrome resulting from the combination of a left anteromedian thalamic infarct and a right hippocampal hemorrhage. We also review the literature relevant to the neuropathology and pathophysiology of Korsakoff syndrome and anterograde amnesia.

Korsakoff syndrome has been classically attributed to thiamine deficiency from chronic alcoholism or severe malnutrition.¹ However, Korsakoff-like syndromes have been occasionally reported following focal structural lesions of the central nervous system.²⁻⁶ We present a case of Korsakoff-like amnestic syndrome resulting from the combination of a left anteromedian thalamic infarct and a right hippocampal hemorrhage.

Case Report

A 63-year-old right-handed nonalcoholic diabetic male patient presented for sudden-onset headache with vomiting. Findings of a neurologic examination were unremarkable. CT (Fig 1A), and MR imaging (Fig 1B) of the brain revealed a localized hemorrhage in the right hippocampal formation extending into the temporal and occipital horns of the lateral ventricle and the deep Sylvian fissure. The patient was admitted to the hospital and managed conservatively. He remained stable until 2 weeks later when he suddenly developed confusion. At that time, he was found to be awake and alert but disoriented as to time and place. Neurologic assessment revealed severe anterograde amnesia, altered temporal order recall, florid confabulations, impaired recognition, and loss of insight. The patient could not remember recent conversations and kept repeating the same questions. He could not recognize the medical and nursing staff, though he recognized and knew the names of his wife and children. In addition, he exhibited retrograde memory loss with a 25-year temporal gradient. Although he knew he was a taxi driver, he could not remember having stopped since his hospital admission. He recalled his date of birth and brought up genuine memories from childhood but kept asking about his parents, who had died 25 years ago. He was attentive with good immediate recall and did not have executive dysfunction, aphasia, or apraxia. Findings of the rest of the neurologic examination were normal. CT and MR imaging of the brain revealed a left anteromedian thalamic infarct, not seen on the initial films: hypoattenuation on CT (Fig 1C), low signal intensity on T1-weighted images, and high signal intensity on T2-weighted (T2WI) (Fig 1D) and fluid-attenuated inversion recovery images. The right-sided hemorrhage had

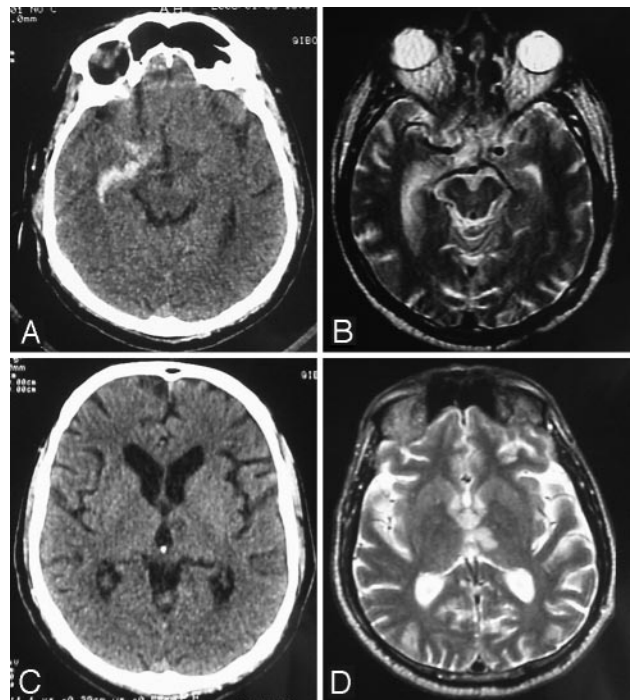


Fig 1. A and B, Head CT (A) and T2WI brain MR imaging (B) revealing a localized hemorrhage in the right hippocampal formation, extending into the temporal and occipital horns of the right lateral ventricle and the right deep Sylvian fissure. C and D, Head CT (C) and T2WI brain MR imaging (D) performed 2 weeks later, demonstrating a left anteromedian thalamic infarction that was not present on the initial films.

started to resolve, and there was no hydrocephalus; the remaining limbic structures, including mamillary bodies, were intact. Findings of echocardiography, carotid Doppler, and cerebral angiography were nonrevealing. The patient was managed conservatively, but his symptoms persisted. He was discharged home 2 weeks later. Repeat mental status assessment 4 weeks after discharge did not show improvement. He was unable to remember events of the day, kept confabulating and repeating the same questions, constantly asked about his parents, and had difficulty recognizing his friends and neighbors.

Discussion

Korsakoff syndrome is diagnosed in an alcoholic or malnourished patient presenting with anterograde amnesia, impaired

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From the Departments of Neurosurgery (R.R., R.M., I.I., Y.A., J.M., T.R., G.N., N.O., E.S.) and Neurology (A.A.), Hôtel-Dieu de France and Saint-Joseph University Faculty of Medicine, Beirut, Lebanon.

Please address correspondence to Ralph Rahme, MD, Department of Neurosurgery, Hôtel-Dieu de France, Ashrafieh, Beirut, Lebanon; e-mail: rrahme@waln.org

temporal order recall, confabulations, impaired recognition, loss of insight, and temporally graded retrograde amnesia.^{1,7} The patient described in this report presented with a very similar syndrome, but no history of alcoholism or malnutrition. His symptoms resulted from a left anteromedian thalamic infarct following a right hippocampal hemorrhage. We believe that this thalamic lesion, either alone or in combination with the contralateral hippocampal injury, was responsible for the patient's clinical picture.

The critical lesion sites for the memory disorder in Korsakoff syndrome have been extensively debated. Victor et al⁷ pointed out that the dorsomedial thalamic nucleus was constantly affected in these patients. Mair et al⁸ described 2 patients whose autopsies showed lesions in the mammillary bodies and the midline and anterior portions of the thalamus, but not in the dorsomedial nuclei. Their findings were closely replicated by Mayes et al.⁹ Visser et al¹⁰ showed that anterograde amnesia in this syndrome is associated with atrophy of the midline thalamic nuclei, but not of the mammillary bodies, hippocampus, or parahippocampal gyrus. Harding et al¹¹ concluded that neuronal loss in the anterior thalamic nuclei, but not in the mammillary bodies or dorsomedial thalamic nuclei, was characteristic of Korsakoff psychosis. Sullivan and Marsh¹² demonstrated that amnesia was related to hippocampal-volume deficits in patients with Korsakoff syndrome.

Anterograde amnesia and Korsakoff-like syndrome have been reported following injury to various limbic structures, including the medial temporal lobe and hippocampal formation,⁵ fornix,³ mammillary bodies,² mammillothalamic tract,⁶ anterior and dorsomedial thalamus,^{4,13-15} and retrosplenial cortex.¹⁶ Mishkin¹⁷ demonstrated that there are 2 limbic circuits in which combined lesions are required to produce severe amnesia: the medial circuit connecting the hippocampus and anterior thalamic nuclei and the basolateral circuit connecting the amygdala and dorsomedial thalamic nucleus. The anterior and dorsomedial thalamus and medial temporal lobe are the "nodal points" where these 2 circuits converge and are, therefore, particularly vulnerable to the effect of discrete structural lesions.^{13,16,18-20}

Bilateral infarcts in the anterior, paramedian, or anteromedian thalamus can result in Korsakoff-like amnesia from injury to the anterior and dorsomedial thalamic nuclei.^{14,15} Unilateral left-sided thalamic injury presenting with amnesia has been rarely reported in the literature.^{4,13} This patient might represent a similar case because his symptoms immediately

followed his thalamic infarction. Alternatively, this thalamic injury might have acted as the trigger for the amnesic syndrome, which would have been produced by bilateral interruption of the Papez circuit: a 1st injury to the right hippocampal formation and a 2nd injury to the left anteromedian thalamus. Welch et al⁵ attributed Korsakoff syndrome in an alcoholic patient to the combination of right hippocampal atrophy and left thalamic dysfunction. Although the exact etiology of this patient's strokes remains unclear, this is, to the best of our knowledge, the first report of Korsakoff-like amnesic syndrome resulting from the combination of asymmetric bilateral structural injuries to the limbic system.

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