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Korsakoff Syndrome: Underdiagnosed and Underserved

Rajkumar Patel, MBBS[1*], Olaniyi David Ogundeji, MBBS[2], Oluwafeyikemi Fasina, MBBS[3], Hafiz Olatunde, MD, MPH[1]

[1]Psychiatry Department, Meharry Medical College

[2]Department of Mental Health, Bloomberg School of Public Health, Johns Hopkins University

[3]Psychiatry Department, Larkin Community Health Hospital

*Corresponding author details: Rajkumar Patel, MBBS; Rpatel@mmc.edu

ABSTRACT

Korsakoff syndrome (KS) is a late neuropsychiatric manifestation of Wernicke encephalopathy (WE) in which there is a disorder of selective anterograde and retrograde amnesia. Although this can happen in a variety of conditions that lead to thiamine deficiency and, as a result, damage the medial temporal lobes, KS is most frequently seen in individuals with alcohol use disorder after an episode of WE. In some individuals, like in this case report, KS develops without a recognized acute episode of WE. It has been suggested that in these cases, KS may result from a series of subclinical or unrecognized episodes of WE.

Keywords: Korsakoff syndrome; Wernicke–Korsakoff; Thiamine deficiency; Amnesia; Amnestic disorders.

INTRODUCTION

Korsakoff syndrome is an amnestic syndrome caused by thiamine deficiency, most associated with the poor nutritional habits of people with chronic alcohol use disorder. Other causes of poor nutrition (e.g., starvation), gastric carcinoma, hemodialysis, hyperemesis gravidarum, prolonged IV hyperalimentation, and gastric plication can also result in thiamine deficiency [2]. The biologically active form of thiamine is thiamine pyrophosphate, which is metabolized by the liver and is essential in biochemical pathways involved in carbohydrate metabolism. Long-term consumption of alcohol interferes with the absorption of thiamine in the intestine, and this can worsen thiamine deficiency in people who already suffer from malnutrition [3]. Thiamine deficiency causes cytotoxic and vasogenic destruction of the astrocytes first and subsequently causes disruption of the blood-brain barrier and local petechial hemorrhages in the areas vulnerable to thiamine deficiency [4].

Korsakoff syndrome is often associated with Wernicke encephalopathy, which is associated with a syndrome of confusion, ataxia, and ophthalmoplegia. Although delirium clears up within a month or so, the amnestic syndrome either accompanies or follows untreated Wernicke encephalopathy in approximately 85 percent of all cases [2].

Patients with Korsakoff syndrome typically demonstrate a change in personality as well, such that they display a lack of initiative, diminished spontaneity, and a lack of interest or concern. These changes appear frontal lobe-like, similar to the personality change ascribed to patients with frontal lobe lesions or degeneration. Indeed, such patients often demonstrate executive function deficits on neuropsychological tasks involving attention, planning, set-shifting, and inferential reasoning consistent with frontal pattern injuries. For this reason, Korsakoff syndrome is not a pure memory disorder, although it certainly is a useful paradigm of the more common clinical presentations of the amnestic syndrome [2].

The onset of Korsakoff syndrome can be gradual. Recent memory tends to be affected more than remote memory, but this feature is variable. Confabulation, apathy, and passivity are often prominent symptoms of the syndrome. With treatment, patients may remain amnestic for up to 3 months and then gradually improve over the ensuing year. Administration of thiamine may prevent the development of additional amnestic symptoms, but the treatment seldom reverses severe amnestic symptoms when they are present. Approximately one-third to one-fourth of all patients recover completely, and approximately one-fourth of all patients have no improvement at all [2].

The neuropathologic lesions in KS are symmetrical and paraventricular, involving mammillary bodies, the thalamus, the hypothalamus, the midbrain, pons, medulla, fornix, and cerebellum [2]. Mammillary body shrinkage, which is observed in upwards of 60-80% in postmortem neuropathology studies, has

been proposed as a specific macroscopic lesion for chronic WE and KS. Neuronal loss in the anterior thalamic nuclei is the best predictor of memory impairment in KS. Structural lesion in the mammillothalamic tract- white matter projections from the mammillary body to the anterior thalamus- results in KS [5].

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CASE PRESENTATION

A 55-year-old Caucasian male presented to the emergency department with complaints of anxiety and confusion. He is unemployed, single, and has experienced housing instability over the past few years, currently residing in an Airbnb arranged by his friend, Amy. Over the past 18 months, he has experienced increasing memory difficulties, including frequent misplacement of personal items (e.g., keys, wallets, IDs), forgetting that appliances like the oven are on, difficulty recalling names, and forgetting recent events such as a friend's visit for dinner earlier in the week. He also became disoriented in his familiar neighborhood. He has a history of severe alcohol use disorder, having consumed alcohol daily since the age of 16, with episodes of withdrawal seizures. His diet has been poor, consisting mainly of frozen pizza and steak, with minimal vegetable intake. He denies other substance use. He has experienced depressive symptoms most days in recent weeks but denies feelings of guilt, suicidal ideation, or amotivation.

Emergency department vital signs were unremarkable. Comprehensive metabolic panel revealed hypokalemia; other values were within normal limits. Urine drug screen was positive for cannabis and benzodiazepines (the latter attributed to a recent short course prescribed during an ER visit). Head CT was unremarkable. He was initiated on a 3-day course of intravenous thiamine in the emergency department. Approximately six weeks prior to this presentation, he was admitted to an inpatient psychiatry unit for alcohol detoxification and evaluation of forgetfulness.

Initial Assessment

During our initial evaluation, the patient identified unemployment and housing instability as his primary concerns. He appeared somewhat apathetic and tended to downplay his problems. A more comprehensive understanding of his situation emerged through collateral information from his friend, Amy. He was unable to specify his current residence and demonstrated poor recall of recent events, indicating he was a poor historian. He appeared indifferent to multiple ongoing stressors. Discrepancies were noted between his account and Amy's reports. Amy described his housing instability as beginning two years prior, with periods of living in hotels. She had recently secured an Airbnb for him, but he forgets the access code daily and contacts her for assistance. When asked, he provided an incorrect code, which Amy confirmed was inaccurate. She also recounted a recent incident where he was found by police near a gas station, displaying inappropriate behavior; he had no recollection of this event. Amy stated that he relies on his 401(k) for income but often forgets making withdrawals, leading to confrontations with bank staff under the belief that his funds were being stolen. Prior to two years ago, he resided with his 83-year-old mother, who asked him to leave due to belligerent behavior associated with alcohol consumption. Amy also reported witnessing seizure episodes followed by periods of altered mental status, including one instance where he bit his tongue, resulting in oral bleeding. He had not sought medical attention for these episodes.

Laboratory Findings

Complete blood count and comprehensive metabolic panel were largely unremarkable, aside from a slightly low hematocrit and mild hyperglycemia. Coagulation profile was normal, except for an activated partial thromboplastin time of 37.2 seconds. Vitamin D level was low at 17.3 ng/mL. Thyroid-stimulating hormone was elevated at 5.44 µIU/mL, with a normal free T4 of 0.86 ng/dL. Urine drug screen was positive for benzodiazepines (attributed to a recent ER prescription for severe anxiety) and THC.

Diagnosis and Management

Diagnoses included major depressive disorder, moderate without psychotic features; mild neurocognitive disorder due to another medical condition; cannabis use disorder; and unspecified anxiety disorder. Medications continued included escitalopram 15 mg daily, donepezil 5 mg at bedtime, and prazosin 2 mg at bedtime. He was managed with symptom-triggered CIWA protocol and received intravenous thiamine, along with oral multivitamins and folate. MRI of the brain with and without contrast showed no acute findings. Upon request, the radiologist assessed the mammillary bodies, reporting probable atrophy of the right mammillary body and the left being barely perceptible, suggesting significant atrophy.

Mental Status Examination

The patient appeared his stated age and was dressed in hospital pajamas. He was calm, cooperative, and pleasant, maintaining fair eye contact throughout the interview. No evidence of psychomotor agitation or retardation was observed. Speech was normal in rate, volume, rhythm, and prosody. He described his mood as "okay," with a depressed affect. Thought processes were superficially logical and goal-directed, without loose associations. He denied suicidal or homicidal ideation, auditory or visual hallucinations, and did not appear to be responding to internal stimuli. Insight and judgment were poor.

He was oriented to person, place, and time but exhibited multiple lapses in recent memory, while remote memory appeared relatively preserved. Inattention was noted, as evidenced by his inability to spell "WORLD" backwards. His fund of knowledge appeared below average. The Saint Louis University Mental Status (SLUMS) examination score was 24 out of 30.

DISCUSSION

Korsakoff syndrome is an amnestic syndrome caused by thiamine deficiency, most commonly associated with poor nutritional habits of people with chronic alcohol use disorder [1], which has been the case with the patient above, who mostly relied on frozen pizza and such items due to severe executive dysfunction and resultant inability to grocery shop or obtain healthy foods.

Korsakoff syndrome is often associated with Wernicke encephalopathy (WE). Although the delirium of WE clear up in a few weeks, the amnestic response either accompanies or follows untreated Wernicke encephalopathy in approximately 85% of all cases. This seems to have been the case with our patient, as Amy described multiple past episodes of confusion and seizure episodes.

Patients also demonstrate significant anterograde amnesia than retrograde as evidenced by losing or misplacing possessions, leaving the oven on, forgetting a recent dinner with a friend, and having a recent encounter with the police. His remote memory was somewhat preserved as he did remember the name of his high school correctly, as confirmed by Amy, and also recalls details of his past marriages somewhat accurately.

This patient also displayed other symptoms of Korsakoff syndrome, like changes in personality, lack of initiative, diminished spontaneity, and lack of interest or concern [2] as evidenced by his nonchalant attitude towards multiple ongoing stressors like housing instability, unemployment, and 401k getting depleted. The patient also exhibited significant confabulation, as evidenced by the patient making up a completely wrong 4-digit Airbnb code in which he had been living for the past few weeks.

While mammillary body atrophy is a nonspecific finding in KS, it is observed in upwards of 60- 80% in postmortem neuropathology studies [5]. MRI in our case also showed bilateral Mammillary body atrophy, which further supported our suspicion of Korsakoff syndrome. Diagnosis of Korsakoff syndrome thus should be based comprehensive evaluation, which should include past history, clinical presentation, and neuroimaging findings.

CONCLUSION

The 55-year-old male patient displays clear signs of Korsakoff syndrome, worsened by chronic alcohol use and poor nutrition. Symptoms such as memory loss, apathy, and confusion suggest progression from Wernicke encephalopathy. Immediate intravenous thiamine treatment is crucial to prevent further neurological damage. A multidisciplinary approach that includes psychiatric support, nutritional care, and social services is vital for addressing his complex needs. Ongoing monitoring and a tailored rehabilitation plan focusing on cognitive function will be essential for improving his quality of life and promoting housing and employment stability. Collaboration among healthcare providers will enhance his recovery and functional outcomes.

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