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Case Report

# From Confusional Syndrome to Korsakoff's Encephalopathy: Understanding, Detecting, Treating in a Case

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## Abstract

Wernicke-Korsakoff encephalopathy (WKE) is a severe neuropsychiatric complication resulting from thiamine deficiency, most often secondary to chronic alcoholism. It comprises two overlapping conditions: Wernicke's encephalopathy, characterized by acute neurological dysfunction, and Korsakoff's syndrome, marked by persistent cognitive deficits, particularly memory impairment. This case report describes a 34-year-old male, chronic alcohol consumer, admitted with acute mental confusion, spatiotemporal disorientation, incoherent speech, and episodes of wandering. Neurological examination revealed no focal motor or sensory deficits but showed severe anterograde amnesia, confabulations, and emotional blunting. Brain MRI demonstrated cortico-subcortical atrophy, and laboratory analysis confirmed thiamine deficiency. The patient received high-dose intravenous vitamin B1 for seven days, broad vitamin supplementation, neuropsychological monitoring, and psychosocial interventions targeting alcohol withdrawal. Within 10 days, orientation and coherence of speech partially improved; however, significant memory deficits persisted at discharge. Neuropsychological evaluation revealed marked impairment in executive function and fixation memory, consistent with Korsakoff's syndrome. This case emphasizes the importance of early recognition of delirium as a warning sign of thiamine deficiency in at-risk individuals, even in the absence of the complete Wernicke triad. Timely administration of parenteral thiamine is critical to preventing irreversible neurological damage. Furthermore, the case underlines the role of structured neuropsychological assessment in documenting the extent of cognitive impairment, guiding rehabilitation, and monitoring recovery. A multidisciplinary approach involving neurology, psychiatry, nutrition, and social services is essential to optimize functional prognosis, reduce morbidity, and prevent recurrence. This report also supports systematic screening for WKE in chronic alcoholics and other at-risk populations, as well as continued education for healthcare providers to improve early detection and management strategies.

## Keywords

Wernicke's Encephalopathy, Korsakoff's Syndrome, Thiamine Deficiency, Chronic Alcoholism, Delirium

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## 1. Introduction

Wernicke's encephalopathy is a neurological syndrome characterized by a triad of symptoms: ocular mobility disturbances (nystagmus and ophthalmoplegia), mental health and gait alterations, which are present in only 16% of reported cases [1]. The etiology is diverse and is associated with thiamine deficiency or malnutrition combined with alcohol abuse. [12, 9]. Not all patients are diagnosed in time and may develop Korsakoff's syndrome or encephalopathy. Patients present with memory impairment (anterograde amnesia), severe temporary disorientation, and in the initial phases, communication disorders and memory loss. [14] These changes, due to neuronal necrosis caused by insufficient protein intake, lead to irreversible structural damage in certain areas of the brain. The diagnosis of Wernicke-Korsakoff syndrome (WKS) is complex and essentially clinical, as laboratory tests are not an absolute reference and diagnosis by magnetic resonance imaging offers only a sensitivity of 53%. [9]. However, each patient requires an individualized approach to nursing care, as symptomatic signs related to cognitive status and anterograde amnesia, as well as learning about the evolution of the disease, constitute an obstacle to patient care.

The objective of this presentation is to highlight the importance of early detection of confusional syndrome, as a fundamental clinical warning signal of Korsakoffs encephalopathy; to underline the complexity of neuropsychological diagnosis in chronic alcoholic patients, where symptoms may be atypical or masked and to insist on the determining impact of rapid and appropriate management, in order to improve the functional prognosis, limit the progression towards irreversible sequelae, and reduce the morbidity linked to these disorders.

Presentation of case

*Patient presentation Initials:* MMNS.

*Gender:* Male *Age:* 34 years old *Profession:* Immigration Officer

*Place of Residence:* Lubumbashi (DRC) *Date of Admission:* July 17, 2025 His History: Chronic excessive alcohol consumption, associated with active smoking (approximately 5 packs per week). No previous psychiatric or neurological follow-up was reported. History of the disease: He was transferred from a hospital center following the onset of memory disorders, mental confusion, temporo-spatial disorientation, and incoherent speech. He also presented episodes of wandering in the ward, reflecting an acute confusional state. These symptoms manifested in a context of chronic alcohol abuse, suggesting a serious neuropsychological complication. Neurological examination revealed a confused patient, disoriented in time and space. There is no motor, sensory, or cerebellar deficit. Gait is unsteady, with a staggering gait, without paralysis or tremor. The search for pathological reflexes is negative. The tongue, cranial nerves, as well as fine motor skills, remain normal. The state of consciousness is altered, with no signs of focal neurological deficit. On the psychological level, the patient presents severe mental confusion, with temporal and spatial disorientation. His speech is incoherent, marked by confabulations to fill his memory losses. He exhibits significant anterograde amnesia, with difficulty retaining new information. His affect is poor; he appears indifferent to his environment and shows little emotional expression. Memory for recent events is severely impaired, while long-term memory seems little affected. No signs of delusions or hallucinations are detected. Cognitive assessment reveals major impairment of executive and memory functions, particularly fixation memory.

***Table 1.*** *Neuropsychological test results.*

| Test | Domain assessed | Score | Impairment level |
|---|---|---|---|
| MMSE | Global cognition | 21/30 | Moderate |
| MoCA | Executive & memory functions | 19/30 | Moderate |
| Trail Making Test (Part B) | Cognitive flexibility | 210 sec | Severe |
| Verbal Fluency (semantic) | Language & executive function | 8 words | Severe |
| Digit Span (forward/backward) | Working memory | 5 / 3 | Mild / Moderate |

Additional examinations: *Brain imaging (TBM)*: Cortico-subcortical atrophy. *Biology:* Thiamine deficiency confirmed.

Diagnostic hypothesis;

Alcohol-induced amnesic syndrome (Korsakoff type)

Alcoholic dementia (Marchiafava-Bignami encephalopathy)

Chronic delusional state or alcohol-induced psychotic disorder

Non-alcoholic deficiency encephalopathy

Acute confusional syndrome (delirium)

Support Management correction of intramuscular vitamin

B1 deficiency, at a rate of 100 mg per day by IV, continuously for at least 5 to 7 days. A broad vitamin supplementation regimen, including vitamins B and C, is also initiated. Symptomatic treatment is prescribed to manage the confusional state, including neuropsychological monitoring and cognitive rehabilitation to alleviate memory deficits. Psychosocial care includes support for reducing or stopping alcohol consumption, with psychological support and possibly addiction treatment. Neurological and psychiatric monitoring.

## 2. Evolution

The evolution was marked by a partial or stable improvement in mental status, but memory deficits persisted.

## 3. Discussion

Delirium, frequently observed in chronic alcoholic patients, is a complex clinical entity to diagnose early, often being a warning signal of Wernicke's encephalopathy or Korsakoff's syndrome. The literature emphasizes the need for systematic screening, particularly in at-risk patients, to avoid progression to irreversible neurological damage. The clinical manifestation of the syndrome Wernicke encephalopathy and Korsakoff syndrome belong to the same spectrum of disorders related to thiamine deficiency. The classic Wernicke triad includes ataxia, oculomotor disturbances and altered mental status, but is rarely complete in the patient. Early detection of ocular disturbances, balance disturbances and confusional state should raise the possibility of Wernicke encephalopathy [15]. Korsakoff syndrome is primarily manifested by anterograde amnesia, confabulations and executive function impairment [9]. The diagnosis of Korsakoff syndrome can be made according to the DSM-5 criteria for major neurocognitive disorder of the confabulating amnestic type [2]. In general, Korsakoff syndrome is characterized by severe anterograde and, to a lesser extent, retrograde amnesia of declarative knowledge [11]. Patients with Patients with Korsakoff syndrome may also have difficulty correctly identifying the temporal sequence of events [16]. In addition, many patients have executive function deficits, such as problems with initiating, planning, organizing, and regulating behavior [10]. Patients themselves often do not recognize their problems in daily functioning due to limited awareness of their illness (anosognosia). Patients with Korsakoff syndrome may experience confabulations, although the intensity and frequency may vary among patients [13]. In addition, Korsakoff syndrome is very often accompanied by peripheral neuropathy [8]. Non-invasive neuroradiological investigation of Wernicke encephalopathy (WE) dates back to the 1970s. Early studies used *computed tomography (CT)*, revealing *ventricular dilation,* particularly of the *third ventricle,* but were largely unable to detect *cerebral edema* or *focal lesions.* [12] The introduction of MRI *(*magnetic resonance imaging), with its increased sensitivity to *tissue water content*, has made it possible to visualize acute and chronic *radiological signs* of neuropathology that were not visible on CT [1]. On some sequences, *specific brain structures* appear *hyperintense*, reflecting a *high water content* associated with the *edematous lesions* characteristic of WE. Treatment of Wernicke's encephalopathy requires *rapid administration of thiamine*, although *ideal doses are controversial.* [6] In *high risk*, a dose of *250 mg/day IM or IV for 3 to 5 days* is recommended, although data are limited [7] For curative treatment, the *EFNS recommends 200 mg IV three times a day*, while *7 out of 9 guidelines* recommend *>500 mg/day*, administered slowly (30 min) as a dilute infusion, 3 times a day for 3 to 5 days, with possible relay to 250 mg/day or oral route [3] *In* the absence of IV access, an *intramuscular injection of 250 mg/day* can be used, and *treatment should not be delayed by blood sampling;* in addition, *hypomagnesemia may prevent response to thiamine* [5] Prevention remains central. Wernicke encephalopathy (WE) is a common complication of alcohol withdrawal, serious but often undertreated. Effective treatment and prophylaxis require the administration of *parenteral thiamine*, because the oral form is poorly absorbed. Despite rare adverse reactions, the risks associated with the absence of treatment are much greater. It is recommended to systematically administer injectable thiamine to all patients hospitalized for withdrawal, and to make a presumptive diagnosis of WE in case of clinical doubt. [4].

## 4. Conclusion

Delirium, often encountered in chronic alcoholic patients, is an essential warning signal for early detection of Wernicke's encephalopathy or Korsakoff's syndrome. The complexity of the diagnosis, reinforced by clinical variability and the limitations of paraclinical examinations, underlines the importance of increased vigilance and systematic screening in at-risk populations. Rapid and appropriate management, particularly through vitamin B1 supplementation, can prevent progression to irreversible damage and improve functional prognosis. Finally, a multidisciplinary approach, integrating management of the cause (alcohol abuse), cognitive rehabilitation and psychosocial support, is essential to limit morbidity and promote patient recovery. Raising awareness among healthcare professionals of these issues remains essential to optimize the management of these often underestimated pathologies.

## Abbreviations

| | |
|---|---|
| WE | Wernicke’s Encephalopathy |
| WKS | Wernicke–Korsakoff Syndrome |
| MRI | Magnetic Resonance Imaging |

| | |
|---|---|
| CT | Computed Tomography |
| DSM-5 | Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition |
| EFNS | European Federation of Neurological Societies |
| IV | Intravenous |
| IM | Intramuscular |

## Conflicts of Interest

The authors declare no conflicts of interest.

## References


[1] Antunez E, Estruch R, Cardenal C, Nicolas JM, Fernandez-Sola J, Urbano-Marquez A. *Usefulness of CT and MR imaging in the diagnosis of acute Wernicke's encephalopathy.* 1998; 171(4): 1131-1137. https://doi.org/10.2214/ajr.171.4.9763009

[2] AP Association. *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision (DSM-IV-TR).* 2000 [cited 28 July 2025]; Available at: https://doi.org/10.1176/appi.books.9780890423349

[3] Boulanger AS, Paquette I, L étourneau G, Richard-Devantoy S. *[Wernicke encephalopathy: Guiding thiamine prescription].* Encephalon. 2017; 43(3): 259-267. https://doi.org/10.1016/j.encep.2016.04.011

[4] Cook CCH. *Prevention and Treatment of Wernicke-Korsakoff Syndrome.* Alcohol and Alcoholism. 2000; 35(Supplement_1): 19-20. https://doi.org/10.1093/alcalc/35.Supplement_1.19

[5] Dingwall K, Delima J, Gent D, Batey R. *Hypomagnesiaemia and its potential impact on thiamine use in patients with alcohol misuse at the Alice Springs Hospital.* 2015; 34(3): 323-328. https://doi.org/10.1111/dar.12237. Epub 2015 Feb 19

[6] Frank L. L. *Thiamin in Clinical Practice.* 2015; 39(5): 503-520. https://doi.org/10.1177/0148607114565245

[7] Galvin R, Br åthen G, Ivashynka A, Hillbom M, Tanasescu R, Leone MA, et al. *EFNS guidelines for diagnosis, therapy and prevention of Wernicke encephalopathy.* Eur J Neurol. 2010; 17(12): 1408-1418. https://doi.org/10.1111/j.1468-1331.2010.03153.x

[8] Hammoud N, Jimenez-Shahed J. *Chronic Neurologic Effects of Alcohol.* Clinics in Liver Disease. 2019; 23(1): 141-155. https://doi.org/10.1016/j.cld.2018.09.010

[9] Isenberg-Grzeda E, Kutner HE, Nicolson SE. *Wernicke-Korsakoff-Syndrome: Under-Recognized and Under-Treated.* Psychosomatics. 2012; 53(6): 507-516. https://doi.org/10.1016/j.psym.2012.04.008

[10] Joyce EM, Robbins TW. *Frontal lobe function in Korsakoff and non-Korsakoff alcoholics: Planning and spatial working memory.* Neuropsychologia. 1991; 29(8): 709-723. https://doi.org/10.1016/0028-3932(91)90067-i

[11] Kopelman MD, Thomson AD, Guerrini I, Marshall EJ. *The Korsakoff Syndrome: Clinical Aspects, Psychology and Treatment.* Alcohol and Alcoholism. 2009; 44(2): 148-154. https://doi.org/10.1093/alcalc/agn118. Epub 2009 Jan 16

[12] NekiNS. *Wernicke's Encephalopathy.* 2015; 5: 107-112.

[13] Schnider A. *Spontaneous confabulation and the adaptation of thought to ongoing reality.* Nat Rev Neurosci. 2003; 4(8): 662-671. https://doi.org/10.1038/nrn1179

[14] Sechi G, Serra A. *Wernicke's encephalopathy: new clinical settings and recent advances in diagnosis and management.* The Lancet Neurology. 2007; 6(5): 442-455. https://doi.org/10.1016/S1474-4422(07)70104-7

[15] Sharp CS, Wilson MP, Nordstrom K. *Psychiatric Emergencies for Clinicians: Emergency Department Management of Wernicke-Korsakoff Syndrome.* Journal of Emergency Medicine. 2016; 51(4): 401-404. https://doi.org/10.1016/j.jemermed.2016.05.044

[16] Shimamura AP, Janowsky JS, Squire LR. *Memory for the temporal order of events in patients with frontal lobe lesions and amnesic patients.* Neuropsychologia. 1990; 28(8): 803-813. https://doi.org/10.1016/0028-3932(90)90004-8