Diagnosis of Wernicke's Encephalopathy in Patients With a Psychiatric History:

A Case Series and Literature Review

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ABSTRACT

Objective: To review the literature on diagnostic criteria and management of Wernicke's encephalopathy (WE) and its application in psychiatric populations.

Evidence Review: A PubMed MEDLINE search was conducted in February 2022 and updated in April 2022 for articles published in English between 2012 and 2022 describing clinical findings and treatment of WE. Reference lists of included articles and treatment guidelines were reviewed. Search terms included *Wernicke's encephalopathy*, *thiamine*, *thiamine administration dosage*, and *prescribing*. Additionally, 2 cases with co-occurring psychiatric and WE manifestations were selected from the consult-psychiatry service between July and December 2021. Pertinent clinical findings and management were extracted from the literature and compared with that of the cases.

Findings: 113 titles were retrieved; 39 studies were excluded. Exclusion criteria included studies done in patients < 18 years old, animal studies, studies with no abstract, and studies with no clinical discussion. Twelve articles were added from the grey literature. Eighty-six articles were included in the review. Only 7 studies discussed WE in psychiatry. The results reaffirm the lack of information regarding diagnosis and treatment of WE in the general population. Clinical cases suggest further increased risk in the psychiatric population.

Conclusion and Relevance: Evidence suggests underdiagnosis and undertreatment of WE in general populations and psychiatric patients at increased risk for malnutrition. An interdisciplinary approach improves time to diagnosis and management of comorbidities. We recommend dosing guided by clinical response; however, evidence suggests lengthier and higher doses of thiamine may be needed. Addition of neuroleptics may be necessary for management of psychiatric symptoms and relapse prevention.

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Wernicke's encephalopathy (WE) diagnosis has been chiefly associated with alcohol use disorder. Recent studies,[1–5] however, have highlighted its prevalence among vulnerable patient populations, such as oncologic patients, pregnant individuals, and surgical cases. While a review of the literature showed WE and psychiatric disorders documented concurrently in a few case reports primarily focusing on depression, there is a lack of content regarding comanagement in these cases and challenges in making the diagnosis. The objective of this article is to add to the very limited body of literature on WE and psychiatric patients and review the latest published findings.

METHODS

A PubMed MEDLINE search was conducted in February 2022 and updated in April 2022 for articles published in English between 2012 and 2022 describing clinical manifestations of WE and its treatment. Search terms included *Wernicke's encephalopathy*, *thiamine*, *thiamine administration dosage*, and *prescribing*.

The search was conducted by 1 reviewer, supplemented by articles from the grey literature (ie, information produced by an organization outside the traditional commercial or academic publishing distribution channels). Exclusion criteria included non-English studies, studies conducted with patients aged < 18 years old, animal studies, studies with no abstract available, and studies with no discussion of clinical manifestations. Inclusion criteria included studies published in English, human subjects aged ≥ 18 years old, and studies that focused on clinical manifestation and management of WE.

The review was conducted in accordance with the Preferred Items for Systematic Reviews and Meta-Analyses (PRISMA) statement. A PRISMA study flow chart is provided in Supplementary Figure 1. From the literature research, 113 titles were retrieved. On initial screening, 39 studies were deemed ineligible due to meeting exclusion criteria or having the wrong diagnosis, population, or focus. Twelve articles were added from the grey literature, including treatment guidelines, and pertinent articles from cross-citation and reference search. A total of 86 articles were reviewed (see Supplementary Table 1 for the complete list of references). Case reports comprised 45% (51) of the articles retrieved, and 59.3% (51) of the articles selected. Only 7 studies involved

Clinical Points

- Wernicke's encephalopathy (WE) can present concomitantly with psychiatric decompensation, often requiring treatment with both neuroleptics and thiamine.
- Comanagement of WE cases can lead to better outcomes and help to avoid relapse due to untreated symptoms.
- Higher doses of intravenous (IV) thiamine could be used to rapidly treat WE, with careful monitoring for relapse upon discontinuation. Intramuscular administration can help increase utilization of parenteral routes when IVs are unavailable.

discussion of WE and psychiatry (see Supplementary Table 2 for the complete list of references).

Two clinical cases with co-occurring psychiatric and WE manifestations were selected from the psychiatry consultation liaison service during July–December 2021. De-identified data were used to protect confidentiality. No quantitative analysis was made. Clinical findings and treatments described in the literature were compared with that of the cases.

CASE SERIES

Case 1

A 53-year-old man with hypertension and no formal past psychiatric history was brought to the emergency department due to alcohol intoxication and suicide attempt by overdose on clonazepam. The initial workup revealed rhabdomyolysis (creatine phosphokinase > 10,000), transaminitis (aspartate aminotransferase of 170 U/L and alanine aminotransaminase of 143 U/L), and an ultrasound showing liver steatosis. The patient was admitted to the medical floor, and the psychiatry department was consulted. He received intravenous (IV) fluids for rhabdomyolysis, clonazepam taper for alcohol withdrawal symptoms, and thiamine 500 mg IV 3 times/d for 3 days.

The psychiatric evaluation revealed the use of alcohol, tobacco, and nonprescribed clonazepam 0.5 mg 3 times/day. He complained of depressed mood and insomnia but minimized suicide attempt. The patient remained alert and fully oriented; his thought process was primarily linear, with occasional circumstantiality.

He was stabilized and transferred to the inpatient psychiatry department for management of major depressive disorder. Upon transfer, IV treatment was discontinued. Mood lability, grandiosity, tangentiality, insomnia, and confusion emerged. He was also noted to have increased energy levels despite documented poor sleep. The patient reported gait imbalance and dizziness; a physical examination showed staggering gait and nystagmus. Confusion progressed to poor memory retention, as evidenced by a Montreal Cognitive Assessment (MoCA)[6] result of 20/30 with deficits in delayed recall, abstraction, and attention domain. Magnetic resonance imaging (MRI) was performed, demonstrating subcortical periventricular white matter changes; periaqueductal area and mamillary bodies were unremarkable.

Treatment in inpatient psychiatry included aripiprazole started on hospital day (HD) 7, thiamine 250 mg via intramuscular route (IM) once a day for 5 days started on HD 9 after nystagmus was noted, and lithium, added as an adjunct for mood stabilization of suspected comorbid bipolar disorder. Alcohol use treatment was initiated with naltrexone. The patient began to respond to the regimen, as evidenced by a marked improvement in sleep, thought process, mood lability, and cognition (repeat alternative version of MoCA results were 27/30 after the second day of thiamine IM regimen and following the addition of lithium). The patient was discharged on lithium, aripiprazole, and naltrexone.

Case 2

A 64-year-old woman with major depressive disorder with psychotic features, prior suicide attempts, hypertension, hyperlipidemia, type 2 diabetes mellitus, and rheumatoid arthritis presented to the psychiatry emergency services for possible admission due to psychosis. The patient endorsed weakness and poor oral intake for the past month but could not provide a good history. She appeared disheveled, confused, and oriented only to self. The patient was evaluated by both medicine and psychiatry personnel and was admitted to the inpatient medicine floor for further workup of toxic metabolic encephalopathy.

Due to her poor nutritional status, she was started on thiamine 500 mg IV 3 times/day. MRI of the brain was consistent with abnormal T2 and diffusion hyperintensity of the bilateral medial thalamus; periaqueductal area and mamillary bodies were unremarkable. A basic infectious workup revealed a positive urinalysis, and she was given a short course of IV antibiotics. Despite treatment of the urinary tract infection, her mental status remained poor, with episodes of paranoia, confusion, and agitation. Electroencephalography indicated mild diffuse cerebral dysfunction with no evidence of epileptiform activity. The patient was started on oral olanzapine for paranoia and agitation from suspected comorbid psychosis but demonstrated minimal improvement in encephalopathy and mood. Olanzapine was switched to risperidone. MoCA results were 12/30 with deficits in visuospatial/executive, identification, attention, language, abstraction, and memory domains. This result was consistent with severe cognitive decline and, given the minimal improvement in mental status, lumbar puncture and autoimmune and paraneoplastic panels were obtained with negative results.

On HD 5, routine laboratory tests revealed hypomagnesemia (1.6–1.9 mg/dL). Aggressive replenishment with IV magnesium and thiamine improved mental status. On HD 6, the patient became more interactive with staff and family members. She was alert and oriented to person, place, and time and could recall events prior to hospitalization. A careful review of the patient's brain imaging with the neurology department and the patient's overall clinical

improvement on thiamine indicated a diagnosis of WE. The patient reported no alcohol abuse, and the family confirmed. She was eventually discharged to subacute rehabilitation with daily oral thiamine replacement and risperidone.

LITERATURE REVIEW

Despite being well described in the literature, most information on WE stems from case reports or systematic reviews conducted over 10 years ago. There is consensus that the clinical diagnosis of WE is missed in 75%–80% of the cases,[7] with postmortem studies showing a higher prevalence than what is clinically reported.[4,5,7–9] Most studies agree that high variability in clinical presentations and WE's biased linkage to alcohol use disorder are a cause of these misdiagnoses. Case reports of WE associated with nutritional deficiency, hyperemesis gravidarum, intestinal obstruction, bariatric surgery, cancer, chemotherapy, hemodialysis, and malignant diseases have expanded clinicians' view of WE.[4,5,7,10]

The neurobiochemical role of thiamine in energy homeostasis comes from its role as a cofactor for enzymes like pyruvate dehydrogenase complex, α-ketoglutarate dehydrogenase complex, transketolase, and α-ketoacid dehydrogenase.[4] The central nervous system is particularly vulnerable to thiamine deficiency due to the lower levels of thiamine in the normal human brain versus other organs.[11]

Besides WE, thiamine deficiency has been linked to other various neurodegenerative processes like Alzheimer's disease, Parkinson disease, and prion-related diseases. The exact mechanism of neurodegeneration remains unclear, but thiamine's role in the metabolism of lipids, glucose, and neurotransmitters and maintaining the antioxidant system in the brain is believed to be the key component. Thiamine depletion leads to release of reactive oxygen species, creating neuroinflammation, axonal death, and neuronal death. Moreover, thiamine deficiency and the subsequent halt in major enzyme function leads to overall gliopathy due to the lowered brain pH, in turn, increasing lactate production, reducing levels of glutamine synthetase, causing swelling, and disrupting the blood-brain barrier.[11] These serious neurobiochemical consequences highlight the importance of early diagnosis and management of WE.

The well-known triad of ataxia, nystagmus, and confusion is said to be present in 10%–30% of WE cases.[4,5,7,12,13] Caine's criteria introduced a fourth component: dietary deficiency. Categories were also broadened to more generalized terms, such as cerebellar dysfunction, oculomotor abnormalities, and altered mental status (AMS) or mild memory impairment.[14] Despite these specific criteria, a more comprehensive list of symptoms has been recognized in the literature, including but not limited to loss of appetite, nausea and vomiting, fatigue, weakness, apathy, giddiness, diplopia, insomnia, anxiety, difficulty in concentration, loss of memory for immediate past, confusion, disorientation, confabulation, hallucinations, and even coma.[4,5,8,15] Of these, the most consistently described in the literature is cognitive change, ranging from mild confusion to delirium.[4,10]

Other than clinical evaluation, no gold standard diagnostic tests exist, but MRI helped confirm the diagnosis in case studies. Hyperintensities on the thalamus, periaqueductal area, and mammillary bodies are observed in only 58% of cases.[4,7,12] Serum studies, such as thiamine and red blood cell transketolase levels, have been explored as diagnostic aids, most commonly using high-performance liquid chromatography to measure free thiamine levels. Results may take 3 to 4 days to be obtained and lack sensitivity and specificity for diagnosing active disease. They may be helpful in detecting individuals with very low levels of thiamine who are at risk of developing WE.[4,13] Ocular signs can help confirm the diagnosis, as they are the first symptoms expected to improve within hours to a day of thiamine administration.[10]

Currently, there are 2 sets of updated treatment guidelines for WE. From the European Federation of Neurologic Societies, one guideline recommends 200 mg of thiamine 3 times/day, preferably via IV route, to be continued until no further improvement of symptoms is observed. The other guideline, from the Royal College of Physicians, recommends IV thiamine 500 mg 3 times/day for 3–5 days if WE is identified, followed by 250 mg IM for 5 days or until clinical improvement stops. Guidelines for thiamine supplementation in alcohol use disorder are equally nonspecific and inconsistent.[16] Several publications describe the clinical benefit of using higher doses of IV (HDIV) thiamine > 200 mg, but existing data are limited to case series. One preliminary randomized, double-blind, multidose study[17] of thiamine in patients with alcohol use disorder agreed with HDIV improving working memory; however, the study was not specific to WE. Because the half-life of thiamine is 96 minutes, most regimens involve administering the drug 2 or 3 times/day. Although there is a lack of formal studies regarding thiamine's safety profile, most studies agree that thiamine has an extremely high safety profile, with low concern for adverse events like anaphylaxis.[4,7,12,13,15,18] Recent retrospective studies have highlighted the underprescribing of HDIV in hospitals despite recommendations by these guidelines.[19–21]

DISCUSSION

WE or Psychiatric Symptoms?

As described in *Adams and Victor's Principles of Neurology*,[10] a universal sign of WE is a global confusional state, ranging from apathy and inattentiveness to frank disorientation. Common psychiatric diagnoses that can present in this manner include mood and psychotic disorders, dementia, and catatonia. The presence of past psychiatric history on initial evaluation can mistakenly attribute changes in behavior to a psychiatric decompensation. Symptoms such as psychosis, apathy, or agitation can mask an underlying encephalopathic state, which routine screening laboratory tests and imaging may not necessarily detect. We were able to observe this in the 2 cases described here.

In the first case, altered mental status was not evident initially, and psychiatric symptoms of insomnia, anxiety, and mood dysregulation in the setting of a suicide attempt were more prominent. Substance use was a confounder when evaluating the patient's cognition, as alterations could also be expected in a benzodiazepine overdose. Time in the psychiatric unit revealed evidence of thought disorganization and progression of confusional state, alongside mild nystagmus and ataxia.

In the second case, an initial psychiatric evaluation was conducted due to chronic history of paranoia and disorganization. Appreciation of altered mental status and poor nutritional status prompted the workup of toxic encephalopathy, including thiamine deficiency, and HDIV thiamine was initiated. Once the patient became more alert, residual paranoia and psychosis pointed toward an underlying psychiatric decompensation overlapping with WE. In both cases, a MoCA test provided objective evidence of memory deficits, helping to distinguish confusional states with memory impairment from thought disorganization.

Increased Risk of WE in the Psychiatric Population

The World Psychiatric Association has emphasized that psychiatric patients are at increased risk for many chronic diseases such as malnutrition, an essential part of Caine's diagnostic criteria for WE, due to biopsychosocial factors.[14,22] Housing instability, isolation, substance use, depression, and acute psychosis can lead to malnutrition.[23] These risk factors should alert a clinician to the possibility of this reversible cause of encephalopathy.

In the cases presented here, a causal relationship between WE and the patient's psychiatric history can be observed in the first case due to worsening depression with suicide attempt and in the second case due to paranoia with isolation. Melchionda et al[24] alluded to this association between poor oral intake in depression leading to WE.

The inherent bias of alcohol-related WE is slowly dissipating as more diverse cases are reported. However, patients with psychiatric history face added stigma from mental illness, resulting in further risk of missed diagnosis. We observe this in case 2, wherein the patient was initially triaged for psychiatry admission in the setting of prominent paranoia and a history of psychosis, overlooking the history of malnutrition.

A retrospective study by Guirguis et al[22] recognized that WE is underdiagnosed and undertreated in inpatient psychiatry. In the inpatient psychiatric unit, patients with possible WE symptoms (1.85%), or at high risk of WE (7%), were identified through chart review. None of the cases received HDIV, and prescriptions were limited to 100-mg oral dosing.

Although not discussed in the study, access to IV treatment in inpatient psychiatry is difficult, possibly due to ligature risk precautions. If permitted, it may require constant observation, creating strains on staffing. In case 1, IM thiamine provided parenteral supplementation inside the psychiatric unit, bypassing this problem. Other case reports of successful treatment with IM thiamine have been documented.[25]

Need for Higher Doses and Prolonged Treatment

Despite the establishment of international treatment guidelines for WE, the underprescribing of IV thiamine is evident in the literature. In many cases, the lack of homogenous consensus in local institutions has led to underprescribing. Current recommendations of treatment for 3–5 days, or until symptom resolution, could lead to an insufficient treatment regimen. Shorter courses of treatment, even if given at an appropriate dosage, can pose the risk of partial resolution with symptom relapse that can be easily overlooked, as observed in case 1. In case 2, a gradual titration in dosage helped confirm adequate thiamine replacement.

Case reports by Infante et al[1] and Hutcheon[26] suggest ongoing treatment for months and support a gradual reduction in dosage prior to discontinuation. In relapse cases, IM administration could be a solution for extended parenteral treatment outside an acute medical setting. A similar case was presented by Shoaib et al[25] with IM use in the phase of relapse on discontinuation of IV administration.

A study by Nakamura et al[20] looked at prescription practices in a teaching hospital. Of 5,236 thiamine orders, 29% (n = 1,531) were IV; only 10% (n = 150) of IV orders met HDIV criteria. Of patients with encephalopathy receiving IV thiamine, only 2.1 received doses consistent with Royal College of Physicians guidelines. They also found an association between HDIV prescription and consultation-liaison psychiatry involvement, supporting our notion that comanagement is associated with better prescription practices.

In another study, Thomson et al[21] audited case notes and local policies of various hospitals in the United Kingdom regarding their management of WE. They found that most hospitals have separate institutional protocols that differ from established guidelines. Most physicians are unaware of these protocols, leading to incorrect prescription practices, with only approximately 25% of patients with WE, or at high risk for WE, undergoing appropriate treatment in 1 audited hospital. Day et al[19] modified the computerized provider order entry system promoting the use of HDIV thiamine prescription, leading to a significant improvement in prescription practices. Systemic interventions like this may be necessary to ensure appropriate management of patients until universal guidelines are available.

Comanagement With Neuroleptics and When to Add

As previously discussed, the presentation of WE and psychiatric decompensation can overlap and have causal relationships with one another. Both cases presented here required close monitoring by consultation-liaison psychiatry and internal medicine teams to optimize their treatment. Comanagement can lead to improvements in thiamine prescription practices and decrease mortality.[20]

The *DSM-5* criteria for most psychiatric diagnoses include a need for the medical cause of disease to be ruled out. In both cases described here, the persistence of psychiatric symptoms such as mood lability, delusions, or hallucinations despite HDIV thiamine pointed toward a comorbid psychiatric illness. This was especially true when considering that their initial presentation pointed toward the prodromal phase of psychiatric illness. Addition of neuroleptics in this case will not only clarify diagnostic questions as more specific WE symptoms persist, but also protect the patient from relapse due to reemergence of poor oral intake as a result of disorganization or mood disturbances. In both cases, long-term treatment with neuroleptics was beneficial.

Despite a lack of evidence regarding the use of neuroleptics in WE, we can infer modalities from similar encephalopathic states like delirium. A study by Bush et al[27] evaluated the use of antipsychotics in delirium, highlighting possible risk factors of decreased seizure threshold, extrapyramidal symptoms, neuroleptic malignant symptoms, and prolongation of the QTC. This should be considered when managing patients with WE who could have a complex medical history. Wu et al[28] published a meta-analysis on the effectiveness and safety of pharmacologic interventions in delirium, which included numerous antipsychotics. Results revealed all-cause mortality is unchanged from placebo with the use of neuroleptics in delirium.[28]

Medical comorbidities that could affect treatment are broad, ranging from metabolic derangements to postsurgical states to lower seizure threshold. As observed in case 2, a finding of hypomagnesemia may have contributed to refractoriness to thiamine treatment in WE. A 1974 study by Traviesa[29] highlighted the degree of delayed transketolase activity, an enzyme directly implicated in the development of WE, in the setting of hypomagnesemia. Transketolase requires both thiamine monophosphate and magnesium as cofactors for proper activity. It was observed that hypomagnesemia may be a major cause of thiamine refractoriness seen occasionally in patients with WE.

Given the lack of broader studies and randomized controlled trials, most supporting evidence stems from case reports, which are numerous despite their heterogeneity. A very limited number of cases associate WE with psychiatry, making the current article a vanguard and new approach for the consultation-liaison perspective.

CONCLUSION

The differential diagnosis of WE should be considered by clinicians not only in patients with alcohol use disorder but also in patients at risk for malnutrition, such as psychiatric patients and others with complicated medical and surgical illnesses. Given its overlap in presentation, comanagement by consultation-liaison psychiatry and the primary team is likely to improve patient outcomes. HDIV continues to be underutilized. Prolonged treatment regimens may be needed and should be dictated by a clinical response with careful monitoring for relapse due to the inconsistency of current guidelines.

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Supplementary Material: See accompanying pages.

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Supplementary material follows this article.

The Primary Care Companion
FOR CNS DISORDERS

Supplementary Material

Article Title: Diagnosis of Wernicke's Encephalopathy in Patients With a Psychiatric History: A Case Series and Literature Review

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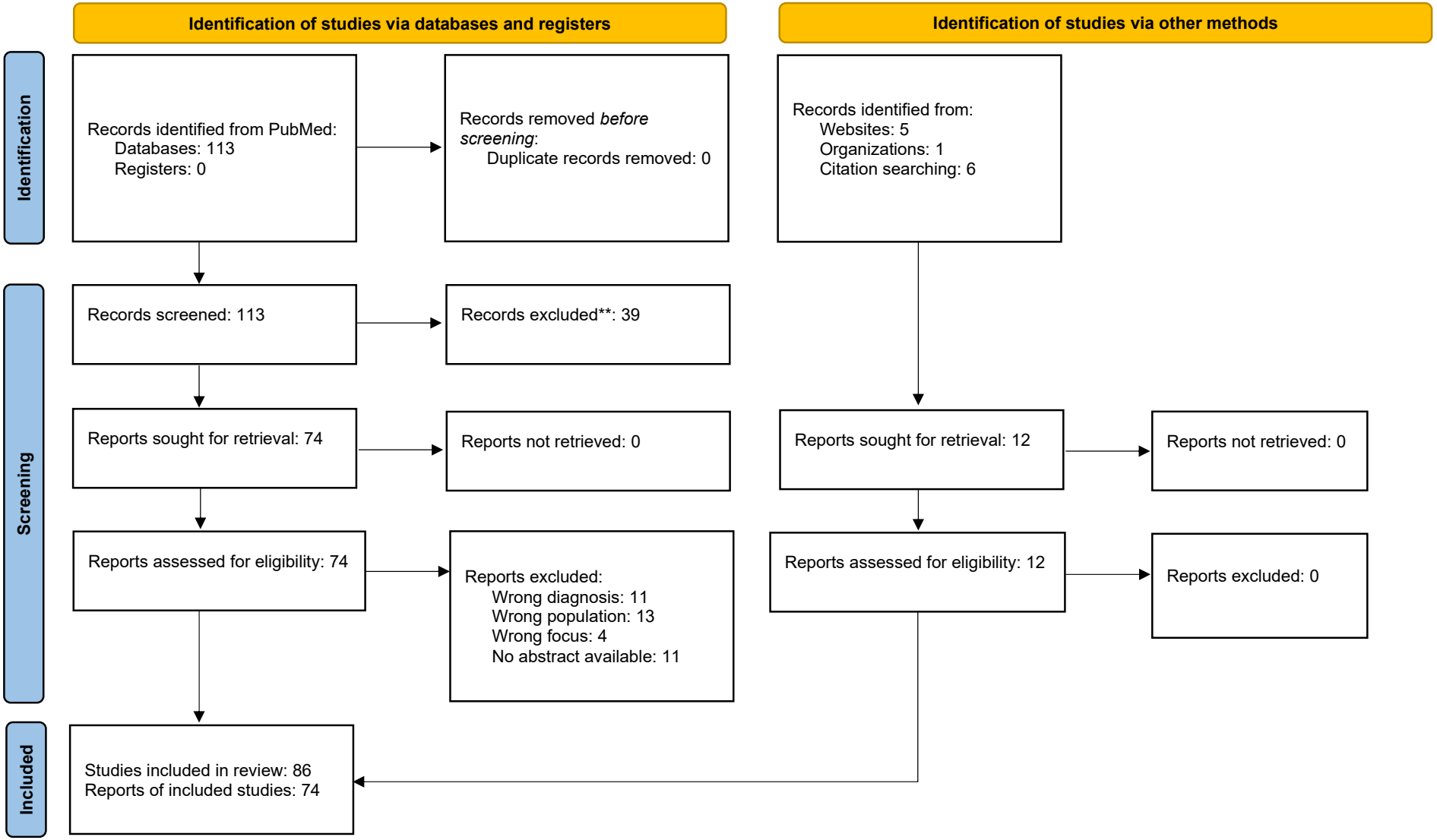
LIST OF SUPPLEMENTARY MATERIAL FOR THE ARTICLE

DISCLAIMER

Figure 1. PRISMA 2020 Flow Diagram for New Systematic Reviews Which Included Searches of Databases, Registers, and Other Sources



*Consider, if feasible to do so, reporting the number of records identified from each database or register searched (rather than the total number across all databases/registers).
**If automation tools were used, indicate how many records were excluded by a human and how many were excluded by automation tools.

From: Page MJ, McKenzie JE, Bossuyt PM, Boutron I, Hoffmann TC, Mulrow CD, et al. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. BMJ 2021;372:n71. doi: 10.1136/bmj.n71. For more information, visit: http://www.prisma-statement.org/

Table 1. List of Reviewed Articles

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*The order of the articles in this table is aleatory.

Table 2. List of Articles on Wernicke's Encephalopathy and Psychiatry

Early detection and successful treatment of Wernicke's encephalopathy in outpatients without the complete classic triad of symptoms who attended a psycho-oncology clinic.Onishi H, Ishida M, Tanahashi I, Takahashi T, Ikebuchi K, Taji Y, Kato H, Akechi T.Palliat Support Care. 2018 Oct;16(5):633-636. doi: 10.1017/S1478951518000032. Epub 2018 Feb 26.PMID: 29478423

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