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Original Article

What is the impact of thiamine deficiency on cognitive function in patients with alcohol use disorder? – A systematic review

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ABSTRACT

Cognitive impairments are frequently observed in patients with Alcohol Use Disorder (AUD). Thiamine deficiency is often found in AUD patients and has been suggested as a possible cause of cognitive impairments. While thiamine deficiency is not consistently present in all AUD patients with cognitive deficits, thiamine is traditionally prescribed to patients with AUD to treat or prevent cognitive impairment.

To better understand the relationship between thiamine levels and cognitive impairments in AUD patients, we conducted a systematic literature review following the Cochrane guidelines and adhering to the PRISMA-P framework. Additionally, this review is registered in PROSPERO under the reference CRD42024522058. Our research question was: "what is the impact of thiamine deficiency on cognitive function in patients with AUD?".

The studies included in this review assessed thiamine levels in AUD patients and found values at or above the threshold for many measures of thiamine deficiency. Despite baseline thiamine levels being above the cutoff for deficiency in these studies, many still identified a correlation between thiamine levels and cognitive function with lower thiamine levels associated with cognitive impairments in AUD patients.

This review indicates that there is a relationship between thiamine levels and cognitive function in AUD patients, even in the absence of thiamine deficit. The cognitive domains particularly affected are visuospatial/executive ability, abstraction, attention, verbal fluency, and memory scores, notably delayed memory. Additionally, studies have demonstrated that thiamine supplementation in AUD patients, even in the absence of thiamine deficit, leads to improvements in cognitive function.

1. Introduction

Cognitive impairments, frequently observed in Alcohol Use Disorder (AUD) patients, range from severe memory impairment with other cognitive functions relatively preserved seen in Korsakoff's Syndrome (KS) [1] and dementia [2] to mild cognitive deficits primarily affecting executive functions and memory [3].

Chronic excessive alcohol consumption is believed to contribute to cognitive impairments in patients with Alcohol Use Disorder. However, the exact mechanisms behind these impairments are not fully understood. Theories range from a direct toxic effect of alcohol on neurons to an indirect effect through thiamine deficiency [4–6].

In fact, alcohol use disorder has been linked to malnutrition and severe vitamin deficiencies, particularly thiamine [7–9] with reports indicating that approximately 30–80 % of AUD inpatients undergoing alcohol detoxification treatment exhibit thiamine deficiency [10,11].

Thiamine deficiency may stem from dietary insufficiencies, impaired thiamine absorption due to alcohol consumption, or storage issues caused by liver disease [12]. Furthermore, impairment of thiamine utilization related to abnormalities in the transketolase protein (which uses thiamine as a co-factor) has also been described in AUD patients [13].

One significant consequence of thiamine deficiency in AUD patients is the onset of Wernicke's Encephalopathy (WE), a neurological condition characterized by confusion, ataxia, and oculomotor abnormalities such as nystagmus and ophthalmoplegia [9,14]. Untreated WE cases can

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progress to Korsakoff's Syndrome (KS), marked by enduring amnesia and ataxia [15]. Although the pathomechanism underlying WE remains unclear [9], prevention and treatment of WE and KS is widely conducted with thiamine supplementation, as examined by Dingwall et al. (2022) [16].

Although thiamine deficiency is not consistently found in AUD patients with cognitive deficits [5,11] Listabarth et al. (2023) [9] found in a secondary analysis of data from a study of thiamine supplementation in AUD that the extent of the response to thiamine (measured as the increase in serum thiamine pyrophosphate level) correlated with improvement in a memory test.

Therefore, despite thiamine supplementation already being widely recommended, we believed the extent of inconsistencies merited a systematic literature review addressing the question: "What is the impact of thiamine deficiency on cognitive function in patients with alcohol use disorder?".

2. Material and methods

Methods: The systematic review was structured according to Cochrane guidelines for systematic literature reviews and adhered to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA-P) framework. Additionally, this review is registered in PROSPERO under the reference CRD42024522058.

Research question: we defined the research question "what is the impact of thiamine deficiency in cognitive function in patients with alcohol use disorder?".

2.1. Inclusion/exclusion criteria

The inclusion/exclusion criteria defined were:

Study design: cross-sectional studies, longitudinal studies, randomized and non-randomized clinical trials, case reports and case-control studies.

Population: adult patients (> 18 years old) with AUD diagnosis, according to the Diagnostic and Statistical Manual of Mental Disorders, Fourth or Fifth Edition, either in inpatient or outpatient treatment. Studies with patients with history of other substance use disorder diagnosis (except nicotine and caffeine) were excluded. Only articles published in English were selected.

Intervention: assessment of both blood thiamine and cognitive function of AUD patients.

Comparison: compare cognitive function between patients with and without thiamine deficiency.

Outcome: to assess specific cognitive domains impaired in thiamine deficiency in AUD patients and to explore the relationship between thiamine levels and cognitive function in patients with AUD.

2.2. Information sources and research strategies

Electronic records from MEDLINE, CENTRAL (Cochrane Library), and Web of Science databases up to December 2023 were searched, along with the reference lists of identified studies.

The research strategy will involve searching for keywords or MeSH terms. Keywords or MeSH terms, along with the Boolean operators "OR" and "AND," will be used to combine the search terms. The keywords to be included are "thiamine deficiency," "cognition," "cognitive function," "cognitive dysfunction," "cognitive impairments," "alcoholism," "alcohol dependence," "alcohol addiction," and "alcohol use disorder." These keywords will be grouped into three combined free-text blocks: one on thiamine deficiency, one on cognition, and another one on alcohol.

Study selection:

The titles of the reports retrieved from various databases were extracted, and duplicate entries were eliminated. Two reviewers independently screened the titles and abstracts to identify reports potentially meeting the inclusion criteria. Full texts of the selected reports meeting the inclusion criteria were then evaluated to confirm their eligibility. Any discrepancies between the reviewers were resolved through consensus. A list outlining the excluded reports along with reasons for their exclusion was compiled.

2.3. Data collection process

One reviewer entered the data from each study into a standardized table, while another reviewer verified the accuracy of the extracted data.

2.4. Data items

The standardized table comprised the author's name, publication year, country, study design, inclusion and exclusion criteria, sample size, patient demographics including age and sex, daily alcohol use, treatment setting, thiamine level evaluation, cognitive function assessment, main outcome (differences in cognitive function between patients with and without thiamine deficiency).

This information was synthesized narratively and supplemented with tables due to the variety of interventions examined.

2.5. Risk of bias assessment

Two reviewers will assess the methodological quality of the selected studies independently. The risk of bias in observational studies will be evaluated using the Newcastle-Ottawa Scale (NOS). The NOS scale awards 4 stars for Selection of exposure and control groups, 2 stars for comparability between exposure and control groups, and 3 stars for outcome evaluation. Disagreements were solved by consensus.

2.6. Ethics

This review utilized anonymized data from previously published studies in literature, exempting it from submission to an ethical committee.

3. Results

3.1. Study selection

A total of 268 records were identified (250 through database searching and 18 additional records through bibliographic review) and after duplicate removed a total of 117 records remained. Out of those 117 articles whose abstracts were reviewed, 21 were selected for full-text assessment. Among these, 7 met the eligibility criteria and were thus included in the review (Fig. 1).

3.2. Synthesis of study characteristics

The synthesis of study characteristics included in the review is depicted in Table 1.

All studies, apart from Pitel [4], were conducted among AUD patients undergoing inpatient treatment. The sample sizes across the studies ranged from 20 to 107 patients.

3.3. Risk of bias within studies

Risk of bias was assessed using the Newcastle-Ottawa Scale for longitudinal studies is presented in Table 2.

3.4. Assessment of thiamine

Out of the 7 selected studies [4,5,9,11,12,15,16], baseline thiamine levels were evaluated in 5 of them [4,9,11,12,15] and 3 of them determined thiamine levels after thiamine supplementation [5,9,16].

Among the 5 studies in which thiamine concentrations were assessed

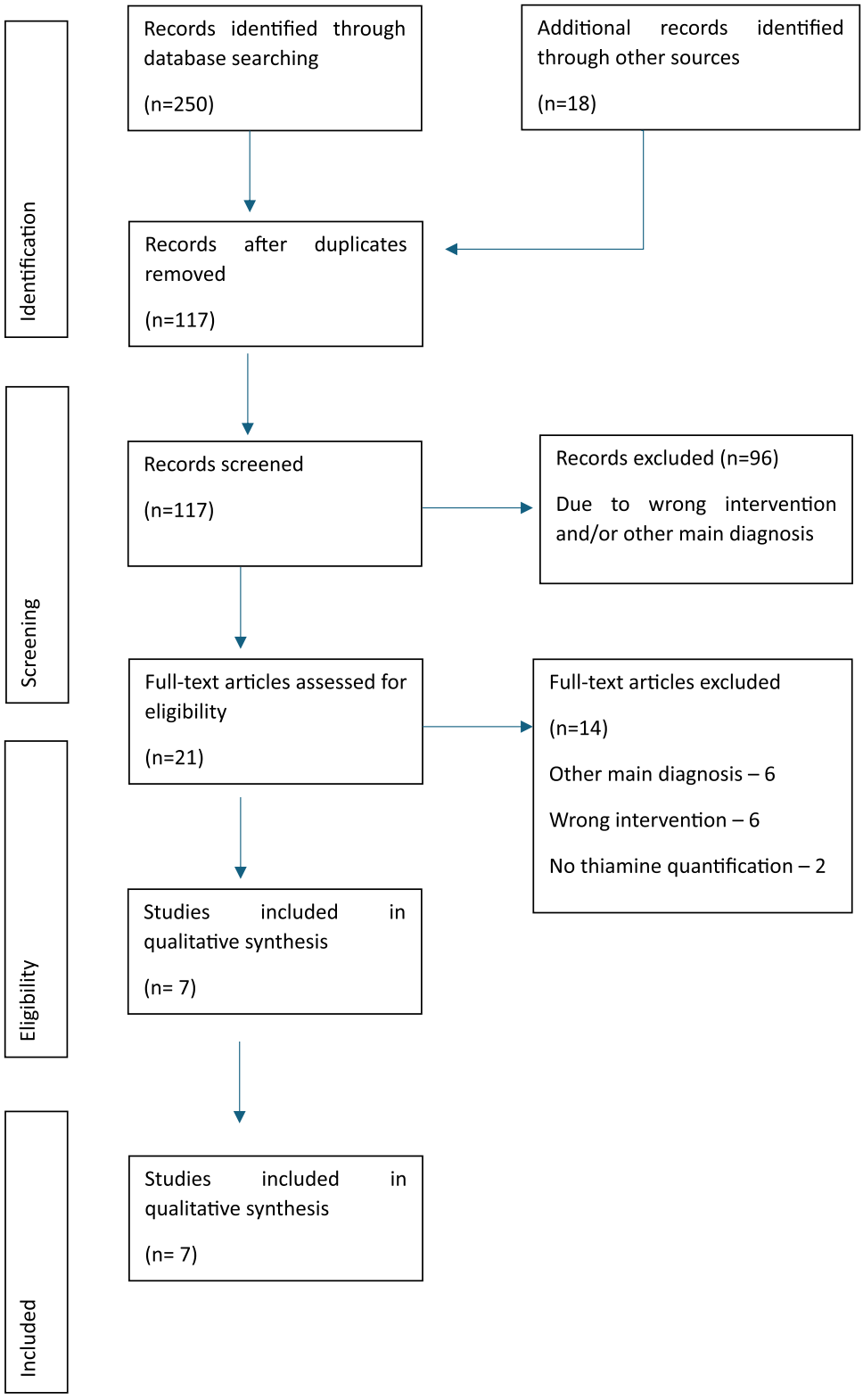

Fig. 1. Flowchart PRISMA of study selection.

at baseline during the first evaluation of AUD patients [4,9,11,12,15], all patients presented thiamine values within or above the reference range, except in the study by Bonnet, U., et al., 2023 [15], where one patient had a value below the reference range (25 ng/mL compared to a cutoff of 28 ng/mL).

In the 5 studies that assessed thiamine levels at baseline [4,9,11,12,15], Pitel [4], Listabarth [9] Gautron [11], Dingwall [12] and Bonnet [15] quantified thiamine pyrophosphate (TPP) blood levels. To clarify, thiamine pyrophosphate (TPP) and thiamine diphosphate (TDP) are two names for the same molecule, which constitutes the biologically active form of thiamine and accounts for approximately 80 % of the total thiamine content in whole blood. For consistency, the TDP nomenclature will be used from this point onward throughout the manuscript, as this is the form officially recognized by the Nomenclature Committee of

Table 1
Synthesis of study characteristics.

Authors	Country	Study design	Inclusion criteria	Exclusion criteria	Sample size	Patient gender	Patient age	Daily alcohol use	Setting of treatment	Thiamine evaluation	Cognitive function assessment	Outcome
Gautron et al., 2018	France	Retrospective study	AUD diagnosis	Decompensated cirrhosis (Child B or C) or hospitalization <3 months ago	94 (thiamine dosing only in 54)	85 % male	49.25 ±12.05	198±111 g/day	Inpatient	All results within RV	MoCA assessed with 10 days of detoxification	No low blood thiamine levels were found. The association with low MoCA score was not tested because there were no low blood thiamine levels.
Bonnet et al., 2023	Germany	Prospective observational cohort study	AUD diagnosis; alcohol dependence for > 5 years; age> 18y; being fluent in Germany; presenting no active comorbid psychiatric or somatic disorder requiring superseding treatment; having no supplemental intake of thiamine prior to admission; presenting no other substance use (apart from tobacco) during the 8-week period prior to admission.	Documented alcohol use during the study (breath analysis or urinalysis); withdrawal of study participation; occurrence of a relevant comorbidity requiring additional intervention and stabilization.	100	79 % male	47.71 ± 10.97	No information	Inpatient	All results within the reference range except one; Mean thiamine blood level at baseline was 62.0 ± 16.1 ng/mL, median value 60.3 ng/mL.	MoCA and FAB	Baseline thiamine blood levels were both significantly associated to cognitive performance (assessed by FAB and MoCA). After analysis including covariates, the association between cognitive performance assessed by MoCA was confirmed but the association between cognitive performance assessed by FAB disappeared. Cognitive function improved significantly after thiamine supplementation.
Pitel AL et al., 2011	USA	Cross-sectional study	AUD diagnosis;	Presence of other DSM-IV diagnoses but mood disorders other than bipolar were not exclusion criteria; history of central nervous system trauma or serious medical conditions. No Korsakoff syndrome diagnosis.	56	73 % male	46.29 ± 11.45	Only information on lifetime alcohol intake (Kg) 1149.00 ± 956.63 Kg	Outpatient	Whole blood was collected from 28 patients, frozen and assessed for TDP.	Wechsler memory scale-Revised, Trail Making Test, semantic fluency and letter fluency tests, Complex Figure Test, digit symbol subtest of WAIS-R, fine finger movement test and grooved pegboard test.	TDP levels did not differ significantly between controls and alcoholics. A selective relation was identified between poorer memory scores and lower TDP levels.
Listabarth et al., 2023	Austria	Longitudinal study	AUD diagnosis; age between 18 and 65 years; have adequate language skills.	Presence of a condition accompanied by impaired cognitive functioning, such as any type of dementia, other neurodegenerative diseases or specific psychiatric or medical conditions. Patients who had received any form of thiamine substitution within the last 4 weeks.	50	54 % male	47.9 +/ 8.5	Median 154.0 g	Inpatient	TDP levels for the sample (184.71± 61.1 nmol/l) correspond to values above the cutoff for the RV	MoCA conducted at 5–7 day of detoxification and repeated at 12–14 day, not at baseline.	Significant positive correlation between the changes in TDP levels after TS and delayed memory (subdomain from MoCA).

(*continued on next page*)

Table 1 (*continued*)

Authors	Country	Study design	Inclusion criteria	Exclusion criteria	Sample size	Patient gender	Patient age	Daily alcohol use	Setting of treatment	Thiamine evaluation	Cognitive function assessment	Outcome
Dingwall et al., 2015	Australia	cross-sectional study	Patients admitted to emergency department for a variety of reasons, including alcohol abuse; AUD diagnosis; age between 18 and 65 years;	Serious medical conditions with need to transfer to the intensive care unit; pregnancy	105	59 % male	40.77 ± 10.32	All patients had > 60 g alcohol use/ day	Inpatient	Blood analysis to assess TDP; all TDP concentrations were above the defined range for deficiency (mean 175.64 ± 47.62)	RUDAS ($n = 21$)	No patient had TDP concentrations below the reference range. No correlation was found between cognitive test scores and TDP concentrations.
Dingwall et al., 2022	Australia	Randomized Clinical Trial	Age between 18 and 65 years, history of heavy alcohol use in the last 3 months defined by AUDIT-C scores above 4 or consumption of greater than 60 g alcohol per day or 80 g per binge.	Pregnancy, acute neurological or cognitive impairment clearly unrelated to presumed thiamine deficiency or WKS, intubation, vasopressor therapy for hypotension, dialysis treatment, acute exacerbation of a psychiatric illness, treatment with parenteral thiamine in the past 4 weeks.	75	64 % male	44.6 ± 11.3	No information	Inpatient	No baseline blood thiamine assessment, only TDP quantification after TS supplementation in 3 different doses (100, 300 and 500 mg)	RUDAS, Cogstate and Story recall memory test, performed on the 3rd and 5th day of alcohol abstinence	No significant differences were observed between any of the dosage of TS on cognitive functioning.
Coulbault et al., 2019	France	Cross-sectional study	AUD diagnosis; inpatient addiction treatment	No information	20	60 % male	45.2 ± 9.4	18.04 ± 7.2	Inpatient	Th, TMP and TDP were determined in serum and whole blood after TS. There was no baseline thiamine assessment.	MoCA and BEARNI performed at 10.0 ± 4 days	Thiamine concentrations were significantly higher in whole blood and in serum of AUD patients with moderate to severe cognitive deficits when compared to controls. Adjusted for covariates, the percentage of thiamine in serum in AUD patients negatively correlated with MoCA and BEARNI.

Table 2
Risk of bias across longitudinal studies.

Author	Selection	Comparability	Outcome
Gautron, 2018	***	**	**
Bonnet, 2023	***	**	**
Pitel, 2011	**	**	**
Listabarth, 2023	***	**	***
Dingwall, 2022	****	**	***
Dingwall, 2015	***	**	**
Coulbault, 2019	***	**	***

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In the 3 studies that assessed thiamine levels after supplementation [5,9,16], Coulbault [5] assessed whole blood and serum concentrations of thiamine and metabolites (thiamine monophosphate (TMP) and TDP) and Listabarth [9] and Dingwall [16] assessed TDP levels.

3.5. Assessment of cognitive function

General cognition tests, such as MoCA, were used to assess cognitive function in four studies [5,9,11,15], and the Rowland Universal Dementia Assessment Scale (RUDAS) in two other [12,16]. Additional neuropsychological screening tests, like the Frontal assessment Battery (FAB), were used by Bonnet [15], and the Brief Evaluation of Alcohol-Related Neuropsychological Impairments (BEARNI) by Coulbault [5]. A more comprehensive battery of neuropsychological tests was used in two studies [4,16]. Pitel [4] utilized the Wechsler Memory Scale-Revised (WMS-R), Trail Making Test (TMT), semantic fluency and letter fluency tests, Complex Figure Test, digit symbol subtest of WAIS-R, fine finger movement test, and grooved pegboard test. Dingwall [16] used CogState that is a computerized cognitive assessment, and a Story Recall Memory Test that is a verbal memory assessment tool like the logical memory subtest of the Wechsler memory scale.

Only five studies specified the timing of neuropsychological test administration to patients [5,9,11,12,16]. One study conducted assessments after 24 h, another between the 5–7th day of alcohol detoxification [9], another on the 3rd and 5th day of alcohol abstinence [16], another on the 10th day of alcohol abstinence [11] and the last one between the 10–14th day [5].

All studies found that some AUD patients included in the sample presented cognitive impairments.

3.6. Relationship between thiamine levels and cognitive function

Among the five studies that evaluated thiamine levels at baseline [4,9,11,12,15], four examined the relationship between thiamine levels and cognitive function in patients with AUD [4,11,12,15]. Bonnet [15] identified a significant association between lower thiamine blood levels and lower MoCA results, with the strongest association observed for visuospatial/executive ability and abstraction MoCA subscales, and smaller effect sizes found for attention, language, and delayed recall/-memory subscales. Pitel[4] identified a statistically significative relationship between poorer memory scores and lower TDP levels and found only a tendency to correlate with executive functions, although TDP levels did not significantly differ between controls and alcoholics. Dingwall [12] found no correlation between RUDAS scores (used to assess cognitive function) with TDP concentrations and one study did not investigate the potential association between thiamine deficit and cognitive function found in neuropsychological assessments because all patients presented thiamine levels within the normal reference value range [11].

In the3 studies that assessed thiamine levels after supplementation and examined the relationship between thiamine levels and cognitive function in patients with AUD [5,9,16], Coulbault [5] found that after adjusting for covariates (education), the percentage of thiamine in serum in AUD patients negatively correlated with cognitive function assessed by MoCA and BEARNI. Listabarth et al. (2023) [9] found in a secondary analysis of data that the extent of the response to thiamine correlated with improvement in a memory test, and Dingwall [16] found no significant differences between any of the dosages of thiamine supplementation on cognitive functioning.

4. Discussion

This systematic literature review aimed to clarify the relationship between thiamine levels and cognitive impairments observed in patients with AUD.

The studies included in this review, which assessed thiamine levels in patients with AUD, reported values at or above the threshold for thiamine deficiency. These findings align with existing literature which indicates that AUD patients do not necessarily exhibit deficient blood thiamine levels [4,5,11,17]. Additionally, most studies included in the review identified cognitive deficits in AUD patients, consistent with previous reports for this population [3,18–22]. This indicates that the presence of cognitive impairments in AUD patients does not necessarily correlate with the presence of thiamine blood deficiency. One possible explanation is that thiamine transport to the brain may be impaired in AUD patients. So, the findings from this review indicate that thiamine deficiency, as measured in whole blood, is uncommon in patients with Alcohol Use Disorder (AUD) and does not appear to predict cognitive impairment in this population.

Indeed, although thiamine blood levels were found to be above the cutoff for deficiency at baseline assessments in some studies included in this systematic review, many of these studies still identified an association between thiamine levels and cognitive function in AUD patients, with lower thiamine levels correlating with impairments on cognitive function. Furthermore, research has demonstrated that thiamine supplementation can benefit AUD patients even in the absence of thiamine deficiency, leading to improvements in cognitive function [9,15]. The association between thiamine intake and cognitive function has also been identified in older adults [23] indicating that enhancing thiamine intake may contribute to improve cognitive performance. One possible explanation is that patients with AUD may have an increased need for thiamine concentrations to compensate for potential impairments in its utilization at the cellular levels or they may require higher thiamine concentrations to enhance its delivery across the blood-brain barrier to the brain. Nevertheless, the review revealed that higher blood thiamine levels, achieved through supplementation, seem to improve the cognitive impairments commonly observed in AUD patients.

Some studies included in this review also aimed to identify which specific cognitive domains were affected by thiamine levels in AUD patients. These studies suggest that thiamine levels influence several cognitive domains, including visuospatial and executive abilities, abstraction, attention, verbal fluency, and memory, with impact on delayed memory [4,9].

The limitations of this review are the lack of a defined cutoff for thiamine, the poor quality of some studies, and the limited number of studies that hinder generalization. Additionally, the heterogeneity of AUD patients in clinical samples and the significant methodological challenges in the literature on this topic in accounting for other influencing factors that contribute to cognitive disturbances, such as withdrawal severity and medications prescribed during inpatient treatment, are noteworthy[24]

This literature review revealed heterogeneity in assessing thiamine deficiency, highlighting the lack of a standardized measurement method used to define thiamine deficiency. Some studies identified thiamine deficiency based solely on the presence of Wernicke's encephalopathy (WE) or Korsakoff syndrome (KS) diagnoses without measuring thiamine levels. Among studies that did quantify thiamine, there was variation in the parameters used and different measurement methods. Some studies measured thiamine in whole blood, while others assessed it in

serum or erythrocyte concentrations or in terms of its active metabolites such as TDP. Another limitation is that although thiamine deficiency has long been associated with AUD there are few studies that have specifically aimed to clarify the exact role of thiamine deficiency on cognitive function in AUD patients, and some studies had poor quality, as they included small samples and no control groups.

Despite thiamine supplementation is recommended in most guidelines for AUD and alcohol withdrawal to prevent WE and KS, even in the absence of evidence of serum deficiency [25], further research is needed to evaluate the effectiveness and optimal dosing of thiamine supplementation in AUD patients to improve their cognitive function.

5. Conclusions

The review indicates that there is a relationship between thiamine levels and cognitive function in AUD patients, even in the absence of thiamine deficit. The cognitive domains particularly affected are visuospatial/executive ability, abstraction, attention, verbal fluency, and memory scores, notably delayed memory. Additionally, studies have demonstrated that thiamine supplementation in AUD patients, even in the absence of thiamine deficit, leads to improvements in cognitive function.

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Author contribution statement

Each author certifies that their contribution to this work meets the standards of the international Committee of Medical Journal Editors.

Declaration of competing interest

The authors have no conflicts of interest to declare.

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