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Research Article

Vitamin E as a Therapeutic Agent for NAFLD: A Systematic Review of Randomized Controlled Trials

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Abstract

Non-alcoholic fatty liver disease (NAFLD) is one of the most common fatty liver diseases, leading to biochemical and histological disorders in NAFLD. It is characterized by kinds of pathologies, ranging from simple non-alcoholic fatty liver (NAFL) to serious non-alcoholic steatohepatitis (NASH) with the complication of steatosis, fibrosis, NASH cirrhosis and hepatocellular carcinoma (HCC). The most important potential risk factors including insulin resistance and increased oxidative stress, especially vitamin E, have significant influence on the treatment for NAFLD. Vitamin E, as a key fat-soluble antioxidant, presents potential therapeutic value in the intervention of NAFLD. At present, there are differences in the intervention measures proposed by various studies, and the efficacy of corresponding intervention measures has not yet reached a unified conclusion. To analyze the relevant research on the combination therapy of vitamin E in NAFLD patients, and to explore the intervention effect of vitamin E combination therapy on their biochemical indicators and histological abnormalities. PubMed, EMBASE, Medline, Cochrane, ScienceDirect, Web of science, and Google Scholar Database were systematically searched to screen studies on the intervention effect of vitamin E combination therapy on NAFLD from January 2005 to April 2025. A total of 9 randomized controlled trials (RCT) were included, which included 885 participants with an average age of 35~48.04 years. The combination therapy of vitamin E had significantly improved liver function indicators (aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase (ALP), and gamma glutamyl transferase (GGT)) in NAFLD patients in most studies, and had also positive effects in improving liver histological parameters (NAFLD activity score (NAS), steatosis, fibrosis score, and inflammation), but there was heterogeneity among these studies. In addition, some studies did not observe significant improvement in ballooning, and only one study showed that combination therapy had no significant effect on liver function testing. The combination therapy of vitamin E has potential benefits in improving liver function indicators and some histological parameters in NAFLD patients, but its efficacy varies among studies. In the future, more high-quality and long-term RCT need to be conducted to further clarify the optimal regimen and applicable population of vitamin E in the treatment of NAFLD, providing a more comprehensive and reliable reference for clinical practice.

Keywords

Vitamin E, Non-alcoholic Fatty Liver Disease, RCTs, Systematic Review

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1. Introduction

Non-alcoholic fatty liver disease (NAFLD), one of the common chronic liver disease, is characterized by hepatic fat accumulation exceeding 5% of liver weight [1], encompassing a disease spectrum from simple non-alcoholic fatty liver (NAFL) to serious non-alcoholic steatohepatitis (NASH), which may further progress to liver fibrosis, cirrhosis, and even hepatocellular carcinoma (HCC) [2]. Epidemiologically, NAFLD affects over 30% of the global adult population, with a higher prevalence in industrialized nations reaching 40-50% due to sedentary lifestyles and high-calorie diets [3-5]. The pathogenesis of NAFLD is multifactorial, with insulin resistance (IR) as a central driver [6]. IR promotes hepatic de novo lipogenesis and impairs triglyceride export, leading to lipid accumulation [7]. Importantly, accumulated lipids in hepatocytes induce mitochondrial dysfunction, which enhances the production of reactive oxygen species (ROS) [8]. Excessive ROS overwhelms the cellular antioxidant defense system, triggering oxidative stress—a key contributor to hepatocellular injury, inflammation, and fibrosis [9]. Oxidative stress mediates lipid peroxidation, generating toxic by-products (e.g., malondialdehyde) that activate hepatic stellate cells, initiating fibrogenesis [10]. NAFLD also exhibits strong comorbidities with metabolic syndrome components: 50-70% of NAFLD patients have type 2 diabetes mellitus (T2DM), 60-80% are obese, and hyperlipidemia is present in 70-80% of cases, creating a bidirectional relationship where metabolic derangements exacerbate liver injury and vice versa [11].

Despite its high prevalence and related metabolic syndrome, there is no standard drug approved for the treatment of NAFLD [12]. Currently, lowering the calorie intake, weight loss, and exercise is recommended for the treatment of NAFLD. In addition, some drugs including Silymarin, symbiotic, alpha-tocopherol and spironolactone have also been tried. However, recommending these agents may be a difficult thing due to their side effects and safety in NAFLD [13, 14]. Consequently, it seems necessary to find relatively safe drugs for the treatment of NAFLD. Beta vulgaris, Silymarin and Spironolactone reduce blood sugar, induce glucose storage as glycogen and has been used to treat different liver disorders, and the level of liver enzymes such as aspartate transaminase (AST), alanine transaminase (ALT), and alkaline phosphatase (ALP) is also decreased [11, 15, 16]. Conjugated linoleic acid (CLA), one of the promising dietary supplements, has anti-diabetic, anti-obesity and anti-inflammatory effects, acting through a variety of mechanisms to prevent and improve NAFLD [17]. However, vitamin E, a potent antioxidant, significant improves biochemical and histological abnormalities related with NAFLD, on account of the absence of adverse effects [18]. And this adjuvant medicine, has also potential benefits for the treatment of NAFLD [19]. Among potential agents, vitamin E (α-tocopherol, the most biologically active isoform) has garnered attention for its robust antioxidant properties and favorable safety profile [20]. As a lipid-soluble antioxidant, vitamin E exerts its effects through multiple mechanisms: (1) It scavenges lipid peroxyl radicals by donating a hydrogen atom from its chromanol ring, terminating lipid peroxidation chains and protecting membrane phospholipids from oxidative damage—critical for preserving hepatocyte integrity [21]. (2) It preserves mitochondrial function by reducing ROS-induced damage to mitochondrial DNA and respiratory chain complexes, thereby decreasing further ROS production [22]. (3) It upregulates the Nrf2-Keap1 pathway, a master regulator of antioxidant genes (e.g., HO-1, SOD), enhancing endogenous antioxidant capacity [23].

Beyond NAFLD, vitamin E's antioxidant actions contribute to its protective role in various oxidative stress-related diseases [24]. In cardiovascular disease, it inhibits low-density lipoprotein (LDL) oxidation, a key step in atherogenesis, reducing the risk of myocardial infarction in high-risk populations [25]. In T2DM, it ameliorates oxidative stress-induced insulin resistance by preserving pancreatic β-cell function and improving insulin signaling in peripheral tissues [26]. In neurodegenerative diseases (e.g., Alzheimer's), it mitigates oxidative damage to neurons, slowing cognitive decline [27]. These pleiotropic effects are clinically relevant for NAFLD patients, who often face comorbid cardiovascular and metabolic risks, making vitamin E a potential multitarget intervention [28]. Although there have been some studies where authors have compared the efficacy of vitamin E vs. placebo or vitamin E combination therapy vs. monotherapy among all population groups [11, 16, 17, 19, 29-33], systematic reviews that concurrently observed the effects of the combined multiple drugs plus vitamin E vs. vitamin E on NAFLD outcomes for the adult population are scarce. Given its established antioxidant mechanisms, favorable safety, and potential to address both liver-specific and systemic oxidative stress, this systematic review aims to synthesize evidence on vitamin E's therapeutic efficacy in adult NAFLD.

2. Methods

2.1. Information Sources

Two independent reviewers conducted an exhaustive literature search via electronic databases, adhering strictly to the PRISMA guidelines for systematic reviews. PubMed, EMBASE, Medline, Cochrane, ScienceDirect, Web of science, Google Scholar and clinical trial directories were searched for peer-reviewed randomized control trials conducted between January 2005 and April 2025. Two search themes were employed to query the databases, with the two themes combined via the Boolean operator 'AND'. The following keywords were used: 'Vitamin E', 'Tocopherols', 'Tocotrienols', 'Non-alcoholic Fatty Liver Disease', 'NAFLD', 'Fatty Liver, Non-alcoholic', 'Liver, Non-alcoholic Fatty', 'Non-alcoholic

Steatohepatitis', 'NASH'.

2.2. Inclusion and Exclusion Criteria

The inclusion criteria were as follows: (a) RCTs researches; (b) studies recruiting adult population (age ≥18); (c) biochemical markers (ALT, AST) and/or histological markers (steatosis, ballooning, inflammation, and fibrosis); (d) NAFLD/NASH must be diagnosed by radiology or histology; (e) published in English.

The exclusion criteria were as follows: (a) all studies conducted on children and adolescent population, (b) other common chronic liver diseases and secondary steatosis (severe alcohol consumption, hepatitis B, autoimmune hepatitis, etc.); (c) studies that were not RCTs were excluded from the study.

2.3. Data Extraction and Study Selection

Two researchers evaluated the studies, and in cases of disagreement, a third researcher was consulted. Relevant information was extracted from and organized in the included studies using Microsoft Excel, encompassing basic details (such as gender and age), intervention methods, serum liver function indicators (ALT, AST, ALP, GGT), and pathological parameters (NAS, ballooning degeneration, fibrosis).

A total of 7,665 studies were identified through the literature search. Of these, 2,142 were excluded due to duplication, and 5,336 were excluded as irrelevant records. The researchers then manually screened 192 studies, with a further 178 excluded after reviewing their titles and abstracts. Ultimately, 9 randomized controlled trials (RCTs) were included in the final analysis.

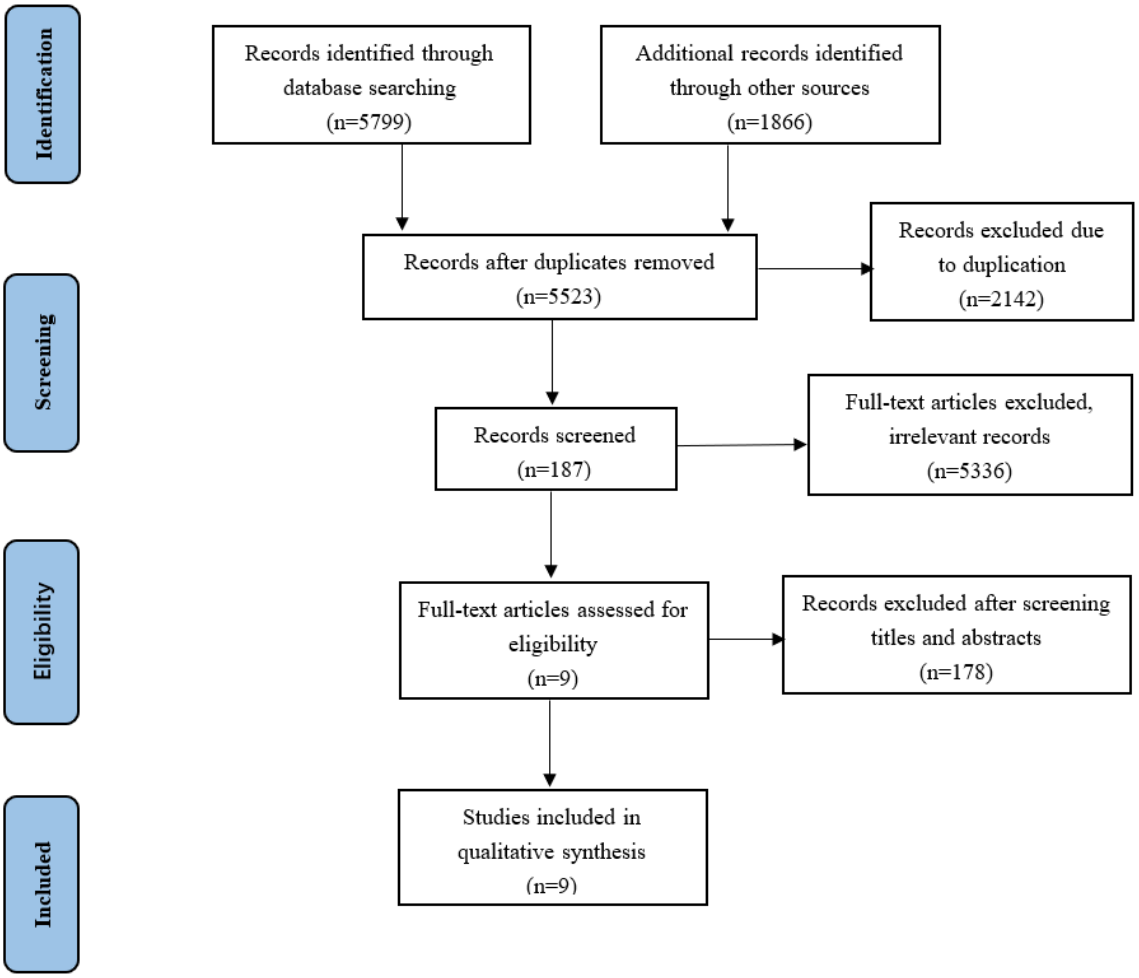

Figure 1. *Describes the literature review process.*

3. Results

A total of 9 RCTs published between January 2005 and April 2025 were identified. All the trials included a total of 867 participants with the mean age ranging between 30 and 65 years. The duration of studies ranged from 2 months to 24 months. Table 1 includes the RCTs added in chronological order and summarizes the population demographics and interventions used.

Table 1. *Summary of patient demographics, study design, and study interventions in the included RCTs.*

Authors and year	Type of study	Duration of study	Sample size	Mean age	Gender	Interventions used
Akhondi-Meybodi et al. 2020 [29]	RCT	4 months	Group1 (Vitamin E): 40 Group2 (Silymarin): 40	Group1: 44.2 Group2: 43.7	Group1: Females: 38% Males: 62% Group2: Females: 33% Males: 67%	Group1: Vitamin E 400 IU once Daily Group2: silymarin 140 mg BID once daily
Ekhlasi et al. 2017 [19]	double-blinded RCT	2 months	Group1 (symbiotic): 15 Group2 (alpha-tocopherol): 15 Group3 (symbiotic+alpha-tocopherol): 15 Group4 (placebo): 15	44	Females: 20% Males: 80%	Group1: symbiotic 1 g once daily Group2: alpha-tocopherol 400 IU once daily Group3: symbiotic 1 g, alpha-tocopherol 400 IU once daily Group4: placebo 400 IU once daily
Polyzos et al. 2017 [16]	RCT	13 months	Group1 (spironolactone+vitamin E): 14 Group2 (Vitamin E): 17	-	Group1: Females: 85.7% Males: 14.3% Group2: Females: 64.7% Males: 35.3%	Group1: spironolactone 25 mg once daily, vitamin E 400 IU/day Group2: Vitamin E 400 IU/day
Abedi et al. 2018 [17]	single-blind RCT	2 months	Group1 (Vitamin E+soft gel CLA): 19 Group2 (vitamin E): 19	Group1: 36.7 Group2: 38.6	Group1: Females: 89.5% Males: 10.5% Group2: Females: 84.2% Males: 15.8%	Group1: Vitamin E 400, soft gel CLA 3000 mg/day Group2: vitamin E 400 mg/day
Afzali et al. 2020 [11]	double-blind RCT	6 months	Group1 (Beta vulgaris): 60 Group2 (Placebo): 57	Group1: 47.5 Group2: 46.4	Group1: Females: 45% Males: 55% Group2: Females: 49.1% Males: 50.9%	Group1: vitamin E pearl (300 IU/twice daily), Livergol tablet (140 mg/daily), Beta vulgaris capsule (400 mg/daily) Group2: vitamin E pearl (300 IU/twice daily), Livergol tablet (140 mg/daily), instead of Beta vulgaris capsule (400 mg/daily)
Chandan et al 2021 [30]	RCT	12 months	Group1 (Pentoxyphylline+Vitamin E): 36 Group2 (Vitamin E): 33	Group1: 40 Group2: 35	Group1: Males/Females 33/3 Group2: Males/Females 28/5	Group1: Pentoxyphylline (400 mg/ thrice daily), vitamin E (400 IU/ twice daily) Group2: vitamin E (400 IU/ twice daily)
Bilal et al 2024 [31]	RCT	6 months	Group1 (Saroglitazar): 44 Group2 (Vitamin E): 41 Group3 (Saroglitazar +Vitamin E): 47 Group4 (control arm): 43	45	Group1: Females: 19 Males: 25 Group2: Females: 17 Males: 24 Group3: Females: 14	Group1: Saroglitazar (4 mg/ daily alone) Group2: vitamin E (800 IU/ daily alone) Group3: Saroglitazar (4 mg/ daily alone), vitamin E (800

Authors and year	Type of study	Duration of study	Sample size	Mean age	Gender	Interventions used
					Males: 33 Group4: Females: 13 Males: 30	IU/ daily alone) Group4: control arm
Khaliq [32]	double-blind RCT	6 months	Group1 (vitamin E): 42 Group2 (pioglitazone): 43 Group3 (ertugliflozin): 44 Group4 (vitamin E + ertugliflozin): 44	30-65	Group1: Females: 11 Males: 31 Group2: Females: 10 Males: 33 Group3: Females: 9 Males: 35 Group4: Females: 10 Males: 34	Group1: vitamin E (800 IU/ once daily) Group2: pioglitazone (30 mg/ once daily) Group3: ertugliflozin (15 mg/ once daily) Group4: ertugliflozin (15 mg/ once daily) vitamin E (800 IU/ once daily)
Yu [33]	double-blind RCT	24 months	Group1 (Vitamin E): 58 Group2 (placebo): 66	Group1: 37.9 Group2: 39.0	Group1: Females: 16 Males: 42 Group2: Females: 16 Males: 50	Group1: vitamin E (300 mg / daily) Group2: placebo (300 mg / daily)

The study by Akhondi-Meybodi et al. did not clearly define the randomization strategy and combine vitamin E with silymarin for the therapy of NAFLD [29]. One double-blinded RCT compared a combination therapy of symbiotic, alpha-tocopherol, symbiotic and alpha-tocopherol, and placebo [19]; the other double-blinded study compared Beta vulgaris therapy and placebo [11]. One RCT compared vitamin E monotherapy and vitamin E combination therapy with low-dose spironolactone [16]. One single-blinded RCT compared a combination therapy of vitamin E and soft gel CLA [17]. One RCT compared vitamin E monotherapy and vitamin E combination therapy with Pentoxyphylline [30]. Another RCT compared control and saroglitazar, vitamin E, as well as combination therapy [31]. One double-blinded RCT compared vitamin E and pioglitazone, ertugliflozin, as well as a combination therapy of vitamin E and ertugliflozin [32]. Another double-blinded RCT compared vitamin E and placebo [33]. All the RCTs used varying doses of vitamin E, ranging from 300 IU to 800 IU [11, 16, 17, 29-33]. Silymarin, symbiotic, alpha-tocopherol, symbiotic and alpha-tocopherol, spironolactone, soft gel CLA, Beta vulgaris, pentoxyphylline, saroglitazar, pioglitazone and ertugliflozin were the common co-intervention used in the nine RCTs [11, 16, 17, 19, 29-33].

Eight of the nine RCTs noted significant improvements in liver function tests [11, 17, 19, 29-33]. Six studies noted a significant improvement in both ALT and AST [11, 19, 29-33]; two noted improvement in ALP [19, 32], and one noted improvement in GGT only [32]. Abedi et al. reported additional improvements in ALT/AST ratio [17]. Polyzos et al. did not report any improvements in liver function tests with a combination therapy of vitamin E and spironolactone [16] (Table 2).

When liver histological parameters were evaluated, the treatment groups showed only limited improvements. Polyzos et al., Bilal et al., Khaliq et al. and Yu et al. reported improvement in steatosis [16, 31-33], and Abedi et al., Chandan et all. and Yu et al. reported an improvement in inflammation [17, 30, 33]. Chandan et all. and Yu et al. showed improvement in NAFLD Activity Score NAS [30, 33], Polyzos et al., Bilal et al. and Khaliq et al. reported improvement in fibrosis score [19]. Polyzos et al. and Yu et al showed no improvement in ballooning [16, 33].

However, it must be noted that several markers of hepatic histological response (such as NAS, steatosis, fibrosis, ballooning, and inflammation) were not recorded in several studies [11, 16, 17, 19, 29-32].

Table 2. *Summarizes the effects of vitamin E on biochemical and histological markers of NAFLD.*

Author and year	Improvement in liver function tests (ALT, AST, ALP, GGT)	Improvement in NAFLD Activity Score (NAS)	Improvement in steatosis	Improvement in fibrosis score	Improvement in ballooning	Improvement in inflammation
Akhondi-Meybodi	significant decrease in	No	No	No	Not recorded	Not recorded

Author and year	Improvement in liver function tests (ALT, AST, ALP, GGT)	Improvement in NAFLD Activity Score (NAS)	Improvement in steatosis	Improvement in fibrosis score	Improvement in ballooning	Improvement in inflammation
et al. 2020 [29]	ALT and AST in the treatment group					
Ekhlasi et al. 2017 [19]	significant decrease in AST, ALT and ALP level	Not recorded	No	No	Not recorded	No
Polyzos et al. 2017 [16]	Not recorded	Not recorded	Yes	Not recorded	No	Not recorded
Abedi et al. 2018 [17]	significant decrease in ALT/AST ratio in the treatment group	Not recorded	No	Not recorded	Not recorded	Yes
Afzali et al. 2020 [11]	significant decrease in AST and ALT in the treatment group	Not recorded	Not recorded	No	Not recorded	Not recorded
Chandan et al 2021 [30]	significant decrease in ALT level	Yes	Not recorded	Yes	Not recorded	Yes
Bilal et al 2024 [31]	significant decrease in AST and ALT level	No	Yes	Yes	Not recorded	Not recorded
Khaliq et al 2025 [32]	significant decrease in AST, ALT, ALP and GGT level	Not recorded	Yes	Yes	Not recorded	Not recorded
Yu et al 2025 [33]	significant decrease in AST and ALT	Yes	Yes	No	No	Yes
No. of results showing improvement	9	2	4	3	0	3
No. of results showing no improvement	0	2	3	4	2	1
No. of results not recorded	1	5	2	2	7	5

GGT: Gamma-Glutamyl Transpeptidase

4. Discussion

NAFLD is closely associated with metabolic syndrome [34]. The relation of NAFLD with insulin resistance, obesity, type II diabetes mellitus and hyperlipidemia is well confirmed. The progression of the disease is attributed to ROS, mitochondrial dysfunction, endotoxin-induced cytokine release, oxidative stress and metabolic changes [35]. According to the progression of NAFLD, pharmacological therapies should be considered to avoid more serious consequences. However, some medicines such as Silymarin, symbiotic, alpha-tocopherol and spironolactone are not widely recommended due to its own side effects and unapproved validity in NAFLD. Interestingly, vitamin E, an anti-oxidant, reduces reactive oxygen species, improves the antioxidants and decreases the hepatic steatosis, fibrosis, ballooning and inflammation, and has been investigated to treat NAFLD [18]. Therefore, vitamin E is broadly recommended for the therapy of NAFLD by the different scientific societies from Europe, America, and Asia-Pacific regions [36].

Research by Afzali et al. reported that Beta vulgaris has positive effects on the biochemical parameters of patients with NAFLD, and lowers ALT and AST levels. Moreover, Beta vulgaris alongside NAFLD therapy such as vitamin E and Silybum marianum has satisfactory clinical results due to its high availability and low cost [11]. Akhondi et al. noted that combination therapy with vitamin E and silymarin can improve the conditions of patients with NAFLD. Silymarin acted as a natural ingredient of milk thistle and an antioxidant which may reduce liver damage by decreasing ALT and AST levels [29]. In the Polyzos et al. study, despite biochemical

markers (ALT and AST levels) in NAFLD did not change, insulin resistance and steatosis were significantly decreased in the combination group of low-dose spironolactone plus vitamin E [16]. Study by Abedi et al. showed that CLA, a mixture of unsaturated isomers of octadecadienoic acid, has an anti-inflammatory effects and supplementation of CLA lead to the decrease of inflammatory level and (ALT/AST) ratio [17]. In addition to CLA, vitamin E has also a strong anti-oxidative property and could positively impact ALT, AST and inflammation in patients with NAFLD [37]. A crucial finding in another study suggested that ALT, AST and ALP levels were significantly lower in patients with NAFLD in the symbiotic alongside alpha-tocopherol intervention groups [19]. Study by Chandan et al. [30] indicated a significant decrease in ALT levels, which further supported the potential of the intervention (e.g., vitamin E supplementation) in improving hepatocellular injury, as ALT, a sensitive marker of liver damage, reflected the alleviation of hepatic inflammation and structural impairment. The significant decreases in AST and ALT levels were observed in the study of Bilal et al [31] and Yu et al [33], collectively supporting that the intervention (e.g., vitamin E supplementation) may exert a protective effect on hepatocellular integrity, as these enzymes were reliable indicators of liver cell damage, and their reduction reflects mitigated hepatic injury and improved liver function. The significant reduction in AST, ALT, ALP, and GGT levels reported by Khaliq et al [32] underscored a comprehensive improvement in liver function, as these enzymes collectively reflected hepatocellular integrity (AST, ALT), bile duct function (ALP), and hepatobiliary detoxification capacity (GGT), thereby indicating that the intervention in question may exert multi-faceted protective effects on hepatic structure and physiological processes.

The studies above indicated that the potential of vitamin E in the NAFLD therapy significantly improves liver function (ALT and AST levels) and histological abnormalities such as steatosis and inflammation in NAFLD. The consequences of our study are in agreement with those of Loguercio et al.[38] and Pietu et al.[39], in which the combined other drugs plus vitamin E reduced biochemical markers in patients with NAFLD, especially ALT and AST levels. However, the impact of vitamin E on biochemical and hepatic histological parameters in NAFLD patients have been represented discrepantly in the relevant research. The present study demonstrated that vitamin E is able to improve liver steatosis and inflammation, yet hepatic fibrosis and ballooning are not significant change. One research by Derakhshandeh-Rishehri et al.[1] reported Realsil intake decreased circulating GGT level and was not associated with the improvement of AST and ALT levels. In that study, mean scores for liver histological markers such as steatosis, inflammation, and ballooning were reduced with the combined vitamin E and pioglitazone [40]. However, a meta-analysis showed all hepatic histological indicators, including liver steatosis, fibrosis, ballooning and inflammation, were significantly improved in patients with NASH [41]. In addition, Fukui et al. [42] reported a significant improvement in liver function such as ALT, AST and GGT with the use of vitamin E. Even so, the evidence indicated that high-dose vitamin E could increase the risk of mortality [43], though the relation was not well confirmed [44, 45]. Also, the present study included only three blinded and two open-label trials in the analysis with different baseline morbidities and three of the five were followed for less than six months [46]. Thus, further investigations are necessary for the safety profile of vitamin E in the clinical practice, especially on long-term follow up.

Limitations

Our systematic review has some limitations; duration of intervention was relatively short in the RCTs. The sample sizes in the current RCTs were relatively small. Moreover, the RCTs did not investigate one or more crucial factors useful for monitoring disease progression in NAFLD. The longer duration of intervention and larger samples are necessary to verify the safety of daily vitamin E alone or combined with other anti-oxidants in future research.

5. Conclusions

Vitamin E is a potential antioxidant useful for the treatment of NAFLD. Our systematic review suggests that vitamin E therapy provides several biochemical and histological improvements in NAFLD. However, the association of vitamin E therapy with liver disease pathology such as NAFLD Activity Score, fibrosis and ballooning are not significantly observed. Additionally, the conclusion on the safety and efficacy of proposed treatments is limited due to the short duration of trials. Thus, vitamin E should be more widely used as a potent antioxidant or an adjuvant due to its potential benefit for the therapy of NAFLD in clinical practice. Future research could explore the underlying mechanisms by which vitamin E exerts its biochemical and histological benefits in NAFLD, as well as investigate whether modifying factors such as dosage regimens, patient genetic backgrounds, or concurrent metabolic conditions might enhance its effects on key pathological features like NAFLD Activity Score, fibrosis, and ballooning, while also extending trial durations to better assess long-term safety and efficacy.

Abbreviations

NAFLD	Non-alcoholic Fatty Liver Disease
NAFL	Non-Alcoholic Fatty Liver
NASH	Non-alcoholic Steatohepatitis
HCC	Hepatocellular Carcinoma
RCT	Randomized Controlled Trials
AST	Aspartate Aminotransferase
ALT	Alanine Aminotransferase
ALP	Alkaline Phosphatase
GGT	Gamma Glutamyl Transferase

NAS	NAFLD Activity Score
IR	Insulin Resistance
ROS	Reactive Oxygen Species
T2DM	Type 2 Diabetes Mellitus
CLA	Conjugated Linoleic Acid
LDL	Low-density Lipoprotein

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Author Contribution

Can Liu: Conceptualization, Formal analysis, Investigation, Methodology, Validation, Writing – original draft

Zeming Bai: Data curation, Software

Jingmin Cheng: Conceptualization, Supervision, Writing – review & editing

Conflicts of Interest

The authors declare that there is no conflict of interest.

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