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REVIEW ARTICLE

Alcohol and metabolic dysfunction interaction: MetALD

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Abstract

In 2023, the concept of metabolic dysfunction-associated steatotic liver disease (MASLD) combined with metabolic dysfunction and alcohol-related liver disease (MetALD) emerged for patients who meet MASLD criteria and have risky alcohol consumption (20-50 g/day in women and 30-60 g/day in men). Although conceptually useful, the new nomenclature faces controversies: alcohol thresholds are variable and poorly supported, the boundary between metabolic and alcohol-related etiology is blurred, and prospective studies defining the specific risks of MetALD are lacking. The prevalence of MetALD is highly variable. Risk is greater in men with visceral obesity and type 2 diabetes, and it is associated with more advanced fibrosis and a higher incidence of hepatocellular carcinoma. In the pathophysiology of MetALD, alcohol metabolism-related injury converges with insulin resistance, lipogenesis, oxidative stress, mitochondrial dysfunction, activation of inflammatory pathways, and hepatic stellate cell activation driving fibrogenesis. Diagnostic evaluation should be comprehensive, documenting the presence of steatosis, confirming MASLD criteria, demonstrating evidence of risky alcohol consumption, and stratifying fibrosis. Treatment focuses on alcohol abstinence, strict control of metabolic risk factors, and surveillance for complications.

Keywords: MetALD. Risky alcohol consumption. MASLD. Nomenclature.

Interacción del alcohol y la disfunción metabólica: MetALD

Resumen

En 2023 surgió el concepto de esteatosis hepática metabólica (MASLD, Metabolic dysfunction Associated Steatotic Liver Disease) aunada a enfermedad hepática relacionada con el alcohol (MetALD, Metabolic dysfunction and Alcohol-related Liver Disease) para pacientes con criterios de MASLD y consumo riesgoso de alcohol (20-50 g/día en las mujeres y 30-60 g/día en los hombres). Aunque útil conceptualmente, la nueva nomenclatura enfrenta controversias: los umbrales de alcohol son variables y poco sustentados, la frontera entre etiología metabólica y relacionada con el alcohol es difusa, y faltan estudios prospectivos que delimiten los riesgos específicos de MetALD. La prevalencia de MetALD es extremadamente variable. El riesgo es mayor en varones con obesidad visceral y diabetes tipo 2, y se asocia a más fibrosis avanzada y a mayor incidencia de carcinoma hepatocelular. En la fisiopatología de la MetALD converge el daño propio derivado del metabolismo del alcohol con Resistencia a la insulina, lipogénesis, estrés oxidativo, disfunción mitocondrial, activación de vías de inflamación y activación de células estrelladas que detonan la fibrogénesis. La evaluación para establecer el diagnóstico debe ser integral, evidenciando la presencia de esteatosis y de criterios de MASLD, así como consumo riesgoso de alcohol, y estratificando la fibrosis. El tratamiento se centra en la abstinencia del alcohol, el control estricto de los factores metabólicos y la vigilancia de las complicaciones.

Palabras clave: MetALD. Consumo riesgoso de alcohol. MASLD. Nomenclatura.

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Introduction

In 2023, non-alcoholic fatty liver disease (NAFLD) was redefined as metabolic dysfunction-associated steatotic liver disease (MASLD), and likewise non-alcoholic steatohepatitis (NASH) was redefined as metabolic dysfunction-associated steatohepatitis (MASH). Within this new classification, a new category was proposed, currently termed MetALD (metabolic dysfunction and alcohol-related liver disease), which encompasses patients who meet criteria for MASLD and who also have risky alcohol consumption[1], defined as 140-350 g/week (20-50 g/day) in women and 210-420 g/week (30-60 g/day) in men[2]. The proposal, according to this new nomenclature, for establishing MetALD criteria is shown in figure 1.

Controversies regarding the new nomenclature and definition of MetALD

There is ambiguity in alcohol consumption thresholds, as the limits currently proposed to define risky alcohol consumption lack scientific robustness, have been mostly defined on an empirical basis, and vary across different publications[3]. On the other hand, the term MetALD represents a conceptually imprecise overlap between MASLD and alcohol-associated liver disease (ALD). The diagnostic distinction between metabolic or alcoholic predominance is not always clear and may depend excessively on subjective clinical judgment[4]. Furthermore, although it is recognized that alcohol consumption potentiates the progression of fibrosis, cirrhosis, and hepatocellular carcinoma, robust prospective studies are still lacking that clearly differentiate the risks in individuals with MetALD compared to MASLD or ALD alone[5].

In the research field, another major challenge is that this new classification requires reevaluating historical databases and ongoing clinical trials, which may complicate longitudinal comparisons and question the validity of previous results[6].

Epidemiology of MetALD

There is significant underreporting regarding alcohol consumption, since generally, to assess it, self-reported questionnaires are applied, which underestimate actual intake by 20-40%, potentially undervaluing the true burden of MetALD[7].

The prevalence of MetALD is higher in Hispanic populations and Eastern Europe, while it is lower in Asia, but with an increasing trend among young people in urban centers[8].

Studies in the general population conducted in the United States and Europe suggest that MetALD affects approximately 3-5% of the adult population[9,10]. In Asia, figures are reported between 1% and 3%, probably related to cultural differences in alcohol consumption[11]. In Latin America, where high obesity prevalence and high alcohol consumption coexist, prevalences range between 8 and 10%[12]. However, the prevalence of MetALD increases when evaluated in patients with a pre-established diagnosis of MASLD, in whom it has been reported that between 15% and 30% meet MetALD criteria[13].

The risk of having MetALD is particularly high in middle-aged men with visceral obesity and type 2 diabetes mellitus[14]. It is also known that patients with MetALD have a higher risk of advanced fibrosis compared to those who only meet MASLD criteria[15]. Finally, studies have shown that the risk of hepatocellular carcinoma is substantially higher (incidence rate of 1.847 per 1000 person-years) in patients with MetALD and fibrosis-4 index (FIB-4) ≥ 1.3, compared to other etiologies or MASLD alone[16].

Pathogenesis of ALD and MASLD: differences and similarities

Although ALD and MASLD differ in their primary triggering factors (excessive alcohol consumption in ALD and insulin resistance/obesity in MASLD), they share multiple common pathophysiological pathways that explain why there is an overlapping clinical spectrum and even the mixed condition MetALD[4]. In MetALD, on the one hand, chronic and excessive ethanol consumption converges, causing direct toxicity in hepatocytes, generating alterations in lipid metabolism, and promoting inflammation[17], and on the other hand, insulin resistance, visceral obesity, and metabolic syndrome, which induces a state of hepatic lipotoxicity[18] (Table 1).

Alterations in lipid metabolism

In both ALD and MASLD, intrahepatic accumulation of triglycerides (steatosis) occurs, and there is an increase in *de novo* lipogenesis and an alteration in mitochondrial β-oxidation. However, in ALD, ethanol oxidation generates an excess of reduced nicotinamide adenine dinucleotide (NADH), which in turn favors lipogenesis and blocks fatty acid oxidation, while in MASLD, hyperinsulinemia stimulates key transcription factors

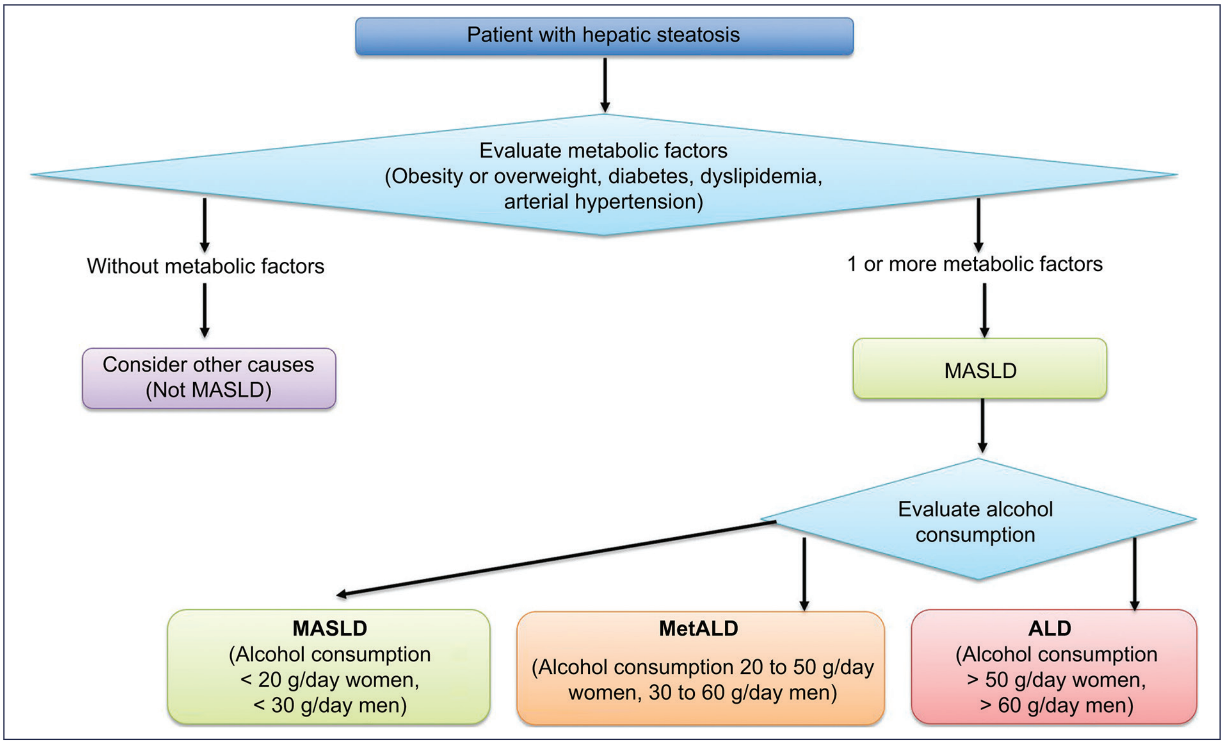

Figure 1. Criteria for considering MetALD within the new nomenclature for hepatic steatosis

Table 1. Comparison of the pathogenesis of ALD and MASLD

Mechanism	ALD	MASLD	Key convergence points
Primary triggering factor	Chronic and excessive alcohol consumption	Insulin resistance, visceral obesity, metabolic syndrome	Both require a sustained external stimulus that alters hepatic homeostasis
Lipid metabolism	Acetaldehyde and excess NADH inhibit β-oxidation and promote lipogenesis	Hyperinsulinemia stimulates SREBP-1c and ChREBP → increases fatty acid synthesis	Accumulation of triglycerides (hepatic steatosis)
Oxidative stress	Ethanol metabolism via CYP2E1 → ROS and acetaldehyde adducts	Lipotoxicity from free fatty acids → lipid peroxidation	Oxidative stress and mitochondrial damage as central axes
Inflammation and innate immunity	Intestinal dysbiosis, increased permeability → LPS activates TLR4 in Kupffer cells → TNF-α, IL-1β	Dysfunctional adipose tissue releases TNF-α, IL-6; decreased adiponectin	Activation of Kupffer cells and chronic inflammatory response
Fibrogenesis	Acetaldehyde and ROS activate hepatic stellate cells	Chronic metabolic signals (lipotoxicity, insulin resistance) activate stellate cells	Progressive fibrosis mediated by extracellular matrix deposition
Carcinogenesis	Direct mutagenesis by acetaldehyde, ROS, and epigenetic alterations	Chronic inflammation, obesity, hyperinsulinemia → activation of IGF/PI3K-AKT pathways	HCC can appear even in the absence of cirrhosis
Clinical spectrum	Steatosis → alcohol-related hepatitis → fibrosis → cirrhosis → HCC	Steatosis → MASH → fibrosis → cirrhosis → HCC	Similar progressive spectrum course

HCC: hepatocellular carcinoma; ChREBP: carbohydrate response element-binding protein; CYP2E1: cytochrome P2E1; IGF/PI3K-AKT: insulin-like growth factor/phosphoinositide 3-kinase – protein kinase B; IL-1β: interleukin 1 beta; IL-6: interleukin 6; LPS: lipopolysaccharide; MASH: metabolic dysfunction-associated steatohepatitis; NADH: reduced nicotinamide adenine dinucleotide; ROS: reactive oxygen species; SREBP-1c: sterol regulatory element-binding protein 1c; TNF-α: tumor necrosis factor alpha; TLR4: toll-like receptor 4.

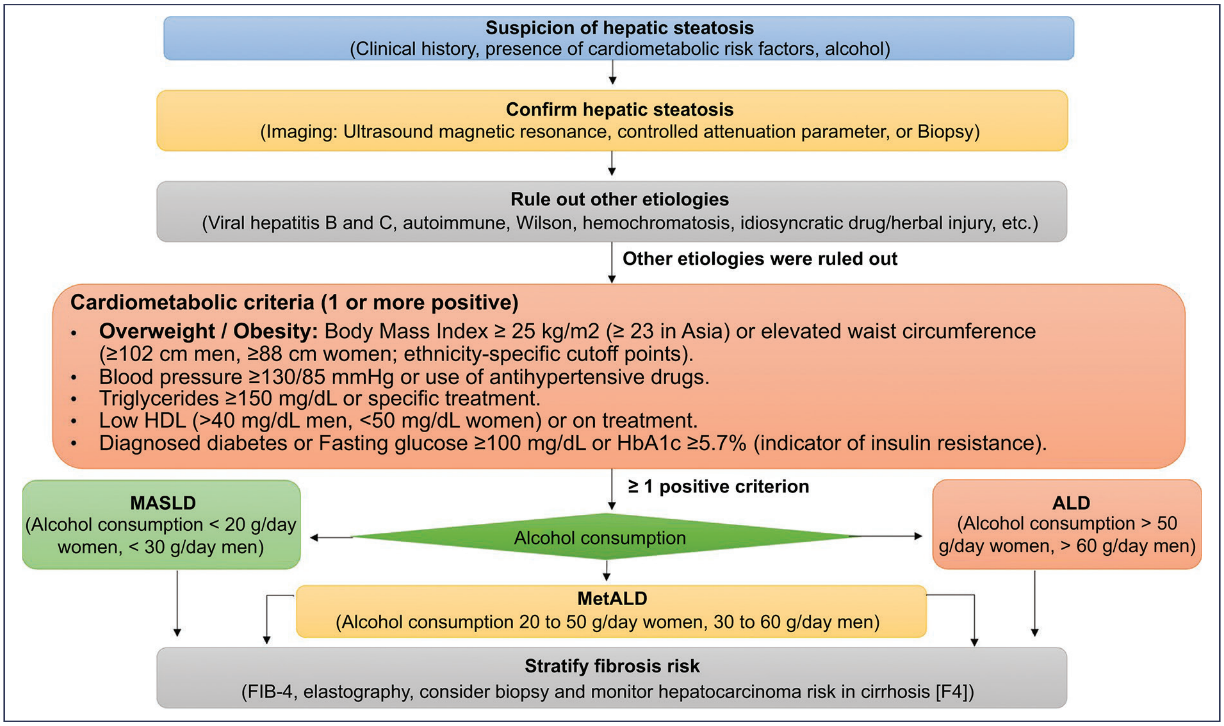

Figure 2. Diagnostic algorithm for MetALD.

that regulate lipogenesis; among the most relevant are sterol regulatory element-binding protein 1c (SREBP-1c) and carbohydrate response element-binding protein (ChREBP), inducing greater fatty acid synthesis[17-20].

Oxidative stress and mitochondrial damage

In ALD, ethanol metabolism via cytochrome P2E1 (CYP2E1) produces reactive oxygen species (ROS) and acetaldehyde, which damage proteins and deoxyribonucleic acid[17]. In MASLD, excess free fatty acids induce lipotoxicity, mitochondrial dysfunction, and lipid peroxidation[20,21]. Thus, in MetALD, oxidative stress and mitochondrial damage are mechanisms that trigger inflammation and progression to fibrosis[17,20-22].

Inflammation and innate immune system

Ethanol increases intestinal permeability, favoring bacterial translocation and activation of toll-like receptor 4 (TLR4) by lipopolysaccharide (LPS), which is capable of triggering an inflammatory response (tumor necrosis factor alpha [TNF-α], interleukin 1β [IL-1β], and interleukin 6 [IL-6)[17,19,22]. On the other hand, in MASLD, insulin resistance and lipid peroxidation favor a proinflammatory profile (TNF-α, IL-6, leptin) and reduction of adiponectin[20,21]. In both conditions, there is activation of Kupffer cells and recruitment of immune cells, which amplifies hepatic inflammation[19,22].

Fibrogenesis

In both ALD and MASLD, chronic inflammation activates hepatic stellate cells, promoting extracellular matrix deposition and favoring fibrosis progression[17,19,21-23]. In ALD, acetaldehyde and ROS act directly on stellate cells[22,23], while in MASLD, fibrosis is more related to chronic metabolic signals (lipotoxicity, insulin resistance) that activate stellate cells[21].

Carcinogenesis

In ALD, there is direct mutagenesis by acetaldehyde and oxidative stress, in addition to epigenetic effects induced by alcohol[17,19,21]. In MASLD, carcinogenesis is associated with obesity, diabetes, and chronic inflammation, even in the absence of cirrhosis. In both diseases, inflammation, mitochondrial dysfunction, and altered insulin or insulin-like growth factor signaling contribute to the development of hepatocellular carcinoma[16,17,21,22].

Table 2. Therapeutic strategies in MetALD[1-4]

Strategy	Specific intervention	Evidence (key)	Expected hepatic effect	Level of recommendation*	Safety considerations
Alcohol	Total abstinence or intensive reduction; structured counseling; management of alcohol use disorder	Meta-analyses and cohorts: reduce decompensation and mortality	Reduce inflammation, fibrosis progression, and HCC risk	A	Assess withdrawal syndrome; refer to addiction services; objective tests (PEth/EtG) when possible
Drugs for alcohol	Naltrexone, acamprosate, baclofen (cirrhosis)	RCTs and series: increase abstinence; baclofen is useful in liver disease	Reduce relapses → indirect hepatic benefit	B	Naltrexone: avoid in acute hepatitis; baclofen: sedation; acamprosate: adjust in kidney impairment
Weight loss	Hypocaloric diet 500-1000 kcal/d; target 7-10%	RCTs and observational studies in MASLD: improve MASH and fibrosis	Reduce steatosis, NASH/MASH, and possibly fibrosis	A	Individualize if sarcopenia; avoid rapid losses in decompensated cirrhosis
Exercise	Aerobic + resistance ≥ 150 min/week	RCTs: reduce hepatic fat independent of weight	Reduces steatosis and increases insulin sensitivity	A	Adapt in cirrhosis; prevent sarcopenia
Dietary pattern	Mediterranean diet; less fructose and trans/saturated fats	RCTs and cohort studies: reduce steatosis and insulin resistance	Reduce lipotoxicity and inflammation	B	Sustained adherence; clinical nutrition support
T2DM: GLP-1R agonists	Semaglutide/ liraglutide	RCTs: MASH resolution; weight loss	Reduces steatohepatitis; possible reduction in fibrosis (under study)	A/B	GI, cholelithiasis; monitor in previous pancreatitis
T2DM: SGLT2 inhibitors	Empagliflozin/ dapagliflozin	RCTs: reduce hepatic fat by imaging	Reduce steatosis and inflammation	B	Fungal UTIs; euglycemic ketoacidosis (rare)
T2DM: metformin	Metabolic first line	Cohort studies: CV benefit; no clear effect on NASH	Systemic metabolic benefit	C	GI; avoid in advanced kidney impairment with acidosis risk
Dyslipidemia	Statins	Trials and cohort studies: safe in compensated liver disease; reduce CV events; signal of HCC reduction	CV benefit; possible HCC reduction	A	Monitor transaminases; safe in compensated cirrhosis
Arterial hypertension	Individualized intensive control	Cohort studies: reduce CV events	Indirect on prognosis	B	ACE-I/ARB with caution in advanced ascites
Procedures	Bariatric surgery (selected)	Cohort studies and RCTs: MASH resolution and fibrosis reduction	Reduce steatohepatitis and fibrosis	B	Rigorous selection; perioperative risk in cirrhosis
Investigational agents	PPAR pan-agonist (lanifibranor), FXR (cilofexor/ obeticholic), ACC/ FASN inhibitors	Phase II–III studies: histological and enzymatic improvement	Reduce inflammatory activity and lipogenesis; potential antifibrotic effect	B (under investigation)	Access in trials; AE pruritus (FXR), lipid profile (ACC)

(Continues)

Table 2. Therapeutic strategies in MetALD[1-4] *(continued)*

Strategy	Specific intervention	Evidence (key)	Expected hepatic effect	Level of recommendation*	Safety considerations
Surveillance and complications	Elastography/FIB-4; US/AFP every 6 months in advanced fibrosis	Guidelines	Reduces mortality through early HCC diagnosis	A	Ensure adherence; referral pathway to liver transplant program
Liver transplantation	Decompensated cirrhosis or incurable HCC	Standard	Long-term survival	A	Selection criteria; addiction support

*Guidance recommendation level (A: strong/high quality; B: moderate quality; C: limited quality/consensus).
ACC: acetyl coenzyme A carboxylase; AFP: alpha-fetoprotein; ARB: angiotensin II receptor antagonists; HCC: hepatocellular carcinoma; CV: cardiovascular; T2DM: type 2 diabetes mellitus; AE: adverse events; RCT: randomized controlled trials; FASN: fatty acid synthase; FIB-4: fibrosis-4 index; FXR: farnesoid X receptor; GI: gastrointestinal; GLP-1R: glucagon-like peptide 1 receptor; ACE-I: angiotensin-converting enzyme inhibitors; kidney impairment: renal insufficiency; UTI: urinary tract infections; PEth/EtG: phosphatidylethanol/ethyl glucuronide; PPAR: peroxisome proliferator-activated receptor; SGLT2: sodium-glucose cotransporter 2; liver transplant program: liver transplant program; US: ultrasound.

Evaluation of MetALD

The diagnosis of MetALD requires the integration of four fundamental aspects: 1) evidence of hepatic steatosis, 2) meeting MASLD criteria, 3) demonstrating risky alcohol consumption, and 4) stratifying fibrosis risk[1,3,4] (Fig. 2).

Therapeutic strategies

The treatment of MetALD is based on alcohol abstinence, strict control of metabolic factors, and surveillance for complications, complemented with therapies under investigation. There is not yet a specific approved drug, but the combination of behavioral strategies, pharmacological treatments for comorbidity, and hepatic surveillance constitutes the current therapeutic basis[4] (Table 2).

Conclusions

- The introduction of the MetALD concept is conceptually valuable but faces significant challenges in its clinical and epidemiological application.
- Prospective studies are needed to validate its prognostic utility and define clinically relevant thresholds with evidence.
- It is important to avoid stigmatizing patients; the ideal approach should integrate multiple risk factors and emphasize patient-centered interventions.
- Until more evidence is available, it would be prudent to consider alcohol as a modulator of continuous risk within MASLD, rather than delimiting rigid categories.
- MetALD already affects millions of individuals worldwide, with prevalences of 3-10% in the general population and up to 30% in patients with metabolic risk.
- Current data are from retrospective studies and self-reports, which limits the precision of estimates.
- There is an urgent need for standardized definitions to estimate the global burden and for objective biomarkers of alcohol consumption and metabolic susceptibility, and prospective multinational studies are required to determine the true burden of advanced fibrosis and hepatocarcinoma attributable to MetALD.
- MetALD should be understood as an emerging disease with significant epidemiological impact, whose early recognition will allow the design of more effective preventive strategies.
- ALD and MASLD share key pathogenic mechanisms (steatosis, oxidative stress, inflammation, fibrogenesis), but differ in the initial stimulus (alcohol or metabolic dysfunction) and in some predominant mediators (acetaldehyde and dysbiosis in ALD, and insulin resistance and adipokines in MASLD). This pathophysiological convergence explains the emergence of the MetALD concept, which recognizes the synergistic interaction between alcohol and metabolic dysfunction in the development and progression of liver damage.
- Current therapy for MetALD is based on strategies focused on maintaining alcohol abstinence and emphasizing metabolic control.

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Conflicts of interest

F. Higuera-de la Tijera is a speaker for Sanfer, Grünenthal, Gilead, Abbott, Medix, and Adium. A. Servín-Higuera has no conflicts of interest.

Ethical considerations

Protection of human subjects and animals. The authors declare that no experiments were performed on human subjects or animals for this research.

Confidentiality, informed consent, and ethical approval. The study does not involve personal patient data nor requires ethical approval. SAGER guidelines do not apply.

Statement on the use of artificial intelligence. The authors declare that they used artificial intelligence for writing this manuscript, specifically ChatGPT and Gemini to create the algorithms, figures, and tables for this article.

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