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Clinical Utility of C-Reactive Protein-Triglyceride Glucose Index (CTI) and hs-CRP for Distinguishing Obese and Non-Obese Phenotypes of NAFLD: NHANES 2017-2020

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Abstract

Background: Non-alcoholic fatty liver disease (NAFLD) is commonly associated with obesity, but emerging evidence suggests significant heterogeneity between obese and non-obese phenotypes. We aimed to evaluate the discriminative ability of high-sensitivity C-reactive protein (hs-CRP) and the C-reactive protein-triglyceride glucose index (CTI) in identifying the obese NAFLD phenotype using data from NHANES 2017-2020. **Methods:** We analyzed 1171 adults with NAFLD from NHANES 2017-2020 (obese, n = 729; non-obese, n = 442). Obesity was defined as BMI ≥ 30 kg/m^2. Logistic regression models (models 1 - 4) assessed associations of hs-CRP and CTI with obesity, progressively adjusted for demographics and metabolic factors. Receiver operating characteristic (ROC) curves evaluated discrimination. **Results:** We enrolled 1171 participants diagnosed with non-alcoholic fatty liver disease (NAFLD) (53.7% male), of whom 62.3% were classified into the obese phenotype group. Univariate logistic regression revealed that both hs-CRP (OR = 1.11, 95% CI: 1.08 - 1.15, p < 0.001) and CTI (OR = 1.70, 95% CI: 1.47 - 1.98, p < 0.001) were significantly associated with the obese and non-obese NAFLD phenotypes. In the multivariate model incorporating both CTI and hs-CRP along with confounders, CTI demonstrated a strong and independent association with the obese phenotype (OR = 2.09, 95% CI: 1.68 - 2.62, p < 0.001). It is noteworthy that under these conditions, the association for hs-CRP was attenuated and

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became non-significant (OR = 1.01, 95% CI: 0.99 - 1.03, p = 0.411). This attenuation suggests that hs-CRP may not provide additional independent predictive value beyond what is already captured by the composite CTI index in this specific model. ROC analysis indicated that the discriminatory ability of hs-CRP alone was significantly superior to that of CTI, with an AUC value of 0.711 compared to 0.628 for CTI (ΔAUC = −0.082; DeLong's test, p < 0.001). Collinearity analysis (VIF = 1.2) indicated a low degree of collinearity between the two markers. The Brier score showed that the calibration accuracy of hs-CRP (0.217) was slightly better than that of CTI (0.224). **Conclusions:** Both CTI and hs-CRP demonstrated significant associations with the obese phenotype of NAFLD. However, after adjusting for multiple confounding factors, only CTI remained an independent risk factor for the obese phenotype, suggesting that, as a composite metric reflecting metabolic-inflammatory interactions, it more accurately captures the core pathophysiological mechanism underlying obese-phenotype NAFLD. Notably, hs-CRP exhibited superior discriminatory power (AUC = 0.711) and calibration accuracy, making it more suitable for use as a tool for rapid clinical screening. Although CTI showed relatively weaker standalone discriminatory ability (AUC = 0.628), its independent and robust association supports its unique value in comprehensive multifactorial risk assessment models. The low collinearity (VIF = 1.2) further indicates that these two markers provide complementary rather than redundant information for phenotypic differentiation. In summary, this study advocates for a hierarchical biomarker application strategy to distinguish NAFLD phenotypes: hs-CRP can be used for initial phenotypic screening, while CTI provides deeper mechanistic insights for precise risk stratification and individualized management of NAFLD. Future multicenter diagnostic studies are needed to validate their clinical utility and define optimal diagnostic cut-off values.

Keywords

NAFLD, Obesity, hs-CRP, C-Reactive Protein-Triglyceride Glucose Index, Phenotype, NHANES

1. Introduction

Non-alcoholic fatty liver disease (NAFLD) is one of the most common chronic liver conditions worldwide, affecting approximately 25% of the global adult population [1]. Characterized by the abnormal accumulation of lipids within hepatocytes, NAFLD is closely linked to metabolic dysfunction and often coexists with other metabolic disorders such as metabolic syndrome, obesity, type 2 diabetes mellitus, and insulin resistance [2] [3]. While NAFLD has traditionally been associated with obesity, recent studies have highlighted its occurrence in individuals with normal body weight or those who are overweight, giving rise to the clinical phenotypes "lean NAFLD" or "non-obese NAFLD" [4]. This growing recognition of non-obese NAFLD is significant, as it often presents with a distinct metabolic

phenotype despite the absence of obesity. Studies have shown that non-obese individuals with NAFLD experience impaired glucolipid homeostasis, contributing to a potentially more detrimental metabolic profile [5]-[8]. Although non-obese NAFLD patients generally exhibit a less severe metabolic phenotype than their obese counterparts, they face higher all-cause mortality rates (as high as 28% in a study of over 1000 patients), more rapid disease progression, and significantly greater risks for cardiovascular disease and malignancies [9]-[12]. Evidence further suggests that the relationship between NAFLD and metabolic complications is stronger in non-obese individuals than in obese patients, underscoring the need for more focused attention on this subtype [13].

Therefore, these findings highlight significant differences in inflammatory and metabolic profiles between obese and non-obese NAFLD patients, underscoring the clinical imperative for precise phenotypic stratification. Implementing such differentiation is crucial for developing tailored management strategies that address the distinct pathophysiological mechanisms underlying each subtype, ultimately advancing toward personalized therapeutic interventions. High-sensitivity CRP (hs-CRP) is one of the most convenient and widely used clinical biomarkers for assessing systemic inflammation [14]. hs-CRP detects low-grade inflammation and is associated with multiple adverse health outcomes, such as cardiovascular disease, metabolic syndrome, insulin resistance, and impaired physical function [15]-[17]. hs-CRP serves as a valid proxy for IL-6 and TNF-α activity, offering a feasible approach for evaluating inflammatory status in large-scale epidemiological studies [14]. Furthermore, the CTI is a composite metric that integrates the TyG index and hs-CRP to concurrently reflect both insulin resistance and the inflammatory pathway [18]. Previous studies have demonstrated the correlation between CTI and conditions such as coronary heart disease [19], depression [20], stroke [21], and NAFLD/liver fibrosis [22].

However, little is known about how inflammatory and metabolic composite biomarkers distinguish NAFLD phenotypes. This study, therefore, aimed to evaluate the discriminative and complementary roles of hs-CRP and CTI in differentiating obese from non-obese NAFLD using NHANES 2017-2020 data. The combined use of these biomarkers offers a more complete perspective on metabolic and inflammatory contributions to NAFLD, supporting the development of tailored treatment approaches. Our goal is to establish practical biomarkers that improve risk stratification and advance precision medicine in NAFLD management.

2. Methods

This study employed a cross-sectional design using NHANES data to evaluate the health and nutritional status of the U.S. population. NHANES adopts a multistage, stratified sampling strategy to ensure national representativeness. Data are collected biennially through questionnaires, physical examinations, and laboratory tests, covering disease status, nutritional indicators, lifestyle behaviors, and socioeconomic information. The study was approved by the NCHS Institutional Re-

view Board, and all participants provided informed consent. Data from the 2017-2020 NHANES cycles were utilized, with an initial cohort of 15,560 individuals [23]. NAFLD was diagnosed based on a controlled attenuation parameter (CAP) value ≥ 274 dB/m. Based on predefined inclusion and exclusion criteria, the following were excluded: 1) aged < 18 years; 2) lacking vibration-controlled transient elastography (VCTE) data or CAP < 274 dB/m [24]; 3) positive for hepatitis B surface antigen (HBsAg) or hepatitis C virus RNA (HCV RNA); 4) excessive alcohol consumption (>2 standard drinks/day for men, >1 standard drink/day for women); 5) missing any parameter required for calculating the C-reactive protein-triglyceride glucose index (CTI), including high-sensitivity C-reactive protein (hs-CRP), triglycerides, or fasting glucose.

2.1. Main Exposures and Outcome Variables

2.1.1. Exposures

hs-CRP (mg/L); The Triglyceride-Glucose Index (TyG) was utilized to evaluate insulin resistance and metabolic function. A novel Composite Triglyceride Index (CTI) was defined by integrating the TyG with high-sensitivity C-reactive protein (hs-CRP). C-reactive protein-triglyceride glucose index (CTI) = 0.412 × ln [hs-CRP (mg/L)] + ln{[triglycerides (mg/dL) × fasting glucose (mg/dL)]/2}. CTI was calculated only if hs-CRP, triglycerides, and fasting glucose were available and >0 [18].

2.1.2. Outcome

The obese phenotype is defined as BMI ≥30 kg/m^2.

2.1.3. Covariates

Age (years), gender (male/female), triglycerides (mg/dL), total cholesterol (mg/dL), HbA1c (%), uric acid (mg/dL), and albumin (g/dL). Covariates were selected a priori for clinical relevance to NAFLD and obesity.

2.2. Statistical Analysis

We summarized baseline characteristics overall and by obesity status (**Table 1**). Logistic regression models were prespecified as: Model 1 (hs-CRP), Model 2 (CTI), Model 3 (hs-CRP, CTI, age, gender), Model 4 (hs-CRP, CTI, age, gender, total cholesterol, HbA1c (%), uric acid, and albumin). Collinearity was assessed by VIF. Discrimination was evaluated by ROC/AUC and Youden-optimized cut-offs.

3. Results

This study conducted a retrospective analysis using data from two cycles (2017-2020) of the National Health and Nutrition Examination Survey (NHANES). The initial study population consisted of individuals aged 18 years and older (n = 9693). Based on exclusion criteria, we removed participants with missing vibration-controlled transient elastography (VCTE) data or CAP < 274 dB/m (n =

6115), those infected with hepatitis B or C (n = 40), excessive alcohol consumption (>2 standard drinks/day for men, >1 standard drink/day for women) (n = 1116), and those lacking CTI or hs-CRP data, individuals with severe infections (n = 1.251). The final analytical sample comprised 1171 participants. (Figure 1)

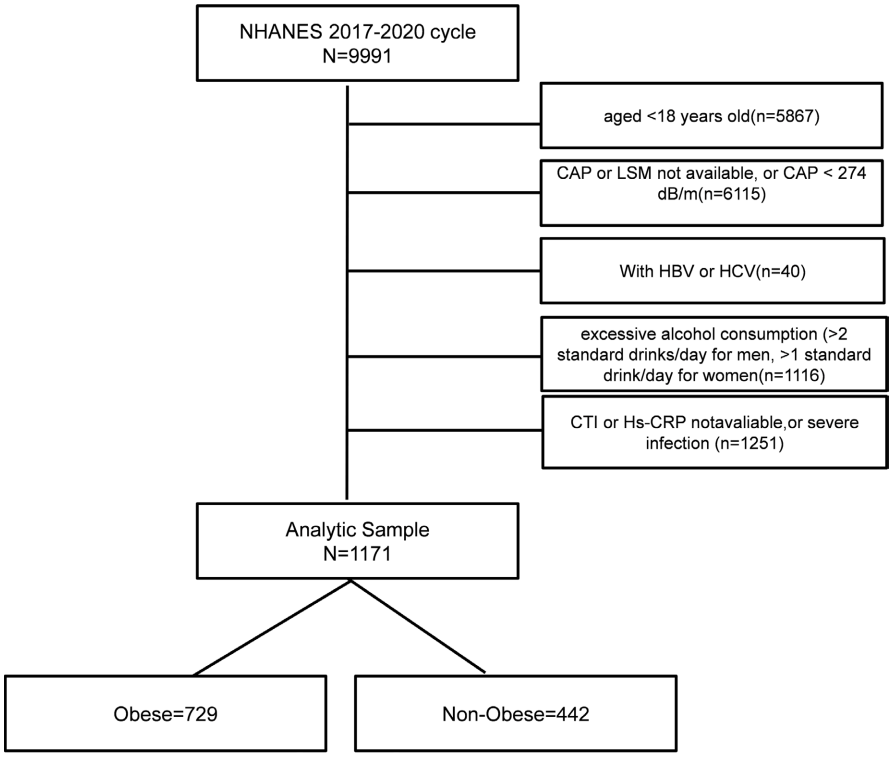

Figure 1. Flow diagram of study participants.

This study included a total of 1171 patients with non-alcoholic fatty liver disease (NAFLD); the mean age of the sample was 53 years (standard deviation (SD) = 19), with a slightly higher proportion of males compared to females (53.7% vs 46.3%). We identified that 729 (62.3%) participants had obesity. Compared with the non-obese group, participants in the obese group were younger (51 ± 19 vs. 55 ± 19 years, $p = 0.002$), had a lower proportion of males (51% vs. 60%, $p = 0.008$), and included more Black/African American individuals (25% vs. 13%, $p < 0.001$). BMI was higher in the obese group (37 kg/m^2 ± 7 kg/m^2) compared to the non-obese group (27 kg/m^2 ± 2 kg/m^2) ($p < 0.001$). In terms of laboratory parameters, the obese group showed higher levels of hs-CRP ((6.1 ± 9.5) mg/L vs. (3.0 ± 7.5) mg/L, $p < 0.001$) and ALT ((26 ± 18) U/L vs. (24 ± 15) U/L, $p = 0.017$), but lower albumin ((39.4 ± 3.0) g/L vs. (41.1 ± 2.9) g/L, $p < 0.001$) and HDL cholesterol ((1.20 ± 0.30) mmol/L vs. (1.30 ± 0.36) mmol/L, $p < 0.001$). No significant differences were detected between groups in fasting glucose, triglycerides, HbA1c, GGT, and AST levels. (Table 1)

Both hs-CRP (OR = 1.11, 95% CI: 1.08 - 1.15) and CTI (OR = 1.70, 95% CI: 1.47 - 1.98) were significantly associated with obesity in unadjusted models (both $p < 0.001$). After adjustment for hs-CRP, CTI, age, and sex, CTI remained strongly

predictive (OR = 1.77, 95% CI: 1.48 - 2.12), whereas the association for hs-CRP was modest (OR = 1.04, 95% CI: 1.01 - 1.07). In the fully adjusted model, CTI retained a robust association with obesity (OR = 2.09, 95% CI: 1.68 - 2.62, p < 0.001), while hs-CRP(OR = 1.01, 95% CI: 0.99 - 1.03, p = 0.411) was no longer significant. (Table 2)

Table 1. Summarizes the baseline characteristics of participants by obesity status.

Variable	Non-Obese N = 442	Obese N = 729	p-value
Age, mean ± SD	55 ± 19	51 ± 19	0.002
Sex, n (%)			0.008
Male	263 (60%)	374 (51%)	
Female	179 (40%)	355 (49%)	
Race, n (%)			<0.001
White	147 (33%)	272 (37%)	
Black/African American	58 (13%)	184 (25%)	
Other	237 (54%)	273 (37%)	
BMI (kg/m^2), mean ± SD	27 ± 2	37 ± 7	<0.001
hs-CRP (mg/L), mean ± SD	3.0 ± 7.5	6.1 ± 9.5	<0.001
Fasting glucose (mg/dL), mean ± SD	122 ± 45	125 ± 46	0.301
Triglycerides (mg/dL), mean ± SD	132 ± 104	140 ± 144	0.295
HbA1c (%), mean ± SD	6.13 ± 1.37	6.25 ± 1.33	0.139
ALT (U/L), mean ± SD	24 ± 15	26 ± 18	0.017
AST (U/L), mean ± SD	21 ± 8	22 ± 11	0.204
GGT (U/L), mean ± SD	31 ± 31	33 ± 31	0.121
Albumin (g/L), mean ± SD	41.1 ± 2.9	39.4 ± 3.0	<0.001
Uric acid (μmol/L), mean ± SD	332 ± 87	357 ± 87	<0.001
HDL cholesterol (mmol/L), mean ± SD	1.30 ± 0.36	1.20 ± 0.30	<0.001
LDL cholesterol (mmol/L), mean ± SD	2.96 ± 0.99	2.74 ± 0.89	<0.001
Total cholesterol (mmol/L), mean ± SD	4.90 ± 1.11	4.63 ± 1.04	<0.001
CTI, mean ± SD	8.95 ± 0.85	9.34 ± 0.87	<0.001

Abbreviations: BMI, Body Mass Index; ALT, Alanine Aminotransferase; AST, Aspartate Aminotransferase; GGT, Gamma-Glutamyl Transferase; CTI, C-Reactive Protein-Triglyceride Glucose Index; SD, Standard Deviation. The p-value was estimated using chi-square for categorical variables and t-tests for continuous variables.

Table 2. Association between hs-CRP, CTI, and the obese phenotype of non-alcoholic fatty liver disease.

Variable	Model 1 OR (95% CI), p	Model 2 OR (95% CI), p	Model 3 OR (95% CI), p	Model 4 OR (95% CI), p
hs-CRP	1.11 (1.08, 1.15), p < 0.001		1.04 (1.01, 1.07), p = 0.016	1.01 (0.99, 1.03), p = 0.411
CTI		1.70 (1.47, 1.98), p < 0.001	1.77 (1.48, 2.12), p < 0.001	2.09 (1.68, 2.62), p < 0.001
Age (years)			0.98 (0.98, 0.99), p < 0.001	0.98 (0.97, 0.99), p < 0.001
Sex: Female (vs Male)			1.36 (1.05, 1.77), p = 0.020	1.47 (1.09, 1.98), p = 0.011
Total cholesterol (mmol/L)				0.65 (0.57, 0.74), p < 0.001
HbA1c (%)				0.89 (0.79, 1.02), p = 0.089
Uric acid (μmol/L)				1.00 (1.00, 1.01), p < 0.001
Albumin (g/L)				0.83 (0.79, 0.87), p < 0.001

Notes: Model 1: Crude model, adjusted for hs-CRP. Model 2: Crude model, adjusted for CTI. Model 3: Adjusted for hs-CRP, CTI, age, and gender. Model 4: Adjusted for Model 3 + total cholesterol, HbA1c (%), uric acid, and albumin. Abbreviations: hs-CRP, high-sensitivity C-Reactive Protein; CTI, C-Reactive Protein-Triglyceride Glucose Index; OR, Odds Ratio; NAFLD, Non-Alcoholic Fatty Liver Disease.

The discriminative ability of the hs-CRP model (AUC = 0.711) was significantly superior to that of the CTI model (AUC = 0.628; ΔAUC = −0.082, DeLong's test p < 0.001). The Brier score for hs-CRP (0.217) was slightly lower than that for CTI (0.224), further supporting its advantage in predictive accuracy and calibration. The optimal cut-off values, as determined by the Youden index, were 2.40 mg/L for hs-CRP and 9.19 for CTI. Furthermore, since the calculation of CTI inherently incorporates hs-CRP, NRI and IDI analyses were not conducted to avoid statistical and interpretative redundancy. (Table 3 and Figure 2)

Table 3. Comparison of discriminative performance between hs-CRP and CTI models for identifying obese phenotype in NAFLD.

Model	AUC (95% CI)	ΔAUC	DeLong p	Brier
hs-CRP (M1)	0.711 (0.680 - 0.741)	Ref	-	0.217
CTI (M2)	0.628 (0.595 - 0.661)	−0.082	<0.001	0.224

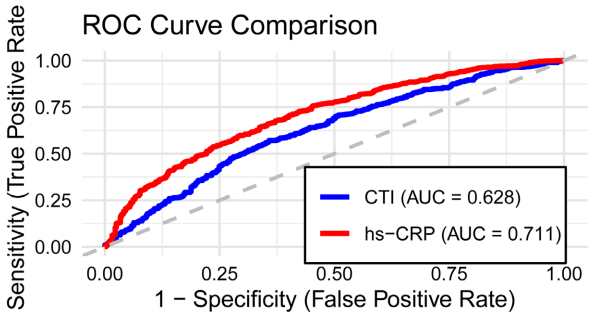

Figure 2. ROC curves for CTl and hs-CRP in identifying obese NAFLD.

4. Discussion

4.1. Principal Findings

In this cross-sectional analysis of 1171 adults with NAFLD from the NHANES 2017-2020 dataset (442 non-obese and 729 obese), both hs-CRP and CTI were associated with the obese phenotype. With respect to discriminative ability, hs-CRP demonstrated superior single-marker performance. After adjusting for multiple variables, CTI remained strongly associated with obesity, whereas hs-CRP was not significant. This result suggests that the predictive value of hs-CRP, a robust marker of obesity-related systemic inflammation, may have been captured by the composite CTI measure and other covariates included in the adjusted model.

hs-CRP exhibits good predictive accuracy (higher AUC) along with practical advantages such as wide availability and low cost, rendering it well-suited for use as a first-line screening tool for NAFLD in clinical practice. Although CTI demonstrated relatively lower standalone discriminative ability, its independent association and robustness support its unique value in multifactorial comprehensive risk assessment models. The low collinearity (VIF = 1.2) between the two markers further suggests their complementary utility in phenotypic stratification. Therefore, we propose a hierarchical biomarker application strategy for NAFLD phenotyping: hs-CRP may serve for initial phenotypic screening, while CTI could provide deeper mechanistic insights for precise risk stratification and individualized management of NAFLD.

The superior discriminative performance of hs-CRP underscores the central role of systemic inflammation in distinguishing NAFLD phenotypes, whereas the sustained association of CTI after multivariable adjustment highlights the importance of metabolic-inflammatory interactions as a defining feature of the obese phenotype.

4.2. Clinical Implications

As an inflammatory marker, hs-CRP demonstrates high discriminatory ability (AUC = 0.711) in distinguishing between obese and non-obese phenotypes of non-alcoholic fatty liver disease (NAFLD), enabling clinicians to rapidly classify NAFLD subtypes. Meanwhile, CTI provides a more accurate assessment of metabolic burden. Although CTI exhibits relatively weaker standalone discriminative performance (AUC = 0.628), it maintains a strong association with the obese phenotype even after adjusting for multiple confounding factors, highlighting its unique value in comprehensively evaluating obese NAFLD.

Therefore, a hierarchical clinical approach is recommended: hs-CRP should be prioritized for initial screening, while CTI can be incorporated subsequently for metabolic management and risk stratification. This strategy allows effective differentiation between obese and non-obese NAFLD patients, facilitating personalized intervention strategies and enabling clinicians to implement more targeted treatment plans.

4.3. Limitations

1) The cross-sectional design precludes the establishment of causal inferences. 2) The use of a non-invasive method (VCTE-CAP ≥ 274 dB/m) for NAFLD diagnosis may introduce potential misclassification bias. 3) Although multiple confounding factors were adjusted for in the multivariate models, residual confounding may remain possible due to the potential influence of unmeasured variables on the observed associations.

4.4. Future Directions

To build on these findings, future studies should take the following steps:

i) Track dynamic changes in hs-CRP and CTI to assess their incremental prognostic value in predicting NASH, fibrosis progression, and cardiovascular outcomes. Longitudinal studies would be instrumental in confirming the temporal role of these biomarkers in disease progression.

ii) Investigate inflammation-related pathways in non-obese NAFLD through histological analysis and multi-omics approaches to deepen our understanding of the mechanistic differences between obese and non-obese phenotypes.

iii) Validate and clinically calibrate the identified thresholds for hs-CRP and CTI in independent cohorts to confirm their generalizability and applicability in clinical settings.

iv) Integrate imaging techniques for adipose distribution with metabolic-inflammatory biomarkers, such as CTI and hs-CRP, to enhance both the biological interpretability and clinical utility of these indices. Such an integration could further improve the precision of phenotyping NAFLD patients and guide personalized management strategies.

5. Conclusions

This study indicates that both CTI and hs-CRP possess distinct advantages in differentiating between obese and non-obese NAFLD phenotypes, with their roles being complementary. hs-CRP demonstrates stronger discriminative ability, making it suitable as a primary indicator for rapid clinical screening. In contrast, CTI maintains a stable association with obese phenotypes even after multivariate adjustments, better reflecting metabolic and inflammatory burden, and thus holds unique value in risk stratification. The low collinearity between the two further supports the fact that they provide mutually complementary information.

Therefore, we recommend adopting a tiered application strategy in clinical practice: using hs-CRP as an initial screening tool, followed by combining CTI for metabolic risk assessment and stratified management. This approach not only facilitates more accurate differentiation between obese and non-obese NAFLD patients but also promotes the development of individualized intervention and treatment plans.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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