European Journal of Clinical and Experimental Medicine

ONLINE FIRST

Authors: Sachin Kumar, Vignessh Raveekumaran, Sethuraj Selvaraj, K.S. Chenthil

Article type: Original Article

Received: 16 September 2025

Accepted: 22 November 2025

Published online: 7 January 2026

eISSN: 2544-1361

Eur J Clin Exp Med

doi:10.15584/ejcem.2026.1.21

Association of the DeRitis ratio with insulin resistance in non-obese adults – a cross-sectional study from South India

Sachin Kumar, Vignessh Raveekumaran, Sethuraj Selvaraj, K.S. Chenthil

Department of General Medicine, Mahatma Gandhi Medical College and Research Institute, Sri Balaji Vidyapeeth (Deemed to be University), Pondicherry, India

Corresponding author: Vignessh Raveekumaran, e-mail: vignesshr@mgmcri.ac.in

ORCID

SK: https://orcid.org/0009-0000-3250-8595

VR: https://orcid.org/0009-0006-6283-6542

SS: https://orcid.org/0009-0009-0867-4070

KSC: https://orcid.org/0009-0002-2699-160X

ABSTRACT

Introduction and aim. Beyond overt obesity, insulin resistance (IR) is increasingly recognized in non-obese individuals, particularly South Asians. Liver enzymes, especially aspartate aminotransferase (AST) and alanine aminotransferase (ALT), and their ratio (DeRitis) have emerged as potential surrogate markers of metabolic dysfunction. To the best of our knowledge, this is the first study to evaluate the DeRitis ratio as a surrogate marker of IR specifically in non-obese South Indian adults, addressing an important evidence gap. With this background, the aim was to estimate the IR prevalence in non-obese adults by homeostasis model assessment of IR (HOMA-IR) and to assess the correlation and diagnostic performance of the DeRitis ratio.

Material and methods. This cross-sectional study included 100 non-obese adults (body mass index (BMI) <25kg/m²) selected using a convenience sampling technique attending a tertiary care hospital in Pondicherry, India. Data collected by structured proforma and biochemical assays of fasting plasma glucose, fasting insulin, and liver enzymes. HOMA-IR ≥2.5 as confirmed IR. The correlation and diagnostic accuracy of the DeRitis ratio for predicting IR was analyzed using SPSS software (V_25.0); $p<0.05$ considered statistically significant.

Results. IR (HOMA-IR 2.5) was present in 13% of participants. Overweight individuals showed significantly higher fasting insulin levels and HOMA-IR values compared to adults with normal BMI. The DeRitis ratio was positively correlated with HOMA-IR ($r=0.516$, $p<0.001$). Using the cut-off AST/ALT

>1.0, the ratio demonstrated good discriminatory ability for IR (AUC=0.778), with 82.5% sensitivity and 83.3% specificity.

Conclusion. The DeRitis ratio shows moderate discrimination for IR and may aid in screening where insulin assays are limited. Validation in larger, multicenter cohorts is warranted.

Keywords. alanine transaminase, aspartate aminotransferase, body weight, insulin resistance, India

Introduction

Obesity is a multifactorial disease that has escalated globally, now affecting roughly one in eight people and surpassing one billion individuals, with adult obesity more than doubling since 1990 and adolescent obesity quadrupling, underscoring a substantial cardiometabolic burden.[1,2] Elevated hepatic enzymes, particularly alanine aminotransferase (ALT), aspartate aminotransferase (AST), and γ-glutamyl transferase (GGT), track closely with adiposity and metabolic dysfunction, reflecting steatosis, inflammation, and hepatocellular injury that accompany obesity.[3,4] Several population-based studies have consistently demonstrated that even modest elevations in liver enzymes are associated with IR, metabolic syndrome, and future diabetes risk across diverse ethnic groups, reinforcing the liver's centrality in obesity-related risk stratification.[3,5,6] These findings support the growth recognition of hepatic biomarkers as early indicators of metabolic dysfunction.

IR is a hallmark of obesity, driven by adipose-tissue inflammation, lipotoxicity, ectopic fat deposition, and endocrine-immune cross-talk that impair insulin-receptor signalling across liver, skeletal muscle, and adipose tissue.[7–9] These processes include cytokine-mediated serine phosphorylation of insulin-signalling intermediates, mitochondrial stress, and altered adipokine profiles, collectively propagating systemic IR and its sequelae, including type-2 diabetes mellitus (T2DM), dyslipidaemia, and non-alcoholic fatty liver disease (NAFLD).[6,7,9]

Uncertainty surrounds the possible mechanism linking obesity to serum levels of liver enzymes. However, some studies have shown that obesity is found to be associated with an increase in DNA methylation in liver tissues, thereby increasing oxidative stress in the tissue, and this ultimately leads to liver destruction.[10] The DeRitis ratio (AST/ALT), originally proposed by Fernando DeRitis in 1957, is widely used as a biochemical index of hepatocellular injury and a versatile prognostic marker across liver and systemic illnesses, reflecting shifts in mitochondrial versus cytosolic enzyme release, necro-inflammatory activity, and extrahepatic sources of AST.[11,12]

Recent studies have shown that the DeRitis ratio is one of the independent markers of mortality.[12,13] Beyond its classical use in liver disease, emerging research highlights its potential role as a metabolic biomarker. Multiple population datasets indicate that transaminase-based indices related to metabolic risk, like in non-obese Japanese adults, the ALT/AST (inverse of De-Ritis) ratio, were the best surrogate of IR (Homeostasis Model Assessment of insulin resistance (HOMA-IR)),[14] while a large Korean cohort analysis showed

DeRitis outperforming single-enzyme measures for predicting dysglycemia and IR.[15] These reports collectively indicate that liver enzyme ratios may capture early hepatic-metabolic interactions even before overt disease manifestations. Earlier work also linked elevated ALT with future T2DM and declining hepatic insulin sensitivity, reinforcing the liver-IR nexus [16]. However, these findings have been predominantly documented in East Asian population,[17,18] and there remains limited evidence from South Asian settings, where metabolic risk occurs at lower BMI thresholds and hepatic fat accumulation is common even among non-obese individuals.

Despite growing evidence that transaminase-based ratios reflect metabolic risk, Indian data in non-obese adults remain limited and none have specifically evaluated the DeRitis ratio as a potential surrogate marker of IR in non-obese adults. Given India's high and heterogenous obesity/overweight burden and the constrained availability of insulin assays in peripheral settings, a simple, inexpensive biochemical marker is clinically valuable.[19] Hence, this study was undertaken as a prospective, single center observational study in Pondicherry to examine the association between the DeRitis ratio and IR in non-obese adults and evaluate the ability of the DeRitis ratio to discriminate IR defined by HOMA-IR.

Aim

The primary objective of this study was to investigate the association between the DeRitis ratio and IR (HOMA-IR ≥2.5) in non-obese South Indian adults, addressing an existing evidence gap in this population. The secondary objectives were to assess the diagnostic performance of the DeRitis ratio in identifying IR and to evaluate its correlation with HOMA-IR.

Material and methods

Study design and setting

This hospital-based cross-sectional observational study was conducted in the Department of General Medicine at a tertiary care center in Pondicherry, India for a period of 12 months after obtaining Institutional Human Ethics Committee approval (MGMCRI/Res/01/2023/115/IHEC/106). All procedures performed in this study were in accordance with the ethical standards of the institutional and national research committee(s) and with the Helsinki Declaration (as revised in 2013). From all the participants, written informed consent was obtained during the data collection.

Study population

Adults aged 18–60 years attending the outpatient clinic with non-obese body mass index (BMI) (BMI<25.0kg/m^2) according to Asian cut-off were eligible.[20] BMI was categorized using Asian BMI cut-offs as normal BMI is 18.5–22.9 kg/m^2; overweight: BMI 23.0–24.9 kg/m^2; and obese: ≥25.0 kg/m^2.[20]

The exclusion criteria were patients with known liver diseases, significant alcohol intake, viral hepatitis or hepatotoxic drug use, known DM, hypertension, or other endocrine disorders, with acute illness or infection at the time of evaluation, and pregnant/lactating women.

Sample size and sampling technique

The sample size was calculated based on the 6.2% prevalence of IR reported in a study by Kawamoto et al. (2012) performed in Japan,[14] among non-obese individuals. Using the formula, $n = \frac{Z^2pq}{d^2}$ with p (prevalence) as 6.2%, 5% alpha error, 20% beta error and allowable error of 5% (d), (substituting as $n=(1.96)^2*0.062*(1-0.062)/(0.05)^2=90$) the sample size calculated was 90 (Open Epi (v3.0)). Allowing 10% for attrition rate, the required sample size was 100 participants. By convenience sampling techniques, patients were selected for the study until the desired sample size was achieved.

Outcomes

Primary – the prevalence of IR (HOMA-IR ≥2.5); secondary – (i) correlation between DeRitis ratio and HOMA-IR, (ii) diagnostic performance of AST/ALT for IR.

Study procedures

Data collection included demographic details, anthropometric measurements, and clinical history, which were recorded using a structured proforma. Then the patients were subjected to various biochemical analyses. Fasting venous blood samples were collected after an overnight fast of 8-10 hours. The following parameters were measured:

- Fasting plasma glucose (FPG) by the glucose oxidase-peroxidase method
- Fasting insulin by chemiluminescent (E-CLIA) immunoassay (Manufacturer – Cobas E411; Assay type ROCHE ELECSYS 2010; quality control at Biorad (Level 3))
- The AST and ALT were prepared using standard enzymatic methods (Manufacturer – Cobas C311; Assay type International federation of clinical chemistry (IFCC) method; quality control at Biorad (Level 3))

The DeRitis ratio[12] was calculated $\text{DeRitis ratio} = \frac{\text{AST}}{\text{ALT}}$ and used to evaluate the liver function and disease severity. An elevated ratio (>1) suggests liver damage, and ≤1 was considered normal.[12]

The HOMA-IR[21] was computed using the formula

$$\text{HOMA} - \text{IR} = \frac{\text{Fasting insulin}\left(\frac{\mu U}{mL}\right) \times \text{Fasting glucose}\left(\frac{mg}{dL}\right)}{405}.$$

IR was defined as HOMA-IR ≥2.5 (based on validated Asian cut-off).

Statistical analysis

The data was entered into Microsoft Excel and analyzed using SPSS version 25.0 (IBM, Armonk, NY, USA). Continuous variables were expressed as mean±standard deviation (SD) or median (interquartile range (IQR)) depending on normality (assessed by the Shapiro-Wilk normality test and Q-Q plot). Categorical variables were presented as proportions. An independent t-test or Mann-Whitney U-test was used to compare the continuous variables, and the homogeneity of variances were performed using the Levene's test, if violated Welch's test was used. Chi-square test or Fisher's exact test was applied for categorical variables. Pearson's correlation coefficient assessed the relationship between the DeRitis ratio and HOMA-IR after confirming its linearity and absence of extreme outliers. The diagnostic performance of the DeRitis ratio for IR was evaluated using Receiver Operating Characteristics (ROC) curve. The primary diagnostic threshold was pre-specified as DeRitis ratio >1.0, reflecting a conventional clinical cut-off used to indicate the elevation of the ratio in liver disease. At this priori cut-off, sensitivity, specificity, predictive values, and overall diagnostic accuracy, were calculated with area under the curve (AUC) at 95% CI. Youden's index was additionally computed to summarize the balance between the sensitivity and specificity. A p-value <0.05 was considered statistically significant.

Results

Participant flow and baseline characteristics are presented in Table 1, and associations with BMI is presented in Table 2.

Table 1. Socio-demographic characteristics and risk factors of the study participants (n=100)*

Variables		**Results n (%)**
Age (in years)	18–30	14 (14.0)
	31–40	20 (20.0)
	41–50	26 (26.0)
	51–60	32 (32.0)
	>60	8 (8.0)
Gender	Male	43 (43.0)
	Female	57 (57.0)
SES	Class I	12 (12.0)
	Class II	16 (16.0)
	Class III	16 (16.0)
	Class IV	24 (24.0)
	Class V	32 (32.0)

Locality	Rural	50 (50.0)
	Urban	50 (50.0)
Smoking	Present	41 (41.0)
	Absent	59 (59.0)
Alcohol consumption	Present	40 (40.0)
	Absent	60 (60.0)
Diet pattern	Vegetarian	49 (49.0)
	Non-vegetarian	51 (51.0)
BMI (kg/m²)	Overweight (23.1 – 24.9)	39 (39.0)
	Normal (<23)	61 (61.0)

* SES – socio-economic status as per the B.G. Prasad scale

Table 2. Association of sociodemographic and risk factors in relation to the BMI (Pearson's chi-square test)

Variables	**BMI (kg/m²)**		**p**
	Normal (< 23) (n=61) n (%)	**Overweight (23.1–24.9) (n=39) n (%)**	
Age (in years)			
18-30	10 (16.4)	4 (10.3)	0.035
31-40	15 (24.6)	5 (12.8)	
41-50	18 (29.5)	8 (20.5)	
51-60	12 (19.7)	20 (51.3)	
>60	6 (9.8)	2 (5.1)	
Gender			
Male	35 (57.4)	8 (20.5)	0.001
Female	26 (42.6)	31 (79.5)	
Socio-economic status (according to the B.G. Prasad scale)			
Class I	10 (16.4)	2 (5.1)	0.021
Class II	12 (19.7)	4 (10.2)	
Class III	10 (16.4)	6 (15.4)	
Class IV	16 (26.2)	8 (20.5)	
Class V	13 (19.7)	19 (48.7)	
Locality			
Rural	40 (65.6)	10 (25.6)	0.001

Urban	21 (34.4)	29 (74.4)	
Smoking status			
Present	10 (16.4)	31 (79.5)	<0.001
Absent	51 (83.6)	8 (20.5)	
Alcohol consumption			
Present	15 (24.6)	25 (64.1)	0.001
Absent	46 (75.4)	14 (35.6)	
Dietary pattern			
Vegetarian	35 (57.4)	14 (35.9)	0.022
Non-vegetarian	26 (42.6)	25 (64.1)	

Overall, 13% of participants were identified with IR (HOMA-IR ≥2.5). Using the DeRitis ratio (>1), approximately 43% of patients were identified as having insulin resistance (IR), with a significantly higher prevalence in the overweight group (33 out of 39; 84.6%) compared to the normal BMI group (10 out of 61; 16.4%), which was statistically significant ($p<0.001$, Table 3).

Similarly, the mean DeRitis ratio among patients with normal BMI was 1.084±0.060, compared to 0.920±0.035 for overweight patients, and the difference between the two groups was statistically significant ($p<0.05$). The rest of the biochemical parameters and their association with BMI are presented in Table 3.

Table 3. Association of biochemical parameters in relation to the BMI*

Variables	**BMI (kg/m²)**		**p**
	Normal (n=61) (< 23)	**Overweight (n=39) (23.1–24.9)**	
ALT	16.28 ± 1.25	23.05 ± 1.61	<0.001
AST	17.61 ± 1.41	21.23 ± 1.61	<0.001
DeRitis ratio	1.08 ± 0.06	0.92 ± 0.03	<0.001
FBS	87.59 ± 3.11	94.92 ± 4.69	<0.001
Fasting insulin	5.34 ± 2.04	8.35 ± 1.90	<0.001
HOMA-IR	1.16 ± 0.53	1.97 ± 0.50	<0.001
HOMA-IR ranges			
<1.6	51 (83.6)	7 (17.9)	<0.001
1.6–2.5	7 (11.5)	22 (56.4)	
≥2.5	3 (4.9)	10 (25.6)	

DeRitis ratio and IR diagnosis			
>1	10 (16.4)	33 (84.6)	<0.001
≤1	51 (83.6)	6 (15.4)	

* data were presented in the form of frequency (percentage) or mean ± standard deviation, based on the type of variables, Student's t-test

A significant positive correlation was observed between the DeRitis ratio and HOMA-IR (Pearson's r=0.516; p<0.001), indicating that higher IR was associated with an altered transaminase ratio. The ROC curve demonstrated an AUC of 0.778 (95% CI: 0.675–0.865), confirming the discriminatory ability of the DeRitis ratio in detecting IR. The overall diagnostic accuracy was 83% (95% CI: 74.3–90.1%), and Youden's index was 66%, suggesting that the DeRitis ratio demonstrated moderate discrimination for identifying IR among non-obese patients and is presented in Table 4 and Figure 1.

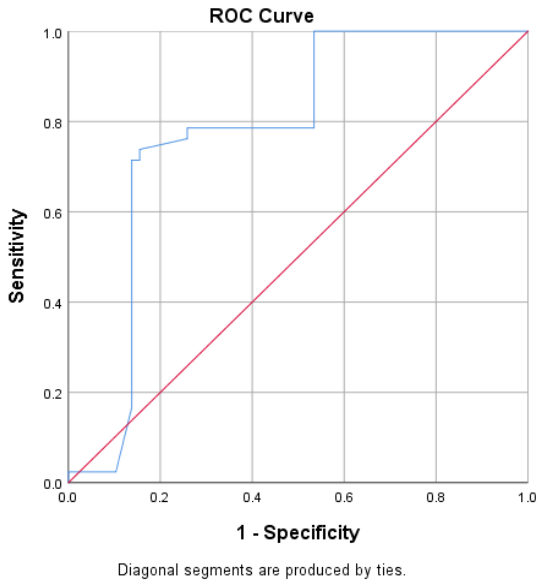

Fig. 1. ROC curve of the DeRitis ratio for the diagnosis of insulin resistance

Table 4. Diagnostic accuracy of DeRitis ratio for insulin resistance diagnosis*

Variables	Result	95% CI
Sensitivity (%)	82.50	57.3–96.2
Specificity (%)	83.33	73.4–90.9
Positive predictive value (%)	76.74	50.1–93.2
Negative predictive value (%)	87.72	78.5–94.0
Accuracy	83	74.3–90.1

Area under the curve*	0.778	0.675–0.865
Youden's index (J) (%)	66	

* the 95% confidence interval (CI) was calculated using DeLong's estimate, with a standard error (SE) of approximately 0.048; diagnostic accuracy was assessed at the pre-specified threshold of a DeRitis ratio greater than 1.0, CI – confidence interval

Discussion

This present cross-sectional study of 100 non-obese adults in Pondicherry demonstrated that 43% had IR, with prevalence markedly higher among overweight individuals (84.6%) compared to participants with normal BMI (16.4%). The research identified a substantial correlation between the DeRitis ratio and HOMA-IR ($r=0.516$; $p<0.001$), with ROC analysis producing an AUC of 0.778 and a diagnostic accuracy of 83%. These findings suggest that the DeRitis ratio can serve as a simple, inexpensive biochemical marker for identifying IR in non-obese adults, complementing or substituting for insulin-based assays in resource-limited settings.

Obesity and overweight are now established as major public health problems. The WHO estimated that obesity affects over a billion people globally, with prevalence doubling since 1990.[2,5] In the present study, the prevalence of overweight was 39%. In India, recent estimates place obesity prevalence between 11.8% and 40.3%, varying by region and gender.[19,22] In this study population, while restricted to non-obese individuals, it highlights that IR is not limited to overt obesity, reinforcing prior evidence that metabolic dysfunction can occur even at lower BMI thresholds in Asian population warranting re-evaluation of risk stratification paradigms for this population.[23,24]

When comparing the ALT and AST levels between the groups, the mean ALT among overweight people is 23.05±1.614 IU/L vs 6.28±1.255 IU/L in normal BMI patients and is statistically significant, and the mean AST among overweight and normal patients (21.23±1.613 IU/L vs 17.61±1.41 IU/L) is statistically significant. Similar to our results, a study done by Jalili et al.,[4] showed that ALT and AST levels were found to be higher among the obese than the non-obese individuals. This finding was further supported by Momo et al.[25] and Song et al.[26], where liver enzymes such as ALT and AST were found to be higher among obese individuals. Although the mechanism is not understood, it is found that increased accumulation of fat in liver cells and disruption of hepatocytes results in elevated liver enzymes.[3,6,10]

The DeRitis ratio, introduced by DeRitis in 1957,[12,27] has long served as a biomarker of hepatic injury, mitochondrial compromise, and systemic inflammatory load. Recently, its prognostic value has expanded to cover NAFLD, cardiovascular outcomes, and malignancies.[11,28] The present study extends its utility, demonstrating that even within non-obese cohorts, shifts in transaminase balance mirror metabolic risk and correlate with IR.[14,15] Mechanistically, IR in obesity arises from ectopic lipid accumulation, inflammatory cytokine cascades, and mitochondrial dysfunction, culminating in hepatic lipotoxicity and altered enzyme

release.[7,8] A reduced AST/ALT ratio reflects relative ALT elevation due to hepatocellular steatosis and mitochondrial injury. In the non-obese population, this pattern hints at subclinical NAFLD. The present study results, revealing lower DeRitis ratios in individuals with IR, align with observed biochemical shifts in early metabolic derangement.

In the present study, the DeRitis ratio between the participants in the overweight (0.920±0.035) and normal groups (1.084±0.060) was compared and found to be statistically significant and higher among the overweight participants. A study by Ndrepepa et al.,[12] found that the DeRitis ratio was >1.0 among obese participants, and >2 was noted among participants with alcoholic fatty liver disease. The DeRitis ratio was also found to be associated with the increased risk of mortality among participants with obesity.[11,13,28]

The HOMA-IR is used to assess IR in individuals; in our study, the mean HOMA-IR among the overweight and normal BMI participants was 1.97±0.51 vs 1.17±0.53. Our finding of a moderate positive correlation between the DeRitis ratio and HOMA-IR (r=0.516) is consistent with earlier surveys demonstrating that ALT/AST or AST/ALT ratios are valid surrogates of IR. The study by Lee et al.,[29] showed that the obese individuals were found to have higher levels of HOMA-IR values than the non-obese individuals. Similarly, a study by Raj et al.,[30] also showed a positive correlation between the HOMA-IR and the BMI. A study by Kawamoto et al.,[14] found that ALT/AST is the best IR surrogate in non-obese Japanese adults, and Han et al.,[15] confirmed this in a large Korean cohort. Vozarova et al., further reported that elevated ALT predicts future T2DM.[16] We are adding to this corpus by validating that the DeRitis ratio performs comparably in predicting IR among the non-obese Indian adults, thus reinforcing its prospective clinical relevance in screening and risk stratification.

Given the low cost and widespread availability of transaminases, the AST/ALT ratio may complement risk screening in situations where insulin assays are unavailable; however, its performance is insufficient for diagnosis and requires external validation. To align claims with the evidence, we intentionally avoid diagnostic language and frame the DeRitis ratio as a screening adjunct rather than a standalone test.

This study is among the few from South India that specifically investigates the association between the DeRitis Ratio and IR in non-obese adults, an often-overlooked subgroup in metabolic research at lower BMI compared with Japanese and Korean cohorts.[14,15,26] By demonstrating a significant association between the DeRitis ratio and IR in this ethnic group, the study fills an important geographic and metabolic evidence gap and extends the applicability of transaminase-based markers to a high-risk South Asian population. Moreover, the use of standardised biochemical methods improved the internal validity of the findings. The inclusion of both normal and overweight participants within the non-obese BMI range allowed meaningful subgroup comparisons. The diagnostic evaluation of the DeRitis Ratio adds quantitative rigour and translational clinical value.

Nonetheless, this study has limited novelty relative to prior enzyme-based markers; the sample size is small, from a single tertiary center, which limits precision and generalizability. The cross-sectional design

precludes causal inference and may introduce spectrum bias. Moreover, the assessment of HOMA-IR has inherent methodological constraints and is considered a surrogate and not a gold-standard clamp. The HOMA-IR index is influenced by pancreatic β-cell function and showed variability with age, sex, and pubertal stages, which might affect its accuracy across different physiological states. Because HOMA-IR provides only a static estimate derived from fasting glucose and insulin, it does not capture dynamic glucose-insulin interactions over a 24-hour period. Additionally, its reproducibility has been reported as moderate and lower for indices such as the Quantitative Insulin Sensitivity Check Index (QUICKI).[31] Recent evidence suggests that the triglyceride-glucose (TyG) index demonstrates superior diagnostic performance for IR[32] and could be explored in future studies for comparison. Although the present study demonstrates a significant association between the DeRitis ratio and IR, it is important to acknowledge the role of potential confounders that might influence the study findings. Factors such as hepatic steatosis by imaging, alcohol intake quantification, diet, physical activity, medications, raising residual confounding and spectrum bias.

Future multicenter studies with larger, more diverse cohorts are recommended to validate the association between the DeRitis ratio and IR in non-obese adults. Comparative analyses using alternative IR indices such as the TyG index, QUICKI, and clamp-based methods could provide more robust evidence and determine which index most accurately reflects metabolic risk in the South Asian population. Incorporating validated dietary assessment, objective alcohol consumption measures, and liver imaging along with longitudinal follow-up could help establish temporal relationships between hepatic enzyme alterations and metabolic dysfunction and yield more precise estimates of the independent relationship between DeRitis ratio and IR. Exploring sex- and age-specific cut-offs for both HOMA-IR and the DeRitis ratio would also improve diagnostic precision in clinical screening.

Conclusion

Given the rising burden of metabolic disorders in India, even among individuals with normal or near-normal BMI, the DeRitis ratio offers a simple, inexpensive, and readily available biochemical marker for early identification of at-risk individuals. The present study demonstrated that the DeRitis ratio is associated with IR and shows moderate discrimination. Findings should be viewed as hypothesis-generating and require confirmation in larger, multicenter cohorts with a comprehensive confounder assessment.

Acknowledgements

We thank all the laboratory technicians, medical interns and nurses for their generous assistance and support. I also extend my gratitude to the patients for their willingness and consistent support.

Declarations

Funding

This research did not receive any specific funding from any public, commercial, or no-for-profit-sector agencies.

Author contributions

Conceptualization, K.S.C., S.S. and V.R.; Methodology, V.R.; Software, S.K.; Validation, K.S.C., and S.S.; Formal Analysis, S.K. and V.R.; Investigation, S.K.; Resources, S.K. and V.R.; Data Curation, V.R; Writing – Original Draft Preparation, S.K. and V.R..; Writing – Review & Editing, K.S.C. and S.S.; Visualization, V.R..; Supervision, K.S.C.; Project Administration, V.R.; Funding Acquisition, S.K.

Conflicts of interest

With respect to the research, authorship, and publication of this article, the authors declared no potential conflicts of interest.

Data availability

This published article includes all the data generated or analyzed during the study.

Ethics approval

This study was approved by the Institutional Ethical Committee (MGMCRI/Res/01/2023/115/IHEC/106).

References

1. NCD Risk Factor Collaboration (NCD-RisC). Worldwide trends in underweight and obesity from 1990 to 2022: a pooled analysis of 3663 population-representative studies with 222 million children, adolescents, and adults. *Lancet*. 2024;403(10431):1027-1050. doi:10.1016/S0140-6736(23)02750-2
2. James WPT. WHO recognition of the global obesity epidemic. *Int J Obes*. 2008;32(suppl 7):S120-S126. doi:10.1038/ijo.2008.247
3. El-Eshmawy MM. Impact of obesity on liver function tests: Is nonalcoholic fatty liver disease the only player? A review article. *Porto Biomed J*. 2023;8(5):e228. doi:10.1097/j.pbj.0000000000000228
4. Jalili V, Poorahmadi Z, Hasanpour Ardekanizadeh N, et al. The association between obesity with serum levels of liver enzymes, alanine aminotransferase, aspartate aminotransferase, alkaline phosphatase and gamma-glutamyl transferase in adult women. *Endocrinol Diabetes Metab*. 2022;5(6):e367. doi:10.1002/edm2.367

5. World Health Organization. One in eight people are now living with obesity. WHO News. https://www.who.int/news/item/01-03-2024-one-in-eight-people-are-now-living-with-obesity. Accessed September 3, 2025.
6. Ghotbi S, Joukar F, Orang Goorabzarmakhi M, et al. Evaluation of elevated serum liver enzymes and metabolic syndrome in the PERSIAN Guilan cohort study population. *Heliyon*. 2024;10(11):e32449. doi:10.1016/j.heliyon.2024.e32449
7. Arneth B. Mechanisms of Insulin Resistance in Patients with Obesity. *Endocrines*. 2024;5(2):153-165. doi:10.3390/endocrines5020011
8. Zhao X, An X, Yang C, Sun W, Ji H, Lian F. The crucial role and mechanism of insulin resistance in metabolic disease. *Front Endocrinol*. 2023;14:1149239. doi:10.3389/fendo.2023.1149239
9. Jin X, Qiu T, Li L, et al. Pathophysiology of obesity and its associated diseases. *Acta Pharm Sin B*. 2023;13(6):2403-2424. doi:10.1016/j.apsb.2023.01.012
10. Geißler C, Krause C, Neumann AM, et al. Dietary induction of obesity and insulin resistance is associated with changes in Fgf21 DNA methylation in liver of mice. *J Nutr Biochem*. 2022;100:108907. doi:10.1016/j.jnutbio.2021.108907
11. Shaikh SM, Varma A, Kumar S, Acharya S, Patil R. Navigating disease management: a comprehensive review of the De Ritis ratio in clinical medicine. *Cureus*. 2024;16(7):e64447. doi:10.7759/cureus.64447
12. Ndrepepa G, Holdenrieder S, Kastrati A. Prognostic value of DeRitis ratio with aspartate aminotransferase and alanine aminotransferase within the reference range. *Clin Chim Acta*. 2023;538:46-52. doi:10.1016/j.cca.2022.11.005
13. Li Q, Lan W. Association between DeRitis ratio (AST/ALT) and mortality in patients with chronic kidney disease: a retrospective cohort study. *Sci Rep*. 2025;15:9649. doi:10.1038/s41598-025-93184-1
14. Kawamoto R, Kohara K, Kusunoki T, Tabara Y, Abe M, Miki T. Alanine aminotransferase/aspartate aminotransferase ratio is the best surrogate marker for insulin resistance in non-obese Japanese adults. *Cardiovasc Diabetol*. 2012;11(1):117. doi:10.1186/1475-2840-11-117
15. Han SK, Seo MJ, Lee T, Kim MY. Effectiveness of the ALT/AST ratio for predicting insulin resistance in a Korean population: A large-scale, cross-sectional cohort study. *PLOS ONE*. 2024;19(5):e0303333. doi:10.1371/journal.pone.0303333
16. Vozarova B, Stefan N, Lindsay RS, et al. High alanine aminotransferase is associated with decreased hepatic insulin sensitivity and predicts the development of type 2 diabetes. *Diabetes*. 2002;51(6):1889-1895. doi:10.2337/diabetes.51.6.1889
17. Narayan KMV, Kanaya AM. Why are South Asians prone to type 2 diabetes? A hypothesis based on underexplored pathways. *Diabetologia*. 2020;63(6):1103-1109. doi:10.1007/s00125-020-05132-5

18. Misra A, Soares MJ, Mohan V, et al. Body fat, metabolic syndrome and hyperglycemia in South Asians. *J Diabetes Complications*. 2018;32(11):1068-1075. doi:10.1016/j.jdiacomp.2018.08.001
19. Venkatrao M, Nagarathna R, Majumdar V, Patil SS, Rathi S, Nagendra H. Prevalence of obesity in India and its neurological implications: a multifactor analysis of a nationwide cross-sectional study. *Ann Neurosci*. 2020;27(3-4):153-161. doi:10.1177/0972753120987465
20. Mahajan K, Batra A. Obesity in adult asian indians- the ideal BMI cut-off. *Indian Heart J*. 2018;70(1):195. doi:10.1016/j.ihj.2017.11.020
21. Minh HV, Tien HA, Sinh CT, et al. Assessment of preferred methods to measure insulin resistance in Asian patients with hypertension. *J Clin Hypertens*. 2021;23(3):529-537. doi:10.1111/jch.14155
22. Ahirwar R, Mondal PR. Prevalence of obesity in India: A systematic review. *Diabetes Metab Syndr Clin Res Rev*. 2019;13(1):318-321. doi:10.1016/j.dsx.2018.08.032
23. Misra A, Khurana L. Obesity and the metabolic syndrome in developing countries. *J Clin Endocrinol Metab*. 2008;93(11 Suppl 1):S9-30. doi:10.1210/jc.2008-1595
24. Chan K, De Souza LR, Kobayashi K, Fuller-Thomson E. Diabetes and diabetes care among non-obese South Asian Americans: Findings from a population-based study. *Diabetes Metab Syndr*. 2019;13(1):96-102. doi:10.1016/j.dsx.2018.08.025
25. Momo FR, Hoque MR, Sarkar S, Faysal MR, Mitu SA, Suchi SS. Comparison of serum alanine aminotransferase and aspartate aminotransferase among obese and non-obese adults. *Mymensingh Med J MMJ*. 2024;33(4):955-961.
26. Song HR, Yun KE, Park HS. Relation between alanine aminotransferase concentrations and visceral fat accumulation among nondiabetic overweight Korean women. *Am J Clin Nutr*. 2008;88(1):16-21. doi:10.1093/ajcn/88.1.16
27. DeRitis F, Coltorti M, Giusti G. An enzymic test for the diagnosis of viral hepatitis: The transaminase serum activities. *Clin Chim Acta*. 2006;369(2):148-152. doi:10.1016/j.cca.2006.05.001
28. Botros M, Sikaris KA. The De Ritis ratio: the test of time. *Clin Biochem Rev*. 2013;34(3):117-130.
29. Lee S, Lacy ME, Jankowich M, Correa A, Wu WC. Association between obesity phenotypes of insulin resistance and risk of type 2 diabetes in African Americans: The Jackson Heart Study. *J Clin Transl Endocrinol*. 2020;19:100210. doi:10.1016/j.jcte.2019.100210
30. Raj A, Vijayakumar V. Correlation of HOMA IR with BMI. *Asian J Med Res*. 2021;10(3):5-8.
31. Mirzaalian Y, Nourian M, Gholamalizadeh M, et al. The association of quantitative insulin sensitivity indices (HOMA-IR and QUICKI) with anthropometric and cardiometabolic indicators in adolescents. *Arch Med Sci Atheroscler Dis*. 2019;4:e32-e37. doi:10.5114/amsad.2019.84411
32. Park MS. Triglyceride-glucose index predicts future metabolic syndrome in an adult population, Korea: a prospective cohort study. *Ann Clin Nutr Metab*. 2024;16(3):168-172. doi:10.15747/ACNM.2024.16.3.168