Journal of Paediatric Surgeons of Bangladesh (2026) Vol. 17 (2)
Official Journal of the Association of Paediatric Surgeons of Bangladesh
https://doi.org/10.3329/jpsb.v17i2.93380

Journal of Paediatric Surgeons of Bangladesh

Original Article

Metabolic and Anthropometric Predictors of Paediatric Metabolic Steatotic Liver Disease: Development of a Clinical Risk Prediction Model

Khan NA[1], Nabi A[2], Haque QUL[3], Tasnim T[4], Nahar ZU[5], Zaman S[6], Ahmed R[3]

Abstract

Background: Paediatric metabolic dysfunction-associated steatotic liver disease (MASLD) is common yet frequently undetected, and access to imaging is limited in many settings. A simple risk tool built from routinely available variables could prioritize children for confirmatory assessment. We aimed to develop and internally validate a multivariable model predicting ultrasound-detected MASLD from metabolic and anthropometric data.

Methods: In a cross-sectional study of 205 children and adolescents (aged 5–17 years) with metabolic risk factors, MASLD was ascertained by abdominal ultrasonography. Candidate predictors comprised demographic, anthropometric, biochemical, and family-history variables. A multivariable logistic regression model was developed using clinical reasoning and parsimony, with continuous predictors transformed where appropriate. Discrimination (C-statistic), calibration (flexible calibration curve, calibration slope, Hosmer–Lemeshow test), and clinical utility (decision curve analysis) were assessed; optimism was estimated by bootstrapping (1000 resamples). A simplified integer points score was derived and stratified into risk categories. The study follows the TRIPOD statement.

Results: Ultrasound-detected MASLD was present in 109 of 205 participants (53.2%). The final model retained three predictors: body mass index (BMI), log-transformed ALT, and family history of dyslipidaemia. BMI was associated with MASLD with an adjusted odds ratio (aOR) of 1.81 (95% CI, 1.25–2.62) per 5 kg/m² increase, while a two-fold increase in ALT was associated with an aOR of 2.62 (95% CI, 1.67–4.10). Family history of dyslipidaemia was also independently associated with MASLD (aOR, 1.87; 95% CI, 1.02–3.43). The apparent C-statistic was 0.741, with an optimism-corrected C-statistic of 0.730 and a bootstrap-corrected calibration slope of 0.95. Graphical assessment suggested generally acceptable calibration across most of the predicted-risk range, although the Hosmer–Lemeshow test indicated statistically significant departure from perfect fit. Decision-curve analysis suggested positive net benefit of the model across clinically relevant threshold probabilities. A simplified points score showed a C-statistic of 0.711 and separated participants into lower-, intermediate-, and higher-predicted-risk groups.

Conclusions: A parsimonious model incorporating BMI, ALT, and family history of dyslipidaemia showed moderate discrimination and generally acceptable calibration for ultrasound-detected MASLD among children and adolescents with metabolic risk factors. The model may have potential as a triage tool to prioritize children for further hepatic assessment where imaging resources are limited. However, the model and its simplified points score require external validation in independent and more representative paediatric populations before clinical application.

Keywords: metabolic dysfunction-associated steatotic liver disease; risk prediction model; children; body mass index; alanine aminotransferase; logistic regression; calibration; decision curve analysis.

1. Naila Atik Khan
Associate Professor, Department of Biochemistry & Molecular Biology, Bangladesh Medical University

2. Ashfaque Nabi
Assistant Professor, Department of paediatric Surgery, Dhaka Medical College

3. Quazi Umme La-sani Haque
Research Assistant
Bangladesh Medical University

4. Torsa Tasnim
Research Assistant
Bangladesh Medical University

5. Zeba-Un-Naher
Associate Professor, Dept. of Biochemistry & Molecular Biology
Bangladesh Medical University

6. Shiblee Zaman
Assistant Professor, Dept. of Biochemistry & Molecular Biology
Bangladesh Medical University

Correspondence to: **Naila Atik Khan**
Associate Professor, Dept. of Biochemistry & Molecular Biology
Bangladesh Medical University, Shahbag, Dhaka, Bangladesh
E-mail: naila.atik@bsmmu.edu.bd; Cell: +8801715-475217

7. Rumana Ahmed
Assistant Professor, Dept. of Biochemistry & Molecular Biology
Bangladesh Medical University

1. Introduction

Metabolically-dysfunction-associated steatotic liver disease (MASLD), the term that replaced non-alcoholic fatty liver disease in 2023, is now the leading chronic liver disorder in children and adolescents, mirroring the global escalation of paediatric obesity [1,2]. Because early MASLD is asymptomatic, it commonly escapes recognition until hepatic fibrosis or cardiometabolic comorbidity has developed, by which time opportunities for reversible intervention have narrowed [3,4]. The clinical challenge is therefore one of efficient case-finding: identifying, among the large population of children with metabolic risk, those most likely to harbor steatosis and warranting confirmatory evaluation.

The reference standards for steatosis—liver biopsy and quantitative magnetic resonance imaging—are invasive or unavailable in most paediatric settings, and even ultrasonography, though widely used, requires equipment and operator expertise that are scarce in primary care and population screening [5,6]. Current paediatric guidance recommends screening at-risk children but offers no validated, quantitative tool to rank individual risk and allocate limited imaging capacity [7]. This gap is particularly consequential in low- and middle-income countries, where the burden of paediatric obesity is rising fastest and diagnostic resources are most constrained [8]. A pragmatic response is a multivariable risk prediction model built from variables already collected during routine assessment of childhood obesity. Anthropometric measures, aminotransferases, lipid profiles, and family history are inexpensive, broadly available, and biologically linked to hepatic steatosis [9]. Although individual markers and insulin resistance indices have been examined as screening tests for paediatric MASLD [10–12], they are typically reported in isolation; combining several routine predictors in a single, calibrated model—and translating it into a usable bedside score may yield greater and more actionable predictive value [13]. Such a model would complement, rather than replace, confirmatory imaging by triggering which children most need it.

We therefore aimed to develop and internally validate a parsimonious clinical risk prediction model for ultrasound-detected MASLD in at-risk children, using only routinely available metabolic, anthropometric, and family-history variables, and to derive a simplified integer points score with defined risk strata to support its use at the point of care [14].

2. Materials and Methods

2.1 Study design, setting, and participants

This cross-sectional model-development study was conducted at Bangladesh Medical University (BMU), Dhaka. Consecutive children and adolescents aged 5–17 years attending the paediatric outpatient department with at least one metabolic risk factor (overweight or obesity, acanthosis nigricans, raised aminotransferases, or a family history of type 2 diabetes or dyslipidaemia) were assessed for eligibility. Children with liver disease of other etiology (viral or autoimmune hepatitis, Wilson's disease, alpha-1 antitrypsin deficiency), type 1 diabetes or unrelated endocrine disorders, current hepatotoxic or insulin-sensitizing medication, intercurrent systemic illness, or incomplete data were excluded. Written parental consent and, where appropriate, child assent was obtained. The study is reported in accordance with the Transparent Reporting of a multivariable prediction model for Individual Prognosis or Diagnosis (TRIPOD) statement [14].

2.2 Outcome

The outcome was MASLD defined by abdominal ultrasonography, read by an experienced radiologist blinded to clinical and biochemical data, based on increased hepatic echogenicity, hepatorenal contrast, and attenuation of vascular and diaphragmatic definition, with secondary causes excluded. The outcome was recorded as present or absent before any predictor modelling.

2.3 Candidate predictors

Candidate predictors were restricted, a priori, to variables obtainable during routine care: age, sex, family history of type 2 diabetes, dyslipidaemia, and liver disease, obesity status, BMI, waist circumference, fasting plasma glucose, fasting insulin, triglycerides, total, high-density lipoprotein (HDL) and low-density lipoprotein (LDL) cholesterol, and alanine aminotransferase (ALT). Anthropometry was measured with calibrated instruments after an 8–12 h fast, when venous blood was also drawn. To avoid undue influence of extreme values, the right-skewed ALT was modelled on the natural-logarithmic scale; collinear adiposity measures (BMI and waist circumference, Spearman $r = 0.76$) were not entered simultaneously.

2.4 Sample size

The available sample of 205 participants, established for the precision of diagnostic estimates [15], yielded 109 outcome events. With a final model of three parameters, this corresponds to approximately 36 events per variable, well above the conventional minimum and supportive of stable coefficient estimation.

2.5 Model development

Predictors were first examined univariately by logistic

regression. The multivariable model was built by combining variables with univariable association ($p < 0.10$) or established clinical relevance, favoring parsimony and avoiding collinearity; candidate models were compared by the Akaike information criterion, and predictors that were neither statistically nor clinically contributory were removed. The final model is reported as adjusted odds ratios (aOR) with 95% confidence intervals (CI) and as a probability equation.

2.6 Model performance and internal validation

Discrimination was quantified by the concordance (C) statistic, equivalent to the area under the receiver operating characteristic curve. Calibration was assessed graphically with a locally weighted (lowess) calibration curve and grouped deciles, by the calibration intercept and slope, and by the Hosmer–Lemeshow test. Internal validation used 1000 bootstrap resamples to estimate optimism in the C-statistic and the calibration slope (uniform shrinkage factor), following the Harrell approach. Clinical utility was evaluated by decision curve analysis, comparing the net benefit of the model with the default strategies of treating (imaging) all or no children across threshold probabilities [16].

2.7 Simplified points score

To support bedside use, the regression coefficients were converted into an integer points system following the method of Sullivan and colleagues [17], assigning points proportional to each predictor's contribution per clinically meaningful increment. Total points were mapped to observed MASLD risk and grouped into low-, intermediate-, and high-risk strata. Analyses were performed in SPSS version 27 (IBM Corp., Armonk, NY) and Python 3 (statsmodels, scikit-learn); two-sided $p < 0.05$ indicated significance.

3. Results

3.1 Participants

Of 205 children and adolescents (mean age 11.6 ± 2.2 years; 53.2% male), 178 (86.8%) were obese, and the remainder were overweight. Ultrasound-defined MASLD was present in 109 (53.2%). In univariable analysis (Table 1), MASLD was associated with higher BMI (odds ratio [OR] 1.73 per 5 kg/m²), larger waist circumference (OR 1.50 per 10 cm), higher ALT (OR 1.39 per 10 U/L), and obesity versus overweight (OR 2.56), with a borderline association for family history of dyslipidaemia (OR 1.63, $p = 0.082$). Age, sex, fasting glucose, fasting insulin, triglycerides, and HDL or LDL cholesterol were not significantly associated.

Table 1. Univariable associations between candidate predictors and ultrasound-defined MASLD. Odds ratios (OR) are expressed per the stated increment.

Predictor	Increment /contrast	OR (95% CI)	P
Age	per year	1.01 (0.89–1.14)	0.90
Male sex	male vs female	1.17 (0.68–2.04)	0.57
Family history of diabetes	Yes vs No	1.15 (0.64–2.08)	0.64
Family history of dyslipidaemia	Yes vs. No	1.63 (0.94–2.84)	0.082
Obesity	obese vs overweight	2.56 (1.09–6.02)	**0.031**
Body mass index	per 5 kg/m²	1.73 (1.22–2.45)	**0.002**
Waist circumference	per 10 cm	1.50 (1.14–1.98)	**0.003**
Fasting glucose	per mmol/L	1.12 (0.86–1.46)	0.39
Triglycerides	per 50 mg/dL	1.06 (0.94–1.20)	0.36
HDL cholesterol	per 10 mg/dL	0.77 (0.54–1.09)	0.14
LDL cholesterol	per 10 mg/dL	1.09 (0.99–1.20)	0.083
Alanine aminotransferase	per 10 U/L	1.39 (1.15–1.68)	**<0.001**

3.2 Final prediction model

The most parsimonious, best-fitting model retained three independent predictors: BMI, log-transformed ALT, and family history of dyslipidaemia (Table 2). Each contributed independently: BMI (aOR 1.81 per 5 kg/m², 95% CI 1.25–2.62), ALT (aOR 2.62 per doubling, 95% CI 1.67–4.10), and family history of dyslipidaemia (aOR 1.87, 95% CI 1.02–3.43). Adding waist circumference, lipids, or insulin resistance indices did not improve fitness. The predicted probability of MASLD is given by:

logit(p) = −8.198 + 0.119 × BMI (kg/m²) + 1.388 × ln [ALT (U/L)] + 0.626 × (family history of dyslipidaemia: 1 = yes, 0 = no)

Table 2. Final multivariable logistic regression model for ultrasound-defined MASLD, with the integer points assigned in the simplified score.

Predictor	Adjusted OR (95% CI)	P	Points
BMI (per 5 kg/m²)	1.81 (1.25–2.62)	**0.002**	—
ALT (per doubling)	2.62 (1.67–4.10)	**<0.001**	—
Family history of dyslipidaemia	1.87 (1.02–3.43)	**0.043**	1

3.3 Discrimination, calibration and internal validation

The model discriminated against children with and without MASLD with an apparent C-statistic of 0.741 (Figure 1). Calibration was checked with the intercept, slope, an assessment and the Hosmer–Lemeshow test. In the development dataset, the apparent calibration intercept was 0.00 and the apparent calibration slope was 1.00. These values come from the data that gave the model coefficients, so they may be overly optimistic. Internal validation used 1,000 bootstrap resamples to measure optimism in the model's performance. The bootstrap optimism for the C-statistic was 0.011, which lowered the corrected C-statistic to 0.730 from the 0.741. The bootstrap-corrected calibration slope was 0.95 showing slight overfitting and a fairly good maintenance of calibration after adjusting for optimism. The calibration graph displayed a match between the model's predictions and the actual outcomes over most risk levels though some differences appeared at the very highest predicted-risk levels (Figure 2). The Hosmer–Lemeshow test was significant ($p < 0.001$) showing a deviation from calibration when probabilities were split into deciles. Because this test is sensitive to the choice and number of risk groups and may detect relatively small departures from calibration, its result was considered together with the calibration plot and bootstrap-corrected calibration slope rather than interpreted in isolation. Overall, the model showed discrimination and generally acceptable calibration after correcting for optimism though some miscalibration was seen, especially at the very low or very high predicted risk. These results come from validation alone and do not prove that the model works in other paediatric population.

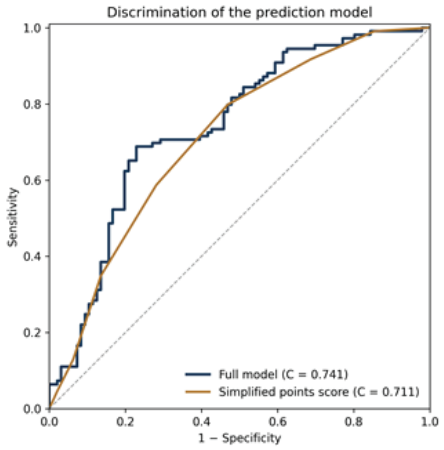


Figure 1. Receiver operating characteristic curves for the full prediction model and the simplified integer points score.

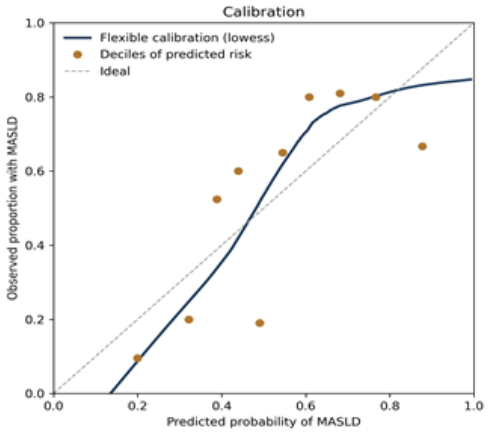


Figure 2. Calibration of the prediction model: flexible (lowess) calibration curve with deciles of predicted risk against the line of identity.

3.4 Simplified points score and risk stratification

Translation of the model into an integer score assigned points for BMI category, ALT category, and family history of dyslipidaemia (Table 3). The simplified score demonstrated moderate discrimination, with a C-statistic of 0.711, compared with 0.741 for the full regression model. Thus, conversion to the simplified score resulted in some loss of discriminative performance but retained substantial separation between participants with and without ultrasound-detected MASLD. Observed MASLD risk rose monotonically across the score, and three pragmatic strata—low (≤2 points), intermediate (3–4 points), and high (≥5 points)—separated children with observed MASLD prevalences of 30.1%, 60.5%, and 74.5%, respectively (Table 3). Because the study population consisted predominantly of children and adolescents already identified as having metabolic risk factors, these risk estimates represent observed risks within an enriched clinical population and should not be interpreted as absolute risks

applicable to the general paediatric population. In particular, the lower-risk category should be interpreted as a relatively lower-risk group rather than a group in whom MASLD has been excluded. The simplified score is therefore best regarded as a preliminary risk-stratification tool intended to support prioritization for further hepatic assessment rather than as a diagnostic instrument..

Table 3. Simplified points score allocation of points and observed MASLD risk by total score and by risk stratum.

Component/ stratum	Category	Points / observed
BMI (kg/m²)	< 25	0
	25–29.9	1
	30–34.9	2
	≥ 35	3
ALT (U/L)	< 25	0
	25–39	2
	40–59	3
	≥ 60	4
Family history of dyslipidaemia	yes	1
Risk stratum	Low (0–2 points)	30.1% (22/73)
	Intermediate (3–4 points)	60.5% (49/81)
	High (≥5 points)	74.5% (38/51)

3.5 Clinical Utility

Decision-curve analysis was performed to explore the potential clinical utility of the prediction model across a range of threshold probabilities (Figure 3). The model showed a higher net benefit than the default strategies of imaging all participants or imaging none across a broad range of threshold probabilities (approximately 0.2–0.8). However, these findings should be interpreted as an exploratory assessment of potential clinical utility rather than evidence of established clinical benefit.

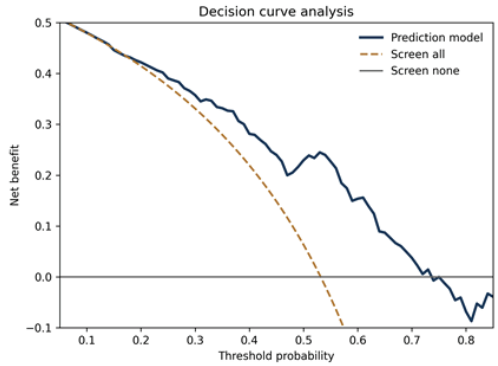


Figure 3. Decision curve analysis comparing the net benefit of the prediction model with screen-all and screen-none strategies across threshold probabilities.

4. Discussion

Using only variables collected during the routine assessment of childhood obesity, we developed and internally validated a three-predictor model BMI, ALT, and family history of dyslipidaemia) that predicted ultrasound-detected MASLD with acceptable discrimination (optimism-corrected C-statistic 0.730), satisfactory calibration, and demonstrable clinical net benefit. A simplified points score preserved most of this performance and stratified children into clinically meaningful risk groups, offering a practical triage tool for settings where imaging is scarce.

The three retained predictors are biologically and clinically coherent. Adiposity is the dominant driver of paediatric steatogenesis, and BMI captures this efficiently [2,3]; ALT, although an imperfect marker of steatosis, reflects hepatocellular involvement and is among the few liver-specific measures available in primary care [9]; and a family history of dyslipidaemia signals heritable cardiometabolic susceptibility that compounds individual risk [4]. That fasting glucose, insulin, and the lipid fractions did not enter the model is consistent with our parallel finding that insulin-resistance indices add little incremental discrimination once adiposity is accounted for, and with reports that such indices largely track body size in paediatric populations [10–12]. The model's reliance on a liver enzyme and an anthropometric measure, rather than on fasting insulin assays, is an advantage for low-resource deployment.

Our approach differs from and complements prior paediatric work. Whereas most studies evaluate single markers or insulin-resistance indices as stand-alone tests [10,13], we combined routine predictors into a calibrated multivariable model, subjected it to bootstrap internal validation, and assessed clinical utility by decision curve analysis steps recommended by the TRIPOD framework but uncommon in this literature [14,16]. The resulting discrimination is comparable to that of established paediatric metabolic risk tools and exceeds that of any single component, while the points score makes the model usable without a calculator. Importantly, decision curve analysis confirmed net benefit over the simplistic alternatives of imaging everyone or no one, which is the decision clinicians face when triaging at-risk children [16]. The study was conducted at a single tertiary-care centre in a clinically enriched population, with 86.8% of participants classified as obese and an overall prevalence of ultrasound-detected MASLD of 53.2%. Consequently, the observed risks associated with the score are likely to reflect the case mix and disease prevalence of the derivation cohort and should not be directly extrapolated to children in primary care, community settings, or populations with a different prevalence of metabolic risk

or MASLD. Although the use of BMI, ALT, and family history of dyslipidaemia makes the model potentially feasible in settings where these variables are routinely available, feasibility of measurement should not be equated with demonstrated clinical effectiveness or transportability. Future validation should include independent cohorts from multiple centres and, where possible, populations with different levels of metabolic risk and disease prevalence. Such studies should assess discrimination, calibration, and clinical utility and determine whether recalibration or modification of the model is required before implementation.

4.1 Strengths and limitations

Strengths include adherence to TRIPOD, a blinded outcome assessment, a favorable events-per-variable ratio, transformation of skewed predictors, formal internal validation with optimism correction, and evaluation of clinical utility rather than discrimination alone [14,16]. Limitations temper the conclusions. First, the reference standard was ultrasonography, which misses mild steatosis relative to magnetic resonance or biopsy; outcome misclassification would attenuate apparent performance. Second, the cohort was single-centre and almost uniformly overweight or obese, so the case mix is narrow, and the 53% prevalence far exceeds that of general paediatric populations—features that limit transportability and inflate predictive values. Third, only internal validation was performed; the optimism estimate, though small, cannot substitute for external testing, and the Youden-independent points thresholds require confirmation. Fourth, the borderline contribution of family history (upper CI near unity) suggests this predictor may not replicate in every setting. External validation in independent, more representative paediatric cohorts, ideally against quantitative imaging, is the essential next step before clinical adoption.

5. Conclusions

A parsimonious model combining BMI, ALT, and family history of dyslipidaemia predicted ultrasound-detected paediatric MASLD with acceptable discrimination, satisfactory calibration, and positive clinical net benefit, and was successfully simplified into a bedside points score with clear risk strata. Because the model uses routinely available variables, it has potential for use in resource-constrained settings; however, its feasibility, transportability, and clinical utility require evaluation in independent and representative paediatric populations before clinical implementation.

Declarations

Ethics approval and consent to participate. The study was approved by the Institutional Review Board of Bangladesh Medical University. Written informed consent was obtained from a parent or legal guardian, with child assent where appropriate.

Consent for publication. Not applicable; no individually identifiable data are presented.

Availability of data and materials. The de-identified dataset analyzed during the current study is available from the corresponding author on reasonable request.

Competing interests. The author declares no competing interests.

Funding. This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Author contributions. N.A.K. conceived and designed the study, supervised data collection and laboratory analyses, performed the statistical modelling, interpreted the data, and drafted and approved the final manuscript.

Acknowledgements. The author thanks the participating children and families, and the staff of the Departments of Paediatrics and Biochemistry & Molecular Biology at BMU.

References

1. Rinella ME, Lazarus JV, Ratziu V, Francque SM, Sanyal AJ, Kanwal F, et al. A multisociety Delphi consensus statement on new fatty liver disease nomenclature. Hepatology. 2023;78(6):1966–1986.
2. Accacha S, Barillas-Cerritos J, Srivastava A, Ross F, Drewes W, Gulkarov S, et al. From childhood obesity to metabolic dysfunction-associated steatotic liver disease (MASLD) and hyperlipidemia through oxidative stress during childhood. Metabolites. 2025;15(5):287.
3. Panganiban J, Kehar M, Ibrahim SH, Hartmann P, Sood S, Hassan S, et al. Metabolic dysfunction-associated steatotic liver disease (MASLD) in children with obesity: an Obesity Medicine Association (OMA) and expert joint perspective 2025. Obes Pillars. 2025; 14:100164.
4. Basil B, Myke-Mbata BK, Eze OE, Akubue AU. From adiposity to steatosis: metabolic dysfunction-associated steatotic liver disease, a hepatic expression of metabolic syndrome – current insights and future directions. Clin Diabetes Endocrinol. 2024;10(1):39.
5. Yodoshi T. Exploring non-invasive diagnostics and non-imaging approaches for pediatric metabolic dysfunction-associated steatotic liver disease. World J Gastroenterol. 2024;30(47):5070–5075.
6. Frączek J, Sowa A, Agopsowicz P, Migacz M, Dylińska-Kala K, Holecki M. Non-invasive tests as a replacement for liver biopsy in the assessment of MASLD. Medicina (Kaunas). 2025;61(4):736.

7. Lin YC, Liao FM, Chao HC, Chen AC, Jeng YM, Lin CC, et al. Consensus statement on metabolic dysfunction-associated steatotic liver disease in children and adolescents from the Joint TASL-TSPGHAN Expert Committee. JGH Open. 2025;9(6): e70137.
8. Mierzwa M, Malczyk Ż, Bik-Multanowski M, Brandt-Heunemann S, Flehmig B, Małecka-Tendera E, et al. High prevalence of metabolic-associated fatty liver disease (MAFLD) in children and adolescents with severe obesity. J Clin Med. 2025;14(10):3565.
9. Abdelhameed F, Kite C, Lagojda L, Dallaway A, Chatha KK, Chaggar SS, et al. Non-invasive scores and serum biomarkers for fatty liver in the era of metabolic dysfunction-associated steatotic liver disease (MASLD): a comprehensive review from NAFLD to MAFLD and MASLD. Curr Obes Rep. 2024;13(3):510–531.
10. Song K, Lee E, Lee HS, Youn YH, Baik SJ, Shin HJ, et al. Identification of pediatric MASLD using insulin resistance indices. JHEP Rep. 2025;7(7):101419.
11. Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. Homeostatic model assessment of insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. Diabetologia. 1985;28(7):412–419.
12. Bello-Chavolla OY, Almeda-Valdes P, Gómez-Velasco D, Viveros-Ruiz T, Cruz-Bautista I, Romo-Romo A, et al. METS-IR, a novel score to evaluate insulin sensitivity, is predictive of visceral adiposity and incident type 2 diabetes. Eur J Endocrinol. 2018;178(5):533–544.
13. Pecani M, Andreozzi P, Cangemi R, Corica B, Miglionico M, Romiti GF, et al. Metabolic syndrome and liver disease: re-appraisal of screening, diagnosis, and treatment through the paradigm shift from NAFLD to MASLD. J Clin Med. 2025;14(8):2750.
14. Collins GS, Reitsma JB, Altman DG, Moons KGM. Transparent reporting of a multivariable prediction model for individual prognosis or diagnosis (TRIPOD): the TRIPOD statement. BMJ. 2015;350: g7594.
15. Flahault A, Cadilhac M, Thomas G. Sample size calculation should be performed for design accuracy in diagnostic test studies. J Clin Epidemiol. 2005;58(8):859–862.
16. Vickers AJ, Elkin EB. Decision curve analysis: a novel method for evaluating prediction models. Med Decis Making. 2006;26(6):565–574.
17. Sullivan LM, Massaro JM, D'Agostino RB Sr. Presentation of multivariate data for clinical use: the Framingham Study risk score functions. Stat Med. 2004;23(10):1631–1660.