Research Square

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Clinical characteristics and factors associated with insulin resistance in children with primary hypertension: a retrospective study

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Abstract

Objectives

To investigate the clinical characteristics and factors associated with insulin resistance (IR) in children with primary hypertension.

Methods

We retrospectively analyzed 612 children aged 10–17 years who were newly diagnosed with primary hypertension at the Capital Center for Children's Health, Capital Medical University. Based on the homeostatic model assessment of insulin resistance (HOMA-IR), participants were classified into the non-IR group (n = 143) and the IR group (n = 469), and their clinical data were compared. Multivariable logistic regression was performed to identify factors independently associated with IR. A nomogram was developed based on the final multivariable model and internally validated using 1000 bootstrap resamples. Multivariable linear regression with log-transformed HOMA-IR was performed as a sensitivity analysis.

Results

IR was identified in 469 of 612 children (76.6%). Compared with the non-IR group, the IR group had higher BMI, a higher prevalence of obesity and stage 2 hypertension, higher levels of fasting blood glucose, fasting insulin, uric acid, total cholesterol, triglycerides, low-density lipoprotein cholesterol, and non-high-density lipoprotein cholesterol, a higher 24-h systolic blood pressure index, and a higher prevalence of cardiac damage. However, age and levels of high-density lipoprotein cholesterol and homocysteine were lower in the IR group (all $p < 0.05$). Multivariable logistic regression showed that younger age (OR = 0.74, 95% CI: 0.64 to 0.86), obesity (OR = 6.08, 95% CI: 3.64 to 10.13), and higher triglyceride levels (OR = 2.62, 95% CI: 1.47 to 4.67) were independently associated with IR in children with primary hypertension (all $p < 0.05$). The nomogram showed good discrimination (AUC = 0.829, 95% CI: 0.789–0.869), with a bias-corrected C-index of 0.823 after bootstrap validation. Calibration curves and decision curve analysis (DCA) indicated good calibration and potential clinical net benefit. The sensitivity analysis yielded consistent results.

Conclusions

IR was common among children aged 10–17 years with primary hypertension. Younger age, obesity, and higher triglyceride levels were independently associated with IR. The nomogram may help identify children with primary hypertension who are at high risk for IR, providing a convenient tool for clinical screening.

1. Introduction

Pediatric primary hypertension is a multifactorial condition associated with obesity, adverse childhood experiences, and unhealthy lifestyle behaviors [1]. The prevalence of sustained hypertension is approximately 3.89% among children and increases to 16.35% among those with obesity, underscoring its growing importance as a global public health concern [2]. Its pathophysiology involves multiple interacting mechanisms, including sympathetic nervous system overactivity, vascular remodeling, increased salt sensitivity, dysregulation of the renin–angiotensin–aldosterone system, and metabolic disturbances [3]. Insulin resistance (IR) is an important feature of the metabolic disturbances. It may contribute to elevated blood pressure through sympathetic activation, increased renal sodium reabsorption, and endothelial dysfunction, and is associated with a range of cardiovascular and metabolic complications [4–6].

IR is characterized by reduced tissue responsiveness to insulin, resulting in compensatory hyperinsulinemia. The homeostatic model assessment of insulin resistance (HOMA-IR) is a widely used surrogate index for assessing IR in children and has demonstrated good correlation with the hyperinsulinemic–euglycemic clamp, the reference method for evaluating insulin sensitivity [7–9]. However, HOMA-IR requires fasting insulin measurement, which may increase testing costs and limit its accessibility in primary healthcare settings. Although IR has been associated with an increased risk of hypertension in adults [10], IR-related endothelial dysfunction and atherosclerotic processes may begin during childhood [6]. Therefore, early identification of IR is important in the management of children with hypertension. Our study aimed to investigate the clinical characteristics and factors associated with IR in children aged 10–17 years with primary hypertension, thereby providing a basis for the early identification of IR and individualized intervention.

2. Materials and Methods

2.1 Study Design and Participants

This retrospective study was conducted in the Department of Pediatric Cardiology, Capital Center for Children's Health, Capital Medical University. We included 612 children newly diagnosed with primary hypertension between January 2015 and December 2024. The inclusion criteria were as follows: (1) age 10–17 years; (2) diagnosis and staging of hypertension according to the 2018 Chinese Guidelines for Prevention and Treatment of Hypertension [11]; and (3) availability of the clinical data required for this analysis. The exclusion criteria were as follows: (1) secondary hypertension; (2) organic heart disease, acute infectious disease, genetic disease, metabolic disease, and immune disease; and (3) previous use of antihypertensive medication. The study was approved by the Ethics Committee of the Capital Institute of Pediatrics (No. SHERLL2025030).

2.2 General Clinical Information and Blood Pressure Measurements

Age, sex, family history of hypertension, stage of hypertension, and body mass index (BMI) were recorded. Obesity was defined as BMI ≥ the 95th percentile for age and sex [12]. Office blood pressure was measured using a standard mercury sphygmomanometer. Ambulatory blood pressure monitoring (ABPM) was performed using a noninvasive ambulatory blood pressure monitor, with measurements scheduled every 30 min from 09:00 to 21:00 and every 60 min from 21:00 to 09:00 the following day. The recorded variables included 24-h, daytime, and nighttime systolic blood pressure (SBP) and diastolic blood pressure (DBP). The 24-h SBP index was calculated as the 24-h SBP divided by the 95th percentile SBP; the 24-h DBP index was calculated in the same way using the 95th percentile DBP.

2.3 Laboratory Measurements and Definition of Insulin Resistance

Morning fasting venous blood samples were analyzed using a Siemens ADVIA 2400 automated biochemical analyzer to measure total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), uric acid (UA), fasting blood glucose (FBG), and homocysteine (Hcy). Fasting insulin (FINS) was measured using a Roche Cobas E601 automated electrochemiluminescence immunoassay analyzer. non-HDL-C was calculated as TC minus HDL-C. HOMA-IR was calculated as FBG (mmol/L) × FINS (mU/L)/22.5. IR was defined as HOMA-IR > 3 [13]. Participants were subsequently classified into the non-IR and IR groups according to this threshold.

2.4 Assessment of Target Organ Damage (TOD)

Cardiac damage was assessed by echocardiography. Left ventricular mass was calculated using the Devereux formula, and the left ventricular mass index (LVMI) and relative wall thickness (RWT) were subsequently calculated. Left ventricular hypertrophy was defined by either sex-specific LVMI thresholds (≥ 37.08 g/m² for boys or ≥ 34.02 g/m² for girls) or RWT > 0.36 [14, 15]. Renal damage was defined as estimated glomerular filtration rate < 90 mL/min/1.73 m² or urinary albumin-to-creatinine ratio ≥ 30 mg/g [16]. Fundus abnormalities were classified according to the Keith–Wagener system [17]. Increased carotid intima-media thickness (CIMT) was defined as CIMT ≥ 0.55 mm in children aged 10–18 years [18].

2.5 Statistical Analysis

Statistical analyses were performed using SPSS version 30.0 and R version 4.5.2. Continuous variables that were approximately normally distributed were presented as mean ± standard deviation and compared using independent-samples t-tests; non-normally distributed continuous variables were presented as median (interquartile range) and compared using the Mann–Whitney U test. Categorical variables were presented as n (%) and compared using chi-square tests.

Spearman correlation analysis was used to assess the relationships between HOMA-IR and continuous clinical variables. To reduce multicollinearity, variables with strong pairwise correlations (|r| ≥ 0.70) or a variance inflation factor > 5 were excluded from the multivariable candidate set. Univariable logistic regression analyses were performed first, followed by multivariable logistic regression to identify factors

independently associated with IR among children with primary hypertension. A nomogram was developed based on the final multivariable model. Internal validation was performed using 1000 bootstrap resamples. The bias-corrected C-index was used to assess discrimination, calibration curves were used to evaluate agreement between predicted and observed probabilities, and decision curve analysis (DCA) was used to examine net benefit. As a sensitivity analysis, HOMA-IR was natural log-transformed and analyzed as a continuous outcome in multivariable linear regression. A two-sided p value < 0.05 was considered statistically significant.

3. Results

3.1. Clinical and laboratory characteristics of the non-IR and IR groups

A total of 612 children with primary hypertension were included, of whom 483 (78.9%) were male. The median age was 13 (11, 14) years and the median BMI was 27.78 (24.21, 31.52) kg/m². Overall, 73.4% of the children had obesity, and 60.5% had stage 2 hypertension. IR was identified in 469 children (76.6%), while the remaining 143 were classified as non-IR. Compared with the non-IR group, the IR group had higher BMI, a higher prevalence of obesity and stage 2 hypertension, and higher levels of FBG, FINS, UA, TC, TG, LDL-C, and non-HDL-C. Age and levels of HDL-C and Hcy were lower in the IR group. Sex and family history of hypertension did not differ significantly between the groups (Table 1).

Table 1
Clinical and laboratory characteristics of the non-IR and IR groups.

Variable	non-IR (n = 143)	IR (n = 469)	*p Value*
Age, years	14.00 (13.00, 15.00)	13.00 (12.00, 14.00)	< 0.001
BMI, kg/m²	23.35 (20.77, 26.75)	29.00 (25.69, 32.24)	< 0.001
Male, n (%)	117 (81.82)	366 (78.04)	0.332
Family history of hypertension, n (%)	119 (83.22)	390 (83.16)	0.986
Obesity, n (%)	51 (35.66)	398 (84.86)	< 0.001
Stage 2 hypertension, n (%)	73 (51.05)	297 (63.33)	0.009
FBG, mmol/L	4.50 (4.15, 4.69)	4.63 (4.35, 4.98)	< 0.001
FINS, mU/L	11.10 (8.90, 12.95)	25.50 (19.90, 35.10)	< 0.001
UA, μmol/L	431.00 (369.50, 477.00)	458.00 (395.00, 529.00)	< 0.001
TC, mmol/L	3.75 (3.33, 4.20)	3.94 (3.52, 4.42)	0.014
TG, mmol/L	0.93 (0.74, 1.26)	1.29 (0.94, 1.74)	< 0.001
HDL-C, mmol/L	1.13 (0.96, 1.29)	1.01 (0.88, 1.16)	< 0.001
LDL-C, mmol/L	2.35 (1.99, 2.90)	2.66 (2.23, 3.16)	< 0.001
non-HDL-C, mmol/L	2.64 (2.21, 3.01)	2.89 (2.44, 3.37)	< 0.001
Hcy, μmol/L	11.24 (9.32, 16.76)	10.02 (8.28, 13.32)	< 0.001

IR, insulin resistance; non-IR, no insulin resistance; BMI, body mass index; FBG, fasting blood glucose; FINS, fasting insulin; UA, uric acid; TC, total cholesterol; TG, triglycerides; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; non-HDL-C, non-high-density lipoprotein cholesterol; Hcy, homocysteine.

3.2. Blood pressure measurements and prevalence of TOD in the non-IR and IR groups

The IR group had a higher 24-h SBP index and a higher prevalence of cardiac damage than the non-IR group. There were no statistically significant differences between groups in 24-h SBP/DBP, daytime SBP/DBP, nighttime SBP/DBP, the 24-h DBP index, or overall TOD, including renal damage, fundus abnormalities, and increased CIMT (Table 2).

Table 2
Blood pressure measurements and prevalence of TOD in the non-IR and IR groups.

Variable	non-IR (n = 143)	IR (n = 469)	*p* Value
24-h SBP, mmHg	131.00 (125.00, 136.50)	132.00 (125.00, 139.00)	0.083
24-h DBP, mmHg	73.00 (69.00, 78.00)	73.00 (69.00, 79.00)	0.653
Daytime SBP, mmHg	133.00 (126.50, 139.50)	135.00 (127.00, 142.00)	0.065
Daytime DBP, mmHg	75.00 (69.00, 80.00)	75.00 (70.00, 81.00)	0.250
Nighttime SBP, mmHg	125.00 (119.00, 130.00)	126.00 (117.00, 133.00)	0.148
Nighttime DBP, mmHg	68.00 (64.00, 72.00)	68.00 (63.00, 74.00)	0.968
24-h SBP index	1.01 (0.97, 1.05)	1.03 (0.98, 1.09)	< 0.001
24-h DBP index	0.92 (0.86, 0.98)	0.94 (0.87, 1.00)	0.148
TOD, n (%)	64 (44.76)	249 (53.09)	0.081
Cardiac damage, n (%)	25 (17.48)	123 (26.23)	0.033
Renal damage, n (%)	35 (24.48)	144 (30.70)	0.152
Fundus abnormalities, n (%)	13 (9.09)	31 (6.61)	0.315
Increased CIMT, n (%)	8 (5.59)	28 (5.97)	0.867

IR, insulin resistance; non-IR, no insulin resistance; SBP, systolic blood pressure; DBP, diastolic blood pressure; TOD, target organ damage; CIMT, carotid intima-media thickness.

3.3. Correlations between HOMA-IR and clinical variables

Spearman correlation analysis showed that HOMA-IR was positively correlated with BMI, UA, TC, TG, LDL-C, non-HDL-C, 24-h SBP, daytime SBP, daytime DBP, nighttime SBP, the 24-h SBP index, and the 24-h DBP index. HOMA-IR was negatively correlated with age, HDL-C, and Hcy (all $p < 0.05$).

3.4. Univariable and multivariable logistic regression analyses of IR in children with primary hypertension

After strongly correlated or collinear variables were excluded, age, sex, family history of hypertension, obesity, stage 2 hypertension, UA, TG, HDL-C, non-HDL-C, Hcy, 24-h SBP index, and cardiac damage were evaluated using univariable logistic regression. Variables with $p < 0.05$ were subsequently entered into the multivariable model. After adjustment for sex and family history, age, obesity, and TG remained statistically significant (Table 3).

Table 3
Univariable and multivariable logistic regression analyses of IR in children with primary hypertension.

Analysis	Variable	OR	95% CI	*p* value
Univariable	Age	0.68	0.60–0.76	< 0.001
	Sex (female)	1.27	0.79–2.04	0.333
	Family history of hypertension	1.00	0.60–1.64	0.986
	Obesity	10.11	6.61–15.47	< 0.001
	Stage 2 hypertension	1.66	1.13–2.42	0.009
	UA	1.01	1.00–1.01	< 0.001
	TG	4.47	2.82–7.08	< 0.001
	HDL-C	0.14	0.06–0.31	< 0.001
	non-HDL-C	1.77	1.32–2.37	< 0.001
	Hcy	0.98	0.96–0.99	0.019
	24-h SBP index	66.37	5.79–760.99	< 0.001
	Cardiac damage	1.68	1.04–2.71	0.034
Multivariable	Sex (female)	1.17	0.62–2.21	0.628
	Family history of hypertension	1.25	0.69–2.28	0.460
	Age	0.74	0.64–0.86	< 0.001
	Obesity	6.08	3.64–10.13	< 0.001
	TG	2.62	1.47–4.67	0.001

IR, insulin resistance; UA, uric acid; TG, triglycerides; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; non-HDL-C, non-high-density lipoprotein cholesterol; Hcy, homocysteine.

3.5. Nomogram development and internal validation

A nomogram based on the final multivariable logistic regression model was developed to estimate the probability of IR in children with primary hypertension (Fig. 1). The model had an apparent AUC of 0.829 (95% CI: 0.789–0.869), with a sensitivity of 74.1% and a specificity of 79.5% (Fig. 2a). Internal validation using 1000 bootstrap resamples yielded a bias-corrected C-index of 0.823, indicating good discrimination. The calibration curve showed good agreement between the predicted and observed probabilities (Fig. 2b). DCA showed positive net benefit across a relatively broad range of threshold probabilities, suggesting potential clinical utility (Fig. 2c).

3.6. Sensitivity analysis

In the sensitivity analysis, HOMA-IR was natural log-transformed and analyzed as a continuous outcome in multivariable linear regression. Age (β = −0.08), obesity (β = 0.47), and TG (β = 0.21) remained independently associated with log-transformed HOMA-IR (all $p < 0.001$). The directions of these associations were consistent with the primary logistic regression analysis.

4. Discussion

Pediatric primary hypertension is often accompanied by metabolic disturbances and blood pressure tends to track from childhood into adulthood, increasing the risk of later cardiovascular events [19]. With the increasing prevalence of childhood obesity, IR has become an important concern, although its reported prevalence varies across studies [6]. Previous evidence has linked IR to increased cardiometabolic risk, particularly among adolescents with obesity [20]. Therefore, identifying simple and effective indicators for IR screening is of considerable clinical importance. In this study of children aged 10–17 years with primary hypertension, IR was common. Younger age, obesity, and higher TG levels were independently associated with IR, and the resulting nomogram showed good discrimination and calibration on internal validation.

Previous studies have reported that the prevalence of IR among children ranges from 3.1% to 44% [21]. In our study, IR prevalence was 76.6%, indicating a substantial burden among children with primary hypertension. This difference may reflect variations in study populations, underlying metabolic risk, and the diagnostic thresholds used to define IR. Puberty is a critical physiological period for changes in insulin sensitivity. Increases in growth hormone and sex hormone levels during puberty may contribute to transient physiological IR, which may be exacerbated by obesity and unhealthy lifestyle behaviors [6]. Using data from the Korean National Health and Nutrition Examination Survey, Kim et al. [22] reported age-related variation in IR, with IR peaking at 12–13 years in boys and 11–13 years in girls. A life-course study of individuals aged 5–80 years found that IR increased during puberty and that recovery in insulin sensitivity could extend into adulthood [23]. In our study, children with IR were younger, and age was negatively correlated with HOMA-IR. These findings suggest that younger age may be associated with a higher likelihood of IR among children with primary hypertension. However, because pubertal stage was not directly assessed, the contribution of pubertal development to this association remains uncertain.

Obesity-associated IR is considered an important link in the progression from metabolic dysfunction to elevated blood pressure [5]. In the present study, children with IR had a higher BMI and a higher prevalence of obesity, and HOMA-IR was positively correlated with BMI. Obesity may promote IR by activating inflammatory pathways, including nuclear factor-κB (NF-κB) and c-Jun N-terminal kinase (JNK) signaling, thereby contributing to adipose tissue inflammation and impaired insulin signaling [24]. Children with IR also exhibited higher UA levels and a less favorable lipid profile, characterized by higher TC, TG, LDL-C, and non-HDL-C levels and lower HDL-C levels. These findings, together with the positive correlations between HOMA-IR and several metabolic parameters, indicate a greater burden of metabolic

abnormalities in hypertensive children with IR. Previous studies by Chung et al. [25] and Cheng et al. [26] similarly linked IR to an atherogenic lipoprotein profile and hyperuricemia, respectively. Mechanistically, triglyceride-rich lipoproteins can release free fatty acids through lipolysis. Excessive fatty acid accumulation in non-adipose tissues, including the liver and skeletal muscle, may impair insulin signaling through lipotoxic mechanisms and further aggravate IR [27]. Altered purine metabolism and reduced renal uric acid excretion may also contribute to elevated UA levels in individuals with IR [26]. Collectively, these findings support the importance of screening for IR in children with primary hypertension to identify those at increased risk and facilitate timely, individualized intervention.

IR may also be associated with blood pressure abnormalities in children with primary hypertension. A Mendelian randomization study of Chinese children aged 6–18 years found that BMI may indirectly contribute to higher blood pressure and an increased risk of hypertension by increasing insulin levels, suggesting that insulin may partly mediate the association between BMI and hypertension [28]. In our study, the IR group had a higher prevalence of stage 2 hypertension and a higher 24-h SBP index than the non-IR group, whereas other ambulatory blood pressure measures did not differ significantly between groups. These findings suggest that IR may be predominantly associated with relative SBP elevation rather than a consistent difference across all ambulatory blood pressure measures. Arterial stiffness has been proposed as one potential link between IR and cardiovascular risk [29]. In adolescents, Agbaje reported that body composition and IR mediated part of the association between arterial stiffness and blood pressure [30]. In a subsequent 7-year follow-up study, the same research group reported that SBP and IR partially mediated the association between arterial stiffness and left ventricular hypertrophy [31]. These studies offer a possible context for our findings, but our retrospective data cannot establish the direction or causality of the relationship between IR and blood pressure.

Although cardiovascular events are uncommon in children with primary hypertension, TOD is not rare. In our study, cardiac damage was more prevalent in the IR group, whereas the overall prevalence of TOD and the prevalence of other individual forms of TOD did not differ significantly between groups. The absence of statistically significant differences in some outcomes may reflect limited statistical power, particularly for less frequent forms of damage. The relationship between IR and TOD remains incompletely understood. Studies in adults have linked IR to adverse cardiac structure and function [32–34]. Among adolescents with blood pressure between the 75th and 95th percentiles, IR was associated with diastolic dysfunction independently of blood pressure and other metabolic factors [35]. A study of adolescents with obesity also reported an association between IR and higher CIMT [25]. In contrast, the ALSPAC cohort study, which followed 1,779 healthy adolescents for 9 years, found no association between IR and CIMT progression [36]. Animal studies have shown that prolonged exposure to high insulin levels may induce pathological changes in target organs, including glomerular basement membrane thickening, mesangial expansion, and hepatic lipid accumulation [37]. These observations provide biological plausibility but do not show that IR directly causes TOD in hypertensive children. Differences in study populations, IR definitions, and methods of assessing TOD may also explain variation across studies. Given the higher prevalence of cardiac damage in our IR group, careful clinical assessment is warranted.

In multivariable logistic regression, younger age, obesity, and higher TG levels were independently associated with IR in children aged 10–17 years with primary hypertension. The sensitivity analysis, in which HOMA-IR was analyzed as a continuous outcome after natural log transformation, yielded associations in the same directions, supporting the robustness of these findings. Evidence that early-life metabolic disturbances may have persistent effects on insulin sensitivity and blood pressure further underscores the importance of identifying metabolic risk during childhood [38, 39]. However, our retrospective design cannot establish when IR developed or how risk accumulated over time. We also developed a nomogram based on the multivariable logistic regression model to estimate the probability of IR. The model demonstrated good discrimination and calibration in internal validation, while DCA suggested a potential net benefit across a range of threshold probabilities. The model may help clinicians identify children who are more likely to have IR using routinely available clinical information and prioritize further metabolic assessment. However, the nomogram should be regarded as a preliminary risk assessment tool rather than a substitute for biochemical evaluation. Where fasting insulin testing is not readily available, the nomogram may help inform decisions about further assessment in the context of the individual child and local practice. External validation is necessary before routine clinical use can be recommended.

This study has several limitations. First, its single-center retrospective design limits causal inference regarding the relationship between primary hypertension and IR in children. Second, although a HOMA-IR cutoff of > 3 is commonly used to define IR, HOMA-IR varies with age and ethnicity, and no universally accepted diagnostic threshold for IR in children has been established. Third, the nomogram underwent internal validation only and has not been externally validated; its clinical applicability therefore requires further evaluation. Future multicenter prospective cohort studies are needed to validate these findings and assess the effects of targeted interventions on IR, blood pressure, and TOD progression in children with primary hypertension.

5. Conclusions

In this study of children aged 10–17 years with primary hypertension, IR was common. Younger age, obesity, and higher triglyceride levels were independently associated with IR. The nomogram showed promising performance in internal validation and may help identify children who warrant further assessment for IR. External validation is needed before its clinical utility can be established.

Abbreviations

The following abbreviations are used in this manuscript:

ABPM	Ambulatory blood pressure monitoring
AUC	Area under the curve
BMI	Body mass index
CI	Confidence interval
CIMT	Carotid intima-media thickness
DBP	Diastolic blood pressure
DCA	Decision curve analysis
FBG	Fasting blood glucose
FINS	Fasting insulin
HDL-C	High-density lipoprotein cholesterol
Hcy	Homocysteine
HOMA-IR	Homeostatic model assessment of insulin resistance
IR	Insulin resistance
JNK	c-Jun N-terminal kinase
LDL-C	Low-density lipoprotein cholesterol
LVMI	Left ventricular mass index
NF-κB	Nuclear factor kappa B
non-HDL-C	Non-high-density lipoprotein cholesterol
non-IR	No insulin resistance
OR	Odds ratio
ROC	Receiver operating characteristic
RWT	Relative wall thickness
SBP	Systolic blood pressure
TC	Total cholesterol
TG	Triglycerides
TOD	Target organ damage
UA	Uric acid

Declarations

Ethics approval and consent to participate

The study was conducted in accordance with the Declaration of Helsinki and was approved by the Ethics Committee of the Capital Institute of Pediatrics (No. SHERLL2025030). Written informed consent was obtained from the participants' parents or guardians.

Consent for publication

Not applicable.

Availability of data and materials

The data presented in this study are available from the corresponding author upon reasonable request. They are not publicly available because of institutional restrictions.

Competing interests

The authors declare that they have no competing interests.

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Author Contributions

Yao Li collected the data, performed the statistical analyses, and drafted the manuscript. Yaqi Li, Chang Wu, and Tingjuan Lin contributed to data collection and statistical analysis. Yao Lin, He Jiang, and Lin Shi reviewed and revised the manuscript. Yao Lin and Lin Shi conceived and supervised the study, oversaw quality control, and critically revised the manuscript. All authors have read and agreed to the published version of the manuscript.

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Figures

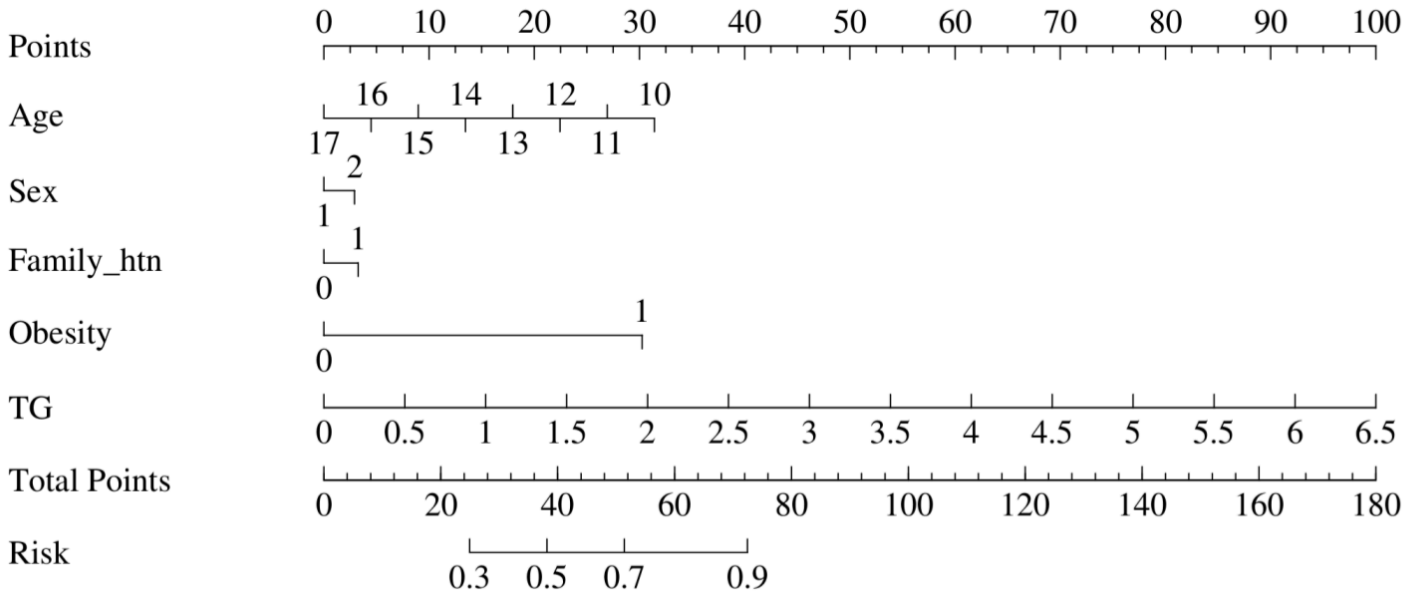
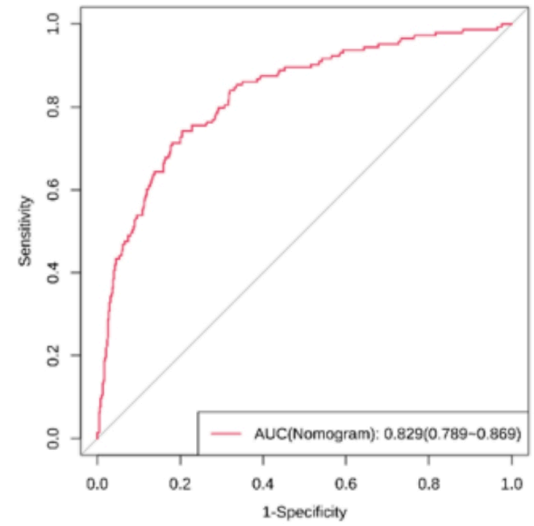
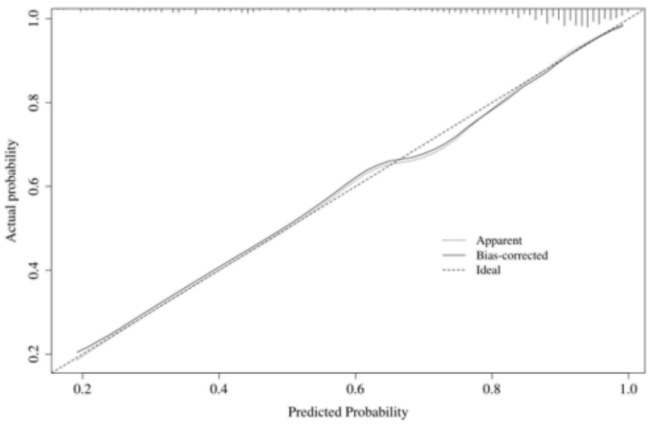


Figure 1

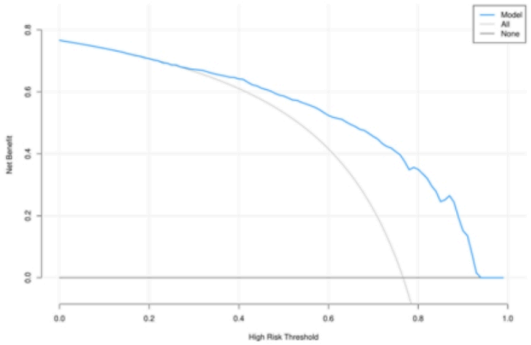
Nomogram for estimating the probability of IR in children with primary hypertension



(**a**)



(**b**)



(c)

Figure 2

Internal validation of the nomogram: (**a**) receiver operating characteristic (ROC) curve; (**b**) calibration curve; and (**c**) decision curve analysis. AUC, area under the curve.