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ORIGINAL ARTICLE

Red cell distribution width-to-platelet ratio (RPR) as a simple non-invasive predictor of liver cirrhosis in patients with chronic hepatitis B: a cross-sectional study

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ABSTRACT

Aim Chronic hepatitis B (CHB) is a major cause of liver cirrhosis worldwide. This study aimed to evaluate the red cell distribution width-to-platelet ratio (RPR) as a simple non-invasive predictor of cirrhosis in CHB patients.

Methods A cross-sectional study was conducted in 221 CHB patients at H. Adam Malik General Hospital, Medan, Indonesia (May 2024–May 2025). Cirrhosis was diagnosed by FibroScan (≥ 14 kPa) or ultrasonography findings. RPR was calculated from routine haematology data. Bivariate analysis was used to compare the cirrhotic and non-cirrhotic groups. ROC curve analysis was used to evaluate the diagnostic accuracy of RPR and determine the optimal cut-off values in terms of sensitivity and specificity.

Results Of 221 patients, 64 (29.0%) had cirrhosis. Cirrhotic patients were older (54.5 vs. 48 years; $p = 0.001$) and predominantly male (77.6% vs. 56.1%; $p = 0.004$). RPR was significantly higher in the cirrhotic group (0.16 vs. 0.05; $p < 0.001$). ROC analysis showed that RPR had an AUC of 0.938 (95% CI: 0.906–0.970; $p < 0.001$), with an optimal cut-off value of 0.085, yielding sensitivity of 87.5% and specificity of 87.3% for detecting cirrhosis.

Conclusion RPR is a simple, inexpensive, and widely available haematological index with excellent diagnostic accuracy for predicting cirrhosis in CHB patients. It may serve as an effective screening tool in settings with limited access to advanced diagnostic modalities.

Keywords: erythrocyte indices, hepatitis B, chronic, liver cirrhosis, platelet count

INTRODUCTION

Hepatitis B, an infection caused by the Hepadnavirus, is a major cause of acute and chronic hepatitis that can progress to cirrhosis and hepatocellular carcinoma (HCC) (1). Globally, more than 296 million people are living with chronic hepatitis B (CHB), with about 1.5 million new cases and 820,000 deaths each year, mainly due to complications such as liver cirrhosis and HCC (2,3). The prevalence of CHB varies across regions, with the highest rates found in East Asia and Sub-Saharan Africa (2). In Indonesia, the prevalence of CHB decreased from 9.4% in 2007 to 7.1% in 2013, reflecting the effectiveness of universal hepatitis B immunization in newborns. Nevertheless, vaccination coverage remains uneven, particularly in Eastern Indonesia, and the prevalence of HBsAg positivity among pregnant women is still high, contributing to ongoing perinatal transmission (4).

Chronic CHB leads to liver fibrosis, which can progress to cirrhosis and hepatocellular carcinoma (HCC). Fibrosis progression is influenced by CHB clinical phase, HBsAg clearance, age, the presence of metabolic syndrome or fatty liver, and baseline liver stiffness (5). Inflammation induces activation of Kupffer cells and the release of proinflammatory cytokines, which activate hepatic stellate cells (HSC). These cells subsequently transform into myofibroblast-like cells and produce excessive extracellular matrix, leading to fibrosis (6). Advanced fibrosis disrupts the normal hepatic architecture through scar tissue and regenerative nodule formation, a condition known as liver cirrhosis. Hepatitis B is reported to be the most common cause of liver cirrhosis in Indonesia, accounting for 37.3–73.7% of cases (7).

The gold standard for diagnosing fibrosis is liver biopsy, but the procedure is invasive and expensive (3). Non-invasive alternatives, such as elastography, remain limited, especially in

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primary care, and can be affected with some technical factors such as obesity and ascites (8,9). Therefore, simple non-invasive laboratory tests such as routine haematology and liver function test are often considered. Blood-based non-invasive index such as aspartate-to-platelet ratio index (APRI) and FIB-4 score have been proven to have a good diagnostic value (10).

Thrombocytopenia is a common manifestation of chronic hepatitis B, caused by hypersplenism, reduced thrombopoietin production, impaired haematopoiesis, and immune-mediated destruction (6,11). Platelet indices derived from complete blood count, such as mean platelet volume (MPV), platelet distribution width (PDW), platelecrit (PCT), platelet-to-lymphocyte ratio (PLR), and red cell distribution width-to-platelet ratio (RPR), are associated with inflammatory processes and the progression of liver fibrosis (11–13). Previous studies have reported significant associations of MPV, PDW, and platelet-large cell ratio (P-LCR) with liver fibrosis (13).

This study aimed to evaluate the red cell distribution width-to-platelet ratio (RPR) as a simple non-invasive predictor of cirrhosis in CHB patients.

MATERIAL/PATIENTS AND METHODS

Patients and study design

This single-centre, cross-sectional observational study was conducted at H. Adam Malik General Hospital, Medan, Indonesia. The study followed the STROBE reporting guideline for cross-sectional studies. Data were collected between May 2024 and May 2025.

The study included 221 adult patients diagnosed with chronic hepatitis B (CHB), defined as hepatitis B surface antigen (HBsAg) positivity for more than six months.

Inclusion criteria were: age ≥ 18 years, complete laboratory data (complete blood count, alanine aminotransferase - ALT, aspartate aminotransferase - AST, red cell distribution width - RDW), and availability of FibroScan or abdominal ultrasonography results. Patients were excluded if they had co-infections with hepatitis C, hepatitis D, or HIV, other chronic liver diseases (e.g., hemochromatosis, alcoholic liver disease, drug-induced liver injury), hepatocellular carcinoma or other malignancies, or a history of liver transplantation.

This study was approved by the Health Research Ethical Committee of Universitas Sumatera Utara and H. Adam Malik General Hospital (Approval No: 871/KEPK/USU/2025). All personal identifiers were removed to ensure confidentiality.

Methods

Demographic, clinical, and laboratory data were collected from hospital medical records. Laboratory parameters were measured using standard automated haematology and biochemistry analysers in the hospital's central laboratory. FibroScan examinations were performed by trained hepatologists, while abdominal ultrasonography was performed by experienced radiologists using standard protocols. To reduce measurement bias, only patients with complete clinical, laboratory, and imaging data were included. Misclassification bias was minimized by using standardized diagnostic criteria for cirrhosis (FibroScan cut-off ≥ 14 kPa or typical ultrasonography features).

The primary outcome was liver cirrhosis, defined as liver stiffness ≥ 14 kPa on FibroScan or ultrasonography findings consistent with cirrhosis (coarse echotexture, nodular surface, altered lobe morphology, splenomegaly, or portal vein dilatation). The primary predictor variable was red cell distribution width-to-platelet ratio (RPR), calculated as RDW (%) divided by platelet count (10 /L). Other variables included age, sex, HBeAg status, platelet indices (mean platelet volume - MPV, platelet distribution width - PDW, plateletcrit - PCT, platelet-to-lymphocyte ratio - PLR, platelet-to-monocyte ratio - PMR), aspartate aminotransferase-to-platelet ratio index - APRI, and FIB-4 score.

Statistical analysis

Continuous variables (e.g., RPR, APRI, FIB-4, platelet indices) were analysed as continuous measures, expressed as means with standard deviations (SD) or median with interquartile ranges (IQR), depending on data distribution. No arbitrary categorization was applied, except when calculating receiver operating characteristic (ROC) curve cut-offs. Descriptive statistics were used to summarize baseline characteristics. Bivariate analyses were performed to compare cirrhotic and non-cirrhotic groups using χ^2 tests for categorical variables, and independent t-test or Mann–Whitney U test for continuous variables, depending on normality. ROC curve analysis was performed to evaluate the diagnostic accuracy of RPR, calculating the area under the curve (AUC), sensitivity, specificity, and Youden index for optimal cut-off values. The DeLong method for correlated ROC curves was used to compare the AUC values between RPR, APRI, and FIB-4 pairwise. The easyROC web-based tool (version 1.3.1; Hacettepe University, Ankara, Türkiye) was used to perform ROC analyses and comparative AUC testing. A $p < 0.05$ was considered statistically significant. Missing data were excluded from analysis.

RESULTS

During the study period (May 2024–May 2025), 245 patients with chronic hepatitis B were screened for eligibility. A total of 24 patients were excluded: 10 due to incomplete laboratory data, six with hepatitis C or HIV co-infection, and eight with hepatocellular carcinoma leaving 221 patients who fulfilled the eligibility criteria and were included in the analysis (Figure 1). No patients were lost to follow-up, as this was a cross-sectional study.

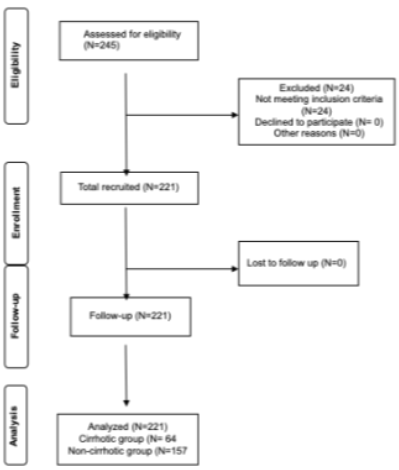

Figure 1. Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) flowchart

Of the 221 patients, 64 (29.0%) were diagnosed with cirrhosis and 157 (71.0%) were non-cirrhotic. The cirrhotic group had a higher median age compared to the non-cirrhotic group (54.5 vs. 48 years; p = 0.001) and consisted predominantly of males (77.6% vs. 56.1%; p = 0.004). Most patients were hepatitis B e-antigen (HBeAg)-negative in both groups, with no significant difference (p > 0.05). (Table 1). No missing data were reported for the main variables analysed (age, sex, HBeAg status, haematological parameters, APRI, FIB-4, RPR). Among the 221 participants, 64 (29.0%) cases of cirrhosis were identified based on FibroScan (≥ 14 kPa) or typical ultrasonographic findings. The remaining 157 (71.0%) patients were classified as non-cirrhotic.

Bivariate analysis (Table 2) showed that the platelet count, PCT, PLR, and PMR were significantly lower in the cirrhosis group, while MPV, PDW, RPR, APRI, and FIB-4 were significantly higher (all p < 0.05). In multivariate logistic regression analysis, RPR remained independently associated with cirrhosis after adjusting for age, sex, and other haematological indices (B = 1.404; p < 0.001). FIB-4 exhibited a robust independent correlation with cirrhosis (B = 0.046; p < 0.001). In the multivariate model, plateletcrit (PCT) remained statistically significant (p < 0.05). These results substantiate that RPR and FIB-4 are the most robust non-invasive indicators of cirrhosis in chronic hepatitis B.

ROC curve analysis revealed that RPR had excellent diagnostic accuracy for cirrhosis, with an AUC of 0.938 (95% CI: 0.906–0.970; p < 0.001). The optimal cut-off value of RPR was 0.085, which provided a sensitivity of 87.5% and specificity of 87.3% for detecting cirrhosis (Youden index = 0.748). The DeLong method's pairwise AUC comparisons revealed no statistically significant differences between APRI and RPR (p = 0.923) or FIB-4 and RPR (p = 0.367). This suggests that RPR's diagnostic performance was statistically equivalent to that of both APRI and FIB-4, despite FIB-4's somewhat higher AUC (Table 3, Figure 2).

DISCUSSION

This study demonstrated that RPR was significantly higher in cirrhotic patients compared to non-cirrhotic patients with chronic hepatitis B (0.16 vs. 0.05; p < 0.001). ROC curve analysis confirmed its excellent diagnostic accuracy, and an optimal cut-off of 0.085, yielding a sensitivity of 87.5% and a specificity of 87.3%. Multivariate analysis further supported that RPR remained independently associated with cirrhosis, consistent with the study objective to evaluate RPR as a simple non-invasive predictor of liver cirrhosis. These findings are aligned with pre-

Table 1. Baseline characteristics of patients with and without cirrhosis

Variable	Cirrhosis (N = 64)	No cirrhosis (N = 157)	Total (N = 221)	p
Median age (range) (years)	54.5 (35–80)	48 (18–78)	50 (18–80)	0.001
		No. (%) of patients		
Gender				0.004
Male	47 (73.4%)	82 (52.2%)	129 (58.4%)	
Female	17 (26.6%)	75 (47.8%)	92 (41.6%)	
HBeAg status				0.581
Negative	53 (82.9%)	123 (78.3%)	176 (79.6%)	
Positive	11 (17.1%)	34 (21.7%)	45 (20.4%)	
Hepatitis B phase				0.566
HBeAg (+), chronic infection	7 (10.9%)	27 (17.2%)	34 (15.4%)	
HBeAg (+), chronic hepatitis	4 (6.3%)	7 (4.5%)	11 (5.0%)	
HBeAg (−), chronic infection	47 (73.4%)	113 (72.0%)	160 (72.4%)	
HBeAg (−), chronic hepatitis	6 (9.4%)	10 (6.4%)	16 (7.2%)	

HBeAg, hepatitis B e-antigen.

Table 2. Univariate and multivariate analysis of haematological indices associated with liver cirrhosis in patients with chronic hepatitis B

Variable	p^*	$B^\dagger$	Standard Error	95% CI	p
Platelet count	0.004				
MPV	<0.001				
PCT	<0.001	−0.998	0.276	−1.538 to −0.458	<0.001
PDW	0.002				
RPR	<0.001	1.404	0.607	0.214 to 2.594	0.022
PLR	0.001				
PMR	<0.001				
APRI	<0.001				
FIB-4	<0.001	0.046	0.012	0.022 to 0.070	<0.001

* Univariate analysis was performed using independent t-test or Mann–Whitney U test, as appropriate.
† Multivariate analysis was conducted using backward stepwise logistic regression. Only variables retained in the final multivariate model are presented with regression coefficients.
MPV, mean platelet volume; PCT, plateletcrit; PDW, platelet distribution width; RPR, red cell distribution width-to-platelet ratio; PLR, platelet-to-lymphocyte ratio; PMR, platelet-to-monocyte ratio; APRI, aspartate aminotransferase-to-platelet ratio index; FIB-4, fibrosis-4 score; CI, confidence interval.

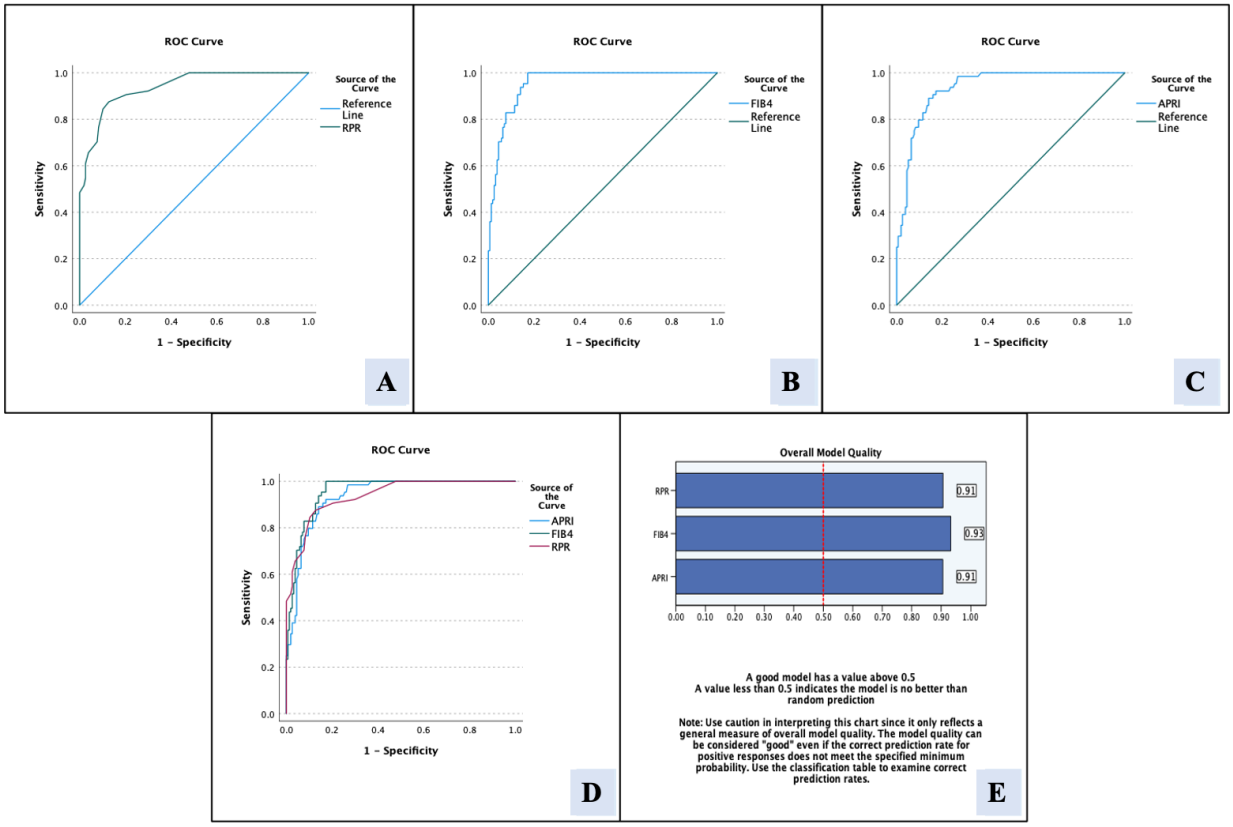

Figure 2. Receiver operating characteristic (ROC) analysis of non-invasive indices for predicting liver cirrhosis. A) ROC curve of the red cell distribution width-to-platelet ratio (RPR); B) ROC curve of the fibrosis-4 score (FIB-4); C) ROC curve of the aspartate aminotransferase-to-platelet ratio index (APRI); D) Combined ROC curves for RPR, APRI, and FIB-4, demonstrating comparative diagnostic performance; E) Overall model quality values for each index, calculated as the proportion of correct predictions relative to random classification performance

Table 3. Diagnostic performance and pairwise comparison of non-invasive indices for predicting liver cirrhosis

A. Diagnostic accuracy based on ROC analysis								
Parameter	**AUC**	**Standard Error**	***p***	**95% CI (Lower–Upper)**	**Cut-off**	**Sensitivity (%)**	**Specificity (%)**	**Youden Index**
RPR	0.938	0.016	<0.001	0.906–0.970	≥0.085	87.5	87.3	0.748
FIB-4	0.956	0.012	<0.001	0.933–0.979	≥2.11	98.4	82.8	0.812
APRI	0.936	0.015	<0.001	0.906–0.966	≥0.77	89.1	86.0	0.750
B. Pairwise comparison of AUC values using the DeLong method								
Marker 1	**Marker 2**	**AUC_1**	**AUC_2**	**AUC Difference**	**SE Difference**	***z***	***p***	
RPR	APRI	0.9380	0.9359	0.0021	0.0222	0.0962	0.9233	
RPR	FIB-4	0.9380	0.9560	0.0180	0.0200	0.9020	0.3671	

ROC, receiver operating characteristic; AUC, area under the curve; AUC^1, area under the curve of marker 1; AUC^2, area under the curve of marker 2; AUC difference, area under the curve difference; CI, confidence interval; RPR, red cell distribution width-to-platelet ratio; APRI, aspartate aminotransferase-to-platelet ratio index; FIB-4, fibrosis-4 score.

vious studies and meta-analyses that reported RPR as a reliable marker for liver fibrosis in CHB patients (14–16).

Pairwise ROC comparisons using the DeLong method showed that the diagnostic performance of RPR was statistically comparable to both APRI and FIB-4, despite FIB-4 demonstrating a slightly higher numerical AUC. This suggests a simple hematological marker is able to reflect the key mechanism underlying advanced liver fibrosis. Although FIB-4 includes age and aminotransferase value, which are associated with necroinflammatory activity in chronic hepatitis B, these additional components did not translate into a meaningful improvement in discriminatory ability. RPR combines inflammation-related changes in erythrocyte morphology with fibrosis-associated thrombocytopenia, both of which are central features of cirrhosis pathogenesis. Thus, RPR may represent as an alternative for fibrosis screening, particularly in settings where access to elastography or other biochemical indices is limited (10,15,16).

The slightly higher numerical AUC of FIB-4 may be attributed to the incorporation of age and transaminase levels, which index both fibrosis severity and necroinflammatory activity (17). The lack of substantial changes in the DeLong test indicates that these further biochemical components do not provide enhanced discriminative capability relative to RPR. There are several pathophysiologic and clinical reasons why RPR works so well as a non-invasive diagnostic for fibrosis. RDW rises in chronic HBV infection due to ongoing inflammation, oxidative

stress, red blood cell production abnormalities, and changes in red blood cell membrane shape. These changes worsen as fibrosis progresses (14,18). Platelet counts fall progressively with worsening fibrosis due to portal hypertension, splenic sequestration, and reduced thrombopoietin synthesis, all of which are hallmark processes in cirrhosis (19,20). By combining these two opposing directions, rising RDW and declining platelet counts, RPR amplifies the biological signal of fibrosis more strongly than either parameter alone.

Recent studies have also shown that RPR is significantly related with to HBV. Yu et al. identified RPR as a significant and independent marker of cirrhosis in a biopsy-verified cohort, demonstrating diagnostic performance akin to APRI and FIB-4 (15). Similarly, it was demonstrated that RPR consistently increased across fibrosis stages and accurately differentiated severe fibrosis (F3–F4) in HBV patients (18). A more recent meta-analysis also reported an AUC of 0.80 for detecting HBV-related cirrhosis, supporting its use as a haematology-based index (16). These data collectively suggest that RPR represents both inflammation-induced erythrocyte abnormalities and portal hypertension-induced thrombocytopenia, two essential components of cirrhosis pathogenesis.

Several limitations should be acknowledged. First, the cross-sectional design does not allow causal inference or assessment of fibrosis progression over time. Second, the study was conducted at a single centre, which may limit the representativeness of the findings. Third, although multivariate analysis was performed, residual confounding could not be excluded. Fourth, factors unrelated to fibrosis—such as haematological disorders, hypersplenism, or chronic inflammation—may also influence platelet and RDW levels, potentially biasing the results. Finally, data regarding antiviral therapy were not included, which may introduce bias since the treatment can affect platelet counts and fibrosis progression (5,21).

Overall, the results suggest that RPR, a simple parameter derived from complete blood count, may serve as an inexpensive and accessible tool for detecting cirrhosis in CHB patients. The biological plausibility of RPR lies in two key mechanisms: increased RDW due to chronic inflammation and dysregulated haematopoiesis, and decreased platelet counts resulting from portal hypertension and reduced thrombopoietin production (11,22). These findings are consistent with previous reports and meta-analyses showing that RPR has good diagnostic value for predicting advanced fibrosis and cirrhosis (15,16,18).

Considering the limitations, these results should be interpreted cautiously, but they provide further evidence supporting the role of haematology-based indices as practical non-invasive alternatives to FibroScan or biopsy in clinical practice. This study was conducted in a tertiary referral hospital in Indonesia, a country with intermediate hepatitis B endemicity. The use of routine laboratory parameters such as RPR increases its applicability in primary and secondary healthcare facilities, particularly in resource-limited settings where advanced diagnostic modalities are not available. However, external validation in multicentre cohorts and different populations is necessary to ensure generalizability and confirm the robustness of these findings.

This study revealed that the red cell distribution width-to-platelet ratio (RPR) is markedly elevated in patients with chronic hepatitis B and cirrhosis compared with those without cirrhosis. The ROC curve study validated its superior diagnostic efficacy, with high sensitivity and specificity at a threshold of 0.085. A multivariate study also confirmed RPR as an independent predictor of cirrhosis, in combination with FIB-4. The similar AUC values of RPR and FIB-4 underscore the potential therapeutic use of RPR as a substitute for biochemical fibrosis scores, particularly in resource-constrained environments where access to FibroScan or comprehensive liver panels is restricted. External validation in multicentre cohorts is necessary, along with longitudinal studies to assess the relevance of RPR in monitoring fibrosis progression.

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CONFLICTS OF INTEREST

None to declare.

ETHICS STATEMENT

The study was conducted in accordance with the Declaration of Helsinki and was approved by the Ethics Committee of Universitas Sumatera Utara (No: 871/KEPK/USU/2025).

DATA AVAILABILITY STATEMENT

The data supporting the findings of this study are available from the corresponding author upon reasonable request and with institutional permission.

AUTHOR CONTRIBUTIONS (CRediT)

Conceptualization: A. Y.; **Data curation:** A. Y.; **Formal analysis:** A. Y.; **Funding acquisition:** A. Y.; **Investigation:** A. Y.; **Methodology:** A. Y.; **Project administration:** A. Y.; **Resources:** A. Y.; **Software:** A. Y.; **Supervision:** T. S., I. I.; **Validation:** A. Y.; **Visualization:** A. Y.; **Writing – original draft:** A. Y.; **Writing – review & editing:** A. Y., T. S., I. I. All authors have read and agreed to the published version of the manuscript.

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