Original Article

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Assessment of Serum Interferon Gamma-Induced Protein 10 in Chronic Hepatitis B Patients

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Abstract:

Objective: This study assessed the serum levels of IP-10 alongside standard liver function parameters, including Alanine Transaminase (ALT), Aspartate Transaminase (AST), Total Serum Bilirubin (TSB), Albumin, and Prothrombin Time (PT), in patients with Chronic hepatitis B (CHB). Additionally, the study sought to analyze the correlations among these variables.

Material and Methods: A case-control study was conducted with a total of 80 participants, comprising 40 patients diagnosed with CHB and 40 healthy controls. Serum IP-10 levels were quantified utilizing a commercial Enzyme-Linked Immunosorbent Assay (ELISA) kit. The standard automated methods were employed to conduct liver function tests.

Results: Patients with CHB demonstrated significantly elevated levels of IP-10 (221.6±68 vs. 111.2±32.1 pg/mL; p-value<0.001), ALT, AST, and TSB, while showing significantly reduced albumin levels compared to the control group (p-value<0.001). IP-10 exhibited remarkable diagnostic performance, achieving an Area Under the Curve of 0.96, with sensitivity at 92.5%, and specificity at 87.0% when using a cut-off of >156.2 pg/mL. A significant negative correlation was observed between IP-10 and albumin (r=-0.41, p-value<0.01), alongside a positive correlation with TSB (r=0.32, p-value<0.05).

Conclusion: Serum IP-10 is a highly sensitive and specific biomarker for CHB, demonstrating a significant correlation with the markers of liver dysfunction and synthetic function. Its strong inverse relationship with albumin suggests its potential role in indicating impaired hepatic synthesis, supporting its utility in the clinical evaluation and treatment of CHB.

Keywords: alanine transaminase, albumin, aspartate aminotransferases, chronic, Chemokine CXCL10, hepatitis B, Iraq

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Introduction

Chronic hepatitis B (CHB) poses a significant public health challenge globally, impacting approximately 254 million individuals, and continues to be a leading cause of liver-related morbidity and mortality[1,2]. This viral disease is caused by the hepatitis B virus (HBV) and can lead to prolonged inflammation in the liver. This condition results in various clinical phenotypes, from asymptomatic chronic carriers to a fulminant disease, including chronic liver disease (cirrhosis) and hepatocarcinoma[3,4]. The complex pathogenesis of CHB involves the intricate interactions of viral factors, host immune responses, and inflammatory mediators, all of which influence disease progression and treatment outcomes[5].

The precise assessment of disease activity and prognosis in CHB remains challenging, requiring dependable indicators for effective clinical management. Standard liver function tests serve as crucial indicators of hepatocellular damage[6]. Liver-synthesized proteins are involved in numerous physiological processes that occur within the liver, such as creating bile, metabolizing food and toxins, making blood clots, and supporting the immune system[7]. Increased amounts of these enzymes generally indicate liver insult or injury, and Alanine Transaminase (ALT) and Aspartate Transaminase (AST) are particularly sensitive markers for hepatocellular toxicity[8]. Bilirubin, generated during the breakdown of red blood cells, serves as a crucial component in evaluating liver function[9]. Elevated bilirubin levels may suggest damage to hepatic cells, obstruction in the intrahepatic or extrahepatic biliary tract, or hemolysis, which could result in jaundice[10]. In hepatobiliary failure, serum bilirubin may rise progressively and possibly by one or more times the upper limit of normal each day. Hence, serum albumin, being primarily produced by the liver, serves as one of the most important indicators to evaluate synthetic liver function and can be a marker for the dysfunction of either the liver or kidneys[11].

Interferon gamma-induced protein 10 (IP-10 (CXCL10) has been identified as a potential biomarker for CHB. This chemokine plays a crucial role in both innate and adaptive immune responses by guiding T cells to the sites of inflammation[12]. IP-10 is secreted by various cell types, including microglia, macrophages, and astrocytes, in response to stimuli like interferon-α, interferon-β, interferon-γ, or viral infections. Within the hepatic environment, IP-10 is specifically produced by hepatocytes in inflammatory areas, facilitating the recruitment of T cells to hepatic lesions during chronic viral hepatitis[13]. Despite the promising potential of IP-10 as a biomarker for CHB, there are still several important research gaps that need to be addressed. Initially, the standardization of measurement techniques for IP-10 across various laboratories and populations poses a significant challenge[14].

The role of IP-10/CXCL10 in the immunopathogenesis of CHB has gained global recognition, yet notable significant research gaps remain, especially concerning the Iraqi population[15]. Current international studies have shown the significance of IP-10 as a biomarker for diagnosing CHB, assessing disease severity, and predicting treatment response[16]. Nonetheless, applying these findings to the Iraqi population is limited by various factors: a shortage of local epidemiological data on IP-10 levels, the absence of established population-specific reference ranges, and an insufficient understanding of how regional genetic, environmental, and viral factors might affect its expression and utility. This study aimed to evaluate the clinical relevance of serum IP-10 in Iraqi patients with CHB by quantifying its levels alongside standard liver function tests (ALT, AST, Total Serum Bilirubin (TSB), Albumin, Prothrombin Time (PT)) and comparing them with healthy controls (HCs) to determine its diagnostic efficacy and investigate its correlation with the markers of liver damage and synthetic function.

Material and Methods

Study design

This case-control study was carried out at the Gastroenterology Center of Marjan Teaching Hospital over a six-month period (February-July 2025). A total of eighty subjects were recruited and categorized into two groups. The case group (n=40) included adults aged 21 to 57 years with a confirmed diagnosis of CHB. The diagnostic criteria for CHB required the persistent presence of HBsAg for more than six months, detectable serum HBV DNA, and corroborating clinical and biochemical evidence of chronic liver disease. The control group (n=40) consisted of healthy volunteers who were individually matched to cases by age (±5 years) and sex. Controls were required to be seronegative for HBsAg, anti-HCV, and anti-HIV, have no known history of liver disease, and present with normal baseline liver function tests (ALT, AST, TSB within laboratory reference ranges).

Ethical considerations

This research adhered to the ethical standards outlined in the Declaration of Helsinki. The Local Ethics Committee at Babylon Medical College evaluated and sanctioned the research protocol (Approval Reference: BMC-LEC-2024-067; Date: June 7, 2024). Informed written permission was acquired from all the participants after explanation of the methods.

Inclusion and exclusion criteria

Participants were recruited into the CHB cohort subsequent to a verified diagnosis of CHB. To mitigate the potential impact of comorbid conditions, the study's exclusion criteria included co-infection with hepatitis C virus (HCV), hepatitis D virus (HDV), or human immunodeficiency virus (HIV), diagnosed renal failure, autoimmune disorders such as rheumatoid arthritis or autoimmune hepatitis, clinical or histological evidence of liver cirrhosis or hepatocellular carcinoma, a history of significant alcohol consumption, metabolic dysfunction-associated steatotic liver disease (MASLD), and obesity, defined as a body mass index (BMI) of 30 kg/m^2 or higher.

Sample collection and processing

Venous blood samples (~5 mL) were obtained from all participants after an overnight fast using standard sterile phlebotomy procedures. Blood was drawn into two types of vacuum tubes: serum-separating tubes and sodium citrate tubes for prothrombin time assessment. Serum was isolated by centrifuging samples at 3000 rpm for 10 minutes within a half-hour of collection. The resulting serum aliquots were rapidly frozen at -80 °C for preservation and stored until a subsequent batch analysis was performed to ensure analyte integrity.

Biochemical and immunoassays

Liver Enzymes Serum ALT, AST, total serum bilirubin (TSB), and albumin were measured with standard spectrophotometric procedures on a fully automatic clinical chemistry analyzer (Cobas c501, Roche Diagnostics). PT measurements were performed on citrated plasma with a coagulation analyzer.

IP-10 Serum (IP-10/CXCL) levels were measured using an ELISA kit for human IP-10/CXCL (Elabscience® Human IP-10/CXCL, Cat: No: E-EL-H0050), following the manufacturer's guidelines. Calculated average concentrations after duplicate analyses of each sample. Intra- and inter-assay coefficients of variation were maintained ≤10.0% to ensure accuracy.

Statistical analysis

Data analysis was performed using GraphPad Prism® (Version 9.3.1). Continuous variables were first

tested for adherence to a normal distribution via the Shapiro-Wilk test. Results are presented as mean±standard deviation (S.D.) for parametric data; all dataset parameters in this study were normally distributed. Differences between the independent CHB and control groups were assessed with an Independent Samples T-test. Within the CHB group, a comparative analysis based on albumin level stratification (<3.0 g/dL vs. ≥3.0 g/dL) was also conducted using an Independent Samples T-test. The efficacy of IP-10 as a diagnostic biomarker was evaluated by measuring the Area Under the Curve (AUC) from an Receiver Operating Characteristic Curve (ROC) analysis.

Results

The study enrolled 80 participants, divided equally into a cohort of 40 CHB patients and 40 HCs; their baseline characteristics are detailed in Table 1. Demographic factors, including age, sex, and BMI, did not differ significantly between groups (p-value>0.05), supporting their comparability. However, liver function tests revealed expected and significant deviations in the CHB group. This included markedly higher mean levels of AST, ALT, and TSB, alongside lower mean albumin levels (p-value<0.001 for all). The mean PT was also significantly shorter in patients (p-value<0.001). A key finding was that the mean serum concentration of IP-10 was significantly elevated in patients, measuring approximately double that of the control group (p-value<0.001).

Table 2 categorizes CHB patients based on serum albumin levels using a clinical cut-off of <3.0 g/dL to identify those with hypoalbuminemia, a marker of impaired synthetic liver function. The group with albumin <3.0 g/dL (n=18) exhibited significantly higher serum levels of IP-10 than those with albumin ≥3.0 g/dL (n=22) (248±70.4 pg/mL vs. 203.6±60.4 pg/mL, p-value=0.037).

The ROC curve analysis for serum IP-10 levels demonstrated its strong diagnostic performance in distinguishing CHB patients from HCs. The AUC was 0.96 (95.0% CI: 0.94–0.99; p-value<0.001), indicating excellent discriminatory power. At an optimal cut-off value of >156.2 pg/mL, IP-10 exhibited high sensitivity (92.5%) and specificity (87.0%), indicating its potential as a reliable biomarker for the identification of CHB and facilitating clinical decision-making.

Table 1 Clinical and demographic features of chronic hepatitis B vs. healthy controls

Parameter	Patients (n=40)	Healthy controls (n=40)	p-value
Age (years)	40.4±11.8	39.6±13.2	0.78
Sex			
Male	18 (45.0%)	23 (58.0 %)	0.4
Female	22 (55.0%)	17 (42.0%)	
BMI (kg/m^2)	23.9±1.2	23.6±1.7	0.36
AST (IU/L)	30.4±5.9	16.2±3.1	<0.001*
ALT (IU/L)	45.2±8.7	19.8±3.6	<0.001*
Total serum bilirubin (µmol/L)	25.4±4.3	11.2±1.9	<0.001*
Albumin (g/dl)	3.09±0.28	3.97±0.36	<0.001*
Prothrombin time (seconds)	11.7±0.51	12.8±0.65	<0.001*
IP-10 (pg/mL)	221.6±68	111.2±32.1	<0.001**

*=p-value<0.05, BMI=body mass index, AST=Aspartate Transaminase, ALT=Alanine Transaminase

Table 2 Comparison of liver injury and inflammatory markers in chronic hepatitis B patients stratified by albumin level

Parameter	Albumin <3.0 (n=18)	Albumin ≥3.0 (n=22)	p-value
AST (IU/L)	29.8±5.8	31.8±5.9	0.29
ALT (IU/L)	46±9.2	43.1±8.2	0.4
TSB (µmol/L)	26.5±4.2	24.1±4.1	0.09
Prothrombin time (seconds)	11.89±0.26	11.6±0.6	0.17
IP-10 (pg/mL)	248±70.4	203.6±60.4	0.037**

Chi-square test, Independent Samples T-Test, Bold p-values indicate statistical significance (p-value<0.05), *=p-value<0.05, AST=Aspartate Transaminase, ALT=Alanine Transaminase, TSB=Total Serum Bilirubin, IP-10=Interferon Gamma-Induced Protein 10

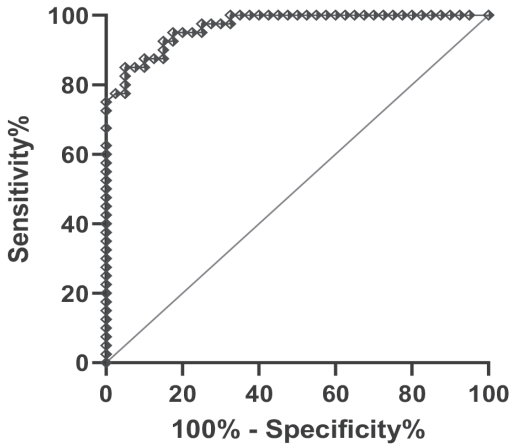

Figure 1 ROC curve analysis for IP-10 between the patients and control group

Table 3 presents the frequency and percentage of CHB patients exhibiting abnormal clinical values relative to the established cutoff values. Elevated serum IP-10 levels (>156.2 pg/mL) and hypoalbuminemia (<3.5 g/dL) were the most prevalent abnormalities, occurring in 82.5% of the patients (n=33), followed by elevated TSB (>21 µmol/L) in 80.0% (n=32). Over half of the patients (52.5%, n=21) showed elevated ALT levels (>45 IU/L), whereas elevated AST (>40 IU/L) and prolonged prothrombin time (>13 s) were less common, observed in only 15% (n=6) and 7.5% (n=3) of patients, respectively.

Table 4 compares the serum levels of IP-10, AST, ALT, and albumin between CHB patients and HCs, stratified by age group (<40 and ≥40 years). The results demonstrated that CHB patients in both age subgroups exhibited significantly elevated levels of IP-10, AST, and ALT, along with significantly reduced albumin levels, compared to age-matched HCs (all p- This confirms that the significant differences are consistent in both younger and older populations.

Table 3 Frequency of abnormal clinical parameters in the CHB patient group

Abnormal Parameter	Cut-off value	Number of patients (n)	Percentage (%)
Elevated IP-10	>156.2 pg/mL*	33	82.5
Elevated AST	>40 IU/L	6	15.0
Elevated ALT	>45 IU/L	21	52.5
Elevated TSB	>21 µmol/L	32	80.0
Hypoalbuminemia	<3.5 g/dL	33	82.5
Prolonged PT	>13 seconds	3	7.5

*The IP-10 cut-off was derived from the ROC analysis performed in this study, AST=Aspartate Transaminase, ALT=Alanine Transaminase, TSB=Total Serum Bilirubin

Table 4 Comparison of key biomarkers between patients and controls stratified by age group

Age Group	Group	n	IP-10 (pg/mL)	AST (IU/L)	ALT (IU/L)	Albumin (g/dl)
<40 years	Patients	16	229.3±82.4	28.7±6.2	45.7±9.5	3.05±0.35
	Healthy controls	17	98.7±23.8	16.8±3.3	20.1±3.4	4.12±0.41
p-value			<0.001	<0.001	<0.001	<0.001
≥40 years	Patients	24	218.9±72.5	31.8±5.6	44.5±8.1	3.16±0.32
	Healthy controls	23	119.7±32.6	16.8±2.8	19.1±3.7	3.94±0.38
p-value			<0.001	<0.001	<0.001	<0.001

This confirms that the significant differences are consistent in both younger and older populations, AST=Aspartate Transaminase, ALT=Alanine Transaminase, TSB=Total Serum Bilirubin, IP-10=Interferon Gamma-Induced Protein 10

Correlation matrix assessing the relationships among clinical parameters in patients with CHB. Serum IP-10 levels demonstrated a statistically significant positive correlation with (TSB; r=0.32, p-value<0.05) and a significant negative correlation with albumin levels (r=–0.41, p-value<0.01). IP-10 did not show significant correlation with AST, ALT, or PT. However, strong intercorrelations were found among the traditional liver markers: AST exhibited a positive correlation with ALT (r=0.78, p-value<0.01), TSB (r=0.45, p-value<0.01), and PT (r=0.51, p-value<0.01), while showing a negative correlation with albumin (r=–0.62, p-value<0.01). Albumin levels exhibited an inverse correlation with all injury markers (AST, ALT, TSB, and PT), thereby reinforcing its significance as an indicator of synthetic liver function.

Discussion

The findings of this study provided substantial evidence that supported the clinical relevance of IP-10 as a biomarker for CHB. The results demonstrated a significant elevation of serum IP-10 levels among CHB patients when compared to HCs, showcasing excellent diagnostic performance characteristics that were consistent with previous research findings.

The approximately two-fold higher serum IP-10 levels in patients with CHB compared to HCs (221.6±68 pg/mL vs. 111.2±32.1 pg/mL) highlighted its significant role in the immunopathogenesis of HBV infection. This increase reflects not merely a correlation but indicates the fundamental inflammatory processes driving liver disease.

IP-10 serves as a crucial chemokine induced by interferon-γ, predominantly synthesized by hepatocytes in response to inflammatory stimuli[17]. It served as a strong chemoattractant for activated T-cells, natural killer (NK) cells, and monocytes, effectively coordinating the recruitment of immune cells to sites of hepatic inflammation[18]. This mechanistic role was supported by histological studies demonstrating that IP-10 mRNA was predominantly synthesized by hepatocytes in the periportal areas of inflammation, with its expression directly correlating to the severity of liver necroinflammation[19].

In addition to its pathophysiological role, the results aligned with previous studies and highlighted the significant clinical value of IP-10 as a noninvasive biomarker for liver disease staging. Serum IP-10 levels exhibited a significant ability to differentiate CHB patients from healthy individuals, as well as to categorize disease severity in the patient group. Specifically, IP-10 levels were significantly elevated in patients exhibiting significant liver fibrosis compared to those with mild fibrosis, demonstrating strong diagnostic sensitivity and specificity for the identification of advanced disease[20]. The prognostic significance of this finding extended to the identification of cirrhosis, as cirrhotic patients demonstrated significantly elevated levels of IP-10 compared to non-cirrhotic patients, indicating its potential use in predicting this critical complication[21].

The clinical significance of IP-10 was highlighted in relation to therapeutic decision-making. Evidence indicated that a high baseline CXCL10 to BAFF (B-cell activating factor) ratio (≥0.45) served as a predictor for a combined response to pegylated-interferon (PEG-IFN) therapy in HBeAg-positive CHB patients, demonstrating a high negative predictive value of 89.7%[22]. This suggested that a profile defined by low BAFF and high CXCL10 levels correlated with a stronger antiviral immune response, establishing a significant tool for identifying patients who are most likely to benefit from IFN-based therapy.

Furthermore, the strong correlation between elevated circulating IP-10 levels and markers of liver injury reinforced its role as a reliable marker of hepatocellular damage[23]. IP-10 levels were consistently elevated in patients with CHB who exhibited abnormal liver function compared to those with normal function, emphasizing its significance within the CXC chemokine family as a dependable biomarker for assessing and monitoring the degree of liver damage in chronic HBV infection[23,24].

This study demonstrated the most significant finding, which involved the strong diagnostic performance of IP-10, evidenced by an area under the ROC curve (AUC) of 0.96, sensitivity of 92.5%, and specificity of 87.0% at an optimal cut-off value of >156.2 pg/mL. This diagnostic accuracy exceeded that of numerous traditional liver function markers and aligned with previous meta-analytic findings, indicating that patients with CHB exhibited significantly higher serum IP-10 levels compared to HCs[19]. The cut-off value identified in this study (156.2 pg/mL) aligned with previously reported thresholds, yet it differed from some studies that indicated higher cut-offs of approximately 300 pg/mL for predicting liver fibrosis. This variation suggested that IP-10 cut-off values may vary based on the specific clinical endpoint being evaluated[25,26].

The correlation analysis conducted in this study revealed significant relationships between IP-10 and the key conventional liver function markers, offering valuable insights into its potential clinical applications. A significant positive correlation was observed between IP-10 and TSB ($r=0.32$, p-value<0.05), while a stronger negative correlation was detected with albumin levels ($r=-0.41$, p-value<0.01)[27].

The correlations observed suggested that IP-10 levels reflect both inflammation and a particular aspect of liver pathophysiology. The negative correlation with albumin was particularly important as the synthesis of albumin had been considered a basic test of the synthetic function of the liver. The reduction presented a reliable marker for the

monitoring of disease progression and prognosis in chronic liver patients[28]. Accordingly, the relationship between high IP-10 levels and hypoalbuminemia may be an indicator of abnormal synthetic function in CHB. The positive association with bilirubin, a cholestasis marker, indicated that IP-10 was related to both cholestatic and synthetic liver dysfunction. Such a relationship may be additive in nature to other biomarkers, enabling an extensive assessment of disease activity.

This study revealed an intriguing observation: there was no significant correlation found between IP-10 and aminotransferase levels (ALT and AST). This contrasted with several previous studies that demonstrated strong positive correlations (r=0.546 and 0.644 for ALT and AST, respectively; p-value<0.001)[24]. Several factors may have contributed to this discrepancy. Variations in patient groups, such as differences in disease phase, severity, and treatment status, had likely influenced these relationships[18]. Additionally, the temporal dynamics of biomarker elevation may be significant; findings indicated that IP-10 levels can stay elevated even when aminotransferase activity returns to normal[19,26]. This suggested that IP-10 could serve as a more sensitive and durable marker of underlying immune-mediated inflammation compared to ALT or AST, which are both direct surrogates for acute hepatocyte necrosis. This feature could make IP-10 especially valuable for identifying subclinical or smoldering disease activity that serum aminotransferases might fail to detect.

The strong correlation between elevated IP-10 levels and hypoalbuminemia (albumin <3.0 g/dL) warrants further examination. Patients exhibiting low albumin levels demonstrated significantly higher IP-10 levels, suggesting a potential correlation between immune stimulation, as indicated by IP-10 levels, and impaired hepatic synthesis. This association carried significant clinical implications, as hypoalbuminemia is a well-established prognostic factor for mortality and morbidity in liver disease[29]. Therefore, the combination of elevated IP-10 and hypoalbuminemia may help identify patients at a higher risk for disease progression and adverse clinical outcomes[27].

The findings of this study supported the established understanding of the role of IP-10 in the pathogenesis of CHB. The elevated chemokine indicated the host's immune response to persistent HBV infection, facilitating the recruitment of immune effector cells to the liver. Nevertheless, this immune activation, while potentially beneficial for managing viral infections, could also lead to hepatocellular damage and the progression of fibrosis. Previous studies have demonstrated that IP-10 levels correlated with the degree of liver fibrosis and can accurately predict significant fibrosis[18,25]. The elevation of IP-10, which was consistent regardless of age in both younger (<40 years) and older (≥40 years) CHB patients, indicated that this biomarker's effectiveness was not influenced by age-related factors, thereby improving its clinical relevance across various patient groups. This finding aligned with earlier studies that demonstrated the reliability of IP-10 as a biomarker across different patient populations[26].

Study limitations and future directions

Although this study provides valuable insights into IP-10 diagnostic utility in CHB, several limitations should be acknowledged. Exclusion of patients with cirrhosis and HCC and maintaining a homogenous study population may restrict the applicability of these findings to all CHB patients. Large prospective longitudinal studies need to explore the value of IP-10 in monitoring disease course, predicting responses to therapy, and recognizing patients at risk of complications. Combining IP-10 measurement with other promising biomarkers, such as hepatitis B core-related antigen (HBcrAg) or M2BPGi, might also improve the diagnostic and prognostic performance. Furthermore, harmonisation of the assay measurement procedures for IP-10 and the definition of universally accepted reference.

Conclusion

This study demonstrated that serum IP-10 has the potential to be an effective biomarker for the diagnosis and evaluation of CHB, exhibiting high diagnostic accuracy and a significant correlation with the biochemical markers indicative of hepatic injury. The association between elevated levels of IP-10 and hypoalbuminemia in patients with CHB and the relationship between immune activation and synthetic liver function in CHB patients have been newly revealed through the association of elevated IP-10 levels and hypoalbuminemia. The results support further investigation of IP-10 as a significant addition to the current biomarkers panel utilized in the management of CHB, with potential applications in diagnosis, disease monitoring, and treatment guidance. The robust diagnostic performance of IP-10, along with its relevance to pathophysiological and age-independent utility, serves as a valuable tool for healthcare professionals treating patients with CHB.

Authors' contributions:

SHA: Conceptualization, Methodology, Formal analysis, investigation, writing – Original Draft, Writing – Review & Editing. KAS: Investigation, Data Curation, Resources, Writing – Review, and Editing. ZAA: Methodology, Validation, Formal analysis, Supervision, Project administration, writing – Review & Editing.

Availability of data and materials

The datasets generated and analyzed during the current study are not publicly available because of participant privacy and confidentiality concerns but are available from the corresponding author upon reasonable request.

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Conflict of interest

The authors declare that they have no competing interests.

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