



Lower Serum Kindlin-2 Levels Are Associated with Liver Cirrhosis: A Preliminary Study

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Abstract

Background: Kindlin-2 is a focal adhesion protein involved in cell-cell and cell-extracellular matrix adhesion. These functions influence cell behavior and processes such as fibrosis. Kindlin-2 has recently been reported to play important roles in fibrotic diseases.

Objectives: This study aimed to assess the association between serum kindlin-2 levels and liver cirrhosis.

Methods: The study included 48 individuals with liver cirrhosis and 40 healthy controls. Serum kindlin-2 levels were measured using an enzyme-linked immunosorbent assay (ELISA). Complete blood count parameters and liver function tests were assessed. Liver fibrosis was evaluated using noninvasive indices, including the aspartate aminotransferase-to-platelet ratio index (APRI) and fibrosis-4 (FIB-4) index. Mortality risk was evaluated using the Model for End-Stage Liver Disease-Sodium (MELD-Na) score.

Results: Serum kindlin-2 levels were lower in the liver cirrhosis group ($P = 0.014$). Kindlin-2 levels were inversely associated with alanine aminotransferase (ALT), aspartate aminotransferase (AST), and aminotransferase-to-platelet ratio index (APRI) scores and positively associated with platelet count. Low kindlin-2 levels were independently associated with liver cirrhosis.

Conclusions: Low serum kindlin-2 levels were associated with liver cirrhosis. Correlations were observed between kindlin-2 levels and markers of liver fibrosis, as well as certain liver function tests. These findings may be a consequence of the cirrhosis process.

Keywords: Kindlin-2, Liver Fibrosis, Chronic Liver Diseases

1. Background

Liver cirrhosis is a serious public health problem that causes approximately 2 million deaths annually (1). Advanced fibrosis is a feature of liver cirrhosis. Liver fibrosis is a wound-healing response to hepatic tissue damage caused by various etiologies, including infections, metabolic disorders, and autoimmune conditions. This process results in excessive extracellular matrix (ECM) deposition and impaired hepatic function (2).

Kindlins are focal adhesion proteins that mediate adhesion between cells and the ECM. The kindlin family

comprises kindlin-1, kindlin-2, and kindlin-3. The fermitin family homologue 2 (FERMT2) gene encodes kindlin-2, which is expressed ubiquitously in all cell types in the body (3). Kindlin-2 has been implicated in cell and ECM adhesion, apoptosis, differentiation, and carcinogenesis (3). Important effects of kindlin-2 have also recently been reported in fibrotic diseases. Its effects on fibrosis are hypothesized to involve integrin-dependent and integrin-independent pathways in various tissues. Kindlin-2 mediates integrin activation and integrin-ligand binding (4, 5). It is also thought to play a role in cirrhosis through oxidative stress, cell death, inflammation, and increased

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ECM deposition via integrin-independent intracellular signaling in the liver (6, 7).

Research indicates that kindlin-2 has organ-specific roles in fibrogenesis. Cardiomyocyte-specific deletion of kindlin-2 has been shown to cause progressive cardiac fibrosis (8, 9). Conversely, inhibition of the kindlin-2 gene in renal tissue attenuated fibrosis (10). Kindlin-2 expression is also elevated in lung tissue during pulmonary fibrosis (11). Its role in liver fibrosis is not fully understood, and studies have yielded conflicting findings. Few in vivo and in vitro studies have addressed this relationship (6, 7, 12). Loss of kindlin-2 expression caused liver fibrosis, whereas kindlin-2 was also reported to protect against liver fibrosis (6, 7). Conversely, increased kindlin-2 expression has been reported in fibrotic liver tissue from both mice and humans (12).

2. Objectives

Based on these findings, this study was the first clinical study to examine changes in kindlin-2 levels in patients with liver cirrhosis and their relationships with laboratory parameters.

3. Methods

3.1. Ethical Approval

This cross-sectional case-control study adhered to the ethical principles of the Declaration of Helsinki. The study protocol was approved by the Hospital Ethics Committee (Protocol No. 151 - 2021) before the study began. Written informed consent was obtained from all participants before enrollment.

3.2. Study Design and Population

The study included 88 participants who attended the Gastroenterology and Internal Medicine Outpatient Clinics of Prof. Dr. Cemil Taşcıoğlu Hospital between June 2021 and June 2022. The case group comprised 48 patients older than 18 years with liver cirrhosis: 15 with nonalcoholic steatohepatitis (NASH)-related cirrhosis, 6 with alcohol-related cirrhosis, 16 with hepatitis B-related cirrhosis, 4 with hepatitis C-related cirrhosis, 2 with primary biliary cirrhosis, and 5 with cryptogenic cirrhosis. Gastroenterology and internal medicine physicians diagnosed and followed these patients using radiological, endoscopic, laboratory, and clinical methods.

Clinical severity was assessed using the Child-Pugh classification. A total of 26 patients were classified as Child-Pugh class A, 14 as class B, and 8 as class C. The patients were stable outpatients attending routine checkups, rather than

patients hospitalized with decompensated cirrhosis. Patients with hepatocellular carcinoma, chronic obstructive pulmonary disease (COPD), heart failure, renal failure, or malignancies were excluded from the case group.

The control group comprised 40 healthy participants older than 18 years without liver cirrhosis, chronic renal failure, COPD, heart failure, or malignancy. Demographic characteristics (gender and age) and laboratory results were recorded for all participants. Laboratory assessments included aspartate aminotransferase (AST), alanine aminotransferase (ALT), gamma-glutamyl transferase (GGT), alkaline phosphatase (ALP), total bilirubin, direct bilirubin, albumin, C-reactive protein (CRP), prothrombin time (PT), activated partial thromboplastin time (aPTT), international normalized ratio (INR), and complete blood count parameters (hemoglobin, platelet count, and leukocyte count). Blood samples from all participants were collected in biochemistry tubes and centrifuged at 4000 rpm for 10 minutes. Serum was then collected and stored at -80 °C until analysis.

The AST-to-platelet ratio index (APRI) predicts advanced hepatic fibrosis. Its formula is as follows: $APRI = [(AST / \text{Upper limit of normal for AST}) / \text{Platelets (109/L)}] \times 100$ (13).

The fibrosis-4 (FIB-4) index is another noninvasive score for liver fibrosis. Its formula is: $FIB4 = [\text{Age} \times \text{AST (IU/L)}] / [\text{platelet count (109/L)} \times \sqrt{\text{ALT (IU/L)}}]$ (14).

Values for PT, INR, albumin, total bilirubin, urea, creatinine, and sodium were analyzed to determine baseline Model for End-Stage Liver Disease-Sodium (MELD-Na) scores for all patients. The MELD score was calculated as follows: $MELD \text{ score} = 3.8 \times \log_e(\text{serum bilirubin [mg/dL]}) + 11.2 \times \log_e(\text{INR}) + 9.6 \times \log_e(\text{serum creatinine [mg/dL]}) + 6.4$. A lower limit of 1 was used for all variables, and creatinine was capped at 4. Creatinine was set to 4 for patients receiving renal replacement therapy. The MELD-Na score is used when the sodium level is between 125 and 140 mmol/L. $MELD\text{-}Na = [MELD\text{-}Na - 0.025 \times MELD \times (140 - Na)] + 140$ (15).

3.3. Measurement of Kindlin-2

The assay kit and samples were brought to room temperature. Serum kindlin-2 levels were measured using an enzyme-linked immunosorbent assay (ELISA) kit (catalog number: E2774Hu). The measurement range was 0.05 - 24 ng/mL, and the reported lower limit was 0.022 ng/mL. The reported intra-assay and inter-assay coefficients of variation were 8% and 10%, respectively.

3.4. Statistical Analysis

Data were analyzed using IBM SPSS Statistics 27.0 and summarized as mean $\pm$ standard deviation or median (25th - 75th percentile), according to their distribution. Categorical variables were reported as frequencies and percentages. Comparisons between 2 independent groups were performed using the independent-samples t-test or Mann-Whitney U test, according to the data distribution. Univariate and multivariate regression models were used to investigate the determinants of serum kindlin-2 concentrations. Because kindlin-2 values were skewed, they were logarithmically transformed before analysis. A separate binary logistic regression model was constructed to identify variables independently associated with liver cirrhosis.

4. Results

Serum kindlin-2 levels were significantly lower in patients with liver cirrhosis than in healthy controls ($P = 0.014$). AST levels were higher in the cirrhosis group ($P = 0.003$). ALT, ALP, GGT, total bilirubin, direct bilirubin, INR, PT, aPTT, CRP, APRI, FIB-4, and MELD-Na values were significantly higher in the cirrhosis group ($P < 0.001$). Platelet counts and albumin levels were significantly lower in the cirrhosis group ($P < 0.001$). Leukocyte counts did not differ significantly between groups ($P = 0.730$). Patients with cirrhosis were older than controls ($P < 0.001$) (Table 1).

Univariate and multivariate regression analyses were performed to identify factors associated with kindlin-2 levels. Univariate analysis showed that kindlin-2 levels were negatively correlated with liver cirrhosis, AST, ALT, and APRI ($P < 0.001$, $P = 0.022$, $P = 0.008$, and $P < 0.001$, respectively) and positively correlated with platelet count ($P = 0.015$). Multivariate analysis identified significant associations of kindlin-2 levels with liver fibrosis and APRI ($P = 0.036$ and $P = 0.011$, respectively). No correlation was found between kindlin-2 levels and age or gender (Table 2).

Serum kindlin-2 levels were significantly associated with the presence of cirrhosis independently of other variables, including age, gender, and clinically significant liver function tests. Lower kindlin-2 levels were independently associated with liver cirrhosis (odds ratio [OR] = 0.714; $P = 0.017$). Liver cirrhosis was also associated with lower platelet counts and albumin levels ($P = 0.004$ and $P = 0.025$, respectively). Age, gender, and ALT levels were not associated with liver cirrhosis (Table 3).

5. Discussion

Liver cirrhosis is characterized by advanced fibrosis and loss of hepatic function through various pathophysiological mechanisms (2). Given the reported association between kindlin-2 and fibrosis (6), we

measured serum kindlin-2 levels in patients with liver cirrhosis. Kindlin-2 levels were lower, and this reduction was independently associated with liver cirrhosis after adjustment for other factors.

Previous *in vivo* studies have reported findings similar to ours (6, 7). Gao et al. reported that kindlin-2 loss may lead to hepatocyte apoptosis and ECM accumulation, potentially resulting in severe liver fibrosis. One possible intracellular mechanism linking low kindlin-2 levels with cirrhosis is activation of the tumor necrosis factor- α /nuclear factor- κ B (TNF- α /NF- κ B) pathways in the absence of kindlin-2. TNF- α activation has been associated with increased hepatic inflammation, hepatocyte death, and fibrosis (6).

In our study, kindlin-2 levels were inversely associated with APRI, a noninvasive marker of liver fibrosis, independently of its other components. The APRI score increased as kindlin-2 levels decreased. ALT and AST levels, which are markers of hepatocyte parenchymal damage, were inversely correlated with kindlin-2 levels. Kindlin-2 levels were also positively associated with platelet count, one of the components of APRI. Liver cirrhosis was inversely associated with kindlin-2 levels. Consistent with our findings, Gao et al. (6) observed increased ALT and AST levels in the livers of kindlin-2 knockout mice. They also reported that kindlin-2 loss led to severe splenomegaly. Kindlin-2 loss has been reported to increase profibrotic molecules, including collagen, transforming growth factor- β (TGF- β), and tissue metalloproteinase inhibitor-1 (TIMP-1), which in turn lead to severe liver cirrhosis.

Zhong et al. (7) provided further molecular evidence supporting our clinical findings. They reported that kindlin-2 loss reduces the expression of glutathione-S-transferase P1 (GSTP1), an antioxidant enzyme in hepatocytes. Kindlin-2 deletion has also been reported to contribute to liver damage by increasing the production of osteopontin (OPN), which is elevated in fibrotic liver tissue (7, 16).

A third possible mechanism linking kindlin-2 to fibrosis in cirrhosis involves integrins. Kindlin-2 is a cytoplasmic adhesion molecule associated with integrin β 1, which requires kindlin-2 for activation. Kindlin-2 deletion impairs integrin activation (4-5, 17). Integrin β 1 deficiency may contribute to liver fibrosis by impairing hepatocyte-ECM interactions and increasing TGF- β , matrix metalloproteinase 13 (MMP-13), and TIMP-1 levels. Integrin β 1 deficiency also increases liver damage and reduces regeneration (4, 18). Thus, low kindlin-2 levels may lead to functional integrin β 1 deficiency and increased susceptibility to fibrosis.

Zhang et al. (19) demonstrated that kindlin-2 interacts with irisin to exert protective effects against hepatic

Table 1. Demographic and Laboratory Data for Patient and Control Groups ^a

Variables	Control Group (n = 40)	Liver Cirrhosis Group (n = 48)	P-Value
Age (y)	43.95 ± 12.4	59.63 ± 13.8	< 0.001 ^b
Gender			< 0.001 ^c
Female	28 (70)	17 (35)	
Male	12 (30)	31 (65)	
Platelet (10 ³ /μL) median (25th - 75th percentile)	231 (208 - 277)	109 (81 - 159)	< 0.001
Leukocyte (10 ³ /μL) (mean ± SD)	6.64 ± 1.8	6.45 ± 1.8	0.730 ^b
ALT (IU/L) median (25th - 75th percentile)	15 (11 - 23)	23 (15 - 41)	< 0.001
AST (IU/L) median (25th - 75th percentile)	17 (14 - 20)	34 (22 - 63)	0.003
ALP (IU/L) median (25th - 75th percentile)	68 (49 - 81)	106 (62 - 146)	< 0.001
GGT (IU/L) median (25th - 75th percentile)	14 (11 - 23)	57 (31 - 102)	< 0.001
Total bilirubin (mg/dL) median (25th - 75th percentile)	0.45 (0.32 - 0.58)	1.04 (0.72 - 1.98)	0.001
Direct bilirubin (mg/dL) median (25th - 75th percentile)	0.14 (0.09 - 0.21)	0.45 (0.36 - 0.99)	0.001
INR median (25th - 75th percentile)	0.95 (0.9 - 1.00)	1.36 (1.18 - 1.90)	< 0.001
PT (s) median (25th - 75th percentile)	12.4 (12.0 - 13.2)	16.8 (14.3 - 19.9)	< 0.001
aPTT (s) median (25th - 75th percentile)	28.6 (25 - 30.3)	35.9 (30.9 - 57.5)	< 0.001
Albumin (g/L) median (25th - 75th percentile)	45.0 (42.2 - 46.9)	34.0 (25.4 - 39.7)	< 0.001
CRP (mg/dL) median (25th - 75th percentile)	1.23 (0.63 - 4.45)	8.24 (1.92 - 18.97)	< 0.001
APRI median (25th - 75th percentile)	0.22 (0.17 - 0.27)	0.95 (0.66 - 1.74)	< 0.001
FIB-4 median (25th - 75th percentile)	0.71 (0.55 - 1.00)	4.13 (3.1 - 6.4)	< 0.001
MELD-Na median (25th - 75th percentile)	4.8 (4.3 - 5.6)	7.8 (6.4 - 10.6)	< 0.001
Kindlin-2 (ng/mL) median (25th - 75th percentile)	2.24 (1.82 - 11.30)	2.17 (0.84 - 3.42)	0.014

^a Values are expressed as mean ± SD or No. (%) unless otherwise indicated. Abbreviations: ALP, alkaline phosphatase; ALT, alanine aminotransferase; APRI, aspartate aminotransferase-to-platelet ratio index; aPTT, activated partial thromboplastin time; AST, aspartate aminotransferase; CRP, C-reactive protein; FIB-4, fibrosis-4 index; GGT, gamma-glutamyl transferase; INR, international normalized ratio; MELD-Na, Model for End-Stage Liver Disease-Sodium; PT, prothrombin time; SD, standard deviation.

^b Independent-samples t test;

^c Chi-square test; all other comparisons: Mann-Whitney U test.

steatosis. Kindlin-2 suppression reduced the beneficial effects of irisin on hepatocyte reactive oxygen species production, mitochondrial function, and endoplasmic reticulum stress. This pathway may represent another mechanism linking reduced kindlin-2 levels with hepatic fibrotic processes. The molecular and clinical findings suggest a potential association between kindlin-2 and liver fibrosis. Kindlin-2 may be a protective molecule essential for liver physiology, and low levels may be a consequence of the cirrhosis process.

Studies have also associated fibrosis in tissues other than the liver with low kindlin-2 levels. Qi et al. reported that kindlin-2 deficiency led to progressive cardiac fibrosis in animal studies (8). Similarly, cardiac kindlin-2 deficiency leads to cardiomyopathy and death through reduced integrin β₁ levels (9). In the kidney, loss of kindlin-2 in podocytes has been shown to cause glomerular fibrosis (20). However, a few independent studies have reported findings that conflict with ours. Elevated kindlin-2 levels have been reported to increase renal and pulmonary

fibrosis (10, 11). A tissue/cellular and experimental fibrosis study reported increased kindlin-2 expression in fibrotic liver tissue via TGF-β (12), in contrast to our finding of lower serum levels. Other experimental or clinical studies did not support this pathway. Unlike previous in vivo studies using different transgenic and fibrotic animal models, which reported inconsistent findings regarding kindlin-2 levels, our study offers a different perspective (10-12). These initial clinical findings in cirrhosis and the differing reports from other tissues are particularly noteworthy.

The cross-sectional case-control design is a limitation of our study. Cross-sectional studies have limited ability to establish causal relationships and reflect only a specific point in time. Further studies of liver cirrhosis and the preceding stage of hepatosteatosis may clarify the relationship between kindlin-2 and cirrhosis. Long-term changes in serum kindlin-2 levels and their relationship with clinical outcomes have not been studied; therefore, the diagnostic and prognostic value of kindlin-2 remains

Table 2. Univariate and Multivariate Regression Analyses of Factors Associated with Kindlin-2 Levels ^a

Variables	Univariate ^b		Multivariate ^b					
	β	P Value	B	SE	β	95% CI Lower	95% CI Upper	P Value
Age	-0.137	0.214						
Gender	-0.188	0.087						
Liver fibrosis	-0.362	< 0.001	-0.277	0.130	-0.238	-0.535	-0.019	0.036
ALT	-0.251	0.022						
AST	-0.287	0.008						
ALP	-0.174	0.121						
GGT	-0.070	0.535						
Direct bilirubin	-0.165	0.115						
Total bilirubin	-0.194	0.080						
Albumin	-0.105	0.387						
INR	-0.213	0.055						
Platelet	0.266	0.015						
Leukocyte	0.135	0.219						
CRP	-0.066	0.557						
APRI	-0.393	< 0.001	-0.096	0.037	-0.288	-0.170	-0.022	0.011
FIB-4	-0.197	0.080						
MELD-Na	-0.216	0.051						

^a Abbreviations: ALP, alkaline phosphatase; ALT, alanine aminotransferase; APRI, aspartate aminotransferase-to-platelet ratio index; AST, aspartate aminotransferase; CI, confidence interval; CRP, C-reactive protein; FIB-4, fibrosis-4 score; GGT, gamma-glutamyl transferase; INR, international normalized ratio; MELD-Na, Model for End-Stage Liver Disease-Sodium; SE, standard error.

^b Serum kindlin-2 levels were logarithmically transformed before statistical analysis to improve their distributional properties.

Table 3. Logistic Regression Analysis of Variables Associated with Liver Cirrhosis ^a

Variables	B	SE	OR	95% CI Lower	95% CI Upper	P Value
Kindlin-2	-0.337	0.141	0.714	0.541	0.942	0.017
ALT	0.016	0.063	1.016	0.897	1.150	0.063
Platelet	-0.027	0.000	0.973	0.956	0.991	0.004
Albumin	-0.311	0.147	0.718	0.538	0.959	0.025
Age	0.083	0.047	1.086	0.990	1.192	0.081
Gender	-0.112	1.201	0.894	0.085	9.402	0.925

^a Abbreviations: ALT, alanine aminotransferase; B, beta coefficient; CI, confidence interval; OR, odds ratio; SE, standard error.

uncertain. In our study, patients with cirrhosis were significantly older and more often male than controls. However, univariate regression analysis showed no association between kindlin-2 levels and age or gender, and logistic regression analysis showed no association between age or gender and cirrhosis.

5.1. Conclusions

Low serum kindlin-2 levels were associated with liver cirrhosis. Correlations were observed between kindlin-2 levels and markers of liver fibrosis, as well as certain liver function tests. These findings may be a consequence of the

cirrhosis process. Our findings will guide future research aimed at clarifying the role of kindlin-2 in cirrhosis.

Footnotes

AI Use Disclosure: The authors declare that no generative AI tools were used in the creation of this article.

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