Relationship Between Fibrinogen, ISKDC Pathological Grading, and Nephron Microscopic Lesions in Children with Henoch-Schönlein Purpura Nephritis: A Postprint Study

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Abstract

Background: In clinical practice, children with Henoch-Schönlein purpura nephritis (HSPN) frequently present with elevated fibrinogen (FIB) levels; however, investigations into the correlation between FIB and renal pathological changes remain limited.

Objective: To explore the correlation between FIB and the International Study of Kidney Disease in Children (ISKDC) pathological grading as well as microscopic pathological alterations in renal units, and to ascertain whether FIB can evaluate the severity of renal injury in pediatric HSPN patients.

Methods: A cohort of 922 children with HSPN who underwent renal biopsy and were hospitalized in the Pediatric Nephrology Ward of the First Affiliated Hospital of Henan University of Chinese Medicine between December 2017 and December 2022 was retrospectively collected. Clinical data, FIB levels, and renal pathological information during the renal biopsy period were compiled. Patients were stratified into Group A (low) <2.38 g/L, Group B (standard) 2.38–4.98 g/L, and Group C (high) >4.98 g/L based on FIB levels. Spearman rank correlation analysis was employed to investigate the association between FIB and ISKDC pathological grading, glomerular mesangial hyperplasia proportion, and crescent proportion. Receiver operating characteristic (ROC) curve analysis was subsequently utilized to assess the predictive performance of FIB for microscopic pathological changes in renal units.

Results: Among the 922 HSPN children who underwent renal biopsy, the mean FIB level was (3.48$±$1.01) g/L. Group A comprised 113 cases, representing a low FIB rate of 12.26%; Group B comprised 734 cases, representing a standard FIB rate of 79.61%; and Group C comprised 75 cases, representing a high

FIB rate of 8.13%. Regarding ISKDC pathological classification, there were 173 cases of type IIa (18.76%), 29 cases of type IIb (3.15%), 466 cases of type IIIa (50.54%), 232 cases of type IIIb (25.16%), and 22 cases of type IV and above (2.39%) (including 2 cases of type IVa, 18 cases of type IVb, and 2 cases of type V). Spearman rank correlation analysis revealed that both FIB levels and FIB grouping were positively correlated with renal pathological ISKDC grading (rs=0.146, P<0.001; rs=0.129, P<0.001). Among the 922 HSPN children, 911 cases (98.9%) exhibited mesangial cell proliferation and 655 cases (71.04%) exhibited crescent formation. Spearman rank correlation analysis demonstrated that both FIB and FIB grouping showed weak positive correlations with mesangial cell proliferation rate (rs=0.092, P=0.005; rs=0.096, P=0.003) and positive correlations with crescent formation rate (rs=0.132, P<0.001; rs=0.83, P=0.012). Among the 922 HSPN children, 763 cases (82.75%) presented with acute glomerular lesions, 97 cases (10.52%) with acute-chronic lesions, and 62 cases (6.73%) with chronic lesions. FIB levels in HSPN children were positively correlated with the acute-chronic status of glomerular lesions (rs=0.145, P<0.001). Additionally, comparisons between FIB and certain renal biopsy pathological indicators (crescent formation, mesangial hyperplasia with lobulation, glomerular sclerosis, capsular adhesion, tubulitis or regeneration, tubular cell granular degeneration, tubular interstitial edema, tubular inflammatory cell infiltration, tubular atrophy, tubular interstitial fibrosis, tubular lumen red blood cell casts, and renal interstitial vessels) revealed statistically significant differences (P<0.05). ROC curve analysis indicated that FIB exhibited the highest sensitivity for glomerular sclerosis (sensitivity=0.900, specificity=0.303), with an optimal cutoff value of 2.835 mg/L. The area under the ROC curve (AUC) for FIB in positively predicting tubular interstitial fibrosis was 0.623, while the AUC for negatively predicting tubular cell granular degeneration was 0.641.

Conclusion: FIB can serve as a laboratory indicator that reflects the severity of renal pathological changes in children with HSPN. It can reflect the severity of renal pathological grading and is closely associated with microscopic indicators of renal units such as glomerular sclerosis and capsular adhesion, thereby assisting in clinical diagnosis and treatment.

Full Text

The Relationship between Fibrinogen and International Pediatric Nephrology Study Group Pathologic Grading and Microscopic Lesions of Renal Units with Henoch-Schönlein Purpura Nephritis in Children

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Abstract

Background: Fibrinogen (FIB) is frequently elevated in children with Henoch-Schönlein purpura nephritis (HSPN), yet the correlation between FIB levels and renal lesions remains understudied. **Objective:** This study investigates the relationship between FIB and International Study of Kidney Disease in Children (ISKDC) pathological grading and micropathological changes in renal units, aiming to determine whether FIB can assess the severity of renal injury in pediatric HSPN patients. **Methods:** We enrolled 922 children with HSPN who underwent renal biopsy in the pediatric nephrology ward of the First Affiliated Hospital of Henan University of Traditional Chinese Medicine between December 2017 and December 2022. Clinical data, FIB levels, and renal pathology findings were collected during the biopsy period. Patients were stratified into three groups based on FIB levels: Group A (low) <2.38 g/L, Group B (standard) 2.38–4.98 g/L, and Group C (high) >4.98 g/L. Spearman rank correlation analysis examined associations between FIB and ISKDC grading, glomerular mesangial hyperplasia ratio, and crescent formation ratio. Receiver operating characteristic (ROC) curves evaluated FIB's predictive value for micropathological changes. **Results:** Among 922 biopsied HSPN patients, mean FIB was (3.48$±$1.01) g/L. Group A comprised 113 cases (12.26% low FIB), Group B 734 cases (79.61% standard FIB), and Group C 75 cases (8.13% high FIB). ISKDC pathology distribution was: Type IIa 173 cases (18.76%), Type IIb 29 cases (3.15%), Type IIIa 466 cases (50.54%), Type IIIb 232 cases (25.16%), and Type IV or above 22 cases (2.39%) (including 2 cases of Type IVa, 18 cases of Type IVb, and 2 cases of Type V). Spearman analysis revealed positive correlations between FIB (and FIB grouping) and ISKDC grading (rs=0.146, P<0.001; rs=0.129, P<0.001). Mesangial proliferation occurred in 911 cases (98.9%), and crescent formation in 655 cases (71.04%). FIB showed weak positive correlation with mesangial hyperplasia rate (rs=0.092, P=0.005; rs=0.096, P=0.003) and positive correlation with crescent formation rate (rs=0.132, P<0.001; rs=0.83, P=0.012). Glomerular lesions were acute in 763 cases (82.75%), acute-chronic in 97 cases (10.52%), and chronic in 62 cases (6.73%). FIB correlated positively with lesion chronicity (rs=0.145, P<0.001). Multiple biopsy indicators (crescent formation, mesangial hyperplasia lobulation, glomerular sclerosis, capsular adhesion, tubular inflammation/regeneration, tubular cell granular degeneration, tubular interstitial edema, tubular inflammatory cell infiltration, tubular atrophy, tubulointerstitial fibrosis, tubular red blood cell casts, renal interstitial vascular abnormalities) showed significant associations with FIB (P<0.05).

ROC analysis demonstrated highest sensitivity for glomerulosclerosis (sensitivity=0.900, specificity=0.303) at an optimal FIB cutoff of 2.835 mg/L. The area under the curve (AUC) was 0.623 for positive prediction of tubulointerstitial fibrosis and 0.641 for reverse prediction of tubular cell granular degeneration. **Conclusion:** FIB serves as a valuable laboratory indicator reflecting the severity of renal pathological changes in HSPN patients. It correlates with pathological grading and shows strong associations with irreversible microscopic lesions such as glomerular sclerosis and capsular adhesion, offering clinical utility for diagnosis and treatment guidance.

Keywords: Nephritis; Henoch-Schönlein purpura nephritis; Pathological grade; Coagulation index; Fibrinogen

Introduction

Henoch-Schönlein purpura nephritis (HSPN) represents one of the more common secondary glomerular diseases in childhood, characterized primarily by renal damage manifesting as hematuria and proteinuria. Research indicates that 1–7% of HSPN patients develop renal insufficiency, with some progressing to end-stage renal disease (ESRD), posing serious threats to pediatric health and representing a high-risk population for chronic kidney disease and ESRD in adulthood. This creates substantial psychological and economic burdens for families and society. Renal biopsy remains the gold standard for diagnosing HSPN, determining pathological classification, and predicting prognosis. However, the procedure carries high costs, bleeding risks, and technical challenges, particularly in children with small kidneys and poor cooperation, limiting its universal clinical application. Consequently, identifying high-diagnostic-efficiency laboratory indicators that can supplement renal pathological diagnosis has become a research priority.

Hemorheological abnormalities may contribute to HSPN pathogenesis. Early domestic studies demonstrated that coagulation indices such as fibrinogen (FIB) could reflect renal injury severity, while other research reported significant correlations between systemic hypercoagulability during the acute phase of Henoch-Schönlein purpura and renal damage. Clinical observations suggest coagulation indicators correlate with pathological changes in HSPN patients. As a crucial precursor to thrombus formation, elevated FIB indicates hypercoagulability. This study examines the relationship between FIB and renal pathological indices to further elucidate how hypercoagulability affects renal pathology and glomerular injury.

Methods

Study Design and Population

We retrospectively collected data from 922 HSPN children who underwent renal biopsy in the pediatric nephrology ward of the First Affiliated Hospital of

Henan University of Traditional Chinese Medicine between December 2017 and December 2022. From an initial cohort of 1,056 patients with renal pathology records, we excluded 134 cases. The final sample included 533 males and 389 females, with a mean age of (10.5$±$3.3) years. We compiled comprehensive data including demographic information, renal biopsy pathology reports, traditional Chinese and Western medicine diagnoses, and six coagulation parameters (focusing on FIB) measured during hospitalization. Pathological specimens were evaluated using ISKDC grading standards.

Inclusion and Exclusion Criteria

Inclusion criteria: (1) Age 3–18 years; (2) Met diagnostic criteria for HSPN in both traditional Chinese and Western medicine; (3) Complete laboratory indicators; (4) Underwent renal biopsy with complete pathological data.

Exclusion criteria: (1) Incomplete clinical or pathological data; (2) Renal biopsy specimens containing $ $5 glomeruli; (3) Recent use of glucocorticoids, anticoagulants, or antiplatelet drugs like aspirin, or history of coagulation disorders; (4) Primary IgA nephropathy, thrombocytopenic purpura, systemic lupus erythematosus, or other systemic diseases; renal damage caused by hepatitis B, hepatitis C, syphilis, HIV; ANCA-associated small vasculitis, or other secondary renal diseases.

Diagnostic Criteria

Clinical diagnosis followed the 2016 Evidence-Based Guidelines for HSPN Diagnosis and Treatment: (1) Hematuria and/or proteinuria occurring during Henoch-Schönlein purpura course or within six months of purpura resolution; (2) Renal pathology consistent with HSPN; (3) Exclusion of renal damage from lupus, vasculitis, or other diseases.

Pathological diagnosis used ISKDC grading [8], which classifies glomerular crescentic lesions into grades I–VI. Grade I shows minimal glomerular abnormalities; Grade II shows pure mesangial proliferation; Grade III shows <50% of glomeruli with crescent formation and/or segmental lesions with focal segmental or diffuse mesangial proliferation; Grade IV shows similar lesions involving 50–75% of glomeruli; Grade V involves >75% of glomeruli; and Grade VI indicates membranoproliferative glomerulonephritis. Focal segmental lesions affect <50% of glomeruli, while diffuse lesions exceed 50%. All pathological specimens were re-evaluated by experienced pathologists from the pediatric laboratory (national tertiary laboratory) at our hospital, blinded to clinical information.

Detection Methods

Coagulation Index Detection: Fasting venous blood (2 mL) was collected in sodium citrate anticoagulation tubes from hospitalized HSPN children. After centrifugation at 3,000 r/min for 15 minutes, plasma FIB levels were measured using the STA-REvolution automated coagulation analyzer with 配套试剂. The

reference range at our hospital laboratory is 2.38–4.98 g/L, forming the basis for group stratification.

Renal Pathology Detection: Renal biopsy specimens obtained during hospitalization were processed according to standard requirements. Samples underwent hematoxylin-eosin staining, periodic acid-Schiff staining, periodic acid-silver methenamine staining, and MASSON staining. Light microscopy examined glomerular number, mesangial cell proliferation, crescent formation, glomerular sclerosis, podocyte hypertrophy, endothelial cell proliferation, endothelial thrombosis, basement membrane thickening, capsular thickening, capsular stratification, capsular rupture, and extraglomerular fibrosis. Each patient had calculated mesangial proliferation rate (proliferating glomeruli/total biopsied glomeruli $×$100%) and crescent formation rate (cellular + cellular fibrous + fibrocellular + fibrous crescents/total biopsied glomeruli $×$100%).

Statistical Analysis

We performed statistical analysis using SPSS 26.0 and GraphPad Prism 8.0. Continuous variables underwent normality testing; normally distributed data were expressed as mean±standard deviation ($\bar{x}\pm s$) and compared using t-tests, while non-normally distributed data were expressed as median (P25, P75) and analyzed using Mann-Whitney U test for two groups or Kruskal-Wallis test for three or more groups. Categorical data were expressed as percentages. Correlation analysis employed Spearman rank correlation. ROC curves determined optimal diagnostic cutoffs for FIB in predicting glomerular lesions, calculating sensitivity, specificity, and accuracy. Statistical significance was defined as $P<0.05$.

Results

FIB Levels in Relation to Age and Sex

Among 922 biopsied HSPN patients, no significant sex difference in FIB levels was observed. However, age-related differences were significant ($P<0.001$). Children aged 3–8 years had lower FIB than those aged 9–14 years and 14–18 years ($P<0.001$ and $P=0.009$, respectively). Spearman analysis confirmed positive correlation between FIB and age distribution ($rs=0.151$, $P<0.001$).

Correlation between FIB and ISKDC Pathological Grading

Mean FIB was (3.48$±$1.01) g/L. Group A (low FIB) included 113 cases (12.26%), Group B (standard) 734 cases (79.61%), and Group C (high) 75 cases (8.13%). ISKDC pathology distribution showed Type IIa in 173 cases (18.76%), Type IIb in 29 cases (3.15%), Type IIIa in 466 cases (50.54%), Type IIIb in 232 cases (25.16%), and Type IV or above in 22 cases (2.39%) (including 2 Type IVa, 18 Type IVb, and 2 Type V). Significant differences in ISKDC grading existed across FIB groups ($P<0.001$). Spearman analysis revealed positive

correlations between FIB (and FIB grouping) and ISKDC grading (rs=0.146, P<0.001; rs=0.129, P<0.001).

Correlation with Mesangial Cell Proliferation and Crescent Formation Rates

Mesangial proliferation occurred in 911 cases (98.9%), and crescent formation in 655 cases (71.04%). Both mesangial proliferation rate and crescent formation rate differed significantly across FIB groups (P<0.05). Further Spearman analysis showed weak positive correlation between FIB (and FIB grouping) and mesangial proliferation rate (rs=0.092, P=0.005; rs=0.096, P=0.003), and positive correlation with crescent formation rate (rs=0.132, P<0.001; rs=0.83, P=0.012).

Correlation with Acute and Chronic Glomerular Lesions

Glomerular lesions were acute in 763 cases (82.75%), acute-chronic in 97 cases (10.52%), and chronic in 62 cases (6.73%). Distribution across FIB groups differed significantly (P=0.007). Spearman analysis confirmed positive correlation between FIB and lesion chronicity (rs=0.145, P<0.001).

FIB and Renal Injury Indicators

Various microscopic lesions showed significant FIB differences (P<0.05): crescent formation (654 cases, 70.93%), mesangial hyperplasia lobulation (361 cases, 39.15%), glomerular sclerosis (100 cases, 10.85%), capsular adhesion (246 cases, 26.68%), tubular inflammation/regeneration (61 cases, 6.62%), tubular cell granular degeneration (895 cases, 97.07%), tubular interstitial edema (299 cases, 32.43%), tubular inflammatory cell infiltration (352 cases, 38.18%), tubular atrophy (137 cases, 14.86%), tubulointerstitial fibrosis (163 cases, 17.68%), tubular red blood cell casts (360 cases, 39.05%), and renal interstitial vascular abnormalities (33 cases, 3.58%). No significant FIB differences were found for fibrous crescents, podocyte hypertrophy, endothelial thrombosis, basement membrane thickening, capsular thickening, or tubular protein casts (P>0.05).

ROC Curve Analysis

ROC curves for FIB predicting various lesions showed highest sensitivity for glomerulosclerosis (sensitivity=0.900, specificity=0.303) with an optimal cutoff of 2.835 mg/L. The AUC was 0.623 for positive prediction of tubulointerstitial fibrosis and 0.641 for reverse prediction of tubular cell granular degeneration.

Discussion

The etiology and pathogenesis of HSPN remain incompletely understood, with proposed mechanisms including hemorheological abnormalities, inflammatory responses, endothelial injury, immune dysfunction, and complement activation.

Professor Ding Ying's clinical observations revealed hypercoagulability in many HSPN patients. FIB, the precursor to fibrin, plays a critical role in the final coagulation stage by converting soluble FIB to insoluble fibrin. Our analysis demonstrates that FIB levels increase with worsening ISKDC pathology and patient age, correlating positively with mesangial proliferation and crescent formation rates.

Crescents form when parietal epithelial cells (PECs) lining Bowman's capsule proliferate extensively, creating crescent-shaped masses around capillary tufts. While plasma components may promote PEC proliferation, only FIB formation has been consistently demonstrated across studies. Hypercoagulability from elevated FIB slows blood flow in glomerular capillaries, and when capillary loops are damaged, FIB leaks into Bowman's space, stimulating PEC proliferation and mononuclear macrophage infiltration that drives crescent formation. Early intervention can promote lesion resolution and delay progression; without timely treatment, these lesions progress to fibrous crescents, leading to irreversible changes including glomerulosclerosis, capsular adhesion, tubulointerstitial fibrosis, and tubular atrophy.

Mesangial hyperplasia lobulation occurs when proliferating mesangial tissue invades capillaries, thickening walls and narrowing lumens, creating lobulated glomerular tufts. Hypercoagulability may also promote adhesion between Bowman's capsule and glomerular capillaries (capsular adhesion), stretching capillary loops. Studies show elevated FIB increases cardiovascular disease risk and may worsen kidney disease. Our data suggest increased FIB may heighten capsular adhesion risk in HSPN. Glomerulosclerosis, an irreversible outcome of various glomerular diseases, results from capillary lumen narrowing, native cell loss, and collagen deposition. Research indicates glomerular FIB immune complex deposition reflects IgA nephropathy severity, activity, and long-term prognosis. Our findings show FIB increases as glomerular lesions transition from active to chronic, with chronic stages showing more irreversible lesions like fibrous crescents and glomerulosclerosis, supporting FIB's utility for assessing irreversible renal damage. Notably, FIB showed high sensitivity (0.900) for detecting glomerulosclerosis, highlighting its importance for timely identification and prevention.

Xie et al. reported FIB levels independently correlate with crescent formation and tubular atrophy. Our data similarly demonstrate FIB's independent associations with both glomerular lesions (crescent formation, glomerulosclerosis, capsular adhesion) and tubular lesions (tubular inflammation/regeneration, tubular cell granular degeneration, tubular interstitial edema, tubular inflammatory cell infiltration, tubular atrophy, tubulointerstitial fibrosis, tubular red blood cell casts, renal interstitial vascular abnormalities).

The complex renal architecture and limited Bowman's space mean hypercoagulability and slowed filtrate flow exacerbate intraglomerular pathology. FIB and renal microlesions appear mutually reinforcing—lesions worsen hypercoagulability, which in turn aggravates pathology. Glomerular reperfusion occurs

via mechanical and charge barriers, and capillary collapse may delay plasma protein uptake, indirectly explaining positive correlations between FIB and microlesion indicators. HSPN pathology primarily involves IgA-dominant immune complex deposition in mesangial and capillary regions, and mesangial proliferative glomerulonephritis may relate to immune complex deposition influenced by FIB.

Our ROC analysis revealed optimal FIB cutoffs of 2.83–4.78 g/L for various microlesions—all within the normal reference range (2.38–4.98 g/L). This suggests that even "normal" FIB levels in HSPN patients warrant anticoagulant and blood-activating therapy to prevent progression to irreversible lesions and reduce microscopic damage.

In conclusion, FIB serves as a valuable laboratory indicator for assessing HSPN pathological classification and renal microlesions. Clinicians can monitor FIB levels in non-biopsied patients to adjust treatment strategies, reduce nephron injury, and prevent deterioration toward irreversible pathology.

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Note: Figure translations are in progress. See original paper for figures.