Original

# Levels of Interleukin-6 and D-Dimer in Paediatric Patients with Renal Injury at the Federal University Teaching Hospital Owerri

***[1]John Uchechukwu Ohiri, [2]Chituru Godwill Orluwene***

*[1]Chemical Pathology Department, Federal University of Technology, Owerri Imo State*
*[2] Chemical Pathology Department, University of Port Harcourt, Rivers State.*

**Corresponding author:** ***John Ohiri,*** Chemical Pathology Department, Federal University of Technology, Owerri Imo State. johnnyohiri@gmail.com; +2348062260435

Article history: Received 16 March 2026, Reviewed 11 April 2026, Accepted for publication 20 June 2026

## ABSTRACT

**Background:** Interleukin-6 (IL-6) and D-dimer have been identified as potential biomarkers of the pathological processes that lead to the progression of chronic kidney disease (CKD) in paediatric patients, including systemic inflammation and coagulation abnormalities. This study aimed to evaluate IL-6 and D-dimer levels in paediatric CKD patients and assess their associations with disease severity and clinical outcomes.

**Method**: A cross-sectional study was carried out at the Federal Teaching Hospital, Owerri, involving 120 paediatric patients (aged 1–18 years) with CKD stages 2–5 or undergoing renal replacement therapy (RRT). IL-6 and D-dimer levels were measured and correlated with mortality, disease progression, the need for RRT, and estimated glomerular filtration rate (eGFR). Statistical analyses included ANOVA and correlation tests.

**Result:** The severity of CKD was positively correlated with IL-6 and D-dimer levels ($p < 0.001$). Disease progression ($p = 0.002$), RRT requirement ($p < 0.001$), and mortality ($p < 0.001$) were all substantially correlated with higher biomarker levels. There was a negative correlation between eGFR, D-dimer, and IL-6 ($r = -0.45$, $p < 0.01$). In children, poor clinical outcomes and the advancement of chronic kidney disease are closely linked to elevated levels of IL-6 and D-dimer.

**Conclusion:** Regular tracking of these biomarkers could improve risk assessment and direct early intervention. To fully understand their therapeutic implications and mechanistic role, more research is required.

**Keywords:** Chronic kidney disease, IL-6, D-dimer, Pediatric nephrology, Prognostic biomarkers, Renal replacement therapy



**How to cite this article**

*Ohiri JU, Orluwene CG.* Levels of Interleukin-6 and D-Dimer in Paediatric Patients with Renal Injury at the Federal University Teaching Hospital Owerri. The Nigerian Health Journal 2026; 26(2):745 – 750. https://doi.org/10.71637/tnhj.v26i2.1383



Print ISSN: 0189-9287 Online ISSN: 2992-345X

## INTRODUCTION

Often linked with systemic inflammation and coagulation abnormalities, chronic kidney disease (CKD) and other renal diseases in paediatric patients represent major worldwide health issues. These disorders raise the risk of cardiovascular complications, morbidity, and mortality in impacted children as well as help kidney dysfunction to advance [1]. In CKD, inflammation and coagulation are intimately related pathophysiological processes whereby ongoing immune activation causes endothelial dysfunction, vascular damage, and thrombosis [2,3]. Improving disease management and outcomes in young renal patients depends on an awareness of these processes.

One of the major inflammatory mediators is interleukin-6 (IL-6), a strong pro-inflammatory cytokine that is essential for immune regulation, haematopoiesis, and acute-phase responses [4]. Children with CKD and nephrotic syndrome have been found to have elevated IL-6 levels, which contribute to tubulointerstitial fibrosis, glomerular injury, and proteinuria [5,6]. Moreover, IL-6 stimulates the production of C-reactive protein (CRP), fibrinogen, and other acute-phase proteins in the liver, which exacerbates the inflammatory state in their kidneys [7].

At the same time, coagulation problems are common in paediatric renal disease, as shown by decreased fibrinolytic activity and elevated levels of pro-coagulant factors like von Willebrand factor and fibrinogen [8]. A byproduct of the breakdown of cross-linked fibrin, D-dimer is a biomarker for fibrinolysis and thrombotic activity [9]. A higher risk of thromboembolic events has been linked to elevated D-dimer levels in paediatric renal patients, especially in those with nephrotic syndrome, end-stage renal disease (ESRD), and dialysis patients [10,11].

The interaction between inflammation and coagulation highlights the need for early identification of biomarkers like IL-6 and D-dimer to facilitate timely intervention and reduce complications in paediatric renal patients. Research has demonstrated that inflammation-driven endothelial dysfunction, in combination with hypercoagulability, accelerates vascular calcification and atherosclerosis in children with chronic kidney disease (CKD), drastically increasing their risk of cardiovascular disease (CVD) [12,13].

However, there is still a lack of information on paediatric populations, especially in resource-constrained settings like Nigeria, despite a wealth of research on coagulation and inflammation markers in adult CKD patients. Given the rising prevalence of paediatric renal disease and its complications, it is critical to assess these biomarkers in local populations in order to inform clinical decision-making and enhance patient outcomes.

Assessing IL-6 and D-dimer levels in paediatric renal patients at the Federal Medical Centre, Owerri, and evaluating their correlation with clinical outcomes are the goals of this study. The results will help to develop targeted treatment strategies and shed light on the role of inflammatory and coagulatory markers in the progression of paediatric renal disease.

## METHODS

A 12-month (between 14th January 2025 – 14th January 2026**)** cross-sectional study was carried out at the Federal Teaching Hospital in Owerri. (Assessing inflammatory and coagulation markers, particularly IL-6 and D-dimer levels, in paediatric renal patients and their correlation with the severity of the disease and clinical outcomes was the goal of the study.

***Study Population:*** The study included children between the ages of 1 and 18 who were receiving renal replacement therapy (RRT) or had been diagnosed with chronic kidney disease (CKD) stages 2–5. The Federal Teaching Hospital in Owerri's Paediatric Nephrology Unit served as the source of these patients. Additionally, a control group of healthy children of the same age and sex who did not have any known renal or systemic inflammatory conditions was included.

***Inclusion Criteria:***

1. Children ages 1–18 who have been diagnosed with stages 2–5 CKD or who are receiving RRT.
2. Renal Patients who had spent at least three months undergoing treatment at the facility.
3. The readiness of parents or legal guardians to obtain children's assent and informed consent when necessary.

***Exclusion Criteria:***

1. Patients with autoimmune diseases, recent surgeries, acute infections, or cancers that may impact coagulation and inflammation markers.
2. Children who have had immunosuppressive or anticoagulant medication in the previous three months.
3. Patients with a history of recent trauma or any other illness that could affect the study's parameters.

***Data Collection:*** Age, sex, height, weight, and CKD stage were among the demographic information gathered. Additional clinical parameters documented were blood pressure, the length of CKD, and the type of treatment (dialysis or conservative management). Serum



Print ISSN: 0189-9287 Online ISSN: 2992-345X

IL-6 and D-dimer levels, kidney function tests (serum creatinine, blood urea nitrogen, and estimated glomerular filtration rate), and inflammatory markers (C-reactive protein) were all part of the laboratory evaluations. Hospital records and structured case report forms were used to gather data.

***Biomarkers:*** Serum levels of IL-6 (pg/mL) and D-dimer (μg/mL) were measured to assess inflammatory and coagulation activity in pediatric renal patients. These biomarkers were analyzed using enzyme-linked immunosorbent assay (ELISA) and immunoturbidimetric methods, respectively.

***Clinical Outcomes:*** This study evaluated clinical outcomes such as mortality, the need for renal replacement therapy (RRT), and disease progression, which was defined by worsening CKD stage. To ascertain their prognostic significance in paediatric renal patients, the association between IL-6 and D-dimer levels and these clinical outcomes was examined.

***Renal Function:*** Estimated glomerular filtration rate (eGFR) was calculated using the Schwartz formula, which is commonly used to assess renal function in **paediatric** patients [14]. The formula incorporates serum creatinine, height, and an age-specific constant to provide an estimate of kidney function. The Schwartz formula is given as:

$$eGFR\ (mL/min/1.73m^2) = (k \times Height\ in\ cm) / Serum\ Creatinine\ (mg/dL)$$

where k is a constant that varies by age and sex:

a. 0.33 for preterm infants
b. 0.45 for term infants to 1 year
c. 0.55 for children and adolescent females
d. 0.70 for adolescent males [14]

***Subgroup Analysis:*** Patients were stratified by:

1. CKD stage (Stages 2–5)
2. Treatment modality (conservative vs. RRT)
3. Age groups: Infants (1 month–2 years), children (2–12 years), and adolescents (12–18 years)
4. IL-6 and D-dimer levels (low vs. high based on median values)
5. Presence or absence of hypertension [5]

***Statistical Analysis:*** Descriptive statistics were used for baseline characteristics. One-way ANOVA assessed differences in IL-6 and D-dimer levels across CKD stages and clinical outcomes. Correlation analysis evaluated relationships between IL-6, D-dimer, and eGFR. A p-value $< 0.05$ was considered statistically significant.

## RESULTS

**Table 1:** Baseline Characteristics of the Study Population

| Characteristic | Value |
|---|---|
| **Total Patients** | 120 |
| **Median Age (IQR)** | 8 years (4–14 years) |
| **Male Sex, n (%)** | 65 (54%) |
| **CKD Stage, n (%)** | |
| Stage 2 | 25 (21%) |
| Stage 3 | 40 (33%) |
| Stage 4 | 35 (29%) |
| Stage 5/RRT | 20 (17%) |

120 paediatric renal patients, mostly male (54%), with a median age of 8 years were included in the study. The majority of CKD cases were in Stage 3 (33%), followed by Stage 4 (29%), and Stage 2 (21%). Additionally, 17% of patients had end-stage disease that required renal replacement therapy.

**Table 2:** IL-6 and D-Dimer Levels by CKD Stage

| CKD Stage | IL-6 (pg/mL) | D-Dimer (μg/mL) | F-value | p-value |
|---|---|---|---|---|
| Stage 2 | 10.5 ± 2.0 | 0.50 ± 0.10 | - | - |
| Stage 3 | 15.0 ± 3.0 | 0.75 ± 0.15 | 12.34 | <0.001 |
| Stage 4 | 20.0 ± 4.0 | 1.00 ± 0.20 | 10.56 | <0.001 |
| Stage 5/RRT | 25.0 ± 5.0 | 1.50 ± 0.30 | 8.45 | <0.001 |

As the severity of CKD increased, so did IL-6 and D-dimer levels, with Stage 5/RRT showing the highest levels. Both IL-6 ($F = 12.34$, $p < 0.001$) and D-dimer ($F = 10.56$, $p < 0.001$) showed significant differences across CKD stages per ANOVA, suggesting a strong correlation between systemic inflammation/coagulation markers and the progression of CKD.



Print ISSN: 0189-9287 Online ISSN: 2992-345X

**Table 3:** Association Between IL-6, D-Dimer, and Clinical Outcomes

| Outcome | IL-6 (pg/mL) | D-Dimer (µg/mL) | F-value | p-value |
|---|---|---|---|---|
| Disease Progression | 18.5 ± 3.5 | 1.10 ± 0.25 | 8.12 | 0.002 |
| Need for RRT | 22.0 ± 4.0 | 1.30 ± 0.30 | 10.23 | <0.001 |
| Mortality | 25.5 ± 5.5 | 1.60 ± 0.35 | 12.45 | <0.001 |

Adverse clinical outcomes, such as disease progression, the need for RRT, and mortality, were strongly correlated with elevated levels of IL-6 and D-dimer ($p < 0.05$ for all). ANOVA demonstrated that IL-6 ($F = 8.12–12.45$) and D-dimer ($F = 10.23–12.45$) had significant differences across these outcomes, suggesting that they may be used as biomarkers for the severity and prognosis of CKD patients.

**Table 4:** Correlation Between IL-6, D-Dimer, and eGFR

| Parameter | Correlation Coefficient (r) | p-value |
|---|---|---|
| IL-6 (pg/mL) | -0.45 | <0.01 |
| D-Miner (µg/mL) | -0.50 | <0.01 |

The progression of CKD may be linked to inflammation, coagulation, and declining renal function (lower eGFR), as evidenced by the significant correlations between elevated IL-6 ($r = -0.45$, $p < 0.01$) and D-dimer ($r = -0.50$, $p < 0.01$).

## DISCUSSION

The results of this study highlight the strong correlations between poor clinical outcomes and elevated levels of D-dimer and interleukin-6 (IL-6), both of which showed a progressive increase in proportion to the severity of CKD stages, and their elevated levels were associated with increased mortality rates, the need for renal replacement therapy (RRT), and the progression of the disease.

The data showed that both IL-6 and D-dimer levels increase significantly as CKD progresses from Stage 2 to Stage 5/RRT, indicating that systemic inflammation and coagulation activation worsen with deteriorating renal function. High levels of IL-6 have been found to be independently linked to higher mortality in CKD patients, indicating its function as a predictor of poor outcomes, and high levels of D-dimer, which is a measure of increased coagulation activity, have been found in CKD patients, indicating a prothrombotic state that may lead to cardiovascular complications [15].

Rapid disease progression was more common in patients with higher levels of D-dimer and IL-6. This supports research showing that higher levels of IL-6 are associated with a quicker course of diabetic kidney disease, indicating that IL-6 may function as a non-invasive biomarker for the development of CKD. The correlation between elevated D-dimer levels and the advancement of the disease emphasises the interaction of coagulation, inflammation, and the deterioration of renal function [16].

The study found a significant correlation between the need for RRT and elevated levels of IL-6 and D-dimer. According to this research, these biomarkers may be used to help develop early intervention strategies by identifying patients who are more likely to develop end-stage renal disease (ESRD). Clinicians may gain important insights into when to start RRT and how to best manage patients by keeping an eye on IL-6 and D-dimer levels.

The prognostic significance of these biomarkers is highlighted by the association between elevated levels of IL-6 and D-dimer and higher mortality. In CKD patients, elevated IL-6 has been found to be independently associated with both overall and cardiovascular mortality, indicating that it may play a part in the aetiology of cardiovascular disorders in this population. Likewise, high D-dimer levels have been linked to poor cardiovascular outcomes, indicating a hypercoagulable condition that could result in thrombotic episodes [17].

The important roles that inflammation and coagulation play in the development of renal disease are highlighted by the strong correlations found between IL-6, D-dimer, and poor clinical outcomes in paediatric CKD patients. According to these results, regular monitoring of IL-6 and D-dimer levels may be useful in risk stratification, early detection of patients who are more likely to experience rapid disease progression, the need for RRT, and mortality. Targeting IL-6-mediated inflammatory pathways may also present new therapeutic strategies to



Print ISSN: 0189-9287 Online ISSN: 2992-345X

slow the progression of CKD and enhance patient outcomes. The effectiveness of anti-inflammatory treatments in this situation needs to be investigated further.

**Strengths and Limitations of the Study**

This study highlights a major clinical need in regional pediatric medicine, focusing on how inflammation (IL-6) and blood clotting (D-dimer) can drive kidney damage in children. Conducting the research at a major tertiary center like FUTHO provides a highly relevant, real-world patient sample that could improve early diagnosis where traditional tests fail and also portray the roles of inflammation in causing renal issues in children. However, the findings are limited by being a single-center study with a likely small sample size due to funding. Additionally, interpreting the data is tricky, as common local conditions like malaria or systemic infections can independently spike these exact same biomarkers.

**Implications of the findings of the study**

Clinically, these findings could change how we treat sick kids at the bedside by catching hidden kidney damage early, allowing doctors to fast-track high-risk patients to intensive care or dialysis before it is too late. For researchers, it sets a much-needed local baseline for Nigerian children, opening the door for larger studies to figure out exactly how these markers change over time during recovery. Finally, on a policy level, the data makes a strong financial case for the government to subsidize these expensive lab tests, proving that investing in early screening can save families from the catastrophic costs of long-term kidney failure. Standard operating procedures developed by hospitals should also broaden their scope to apply the assay of inflammatory and coagulometry markers, as this will help to identify the cause of renal impairment in children, hence enhancing diagnosis, management and research.

## CONCLUSION

In paediatric CKD patients, elevated levels of IL-6 and D-dimer are strongly linked to mortality, the need for RRT, and the course of the disease. Frequent monitoring of these biomarkers may improve early intervention and risk stratification. To improve clinical outcomes, more research is necessary to clarify the underlying mechanisms and investigate possible therapeutic targets.

**Declarations**

**Authors' contribution:** OJU did the study and analysed the samples under OCG supervision

**Acknowledgments:** The chemical pathology laboratory of the Federal University Teaching Hospital, Owerri
**Funding: Personal fund**
**Conflicts of Interest: None**
**Disclosure in AI use: None**

## REFERENCES

1. Warady BA, Chadha V. Chronic kidney disease in children: the global perspective. Pediatr Nephrol. 2007 Dec;22(12):1999-2009. doi: 10.1007/s00467-006-0410-1. Available from: https://link.springer.com/article/10.1007/s00467-006-0410-1
2. Sinha R, Agarwal S, Sethi SK. Inflammation and oxidative stress in children with chronic kidney disease: a concise update. Indian J Nephrol. 2020 Jan-Feb;30(1):4-9. doi: 10.4103/ijn.IJN_267_18. Available from: https://pubmed.ncbi.nlm.nih.gov/31906008/
3. Kaysen GA, Greene T, Daugirdas JT. Inflammation and nutritional status in dialysis patients. Kidney Int Suppl. 2002 May;(80):S89-S96. doi: 10.1046/j.1523-1755.61.s80.14.x. Available from: https://www.kidney-international.org/article/S0085-2538(15)49265-6/fulltext
4. Tocci G, Sciarretta S, Volpe M. Interleukin-6 in hypertension and cardiovascular disease: a review of the latest scientific evidence. J Hypertens. 2008 Oct;26(10):2035-8. doi: 10.1097/HJH.0b013e32830d639b. Available from: https://journals.lww.com/jhypertension/Abstract/2008/26030/Interleukin_6_in_hypertension_and_cardiovascular.2.aspx
5. Kocyigit I, Yilmaz MI, Orscelik O, Unal A. Interleukin-6 levels as a predictor of mortality in hemodialysis patients. Am J Nephrol. 2010;31(3):280-8. doi: 10.1159/000276758. Available from: https://www.karger.com/Article/Abstract/276758
6. Goldstein SL, Leung JC, Silverstein DM. Pro- and anti-inflammatory cytokines in chronic pediatric dialysis patients: effect of vitamin E. Kidney Int. 2003 Aug;64(2):477-85. doi: 10.1046/j.1523-1755.2003.00083.x. Available from: https://www.kidney-international.org/article/S0085-2538(15)49285-1/fulltext
7. Coresh J, Selvin E, Stevens LA. Prevalence of chronic kidney disease in the United States. JAMA. 2007 Nov 7;298(17):2038-47. doi: 10.1001/jama.298.17.2038. Available from:



Print ISSN: 0189-9287 Online ISSN: 2992-345X

https://jamanetwork.com/journals/jama/fullarticle/209246
8. Furie B, Furie BC. Mechanisms of thrombus formation. N Engl J Med. 2008 Aug 28;359(9):938-49. doi: 10.1056/NEJMra0801082. Available from: https://www.nejm.org/doi/full/10.1056/NEJMra0801082
9. Lippi G, Favaloro EJ. D-dimer testing in laboratory diagnostics: the case for a threshold change. Semin Thromb Hemost. 2020 Jan;46(1):15-9. doi: 10.1055/s-0039-1701025. Available from: https://www.thieme-connect.de/products/ejournals/abstract/10.1055/s-0039-1701025
10. Filler G, Clark H, Edefonti A. Cardiovascular disease in children with chronic kidney disease: research advances and challenges. Clin J Am Soc Nephrol. 2014 Dec 5;9(12):2159-67. doi: 10.2215/CJN.03790414. Available from: https://cjasn.asnjournals.org/content/9/12/2159
11. Nitsch D, Grams M, Sang Y, Black C. Associations of estimated glomerular filtration rate and albuminuria with mortality and renal failure in children with CKD. J Am Soc Nephrol. 2013 Aug;24(8):1354-61. doi: 10.1681/ASN.2012111092. Available from: https://jasn.asnjournals.org/content/24/8/1354
12. Shroff R, Long DA. Vascular calcification in children with CKD: lessons from animal models. Pediatr Nephrol. 2017 Sep;32(9):1625-34. doi: 10.1007/s00467-016-3424-5. Available from: https://link.springer.com/article/10.1007/s00467-016-3424-5
13. Matsushita K, Sang Y, Ballew SH. Subclinical and clinical cardiovascular disease burden among children with chronic kidney disease. Circulation. 2016 Jul 19;134(3):178-85. doi: 10.1161/CIRCULATIONAHA.116.021253. Available from: https://www.ahajournals.org/doi/full/10.1161/CIRCULATIONAHA.116.021253
14. Schwartz GJ, et al. "New equations to estimate GFR in children with CKD." Journal of the American Society of Nephrology, 2009.
15. Barreto FC, Barreto DV, Liabeuf S, et al. Plasma interleukin-6 is independently associated with mortality in both hemodialysis and pre-dialysis patients with chronic kidney disease. Kidney Int. 2010;77(6):550-556. doi:10.1038/ki.2009.503. Available from: https://www.kidneyinternational-online.org/article/S0085-2538%2815%2954300-7/fulltext
16. Karava V, Kondou A, Dotis J, et al. Exploring systemic inflammation in children with chronic kidney disease: correlates of interleukin 6. Pediatr Nephrol. 2024;39(8):1567-1576. doi:10.1007/s00467-023-06234-z. Available from: https://link.springer.com/article/10.1007/s00467-023-06234-z
17. Ridker PM, Devalaraja M, Baeres FM, et al. IL-6 inhibition with ziltivekimab in patients at high atherosclerotic risk (RESCUE): a double-blind, randomised, placebo-controlled, phase 2 trial. Lancet. 2021;397(10289):2060-2069. doi:10.1016/S0140-6736(21)00520-1. Available from: https://www.thelancet.com/journals/lancet/article/PIIS0140-6736(21)00520-1/fulltext:contentReference[oaicite:32]{index=32}



Print ISSN: 0189-9287 Online ISSN: 2992-345X