LIFE SCIENCE REVIEW

Review Article

Journal homepage: https://lsr.crcjournals.org/

p-ISSN 3117-3535
e-ISSN 3117-3543

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Diagnostic Significance of Urinary Biomarkers in Congenital Upper Urinary Tract Obstruction

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Received 15 April 2026 | Revised 12 May 2026 | Accepted 10 June 2026 | Available Online 09 July 2026

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Citation: Shivani Chaudhary, Archika Gupta, Piyush Kumar and Shiv Narain Kureel (2026). Diagnostic Significance of Urinary Biomarkers in Congenital Upper Urinary Tract Obstruction. ***Life Science Review***.
DOI: https://doi.org/10.51470/LSR.2026.10.02.25

Abstract

Congenital obstructive uropathy represents an important contributor to morbidity in the paediatric population and may result in long-term renal impairment if not recognized and managed appropriately. Despite advances in imaging and functional assessment, an accepted gold standard for accurately diagnosing renal obstruction in children is still lacking. Conventional diagnostic modalities often have limitations in predicting the severity of obstruction and the extent of renal damage. In this context, the non-invasive assessment of urinary biomarkers has gained increasing attention, as it offers a safe, repeatable, and child-friendly approach for evaluating renal injury. Assessment of biomarker concentrations in urine collected through normal voiding holds promise for improving early diagnosis, guiding clinical decision-making, and monitoring disease progression or recovery following intervention. The purpose of this review is to examine recent research on the usefulness of urinary biomarkers for diagnosing obstructive uropathy and assessing patient outcomes during follow-up in children.

Methods: *Recent studies highlight several biomarkers such as NGAL, KIM-1, MCP-1, PAI-1, and L-FABP, which indicate early tubular injury, inflammation, and fibrosis. These biomarkers were reviewed for their diagnostic accuracy, correlation with obstruction severity, and their value in monitoring response after surgical interventions like valve fulguration or pyeloplasty.*

Results*: Biomarkers such as NGAL and KIM-1 rise significantly in early tubular injury, even when serum creatinine is normal. MCP-1 and PAI-1 reflect inflammation and future fibrosis, helping predict long-term renal outcomes. L-FABP increases in renal hypoxia seen in severe obstruction. Many studies show that biomarker levels decrease after surgical relief of obstruction, indicating kidney recovery. Biomarkers also help differentiate true obstruction from non-obstructive hydronephrosis.*

Conclusion*: Biomarkers provide a non-invasive, sensitive, and early method to detect renal injury in congenital urinary obstruction. Their use can improve diagnosis, guide treatment decisions, and predict long-term renal outcomes better than traditional tests. Incorporating biomarkers into routine evaluation may help prevent chronic kidney disease in affected children.*

Keywords: *Urinary Biomarkers, Congenital Upper Urinary Tract Obstruction, Pediatric Urology, Renal Function, Biomarker- based Diagnosis.*

INTRODUCTION

In the pediatric population, ureteropelvic junction obstruction is the predominant underlying cause of hydronephrosis [1]. Evidence accumulated over many years suggests that the presence of hydronephrosis does not necessarily confirm urinary tract obstruction [2]. Distinguishing an obstructed dilated kidney from a dilated collecting system without true obstruction continues to be a major diagnostic challenge in pediatric urology.

There is currently no universally accepted gold-standard test that can accurately confirm urinary tract obstruction. As a result, clinicians generally depend on a combination of serial imaging techniques, such as diuretic ultrasonography, radionuclide renography, and excretory urography, to establish the diagnosis and guide patient management [3]. However, these imaging modalities are associated with several limitations, such as exposure to ionizing radiation and, in some cases, the need for intravenous administration of radiocontrast agents or radioisotopes.

The management of UPJO is further complicated by the variable natural history of the disease. While spontaneous resolution occurs in a proportion of affected children, others demonstrate progressive deterioration in renal function and ultimately require surgical intervention [1–3]. Therefore, identifying which patients with UPJO are likely to improve without intervention and which may require necessitate surgery remains an important clinical problem worthy of investigation.

In this context, urinary biomarkers have gained increasing attention because of their non-invasive nature and potential applicability in pediatric patients. Measurement of biomarkers in voided urine may offer a valuable adjunct for improving diagnostic accuracy, assessing the severity of obstruction, and monitoring disease progression or response to treatment. This review examines the available evidence on the clinical utility of urinary biomarkers for the diagnosis and longitudinal monitoring of pediatric patients with upper urinary tract obstruction.

MATERIALS AND METHODS

The literature search was performed using PubMed to retrieve articles published between 2021 and 2023 that investigated the diagnostic and follow-up applications of urinary biomarkers in pediatric congenital obstructive uropathy.

RESULTS

The review analyzed a total of 56 studies, comprising 23 experimental investigations and 33 prospective controlled clinical trials. Multiple cytokines, peptides, enzymes, and low–molecular-weight proteins were identified as key factors either contributing to or resulting from renal fibrosis and apoptotic processes triggered by urinary obstruction. Among the most significant biomarkers were transforming growth factor-β1 (TGF-β1), epidermal growth factor (EGF), endothelin-1 (ET-1), urinary tubular enzymes—namely N-acetyl-β-D-glucosaminidase (NAG), γ-glutamyl transferase (GGT), and alkaline phosphatase (ALP)—as well as microproteins including β2-microglobulin (β2M), microalbumin (M.Alb), and micrototal protein (M.TP). Each of these biomarkers is evaluated in terms of its pathophysiological association with urinary tract obstruction and its utility in the diagnosis and monitoring of upper urinary tract obstruction, as demonstrated in both experimental models and clinical studies.

Transforming growth factor-β1 (TGF-β_1)

Pathophysiologic background

Transforming growth factor-β1 (TGF-β1) plays a central role in the regulation of tissue repair following injury. Under normal physiological conditions, its expression is downregulated through feedback mechanisms once the healing process is complete.

However, sustained or dysregulated release of TGF-β1 leads to excessive deposition of extracellular matrix components, ultimately resulting in tissue fibrosis [4]. Increased synthesis of TGF-β1 within the kidney is associated with collagen accumulation and progressive renal scarring [5]. TGF-β1 is therefore considered a key mediator in the shared pathogenic pathways underlying tissue fibrosis and the progression to advanced chronic kidney disease arising from diverse aetiologies [6]. Experimental studies have further demonstrated that administration of specific antisera against TGF-β1 can attenuate renal injury, supporting its pathogenic role in renal damage [7].

Obstruction of the upper urinary tract triggers a complex cascade of molecular and histopathological alterations, including activation of the renin–angiotensin system, which subsequently leads to increased intrarenal expression of TGF-β1 [8,9]. Honkanen et al. [10] reported that persistently elevated urinary excretion of TGF-β1 was associated with morphological indicators of chronic renal damage. Markedly increased urinary levels were suggestive of an ongoing or progressive disease course, whereas lower levels were indicative of disease remission or normal renal status. These observations support the concept that urinary TGF-β1 reflects active sclerotic and fibrotic processes within the kidney and may serve as a valuable non-invasive biomarker for assessing disease progression and monitoring therapeutic response.

Experimental studies

Experimental studies have consistently demonstrated an association between urinary tract obstruction and increased renal expression of transforming growth factor-β1 (TGF-β1). Walton et al. [11] reported a time-dependent rise in TGF-β1 expression in obstructed kidneys following unilateral ureteral obstruction (UUO) in adult Sprague–Dawley rats. Similarly, Chuang et al. [9] observed a linear increase in renal TGF-β1 gene expression during the first month of life in neonatal rats subjected to UUO. Supporting these findings, studies conducted in fetal sheep with hydronephrosis revealed significantly higher levels of TGF-β1 mRNA in hydronephrotic kidneys compared with normal renal tissue [12]. Furthermore, Seseke et al. [13] demonstrated markedly elevated TGF-β1 expression in hydronephrotic kidneys in a rat model, while the contralateral non-obstructed kidneys showed no significant difference when compared with control animals.

Clinical studies

Several clinical investigations have reported significantly higher urinary levels of transforming growth factor-β1 (TGF-β1) in samples obtained from the dilated renal pelvis compared with bladder urine in children diagnosed with ureteropelvic junction obstruction (UPJO) [14–16]. These findings suggest a localized increase in TGF-β1 production in the obstructed renal unit.

In a recent clinical study, Taha et al. [16] demonstrated that a cutoff value of 190 pg/mg creatinine for urinary TGF-β1 measured in voided urine achieved a sensitivity of 100%, a specificity of 80%, and an overall diagnostic accuracy of 90.8% for identifying UPJO in pediatric patients. The same study further indicated that urinary TGF-β1 may serve as a useful noninvasive biomarker for long-term postoperative follow-up in children undergoing pyeloplasty for UPJO.

Despite these promising findings, TGF-β1 lacks disease specificity for obstructive uropathy. Elevated urinary excretion of TGF-β1 has also been documented in other renal disorders, including IgA nephropathy [17], membranous nephropathy presenting with nephritic features [10], as well as in patients with both insulin-dependent and non-insulin-dependent diabetes mellitus [18,19]. These observations highlight the importance of interpreting TGF-β1 levels in conjunction with clinical findings and other diagnostic modalities.

Epidermal growth factor

Pathophysiologic background

Epidermal growth factor (EGF) is a well-characterized polypeptide growth factor that plays a crucial role in regulating cellular proliferation and differentiation [20]. Within the kidney, EGF acts as a mitogenic stimulus for multiple renal cell types and exerts significant functional effects on intact glomeruli, proximal tubules, and collecting ducts. It is considered a potent trophic factor for tubular epithelial cells [21,22]. Under physiological conditions, EGF is primarily synthesized by distal tubular cells, and its expression increases progressively during renal maturation [23].

Renal cell growth and differentiation are mediated largely through the interaction between EGF and its receptor, the epidermal growth factor receptor (EGFR). The coordinated activity of EGFR and its ligand is essential for cell cycle regulation and proliferative responses in the developing and mature kidney. Lin et al. [24] proposed that EGFR and EGF may act together as a transactivation complex capable of binding to specific DNA sequences, thereby initiating gene expression necessary for high levels of cellular proliferation. Consequently, a reduction in EGF expression may indicate impaired EGFR-mediated signaling pathways and diminished renal cellular growth or repair mechanisms.

Experimental studies

Experimental studies have shown that chronic unilateral ureteral obstruction (UUO) leads to suppression of renal epidermal growth factor (EGF) production in neonatal rats [23]. Administration of exogenous EGF in this model has been reported to significantly reduce tubular epithelial cell apoptosis by approximately 80%—and to facilitate renal recovery following relief of the obstruction. Similarly, in adult rats subjected to UUO, exogenous EGF has been shown to inhibit tubular apoptosis.

In contrast, studies in neonatal wild-type mice have demonstrated differing effects, with exogenous EGF promoting apoptotic pathways rather than enhancing cell survival in the obstructed kidney [25].

Clinical studies

Studies have demonstrated that children with ureteropelvic junction obstruction (UPJO) exhibit a significant reduction in epidermal growth factor (EGF) gene expression in renal tissue samples obtained at the time of surgery when compared with control subjects [26,27]. In addition, Grandaliano et al. [28] reported markedly lower urinary EGF levels in children with UPJO relative to healthy controls. However, these findings were not corroborated by a more recent study conducted by Taha et al. [16].

According to Taha et al. [16], using a cutoff value of 40 ng/mg creatinine for urinary EGF measured in voided urine resulted in a sensitivity of 40%, a specificity of 80%, and an overall diagnostic accuracy of 58.5% for the detection of UPJO in pediatric patients. Based on these limited diagnostic performance characteristics, urinary EGF is currently regarded as having low clinical utility in the diagnosis of upper urinary tract obstruction [16].

Endothelin - 1 (ET-1)

Pathophysiologic background

Endothelin-1 (ET-1) is recognized as one of the most potent endogenous vasoconstrictor peptides identified to date, exhibiting approximately tenfold greater vasoconstrictive activity than angiotensin II [29]. Accumulating evidence has implicated ET-1 in the pathogenesis of tissue injury and functional impairment associated with unilateral ureteral obstruction (UUO) [30]. Kelleher et al. [31] demonstrated that ET-1 plays a dominant role in the development of preglomerular arteriolar narrowing in obstructed upper urinary tract systems. Furthermore, experimental findings suggest that ET-1 contributes to the progression of renal interstitial fibrosis following ureteral ligation [32].

Additional studies have shown that ET-1 concentrations are significantly higher in the renal venous outflow compared with the corresponding arterial inflow in models of UUO, indicating that ET-1 production is predominantly of renal origin rather than systemic [33]. Importantly, pharmacological blockade of ET-1 receptors has been shown to preserve renal function and to attenuate the reduction in renal plasma flow and glomerular filtration rate in rats subjected to ureteral obstruction [34].

Experimental studies

Hegarty et al. [35] conducted a semiquantitative evaluation of endothelin-1 (ET-1) expression in a rat model of unilateral ureteral obstruction (UUO) and demonstrated a significant increase in ET-1 expression in the obstructed kidney, accompanied by reduced expression in the contralateral kidney, when compared with sham-operated controls.

In the same study, administration of bosentan, a dual endothelin receptor antagonist, to a subset of obstructed animals resulted in effective ET-1 receptor blockade. This intervention was associated with restoration of renal blood flow in the obstructed kidney and a marked reduction in tubular apoptosis to levels comparable with those observed in control kidneys. The extent of these functional and cellular improvements supports the concept that ET-1 is a key mediator of vascular and cellular injury in UUO [35].

In a separate experimental investigation, Miller et al. [36] analyzed endothelin-1 gene expression in rats with congenital unilateral ureteropelvic junction obstruction (UPJO). Their findings demonstrated significantly elevated ET-1 gene expression in both the renal pelvis and ureteropelvic junction of obstructed kidneys compared with corresponding regions in healthy control animals. Based on these results, the authors suggested that increased ET-1 expression may play a pathogenic role in the development and progression of ureteral obstruction [36].

Clinical studies

Knerr et al. [37] investigated endothelin-1 (ET-1) gene expression in stenotic tissue obtained from children with congenital ureteropelvic junction obstruction (UPJO) and demonstrated significantly higher ET-1 expression in the obstructed UPJ compared with control tissue. These findings provide evidence for local upregulation of ET-1 in the obstructed segment.

Taha and colleagues [38] were the first to evaluate urinary ET-1 levels in pediatric patients with UPJO. Their study revealed that ET-1 concentrations measured in voided urine were markedly elevated—up to fourfold—compared with levels observed in healthy controls. These results suggest that urinary ET-1 measured in bladder urine may serve as a useful noninvasive biomarker for confirming the diagnosis of UPJO in children. Using a cutoff value of 3 fmol/mg creatinine, urinary ET-1 demonstrated a sensitivity of 74.3%, a specificity of 90%, and an overall diagnostic accuracy of 81.5% [38].

Urinary enzymes

Pathophysiologic background

Obstructive nephropathy is characterized by progressive injury to the proximal tubules of the affected kidney, resulting in disruption of cellular membranes and subsequent release of intracellular enzymes. This tubular damage leads to increased urinary excretion of lysosomal enzymes, such as N-acetyl-β-D-glucosaminidase (NAG), as well as brush border enzymes including γ-glutamyl transferase (GGT) and alkaline phosphatase (ALP). The presence of these enzymes in urine has been shown to serve as a valuable indicator of proximal tubular injury [39].

Among urinary enzymes, NAG is the most extensively studied and commonly measured marker for the detection of renal tubular damage and the diagnosis of renal disease. Its widespread use is attributed to several properties, including its stability in urine, its relatively large molecular weight (approximately 130 kDa), which prevents glomerular filtration, and its high concentration within lysosomes of proximal tubular cells [40]. Consequently, increased urinary NAG activity reflects tubular injury, or more specifically, loss of lysosomal membrane integrity [41]. Similarly, because γ-glutamyl transferase is localized predominantly to the brush border membrane of proximal tubular cells, measurement of urinary GGT has proven to be a reliable marker of luminal membrane dysfunction in cases of obstructive nephropathy [42].

Experimental Studies

Experimental investigations in Wistar rat models with stable partial ureteral obstruction have demonstrated significantly elevated urinary N-acetyl-β-D-glucosaminidase (NAG) activity in the obstructed kidneys compared with the contralateral non-obstructed control kidneys during the first two weeks following the induction of obstruction [43,44]. In contrast, urinary γ-glutamyl transferase (GGT) activity did not exhibit similarly distinct differences between hydronephrotic obstructed kidneys and contralateral control kidneys in the same experimental settings [43].

Clinical studies

Multiple clinical studies have demonstrated that the activities of tubular enzymes—namely N-acetyl-β-D-glucosaminidase (NAG), γ-glutamyl transferase (GGT), and alkaline phosphatase (ALP)—measured in urine aspirated from the dilated renal pelvis during surgery in children with ureteropelvic junction obstruction (UPJO) are consistently higher than those detected in bladder urine samples from the same patients [45,46].

Further evidence from a study published in the Hungarian literature revealed that urinary activities of NAG, ALP, and GGT in children with upper obstructive uropathy were elevated by two- to tenfold when compared with values observed in healthy children [47]. Similarly, Taha et al. [46] reported significantly increased activities of these three enzymes in voided urine samples from children with UPJO, with levels reaching up to 2.34 times those observed in children with dilated but non-obstructed kidneys. These findings suggest that measurement of tubular enzymes in voided urine may provide clinically useful support for the diagnosis of UPJO in pediatric patients.

In the same study, optimal cutoff values for urinary NAG, ALP, and GGT were established to maximize diagnostic performance in children with UPJO [46]. A cutoff value of 7.8 mU/mg creatinine for NAG yielded a sensitivity of 97.1%, a specificity of 80%, and an overall diagnostic accuracy of 92%. For ALP, a cutoff value of 34.5 IU/g creatinine resulted in a sensitivity of 91.4%, a specificity of 100%, and an accuracy of 94%. A GGT cutoff value of 54 IU/g creatinine provided a sensitivity of 62.9%, a specificity of 100%, and an accuracy of 74%.

When NAG and ALP were combined, diagnostic performance improved further, achieving a sensitivity of 100%, a specificity of 80%, and an overall accuracy of 94%. Notably, despite their shared tubular origin, ALP and GGT exhibited differing sensitivities, potentially reflecting their distinct localization within the brush border membrane, with ALP situated more superficially and GGT embedded more deeply within the membrane structure [48].

Overall, these tubular enzymes demonstrate high sensitivity but only moderate specificity for obstructive uropathy. It is important to recognize that elevated urinary NAG levels have also been reported in several other renal and systemic conditions, including high-grade vesicoureteral reflux, urinary tract infection, glomerulonephritis, and diabetes mellitus [49].

The marked elevation of urinary NAG, ALP, and GGT in children with UPJO compared with those with dilated but non-obstructed pelvicalyceal systems is clinically significant, as it aids in distinguishing obstructive from non-obstructive hydronephrosis in children with congenital urinary tract dilation. This differentiation is critical for guiding clinical decision-making between conservative management and surgical intervention, although no absolute criteria currently exist to mandate surgery. Further large-scale, prospective, comparative studies are required to better define the role of these urinary enzymes in the diagnostic evaluation of UPJO in children.

Regarding postoperative and longitudinal follow-up, Tataranni et al. [42] demonstrated that urinary NAG excretion remained elevated for up to 45 days following relief of obstruction in adults, despite the restoration of urine flow. Similarly, Taha et al. [46] observed that a period of three to six months was necessary for significant reductions in urinary NAG, ALP, and GGT activities to occur in children after pyeloplasty for UPJO. These findings indicate that renal functional and ultrastructural recovery following relief of obstruction is a gradual process. Additionally, a recent study identified a strong negative correlation between the function of the affected kidney and levels of urinary tubular enzymes, suggesting that measurement of these biomarkers in voided urine may serve as a useful noninvasive tool for long-term follow-up of children with UPJO, whether managed surgically or conservatively [46]. In contrast, Carr et al. [45] reported that radiographic assessment of obstruction severity did not always correlate with urinary NAG activity; among imaging modalities, renal ultrasonography showed the closest agreement with biochemical findings.

Microproteins

Pathophysiologic background

One of the primary physiological functions of the glomerulus is the selective filtration of plasma proteins. Low–molecular weight proteins, such as β2-microglobulin, are freely filtered through the glomerular membrane and are almost completely reabsorbed by the renal tubules under normal conditions. Consequently, the appearance of β2-microglobulin in urine is considered an indicator of tubular dysfunction. In contrast, high–molecular weight proteins exceeding 40 kDa, including microalbumin and total urinary proteins, are normally retained within the circulation and are not filtered by the intact glomerular barrier. In obstructive uropathy, inflammatory processes and damage to the glomerular basement membrane lead to increased glomerular permeability, resulting in enhanced filtration of these larger proteins. Therefore, elevated urinary levels of microalbumin and total protein are regarded as markers of glomerular dysfunction in this setting [50–52].

Experimental studies

Measurement of urinary β2-microglobulin has been widely used for the evaluation of renal tubular dysfunction. Experimental studies have demonstrated that the urinary β2-microglobulin–to–creatinine ratio is significantly increased as early as one week following the induction of unilateral complete ureteral obstruction in Wistar rats when compared with control animals [39]. Additionally, significant elevations in urinary β2-microglobulin levels have also been observed in rat models subjected to both unilateral and bilateral partial ureteral obstruction, further supporting its role as a marker of tubular injury [53].

In contrast, increased urinary excretion of high–molecular weight proteins reflects glomerular dysfunction, with glomerular proteinuria being regarded as the most clinically significant form of proteinuria [51]. Consistent with this, experimental studies have shown that the urinary microalbumin–to–creatinine ratio is significantly elevated one week after the onset of unilateral ureteral obstruction in Wistar rats compared with controls, indicating early impairment of glomerular permeability in obstructive uropathy [39].

Clinical studies

Studies have shown that urinary β2-microglobulin concentrations measured in samples obtained from the dilated renal pelvis during surgery in children with ureteropelvic junction obstruction (UPJO) are consistently higher than those measured in bladder urine from the same patients [45]. In addition, urinary β2-microglobulin levels in children with UPJO have been reported to be significantly elevated compared with healthy control subjects. Notably, this elevation persists for up to three months following surgical relief of obstruction and subsequently demonstrates a marked and rapid decline between three and four months postoperatively, reflecting gradual recovery of tubular function [42].

With regard to glomerular markers, Lama et al. [54] demonstrated that urinary levels of microalbumin and total urinary proteins were significantly higher in voided urine samples obtained from children with UPJO compared with those from non-obstructed controls.

Furthermore, microalbumin levels were observed to continue rising during the early postoperative follow-up period, with a gradual decline becoming evident approximately 18 months after pyeloplasty, suggesting a prolonged course of glomerular recovery following relief of obstruction [54].

DISCUSSION

Congenital ureteropelvic junction obstruction (UPJO) represents an important cause of morbidity in the paediatric population and manifests across a broad clinical spectrum, ranging from mild hydronephrosis to severe renal impairment. The condition induces diverse structural and functional alterations within the renal parenchyma, some of which may be related to underlying developmental abnormalities. If left untreated, congenital UPJO can interfere with normal nephron maturation and function, ultimately leading to progressive renal deterioration [54].

At present, there is no universally accepted gold standard for the evaluation of renal obstruction against which individual cases can be definitively assessed. In clinical practice, diagnosis frequently relies on serial investigations and longitudinal assessment of changes in functional and anatomical parameters over time. Commonly employed imaging modalities include gray-scale ultrasonography, Doppler ultrasonography, radionuclide renography, excretory urography, contrast-enhanced computed tomography, and magnetic resonance urography. Although each technique offers specific advantages, none provides a completely reliable or comprehensive assessment when used in isolation [55].

The identification of a urinary biochemical marker capable of distinguishing obstructive from non-obstructive hydronephrosis would substantially reduce the invasiveness, subjectivity, and operator dependency associated with current radiological approaches [14]. Therefore, the development of a reliable bladder urine biomarker to assist in the diagnosis of upper urinary tract obstruction is highly attractive from a clinical perspective.

The nephron exhibits marked structural and functional heterogeneity across its different segments, which are composed of at least 13 distinct cell types, each with specialized physiological roles [56]. Owing to this regional specialization, injury affecting a particular nephron segment is expected to produce specific alterations in the urinary biomarker profile. As renal damage progresses and becomes more extensive, the urinary biomarker pattern tends to reflect involvement of multiple nephron segments, resulting in a broader and more uniform biomarker signature indicative of widespread renal injury [57].

In this review, the diagnostic performance of various urinary biomarkers has been examined, demonstrating variable degrees of clinical utility. Among them, transforming growth factor-β1 (TGF-β1), endothelin-1 (ET-1), and panels of tubular enzymes appear to be the most promising candidates. These biomarkers have demonstrated sensitivities ranging from 74.3% to 100%, specificities between 80% and 90%, and overall diagnostic accuracies from 81.5% to 94% in the evaluation of congenital obstructive uropathy in children. Furthermore, certain biomarkers have shown value in distinguishing dilated but non-obstructed kidneys suitable for conservative management from obstructed kidneys requiring surgical intervention. Additionally, several studies indicate that urinary biomarkers may contribute to the assessment of therapeutic response and postoperative recovery in children with congenital renal obstruction.

CONCLUSIONS

Urinary biomarkers represent a promising non-invasive approach for the assessment of congenital renal obstruction in the paediatric population. Among the biomarkers investigated to date, transforming growth factor-β1 (TGF-β1), endothelin-1 (ET-1), and panels of tubular enzymes appear to be the most informative. These biomarkers have shown potential not only for the diagnosis of congenital obstructive uropathy but also for distinguishing between dilated, non-obstructed kidneys suitable for conservative management and obstructed kidneys that require surgical intervention. In addition, several studies have suggested that urinary biomarkers may be useful in monitoring treatment response and evaluating postoperative recovery in children with congenital renal obstruction.

Despite these encouraging findings, the existing body of literature is limited by relatively small sample sizes and a lack of appropriately matched control groups. Therefore, well-designed studies involving larger patient populations and diverse control cohorts are required to validate the clinical utility of urinary biomarkers in the diagnosis and long-term follow-up of children with congenital obstructive uropathy.

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