Urinalysis in Hypertensive Adolescents: From Routine Abnormalities to Early Markers of Kidney and Cardiovascular Risk

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Abstract

Background: Adolescent hypertension is increasingly recognized as an early manifestation of lifelong cardiovascular and renal risk rather than an isolated abnormal blood pressure measurement. The growing prevalence of obesity, insulin resistance, sedentary behavior, and other cardiometabolic disturbances has contributed to a shift toward primary hypertension during adolescence; nevertheless, kidney disease remains an important cause of secondary hypertension in this age group. Urinalysis therefore occupies a unique position in the evaluation of hypertensive adolescents because it may identify an underlying renal disorder while simultaneously providing evidence of early hypertension-mediated kidney injury. Routine abnormalities include proteinuria, albuminuria, microscopic hematuria, glycosuria, leukocyturia, and alterations in urine concentration, although their clinical significance varies considerably. Among these findings, persistent albuminuria and proteinuria are particularly relevant because they may reflect glomerular endothelial dysfunction, increased intraglomerular pressure, or established kidney disease and may coexist with other markers of cardiovascular target-organ damage. Conversely, transient or orthostatic proteinuria and isolated microscopic hematuria are relatively common during adolescence and require confirmation before being attributed to hypertension. Emerging urinary biomarkers, including neutrophil gelatinase-associated lipocalin and N-acetyl-β-D-glucosaminidase, may detect tubular injury before conventional markers become abnormal, but their clinical role remains investigational. This review examines the spectrum, pathophysiology, interpretation, and prognostic significance of urinalysis abnormalities in hypertensive adolescents and proposes a clinically integrated approach to distinguishing benign findings, secondary renal hypertension, and early hypertension-mediated kidney damage.

Keywords: adolescent hypertension, urinalysis, albuminuria, kidney injury

Introduction

Hypertension during adolescence has evolved from a condition traditionally associated with secondary renal or cardiovascular disease into an increasingly common manifestation of primary cardiometabolic disease. Contemporary evidence suggests that hypertension affects approximately 2%–5% of children and adolescents, with primary hypertension now predominating among older children, particularly those with overweight, obesity, adverse dietary patterns, or a family history of hypertension. This transition is clinically important because elevated blood pressure during youth tends to track into adulthood and is associated with early vascular abnormalities, left ventricular remodeling, and future cardiovascular disease. Adolescence therefore represents a critical period during which apparently asymptomatic hypertension may already be accompanied by subclinical target-organ injury. [1]

The kidney is central to this process. Renal sodium handling, activation of the renin-angiotensin-aldosterone system, sympathetic activity, nephron number, glomerular hemodynamics, and obesity-related hyperfiltration all influence blood pressure, while sustained hypertension can itself injure the renal microcirculation. Urinalysis consequently has a dual role in hypertensive adolescents: abnormal urinary findings may reveal kidney disease responsible for secondary hypertension, or they may represent downstream evidence of renal injury from established hypertension. Current pediatric hypertension guidance therefore includes urinalysis, serum electrolytes, blood urea nitrogen, and creatinine among the basic investigations in children and adolescents with confirmed hypertension. [2]

Despite this established diagnostic role, an important research gap remains. Most evidence linking urinary albumin excretion with cardiovascular and renal outcomes originates from adult populations, whereas pediatric studies are smaller, heterogeneous, and often cross-sectional. Moreover, adolescents frequently demonstrate transient proteinuria, orthostatic proteinuria, exercise-associated albuminuria, or microscopic hematuria unrelated to progressive renal disease. The clinical challenge is therefore not merely to detect an abnormal urine result but to interpret it in relation to blood pressure phenotype, obesity, metabolic risk, kidney function, and the probability of secondary disease. The aim of this review is to integrate current nephrological and cardiovascular evidence regarding the pattern and significance of urinalysis abnormalities in hypertensive adolescents and to clarify how routine and emerging urinary markers may contribute to early risk stratification.

Epidemiologic Context of Hypertension in Adolescence

Estimates of pediatric hypertension vary according to population characteristics, measurement methodology, number of blood pressure visits, and diagnostic thresholds. A major systematic review and meta-analysis demonstrated that hypertension is not uncommon among children and adolescents worldwide and that prevalence increases substantially in populations with obesity. Repeated blood pressure measurements are essential because a single elevated office reading considerably overestimates true sustained hypertension. The increasing burden of adiposity among adolescents has therefore created a population in whom high blood pressure, dyslipidemia, insulin resistance, fatty liver disease, hyperuricemia, and early renal abnormalities frequently coexist rather than occurring as isolated disorders. [3]

Blood pressure phenotype is equally important. Ambulatory blood pressure monitoring (ABPM) improves discrimination among sustained hypertension, white-coat hypertension, and masked hypertension and provides information regarding nocturnal blood pressure and circadian patterns. These distinctions have direct relevance to urinary abnormalities because an adolescent with apparent office hypertension and an abnormal urine test may have a fundamentally different risk profile from a patient with white-coat hypertension and transient proteinuria. Conversely, masked hypertension may expose the kidneys and cardiovascular system to substantial blood pressure load despite apparently normal clinic measurements. ABPM therefore strengthens the interpretation of renal findings by establishing whether meaningful hypertension is truly present. [4]

Adolescent hypertension also deserves attention because its consequences extend beyond youth. Long-

term cohort data have demonstrated that elevated blood pressure in childhood and adolescence predicts adult hypertension, arterial stiffness, increased left ventricular mass, and other subclinical cardiovascular abnormalities. More recent population-level evidence indicates that individuals diagnosed with hypertension during childhood have higher rates of major adverse cardiovascular outcomes during subsequent decades. Accordingly, renal abnormalities identified during adolescence should not be viewed exclusively through the lens of immediate nephrological diagnosis; they may represent components of an accumulating lifetime cardiovascular risk phenotype. [5]

The Kidney–Blood Pressure Axis in Adolescence

The relationship between hypertension and kidney disease is bidirectional. Congenital anomalies of the kidney and urinary tract, reflux nephropathy, glomerular disorders, tubulointerstitial disease, hereditary nephropathies, and chronic kidney disease may all produce hypertension through sodium retention, volume expansion, activation of the renin-angiotensin system, and increased sympathetic activity. In such patients, urinary protein, blood, or other abnormalities may precede recognition of impaired glomerular filtration. Consequently, an abnormal urinalysis in a hypertensive adolescent should first raise the question of whether kidney disease is the cause rather than the consequence of hypertension. [2]

The reverse pathway is equally relevant. Persistent systemic hypertension raises glomerular capillary pressure and contributes to endothelial dysfunction, glomerular basement membrane stress, podocyte injury, and eventually increased permeability to albumin and other proteins. Obesity may amplify this process through renal vasodilation, glomerular hyperfiltration, insulin resistance, inflammation, oxidative stress, and maladaptive activation of the renin-angiotensin-aldosterone system. The combination of obesity and hypertension can therefore create renal injury before conventional serum creatinine becomes abnormal. Because serum creatinine is relatively insensitive to mild nephron loss in young individuals, urinary abnormalities may provide an earlier indication that the kidney is participating in the target-organ consequences of hypertension. [6]

This kidney–blood pressure interaction is especially important during adolescence because physiological changes of puberty alter body composition, insulin sensitivity, renal hemodynamics, and urinary protein excretion. Interpretation must therefore incorporate age, sex, body size, exercise, hydration, fever, menstruation, and posture. A urinary abnormality should not automatically be labeled “hypertensive nephropathy”; instead, persistence, magnitude, associated sediment findings, kidney function, and the blood pressure phenotype should determine its significance.

Routine Urinalysis: Diagnostic Value and Limitations

Routine urinalysis provides information regarding urinary protein, blood, glucose, ketones, leukocyte esterase, nitrite, pH, and specific gravity. Dipstick analysis is inexpensive and immediately available, but it is semiquantitative and susceptible to preanalytical and physiological variation. Concentrated urine, alkaline urine, contamination, exercise, fever, and menstrual blood may produce misleading results. A single abnormal result therefore rarely establishes renal disease, particularly in an otherwise asymptomatic adolescent. Abnormal findings should be confirmed using a properly collected specimen, ideally a first-morning sample when protein excretion is being assessed. [7]

Dipstick protein testing is principally sensitive to albumin and may fail to detect low-grade albuminuria or some forms of tubular proteinuria. Similarly, a positive “blood” reaction detects heme activity rather than intact erythrocytes and may represent hematuria, hemoglobinuria, or myoglobinuria. Microscopy is therefore essential when persistent blood is detected. Urinalysis should be considered the beginning of renal characterization rather than the endpoint of investigation. The diagnostic yield comes from integrating the urine result with quantitative protein assessment, kidney function, blood pressure severity, history, examination, and imaging when indicated. [8]

Current pediatric hypertension recommendations illustrate this principle. The American Academy of Pediatrics includes urinalysis in the screening evaluation of all hypertensive children and adolescents because renal parenchymal disorders remain important secondary causes. Renal ultrasonography is particularly indicated in younger children and in patients with abnormal renal function or abnormal

urinalysis. An adolescent with proteinuria, hematuria, reduced estimated glomerular filtration rate, edema, systemic symptoms, or severe hypertension should therefore receive a more intensive nephrological evaluation than an obese adolescent with uncomplicated primary hypertension and repeatedly normal urine studies. [2]

Proteinuria: From Benign Adolescent Phenomenon to Renal Warning Signal

Proteinuria is one of the most important urinary abnormalities in the hypertensive adolescent but also one of the easiest to misinterpret. Temporary protein excretion may occur after exercise, fever, emotional stress, dehydration, or intercurrent illness. Orthostatic proteinuria is particularly characteristic of adolescents and is defined by increased protein excretion during upright activity with normalization during recumbency. Because prolonged standing and physical activity can markedly influence urinary protein excretion, an abnormal random daytime protein result should generally be reassessed using a first-morning urine sample before extensive investigation is initiated. [9]

Persistent proteinuria requires a different approach. Quantification with a spot urine protein-to-creatinine ratio is more informative than dipstick grading alone. Significant persistent proteinuria may indicate glomerular disease, tubulointerstitial disease, structural renal abnormalities, or chronic kidney disease and may therefore provide the clue to secondary hypertension. The combination of proteinuria and hematuria is particularly concerning because it increases the likelihood of glomerular pathology. When accompanied by edema, reduced glomerular filtration, low complement, systemic manifestations, or rapidly increasing blood pressure, the differential diagnosis should include glomerulonephritis and other active renal diseases rather than primary hypertension. [10]

From a prognostic perspective, proteinuria also acts as a marker and mediator of kidney disease progression. In children with established chronic kidney disease, higher protein excretion identifies a population at increased risk of loss of kidney function, and blood pressure control is a central component of renoprotection. Evidence from pediatric chronic kidney disease studies has shown that intensive blood pressure control, commonly in association with blockade of the renin-angiotensin system, can reduce renal progression risk. Although these data cannot be directly extrapolated to otherwise healthy adolescents with primary hypertension, they emphasize that persistent proteinuria plus hypertension represents a high-risk combination requiring active evaluation and follow-up. [11]

Albuminuria and Early Glomerular Injury

Albuminuria deserves separate consideration because small increases in urinary albumin excretion may occur before conventional dipstick protein becomes positive. The preferred quantitative measure is the urinary albumin-to-creatinine ratio (ACR), ideally from a first-morning specimen. Contemporary kidney disease guidelines classify persistent albuminuria according to its magnitude and recognize it as a marker of kidney damage even when estimated glomerular filtration rate remains preserved. The older term “microalbuminuria” is increasingly replaced by “moderately increased albuminuria,” reflecting the fact that albumin molecules are not structurally different at lower concentrations. [6]

Pediatric evidence suggests that albuminuria may identify a subset of young hypertensive patients with early renal vascular injury. In a study comparing children with primary hypertension and white-coat hypertension, moderately increased albumin excretion was present in approximately one fifth of those with primary hypertension but was absent among those with white-coat hypertension. This observation supports the biological plausibility that sustained rather than episodic office blood pressure elevation contributes to renal endothelial or glomerular stress. Nevertheless, the relatively small sample size and limited longitudinal outcome data prevent albuminuria from being interpreted in children exactly as it is in adult hypertension. [12]

Albuminuria also interacts strongly with obesity and metabolic status. Analysis of a nationally representative adolescent population found that low-grade albuminuria was not uniformly associated with cardiovascular risk in all adolescents. Among overweight adolescents, however, albuminuria clustered with hypertension, insulin resistance, impaired fasting glucose, diabetes, and smoking. This suggests that urinary albumin may be most informative when interpreted within the broader cardiometabolic phenotype rather than as an isolated laboratory abnormality. In an obese hypertensive

adolescent, persistent albuminuria should therefore intensify attention to glucose metabolism, lipid abnormalities, dietary sodium intake, sleep-disordered breathing, and other contributors to vascular risk. [13]

Importantly, guidelines do not entirely agree regarding universal ACR testing in uncomplicated pediatric primary hypertension. The 2017 AAP guideline concluded that evidence was insufficient to recommend routine microalbuminuria testing in all children with primary hypertension because albumin excretion in youth can be affected by exercise, obesity, insulin resistance, diabetes, and other factors. In contrast, European-oriented recommendations have placed greater emphasis on ACR as a component of assessing hypertension-mediated kidney injury. A rational clinical synthesis is to regard urinalysis as mandatory in the initial evaluation while using quantitative ACR selectively or more broadly in patients with persistent hypertension, obesity, diabetes, suspected kidney disease, abnormal dipstick protein, reduced kidney function, or evidence of other target-organ damage. [2]

Microscopic Hematuria in the Hypertensive Adolescent

Microscopic hematuria is relatively common in childhood and adolescence and is frequently transient. A positive dipstick for blood should be confirmed by urine microscopy because dipsticks cannot distinguish erythrocytes from free hemoglobin or myoglobin. Menstruation, strenuous exercise, urinary infection, hypercalciuria, nephrolithiasis, trauma, and contamination may all produce blood in the urine. Consequently, isolated microscopic hematuria on a single sample in a hypertensive adolescent does not establish hypertensive renal injury. [8]

The clinical concern increases when hematuria persists or is accompanied by proteinuria, hypertension, reduced kidney function, or a family history of chronic kidney disease. Persistent microscopic hematuria may reflect IgA nephropathy, Alport syndrome, thin basement membrane disorders, postinfectious processes, or other glomerular diseases. Dysmorphic erythrocytes or red-cell casts further support a glomerular source. In a hypertensive adolescent, these findings fundamentally change the diagnostic direction because the hypertension may be secondary to renal disease rather than primary. [14]

Thus, the combination of hypertension and hematuria should be interpreted according to context. An adolescent with obesity, mild primary hypertension, normal renal function, and transient hematuria after exercise has a very different risk profile from an adolescent with sustained stage 2 hypertension, persistent microscopic hematuria, proteinuria, edema, and impaired kidney function. Urinalysis becomes clinically powerful when patterns rather than individual analytes are recognized.

Glycosuria, Ketonuria, Leukocyturia, and Other Findings

Glycosuria in a hypertensive adolescent should prompt evaluation of plasma glucose and glycated hemoglobin, especially when obesity is present. Type 2 diabetes is increasingly encountered during adolescence and commonly clusters with obesity and hypertension. Persistent glycosuria in the presence of normal blood glucose, however, raises a different differential diagnosis that includes renal tubular disorders or isolated renal glycosuria. Ketonuria may accompany fasting, dehydration, acute illness, or uncontrolled diabetes and should therefore be interpreted in conjunction with metabolic status rather than considered a direct manifestation of hypertension.

Leukocyte esterase, pyuria, and nitrite positivity generally suggest urinary infection rather than hypertension-mediated injury. Recurrent urinary tract infection, however, can be clinically relevant because renal scarring and reflux nephropathy may contribute to secondary hypertension. Persistent sterile pyuria also warrants consideration of tubulointerstitial inflammation, nephrolithiasis, medication-associated interstitial nephritis, or systemic disease. As with protein and blood, the significance of leukocyturia depends upon persistence, symptoms, culture findings, and associated renal abnormalities. [7]

Urine specific gravity and pH are less specific but can still provide useful physiological information. A persistently low specific gravity in the setting of polyuria, impaired renal function, or structural kidney disease may suggest a concentrating defect, whereas a highly concentrated specimen can exaggerate dipstick protein results. Urinary interpretation should therefore remain integrated with volume status, medication exposure, diet, and renal function.

Obesity, Metabolic Dysfunction, and the Urinary Phenotype

Obesity has transformed the epidemiology of adolescent hypertension and complicates interpretation of renal biomarkers. Obesity-related glomerular hyperfiltration increases renal plasma flow and intraglomerular pressure and may eventually result in glomerular enlargement, podocyte stress, and albumin leakage. Insulin resistance, sympathetic activation, hyperuricemia, inflammation, and sodium retention provide additional mechanisms connecting adiposity with hypertension and kidney injury. Consequently, an obese hypertensive adolescent may demonstrate renal stress long before serum creatinine rises. [1]

The relationship is nevertheless nonlinear. Albuminuria is not present in every obese adolescent, and some population studies have reported low-grade albuminuria even among otherwise healthy, lean adolescents. More recent work in large cohorts of adolescents with obesity has demonstrated structural renal enlargement and changes in estimated filtration despite a relatively low prevalence of albuminuria, suggesting that ACR alone may not capture all early obesity-associated renal remodeling. Accordingly, a normal ACR should be reassuring but should not be interpreted as proof that the kidneys are entirely unaffected by severe obesity and hypertension. [15]

Recent data also highlight a relationship between albuminuria and masked hypertension among overweight and obese young people. In a multicenter cohort, albuminuria was associated with ambulatory hypertension and was particularly informative in individuals with masked hypertension. Although such cross-sectional observations do not establish causation, they strengthen the argument for combining urinary assessment with ABPM when office blood pressure and clinical risk appear discordant. [16]

Urinalysis as a Clue to Secondary Hypertension

Primary hypertension is increasingly common during adolescence, but secondary hypertension must not be overlooked. The probability of an underlying secondary cause increases with younger age, severe hypertension, absence of obesity, abnormal kidney function, unusual physical findings, or suggestive family and medical history. Renal parenchymal disease remains one of the most important secondary causes. An abnormal urinalysis therefore carries substantially more diagnostic weight in a hypertensive adolescent than it does in routine screening of an otherwise healthy population. [17]

Persistent proteinuria, albuminuria, hematuria, or active urinary sediment should prompt targeted nephrological assessment. Depending on the pattern, this may include repeat first-morning urinalysis, urine protein-to-creatinine and albumin-to-creatinine ratios, serum creatinine and cystatin C where appropriate, complement studies, autoimmune serology, evaluation for postinfectious disease, urinary calcium assessment, and renal ultrasonography. The diagnostic strategy should remain directed by clinical findings rather than relying on indiscriminate testing. Recent pediatric studies examining the diagnostic yield of secondary hypertension investigations reinforce the value of a focused approach based on age, phenotype, abnormal urine findings, renal function, and imaging. [18]

Conversely, a normal urinalysis does not exclude all secondary causes. Renovascular disease, coarctation of the aorta, endocrine hypertension, obstructive sleep apnea, medication- or substance-associated hypertension, and monogenic disorders may present without urinary abnormalities. Urinalysis therefore functions as one branch of a broader diagnostic framework rather than as a standalone screening tool capable of distinguishing all primary from secondary hypertension.

Urinary Abnormalities and Cardiovascular Target-Organ Damage

The clinical significance of albumin leakage extends beyond the kidney. In adults, albuminuria is widely recognized as an indicator of systemic endothelial dysfunction and increased cardiovascular risk. Pediatric data are more limited but support a similar biological relationship. A study of children and adolescents with essential hypertension found that microalbuminuria was associated with left ventricular hypertrophy, suggesting that renal and cardiac target-organ changes may occur in parallel under the influence of elevated blood pressure and cardiometabolic stress. [19]

This concept is important because pediatric hypertension rarely produces myocardial infarction, stroke, or end-stage kidney disease during adolescence itself. Risk assessment therefore relies on intermediate

phenotypes, including left ventricular hypertrophy, vascular stiffness, increased carotid intima-media thickness, and renal abnormalities. Longitudinal evidence now demonstrates that childhood cardiovascular risk factors—including systolic blood pressure and obesity—predict clinical cardiovascular events decades later. Urinary markers that identify early vascular or renal injury may therefore eventually contribute to a multidimensional risk model extending beyond conventional blood pressure classification. [20]

Nevertheless, urinary albumin should not currently replace established cardiovascular assessment. Echocardiography remains the principal clinical method for identifying left ventricular hypertrophy when pharmacological therapy is being considered, whereas ABPM provides superior characterization of blood pressure exposure. Albuminuria is best regarded as complementary information that may increase concern when it accompanies sustained hypertension, obesity, diabetes, chronic kidney disease, or cardiac target-organ injury.

Emerging Urinary Biomarkers of Early Kidney Injury

Conventional markers have important limitations. Serum creatinine may remain normal despite early nephron injury, while albuminuria primarily reflects glomerular permeability and may not identify tubular damage. Investigators have therefore examined biomarkers such as neutrophil gelatinase-associated lipocalin (NGAL), kidney injury molecule-1, interleukin-18, retinol-binding protein, and N-acetyl-β-D-glucosaminidase (NAG) as potentially earlier signals of hypertension-related renal injury.

In children with primary hypertension and normal conventional renal indices, increased urinary and circulating NGAL and higher urinary retinol-binding protein have been reported compared with healthy controls. Urinary NGAL also showed relationships with ambulatory systolic blood pressure measures, raising the possibility that subclinical tubular injury occurs before albuminuria or a decline in estimated glomerular filtration rate becomes evident. However, the study populations were small, and NGAL is influenced by inflammation, infection, acute kidney injury, age, and other factors. These findings therefore remain hypothesis-generating rather than sufficient for routine clinical testing. [21]

Similarly, urinary NAG has been investigated in children evaluated with ABPM. One study found higher urinary NAG concentrations among hypertensive and white-coat hypertensive patients than among normotensive controls, despite no significant difference in urinary microalbumin excretion. This suggests that tubular biomarkers might reveal renal stress not detected by conventional glomerular markers. Yet reproducibility, age-specific reference ranges, assay standardization, incremental prognostic value, and cost-effectiveness must be established before these tests can be incorporated into routine adolescent hypertension care. [22]

Integrating Urinalysis Into Clinical Risk Stratification

A practical approach begins with confirmation that hypertension is genuine. Appropriate cuff size, repeated auscultatory measurements, and ABPM when indicated should precede attribution of renal abnormalities to sustained high blood pressure. Once hypertension is established, routine urinalysis and renal function assessment should be interpreted together. A completely normal urinalysis and preserved kidney function in an obese adolescent with a strong family history and mild hypertension favor primary hypertension, whereas persistent proteinuria, albuminuria, hematuria, or impaired filtration substantially lowers the threshold for nephrology referral and investigation of secondary renal disease. [4]

When dipstick protein is detected, a first-morning specimen is crucial. Persistent protein should be quantified with a protein-to-creatinine ratio, and ACR is useful when low-grade albumin leakage is suspected. Hematuria should be confirmed microscopically and assessed for glomerular features. Evidence of urinary infection should be cultured when clinically appropriate. Simultaneously, clinicians should assess obesity, glucose metabolism, lipids, sodium exposure, sleep-disordered breathing, medications, supplements, stimulant use, and family history because urinary findings frequently occur within a broader cardiometabolic or systemic disease phenotype.

Risk increases substantially when abnormalities cluster. Persistent hypertension plus albuminuria or proteinuria is more concerning than either finding alone; hypertension plus proteinuria and hematuria is more concerning still. The addition of reduced eGFR, abnormal renal imaging, edema, low complement,

or systemic features should prompt timely specialist evaluation. Conversely, transient isolated abnormalities should be confirmed before the adolescent is labeled as having kidney disease. This graduated strategy prevents both under-recognition of renal pathology and unnecessary anxiety or investigation from physiological adolescent urinary variation.

Therapeutic and Follow-up Implications

Management ultimately depends on whether the urine abnormality represents primary renal disease, hypertension-mediated target-organ injury, or an unrelated transient finding. Lifestyle treatment remains foundational for primary hypertension and should include weight optimization when appropriate, dietary sodium reduction, regular physical activity, adequate sleep, and attention to overall diet quality. When pharmacological therapy is required, drug selection is influenced by comorbid disease. In patients with chronic kidney disease, diabetes, or clinically significant proteinuria, blockade of the renin-angiotensin system is often particularly attractive because of its blood pressure-lowering and antiproteinuric effects, provided kidney function and serum potassium are appropriately monitored. [11]

Follow-up should assess both blood pressure response and evolution of the urinary abnormality. Resolution of transient proteinuria after recovery from illness or avoidance of strenuous exercise is reassuring. Persistence or progression of albuminuria despite improved blood pressure control suggests the need to reconsider underlying kidney disease, metabolic contributors, medication adherence, and adequacy of blood pressure assessment. Serial trends are generally more informative than isolated measurements because both blood pressure and urinary protein excretion exhibit substantial biological variability during adolescence.

Several unanswered questions limit the clinical interpretation of urinary abnormalities in hypertensive adolescents. First, large prospective cohorts are needed to determine whether modest elevations in ACR during adolescence independently predict chronic kidney disease, cardiovascular events, or sustained hypertension in adulthood. Second, pediatric thresholds for clinically meaningful albuminuria may require greater consideration of sex, pubertal stage, muscle mass, exercise, and obesity because urinary creatinine excretion changes markedly during adolescence. Third, the incremental value of albuminuria beyond ABPM, obesity measures, metabolic markers, and echocardiographic evidence of target-organ damage remains incompletely defined.

Emerging tubular biomarkers also require rigorous validation before clinical implementation. Their greatest future value may lie not in replacing urinalysis or ACR but in identifying a phenotype of very early renal stress among hypertensive adolescents whose conventional kidney tests remain normal. Studies integrating urinary proteomics, tubular injury markers, ambulatory blood pressure phenotypes, vascular imaging, and longitudinal kidney outcomes could eventually establish whether a multibiomarker strategy improves risk prediction. Until such evidence exists, conventional urinalysis, quantitative protein assessment when indicated, kidney function, and careful blood pressure phenotyping remain the most clinically defensible approach.

Conclusion

Urinalysis is a simple but clinically powerful component of the assessment of adolescents with hypertension because it can reveal both occult renal causes of secondary hypertension and early evidence of hypertension-mediated kidney injury. Persistent proteinuria or albuminuria carries the greatest potential significance, particularly when accompanied by sustained or severe hypertension, obesity, metabolic dysfunction, hematuria, reduced kidney function, or other manifestations of target-organ damage. In contrast, transient proteinuria, orthostatic proteinuria, and isolated microscopic hematuria are common in adolescence and should be confirmed before pathological significance is assigned. The optimal interpretation of urinary abnormalities therefore depends not on the presence of an isolated dipstick finding but on persistence, quantitative assessment, urinary sediment, kidney function, ABPM phenotype, and overall cardiovascular risk. Integrating these elements allows urinalysis to evolve from a routine screening test into a practical tool for identifying adolescents in whom hypertension may already be affecting—or originating from—the kidney.

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