





Hematuria in children: causes and evaluation

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Hematuria is the presence of blood in the urine and is classified as either gross hematuria or microscopic hematuria. There are many causes of hematuria, and the differential diagnosis depends on the presence or absence of comorbidities and whether it is glomerular or non-glomerular. When hematuria in children is symptomatic or persistent, an evaluation of the cause is essential. The causes of hematuria and basic approaches to its diagnosis are discussed in this review.

Keywords: Hematuria; Kidney; Pediatrics; Urinary tract

Introduction

Hematuria is the presence of blood in the urine, defined as five or more red blood cells (RBCs) per high-power field of view ($\times 400$) [1]. Even if the urine is red in color or the urine dipstick detects urinary occult blood in the urine, the urine may not contain RBCs; therefore, microscopic examination is required to confirm hematuria. Hematuria may be visible to the naked eye (gross) or detectable only by urinalysis (microscopic), and

can be divided into intermittent and persistent hematuria based on duration, and asymptomatic and symptomatic hematuria based on the presence or absence of symptoms [1,2]. Any symptomatic or persistent hematuria in children should be evaluated for a cause, and asymptomatic microscopic hematuria is clinically significant if the microscopic hematuria persists on three or more urine examinations, usually 2 weeks apart [1]. This review describes hematuria in children, its causes, and basic approaches to diagnosis, including gross hematuria, mi-

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microscopic hematuria with clinical symptoms, asymptomatic microscopic hematuria, and microscopic hematuria with proteinuria.

Causes of hematuria in children

The causes of hematuria in children are numerous, and the differential diagnosis can range from infections to hypercalciuria, nephrolithiasis, vascular abnormalities including nutcracker syndrome, acute and chronic glomerular diseases, kidney and urinary tract malformations, and tumors. And the differential diagnosis depends on whether the hematuria is glomerular or non-glomerular, and on the presence of comorbidities, such as pain (Table 1). Glomerular hematuria is reddish-brown or cola-colored without blood clots, may show dysmorphic RBCs or RBC casts, and may be associated with proteinuria, hypertension, and decreased kidney function [3]. Asymptomatic microscopic hematuria, if persistent, has clinical significance and requires differential diagnosis of the cause (Fig. 1) [1].

Gross hematuria

The most common causes of gross hematuria are urinary tract infections (UTI) and hypercalciuria, but urethral hemorrhage, trauma, congenital anomalies of kidney and urinary tract (CAKUT), coagulopathy, nephrolithiasis, and glomerulonephritis, such as immunoglobulin A nephropathy (IgAN), can also be

responsible. Gross hematuria with pain is most often non-glomerular hematuria due to infection, stones, or structural abnormalities of the kidney and urinary tract, whereas hematuria due to glomerular causes is painless. UTI should be considered in the presence of gross hematuria, fever, dysuria, and lower urinary tract symptoms (urgency, frequency, etc.), while intermittent sharp flank pain and gross hematuria should raise the suspicion of urinary tract stones or renal vein thrombosis. In addition, some cases of nutcracker syndrome may be associated with pain, and hypercalciuria may cause recurrent episodes of gross or microscopic hematuria without stones. Rarely, tumors of the kidney and urinary tract may be found as gross hematuria with palpable masses [4]. The most common causes of glomerulonephritis are IgAN and poststreptococcal glomerulonephritis (PSGN) [4]. Gross hematuria in a patient with symptoms of an upper respiratory tract infection (within 1 week) suggests IgAN, and gross hematuria in a patient with a recent (more than 1–2 weeks ago) upper respiratory tract infection suggests PSGN.

In a pooled analysis of three U.S. studies and one Indian study investigating the causes of asymptomatic gross hematuria in a total of 710 patients, hypercalciuria and nephrolithiasis were identified in 13.7%, UTI in 8.4%, CAKUT in 7.0%, and trauma in 6.8%, with glomerular causes in 18.5%, followed by IgAN, PSGN, and other glomerulopathies. The cause of gross hematuria could not be determined in 33.3% of cases (Table 2) [5–8].

Table 1. Causes of hematuria in children

Glomerular causes		Non-glomerular causes
Kidney	Systemic	
PSGN/PIGN ^{a)}	Systemic lupus erythematosus	Hypercalciuria ^{a)}
IgAN ^{a)}	IgA vasculitis (HSPN) ^{a)}	Urinary tract infections ^{a)}
TBMD ^{a)}	Hemolytic uremic syndrome ^{a)}	Tubulointerstitial nephritis
MPGN/C3 glomerulopathy ^{a)}	Granulomatosis with polyangiitis ^{a)}	CAKUT ^{a)}
Membranous glomerulonephritis	Goodpasture syndrome ^{a)}	Vascular malformations/abnormalities (thrombosis) ^{a)}
FSGS	Polyarteritis nodosa	Nutcracker syndrome ^{a)}
Alport syndrome ^{a)}	Sickle cell nephropathy ^{a)}	Nephrolithiasis ^{a)}
Other chronic glomerulonephritis		ADPKD ^{a)}
		Trauma ^{a)}
		Tumors ^{a)}
		Coagulopathy ^{a)}
		Urethral hemorrhage ^{a)}
		Exercise-induced hematuria ^{a)}

PSGN, poststreptococcal glomerulonephritis; PIGN, postinfectious glomerulonephritis; IgAN, immunoglobulin A (IgA) nephropathy; TBMD, thin basement membrane disease; MPGN, membranoproliferative glomerulonephritis; C3, complement 3; FSGS, focal segmental glomerulosclerosis; HSPN, Henoch–Schönlein purpura nephritis; CAKUT, congenital anomalies of kidney and urinary tract; ADPKD, autosomal dominant polycystic kidney disease.

^{a)}Gross hematuria may occur.

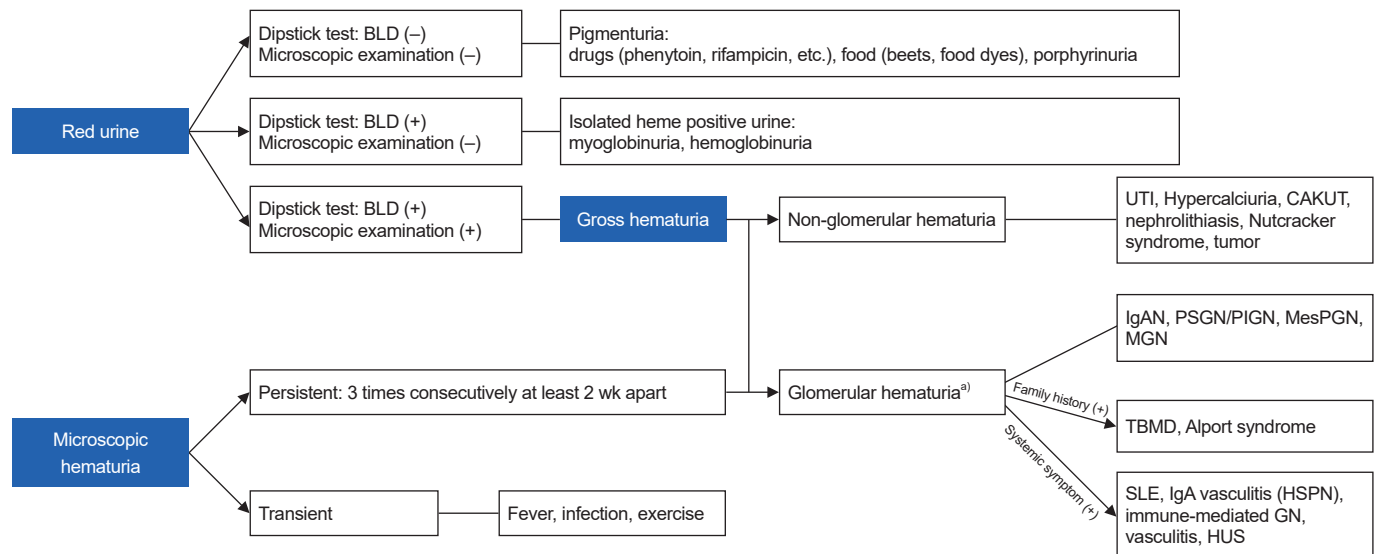


Fig. 1. Approach to a child with hematuria. BLD, blood; UTI, urinary tract infection; CAKUT, congenital anomalies of kidney and urinary tract; IgAN, immunoglobulin A (IgA) nephropathy; PSGN, poststreptococcal glomerulonephritis; PIGN, postinfectious glomerulonephritis; MesPGN, mesangial proliferative glomerulonephritis; MGN, membranous glomerulonephritis; TBMD, thin basement membrane disease; SLE, systemic lupus erythematosus, HSPN, Henoch–Schönlein purpura; GN, glomerulonephritis; HUS, hemolytic uremic syndrome. ^{a)}Possible occurrence of dysmorphic red blood cells (RBCs), formation of RBC casts, hypertension, proteinuria, and decreased renal function

Microscopic hematuria with clinical symptoms

Persistent microscopic hematuria should be evaluated for systemic symptoms such as fever, malaise, abdominal/back pain, high blood pressure, and edema (glomerulonephritis); urinary tract symptoms such as urinary frequency, dysuria, and nocturia (UTI, nephrolithiasis); or other non-urinary symptoms such as rash, purpura, and joint pain (systemic lupus erythematosus [SLE] or IgA vasculitis [Henoch–Schönlein purpura nephritis]). In particular, a family history of kidney disease and the presence of hearing loss or eye abnormalities may raise suspicion for Alport syndrome. Formerly known as benign familial hematuria, thin basement membrane disease (TBMD), but some of them showed defects in type 4 collagen $\alpha 3$ or $\alpha 4$ chain and will develop end-stage kidney disease later in life [9]. Therefore, recent articles have suggested that all cases with carriers of variants in these genes should be classified as autosomal dominant Alport syndrome (Table 3) [9].

Asymptomatic microscopic hematuria

Intermittent microscopic hematuria can be caused by UTI, trauma, fever, or strenuous exercise. The most common glomerular causes of persistent asymptomatic hematuria are IgAN, TBMD or Alport syndrome, and various forms of chronic glomerulonephritis, including mesangial and membranous glomerulonephritis. Non-glomerular causes predominantly

include hypercalciuria, followed by nephrolithiasis, CAKUT, and nutcracker syndrome, but in many cases no clear cause can be found [5]. One systematic review of 857 children with asymptomatic isolated microscopic hematuria found no cause for the hematuria in 57.6% of all patients. Among patients with an identified cause, 15.7% had Alport syndrome/TBMD, 10.4% had IgAN, 9.4% had hypercalciuria or nephrolithiasis, and less than 3% had other causes [10]. In a pooled analysis of eight studies that examined the causes of microscopic hematuria, 54.9% of the 1,210 patients had no identifiable cause, with TBMD accounting for 11.7%, IgAN for 9.92%, SLE for 7.02%, and hypercalciuria for 7.02%, and other causes including other glomerulonephritis, UTI, and nephrolithiasis (Table 3) [5,11–17].

Microscopic hematuria with proteinuria

If microscopic hematuria persists with proteinuria, it is likely to be of glomerular origin. In a pooled analysis of five studies investigating the causes of microscopic hematuria with proteinuria, 31.1% of 425 patients had TBMD, 27.3% had IgAN, 13.9% had other glomerulonephritis, 7.29% had SLE, 2.35% had IgA vasculitis, 1.88% had orthostatic proteinuria, 1.41% had PSGN, and 1.18% had Alport syndrome. No diagnosis was made in 12.2% of cases (Table 4) [11,14,15,17,18].

Table 2. Causes of gross hematuria

Cause	Bergstein et al. (2005) [5]	Youn et al. (2006) [6]	Greenfield et al. (2007) [7]	Mishra et al. (2022) [8]	Summary, No. (%) ^{b)}
No diagnosis/normal	86	26	118	7	237 (33.3)
Hypercalciuria	55	9	0	7	71 (10.0)
IgAN	34	13	^{a)}	2	49 (6.89)
PSGN	21	3	^{a)}	17	41 (5.77)
Other GN	5	1	^{a)}	11	17 (2.39)
Urinary tract infection	1	8	48	3	60 (8.44)
Alport syndrome	3	6	0	0	9 (1.27)
Urethrorrhagia	0	8	52	0	60 (8.44)
Nephrolithiasis	0	3	18	5	26 (3.66)
Exercise	8	0	0	0	8 (1.13)
IgA vasculitis	0	1	0	4	5 (0.70)
Sickle cell trait	3	2	0	0	5 (0.70)
CAKUT	5	0	45	0	50 (7.03)
ADPKD	3	1	0	0	4 (0.56)
TBMD	3	0	0	0	3 (0.42)
Chronic kidney disease	0	0	0	2	2 (0.28)
Tumor	1	1	7	0	9 (1.27)
Trauma	0	0	48	0	48 (6.75)

IgAN, immunoglobulin A nephropathy; PSGN, poststreptococcal glomerulonephritis; GN, glomerulonephritis; CAKUT, congenital anomalies of kidney and urinary tract; ADPKD, autosomal dominant polycystic kidney disease; TBMD, thin basement membrane disease.

^{a)}The value of IgAN, PSGN, and other GN are 7; ^{b)}Greenfield et al. cases were excluded from the summary values for IgAN, PSGN, and other GN.

Table 3. Causes of persistent microscopic hematuria

	Vehaskari et al. (1979) [11]	Trachtman et al. (1984) [12]	Piqueras et al. (1998) [13]	Lin et al. (2001) [14] ^{a)}	Bergstein et al. (2005) [5]	Lee et al. (2006) [15]	Moghtaderi et al. (2014) [16]	Güven et al. (2016) [17]	Summary No. (%)
No diagnosis/ normal	22	25	32	73	274	136	5	97	664 (54.9)
TBMD	0	10	23	9	0	97	0	2	141 (11.7)
IgAN	2	1	39	31	1	46	0	0	120 (9.92)
Hypercalciuria	0	0	4	17	57	0	7	0	85 (7.02)
Lupus	0	0	0	84	0	1	0	0	85 (7.02)
Other GN	1	0	16	6	1	3	0	0	27 (2.23)
Hilar vasculopathy/vascular C3	0	5	15	0	0	0	0	0	20 (1.65)
UTI	0	0	0	19	0	0	0	0	19 (1.57)
PSGN	0	0	0	5	4	4	0	0	13 (1.07)
Nephrolithiasis	0	0	0	0	0	0	13	0	13 (1.07)
CAKUT	2	0	0	3	5	1	0	0	11 (0.91)
Alport syndrome	0	1	2	0	0	1	0	2	6 (0.50)
IgA vasculitis	0	0	0	3	0	0	0	0	3 (0.25)
Tumor	0	0	0	0	0	0	1	0	1 (0.08)
Blunt injury	0	0	0	1	0	0	0	0	1 (0.08)
Hemophilia	0	0	0	1	0	0	0	0	1 (0.08)

TBMD, thin basement membrane disease; IgAN, immunoglobulin A (IgA) nephropathy; GN, glomerulonephritis; C3, complement 3; UTI, urinary tract infection; PSGN, poststreptococcal glomerulonephritis; CAKUT, congenital anomalies of kidney and urinary tract.

^{a)}Lin's study was a school screening study of Taiwanese, where most were asymptomatic, but some lupus patients developed symptoms during follow-up.

Evaluation of hematuria in children

Persistent hematuria in children should be differentiated by careful history taking, physical examination, blood and urine tests, and imaging studies, if indicated [19]. No cause of hematuria can be found in 40% to 70% of patients [20].

History taking

A detailed description of the urine is important to identify the origin of hematuria. Urine color, timing (initial, terminal, throughout the urinary stream), persistence, and presence of blood clots should be assessed. The history taking should include recent trauma, fever, strenuous exercise, recent respiratory or skin infection, menstruation, etc. at the time of urinalysis. Look for decreased urine output or weight change, lower urinary tract symptoms, flank pain, and fever, as well as any associated extra-renal symptoms such as rash, joint pain, abdominal pain/blood in stool, and eye/hearing problems. Be sure to check for recurrent gross hematuria, purpura or blood clotting disorders, infections, and medications such as antibiotics, anticancer drugs, anti-inflammatory drugs, anticonvulsants, and calcium/vitamin D supplements. It is also important to check for a family history of kidney disease (persistent hematuria, chronic kidney disease, glomerulonephritis, cystic kidney disease, hearing loss, etc.).

Physical examination

The patient's physical examination should include measurement of blood pressure, height, weight and assessment for generalized or localized edema. Furthermore, patients with a family history of kidney disease should undergo comprehensive examinations of their hearing and ocular systems.

Urinalysis

The dipstick uses a chromogenic reaction (occult blood) to detect the presence of heme protein in urine. Urine can also test positive for occult blood in the presence of hemoglobinuria or myoglobinuria. The stick should be dipped into the urine, the excess urine should be shaken off, and the results can be read in 1 minute. Care should be taken as false negatives (diluted urine, vitamin C overdose, reducing agents such as formalin) and false positives (hemoglobinuria, myoglobinuria, concentrated urine, etc.) are possible. A result of 1+ is considered a positive occult blood reaction. If the occult blood test is positive, microscopic examination will reveal the presence of RBCs in the urine. Urine microscopy can reveal RBC morphology, RBC casts and urinary crystals. A predominance of dysmorphic erythrocytes (>25%–75%), acanthocytes (>5%), or RBC casts suggests glomerular hematuria, and urinary crystals (crystalluria-calcium oxalate crystals) suggest non-glomerular causes, such as hypercalciuria [21,22]. The single voided urine calcium to creatinine (Cr) ratio (urine calcium [mg/dL]/urine Cr [mg/dL]) is considered

Table 4. Causes of persistent microscopic hematuria with proteinuria

Cause	Vehaskari et al. (1979) [11]	Hisano and Ueda (1989) [18]	Lin et al. (2001) [14]	Lee et al. (2006) [15]	Guyen et al. (2016) [17]	Summary, No. (%)
TBMD	0	0	1	130	1	132 (31.1)
IgAN	0	29	12	75	0	116 (27.3)
Other GN ^{a)}	1	25	13	18	2	59 (13.9)
No diagnosis/normal	4	0	7	40	1	52 (12.2)
Lupus	0	1	30	0	0	31 (7.29)
IgA vasculitis	0	10	0	0	0	10 (2.35)
Orthostatic proteinuria	0	0	8	0	0	8 (1.88)
PSGN	0	0	2	4	0	6 (1.41)
Alport syndrome	0	1	0	3	1	5 (1.18)
VUR	0	0	2	0	0	2 (0.47)
ATN	0	0	1	0	0	1 (0.24)
Polyarthritis	1	0	0	0	0	1 (0.24)
Hypercalciuria	0	0	1	0	0	1 (0.24)
Blunt injury	0	0	1	0	0	1 (0.24)

TBMD, thin basement membrane disease; IgAN, immunoglobulin A (IgA) nephropathy; GN, glomerulonephritis; PSGN, poststreptococcal glomerulonephritis; VUR, vesicoureteral reflux; ATN, acute tubular necrosis.

^{a)}Membranoproliferative glomerulonephritis, mesangial proliferative glomerulonephritis, focal segmental glomerulosclerosis, minimal change disease, complement 3 glomerulonephritis, hemolytic uremic syndrome.

positive for calciuria if the results are ≥ 0.2 in patients 2 years of age or older, ≥ 0.4 in patients 1–2 years of age, ≥ 0.6 in patients 6–12 months of age, and ≥ 0.8 mg/mg in patients younger than 6 months of age [23]. The 24-hour urine test is considered positive for calciuria if the calcium excretion is ≥ 4 mg/kg [23].

If persistent hematuria is accompanied by proteinuria, it is likely to be of glomerular origin and should be evaluated by qualitative and quantitative proteinuria testing. However, orthostatic proteinuria is common in children, so urine should be collected immediately upon awakening to rule this out. The dipstick test is a qualitative test for proteinuria where the positive threshold depends on the urine specific gravity; if the specific gravity is <1.010 , it shows trace amounts of protein, and if the specific gravity is >1.015 , proteinuria should be suspected at $>1+$. In this case, a quantitative test should be performed to confirm proteinuria. There are two ways to quantify protein in urine: a single urine collection and a 24-hour urine collection. A single voided urine protein to Cr ratio (urine protein [mg/dL]/urine Cr [mg/dL]) is considered positive for proteinuria if the results are ≥ 0.2 in patients 2 years of age or older and ≥ 0.5 in patients younger than 2 years of age. Single voided urine albumin to Cr

ratio (urine albumin [mg/dL]/urine Cr [mg/dL]) is considered positive for proteinuria if the results are ≥ 30 mg/g Cr. The 24-hour urine test is considered positive for proteinuria if the results are ≥ 100 mg/m²/day (or ≥ 4 mg/m²/hr) or ≥ 150 mg/day [22]. However, due to the mixing of urine during the daytime, protein above the reference value may be detected even in cases of orthostatic proteinuria.

Estimating kidney function

Serum Cr or cystatin C can be used to estimate glomerular filtration rate. A rate of more than 90 mL/min/1.73 m² indicates normal function. Typically, the Bedside Schwartz equation is used, which uses Cr and height, and sometimes an equation that takes cystatin C into account is used as well (Table 5) [24,25].

Additional tests

Depending on the results of the initial assessment, the following optional tests may be performed (Fig. 2) [26]. For glomerular hematuria, serum complements 3 and 4, antinuclear antibody, double-stranded DNA antibody, anti-neutrophil cytoplasmic antibody, hepatitis B virus antigen/antibody, kidney biopsy, and

Table 5. Estimated glomerular filtration rate in children

1. Bedside Schwartz equation
$eGFR = 0.413 \times (\text{Height [cm]}/\text{Serum Cr [IDMS Cr, mg/dL]})$
2. Cr-cystatin C-based CKiD equation
$eGFR = 39.8 \times (\text{Height [cm]}/\text{serum Cr} \times 100)^{0.456} \times (1.8/\text{CysC}^a [\text{mg/L}])^{0.418} \times (30/\text{BUN [mg/dL]})^{0.079} \times (1.076^{\text{male}})(1.00^{\text{female}}) \times (\text{Height [cm]}/140)^{0.179}$
eGFR, estimated glomerular filtration rate; Cr, creatinine; IDMS, isotope dilution mass spectrometry; CKiD, The Chronic Kidney Disease in Children Cohort Study; CysC, cystatin C; BUN, blood urea nitrogen.
^a If the cystatin C measurement is calibrated according to the International Federation for Clinical Chemistry and Laboratory Medicine calibration, use the cystatin C/1.17 value.

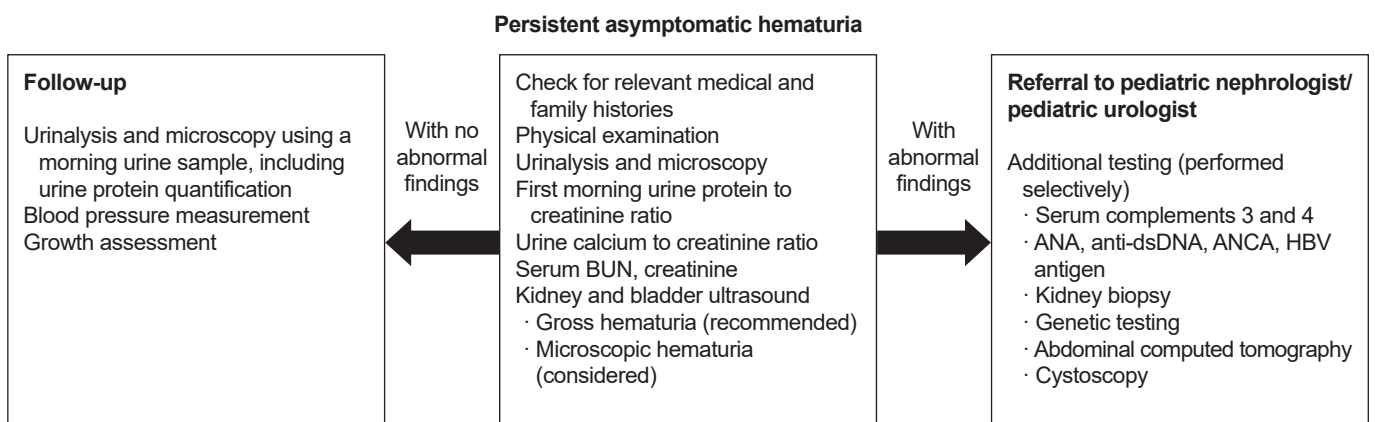


Fig. 2. Initial diagnostic process for children with persistent asymptomatic hematuria. BUN, blood urea nitrogen; ANA, antinuclear antibody; anti-dsDNA, anti-double-stranded DNA; ANCA, anti-neutrophil cytoplasmic antibody; HBV, hepatitis B virus. Modified from Park et al. *Kidney Res Clin Pract* 2024 Mar 7 [Epub]. <https://doi.org/10.23876/j.krcp.23.231> [26].

genetic testing may be performed to identify the underlying disease; for non-glomerular hematuria, Doppler ultrasound, abdominal computed tomography, genetic testing, and cystoscopy may be considered.

Conclusion

There are many causes of hematuria in children, and if it persists, the diagnostic process includes taking a detailed history, physical examination, and laboratory and imaging studies. Children with findings suggestive of glomerulonephritis, such as proteinuria, hypertension, persistently decreased kidney function, or decreased serum complement 3, a family history of kidney disease, or congenital or acquired structural abnormalities, should be referred to a pediatric nephrologist. Furthermore, even if the cause of hematuria is not identified at the time of initial diagnosis, regular follow-up is imperative, as unresolved hematuria can potentially lead to chronic kidney disease.

Conflicts of interest

Eujin Park, Myung Hyun Cho, Hyun Kyung Lee, Hee Gyung Kang, Jin-Soon Suh, and Eun Mi Yang are editorial board members of the journal but were not involved in the peer reviewer selection, evaluation, or decision process of this article. No other potential conflicts of interest relevant to this article were reported.

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Conceptualization: HGK

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