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4th Edition



IAP Specialty Series on Pediatric Nephrology

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CHAPTER 9

Hematuria in Children

Mukta Mantan, Akanksha Mahajan

INTRODUCTION

Hematuria is an uncommon condition in childhood. However, it is frightening to the parents and results in an almost immediate referral to a pediatrician. Children are referred for the presence of gross hematuria, persistent isolated microscopic hematuria, and microscopic hematuria with clinical symptoms or with proteinuria. An approach to these entities will be discussed in this chapter.

The incidence of gross hematuria in children is estimated to be around 0.13%. Asymptomatic microscopic hematuria is common in unselected populations of children, with a prevalence that ranges from 0.4 to 4.1%. However, on repeated evaluations, the prevalence of persistent microscopic hematuria is about 0.5% based on the surveys of school-going children.

DEFINITION

Hematuria is defined as the presence of five or more red blood cells (RBCs) per high power field (hpf) in multiple centrifuged urine examinations (at least 3; 1 week apart).

CONFIRMATION OF HEMATURIA

Gross hematuria should be differentiated from discolored urine. Ingestion of many substances imparts a reddish color to urine **(Box 1)**. Presence of RBCs in urine should be confirmed by the following methods.

Dipsticks

This test is based on the peroxidase activity of hemoglobin that catalyzes a reaction between hydrogen peroxide and the chromogen tetramethylbenzidine to produce an oxidized chromogen, which has a green-blue color. It can detect trace amounts of hemoglobin (rather than the presence of RBCs) and myoglobin. It is important to briefly dip the strip in the urine, tap off excess urine, and read the strip at the recommended time (usually 1 minute). Dipsticks have a sensitivity of 100% and a specificity of 99% in detecting 10 RBC/μL of urine. False positive dipstick results may occur due to urinary contaminants, like bleach and microbial peroxidases in urinary tract infections (UTIs). False negative reports point to very dilute urine or the presence of reducing substances like ascorbic acid.

Microscopy

Microscopic examination of urine requires centrifuging 10 mL of a fresh urine sample at

BOX 1: Causes of red urine.

- *Food items:* Ingestion of beet root, blackberries, and food-coloring agents
- *Drugs:* Nitrofurantoin, phenolphthalein, pyridium, rifampicin, phenothiazines, sulfasalazine, and metronidazole
- *Metabolites:* Homogentisic acid, melanin, methemoglobin, porphyrins, urates, tyrosinosis, and alkaptonuria

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2,000 rpm for about 5 minutes. The supernatant is decanted, and the sediment is resuspended in 0.5 mL of urine. This sample is then carefully examined under high power of light microscope counting RBCs in at least 20 fields, and the average is reported.

GLOMERULAR AND EXTRA-GLOMERULAR HEMATURIA

Hematuria may occur due to bleeding from the urinary tract or as a manifestation of glomerulonephritis. Bright-red urine, visible clots, or crystals with normal-looking eumorphic RBCs suggest bleeding from the urinary tract while cola-colored or brownish urine, presence of RBC casts, and deformed (dysmorphic) RBCs suggest glomerular bleeding. The presence of >20% dysmorphic RBCs on microscopy is suggestive of glomerular hematuria. Phase contrast microscopy helps to detect dysmorphic RBCs better than Wright's stain or light microscopy. Presence of acanthocytes of >5% also favors a diagnosis of glomerular hematuria. Acanthocytes are identified by their unique ring form with vesicle-shaped protrusions. The presence of RBCs with granular casts is suggestive of glomerular disease whereas the presence of RBCs with white blood cells is suggestive of infection or interstitial or glomerular inflammation. The presence of eosinophils makes the diagnosis of interstitial nephritis more likely. Crystals like calcium oxalate, urate, calcium phosphate, struvite and rarely cystine indicate nephrolithiasis. Features differentiating glomerular from extraglomerular hematuria are given in **Table 1**.

TABLE 1: Differentiation of glomerular and extra-glomerular hematuria.

Glomerular hematuria	*Extraglomerular hematuria*
Urine color red or brown	Urine bright red usually
Clots are absent	Clots present
Proteinuria (2+ or more)	No proteinuria or up to 1+
Red cell casts	Absent cellular casts
Dysmorphic RBC >20%	Dysmorphic RBC <20%
Acanthocytes >5%	No acanthocytes
Usually, painless	Often associated with dysuria, urgency, and frequency

(RBC: red blood cell)

HISTORY AND PHYSICAL EXAMINATION

The first step in the evaluation of hematuria is a detailed history and a thorough physical examination. A history of periorbital puffiness, oliguria, presence of painless, dark-colored urine, and the presence of edema or hypertension suggest a glomerular cause. Presence of fever, abdominal pain, dysuria, frequency, and recent enuresis in older children suggest UTI. Recurrent abdominal pain with hematuria occurs in urolithiasis and hypercalciuria. Family history of hematuria, hypertension, and deafness may point to hereditary nephropathies. An attempt should be made to distinguish between glomerular and extraglomerular hematuria. Some clinical pointers that enable us to make a differential diagnosis and help in further planning the investigations are given in **Table 2**. A history of a recent throat or skin infection suggests infection related glomerulonephritis (IRGN). Information regarding exercise, menstruation, recent bladder catheterization, intake of certain drugs or toxic substances, or passage of a calculus may also help in establishing the differential diagnoses.

In the general physical examination, it is important to measure the blood pressure (with an appropriate-sized cuff), look for edema, purpura, other rashes, and arthritis. Ocular manifestations like recurrent corneal erosions, cataract, macular flecks, and anterior lenticonus have been reported

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TABLE 2: Clinical pointers in history to identify the cause of hematuria.

Clinical signs/symptoms	*Likely diagnosis*
Edema, hypertension, and oliguria	Glomerulonephritis
Dysuria, loin pain, and colic	Calculi and hypercalciuria
Joint pains, rash, and edema	Systemic lupus erythematosus, Henoch–Schönlein purpura, and vasculitis
Positive family history and deafness	Alport disease and thin basement membrane disease
Renal lump	Polycystic kidney disease, Wilm's tumor, and hydronephrosis
Bleeding tendency	Hemophilia, idiopathic thrombocytopenic purpura, disseminated intravascular coagulation, and von Willebrand disease

TABLE 3: Causes of hematuria.

Glomerular	*Extraglomerular*
• *Acute onset:* – Acute glomerulonephritis (infection related glomerulonephritis and others*) – Rapidly progressive glomerulonephritis – Hemolytic uremic syndrome • *Chronic glomerulonephritis:* – Membranous and membranoproliferative glomerulonephritis – IgA nephropathy – Thin basement membrane disease – Alport syndrome – Focal segmental glomerulosclerosis – Goodpasture syndrome • *Multisystem disease:* Systemic lupus erythematosus, IgA vasculitis associated nephropathy, and vasculitis	• Urinary tract infections • Urolithiasis and hypercalciuria • Cystic kidney disease • Vascular malformations (kidney or bladder) • Wilm's tumor • Renal vein thrombosis • Renal artery thrombosis • Trauma • Bleeding and clotting disorders • Drugs (nonsteroidal anti-inflammatory drugs) • Hemorrhagic cystitis (viral or cyclophosphamide induced) • Urethritis • Tuberculosis • Nutcracker syndrome • Menarche

*Typhoid, pneumococcal pneumonia, meningococcemia, shunt nephritis, bacterial endocarditis varicella, and mumps associated.

in Alport syndrome. A per abdomen examination should be done to detect any renal lumps or hepatomegaly; local examination of the genitalia to rule out meatal stenosis and ulceration.

CAUSES

Gross hematuria occurs infrequently, though in most patients a cause can be identified. Microscopic hematuria is more common, but it is benign on most occasions. Studies have shown that in almost 75% cases, the microscopic hematuria would disappear on repeated evaluation over a period of 1 year. Some common causes of hematuria are listed in **Table 3**.

The most common cause of glomerular hematuria in children is PIGN now known as infection-related glomerulonephritis (IRGN). It occurs most commonly after a streptococcal infection. However, other bacterial infections, like infective endocarditis, shunt nephritis, pneumococcal pneumonia, *Salmonella* and meningococcemia, can also result in IRGN. Viral infections, such as hepatitis B and C, infectious mononucleosis, mumps, measles, varicella,

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echovirus, parvovirus, and coxsackievirus, can also cause PIGN. Parasitic infections, such as malaria and toxoplasmosis, have been rarely associated with IRGN.

Diagnosis and Management of Infection-related Glomerulonephritis

The investigations useful in confirming the diagnosis are urinalysis, complete blood count, antistreptolysin O (ASLO) titers, blood urea, serum creatinine, and complement C3 levels. Most patients with IRGN would have elevated ASLO titers (>200 IU/L), low complement levels (<60 mg/dL), and mildly deranged kidney function tests. Urinalysis always shows glomerular hematuria, presence of RBC casts, and leukocyturia. Proteinuria is usually <2+. If these tests are negative and there is resolution of symptoms, a presumptive diagnosis of IRGN can be made. Most patients with an acute IRGN would resolve their hematuria and edema within 10–14 days; the kidney functions also normalize by this time. Persistence of gross hematuria or proteinuria beyond 4 weeks is unusual and often becomes an indication for kidney biopsy in these patients. Hypertension and fluid overload can be managed by use of diuretics, like furosemide, in doses of 2–4 mg/kg/day in two or three divided doses. If hypertension persists, it can be controlled with calcium-channel blockers or use of β-blockers. Amlodipine in doses of 0.2–0.5 mg/kg/day; labetalol in doses of 2–5 mg/kg/day is often sufficient in controlling elevated blood pressure. Use of angiotensin-converting enzyme inhibitors and angiotensin-receptor blockers is not recommended as most patients with acute glomerulonephritis have a transient reduction in their glomerular filtration rate. The duration for use of diuretics and antihypertensives is usually <2 weeks. Patients with severe fluid overload and severe acute kidney injury may require kidney replacement therapy in the form of peritoneal or hemodialysis. This occurs in <10% of patients with acute glomerulonephritis.

Recurrent Gross Glomerular Hematuria

Most patients with IRGN do not have a recurrence of hematuria. Patients with chronic glomerulonephritis, such as membranous nephropathy, membranoproliferative disease, are likely to have recurrence of hematuria. However, most of these patients have concomitant hypertension, edema, and proteinuria. Patients with multisystem involvement, like immunoglobulin A (IgA) vasculitis related nephropathy known as Henoch-Schönlein purpura in past, systemic lupus erythematosus (SLE), other vasculitis, may have fever, joint pains, and rash; sickle cell nephropathy may have associated bone pains and hepatomegaly. Most of these conditions would require a kidney biopsy for identification of the underlying condition. If episodes of painless hematuria are recurring, a possibility of IgA nephropathy or Alport disease should also be kept.

Immunoglobulin A nephropathy is the most common cause of glomerulonephritis in the world among adults though less common in pediatric age groups. It typically presents recurrent synpharyngitic hematuria but may also manifest as persistent microscopic hematuria. The degree of proteinuria and histological findings determine the treatment and outcome. The disease is managed with angiotensin-converting enzyme inhibitors to control blood pressure and reduce proteinuria and immunosuppression in selected cases.

Extraglomerular Causes

Local infection, perineal irritation, or trauma to genitalia can cause gross hematuria. UTI, urolithiasis, and disorders of coagulation can also result in hematuria. Hypercalciuria,

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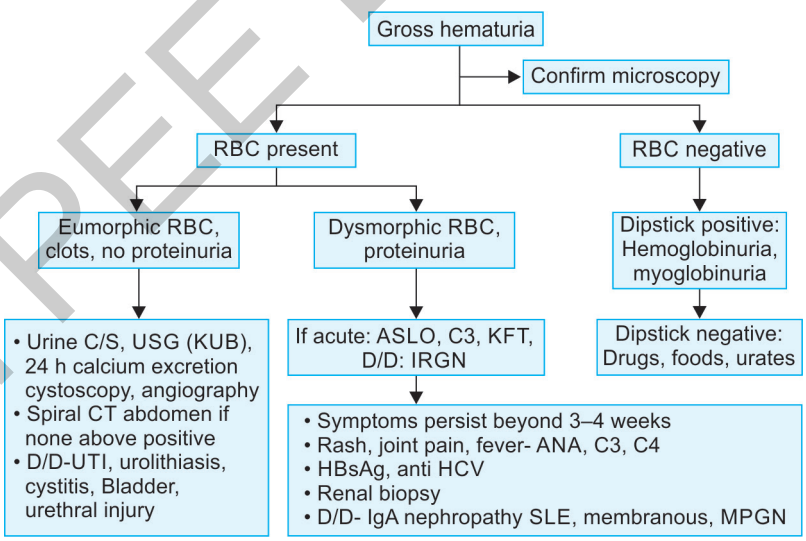
urolithiasis, and cystic disease of kidneys can result in recurrent gross hematuria. Children with nephrolithiasis present with colicky abdominal or loin pain, dysuria, and fever. Most have microscopic hematuria, while 30–50% may have gross hematuria. Eosinophilic cystitis presenting with terminal hematuria and irritative urinary symptoms is a rare diagnosis in children.

INVESTIGATIONS

Gross hematuria from the bladder or urethra is pink or bright red in color and may have visible clots; proteinuria is insignificant, and microscopic examination reveals eumorphic RBCs. Macroscopic hematuria of glomerular origin is associated with significant proteinuria. In a child with hematuria, urine should be examined for proteins, RBCs, and RBC casts. Differentiation of glomerular from extraglomerular hematuria is given in **Table 1**. Further evaluation will be based on clinical signs and symptoms **(Table 2)**.

An episode of gross hematuria needs evaluation. After confirming hematuria, the algorithm of **Flowchart 1** can be followed. If the onset is acute and proteinuria is present, the likelihood of an episode of IRGN is high. The relevant investigations, like ASLO titers, complement C3 levels, and kidney function test (KFT) should be done. An elevated ASLO (>200 IU/L), low C3 levels with normal or deranged KFT are confirmative of IRGN. If the history is of prolonged (3–4 weeks) duration or recurrent, other causes like IgA nephropathy, membranous or membranoproliferative disease are more likely. Presence of fever, joint pains, and photosensitive rash points toward a diagnosis of SLE. Most of these conditions can be identified on a kidney biopsy specimen. The specimen should include light microscopy, immunofluorescence, and electron microscopy (if available). Serological tests that help in identifying the underlying cause are hepatitis B surface antigen (HBsAg)

Flowchart 1: Diagnostic approach to gross hematuria.



(ANA: antinuclear antibody; ASLO: antistreptolysin O; C/S: culture and sensitivity; D/D: differential diagnosis; HBsAg: hepatitis B surface antigen; HCV: hepatitis C virus; IgA: immunoglobulin A; KFT: kidney function test; KUB: kidney, ureter, and bladder; MPGN: membranoproliferative glomerulonephritis; IRGN: infection-related glomerulonephritis; RBC: red blood cell; SLE: systemic lupus erythematosus; UTI: urinary tract infection; USG: ultrasonography)

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and anti-hepatitis C virus (anti-HCV) for related nephropathy (membranous and membranoproliferative glomerulonephritis). Antinuclear antibodies (ANA) and anti-double stranded deoxyribonucleic acid for lupus nephritis; antineutrophilic cytoplasmic antibodies [ANCA; p-ANCA (perinuclear pattern) and c-ANCA (cytoplasmic pattern); these are both immunofluorescence-based tests] for identification of vasculitis (microscopic polyangiitis, Wegener and Churg–Strauss disease) and anti-GBM antibodies for detection of Goodpasture syndrome. Identification of ANCA can also be done by enzyme-linked immunosorbent assay-based tests (antimyeloperoxidase and antiproteinase-3) which are simpler and yield faster results. A formal ophthalmic examination and audiometry to detect high frequency of sensorineural hearing loss is imperative in Alport syndrome.

Presence of extraglomerular hematuria points toward local causes, polycystic kidneys, hydronephrosis, urolithiasis, and hypercalciuria. Vascular abnormalities of urinary bladder can also cause hematuria rarely. An ultrasound kidney, ureter, and bladder (KUB) would identify any structural abnormalities of kidney and/or stones in kidney. Ureteric stones are usually not identified with ultrasound and require a radiograph of KUB region. If calculi are identified, the passed stone can be analyzed using Fourier transform infrared spectroscopy and a complete urinary biochemistry needs to be done (24-h urinary excretion of creatinine, calcium, oxalates, sodium, phosphorus, urates, and cystine). If there is a difficulty in collecting 24-hours sample, spot urine sample can be used for determining urinary calcium/creatinine ratio and oxalate/creatinine ratio. Hypercalciuria is defined as urine calcium to creatinine ratio >0.2 (under 6 months of age: >0.8; 6–12 months: >0.6) or a 24-hours calcium excretion exceeding 4 mg/kg/day. Doppler USG is useful in suspected cases of renal vein thrombosis. High resolution computed tomography may be required to locate calculi in children with hypercalciuria or positive family history of renal calculi. Few patients require whole exome study, if there is syndromic morphology or family history of consanguinity with more than one affected children or for confirmation of familial disorders like *MYH9* nephropathy and Alport disease.

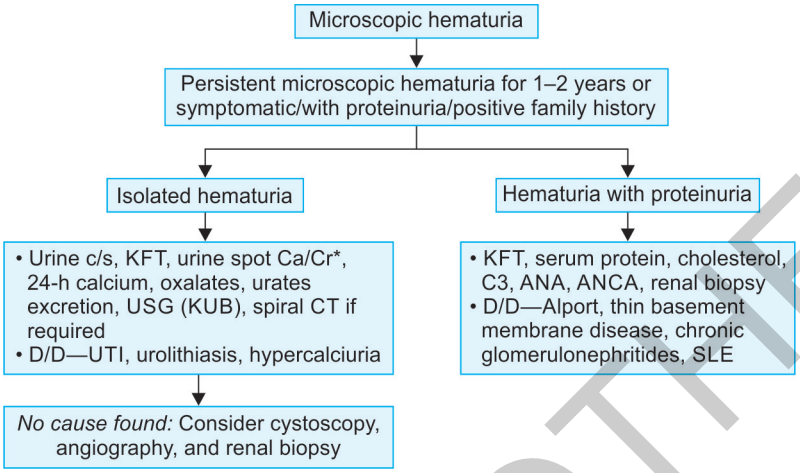
Referral to a pediatric surgeon/urologist will be required when clinical evaluation and work up indicate a structural urogenital abnormality, an obstructing calculus, or a tumor. The most common malignant renal tumor in childhood is Wilms' tumor and is associated with a palpable abdominal mass and hematuria in most patients. Other primary urinary tract tumors, e.g., hemangiomas, rhabdomyosarcomas, and lymphomas, resulting in hematuria are rare. Cystoscopy to identify the source of bleeding is best performed during active bleeding; a biopsy may be required sometimes.

INDICATIONS FOR KIDNEY BIOPSY

Proteinuria along with glomerular hematuria persisting for >4 weeks after the onset of IRGN is an indication for kidney biopsy. Also, decreased complement levels beyond 3 months, suspected systemic disorder and recurrent gross hematuria indicate a need for kidney biopsy to rule out other causes like IgA nephropathy, membranous or membranoproliferative glomerulonephritis nephropathy. Patients with deranged kidney functions (urea and creatinine), and/or presence of proteinuria also need a kidney biopsy to detect the presence of chronic glomerulonephritis. Patients with rapid worsening of kidney functions presenting rapidly progressive glomerulonephritis should have an early biopsy to detect crescentic glomerulonephritis. Presence of ANA, HBsAg, anti-HCV, or ANCA positivity is also an indication for kidney biopsy.

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Flowchart 2: Diagnostic approach to microscopic hematuria.



*Urine spot calcium/creatinine ratio (>0.2 mg/mg abnormal).
(ANA: antinuclear antibody; ANCA: antineutrophilic cytoplasmic antibody; Ca/Cr: calcium-creatinine ratio; CT: computed tomography; D/D: differential diagnosis; HCV: hepatitis C virus; KFT: kidney function test; KUB: kidney, ureter, and bladder; SLE: systemic lupus erythematosus; USG: ultrasonography; UTI: urinary tract infection)

MICROSCOPIC HEMATURIA

Isolated microscopic hematuria is benign on most occasions, like after vigorous exercise and fever. Transient microscopic hematuria can occur with UTIs, fever, trauma, and heavy exercise in children. However, it needs to be evaluated if it is associated with edema, hypertension, rash, fever, joint pains, or deranged KFT. Also, persistent microscopic hematuria (of >1–2 years of duration) needs an evaluation. The algorithm for evaluation is given in **Flowchart 2**.

If the child has persistent hematuria over 2–3 weeks, then urine culture should be done, particularly in the younger child where symptoms of a UTI may not be apparent. Renal ultrasonography is noninvasive and provides information on structural anomalies and urolithiasis. Though on most occasions, sonography is normal in children with asymptomatic microscopic hematuria, it helps in providing reassurance to the parents. A family history of hematuria or hearing loss should raise the suspicion for Alport disease. Serology can be done for gross hematuria, and finally, a kidney biopsy may give the cause of underlying hematuria.

Treatment of each condition would be different. We have discussed the management of IRGN and IgAN briefly. However, the management of other conditions, such as chronic glomerulonephritis, hypercalciuria, and urolithiasis, will be dealt in the relevant sections. On most occasions, these patients would require a consultation with pediatric nephrologists.

CONCLUSION

Hematuria is an uncommon condition in childhood. An episode of gross hematuria or symptomatic microscopic hematuria should always be evaluated. Asymptomatic microscopic hematuria needs evaluation only if it persists on repeated microscopic examination of urine (beyond 1 year) or is associated with proteinuria. A step-wise approach should be followed to identify the etiology. Treatment of the cause results in resolution of hematuria on most occasions.

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This is an official publication of the Indian Academy of Pediatrics (IAP). Several luminaries in the field of Pediatric Nephrology, from home and abroad, have contributed their might in bringing out this book. The book has been designed with a practical approach to renal problems in children, faced by treating clinicians in their routine pediatric practice. The book has been kept concise with simple language, making it more reader friendly.

The book has 30 chapters, dealing with basic renal functions, common renal conditions in day-to-day pediatric practice, and some of the more complex renal problems in children. Drug dosing guidelines for commonly used drugs in renal failure and a few useful appendices are also incorporated.

The two-color format will be pleasing to the eye and will facilitate easy reading by highlighting boxes, tables, and key messages.

The book is ideally suited to the needs of pediatricians, pediatric nephrologists, postgraduate students, nephrologists, and urologists interested in pediatric aspects of the discipline.

Queries and suggestions may be sent to Dr Anand S Vasudev at *asvasudev@gmail.com* and *drabhijeetsaha@gmail.com*.

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