

# Haematuria in children

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## Abstract

Haematuria is a common finding in children and can be macroscopic or microscopic. In contrast to adults, haematuria in children very rarely indicates an underlying malignant pathology. The differential diagnosis is broad, with the most common underlying causes being infection, glomerulonephritis and hypercalciuria. It is useful to distinguish between nephrological or upper urinary tract and lower urinary tract pathologies, as this will guide investigations and referral. This review discusses the causes of haematuria in the paediatric population.

**Key words:** Haematuria, Paediatric

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## Introduction

Haematuria is a common finding in children and can be macroscopic or microscopic. In contrast to adults, haematuria in children very rarely indicates an underlying malignant pathology. Studies indicate that 1% of school children will have a positive urine dipstick, but this will only persist in one third of the positive cases (Vehaskari et al, 1979). The differential diagnosis is broad, with the most common underlying causes being infection, glomerulonephritis and hypercalciuria. It is important to remember that there are a number of conditions which can mimic haematuria in children, including rhabdomyolysis and porphyria. Investigations performed should reflect pertinent findings in the history and examination. Basic investigations include blood pressure recording, urine culture and microscopy, ultrasound and blood tests including full blood count, urea, creatinine and electrolytes.

It is useful to distinguish between nephrological or upper urinary tract and lower urinary tract pathologies, as this will guide investigations and referral. Glomerular bleeding creates brown 'coca-cola' coloured haematuria. This is more likely to be painless and can be associated with proteinuria, hypertension or signs of fluid overload. Cellular casts and dysmorphic red cells on urine microscopy are also indicative of a glomerular source of bleeding. Bleeding from the lower urinary tract is usually bright red or burgundy coloured and may contain clots. This review discusses the causes of haematuria in the paediatric population, which are summarised in [Table 1](#).

## Upper urinary tract: haematuria resulting from glomerular abnormalities

### Glomerulonephritis

Glomerular inflammation can result from a primary renal condition or be secondary to a systemic disease, eg systemic lupus erythematosus. There is often an associated proteinuria. The spectrum of clinical presentations ranges from asymptomatic microscopic haematuria to sudden onset of gross haematuria with acute kidney injury, hypertension, oliguria and oedema; this is a nephrological emergency. The most common causes of glomerulonephritis in children are acute post-streptococcal, primary IgA nephropathy and Henoch–Schönlein purpura.

Post-streptococcal glomerulonephritis is the most common cause of glomerulonephritis in children, resulting from glomerular immune-complex deposition following a group A streptococcal infection, usually of the throat or skin (Rodriguez-Iturbe and Haas, 2016). Children classically present with acute nephritic syndrome, comprising a triad of haematuria, oedema and hypertension. There is often an associated acute kidney injury. Diagnosis is made following a classical history and evidence of a preceding streptococcal infection, either from a throat swab or serum antibodies. Treatment is supportive and focuses on

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**Table 1. Causes of haematuria in children**

Nephrological	Acute post-streptococcal glomerulonephritis
	Primary IgA nephropathy
	Henoch–Schönlein purpura
	Alport syndrome
	Thin basement membrane nephropathy
	Exercise
	Pyelonephritis
	Renal tumours
	Renal trauma
	Sickle cell disease
Urinary tract	Calculi (renal, ureteric, bladder)
	Hypercalciuria
	Cystitis including urinary tract infection
	Schistosomiasis
	Trauma
	Bladder tumours
	Posterior urethritis
Haematuria ‘mimics’	Rhabdomyolysis
	Medications including nitrofurantoin
	Ingestion of beetroot
	Porphyria

the management of oedema and hypertension. Management of oedema includes strict input and output monitoring, measurement of daily weights and fluid and salt restriction. Pharmacologically, loop diuretics are the agent of choice when managing mild to moderate hypertension and a course of penicillin-based antibiotic is administered to aid eradication of the streptococcus. Long-term prognosis is excellent as the disease usually follows a benign course, with less than 2% of children having persistent hypertension beyond 2 years (Shulman and Bisno, 2014).

IgA nephropathy, also known as Berger’s disease, results from deposition of IgA immunoglobulins in the glomerular mesangium. It is more common in teenagers but can be seen in younger cohorts. Classical presentation is with intermittent haematuria within 2–3 days of an upper respiratory tract infection. Rarely, acute nephritic syndrome may be present. A renal biopsy is required for diagnosis and will demonstrate IgA deposits. Early referral to a nephrologist is suggested as the clinical course and management of the disease is variable, with up to 25% of paediatric patients having progressive disease eventually leading to end-stage renal failure (Coppo, 2008). The Oxford classification of IgA nephropathy is often used to classify prognosis and identify those who would benefit from immunosuppression (Soares and Roberts, 2017).

Henoch–Schönlein purpura is a systemic, small vessel vasculitis which causes nephritis in up to 45% of cases (Watson et al, 2012). The condition is very similar to IgA nephropathy, but Henoch–Schönlein purpura is systemic, affecting organs other than the kidney including the skin, joints and gastrointestinal tract. Patients with Henoch–Schönlein purpura present with a purpuric rash, arthralgia and abdominal pain. Renal involvement varies from asymptomatic microscopic haematuria and proteinuria to a severe nephritis. Henoch–Schönlein purpura is self-limiting in the majority of cases but those with renal involvement are more likely to have

recurrent disease. Treatment is supportive and can include administration of corticosteroids. Around 1% of patients will progress to end-stage renal failure (Roberts et al, 2007).

### Alport syndrome

This hereditary nephritis results from a mutation in the genes that encode for type IV collagen, which is primarily located in the basement membrane of the kidney, eyes and cochlea (Watson et al, 2020). Approximately 80% of cases are inherited in an X-linked manner, and the syndrome comprises hearing loss, ectopia lentis and renal involvement. Diagnosis is via skin or renal biopsy and genetic testing may be used. There is no specific treatment, but efforts are made to limit the progression of renal disease, with renal transplant being offered to those with end-stage renal failure.

### Thin basement membrane nephropathy

This benign condition affects at least 1% of the population and presents with persistent, asymptomatic microscopic haematuria (Tryggvason and Patrakka, 2006). It is inherited in a dominant fashion, and therefore runs in families. Histologically, the condition will demonstrate uniformly thin basement membranes; however, biopsies are rarely performed as the condition does not result in progressive renal disease. If macroscopic haematuria, hypertension or proteinuria are present, other diagnoses must be considered. No treatment is required, although patients should be monitored for the appearance of proteinuria, hypertension or renal deterioration, in case of misdiagnosed Alport syndrome.

### Exercise

Exercise-induced haematuria can occur in children (particularly adolescents) as well as adults, with red blood cells appearing in the urine after active sports or vigorous activity at school. It is benign and disappears once the child is rested.

## Upper urinary tract: haematuria resulting from tubulo-interstitial abnormalities

### Pyelonephritis

Urinary tract infection is one of the most common causes of haematuria in children. Older children will present with classical symptoms of pyelonephritis – loin pain, fever and urinary symptoms. It is more difficult to diagnose in infants who often present with a non-specific febrile illness, so a urine sample should always be taken. Infants with a fever and bacteriuria should be presumed to have pyelonephritis and treated with intravenous antibiotics accordingly (National Institute for Health and Care Excellence, 2018). Children with bacteriuria but no systemic symptoms should be presumed to have a lower urinary tract infection or cystitis. Complications of pyelonephritis include renal scarring, hypertension and renal failure.

### Sickle cell disease or trait

Haematuria in sickle cell disease results from red cells sickling in the renal vasculature, leading to corticomedullary hypoperfusion with red cell extravasation (Nath and Hebbel, 2015). Mild haematuria is usually self-limiting and management consists of bedrest and hydration. Persistent haematuria in sickle cell disease is considered a prognostic indicator of progression to end-stage renal failure, which usually occurs in the second decade of life.

### Renal trauma

Renal trauma can result from a blunt or penetrating injury. The kidney is more vulnerable to injury in the paediatric patient because of several anatomical factors: scarce perinephric fat, less protection from the thoracic rib cage and a proportionally larger size of the kidney. Signs that may indicate renal injury include haematuria, flank bruising and fracture of the lower ribs. It is important to note that the degree of haematuria does not correlate with the severity of the injury. Renal injuries are graded according to the American Association for the Surgery of Traumas Organ Injury Scale (Table 2) (Moore et al, 1989). An estimated 80% of renal injury in children will be low grade (grade 1, 2 or 3) (Grimsby et al, 2014).

**Table 2. American Association of Surgery of trauma grading of renal injury**

Grade I	Contusion with haematuria, normal urological studies Non-expanding subcapsular haematoma, no parenchymal laceration
Grade II	Non-expanding perirenal haematoma contained within the retroperitoneum Laceration of <1 cm of renal cortex parenchyma without urinary extravasation
Grade III	Laceration of >1 cm depth of renal cortex parenchyma without urinary extravasation or collecting system rupture
Grade IV	Laceration extending through the renal cortex parenchyma, medulla and collecting system Main renal artery or vein injury with controlled haemorrhage
Grade V	Shattered kidney or hilar avulsion which removes blood supply to kidney

From Kozar et al (2018)

Investigation of renal trauma tends to involve computed tomography scanning with intravenous contrast. In stable children with haematuria, a history of mild trauma and a low suspicion of renal injury, an ultrasound may be appropriate. Management is usually conservative, even for grade IV and V injuries (Fernandez-Ibieta, 2018). All grade I–III injuries can be managed non-operatively with bed rest and urethral catheterisation. Surgical exploration will be required in those with haemodynamic instability or deep penetrating injuries. Interventional radiology can be used for selective artery embolisation in cases of bleeding when the patient is stable.

Haematuria following a history of minor trauma may be a result of underlying renal pathology (eg hydronephrosis or tumour). Vigilance for non-accidental injury should be maintained.

### Renal tumours

Wilms tumour is the most common paediatric renal tumour, with an incidence of 10 per million. It usually presents with an abdominal mass and 18% of cases will have frank haematuria. Ultrasound is the first line investigation, followed by cross-sectional imaging. Treatment is with nephrectomy and chemotherapy, be it adjuvant or neoadjuvant (Aldrink et al, 2019).

Rhabdoid renal tumours are aggressive, malignant tumours, and represent 2% of renal malignancies in children. Initial presentation may be as a result of metastatic disease, but they can also present with haematuria (in 60% of cases) or an abdominal mass. Prognosis is poor, with an 18-month survival rate of only 20% (Lowe et al, 2000).

### Nutcracker syndrome

This rare syndrome is caused by a vascular anatomical variation, whereby the left renal vein is compressed between the aorta and superior mesenteric artery. Symptoms include flank pain and chronic fatigue. The precise mechanism of haematuria from this cause remains unclear. It has been postulated that compression of the left renal vein results in the development of collaterals with intrarenal varicosities, causing haematuria when the thin walled septum between varicose vessels and the renal collecting system ruptures (Hanna et al, 1997).

## Haematuria resulting from abnormalities of the lower urinary tract

### Calculi

Urinary tract calculi are far less common in children than in adults. A metabolic cause is more common in children than adults, accounting for a third of cases. Hypercalciuria is the most common, followed by hypocitraturia, hyperoxaluria and cystinuria. Infective calculi account for 20% of cases, caused by urease producing bacteria such as *Klebsiella* spp. or *Pseudomonas* spp. Metabolic investigations should include blood levels of calcium, bicarbonate and parathyroid hormone as well as urea, creatinine and electrolytes. Spot and 24-hour urine collection should be sent for measurement of calcium/creatinine, oxalate/

creatinine, urate/creatinine and citrate/creatinine ratios and levels of amino acids. Stones removed or passed should be analysed.

The investigation of choice is ultrasonography. This will identify the majority of calculi, in addition to identifying underlying congenital renal tract abnormalities. Second-line investigation is non-contrast computed tomography.

Treatment depends on the site and size of the stone and on the local availability of treatments. Renal calculi may be treated with external shock wave lithotripsy where available, although this requires a general anaesthetic. Success rates of up to 90% have been reported, but this depends on stone size, site and type (Choong et al, 2000). Percutaneous nephrolithotomy is effective for removing large calyceal stones. Ureteroscopy can be used in children, as in adults, to retrieve ureteric stones.

Dietary changes are important to prevent recurrence, including a fluid intake of 2 litres per day and a reduced salt diet. Medical management includes potassium citrate for hypercalciuria, hypocitraturia and cystinuria.

Children with bladder augmentations are at risk of developing bladder stones from the mucus produced by the bowel segment used in the augmentation. The method of prevention is regular bladder washouts and yearly ultrasound screening. Bladder stones can be removed with minimally invasive percutaneous cystolithotomy.

### Hypercalciuria

Hypercalciuria can lead to asymptomatic microscopic haematuria in the absence of any calculi formation. It can be a precursor of urolithiasis and should be suspected when there is a family history of urolithiasis. It is detected on a 24-hour urinary calcium excretion test. Management is with increased fluid intake and reduced salt diet. Rarely, thiazide diuretics are used to control serum calcium levels (Stapleton et al, 1984).

### Cystitis

Lower urinary tract infections are one of the most common causes of haematuria in children. Pre-verbal children and infants with lower urinary tract infections are more likely to present with a non-specific febrile illness with feed refusal, irritability, vomiting and lethargy. Verbal children can present with dysuria, frequency, urgency and a deterioration in continence. The work-up of febrile illness in children should always include urinalysis. The presence of nitrites and leucocytes on a dipstick is indicative of infection and the urine should be sent for microscopy and culture. Antibiotic treatment should be in accordance with local microbiology guidelines.

Investigation and management are according to National Institute for Health and Care Excellence guidelines, which are outlined in [Table 3](#). A urinary tract infection is defined as ‘recurrent’ if there are three or more episodes of lower urinary tract infection, or two or more episodes of upper urinary tract infection. It is defined as ‘atypical’ if there are indicators of urinary flow obstruction – for example poor urine flow, raised creatinine, septicaemia, non-*Escherichia coli* urinary tract infection or an abdominal mass (National Institute for Health and Care Excellence, 2018). Advice regarding urinary tract infection prevention should be given to parents, ensuring adequate fluid intake, regular voiding, treatment of any associated constipation and perineal hygiene advice.

Viral haemorrhagic cystitis particularly affects immunocompromised children, including adenovirus, cytomegalovirus, Epstein–Barr virus and BK virus. A respiratory viral screen should be performed and the haematuria is usually self-limiting (Allen and Alexander, 2005).

### Schistosomiasis

Schistosomiasis of the urinary tract is a result of infection by the *Schistosoma haematobium* trematode, a type of flatworm. Humans are at risk of infection when swimming or wading in freshwater in endemic areas, such as sub-Saharan Africa. Symptoms are caused by a strong immune reaction to the eggs, with granuloma formation along the entire urinary tract. Symptoms include terminal haematuria, dysuria and frequency. Urine microscopy showing parasitic eggs is diagnostic and serum antibodies/antigens are also indicative of infection. Praziquantel is the usual course of treatment. Chronic infection is associated with an increased risk of squamous cell metaplasia and bladder cancer (Osakunor et al, 2018).

**Table 3. Investigation of urinary tract infections**

	Typical urinary tract infection	Atypical urinary tract infection	Recurrent urinary tract infection
<6 months	Ultrasound 6 weeks following infection	Ultrasound during infection	Ultrasound during infection
		Micturating cystourethrogram	Micturating cystourethrogram
		DMSA scan	DMSA scan
6 months–3 years	None	Ultrasound during infection	Ultrasound at 6 weeks
		DMSA scan	DMSA scan
>3 years	None	Ultrasound during infection	Ultrasound at 6 weeks
			DMSA scan or indirect MAG3

DMSA = dimercapto succinic acid, MAG3 = mercaptoacetyltriglycine

### Urinary tract vascular malformations

Isolated genitourinary vascular malformations are extremely rare, but they are seen in up to 30% of patients with Klippel–Trenaunay syndrome, which is a rare congenital condition comprising complex venous and lymphatic malformations with hypertrophy of bony and soft tissues, usually affecting one limb. Bladder lesions are most commonly found in the dome and anterior wall of the bladder and present with painless haematuria. Magnetic resonance or computed tomography angiography is the investigation of choice and treatment is usually with embolisation or sclerotherapy (Rubenwolf et al, 2006).

### Trauma

The bladder is anatomically more susceptible to injury in children, as the pelvic bones are less developed and the bladder is an intra-abdominal organ. Blood at the urinary meatus, especially in the presence of pelvic fractures, provides an alert for a potential bladder injury. Diagnosis is done with a triphasic computed tomography with intravenous contrast. Extravasated contrast will be visualised in the pelvis if the bladder has ruptured and distribution will indicate if this is intraperitoneal or extraperitoneal. Intraperitoneal rupture requires urgent surgical repair. Extraperitoneal rupture can be treated conservatively with a urethral catheter on free drainage for 10 days, followed by a cystogram to confirm the absence of ongoing contrast leak (Gomez et al, 2004).

Anterior urethral injuries are usually secondary to straddle injuries, such as when a child forcefully straddles a bicycle. Posterior urethral injuries are associated with high velocity accidents resulting in associated pelvic fractures. Urethral injury should be suspected with the presence of blood at the urinary meatus, perineal bruising or scrotal haematoma. Diagnosis is via retrograde urethrography and immediate treatment involves placement of a suprapubic catheter. In complete anterior urethral disruption and in posterior urethral injuries in children with multiple injuries, surgical repair should be deferred for 3 months as repair in the acute setting is associated with higher rates of impotence and incontinence (Da Silveria Ugino et al, 2017). In contrast, primary repair should be attempted where possible in females to prevent urethrovaginal fistula formation. Long-term consequences of untreated or unrecognised urethral injuries include stricture or diverticula formation, fistula, incontinence and impotence.

### Bladder tumours

Bladder malignancy is extremely rare in children. The most common tumour affecting the lower genitourinary tract in the paediatric population is rhabdomyosarcoma, a soft tissue sarcoma of primitive muscle cells (Abouheba and Shehata, 2019). Genitourinary rhabdomyosarcoma can arise in the prostate, bladder, vagina or paratesticular regions. Bladder rhabdomyosarcoma can present with haematuria, dysuria, urinary tract infection, urinary retention or an abdominal mass. A pelvic mass will be seen on ultrasound scan and is further characterised with cross-sectional imaging and a biopsy. Treatment involves a combination of chemotherapy, surgery and radiotherapy.

## Key points

- A thorough history and examination is essential in all children with haematuria, with an aim of establishing the precise location of the bleeding – either the renal or upper urinary tract or the lower urinary tract.
- Hypertension, proteinuria and fluid overload in a patient with haematuria indicates a renal cause for the bleeding.
- Haematuria following recent viral infection can represent post-streptococcal glomerulonephritis, IgA nephropathy or adenovirus haemorrhagic cystitis.

## Posterior urethritis

Posterior urethritis is an inflammation of the bulbar urethra in prepubertal boys, which classically presents as terminal haematuria or blood spotting in the underwear. The mean age at presentation is 10 years old. The aetiology remains unknown, although postulated theories include viral infection, hormonal imbalance and dysfunctional elimination syndrome. Radiological and laboratory investigations will be normal, except for microscopic haematuria in around half of cases (Walker et al, 2001). Cystoscopy is only required in those with atypical presentations and may demonstrate inflammation or hyperaemia of the bulbar urethra. Posterior urethritis usually resolves without treatment within 6 months. A small subset of patients will develop urethral strictures. Therefore, it is prudent to perform baseline uroflowmetry and if abnormal or worsening at follow up, or if symptoms are prolonged beyond 2 years, to proceed to cystoscopy (Poch et al, 2007).

## Conclusions

The differential diagnosis of haematuria in children is broad, so the importance of thorough history-taking and examination should be highlighted. The majority of patients with microscopic haematuria have a benign cause that will resolve without further treatment. Red-coloured urine does not always indicate the presence of blood and can be indicative of myoglobinuria or be secondary to medications, and any blood in the urine may be a result of vaginal or anal bleeding.

Patients presenting with dark brown haematuria associated with proteinuria, hypertension and oedema are more likely to have a renal glomerular or tubulo-interstitial abnormality. Common causes include post-streptococcal glomerulonephritis and Henoch-Schönlein purpura. Bleeding from the lower urinary tract is typically bright red and can be associated with urinary symptoms including urgency, frequency and dysuria. This is most commonly as a result of urinary tract infection or hypercalciuria.

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### Conflicts of interest

The authors declare that they have no conflicts of interest.

## References

- Abouheba M, Shehata S. Endoscopic management of bladder tumors in children. *ESPE Manual Pediatric Minimally Invasive Surgery*. Springer, Cham; 2019:473–479
- Aldrink JH, Heaton TE, Dasgupta R et al. American pediatric surgical association cancer committee update on wilms tumor. *J Pediatric Surg*. 2019;54(3):390–397. <https://doi.org/10.1016/j.jpedsurg.2018.09.005>
- Allen CW, Alexander SI. Adenovirus associated haematuria. *Arch Dis Child*. 2005;90(3):305–306. <https://doi.org/10.1136/adc.2003.037952>
- Choong S, Whitfield H, Duffy P et al. The management of paediatric urolithiasis. *BJU Int*. 2000;86(7):857–860. <https://doi.org/10.1046/j.1464-410x.2000.00909.x>

- Coppo R. Pediatric IgA nephropathy: clinical and therapeutic perspectives. *Semin Nephrol.* 2008;28(1):18–26. <https://doi.org/10.1016/j.semnephrol.2007.10.003>
- Da Silveira Ugino RT, Pasqual S, Farias AK et al. Management of traumatic urethral injuries in children using different techniques: a case series and review of literature. *Int J Surg Case Rep.* 2017;40:85–89. <https://doi.org/10.1016/j.ijscr.2017.08.062>
- Fernandez-Ibieta M. Renal trauma in pediatrics: a current review. *Urology.* 2018;113:171–178. <https://doi.org/10.1016/j.urology.2017.09.030>
- Gomez RG, Ceballos L, Coburn M et al. Consensus statement on bladder injuries. *BJU Int.* 2004;94(1):27–32. <https://doi.org/10.1111/j.1464-410X.2004.04896.x>
- Grimsby GM, Voelzke B, Hotaling J et al. Demographics of pediatric renal trauma. *J Urol.* 2014;192(5):1498–1502. <https://doi.org/10.1016/j.juro.2014.05.103>
- Hanna HE, Santella RN, Zawada ET Jr, Masterson TE. Nutcracker syndrome: an underdiagnosed cause for hematuria. *S D J Med.* 1997;50(12):429–436
- Kozar RA, Crandall M, Shanmuganathan K et al; AAST Patient Assessment Committee. Organ injury scaling 2018 update: Spleen, liver, and kidney. *J Trauma Acute Care Surg.* 2018;85(6):1119–1122. <https://doi.org/10.1097/TA.0000000000002058>
- Lowe LH, Isuani BH, Heller RM et al. Pediatric renal masses: wilms tumor and beyond. *Radiographics.* 2000;20(6):1585–1603. <https://doi.org/10.1148/radiographics.20.6.g00nv051585>
- Moore EE, Shackford SR, Pachter HL et al. Organ injury scaling: spleen, liver, and kidney. *J Trauma.* 1989;29(12):1664–1666. <https://doi.org/10.1097/00005373-198912000-00013>
- Nath KA, Hebbel RP. Sickle cell disease: renal manifestations and mechanisms. *Nat Rev Nephrol.* 2015;11(3):161–171. <https://doi.org/10.1038/nrneph.2015.8>
- National Institute for Health and Care Excellence. Urinary tract infection in under 16s: diagnosis and management. Clinical guideline CG54. 2018. <https://www.nice.org.uk/guidance/cg54> (accessed 1 March 2021)
- Osakunor DN, Woolhouse ME, Mutapi F. Paediatric schistosomiasis: what we know and what we need to know. *PLoS Negl Trop Dis.* 2018;12(2):e0006144. <https://doi.org/10.1371/journal.pntd.0006144>
- Poch MA, Handel LN, Kaplon DM et al. The association of urethrorrhagia and urethral stricture disease. *J Pediatr Urol.* 2007;3(3):218–222. <https://doi.org/10.1016/j.jpuro.2006.07.007>
- Roberts PF, Waller TA, Brinker TM et al. Henoch-Schonlein purpura: a review article. *South Med J.* 2007;100(8):821–824. <https://doi.org/10.1097/SMJ.0b013e3180f62d0f>. PMID: 17713309.
- Rodriguez-Iturbe B, Haas M. Post-streptococcal glomerulonephritis. In: Ferretti JJ, Stevens DL, Fischetti VA (eds). *Streptococcus pyogenes: Basic Biology to Clinical Manifestations.* Oklahoma City (OK): University of Oklahoma Health Sciences Center; 2016
- Rubewolf P, Roosen A, Gerharz EW et al. Life-threatening gross hematuria due to genitourinary manifestation of Klippel–Trenaunay syndrome. *Int Urol Nephrol.* 2006;38(1):137–140. <https://doi.org/10.1007/s11255-005-3154-0>
- Shulman ST, Bisno AL. Nonsuppurative poststreptococcal sequelae: Rheumatic fever and glomerulonephritis. In: Bennett J, Dolin R, Blaser M (eds). *Mandell, Douglas, and Bennett's principles and practice of infectious diseases.* 8th edn. 2014. Elsevier; 2300–2309
- Soares MF, Roberts IS. IgA nephropathy: an update. *Curr Opin Nephrol Hypertens.* 2017;26(3):165–171. <https://doi.org/10.1097/MNH.0000000000000312>
- Stapleton FB, Roy S, III Noe HN, Jerkins G. Hypercalciuria in children with hematuria. *N Engl J Med.* 1984;310(21):1345–1348. <https://doi.org/10.1056/NEJM198405243102102>
- Tryggvason K, Patrakka J. Thin basement membrane nephropathy. *J Am Soc Nephrol.* 2006;17(3):813–822. <https://doi.org/10.1681/ASN.2005070737>
- Vehaskari VM, Rapola J, Koskimies O et al. Microscopic hematuria in school children: epidemiology and clinicopathologic evaluation. *J Pediatr.* 1979;95(5):676–684. [https://doi.org/10.1016/S0022-3476\(79\)80710-6](https://doi.org/10.1016/S0022-3476(79)80710-6)
- Walker BR, Ellison ED, Snow BW, Cartwright PC. The natural history of idiopathic urethrorrhagia in boys. *J Urol.* 2001;166(1):231–232. [https://doi.org/10.1016/S0022-5347\(05\)66132-0](https://doi.org/10.1016/S0022-5347(05)66132-0)
- Watson L, Richardson AR, Holt RC, Jones CA, Beresford MW. Henoch schonlein purpura—a 5-year review and proposed pathway. *PLoS One.* 2012;7(1):e29512. <https://doi.org/10.1371/journal.pone.0029512>
- Watson S, Padala SA, Bush JS. Alport syndrome. 2020. <https://www.ncbi.nlm.nih.gov/books/NBK470419/> (accessed 26 April 2021)