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Clinico-etiological profile of haematuria in children: A retrospective analysis

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ABSTRACT

Objectives: The objectives of this study were to study demographic, etiological and clinical profiles of children presenting with haematuria.

Material and Methods: This was a retrospective hospital-based study conducted by referring medical records of patients (aged 1 month–16 years) admitted to Yenepoya Medical College, Mangalore, who were treated for haematuria between January 2018 and December 2022.

Results: Of the 84 children (40 males, mean age 7.3 ± 4.94 years) who presented with haematuria, 48 were microscopic, 36 were with gross haematuria, and 56 were with non-glomerular, 28 were with glomerular haematuria. Common causes included urinary tract infections (47%), post-infectious glomerulonephritis (22.6%), renal calculi and hydronephrosis. Deranged renal function (17.6%) and dyselectrolytemia (53.5%) were frequent complications. Anaemia (61.9%) was the most prevalent associated comorbidity. Acute renal failure occurred in 7.14% and mortality in 2.4%. Glomerular haematuria was associated with hypertension, oliguria and oedema.

Conclusion: This study highlights the diverse aetiologies of haematuria in children, emphasising the need for a comprehensive evaluation and prompt management to prevent long-term renal damage. The findings inform clinical practice and guide further research into paediatric haematuria. The distinction between glomerular and non-glomerular aetiology will help in the proper investigation of the child and further aid in effective management.

Keywords: Haematuria, Glomerulonephritis, Urinary tract infection, Glomerular

INTRODUCTION

Haematuria is described as the continuous presence of more than five red blood cells (RBCs)/ high-power field (HPF) in urine that have not been centrifuged.[1] Although underlying disease may be indicated by 10–50 RBCs/μL, severe haematuria is typically defined as >50 RBCs/HPF.[1] Persistent microscopic haematuria occurs in <1% of children. Gross haematuria (visible discoloration noted in urine due to RBCs) occurs much less often (incidence <0.1%).[2]

According to whether the haematuria is glomerular or non-glomerular in origin, the causes of gross haematuria are divided into different categories.[3] RBCs that are dysmorphic or eumorphic in glomerular and non-glomerular haematuria, respectively, can be identified by microscopic analysis of the urine.[4]

Gross haematuria is more frequently seen as a presenting symptom of acute post-streptococcal glomerulonephritis, immunoglobulin A (IgA) nephropathy, and Alport syndrome, even though

glomerular disorders are almost invariably linked to some degree of haematuria.[5]

Idiopathic hypercalciuria, bacterial or viral urinary tract infections (UTIs), urolithiasis, urinary tract structural abnormalities and sickle cell trait are the most common causes of non-glomerular haematuria.[6]

When evaluating a child with haematuria, it is important to rule out any major underlying renal illness that may have a dire prognosis.[7]

Gross haematuria usually prompts emergency care, but little is known about the long-term effects, clinical features, aetiology and associated symptoms in children from this geographical area. This retrospective review was performed with the aim to study the demographic, etiological and clinical profile of children presenting with haematuria, with the following objectives: To elucidate the underlying aetiology of microscopic and macroscopic haematuria in children and to characterise clinical and laboratory profile of children presenting with macroscopic and microscopic haematuria and its outcome.

MATERIAL AND METHODS

This retrospective study was conducted in Yenepoya Medical College hospital by retrieving case records of patients (aged 1 month to 16 years) presented with haematuria (from 01 January 2018 to 31 December 2022). Ethical clearance was obtained from the Institutional Ethics Committee before the study.

Case files were obtained from the medical records department, using the international classification of diseases (ICD) codes of 'haematuria' and all probable causes of haematuria, which includes post-infectious glomerulonephritis, IgA nephropathy (Berger disease), thin glomerular basement membrane disease, membranoproliferative glomerulonephritis, membranous nephropathy, Alport syndrome, focal segmental glomerulosclerosis, antiglomerular basement membrane disease, Henoch-Schönlein purpura nephritis, systemic lupus erythematosus (SLE) nephritis, haemolytic uremic syndrome, Wegener granulomatosis, polyarteritis nodosa., Goodpasture syndrome, sickle cell glomerulopathy, pyelonephritis, interstitial nephritis, acute tubular necrosis, hydronephrosis, papillary necrosis, nephrocalcinosis, polycystic kidney disease, tumours (Wilms, angiomyolipoma and rhabdomyosarcoma), urolithiasis/hypercalciuria and coagulopathy using respective ICD 10 codes.

Demographic details, clinical presentation and relevant laboratory/radiological investigation, which contribute to making a diagnosis, were noted down.

The outcome of the patient, including mortality and morbidity associated with disease (such as persistent hypertension/deranged renal function and persistent urinary abnormalities) at the time of discharge, was noted down.

Reports of renal biopsy or any other invasive procedures were also noted.

Glomerular haematuria was defined as microscopic urine examination with >5 RBC/HPF and >20% dysmorphic RBC, combined with moderate proteinuria (dipstick 2+) with or without casts.[8] Based on information from the history, clinical examination and urinalysis, the condition was finally classified as either glomerular or non-glomerular haematuria.[8]

Statistical analyses were conducted using the Statistical Package for the Social Sciences 27.0 program. The Fisher exact/Mann–Whitney U-test was used to compare continuous variables, while the Chi-square test was used to analyse categorical variables. $P < 0.05$ was regarded as significant.

RESULTS

Total of 84 children, consisting of 40 boys, with average age of 7.3 ± 4.94 years, (ranging from 3 months to 16 years) were assessed for microscopic and macroscopic haematuria.

The most common presenting complaints were fever, which occurred in 60.7% (n = 51) of patients, dysuria, affected 33.3% (n = 28) of patients, oedema, present in 34.5% (n = 29) of patients, lower abdominal pain, experienced by 27.4% (n = 23) of patients, reduced urine output, reported by 21.4% (n = 18) of patients, excessive cry/irritability, observed in 11.9% (n = 10) of patients and hypertension, affecting 28.5% (n = 24) of patients.

The symptoms appeared 1 day–2 years before the cause of haematuria was determined. Fourteen cases presented with haematuria were known cases of renal illnesses such as nephrotic/nephritic syndrome, end-stage renal disease, chronic kidney disease, mesangioproliferative glomerulonephritis and focal segmental glomerulosclerosis. There were five known cases of SLE and one case of Wiskott–Aldrich syndrome; gross haematuria was observed in 42.4% (n = 36) of patients, whereas microscopic haematuria was seen in 57.6% (n = 49). Glomerular haematuria accounted for 32.9% (n = 28) of cases, while non-glomerular haematuria was seen in 67.1% (n = 57) of cases.

In children with non-glomerular haematuria, UTI (40, 47%), hydronephrosis (5, 5.9%) and renal calculi (4; 4.7%) constituted the most common causes, whereas post-infectious glomerulonephritis (19, 22.6%), lupus nephritis and membranoproliferative glomerulonephritis constituted most common causes among glomerular haematuria [Tables 1 and 2].

UTI nephrotic syndrome constituted the most common cause among microscopic haematuria, whereas post-infectious glomerulonephritis, hydronephrosis and UTI constituted the most common causes among macroscopic haematuria [Tables 3 and 4]. A few cases of acute glomerulonephritis also presented with microscopic haematuria.

Anaemia was present in 61.9% (n = 52) of patients. Severe anaemia necessitating a packed-cell transfusion was observed in 10.6% (n = 9) of patients. Moderate anaemia was reported in 28.2% (n = 24) of patients, while mild anaemia was seen in 22.4% (n = 19) of patients – predominantly due to nutritional causes.

Deranged renal function tests were observed in 17.6% (n = 15) of patients; five of them had chronic kidney disease (three children were on maintenance haemodialysis), rest with acute kidney injury. Six children, representing 7.14% of the study population, developed acute renal failure requiring dialysis. Rapidly progressive glomerulonephritis was observed in two children who had glomerular haematuria.

Dyselectrolytemia was noted in 53.5% (n = 45) of patients. Thirty patients (35.7%) had hyponatremia (Na <134), and one patient with hypernatremia (Na-152). Hypokalaemia (K <3.4) was observed in 4 children (4.8%) and 2 children (2.4%) had hyperkalaemia (K >5.5). Hypercalcemia (Ca >10.5) and hypocalcemia (Ca <8.5) were observed in 15 children (17.8%) and 2 children (2.4%), respectively. Similarly, 4 children (4.8%) had hypophosphatemia (P < 3.5) and 8 children (9.5%) had hyperphosphatemia (P > 6).

Fourteen children (16.6%) had raised antistreptolysin O (ASO) antibody titres, antinuclear antibody (ANA) immunofluorescence (IF) was positive for eight children (9.5%) and 21 children (25%) had low C3 levels. Mean (SD) C3 levels noted in glomerular haematuria were 40.5 (37.3) mg/dL.

Renal biopsy was performed in three children, revealing membranoproliferative glomerulonephritis, renal cortical necrosis and tubulointerstitial nephritis.

Mortality was observed in 2 (2.4%) cases, died due to SLE lupus nephritis with multi-organ dysfunction and haemolytic uremic syndrome with uremic encephalopathy.

Thirty-four cases (40.4%) had persistent abnormal urine analysis at the time of discharge, two cases were discharged with persisting hypertension and 12 cases (14.3%) with deranged renal function tests.

Table 1: Diagnosis in children with haematuria (glomerular vs. non-glomerular).

Glomerular origin	
Diagnosis	**No (%)**
Post-infectious glomerulonephritis	12 (14.2)
Acute glomerulonephritis	7 (8.3)
Nephrotic syndrome	5 (5.9)
Membranoproliferative glomerulonephritis	3 (3.5)
Henoch-Schonlein purpura	1 (1.2)
Lupus nephritis	5 (5.9)
IgA nephropathy	1 (1.2)
Haemolytic uremic syndrome	3 (3.5)

IgA: Immunoglobulin A

Table 2: Diagnosis in children with haematuria (glomerular vs. non-glomerular).

Non-glomerular origin	
Diagnosis	**No (%)**
Urinary tract infection	40 (47)
Renal calculi	4 (4.7)
Hydronephrosis	5 (5.9)
Benign haematuria	1 (1.2)
Haemolytic anaemia	3 (3.5)
PUJ obstruction	1 (1.2)

PUJ: Pelvi-ureteric junction

Table 3: Diagnosis in children with haematuria (microscopic vs. macroscopic).

Microscopic haematuria	
Diagnosis	**No (%)**
Urinary tract infection	33 (39.2)
Post-infectious glomerulonephritis	5 (5.9)
Hydronephrosis	1 (1.2)
Haemolytic anaemia	2 (2.4)
Nephrotic syndrome	4 (4.7)
Renal calculi	1 (1.2)
Henoch-Schonlein purpura	1 (1.2)
Lupus nephritis	3 (3.5)
IgA nephropathy	1 (1.2)
PUJ obstruction	1 (1.2)

IgA: Immunoglobulin A, PUJ: Pelvi-ureteric junction

Table 4: Diagnosis in children with haematuria (microscopic vs. macroscopic).

Macroscopic haematuria	
Diagnosis	**No (%)**
Urinary tract infection	7 (8.3)
Post-infectious glomerulonephritis	14 (16.6)
Haemolytic uremic syndrome	3 (3.5)
Renal calculi	3 (3.5)
Hydronephrosis	4 (4.7)
Benign haematuria	1 (1.2)
Haemolytic anaemia	1 (1.2)
Nephrotic syndrome	1 (1.2)
Membranoproliferative glomerulonephritis	3 (3.5)
Lupus nephritis	2 (2.4)

On analysis between parameters [Tables 5.1 - 5.3 and 6.1 - 6.3], it is noted that hypertension, oliguria and oedema were significantly more in glomerular haematuria. Urine-specific gravity was significantly lower in microscopic and non-glomerular haematuria. Renal functions showed elevated serum creatinine levels in macroscopic (P = 0.046) and glomerular haematuria (0.076). Hypoalbuminemia, hypertriglyceridemia and low complement levels were also noted significantly in glomerular haematuria.

Significant associations were also noted between glomerular haematuria with macroscopic haematuria, and non-glomerular haematuria with microscopic haematuria.

Patients with macroscopic/glomerular haematuria had more associated complications (acute kidney injury, dyselectrolytemia, sepsis, multiple organ dysfunction syndrome [MODS], hypertension, renal failure, posterior reversible encephalopathy syndrome, uremic encephalopathy and pulmonary oedema) and morbidity at the time of discharge and had more hospital stay.

Table 5.1: Analysis between glomerular and non-glomerular haematuria (clinical features).

Clinical features	Non-glomerular	Glomerular	*P*-value
Microscopic haematuria	42	7	<0.001
Macroscopic haematuria	15	21	
Fever			
Present	38	13	0.096
Absent	18	15	
Oedema			
Present	10	19	<0.001
Absent	47	9	
Blood pressure			
Normal	46	14	0.006
Hypotension	0	1	
Hypertension	11	13	
Urine output*			
Normal	41	7	<0.001
Increased	1	0	
Decreased	8	10	
Per abdomen examination			
Normal	39	13	0.036
Hepatomegaly	4	1	
Splenomegaly	3	0	
Fluid thrill	0	2	
Shifting dullness	4	5	
Renal angle tenderness	0	1	
Known case of systemic illness/renal disease**			
No	49	16	0.006
Yes	8	12	

*Documented cases only considered. **Systemic illness includes - Wiskott Aldrich syndrome, chronic kidney disease, end stage renal disease, nephrotic syndrome, mesangioproliferative glomerulonephritis, hemolytic anemia, systemic lupus erythematosus, focal segmental glomerulosclerosis

Comparison of parameters showed age, gender, birth history, vital signs (temperature, pulse rate and respiration rate), urine analysis (pH, colour, pus cells and epithelial cells), urine culture between glomerular and non-glomerular groups, microscopic haematuria and macroscopic haematuria group did not render significant association.

Similarly, a comparison of haemoglobin, total leucocyte counts, platelet counts, C-reactive protein levels, blood urea, serum electrolytes (Sodium, potassium, calcium and phosphorus), liver function test (serum glutamic-oxaloacetic transaminase [SGOT], serum glutamic pyruvic transaminase [SGPT], alkaline phosphatase [ALP] and bilirubin), blood culture, ASO, ANA-IF, chest X-ray and ultrasound abdomen also did not render any significant associations.

Table 5.2: Analysis between glomerular and non-glomerular haematuria (urine analysis).

Urine analysis	Non-glomerular	Glomerular	*P*-value
Urine appearance			
Clear	13	0	0.001
Slight turbid	44	26	
Turbid	0	2	
Urine specific gravity			
1.005	7	1	0.005
1.010	15	8	
1.015	24	5	
1.020	5	12	
1.025	5	2	
1.030	1	0	
Urine RBC			
1–2	15	1	<0.001
2–4	17	3	
4–6	10	3	
6–10	6	4	
10–15	2	2	
15–20	3	2	
Numerous	4	13	
Urine protein			
Negative	30	0	<0.001
Trace	4	0	
1+	23	3	
2+	0	14	
3+	0	6	
4+	0	5	
Urine casts/crystals			
Nil	53	14	<0.001
Granular casts	2	14	
Calcium oxalate crystals	1	0	
Uric acid crystals	1	0	
Urine culture			
Positive	19	8	0.806
Negative	38	19	

RBC: Red blood cell

Table 5.3: Analysis between glomerular and non-glomerular haematuria (blood investigations/complications).

Blood investigations/ complications	Non Glomerular	Glomerular	*P*-value
Renal function tests			
Normal	50	20	0.076
Elevated creatinine	7	8	
S. albumin			
<2	0	3	0.033
2–2.4	1	4	
2.5–2.9	3	8	
3–3.4	10	3	
3.5–3.9	14	4	
>4	25	4	
Lipid profile*			
Normal	3	3	0.012
Hypertriglyceridemia	2	6	
Venous blood gas*			
Normal	4	9	0.002
Metabolic acidosis	6	3	
Metabolic alkalosis	1	0	
Respiratory acidosis	0	2	
Complement levels*			
Normal	4	3	<0.001
Low C3	3	18	
Low C3, Low C4	0	1	
Complications#			
No	46	12	0.001
Yes	11	16	
Duration of hospital stay			
<7 days	35	8	0.006
8–14 days	16	11	
>15 days	5	9	
Morbidity at discharge			
Abnormal urine analysis	12	22	<0.001
Hypertension	1	1	
Deranged RFT	6	6	
Renal scarring	1	0	
MODS	1	0	

*Documented cases only considered. #Complications include - acute kidney injury, dyselectrolytemia, sepsis, mods, hypertension, renal failure, posterior reversible encephalopathy syndrome, uremic encephalopathy, pulmonary oedema. RFT: Renal function test; MODS: Multiple organ dysfunction syndrome

Table 6.1: Comparison between microscopic and macroscopic haematuria (clinical features).

Clinical features	Microscopic	Macroscopic	*P*-value
Non-glomerular haematuria	42	15	<0.001
Glomerular haematuria	7	21	
Fever			
Present	34	17	0.042
Absent	14	19	
Oedema			
Present	9	20	<0.001
Absent	40	16	
Blood pressure			
Normal	39	21	0.028
Hypotension	1	0	
Hypertension	9	15	
Urine output*			
Normal	32	16	0.109
Increased	1	0	
Decreased	8	10	
Per abdomen examination			
Normal	33	19	0.028
Hepatomegaly	3	2	
Splenomegaly	2	1	
Fluid thrill	2	0	
Shifting dullness	1	8	
Renal angle tenderness	0	1	
Known case of systemic illness/renal disease**			
Yes	10	10	0.450
No	39	26	

*Documented cases only considered. **Systemic illness includes - Wiskott Aldrich syndrome, chronic kidney disease, end stage renal disease, nephrotic syndrome, mesangioproliferative glomerulonephritis, hemolytic anemia, systemic lupus erythematosus, focal segmental glomerulosclerosis

DISCUSSION

In our retrospective study of 84 children presented with microscopic/macroscopic haematuria, we found that non-glomerular causes were more common. However, glomerular causes were predominant in patients presented with macroscopic haematuria. These results were consistent with previous findings noted by Mishra *et al.*[7] Our study showed that UTI was the most common cause of microscopic/non-glomerular haematuria, and infection-related glomerulonephritis was the most common cause of glomerular/macroscopic haematuria. These findings were in agreement with studies by Mishra *et al.*[7] but were contradicting in comparison to some of the existing studies, where IgA nephropathy was the most common cause for macroscopic haematuria.[3,9-11]

Our results show that children with glomerular haematuria were more likely to present with high blood pressure, oedema and oliguria compared to those with non-glomerular haematuria. This is in agreement with the previous studies.[12] According to research by Ashraf *et al.* (2013), children having glomerular diseases often present with significant proteinuria and dyslipidaemia[12], which was in agreement with our findings.

The high incidence of anaemia (61.9%) and dyselectrolytemia (53.5%) underscores the importance of thorough laboratory evaluation, including complete blood counts, electrolyte panels and renal function tests. Anaemia was the most commonly

Table 6.2: Comparison between microscopic and macroscopic haematuria (urine analysis).

Urine analysis	Microscopic	Macroscopic	*P*-value
Urine appearance			
Clear	12	1	0.003
Slight turbid	37	33	
Turbid	0	2	
Urine specific gravity			
1.005	5	3	0.946
1.010	12	11	
1.015	17	12	
1.020	9	8	
1.025	5	2	
1.030	1	0	
Urine RBC			
1–2	16	0	<0.001
2–4	20	0	
4–6	13	0	
6–10	0	10	
10–15	0	4	
15–20	0	5	
Numerous	0	17	
Urine protein			
Negative	22	8	0.001
Trace	4	0	
1+	17	9	
2+	3	11	
3+	1	5	
4+	2	3	
Urine casts/crystals			
Nil	43	24	0.019
Granular casts	5	11	
Calcium oxalate crystals	0	1	
Uric acid crystals	1	0	
Urine culture			
Positive	20	7	0.059
Negative	29	28	

RBC: Red blood cell

Table 6.3: Comparison between microscopic and macroscopic haematuria (blood investigations/complications).

Blood investigations/ complications	Microscopic	Macroscopic	*P*-value
Renal function tests			
Normal	44	26	0.046
Elevated creatinine	5	10	
S. albumin			
<2	1	2	0.652
2–2.4	3	2	
2.5–2.9	4	7	
3–3.4	5	9	
3.5–3.9	11	8	
>4	23	6	
Lipid profile*			
Normal	2	4	0.214
Hypertriglyceridemia	4	4	
Venous blood gas*			
Normal	5	8	0.233
Metabolic acidosis	3	6	
Metabolic alkalosis	1	0	
Respiratory acidosis	1	1	
Complement levels*			
Normal	4	3	<0.001
Low C3	3	18	
Low C3, Low C4	0	1	
Complications#			
No	41	17	<0.001
Yes	8	19	
Duration of hospital stay			
<7days	30	14	0.077
8–14 days	11	16	
>15days	8	6	
Morbidity at discharge			
Abnormal urine analysis	7	27	<0.001
Hypertension	1	1	
Deranged RFT	4	8	
Renal scarring	1	0	
MODS	1	0	

*Documented cases only considered. #complications include – acute kidney injury, dyselectrolytemia, sepsis, multiple organ dysfunction syndrome (MODS), hypertension, renal failure, posterior reversible encephalopathy syndrome, uremic encephalopathy and pulmonary oedema. RFT: Renal function test

associated comorbidity, predominantly due to nutritional causes. The significant correlation between glomerular haematuria and hypoalbuminemia highlights the need for close observation of kidney function and consideration of renal biopsy.

Our study demonstrated that children with glomerular haematuria had a longer hospital stay and higher morbidity at discharge compared to those with non-glomerular haematuria. This emphasises the value of early detection and management of glomerular diseases to prevent long-term complications. A study by Youn *et al*. reported similar findings, highlighting the need for prompt intervention in children with glomerular haematuria.[3]

Our findings are consistent with the previous studies[8] that reported similar aetiologies and clinical profiles of haematuria in children. However, our study highlights the need for increased awareness of UTI as a common cause of haematuria in children.

Our research highlights the significance of promptly evaluating children who have haematuria for identifying underlying aetiologies, consideration of UTI as a potential cause of haematuria and aggressive management of glomerular diseases to prevent long-term complications.

Our study was limited by its single-centre design and rather a small sample size, which may limit generalizability.

CONCLUSION

There are various causes for haematuria in children, both glomerular and non-glomerular. The clinical presentation and laboratory findings can differ based on the underlying reason. The findings highlight the importance of the early recognition and management of glomerular diseases and UTIs as a common cause of haematuria. The distinction between glomerular and non-glomerular aetiology will help in the proper investigation of the child and further aid in effective management. More research is required to comprehend the pathophysiology and management of haematuria better.

Ethical approval

The research/study approved by the Institutional Review Board at Yenepoya Deemed to be university, number YEC-1/2023/286, dated 7th October 2023.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Conflicts of interest

There are no conflicts of interest.

Use of artificial intelligence (AI)-assisted technology for manuscript preparation

The authors confirm that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

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