

NEPHROLOGY AND UROLOGY HISTORY OSCE STATION

Red urine

HEMATURIA

Urine which is red in colour or tests positive for haemoglobin on urine sticks should be examined under the microscope to confirm haematuria (>10 red blood cells per high-power field).

Causes of 'red' or dark-colored urine

heme-negative / no RBC

Drugs

- Chloroquine
- Deferoxamine
- Iron sorbitol
- Metronidazole
- Nitrofurantoin
- Rifampin

Dyes (vegetable/fruit):

- Beets
- Blackberries
- food coloring

Metabolites

- Methemoglobin
- Porphyrin
- Urates

heme-positive/ no RBC (RBC < 5)

- Hemoglobin
- Myoglobin

heme-positive (RBC > 5)

- glomerular diseases: acute GN, IgA nephropathy, HUS, HSP, SLE nephritis.
- stones & hypercalciuria.
- infection.
- bleeding tendency.
- HB pathology: sickle cell anemia, G6PD, PNH.
- benign = familial, idiopathic, postural & recurrent.
- congenital malformation, cystic diseases, stenosis
- trauma.
- tumor.

Red urine history taking

1. Initial approach to the patient

2. personal data , Refer to the child by name

3. Open question about the chief Complaint (Red urine)

- when start - duration of the problem
- 1st time (GN) or intermittent microscopical in alport syn , gross H. urea in berger
- painful (renal stone) , painless (GN)
- color {pink to red or clots (extra renal) , brown –cola color (renal)
- At which phase of urination (early or terminal)
- time at night or with activity
- frequent & amount in each episode of urination or not
- Recent history of :
 - ✓ Recent febrile illness
 - ✓ Recent pharyngitis
 - ✓ Recent skin infection
 - ✓ Recent trauma, menstruation or strenuous exercise

4. association symptoms

fever , dysurea, abd pain, edema ,weight gain , headache , fatigue, FTT, pallor, jaundice

5. Systemic review:

- Respiratory system: URTI (IgA GN ,Alport ,HUS ,PSGN),hemoptysis (GPS)
- CVS: Exercise intolerance, fatigue Shortness of breath ,Edema (acute HTN).
- CNS: headache, LOC, convulsion (hypertensive encephalopathy)
- GIT: bloody diarrhea (HUS) ,abdominal mass (wilim tumor).
- Locomotor: Arthritis (SLE,HSP).
- Skin: rash (HSP) ,photosensitivity (SLE), petechial (bleeding tendency)
skin infection (post strept), Jane way & Osler nodules (infective endocarditis)
- Eyes & Ear: deafness, cataract & keratoconus (Alport syndrome)
- Hematology: bleeding tendency ,sickle cell anemia ,G6PD .

6. past history

Medical history : previous similar problem ,kidney diseases, URTI, renal stone, operation

Drug hx & food intake:

- urate/ rifampicin/ flagyl = orange urine.
- methylene blue = green urine.
- alcapronuria = black urine.
- cyclophosphamide = hemorrhagic cystitis.
- anticoagulants, chronic salicylate, desferrioxmine.
- food

7. Family History (H.urea, renal diseases, renal stones, deafness /alport syndrome.)

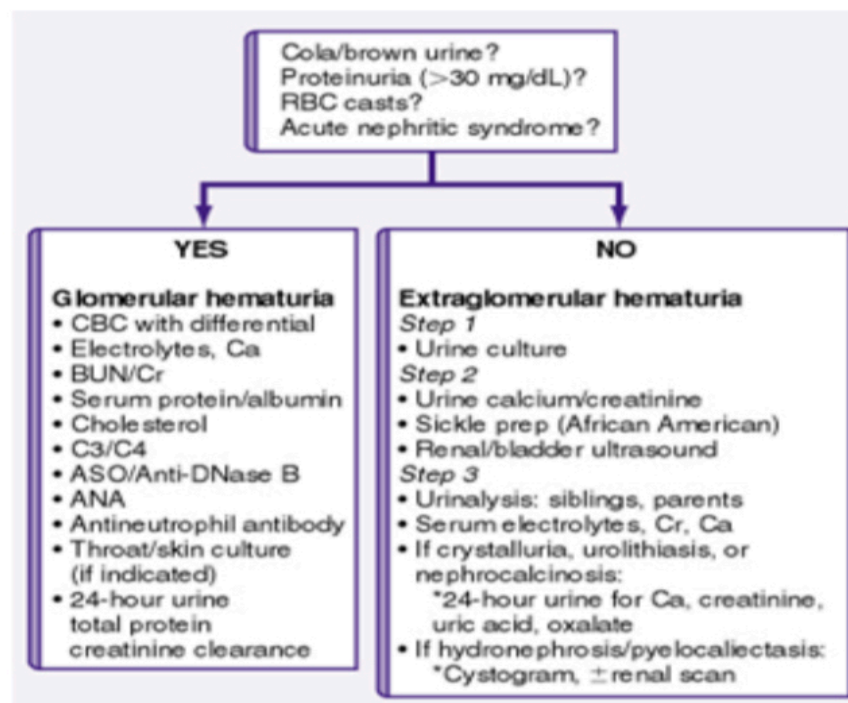
If bright H.urea or painfull without odema / wt gain this extraglomerular H.urea

- If painfull red urination ,recurrent and family history of renal stone **this** renal stone
- If 1st time painless acute H.urea ,history of ingestion **this** Dyes or drugs discoloration
- If painless H.urea with activity ,disappear at nigh ,**this** myoglobinurea
- If painless H.urea with petechia, aspirin or warfarin ingestion this bleeding tendency
- If painless H.urea with recent trauma **this** traumatic h.urea
- If painless H.urea with abdominal mass **this** wilim tumer.
- If painless H.urea with acute pallor , juandice **this** H.globinurea - G6PD
- If painless H.urea with SCD **this** sickle cell nephropathy .

If painless cola urination, oliguria , odema / wt gain this glomular H.urea

- With oliguria, odema **this** ARF
- With headache , coma **this** GN with Ht. encephalopathy
- with DOB **this** GN with CCF
- With Acute H. urea ,URTI - tonsillitis or skin infection **this** PSGN
- With Acute H. urea + URTI , Arthritis and purpura **this** HSP GN.
- With Acute H. urea + bloody diarrhea , skin rash **this** HUS GN
- With Acute H. urea + Arthritis , skin rash (multisystemic symptom) **this** SLE GN.
- With Jane way & Osler nodules **this** infective endocarditis GN
- With hemoptysis **this** good pasture GN
- Recurrent gross hematuria syndrome
 - With URTI (minimal systemic symptom) **this** IgA berger GN
 - With URTI may asymptomatic microscopic hematuria +, deafness, lenticonus, macular flecks ,corneal erosions, family H.urea **this** alport syndrome
 - Benign familial hematuria

start with urine analysis



Others: stones, bleeding (PT, PTT, INR, bleeding time), recurrent infection MCUG.

Manifestation of nephritic syndrome

- Hematuria – dysmorphic red blood cells, red blood cell casts
- Azotemia
- Oliguria
- Hypertension
- Variable proteinuria (nephrotic syndrome 10%)

Conditions that present as acute nephritic syndrome

- Acute postinfectious glomerulonephritis
- Membranoproliferative glomerulonephritis
- IgA berger nephropathy
- Henoch–Schönlein purpura nephritis
- Systemic lupus erythematosus nephritis
- Rapidly progressive glomerulonephritis
- Vasculitis : Wegener's granulomatosis

Investigation

Start with urine analysis

if cola –brown color urine + proteinuria +RBC cast = this acute nephritic syndrome

Evaluation of acute nephritic syndrome

Initial evaluation

- Blood count (rule out HUS)
- BUN, creatinine, electrolytes, serum albumin
- Quantify proteinuria (urine protein:creatinine ratio)
- Culture of throat and skin lesions
- Streptococcal antibody titers (ASO, anti DNase B, antihyaluronidase)
- Complement C3, C4

Further evaluation (If the diagnosis of APSGN is ruled out)

- Hepatitis B panel, hepatitis C titer for Membranous GN
- ANA (full lupus panel if ANA positive) ,ANCA
- Anti-GBM antibody titer (if pulmonary involvement)
- Renal biopsy

Low serum complement level

- SLE low C3 and C4
- Acute poststreptococcal glomerulonephritis low C3
- Membranoproliferative glomerulonephritis 2/3 of case low C3 and C4
- Subacute bacterial endocarditis low C3
- Shunt nephritis low C3

Normal serum complement level

- HSP
- IgA berger nephropathy
- Membranoproliferative glomerulonephritis 1/3 of case low C3
- ANCA GN

Acute Poststreptococcal Glomerulonephritis

most common in children aged 5-12 yr and uncommon before the age of 3 yr.

The typical patient develops an acute nephritic syndrome 1-2 wk after an antecedent streptococcal pharyngitis or 3-6 wk after a streptococcal pyoderma.

Heart failure, hyperkalemia, hyperphosphatemia, hypocalcemia, acidosis, seizures, uremia.

Dx of nephritic syndrome (H.urea, edema, AHT and oliguria) with

- +ve culture of throat and skin lesions
- +ve Streptococcal antibody titers (ASO, anti DNase B, antihyaluronidase)
- Low Complement C3
- Renal biopsy should be considered only in
 - presence of ARF+ nephrotic syndrome but no evidence for PSGN infection with normal C3
 - a low C3 level persist more than 2 mo after onset.

Complications.

Acute hypertension in 60% and with HT encephalopathy in 10% of cases.

acute renal dysfunction.

Management

The treatment of AGN consists of supportive therapy only. Children without volume expansion, hypertension, electrolyte problems, or decreased kidney function need only close follow up

Patients who develop significant renal impairment or hyperkalemia generally require hospitalization for monitoring and appropriate fluid and electrolyte management

Antibiotics

10-day course of systemic antibiotic therapy with **penicillin** is recommended to limit the spread of the nephritogenic organisms

Antibiotic treatment after the onset of APSGN does not alter the course of the disease.

Antibiotic prophylaxis given to family members may reduce the risk of spread of APSGN.

Fluid overload and hypertension

sodium restriction and diuretic therapy, in addition to antihypertensive medication, if necessary, combination of a loop or thiazide diuretic with a calcium channel blocker

Caution should be exercised with the use of angiotensin-converting enzyme inhibitors or angiotensin receptor blockers because of the risk of hyperkalemia or renal failure

Hyperkalemia

Potassium intake should be restricted.

If serum potassium is rising, potassium exchange resin, (Kayexalate), administered orally.

Life-threatening hyperkalemia should be treated as an emergency using intravenous bicarbonate, glucose, and insulin, or calcium gluconate as appropriate.

Rare cases of severe or symptomatic hyperkalemia should be treated with dialysis.

Dialysis

in severe uremia, especially if diuretic unresponsive anuria or oliguria is present

Prognosis (edema / proteinuria disappear within 10 days, low C3 2 mon, H.urea 12 mon)

Complete recovery occurs in more than 95% of children

Mortality in the acute stage can be avoided by appropriate management of ARF, CCF, AHT

Infrequently, glomerular hyalinization and chronic renal insufficiency.

in patients with chronic renal dysfunction consider other diagnoses such as MPGN

Recurrences are extremely rare

