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CLINICAL QUIZ

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Clinical Case Scenario

A 4-year-old boy of Nigerian descent is brought to the Paediatric Emergency department with a 5-day history of progressive periorbital puffiness, abdominal distension, and bilateral pitting lower-limb oedema. His parents report that he has been increasingly lethargic and has had significantly reduced urine output over the past 48 hours. He had a mild upper respiratory tract infection two weeks ago, but has no history of gross haematuria, skin rashes, or joint pain. He was born at full term with normal birth weight, and his developmental milestones are age-appropriate. There is no family history of renal disease.

On physical examination, his temperature is 36.8°C, heart rate is 154 beats per minute, blood pressure is 70/42 mmHg (below the 5th percentile for his age and height), respiratory rate is 30 breaths per minute, and oxygen saturation is 98% on room air. He is irritable but cooperative. He exhibits generalised anasarca, including marked scrotal oedema. His abdomen is tense, shifting dullness is present, and his capillary refill time is 3.5 seconds peripherally with cool extremities.

Initial Laboratory Investigations:

- Urinalysis: 4+ protein; 0–2 RBCs/high-power field; no casts.
- Spot Urine Protein-to-Creatinine Ratio (uPCR): 1402 mg/mmol (Normal: < 20 mg/mmol)
- Serum Albumin: 11 g/L
- Serum Creatinine: 90 µmol/L (Baseline expected: ~34 – 49 µmol/L – Jaffe method)
- Blood Urea Nitrogen (BUN): 11.4 mmol/L
- Serum Sodium: 128 mmol/L
- Serum Potassium: 4.1 mmol/L
- Total Serum Cholesterol: 9.2 mmol/L

Part 1: Multiple-Choice Questions (Best-of-Four)

Question 1: Given the patient's current hemodynamic status, physical findings, and laboratory profile, which of the following is the most appropriate immediate step in fluid management?

a) Rapid intravenous bolus of 20 mL/kg of 0.9% Normal Saline over 15 minutes to restore intravascular volume.
b) Infusion of 20% human salt-poor albumin at 1 g/kg over 4 hours, co-administered with intravenous furosemide 1 mg/kg at the midpoint.
c) Strict fluid restriction to 400 mL/m²/day (insensible losses) and initiating high-dose oral prednisolone at 60 mg/m²/day immediately.
d) Intravenous fluid resuscitation with 10 mL/kg of 5% human albumin solution over 30 to 60 minutes, with close monitoring of blood pressure and heart rate.

Question 2: The patient's serum sodium is 128 mmol/L. Which of the following mechanisms best explains this electrolyte derangement, and what is the correct therapeutic implication?

a) True sodium depletion due to urinary wasting of sodium; requires immediate correction with 3% hypertonic saline to prevent neurological complications.
b) Hypervolemic hyponatremia driven by non-osmotic release of antidiuretic hormone (ADH) secondary to intravascular volume depletion; requires cautious intravascular volume expansion rather than primary sodium supplementation.
c) Hyperlipidaemia-induced pseudohyponatremia due to high cholesterol; the true serum osmolality is normal, and no fluid or sodium alterations are required.
d) Artefactual hyponatremia caused by high serum albumin concentrations expanding the non-aqueous phase of plasma; requires calculation of corrected sodium.

Question 3: The patient's serum creatinine is elevated at 90 µmol/L, and his BUN is 11.4 mmol/L. Which diagnostic index or physiological finding would most reliably differentiate a functional, pre-renal Acute Kidney Injury (AKI) from structural Acute Tubular Necrosis (intrinsic AKI) in this specific child?

a) A urinary fractional excretion of sodium (FE_{Na}) of less than 1.0%, indicating intact tubular sodium reabsorption.
b) A urinary sodium concentration (U_{Na}) of less than 20 mmol/L, reflecting intense proximal tubular sodium conservation.
c) A urinary fractional excretion of urea (FE_{Urea}) of less than 35%, bypassing the confounding effects of altered tubular sodium kinetics.
d) A urinary urea nitrogen/creatinine ratio of greater than 20:1, with the presence of hyaline and granular casts on fresh urine sediment microscopy, confirms structural tubular damage.

Question 4: On day 3 of admission, while still heavily proteinuric, the child develops a sudden fever of 38.9°C, accompanied by worsening abdominal tenderness with subtle rebound and guarding. An ultrasound-guided paracentesis confirms bacterial peritonitis. Given the child's age and condition, what is the most appropriate empirical antibiotic regimen, antibiotic prophylaxis and most evidence-based vaccination plan?

a) Intravenous Ciprofloxacin, followed by lifelong daily oral Amoxicillin prophylaxis upon discharge.
b) Intravenous Cefotaxime or Ceftriaxone; followed by pneumococcal vaccination (PCV10 per Nigerian NPI schedule if completed, PLUS 23-valent pneumococcal polysaccharide vaccine (PPSV23) when in remission ≥3 months) for encapsulated bacteria.
c) Intravenous Vancomycin and Piperacillin-Tazobactam, followed by daily Penicillin prophylaxis until remission per routine practice.
d) Intravenous Ceftriaxone followed by NO antibiotic prophylaxis and NO additional pneumococcal vaccination beyond routine 3 doses of PCV in infancy according to NPI Schedule.

Question 5: The child is started on empiric corticosteroid therapy for presumed Minimal Change Disease (MCD). After 4 consecutive weeks of daily oral prednisolone at 60 mg/m²/day, his morning urine samples consistently show 2+ or 3+ proteinuria, and his generalised oedema persists. What is the most appropriate next step in clinical and diagnostic management?

a) Declare the patient Steroid-Resistant Nephrotic Syndrome (SRNS), perform a genetic panel for podocyte genes, and arrange a renal biopsy before initiating a calcineurin inhibitor.
b) Extend the daily oral prednisolone therapy at 60 mg/m²/day for an additional 2 weeks, as up to 15% are late responders and achieve remission by week 6.
c) Pulsed intravenous methylprednisolone (30 mg/kg, maximum 1g) for 3 consecutive days to overcome glucocorticoid receptor resistance before declaring failure.
d) Extend the daily oral prednisolone therapy at 60 mg/m²/day for an additional 2 – 4 weeks, as up to 20% of late responders achieve remission by week 6 – 8.

Question 6: During his hospital course, the child develops an asymptomatic, progressive increase in D-dimer levels and a further decline in serum albumin to 9 g/L. Based on current evidence-based consensus guidelines for managing thromboembolic risks in paediatric nephrotic syndrome, which strategy is indicated?

a) Prophylactic anticoagulation with Low-Molecular-Weight Heparin (LMWH) should be initiated because the serum albumin is <20 g/L, and he has profound intravascular volume depletion.
b) Routine prophylactic anticoagulation is contraindicated due to bleeding risks; management should focus entirely on avoiding haemoconcentration, encouraging mobilisation, and treating active infections.
c) Administer Low-Dose Aspirin (2–3 mg/kg/day) to inhibit increased platelet hyperaggregability, while avoiding systemic anticoagulation unless a definitive thrombosis is radiologically proven.
d) Therapeutic-dose Unfractionated Heparin (UFH) should be initiated immediately, targeting an activated partial thromboplastin time (aPTT) of 1.5–2.5 times the control baseline.

Question 7: The child's parents travelled to Asia for a holiday and returned after having a genetic test performed due to your expressed concern during a counselling session; a homozygous pathogenic missense mutation in the *NPHS2* gene (encoding podocin) was identified. How should this finding directly alter the child's long-term management strategy?

a) Perform a kidney biopsy and escalate immunosuppression by combining Tacrolimus with High-Dose Intravenous Methylprednisolone, as *NPHS2* mutations retain partial immunological responsiveness.
b) Taper off all immunosuppressive therapies to avoid unnecessary toxicity, focus management on strict renin-angiotensin-aldosterone system (RAAS) blockade, and prepare for pre-emptive live-donor renal transplantation.
c) Review with a geneticist and avoid renal transplantation because *NPHS2*-mutated nephrotic syndrome carries an extremely high risk (>80%) of immediate post-transplant recurrence driven by circulating permeability factors.
d) No need for a kidney biopsy; refer to a paediatric nephrologist to initiate maintenance therapy with Rituximab (anti-CD20 monoclonal antibody), as it directly stabilises the podocyte actin cytoskeleton independent of its B-cell depleting effects.

Part 2: Answer Key & Detailed Explanations

Question 1:

Aim: Immediate Resuscitation and Hemodynamic Optimisation

Correct Answer: D

Why is it correct: This patient is presenting in hypovolemic shock secondary to intravascular volume depletion (underfill mechanism), evidenced by tachycardia, hypotension, cold extremities, and a prolonged capillary refill time (3.5 seconds). In the setting of severe hypoalbuminemia (11 g/L), standard crystalloid fluid boluses (0.9% Normal Saline or *Ringer's lactate*) will rapidly shift into the interstitial space, worsening anasarca and ascites without maintaining intravascular volume. According to international guidelines, volume resuscitation must be achieved using an iso-oncotic or mildly hyperoncotic colloid solution like 5% human albumin (10–20 mL/kg) administered cautiously to restore perfusion without precipitating acute pulmonary oedema. This is obtained by using 25% albumin and diluting it to 1:5 parts with normal saline.

Why others are incorrect:

Option A is incorrect because 0.9% Normal Saline or Ringer's lactate will rapidly leak into the interstitium – because they lack oncotic pressure, worsening tissue oedema and abdominal compartment risk while failing to correct shock.

Option B describes the management of *stable* fluid overload with compromised skin or respiratory integrity. Giving loop diuretics to an already hypotensive, tachycardic patient in hypovolemic shock will worsen intravascular depletion and precipitate circulatory collapse.

Option C fails to address the medical emergency of hypovolemic shock.

Question 2

Aim: Pathophysiology and Interpretation of Electrolyte Derangements

Correct Answer: B

Why is it correct: The patient has hypervolaemic hyponatraemia (systemic fluid overload with intravascular depletion). The severe drop in oncotic pressure causes fluid to shift into the interstitium, triggering profound arterial underfilling. This stimulates the non-osmotic release of Antidiuretic Hormone (ADH) via baroreceptors, alongside activation of the RAAS. Excessive ADH causes free water retention in the collecting ducts that outpaces sodium retention, causing dilutional hyponatraemia. The management requires restoring intravascular volume (which shuts off the non-osmotic ADH trigger), not primary sodium replacement. So, the key teaching point: Hyponatremia in nephrotic syndrome is dilutional (water excess), not sodium depletion. Treatment is intravascular volume restoration (albumin), not sodium supplementation. Rapid Na^+ correction risks osmotic demyelination syndrome (max 8-12 mmol/L/24 h).

Why others are incorrect:

Option A is incorrect because total body sodium is actually *elevated* due to intense renal retention; hyponatremia is dilutional. Hypertonic saline is contraindicated unless life-threatening seizures occur.

Option C is incorrect because while severe hyperlipidaemia can cause pseudohyponatremia, the degree of cholesterol elevation seen here (9.2 mmol/L) is insufficient to cause a drop to 128 mmol/L. The physiological drive is primarily ADH-mediated free-water retention.

Option D is incorrect because hyperalbuminemia causes pseudohyponatremia, whereas this patient has severe *hypoalbuminemia*.

Question 3

Aim: Interpretation of Renal Impairment and Volumetric Status

Correct Answer: C

Why it is correct: In paediatric nephrotic syndrome, the massive glomerular filtration of albumin and subsequent proximal tubular reabsorption processes completely alter traditional urinary markers. Proximal tubules reabsorb sodium aggressively alongside protein, which can drop both FE_{Na} and U_{Na} to extremely low levels (<1% and <20 mmol/L, respectively) even during structural ATN. Fractional excretion of urea (FEUrea) is independent of sodium transport mechanisms and is the most reliable index to differentiate functional pre-renal impairment FEUrea < 35 from structural injury FEUrea > 35 in a nephrotic patient.

Why others are incorrect:

Options A and B are unreliable in nephrotic syndrome because intense baseline sodium avidity yields pre-renal parameters even when structural acute tubular necrosis is underway.

Option D is incorrect because the urine urea nitrogen/creatinine ratio does not exist – what is obtainable is the blood urea nitrogen/Creatinine ratio. A BUN/Cr ratio > 20, and hyaline and granular casts can frequently be seen in severe pre-renal states due to highly concentrated urine and protein precipitation and are not specific to ATN in this context.

Question 4

Aim: Managing Acute Infectious Triggers, Antibiotic Prophylaxis and vaccination in a child with Nephrotic Syndrome

Correct Answer: B

Why is it correct: For bacterial peritonitis in paediatric nephrotic syndrome:

Empirical treatment should cover the most common pathogen - Streptococcus pneumoniae (as well as other encapsulated organisms) [The International Paediatric Nephrology Association (IPNA) Grade A: strong recommendation] – so, Ceftriaxone OR Cefotaxime are suitable options as they provide excellent coverage. Caveat – antibiogram sensitivity is still the standard.

Antibiotic prophylaxis: DO NOT give routinely [IPNA Grade C: weak]

"Antibiotic prophylaxis should not be given routinely to children with SSNS"

IPNA recommendation Grade A (strong):

"We recommend reviewing the child's vaccination status at disease onset and completing all inactivated vaccinations following the vaccination schedule that is recommended for healthy children without delay, especially for encapsulated bacteria (pneumococcus, meningococcus, haemophilus influenzae)"

PCV10: Nigeria's routine NPI (6, 10, 14 weeks), child should have received

PPSV23: When in remission ≥3 months (93% seroconversion)[5]

Why PPSV23: PCV10 doesn't cover serotypes 3, 6A, 19A (major invasive strains)
So, the key teaching point: the IPNA guideline prioritises vaccination over prophylaxis for preventing peritonitis recurrence in children with nephrotic syndrome.
Why others are incorrect
Option A:
1. Antibiotic (Ciprofloxacin): Not recommended for peritonitis treatment and poor pneumococcal coverage: Fluoroquinolones have inconsistent activity against *S. pneumoniae* (38% of cases)
2. Prophylaxis (Lifelong Amoxicillin): Directly contradicts IPNA 2022: "antibiotic prophylaxis should not be given routinely to children with SSNS". Wrong duration: Penicillin prophylaxis, if any, should be until remission, NOT lifelong. Increased antibiotic resistance risk without added benefit

Option C:
1. Antibiotics (Vancomycin + Piperacillin-Tazobactam): Over-treatment for empirical therapy of community-acquired peritonitis. Vancomycin only covers Gram-positive (MRSA) — unnecessary without blood culture evidence. Not recommended by IPNA for peritonitis
2. Prophylaxis (Penicillin until remission): Contradicts IPNA 2022: "antibiotic prophylaxis should not be given routinely to children with SSNS". While some clinicians use penicillin until remission empirically, IPNA does NOT recommend routine prophylaxis. Key correction: IPNA prioritises vaccination over prophylaxis

Option D:
1. Antibiotic (Ceftriaxone): INCOMPLETE: While Ceftriaxone is appropriate, the option lacks the vaccination strategy
2. NO Additional Pneumococcal Vaccination (PSV23): Directly contradicts IPNA 2022 Grade A recommendation: "We recommend completing all inactivated vaccinations...especially for encapsulated bacteria (pneumococcus)"
PCV10 alone is insufficient: Nigerian routine PCV10 does not cover serotypes 3, 6A, 19A (major invasive strains in nephrotic syndrome)
2025 Evidence: PPSV23 provides 93% seroconversion in SSNS during remission
PPSV23 is critical in children with nephrotic syndrome: Covers 23 serotypes (including 3, 6A, 19A + 10 additional strains not in PCV10) and is already in use in more resourced countries.
3. NO Antibiotic Prophylaxis: CORRECT. This aligns with IPNA ("should not be given routinely"), BUT the omission of PPSV23 makes this option incorrect.

Question 5
Aim: Initial Immunosuppressive Stratification and Diagnostic Stewardship
Correct Answer: A
Why is it correct: International guidelines (KDIGO and the International Pediatric Nephrology Association - IPNA) define Steroid-Resistant Nephrotic Syndrome (SRNS) in children as the absence of complete remission after 4 weeks of daily oral prednisolone at 60 mg/m²/day (or 2 mg/kg/day). Once SRNS is confirmed, immediate evaluation via a genetic panel (to rule out structural podocyte mutations) and a kidney biopsy are indicated before starting second-line therapies like calcineurin inhibitors (Cyclosporine or Tacrolimus). The key teaching point: the absence of complete remission after 4 weeks of daily oral prednisolone at 60 mg/m²/day (or 2 mg/kg/day, max. 60mg) is the new definition of steroid-resistant nephrotic syndrome (SRNS). Such children should be referred to a paediatric nephrologist or managed in conjunction with one.

Why others are incorrect:
Option B is incorrect because older protocols allowed extension to 6 weeks, but updated consensus guidelines recommend capping standard initial therapy at 4 weeks to mitigate severe steroid toxicity.
Option C is an alternative intensive strategy used in some centres, but the updated international guidelines state that failing a 4-week daily regimen is sufficient to formally declare primary steroid resistance and initiate the biopsy/genetic workflow. In addition, this intensified steroid / antiproteinuric regimen is ONLY used during the 4–6-week confirmation phase.
Option D is incorrect because older protocols like the Ibadan Consensus allowed extension to 6 - 8 weeks, but updated guidelines now cap standard initial therapy at 4 weeks to mitigate steroid toxicity. Extension to 6 weeks – now called the confirmatory period – is only considered when there is evidence of partial remission, (serum albumin > 30g/L and spot urine protein

creatinine ration > 20mg/mmol but < 200mg/mmol), while on the initial daily oral prednisolone therapy at 60 mg/m²/day or 2 mg/kg (max. 60 mg) during the first 4 weeks. Such children, termed late responders, should achieve complete remission by week 6, or are otherwise still classified as Steroid Resistant Nephrotic Syndrome (SRNS).

Question 6

Aim: Managing Hypercoagulability and Prothrombotic Risk

Correct Answer: B

Why is it correct: Routine prophylactic anticoagulation (with LMWH or warfarin) is not recommended in asymptomatic paediatric nephrotic patients, regardless of severe hypoalbuminemia or elevated D-dimers, due to the high risk of haemorrhage and lack of data showing a net clinical benefit. Paediatric consensus guidelines emphasise that thromboprophylaxis should rely on non-pharmacological measures: maintaining hydration, avoiding unnecessary immobilisation, aggressive treatment of infections, and minimising central venous line placements. So, the key teaching point is:

No consensus supports prophylactic anticoagulation in paediatric nephrotic syndrome based solely on low serum albumin. Thrombosis risk is low in children despite hypoalbuminemia: Children: <5% develop thrombosis, even with severe hypoalbuminemia and Clinical thrombosis: Rare in paediatric nephrotic syndrome (<5% incidence)

Why others are incorrect:

Option A is incorrect because international guidelines explicitly advise against using arbitrary albumin thresholds (like <20 g/L) to trigger pharmacologic prophylaxis in children, unlike adult guidelines.

Option C is incorrect as routine antiplatelet therapy with aspirin is not standard or recommended without evidence of underlying thromboembolic complications or additional severe genetic prothrombotic risks.

Option D describes full therapeutic anticoagulation, which is strictly contraindicated unless a thromboembolic event has been confirmed via objective diagnostic imaging.

Question 7

Aim: Advanced Pharmacogenomics and Precision Management

Correct Answer: B

Why is it correct: Genetic mutations in structural podocyte proteins (such as *NPHS2* encoding podocin) cause an inherited, structural defect of the glomerular filtration barrier. These structural forms of SRNS do not respond to immunosuppressive therapies. Continuing steroids or adding alkylating agents/calcineurin inhibitors exposes the infant to profound toxicity without therapeutic benefit. The standard of care is to taper off immunosuppression, maximise antiproteinuric therapy via RAAS blockade (ACE inhibitors/ARBs), and plan for renal transplantation.

Why others are incorrect:

Option A is incorrect because structural genetic podocytopathies are non-immune-mediated and do not respond to escalated immunosuppression.

Option C is incorrect; genetic forms of SRNS have an exceptionally low risk of post-transplant recurrence (<5%) because the donor kidney will possess normal, wild-type podocin proteins. High recurrence risks are seen in non-genetic, circulating-factor-mediated FSGS.

Option D is incorrect because Rituximab is ineffective in primary genetic podocytopathies.

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