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Case Study: Tracing the Clues – A Child with Hematuria and Proteinuria

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Abstract

Hematuria and proteinuria in pediatric patients can be indicative of various renal pathologies, necessitating a structured diagnostic approach. This case study examines a 9-year-old male presenting with dark-colored urine, mild facial puffiness, and fatigue. Urinalysis revealed significant hematuria with dysmorphic RBCs and proteinuria, prompting further investigations. Laboratory findings, including low C3 levels and elevated ASO titers, led to a diagnosis of Post-Streptococcal Glomerulonephritis (PSGN). The case underscores the importance of early detection, targeted investigations, and supportive management in ensuring positive clinical outcomes.

Keywords: *Hematuria, Proteinuria, Post-Streptococcal Glomerulonephritis, Pediatric Nephrology, RBC Casts, Complement Levels, Streptococcal Infection, Urinalysis*

Patient Presentation

A 9-year-old boy was brought to the pediatric nephrology clinic by his parents due to dark-colored urine noticed over the past week. The parents also reported mild facial puffiness, especially in the mornings, and occasional complaints of fatigue. There was no significant past medical history, no recent infections, and no family history of kidney disease.

Clinical Examination and Initial Assessment

On physical examination, the child appeared well-nourished and was normotensive (blood pressure 105/70 mmHg). No edema was noted beyond mild facial puffiness. Cardiovascular and respiratory examinations were unremarkable. Abdominal examination showed no organomegaly.

Laboratory and Urinalysis Findings

- **Urinalysis**:
  - Color: Dark reddish-brown
  - Protein: 2+
  - Blood: 3+
  - RBCs: Dysmorphic
  - WBCs: Normal range
  - Casts: RBC casts present
- **Serum Studies**:
  - Creatinine: 0.5 mg/dL (Normal for age)
  - BUN: 15 mg/dL
  - Albumin: 3.5 g/dL
  - ASO (Anti-Streptolysin O) Titer: Elevated

- C3 Complement: Low
- ANA and ANCA: Negative

Differential Diagnosis Considered

1. **Post-Streptococcal Glomerulonephritis (PSGN)** – Strongly suggested by hematuria, proteinuria, low C3, and elevated ASO titer.
2. **IgA Nephropathy** – A possibility but usually presents with normal complement levels.
3. **Alport Syndrome** – Typically associated with family history and hearing loss, which were absent.
4. **Lupus Nephritis** – Less likely given negative ANA and absence of systemic symptoms.

Final Diagnosis

The findings were consistent with **Post-Streptococcal Glomerulonephritis (PSGN)**, a common cause of hematuria and proteinuria in children following a streptococcal infection.

Pathophysiology of PSGN

PSGN occurs due to immune complex deposition in the glomerular basement membrane following a group A beta-hemolytic streptococcal infection. The antigen-antibody immune response leads to inflammation and complement activation, causing glomerular injury. This manifests clinically as hematuria, proteinuria, edema, and sometimes hypertension.

Management and Outcome

- **Supportive Care**: The child was advised rest, and a low-sodium diet was recommended.
- **Blood Pressure Monitoring**: Though initially normal, the child was monitored for any signs of hypertension.
- **Antibiotic Therapy**: Although PSGN is post-infectious, a 10-day course of penicillin was prescribed to eradicate any residual streptococcal infection.
- **Fluid and Electrolyte Balance**: The child was encouraged to maintain adequate hydration, with monitoring for any electrolyte imbalances.
- **Follow-Up Plan**: Weekly monitoring of blood pressure and urine protein levels was initiated.

At a 6-week follow-up, hematuria had resolved, and proteinuria had significantly decreased. Serum C3 levels had normalized, confirming recovery from the acute phase.

Conclusion

This case highlights the importance of urinalysis in evaluating pediatric kidney conditions. The presence of hematuria with RBC casts and proteinuria, combined with low C3 and an elevated ASO titer, provided a strong diagnostic clue for PSGN. Early identification and appropriate management ensured a good prognosis for the child. Pediatricians and nephrologists must recognize these clinical patterns to guide timely intervention and prevent complications.

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