Case Report

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A Case Report Of Haemolytic Uraemic Syndrome Following An Episode Of Gastroenteritis.

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Abstract

Haemolytic uraemic syndrome (HUS) is a serious condition characterized by haemolytic anemia, thrombocytopenia, and acute kidney injury, frequently following an infection with Shiga toxin-producing Escherichia coli. We present the case of an 11-year-old female who developed HUS two weeks after an episode of gastroenteritis. She presented with decreased urine output, pallor, and hypertension. Laboratory findings revealed microangiopathic haemolytic anemia, thrombocytopenia, and acute renal failure, confirming the diagnosis of HUS. She was managed with supportive care, including intravenous fluids, blood transfusions, and renal replacement therapy. Her condition gradually improved, and she was discharged after three weeks with normalized renal function and hematologic parameters. This case highlights the critical importance of early recognition and management of HUS in pediatric patients presenting with post-diarrheal symptoms and acute kidney injury. Reviewing similar cases underscores the variability in clinical presentations and outcomes, emphasizing the need for prompt, individualized care strategies.

Keywords:- Haemolytic uraemic syndrome,Gastroenteritis,Pediatric nephrology, Microangiopathic hemolytic anemia

INTRODUCTION

Haemolytic uraemic syndrome (HUS) is a severe, life-threatening condition characterized by the triad of haemolytic anemia, thrombocytopenia, and acute kidney injury. It primarily affects children and is often precipitated by an infection with Shiga toxin-producing Escherichia coli (STEC), most commonly E. coli O157.[1]

HUS can also follow other infections or occur in association with certain genetic mutations. This condition necessitates prompt recognition and intervention due to its potential for significant morbidity and mortality. [2]

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HUS has a global incidence rate of approximately 2-3 cases per 100,000 children under the age of five each year, making it a relatively rare but critical pediatric condition. The pathophysiology of HUS involves endothelial injury, particularly in the kidneys, due to Shiga toxin or other triggers, leading to microangiopathic haemolytic anemia, platelet aggregation, and microvascular thrombosis. This endothelial damage disrupts normal blood flow, resulting in ischemia and the characteristic renal impairment seen in HUS.[3]

Clinically, HUS typically presents with nonspecific prodromal symptoms such as diarrhoea, often bloody, followed by pallor, fatigue, irritability, and signs of acute renal failure like oliguria or anuria. Laboratory findings are crucial for diagnosis, revealing microangiopathic haemolytic anemia with schistocytes on a blood smear, thrombocytopenia, and elevated serum creatinine levels. Other important findings can include elevated lactate dehydrogenase (LDH) levels, indirect hyperbilirubinemia, and decreased haptoglobin levels, which support the diagnosis of hemolysis.[4]

In this report, we discuss the case of an 11-year-old female who developed HUS following an episode of gastroenteritis, highlighting the critical importance of recognizing this syndrome early in patients presenting with post-diarrheal symptoms and acute renal failure.

CASE REPORT

An 11-year-old female, previously healthy, presented to the emergency department with complaints of decreased urine output and generalized weakness. Her symptoms began approximately two weeks after recovering from an episode of gastroenteritis characterized by diarrhoea, nausea, and abdominal cramps, which resolved without antibiotic treatment.

On examination, the patient appeared pale and lethargic. Vital signs were notable for hypertension (blood pressure 145/95 mmHg), and physical examination revealed periorbital edema and mild abdominal tenderness. There was no hepatosplenomegaly or lymphadenopathy. Initial laboratory investigations revealed hemoglobin of 7.2 g/dL, platelet count of 45,000/µL, and serum creatinine of 3.5 mg/dL, indicating significant renal impairment. Blood smear showed numerous schistocytes, consistent with microangiopathic haemolytic anemia. Lactate dehydrogenase (LDH) was elevated at 1,200 U/L, haptoglobin was undetectable, and bilirubin levels were elevated, supporting the diagnosis of haemolysis.

Given the history of recent gastroenteritis and the laboratory findings, a diagnosis of HUS was considered. Further tests for Shiga toxin and stool culture for E. coli O157

were performed. The patient was referred to the pediatric intensive care unit for close monitoring and supportive care, including intravenous fluids, blood transfusions, and antihypertensive medications. Renal replacement therapy was initiated due to worsening renal function and oliguria.

Parameter	Value	Reference Range
Hemoglobin (g/dL)	7.2	11.5-15.5
Platelet count (/µL)	45,000	150,000-450,000
Serum creatinine (mg/dL)	3.5	0.5-1.0
LDH (U/L)	1,200	140-280
Haptoglobin (mg/dL)	<10	30-200
Total bilirubin (mg/dL)	3.0	0.1-1.2

Table 1:- Relevant Laboratory Findings at Admission.

DISCUSSION

HUS is a critical condition in paediatrics, often following an episode of gastroenteritis. The classic post-diarrheal HUS is predominantly associated with STEC infections, but other aetiologies can also trigger this syndrome.[5] In this case, the temporal relationship between the patient's gastroenteritis and the onset of HUS symptoms underscores the need for vigilance in post-diarrheal patients, particularly children.[6,7]

Previous case reports have documented similar presentations. For instance, a study by Tarr et al. described a series of pediatric HUS cases following E. coli O157infections, highlighting the importance of early diagnosis and supportive care . Another report by Wong et al emphasized the variability in clinical outcomes based on the severity of renal impairment and the promptness of intervention.[8] A comprehensive review by Wijnsma et al discussed various therapeutic strategies, including the role of plasmapheresis and eculizumab in severe cases, although supportive care remains the cornerstone of management.[9]

The primary focus in managing HUS is supportive care, which includes maintaining fluid and electrolyte balance, managing hypertension, and providing renal support as needed. In severe cases, interventions like plasmapheresis or eculizumab have been used, although their efficacy varies. Preventing HUS involves proper food handling and hygiene to reduce the risk of STEC infections, which are the most common cause.[10]

.CONCLUSION

The antenatal diagnosis of hypoplastic left heart syndrome at 22 weeks of gestation is critical for the effective management of this congenital heart defect. Early detection through fetal echocardiography, multidisciplinary counselling, and planned delivery at specialized centres significantly improve postnatal outcomes. This case underscores the importance of comprehensive prenatal care and coordinated management in improving the prognosis for infants with HLHS.

Conflict of interest
None

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