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Case Report

Uncommon but Critical Hemolytic Uremic Syndrome in an Adult

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Abstract

Hemolytic Uremic Syndrome (HUS) associated with acute gastroenteritis is widely recognized as a common cause of acute renal failure in children. However, reports of HUS associated with gastroenteritis in adult patients are rare. The most frequently implicated pathogen is *Escherichia coli* O157:H7. Supportive and replacement therapy form the cornerstone of managing complications. The limited number of studies on HUS linked to gastroenteritis motivated us to present this case in order to highlight its rare symptoms. So, our aims are to present a case of HUS in an adult patient and to attract attention to clinicians to be careful in the rapid diagnosis and management of cases like this.

Keywords: Gastroenteritis; Hemolytic-Uremic Syndrome (HUS); Escherichia coli O157; Shiga-toxigenic Escherichia coli

Introduction

The term Hemolytic Uremic Syndrome (HUS) was first used in 1955 by Gasser et al. HUS is a life-threatening condition characterized by Microangiopathic Hemolytic Anemia (MAHA), thrombocytopenia, and acute renal failure (AKI) [1-7]. HUS is primarily classified as either typical (associated with Shiga toxin-producing *E. coli* [STEC-HUS]) or Atypical (aHUS), which results from dysregulated complement activation due to genetic mutations or autoimmune processes [2,3,5,7]. HUS connected infections caused by *E. coli* O157:H7 producing Shiga toxin, occurs in approximately 6-9%. While STEC-HUS is a well-recognized cause of AKI in children, adult cases are rarely reported [4,6]. Gastroenteritis is known to be a triggering factor for HUS, which is commonly associated with abdominal pain, vomiting, and bloody stools. Additional key symptoms for diagnosing HUS include AKI and neurological impairment [6-8]. A major outbreak in Germany in 2011 associated with *E. coli* O104:H4 had a disproportionately high mortality rate in adults over 60 years old [4,9]. Although HUS is much less common in adults than in children, the majority of fatalities related to HUS caused by STEC occur in people over 60 years old [4,6]. The rarity of adult cases often leads to delayed diagnosis and limited treatment options. Our aims are to present a case of HUS in an adult patient and to attract attention to clinicians to be careful in the rapid diagnosis and management of these cases.

Case Presentation

A 57-year-old female presented to the hospital with complaints of profuse diarrhea (10-15 episodes within 24 hours), nausea, vomiting, abdominal pain, severe weakness, headache, and myalgia. The

patient reported experiencing these symptoms for 4-5 days, with worsening severity. She also mentioned consuming bovine liver a day before the onset of symptoms. A routine check-up 10 days prior had shown normal laboratory parameters. The patient had a known history of hypertension under medication but had no previous infectious, autoimmune, or haematological diseases. On physical examination, the patient appeared unwell, with icteric sclera and mildly jaundiced skin. Cardiovascular examination revealed clear heart sounds with a rhythmic pulse, and blood pressure was 90/60 mmHg with a heart rate of 105 bpm. The abdomen was non-tender, with no splenomegaly, and renal angles were free of tenderness. The extremities showed no edema, and urine output was preserved but had decreased slightly over the previous two days. Laboratory findings indicated elevated creatinine, high Lactate Dehydrogenase (LDH), increased ferritin levels, and thrombocytopenia (Table 1).

Table 1: Laboratory Findings.

Hospitalization Days / Laboratory Test and Normal Range	D 0	D 1	D 2	D 4	D 5	D 6	D 7	D 10
Red blood cells count 4x5.6x10^6/UL	3.71	3.48	2.52	3.4	2.9	2.92	3.16	3.03
Hemoglobin (12.1-15.9g/dL)	11.2	10.1	7.4	10.6	8.8	9	9.3	9.3
Hematocrit (37-46%)	31.2	28.7	20.6	29.1	25.4	25.7	26.6	27.5
Platelets (150-400 × 10^3/μL)	46	25	33	51	81	130	214	338
Urea blood nitrogen (15-40mg/dl)	101.3	150.3	197	198.4	190	175.1	142.5	66.3
Creatinine (0.57-1.11md/dL)	2.48	2.9	4.8	5.4	5.31	4.72	3.65	1.96
Tot. bilirubin (0.3-1.2mg/dL)	5.18	4.55	4.81	3.35	1.2	0.72	0.7	0.46
Dir. Bilirubin (0.1-0.5mg/dL)	0.69	0.7	0.6	0.56	0.5	0.4	0.3	0.14
Dehydrogenase lactic (125-220U/L)	1397	1002	1132	1014	920	745	644	458
Reactive C-protein (<0.5mg/dL)	1.9	2.26	1.18	1.05	1.07	1.1	0.97	0.44
Albuminemia (3.5-4.8g/dL)	2.8	2.7	2.8	3.1	3.2	3.2	3.4	3.4
Total protein (5.8-7.3g/dL)	5.7	5.6	5.9	5.9	6	6.2	6	6.3
D-dimer (<0.5ug/mL)	1.87	5.3	6	6.49	8.2	11.96	8.98	4
Ferritin (5-204ng/mL)	1400	1455	1400	1340	1220	950	870	720
Fibrinogen (200-400mg/dL)	470	485		455			370	350

Laboratory Findings

Stool sample testing was positive for STEC O157:H7, confirming the diagnosis of STEC-HUS. Additional serological tests for leptospirosis, Hemorrhagic Fever with Renal Syndrome (HFRS), viral hepatitis B and C were negative. Urinalysis revealed hematuria and significant proteinuria (albumin+++), while faucal occult blood was positive. On the third day patients referred to irritability, tachycardia (110-125 rate/min) and hypertension. The patient received supportive therapy, including isotonic fluids, blood transfusion on day 2, fresh frozen plasma, albumin, electrolyte correction, anticoagulants, and symptomatic treatment. The patient's clinical and laboratory parameters improved, and she was discharged in stable condition after 10 days. A follow-up two-week post-discharge showed complete normalization of laboratory values.

Discussion

The incidence of HUS in individuals infected with STEC ranges from 5-15%, with the highest risk in children under five years old. Symptoms typically begin with bloody diarrhea 2-3 days post-exposure, progressing to HUS within 3-10 days, like in our case. Seasonal trends indicate higher incidence during warmer months, correlating with increased bacterial colonization in livestock [3]. Outbreaks of STEC-HUS have been linked to undercooked meat, unpasteurized dairy products, contaminated produce, and waterborne transmission [4,6]. In contrast, Atypical HUS (aHUS) is significantly less common but carries a higher morbidity and mortality rate. Atypical HUS does not resolve spontaneously and frequently leads to end-stage renal disease without treatment [6]. Rarely, secondary HUS can occur in association with infections (Streptococcus pneumoniae being the most common), autoimmune diseases, malignancies, pregnancy, and drug-induced endothelial injury [1,6,7].

Shiga toxin enters the bloodstream and primarily affects the kidneys and brain, causing endothelial damage, blood clot formation in small vessels, and multi-organ dysfunction. This leads to MAHA, thrombocytopenia, and AKI, often presenting with blood in the urine and proteinuria [1,2,4,6,8]. The same symptoms of our patient. The distinctive signs that guided us to the diagnosis included bloody diarrhea, widespread abdominal pain that developed after food consuming, and abnormal blood test findings. The disease usually starts with diarrhea, abdominal pain, and vomiting,

followed by the onset of HUS within 5-13 days, characterized by anemia, AKI, and in adults, severe neurological symptoms like confusion and seizures [4,8,10]. Diagnosis relies on the classic triad of anemia, thrombocytopenia, and AKI, supported by laboratory tests such as LDH, bilirubin levels, and stool PCR for *E. coli* O157:H7.

Supportive care remains the primary treatment for STEC-HUS [3,4,7,8]. Fluid management is crucial, with close monitoring of renal function and electrolyte balance. Blood transfusions and plasma exchange may be necessary. We had blood transfusion on the second day of hospitalization. The role of antibiotics remains controversial. In contrast, early antibiotic treatment improves outcomes in S. pneumoniae-induced HUS [6]. For aHUS, targeted therapy with eculizumab, a C5 complement inhibitor, has significantly improved survival rates, reducing end stage renal diseases progression from 60% to 6-15% in adults. Plasmapheresis is a second-line option, while steroids and antithrombotic therapy are no longer recommended due to a lack of proven benefit [6].

Conclusion

STEC-HUS remains a rare but severe cause of AKI in adults. Clinicians should consider *E. coli* O157:H7 when evaluating patients with diarrhea, especially those with a history of bloody diarrhea, because early recognition and aggressive supportive care are essential. However, further research is needed to refine treatment protocols, particularly for adult patients, to optimize prognosis and reduce morbidity and mortality. On the other hands we do not have a dedicated surveillance system for STEC-associated HUS in adults in Albania, so maybe this case will draw attention to clinicians and health politics makers.

Acknowledgement

None.

Conflict of Interest

None.

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