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Case study *Medical research*

A case study on hemolytic uremic syndrome- Leading to acute renal failure

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ABSTRACT

A 65 yrs female patient was presented with complaints of vomiting, loose stools, pain, fever, high grade chills, giddiness, loss of appetite acute renal failure, hemolytic anemia and thrombocytopenia She was diagnosed with Escherichia coli associated hemolytic-uremic syndrome and treated with plasmapheresis and other medications for 3 weeks. She recovered without sequelae.

Keywords: Hemolytic uremic syndrome; Atypical hemolytic uremic syndrome; Acute kidney injury; Shiga toxin-producing E.coli, Complement factor H, Therapeutic plasma exchange.

INTRODUCTION

Hemolytic uremic syndrome is a common cause of acute kidney injury. In children, and adults hemolytic uremic syndrome is most commonly associated with gastrointestinal infections caused by Shiga toxin-producing Escherichia coli or other enteric organisms [1][3]. Although less common, atypical hemolytic uremic syndrome is triggered by multiple factors and portends a significantly worse prognosis with a high rate of recurrence. Infections of the gastrointestinal tract (your stomach and intestines) are the most common cause of this disorder. Toxins released during an intestinal bacterial infection can destroy the lining of small blood vessels in your stomach or intestines[2]. This, in turn, causes damage and destruction to blood cells as they circulate through the blood vessels. This destruction affects red blood cells (RBC) and platelets, causing them to die prematurely. A buildup of these destroyed cells clogs the kidneys' filtering system, reducing the blood flow to the kidneys. Your kidneys are responsible for filtering out waste products and toxins so they can be eliminated from your body.

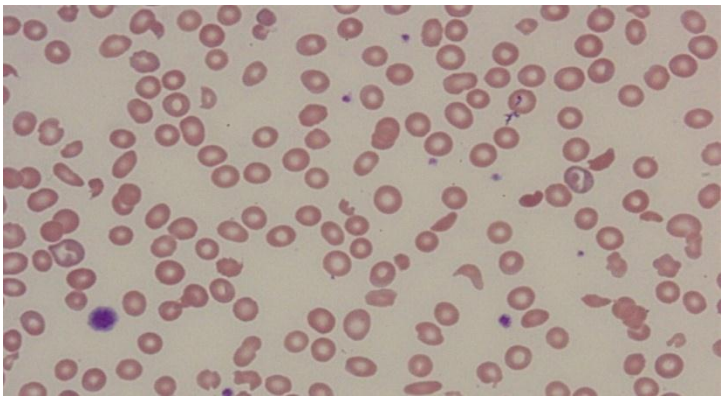
The damage to your health and kidney function can be quite serious if left untreated, or if complications arise. Kidney failure, high blood pressure, heart problems, and stroke are all concerns if HUS advances without prompt treatment.[4][5]

CASE STUDY

A 65 yrs female patient was presented with chief complaints of Vomiting since 2 days(several episodes/day), Loose stools since 2days(several episodes/day) Greenish Mucus, Abdominal Pain since 3days, Fever since 1 day, High grade chills, Giddiness on and off since 2 days, Loss of appetite since 3days. Medical history: k/c/o Type 2DM- 9yrs on OHA'S, Admitted with UTI in Feb.

medication history: Tablet glybovin 25mg 3-0-1 Tablet metadose ipr 850 mg social history: mixed diet, post-menopausal. Upon admission pulse rate was -110/min blood pressure was -90/60mm/hg respiratory rate was found to be -20/min and Temperature is -100F.

Laboratory tests showed Urea: 97 mg/dL (8-35mg/dl) Serum Creatine : 3.1 mg/dL (0.6-1.6mg/dl) , Na: 132 Meq/L (136-148Meq/L),K:>7Meq/L (3.5-5.0Meq/L), Random blood Sugar: 331 mg/dL (60-50mg/dl) Albumin: 4.0 g/dL (3.5-5g/dl), hemoglobin: 10.6 g%, ESR: 62(0-20mm/hr), platelet count 19,000/mm3, Culture Test:- E.coli in blood was identified in the stool culture performed at the hospital where she was first admitted



*Peripheral blood smear showed polychromasia, anisocytosis and schistocytes.

Microangiopathic hemolytic anemia

A diagnosis was made based on acute renal failure, hemolytic anemia, and thrombocytopenia and it was conformed as hemolytic uremic syndrome. She received hemodialysis for 10 days, and plasma exchange and fresh frozen plasma for two weeks.

The following medications was given in the stay of the hospital ivf ns@ 125ml/hr, h.insulin-r sc ½ hr bf

Table: 1 Treatment schedule

Drug	Dose	ROA	Frequency
Inj optineuron	1amp	IV	STAT
Inj ciprofloxacin	200mg	IV	BD
Inj pantoprazole	40mg	IV	OD
Inj ondansetron	4mg	IV	STAT
Inj piperacillin + tazobactam	4.5mg + 2.25mg	IV	STAT
Inj fursosemide	40mg	IV	STAT
Inj. pheniramine	1amp	IV	STAT
Inj paracetamol	1amp	IV	STAT
Inj hydrocortisone	100mg	IV	STAT
Inj haloperidol	2.5mg	IV	SOS
Tab quetiapine	25mg	PO	HS
Tab bisacodyl	10mg	PO	HS
Syp kmac	10ml	PO	TID

After day 16 three successive stool specimens resulted negative results for Escherichia coli. After the treatment platelet count increased to 1 lakh, hemoglobin raised to 14.5 g%, albumin to normal range, urea to 34mg/dl, k value to 4.0 meq/l, and serum creatinine 1.2mg/dl. On hospital day 18, she was discharged and received follow-up care as an outpatient.

DISCUSSION

The patient in this case presented with gastroenteritis symptoms with severe systemic upset. So the etiologic agent causing HUS this time is probably due to enteric pathogen, and the most likely candidate is E. Coli [6].The patho types of intestinal pathogenic Escherichia coli are classified as follows : Shiga toxin-producing Escherichia coli (STEC)/entero - hemorrhagic Escherichia coli (EHEC), enterotoxigenic Escherichia coli (ETEC), entero pathogenic Escherichia coli (EPEC), enteroinvasive Escherichia coli (EIEC), entero aggregative Escherichia coli (EAEC), and diffusely adherent Escherichia coli (DAEC). STEC/EHEC strains can cause hemorrhagic colitis and Hemolytic uremic syndrome (HUS). EHEC has since been documented as the cause of both large outbreaks and sporadic infections in the United States and around the world. Several large outbreaks resulted from the consumption of undercooked ground beef and other foods [7]. Our patient frequently ate fast food, particularly Chinese noodles. We could not exclude the possibility that EHEC was transmitted by contaminated food consumed by our patient. However, we did not investigate the origins of the EHEC infection Hemorrhagic colitis associated with EHEC and others is characterized by grossly bloody diarrhea, often with remarkably little fever or inflammatory exudate in the stool. Although the diarrheal illnesses have been self-limiting, a significant number of children and adults have subsequently developed a potentially fatal hemolytic uremic syndrome or thrombotic thrombocytopenic purpura[8]. The clinical manifestations of post-diarrheal HUS include renal failure, microangiopathic hemolytic anemia, and thrombocytopenia. Several studies reported that the culture rate of EHEC was 2% to 51% in post-diarrheal HUS. HUS complicates 10% of bloody diarrhea induced by EHEC. Some estimate that EHEC causes at least 70% of post-diarrheal HUS in the India and that 80% of these are caused by EHEC, and the other 20% by a different serotype of EHEC. However, EHEC infections are rare in patients with post-diarrheal HUS and the majorities are caused by non EHEC. The use of antimotility agents in children under 10 years of age or in elderly patients should be avoided as it increases the risk of HUS with EHEC infections. The incubation period has usually been 3 to 5 days after bloody diarrhea, but HUS infrequently develops no prodromal symptom [9]. Although the illness is usually self-limited, the mortality rate is 3% to 5% in young children and 30% of patients with HUS develop permanent renal failure, hypertension or neurologic sequelae.

Thrombocytopenia occurs as a consequence of platelet consumption and hemolytic anemia results from intravascular fibrin deposition, increased red blood cell fragility, and fragmentation. Plasma exchange, which removes the plasma with the shiga-like toxin and its breakdown products, may decrease the effects of the toxin. Plamapheresis may not be necessary in mild cases if water and electrolyte balance are well maintained. Fresh frozen plasma can be administered to the patient to replace the loss of plasma proteins and coagulation factors.[10]

CONCLUSION

This case highlights the clinical challenges in diagnosing and managing patients with hemolytic uremic syndrome. Because of similarity in symptoms, differentiating Shiga toxin-producing Escherichia coli associated hemolytic uremic syndrome and atypical hemolytic uremic syndrome can be challenging. However, because of the increased morbidity and mortality of atypical hemolytic uremic syndrome, early detection and initiation of therapy are critical. Providers must have a heightened suspicion in order to initiate supportive care or disease directed therapy in the case of atypical hemolytic uremic syndrome.

REFERENCES

[1] Scheiring J, Andreoli SP, Zimmerhackl LB: Treatment and outcome of Shiga-toxin-associated hemolytic uremia syndrome (HUS). Pediatr Nephrol 2008, 23:1749–1760.

[2] Michael M, Elliott EJ, Ridley GF, Hodson EM, Craig JC: Interventions for haemolytic uraemic syndrome and thrombotic thrombocytopenic purpura. Cochrane Rev 2009. doi:10.1002/14651858.CD003595.pub2. [http://thecochranereviewlibrary.com]

[3] Legendre CM, Licht C, Greenbaum LA, Babu S, Bedrosian C, Bingham C, Cohen DH, Delmas Y, Douglas K, Eitner F, Feldkamp T, Fougue D,Furman RR, Gaber O, Herthelius M, Hourmant M, Karpman D, Lebranchu Y, Mariat C, Menne J, Moulin B, Nurnberger J, Ogawa M, Remuzzi G,Richard T, Sberro-Soussari R, Severino B, Sheerin NS, Trivelli A, Zimmerhackl LB, et al: Terminal Complement inhibitor eculizumab in atypical Hemolytic-Uremia Syndrome. N Engl J Med 2013, 368:2169–2181.

[4] Noris M, Giuseppe R: Atypical hemolytic uremic syndrome. N Engl J Med 2009, 361:1676–1687.

[5] Gould LH, Bopp C, Strockbine N, Atkinson R, Baselski V, Body B, Carey R, Crandall C, Hurd S, Kaplan R, Neill M, Shea S, Somsel P, Tobin-D'Angelo M, Griffin PM, Gener-Smith P: Recommendations for diagnosis of Shiga Toxin producing Escherichia coli infections by clinical laboratories. MMWR 2009, 58:1–14.

[6] Fremeaux-Bacchi V, Fkahouri F, Garnier A, Bienaime' F, Drgaon-Durey M-A, Ngo S, Moulin B, Servais A, Provot F, Rostaing L, Burtey S, Niaudet P, Deschenes G, Lebrachu Y, Zuber J, Loirat C: Genetics and outcome of atypical hemolytic uremic syndrome: A nationwide French series comparing children and adults. Clin J Am Soc Nephrol 2013, 8:554–562.

[7] Hofer J, Janecke AR, Zimmerhackl LB, Riedl M, Rosales A, Giner T, Cortina G, Haindl CJ, Petzelberger B, Pawlik M, Jeller V, Vester U, Gadner B, van Husen M, Moritz ML, Wurzner R, Jungraithmayr T, German-Austrian HUS Study Group: Complement factor H-related protein 1 deficiency and factor H antibodies in pediatric patients with atypical hemolytic uremic syndrome. Clin J Am Soc Nephrol 2013, 8:407–415.

[8] Caprioli J, Castelletti F, Bucchioni S, Bettinaglio P, Bresin E, Pianette G, Gamba S, Brioschi S, Daina E, Remuzzi G, Noris M, International registry of Recurrent and Familial HUS/TTP: Complement factor H mutations and gene polymorphisms in haemolytic uraemic syndrome: the C-257T, the A2089G and the G2881T polymorphisms are strongly associated with the disease. Human Molecular Genetics 2003, 12:3385–3395.

[9] Kavanagh D, Goodship T: Genetics and complement in atypical HUS. Pediatr Nephrol 2010, 25:2431–2442.

[10] Bresin E, Rurali E, Caprioli J, Sanchez-Corral P, Fremeaux-Bacchi V,de Cordoba R, Pinto S, Goodship THJ, Alberti M, Ribes D, Valoti E, Remuzzi G, Noris M, on behalf of the European Working Party on Complement Genetics in Renal Disease: Combined complement gene mutations in atypical hemolytic uremic syndrome influence clinical phenotype. J Am Soc Nephrol 2013, 24:475–486.