

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 850mg

social history: mixed diet, post-menopausal.

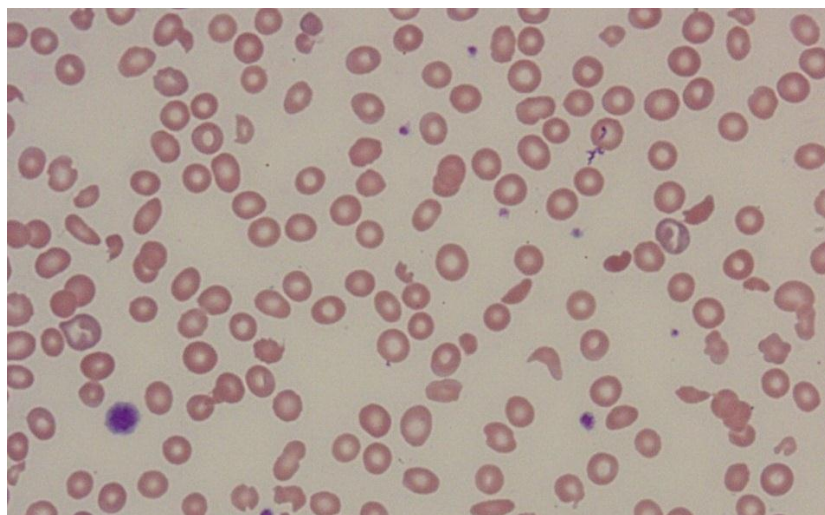
Upon admission pulse rate was -110/min
blood pressure was -90/60mm/hg respiratory

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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 (0.6-1.6 mg/dl), **Na:** 132 Meq/L (136-148Meq/L), **K:** >7 Meq/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.



[11]*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

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 furosemide	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 14.5g% ,albumin to normal range , urea to 34mg/dl, k value to 4.0meq/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 pathotypes of intestinal pathogenic Escherichia coli are classified as follows : Shiga toxin-producing Escherichia coli (STEC) / enterohemorrhagic Escherichia coli (EHEC), enterotoxigenic Escherichia coli (ETEC), enteropathogenic Escherichia coli (EPEC), enteroinvasive Escherichia coli (EIEC), enteroaggregative 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 majority 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. Plasmapheresis 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

In this case study a 65 yrs old patient was reported with hemolytic uremic syndrome which is life threatening disorder which can even lead to death .we conclude in this study that Hemolytic uremic syndrome is another

potential complication. It is rare and is usually associated with gastroenteritis caused by a certain type of E. coli infection. It is a condition where there is anemia, a low platelet count in the blood and kidney failure is well established, one of the common etiological factor observed

was Improper sanitation practices, Therefore advising the patient about Maintain good personnel hygiene and sanitary conditions its early detection, treatment followed by proper management can decrease the morbidity and mortality rate to a certain extent.

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11. Peripheral smear in hemolytic uremic syndrome, with findings of microangiopathic hemolytic anemia. Note schistocytes/helmet cells, as well as decrease in platelets. Image courtesy of Emma Z Du, MD, *N Engl J Med*. Jan 10 1985; 312(2):116-8.