Case Report:

Hemolytic uremic syndrome after acute gastroenteritis – treated conservatively

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Abstract:
A 60-year-old female presented to our department with gastroenteritis. She was found to have decreased platelets and deranged renal function. A diagnosis of hemolytic uremic syndrome was eventually made. The disease usually presents nonspecifically with gastroenteric symptoms and systemic upset at the initial stage. Without a high index of suspicion, the diagnosis can easily be missed. Patient was treated conservatively and successfully without plasmapheresis.

Keywords: E. Coli O157:H7, gastroenteritis, Hemolytic Uremic Syndrome, plasmapheresis, verocytotoxin

Introduction
Hemolytic Uremic Syndrome (HUS) is a life threatening condition. It is characterized by haemolysis, thrombocytopenia, and acute renal failure. Organs including the brain, intestine, pancreas, heart, and lungs may be affected. It is frequently associated with gastroenteritis caused by E. Coli O157:H7 infection.

Case Presentation
60 year old female patient presented to ICU with complaints of loose motions about 3 episodes in last 12 hours which were watery in nature not associated with blood and was associated with mild pain in abdomen which was diffuse in nature not accompanied by vomiting. Patient had signs of mild dehydration and her look was normal. Systemic and general examination didn’t reveal any significant abnormality. Patient was admitted in general ward and was treated as a case of acute gastroenteritis. Patient was normal throughout the day.

On next day morning patient started with more severe pain in abdomen which was diffuse in nature and was associated with 5-6 loose stools all were associated with fresh blood but her vitals were stable throughout the day. Her routine investigations were sent to laboratory. All her investigations were normal with mildly deranged renal function test with serum creatinine level 1.3mg% and blood urea level 46mg%. All other investigations were normal.

On day 3 patient started with sudden onset breathlessness with acidotic breathing. Patient had tachycardia, tachypnea at that time. Her look was toxic and was severely dehydrated. She had complaints of decreased urine output in last 12 hrs and development of icterus. Patient was then shifted to intensive care unit. All her investigations were repeated immediately including stool culture. Her hemogram showed fragmented RBCs on peripheral smear with leucocytosis and reduced platelet count of 78,000cu/mm. Her hemoglobin was 6.4 gm%. Her packed cell volume was reduced to 20%. Arterial blood gas analysis was suggestive of severe metabolic acidosis with reduced anion gap. Her renal
function tests were deranged with serum creatinine 4.4mg% and blood urea level 160mg%.

Her stool came positive for Escherichia coli. The strain of E.coli was not traced out. In accordance to history and progression patient was labelled as a case of hemolytic uremic syndrome. After conformation of diagnosis, all antibiotics on which patient was, were hold on. Central venous pressure guided fluid were given to patient through central line and correction of acidosis was done by infusion of bicarbonates. Packed red blood cells were given to counteract anemia due to hemolysis. No additional platelets were given to patient. Patient was managed conservatively with improvement of signs and symptoms. No further clinical deterioration occurred. Over the period of next 4 days patient had improvement in hematocrit with correction of anemia and normalization of platelet count. Her renal function tests were normal after 4 days. Her arterial blood gas analysis was normal. Patient was then discharged to home after total stay in hospital for 7 days.

Discussion
HUS most commonly occur after colitis resulting from E. ColiO157:H7 infection, but it may also emerge after malignant hypertension, scleroderma, systemic lupus erythematosus, chemotherapy, radiation, HIVinfection, and eclampsia.(2,3) E.ColiO157:H7 bacteria are usually ingested through contaminated food or water, but they can also be transmitted person to person through the faecal-oral route.

Most strains of E. Coli are harmless, but the O157:H7 strain produces a powerful toxin that can cause severe diarrhoea, abdominal cramping, nausea, and vomiting. Though illness usually resolves completely in 5 to 10 days, it may progress to haemorrhagic colitis and HUS, especially in young children and the elderly. HUS is similar to thrombotic thrombocytopenic purpura (TTP), a disease that also causes micro-angiopathic haemolytic anaemia. But whereas HUS is most common in children, and is almost always associated with an E. Coli O157:H7 infection resulting in diarrhoea and renal failure, TTP is most common in adults and usually involves the neurologic system, although the kidneys are sometimes affected. Because HUS and TTP are so similar, some experts suggest that the two are actually the same syndrome with different manifestations.(4,5)

The patient in this case presented with gastroenteritis symptoms with severe systemic upset. So the aetiologic agent causing HUS in this case was E. Coli most probably O 157:H7 strain. It can produce a verocytotoxin that cause haemorrhagic colitis. The presence of bloody diarrhoea in the history made the picture typical. When E. Coli O157:H7 is suspected, a specific enrichment culture should be ordered to isolate the bacteria. The sample should also be collected as early as possible because the culture would be negative 6 days after the onset of diarrhoea. So the positive stool culture in this case is expected because the specimen was collected early in the course of the disease. Verocytotoxin targets the endothelial cells in the kidney and other organs with glycoprotein genotype Gb3 receptor.(6) Children under the age 2 have more of this kind of receptor and therefore are more prone for renal failure caused by HUS.(7)

When endothelial cells of the kidney are attacked by verocytotoxin, protein synthesis will be inhibited and the cells will die. An inflammatory process will be induced and the glomerular capillary lumens become narrowed. This
will lead to destruction of platelets and RBCs as they squeeze through the lumen. There will also be platelet activation and clotting, causing further depletion of platelets.(3)

The damaged platelets and RBCs would then be removed by the spleen resulting in decrease in platelet count, haemoglobin, haematocrit and RBC count. LDH, conjugated and unconjugated bilirubin levels will be raised as a result of hemolysis. The narrowing of the glomerular lumens cause the decrease in glomerular filtration rate. Urine output decreases while BUN and serum creatinine levels rise.(7)

Plamapheresis and corticosteroids have been proven to be effective in the treatment of haemolytic uraemic syndrome.(7,8) 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. When there is a suspicion of E. Coli O157:H7 infection, antimotility agents and antibiotics should be withheld. Antimotility agents can slow the transit time of the bacteria in the intestine and cause increased toxin absorption. Antibiotics may destroy the normal bowel flora, leading to overgrowth of E. Coli. They may also lyse the E. Coli organisms, causing additional toxins to be released.1,8

**Conclusion:**
Diagnosing hemolytic uremic syndrome requires high index of suspicion. If acute gastroenteritis patient develops acidic breathing with deranged renal function test and hemolysis, hemolytic uremic syndrome should be suspected and treated accordingly. Conservative management in such patients can be life saving and plasmapheresis can be avoided.

**Conflict of interest:** none

**References**