

# Case study

## Case Report on Hemolytic Uremic Syndrome

### ABSTRACT:-

#### Background:

In the United States, the hemolytic–uremic syndrome of childhood typically follows gastrointestinal infection with *Escherichia coli* O157:H7. It is presumed that the absorption from the gastrointestinal tract of Shiga toxins 1, 2, or both (formerly called Shiga-like toxins) produced by *E. coli* O157:H7 causes microangiopathic hemolytic anemia as a result of endothelial-cell injury. Shiga-toxin–producing *E. coli* belonging to serotypes other than O157:H7 can also cause the hemolytic–uremic syndrome. However, even though such organisms have been implicated as causes of sporadic cases or outbreaks of gastroenteritis, they are not believed to be important causes of the hemolytic–uremic syndrome in this country. Hemolytic uremic syndrome (HUS) is a kidney disease characterized by damaged and inflamed small blood vessels. As a result of the damage, clots in the vessels may form. Clots impede the filtration function of the kidneys, causing renal failure, which can be deadly.

**Clinical Finding:** Hemolytic uremic syndrome, nephropathy and thrombocytopenia, pallor, jaundice, edema. A previously healthy six-year-old girl was seen after eight hours of abdominal and left-flank pain, vomiting, and dysuria. Constipation had been treated by enema on the preceding day. The child had not had diarrhoea during the two weeks before evaluation. On examination, the patient had an oral temperature of 39.8°C, a pulse rate of 120 per minute, and a blood pressure of 111/59 mm Hg. There was tenderness of the suprapubic area and left costovertebral angle. The white-cell count was 23,400 per cubic millimeter (58 percent neutrophils, 23 percent band forms, 9 percent lymphocytes, and 10 percent monocytes), the hematocrit was 40 percent, and the platelet count was 293,000 per cubic millimeter. Urine obtained by catheterization had a specific gravity of 1.028 and a pH of 5, and dipstick analysis was positive for leukocyte esterase (++) , protein (++) , ketones (++) , and blood (+++). The urinary sediment contained 21 to 100 red cells, more than 100 white cells, 1 to 5 renal tubular cells, and 1 to 5 granular casts per high-power field. Many bacteria were observed by microscopy. Abdominal ultrasonography demonstrated attenuation of fat and a probable collection of fluid around the left kidney; the right kidney was normal. Blood was obtained for culture before treatment with ceftriaxone was begun.

**Diagnostic Evaluation:** HB-5.7 g/dL, total protein-7.4 g/dL, total Alkaline phosphatase -123 U/L , total platelet count-2,93,000 per cubic millimeter, total bilirubin-3.2 mg/dl, WBC 23400 per cubic meter, CRP 5.58 mg/L, Sodium level 136 mg/dl, potassium level 3.92mg/dl.

**Therapeutic Intervention** – Tab Amlodepin 10 mg BD, Tab Prednisolone 10 mg TDS, Tab Envas 2.5 mg OD, Metropenum 340 mg BD, Inj. Pantop 20 mg OD.

**Outcome**-The medication has started for thrombocytopenia, the patient is on dialysis and If is given to patient for movement of body because of patient is obese. The patient was sent home after 20 days of hospitalization. A voiding cystourethrogram was normal three weeks later, and ultrasonography six weeks after discharge demonstrated that the focal abnormality in the left kidney had resolved. As of this writing, two years later, the patient has normal blood urea nitrogen and serum creatinine concentrations, iothalamate clearance, and blood pressure, and her urine is free of protein and blood. She has not had a recurrence of either a urinary tract infection or the hemolytic–uremic syndrome.

**Conclusion**- The atypical hemolytic uremic syndrome is a rare disease entity requiring a high index of suspicion to diagnose. It is a diagnosis of exclusion. Early diagnosis with prompt treatment will render a better outcome. The atypical hemolytic uremic syndrome needs to be considered in all patients with thrombotic microangiopathy.

**Keywords**- HUS, Thrombocytopenia, Uremia, Acute Kidney Failure, E. coli 0157:H7 variant

### **Introduction:**

HUS is defined by low platelets, acute renal failure and low red blood cell. It presents clinically with: 1. Bloody diarrhoea, fever, vomiting, and weakness. 2. As the diarrhoea progresses, kidney failure and low platelets become more prevalent. 3. Adults may have worse outcomes than children, despite the fact that children are more typically affected. The causative agents to HUS include E. coli (0157:H7 variant), Salmonella, Shigella, and S. pneumonia and certain medications. The bacteria's synthesis of Shiga toxin is usually the underlying mechanism. 4. It manifests itself in a distinctive manner. Thrombotic microangiopathy is a condition that results in inflammation and blood clots in the small blood vessels. 5. HUS is caused by eating bacteria that generate Shiga toxin, such as enterohemorrhagic Escherichia coli (EHEC), the most frequent of which being E. coli O157:H. 6. STEC- a frequent strain serotype that is not typical Hemolytic uremic syndrome; (HUS) is a kind of hemolytic uremic disease that affects the kidneys. 7. The initial signs of illness might emerge anywhere from 1 to 10 days after eating infected food, although they usually appear 3 to 4 days after that. 8. Bloody diarrhoea, stomach pains, a mild fever, or dehydration from vomiting are some of the early signs and symptoms. HUS generally shows 5–10 days after the first symptoms, although it might take up to a month. 9. The diarrhoea is progressively improving, and the tiredness is decreasing. Kidney failure can be identified by reduced urine output, blood in the urine, and renal failure In HUS, there is reduced platelet count(i.e., the blood component required for blood coagulation) and red blood cell haemolysis (destruction of red blood cells occasioned by microangiopathic haemolytic anaemia).Jaundice (a yellow tint to the complexion) causes seizures, elevated blood pressure, and skin bleeding. 10. There are noticeable neurologic abnormalities in certain situations.

**Clinical Historical:**

A 6-year-old female patient was hospitalized to AVBRH in February 2021 with pallor, edema, hypertension, nausea, and vomiting, and her relative stated that she was unable to walk due to obesity. Hemolytic uremic syndrome was the subject of some inquiry.

**Family history:** She has 3 siblings and there was no family history of similar illness

**Past history:** Abdominal and left-flank pain, vomiting, and dysuria. Constipation had been treated by enema on the preceding day. The child had not had diarrhoea during the two weeks before evaluation.

**Clinical finding:** Haemolytic anaemia, nephropathy, thrombocytopenia.

**Etiology:**

The most common cause of hemolytic uremic syndrome in children is an infection of the digestive system with *Escherichia coli* (*E. coli*). The gastrointestinal tract, often known as the GI tract, is a series of tubes that runs through the digestive system consists of hollow organs connected in a long, twisting tube from the mouth to the anus—along with other organs that help in food digestion and absorption. *E. coli* strains are typically harmless are common in the intestines and play an important role in digesting. If a child is infected, however, the germs will become lodged in the digestive tract. Toxins are produced, which can enter the circulation. Toxins pass through the circulation and have the potential to kill red blood cells. *E. coli* O157:H7 can be found in raw, unpasteurized ground beef, unwashed milk, and infected raw fruits and vegetables, polluted juice, contaminated lakes or swimming pools.

**On physical examination of the patient,** there was abdominal pain, edema, and un-coordinated movement.

**Laboratory assessment revealed** HB-5.7 g/dL, total protein-7.4 g/dL, total Alkaline phosphatase 123 U/L, total platelet count- 2,93,000 per cubic millimetre, total bilirubin-3.2 mg/dL.

**Therapeutic Interventions were Per oral:** Amlodipin 10 mg BD, per oral Prednisolone 10mg TDS, per oral Envas 2.5 mg OD, Parenteral Metropenum 340 mg BD and parenteral Pantop 20 mg OD.

**Outcome-** The medication was started for thrombocytopenia, the patient is on dialysis and physiotherapy is given to patient for movement of body because of patient is obese.

## Discussion:

Kidney problem may develop when the diarrhoeal disease progresses in HUS. Despite the fact that children are more commonly affected, adults may have poorer results than children. Two other possible consequences of HUS include neurological problems and cardiac failure. 11. The majority of cases are caused by the infectious diarrhoea-causing E. coli strain O157:H7.4, S. pneumoniae, Shigella, Salmonella, and certain medications. 12. When a Shiga toxin-producing bacteria is found, it is called a Shiga toxin-producing bacterium. A genetic mutation causes atypical hemolytic uremic syndrome (aHUS), which can manifest itself in many ways. 5 A disease known as thrombotic microangiopathy occurs when tiny blood arteries become irritated and blocked with blood clots. It might take anywhere from one to 10 days, although it generally happens within three to four days. Dehydration and reduced urine output owing to diarrhoea are the early signs.s

**Conclusion** - The atypical hemolytic uremic syndrome is a rare disease entity requiring a high index of suspicion to diagnose. It is a diagnosis of exclusion. Early diagnosis with prompt treatment will render a better outcome. The atypical hemolytic uremic syndrome needs to be considered in all patients with thrombotic microangiopathy.

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