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Case Report on Hemolytic Uremic Syndrome

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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Case Study

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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. After eight hours of stomach and left-flank pain, vomiting, and dysuria, a previously healthy six-year-old girl was seen. The day before, an enema had been used to alleviate constipation. 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

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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. The left kidney showed attenuation of fat and a possible collection of fluid on abdominal ultrasonography; the right kidney was normal. Before starting ceftriaxone medication, blood was taken for culture.

Diagnostic Evaluation: HB-5.7 g/dL, total protein-7.4 g/dL, total Alcaline 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. Three weeks later, a voiding cystourethrogram was normal, and six weeks later, ultrasonography revealed that the focal abnormality in the left kidney had disappeared. The patient's blood urea nitrogen and serum creatinine concentrations, iothalamate clearance, and blood pressure are all normal two years later, and her urine is protein and blood-free. She has not experienced a recurrence of a urinary tract infection or hemolytic–uremic syndrome.

Conclusion: The atypical hemolytic uremic syndrome is an uncommon condition that must be diagnosed with a high index of suspicion. It's an exclusionary diagnosis. A better outcome will be achieved if the disease is detected early and treated promptly. In all patients with thrombotic microangiopathy, the atypical hemolytic uremic syndrome should be evaluated.

Keywords: HUS, thrombocytopenia; ureamia; acute kidney failure; E. coli 0157:H7 variant.

1. 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 abnormalities neurologic in certain situations.

1.1 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.

1.2 Family History

She has 3 siblings and there was no family history of similar illness.

1.3 Past History

Pain in the abdomen and left side, vomiting, and dysuria. The day before, an enema had been used to alleviate constipation. During the two weeks prior to the evaluation, the youngster had not had diarrhoea.

1.4 Clinical Finding

Haemolytic anaemia, nephropathy, thrombocytopenia.

2. ETIOLOGY

Infection of the digestive system with Escherichia coli (E. coli) is the most prevalent cause of hemolytic uremic syndrome in children. 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-co-ordinated movement.

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

2.1 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.

2.2 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.

3. 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, pneumoniae, Shigella, S. Salmonella, and certain medications [12,13]. 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

4. CONCLUSION

The atypical hemolytic uremic syndrome is a rare disease entity that must be diagnosed with a high level of suspicion. It is an exclusionary diagnosis. Early detection and treatment will result in a better outcome. All patients with thrombotic microangiopathy should be evaluated for atypical hemolytic uremic syndrome.

CONSENT

As per international standard or university standard, patients' written consent has been collected and preserved by the author(s).

ETHICAL APPROVAL

As per international standard or university standard written ethical approval has been collected and preserved by the author(s).

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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