Enterohemorrhagic colitis with disseminated intravascular coagulation

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ABSTRACT

Gastroenteritis is a common illness in the pediatric population. Clinicians must be able to recognize severe disease in the milieu of common presentations. All forms of gastroenteritis can lead to significant dehydration and sequelae when considering causes. However, severe complications can occur with certain bacterial etiologies. We present a case of pediatric enterohemorrhagic colitis leading to disseminated intravascular coagulation and patient demise.

KEYWORDS Disseminated intravascular coagulation; enterohemorrhagic *Escherichia coli*; hemolytic uremic syndrome; Shiga toxin–producing *Escherichia coli*

Most enterohemorrhagic *Escherichia coli* illnesses, also referred to as Shiga toxin–producing *E. coli* (STEC), are self-limited. In some cases, further supportive care is indicated. STEC leads to hemolytic uremic syndrome (HUS) in 15% of children under the age of 10.^{1–4} A small percentage of STEC HUS cases lead to mortality. This case discusses a patient who died within 12 hours of presentation to the hospital.

CASE DESCRIPTION

A 2-year-old girl, up-to-date with immunizations with no past medical history, presented to the emergency department by ambulance. She had multiple episodes of diarrhea over the prior 3 days and one episode of vomiting. One day prior, the patient became more lethargic and her stools became darker. The family had given acetaminophen and loperamide at home. An ambulance was called for tonic-clonic seizure activity. Upon their arrival, the paramedics noted mottled skin and minimal responsiveness. Seizures were intermittent and less than 5 minutes each. A saline bolus was given intraosseously for systolic blood pressure in the 60s.

When the patient arrived, her temperature was 99.4°F (axillary); heart rate, 163 beats/min; blood pressure, 77/55 mm Hg; respiratory rate, 29 breaths/min; and pulse oximetry, 100% on nonrebreathing mask. Her blood glucose level was 119 mg/dL. Other laboratory results are shown in *Table 1*. She appeared toxic. Mucous membranes were moist. Both eyes deviated leftward. Her left pupil was nonreactive at 4 mm, and her right pupil was minimally reactive at 3 mm. On inspiration, there

was decreased air movement and poor chest rise. The abdomen was soft and nondistended, and feces was guaiac positive. She had a Glasgow Coma Scale score of 4, increased tone in all extremities, and seizures that were more prominent on the right than the left. Her skin was warm, with a capillary refill of 3 to 5 seconds and with no petechiae or rash.

Computed tomography of the head, chest, and abdomen showed no acute findings in the head. There was consolidation of the right lung with a mediastinum shift to the right. A bowel loop with wall thickening ran from the right upper quadrant to the left upper quadrant.

Lorazepam, fosphenytoin, and further fluid boluses were given with improvement of hemodynamics. The patient was intubated for airway protection and decreased respiratory drive and was admitted to the intensive care unit. Later that day she went into ventricular fibrillation with return of spontaneous circulation after 9 minutes of resuscitation.

A stool polymerase chain reaction assay came back positive for STEC. Due to a worsening abdominal examination, an emergency bedside exploratory laparotomy was performed that found an ischemic and necrotic colon that required total colonic resection. After the arrest, her pupils were fixed and dilated. There was no movement or response to pain. Repeat computed tomography of the head revealed interval development of venous sinus thromboses and bilateral frontal lobe parenchymal hemorrhage along with diffuse cerebral edema. A marked worsening of pulmonary compliance and oxygenation occurred. Physical examination was consistent with brain death >24 hours from arrest. The family elected to withdraw care.

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Table 1. Laboratory results

Test	Result
White blood cell count ($\times 10^9/L$)	41.8
Hemoglobin (g/dL)	10.0
Platelets ($\times 10^9/L$)	45
Blood urea nitrogen (mg/dL)	70
Creatinine (mg/dL)	1.03
Sodium (mEq/L)	130
Potassium (mEq/L, hemolyzed)	6.5
Carbon dioxide (mEq/L)	15
Calcium (mg/dL)	6.0
Albumin (g/dL)	1.5
Prothrombin time (s)	20.0
International normalized ratio	1.7
D-dimer	16.45
Fibrin monomer	Positive
Fibrin split products ($\mu g/mL$)	>20
Lactic acid (mmol/L)	5.6
Venous pH	7.07
Venous pCO_2 (mm Hg)	64
Venous HCO_3 (mmol/L)	18
Venous BE (mmol/L)	10.7

pCO_2 indicates partial pressure of carbon dioxide; HCO_3 bicarbonate; BE, base excess.

DISCUSSION

STEC infection initially starts as a nonbloody diarrhea, and about 90% of children develop bloody diarrhea between day 2 and day 3. HUS includes microangiopathic hemolytic anemia, thrombocytopenia, and acute kidney injury. It develops in about 15% of children with STEC.⁴ A study from 1997 to 2004 showed that the rate of in-hospital death was 2.9%.⁵ Causes of death in HUS are secondary to central nervous system injury, hyperkalemia, coagulopathy, sepsis, heart failure, pulmonary hemorrhage, and injury to the gastrointestinal tract.^{5,6} Our case is unique considering the component of disseminated intravascular coagulation that led to rapid onset of bowel ischemia and venous sinus thromboses.

In general, long-term sequelae are due to kidney injury. Patients at risk for this will often have the following during the acute phase of the disease: white blood cell count $>20,000$ per mm^3 ; initial oliguria (>10 days) or anuria (>5 days); renal histology with glomerular microangiopathy affecting $>50\%$ of glomeruli, arterial microangiopathy, and/or cortical necrosis.^{7–11}

There seems to be no certain way to prevent progression to HUS. However, it is recommended to not treat the infection with antibiotics and antimotility agents, which may put children at increased risk of HUS. Early intravenous fluids may limit renal hypoperfusion and reduce the risk of acute kidney injury.

Special attention should be paid to anemia. Red blood cell transfusion is indicated at hemoglobin levels <6 g/dL with a goal of 8 to 9 g/dL.¹² Platelet transfusion is rarely necessary in HUS unless there is significant bleeding or invasive procedures must be performed when platelets are $<30,000/mm^3$.⁴ Electrolyte imbalances should be closely evaluated and treated. Specifically, hyperkalemia, hyperphosphatemia, and metabolic acidosis should be monitored. It is essential to renally dose medications and use dialysis as indicated. Plasma exchange has been utilized with central nervous system involvement, but the 2011 *E. coli* O104:H4 outbreak showed no benefit with plasmapheresis.¹³

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