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An Unprovoked Haemolytic-Uremic Syndrome; A Unique Case Report

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Abstract

Haemolytic-uremic syndrome is a rare but serious condition, which is featured by the triad of: acute renal failure, microangiopathic haemolytic anemia and thrombocytopenia associating with viral or bacterial etc. etiologies. However, we present an unprovoked case of HUS in a 2-year-old male case.

Keywords: Case Report, Hemolytic-Uremic Syndrome

Introduction

Hemolytic Uremic Syndrome (HUS) is a rare life-threatening disorder. It is recognized by hemolysis, thrombocytopenia, and acute renal failure. Organs comprising the brain, intestines, pancreas, heart, and lungs might be influenced and damaged during the condition, [1–4]. It is frequently precipitated by gastroenteritis but provoked by bacterial or viral infection especially in pediatric cases. An etiological review of previous reported cases with HUS will be given in the following as well [3-5]. Even if we dedicated attention on HUS risk related with bacterial/viral infections, we identified a 2-year-old male baby compatible with HUS features, most probably without any potential known endo/exogenous triggers whose interesting report will be narrated as below.

Case Presentation

We are reporting the case of a 2-year-old male Caucasian baby who was hospitalized with 8 days history acute severe watery diarrhea gradually reduced into a bloody low-volume diarrhea, accompanied with vomiting, intermittent fever and weakened general condition but no coriza symptoms; unresponsive to home-administered medication-ibuprofen, acetaminophen except for fever. He was born from a consanguineous marriage with no significant past medical history. At the time of admission, his physical examination revealed pale skin, dry lips, decreased skin turgor, no pharyngeal hyperemia, no budging fontanel, respiratory rate of 28 breaths per minute, blood pressure (BP): 67 / 50mmHg, heart rate (HR) of 122 beats per minute (bpm), food refusal, and bloody stools. No lymphadenopathy nor neurologic sings were detected neither. Laboratory tests showed the following results as: leukocytosis: 12400 (Poly: 65%), hemoglobin: 9.2g/L, hematocrit 32%, BUN: 128 mg/dl, Cr: 4.4mg/dl and Normal hepatic tests on admission. In progress: persistent leukocytosis of 20430 with Poly of 63%, decreasing anemia 9.2–10.1g/L, lactate dehydrogenase 5834U/L (references value = 615U/L), changes indicative for an acute renal failure-blood urea nitrogen 128 to 154mg/dL, creatinine 4.10 to 4.72mg/dL, hepatocytolysis, gamma-glutamyl transferase (gGT) 135U/L(references value 0–39U/L), and a C-reactive protein of 17mg/ L (references value 0–10mg/L). The complement tests and ANA and anti-ds DNA were: C3: 0.68, C4: 0.11, CH50: 16; Anti-ds – DNA: 3.4, ANA: 1.2.his hepatitis Ag tests and ant phospholipid tests were negative. His peripheral blood smear showed poikilocytosis, anisocytosis, and schizocytes.

Stool examinations for bacterial organisms or relevant antigens was not positive for Salmonella, Shigella, Campylobacter, Yersinia, E coli 0157, enterohemorrhagic E coli; virulence molecular markers for verocytotoxin-producing E coli (VTEC/vtx1, vtx2), enteropathogenic E coli (EPEC/eae), enterotoxigenic E coli (ETEC elt, est) and enteroinvasive E coli (EIEC/ipaH) were also negative. Repeated check of stool sample for viruses including rotavirus, adenovirus and norovirus were done; this was also unremarkable. Other systemic examinations and imagings were negative for any pathologic evidence as well. while hospitalization for appropriate monitoring of urine output, catheterization was implemented,

observing oligoanuria with a volume of 12mL urine/24 hours under treatment with serum mannitol and lasix. Naturally, there was an augmented nitrogen retention, and anemia related with thrombocytopenia which was improved with tight control of hydration. The patient needed cardiovascular watching one month after his discharge. His renal function became adjusted on discharge.

Discussion

HUS in pediatric is mainly triggered by STEC O157, followed by O26. Since 2010, it was told that 2350 cases initiated by STEC O26 till 2014, [5-7] with a peak in 2015, respectively, 463 cases. In the United States, the incidence of HUS in children under six years is of 6.1/100,000 population/year. In 2012, 274 cases associated with diarrhea were described [1,8]. Other potential etiologies of HUS are: other bacterial infections (Salmonella, Shigella dysenteriae, Campylobacter, Streptococcus pneumoniae, Mycoplasma, Legionella, etc.), viral infections (adenoviruses, enteroviruses, HIV, Epstein Barr virus, herpes simplex, Portillo virus, etc.), but there are also noninfectious causes: as the advance of a neoplasia (pancreatic, gastric, and prostate cancer), drug-induced (anticancer medication, quinine, antiplatelet drugs, contraceptives), post-transplantation (bone marrow, lung, kidney, etc.) and during pregnancy [9] or linked with the antiphospholipid syndrome, systemic lupus erythematosus or hierarchy origins with autosomal dominant or recessive heritage.

Regarding pathophysiology, HUS is the result of endothelial damage, ordinarily being an outcome of the effect of Shiga toxin which attaches to the cell membrane receptor, [1,10] and other infectious or noninfectious triggers, with the release of vasoactive affluences, glomerular microangiopathy and of small renal arterioles [11]. Acute renal failure in the advancement of HUS is correctable in 85% of cases under supportive care. The risk factors for the development into HUS are the age of more the five years old, different etiologies from STEC, insistent oligoanuria, glomerular impairment and central nervous system (80%). The combined leukocytosis and fever characterize an augmented threat for Hemolytic uremic syndrome. HUS is blamable for seven percentage of cases with hypertension in infants, being the principal cause in chronic renal derangement in pediatric, and a main cause of important kidney injury in adults. This current case is unique as no established potential etiology was found for HUS with occurrence of loose stools which could have masked diminished urine output and resulted in acute renal failure, linking other harsh prognostic features such as leukocytosis, fever and non-STEC etiology. Observing renal function, blood pressure, and heart rate will be obligatory afterward.

Conclusion

Although the most common etiology of HUS remains STEC, other etiologies like viral causes should not be neglected, keeping in mind the fact that there might be further unrecognized etiologies responsible for developing HUS.

Declaration

An informed consent was obtained from patients' parents; there is no conflict of interest regarding submission of manuscript.

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