Rev Argent Microbiol. 2014;**46(2)**:103-106

REVISTA ARGENTINA DE MICROBIOLOGÍA

www.elsevier.es/ram

BRIEF REPORT

Hemolytic uremic syndrome with mild renal involvement due to Shiga toxin-producing *Escherichia coli* (STEC) O145 strain

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Received 12 February 2014; accepted 7 April 2014

KEYWORDS
HUS;
Non-O157 STEC;
Renal failure;
Schizocytes

Abstract
Hemolytic uremic syndrome (HUS) is a disorder characterized by the presence of the classic triad: microangiopathic hemolytic anemia, thrombocytopenia and acute renal injury. HUS without acute renal failure can be confused with other hematologic diseases. An infantile HUS caused by a Shiga-toxin-producing *Escherichia coli* (STEC) O145 strain carrying genotype *stx*2, *ehxA*, *eae* subtype β1 is herein reported. The infant did not require dialysis during the acute stage of HUS, evolved favorably, maintained normal blood pressure and normal renal function and had no recurrence until the last control. This could be due to several factors, such as the characteristics of infecting STEC strain and a reduction in host susceptibility to renal injury. This report highlights the regional participation of non-O157 STEC in childhood diseases and the importance of performing active surveillance for all forms of HUS.

PALABRAS CLAVE
SUH;
STEC no-O157;
Falla renal;
Esquizocitos

Síndrome urémico hemolítico con compromiso renal leve debido a una cepa de *Escherichia coli* productora de toxina Shiga [*Shiga-toxin-producing Escherichia coli* (STEC)] O145

Resumen
El síndrome urémico hemolítico (SUH) es una afección caracterizada por la presencia de la tríada clásica: anemia hemolítica microangiopática, trombocitopenia y compromiso renal agudo. Los casos de SUH sin insuficiencia renal pueden confundirse con otras enfermedades hematológicas. Presentamos un caso de SUH pediátrico causado por una cepa de *Escherichia coli* productora de toxina Shiga [*Shiga-toxin-producing Escherichia coli* (STEC)] O145 con el genotipo *stx*2, *ehxA*, *eae* subtipo β1. El niño no requirió diálisis

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durante la etapa aguda del SUH, evolucionó favorablemente y no tuvo recurrencias hasta el último control; además, mantuvo cifras normales de presión arterial y función renal normal. Esto puede deberse a varios factores: características de la cepa STEC infectante y susceptibilidad del hospedero al daño renal, entre otros. Este hallazgo destaca la participación regional de STEC no-O157 en enfermedades de la infancia y la importancia de realizar una vigilancia activa de todas las formas de SUH.

Hemolytic uremic syndrome (HUS) is a disorder characterized by the presence of the classic triad: microangiopathic hemolytic anemia, thrombocytopenia, and acute renal injury[8,10]. There are 2 forms of HUS: typical (with diarrhea in the prodromal period) and atypical (without diarrhea in that period). In addition, HUS may be "complete" (complete triad: microangiopathic hemolytic anemia, thrombocytopenia, and acute renal involvement) or "incomplete" (when the patient does not have any of the manifestations of the classical triad). The typical one, also known as post-enteric HUS or STEC-HUS is the most common, being responsible for 90-95% of cases and occuring after intestinal infection by strains of Shiga-toxin-producing *Escherichia coli* (STEC). It occurs mainly in children under 5 years of age and is usually preceded by episodes of bloody diarrhea or hemorrhagic colitis. STEC-HUS is the leading cause of acute renal failure and the second leading cause of chronic renal failure and renal transplantation in children under 5 years of age[10].

Sometimes, HUS cases present thrombocytopenia and anemia without acute renal failure, making diagnosis difficult[4].

We here describe a case of a child with HUS, with the aim of alerting healthcare professionals, especially pediatricians, hematologists, nephrologists and microbiologists in this regard,

On January 16, 2013 a 1 year-old infant from "Las Piedras" (in the urban area of Canelones Department, Uruguay) was brought to the emergency room with vomiting and fever. During the preceding 48 hours, he had fever without diarrhea and had vomited on several occasions. On examination, he was febrile (38.5 ºC axillary), depressed, with intense mucocutaneous pallor and upper right foot petechiae. He was well hydrated and perfused with a heart rate of 150 cpm, no murmurs and PA records of 110/86 Hg mm. Edema was not present, and the remaining examination was normal. Hematological investigations revealed: RBC: 2,200,000/μl; WBC: 24,500/μl; neutrophils: 36%; lymphocytes: 44%; hemoglobin: 6.6 g/dl; MCV: 75 fL; hematocrit: 18.7%; platelet count: 54,000/μl. Biochemical analysis displayed the following values: azotemia: 0.51 g/l; creatinine: 0.47 mg/dl (estimates of glomerular filtration: 99 ml/min per 1.73 m^2); serum sodium, 143 mEq/l and serum potassium, 4.1 mEq/l. The urine analysis contained 20 erythrocytes per field with hyaline cylinders and showed proteinuria, intense hemoglobinuria and ketonuria.

The infant was admitted to a pediatric ward with a diagnosis of hemolytic anemia. He remained hemodynamically stable with tachycardia and preserved diuresis. He was examined by a hemato-oncologist, who performed a new peripheral blood count and a peripheral blood smear, showing: Hb 5.2 g/dl, RBC 2,000,000/μl, WBC 22,400/μl, reticulocytes 5%, platelets 71,000/μl and 5-10 fragmented erythrocytes (schizocytes) every 40 erythrocytes, respectively (Fig. 1) With these results, HUS without renal failure was diagnosed.

The stool sample collected on January 17th was sent to the Bacteriology and Virology Department for STEC screening according to the previously described procedure[11]. PCR was performed on 20 suspected *E. coli* colonies to detect *stx*1 and *stx*2 genes.

Confirmation of isolates as *E. coli* was performed through biochemical tests and serotyping was conducted using commercial antisera (Statens Serum Institute, Denmark) for serogrouping and our own antibodies for H antigens.

The presence of sequences related to the *ehxA* and *eae* genes was determined by PCR[7]. Subtyping of the *eae* gene was also performed by PCR[1]. Antimicrobial susceptibility to amikacin, ampicillin, ceftriaxone, cefuroxime, ciprofloxacin, cloramphenicol, colistin, gentamicin, nalidixic acid, nitrofurantoin, streptomycin, tetracycline, and trimethoprim-sulfamethoxazole was established according to the Clinical Laboratory Standards Institute (CLSI) guidelines[3].

The 20 colonies of *E. coli* analyzed corresponded to STEC serotype O145:HNT, with genotype: *stx*2, *ehxA*, *eae* subtype β1, which were susceptible to all tested antibiotics.

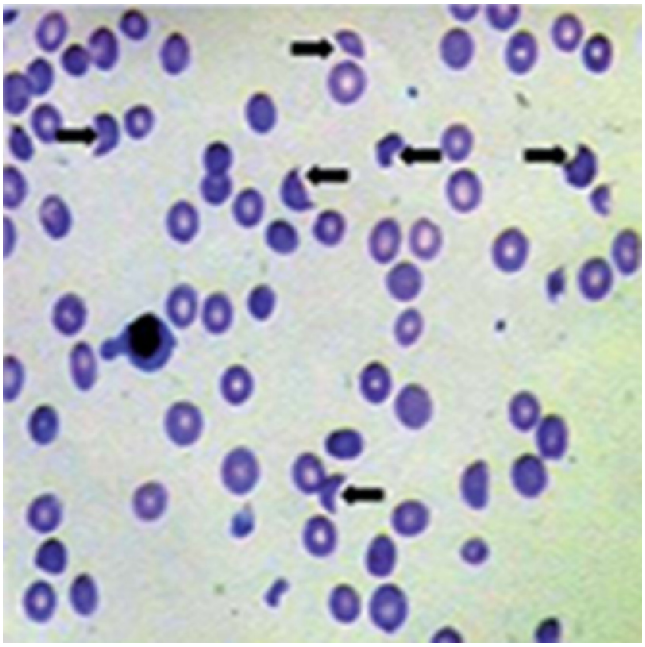

Figure 1 Peripheral blood smear stained with the Giemsa procedure. The black arrows indicate the schizocytes.