



Prevalence of Shiga Toxin in the Stools of HUS Patients

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Dedication

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Abstract

The study included 14 Hemolytic Uremic Syndrome patients who were admitted at Nephrology department of Abulrish El Mouneera Hospital along 6 months (October 2012-March 2013)

The study summarizes the clinical and laboratory findings in these patients as well as the treatment received and outcome with focusing on the prevalence of Shiga Toxin in the stools of the study cases.

The study showed presence of Shiga Toxin in the stools of 3 patients out of 14 resulting in percentage of (21.4%)

Key words:

(HUS, Shiga Toxin, Pediatric Nephrology Unit)

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List of Abbreviations

aHUS	atypical hemolytic uremic syndrome
AKI	Acute Kidney Injury
ANA	Anti-nuclear Antibody
AP	Alternative Pathway
APLS	AntiphospholipidAntibody Syndrome
cbIC	Cobalamine C
cbIG	Cobalamine G
CFH	Complement factor H
CFHR	Complement factor H-related
CFI	Complement factor I
CIC	Circulating immune complex
CNS	Central Nervous System
CT-SMAC	cefixime tellurite-sorbitol MacConkey agar
D+HUS	Diarrhoea associatedHaemolytic Uremic Syndrome
D-HUS	Diarrhoeanot associatedHaemolytic Uremic Syndrome
DAG	Diacylglycerols
E.Coli	Escherichia coli

EHEC	Enterohaemorrhagic E. Coli
EIA	ELISA Immune Assay
ESKD	End Stage Kidney Disease
FDA	Food and Drug Administration
FFP	Fresh Frozen Plasma
GFR	Glomerular Filtration Rate
GI	Gastrointestinal
HELLP	Hemolysis, Elevated liver enzymes, Low platelets
HIV	Human Immunodeficiency Virus
HSCT	hematopoietic stem cell transplantation
HUS	Haemolytic Uremic Syndrome
LDH	Lactate Dehydrogenase
MAC	Membrane Attack Complex
MCP	Membrane Complement Protein
MRI	Magnetic Resonance Imaging
No.	Number of cases
PCR	Polymerase Chain Reaction
PE	Plasma Exchange
PKC	protein kinase C

PNH	Proxysmal Nocturnal Hemoglobinuria
P-TMA	Pregnancy-associated thrombotic microangiopathy
RBCs	Red Blood Corpuscles
SD	Standard Deviation
SLE	Systemic Lupus Erythematosus
SMAC	Sorbitol-MacConkey Agar
S.Pneumoniae	Streptococcus Pneumoniae
STEC	Shiga-toxin-producing E. Coli
Stx	Shiga Toxin
T antigen	Thomsen-Friedenreich antigen
THBD	Thrombomodulin
TCRF	Terminal Chronic Renal Failure
tHUS	Typical Hemolytic Uremic Syndrome
TMA	Thrombotic Microangiopathy
TTP	Thrombotic Thrombocytopenic Purpura
USA	United States of America
VEGF	Vascular endothelial growth factor
VWF	Von Willebrand Factor
WBC	White Blood Cells

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Introduction

Hemolytic uremic syndrome is defined by the characteristic triad of microangiopathic hemolytic anemia, thrombocytopenia, and acute renal failure. In children, most cases of HUS are caused by Shiga-toxin-producing bacteria, especially *Escherichia coli* O157:H7. Common vehicles of transmission include ground beef, unpasteurized milk, and swimming water (*Boyer and Niaudet, 2011*).

STEC is an important cause of acute kidney injury (AKI) in children worldwide and is responsible for 70–90% of all cases of HUS in this age group in countries where STEC are endemic (*Bitzan, 2009*).

Over the past 10 years, it has become increasingly apparent that HUS is one clinical manifestation of a larger entity called thrombotic microangiopathy (TMA). This lesion is a well-defined pathological phenotype that reflects injury to the vascular endothelium. TMA can result from bacterial toxins, medications, systemic illnesses, and abnormalities in the regulation of the alternate complement system, and diminished activity of the von Willebrand factor cleaving protease (*Trachtman, 2008*).

Aim of Work

The aim of the current study is to understand the causes of HUS, patient characteristics, laboratory findings, management and outcome in Egypt's patients population with focusing on prevalence of Shiga Toxin as a major cause of HUS in Egypt as being an endemic area for E.Coli.

Definition of HUS

Haemolytic uremic syndrome (HUS) is a severe, acute and dramatic disease affecting previously healthy children. HUS is defined as a triad of acute kidney injury, microangiopathic haemolytic anemia and thrombocytopenia in patients with no other explanation for coagulopathy e.g. thrombotic thrombocytopenic purpura. More than 90% of the cases are due to Shiga toxin-producing *E. coli* (STEC) infections; termed typical HUS or diarrhea associated HUS (D+HUS). Many different serotypes can cause HUS, the most prevalent in Europe and USA being O157:H7. A broad spectrum of extrarenal complications may occur in HUS, the most common are gastrointestinal and cerebral. Extrarenal involvement at an early stage is associated with increased morbidity and mortality. Although several epidemics, caused by O157 and other serotypes have been reported, the majority of HUS cases appear sporadic or in small clusters (***Krogvold et al., 2011***).