Opinion Article

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The differential diagnosis :Hemolytic uremic syndrome

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Introduction

Hemolytic uremic syndrome (HUS) is a clinical disease that includes thrombotic microangiopathy, thrombocytopenia, and acute kidney damage as a triad. Hemolytic uremic syndrome is a set of illnesses with a variety of etiologies, resulting in variations in presentation, therapy, and prognosis. In recent years, improved knowledge of HUS, particularly those caused by genetic abnormalities in the alternative complement pathway, has resulted in revisions to the disease's nomenclature, categorization, and therapy. In addition to STEC-HUS, which is the most frequent cause of HUS in children, this review will present an updated categorization of the condition as well as current diagnostic and therapeutic options for complement-mediated HUS. The triad of microangiopathy hemolytic anaemia, thrombocytopenia, and acute renal injury characterises hemolytic uremic syndrome (HUS). One of the most prevalent causes of acute renal failure in children is this condition. The clinical manifestations of hemolytic uremic syndrome are caused by thrombotic microangiopathy (TMA). Thickening of arterioles and capillary walls, as well as endothelial enlargement and separation, are pathological lesions. The arterial lumen is obstructed by fibrin and platelet-rich thrombi. Many tissues and organs are impacted, most notably the kidney. Thrombotic microangiopathies are a collection of illnesses with a variety of etiologies and pathologies. HUS and thrombotic thrombocytopenic purpura (TTP) are the two primary disease groupings. For many years, these two disorders were used interchangeably and tried to be distinguished based on their clinical characteristics. According to current understanding, both illnesses have distinct etiologies and pathophysiology. In the absence of a metalloproteinase that cleaves von Willebrand factor in plasma or in the presence of an antibody against it, thrombotic thrombocytopenic purpura develops. HUS was formerly classed as a diarrhea-causing virus. Negative for HUS (D+HUS) and diarrhoea HUS (D-HUS) and D+HUS were considered to be the same as typical HUS, while D-HUS was considered to be the same as atypical HUS. However, because diarrhoea is the triggering event in 25–30% of atypical HUS cases, the distinction between D+HUS and D-HUS has been dropped. In the literature, there are several alternative etiological categories for hemolytic uremic syndrome. In terms of underlying disorders, several of these classifications overlap with TMA classifications.

The conventional and atypical HUS classifications are extensively employed in clinical practise. For STEC-HUS, standard HUS is utilised. On the other hand, there are significant disagreements in the literature over the definition of atypical HUS (aHUS). Until recently, the term aHUS was used to describe all HUS instances save STEC-HUS. Some writers have begun to utilise it exclusively for complement-related HUS in recent years. EHEC is mostly produced by cattle and sheep. Infection in humans is usually caused by eating food that has been contaminated with animal faeces. Undercooked meat, unpasteurized milk and dairy products, fruit juices, water, fruits and vegetables are the most typical causes. Transfer by animal contact, direct transmission from human to human, and transmission from mother to child are all possibilities. It's particularly frequent in the summer and autumn months. Diarrhea starts in 3–8 days after ingesting tainted food. Diarrhea starts off watery and then turns bloody. Diarrhea is accompanied by stomach pains, nausea, and vomiting. Fever isn't as frequent as it used to be. Following enterohemorrhagic E. coli diarrhoea, HUS develops in 5–15 percent of the patients. The bacteria's serotype (O157:H7), the toxin type (Stx 2), the patient's age (five years), the use of antibiotics and antimotility medicines, fever, severe diarrhoea, female gender, an elevated leukocyte count, and genetic variables are all risk factors for the development of HUS . None of them, however, are supported by substantial data. The major clinical picture of the illness is made up of haematological and renal abnormalities. The data generated by this clinical picture are presented to patients. Paleness, lethargy, lack of appetite, nausea, and vomiting are all possible symptoms. Some individuals may notice a decrease in urine flow, or edoema may be a major complaint. There are laboratory abnormalities that correspond to the disease's typical triad (microangiopathic hemolytic anaemia, thrombocytopenia, and acute renal injury). Hemolytic anaemia, thrombocytopenia,

and renal impairment develop abruptly in a patient who has had diarrhoea in the previous two weeks, and the diagnosis is determined clinically. STEC infection must be verified for a definitive diagnosis (demonstration of Stx with stool serologic tests or stool cultures). Renal biopsy isn't required for a definitive diagnosis