A Case of Ulcerative Colitis Accompanied by Hemolytic Uremic Syndrome

Ülseratif Kolit Hemolitik Üremik Sendrom Birlikteliği

ABSTRACT

Hemolytic uremic syndrome (HUS) is a disease presenting with nonimmune hemolytic anemia, renal impairment and low platelet count. Escherichia coli serotype O157:H7 is the most common bacterial strain in typical HUS. Ulcerative colitis can be activated after E. coli infection and complications may occur afterwards. Toxic megacolon, necrosis and perforation of bowel, peritonitis, and severe hemorrhagic colitis are the complications of ulcerative colitis. In this paper, we report a typical Shiga toxin-associated HUS case accompanied by ulcerative colitis activation and toxic megacolon.

KEY WORDS: Hemolytic uremic syndrome, Toxic megacolon, Ulcerative colitis

ÖZ

Hemolitik üremik sendrom (HÜS) nonimmün hemolitik anemi, böbrek yetmezliği ve düşük trombosit sayısı ile prezante olan bir hastalıktır. Escherichia coli O157: H7 serotipi tipik HUS için en yaygın bakteriyel suşudur. Ülseratif kolit e.coli enfeksiyonuna sekonder aktive olabilir ve aktivasyon sonrası komplikasyon gelişebilir. Toksik megakolon, bağırsak perforasyonu ve nekrozu, peritonit, şiddetli hemorajik kolit ülseratif kolitin komplikasyonlarındandır. Bu yazımızda ülseratif kolit aktivasyonu ve toksik megakolon komplikasyonu sonrasında ortaya çıkan ve buna eşlik eden Shiga toksin ilişkili tipik HÜS olgusu sunulmuştur.

ANAHTAR SÖZCÜKLER: Hemolitik üremik sendrom, Toksik megakolon, Ülseratif kolit

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INTRODUCTION

Hemolytic uremic syndrome (HUS) is a disease presenting with microangiopathic hemolytic anemia, thrombocytopenia, and acute renal failure (1). There are many reasons in the etiology such as infections, systemic diseases and drugs but most of them occur after diarrhea of Shiga-like toxin-producing E. coli (2). In this paper we present a case of hemolytic uremic syndrome accompanied by ulcerative colitis activation and toxic megacolon.

CASE

A 50-year-old female patient was admitted with complaints of abdominal pain, and bloody diarrhea 4-5 times daily for five days. Her medical history revealed that ulcerative colitis had been diagnosed with colonoscopy at an external centre (Figure 1). On physical examination, there was tenderness in the abdomen but no rigidity. Laboratory tests revealed Creatinine: 0.6 mg/dl, Urea: 40 mg/dl, Leukocyte: 19000 10^9/L, hemoglobin: 14 g/dl, Thrombocyte: 267000 10^9/L, Sedimentation: 111/hr, CRP: 151 (0-5), and LDH: 478 U/L. Leukocyte and erythrocytes were seen on stool microscopy. X-ray films of the abdomen showed increased diameter of the transverse colon (Figure 2) and computed tomography revealed that the widest part of the colon was 7 cm (Figure 3). These findings were consistent with toxic megacolon. A nasogastric tube was inserted to the patient and metronidazole, ciprofloxacin, mesalazine tablets, mesalazine enemas, methyl prednisolone were started.

On the 4th day of admission creatinine was 2.26 mg/dL, urea 138 mg/dL, hemoglobin 9.8g/dL, platelets 25.000 10^9/L, and LDH 2176 U/L. Schistocytes were seen in the patient's peripheral blood smear, direct and indirect Coombs tests were negative, E. coli O157-H7 antigen was positive and ADAM TS 13 activity was detected at 81%. Cryptitis was seen in colon biopsy, and the pathology findings were consistent with infectious colitis.

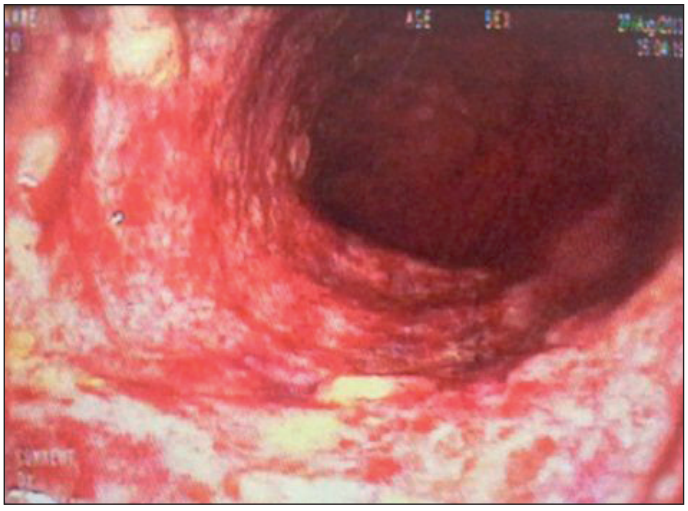

Figure 1: *Endoscopic image of an infectious colitis confused with active ulcerative colitis.*

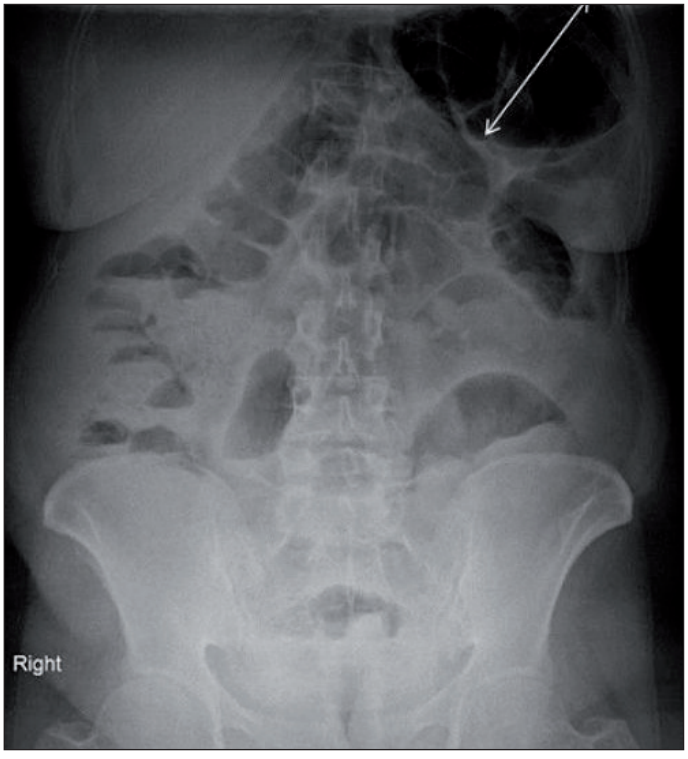

Figure 2: *X ray image of increased diameter in the transverse colon consistent with toxic megacolon.*

HUS was therefore considered. During follow-up, the transverse colon width had decreased and on the 11th day of admission the creatinine level decreased to 1.22 mg/dL, the platelet level increased to 213.000 and LDH level decreased to 817 U/L.

DISCUSSION

HUS is defined with a triad of microangiopathic hemolytic anemia, thrombocytopenia and renal failure. Typical HUS occurs as a result of Shiga toxin and follows diarrhea or upper respiratory tract infections in 90% of the cases (1). Atypical HUS occurs as a result of a malfunction in the alternative complement pathway. It can be genetic, acquired or idiopathic (3). In addition to the kidneys, HUS can affect the nervous system, gastrointestinal tract, lungs, myocardium and liver. Every location from the esophagus to the perianal area can be involved in the gastrointestinal tract. More seriously, it may present as peritonitis, perforation, necrosis and hemorrhagic colitis (2). In this paper the patient present as hemorrhagic colitis and it was diagnosed as ulcerative colitis according to the colonoscopy image. E. coli infection occurred in patient and ulcerative colitis activation developed after this infection. Toxic megacolon, colonic perforation, recurrent signs of obstruction or colonic stricture can also be seen as complication of ulcerative colitis. Patients with fulminant colitis are at high risk of developing toxic megacolon (4). Toxic megacolon developed as a complication in this case and the colon biopsy showed that it was consistent with infectious colitis. The microangiopathic hemolytic anemia, thrombocytopenia and renal failure confirmed the diagnosis of HUS. There are also similar cases with the complications of HUS in the literature (2,5). In conclusion, we want to emphasize that HUS can accompanied by complications of ulcerative colitis and one must therefore be careful.

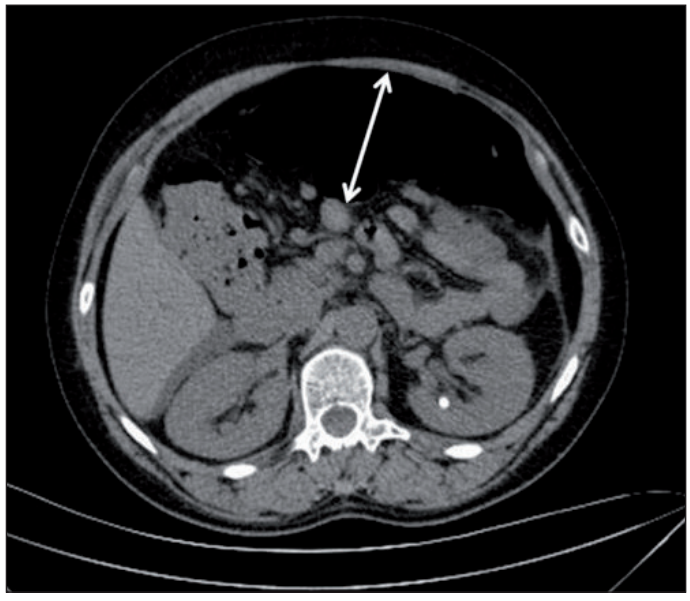

Figure 3: *Computed tomography scan of the abdomen showing toxic megacolon.*

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