Bloody diarrhea caused by enterohemorrhagic *Escherichia coli* (EHEC)

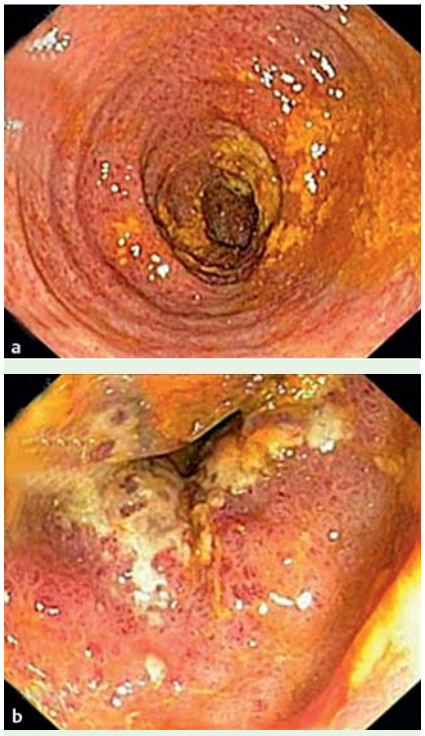

Fig. 1 **a** Sigmoidoscopy showing ulcerated mucosa with diffuse reddening, edema, and numerous petechiae. **b** Bowel obstruction in the proximal sigmoid colon due to marked swelling of the ulcerated mucosa.

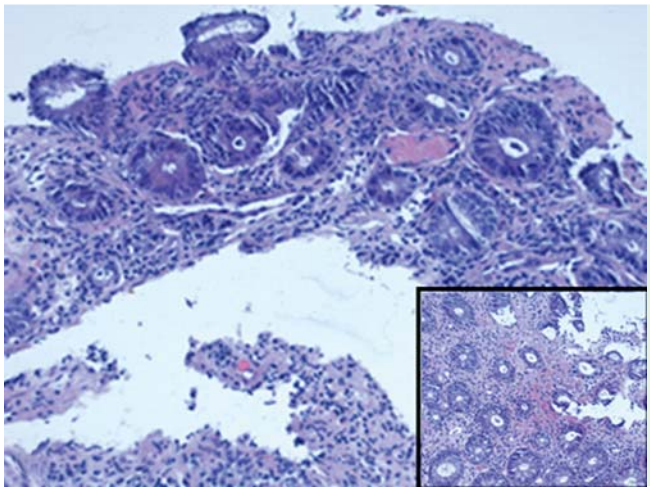

Fig. 2 Histologic section showing patchy eosinophilic cytotoxic necrosis. There is complete destruction of the glandular structures, with scattered infiltrates of neutrophilic granulocytes. The inset shows early-stage lesions with fibrin thrombi within the capillaries.

Infection with enterohemorrhagic *Escherichia coli* (EHEC) has been spreading in Germany since the second week of May and causing considerable concern among the population. At the time of writing, nine deaths have been reported due to the subsequent development of hemolytic uraemic syndrome (HUS), but the source of the outbreak is still unknown. As of May 31, 2011, the Robert Koch Institut, Berlin, Germany, has confirmed a total of 470 cases of HUS and stated that this epidemic has not yet reached its climax [1].

A 72-year-old man presented with massive bloody diarrhea for 3 days. On physical examination, he had a pulse rate of 102 beats per minute, the blood pressure was 110/50 mm Hg, hemoglobin 15 g/dl (reference values 13.5 – 17.5), and hematocrit 43.5 % (40 – 53). Remaining laboratory parameters, including renal function parameters, were unremarkable. We carried out emergency endoscopy to rule out acute gastrointestinal bleeding. Sigmoidoscopy (Olympus, Tokyo, Japan) revealed a markedly abnormal, ulcerative mucosa with diffuse reddening, edema, and numerous petechiae (**Fig. 1 a**). In addition, bowel obstruction was noted in the proximal sigmoid colon, due to marked swelling of the ulcerated mucosa (**Fig. 1 b**).

The differential diagnosis included ischemic colitis, nonsteroidal anti-inflammatory drug (NSAID)-colonopathy, and infectious colitis. Histopathologic examination revealed patchy eosinophilic cytotoxic necrosis with complete destruction of glandular structures and scattered infiltrates of neutrophilic granulocytes (**Fig. 2**).

Early-stage lesions showed fibrin thrombi within the capillaries (**Fig. 2**, **inset image**). The discontinuous pattern of inflammation and necrosis, and presence of microthrombi and granulocytic infiltration, ruled out ischemic colitis and infection caused by common cytotoxic bacteria (i. e., *Clostridium difficile*) [2,3]. On day 4 of hospitalization EHEC was detected in stool samples. However, owing to development of HUS, which signifies unfavorable disease progression, the patient was admitted to the intensive care unit and put on renal replacement therapy. At the time of going to press the patient's situation was still critical.

Our case demonstrates the clinical presentation and the endoscopic and histological characteristics of EHEC infection. As EHEC is transmitted via the oral-fecal route, increased precautions are warranted in the endoscopy suite to reduce the risk of transmission during an epidemic. Physicians should be aware of the currently increasing incidence of EHEC and should consider EHEC infection in the differential diagnosis of patients admitted with bloody diarrhea.

Endoscopy_UCTN_Code_CCL_1AD_2AZ

Competing interests: None

H. Neumann[1], M. Hunstiger[2], C. Langner[3], M. F. Neurath[1], M. Vieth[4]

[1] Department of Medicine I, University of Erlangen-Nuremberg, Erlangen, Germany
[2] Diako Stadtklinik Augsburg, Augsburg, Germany
[3] Institute of Pathology, Medical University of Graz, Graz, Austria
[4] Institute of Pathology, Klinikum Bayreuth, Bayreuth, Germany

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Bibliography

DOI 10.1055/s-0030-1256596
Endoscopy 2011; 43: E229 – E230

ISSN 0013-726X

Corresponding author

H. Neumann, MD
Department of Medicine I
University of Erlangen-Nuremberg
Ulmenweg 18
91054 Erlangen
Germany
Fax: +49 9131 85-35209
helmut.neumann@uk-erlangen.de