Reply

To the Editor,

We were pleased to read Dr. Özsoylu's Letter to the Editor regarding our paper titled "Hemolytic uremic syndrome outbreak in Turkey in 2011"[1]. We would like to comment on two very important points that Özsoylu made about Shiga toxin-producing Escherichia coli (STEC) hemolytic uremic syndrome (HUS).

Rock et al.[2] proposed that molecular similarities between membrane antigen CD36 and Shiga toxin lead to the development of antibodies, and it was this that caused the events in STEC-HUS in 2005. Since then, no other evidence of immune-mediated diseases has been reported in the pathophysiology of STEC-HUS.

The current pathophysiology of STEC-HUS is explained by the binding of Shiga toxins to the Gb3 expressed by glomerular endothelial cells, causing direct endothelial injury through inhibition of protein synthesis. Increased cytokines and chemokines that cause inflammation also take part in this process. Another pathophysiological process during STEC-HUS was shown on the alternate complement system. Purified Shiga toxin-2 caused direct activation of complement in the fluid phase, and cofactor activity of factor H was delayed on the cell surface when bound to Shiga toxin-2[3]. In light of this information, megadose prednisolone is currently not an alternative treatment modality in STEC-HUS.

The other important point that we would like to comment on is the subject of antibiotic usage during enterohemorrhagic *E. coli* infections. Previous reports and expert opinions have given rise to an extensive idea that antibiotic usage increases Shiga toxin excretion from the bacteria, and the incidence of STEC-HUS increases if a child with STEC infection is treated with antibiotics[4-6]. However, recent reports during the German outbreak established new evidence regarding antibiotic usage[7-9]. Randomized controlled trials are necessary before reaching a final decision on this subject.

REFERENCES

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Zelal Ekinci, MD

Professor of Pediatrics, Nephrology and Rheumatology
Kocaeli University Faculty of Medicine

Oğuz Söylemezoğlu, MD

Professor of Pediatrics, Nephrology and Rheumatology
Gazi University Faculty of Medicine