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LETTER TO THE EDITOR

The Significance of Interleukin 12 and Interferon-Gamma in Thrombotic Thrombocytopenic Purpura/Hemolytic Uremic Syndrome and Crohn's Disease

Comment on: A patient with Crohn's disease who presented with thrombotic thrombocytopenic purpura/hemolytic uremic syndrome (Ren Fail. 2011;33(2):244–245)

Dear Editor

We read with great interest the contribution by Unverdi et al. They reported a case of thrombotic thrombocytopenic purpura (TTP)/hemolytic uremic syndrome (HUS) that presented with Crohn's disease (CD) and presumed microthrombosis triggered by autoimmune disorders to be the main cause in the development of TTP/HUS and CD. The first report of HUS in patients with CD also described thrombosis at the microvascular level and increased coagulability as an important mechanism involved in the two diseases. However, we would like to add another possible pathomechanism of TTP/HUS associated with CD.

According to a previous report by Takatsuka et al.,³ interleukin (IL)-12, the T helper (Th) cell-associated cytokine, significantly increased in the patients with thrombotic microangiopathy after bone marrow transplantation (BMT) (p < 0.05), while those without microangiopathy did not show an increase in IL-12. Kakishita⁴ also showed that TTP and HUS after BMT could be predicted at an early stage by any increase in plasma IL-12, assuming that TTP might be related to inflammation or autoimmunity.

Recently, Strober et al.⁵ studied the role of proinflammatory cytokines responsible for the inflammation underlying CD and announced that not only IL-12 but also interferon-gamma (IFN-γ) induced by IL-12-driven Th1 cells during the development of Th17 differentiation is an important proinflammatory cytokine in CD. This explicates that anti-IL-12p40, an antibody

that inhibits both the Th1 and Th17 responses, and anti-IFN- γ administration can logically be the most potent agents of anti-inflammatory cytokine for the treatment of CD.

Therefore, it is possible that IL-12 and Th1-mediated IFN-γ may play the central role in the pathogenesis of TTP/HUS associated with CD, and it would be helpful to measure IL-12 and IFN-γ levels during the course of the disease. However, further studies are necessary to elucidate which cytokines between Th1 and Th17 response are more active in the inflammation of TTP/HUS associated with CD. The relationship between IL-12 and IFN-γ levels and the severity of TTP/HUS or CD should also be further evaluated in the future.

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