Tumor Necrosis Factor and Splenectomy

Tümör Nekrozis Faktör ve Splenektomi

İrfan Yavaşoğlu

Adnan Menderes University Faculty of Medicine, Division of Hematology, Aydın, Turkey

To the Editor,

The article entitled "Effect of Tumor Necrosis Factor-Alpha (TNF-α) on Erythropoietin- and Erythropoietin Receptor-Induced Erythroid Progenitor Cell Proliferation in β-Thalassemia/Hemoglobin E Patients", written by Tanyong et al. [1] and published in a recent issue of your journal, was quite interesting. Here we would like to emphasize some relevant points.

Splenectomy can increase the release of TNF-α and cell apoptosis in experimental and clinical studies in different diseases [2,3,4].

Increased serum TNF-α was reported in E/b-Thal patients, particularly after splenectomy [3,4]. In sickle cell disease presenting with functional asplenia, increased amounts of TNF-α, indicative of monocyte activation, and increased serum C-reactive protein levels were reported [5].

Banyatsuppasin et al. suggested the role of the spleen in controlling mononuclear phagocytic activity in E/b-Thal patients [6]. TNF-α play roles as an inducer and effector of monocyte activation [6]. Additionally, TNF-α returned to normal after 12, 6, and 3 months of deferiprone treatment [7]. Therefore, chelation treatment can affect apoptosis independently of splenectomy. It might be important to know the effect of chelation treatment and splenectomy on tumor necrosis factor in the study of Tanyong et al. [1] based on all these investigations stated above [2,3,4,5,6,7].

Keywords: Thalassemia, Tumor necrosis factor, Splenectomy

Anahtar Sözcükler: Talasemi, Tümör nekrozis faktör, Splenektomi

Conflict of Interest: The author of this paper has no conflicts of interest, including specific financial interests, relationships, and/or affiliations relevant to the subject matter or materials included.

References

1. Tanyong D, Panichob P, Kheansaard W, Fucharoen S. Effect of tumor necrosis factor-alpha on erythropoietin- and erythropoietin receptor-induced erythroid progenitor cell proliferation in β-thalassemia/hemoglobin E patients. Turk J Hematol 2015;32:304-310.
2. Hiroyoshi T, Tsuchida M, Uchiyama K, Fujikawa K, Komatsu T, Kanaoka Y, Matsuyama H. Splenectomy protects the kidneys against ischemic reperfusion injury in the rat. Transpl Immunol 2012;27:8-11.
3. Chuncharunee S, Archararit N, Hathirat P, Udomsubpayakul U, Atichartakarn V. Levels of serum interleukin-6 and tumor necrosis factor-a in postsplenectomized thalassemic patients. J Med Assoc Thai 1997;80(Suppl 1):S86-S91.
4. Wanachiwanawin W, Wiener E, Siripanyaphinyo U, Chinprasertsuk S, Mawas F, Fucharoen S, Wickramasinghe S, Pootrakul P, Visudhiphan S. Serum levels of tumor necrosis factor-α, interleukin-1, and interferon-γ in β0-thalassemia/HbE and their clinical significance. J Interferon Cytokine Res 1999;19:105-111.
5. Belcher JD, Marker PH, Weber JP, Hebbel RP. Activated monocytes in sickle cell disease: potential role in the activation of vascular endothelium and vaso-occlusion. Blood 2000;96:2451-2459.
6. Banyatsuppasin W, Butthep P, Atichartakarn V, Thakkinstian A, Archararit N, Pattanapanyasat K, Chuncharunee S. Activation of mononuclear phagocytes and its relationship to asplenia and phosphatidylserine exposing red blood cells in hemoglobin E/β-thalassemia patients. Am J Hematol 2011;86:89-92.
7. Del Vecchio GC, Schettini F, Piacente L, De Santis A, Giordano P, De Mattia D. Effects of deferiprone on immune status and cytokine pattern in thalassaemia major. Acta Haematol 2002;108:144-149.

Address for Correspondence/Yazışma Adresi: İrfan YAVAŞOĞLU, M.D.,
Adnan Menderes University Faculty of Medicine, Division of Hematology, Aydın, Turkey
Phone : +90 256 212 00 20
E-mail : dr_yavas@yahoo.com

Received/Geliş tarihi: January 23, 2016
Accepted/Kabul tarihi: January 26, 2016
DOI: 10.4274/tjh.2016.0040