Homocysteine-Related Venous Ulcer: Editorial

Homosisteine Bağlı Venöz Ülser

Ahmet AKGÜL, MD[a]
Publication Editor
Turkiye Klinikleri Journal of Cardiovascular Sciences
Member of Council of Science Editors

[a]Clinics of Cardiovascular Surgery, Türkiye Yüksek İhtisas Hospital, Ankara

Yazışma Adresi/*Correspondence:*
Ahmet AKGÜL, MD
Türkiye Yüksek İhtisas Hospital, Clinics of Cardiovascular Surgery, Ankara
TÜRKİYE/TURKEY
aakgul@turkiyeklinikleri.com

Key Words:
Homocysteine, venous ulcer

Anahtar Kelimeler:
Homosistein, venöz ülser

Venous ulcers still remain a common health burden for patients, health professions and health insurance companies as well.[1] The responsible mechanisms of ulcer formation, although have been highly evaluated, are found as fibrin cuff, leucocyte trapping and chronic inflammation.[2] As presented by Simon and coworkers, by an excellent review in *BMJ*, the treatment for venous ulceration is almost treating the elevated venous hypertension.[2] Although this may be the case in the treatment of many venous ulcers, the other etiologies may be kept in mind in evaluating the wound. In this Editorial, besides of many etiologies, the point should be given in dietary habits while managing venous ulcers. Dietary habits remain the basis of all vascular type of pathologies.[3] As Napoli and associates presented in their Editorial in *Cardiovascular Research,* an expanding efford has been made to investigate and evaluate the mechanisms of vacular lesions with nutrition.[3]

Homocysteine (Hcy) is a sulphydryl-containing metionine which leads endothelial damage, inhibition of nitric oxide and oxidation.[4] Plasma Hcy levels differ among populations accepted avarage level: 5-15 μmol/L).[4] Although genetic defects, as enzyme or related factors deficiencies, are higly responsible factor for its metabolism, nutritional habits (i.e. vitamin insufficiencies) remain one of the common acquired factor for defective metabolism.[4] Years of 1960s were the time when Hcy deserves required attention in the pathology of vascular diseases.[5] McCully presented experiences in patients with Hcy in the excellent review, published in *The American Journal of Clinical Nutrition*.[6] Based on Framingham Heart Study[7] and 3rd National Health and Nutrition Examination Survey,[8] McCully proposed dietery defieciencies of vitamines (B-6,B-10,B-12) in patients with vascular diseases associate with elevated levels of Hcy.[6] Hyperhomocysteinemia was finally found an important risk factor for not only arterial diseases but venous pathologies as well.[9] The main key point for venous diseases is the endothelial damage, eventually leads to the development of ulcer, which might be beter modulated by B-vitamines, by decreasing the level of Hcy and maintai-

ning nitric oxide synthesis, cyclooxgenase inhibition, leading to repairing endothelial function.[10]

Shankar and associates treated a patient with venous ulcer by administration of B-type vitamins.[11] Endothelium damage with high levels of Hcy in patients with venous ulcer, might be treated with not only by developing strategies to lowering venous pressure, using expensive surgical wound dressings, establishing invasive surgical approach but by dietary supplements as well.

REFERENCES

1. Ruckley CV. Socioeconomic impact of chronic venous insufficiency and leg ulcers. Angiology 1997;48:67-9.
2. Simon DA, Dix FP, McCollum CN. Management of venous leg ulcers. BMJ 2004;328: 1358-62.
3. Napoli C, Stanley WC, Ignarro LJ. Nutrition and cardiovascular disease: putting a pathogenic framework into focus. Cardiovasc Res 2007;73:253-6.
4. Dikmen M. [Homocysteine metabolism and association with diseases: A Review]. Turkiye Klinikleri J Med Sci 2004;24:645-52.
5. McCully KS. Vascular pathology of homocysteinemia: implications for the pathogenesis of arteriosclerosis. Am J Pathol 1969; 56:111-28.
6. McCully KS. Homocysteine, vitamins, and vascular disease prevention. Am J Clin Nutr 2007;86(Suppl):1563S-8S.
7. Selhub J, Jacques PF, Wilson PW, Rush D, Rosenberg IH. Vitamin status and intake as primary determinants of homocysteinemia in an elderly population. JAMA 1993;270:2693-8.
8. Giles WH, Croft JB, Greenlund KJ, Ford ES, Kittner SJ. Association between total homocyst(e)ine and the likelihood for a history of acute myocardial infarction by race and ethnicity: Results from the Third National Health and Nutrition Examination Survey. Am Heart J 2000;139:446-53.
9. Ray JG. Meta-analysis of hyperhomocysteinemia as a risk factor for venous thromboembolic disease. Arch Intern Med 1998;158:2101-6.
10. Halliwell B. Dietary polyphenols: good, bad, or indifferent for your health? Cardiovasc Res 2007;73:341-7.
11. Krupa Shankar DS, Akhib S. Leg ulcers due to hyperhomocysteinemia. Indian J Dermatol Venereol Leprol 2006;72:367-9.