

Abstract

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Treatment of erectile dysfunction due to C677T mutation of the MTHFR gene with vitamin B6 and folic acid in patients non responders to PDE5i.

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INTRODUCTION: Epidemiological studies conducted on erectile dysfunction (ED) have demonstrated its close correlation with cardiovascular disease. Since hyperhomocysteinemia is considered an important cardiovascular risk factor, it could also be involved in the pathogenesis of ED.

AIM: To study the role of the C677T MTHFR mutation with subsequent hyperhomocysteinemia in the determination of ED.

METHODS: We studied 75 consecutive patients presenting with ED. Patients were interviewed using the International Index of Erectile Function. Blood samples were drawn for determination of MTHFR gene C677T mutation, homocysteine (Hcy) and folate levels. Penile color Doppler was also performed.

MAIN OUTCOME METHODS: Patients were administered sildenafil citrate for 2 months. The nonresponders were treated with combination of sildenafil, vitamin B6, and folic acid for 6 weeks. Patients were split into three groups, A, B, and C on the basis on their MTHFR genotype, and in a further group defined as "sildenafil nonresponders" (NR).

RESULTS: We found 20 patients homozygous for mutant MTHFR 677T, 36 heterozygous, and 19 wild type. Difference in baseline values for Hcy and folic acid was found between groups A and B, and A and C. The NR group (18 patients from group A and B), presented high levels of Hcy and low levels of folic acid. After combination treatment 16 of them (88.9%) revealed an improvement in the IIEF questionnaire. Moreover, it was measured a significant difference between the values of Hcy and folic acid at the baseline and at the end of the study for the nonresponders.

CONCLUSIONS: Hyperhomocysteinemia in patients homozygotes for the C677T mutation may interfere with erection mechanisms and thus be responsible for ED. In case of hyperhomocysteinemia associated with low levels of folates, the administration of PDE5 inhibitors may fail if not preceded by the correction of the altered levels of Hcy and folates.

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