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Authors' response

Sir,

We are extremely grateful for the letter[1] to editor in response to our article. The authors have raised two issues and from the content of their letter, there seems to be a requirement for clarifying some aspects of the study.

Table I in our study[2] shows that homocysteine (Hcy) in cases was 22.14±10.62 and in controls was 17.38 ± 8.46 μmol/l and difference was statistically significant (*P*=0.001). The Hcy levels of controls in our study were higher than western values and this

was attributed to probably folate and vitamin B12 deficiency. As far as values of folate and B12 are concerned, the measurement of these was not a part of our study and the last statement of conclusion in the abstract mentions this very clearly that further studies are required in this direction.

Confounding role of MTHFR can also be understood clearly by referring to Table I. Among cases, there was no significant difference in Hcy level when wild type MTHFR (CC) of mutation was compared with heterozygous cases. Similar finding was seen among controls. As far as homozygous mutations were concerned, there were only three cases with mutation and one control, among them the Hcy values were very high, *i.e.* very few mutant cases. Thus in our study we did not find higher Hcy in cases with mutation as compared to those without mutation. Significant post treatment decrease was found in Hcy by vitamin intervention irrespective of their having mutation or not. These vitamins are known to help bypass the effect of labile methylene tetrahydrofolate reductase enzyme which occurs in patients with mutation. Thus, irrespective of the cause for increase of Hcy, vitamin intervention was helpful in lowering its levels and this has been clearly shown in our study.

The second point raised regarding values of cholestrol and LDL in the controls in Table III is valid and is due to typographical error. The correct value of LDL amongst controls was 82.0 ± 36.24 mg/dl. The *P* value remains the same.

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