It is today well known that vitamin B\textsubscript{12} deficiency can be associated with neuropsychiatric symptoms. Several studies have previously demonstrated that vitamin B\textsubscript{12} deficiency is more common in patients with dementia symptoms than in the cognitively non-impaired. This topic is touched upon in the article “Clinical and laboratory features and response to treatment in patients presenting with vitamin B\textsubscript{12} deficiency-related neurological syndromes” in the current issue of Neurology India. Vitamin B\textsubscript{12} deficiency increases with age and is present in 5-40% of the elderly population. However, the mechanism of neurological damage induced by a quantitative or functional vitamin B\textsubscript{12} deficiency is still unclear.

Vitamin B\textsubscript{12} exerts its physiological effect on two major enzymatic pathways: the conversion of homocysteine to methionine and the conversion of methylmalonyl coenzyme A to succinyl coenzyme A. Disruption of either of these pathways due to vitamin B\textsubscript{12} deficiency results in an elevation of both serum homocysteine and methylmalonic acid. Homocysteine levels are also elevated in the case of folate deficiency. Serum homocysteine is proposed to be more sensitive for functional intracellular vitamin B\textsubscript{12} deficiency than analysis of vitamin B\textsubscript{12} in serum.\textsuperscript{1} Hence, homocysteine, vitamin B\textsubscript{12}, and folate are closely linked together in the so-called one-carbon cycle. The proposed mechanism relates to the methylation reactions involving homocysteine metabolism in the nervous system.\textsuperscript{2} Smith\textsuperscript{3} has suggested that the brain suffers from a double whammy from hyperhomocysteinaemia: cerebrovascular damage that triggers or potentiates the effect of Alzheimer pathology combined with a direct neurotoxic effect of homocysteine.

There is an ongoing debate about the contribution of vitamin B\textsubscript{12} deficiency to the dementia syndrome. The current article adds to those finding an effect on cognitive function as a result of vitamin B\textsubscript{12} treatment in patients with cognitive impairment and co-occurring vitamin B\textsubscript{12} deficiency. One of the most intriguing findings in the study is the low mean age of the investigated subjects. It must be considered unexpected to find such a high percentage of cases with cognitive symptoms in ages below 65 years. It would be very interesting to see added neuropsychological data on the dementia cases, to elucidate exactly in what cognitive domains there was an improvement. It is possible that such a study might contribute to a better understanding of role of vitamin B\textsubscript{12} deficiency in dementia evolution.

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References
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