

Serum Vitamin B12 Levels in Children Presenting with Vasovagal Syncope

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Objectives: Vasovagal syncope is usually defined as sympathetic-parasympathetic imbalance but its precise pathophysiology remains obscure. Vitamin B12 deficiency may cause neurologic deficits and affect the autonomic nervous system. This study aims to determine the serum concentrations of vitamin B12 in children presenting with vasovagal syncope.

Methods: This is a prospective review of 160 children presenting with vasovagal syncope. Patients with cardiac, neurologic, and psychiatric illness, chronic disease and arrhythmia, and any drug use that can alter cardiac conduction velocity heart rate and blood pressure were excluded. Subgroup analysis was done based on the results of head up tilt test.

Results: Head up tilt test gave positive results in 80 children and this test yielded negative results in the remaining 80 children. The tilt test positive children had significantly lower TSH concentrations ($p=0.06$), total iron binding capacity ($p=0.04$) and vitamin B12 levels ($p=0.01$). The prevalence of vitamin B12 deficiency was significantly higher in the tilt positive group (80% vs 52.5%, $p=0.001$). Out of 80 children with positive tilt test, 8 children (10%) showed cardioinhibitory response, 22 children (27.5%) demonstrated vasodepressor response, 24 children (30%) displayed mixed response and 26 children (32.5%) had postural orthostatic tachycardia syndrome (POTS). Erythrocyte sedimentation rate was significantly lower in the mixed response group than in the vasodepressor group (6.2 ± 0.8 mm/h vs 14.3 ± 2.5 mm/h, $p=0.001$). Serum vitamin B12 concentrations were significantly lower in the POTS group than in the vasodepressor group (240.8 ± 38.2 pg/ml vs 392.7 ± 27.1 pg/ml, $p=0.001$). The prevalence of serum vitamin B12 deficiency was significantly higher in the POTS group than in the vasodepressor group (92.3% vs 45.5%, $p=0.001$).

Conclusion: Vitamin B12 acts as a co-factor for three enzymes: (1) phentolamine N-methyltransferase which is needed for the conversion of noradrenaline to adrenaline, (2) catecholamine-O-methyltransferase which is required for the degradation of catecholamines, (3) methylmalonyl coenzyme A (CoA) mutase which catalyzes the conversion of methylmalonyl-CoA to succinyl-CoA in myelin synthesis. Vitamin 12 deficiency causes reduction in myelination, deceleration in nerve conduction and elevation in serum concentrations of noradrenaline. These factors may contribute to the impairment of autonomic functions which are involved in the pathogenesis of vasovagal syncope.