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SCIENTIFIC LETTER

Infantile Vitamin B12 Deficiency with Reversible Acquired Vision Loss

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To the Editor: We report three children (two boys, one girl) with developmental delay and acquired vision loss. They presented with loss of ability to visually fixate and follow for 2-4 wk (2, 3, 4 wk respectively). All babies were exclusively breast fed by vegetarian mothers. On examination, they had skin and hair changes reminiscent of the *"infantile tremor syndrome"*. They had no visual fixation or tracking. Pupils were normally reacting to light. They had temporal disc pallor on fundus examination. Their serum vitamin B12 levels (normal >211 pg/ml) were low (68, 144, 92 pg/ml), while plasma homocysteine was high (44, 90, 48 mmol/L). Their MRI brains showed frontotemporal dominant cortical atrophy and thinning of corpse callosum of varying severity. The P100 latency on visually evoked potentials was prolonged in all. Two of the mothers tested were also found to be vitamin B12 deficient. The children were treated with injection hydroxocobalamin 1 mg/d for 2 wk. The follow up vitamin B12 and homocysteine levels were normal. The babies recovered visual fixation and following by 4 wk of treatment. All had recovery in developmental milestones at their last follow up more than a year after treatment.

Visual loss due to Vitamin B12 deficiency usually presents as bilateral, painless, progressive vision loss. Vitamin B12 is crucial for myelin synthesis, and its deficiency is said to lead to an incorporation of abnormal fatty acids into neuronal lipids, altering nerve transmission [1]. Degeneration of optic nerve myelin lamellae and the damage of retinal ganglion cell axons is said to occur [2]. It has been suggested that cobalamin may function as an endogenous neuroprotectant for retinal ganglion cells through a superoxide-associated mechanism and its deficiency hence results in damage to the ganglion cells [3]. Rapid vision recovery following correction of deficiency makes this an important cause to remember.

Declarations

Conflict of Interest None.

References

1. Briani C, Dalla Torre C, Citton V, et al. Cobalamin deficiency: clinical picture and radiological findings. Nutrients. 2013;5:4521–39.
2. Roda M, di Geronimo N, Pellegrini M, Schiavi C. Nutritional optic neuropathies: state of the art and emerging evidences. Nutrients. 2020;12:2653.
3. Chan W, Almasieh M, Catrinescu MM, Levin LA. Cobalamin-associated superoxide scavenging in neuronal cells is a potential mechanism for vitamin B12-deprivation optic neuropathy. Am J Pathol. 2018;188:160–72.

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