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Karnataka Paediatric Journal

Letter to the Editor

Vitamin B12 deficiency presenting as infantile meningoencephalitis

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Received: 25 October 2022
Accepted: 14 November 2022
EPub Ahead of Print: 17 December 2022
Published: 08 March 2023

DOI
10.25259/KPJ_30_2022

Quick Response Code:

Dear Editor,

Various neurological manifestations described in vitamin B12 deficiency are developmental delay, neuroregression, involuntary movements, seizures, irritability, personality changes and encephalopathy.[1-3] We are reporting a rare case of B12 deficiency masquerading as infantile encephalitis.

A 3-month-old boy presented with fever in the past 10 days, seizures in the past 1 day, refusal of feeds, vomiting and reduced activity. On examination, the baby was febrile, lethargic, pale, with petechial rashes present all over the body, hepatomegaly with a span of 7 cm and increased tone with brisk reflexes. The initial clinical impression was sepsis with meningoencephalitis.

Investigations showed haemoglobin of 8.9 g/dL, normal total counts with thrombocytopenia (platelets: 70,000 µ/L), elevated mean corpuscular volume (98 fL), normal random blood sugar, electrolytes, liver function and renal function. The cerebrospinal fluid (CSF) analysis shows plenty of red blood cells with protein of 95 mg/dL, the glucose of 66 mg/dL and 140 leukocytes with 96% lymphocytes. The acute encephalitis panel for 17 pathogens of viral, bacterial and scrub typhus was negative. The C-reactive protein (56 mg/L) increased in the serum. The baby was treated with intravenous antibiotics; however, no clinical improvement was seen with treatment. MRI brain done showed diffuse cortical atrophy, predominantly in frontal and temporal lobes, bilateral symmetrical periventricular hyperintensities and bilateral temporal hyperintensities. Peripheral smear showed a megaloblastic anaemia picture, repeat investigations showed persistently elevated MCV. Serum vitamin B12 levels were 65.14. The baby was noted to have hypopigmented hair. The history reviewed revealed a vegetarian diet in the mother. Maternal examination showed mild pallor with hyperpigmented knuckles. Maternal investigations done showed low vitamin B12 levels of 123, with peripheral smear showing megaloblastic anaemia. With positive evidence of vitamin B12 deficiency in the mother and the baby, with supportive MRI, the baby was treated with intravenous vitamin B12, 1000 micrograms for 14 days, improved clinically with the return of social smile, the disappearance of petechial lesions, resolved thrombocytopenia and discharged on oral Vitamin B12 supplements. The baby was followed up subsequently and noted developmental delay. At present, baby is 10 months and has achieved sitting without support, immature grasp, monosyllables and stranger anxiety. Vitamin B12 levels have normalized.

Vitamin B12 acts as a cofactor in the conversion of methylmalonyl-CoA to succinyl-CoA, failing which leads to the accumulation of methyl malonyl-CoA which is responsible for neurological manifestations in B12 deficiency.[4] Vitamin B12 deficiency can also present as acute encephalitis in

infants, which is precipitated by infection. A clue to the diagnosis lies in the history of prolonged breastfeeding, history of a vegetarian diet in the mother with evidence of b12 deficiency in blood tests, physical examination of the mother and infant for hair and skin changes and organomegaly. Excellent response to B12 supplements, in our case, confirmed the diagnosis and helped in differentiating from infective encephalitis.

Vitamin B12 deficiency usually manifests with recurrent infection; sometimes, it will be recognised when children present with infections. In this case, vitamin B12 deficiency presented with infection with predominant neurological features of seizures and encephalopathy. Vitamin B12 deficiency can cause encephalopathy and seizures. The presence of pleocytosis in the traumatic CSF with normal sugar should be interpreted carefully and may require repeat lumbar puncture before labelling as neuroinfections. Vitamin B12 deficiency in infants can present as acute encephalitis. Hence, vitamin B12 deficiency should be included in the differential diagnosis of acute infantile encephalitis.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. İncecik F, Hergüner MÖ, Altunbaşak Ş, Leblebisatan G. Neurologic findings of nutritional Vitamin B 12 deficiency in children. Turk J Pediatr 2010;52:17-21.
2. Black MM. Effects of Vitamin B12 and folate deficiency on brain development in children. Food Nutr Bull 2008;29:S126-31.
3. Korenke GC, Hunneman DH, Eber S, Hanefeld F. Severe encephalopathy with epilepsy in an infant caused by subclinical maternal pernicious anemia: Case report and review of the literature. Eur J Pediatr 2004;163:196-201.
4. Gibson RS. Principles of Nutritional Assessment. 2nd ed. New York, USA: Oxford University Press; 2005.

How to cite this article: Gowda VK, Ashwini KR, Srinivasan VM. Vitamin B12 deficiency presenting as infantile meningoencephalitis. Karnataka Paediatr J 2022;37:96-7.