CASE REPORT

Delay Neurological Development in Two Children with Megaloblastic Anemia

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ABSTRACT:.

Vitamin B12 deficiency in infants often produces haematological and neurological deficits, including macrocytic anaemia ،neurodevelopmental delay or regression, irritability, weakness ،hypotonia ، ataxia, apathy, tremor, and seizures. The diagnosis of vitamin B12 deficiency can be difficult when the typical macrocytic anaemia is absent .(١))Infantile vitamin B-12 deficiency with ineffective haematopoiesis and degeneration of nervous tissue has been reported in breast fed infants of mothers on strict vegetarian diets(٢) Adults may tolerate vitamin B-12 deficient diets for many years without apparent symptoms due to their endogenous stores. In contrast, infants have very limited hepatic reserves of vitamin B-12 and so may develop symptoms of deficiency within month's.(٢)we report two cases in which they were diagnosed as cerebral palsy in early life. first case was 9 month old female baby presented with pallor for last 3 months of age, floppiness ,regress of acquired millstone ,exclusive breast feed for a vegan mother ,diagnosed as megaloblastic anemia second case was 16 months old male baby presented with apathiness، he was exclusively breast feed till this age with minimum complementary food.

KEY WORDS : megloblstic anemia ،breast feed ز

CASE REPORT:

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A nine month old female admitted to Raparin teaching hospital in Erbil, Iraq .because of sever anemia and floppiness, she was normal till 6 month of age. The patient was exclusively breast fed until the age of 8 months, when her mother started to feed her cereals ,She took only small amounts of these complementary foods, for example, one date per day. ,her mother was vegan and eat small amount of chicken meat once in week ,she was completely vegetarian during pregnancy ,and without tonic supplementation this is her first child, She reported that her child developed normally during the first 6months; she could sit with support ,later she start to regress in her acquired millstone over these 3 months ,she had complete head lag and unable to sit with support, and diagnosed as cerebral palsy and sent for special center for handicapped child for rehabilitation. On admission her weight was 6 kg and length 65cm (both below 10th percentile) Neurological examination revealed apathy and profound hypotonia with complete head lag when pulled to sitting position and areflexia .Laboratory analyses revealed a hemoglobin level of 5g/dL and mean corpuscular volume of 100fL blood film showed pancytopenia bone marrow aspiration revealed megaloblastic change and . both B12 and folate level were estimated ,B12 level (80 Pg/L). CT scan of brain shows evidence of brain atrophy ,B12 supplementation started, improvement of tone and alertness observed in first week and the patient was followed, she has completely recovered.

CASE 2:

A 16 month old male presented with pallor for the last 2 months, associated with vomiting, poor feeding, failure to thrive, decreased activities, loss of interest, delayed and regressed milestone, he was normal before that. The baby is exclusively breast feeding since birth till now no any complementary food added, his mother restricted vegan and also appear pale .he had history of tonic clonic convulsion last more than 10 minute , and EEG recorded anbonrmal epileptiform pattern so treated by Depakin

His weight height and OFC was 9kg, 71 cm, and 43 cm respectively (all below 3rd percentile(Round unexpressive face, Pale skin, conjunctiva and mucous membrane .

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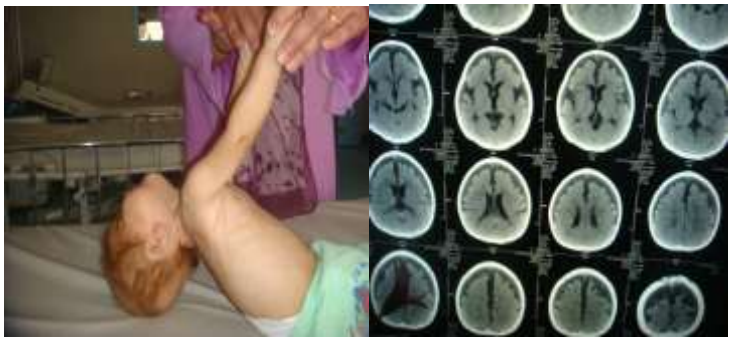
Fair hair but not easily detached, Neurological examination revealed apathy and mild hypotonia (he had mild head lag between 30-45^{o} during pulling to sitting position) and hyporeflexia . Laboratory analyses revealed a hemoglobin level of 5.7/dL and mean corpuscular volume of 110fL ،reticulocyte count was (0.5 %) blood film showed macrocytosis ،serum ferritin was 75 mg/dl , bone marrow aspiration revealed megaloblastic change . both B12 and folate level estimated for both mother and baby ,B12 level (95 Pg/L)normal serum ferrittin level .and CT scan of brain shows widening of subarachnoid space at temporo-parieto-frontal region due to localized brain atrophy, B12 level estimated in mother also and was very low ,B12 supplementation started , for both mother and baby ,baby 's alertness improvement was observed in day 4 after establishment of parenteral B12، reticulocytosis) .(%۹Serum ferritin started to decline and iron supplementation start concomitantly.

DISCUSSION :

The most common cause of cobalamin deficiency in infants and young children is maternal dietary deficiency, which generally manifests in breastfed infants at age 4--8 months. This deficiency is difficult to diagnose because of nonspecific symptoms .[۹] Serious hematological, metabolic and neurological complications owing to the nutritional deficiency of vitamin B12 may occur in infants of mothers on a strict vegetarian diet. The apparent morphological changes of neural tissues induced by vitamin B12 deficiency resolve rapidly with treatment. Atrophy of the optic nerve resolved completely after six months of treatment .[۳] Stollhoff and Schulte reported a case of a patient with severe neurological changes, abnormal EEG, and marked frontal lobe atrophy on cranial computed tomography (CCT .[٤،٢] (Vitamin B12 treatment resulted in rapid clinical response, a normal EEG after five weeks, and a normal CCT after 10 weeks .Neurological involvement often occurs along with macrocytic anemia but can occur in the absence of anemia or macrocytosis .It is unclear why vitamin B12 deficiency leads to neurological disease in some and hematological disease in others .Methylenetetrahydrofolate reductase) MTHFR) polymorphism has been postulated to protect the vitamin B12-deficient patients against anemia and homozygosity for MTHFR C677T gene could cause the dissociation between hematological and neurological disease seen in some patients with vitamin B12 deficiency. The exact mechanism involved in epileptogenesis due to cobalamin deficiency is not clear. It is likely that cerebral neurons with destroyed myelin sheaths are more susceptible to the excitatory effects of glutamate [٧،٦،٥,8].

CONCLUSION:

Early diagnosis of vitamin B12 deficiency is very important because. Psychomotor and mental damage could be reversible with appropriate treatment.



Baby 1

Baby 2

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