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A RARE CASE OF ELDERLY SUDANESE WOMEN WITH CHOREA RESPONSE TO VITAMIN B12 INJECTION

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ABSTRACT

Introduction: Deficiency of vitamin B12 produces protean effects on the nervous system, most commonly neuropathy, myelopathy, cognitive and behavioral symptoms, and optic atrophy. Involuntary movements comprise a relatively rare manifestation of this readily treatable disorder. Both adults and infants deficient in vitamin B12 may present with chorea, tremor, myoclonus, Parkinsonism, dystonia, or a combination of these, which may precede diagnosis or become apparent only a few days after parenteral replacement therapy has begun. The pathogenesis of these movement disorders shows interesting parallels to certain neurodegenerative conditions. The clinical syndrome responds well to vitamin B12 supplementation in most cases, and an early diagnosis is essential to reverse the haematological and neurological dysfunction characteristic of this disorder **Case Study:** An 86-year-old woman with a history of cerebrovascular accident (CVA) and atrial fibrillation was admitted to our hospital Almak Nemer University hospital complaining of disturbance of consciousness and chorea-like involuntary movements of the extremities. The patient was diagnosed with encephalopathy due to vitamin B12 deficiency. Involuntary movements improved with parenteral vitamin B12 supplementation. **Discussion:** Vitamin B12 serve as cofactor for forming methionine from homocystiene. Decrease Vitamin B12 level lead to high level of homocystiene which appear to be toxic to endothelial cells. Methionine is further metabolized to S-adenosylmethionine (SAM). S-adenosylmethionine SAM influences serotonin, norepinephrine, and dopamine synthesis. This suggests that, in addition to structural consequences of vitamin B-12 deficiency, functional effects on neurotransmitter synthesis that may be relevant to mental status changes may occur. **Conclusion:** The present case is considered valuable in identifying the pathophysiology of involuntary movement due to vitamin B12 deficiency.

KEYWORD: Chorea, vitb12, elderly women.

1-5 INTRODUCTION

Megaloblastic anaemia is characterized by the presence in the bone marrow of erythroblasts with delayed nuclear maturation because of defective DNA synthesis (megaloblasts).

Megaloblasts are large and have large immature nuclei. The nuclear chromatin is more finely dispersed than normal and has an open stippled appearance. In addition, giant metamyelocytes are frequently seen in megaloblastic anemia. These cells are about twice the size of normal cells and often have twisted nuclei.

Megaloblastic changes occur in:
- Vitamin B12 deficiency or abnormal vitamin B12 metabolism
- Folic acid deficiency or abnormal folate metabolism
- Other defects of DNA synthesis, such as congenital enzyme deficiencies in DNA synthesis (e.g. erotic aciduria), or resulting from therapy with drugs interfering with DNA synthesis (e.g. hydroxycarbamide (hydroxyurea), azathioprine, zidovudine – AZT)
- Myelodysplasia due to dyserythropoiesis.

2-5 CASE REPORT

An 86-year-old woman was admitted to our hospital Almek Nimer University hospital complaining of disturbance of consciousness and chorea-like involuntary movements of the extremities.

Patient known to have A.F and CVA which lead to the left side weakness.Also had decrease in her oral intake for weeks.

On examination patient subconscious and disoriented and his right side involuntary movement (chorea like movement). Also the patient pale and slightly jaundice.

Had irregular irregular pulse 70 bpm, blood pressure 110/70 .

Left side upper and lower limbs motor grade 0 hyper-reflxia, sensory also lssing on the right side Right side involuntary movement motor grade 5 and sensory intact with hyperreflexia.

Other systems are intact.
The investigations complete blood picture (CPC) showed low hemoglobin 9.3 g/dl, high MCV 99.1 fl high MCH 32.6 PG, peripheral blood picture showed hypersegmented neutrophils where the others within the normal range.

The patient was diagnosed with chorea due to vitamin B12 deficiency.

Involuntary movements improved with parenteral vitamin B12 supplementation. The present case is considered valuable in identifying the pathophysiology of involuntary movement due to vitamin B12 deficiency.

3-5 DISCUSSION

Vitamin B12 is synthesized by certain microorganisms, and humans are ultimately dependent on animal sources. It is found in meat, fish, eggs and milk, but not in plants. Vitamin B12 is not usually destroyed by cooking. The average daily diet contains 5–30 μg of vitamin B12, of which 2–3 μg is absorbed. The average adult stores some 2–3 mg, mainly in the liver, and it may take 2 years or more after absorptive failure before B12 deficiency develops, as the daily losses are small (1–2 μg).

Vitamin B12 deficiency

There are a number of causes of B12 deficiency and abnormal B12 metabolism. The most common cause of vitamin B12 deficiency in adults is pernicious anaemia. Malabsorption of vitamin B12 because of pancreatitis, coeliac disease or treatment with metformin is mild and does not usually result in significant vitamin B12 deficiency.[1]

Chorea

The ad hoc Committee on Classification of the World Federation of Neurology has defined chorea as "a state of excessive, spontaneous movements, irregularly timed, non-repetitive, randomly distributed and abrupt in character. These movements may vary in severity from restlessness with mild intermittent exaggeration of gesture and expression, fidgeting movements of the hands, unstable dance-like gait to a continuous flow of disabling, violent movements."[2]

There are a wide variety of possible causes of chorea. These include:
1-Hereditary
• Huntington's disease (HD) and HD-like syndromes
• Wilson's disease
• Neuroacanthocytosis
• Dentato-rubro-pallidoluysian atrophy
• Benign hereditary chorea
• Paroxysmal dyskinesias
2- Cerebral birth injury (including kernicterus)
3- Cerebral trauma
4- Drugs
• Levodopa
• Antipsychotics
• Anticonvulsants
• Oral contraceptive
5-Metabolic
• Disorders affecting thyroid, parathyroid, glucose, sodium, calcium and magnesium balance
• Pregnancy
6- Autoimmune
• Post-streptococcal(Sydenham's chorea)
• Antiphospholipid antibody syndrome
• Systemic lupus erythematous (SLE)
7- Structural lesions of basal ganglia (usually caudate)
• Vascular
• Demyelination
• Brain tumor.[2]

In the cytoplasm, methylcobalamin (figure 1) serves as cofactor for methionine synthesis by allowing transfer of a methyl group from 5-methyl-tetrahydrofolate (5-methyl-THF) to homocysteine (HC), forming methionine and demethylatedtetrahydrofolate (THF). This results in reduction in serum homocysteine, which appears to be toxic to endothelial cells. Methionine is further metabolized to S-adenosylmethionine (SAM).

S-adenosylmethionine SAM influences serotonin, norepinephrine, and dopamine synthe sis. This suggests that, in addition to structural consequences of vitamin B-12 deficiency, functional effects on neurotransmitter synthesis that may be relevant to mental status changes may occur. Parenthetically, S-adenosylmethionine SAM is being studied as a potential antidepressant.

Another possible cause of neurologic manifestations involves the other metabolically active form of cobalamin, adenosylcobalamin, a mitochondrial cofactor in the conversion.

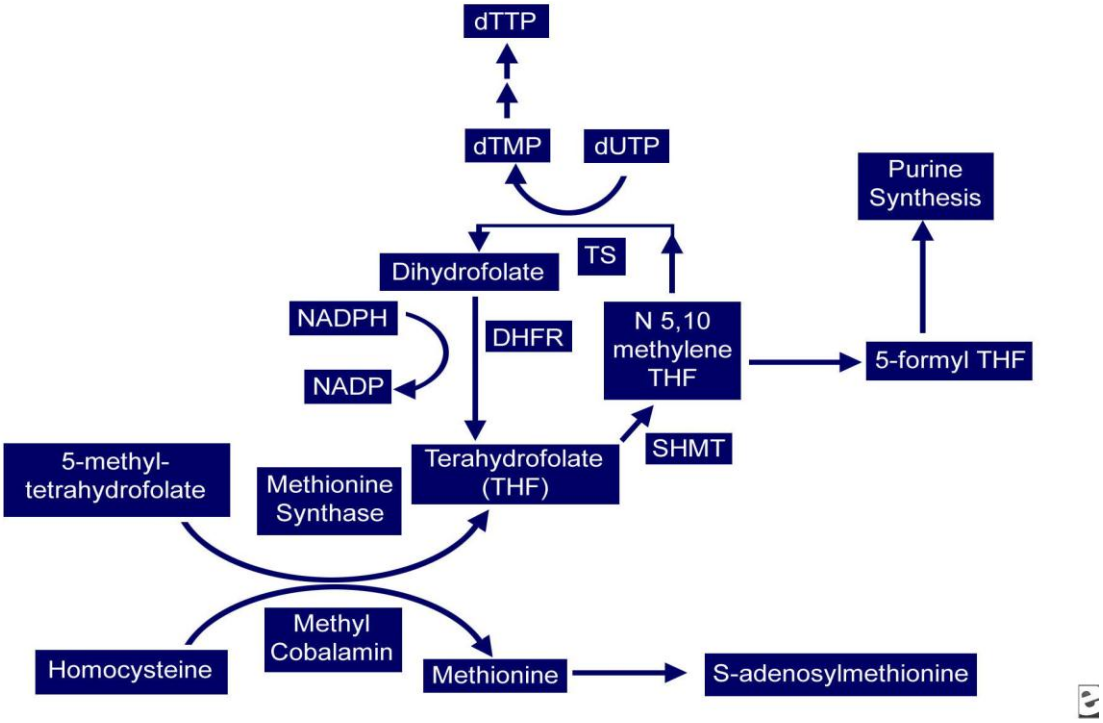

Figure 1.

of L-methylmalonyl CoA to succinyl CoA. Vitamin B-12 deficiency leads to an increase in L-methylmalonyl-CoA, which is converted to D-methylmalonyl CoA and hydrolyzed to MMA. Elevated MMA results in abnormal odd chain and branched chain fatty acids with subsequent abnormal myelination, possibly leading to defective nerve transmission.[3]

A simple model of basal ganglia function states that dopaminergic and GABAergic impulses from the substantianigra and motor cortex, respectively, are funneled through the pallidum into the motor thalamus and motor cortex. These impulses are modulated in the striatum via two segregated, parallel, direct and indirect loops through the medial pallidum and lateral pallidum/subthalamic nucleus. Subthalamic nucleus activity drives the medial pallidum to inhibit cortex-mediated impulses, thereby inducing parkinsonism. Absent subthalamic nucleus inhibition enhances motor activity through the motor thalamus, resulting in abnormal involuntary movements such as dystonia, chorea, and tics.[4]

4-5 CONCLUSIONS

Vitamin B12 deficiency has complex pathogenesis and possible multiple causes with different Presentations. Vitamin B12 deficiency cause disturbed neurotransmitter and hence lead to involuntary movement. Same treatment should be given for all, after exclusion of other causes. This case illustrate the association between. megaloblastic anemia and chorea.

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