Vitamin B-12 and Psychiatric Disorders

J Indian Med Assoc. 2007 Jul;105(7):395-6.

Psychiatric presentations of vitamin B 12 deficiency.

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Vitamin B12 deficiency has been implicated in various psychiatric conditions for a long time. The association could be primary, secondary to the psychiatric disorder, or even just coincidental. However, left untreated, the deficiency can delay or preclude recovery. Hence early recognition is important, especially when the traditional manifestations of B12 deficiency like anaemia, macrocytosis or spinal cord symptoms are not prominent. Three cases are presented here where vitamin B12 deficiency and psychiatric symptomatology were coexistent, and the patients recovered only on a combination of B12 supplementation and psychiatric medication.

PMID: 18178994 [PubMed - indexed for MEDLINE]

J Assoc Physicians India. 2008 Feb;56:115-6.

Chronic psychosis associated with vitamin B12 deficiency.

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B12 deficiency is widely prevalent and usually presents with haematologic and neuropsychiatric manifestations. Psychiatric symptoms seldom precede anaemia and present as the principal manifestation of B12 deficiency. A report an unusual presentation of long standing psychotic symptoms without anaemia in a 31 year old male, who presented to a tertiary care psychiatric facility. His physical examination revealed hyper pigmentation of extremities and posterior column involvement. Laboratory investigations confirmed normal haemoglobin and low serum B12 levels. He recovered dramatically with short term anti psychotic medication and intramuscular cobalamin supplementation. He remained asymptomatic and functionally independent at two years follow up.

Publication Types:

- Case Reports

PMID: 18472513 [PubMed - indexed for MEDLINE]

Psychiatr Clin North Am. 2007 Dec;30(4):739-59.

Hematologic problems in psychosomatic medicine.

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Vitamin B12 deficiency is associated with problems in cognition, mood, psychosis, and less commonly, anxiety. Folate deficiency primarily is associated with problems in mood. Patients who have sickle cell disease, a disease of chronic pain, experience difficulties with depression, anxiety, stigma, and are at risk for substance abuse and dependence. Patients with hemophilia have benefited from advances in treatment; however, their morbidity and mortality were compounded in those who received blood products contaminated with HIV, or hepatitis B and C. Psychiatrists who practice psychosomatic medicine should expect to encounter patients with the above problems, as they are frequently seen in medical settings. Finally, most of the commonly used psychotropic medications have uncommon but potentially important hematologic side effects or may interact with the anticoagulants used in medically ill patients.

Publication Types:

- Review

PMID: 17938043 [PubMed - indexed for MEDLINE]

Prim Care Companion J Clin Psychiatry. 2007;9(3):238.

Vitamin b(12) deficiency manifested as mania: a case report.

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PMID: 17632664 [PubMed - in process]

PMCID: PMC1911186

Gen Hosp Psychiatry. 2008 Mar-Apr;30(2):185-6.

Role of vitamin B12 in depressive disorder--a case report.

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Vitamin B12 deficiency anemia may have psychiatric manifestations preceding the hematological symptoms. Although a variety of symptoms are described, there are only sparse data on the role of vitamin B12 in depression. We report a case of vitamin B12 deficiency presenting with recurrent episodes of depression.

Publication Types:

- Case Reports

PMID: 18291301 [PubMed - indexed for MEDLINE]

Pediatr Emerg Care. 2008 Aug;24(8):538-41.

Neurological consequences of vitamin B12 deficiency and its treatment.

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In developed countries, the vitamin B12 deficiency usually occurs in children exclusively breast-fed, whose mothers are vegetarians, causing low stores of vitamin B12. Symptoms of vitamin B12 deficiency appear during the second trimester of life and include failure to thrive, lethargy, hypotonia, and arrest or regression of developmental skills. A megaloblastic anemia can be present. One half of the infants exhibit abnormal movements before the start of treatment with intramuscular cobalamin, which disappear 1 or 2 days after. More rarely, movement disorders appear a few days after treatment, whereas neurological symptoms are improving. These abnormal movements can last for 2 to 6 weeks. If not treated, vitamin B12 deficiency can cause lasting neurodisability. Therefore, efforts should be directed to preventing deficiency in pregnant and breast-feeding women on vegan diets and their infants by giving them vitamin B12 supplements. When preventive supplementation has failed, one should recognize and treat quickly an infant presenting with failure to thrive and delayed development.

Publication Types:

- Case Reports

PMID: 18708898 [PubMed - indexed for MEDLINE]

Nutr Rev. 2008 May;66(5):250-5.

Effect of vitamin B12 deficiency on neurodevelopment in infants: current knowledge and possible mechanisms.

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Severe vitamin B(12) deficiency produces a cluster of neurological symptoms in infants, including irritability, failure to thrive, apathy, anorexia, and developmental regression, which respond remarkably rapidly to supplementation. The underlying mechanisms may involve delayed myelination or demyelination of nerves; alteration in the S-adenosylmethionine:S-adenosylhomocysteine ratio; imbalance of neurotrophic and neurotoxic cytokines; and/or accumulation of lactate in brain cells. This review summarizes the current knowledge concerning infantile vitamin B(12) deficiency, including a pooled analysis of case studies of infants born to mothers with untreated pernicious anemia or a strict vegetarian lifestyle and a discussion of the mechanisms that may underlie the manifestations of deficiency.

Publication Types:

- Review

PMID: 18454811 [PubMed - indexed for MEDLINE]

Med Secoli. 2007;19(1):9-18.

New pathogenesis of the cobalamin-deficient neuropathy.

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Subacute combined degeneration (SCD) is considered the neurological counterpart of pernicious anaemia because it is the paradigmatic neurological manifestation of acquired vitamin B12 (cobalamin (Cbl)) deficiency in adulthood. Hitherto, the theories advanced to explain the pathogenesis of SCD have postulated a causal relationship between SCD lesions and the impairment of either or both of two Cbl-dependent reactions. We have identified a new experimental model, the totally gastrectomised (TGX) rat, to reproduce the key morphological features of the disease, and found new mechanisms responsible for the pathogenesis of SCD. We have demonstrated that the neuropathological lesions in TGX rats are not only due to mere vitamin withdrawal but also to the overproduction of the myelinolytic tumour necrosis factor (TNF)-alpha, nerve growth factor, the soluble(s) CD40:sCD40 ligand dyad, and the reduced synthesis of the neurotrophic agents, epidermal growth factor and interleukin-6. Cbl replacement treatments normalised all of these abnormalities.

Publication Types:

- Historical Article

PMID: 18447164 [PubMed - indexed for MEDLINE]

J Med Case Reports. 2008 Mar 21;2:90.

Neuropathy caused by B12 deficiency in a patient with ileal tuberculosis: A case report.

Toosi TD, **Shahi F**, **Afshari A**, **Roushan N**, **Kermanshahi M**.

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ABSTRACT: INTRODUCTION: Vitamin B12 deficiency can result in macrocytic anemia. Neurologic abnormalities of B12 deficiency include sensory deficits, loss of deep tendon reflexes, movement disorders, neuropsychiatric changes and seizures. Segmental involvement of the distal ileum, such as in tuberculosis, can cause vitamin B12 deficiency. To our knowledge, macrocytic anemia with unusual manifestations such as brain atrophy and seizures due to intestinal tuberculosis has not been reported in the literature. CASE PRESENTATION: A 14-year-old girl presented with complaints of paraplegia, ataxia, fever and fatigue that had started a few months earlier and which had been getting worse in the last three weeks. Her laboratory results were indicative of macrocytic anemia with a serum B12 level <100 (normal, 160-970) pg/ml and hypersegmented neutrophils. Her MRI findings showed brain atrophy. Her fever workup eventually led to the diagnosis of tuberculosis which was documented by bone marrow aspiration smear & culture. A small bowel series showed that tuberculosis had typically involved the terminal ileum which had resulted in vitamin B12 deficiency. She was treated for vitamin B12 deficiency and tuberculosis. Her fever ceased and her hemoglobin level returned to normal. At present, she can eat, write, and speak normally as well as walk and ride a bicycle. CONCLUSION: Vitamin B12 deficiency should be considered in patients with neurologic features such as paresthesia, sensory deficits, urinary incontinence, dysarthria, and ataxia. The underlying cause of B12 deficiency should be determined and treated to obviate the patients' need for long term vitamin B12 therapy.

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PMCID: PMC2329654

Dev Med Child Neurol. 2007 Oct;49(10):774-6.

West syndrome in an infant with vitamin B12 deficiency in the absence of macrocytic anaemia.

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Vitamin B(12) 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 B(12) deficiency can be difficult when the typical macrocytic anaemia is absent. We report the case of a 10-month-old female diagnosed with West syndrome associated with vitamin B(12) deficiency but without macrocytic anaemia caused by nutritional inadequacy in the mother. The patient's motor skills and cognitive development were normal until she was 9 months old, when she began to exhibit a series of sudden flexions of the head, trunk, arms, and legs. She was exclusively breast-fed and had received no vitamin supplementation. Results of electroencephalography (EEG) indicated modified hypsarrhythmia and the patient was diagnosed as having West syndrome. Synthetic adrenocorticotropic hormone was administered and although her spasms had resolved, the patient remained apathic and could not sit without assistance. EEG results indicated generalized slow activity. After she was diagnosed as having vitamin B(12) deficiency, parenteral treatment with vitamin B(12) was initiated. Her symptoms resolved and EEG was completely normal. When she was 20 months old she exhibited an age-appropriate developmental and neurological profile. To our knowledge, this is the first report of West syndrome as a presenting symptom of vitamin B(12) deficiency.

Publication Types:

- Case Reports

PMID: 17880648 [PubMed - indexed for MEDLINE]

Fortschr Neurol Psychiatr. 2007 Sep;75(9):515-27.

[Review of the role of hyperhomocysteinemia and B-vitamin deficiency in neurological and psychiatric disorders--current evidence and preliminary recommendations]

[Article in German]

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Elevated concentration of total homocysteine (Hcy) in plasma (> 12 micromol/l) is a risk factor for several diseases of the central nervous system. Epidemiological studies have shown a dose-dependent relationship between concentrations of Hcy and the risk for neurodegenerative diseases. Hcy is a marker for B-vitamin deficiency (folate, B12, B6). Hyperhomocysteinemia (HHcy) causes hypomethylation which is an important mechanism that links Hcy to dementia. Supplementation with vitamins B aims at reducing the risk of neurodegenerative diseases. Current evidence suggests that Hcy-lowering treatment has a positive effect for the secondary and primary prevention of stroke. HHcy is very common in patients with Parkinson disease particularly those who receive L-dopa treatment. Furthermore, a positive association has been reported between HHcy and multiple sclerosis. Moreover, HHcy and vitamin B deficiency are reported to have a causal role in depression, and epilepsy. In addition several anti-epileptic drugs cause secondary HHcy. Therefore, sufficient intakes of the vitamins are recommended for patients who have already developed neuropsychiatric diseases. Vitamin B deficiency should be suspected in children with development disorders, failure to thrive and unexplained neurological manifestations. Elderly people are also an important at-risk group where vitamin B deficiency and HHcy have been linked to neurodegenerative diseases. Treatment with folate, B12, and B6 can improve cerebral function. Preventive vitamin B supplementation and sufficient intake seem very important for secondary and primary prevention of neuropsychiatric disorders, especially in subjects with a low intake or status of the vitamins.

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Clin Chem. 2007 Feb;53(2):326-33. Epub 2007 Jan 2.

Biomarkers of folate and vitamin B12 are related in blood and cerebrospinal fluid.

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BACKGROUND: B-vitamins (folate, B(12)) are important micronutrients for brain function and essential cofactors for homocysteine (HCY) metabolism. Increased HCY has been related to neurological and psychiatric disorders. We studied the role of the B-vitamins in HCY metabolism in the brain. METHODS: We studied blood and cerebrospinal fluid (CSF) samples from 72 patients who underwent lumbar puncture. We measured HCY, methylmalonic acid (MMA), and cystathionine by gas chromatography-mass spectrometry; S-adenosylmethionine (SAM) and S-adenosylhomocysteine (SAH) by liquid chromatography-tandem mass spectrometry; and the B-vitamins by HPLC or immunoassays. RESULTS: Concentrations were lower in CSF than serum or plasma for HCY (0.09 vs 9.4 micromol/L), SAH (13.2 vs 16.8 nmol/L), cystathionine (54 vs 329 nmol/L), and holotranscobalamin (16 vs 63 pmol/L), whereas concentrations in CSF were higher for MMA (359 vs 186 nmol/L) and SAM (270 vs 113 nmol/L; all P <0.05). CSF concentrations of HCY correlated significantly with CSF folate (r = -0.46), CSF SAH (r = 0.48), CSF-albumin (r = 0.31), and age (r = 0.32). Aging was also associated with lower concentrations of CSF-folate and higher CSF-SAH. The relationship between serum and CSF folate depended on serum folate: the correlation (r) of serum and CSF-folate was 0.69 at serum folate <15.7 nmol/L. CSF concentrations of MMA and holotranscobalamin were not significantly correlated. CONCLUSIONS: CSF and serum/plasma concentrations of vitamin biomarkers are significantly correlated. Older age is associated with higher CSF-HCY and CSF-SAH and lower CSF-folate. These metabolic alterations may be important indicators of low folate status, hyperhomocysteinemia, and neurodegenerative diseases.

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