Nutritional Deficiencies and Neurological Diseases

By

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Vitamin B_{12} Deficiencies

- refers to low blood levels of vitamin B_{12}.
- A wide variety of signs and symptoms may occur.
- High prevalence among elderly (15%-25%) and persons with gastrointestinal disease or malnutrion

Syndrome of subacute combined degeneration

1) Insidious onset of paresthesias in hands and feet, sensation of generalized weakness and fatigue.

2) With progression: gait abnormalities due to combination of sensory ataxia (posterior column involvement) and spastic paraparesis (spasticity, hyperreflexia, extensor plantar responses accompanying spastic paraparesis)

3) Weakness most prominent in legs

- Cognitive decline, mental slowing, personality changes, psychiatric disease (depression, psychosis)

- Systemic features: megaloblastic anemia (may lead to pancytopenia), hyperpigmented fingernails, diarrhea, glossitis

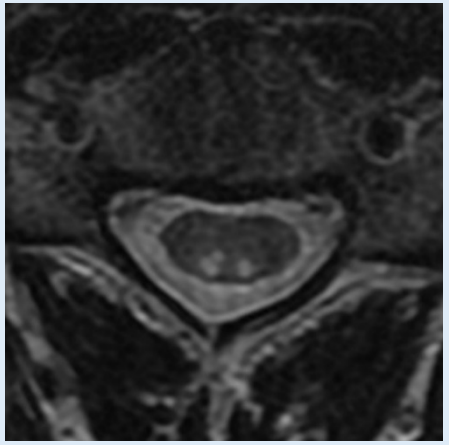
Diagnosis

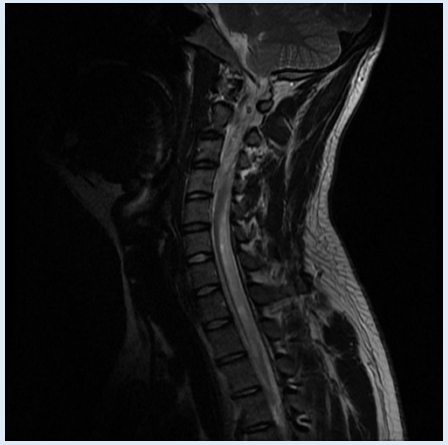
1) Megaloblastic anemia

2) Reduced serum levels of vitamin B12 (may be normal or borderline low)

3) Increased serum levels of homocysteine and methylmalonic acid

4) Hyperintense T2 abnormalities involving posterior columns, predominantly in lower cervical and upper thoracic cord





Treatment

Intramuscular cyanocobalamin

Vitamin E Deficiency

Spinocerebellar syndrome (cerebellar ataxia and large-fiber sensory loss), areflexia, ataxic dysarthria, cerebellar and sensory gait ataxia, extensor plantar responses, hemolytic anemia, retinitis pigmentosa

Vitamin B6

- Often due to intake of isoniazid, hydralazine, or penicillamine
- Distal sensorimotor peripheral neuropathy (predominant features of sensory symptoms and axonal degeneration)
- Treatment: slow improvement with withdrawal of offending drug or initiation of low-dose vitamin B6 supplementation
- Primary prevention: coadministration of vitamin B6 prevents this neuropathy

Thiamine Deficiency

- Thiamine deficiency refers to the lack of thiamine pyrophosphate, the active form of vitamin B-1.

- Thiamine pyrophosphate acts as a coenzyme in carbohydrate metabolism.

- Thiamine requirements are greatest during periods of high metabolic demand and/or high glucose intake: this may partly explain occurrence of Wernicke’s encephalopathy with administration of intravenous glucose to a thiamine-deficient patient

Laboratory diagnosis

1) Decreased serum and urine levels of thiamine
2) Decreased erythrocyte transketolase activity (functional assay)
3) Increased serum pyruvate levels
4) Electromyographic evidence of axonal, distal peripheral neuropathy

Beriberi

a. Length-dependent, axonal, sensorimotor peripheral neuropathy with distal sensory loss, paresthesias, weakness (termed “dry beriberi” in absence of cardiac involvement)

b. Associated with cardiac involvement: cardiomyopathy, arrhythmias, congestive heart failure (termed “wet beriberi”)

Wernicke-Korsakoff Syndrome

- Usually in setting of chronic alcoholism or non-alcohol related malnutrition

Wernicke's encephalopathy triad

- Acute presentation of delirium and global confusional state
- ataxia (axial, not appendicular)
- nystagmus and ophthalmoparesis (especially cranial nerve VI palsy, commonly bilateral ± other cranial nerves), sometimes followed by Korsakoff's syndrome

Classic triad is not present in up to 90% of cases, so it is important to have a low threshold for treating alcoholics with any *one* of the following symptoms:

1) Alteration of consciousness (ranging from acute confusion to coma)
2) Ataxia
3) Ophthalmoparesis
4) Nystagmus
5) Hypothermia
6) Hypotension
7) Memory deficits

- Treatment of Wernicke’s encephalopathy with thiamine supplements reverses ophthalmoparesis and ataxia within minutes to hours, with a more gradual recovery of the nystagmus; impairment of memory is slower and often incomplete

Korsakoff ’s syndrome

a. Chronic amnestic syndrome, both anterograde and retrograde components for events as far back as several years

b. Is often the “chronic phase” of acute Wernicke’s encephalopathy

- Amnestic syndrome associated with Korsakoff’s syndrome is not reversed with thiamine supplementation, but further progression is usually halted