Neurological Manifestations of Nutritional Deficiencies

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Abstract

Vitamins and trace minerals are essential constituents of human diet as they are either not synthesized or synthesized in only small quantities in the human body. Only small amount of these substances are needed for carrying out essential biochemical reactions. Deficiency of these nutrients can cause protean manifestations. Nervous system occupies a position of special interest and importance in these deficiency disorders .Nutritional deficiency is responsible for significant morbidity and considerable mortality if not detected early Neurological disorders .because of nutritional deficiencies are important as they can be preventable and curable if treated in time .Majority of vitamins can affect nervous system by one or other way but those of special importance are components of vitamin-B complex ,vitamin-E and vitamin-D .Inspite of great advances in the medicine and increased awareness of importance of proper nutrition in health these disorders are still of major concern in developing countries .Because of enrichment of food with these essential nutrients there is considerable reduction in prevalence of these disorders in developed countries .It is extremely essential to have high index of suspicion for detecting these disorders .

Introduction

Nutritional deficiency is the problem of global concern. Despite the great advances since the turn of 20th century nutritional deficiency is still a serious world wide problem & it is almost endemic in many parts of world. Poverty, dietary deficiency & coexisting infections contribute to nutritional deficiency in developing countries while chronic alcoholism is of main concern in developed world. Another very important cause of malnutrition which is becoming common nowadays in western world is bariatric surgery which is increasingly used for treating obesity.

Under nutrition causes a wide spectrum of neurological diseases. Although deficiency of almost any nutrient can lead to some kind of neurological symptoms , the B vitamin,(thiamine , pyridoxine , cyanocobalmine) vitamin E, Folic acid are the most important to nervous system Nutritional deficiencies are often multiple and may have multi organ involvement .All the parts of the nervous system such as cerebrum, cerebellum, spinal cord, peripheral nerves and muscles may be affected solely or in combination

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TABLE 1* - Neurological Manifestations in Deficiency Diseases (16)

Neurological Manifestations	Nutritional Deficiencies
Seizures	Pyridoxine
Myelopathy	Vitamin B12, vitamin E, folate
Myopathy	Vitamin D, vitamin E
Peripheral neuropathy	VitaminB1,B12 B6, vitamin E, folate
Optic neuropathy	Vitamin B12, thiamine, folate,
Dementia, encephalopathy	Vitamin B12, nicotinic acid, thiamine, folate

Vitamin B1 (Thiamine) deficiency :-[1, 2, 5, 17]

Thiamine deficiency is the first identified human

nutritional deficiency disorder. Introduction of milled rice during industrial evolution in 19th country was accompanied by epidemics of beriberi & universal enrichment of rice grains & flour products with thiamine lead to significant world wide control.

Function:-

Thiamine is precursor of coenzyme thiamine pyrophosphate which catalizes the oxidative decarboxylation of pyruvate & ketoglutarate with the eventual production of acetyl co-enzyme. A and succinate respectively Thiamine pyrophosphate also serves as a cofactor for transketolase reaction in HMP shunt. A defect resulting from deficiency of thiamine can lead to reduction i the generation of ATP and impaired acetyl choline synthesis. Thiamine also may have a direct effect on excitable membrane.

Etiology:-

Chronic alcoholism is the commonest cause of thiamine deficiency. Nonalcoholic conditions which can precipitate thiamine deficiency are hyper emesis of pregnancy, gastrointestinal surgery, Prolonged intravenous feeding,

Re-feeding after prolonged fasting or starvation , anorexia nervosa and dieting .

Neurological manifestations

Dietary depletion of thiamine can lead to dysfunction of both central and peripheral nervous system Major manifestations of thiamine deficiency are 1.Neuropathic beriberi 2. Wernicke korsakoff psychosis.

Neuropathic beriberi

It is also known as dry beriberi. It is characterized by insidious onset, progressive sensori motor neuropathy. Patient presents with paresthesia & pain in the feet. Patient has progressive difficulty in walking, distal weakness & foot drop. Patient can have muscle tenderness & cramps in calves. Often dry beriberi is associated with cardiovascular symptoms. Objectively patient has glove & stocking type of sensory loss & distal weakness. DTR are usually lost .Cranial nerves which can be involved are laryngeal nerve palsy causing hoarseness of voice & sub-acute optic neuropathy in patients taking ketogenic diet in epilepsy.(5) l

Excessive sweating of the soles & dorsal aspects of feet & volar aspects of hands & fingers is a common manifestation of alcohol induced neuropathy. Postural hypotension may be present. These symptoms are indicative of involvement of sympathetic nerve fibers.

Neuropathology

There occur wallerian type of axonal degeneration & secondary De-myelination.

Investigations

- Serum thiamine & urinary thiamine levels are decreased
- Erythrocyte transketolase activity is decreased and Serum pyruvate level is increased
- EMG & NCS. Axonal neuropathy with reduced amplitude of sensory & motor responses, normal or slightly decreased conduction velocity & neurogenic changes in EMG.

Treatment:-

Thiamine 50.100 mg/day orally & parenterally if malabsorption is suspected. Initiate balanced diet and Physiotherapy.

Wernicke korsokoff psychosis:-

Wernicke in 1881 described symptom complex– consisting of ocular abnormalities, ataxia, and mental confusion in association with deficiency of thiamine in alcoholics, Korsakoff in 1887 described an amnestic state called. Korsakoff' psychosis also occurs in association with thiamine deficiency. According to him few of the patients had associated neuropathy. Interesting fact is that the relationship between wernicke disease & korsakoff polyneuritis psychosis was not appreciated by either of them. The intimate clinical relationship was established by Bonhoeffer in 1904. (6)

Clinical features:-

Wernicke korsakoff psychosis usually occurs in chronic alcoholics or can occur in non alcoholics with poor nutritional status. Overall incidence is 8 – 2.8 % amongst Indian males and almost 27%

patients remain undiagnosed.

Wernicke encephalopathy

It is often precipitated acutely in at risk patients by intravenous glucose administration or carbohydrate loading.

Triad of symptoms is hallmark of this condition i.e.. Confusion opthalmoplegia and ataxia. In Wernicke encephalopathy. Confusion i.e. disturbances in mentation occurs in 90% of patients, the commonest being global confusional state .The patient is apathetic, inattentive & indifferent. Drowsiness is commonly seen which sometimes progress to stupor or coma. Ophthalmoplegia. Involves both lateral recti either in isolation or together, with palsies of other extraocular muscles. Patients have horizontal nystagmus on lateral gaze & may have vertical nystagmus on upward gaze. Patient will have sluggish reaction to light and paralysis of conjugate gaze. Ataxia may vary in severity and affects predominantly gait. Truncal ataxia is more common.(16)

Hypothermia & postural hypotension may be present and reflects involvement of hypothalamus & brainstem autonomic pathways. Patients can have impaired olfactory discrimination

Korsakoff psychosis

It is characterised by amnestic confusional state where memory is impaired out of proportion to cognitive function. Most patients are disoriented to time, place & person. The core of amnestic disorder is a defect in learning (anterograde amnesia). The defect in learning (Memorization) can be remarkably severe. Anterograde amnesia is always coupled with disturbance of past or remote memory i.e. retrograde amnesia. Memories of recent past are more severely affected than the remote past Characteristically a few isolated events from the past are retained but these are related without regard for the proper temporal sequence. Usually patient "telescopes" events. Often the lapses in memory is filled by confabulation.

Neuropathology Symmetrical. Paraventricular lesions involving thalamus, hypothalamus, mammilary bodies, periaqueductal, region & floor of the 4th ventricle showing necrosis & partial loss of neurons are seen.

Investigations:-

- Blood pyruvate level is increased and RBC transketolase activity is decreased
- EEG – Slow activity of mild to moderate degree is seen in 50% patients
- MRI- MRI may show signal abnormalities on T_2-weighted,fluid-attenuated inversion recovery, and diffusion-weighted images in the periaqueductal regions, medial thalami, and bilateral mammillary bodies(17)

Treatment:

It is a medical emergency Thiamine 50mg IV & 50 mg Im will reverse most of the features and prevent development of irreversible psychosis . It has to be continued for several days .

Nicotinic acid deficiency (Pellagra) :[2, 15,17,]

Pellagra is a disorder characterized by classic triad of diarrhea, dermatitis & dementia. It results from dietary deficiency of niacin and it's precursor tryptophan.

Function:-

Niacin in human beings is an end product of tryptophan metabolism. It is converted into the body to nicotinamide adenine dinucleotide (NAD) and nicotinamide adenine dinucleotide phosphate (NADP), coenzymes that have role in carbohydrate metabolism. Deficiency of of niacin brings about visible changes in central & peripheral nervous system. The mechanisms involved in these changes are not clearly understood.

Epidemiology and etiology

Pellagra was world wide in distribution. But presently it disappeared from developed world as a result of enrichment of cereal flour with niacin. Pellagra is now mostly confined to India, Africa & central America. In India pellagra is endemic is deccan plateau & Udaipur especially in maize and millet (Jowar) eating population. -Common causes of pellagra are eating corn or maize as staple diet ,or diet rich is leucine , chronic

alcoholism and malabsorption

Neurological manifestations

Neurological syndrome is not well characterized Primary symptoms are neuro psychiatric like irritability, apathy, depressed mood, inattentiveness, memory loss, organic psychosis & may progress to stupor & coma. Unexplained progressive encephalopathy in alcoholic patients not responding to thiamine should raise possibility of pellagra. Acute syndrome of clouding of consciousness cogwheel rigidity & uncontrollable grasping & sucking reflexes may be seen sometimes which respond to niacin. Other neurological manifestations are peripheral neuropathy, and myelopathy i.e. spastic paraplegia or sub acute combined degeneration.

Neuropathology:-

Diffuse neuronal chromatolysis is neuro pathological hallmark of neurological pellagra. It is seen in betz cells, pontine nuclei and other areas of brainstem. There can be degeneration of posterior & lateral columns of spinal cord.

Investigations:-

Clinical picture & response to niacin usually clinches the diagnosis there are no direct sensitive & specific markers of niacin deficiency.

- Erythrocyte NAD & plasma metabolites of niacin are indirect markers.
- Urinary excretion of niacin metabolite N: methyl nicotinamide and its 2 pyridone derivative are more reliable measures.

Treatment-

Oral nicotinic acid 50mg 3 times a day or parenteral 25mg 3 times a day Nicotinamide has comparable therapeutic efficacy but it lacks vasodilatory & cholesterol lowering property of nicotinic acid Advanced stages of pellagra can be treated by intramuscular nicotinamide 50 – 100 mg 3 times a day for 3-4 day followed by orally.

Vitamin B6: (Pyridoxine)-deficiency [2, 15, 17]

Vit B6 refers to family of compounds including pyridoxine, pyridoxal, pyridoxamine and their 5 phospate derivatives. Pyridoxine is readily converted into pyridoxal phosphate PLP. This PLP serves as coenzyme. important for metabolism of many amino acids , lipids & nucleic acids Vitamin B6 is also involved in synthesis of neurotransmitter.

Etiology:-

Deficiency usually occurs when treatment with B6 antagonist such as INH, hydralazine or penicillamine is given. It is also seen in chronic alcoholics. Pregnant & lactating women & elderly individuals.

Neurological Manifestations :-

In adults pyridoxine responsive peripheral neuropathy occurs during treatment with B6 antagonist drugs, most commonly INH .It is seen in 50% of slow acetylators. Patient usually presents with painful distal paresthesias that can rapidly progress to limb weakness and sensory ataxia. Pyridoxine deficiency is a contributory factor in peripheral neuropathies secondary to dietary deficiency.

Neuropathology There occur degeneration and regeneration in myelinated and unmyelinated fibers.

Investigations

- Blood and urinary B6 levels by microbiologic assay Plasma PLP levels
- Methionine load test is used as functional indicator of vit B6 status .B6 deficiency leads to higher homocysteine concentration after methionine load.

Treatment

Neuropathy reverses after B6 supplementation 50 – 100 mg per day

Vitamin B5(Pantothenic acid)deficiency: [2,6,15]

Burning feet syndrome: -

A distinct clinical entity reported among malnourished patients in tropical countries. It is more common in female and often seen is rice eating zones.

Patient presents with burning sensation in extremities. It starts in ball of big toe and later spreads to the sole and progresses proximally. There may be aching, throbbing or lightening type of pain. Symptoms are maximum when

retiring to bed in night & decreases after walking around. Objective signs of peripheral neuropathy are surprisingly absent. Hyperalgesia & hyperhydrosis is also seen.

Treatment:-

Exact etiology is not known but patients respond dramatically with intramuscular injection of calcium pantothenate.

Vitamin B_{12} (cobalmine) deficiency [3,7,11,12,14,]

B 12 is a water soluble vitamin whose major source is animal tissues. Milk also contains significant amount of Vit B 12. Plant food lacks Vit. B12. True vegans who don't's even take milk are prone to develop B12 deficiency. Major manifestations of Vit B12 deficiency are related to nervous system and haemopoietic system leading to megaloblastic anemia. Prevalence of Vitamin B12 detected in Indian men was found to be 67 % (Yojnik etal)

Functions:-

Vit. B12 or cobalmine occurs in two active forms methylcobalmine & adenosylcobalmine.

Methylcobalmine is a cofactor for a cytosolic enzyme, methionine synthetase to convert homocysteine to methionine in presence of folic acid. Methioine is adenosylated to S adenosyl methionine (SAM). A methyl group doner required for methylation reaction involving proteins, neurotransmitters, and phospholipids. Decreased SAM production leads to reduced myelin basic protein methylation ', white matter vacuolization in cobalmine deficiency. During the process of methionine formation methyltetrahydrofolate (methyl THF) donates the methyl group and is converted into THF which is precursor of purine & pyamidine synthesis. Impaired DNA synthesis leads to defect in oligodendrocyte growth & myelin production.

Adenosylcobalmine is a cofactor for methylmelonyl CoA mutase, which catalizes conversion of methylmelonyl CoA to succinyl CoA which later enters Krebs cycle. Accumulation of methylmelonate and propionate may provide abnormal substrate for fatty acid synthesis. The branched chain of abnormal odd number fatty acids may be incorporated into myelin sheath. This biochemical abnormality is suppose to be responsible for lesion in myelinated fibers in this disease.

Etiology:-

Defective intake or defective absorption is responsible for B12 deficiency Malabsorbtion because of– intrinsic factor deficiency, atrophic gastritis, acholrhydria , gastrectomy, bariatric surgery ,ileal disease/ resection blind loop syndrome ,Pancreatic disease tropical sprue Helicobacter pylori infection and fish tapeworm infestation are the important causes B12 deficiency

Neurological manifestation: -

CNS manifestations Dementia And Acute Psychosis (Megaloblastic Madness)Neuropsychiatric manifestations such as memory loss, depression, hypomania, paranoid psychosis with auditory and visual hallucinations the so called megaloblastic madness have been described with vitamin B-12 deficiency. Patients may present with violent behavior or more subtle personality changes. They may also present with vague complaints typical of aging such as fatigue, generalized weakness and loss of memory. Cognitive testing may reveal frank dementia. . It is still not clear if mild or moderate B12 deficiency can cause dementia and whether supplementation of the diet with B12 can prevent or delay the onset of dementias like Alzheimer's disease. Patients who are demented usually show little to no cognitive improvement with B12 supplementation.

Cerebrovascular Disease

Homocysteine is an atherogenic and thrombophilic agent, thus an increase in the plasma homocysteine represents an independent risk factor for cerebrovascular disease. It is one of the risk factors for cerebrovascular disease in the young. Deficiency of vitamin B-12 inhibits the conversion of homocysteine to methionine. Homocysteine plays an important role in cross linking of collagen and thus its excess in the vascular walls may predispose to both arterial and venous thrombosis. Cobalamin deficiency thus might be the cause of an otherwise unexplained

ischemic stroke or cranial artery dissection. Patient can have brain stem or cerebellar signs.

Spinal Cord Manifestations

Classical entity described with cobalmie deficiency is subacute combined degeneration onset is often insidious clinically this myelopathy presents with paraesthesias in the hands and feet with early loss of vibration and position sense leading to a progressive disturbance with gait. Ankle jerks are lost early in the course due to a superimposed peripheral neuropathy . Loss of vibration or position sense is always found and may be the first to appear, whereas other modalities of sensation may be affected in advanced disease. In mildly affected cases the activity of the knee and ankle jerks may be either increased or diminished but in severely affected patients these reflexes are always decreased or absent. Extensor plantar responses may also be present. Myelopathic signs tend to be symmetric and reflect the predominant involvement of posterior and lateral columns of the spinal cord.Cervical & upper thoracic regions are most dominantly involved.

Peripheral Neuropathy

The most frequent manifestations of peripheral neuropathy are paresthesias and numbness. The paresthesias usually first occur in the lower extremities. At this early stage there might be no objective signs on the neurological examination. As posterior column disease may give rise to exactly the same signs and symptoms it is difficult to distinguish the early signs of myelopathy from that of peripheral neuropathy .So a patient presenting with depressed ankle jerks, position and joint sense abnormalities, impaired vibration sense and blunting of pin-prick perception may be erroneously diagnosed to have a neuropathy rather than a myelopathy of the posterior columns. Pathological studies have documented a demyelinating neuropathy in some and shown an axonal or mixed picture in others.

Optic Neuropathy

Optic neuropathy is a rare manifestation of cobalamin deficiency. Patients present with complaints of diminished visual acuity. The optic neuropathy in cobalamin deficiency is of retrobulbar type and demonstrates a central or cecocentral scotoma.

Neuro pathology :-

Microscopically there occur spongiform changes and foci of myelin and axon destruction in white matter of spinal cord especially in posterior column at cervical and thoracic level & also in lateral column. Peripheral nerves show mostly axonal degeneration or demyelination.

Investigations

- Peripheral Smear- Macrocytosis & hyper segmented neutrophils
- Serum B12 assay influenced by many factors Radio isotope dilution assay is preferred technique.
- Homocysteine and methylmelonic acid assay -Levels increase in > 90% patient with Vit B12 deficiency
- Schilling test for cause of malabsorption
- Nerve conduction study – It is normal in early course of sub acute combined degeneration. There can be slowing of distal sensory conduction, reduced amplitude & minor sings of denervation suggestive of axonal change
- MRI- There occur abnormalities in posterior column and may be in lateral column. Deep white matter lesions are seen on T 2 weighted image which become confluent with disease progression.

Treatment :-

Intramuscular cyanocobalmine 1000 microgram daily for few days then weekly for a month and then monthly for remainder of life. In recent years oral cobalamine it suggested for maintenance treatment. Earlier the institution of therapy better and complete is the response.

Vitamin E Deficiency[10, 16]

Vit. E is a family of fat soluble vitamins that are active throughout the body some members are called tocopherols ($\alpha, \beta, \gamma, \delta$) Other members are called tocotrienols ($\alpha, \beta, \gamma, \delta$)

Function:-

It is powerful biological antioxidant which protects cells including nerve cells against the effect of free radicals.

Aetiology:

Vitamin E Deficiency occurs in patients having fat malabsorption like biliary atresia, chronic cholestasis, intestinal resection, Crohn's disease Pancreatic insufficiency, blind loop syndrome and bacterial overgrowth Celiac disease and hereditary diseases: abetalipoproteinemia.

Neurological Manifestations:-

Patients usually present with weakness or unsteadiness of gait. Neurological examination reveals spino-cerebellar syndrome along with variable degree of peripheral neuropathy. Patient commonly have limb ataxia, areflexia, severe loss of vibration and joint position. Cutaneous sensatios may be affected patients can have proximal muscle weakness.50% patiets have nystagmus ptosis or partial external ophthalmoplegia.

Neuro pathology :-

There is degeneration of large myelinated fibers of peripheral nerves, posterior column & sensory root. Accumulation of lipoproteins is seen in neurons of spinal cord, cerebellum and endothelial cells.

Investigations:

- Serum vitamin E – decreased and Serum CK levels are increased
- NCS – Mild axonal neuropathy and
- Somatosensory & visual evoked potential are abnormal

Treatment:-

Oral Vitamin E 50-100mg/day if started early.

In severe cases higher & parenteral dose is used.

Vitamin D deficiency :-[13, 17]

Vitamin D is a fat soluble vitamin. Neurological manifestations are described since ancient times. & it is referred as osteomalacic myopathy. It occurs due to dietary deficiency, malabsorption states, lack of exposure to sunlight or increased demand as in multiple pregnancies or lactating mothers.

Functions:

Vitamin D behaves as a neuroactive compound (neurosteroid) largely implicated in the control of brain homeostasis. Neuroprotective effects of vitamin D, acheived by it's action on levels of nerve growth factors in treatment of neurodegenerative disorders. Vitamin D appears to regulate development and homeostasis of nervous system and skeletal muscles.

Neurological manifestations:-

Bone pains & tenderness occur in almost all vitamin D deficient patients Neurologically patient usually presents with muscle tenderness particularly in thigh muscles, easy fatigability and weakness of proximal muscles of lower limbs. Classical waddling gait is noted in 40 to 65% patients. Weakness of proximal muscles of upper limbs occurs in 40 to 60% patients. Patients does not have subjective or objective sensory system involvement. DTR are brisk or normal.

Neuropathology:-

Histopathological changes in muscles are not much remarkable. There are atrophic changes in muscle fibers scattered degeneration of muscle fibers with crowding of sarcolemmal nuclei with mild fat infiltration & fibrosis.

Investigations

- Serum CK is increased, Serum Calcium & serum Phosphorus decreased, serum Alkaline phosphotase increased ,Serum 25(OH) D decreased
- Radiological picture reveals features of osteomalacia
- EMG Short duration low amplitude and polyphasic motor unit potentials with normal interference pattern

Treatment

Calciferol 2000 -4000 IU for 6 – 12 weeks followed by 200 – 400 IU per day along with Calcium 500 – 1000 mg daily.

Vitamin A deficiency [13, 17]

Deficiency may occur rarely in fat malabsorption syndromes, such as sprue, biliary atresia, and

cystic fibrosis. . The earliest sign of deficiency is reduced ability to see in dim light. Retinol, an aldehyde form of vitamin A, binds with the protein opsin to form rhodopsin, which is responsible for vision at low light level.

Copper Deficiency :-[10, 17]

Copper deficiency is also known to cause significant neurological problems.

Functions

Copper functions as a prosthetic group in several metalloenzymes, which act as oxidases many of these have a critical role in maintaining the structure and function of the nervous system.

Etiology

The most common cause is impaired absorption of dietary copper after gastric surgeries; including bariatric surgery Excessive consumption of zinc and iron may impair the absorption of copper. Menkes' disease is due to an inherited disorder of intestinal copper absorption.

Neurological manifestations

Deficiency of copper leads to a myelopathy characterized by sensory ataxia and gait difficulty. Many patients also have clinical or electrophysiological evidence of peripheral neuropathy.

Investigations

- Low serum copper level.
- Hematological manifestations, such as anemia and neutropenia.
- MRI Nonspecific T2-signal abnormalities may be seen on MRI of the spinal. Cord The paramedian cord is most commonly affected.

Treatment

Even in cases of malabsorption, dietary supplement of 2 to 6 mg of copper salt per day is usually sufficient to reverse a deficiency state. Intravenous infusion may be used if needed.

Nutritional Amblyopia[2, 6, 17]

This is the condition characterized by progressive visual impairment due to optic neuropathy resulting from nutritional deficiency , although the specific factor is not yet identified This condition is commonly seen in Africa , Asia and South America It is seen mostly among persons addicted to alcohol and tobacco hence it is also known as alcohol tobacco amblyopia.

Clinical features

It occurs usually in middle aged men Patient usually presents with blurring of vision for near and far objects which gradually progresses over several weeks or months. The reduction in visual acuity is due to presence of central or centrocecal scotoma which are larger for coloured than white objects .The optic disk is hyperemic initially ,but later on temporal pallor may develop .Abnormalities are bilateral and symmetrical . It can progress to blindness if not treated .The specific nutrient responsible for this condition is not known.

Neuropathology

There is symmetrical loss of myelinated fibers in the .central parts of optic nerves, chiasma and .optic tracts .Affected fibers correspond to papillomacular bundle

Treatment

Oral or parenteral B vitamin and nutritious diet

Strachan's Syndrome and Related Disorders[2, 6, 17]

In 1887, Strachan, described a syndrome in Jamaicans characterized by severe painful polyneuropathy, sensory ataxia, vision loss, and mucocutaneous excoriations. The condition later recognized in undernourished populations of other parts of world. The majority of these patients likely to have deficiencies of multiple vitamins, especially thiamine. The essential features are (1) a polyneuropathy that is often sufficiently severe to produce sensory ataxia; (2) amblyopia with optic atrophy; (3) tinnitus, hearing loss, and sometimes vertigo; and Gait ataxia and loss of sensation to vibration and joint position are prominent findings.

Treatment:

Is directed toward establishing adequate diet and replenishing vitamins.

Complications after Bariatric Surgery[4, 8, 9]

The epidemic of obesity and limited efficacy of

medical treatments have led to increasing use of bariatric surgical procedures for the treatment of medically complicated obesity especially in developing countries As many as half of these patients may become deficient in at least one of the micronutrients (especially B vitamins and folate, and less commonly vitamin E, vitamin D, and trace elements such as copper). Clinically significant malabsorption and vitamin deficiency syndromes are common. The most commonly reported neurological complication is peripheral neuropathy. Most of these patients have a sensory-motor polyneuropathy. Wernicke's encephalopathy is the most common central nervous system disorder. Other less common complications include optic neuropathy, myelopathy, and myopathy. All patients should have long-term medical follow-up, dietary counseling, and periodical laboratory evaluations. They should all take dietary supplements on an indefinite basis. Supplements should include multivitamins, folic acid, iron, calcium, and additional oral vitamin B12 supplement

Conclusion

Nutritional deficiency disorders are quite frequently encountered in clinical practice. Many of the vitamins are known to produce clinically significant neurological problems. It is extremely vital to detect these conditions early in their course, as if left untreated they can have disastrous effects.

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