Water soluble-Vitamin

I. Introduction

Vitamins are organic substances present in small amounts in food. They are required for carrying out vital functions of the body. They must be supplied in the diet if the body is unable to synthesize them. Vitamins are considered essential, the word vita means 'life' in Latin. Their discovery often came about because of their absence in the diet. They are involved in the utilization of the major nutrients like proteins, fats and carbohydrates. Though needed in small amounts, they are essential for health and well-being of the body. Vitamins were discovered based on their function and chemical nature and were designated as A, B, C, D. Vitamins are classified based on their solubility as fat-soluble and water-soluble vitamins. Water-soluble vitamins are not accumulated in the body, but are readily excreted. In the case of a deficiency, the clinician should be able to recognize the syndrome caused by a lack of the particular vitamin. In this country of abundant and varied food supply, the individual should instead think in terms of what a specific vitamin does rather than what disease it prevents.

Water-soluble vitamins are members of the B-complex and vitamin C (ascorbic acid). Most of the B-complex group can be further divided according to general function: energy releasing or hematopoietic. Other vitamins cannot be classified this narrowly because of their wide range of functions. Figure 1 shows the classification of water-soluble vitamins. Table 1 contains a summary of the functions, deficiency syndrome, those at risk for deficiency, sources and recommended dietary allowance (RDA) of each of the water-soluble vitamins.

Deficiencies caused due to lack of the following vitamins

Vitamin-C
Thiamine
Riboflavin
Niacin
Pantothenic acid
Pyridoxine (B6)
Biotin
Folic Acid
Vitamin B12

1. Vitamin C

The chemical name for Vitamin C is ascorbic acid. It was discovered in 1747 by demonstrating that citrus fruit juices prevented and cured scurvy. Vitamin C functions in oxidation-reduction reactions and is synthesized from glucose and galactose by plants and most animals. However, humans and other primates, lack the enzyme l-gulonolactone oxidase and thus cannot biosynthesize the factor, which for them consequently is a vitamin.

1.1. Effects of Deficiency:

Prolonged deficiency of vitamin-C produces a disease condition called as 'scurvy' in both infants and adults. Scurvy is typically manifested when the total body vitamin-C pools fall below about 300mg and plasma vitamin-C concentrations drop to <0.2mg/dL. Scurvy is fatal if untreated. The four Hs—

hemorrhagic signs, hyperkeratosis of hair follicles, hypochondriasis (psychological manifestation), and hematologic abnormalities (associated with impaired iron absorption)—are often used as a mnemonic device for remembering scurvy signs

a. Infantile scurvy:
There will loss of appetite, failure to gain weight, irritability, palor, defective growth of bones. Haemorrhage occurs under the skin. There will be defective formation of teeth and gums will be swollen (Figure 2a). The ends of the ribs become prominent resulting in beaded appearance called scorbutic rosary.

b. Adult Scurvy:
i.General manifestations are fever, susceptibility to infection, lethargy, fatigue, rheumatic pains in the legs, muscular atrophy and delayed wound healing.
ii.Anaemia: Microcytic hypochromic anaemia develops due to failure of absorption of iron.
iii.Gums become spongy and bleed easily. Gums become swollen, ulcerated and eventual tooth loss.
iv.The blood vessels become fragile and porous due to defective formation of collagen. Joints become swollen and tender.
v.Clinical symptoms appear when total body pool of vitamin-C decreases. Skin becomes rough and dry. There will be small petechial hemorrhages around hair follicles.

2. Thiamine
Thiamine is known as Vitamin B1. Deficiency of thiamine leads to beriberi. This condition is widely prevalent among population whose diet contains more of polished cereals.
2.1. Effects of Deficiency
Deficiency of thiamine is associated with low calorie intake. Severe deficiency of thiamine produces a disease known as beriberi (beri means “weakness”) One of the first symptoms of thiamin deficiency is a loss of appetite (anorexia) and thus weight loss. As the deficiency worsens, cardiovascular system involvement (such as hypertrophy and altered heart rate) and neurological symptoms (such as apathy, confusion, decreased short-term memory, and irritability) appears.
There are three types of beriberi
a.Dry beriberi
b.Wet beriberi
c.Acute/Infantile beriberi

a.Dry beriberi:
Found predominantly in older adults. Deficiency is of result from a chronic low thiamin intake, especially if coupled with a high carbohydrate intake. Dry beriberi is characterized by muscle weakness and wasting, loss of appetite, tingling numbness and burning sensation in hands and feet. Calf muscles will become tender. Knee and ankle jerks will be sluggish. In later stages complete loss of sensation in hands and legs will occur. It is characterized by foot and waist drop. Mental depression and confusion occurs.
b.Wet beriberi :
In this case, there is enlargement of heart and the cardiac output is high. Oedema or accumulation of

fluid in legs, face and trunk is observed. Palpitations are marked.

c.Acute/Infantile beriberi:
It occurs in first few months of life if the diet of the mother is deficient in thiamine. Symptoms are anorexia, vomiting, restlessness, sleeplessness, constipation, enlargement of the heart and breathlessness.

Thiamin deficiency is often associated with alcoholism. Wernicke's encephalopathy or Wernicke-Korsakoff syndrome, a neuropsychological complication, is also commonly found in those with alcoholism and AIDS, and in those receiving parenteral nutrition that is high in dextrose and low or absent in thiamin. People with alcohol dependency are particularly prone to thiamin deficiency because of:
decreased intake of the vitamin from decreased food consumption
increased requirement for the vitamin because of liver damage
decreased thiamin absorption
Wernicke's encephalopathy is characterized by ophthalmoplegia (paralysis of the ocular muscles), nystagmus (constant, involuntary eyeball movement), ataxia (impaired muscle coordination), loss of recent memory and confusion.

II. 3. Riboflavin
Riboflavin or Vitamin B2 is the yellow enzyme which is heat stable unlike other B Vitamins. Riboflavin in the combined form with proteins form flavo proteins or yellow enzymes.

3.1. Effects of Deficiency
A deficiency of riboflavin, known as ariboflavinosis, rarely occurs in isolation but most often is accompanied by other nutrient deficits. Riboflavin deficiency is prevalent mainly among the low income groups particularly the vulnerable group and the elderly adults. Riboflavin deficiency becomes manifest after several months of deprivation of the vitamin. Riboflavin deficiency is characterized by
i.Soreness and burning of the mouth and tongue.
ii.Lesions at the angles of the mouth called Angular Stomatitis (cracks in the skin at the corners of the mouth-Figure 4a).
iii.The inflammation of the tongue called glossitis
iv.Dry chapped appearance of the lip with ulcers termed cheilosis.
v.The skin becomes dry and results in seborehoeic dermatitis.
vi.Photophobia, lacrimation, burning sensation of the eyes and visual fatigue.
vii.Decreased motor co-ordination
viii.Normocytic anaemia

4. Vitamin Niacin
The term niacin (vitamin B3) is considered a generic term for nicotinic acid and nicotinamide. Niacin is required by all the cells of our body. Like thiamine and riboflavin it plays a vital role in the release of energy from carbohydrates, protein, fat and alcohol. Like thiamin, which was discovered through its

deficiency disorder beriberi, niacin was discovered through the condition pellagra in humans and a similar condition, called black tongue, in dogs.

4.1. Effects of Deficiency

Deficiency of niacin causes a disease known as pellagra (Figure 5). It is characterized by three D's - Dermatitis, Diarrhoea and Dementia.

a.Dermatitis–Name pellagra comes from pelle-skin and agra-rough. Marked changes occur in the skin especially in the skin exposed to sun and friction areas like elbows, surfaces of arms, knees. Lesions are symmetrically distributed, in the affected parts. At first there is reddening, thickening and pigmentation of the skin. Later on there is exfoliation leading to ultimately parchment of skin –butterfly like appearance.

b.Diarrhoea–Diarrhoea enhances the deficiency state. There are structural and absorptive defects in the small intestine. Tongue appears raw, and mucous membrane of the tongue is inflammed. Gastrointestinal manifestations include glossitis, cheilosis, stomatitis, nausea, vomiting, and diarrhea or constipation.

c.Dementia–There is irritability, depression, poor concentration and loss of memory. Delirium is a common mental disturbance.

III. 5. vitamin Pantothenic acid

Pantothenic acid consists of β-alanine and pantoic acid joined by a peptide bond/amide linkage. The vitamin was once called vitamin B5.

5.1. Effects of Deficiency

"Burning feet syndrome" is characterized by numbness of the toes and a sensation of burning in the feet. The condition is exacerbated by warmth and diminished with cold and is thought to result from pantothenic acid deficiency. Other symptoms of deficiency include vomiting, fatigue, weakness, restlessness, and irritability. Deficiency of pantothenic acid is thought to occur more often in conjunction with multiple nutrient deficiencies, as for example in malnutrition. Some conditions that may increase the need for the vitamin include alcoholism, diabetes mellitus, and inflammatory bowel diseases. Increased excretion of the vitamin has been shown in people with diabetes mellitus.

6. Vitamin Pyridoxine (B6)

Pyridoxine is unique among B–complex Vitamins in that it functions primarily in protein metabolism. Pyridoxine denotes related substances such as Pyridoxine, Pyridoxal and Pyridoxamine are three forms in which it is present in our body. Pyridoxine represents the alcohol form, pyridoxal the aldehyde form, and pyridoxamine the amine form. Some of the initial research was aimed at correcting dermatitis in rats.

6.1 Effects of Deficiency

Vitamin B6 deficiency leads to abnormalities in protein metabolism which is manifested as poor growth, convulsions, anaemia, and skin lesions. Severe deficiency leads to microcytic hypochromic anaemia. Symptoms such as weakness, nervousness, irritability, insomnia and difficulty in walking is predominant. Deficiency also alters calcium and magnesium metabolism, impairs niacin synthesis from tryptophan, and inhibits metabolism of homocysteine. The last results in hyperhomo cysteinemia, a risk factor for heart disease. Groups particularly at risk for vitamin B6 deficiency are the elderly, who have a poor

intake of the vitamin; people who consume excessive amounts of alcohol and people on a variety of drug therapies.

IV. 7. Vitamin Biotin

Biotin's discovery was based on the research investigating the cause of what was called "egg white injury." Eating raw eggs was known to result in hair loss, dermatitis, and various neuromuscular problems. In 1931 a substance was found (now called biotin) in liver that could cure and prevent the condition. Biotin was once called vitamin H (the H refers to haut in German and means "skin") as well as vitamin B7.

7.1 Effects of Deficiency

Biotin deficiency in humans is characterized by lethargy, depression, hallucinations, muscle pain, paresthesia in extremities, anorexia, nausea, alopecia (hair loss), and scaly, red dermatitis. A diet devoid of biotin can result in decreased plasma biotin and in reduced biotin excretion in about 2-4 weeks. Biotin deficiency or poor biotin status, though fairly rare, occurs in various populations. People who ingest raw eggs in excess amounts are likely to develop biotin deficiency because of impaired biotin absorption. Impaired biotin absorption also may occur with gastrointestinal disorders such as inflammatory bowel disease and achlorhydria (lack of hydrochloric acid in gastric juices), in people on anticonvulsant drug therapy, or in chronic consumers of excessive amounts of alcohol. Biotin status has been shown to decline in some women during pregnancy.

. Vitamin Folic Acid

Folic acid is the term used to refer to the oxidized form of the vitamin found in fortified foods and in supplements. Folate refers to the reduced form of the vitamin found naturally in foods. The Latin word folium means "leaf," and the word folate from Italian means "foliage". Folic acid was first extracted from dark green leafy vegetables. Folate's and vitamin B12's discovery resulted from the search to cure the disorder megablastic anemia, a problem in the late 1870s and early 1880s. As with many of the other vitamins, eating liver was shown to cure the condition.

8.1. Effects of Deficiency

Simple folate deficiency results in the bone marrow producing immature cells (megaloblasts cells) and few matured red blood cells. This results in reduced oxygen–carrying capacity causing anaemia termed-Megaloblastic anaemia. Folate deficiency during pregnancy causes neural tube disorders of the foetus. Folate deficiency impairs the ability of the immune system to fight infection.

9. Vitamin B12 (Cyanocobalamin)

Vitamin B12, also called cobalamin, is considered a generic term for a group of compounds called corrinoids because of their corrin nucleus. Vitamin B12 was the last vitamin to be discovered. It was isolated in 1940. Eating large amounts of liver could help correct pernicious anemia associated with deficiency of this vitamin. It took about two decades to identify the vitamin in liver.

9.1. Effects of Deficiency

Pernicious amaemia is the major problem arising from an inadequate amount of vitamin B12. Pernicious

amaemia is a condition characterized by very large, immature red blood cells with normal amounts of haemoglobin. Most deficiency signs and symptoms are of neurologic and hematologic origin; some signs and symptoms include skin pallor, fatigue, shortness of breath, palpitations, insomnia, tingling and numbness (paresthesia) in extremities, abnormal gait, loss of concentration, memory loss, disorientation, swelling of myelinated fibers, and possibly dementia. Neurological problems occur in about 75% to 90% of deficient people.

V. 10. Conclusion

Vitamins are required for carrying out vital functions of the body. Though needed in small amounts, they are essential for health and well being of the body. Vitamins are classified based on their solubility as fat-soluble and water-soluble vitamins. Water-soluble vitamins are members of the B complex and vitamin C. Deficiency of vitamin-C produces a disease condition called as 'scurvy'. The four Hs— hemorrhagic signs, hyperkeratosis of hair follicles, hypochondriasis, hematologic abnormalities are often used as a mnemonic device for remembering scurvy signs. Deficiency of thiamine leads to beriberi. There are three types of beriberi and they are dry beriberi, wet beriberi and acute/infantile beriberi. A deficiency of riboflavin is known as ariboflavinosis and of niacin is known as pellagra. Pellagra is characterized by three D's -Dermatitis, Diarrhoea and Dementia. Pantothenic acid deficiency leads to Burning feet syndrome and is characterized by numbness of the toes and a sensation of burning in the feet. Vitamin B6 deficiency leads to abnormalities in protein metabolism and microcytic hypochromic anaemia. Biotin deficiency in humans is characterized by lethargy, depression, hallucinations, muscle pain, paresthesia in extremities, anorexia, nausea, alopecia, and scaly, red dermatitis. Folate deficiency causes Megaloblastic anaemia while, Pernicious anaemia is caused due to vitamin B12 deficiency.