

## **Diseases caused by water soluble vitamins**

### **Vitamin C**

Vitamin C is synthesized by all species and is not an important dietary essential in any of the domestic animals. The synthesis occurs in tissues and although blood level fall after birth, in the newborn calves they begin to rise again at about 3 weeks of age. However a dermatosis of young calves has been associated with low levels of ascorbic acid in their plasma and responds to a single injection of 3 gram of ascorbic acid. A heavy dandruff followed by waxy crust, alopecia and dermatitis commences on the ears and spread over the cheeks down the crest of neck and over the shoulders. Some deaths have been recorded but spontaneous recovery is more usual.

There is some interest in the administration of high doses of ascorbic acid orally to horses to counteract the effect of stress and minimize the effect of infection. A single oral dose of 20 gr of ascorbic acid does not result any effect in plasma concentration. However daily administration of either 4.4 gr or 20 gr result in significant increase in plasma concentration.

## **Thiamin deficiency**

The disease caused by deficiency of thiamin in tissues is characterized by nervous sings.

### **Etiology**

Thiamin deficiency may be primary due to deficiency of the vitamin in the diet or secondary because of destruction of the vitamin in the diet by thiaminase . The primary deficiency is unlikely under natural condition because most plants specially seeds, yeast and milk contain adequate amounts.

Thiamin is normally synthesized in adequate amount in rumen of cattle and sheep on a well balanced roughage diet. The degree of synthesis governed to some extend by the composition of the ration, a sufficiently of readily fermentable carbohydrate causing increase of synthesis of most vitamins of the B complex and high intake in the diet reducing synthesis

### **Epidemiology :**

One of the best example of secondary thiamin difiecnycy is inclusion of excess raw fish in the diet of carnivores resulting in destruction of thiamin because of the high content of thiaminase in the fish. Two major occurrence of secondary thiamin deficiency are recorded. In horses the ingestion of excessive quantities of barken fern and horsetail cause nervous sings

because of the high concentration of thiaminase of these plants. The second important occurrence of the thiamin deficiency is in the etiology of polioencephalomalacia.

Thiamin deficiency occurs in sheep being subjected to live export from Australia to the middle east.

### **Pathogenesis:**

The Only known function of thiamin its activity as a carboxylase in the metabolism of fats, carbohydrates and proteins and its deficiency lead to accumulation of endogenous pyruvates. Although the brain is known to depend largely on carbohydrates as a source of energy, there is no obvious relationship between the deficiency of thiamin and the development of nervous signs which characterized it.

### **Clinical findings:**

**Barcken fern and horsetail poisoning in the horse.** Incoordination and falling and bradycardia due to cardiac irregularity are the cardinal clinical sings of barcken fern poisoning in horse. The design disappear after the Parentral administration of thiamin. Similar clinical effects occur with horsetail. Swaying from side to side occurs first followed by pronounced incoordination including crossing of the forelegs and wide action of the hindlegs . When standing the legs are placed well a part and crouching and arching of the back are evident. Muscle tremor develops and eventually the

horses is unable to rise, clonic convulsion and opsthotonus are the terminal stage.

**Clinical pathology :**

- ❖ Blood pyruvic acid level in horses are raised from normal levels of 2-3 Mg/dl to 6-8 Mg/dl.
- ❖ Blood thiamin levels are reduced from normal of 8-10 Mg/dl to 2.5 - 3 Mg/dl.
- ❖ Electrocardiograms show evidence of myocardial insufficiency.
- ❖ In sheep subjected to export liver and rumen thiamin concentration and erythrocyte transketolase activities were below level found in clinically normal sheep.

**Differential diagnosis :**

- \* Similar syndrome may occur with plant poisonings.
- \* Hepatic necrosis and fibrosis.

**Treatment**

- ❖ In clinical cases the injection of a solution of the vitamin produces dramatic results ( 5 mg/kg B.W every 3h) the initial dose is usually given IV following by 1M injections for 2 - 4 days.
- ❖ An oral source of thiamin should be given daily for 10 days and any dietary abnormalities corrected.

## **Control**

The daily requirement of thiamin for monogastric animals is in general 30 - 60 mg/kg B.W. The addition of yeast, cereals, grains, liver and meat to the ration usually provides adequate thiamin.

## **Riboflavin Deficiency ( Vit B<sub>2</sub>)**

Although riboflavin is essential for cellular oxidative processes in all animals the occurrence of deficiency under natural condition is rare in domestic animals because activity growing green plants and animal protein are good source and some synthesis by alimentary tract microflora occurs in all species. Synthesis by microbial activity is sufficient for the need of ruminants but dietary source is required in the these animals in the pre-ruminant stage. Milk is very good source. On experimental diets the following syndrome have been observed in calves. Anorexia, poor growth , scour, excessive salivation and lacrimation and alopecia occur. Area of hyperemia develop at the oral commensures, on the edge of the lips and around the navel. There are no ocular lesion.

### **Pantothenic acid deficiency ( Vit B<sub>3</sub>)**

Pantothenic acid is a dietary essential in all species rather than ruminants, which synthesize it in the rumen. Deficiency under natural condition had been reported mainly in pigs based on corn.

Experimentally induced Pantothenic acid deficiency in calves is manifested by rough hair coat, dermatitis under lower jaw, excessive nasal mucus, anorexia and reduced growth rate and eventually fatal.

### **Niacin and Nicotinic acid deficiency ( Vit B<sub>4</sub> & B<sub>5</sub>)**

Nicotinic acid or niacin is essential for normal carbohydrate metabolism. Because of the high content in most natural animal feed deficiency status are rare in ordinary circumstances, except in pigs.

In ruminants synthesis within the animal provides an adequate source. Even in young calves signs of deficiency do not occur and because rumen microflora activity is not yet of any magnitude, extra ruminal synthesis appear probable. The oral supplementation of niacin in the diet of periparturient dairy cows may result in an increase in serum potassium, niacin and sodium concentration. The oral therapeutic doses rate of nicotinic acid in pigs is 100-200mg, 10-20 g/ ton of fed.

### **Pyridoxine ( Vit B<sub>6</sub> ) deficiency**

A deficiency of pyridoxine in diet is not known to occur under natural condition. Experimental deficiency in pigs is characterized by periodic epileptiform convulsions. The daily requirement of pyridoxine in the pigs is of the 100 mg/kg BW or 1 mg/kg of solid food . Experimentally induced deficiency in calves is characterized by anorexia, poor growth, a pathy , dull coat and alopecia, severe fatal epileptiform seizures occur in some animals. Anemia with poikilocytosis is characteristics of this deficiency in cow and calves.

### **Biotin ( Vit H) deficiency**

Biotin or Vit H has been several important biochemical functions. It is a cofactor in several enzyme systems involved in carboxylation and trans carboxylation reaction and consequently has a significant effect on carbohydrate metabolism, fatty acid synthesis, amino acid deamination, purin synthesis and nucleic acid metabolism. Biotin is found in almost all plants and animal materials and being required in very small quantities, is unlikely to be deficient in diet under the natural condition, especially as microbial synthesis occurs in alimentary tract. Biotin is now considered as significant factor in lameness of cattle. Biotin is important for all

differentiation of epidermal cells which are required for normal production of keratin and hoof horn tissue. Biotin also act as cofactor in carboxylase enzymes and is an important factor in the both gluconeogenesis and fatty acid synthesis. Biotin is synthesized in rumen and absolute biotin deficiency has not been recognized.

### **Folic acid deficiency**

Folic acid ( pteroylglutamic acid) is necessary for nucleic acid metabolism and its deficiency in humans lead to the development of pernicious anaemia. A dietary source is necessary for all animal species and an adequate intake is provided by pasture. Although naturally occurring deficiencies have not been diagnosed in domestic animals. Folic acid has numerous and complex interrelationship with other nutrient and the possibility of a deficiency playing a part in inferior animal performance should not be overlooked. The vitamin has a particular interest for equine. Permanently stabled horses and some horses in training may require additional folic acid preferably on a daily basis by the oral route. Folic acid deficiency can be induced in fetal foals and adult horses by administration of inhibitor of folate metabolism ( pyrimethamine, trimethoprim, sulfonamides). Folic acid at dose of 1 mg/kg B.W orally daily for 2 weeks was used successfully for the treatment of acquired alopecia in a 3 weeks old Charolais calf but spontaneous recovery without treatment was a possibility.



### **Choline deficiency**

Choline is dietary essential for pigs and young calves. Calves fed on synthetic choline deficient diet from the second day of life develop an acute syndrome in about 7 days. There is marked weakness and inability to get up, labored or rapid breathing, and anorexia. Recovery follows treatment with choline. Older calves are not affected.

Supplementation of 20 gr/day of rumen protected choline to dairy cows 14 days before parturition increased milk production during the first month of lactation and the concentration of choline in milk but did not affect fat or protein concentration in the milk.

### **Vit B<sub>12</sub> deficiency ( Cyanocobalamin )**

Vit B<sub>12</sub> deficiency is unlikely to occur under natural conditions other than because of primary dietary deficiency of cobalt which is an important disease in many countries.

Although microbial synthesis of the vitamin occurs in the rumen in the presence of adequate cobalt and in the intestines of other herbivores such as the horses.

A deficiency syndrome has been produced in young calves on a synthetic ration, signs include anorexia, cessation of growth, loss of

condition and muscular weakness. Daily requirement under these condition is 20 - 40 mg of Vitamin B<sub>12</sub>.