

Vitamin A Deficiency **(Hypovitaminosis A)**

Etiology

Vitamin A deficiency may be primary disease due to an absolute deficiency of Vit A or its precursor carotene in the diet or a secondary disease in which the dietary supply of Vit A or carotene is adequate but their digestion, absorption or metabolism is interfered with to produce a deficiency at the tissue level.

Pathogenesis

Vit A is essential for the regeneration of the visual purple necessary for dim light vision, for normal bone growth, and for maintenance of normal epithelial tissue. Deprivation of the vitamin produces effects largely attributable to disturbance of these functions. The same tissue are affected in all species.

Night Vision

Ability to see in dim light is reduced because interference with regeneration of visual purple.

Cerebrospinal fluid pressure

An increase in CSF pressure is one of the first abnormalities to occur in Hypovitaminosis A in calves. It's a more sensitive indicator than ocular changes and in the calves it occurs when the vit A intake is about twice that need to prevent night blindness. The increase in CSF pressure is due to impaired absorption of CSF due to reduced tissue permeability of the arachnoids villi and thickening of the connective tissue matrix of the cerebral dura mater. The increased CSF pressure responsible for syncope and convulsions which occur in calves in the early stage of vit A deficiency. The syncope and convulsions may occur spontaneously or be precipitated by excitement and exercise.

Bone growth

Vit A is necessary to maintain normal position and activity of osteoblast and osteoclasts. When deficiency occur there is no retardation of endochondral bone growth but there is incoordination of bone growth in that shaping, especially in final molding of bones, dose not processed normally.

Epithelial tissues

Vitamin A deficiency leads to atrophy of all epithelial cells but the important effect are limited to those of epithelial tissue with a secretory as well as covering functions. The secretory cells are without power to divide and develop from undifferentiated basal epithelium. In Vit A deficiency these secretory cells are gradually replaced by the stratified keratinizing epithelial cells common to non secretory epithelial tissues. The occurs chiefly in the salivary glands, urogenital tracts but not ovaries or renal tubules and the paraocular glands and teeth. The secretion of thyroxine is

markedly reduced. The mucosa of the stomach is not markedly affected. These changes in epithelium leads to clinical signs of placental degeneration, xerophthalmia and corneal changes.

Embryological development

Vit A is essential for organ formation during growth of the fetus. Multiple congenital defects occur in pigs and rats and congenital hydrocephalus in rabbits.

Immune mechanism

The effects of Vit A and B carotene on **host defense mechanism** have been uncertain and controversial for many years. Some workers claim that the incidence and severity of infectious diseases are higher in vit A deficiency.

Clinical findings:

In general similar syndrome occur in all species but because of the species differences in tissue and organ response, some variations are observed. The major clinical findings are.

Night blindness

Inability to see in dim light is the earliest signs in all species except in the pig which is not evident until plasma vit A levels are very low. This is an important diagnostic sign.

Xerophthalmia

True xerophthalmia with thickening and clouding of the cornea occurs only in the calves. In other species a thin, serious mucoid discharge from the eyes occur followed by **corneal keratinization**, clouding and sometime ulceration and photophobia.

Changes in skin

A rough , dry coat with shaggy appearance and splitting of the bristle tips in pigs is characteristics , but excessive **keratinization** such as occur in cattle poisoned with **chlorinated naphthalene** dose occur under natural condition of Vit A deficiency. Heavy deposits of bran like scales on the skin are seen in a affected cattle. Dry scaly hooves with multiple vertical cracks are another manifestation of skin changes especially in horses. **A seborrheic dermatitis** may also observed in pigs but is not specific for Vit A deficiency.

Body weight

Under natural conditions, a simple deficiency of vitamin A is unlikely to occur and the emaciation commonly attributed to vit A deficiency may be largely due to multiple deficiency of **protein and energy**. Although inappetence, weakness stunted growth and emaciation occur under experimental condition of severe deficiency.

Reproduction efficiency

Loss of reproductive function is one of the major causes of loss in Vit A deficiency. Both male and female are affected. In male libido is retained but **degeneration of germinative epithelium of seminiferous tubules causes**

reduction in the number of motile, normal spermatozoa produced. In young rams the testicles may be visibly smaller than normal. In female conception is usually not interfered with but placental degeneration leads to abortion and the birth of dead or weak young. Placental retention is common.

Nervous system

Sings related to damage of the nervous system include

- Paralysis of skeletal muscle due to damage of peripheral nerve roots.
- Encephalopathy due to constriction of the optic nerve canal.

Congenital defects

These have been observed in piglets, and calves. In calves the defects are limited to congenital blindness due to optic nerve constriction and encephalopathy.

Clinical pathology :

*** Plasma Vit A concentration**

Normal plasma Vit A concentration in cattle range from 25 - 60 mg/dl , clinical signs can be expected when the levels fall to 5 mg/dl.

*** Plasma retinol**

Normal in horse 16.5 mg/dl

*** Plasma carotene**

In cattle levels of 150 mg/dl are optimum. The clinical signs appear when the levels fall to 9 mg/dl.

- **Hepatic Vit A**

Differential diagnosis:

- 1- Polio encephalomalacia .
- 2- Hypomagnesemic tetany.
- 3- Lead poisoning .
- 4- Rabies .
- 5- Menignoencephalitis .
- 6- All disease caused low growth and poor reproduction functions.

Treatment

- **Vitamin A**

440 IU/Kg b.w daily for 7 days

Parental injection is an aqueous rather than oily.