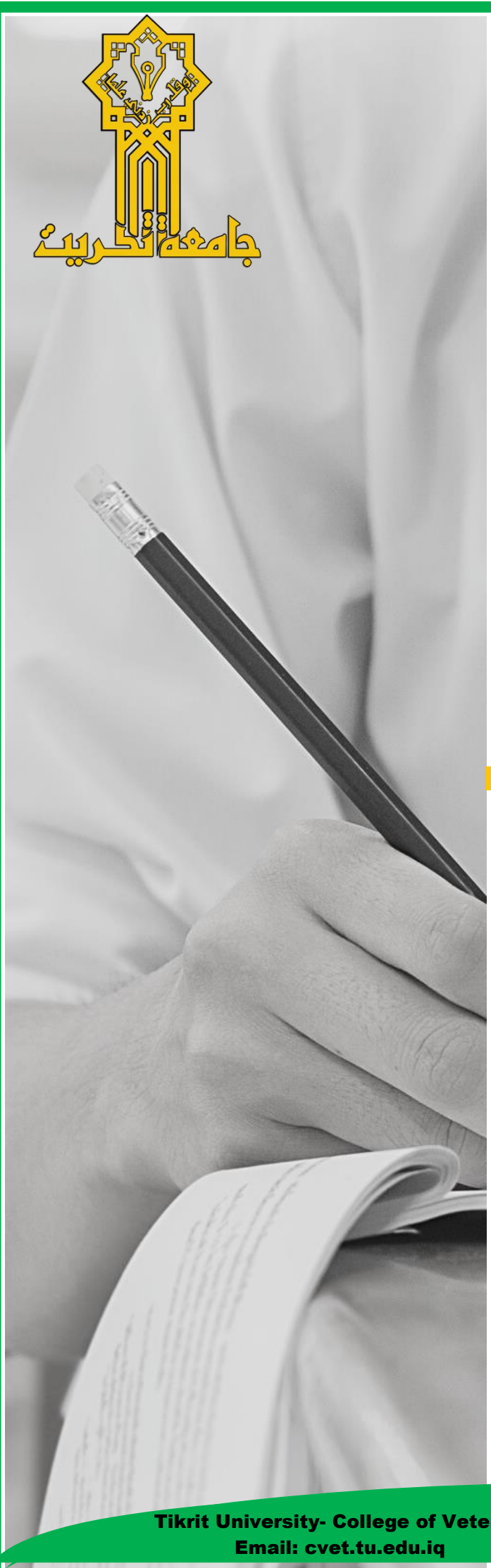

Tikrit University
College of Veterinary Medicine

Diseased Caused By Nutritional Deficiencies

Subject name: internal medicine

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Vitamin A Deficiency (Hypovitaminosis A)
Fat-Soluble vitamins

- ❖ Hypovitaminosis A may cause by an insufficient supply of vitamin in ration or defective in absorption from alimentary canal.

The main effects of vitamin A deficiency in animals:-

- Young animals: Compression of the brain and spinal cord.
- Adult: Night blindness, corneal keratinization, pityriasis, defect in hooves, loss of weight and infertility.
- Pregnant females: Congenital defects, abortion.

Etiology

a. Primary disease: absolute deficiency of vitamin A or it precursor carotene in diet.

b. Secondary disease: Defect in digestion, absorption or metabolism cause deficiency.

Function of vitamin A:-

- Some metabolic functions of vitamin A are not yet known.
- A chief role is maintenance of epithelial tissue (skin and lining of respiratory, digestive and reproductive tract) in a healthy condition.
- It also functions in visual purple, a compound in the eye needed for sight when an animal adapts from light to dark.
- Vitamin A is essential for proper kidney function and normal development of bones, teeth and nerve tissue.

Factors that effect of vitamin A requirements:-

- **Species**

  Rats are much more efficient than farm animals in converting carotene to vitamin A. (One millogram of beta-carotene is considered to have 1,667 IU of vitamin A value for the rat), 400 IU for cattle, 400 to 500 IU for sheep, and 500 IU for dogs.

- **Breed**

Some dairy breeds are more efficient than others at converting carotene to vitamin A. The yellow milk and carcass fat of Guernseys and Jerseys indicate their poor utilization of carotene for vitamin A. Some studies indicated Holsteins were twice as efficient as Guernseys in changing carotene to vitamin A.

There does not appear to be any difference among beef breeds in ability to convert carotene to vitamin A.

- **Animal condition or status**

The dam is reflected in that of the fetus specially in certain circumstances, EX. The carotene as it occur in green feed, it does not pass placental barrier and high intake of green pasture before parturition does not increase the hepatic stores of vitamin A in newborn calves, lambs, kids. While Vit. A in ester form (as fish oils) will pass placental barrier in cow. Or parenteral administration of Vit. A injection preparation before parturition will increase the hepatic stores of vitamin A in newborn.

- **Carotene**

Beta-carotene makes up a larger percent of the total carotene in some plants than in others. Other carotenes yield less vitamin A activity than beta-carotene.

- **Depletion**

Cattle depleted of vitamin A are less efficient in converting carotene to vitamin A. This supports the practice of administering **pre-formed vitamin A** in the diet or by injection instead of depending upon **carotene in the feed** to replenish cattle severely deficient of vitamin A.

- **High-carotene feeds**

Cattle maintained on high-carotene diets convert carotene less efficiently to vitamin A. This condition could accelerate يسرع the of liver stores of vitamin A. While, when cattle are abruptly نحو مفاجئ changed to diets with less carotene.

- **Thyroid depression**

Hot weather or components in the diet may cause thyroid depression, which is thought to decrease conversion of carotene to vitamin A.

- **Stresses**

Hot weather, disease, parasites and other stresses are believed to interfere with the animal's ability to convert carotene to vitamin A and to depress the efficiency with which vitamin A can be used to meet needs. Also, these and other factors may increase the animal's requirements for vitamin A. Inflammation and damage of the intestinal wall by diarrhea or parasites interfere with the absorption of carotene and vitamin A and the conversion of carotene to vitamin A.

- **Silages, haylage, pasture**

Cattle consuming rations high in corn silage, sorghum ذرة or oat silage and grass-legume haylage have been found to deplete normal stores of vitamin A in the liver, even though these feeds contained medium to high levels of what was thought to be beta-carotene.

Cattle full-fed grain on pasture have benefited from vitamin A supplementation in some trials.

Pathogenesis

Vitamin A essential for regeneration of the followings:- Visual purple necessary for dim-light vision, Bone growth and Maintenance of normal epithelial tissue.

1- Night Vision

Due to interference with regeneration of visual purple. Ability to see in dim light is reduce.

2- Cerebrospinal Fluid Pressure

- Increase in CSF (In calves), lead to ocular changes.
- It is occur due to reduce tissue permeability of the arachnoids villi and thickening of the connective tissue matrix of the cerebral duramater.

Clinical signs: The increase in CSF pressure lead to syncope and convulsions in calves (It may be precipitated by excitement and exercise).

3- Bone Growth

- Vitamin A essential for normal position and activity of osteoblast and osteoclast.
- Retardation of endochondral bone growth. This lead to distortion and herniation of the brain and increase CSF pressure 4-6 times.

Clinical signs:

1. Papilledema, incoordination and syncope, weakness and ataxia.
2. Spinal cord: facial paralysis , blindness due to constriction of the optic nerve.

4- Epithelial Tissue

- Atrophy of epithelial cells especially secretory and covering function and replaced by the stratified, keratinizing cell (salivary gland, Urogenital tract, paraocular gland and teeth).
- Decrease of thyroxine
- Placental degeneration
- Xerophthalmia and Corneal changes

5- Embryonic Development

- ❖ Vit. A appears to be essential for organ formation during growth of fetus. Multiple congenital defects fetus borne from deficient dam

- 1. Congenital defects (Pig and Rats)
- 2. Hydrocephalus (Rabbits).

Clinical Signs

1. Night Blindness.

2. Xerophthalmia (Dog and Cattle).

- Thickening and clouding of the cornea.
- Cornial keratinization and ulceration and photophobia.

3. Change in the skin

- Rough dry coat.
- Bran-like scales on the skin.
- Keratinization.
- Deformities in hooves (Cracks).
- Seborrheic dermatitis.

4. Body Weight

- Weakness,
- Stunted growth,
- Emaciation.

5. Reproductive Efficiency

- Loss of productive function (Both, male and Female).
- Decrease libido.
- Reduction in the number of motile spermatozoa due to degeneration of the germinative epithelium of the seminiferous tubules.
- Testicles may be visibly smaller than normal.
- Female, Placenta degeneration lead to abortion and birth of dead or weak .
- Retented placenta.

6. Nervous Signs

- Paralysis (CNS damage).
- Blindness due to constriction of the optic nerve canal.
- Incoordination in hind legs.
- Knuckling of the feet.
- Convulsion due to increase in CSF pressure.

7. Congenital Defects

- Blindness due to constriction of the optic nerve canal.
- Absence of the eye (Anophthalmous).
- Small eyes (Microphthalmous).
- Degenerative changes in lens and retina.
- Cleft palate (Pigs).
- Diaphragmatic hernia.
- Aplasia of genitalia.
- Herniation of spinal cord.

8. Other Diseases

- Increase susceptibility to infection.
- Increase incidence of otitis media.
- Enteritis.

Clinical Pathology

- 10 µg/dl (necessary for normal growth).
- 5 µg/ dl ……..clinical signs.
- 25 µg/ dl or above …….(normal).
- Hepatic level of vitamin A …….6 µg/g.
- Cerebro spinal fluid pressure …….Normal 100 mm of water increased to 200 mm.

Necropsy Findings

- Lesions in optic nerve.
- Pneumonia and otitis media.
- Sequamous metaplasm of the inter lobular duct of the parotid salivary.
- Degenerative muscle.
- Hyperkeratenization of reticulum and rumen

In general Symptoms of vitamin A deficiency

- One of the first easily detected signs of vitamin A deficiency in cattle is night blindness. An easy way to check for this condition is to place an obstacle in the pathway of cattle and notice if they stumble over it at twilight.
- Other early signs are loss of appetite, rough hair coat, dull eyes, slowed gains and reduced feed efficiency.
- Diarrhea and pneumonia may be the first indicators, especially in young animals.
- Later developments include excessive watering of the eyes, staggering gait, lameness or stiffness in knee and hock joints, and swelling of the legs and brisket (and sometimes in the abdominal region).
- Feedlot cattle with advanced vitamin A deficiency often pant excessively at high temperatures and go into convulsions when excited.

- Signs of vitamin A deficiency in breeding herds include lowered fertility and calving percentage. Cows abort, drop dead and weak calves.

Treatment

1-Affected animals as early as possible treatment with Vit. A paranteral injection an equeous rather than oily solution is preferred (the dose rate used 440 I.U /Kg B.Wt. It give rapid respond).

2-It was necessary to repeat an intramuscular injection of 1 million IU of vitamin A every 28 days to maintain safe vitamin A liver stores for cattle fed corn silage

3- The intramuscular injection of 500,000 to 6 million IU of vitamin A in cows two months before calving has been used in numerous experiments with range and farm herds. In calves with signs of increase of CSF pressure will usually respond to treatment and return to normal in 48 hours.

4-In chronic cases there is no respond to treatment(Irreversible)

5-In cattle with ocular form of deficiency will no respond to treatment and should slaughter.

Control

- The minimum daily requirement in all species is 40IU/Kg (daily or weekly in green or concentrated feed) diatory supplementation.
- During pregnancy, lactation or rapid growth , should feed double dose.
- An alternative method is IM. Injection of Vit. A intervals of 50-60 day, at the rate of 3000-6000IU/Kg.B.Wt.