Copper deficiency

Copper deficiency may be:

1- primary when the intake in the diet is inadequate.

2- secondary when the dietary intake is sufficient but the utilization of the copper by the tissue is impeded.

Primary copper deficiency

The amount of copper in diet is adequate, but conditioning dietary factors interfere with utilization of the copper which include:

a- Dietary excess of molybdenum

b- Zink, iron, lead and calcium carbonate are also conditioning factors.

c- Administration of selenium to sheep on copper deficient pastures increase copper absorption and improve the growth of lambs.

d- Dietary inorganic sulfate in combination with molybdenum has a profound effect in the uptake of copper in ruminant.

Pathogenesis

Effect on tissue

Copper is necessary in tissue oxidation by either supplementation of cytochrome oxidase system or entering into their formation.

Ceruloplasmin in the copper – containing enzymes through which copper exerts its physiological functions.

The pathogenesis of most lesion has been explained in term of faulty tissue oxidation because of failure of these systems.

This role is the exemplified early stage of copper deficiency by the changes in this wool of the sheep.

a- Wool

The straightness and stringiness of this wool is due to inadequate keratinization probably due to imperfect oxidation of free thiol group.

b- Body weight

In the latter stage of copper deficiency the impairment of tissue oxidation cause interference with intermediary metabolism and loss of condition or failure to grow.

c- Diarrhea

The pathogenesis of copper deficiency in causing diarrhea is uncertain and there is little evidence that a naturally occurring primary copper deficiency cause diarrhea.

d- Anemia

The known importance of copper in the formation of haemoglobin account for the anaemia in copper deficiency.

The presence of haemosiderin deposits in tissue of the copper deficient animals suggest that copper in necessary for the reutilization of iron liberated from the normal breakdown of haemoglobin.

e- Bone

The osteoporosis that occurs in some natural cases of copper deficiency is caused by the depression of osteoplastic activity.

f- Connective tissue

Copper is a component of the enzyme lysyl oxidase, secreted by the cells involved in the synthesis of the elastin component of connective tissue and has important functions in maintaining the integrity of tissues such as capillary beds, ligaments and tendons.

g- Hart

The myocardial degeneration of **falling disease** may be terminal manifestation of **anemic anoxia** or due to interference with tissue oxidation.

h- Nervous tissue

Copper deficiency cause decrease in the formation of **myelin** and cause **demyelination in lambs.**

Clinical findings

The general effect of copper deficiency is the same in sheep and cattle.

Cattle

1- Sub clinical hypocuprosis

No clinical sings occur, blood copper levels are marginal or below 57 mg/dl (9.0 m mol/L).

2- General syndrome

a- primary copper deficiency

1- Primary copper deficiency cause unthriftness, loss of milk production, and anaemia in adult cattle

2- The **coat colure** is affected, red and black cattle changing to **rusty red** and coat becomes rough.

3- In severely deficient states, calves grow poorly and there is an increased tendency for bones to fracture particularly the limb bones and scapula.

4- Ataxia may occur after exercise with a sudden loss of control of the hind limbs and the **animal falling** or assuming the sitting posture.

5- Itching and hair licking also recorded.

6- Although diarrhea is occur persistence diarrhea is not characteristic of primary copper def.

7- In some cases the calves develop stiffness and enlargement of the joints an contraction of the flexor tendon causing the affected animal to stand on their toes.

b- Secondary copper deficiency

This syndrome include the sings of primary copper deficiencies except that **anaemia** occur less commonly probably due to relatively better copper status in the secondary state.

c- Falling disease (primary deficiency)

The characteristic behavior in falling disease is for cow in apparently good health to throw up their head, bellow and fall.

Death is occur in most cases. Rare cases show sings of for up to 24 hours or more. These animals periodically lower their heads and pivot on the front legs. Sudden death usually occurs during one of these episodes.

d- Peat scours (treat) (Secondary deficiency)

Persistent diarrhea with the passage of watery , yellow green to black feces with an inoffensive odor occurs soon after the cattle go on to affected pasture, in some cases within 8-10 days. The feces are released without effort, often without lifting the tail. Severe debilitation is common, although the appetite is good.

The hair coat is rough and depigmentation is manifested by reddening or gray flecking , especially around the eyes in black cattle. Animal affected is usually recover in few days following treatment with copper.

e- Unthriftness (Pine) of the calves

The earliest sings are stiffness of gait and unthriftness. The epiphysis of the distal ends of metacarpus and metatarsus may be enlarged and resemble the epiphysis of rapidly growing calve deficient in calcium,

phosphorus or vit D. The epiphysis are painful on palpation and some calves are severely lame.

The pastern are upright and the animal may appear to have contracted flexor tendon. The unthriftness and emaciation are progressive and death may occur in 4-5 months.

Grayness of the hair especially around the eyes in black cattle is apparent. Diarrhea may occur in few cases.

Sheep

General syndrome

1- primary copper deficiency

a- **Abnormalities of the wool** are the first observed signs and may be the only sign in area of marginal copper deficiency**. Fine wool** become limp, glossy and loss its crimp developing a straight, steely appearance. Black **wool show depigmentation** to gray or white.

b- Anemia, scouring, unthriftness and infertility may occur in condition of extreme deficiency.

2- Enzootic ataxia (primary def.)

Affect only unweaned lamb. In severe outbreaks, the lambs may affected at birth, but most cases occur in the 1-2 months age group.

The severity of the paresis decrease with increasing age at onset.

Lamb affected at birth or within the first month usually die within 3-4 days. The disease of older lamb may be last for 3-4 weeks and survival is more likely.

The first sings appear in **enzootic ataxia** is in coordination of the hind limbs (appearing when the lambs are driven).

As the disease progress the incoordination become more severe and may be apparent after walking only few yards. There is **excessive flexion** in **joints, knuckling over of the** fetlock , **wobbling of the hind quarters** and finally falling.

The hind leg are affected at first and the lamb may be able to drag itself about in a sitting posture. When the fore legs eventually become affected, **recombancy, paresis** and the lamb dies of inanition. There is no true paralysis, the lambs being able to kick vigorously even in recumbent stage. The appetite is remaining unaffected.

Clinical pathology

1- measurement of plasma copper conc.

Normal in cattle 0.07-0.17 mg/ml → 0.01 - 0.02

In sheep 0.07- 0.13 mg/ml ↗

2- Conc. copper in liver

Normal cattle more than 100 ppm → 20 - 10

Sheep more than 200 ppm → 20 - 15

3- Hb (8-5 gr% /L) normal (8-15gr %).

4- Estimations of copper in pasture and soil

Differential diagnosis

1- Jhones disease
2- Salmonellosis
3- Coccidiosis
4- Mucosal diseases

Diarrhea

5- Vit E deficiency (enzootic ataxia)
6- Sudden death disease (Falling disease)
7- Riktes
8- Cobalt def.

Daily requirement of copper 10mg/kg Dm in cattle and 5mg/kg Dm in sheep

Treatment

- Oral dosage of 4gr of copper sulfate for calves from 2 - 6 months of age and 8 - 10 gr for mature cattle given weekly for 3-5 weeks, 1.5gr weekly for sheep.
- Parentral injection of copper glycenate may also be used the dosage are given under control .
- The diet of affected animals should be supplemented with copper as copper sulfate 10 ppm for cattle and 5 ppm for sheep.