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 secretary as well as covering functions. The secretary cells are without power to divide and develop from undifferentiated basal epithelium. In Vit A deficiency these secretary cells are gradually replaced by the stratified keratinizing epithelial cells common to non secretary epithelial tissues. The occurs chiefly in the salivary glands, urogental tracts but not ovaries or renal tubules and the paraocular glands and teeth. The secretion of thyroxin is markedly reduced. The mucosa of the stomach is not markedly affected. These changes in epithelium leads to clinical of sings of placental degeneration, xerophthalmia and corneal changes.

Embryological development

Vit A is essential for organ formation during growth of the fetus. Multiple congenital defects occurs 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 sings 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 ad 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 inappettence, 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 somniferous 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 claves. In claves 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 form 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 sings 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

Parentral injection is an aqueous rather than oily.

Vitamin D deficiency

Vitamin D deficiency is usually caused by insufficient solar irradiation of animals or their feed and is manifested by poor appetite and growth and in advanced cases by osteodystrophy .

Etiology:

A lack of ultraviolet solar irradiation of the skin, coupled with deficiency of preformed vitamin D complex in the diet , leads to deficiency of vitamin D in tissues

Epidemiology:

A – Ultraviolet irradiation:

The lack of ultraviolet irradiation becomes important as distance from equator increases and the sun rays are filtered and refracted by an increasing depth of the earths atmosphere . Cloudy , overcast skies , smoke laden atmosphere , and winter months exacerbate the lack of irradiation . The effects of poor irradiation are felt first by animals with dark skin or heavy coats , by rapidly growing animals and by those that are housed indoors for long periods . The concentration of plasma vit D3 recorded in grazing sheep varies widely throughout the year . During the winter months the level in sheep fall below what is considered optimal , while in the summer months the level are more than adequate . There is marked differences in vit D status between sheep with long fleece and those that have been recently shorn , especially in the periods of maximum sunlight . The higher blood levels of vit D in the later group probably due to their greater exposure to sunlight .

B – Dietary vit D :

The important of dietary source of preformed vit D must not be under estimated . Irradiated plant sterols with anti – rachitic potency occur in the dead leaves of growth plants . Variation in vit D content of hay can occur with different methods of curing . exposure to irradiation by sunlight for long periods causes marked increase in anti – rachitic potency of cut fodder , whereas modern hay making technique with is emphasis of rapid curing tends to keep vit D levels at minimum .

C – Grazing animals

The grazing of animals , especially in winter time , in lush green feed including cereal crops leads to high incidence of rickets in the young .

Pathogenesis :

Vit D is a complex of substances with anti – rachitogenic activity . The important components are as follow .

A – Vit D3 (cholecalceferol) is produced from its precursor 7- dehydrocholesterol in mammalian skin by natural irradiation with ultraviolet light .

B – Vit D2 is present in sun – cured hay and is produced by ultraviolet irradiation of plant sterols . Calciferol or viosterol is produced commercially by the irradiation of yeast . Ergosterol is a provitamin .

C- Vit D4 and D5 occur naturally in the oils of some fish.

Vit D produced in the skin or ingested with diet and absorbed by small intestine is transported to the liver . In the liver 25- hydroxycholecalceforol is produced which is then transported to the kidney where at least two additional derivatives are formed by 1-α hydroxylase . One is 1 , 25 – dihydroxycholecalciferol (DHCC) and the other is 24 ,25 DHCC . Under conditions of calcium need or calcium deprivation the form predominantly produced by the kidney is 1,25 DHCC . At present , it seems likely that 1,25-DHCC is the metabolic form of vit D most active in eliciting intestinal calcium transport and absorption The metabolite also functions in regulating the absorption and metabolism of phosphate ion and especially its loss from the kidney . A deficiency of metabolite may occur in animals with renal diseases , resulting in decreased absorption of calcium and phosphorus , decreased mineralization of bone , and excessive losses of minerals through the kidney.

Clinical findings :

The most important effect of lack of vit D in farm animals is reduced productivity. A decrease in appetite and efficiency of food utilization cause poor weight gains in growing stock and poor productivity in adults. Reproductive efficiency is also reduced and the overall effect on the animal economy may be severe.

In the late stages lameness which is most noticeable in the forelegs is accompanied in young animals by bending of the long bones and enlargement of the joints . This later stage of clinical rickets may occur simultaneously with cases of oseomalacia in adults .

Clinical pathology :

A- Serum calcium and phosphorus :

Typical figure for beef cattle kept indoors are serum calcium 8.7 mg / dl , serum inorganic phosphate 4.3 mg / dl and alkaline phosphatase 5.7 unites .

B – Plasma Vit D

The normal range of plasma concentration of vit D and its metabolites in farm animals are know available and can be used to monitor the response to administration of vit D parenterally or orally in sheep .

Treatment :

It is usual to administer Vit D in the dose rate of 7 – 12 IU / kg BW . Affected animals should be also receive adequate calcium and phosphorus in the diet

Rickets

Rickets is a disease of young growing animals characterized by defective calcification of growing bones .

Etiology :

Rickets is caused by an absolute or relative deficiency of any or a combination of calcium , phosphorus or vit D in young growing animals . The effect of deficiency are also exacerbated by a rapid growth rate .

Pathogenesis :

Dietary deficiencies of calcium , phosphorus , and vit D result in defective mineralization of the osteoid and cartilaginous matrix of developing bones . There is

persistence and continued growth of hypertrophic epiphyseal cartilage , increasing width of the epiphyseal plate . Poorly calcified specules of diphyseal bones and epiphyseal cartilage yield to normal stresses , resulting in bowing of the long bones and broadening of the epiphyses with apparent enlargement of the joints . Rapidly growing animals on an otherwise good diet will be first affected because of their higher requirement of the specific nutrients.

Clinical findings:

Clinical rickets is characterized by

A- Stiffness of the gait .

B- Enlargement of the limb joints especially in the foreleg

C - Enlargement of the costochondral junction.

D – Long bones show abnormal curvature, usually forward and outward the carpus in the sheep and cattle.

E – Lameness and tendency to lie down for long periods.

Outbreaks affecting 50 % of the group of the lambs have been described . Affecting of the back and contraction , often to the point of virtual collapse , of the pelvis occur and there is an increased tendency for bone to fracture .

Eruption of the teeth is delayed and irregular and the teeth are poorly calcified with pitting , grooving and pigmentation . They are often badly aligned and wear rapidly and unevenly . These dente abnormalities together with the thickening and softness of the jaw bones may make impossible for severely affected calves and lambs to close their mouths . The tongue is protrude and there is drooling of saliva and difficulty in feeding . In less severely affected

animals , dental malocclusion may be a significant occurrence . Severe deformity of the chest may in dyspnea and chronic ruminal tympany. In the final stage , the animal show hypersensitivity , tetany , recumbency and eventually dies of inanition .

Clinical pathology:

1 - The plasma alkaline phosphatase is commonly elevated , but the serum calcium and phosphorus levels depend on the causative factors. If phosphorus or vit D deficiencies are the cause , the serum phosphorus level will usually be below the normal lower limit of 3 mg / dl . The serum conc. of 25 hydroxy vit D3 and 25 hydroxy vit D2 are markedly decreased in vit D deficient rickets compared with normal values of more than 5 ng / ml.

2 – The radiographic examination of bones and joints is one of the most valuable aids in the detection of rickets . Rachitic bones have characteristic lack of density compared with normal bones . The ends of the long bones have a woolly or moth-eaten appearance and have a concave or flat instead of the normal convex contour .

Differential diagnosis :

1 – Copper deficiency .

2 – Epiphysitis

3 – Congenital and acquired abnormalities of the bony skeletal system

Treatment and control :

As in Vit D , calcium and phosphorus deficiencies .

Vitamin K deficiency

A primary deficiency of Vit K is unlikely under natural condition in domestic animals because of high content of substances with Vit K activity in most plants and the substantial synthesis of these substances by microbial activity in the alimentary canal. Sporadic cases may occur when impairment of the flow of bile reduces the digestion and absorption of this fat soluble vitamin**.**

Clinical findings :-

A haemorrhagic disease of recently weaned pigs from 6-15 weeks of age is considered to be associated with Vit K deficiency. Affected pigs fail to grow, become pale, develop large subcutaneous heamatomas and exhibit lameness and epistaxis . Excessive and fatal haemorrhage following routine castration may occur is pigs from 30- 40 days of age but not 15-20 day of age. Prothrombine time and activated partial thromboplastine time are prolonged along with decreased levels of Vit K dependent factors II, VI , X1, X . At necropsy haemorrhage are extensive in the muscles of hindlimbs, forelimbs and auxiliary mandibular region.

Treatment

Vit K given at dose of 3 mg/kg B.W as single dose will restore the blood coagulation defects to normal.

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 being to rise again at about 3 weeks of ages. However a dermatosis of young calves has been associated with low levels of ascorbic acid in their plasma and responds to a signal 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 interested 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 dose 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 difiecncy 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 opsthotonous 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 mongastric 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_2)

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 commeasures, on the edge of the lips and around the navel. There are no ocular lesion.

Pantothenic acid deficiency (Vit B_3)

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_4 & B_5)

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 claves sings 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 diary 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_6) deficiency

A deficiency of pyridoxine in diet is not known to occur under natural condition. Experimental deficiency in pigs is characterized by periodic epiliptiform 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 elipletiform 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 gluconeogensis 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 it's 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 possibly 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 rout. 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 Charolias 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 claves are no 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 milk but did not affect fat or protein concentration in the milk.

Vit B_{12} deficiency (Cyanocobalamin)

Vit B_{12} 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, sings include anorexia, cessation of growth, loss of condition and muscular weakness. Daily requirement under these condition is 20 - 40 mg of Vitamin B_{12}.

Calcium deficiency

Calcium deficiency may be primary or secondary, but in both cases the end result is an osteodystrophy , the specific disease depending largely on the species and age of the animals affected.

Etiology :

A primary deficiency due to a lack of calcium in the diet is uncommon, although a secondary deficiency due to marginal calcium intake aggravated by a high phosphorus intake is not uncommon.

Epidemiology

- Sporadic
- Not common if diets adequate

Pathogenesis

- The main physiological functions of calcium are the formation of bone and milk, participation in the clotting of blood and the maintenance of neuromuscular excitability.

- In the development of osteodystrophies, dental defects and tetany, the role of calcium is well understood but the relation between deficiency of the element and lack of appetite, poor growth, loss of condition, infertility and reduced milk flow is not readily apparent. The disclination of the animals to move about and graze and poor dental development may contribute to these effects.

Clinical findings :

The clinical findings are less marked in adult than is young animals, in which there is decreased rate or cessation of growth and dental development. The later is characterized by deformity of the gums, poor development of the incisors, failure of permanent teeth to erupt for periods of up to 27 months and abnormal wear of the

permanent teeth due to defective development of dentine and enamel , occurring principally in sheep.

A calcium deficiency may occur in lactating ewes and suckling lambs whose metabolic requirement for calcium are higher than in dry and pregnant sheep. There is profound fall in serum calcium. Tetany and hyperirritability do not usually accompany hypocalcemia in these circumstances, probably because it develops slowly. However exercise and fasting often precipitate titanic seizures and parturient paresis in such sheep. This is typical of the disease as it occurs in young sheep. Attention is drawn to the presence of the disease by the occurrence of tetany, convulsions and paresis but the important sings are ill- thrift and failure to respond to antihelmentics. Serum calcium levels will be as low as 5.6 mg/dl. There is lameness but fracture are not common even though the bones are soft.

A simple method for assessing this softness is compression of the frontal bones of the skull with the thumbs. In affected sheep, the bones can be felt to fluctuate. Inappetance, stiffness, tendency of bones to fracture, disclination to stand, difficult parturition, reduced milk flow, loss of condition and reduced fertility are all non specific sings recoded in adults.

Primary calcium def.

No specific syndrome are recorded

Secondary calcium def.

Rickets, osteomalacia, osteodystrophia fibrosa of the horses and pigs and degenerative arthropathy of cattle are the common syndrome in which secondary

calcium deficiency is one of the specific causative factors. In sheep, rickets is seldom recognized but there are marked dental abnormalities

Clinical pathology

- Estimation of serum calcium and phosphate levels.
- Radiographic examination of bones.
- Balance studies of calcium and phosphorus retention.

Necropsy findings

- Osteoporosis
- Low ash content of bone

Deferential diagnosis

A close similarity between the dental defects in severe calcium deficiency of sheep and those occurring in chronic fluorosis may necessitate quantitative estimates of fluorine in the teeth or bones to determine the cause.

Treatment

The response of treatment is rapid and the preparations and doses recommended are effective as treatment.

Parentral injection of calcium salts are advisable when tetany is present. The dose is same as in milk fever.

Cobalt deficiency

Is a disease of ruminants ingesting the diet deficient in cobalt which is required for the synthesis of vit B_{12} and characterized clinically by inappetance and loss of body weight .

Etiology :

The disease is caused by a deficiency of cobalt in the diet which result in deficiency of vit B_{12} .

Pathogenesis :

1 - Cobalt is unique as an essential trace element in ruminant nutrition because it is stored in the body in limited amounts only and not in all tissues. In the adult ruminants it is only known function is in the rumen and it must therefore be present continuously in the feed .

2 – The effect of cobalt in rumen is to participate in the production of vit B_{12} (cyanocobalamin) , and compared to other species the requirement for vit B_{12} is very much higher in ruminants .

3 – The essential defect of cobalt deficiency in ruminant is an inability to metabolize the propionic acid lead to ketosis and death of animal .

4 – The pathogenesis of ovine white liver disease is unknown .

Clinical findings :

No specific signs are characteristic for cobalt deficiency .

1 – A gradual decrease of appetite is the only obvious clinical sign . It is accompanied by loss of body weight , emaciation and weakness .

2 – Pica is likely to occur especially in cattle .

3 – Marked pallor of the mucus membranes and affected animals are easily fatigued .

4 – Growth, lactation and milk production are severely retarted .

5 – The wool may be tend to broken.

6 – In sheep , severe lacrimation with profuse outpouring of the fluid sufficient to mat the wool of the face is one of the most important sign in advanced cases .

7 – Signs usually become apparent when animals have been on affected area for about six months and death occur in 3 – 12 months after the first appearance of illness.

8 – Cobalt deficiency in pregnant ewes can result in decreased lambing percentage , increase percentage of stillbirths and increased neonatal mortality .

9 – Lambs from deficient ewes are slower to start sucking , have reduced concentration of serum colostral immunoglobulins and have lower vit B_{12} concentration than lambs from normal ewes .

Clinical pathology :

1 – Reduction of cobalt concentration in blood , normal value is 1 – 3 Mg / dl (0.17 – 0. 51 Mmol / L) reach to 0.03 – 0.41 Mmol / L .

2 – Decrease of vit B_{12} concentration in serum reach to less than 20 mg% .

3 – Decrease of RBCs count .

4 – Estimation of cobalt and vit B_{12} concentration in liver .

5 – Increase of methylmalonic acid concentration in urine and plasma .

Differential diagnosis :

1 – Jhones disease .

2 – Osteomalacia .

3 – Dietary deficiency of copper , selenium and vit D .

Treatment :

1 – Cobalt chloride or cobalt sulfate 1 – 2 mg per sheep and 5 mg per cattle orally with water daily for 3 weeks .

2 - Vit B_{12} 4 – 7 Mg / kg IM once weekly for 3 – 4 weeks .

3 – Addition of cobalt to diet (1 gr cobalt sulfate or chloride / 1000 kg DM).

* Daily requirements of cobalt in cattle is 0. 11 mg / kg DM .

Copper deficiency

Copper deficiency may be primary when the intake in the diet is inadequate or 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 are 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

<u>1- primary copper deficiency</u>

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 remain 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.

Iodine deficiency

Etiology

Iodine deficiency may be due to the deficient iodine intake or secondary by high of calcium.

Occurrence

The importance of sub clinical deficiency as a cause of neonatal mortality could be much greater than clinical disease .

Young animals are most likely to bear goitrous offspring than older ones and this may account for the apparent breed susceptibility of Dorset horn sheep which mate at an earlier age than other breeds.

Pathogenesis

- iodine deficiency result in decrease production of thyroxin and stimulation of the secretion of thyrotropic hormone by the pituitary gland hyperplasia of thyroid ⟶ tissue and enlargement of thyroid gland.
- The primary deficiency of thyroxin is responsible for the severe weakness and hair abnormalities of the affected animals.
- A hyperplastic goiter is highly vascular and the gland can be felt to pulsate with the arterial plus loud murmur may be audible over the gland .
- Iodine is on essential element for normal fetal brain and physical development in sheep. A severe iodine deficiency in pregnant ewes cause reduction in fetal brain and body weight form 70 day of gestation to parturition.

Clinical findings

1- Although loss of condition, decrease milk production and weakness might be anticipated these signs are not usually seen in adults.

2- Loss of libido in the bull, failure to express estrus in cow and high incidence of aborted , stillborn or weak calves have been suggested as manifestations of hypothyroidisim in cattle where as prolonged gestation is reported in mares, ewes and sows.
3- A high incidence of still births and weak newborn animals is the most common manifestation of iodine deficiency.
4- Partial or complete alopecia and palpable enlargement of thyroid gland are the sings that occur most frequently in different species.
5- Excessive flexion of the lower for legs and extension of lower parts of the hind legs has also been observed in affected foals.

6- Defective assification has also been reported , the manifestation in collapse of the central and third tarsal bones leading to laminess and deformity of the hock.
7- Adult sheep in iodine deficiency areas may show a high incidence of thyroid enlargement but are clinical normal in other respects.
8- New born lambs manifest weakness, extensive alopecia and palpable (if not visible) enlargement of thyroid glands.
9- Goat present a similar clinical picture (more severe than sheep).
10- Animal survive the initial danger after birth may recover except partial persistence of goiter.

Clinical pathology

1- Estimation of blood iodine levels.

Normal range (2.4-14 mg/100ml).

2- Estimation the iodine conc. In milk.

The conc. Below 8mg/Lo indicate iodine deficiency.

3- Levels the thyroxin in the blood have not been much used to measure the thyroid gland sufficiency in animals.

4- Blood cholesterol levels have been used as an indicator of thyroid function in human but are not used in the investigation of goiter in animals.

Differential diagnosis

1- Iodine deficiency should be differentiated from other causes of abortion and stillbirth such as brucellosis.

2- Vit A def. (weakness).

3- Cobalt def. and copper def. (coat lesion).

Treatment

1- Treatment of neonates with obvious clinical evidence of iodine deficiency is usually and undertaken because of the high case fatality rate.

2- Addition of iodine to the diet of pregnant dams.

3- Potassium iodine 6-10gr with water orally.

4- Vit A or fish oil (440 1u/kg bw) Vit A daily for 7 days

5- Thyroxin tablet for calves and lambs. On tablet (3mg) daily for 7 days.

Daily requirement of iodine is 0.8-1.0 mg/kg Dm of feed for lactating and pregnant cows and 0.1- 0.3 mg/kg Dm of feed for non pregnant cows and calves.

Manganese deficiency

A dietary deficiency of manganese (Mn) may cause infertility and skeletal deformities both congenitally and after birth .

Etiology:

1- A primary deficiency occurs endemically in some area because of a geological deficiency in the local rock formation.

2- An excess of calcium and, or phosphorus in the diet is known to increase the requirements of manganese in the diet of calves, and is considered to reduce the availability of dietary manganese to cattle generally.

3- Congenital chondrodystrophy in calves has been associated with manganese deficiency , and outbreak of congenital skeletal defects in Holistein calves due to manganese deficiency has been reported .

Epidemiology :

1- Soil containing less than 3 mg / kg of manganese are unlikely to be able to support normal fertility in cattle. In areas where manganese – responsive infertility occurs , soils on farms with infertility problems have contained less than 3 mg / kg of manganese , whereas soils on neighboring farms with no infertility problems have had level more than 9 mg / kg .

2- A secondary soil deficiency is thought to occur and one of the factors suspected of reducing the availability of manganese in the soil to plants is high alkalinity. Thus heavy liming is associated with manganese – responsive infertility . There are three main soil types on which the disease occur.

A- Soils low manganese have low output even when pH is less than 5.5

B- Sandy soils where availability starts to fall .

C-Heavy soils where availability starts to fall at pH of 7.0 .

Pathogenesis :

Manganese plays an active roles in bone matrix formation and in the synthesis of chondroitin sulfate , responsible for maintaining rigidity of connective tissue . In manganese deficiency , these are affected deleteriously and skeletal abnormalities result. Only 1 % of manganese is absorbed from diets and the liver removes most of it , leaving very low blood level of element.

Clinical findings :

In cattle, the common syndrome are infertility, calves with congenital limb deformities, and calves which manifest poor growth, dry coat, and loss of coat color. The deformity include knuckling over the fetlocks, enlarged joints and possibly twisting of the legs . The bones of affected limbs are shorter and weaker than normal and there are signs of joint pain , hoping gait and reluctance to move .

A manganese responsive infertility has been described in ewes and is well known in cattle . In cattle , it is manifested by slowness to exhibit estrus and failure to conceive , often accompanied by subnormal size of one or both ovaries . Subestrus and weak estrus have also been observed .

Clinical pathology :

1- The blood of normal cattle contains 18- 19 Mg / dl (3.3-3.5 Mmol / L) of manganese , although considerably lower level are sometimes quoted .

2- The livers of normal cattle contains 12 mg / kg of manganese and down to 8 mg / kg in newborn calves which also have a lower content in hair

3- The manganese content of hair varies with intake . The normal level is about 12 mg / kg and infertility is observed in association with level of less than 8 mg / kg .

4- In normal cows , the manganese content of hair falls during pregnancy from normal level of 12 mg / kg in the first month of pregnancy to 4.5 mg / kg .

Treatment and control:

The maintenance requirement for Mn represents 82 % of the total Mn requirement for non lactating , late gestation cow and 53 % for a cow producing 40 kg / day of milk .

Recent research has determined that Mn intake had to equal 580 mg / day to meet the metabolic fecal Mn requirement.

Osteomalacia

Osteomalacia is a disease of mature animals affecting bones in which endochondral ossification has been completed. The characteristic lesions are osteoporosis and the formation of excessive uncalcified matrix. Lameness and pathological fractures are common clinical findings.

Etiology

In general the etiology and occurrence of osteomalacia are the same as of rickets except that the predisposing causes is not the increase requirement of growth but the drain of lactation and pregnancy.

Pathogenesis

Increased resorption of bone minerals to supply the need of pregnancy, lactation and endogenous metabolism lead to osteoporosis and weakness and deformity of the bones.

Large amount of uncalcified osteoid are deposited about the diaphysis. Pathological fractures are commonly precipitated by sudden exercise or handling of the animal during transportation.

Clinical Findings:-

In the early stage, the signs are those of phosphorus deficiency, including lowering productivity and infertility and loss of condition. Licking and chewing of inanimate objects begins at this stage and may bring their attendant ills of oral, pharyngeal, and esophageal obstruction, TRP, lead Poisoning and botulism.

The sings specific to osteomalacia are those of a painful condition of the bones and joints and include a stiff gait, moderate lameness. Often shifting from leg to leg, crackling sounds while walking, and arched bock.

The hindlegs are mostly severely affected and the hocks may be rotated inwards. The animals are desalinated to move, lie down for long periods and are unwilling to get up. Fractures of bones and separation of tendon attachments occur frequently often without apparent precipitating stress. In extreme cases deformities of bones occur and when the pelvis is affected Dystocia my result. Finally weakness leads to permanent recombancy and death from starvation.

Clinical pathology :

In general the finding are the same as those for rickets, including increase serum alkaline phosphatase, and decrease serum phosphorus levels. Radiographic examination of long bones shows decrease destiny shadow.

Differential diagnosis

- Vit D deficiency.
- Magnesium deficiency .
- Chronic flourosis .

Treatment and control

Recommendations for the treatment and control of the specific nutritional deficiencies should be done. Some weeks will elapse before improvement occurs and deformities of the bones are likely to be permanent.

Phosphorus deficiency

Phosphorus deficiency is usually primary and characterized by pica, poor growth, infertility and in the later stage osteodystrophy. Hypophosphatemia in dairy cattle is also associated with increased fragility of red blood cells and post parturient haemoglobin urea.

Etiology

Phosphorus deficiency is usually primary under field conditions but may be exacerbated by deficiency of Vit D and possibly by an excess of calcium.

Epidemiology

Primary phosphorus deficiency occurs worldwide. Soils and crops commonly deficient in phosphorus. Primary deficiency may occur in lactating cattle in early lactation. Phosphorus deficiency occurs under rang of condition in beef cattle and sheep.

Pathogenesis

- From 80- 85% of the phosphorus of the body located in the skeleton where it occur as hydroxyapatite in a 1.0 : 1.7 ratio with calcium. The two minerals provide bone strength necessary for normal activities.
- Bone phosphorus also functions as an important phosphorus reservoir for resorption when body requirements exceed dietary intake. From 17-42% of bone reserve could be resorbed in cattle and sheep in times of phosphorus deficiency .
- Phosphorus is also essential for a broad range of enzymatic reactions especially those concerned with energy metabolism and transfer. Phosphorus is also essential for the transfer of genetic information and is a vital component of various buffering system. Phospholipids are necessary for maintenance of cell wall structure and integrity and as a integral component of myeline.
- Rumen microbes have a phosphorus requirements a part of animals requirements which must be meet for optimum rumen microbial activity to occur.
- Phosphorus is essential for the laying down of adequately mineralized bones and teeth and a deficiency will result in their abnormal development. Inorganic phosphorus which may be ingested or liberated from esters during digestion or intermediary metabolism is utilized in the formation of protein and tissue enzymes and is withdrawn from the plasma inorganic phosphate, for this purpose.

Clinical findings

Primary phosphorus deficiency is common only in cattle. Young animals grown slowly and develop rickets. In adults there is an initial sub clinical stage followed by osteomalacia.

In cattle of all age a reduction in voluntary intake of feed is a first effect of phosphorus deficiency and is the basis of most of the general systemic sings. Retarded growth, low milk yield and reduced fertility are the earliest signs of phosphorus deficiency.

* In the experimental production of phosphorus deficiency in beef cows, several months on a deficient diet are necessary before clinical sings develops. The clinical sings included general Unthriftness, marked body weight loss, reduced food consumption, reluctance to move, abnormal stance, bone fractures, and finally impaired reproduction.

* Acute recombancy in high producing dairy cows on a marginally phosphorus deficient diet may become recumbent in early lactation. Affected animals are recumbent and can not stand. They may be bright and alert and their vital sings are within normal range.

Clinical pathology

- Estimation of serum phosphorus. Clinical sings occur when blood levels have fallen from the normal of 4 - 5 mg/dl to 1.5 - 3.5 mg/dl.
- Phosphorus content in diet.

Differential diagnosis

- The disease should be differentiated from those diseases that may be resemble to rickets and osteomalacia.

Treatment

As in PPHb urea.

Selenium and Vit E deficiency

Etiology :

Dietary deficiency of selenium and Vit E and conditioning factors like dietary polysaturated fatty acids.

Epidemiology :

***** Enzootic muscular dystrophy occurs in young growing calves, lambs, goat kids and foals borne to dams in selenium deficient areas and unsupplemented. Occur world wide and common in Australia, UK great planes of north America where soils are deficient to selenium. Vit E deficiency in animals fed poor quality forage and diets high in polyunsaturated fatty acids. Outbreaks of muscular dystrophy precipitated by exercise.

* Mulberg heart disease in finishing pigs.

* Selenium responsive disease occur in Australia and are not obvious clinically but respond to selenium supplementation. Selenium and Vit E deficiency may be involved in reproductive performance, retained placenta in cattle. Resistance to infectious diseases controversial.

Clinical findings :

Muscular dystrophy characterized by groups of animals with stiffness, weakness, recombancy. Myocardial from mulberry heart disease characterized by outbreak of sudden death in finishing pigs.

Acute enzootic muscular dystrophy :

Affected animals may collapse and die suddenly after exercise without any other sings. The excitement associated with the hand-feeding of diary calves may be

precipitate peracute death. In calves under close observation, a sudden onset of dullness and severe respiratory may be observed in some cases.

Affected calves, lambs and foals are usually in lateral recombancy and may be unable to assume sternal recombancy even when assisted. When picked up assisted to stand they feel and appear limp. However their neurological reflexes are normal.

Their eyesight and mental attitude are normal and they are usually thirsty and swallow unless the tongue is affected. The heart rate is usually increased up to 150-200 per minute and often with arrhythmia. The respiratory rate is increased up to 60-72 per minute and loud breath sound are audible over the entire lung field . The temperature is usually normal or slightly elevated. Affected animals commonly dies 6-12 hours after onset of sings in spite of therapy.

Sub acute enzootic muscular dystrophy

This is the most common form in rapidly growing calves (white muscle disease) and in young lambs (stiff lamb disease). Affected animals may be found in sternal recombancy and unable to stand , if they are standing the obvious sings are stiffness, trembling of the limbs. Weakness and in most cases an inability to stand for more than few minutes. The gait in calves is accompanied by rotating movement of the hocks and in lambs a stiff goose – stepping gait. Muscular tremor is evident if the animal is forced to stand. On palpation the dorsolimber, gluteal and shoulder muscles firmer than normal . Most affected animals retain their appetite and will suck if held up to the dam or eat if hand fed. Major involvement of the diaphragm and intercostals muscles causes dyspnea with labored and abdominal type of respiration. The temperature is usually in the normal range but there may be transient fever (41) due to the effect of myoglobenemia and pain. The heart C° rate may be elevated but there are usually no rhythmic irregularities. Following

treatment affected animals usually respond in few days and within 3-5 days they are able to stand and walk unassisted.

Congenital muscular dystrophy has been described in new born calves. The calf will still recumbent 13 hours after birth, had increased serum creatinin kinase and decreased vit E and selenium levels. Recovery occurred following supportive therapy and vitamin E and selenium.

Sub capsular liver rapture in lambs has been associated with Vit E deficiency in lambs usually 4 weeks of age. Affected lambs collapse suddenly, become limp and die within a few minutes or several hours after the onset of weakness.

In foals, muscular dystrophy occurs most commonly during the first few months of life and is common in the first week. The usual clinical findings are failure to suck, recombancy , difficulty in rising, and unsteadiness and trembling when forced to stand. The temperature is usually normal but commonly there is polypnea and tachycardia. The disease is faols may be characterized by an acute fulminate syndrome which is rapidly fatal or subacute syndrome characterized by profound muscular weakness, failure of passive transfer, aspiration pneumonia and stunting are frequent complications. In the subacute form mortality rate may range from 30-45%.

In adult horse with muscular dystrophy a stiff gait , myoglobinuria, depression , inability to eat, holding the head down low and edema of the head and neck are common. The horse may be presented initially with clinical sings of colic.

Mulberry heart disease : usually seen in pigs .

Clinical pathology :

*** Myopathy**

- Plasma creatinin kinase (CK). The enzyme is highly specific for cardiac and skeletal muscle and is released into the blood indicate the myopathy.
  - Aspartames amino transferase .
  - Serum selenium level is low .
  - Serum Vit E level is low .
  - Decrease of glutathione peroxidase activity .

Differential diagnosis :

- Acute muscular dystrophy of calves and yearlings

a- *Haemophilus somnus* septicemia

b- pneumonia

- Sub acute enzootic muscular dystrophy

a- Musculoskeletal disease such as

1- Polyarthritis .

2- Traumatic or infectious myopathies (black leg) .

3- Osteodystrophy .

4- fracture of the long bones .

b- Disease of nervous system

1- Spinal cord compression

2- *H. Somnus* minengoencephalitis

3- Organophosphate insecticide

c- Disease of the digestive tract

- Carbohydrate engorgement resulting in lactic acidosis.
- Shock, dehydration and weakness .

- Muscular dystrophy in lambs and kids

- Enzootic ataxia and swayback disease .

- Muscular dystrophy in foals

1- Traumatic injury of musculoskeletal system.

2- Polyarthritis .

3- Meningitis .

4- traumatic injury of the spinal cord .

Treatment

A mixture containing 3 mg selenium as (potassium or sodium selenite) and 150 IU /ml of vit E given i/m at 2 ml/ 45 kg b.w. is recommended .

Zink deficiency (parakeatosis)

Etiology

1- A primary Zink deficiency due to dietary Zink in ruminate is rare but dose occur.

2- Many factors influence the viability of Zink from soil including compaction of the soil, nitrogen and phosphorus increase.

3- Consumption of immature which gross which affected digestibility, the feeding of late – cute hay which may be poorly digestible and the presence of excessive dietary sulfur. All these cause secondary Zink deficiency.

Pathogenesis

1- Zink is component of the enzyme carbonic anhydrase which is located is red blood cells and parietal cells of the stomach and is related to the transport of respiratory carbon dioxide and the secretion of hydrochloride acid by the gastric mucosa.

2- Zink also associated with RNA function and related to insulin, glucagons and other hormones.
3- Zink also have a role in keratinization , calcification, wound healing and somatic and sexual development.
4- A Zink deficiency results in a decreased feed intake in all species and probably the reason for the depression growing rate in growing animals.
5- Failure of keratinization result in parakeratosis, failure of growth of wool and hair and lesions of the coronary bands probably reflect the importance of Zink in protein synthesis.

Clinical Findings

1- In naturally occurring disease in cattle, in severe cases, parakeratosis and alopecia may affect about 40% of skin area. The lesions are most marked on the muzzle, vulva, anus , tail- head, ears, backs of hind legs, kneefold, flank and neck.
2- Most animal are below average body condition and are stunted in growth.
3- After treatment with Zink improvement is apparent in 1 week and complete in 3 weeks.
4- Experimentally produced cases exhibit the following sings
    a- Poor growth
    b- A stiff gait
    c- Swelling of the coronates, hocks and knees.
    d- Soft swelling containing fluid of the anterior aspect of the hind fetlock.
    e- Alopecia

f- Wrinkling of the skin of the legs, scrotum and on the neck and head especially around the nostrils.

g- Haemorrhage around the teeth.

h- Ulcers of the dental pad.

The natural disease in sheep is characterized by :-

1- Loss of wool and the development of thick, wrinkled skin.
2- Wool eating also occur in sheep.
3- Induced cases in lambs is exhibited reduced growth rate, salivation, swollen hocks, wrinkled skin and open skin lesion around the hoof and eyes.
4- Experimental disease in goats is similar to that in sheep.
5- Impaired testicular growth and complete cessation of spermatogenesis in ram lambs.

Clinical pathology

- Skin biopsy (parakeratosis)
- Estimation of Zink un blood (normal level 8- 120 mg/dl) in sheep and cattle.

Differential diagnosis

1- Mange
2- Exudative epidermitis

Treatment

1- Zink sulfate 300 mg / in water daily for two weeks .
2- The injection of Zink at rate of 2-4 mg/kg bw daily for 10 days also effective.

Zink oxide suspended with olive and given 1/m at dose of 200mg of Zink for lamb.