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In this issue

- Gastro-intestinal parasitism in dairy animals
- Measures to Keep Snakes Away from Animals
- Vitamin and mineral disparities in poultry birds
- Tube cystostomy in bovine calves
- Congenital affections in bovine

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LABORATORY DIAGNOSIS OF GASTRO-INTESTINAL PARASITISM IN DAIRY ANIMALS

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Abstract

Gastrointestinal (GI) parasitic infections constitute a major threat to the health and productivity of livestock throughout the world. The prevalence of these infections is quite high in tropical and subtropical regions including Punjab state, India due to the presence of favourable environmental conditions supporting development and propagation of the non-parasitic stages. The clinical signs exhibited by the animals suffering from GI parasitism are mostly generalised and in the absence of pathognomic signs laboratory diagnosis become very important for the effective control and management of these diseases. Among the various samples utilized for confirmatory diagnosis of GI parasitic infections, examination of faecal samples collected from suspected animals is of utmost importance. The present article presents an overview on the important aspects of faecal examination including collection, macroscopic and microscopic examination, quantification of infection along with common GI parasites prevalent in dairy animals of the region.

Key words: *Gastrointestinal parasitism, faecal examination, EPG.*

Introduction

Parasitism can be defined as an ecological heterogenetic relationship of two distinct organisms in which one is the host and other is a parasite. The word 'parasite' is derived from two Greek words 'para' meaning 'beside' and 'sitos' meaning 'food'. The parasite is an organism that lives inside or on the body of any ecological distinct type of animal called host; is physiologically and metabolically dependent on its host for its habitat, nutrition and is also harmful to the host. Gastrointestinal (GI) parasitism is a disease condition caused by various genera of parasites inhabiting the digestive tract of dairy animals. The harmful effects of GI parasites include competition with hosts for various nutrients present in the food such as carbohydrates, proteins, microelements, etc., decreased utilization & absorption of food nutrients in the digestive tract of hosts, reduction in the feed intake of animals, decreased protein synthesis, increased passage of food without proper digestion in the digestive tract of animals, reduction in the absorptive surface of intestinal microvilli, removal of various body fluids of hosts like lymph and blood and also feeding on solid tissues/exudates, mechanical destruction/obstruction of various tissues of the hosts and variable degree of production losses in affected animals.

The general clinical signs exhibited by the animals suffering from GI parasitism are inappetence, anaemia, diarrhoea, poor growth, and economic losses in the herds. As regards the effect on milk production, it has been reported that cows were able to produce 142 litres of more milk in 100 days after removal of parasites by anthelmintic medication. Furthermore, for a lactation period of 300 days or 10 months, the untreated animals caused monetary losses of approximately Rs. 2980.00 per animal/lactation whereas an increase of 12.83-17.50% milk production in anthelmintic treated groups was observed. All these warrant the importance of laboratory diagnosis of GI parasitism in dairy animals, so that a specific treatment rationale can be prescribed by a clinician, to treat the animals.

Examination of faecal samples for detection of GI parasitism

The examination of faecal samples for detection of GI parasitism constitutes an integral part of clinical diagnosis. It is a well-known fact that in livestock, it is rare to get an animal completely free from parasites and at the same time all animals may not show signs of disease. As regards GI parasitism most of the helminths and coccidia inhabit the gastro-intestinal tract. Therefore, clinical diagnosis, of these gut dwelling parasites depends mostly on identification of their ova/ oocyst/ cyst/ trophozoite in faecal samples.

Collection of faecal samples

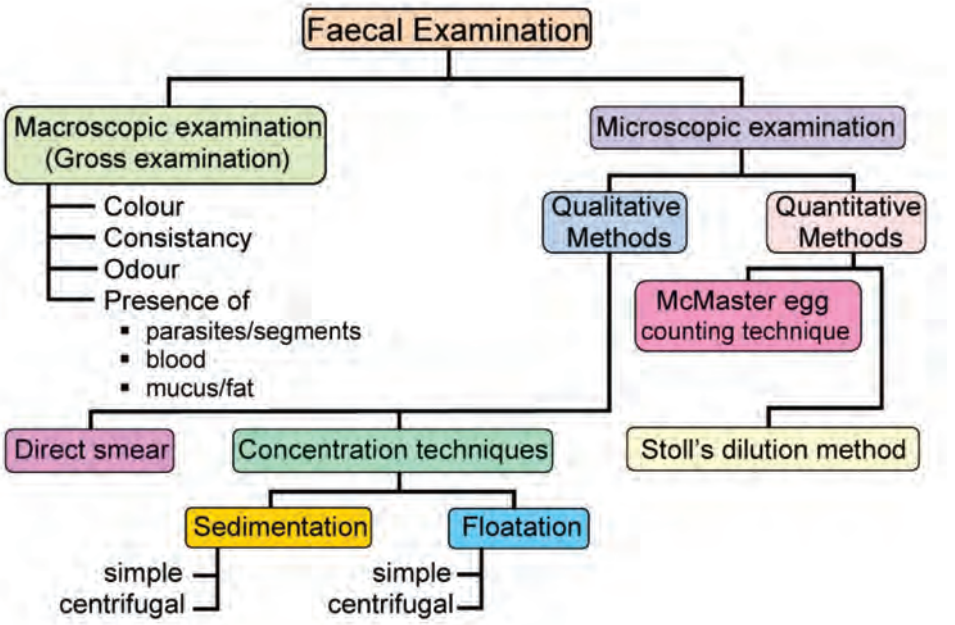
Fresh faecal samples should be collected in a clean and dry stoppered wide mouth bottle. The best way is to collect the sample directly from the rectum manually. For this, use of disposable hand gloves, smear vaseline or paraffin oil on the little finger (for small animals) or entire hand (for large animals) are advisable. Sometimes faecal samples may have to be picked up from the ground (clean pucca floor) but the faeces should be fresh and free from extraneous matters or uncontaminated from urine, soil and dirt. The portion of faeces with mucus alone or with mucus and blood should always be collected for examination, as these often show young or developing stages of the parasites in large numbers (Juyal *et al.,* 2013).

Storage & dispatch of faecal samples

If the faeces cannot be examined within a few hours, the sample should be refrigerated until it can be tested but should not be frozen, because freezing can distort parasite eggs. If a sample needs to be evaluated for the presence of protozoan trophozoites like *Giardia* and trichomonads, it should be examined within 30 min after collection. If fresh faecal material is submitted to another laboratory for examination, it should be packaged with cold packs. Faecal samples suspected for helminthic eggs may also be preserved with an equal volume of 10% formalin while special fixatives, such as polyvinyl alcohol (PVA), are required to preserve protozoan trophozoites.

Examination of faecal sample

The collected faecal samples may be examined as per the flow chart given below:



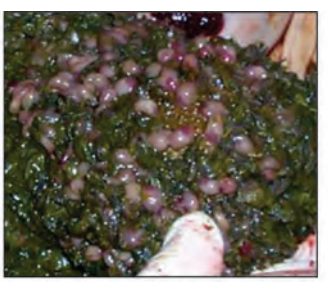
Macroscopic/Gross examination

First, faeces should be examined grossly for detection of living or dead *worms* or for the detection of the segments of tapeworms.

- The ripe segments of *Moniezia* spp. resemble cooked rice grains in the faeces.
- Diarrheic faeces of ruminants should be observed from amphistomes, which appear like pinkish structures like pomegranate in the faeces.
- Sometimes pale yellowish ascarid roundworms may also be seen in faeces that resemble like a spaghetti.



***Moniezia* spp.**
(Source: Internet)



Amphistomes
(Source: Internet)



***Toxocara* spp.**

Furthermore, all the available information like quantity, form, consistency, color, smell, presence of blood/mucus in faeces should also be noted.

- Whipworm infection results in diarrhoea with excess mucus and frank blood.
- Infection with *Toxocara vitulorum* causes mud coloured, fowl smelling diarrhoea along with steatorrhea in calves.

Microscopic Examination

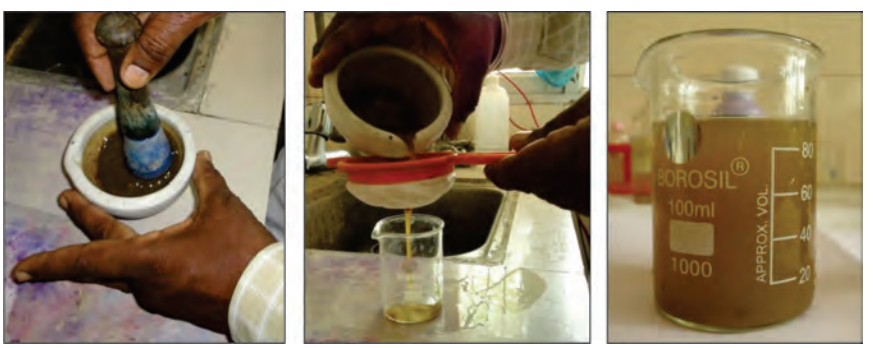
A. Qualitative Examination: It provides information whether a sample is positive or negative for any parasitic infection.

i. **Direct smear method**: In this method, a small quantity of faeces is placed on a glass microscopic slide and mixed with a drop of saline. Slightly tilt the slide to push large particles of debris to the side and place the cover slip on the slide and examined directly under 10x and 40x magnification of the microscope.

It is suitable for very rapid examination, but will usually fail to detect low grade infections. It gives good results for heavy infections. This method though easy, is not reliable and at least three slides should be examined (Zajac *et al.*, 2012).

ii. **Concentration techniques:** The purpose of these methods is to detect light infections as well as others, to save time by concentrating the eggs in a small volume and to eliminate the trouble caused by large faecal particles. Advantage is taken of the specific gravity of helminth eggs to separate them from the faeces.

a. **Simple sedimentation method**: Approximately 1-2 g of faeces is placed in a mortar and little quantity of water is added. Thereafter the material is triturated with pestle to make a suspension. If required, some more quantity of water can be added. The suspension is strained through a sieve and the mixture is transferred to a beaker. It is kept undisturbed for 10-20 min. The supernatant is discarded and sediment is examined under 10x magnification of microscope by transferring a drop of sediment to a glass slide (with a



Simple sedimentation

pipette) and placing a coverslip over it. This method is used to detect heavy eggs like trematode eggs and some cestode eggs (Bhatia *et al.*, 2007).

b. **Simple floatation method**: Approximately 1-2 g of faeces is placed in a mortar and little quantity of saturated salt solution is added. Thereafter the material is triturated with pestle to make a suspension. If required, some more quantity of saturated salt solution can be added. The suspension is strained through a sieve and the mixture is transferred to a clean, dry vial and filled until a convex meniscus is formed. A clean cover slip is slid sideways over the top of the vial so that it is in contact with liquid and is kept undisturbed for 10-15 min. Then the coverslip is gently picked up, placed on glass slide and examined under 10x magnification of microscope. This method is used to detect lighter eggs in faeces e.g., majority of nematode eggs (strongyle) and some cestode eggs.



Simple floatation

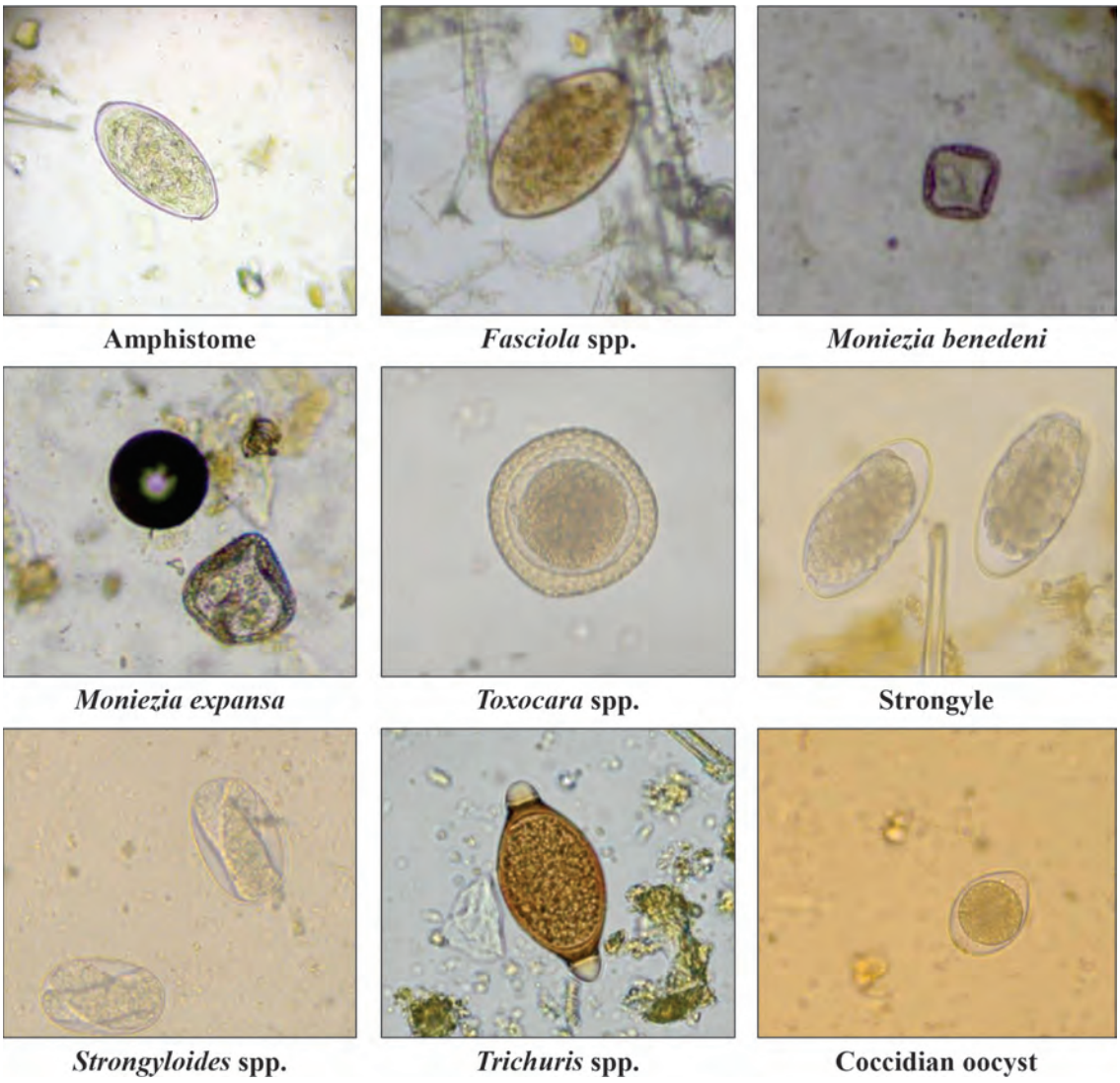
B. Quantitative Examination: This determine the magnitude/severity of infection which is measured as eggs per gram of faeces i.e., EPG.

i. **McMaster's egg counting technique**: The McMaster chamber has two compartments, each with a grid etched onto the upper surface. When filled with faecal suspension, much of debris sinks while eggs float to the surface, where they can easily be seen and counted (Roberts *et al.*, 1950).

Protocol: Three grams of faeces are weighed and triturated in saturated salt solution to make a final volume of 45 mL. After thorough mixing, the suspension is strained through a tea strainer. The chamber of the McMaster slide is filled immediately with the mixture using a pipette or syringe. The entire chamber must be filled, not just the area under the grid. If large air bubbles are present, remove the fluid and refill. The slide is allowed to sit for at least 5 min before examining to allow the flotation process to occur. After focusing with the 10x lens, the number of eggs only within ruled areas is counted. The number of eggs in single ruled area is multiplied by 100, if both areas are counted the average

number of eggs is multiplied by 100 that represents the EPG of the sample.

ii. Stoll's egg counting method: Three grams of faeces are weighed and placed into a 45 mL graduated test tube. The tube is filled with N/10 sodium hydroxide solution to make a final volume up to 45 mL and 10-12 glass beads are added in it. It is shaken vigorously to make a homogenous suspension of the faecal matter. 0.15 mL of suspension is pipetted out and placed on a glass slide, covered with long cover slip and entire eggs (outside & inside) are counted and multiplied by 100, that represents the number of EPG of the sample (Soulsby, 1982).



Important parasitic eggs/oocyst of dairy animals

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BACTERIAL DISEASES OF SMALL RUMINANTS: CAUSES, SYMPTOMS AND DIAGNOSIS

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Abstract

Livestock is an integral part of agriculture sector, that has a huge impact on our country's economy. The major impediment in increasing productivity of livestock is improper management, poor nutrition and disease outbreaks. Animals suffer from a number of infectious diseases thus leading to major economic losses to the farmers. Infectious diseases can be caused by bacteria, viruses and fungi. Here we are focusing on some common infectious bacterial diseases of sheep and goat, so that a better perception of disease can be built up among the livestock farmers and field veterinarians.

Keywords: *Bacterial diseases, goat, infectious diseases, sheep*

Introduction

India holds first rank in livestock population. The current population of livestock is 535.78 million, out of which sheep and goat are 74.26 M and 148.88M respectively. According to 20th livestock census, the highest increase in livestock population is seen in sheep (14.13%) and goat (10.14%). In Indian scenario, sheep and goat farming is considered as a livelihood support system of poor and marginal farmers. The secret behind a profitable livestock farming lies in proper farm management along with formulation of strategies to mitigate and control the diseases. Sheep and goats are mostly affected with infectious agents which include bacterial, viral, fungal and parasitic infections that can lead to great economic losses. Hence, we are aiming to provide a glimpse of some economically important bacterial diseases affecting small ruminants.

Common Bacterial diseases of sheep and goat

Brucellosis

Etiology: It is caused by Gram negative coccobacilli that comes under genus *Brucella*. Though there are many species of Brucella affecting small and large ruminants, but *Brucella ovis* and *Brucella melitensis* are responsible for ovine and caprine brucellosis respectively. *Brucella melitensis* has zoonotic significance which can cause Malta fever or undulating fever in humans.

Clinical manifestation: The foremost clinical feature is abortion in late pregnancy in does and ewes, accompanied by reduced milk yield, still birth, acute fever and septicaemia.

Orchitis, epididymitis is observed in rams. Kids and lambs develop osteoarthritis, hygroma and synovitis.

Diagnosis: Diagnosis can be achieved by direct demonstration of bacteria from vaginal discharge, abomasal content of foetus, placental fluid, by Gram's stain and modified Ziehl-Neelsen staining and Culturing of brucella in 5-10% blood agar or Farrell's medium at 37°c for 24 hrs and the isolates can be confirmed by biochemical and molecular tests. Serological diagnosis like serum agglutination test (gold standard test), Rose Bengal plate test, complement fixation test, ELISA are used. Strauss test is the biological test, in which Guinea pig is inoculated with suspected fluid and orchitis is seen in positive result.

Anthrax: It is a per acute, acute, or subacute fatal disease affecting all species including sheep and goats.

Etiology: It is caused by Gram positive, nonmotile, aerobic, spore forming bacterium *Bacillus anthracis*.

Clinical manifestation: Per acute form is characterized by death within 24 hr. In Acute case, there will be fever, anorexia, rumen stasis, oozing of unclotted blood from natural orifices, convulsion and death. Absence of rigor mortis, rapid putrefaction, bloating of carcass and splenomegaly are the main post-mortem changes.

Diagnosis: Bacteria can be demonstrated from blood smear by polychrome methylene blue stain and can be cultured in 5% sheep blood agar or selective medium like PLET agar. Medusa head colonies can be seen on medium plates and the isolates can be confirmed by molecular tests. Ascoli test is widely used precipitation test for diagnosis of anthrax.

Clostridial diseases

Black quarter: Causative agent is *Clostridium chauvoei*

Clinical manifestations: Fever, increased heart rate, lameness in leg and crepitating sound in the affected muscle, swelling and black discoloration of the leg, death may occur within 1-2 days.

Diagnosis: Demonstration of Gram-positive rods with subterminal spores in stained blood smear. Isolation of bacteria is difficult but it can be cultured in fresh blood agar or semi solid media like cooked medium with thioglycolate. Clostridial species can also be identified by fluorescent antibody technique.

Tetanus: Highly fatal disease affecting nervous system of the animal's body.

Etiology: Gram positive spore producing *Clostridium tetani*. It produces tetanospasmin toxin that cause spastic paralysis.

Clinical manifestation: Stiffness of muscle, tremor, prolapse of 3rd eyelid, uncontrolled

muscle contraction, convulsion, bending of spine, saw horse stance and death within 24 - 48 hrs.

Diagnosis: Demonstration of drum stick shaped Gram-positive rods in Gram staining. Isolation of bacteria in blood agar in anaerobic condition. Identification of toxin is a crucial step in diagnosis of tetanus.

Enterotoxaemia

A severe disease of small ruminants caused by *Clostridium perfringens*. Different strains responsible for occurrence of diseases are mentioned below:

Clostridium perfringens	**Host**	**Diseases**
Type A	Lambs	Enterotoxaemic jaundice
Type B	Lamb	Lamb dysentery
Type C	Lamb and sheep	Haemorrhagic enterotoxaemia and struck
Type D	Sheep, goat	Pulpy kidney disease
Type E	Lamb	Enterotoxaemia

Clinical signs: In per acute stage animal dies suddenly. Acute form is manifested by fever, reduced feed intake, abdominal pain, kicking at its belly, bleating, bloody diarrhoea, coma and death.

Diagnosis: Demonstration of bacteria by Gram's staining, isolation of bacteria and identification by biochemical tests. The organism produces stormy clot in litmus milk medium. Target haemolysis is a characteristic feature of *C. perfringens* in sheep blood agar. In addition, the animals can also be affected by other bacterial diseases like Listeriosis, Leptospirosis, Dermatophilosis or Foot rot, Mastitis, Colibacillosis, Campylobacteriosis, Caseous lymphadenitis, Chlamydiosis or Enzootic abortion of ewe, Contagious caprine pleuropneumonia, Paratuberculosis etc.

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VITAMIN AND MINERAL DISPARITIES IN POULTRY BIRDS

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Abstract

Nutrition in the poultry sector plays an important role in the economy of the farmers and the upliftment of their social and economic status. The vitamins play an important role in various physiological processes such as eggshell formation, muscle mass formation, immune system etc. The micro-minerals such as selenium and choline also play an important role in various physiological processes. The macro-minerals such as calcium and phosphorous plays an important role in the layer production along with skeletal system of the birds. So, the farm poultry manager or owner should have to provide the recommended dietary level of the various vitamins and minerals in the diet of the chicken to enhance the production.

Keywords: *Avian, deficiency diseases, nutrition, supplements*

Introduction

Nutrition plays a fundamental role in determining the health and performance of poultry and a correctly balanced diet is essential to avoid disease associated with a deficiency or toxicity of a particular nutrient. A fairly large number of different elements and compounds are required for the normal nutrition of poultry. If one or more of them are not present in the diet in adequate quantity, or if certain ones are present in an unsuitable ratio, there is a disturbance of nutrition, or of the functioning of the body, which may be referred to as a nutritional disease. Other nutritional diseases may result from harmful elements or compounds in the diet. Poultry require at least 36 dietary nutrients in appropriate concentrations for a balanced diet (Klasing, 2013). The poultry meat also called as white meat, is easily digestible, has higher nutritive value, higher protein, less fat, a good source of vitamin B complex, iron, and phosphorus. Like other food animals such as cattle, buffaloes, sheep, goat, pig, the poultry also require well-balanced diet to keep good health. The poor feeding is commonly observed in backyard poultry. This can result in deficiencies of numerous vitamins and minerals, which are essential for the maintenance of poultry health. Some of the most encountered nutritional disorders in the poultry are as follows:

1) Vitamin A deficiency: The vitamin A deficiency has been seen mainly at the younger phase (1-7 weeks age) of life among poultry causing skeletal as well as neurological disturbances in the young poultry birds. The most encountered form is "Roup" characterized by excessive ocular discharge causing sticking of the eyelids, nasal discharge, poor feathering, poor growth rate, etc. The necropsy findings will include inflamed and adhered eyelids, excessive urates in kidneys and ureters, and pustules in the mouth and pharynx.



Fig.1: Nutritional roup condition: Swelling around the eyes and excessive ocular discharge.

2) Vitamin E and selenium deficiency: The deficiency of vitamin E is associated with muscular as well as neurological disorders in the chicks (15-30 days of age). It is associated with softening of the grey matter of the brain commonly known as nutritional encephalomalacia causing neural signs. Vitamin E deficiency is associated with exudative diathesis leading to the edema of the thorax and abdomen. The vitamin E deficiency will lead to the wide linear areas of the muscle degeneration commonly called as nutritional muscular dystrophy (Kuttappan *et al.*, 2012).

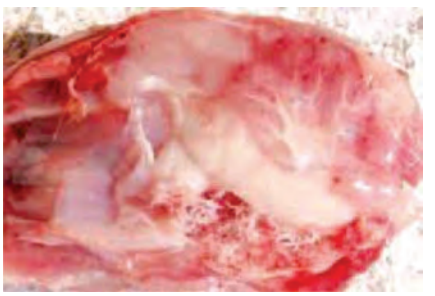


Fig.2: Exudative diathesis condition characterized by gelatinous oedema in the thoracic muscles due to exudation.

3) Vitamin D deficiency: The most common skeletal problems that are associated with Vitamin D deficiency in broilers are tibial dyschondroplasia, chronic painful lameness, chondrodystrophy or angular bone deformities, valgus-varus deformities, spondylolisthesis, rickets, femoral head necrosis, curled toes, and ruptured gastrocnemius tendon (Angel *et al.*, 2007).

4) Vitamin B1 deficiency: Mature chickens show signs 3 weeks after being fed a deficient diet. In young chicks, it can appear before 2 weeks of age. Onset is sudden in young chicks. There is anorexia and an unsteady gait. Later, there are locomotor signs, beginning with an apparent paralysis of the flexor of the toes. The characteristic position is called "star-gazing", meaning a chick "sitting on its hocks and the head in opisthotonos". Polyneuritis in birds



Fig. 3: Star-gazing posture characterized by the specific sitting posture of the chicken on the flexed hock joints and opisthotonos condition of the neck.

represents the later stages of a thiamine deficiency, probably caused by the buildup of the intermediates of carbohydrate metabolism. A marked decrease in appetite is seen in birds fed a thiamine-deficient diet. Poultry are also susceptible to neuromuscular problems, resulting in impaired digestion, general weakness, star-gazing, and frequent convulsions.

5) Vitamin B2 deficiency: Riboflavin deficiency caused a decrease in growth rate, lowers the hatchability of eggs, and egg production decreases (Rasikh, 2019). Many tissues may be affected by riboflavin deficiency, although the epithelium and the myelin sheaths of some of the main nerves are major targets. Changes in the sciatic nerves produce "curled-toe" paralysis in growing chickens (Patel, *et al.*, 2019). Deficient chicks are reluctant to move unless forced and then frequently walk on their hocks with the aid of their wings. The leg muscles are atrophied and flabby, and the skin dry and harsh. In advanced stages of deficiency, the chicks lie prostrate with their legs extended, sometimes in opposite directions. The characteristic sign of riboflavin deficiency is a marked enlargement of the sciatic and brachial nerve sheaths; sciatic nerves usually show the most pronounced effects.



Fig.4: Curled toe paralysis condition characterized by the inward bending of the toes.

6) Choline deficiency: It is usually seen in the growing phase of the chicken. In addition to poor growth, the classic sign of choline deficiency in chicks and poults is perosis. Perosis is first characterized by pinpoint haemorrhages and a slight puffiness about the hock joint, followed by an apparent flattening of the tibiometatarsal joint caused by a rotation of the metatarsus. The metatarsus continues to twist and may become bent or bowed so that it is out of alignment with the tibia (Pour *et al.*, 2017).



Fig. 5: Perosis condition: Enlargement of the hock, twisted metatarsi, and slipped tendons.

7) Manganese deficiency: The most dramatic classic effect of manganese deficiency syndrome is perosis, characterized by enlargement and malformation of the tibiometatarsal joint, twisting and bending of the distal end of the tibia and the proximal end of the tarsometatarsus, thickening and shortening of the leg bones, and slippage of the gastrocnemius tendon from its condyles. In laying hens, reduced egg production, markedly reduced hatchability, and eggshell thinning are often noted. A manganese-deficient breeder diet can result in chondrodystrophy in chick embryos. This condition is

characterized by shortened, thickened legs and shortened wings.

8) Zinc deficiency: In young chicks, signs of zinc deficiency include retarded growth, shortening and thickening of leg bones and enlargement of the hock joint, scaling of the skin (especially on the feet), very poor feathering, loss of appetite, and in severe cases, mortality. Although zinc deficiency can reduce egg production in aging hens, the most striking effects are seen in developing embryos. Chicks hatched from zinc-deficient hens are weak and cannot stand, eat, or drink. They have accelerated respiratory rates and labored breathing (Sahraei, *et al.*, 2012).

9) Iron and copper deficiency: Deficiencies of both iron and copper can lead to anemia. Iron deficiency causes a severe anaemia with a reduction in PCV. In color-feathered strains, there is also loss of pigmentation in the feathers (Ala Al Deen *et al.*, 2007). Young chicks become lame within 2–4 weeks when fed a copper-deficient diet. Bones are fragile and easily broken, the epiphyseal cartilage becomes thickened, and vascular penetration of the thickened cartilage is markedly reduced. Copper is required for cartilage formation. Copper deficiency in birds, and especially in turkeys, can lead to rupture of the aorta (Dibner *et al.*, 2007).

10) Calcium and phosphorus: A deficiency of either calcium or phosphorus in the diet of young growing birds results in abnormal bone development, even when the diet contains adequate vitamin D_3. A deficiency of either calcium or phosphorus results in a lack of normal skeletal calcification. Rickets is seen mainly in growing birds, whereas calcium deficiency in laying hens results in reduced shell quality and subsequently osteoporosis. This depletion of bone structure causes a disorder commonly referred to as "cage layer fatigue" (Liu *et al.*, 2013).



Fig.6: Cage layer fatigue condition, characterized by resting of the layer on the ventral abdomen, fatigue, dull and depressed.

11) Iodine deficiency: Lack of thyroid activity or inhibition of the thyroid by administration of thiouracil or thiourea causes hens to cease laying and become obese. It also results in the growth of abnormally long, lacy feathers. Administration of thyroxine or iodinated casein reverses the effects on egg production, with eggshell quality returning to normal (Hassaan *et al.*, 2015).

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ROLE OF NEFA AND BHBA IN PREDICTING METABOLIC DISEASES DURING TRANSITION PERIOD

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Abstract

Transition period is the most vulnerable period that is associated with peak incidences of metabolic diseases like ketosis, milk fever, fatty liver and results in detrimental effect on cow's performance. By evaluating the serum levels of non-esterified fatty acids (NEFA) and β-hydroxybutyrate (BHBA) in late prepartum and early postpartum period, incidences or occurrence of metabolic diseases can be predicted as well as prevented by timely nutritional intervention which leads to better health and performance of the cow.

Key words: *β-hydroxybutyrate, Metabolic diseases, non-esterified fatty acids.*

Introduction

Advances in genetic techniques has resulted in improvement in dairy cows markedly increasing milk yield over the last three decades. But this increased milk production is often associated with poor reproductive performance and other metabolic disorders. Transition period as described by Grummer, (1995) is the most vulnerable and critical period experienced by cow that extends from three weeks prepartum to three weeks postpartum. During this period cow undergoes major physiological, nutritional, immunological, endocrine and metabolic changes that are necessary for the cow to shift from gestational non-lactating stage to parturition and lactogenesis. Hayirli *et al.* (2002) reported one-third decrease in feed intake experienced by the cow during last three weeks prior to calving and significant reduction in final week before parturition. This is mainly due to less capacity for rumen to expand because of increased size of foetus and high concentrations on estrogens levels. After parturition there is increase in demand for nutrient and energy for lactation but the inability of the cow to cope with the increasing energy demands due to decreased DMI results in negative energy balance. To meet the energy requirements depletion of body fat stores occurs. These processes are accompanied by elevated blood concentrations of non-esterified fatty acids (NEFA) and β-hydroxybutyrate (BHBA) and decreased levels of calcium and phosphorous leading to metabolic disorders.

Production diseases are the result of cow's inability to cope with the high metabolic demands of lactation. The term 'production disease' encompassed metabolic disorders like ketosis, hypocalcaemia (milk fever) and hypomagnesaemia (grass tetany)

but the term has been broadened to include associated clinical diseases like retained placenta, metritis, endometritis, and displaced abomasum (Mulligan and Doherty, 2008). These production diseases are associated with severe economic losses in terms of heavy reduction in milk yield and impaired reproductive performance.

Milk fever (hypocalcaemia)

Incidence rates of clinical hypocalcaemia vary between 3.5 and 7% (De Garis and Lean, 2008). After parturition, there is increased calcium demand for lactation and colostrum production and to meet this demand calcium mobilisation from bones occurs. Milk fever occurs if the calcium homeostasis mechanism which normally maintain blood calcium levels between 9 and 10 mg/dl fails to keep up with the lactational demands resulting in blood calcium levels to fall below 5mg/dl (NRC, 2001). This hypocalcaemia impairs muscle and nerve function to such a degree that the animal is unable to rise. Hypocalcaemia is associated with many other disorders like dystocia, retained placenta, endometritis, infertility, uterine prolapse, mastitis, displaced abomasum, and ketosis (Houe *et al.* 2001). Intravenous calcium treatments (calcium borogluconate) are used to keep the cow with milk fever alive long enough for calcium homeostatic mechanisms to adapt.

Grass tetany (hypomagnesaemia)

Grass tetany occurs due to low level of magnesium in the blood and is often associated with early lactation as 0.15g magnesium is removed from the blood per litre of milk production. Animals grazing on lush green pastures or potassium fertilized pastures are more prone to grass tetany as potassium interfere with the absorption of magnesium thus leading to deficient levels of magnesium in blood.

Fatty liver and ketosis

During early lactation dietary nutrient supply is unable to meet the nutrient and energy demand of lactation and decreased DMI associated with high estrogen levels and less space in rumen to expand due to foetus leads to negative energy balance (NEB). To cope with the negative energy balance mobilisation of non-esterified fatty acids (NEFA) from adipose tissue occurs. The extent of lipid mobilisation depends on the period of negative energy balance experienced by the animal. Uptake of NEFA by liver is proportional to NEFA concentrations in blood (Bell, 1979). Higher rates of lipid mobilisation led to higher intake of NEFA by liver. Non-esterified fatty acids (NEFA) taken up by liver can either be esterified or oxidised. Esterification of NEFA results in the formation of triglycerides which are either exported as very low-density lipoprotein (VLDL) or stored in liver. The rate at which the export of triglyceride occurs is very slow as compared to other species (Kleppe *et al.,* 1988; Pullen *et al.,* 1990). Thus, leading to accumulation of triglycerides (TG) in liver resulting in fatty liver.

Oxidation of Non-esterified fatty acids (NEFA) lead to either formation of carbon dioxide and ATP or ketone bodies like β-hydroxybutyrate (BHBA). Complete oxidation of NEFA in liver leads to production of energy and carbon dioxide where as incomplete oxidation leads to formation of ketone bodies leading to ketosis. Low levels of insulin enhance fatty acid oxidation by decreasing hepatocyte malonyl CoA which in an inhibitor of Carnitine plamitoyltranferase-1 which is responsible for translocation of fatty acid from cytoplasm to mitochondria for oxidation.

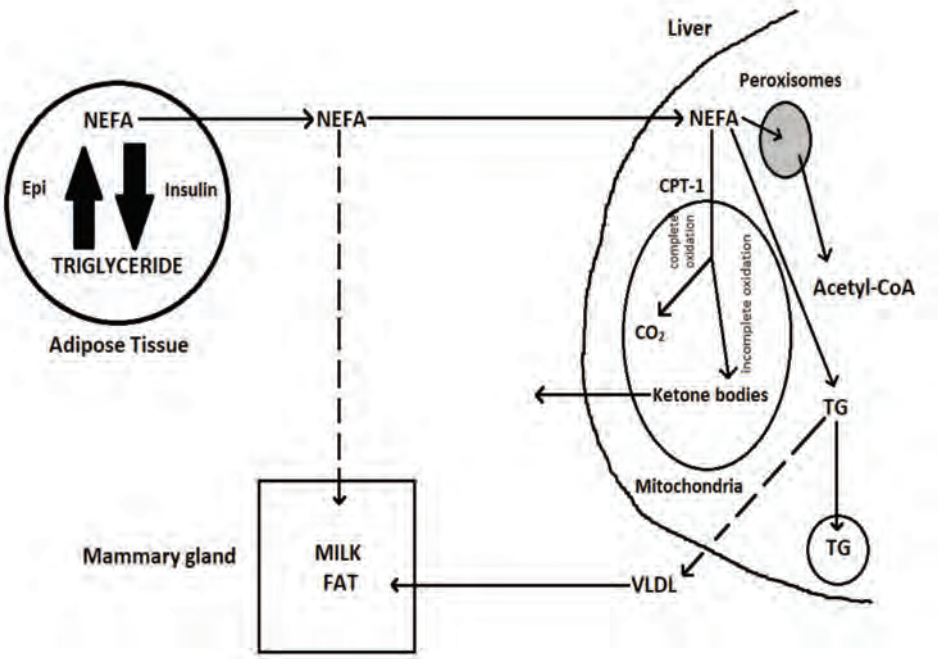


Figure 1: Schematic representation of lipid metabolism in adipose tissue, liver, and mammary gland. Dashed lines indicate processes that occur at low rates or only during certain physiological states. Abbreviations: epi = epinephrine, TG = triglyceride, VLDL = very-low-density lipoproteins, CPT-1 = carnitine palmitoyl transferase-1.

Prediction of clinical diseases through NEFA and BHBA levels

Ospina *et al.* (2010) reported that serum levels of NEFA and BHBA during transition period can be used to predict the occurrence of periparturient diseases like displaced abomasum, ketosis, metritis and retained placenta within 30 days in milk. Ospina *et al.* (2010) calculated threshold values for both NEFA and BHBA and values higher than the threshold values were associated with the incidence of periparturient diseases or production diseases. Ospina *et al.* (2010) reported that NEFA concentrations of ≥ 0.29 mEq/L prepartum and ≥ 0.57 mEq/L postpartum were associated with the risk of developing displaced abomasum, metritis, or retained placenta during the first 30 d

in milk and BHBA concentrations of ≥1.0 mmol/L from day 3 to 14 postpartum were associated with increased risk of clinical ketosis as well as metritis.

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BHB METER AS DIAGNOSTIC KIT FOR KETOSIS IN FIELD

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Abstract

Ketosis is an underdiagnosed metabolic problem dairy animals secondarily associated with many disease conditions. Non-specific clinical signs, inadequate laboratory facilities and unawareness among veterinary practitioners are major hindrance for its diagnosis. Beta Hydroxy Butyrate (BHB) meter is a low cost, small hand held and portable machine that may be used to diagnose ketosis under field conditions and early treatment may be initiated to restore animal health and production.

Keywords*: dairy animals, ketosis, milk production*

Ketosis is a metabolic problem of dairy animals which often goes undiagnosed. The problem arises due to inability of cattle to fulfil energy demands of production. So animal starts using adipose tissue especially from dorsum of body. This further leads to production of NEFA (Non esterified fatty acid) and ketone bodies (acetone, acetoacetic acid and beta hydroxy butyric acid) in liver (Duffield, 2022). This is compensatory mechanism of body of animal to provide energy. Some of these ketone bodies are used to provide energy but after sometime bad effects start to appear. Ketosis is thought to be more common in high yielding animals between 4 to 6 weeks postpartum. Ketosis is a gateway for LDA, ROP, Mastitis and Metritis.

Symptoms

1. Concentrate Refusal
2. Gradual emaciation
3. Decreased Milk Yield
4. Abnormal Licking movements
5. Inappetence



Treatment includes

1. Glycerine
2. 50% Dextrose IV
3. Steroids

Diagnosis is very important because sometimes the disease may go unnoticed. So, it is very important to have accurate diagnostic test for ketosis to restore the health and production of animal. Acetone goes off with expiated air so it can't be measured. Acetoacetic acid can be qualitatively measured in urine and milk. But estimation of BHB (Beta Hydroxy Butyrate) is more accurate method for ketosis diagnosis. So, I used BHB Meter to diagnose ketosis at field level from direct blood. BHB value of more than 1mmol/L is diagnostic for ketosis. I will discuss few cases:

1. Buffalo not taking feed in the evening, decreased milk yield, parturiated 3 months back. BHB 1.8mmol/L, treatment done with dextrose, dexamethasone and glucose precursor. Animal showed recovery after 2 days of treatment.
2. Cow showing gradual emaciation, parturiated 22 days back, decreased feed intake and milk yield (1kg/day). There was history of pneumovagina. BHB 1.6mmol/L. After treatment appetite was restored and milk yield increased to 7kg/day.
3. Cow parturiated 21 days back, showed PPM now showing gradual emaciation and hard faecal balls. BHB 3.4 mmol/L treated with glycerin, steroids and dexamethasone. Animal recovered after 2nd day.
4. Cow calved 1 month back now showing gradual emaciation and decreased milk yield. BHB 1.2mmol/L. The cow showed recovery after 2 days.
5. The cow was showing shivering and abnormal lickings, BHB 2.8mmol/L. Recovery after 3rd day.

These cases showed

1. BHB Meter is quite accurate way to diagnose ketosis.
2. Not only high yielders, but even moderate yielders also suffered from ketosis.
3. Ketosis may occur as early as three weeks postpartum.

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PREGNANCY DIAGNOSIS IS A KEY FOR THE BETTER MANAGEMENT OF DAIRY FARMS

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Abstract

Late Pregnancy Diagnosis is a hallmark of unending losses to dairy farm owners. Scientist explains why it's necessary for every farm owner to get their animals diagnosed for pregnancy after every mating and AI. Maximum output from an animal during its life time by reducing calving interval can only be achieved by early pregnancy diagnosis. Early pregnancy diagnosis can help in removing the major chances of loss due to anestrus or infertility. Thus helping farmers to harvest more monetary gains from their livestocks. Currently per rectal palpations, ultrasonography are the most common methods utilised to determine pregnancy. But still these methods too have limitations as pregenancy determination requires long time interval, which further results in skipping of one estrus. Hence increasing the postpartum barren gap. Other protein and marker based methods are still under exploration and progression in this area is further the necessity to improve the scenario of farms.

Keywords: *Diagnosis, loss, pregnancy and profitable.*

Profitability in the business is the priority to every dairy farm owner. But what can be a prominent reason of keeping farms in profit, how to ensure it and in what ways a farm owner can expect a logarithmic increase in profit, are some of the key questions a farm owner needs to consider. Overall for every businesses, monetary benefit is the key goal for its smooth survival and progress towards continuing a business. Reproductive efficient animals are the prime reason for a good farm. But their reproductive efficiency can get hampered, which results in losses due to extension of the non-productive and dry period, feeding of non-productive animals and associated veterinary costs in the treatment of these animals. Since feeding non-reproductive animal till next season of calving and getting nothing in return, definitely add extra costs to the farm owner.

Generally, absence of estruses after mating or artificial insemination (AI) is considered as an indicator of pregnancy. However, absence of heat signs in animal does not guarantee confirmation of pregnancy. Buffaloes, are silent heat animal, they don't express prominent signs of estrus. Such situations lead to long calving interval, which leads to more investment on livestock than output and ultimately a big loss. In order to have high calving from dairy animals and to enjoy more benefits, pregnancy diagnosis at early stage will act as boon for dairy farm owners.

What is pregnancy diagnosis (PD)? The state of carrying a developing embryo or fetus within the female body is termed as pregnancy. The method of detecting such condition (Pregnancy) by utilizing various test such as urine, blood, ultrasound or detecting first heart beat is pregnancy diagnosis.

There are various methods of pregnancy diagnosis in cattle (Figure 1). Some are well established and common methods of diagnosis, suffers with limitations (Balhara *et al.*, 2013). The oldest and most practiced method of pregnancy diagnosis is the transrectal palpitations of uterus, which is usually performed at the interval of 3o days post insemination. Veterinarians generally rely on palpitations of amniotic vesicle. In buffaloes this is successfully done post 45 days insemination and considered as cheapest method.

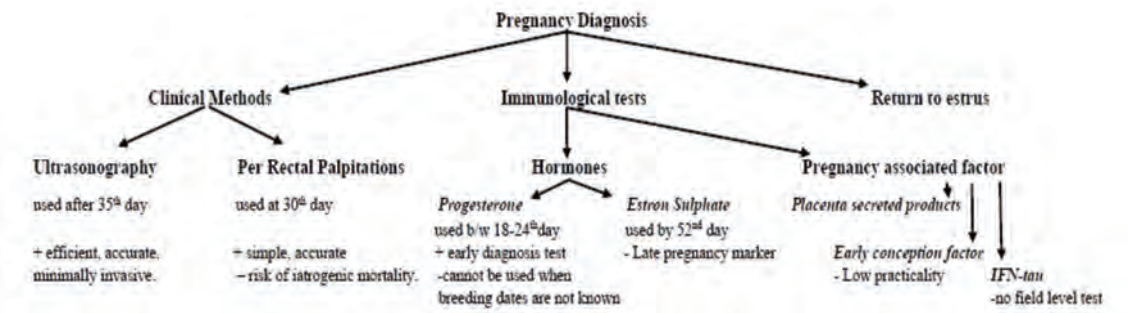


Figure 1: Flow diagram showing different methods of pregnancy diagnosis + ; advantages, - : Disadvantages

By per rectal palpitation an expert can determine whether an animal is pregnant or not accurately only after 45 days (Romano *et al.*, 2007). But with ultrasonography it can be predicted as early as 28 days. By 21st day visible signs of developing foetus can be determined by visualising fetal heartbeat. It is performed transrectally and confirms the pregnancy. It adds on various other advantages such as twins can be identified, fetal viability, age and, sex can be determined. Due to intensive and selective breeding of animals, pregnancy diagnosis also requires interventions. Scientific interventions on successful pregnancy diagnosis relies on early diagnosis as soon as implantation of fetus occurs and reliability of the tests. Recently scientists at NDRI, Karnal developed an early pregnancy diagnosis kit which can confirm the pregnancy by 35th day of gestation within 5 minutes with just 4-5 drops of blood and can be used by farmers on their own. After validation of kit in laboratory, the scientists found 85% accuracy of that kit and now working to increase its accuracy up to 95%. Knowing whether an animal is pregnant or not is of considerable interests since it is associated to monetary value and an important key in dairy farm management. Hence, an early diagnosis of pregnancy is required to identify nonpregnant animals soon after mating or insemination so that production time

lost from infertility may be reduced by appropriate treatment or culling, to reduce the wastage of expensive hormones during breeding, to select animals for sale and insurance.

Veterinarians across the globe are helping farmers by diagnosing pregnancy of their livestock by utilising various methods such as per rectal palpitations, ultrasound and different biochemical methods. In a study of 240 buffalo owners, approximately 71.3% of respondents take their animal for PD at 90 days, 15.4% after 90 days and rest do not take their animal for any PD and it was observed that economic losses was assessed only for respondents who took their animal for PD after 90 days. Average loss due to extra feed was assessed to be Rs 5918. Average quantity of milk loss was assessed to 308 litres per buffalo and average economic loss was assessed to be Rs 23925 per buffalo (Yadav *et al.*, 2019). The consequences of dry period in animals due to late pregnancy diagnosis will not only lead to monetary loss, but also effect the animal efficiency in term of milk production. So, diagnosing animals for pregnancy is a strict need in dairy industry. But current diagnostic approaches do have some limitations such as long-time interval for detection, costly equipment's (ultrasound) and, well established labs when detecting proteins or some pregnancy associated markers (Ferraz *et al.*, 2021, Jain *et al.*, 2012) which act as a major obstacle in well prosperity of dairy industry. Hence all currently available approach can't be called as a best approach. But probably successful work in this area can change the whole scenario of livestock farming.

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OCULAR AFFECTIONS IN BOVINE

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Abstract

The eye is an important special sense organ in vertebrate animals, which depend on it to survive and interact with the environment. Veterinary ocular study is still in infancy in India. It is seen that eye disease(s) remains one of the most important incremental problem(s) which reduce the livestock productivity. This article explores the different etiological factors responsible for ocular lesion(s) in bovine i.e. bacterial, viral, fungal, and parasitic or neoplasm(s). Morexella spp., gram negative organisms are the major contributor to cause ocular lesions in bovine. Among the tumors, squamous cell carcinoma is more common than malignant melanoma, adenocarcinoma and lymphoma.

Keywords: *Bovine, inflammatory, non-inflammatory, ocular affection.*

Introduction

Eye diseases in animals are usually associated with unscientific managemental practices, viz. particularly poor health management and also unhygienic conditions prevailing in the animal houses or due to environmental and the nutritional factors. If any one or both eyes develop impaired vision or get infected, it leads to great economic loss to the farmer(s) and reduced productive economic life of the affected animal. Among the various eye diseases, several congenital and acquired ocular affections can be seen in bovine (Squnders and Rubin 1975). There has been plethora of etiological factors incriminated in causation of ocular lesion(s) in veterinary patients (Mahamune, 2005).

I. Inflammatory affections

Bacterial affections: Amongst, the inflammatory affection(s) ocular bacterial infection(s) occur overwhelmingly due to both Gram- positive and Gram-negative. Gram- positive bacteria includes *Staphylococcus spp., Streptococcus spp., Corynebacterium spp*. and *Listeria spp.* Gram-negative: *Morexella spp., Escherichia coli, Pseudomonas spp., Niesseria spp., Actinobacillus spp., Klebseilla spp.*, and *Proteus spp*.

Listeriosis: Infectious disease caused by Gram- positive asporogenic bacterium, *Listeria monocytogenes,* transmitted by contaminated silage or by organic refuse (poultry litter). Ocular signs characterized by ptosis, medial strabismus, nystagmus, lacrimation, cloudy cornea, keratoconjunctivitis and uveitis.

Moraxella infection: Infectious bovine keratoconjunctivitis or pink eye, an acute contagious disease caused by Gram- negative bacterium *Moraxella bovis* and *Moraxella bovoculi,* characterized by lacrimation, hyperemia of conjunctival vessels, photophobia, serous-purulent discharge, opacity and ulceration of a cornea and partial or total loss of vision in an affected animal's eyes (Sharma *et al.*, 2018).

Viral affections: Ocular lesion as a result of viral etiology can be seen in case of several systemic diseases like malignant catarrhal fever and Bovine herpes-virus.

Malignant catarrhal fever: Fatal systemic infectious disease characterized by ocular symptoms of exophthalmos, blindness, nystagmus, photophobia, lacrimation, muco-purulent discharge, eyelid edema, conjunctivitis, keratitits and anterior uveitis (Whiteley *et al.*, 1985).

Bovine herpes-virus: Bovine herpes-virus type- 1, an infectious agent is associated with infections of ocular, genital, respiratory, enteric and nervous system. Ocular symptoms are characterized by conjunctival edema and corneal vascularization. While, in calves less than 6 months with encephalitic form blindness and convulsions can be seen.

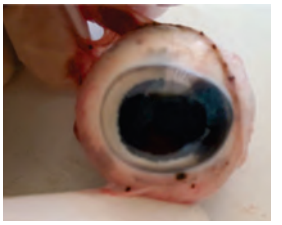


Fig. 1. Corneal pigmentation

Parasitic infection

***Thelazia species*:** Eye worm seen in the conjunctival sac (eyelid) of the eye associated with the warm season activities of the face flies (*Musca spp*.)

***Setaria digitata*:** Thread like filarid nematode commonly found in the peritoneal cavity of cattle, sometimes may also invade into eye. The erratic movement of the worm within the eye cause severe irritation to the cornea leading to corneal opacity and blindness in the affected animal (Mohan et al 2009).

***Toxoplasma gondii*:** Oocysts and cysts of *Toxoplasma gondii* can be seen in retina of young calves affected by oral route with clinical signs of fever, respiratory distress, nasal discharge, and hyperemia of the conjunctivas.

Theleria annulata: there are report of acute bilateral proptosis in a cross bred calf naturally infected with *Theileria annulata* (Sudan *et al.*, 2012).

Fungal infection: *Aspergillus spp.*, *Fusarium spp.,* and *rhizopus spp. in* hyphae and pseudo-hyphae state can be seen in the ocular tissue of bovine.

II Congenital

Congenital anomalies like nuclear cataracts (Fig 3) and posterior lenticonus; iris to lens persistent pupillary membranes and lenticular colobomata have been observed. Furthermore, congenital ocular abnormalities in the production animals, Anopthalmos



Fig. 2: Corneal opacity in eye

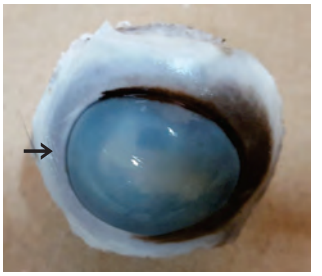


Fig. 3: Corneal opacity with cataract

and micropthalmos, cyclopia, cataract, optic nerve colobomas and several genetic and infectious disease(s) can be seen (Radostitis *et al.*, 2000).

III Vitamin deficiency

Vitamin A deficiency: Bilateral papilledema, diarrhea, blindness and optic pathway degeneration can be seen in calves with hypo-vitaminosis A (Radostitis *et al.*, 2000).

Vitamin B 1 (thiamine) deficiency: B1/thiamine deficiency associated blindness and lack of coordination as a result of high sulfur can be seen in bovine calves.

IV Non-inflammatory affection

Neoplasm: Of the various non-inflammatory affections, tumors outweigh other affections. Ocular growth interferes with vision and the cosmetic look of animal (Shruthi *et al.*, 2018). If these growths are malignant ocular neoplasm, they may cause a threat to life span of animal and also condemnation of the carcasses.

Ocular squamous cell carcinoma: The most common and important reported neoplasm inflicting the bovine eye, characterized by ulcerated, proliferating cauliflower like growth involving the orbital margin and the whole of the globe in some cases. It is usually caused as a result of exposure to de-pigmented skin to UV rays from the sun. Histo-pathologically, keratin pearls, hyperchromatic nuclei, numerous mitotic figures and pleomorphism can be seen (Yavuz and Yumusak, 2017).

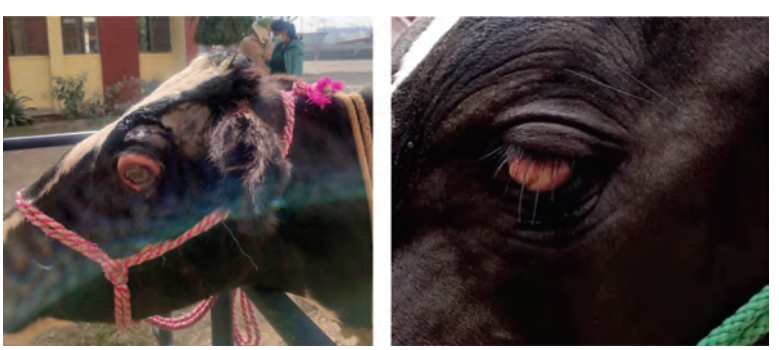


Fig. 4: Tumor of the eyes in cattle

Malignant melanoma: Tumor involving the orbital margin commonly, histopathologically characterized by typical nesting of atypical melanocytes can be seen.

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SURGICAL MANAGEMENT OF CARPAL HYGROMA IN BOVINES

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Abstract

Carpal hygroma is the accumulation of fluid/pus/fibrin in the subcutaneous space in bovines. The present report describes the surgical management of carpal hygroma in bovines.

Key words: *bovine, brucellosis, buffalo, carpal, cow, hygroma, joint, swelling,*

Introduction

Carpal hygroma is the accumulation of inflammatory fluid in the subcutaneous space above carpal joint in bovines. It is a common condition in bovines as they sit or stand by putting weight on the carpal first. Since, most of the bovines are kept on hard flooring (Tyagi & Singh, 2006), it leads to constant irritation to the bursa of the joint. The accumulated fluid may become infected and form pus otherwise with time it become hard/fibrosed with reduced water content (Kumar *et al.*, 2020). Brucellosis in cows is another etiology reported for carpal hygroma (Megid *et al.*, 2010). The present study describes the method of surgical management of carpal hygroma in bovine.

Case history and Clinical examination

The bovine is usually presented with the history of swelling in the carpal joint region, varying in size as per the chronicity of the condition (Fig. 1). It may be unilateral or bilateral. Medicinal treatment with needle drainage, anti-inflammatory, crepe bandage and change to soft/kachha floor may help in early stages (Tyagi & Singh, 2006). However, surgical drainage is required in large and chronic hygromas (Shukla *et al.*, 2020). Proper history of any abortion or placenta retention should be collected from owner, if suspected for brucellosis. A serum sample may be collected for Brucella testing. Cows have more incidence of Brucella induced hygroma than buffaloes.

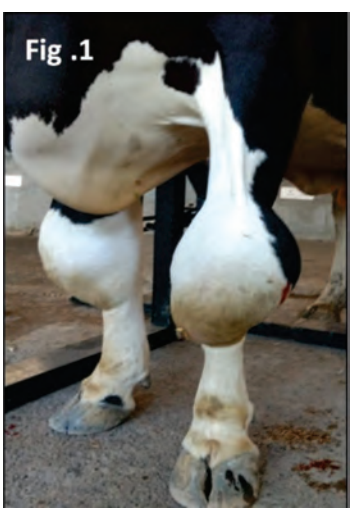


Fig. 1. Photograph showing bilateral hygroma in a cow

The clinical examination of the swelling should include the physical consistency to rule out that the hygroma is not completely fibrosed. A needle aspirate with a 16 gauze

hypodermic needle and a 20ml syringe may be carried out at the dependent portion in standing position after proper restraint. If fluid/pus comes on aspirate it is fit for surgical drainage. If needle aspirate do not reveal fluid/pus and the hygroma appears very hard and extensive with needle not going freely, such case should not be dealt in field with improper facilities. Resection of carpal hygroma should not be done enthusiastically, as there might be lot of bleeding and the stitches breaks off as soon as the animal bent his carpal for sitting.

Surgical management: The surgical drainage of hygroma is recommended in lateral recumbency with the affected limb upwards, as otherwise in standing position; the animal moves the limb a lot, secondly there might be bleeding during drainage and may need application of few sutures. Sedation may be required in few bovines (Inj. Xylazine @0.1mg/kg, Intramuscular) that do not allowing casting, but avoid it in pregnant bovines. The affected limb should be tied separately with ropes.

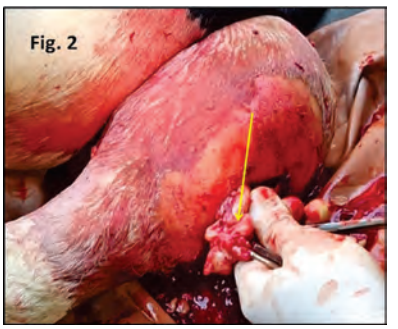


Fig. 2. Photograph showing the drainage of hygroma in lateral recumbency with fibrin being removed (Yellow arrow)

A bold stab incision is made with a 20-24 size new BP blade, in line to the limb axis at the most dependent and soft part of the hygroma. When the fluid starts coming, the incision can be extended dorsally or/and ventrally as per the cavity size. A gloved finger may be put inside the cavity, to assess the contents and remove all the fibrin inside (Fig. 2). There may be some calcified parts in the cavity in chronic hygroma. The cavity when empty is flushed with normal saline solution. At this stage, if there is bleeding from the skin incision site, ligatures may be applied with silk thread and cutting needle. If, the bleeding is more and is not controlled with ligatures, both sides of full thickness skin incision can be separately stitched in continuous manner with silk thread. This will stop bleeding and still there will be space to clean the cavity daily for dressing. Pulv magnesium sulfate is placed inside the cavity and a seton/ cotton bandage is placed to fill the cavity. If more than one bandage is required, it should always be tied with the previous bandage. This tying should be followed later also. As it sometimes happens that while cleaning, one bandage is left inside. Pressure bandaging with cotton,

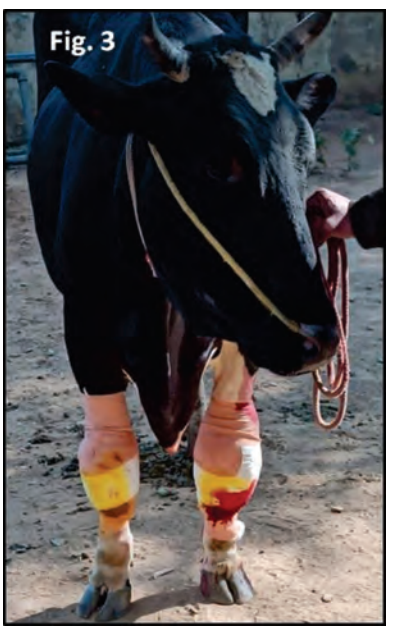


Fig. 3. Photograph of a cow with drained bilateral hygroma after pressure bandaging

cotton bandage and crepe bandage is to be done above it (Fig. 3). This bandaging after flushing with fresh tap water or NSS is to be repeated daily till the wound heals from inside. Postoperative care includes antibiotics and painkillers for 5 and 3 days respectively but the bandaging may take longer depending on the size on the hygroma. The swelling will reduce with time (Fig. 4).

The study reports that carpal hygroma in bovines can be managed successfully using needle aspirate or incisional drainage in early stages in field conditions. The bovine should be kept for at least 20 hrs on kachha/soft floor to avoid development of such conditions.



Fig.4. Photograph of the cow with bilateral hygroma at 75 days

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ABDOMINAL EMERGENCIES IN CATTLE

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Abstract

The article presents the clinical approach and treatment strategies used for cases of abdominal emergencies in cattle. Many of these emergencies can quickly deteriorate into an irreversible state and thus possess a potent risk to the life of the cattle. In such acute conditions, the clinician has to consider the appropriate diagnostic as well as a therapeutic approach in a timely and orderly manner. The decision whether to choose medicinal treatment or go for surgical management depends upon physical examination findings, per rectal findings, and the results of ancillary diagnostic tests. Additional considerations for this decision include the economic and sentimental worth, physical status, and condition of the cow as well as the accessible medicinal or surgical facilities. A definite diagnosis or strong suspicion of some gastrointestinal disorders such as right abomasal displacement and abomasal volvulus immediately necessitates laparotomy and surgical correction.

Keywords*: Abdominal Emergencies; Acute Abdomen; Gastrointestinal Visceral Disease***;** *Cattle*

Introduction

An abdominal emergency is a pathological process in the abdomen that manifests as life-threatening or acute clinical signs (Fecteau *et al.*, 2018). The acute abdomen should be evaluated as soon as possible to take the appropriate action (medical or surgical). Abdominal emergencies encompass a wide range of disorders (Table 1) that cause abdominal pain and distension. Parietal pain (parietal peritoneum, abdominal muscles, rib cage) is a localized pain that shows up in cattle as movement reluctance and reflex tonic contractions of the abdominal muscles. Visceral pain (hollow and solid organs) is a diffuse pain that manifests clinically as severe colic: the animal kicks its abdomen, stretches its body while lying down or standing, and steps on its hind legs (Muiño et al, 2021).

Predisposing Factors to the Development of Acute Abdominal Syndrome

- Age
- Lactation stage
- Season
- Nutritional status

Jejunal hemorrhage syndrome or hemorrhagic jejunitis, an acute enterotoxemic

disorder, occurs most commonly in older dairy cows, early in the second lactation. Torsions of the uterus are prevalent during parturition or the dry period. Abdominal volvulus is more common in dairy cows than in those destined for meat production. The most typical times for caecal dilatation are during the winter months and the periparturient period. During this time, the animal consumes more concentrates, leading to an increase in volatile fatty acid production and subsequent gas accumulation, which predisposes the animal to cecal dilatation and subsequent dislocation. Recent estrus has been linked to hypocalcemia, which may result in paralytic ileus. Feeding of non-fermented energy-rich carbohydrates after the early lactation may lead to subacute ruminal acidosis (Muiño et al, 2021).

Diagnostic Approach to the Acute Abdomen

a) Health History and Physical Examination: To properly approach the situation, a thorough anamnesis is required which should cover the onset of disease along with its duration and progression of the problems, the recent occurrence of any similar signs in the patient's herd, nutrition, and management system, past therapies and feces output, consistency, and appearance. The emergence of similar, recent instances in the herd necessitates a thorough consideration of illnesses influenced by nutritional and infectious factors (Van Metre *et al.*, 2005). Feces presented with mucus, blood, dark colour, and fetid odour are all signs of gastrointestinal disease. Physical examination includes:

- Determination of physiological parameters- In acute pain, heart and respiratory rates show an increase, the pulse becomes feeble, and capillary refill time is prolonged (when hypovolemia is present).
- Rectal palpation- A rectal palpation is a fundamental tool for distinguishing GIT, reproductive, and renal diseases. Intussusception can be detected by palpating distended and firm bowel loops. Stool mixed with fresh blood may indicate hemorrhagic jejunitis or intussusception. Per rectal and vaginal examinations are used to detect uterine torsion.

Table 1: Causes of acute abdomen and common physical examination findings

Gastrointestinal Visceral Disease	**Common clinical signs**
Abomasal volvulus	Dehydration, feeble pulse, increased heart rate, troubled rapid breathing, rarely colic, absent or watery feces, abdominal distension (greater on the right side), and tympanic resonance (ping) extending from the eighth rib to the middle of the paralumbar fossa (PLF) on the right.

Abomasal displacement	Temperature, pulse, and respiratory rates are usually normal. Partial anorexia and reduced milk production are the earliest signs. Other signs include ketosis, reduced rumination, Changes in coat texture (rough), sunken abdomen in PLF, and a high-pitched musical sound (ping) generated by simultaneous percussion and auscultation.
Intestinal atresia (atresia jejuni and atresia coli)	Debilitation, no fecal discharge since birth, abdominal pain, Progressive bilateral distension of abdomen, engorged scleral vessels, cool extremities, dehydration and increased heart and respiration rate.
Intussusception	Dehydration, tachycardia, tachypnea, ±pale mucous membranes, cool extremities, ±severe colic, kicking at the abdomen, gradual distension of abdomen (initially in right side ventral abdomen and generalized in advanced cases), splashing sounds by gentle ballottment of upper right flank and abomasum, auscultation of multiple areas of tympanic resonance over right abdomen, bloody scanty feces per rectally.
Mesenteric torsion	Anorexia, hypothermia, elevated heart rate, severe colic, both-sided flank distension, multiple distended intestine loops per rectally and absence of faecal contents in the rectum. Exploratory laparotomy through the right paralumbar fossa can be used as a diagnostic as well as a means of cure in this condition (Karvountzis, 2016).
Paralytic ileus	Tachycardia, rapid breathing, variable abdominal pain (moderate to severe), moderate abdominal distension in the lower right abdomen, and fluid sounds on auscultation on the right side of the abdomen.
Cecal dilatation	Decreased milk production, lack of appetite, loose feces (whereas animals with cecal volvulus typically do not pass feces), right side abdominal distention, mild abdominal pain, tympanic resonance, and fluid sounds on simultaneous ballottement and auscultation of right paralumbar fossa. Rectal palpation: loose feces, dilated and rounded caecal apex protruding into the pelvic inlet (Fecteau *et al.*, 2018).

Traumatic-reticulitis/ reticulo-peritonitis	Reduced appetite, sudden drop in milk, fever, Tachycardia, Stiff gait, arched back, expiratory grunting or groaning, resistance in extending back with withers pinch, reduced motility or atony of rumen, regurgitation/vomiting (rare), muffled or splashing heart sounds and signs of congestive heart failure if pericardium is involved. Pain response on deep pressure (fist, knee, or metal bar) in the cranial abdomen, especially on the right side. Signs of vagal indigestion may develop if reticular perforation has occurred (Fecteau *et al.*, 2018).
Non-Gastrointestinal Disease	**Clinical signs**
Uroliths	Lethargy, reduced appetite, bruxism, straining to urine, mild colic, grit on preputial hairs, pulsing of urethra, hematuria, and it can progress to the urethra or bladder rupture.
Pyelonephritis	Anorexia, dehydration, colic, tail switching, polyuria, straining to defecate, dysuria, hematuria, ±stranguria, exudate adherent to vulvar hairs, and enlarged kidney per rectally.
Uterine torsion	Abdominal pain, Anorexia, Tachycardia, tachypnea, signs of impending parturition without advancement. vaginal examination: circumferential constriction of the vaginal wall, tensor spiral folds of vaginal mucosa near the cervix. Rectal examination: torsion of the uterine ligament is palpated.

b) Complementary examination methods: Complementary tests are useful as they offer clearer information about the patient's critical issues.

Different complementary examination methods include:

- Haemato-biochemical tests: CBC, L-lactate, chloride, potassium, acute phase proteins, etc. should be assessed in cases with acute abdomen.
- Abdominocentesis: Peritoneal Fluid (PF) should be assessed for turbidity, colour, odour, volume, and cytological examination. The PF is usually clear or light yellow, with low turbidity. However, an increase in turbidity indicates the presence of inflammatory cells, blood, and proteins as seen in strangulating

lesions. The dark or green colour of PF is suggestive of rapture or involvement of the GIT. Reddish-brown discoloration of PF indicates gut necrosis. The presence of a urine-like odour in PF may indicate a case of uroperitoneum (Zadnik, 2010). The cytological examination should be performed to rule out the origin of the condition (Table 3). Uroperitonem is diagnosed when the PF: Serum creatinine concentration is ≥ 1.5-2 (Muiño et al, 2021).

Table 3: Classification and origin of PF according to protein (g/dL) and cell concentration

Peritoneal Fluid	Protein (g/dl) and Nucleated Cell Concentration (cells/µL)	Origin
Transudate	2.5 g protein/dL & <5000 cells/ µL	Hypoproteinemia, renal failure, parasite infestation, chronic diarrhea
Modified transudate	2.5–3 g protein/dL & 5000–10,000 cells/ µL	Displacement of abomasum, pericarditis, neoplasm
Exudate	>3 g protein/dL & >10,000 cells/ µL	Infectious peritonitis

- Abdominal Ultrasonography- The ultrasound examination should always begin on the left side at the level of reticulum and progresses caudally with examination of spleen and rumen. The right side is next examined to visualize small and large intestines, right kidney, liver, omasum, and abomasum. Evaluation of all these viscera for its size, content, wall, and even its functionality is done thoroughly.

The various complementary tests advised in the acute abdomen are listed in table 2 (Muiño et al, 2021).

Table 2: Complementary tests for diagnosing acute abdominal syndrome along with the findings

Gastrointestinal (GIT) Disease	Complementary Diagnostic Methods
Abomasal volvulus	• Serum biochemistry: hypochloremia, metabolic alkalosis, and metabolic acidosis in due course of time • USG: abomasal distension and more cranial orientation of pylorus
Abomasal displacement	• Serum biochemistry: hypochloremia and alkalosis. • USG: pylorus is positioned medial to the right mammary vein or towards the ventral midline

Intussusception	• Biochemical tests: hypochloremia, hyponatremia and hypokalemia. • USG: dilated intestinal loops in right PLF and ventral abdomen, on cross-sectional view ''bulls-eye'' appearance of the lesion may be visible while in longitudinal view, the affected segment has a ''sandwich'' appearance (Fecteau *et al.*, 2018). Laparotomy is recommended unless signs of ruptured bowel are obtained.
Mesenteric torsion	• Serum biochemistry: hyperglycemia, hyponatremia, hypochloremia, hypokalemia and azotemia. • USG: distended bowel loops before the obstruction.
Cecal dilatation	• Serum biochemistry: hypochloremia, hypokalemia and metabolic alkalosis. • USG: thick, echogenic curved cecal wall at right paralumbar fossa. • Exploratory laparotomy
Traumatic-reticulitis/ reticulo-peritonitis	• Serum biochemistry: ±hypochloremia and alkalosis, increased globulin (chronic cases) • USG: reduced reticular motility, peri-reticular fluid of mixed echogenicity in case of concurrent peritonitis. • Reticular radiography: free fluid/gas pockets outside of the reticular margins, metallic foreign body may be visible. • Exploratory laparotomy and rumenotomy
Non-GIT Disease	
Uroliths	• Serum biochemistry: mild azotemia • Transrectal USG: to visualize the state of the bladder and the presence of hyperechoic uroliths.
Pyelonephritis	• Urinalysis: dipstick (hematuria and proteinuria) and sediment examination (bacteriuria and pyuria) • Haemato-biochemical findings: azotemia, anemia (Chronic case), and hypoalbuminemia. • USG: hyperechogenic areas in the calyx and renal sinus.
Uterine torsion	• Vaginal and rectal palpation confirms the diagnosis.

Treatment strategies

The most important criteria for deciding when to proceed with surgery are:

- The rapidity with which clinical signs progress,

- The severity of colic,
- The severity of abdominal distention and absence of fecal output, and
- The level of blood lactates, notably if blood lactate did not normalize despite medical therapy (Lausch *et al.,* 2019).

If surgery is the only possible treatment as in abomasal volvulus, there is no reason to delay the intervention. Unfortunately, unlike in cases of abomasal volvulus, the clinical diagnosis in most acute abdomens is less definite, and medical treatment may appear promising at first. In ruminants, exploratory laparotomy is a significant supplementary diagnostic procedure. Cattle are particularly receptive to exploratory surgery since it is performed standing and is linked with few problems when performed with care for asepsis. If emergency surgery is not required, there is still time to begin medical treatment to improve the patient's overall condition. The purpose of supportive therapy is to rectify hemodynamic and metabolic imbalances, reduce discomfort and prevent or treat the infection. For the treatment of the acute abdomen, fluid therapy, analgesics, anti-inflammatory medications, and antibacterial therapy should always be recommended. Abdominal problems are generally linked to either hypovolemic or septic shock. Tachycardia, pale mucous membranes, slow capillary refill time, and dehydration are all signs of hypovolemic shock. Septic shock is characterized by an increased heart rate and dehydration, as well as hyperemic or bluish mucous membranes and engorged and black scleral vessels. For both hypovolemic and septic shock, intensive fluid therapy is the preferred treatment. Administration of hypertonic saline at the rate of 4 to 5 mL/kg intravenously over 4 to 5 minutes provides a rapid resuscitation in dehydrated or endotoxemic animals but these solutions are generally not used routinely in acute abdomen unless the animal is showing signs of acute blood loss or the animal is recumbent. Initially, crystalloid solutions (0.9 percent sodium chloride, Ringer solution) are used to replace fluid loss and increase circulating blood volume. Because of the cessation of abomasal absorption during acute abdomen, the majority of patients develop metabolic alkalosis with hypochloremia and hypokalemia. In order to maintain electrolyte balance, Ringer's Lactate must be administered. Hypocalcemia is also common in dairy cows with gastrointestinal diseases. The most common IV fluid for acute abdomen is isotonic saline (20 L of 0.9%) in which calcium borogluconate 23% (500 mL) can be added. Because improvement with calcium-rich fluid provides insight into the likelihood of a surgical problem, the response to fluid therapy aids in the diagnosis. The medical strategy may not be sufficient if the patient continues to deteriorate while receiving rapid IV fluids. Repeated monitoring of rectal temperature, pulse or heart rate, respiratory rate, and a brief gastrointestinal motility exam (rumen contractions, gut sounds, ultrasonographic motility and faecal output) is frequently used to determine whether medicinal therapy is beneficial (Fecteau *et al.,* 2018).

To arrive at a diagnosis, the first step is to obtain a clinical history and a thorough physical examination. In order of importance, ultrasound, abdominal fluid analysis, and biochemical tests can aid in the confirmation, localization, and assessment of acute abdomen. Isotonic saline solution is the most commonly used IV fluid for abdominal emergencies.

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STANDARD OPERATING PROTOCOL FOR TUBE CYSTOSTOMY IN CALVES

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Abstract

Present communication delineates standard operating procedure of tube cystotomy technique for surgical treatment of obstructive urolithiasis in bovine calves; with an aim to guide Veterinarians of both institute and field level to learn and perform this simple, fast, and efficient salvation technique to prevent financial loss to farming community.

Keywords: *Bovine, surgery, uroinary bladder rupture, urolithiasis*

Introduction

Urolithiasis is the second highest reported case among ruminants followed by respiratory affections (Videla, & van Amstel, 2016). Urolithiasis is the formation of calculi being lodged anywhere in the urinary system (most frequently at sigmoid flexure) in case of bovine calves (Badamim *et al.*, 2016; Videla, & van Amstel, 2016). Both medicinal and surgical treatments are available for obstructive urolithiasis but surgical interventions are considered more successful. Among surgical interventions, tube cystostomy is the most successful method (Kushwaha *et al.*, 2009; Gugjoo *et al.*, 2013; Tamilmahan *et al.*, 2014). In this procedure, urine is diverted from the urinary bladder with the help of a Foley's catheter.

Indications

- Stranguria/ anuria
- Ruptured urinary bladder,
- urethral obstruction
- urethral rupture.

Prevalence: Male calves are more predisposed (Figure.1) to obstructive urolithiasis in comparison to female calves because of anatomical difference of external genitalia, where male urethra is longer and narrower (Gugjoo *et al.*, 2013; Badamim *et al.*, 2016).

History

- Decrease in urine outflow or urine dribbling (due to partial obstruction)
- Stranguria, anuria more than 24 hours
- Straining and Restlessness



Fig. 1: Showing male calf with clinical signs like abdominal distension and anuria.



Figure 2: Showing gazing towards flank and pear-shaped abdomen filled with urine.

- Futile attempt to void urine (Ruptured urinary bladder)
- Abdominal distension or water belly (Ruptured urinary bladder)
- Inappetence to anorexia, suspended rumination, and scanty feces

Clinical Signs

- To and fro movement of penis showing urge to urinate
- Dry muzzle, sunken & anemic eyes, rough coat
- Gazing towards flank
- Wide stance of hindlimb
- Pear shaped abdomen (Figure.2), if urinary bladder is ruptured.
- Swelling of urethral tract

Physical Examination: Fluid thrills on abdominal ballottement in case of ruptured bladder or distended urinary bladder, if intact, on palpation of caudal abdomen. Paracentesis of ventral abdomen can be done for confirmation

Diagnostic Imaging: Ultrasonography helps in confirming the status of the urinary bladder. If urinary bladder is ruptured, anehcogenic free fluid (urine) will be visible in the abdomen and urinary bladder will be collapsed or partially filled.

Surgical Procedure

- Tube cystostomy is treatment of choice in both intact or ruptured bladder associated with obstructive urolithiasis.
- Animal is positioned in right lateral recumbency (Figure.3) with the left hindlimb extended cranio-dorsally while right hindlimb tied caudally.



Figure 3: Animal positioned in right lateral recumbency

- Area near caudal rudimentary teats is prepared aseptically with local infiltration with 2% lignocaine (5ml to 10ml)
- Incision is made between the teats of left side and parallel to prepuce as caudal as possible, followed by separation of subcutis and muscles (Figure.3).
- Urinary bladder can be confirmed by aspiration of urine with sterilized syringe. Then urinary bladder can be exteriorized (Figure. 4).
- Subcutaneous tunnel is created and Foley's catheter (#FG 16 or, 18) is passed from outside the abdominal wall to inside with the help of artery forceps. Tip of catheter is stabbed directly into the bladder with the help of groove director. Balloon of Foley's catheter is inflated with normal saline solution so that catheter does not comes out of urinary bladder lumen.
- In case of urinary bladder rupture; the collapsed bladder is exteriorized, adhesions removed, necrosed bladder wall trimmed and debrided (Figure.5). Cystorhapphy is performed using absorbable suture materials like PDS, Catgut with inversion suture pattern (Cushing or, Lembert suture pattern) followed by Foley's catheter placement as described earlier. Irrigation with normal saline is important to remove concretions and calculi (Figure.6).

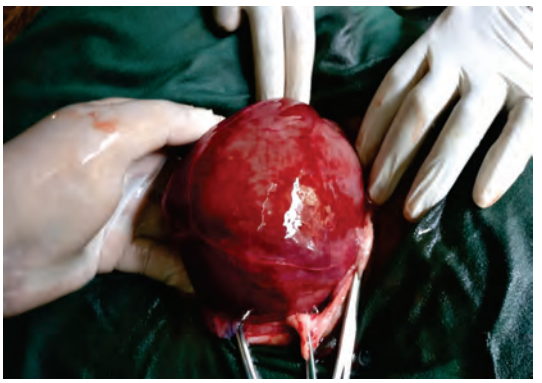


Figure 4: Exteriorization of urinary bladder with inflamed haemorrhagic serosal surface.

- Muscles are closed with absorbable suture materials like PDS, Vicryl or, Catgut (# 0 or, 2) in simple continuous fashion. Skin is closed with horizontal or, cross mattress pattern with non-absorbable suture materials like nylon or braided silk. Foley's catheter is to be fixed to the ventral abdominal wall at multiple sites (Figure.7). Care should be taken not to pierce the Foley's catheter.

Post operative care

Apart from broad spectrum antibiotics and analgesics, calves are prescribed Pulv Ammonium Chloride (@200mg/kg BW q24h) until removal of Foley's catheter. Sutures are removed after 12th post operative day. Antiseptic dressing with povidone iodine and fly repellent is prescribed. The catheter is allowed to drain freely for 4 days after which it is to be clamped on every alternate day to determine the urethral potency. Foley's catheter is removed when calf resumes normal urination through the urethra.

Obstructive urolithiasis prevents excretion of urine from the body leading to dumping of nitrogenous waste in the body. This leads to uremia/ azotemia and concurrent clinical signs in animal described previously. In calves, obstruction of urethra occurs due to sand like concretions made up of calcium apatite predisposed by fattening ration, mineral imbalance, avitaminosis A, concentrated urine and alkaline medium of urine. Tube cystotomy provides an alternative route for urine to excrete along with debris and concretions to prevent further exacerbation due to uremia/ azotemia. In the meantime, animal is provided with Ammonium chloride, which is a urinary acidifier. During post-operative clamping of Foley's catheter, acidified urine courtesy of Ammonium chloride diverted into blocked urethra. Under influence of acidic urine, sandy concretion of urethra dissolves gradually until complete patency of urethra is restored. Unlike other surgical techniques; tube cystotomy not only restores excretory function, but also preserve the reproductive function of male calves (Kushwaha *et al.,* 2009; Gugjoo *et al.,* 2013; Tamilmahan *et al.,* 2014; Badamim *et al.*, 2016).



Figure 5: Photograph showing ruptured and necrosed urinary bladder wall



Figure 6: Concretions removed during the procedure



Figure 7: Suturing of Foley's catheter to ventral abdominal wall.

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CONGENITAL AFFECTIONS IN BOVINE

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Abstract

Congenital affections are commonly encountered in calves. Few abnormities are major and lead to death of the calf soon after birth but many survive little longer and few even live a normal life. The article describes the common type of congenital affections presented in the calves of cow and buffaloes and there surgical correction, if any.

Keywords: *atresia anai, calf, cleft palate, congenital, hernia, cow*

Introduction

Congenital defects are the abnormalities, which are present at birth and can be structural, functional or both. Majority of the abnormalities are minor but certain may be related to more than one organ due to the same developmental germ layers (Camon *et al.*, 1990) and are life threatening. Genetics and the environment in the pre-natal stage may be responsible for these abnormalities. The incidence of congenital defects ranges from 2 to 3.5% of all births in calves, lambs and foals (Islam *et al.*, 2011). Congenital abnormalities occurs maximum in animals during 4–8 weeks of foetal life (Sharma *et al.*, 1986).

There can be many causes for the occurrence of congenital defects, a few are listed below:

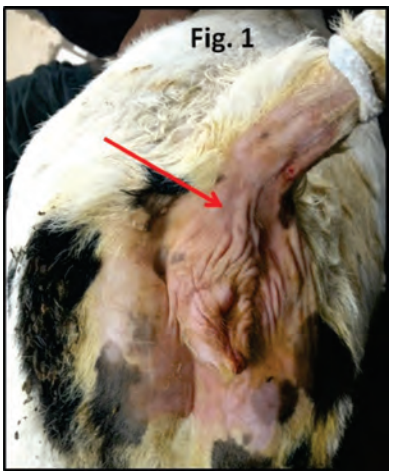
- Hereditary/genetics
- Ingestion of toxic plants by the bovine during gestation.
- Environmental factors like nutritional deficiencies, teratogenic drugs or chemical exposure, viral infections,
- X- rays and per rectal examination during the early stage of organogenesis. (Rafid, 2010)

The various types of common congenital defects are being described below:

1. Atresia Anai, et recti, et coli

Atresia anai alone or et recti or et coli are common congenital gastro-intestinal malformation in large and small ruminants. The condition may occur alone or with agenesis of other parts like vagino urethral agenesis hypospadias, cleft scrota**.**

Probable aetiology: Rectal palpation before 40 days of gestation (Durmus 2009).



Clinical signs: absence of anal opening (Fig. 1), suspended defecation. In atresia anai alone, a bulge is felt and seen at the site of anus when the calf strains to defecate or abdominal pressure is applied manually (Singh *et al.* 2020).

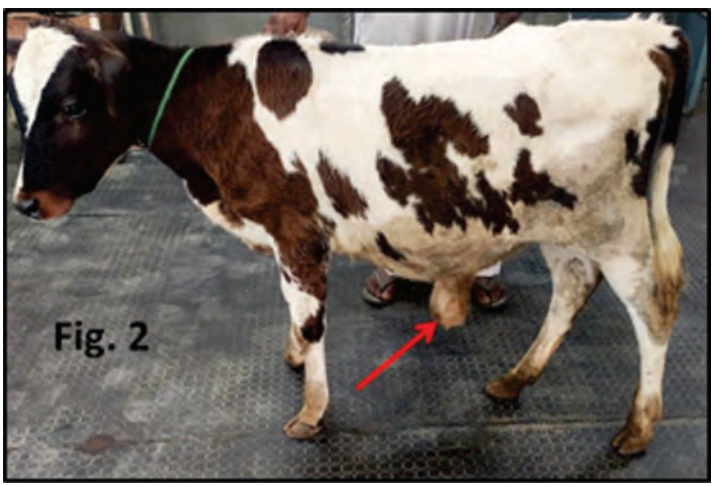
Surgical treatment: Surgical formation of anus can be done under local infiltration anaesthesia in 1-2 day old calf. A X or Round incision of 2-3cm (Singh *et al.*, 2020) is made below the base of tail where bulging is most prominent. The blind end of rectum is identified and opened and confirmed with meconium coming from it. The rectal wall is sutured with the skin at four cardinal sites and if required diagonal also using nylon or silk in interrupted fashion. A syringe barrel of 20 ml may also be placed and sutured with anus outside for few days.

Major complication: Stenosis of anus.

2. Umbilical hernia

Umbilical hernia is the most common form of congenital hernia in calves and many umbilical hernias are secondary to umbilical sepsis (Fig. 2).

Etiology: Umbilical hernia is common in females than males and is primarily hereditary in origin due to dominant gene with low penetrance, autosomal recessive genes or due to environmental factor (Singh *et al.*, 2020).



Inflammation and sepsis of the umbilicus, post-calving infection of umbilical infection, breakage of the umbilicus during manual traction of the foetus, external trauma to the umbilicus, excessive straining, cloned calves (less collagen in the ventral abdominal wall), hypoplasia of the abdominal musculature, and multiple births (twins, triplets, etc).

Surgical Treatment:

- Close the hernia ring by vest over pant sutures using sterile silk thread (no. 2, double).
- The calf should be dorsal recumbency to allow least pressure on the suture line and to avoid interference of the abdominal organs.
- In young calves on milk diet, the surgery can be done without keeping off feed and off water under local (total 5-7ml) and sedation (inj. diazepam (@0.2mg/kg) and inj ketamine (@4mg/kg), IV), if required.
- In adult calves/cows/heifers, as per the status of feed, may need off feed and off water for 12, 24 or 36 hrs. General anaesthesia is required to place the animal in dorsal recumbency.

Major complications: reoccurrence/infection, if sutures are not applied properly or are loose.

3. Pervious urachus

The most common congenital condition of the urinary bladder is the pervious urachus. This condition is more commonly observed in the foals, cow calves and is rare in buffalo calves (Sharma and Singh, 2004).

Etiology: In pre-natal life, the urinary bladder communicates with the allantois through urachus which becomes atrophied and its lumen gets obliterated after parturition. Complete failure of urachal lumen obliteration throughout its length results in development of pervious urachus.

- The disorder is sometimes accompanied by urethral obstruction and uroperitoneum and thus may deteriorate the animal condition and may become life threatening.

Clinical signs: The young one shows dribbling of urine from the umbilicus and the area surrounding it remains wet. The young one may pass urine from normal site or may not pass.

Treatment: Both surgical and conservative treatments are indicated (Singh *et al.*, 2020). Conservative treatment is successful in initial stages or when the condition is partial. A cotton swab dipped in 90% phenol is applied inside the urachus at a distance of 4-5 centimetres towards the urinary bladder.

- The surgical treatment is required when conservative treatment fails or the urine dribbling from the umbilicus is high. The abdomen is opened at umbilicus and the vessels and the urachus is ligated with absorbable sutures. The surgery can be done like umbilical hernia. Necrotic tissue, if any, needs to be removed.

4. Contracted tendon

It is a congenital deformity of locomotor system and is commonly observed in calves, lambs and foals and affects flexor and extensor tendon of fetlock and pastern joints (Sangwan *et al.* 2009). The tendon disorders may be congenital or acquired. Congenital abnormalities include lax tendons, contracted tendons, and displaced tendons. Acquired tendon disorders include lax tendons, contracted tendons, luxated tendons, tendinitis, lacerated tendons, avulsed tendons, ruptured tendons, and tenosynovitis.

Etiology: One school of thought says that the in-utero mal-positioning and overcrowding caused by size of the foetus relative to the dam may lead to contracted tendon condition. But other school of thought is that the condition is due to autosomal recessive gene.

Clinical signs:

At birth, the young one is unable to bear weight on affected limbs due to shortening of superficial and deep digital flexor tendon and associated muscles at fetlock and pastern region. In case of contracture at fetlock, the calf may bear weight on the anterior aspect of fetlock, leading to development of wounds and in chronic case may lead to opening of joint.

Treatment:

- Mild cases may not need surgical corrections and a bandaging with a caudal aluminium splint may be done to keep the limb straight. The bandaging and splint need to involve fetlock and carpal in case of fetlock joint. When the calf will put weight on the hoof, the condition will improve more.
- Surgical correction includes partial or complete tenotomy of the superficial digital flexor and deep digital flexor tendon is done depending on severity of knuckling. They are usually cut under local infiltration anaesthesia at the distal third region of metacarpal in case of fetlock contracture. The tension in the tendon can be felt. With manual pressure the limb is straightened and POP is a pplied with a caudal and cranial aluminium straight splint (Fig. 3).


Fig. 3

The fetlock and carpal joint need to be involved. Antibiotics and pain killers are given and the pop may be repeated after one week.

Complication: If contracture is severe, the limb may get rotated. The tarsal contractures may not be possible to be corrected.

5. Cleft palate

Clefts of the face are the developmental disorders resulting from a failure of closure in facial processes such as the fronto-nasal, maxillary (Fig. 4), and mandibular processes.



Etiology: Primary cleft palate is due to incomplete fusion of fronto-nasal prominence with the maxillary prominence, whereas the secondary cleft palate is due to incomplete fusion or failure of fusion between the lateral palatine processes. The incidence of cleft palate in cattle is estimated to be rare.

Clinical signs: Postprandial bilateral nasal discharge or milk, coughing and dysphagia are the usual clinical signs that can be observed at birth or after suckling has commenced (Smolec *et al.*, 2010).

Treatment: Surgical correction of the defect may be tried under general anaesthesia in cases with mild to moderate defect. Severe cases are difficult to treat.

Complications: Suture dehiscence with re-occurrence

6. Hypospadias

Hypospadias is a rare congenital malformation of the urethra reported in dogs, sheep, goats, cattle, rats, nonhuman primates and humans.

Clinical Sings: In hypospadias the urethra may open anywhere along its length at one or more locations from the perineum to the tip of penis and is often accompanied by the hypoplasia of the corpus cavernosum urethra (Alam *et al.*, 2005).

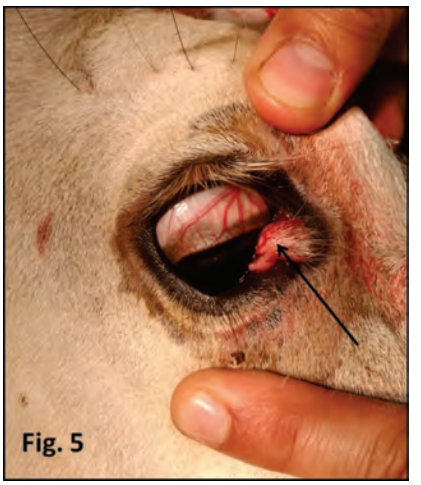
Etiology: It is due to incomplete formation of the penile urethra and imperfect closure of the external male urethra (Radostits *et al.*, 2007). The disturbance in the process of fusion of the paired urethral folds to form complete urethra after rupture of the urogenital membrane (Kluth *et al.,* 1988) or extra/intrauterine factors resulting in abnormalities of androgen metabolism and timing of receptor function during male sexual differentiation at the early period of gestation (Uda *et al.*, 2004) may be the reasons for this defect..

Treatment:

- Surgical correction is not recommended if it co exists with other anomaly (Singh *et al.*, 2020). Perineal urethrostomy can be done, if required.

7. Ocular dermoid cyst

The ocular dermoid cyst may be solitary or multiple, firm to fluctuant, well circumscribed, smooth, and round and usually the overlaying skin is normal (Fig. 5).


Fig. 5

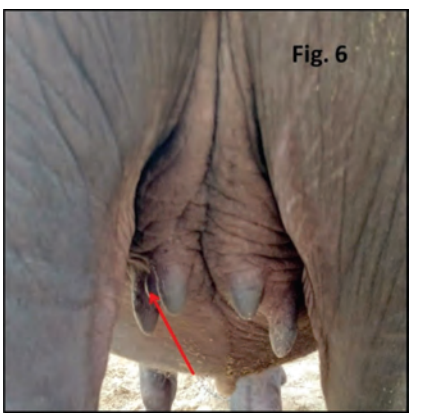
Etiology: Dermoid cysts are formed due to defective epidermal closure along embryonic fissures, which isolates an island of ectoderm in the dermis or subcutis. The cyst usually contains hair, keratin, and sebum, and these materials may produce progressive enlargement of the structure so that it becomes clinically apparent (Edwards, 2002).

Clinical signs: Hair are seen on the cornea or limbus mostly which irritate eye leading to watery discharge initially.

Treatment: Surgical removal of hair projections along with superficial lamellar keratectomy is recommended under auriculopalpebral and Peterson nerves blocks.

8. Supernumerary Teat

Supernumerary, or extra teats in ruminants are defined as any teat that is in excess of the normal number of teats (Fig. 6). The extra teat is usually blind but may sometime have streak canal.

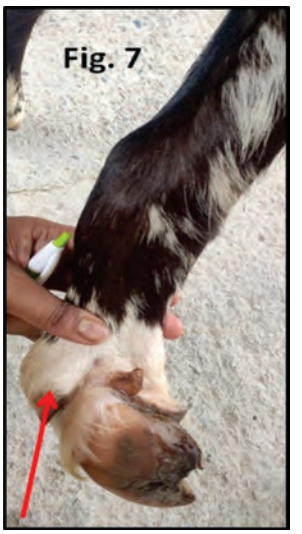

Fig. 6

Treatment: The surgical excision of the extra teat is recommended under local infiltration anaesthesia before getting pregnant for the first time. This is not recommended for con-joint teat.

9. Polydactyl

Polydactyly is an abnormal condition in which cattle are born with one or more extra digits on one or more limbs (Fig. 7). It has been reported to be sex-linked recessive gene with males expressing the trait with one gene but females requiring two genes (similar to the inheritance of red-green colour blindness in humans).

Treatment: Surgical excision of the extra digit is recommended from the base at an early age to avoid deformity in normal digit.



10. Dentigerous cyst

The dentigerous cyst is a malformation derived from deciduous and/or permanent teeth follicle and cell rests in the dental lamina. Dentigerous cysts consist of osseous tissue like enamel, cementum, dentin and pulpal tissue (malformed tooth/ teeth-like structures).

Etiology: During embryogenesis, the maxillae, mandibles, and muscles associated with chewing develop from the first branchial cleft and the teeth develop from the epithelium of the oral cavity and blend with the periodontium, cement, and dental papilla, which are derived from mesenchymal tissue (Schnorr & Stuttgart, 2001).

Clinical Sings: Swelling in the mandibular or maxillary region(s) of the head with discharge may be noticed..

Treatment: Surgical excision of the bony structure under general anaesthesia is recommended with complications of infection and reoccurrence.

11. Wry tail

It a genetic variation in domestic cattle in which the base of the tail is distorted and the tail partially turned to right or left. The defect may also be associated with congenital heart defect, so echocardiography is recommended. No treatment is required for tail.

12. Hydrocephalous

Hydrocephalus is an accumulation of excessive fluid in durameter or ventricles of the brain thereby leading to the swelling of the cranium.

Aetiology: Abnormal development of the fetus during pregnancy; hereditary, infectious, and nutritional factors may predispose. Occasional in the ewe, doe, mare, and sow, and rare cattle and buffalo (Long, 2001).

Clinical Signs: Hydrocephalus may cause increased intracranial cerebral pressure, progressive enlargement of the head, convulsions, mental disability, and even death (Saini *et al.*, 2019). The life span of the affected new born is less.

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INFECTIOUS BOVINE KERATOCONJUNCTIVITIS OR "PINKEYE"

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Abstract

Infectious bovine keratoconjunctivitis is one of the most common cattle eye infections and it has global economic implications. It is a bacterial infection of the eye that produces inflammation as well as temporary or permanent blindness in severe cases. The gram-negative bacterium Moraxella bovis is thought to be the principal cause of the disease in cattle, which affects cattle of all ages and occurs all over the world. Swelling around the affected eyes ranged from mild to severe, with profuse lacrimation. The eyelid skin, conjunctiva, and corneal opacity were commonly involved in lesions that affects both eyes. Maximizing the herd's immunological state, limiting the concentration of Moraxella and maintaining as irritant-free an environment as possible are the best measures for outbreak prevention and control.

Keywords: *Infectious bovine keratoconjunctivitis, Pink eye, Moraxella bovis,*

Introduction

Infectious bovine keratoconjunctivitis (IBK) also known as pinkeye, is found in dairy, beef cattle and water buffalo populations all over the world. As per an estimate, IBK affected 50% of herds with 5% of animals in a herd suffering from the disease (Slatter *et al.*, 1982) Cattle that have been less severely affected can recover with or without corneal scarring but, highly affected cattle can have permanent blindness as a result of corneal rupture and lens or iris prolapse. When both eyes are affected, cattle may die from starvation, thirst and accidents. The infection can progress quickly and the economic impact due to weight loss and lowered milk production can be considerable. Blepharospasm, conjunctivitis, lacrimation, variable degrees of corneal opacity and ulceration are all symptoms of infectious bovine keratoconjunctivitis (IBK) in cattle. Pinkeye can affect up to 80% of a mob, with affected weaner calves losing 10% of their body weight.

Causative agent

It is caused by *Moraxella bovis*, Gram-negative (Figure 1), β-hemolytic, aerobic, rod-shaped bacteria (Holzhauer *et al.,* 2004). When pathogenic, the organism lives as a virulent, hemolytic, rough colony form, while in the conjunctiva of recovered animals or calves, it exists as a non-hemolytic, smooth colony non-virulent form.

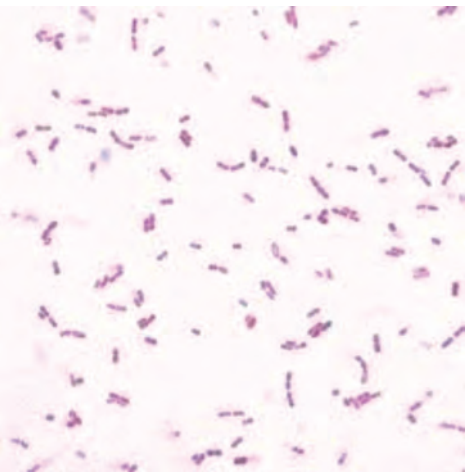


Figure 1: *Moraxella bovis* (Gram stain)

Predisposing factors: Pinkeye outbreaks are more common in the summer and autumn, when flies are more abundant and UV radiation is at its highest. This is also the time of year when mature dry thistles and dusty conditions are more prevalent. Breeds lacking pigment on their eyelids are more susceptible to pinkeye because of their increased sensitivity to sunlight and a decreased immune response in the eye. Calves are more likely to develop the disease than adult cattle, as adult cattle appear to develop protective antibodies on the surface of the eye. Bull calves have a higher incidence of disease than heifer calves. Genetic selection for pigmented eyelids and hooded eye conformation is helping to reduce this susceptibility.

Transmission: The disease is more severe in younger cattle. The bacteria are shed in nasal secretions and cattle may be subclinical carriers. Fomites, flies, aerosols, and direct contact are all methods of transmission. UV radiation and damage from dust or plant debris are two further elements that contribute to infection (Hughes *et al.*, 1990). In hot conditions, the higher incidence is reported.

Clinical Signs: Pinkeye is divided into three stages (Figure 2). The disease may get resolved spontaneously at any of these stages, but if left untreated, the most serious cases will continue through all three.

Stage I: The first sign of pinkeye is an animal with a 'runny eye'. In the first two days, the eye, conjunctiva becomes red and swollen (hence the name 'pinkeye') with a watery discharge causing tear staining and a closed eye. Cattle have excessive tearing and are more sensitive to light. Constant blinking and redness along the eyelid can be seen. Their feed intake is also reduced as a result of the pain associated with pinkeye. Stage I develops into a tiny ulcer in the center of cornea that looks as a small white spot. Because

of the inflammation, the cornea takes on a slightly foggy grey look. It's possible that one or both eyes are affected (Whittier *et al.*, 2009).

Stage II: The area in the center of the eye continues to expand in more severe infections. The cornea erodes during the next one to two weeks, forming an ulcer that spreads and expands, with the majority of the eye turning white to yellow, then red (as white blood cells and blood vessels move into the ulcer). Treatment should begin before the condition progresses to this point. The cornea may break at this point if the ulceration is severe. Once the jelly-like fluid from the center of the eyeball is lost, the sightless eye shrinks back into the eye socket (Whittier *et al.*, 2009).

Stage III: As blood vessels form over the cornea, the majority of the eye becomes red. The blood vessels in the eye begin to shrink as recuperation progresses, and the eye turns a murky blue color before clearing. 3–5 weeks after the initial infection, recovery is usually complete. The majority of damaged eyes heal fully; however, scarring can leave a small bluish-white patch in the cornea. The damaged eye remains blue in around 2% of instances, and the animal is blind in that eye (Beard *et al.*, 1994)



Figure 2: Stages of Pinkeye (Whittier *et al.*, 2009)

Treatment: Animals with IBK should be treated as soon as possible to prevent the disease from spreading to other animals and to reduce the risk of permanent eye damage**.** Sometimes cattle can recover without treatment, younger animals should be treated as soon as an infection is discovered. Topical and subconjunctival antibiotics are used as treatments (Kibar *et al.*, 2006). Gentamicin, first-generation cephalosporins, trimethoprim-sulfonamides, nitrofurans, tetracycline and sulfonamides are all known to be effective against *Moraxella bovis*. Over-the-counter oxytetracycline (LA200, LA300, LA400) is often effective against *Moraxella bovis*. Intramuscular antibiotic therapy is usually effective, especially during the early stages of the disease.

Prevention and control: Because *Moraxella bovis* is so widespread, it's hard to eradicate it, so disease prevention is the only option. The need of early diagnosis by rigorous visual inspection and isolation of affected livestock is critical. Carriers should be removed and the most significant vector should be controlled. Current vaccines provide limited protection

against clinical disease. Chemically inactivated *Moraxella bovis* bacterins containing pili antigens are available, although their efficacy as a protective agent is debatable. New animals should be quarantined and treated prophylactically before introduction to herds. One of the most common treatments for more severe episodes of pinkeye is sub-conjunctival local injection. Recent studies however shows that topical application of cloxacillin and intramuscular injections heal at a similar rate to local penicillin injections (Diagneault and George 1990).

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MEASURES TO KEEP SNAKES AWAY FROM ANIMALS

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Abstract

Diarrhoea and snake bite poisoning were found to be the major causes of calf mortality in Kandi area of Hoshiarpur district. Awareness was created to feed colostrum of first milking to the newly born calves within two hours of their birth. This had led to significant reduction in cases of enteritis in calves. Different methods were worked out for their feasibility to ward snakes off coming near to the livestock and to prevent snake bites.

Key Words: *Livestock, Snake bite, Kandi area, Carbolic acid*

Regional Research and Training Centre, Talwara conducted a survey a few years back regarding calf mortality in *kandi* area of Hoshiarpur district. The results were very disturbing. About half of the calves born alive died before attaining the age of two months because of two major causes-diarrhoea and snake bites. Majority of the dairy farmers in *kandi* area used to feed colostrum to the calves only when the dams had shed placenta. In *kandi* area especially where green fodder is very scarce, placenta is usually shed 10-14 hours after the calf is born. Feeding of colostrum after so much delay often fails to elicit desired immune response in newly born calves. These immunity deprived calves become very much prone to developing gastroenteritis. Paucity of veterinary services in *kandi* area combined with lack of will power of owners to start treatment quickly usually leads to calves dying unnaturally. We, at Regional Research and Training Centre, started a campaign to make the dairy owners aware of the importance of colostrum feeding within first two hours of birth of a calf. The other modes of capacity building of farmers regarding scientific calf management including colostrum feeding have been discussed in detail somewhere else. In the present paper, we will focus on the second cause of calf mortality i.e. preventing snake bites.

In *Kandi* area, snake bites are very common owing to extensive forest cover. Moreover, the animals are, often, kept stanchioned in open area just close to their own dwellings. Whenever, the animal is bitten by a poisonous snake, he/she should immediately receive anti-venom against that particular snake. However, non- availability of specific anti-venom or poly-valent anti-venom often leads to death of the subject in question. Thus, it will be a safe preposition to follow all the preventive measures to avoid snake bites at any cost.

Before implementing measures of preventing snake bites in animals, one must know a few points regarding their behaviour. Majority of the snakes found roaming around human and animal dwellings are non-poisonous. They usually thrive on rats, mice, frogs, insects etc. They do not pose any threat to the animals i.e. they do not attack the animals in the first place. When the animal becomes furious due to their presence, only then, they attack that particular animal. Snakes do not prefer to live among animals or in their dwellings. Since snakes can't chew, therefore, they can never make holes in the walls or burrows in the ground on their own. They just creep or crawl through holes already available in the walls and happen to be seen in the animal premises. Snakes, being poikilotherm, hibernate during colder months of the year. They are seen roaming on the ground during summers and monsoons only.

Snakes can be prevented from coming very close to the animals by following some effective points explained ahead.

1. The grasses around animal premises should not be allowed to grow too much. These should be mowed to about one inch length regularly. Snakes can easily hide/conceal themselves in overgrown grasses or untended bushes around animal sheds. Even the preying birds (like owl, eagle etc.)fail to detect snakes in haphazardly grown grasses. This way the snakes gain easy entry into animal premises.
2. Efforts should be made to attract the preying birds to animal premises. A long bar or bamboo-made-open-roosts should be installed along East-West direction so that the preying bird can easily perch over there. The perching place should be high enough so that the birds can sit over there for hours together without any fear. The height can also prove beneficial for the preying birds since they can have better view of a larger area.
3. Crops that have bushy stems (like Napier grass) should not be grown too close to the animal premises. Rats can make their shelters in such crops and snakes get attracted easily to those rats.
4. In *kandi* area, most of the ladies, as a goodwill gesture, feed cereal grains to wild birds. They usually employ earthen pots for this purpose. The birds often splatter or spill grains while eating from those pots, thereby, pulling in rats or mice. If rats or mice are there, can snakes be far behind!
5. It will be a good practice if the earthen pots full of cereal grains are put at the roof tops.
6. The incandescent bulbs or fluorescent tubes meant for lighting purpose should not be put outside the animal premises. This will attract the moths especially during monsoons. Moths will attract frogs, toads or lizards that will in turn attract

snakes. The lighting arrangements should preferably be switched off as soon as the manual work is over especially during monsoons.

7. The animal premises should not be used for stacking firewood or for storing loose goods or for putting agricultural implements over there. These will act as harbouring places for snakes.

8. The boundary walls of animal premises must be free from any kind of holes or crevices. The holes that are bigger than half an inch diameter should be plastered.

9. In areas where snake bites are frequently encountered, other methods (chemical method) of warding them off should also be followed. The best alternative method is use of phenol or carbolic acid. It is a volatile acid. Its smell is quite offensive for all kinds of snakes. The bottle of carbolic acid with holes in its cap should be installed near the entrance to animal premises in such a way that the bottle remains erect all the time. It can be put in a hole dig up in the ground for the purpose. It can also be secured in a wooden rack with the help of sand or saw-dust or ragged clothes. The wooden rack, then, can be fastened in the wall with the help of long nails.Carbolic acid is highly corrosive. Contact with skin should be avoided at any cost. Efforts should also be made to keep children and wild animals away from the bottle of carbolic acid.

10. Another physical method to keep snakes away from animals is to install a sheet made of wood, tin, plastic or asbestos in a slanting manner. The sheet should make an angle of 30° with the wall. The snake can never be able to climb this slating sheet. Installing of slanting sheets can bring lot of expenditure. To reduce its costs, plants or hedges should be grown instead of walls along slanting sheets.

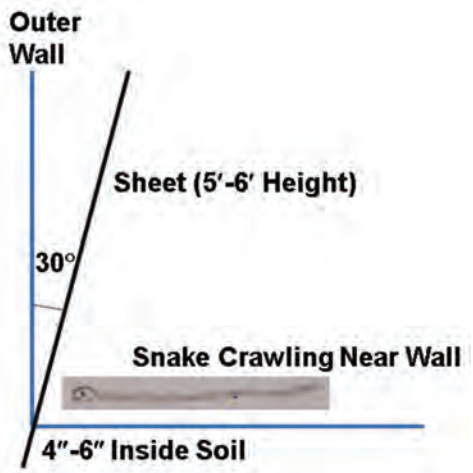


Fig.: Alignment of Some Sort of Sheet along Outer Wall

There are certain other things which should not be encouraged for keeping snakes away from animals. Naphthalene balls should never be used to fend off snakes. These are highly toxic to humans. Their mishandling can lead to accidental ingestion among children and pet animals. Research by San Julian and Woodward in 1985 clearly indicates inability of naphthalene to keep snakes away from animals. Similarly, sulphur, ammonia, lime and insecticides prove useless.

If you have an encounter with a snake, don't get panicky. Just procure a plastic pipe of 3 or 4-inch diameter. Close one end with any sort of cap. If it is not feasible, just put that end in thick plastic or gunny bag and tie it with a string. The pipe, then, should be placed on ground just near to the snake and efforts should be made to allow the snake to enter through the pipe into the bag. Once the snake is inside the bag, remove the pipe and close the mouth of the bag with that string. The snake, thus, can be easily transferred to the forest area.

If you can distinguish a non-poisonous snake, then, it can also be put into a deep bucket with the help of a broom. The path of the snake can also be manoeuvred with the aid of a water jet. The non-poisonous snake has a thin and elongated head while the poisonous snake has broad and triangular head just to accommodate poison glands. The poisonous snakes have small scales over their head and their tails taper suddenly.

When the snake is seen entering the premises but conceals itself quickly somewhere in the vicinity, then, put a wet towel or wet gunny bag or any cloth on the floor just close to the wall. Keep another bigger cloth over the wet cloth. Snakes, often, hide in dark, cool and moist places. After some time, you will notice presence of snake under that wet cloth.

When the snake was noticed entering some burrow or a hole, then, cinnamon oil, pine oil, clove oil or eugenol oil (Sukumaran *et al.*, 2012) can be used to bring the snake out of that hole.

Thus, by employing these methods, one can easily keep animals at a safe distance from snakes and life-threatening incidences involving livestock and humans may be prevented.

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COMMON GENETIC DISORDERS IN CATTLE

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Abstract

The main purpose of the present article is to describe the common genetic defects in bovine and their testing methods. Genetic defect or disease may or may not be heritable as some genetic disorders are passed down from the parent's genes. In bovines, the most common genetic abnormalities include DUMPS (Deficiency of Uridine Monophosphate Synthase), BLAD (Bovine Leukocyte Adhesion Deficiency), Citrullinaemia, Factor XI deficiency syndrome, Syndactylism, Chondrodysplasia, Muscular hyperplasia, Protoporphyria, Complex vertebral malformation (CVM), Holstein haplotypes. As a result, it is vital to study all genetic disorders, including their definitions, genetic causes/bases, screening methods and to combat them and provide economic losses in the dairy industries.

Keywords*: Cattle, Genetic Abnormalities, DNA mutation.*

Introduction

Genetic defects, genetic abnormalities, or genetic disorders are some of the most pressing concerns for animal producers. A genetic disease is an illness caused by inborn abnormalities in genes or chromosomes, which are quite uncommon and affect one animal in every several thousands or millions. The genetic diseases occur in all breeds of cattle however some defects are strongly associated with certain breeds (Weaver Syndrome' in Brown Swiss). Around 200 different genetic defects have been identified in cattle. Genetic abnormalities contribute to poor animal performance, structural unsoundness, semi-lethal disease, lethal disease, etc. New unwanted haplotypes have been found as genetics has progressed. Fertility haplotypes and congenital abnormalities are among them. Some genetic illnesses are passed down through the parents' genes, whereas others are always or almost always produced by new mutations or alterations in the DNA. The majority of them occur seldom and are of modest significance, but others increase in frequency to the point where they constitute a major economic worry and must be avoided. The most prevalent genetic illness inheritance pattern is a simple recessive trait. From its sire and dam, the faulty calf inherits a recessive gene. Genes with partial dominance are known to cause a few hereditary disorders, and others are caused by two or more sets of genes.

Common Genetic Diseases

DUMPS (Deficiency of Uridine Monophosphate Synthase): DUMPS is a genetic deadly autosomal condition that causes early embryonic death upon uterine implantation.

The conversion of orotate to uridine monophosphate synthase (UMPS) is the final step of pyrimidine nucleotide synthesis in mammalian cells, and it is catalysed by the UMP synthase enzyme. A premature stop codon is produced when a gene for UMPS is mutated, resulting in a functionally deficient enzyme. Approximately 40 days following conception, the embryos appear to be aborted or reabsorbed, resulting in recurring breeding issues.

BLAD (Bovine Leukocyte Adhesion Deficiency): Bovine leukocyte adhesion deficiency (BLAD) is an autosomal recessive disease. It is marked by a decrease in the expression of the adhesion molecules - integrins on neutrophils. These proteins aid neutrophil migration to the inflammatory location. Recurrent bacterial infections, pneumonia, ulcerative and granulomatous stomatitis, enteritis with bacterial overgrowth, periodontitis, tooth loss, delayed wound healing, chronic neutrophilia, and early mortality are all symptoms of BLAD in animals.

Citrullinaemia: Citrullinaemia in cattle is an autosomal recessive hereditary urea cycle illness that is fatal in the early postnatal period. It is caused by a lack of argininosuccinate synthase (ASS), which is one of the urea cycle's enzymes. Bovine citrullinaemia causes calves to appear normal right after delivery. The sickness, however, accelerates between the third and fifth day. Within 12 hours of the commencement of these clinical indications, most calves die. The accumulation of ammonia in the brain of the affected calves is thought to cause the clinical manifestations of citrullinaemia.

Factor XI Deficiency Syndrome: Factor XI (FXI) is one of more than a dozen proteins involved in the early blood coagulation cascade. FXI deficiency syndrome is caused by an insertion of a 76bp segment within exon 12 of the FXI gene. Heterozygous individuals with FXI deficiency have serious health problems. On the other hand, both heterozygous and homozygous animals for this mutation have susceptibility to some diseases such as mastitis, metritis, and pneumonia and also have lower calving and survival rates.

Syndactylism: Bovine syndactylism is a heritable disorder also known as mule foot disease. This malformation has an autosomal recessive character, and it occurs differently in each case. This is due to incomplete penetrance and variable expression of this trait (Johnson *et al.* 2006). The defect is due to the deletion and insertion of c.4863-4864delCGinsAT (p.Asn1621Lys; p.G1622C) and point mutation c.4940>T. This genetic disorder is a non-division or fusion of digits, and it mostly appears as synostosis of phalanges. More specifically, the right-front limb is affected predominantly, then left front, right rear, and left rear limb. Syndactylism can be present in both dairy and beef cattle. During studies under mutation causing mule foot disease, there have been many candidates (Duchesne *et al.* 2006), but eventually, one gene has become a strong one—*LRP4* (low-density lipoprotein receptor-related protein 4 gene) (Duchesne *et al.*, 2006).

Chondrodysplasia: Another disproportionate dwarfism, chondrodysplasia (OMIA

000187-9913), is caused by a mutation in the *EVC2* gene on chromosome 6. Causative mutation is 2-bp deletion (c.2993_2994ACdel) was discovered in exon 19 of this gene in Tyrolean Grey cattle. *EVC2*, also named *LBN*, codes protein Ellis-van Creveld syndrome 2 protein that transmembrane compound of the basal body of the primary cilia. The mutation leads to frameshift and premature codon stop and it is still unknown whether the product is not expressed at all or it just loses its functions due to structural alterations. However, this variation results in impaired cranium, vertebra, and limbs development.

Muscular Hyperplasia: Muscular hyperplasia, also known as double muscling, is an inherited condition that results from an increase in the number of muscle fibers. This condition is also, incorrectly, called hypertrophy (Bouyer *et al.*, 2014). Animals with DM phenotype present many clinical syndromes. They are characterized by extremely high carcass yield, which is connected with a reduction in the size of most vital organs such as the heart, lungs, kidneys, as well as, a high frequency of broken bones. Myostatin (MSTN), also known as GDF8 (growth and differentiation factor 8), is included in the superfamily of transforming growth factor β (TGF-β). It functions as a negative regulator of skeletal muscle mass development by inhibiting the Myo5 and MyoD factors, which play a pivotal role in muscle development and maturation.

Protoporphyria: Bovine protoporphyria or bovine ferrochelatase deficiency is inherited as a recessive trait. It is characterized by a decrease in the activity of enzymes taking part in heme biosynthesis ferrochelatase. Protoporphyria accumulation of protoporphyrin occurs and it might be manifested by an increased level of protoporphyrin in blood and feces. Due to photoreactivity of this compound, subsequently, photosensitivity appears in individuals affected by this defect, which causes i.e. ulceration, alopecia (mostly nostrils and earlobes lesions), pain. Mutation, which causes bovine protoporphyria occurs in gene *FECH* that encodes ferrochelatase, which has been mapped to chromosome 24 (NCBI Gene ID: 281158). It is transversion of guanine to thymine appearing in exon 11 at 1250 position that leads to conversion of a stop codon to leucine (TGA→TTA; p.X417L)

Complex Vertebral Malformation: Complex vertebral malformation (CVM) is a lethal syndrome of Holstein cattle, which is premature and mature calves is characterised by congenital growth retardation, malformed vertebrae, and tetrameric arthrogryposis. It is a recessively inherited disorder leading to frequent abortion of fetuses or perinatal death, and vertebral anomalies (Agerholm *et al.*,2004;Nielsen *et al.,* 2003). Statistical analyses of breeding data have demonstrated reduced maternal fertility if carriers of CVM are mated; an observation probably due to extensive intrauterine mortality in CVM affected foetuses.

Holstein Haplotypes: The autosomal codominant Holstein haplotype is linked to highly defective cholesterol metabolism, which has a major impact on human health

and the survival of homozygous haplotype carriers. On bovine chromosomes 1 (HH1), 5 (HH2), and 8, three fatal haplotypes impacting fertility in HF have been found (HH3). Mutations in the *APOB* gene cause a congenital disorder in Holstein cattle that is similar to hypobetalipoproteinemia (HHBL) in humans. It is linked to a phenotype in calves that includes growth retardation, emaciation, and chronic or recurrent diarrhoea, and it manifests itself in several ways.

Diagnosis and control of genetic disorders in cattle

During the selection of animals particularly the bull selection, genetic defects should be identified and animals carrying those defects should be culled out from the herd. Suitable infrastructure should be developed at relevant institution for detecting the genetic defects in cattle.

Polymerase chain restriction-Restriction Enzyme digestion of PCR product (RFLP) methods is the best molecular screening method for obtaining speedy and reliable results. Both homozygous and heterozygous animals can be easily screened. By using such techniques we can prevent the propagation of faulty genes within the population by eliminating carrier heterozygous animals.

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