

Introduction of livestock and poultry diseases

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Anthropogenic - of, relating to, or resulting from the influence of human beings on nature.

Autolysis – spontaneous disintegration of cells or tissues by autologous (self) enzymes, as occurs after death (post mortem) and in some pathologic conditions; lysis, enzymatic digestion, of cells by the enzymes present within them.

Biomarker – any measurable sign (including visual and molecular) indicating the status of an organisms physiology, health, etc. that can be used in the diagnosis of a disease or syndrome⁴; includes a variety of measures of specific molecular, cellular, and physiological responses of key species to either contaminant exposure or poor health.

Bleach – to make or become white or colorless by means of chemicals, by exposure to sunlight, etc. In coral – *bleaching* or *bleached* – the reduction in intensity or complete absence of coloration within living coral, due to loss of pigmentation, death, or expulsion of endosymbiotic zooxanthellae.

Bleaching - the loss or expulsion of symbiotic dinoflagellates (*Symbiodinium* spp.) by corals; can be caused by various stressors, including increased temperatures and toxins produced by bacterial pathogens. During temperature-induced bleaching, caspase-mediated apoptosis and autophagy are reciprocally activated to expel the dinoflagellate symbionts.

Degeneration – a nonspecific term applied to retrogressive but sometimes reversible pathological change in cells or tissues, resulting in impairment or destruction of functions; deterioration; preferably the specific changes observed should be fully described.

Degradation – conversion of a chemical compound to one less complex as by splitting off one or more groups of atoms. Degeneration, not degradation, should be used to describe the deterioration or change of functionality of living tissue.

Diagnosis – the determination of the nature of a disease.

Field Diagnosis – made from the study of the macroscopic changes of a coral disease observed in the field.

Laboratory Diagnosis – made by a chemical, virological, parasitological, microbiological, or immunological study of secretions, discharges, or tissues.

Morphologic Diagnosis – made from an anatomical or histological study of the lesions present.

Etiologic Diagnosis – the determination of the cause of the disease.

Differential Diagnosis – a systematic comparison and contrasting of similar disease signs and findings to determine which of two or more diseases is considered to be most likely present in the organism, although one or more other diseases are considered less likely to be present but possible. (See also Pathognomonic) A differential diagnosis is also made to distinguish between closely related species in taxonomy.

Disease – any deviation from, or interruption of, the normal structure or function of any body part, organ, or system that is manifested by a characteristic set of signs and whose etiology, pathology, and prognosis may be known or unknown; any impairment that interferes with or modifies the performance of normal function, including responses to environmental factors such as nutrition, toxicants, and climate; infectious agents; inherent or congenital defects, or combinations of these factors.

Epizootic – attacking many animals in any region at the same time; widely diffused and rapidly spreading; a disease of high morbidity which is only occasionally present in an animal community.

Epizootiology – the scientific study of factors determining the frequency and distribution of diseases among animals; animal epidemiology.

Etiology – the study or theory of the factors that cause disease and the method of their introduction to the host; the causes or origin of a disease or disorder.

Hypertrophy – The enlargement or overgrowth of an organ or part due to an increase in size of its constituent cells

Incidence – the rate at which a certain event occurs; e.g., the number of new cases of a specific disease occurring during a certain time period in a population at risk.

Infection – invasion and multiplication of parasitic organisms within the body.

Koch's Postulates – to establish the specificity of a pathogenic microorganism, it must be present in all cases of the disease, inoculations of its pure cultures must produce disease in animals, and from these it must be again obtained and be propagated in pure culture.

Lesion – a wound or injury, or any pathologic change in the tissues.

Lesion Distribution – distinguished on the basis of number of that particular type of lesion (focal: single, localized area; multifocal: relating to, arising from, or occurring in more than one place; diffuse: spread about, not restricted; systemic: spread throughout the entire organism). Lesion Severity – semiquantitative, subjective ranking of the degree of damage or extent of pathological change seen in tissues or an organism (ranging from minimal: smallest amount or lowest limit; mild; moderate; marked; to severe: intensely or extremely bad, very poor condition, or greatest in degree or extent). Morbidity – a diseased condition or state; the incidence or prevalence of a disease, or of all diseases, in a population.

Morphology – the form and structure of a particular organism, organ or part.

Mortality – the death rate; the ratio of the total number of deaths to the population of a specified area in a given time period.

Necrosis – cell death characterized by irreversible damage, the earliest of which is mitochondrial. Changes visible with light microscopy are nuclear (pyknosis, karyolysis, or karyorrhexis) and generally accompanied by cytoplasmic hyper-eosinophilia, shrinkage, or fragmentation. After such changes, the outlines of individual cells are indistinct and affected cells may become merged, sometimes forming a focus of coarsely granular, amorphous, or hyaline material.

Pathogen – any virus, microorganism, or other substance causing disease. pathogenic, adj.

Pathogenicity – the quality of producing or the ability to produce pathologic changes or disease.

Pathophysiology – the physiologic changes in the host as the disease progresses. Prevalence – the number of cases of a specific disease that are present in a given population at a specified time.

Sign – an indication of the existence of something; any objective evidence of a disease, i.e., such evidence as is perceptible to the examiner, as opposed to the subjective sensations of the patient (symptom). *Sign, not symptom should be used to describe lesions or conditions observed in coral, as the ability to convey sensations (feelings) is not a characteristic of coral.*

Syndrome – a set of symptoms occurring together; the sum of signs of any morbid (disease) state.

Ulceration – The formation or development of an ulcer, a local defect or excavation, of the surface of an organ or tissue, which is produced by the sloughing of inflammatory necrotic tissue.

Term	Definition
INFECTIOUS DISEASE	caused by living agents such as bacteria, vi fungi, and parasites that invade the body c illness; can be spread to other animals Ex: Feline upper respiratory infection

Term INFECTIOUS DISEASE	Definition caused by living agents such as bacteria, viruses, fungi, and parasites that invade the body causing illness; can be spread to other animals Ex: Feline upper respiratory infection
Term NON-INFECTIOUS DISEASE	Definition Not spread to other animals but cause of illness or symptoms in host animal. Ex: Kidney disease
Term CONTAGIOUS DISEASE	Definition Diseases transmitted directly or indirectly from an infected animal to a healthy animal; infectious diseases are usually contagious
Term ZOONOTIC	Definition diseases spread from animals to humans or vice versa. Ex: Rabies
Term CONGENITAL DISEASE / CONDITION	Definition a disease/condition present at birth. Ex: cleft palate
Term VECTOR	Definition A living organism (flea, tick, mosquito) that transmits disease
Term FOMITE	Definition inanimate objects (bowls, blankets, doors, shoes) that provide a means of infectious disease transmission
Term	Definition

<p style="text-align: center;">CARRIER</p>	<p style="text-align: center;">animals that harbor contagious disease without showing clinical signs.</p> <p style="text-align: center;">Ex: Feline Aids</p>
<p>Term</p> <p style="text-align: center;">DIAGNOSIS</p>	<p>Definition</p> <p style="text-align: center;">process of identifying or determining the cause and nature of a disease or condition</p> <p style="text-align: center;">Ex: Dr. says the dog has a fractured femur</p>
<p>Term</p> <p style="text-align: center;">PROGNOSIS</p>	<p>Definition</p> <p style="text-align: center;">prediction of the probable course of a disease condition and the probability of recovery</p> <p style="text-align: center;">Ex: Dr says, "with surgery the dog with fractured femur will make a full recovery"</p>
<p>Term</p> <p style="text-align: center;">MORBIDITY</p>	<p>Definition</p> <p style="text-align: center;">The <i>number of sick animals</i> within a given group/herd</p>
<p>Term</p> <p style="text-align: center;">MORTALITY</p>	<p>Definition</p> <p style="text-align: center;">the <i>number of animals that die</i> from disease in a given group/herd</p>
<p>Term</p> <p style="text-align: center;">NECROPSY</p>	<p>Definition</p> <p style="text-align: center;">examination of a dead animal body by a veterinarian; an <i>autopsy</i> of an animal</p>
<p>Term</p> <p style="text-align: center;">PATHOGENS</p>	<p>Definition</p> <p style="text-align: center;">disease causing organism</p> <p style="text-align: center;">Ex: AIDS</p>
<p>Term</p> <p style="text-align: center;">ACUTE DISEASE</p>	<p>Definition</p>

	<p align="center">Disease with a short duration</p> <p align="center">Ex: flu</p>
<p>Term</p> <p align="center">CHRONIC DISEASE</p>	<p>Definition</p> <p align="center">disease with a <i>long</i> duration</p> <p align="center">Ex: arthritis</p>
<p>Term</p> <p align="center">SUBCLINICAL</p>	<p>Definition</p> <p align="center">does not develop full-scale clinical signs</p>
<p>Term</p> <p align="center">LOCAL</p>	<p>Definition</p> <p align="center">Disease or condition is confined to a small area of the body</p> <p align="center">Ex: ringworm; poison ivy</p>
<p>Term</p> <p align="center">SYSTEMIC</p>	<p>Definition</p> <p align="center">diseases that affect the whole body or a large part of the body</p>

1. Black Quarter (BQ)

Caused by *Clostridium chauvei*

An acute disease of cattle characterized by emphysematous swelling usually in heavy muscles.

Buffaloes usually suffer from a milder form.

Contaminated pasture appears to be major source of infection.

Healthy animals in the age group 6 months to 2 years are generally affected

SYMPTOMS

Sudden high fever (107°F-108°F) and the animal stops eating and ruminating.

Characteristic hot and painful swelling develops on loin and buttocks causing lameness. Swelling sometimes affects shoulders, chest and neck also. When pressed, a crackling sound is heard because of the gas accumulation in the swellings.

Animal dies within 24-48 hrs of appearance of symptoms. At this juncture, swellings become cold and painless.

PREVENTION

Vaccinate all animals which are 6 months and above of age annually before the on-set of monsoon in endemic areas.

Burning the upper layer of soil with straw to eliminate spores may be of help in endemic areas.

Sprinkle lime over carcass at the time of burial.

TREATMENT

Treatment may be effective in initial stages of infection. However in most cases treatment is not worth the while.

2. FMD

Foot-and-mouth disease (FMD) is a highly communicable viral disease caused by an Aphthovirus of the family Picornaviridae. There are 7 serotypes: A, O, C, Asia 1, and SAT (Southern African Territories) 1, 2, and 3. Further diversity is found in strains within each serotype. It primarily affects cloven-hooved animals of the order Artiodactyla. Livestock hosts include cattle, pigs, sheep, goats, and experimental infections in alpacas and llamas. FMD virus has also been reported in >70 species of wild artiodactyls, including bison, giraffes, Indian elephants, and several species of deer and antelope.

The virus survives in lymph nodes and bone marrow at neutral pH, but is destroyed in muscle when pH is less than 6.0, i.e., after rigor mortis. The virus can persist in contaminated fodder and the environment for up to one month, depending on the temperature and pH conditions. Airborne spread of the disease can take place and under favourable weather conditions the disease may be spread considerable distances by this route.

Animals pick up the virus either by direct contact with an infected animal or by contact with foodstuffs or other things which have been contaminated by such an animal, or by eating or coming into contact with some part of an infected carcass. Outbreaks have been linked with the importation of infected meat and meat products. The disease can also be spread by people, vehicles and other objects that have been contaminated by the virus.

Symptoms

Fever, Blisters in the mouth and on feet, Drop in milk production, Weight loss, Loss of appetite, Quivering lips and frothing of mouth, Cows may develop blisters on teats, Lameness

Treatment

Treatment is not given. Affected animals will recover. However because of the loss of production and the infectious state of the disease, infected animals are usually culled.

Prevention

- FMD is one of the most difficult animal infections to control. Because the disease occurs in many parts of the world, there is always a chance of its accidental introduction into an unaffected country. Export restrictions are often imposed on countries with known outbreaks. FMD outbreaks are usually controlled by quarantines and movement restrictions, euthanasia

of affected and in-contact animals, and cleansing and disinfection of affected premises, equipment and vehicles.

- Infected carcasses must be disposed of safely by incineration, rendering, burial or other techniques. Milk from infected cows can be inactivated by heating to 100°C (212°F) for more than 20 minutes. Slurry can be heated to 67°C (153°F) for three minutes. Rodents and other vectors may be killed to prevent them from mechanically disseminating the virus.
- Good biosecurity measures should be practiced on uninfected farms to prevent entry of the virus.

Vaccination

- Vaccination can be used to reduce the spread of FMD or protect specific animals. Vaccines are also used in endemic regions to protect animals from clinical disease. FMDV vaccines must closely match the serotype and strain of the infecting strain. Vaccination with one serotype does not protect the animal against other serotypes, and may not protect the animal completely or at all from other strains of the same serotype. Currently, there is no universal FMD vaccine.

3. Mastitis

Mastitis is an infectious disease condition resulting in an inflammatory reaction in the mammary gland of the cow. It is the most common disease in dairy cattle characterized by various degrees of severity - ranging from a mild disease with no gross changes in the secretion (milk) but an increase in inflammatory cells (somatic cells) in the milk, to a moderate disease with an increase in inflammatory cells and gross changes in the milk. It may be accompanied by signs of inflammation in the mammary gland including swelling, redness, and painfulness. Mastitis may progress to a severe disease with all of the above changes in the milk and systemic signs including fever, depression, and “off-feed” and occasionally even death in the most severe cases. Mastitis reduces milk production and milk quality.

Etiology

Mastitis causing pathogens include bacteria (mostly *Staphylococcus aureus*, coagulase-negative staphylococcus, *Streptococcus uberis*, *Streptococcus dyslactiae*, *Streptococcus agalactiae*, enterococci and coliform bacteria including *Escherichia coli*) and *Mycoplasmas*.

Mastitis can be subdivided into two categories based on the source of infections: 1) Contagious mastitis infections acquired by transmission of contagious bacteria from cow to cow during the milking process; and, 2) Environmental infections acquired from bacteria in the environment of the cow.

Symptoms

Subclinical mastitis: Most commonly associated with *S. aureus*, *Strep. Spp.* Milk appears normal and there is no visible sign of inflammation of the mammary gland. Diagnosis will be made on the basis on an increase in somatic cell counts in the milk.

Clinical mastitis : Depending on the type of pathogens involved, fever and depression could be associated with the disease. Evidence of mammary gland inflammation (redness, heat, swelling, pain) Physical changes in the milk (from a few milk clots to appearing like serum with clumps of fibrin).

Acute mastitis (organisms most commonly associated: coliform organisms including E. coli and Klebsiella, and Strep. Spp .and enterococci). Most commonly associated with Coliform organisms such as E. coli and Klebsiella and with Strep Spp

Clinical signs (fever, depression, loss of appetite) are severe. The udder is swollen, hard and painful. The milk may contain clots or flakes and can be watery, serous or purulent.

Acute gangrenous mastitis- Most commonly associated with S. aureus, Cl. Perfringens, and E. coli. Anorexia, dehydration, depression, fever and signs of toxæmia, sometimes leading to death. Early in the disease, the gland is red, swollen and warm Within a few hours the teat becomes cold. The secretions become watery and bloody. The mammary gland becomes necrotic.

Chronic mastitis (organisms most commonly associated: coagulase-negative staphylococci, S.aureus, S. uberis). Most commonly associated with coagulase-negative staphylococci, S. aureus, and S. uberis. Clinical signs of an acute infection from time to time with no clinical signs for prolonged intervals. Milk periodically contain clots, flakes or shreds of fibrin. The Somatic Cell Count is elevated.

Diagnosis

Diagnosis is made on clinical signs of abnormal milk, swelling of the udder (tender to the touch) and general signs of illness (fever, depression, loss of appetite) and in many cases a reduction in milk production. For subclinical mastitis, the diagnosis will be made on the basis of an increase in somatic cells in the milk. Bacteriology provides useful information about the likely source of the bacteria and aids in developing a mastitis control plan.

Treatment

Dry cow intra-mammary antibiotic treatment consists of a dry tube in each quarter at dry off Lactating mastitis therapy should be based upon the severity of the mastitis and knowledge of the bacteria causing the mastitis. For mild to moderate cases of mastitis, the preferred route of treatment is with an intra-mammary mastitis tube. If cases are being treated without the results of a bacteriological culture, a tube with a broad spectrum of activity should be selected. If bacteriological results are known, the tube should be selected based on culture and sensitivity results. The antibiotics in mastitis tubes kill the bacteria that are exposed to adequate concentrations of antibiotic. Some mastitis tubes allow the use of extended therapy (up to 8 days). Follow all label directions when using tubes. For cows with severe clinical mastitis with systemic signs, an appropriate systemic antibiotic should be part of the therapy.

Prevention

Control of mastitis is based on prevention of infections. A sound mastitis control program should implement the following practices: Proper use of a functional milking machine with appropriate

milking machine maintenance. Teat dipping, pre- and post-milking with an effective, approved teat dip.

Early treatment of clinical cases.

Dry cow therapy at the end of lactation with one of a Dry cow mastitis products. Use of an internal teat sealant. Culling of cows with chronic mastitis. Appropriate vaccination with a Gram-negative core antigen vaccine to prevent coliform infections.

4. Haemorrhagic Septicaemia (HS)

It is caused by *Pasteurella multocida*. This is an acute bacterial disease of cattle and buffaloes which usually occurs during monsoon. Mortality rate may be as high as 80 %. Germs of this disease survive longer in humid and waterlogged conditions.

SYMPTOMS

High temperature, sudden decrease in milk yield. Salivation and serous nasal discharge. Severe oedema of the throat region. Difficulty in breathing, animal produces a grunting sound. Animal usually dies within 1-2 days of showing symptoms. Buffaloes are generally more susceptible than cattle. Animals with clinical signs, particularly buffalo, rarely recover. In endemic areas, most deaths seen in older calves and young adults.

PREVENTION

Segregate the sick animal from healthy ones and avoid contamination of feed, fodder and water. Avoid crowding especially during wet seasons. Vaccinate all animals which are 6 months and above of age annually before the on-set of monsoon in endemic areas.

TREATMENT

Treatment is usually ineffective unless treated very early, that is during the stage when fever sets in. Few animals survive once clinical signs develop. Case fatality approaches 100% if treatment is not followed at the initial stage of infection.

5. Salmonellosis'

Salmonellosis is an infection of the digestive tract caused by the bacterium, *Salmonella enterica*. *Salmonella enterica* has over 2,000 strains. Fortunately cattle are usually clinically infected by less than 10 of them. The majority of *Salmonella* that infect cattle are in groups B (species example - *S. Typhimurium*), C (example - *S. Montevideo*), D (example - *S. Dublin*), or E (example - *S. Anatum*). The type that most commonly infects cattle in the Northeast is *Salmonella Typhimurium*.

Infection can range from apparently "healthy" carrier animals to those that show acute signs of the infection. A spectrum of symptoms occurs with *Salmonella* infection, from inapparent or subclinical infections to obvious or clinical disease. In calves, the disease most commonly affects colostrum-deprived or deficient calves, and may cause a fever (105°–107° F), diarrhea (yellow with or without flecks of blood and mucus), rapid dehydration and death within 24–48 hours.

In adult cattle, severe intestinal disease is often brought on by some stress factor(s). Clinical signs include: fever (104°–106° F), followed by going off feed, depression, and foul-smelling diarrhea

with varying amounts of blood, mucus, and shreds of intestinal lining. In milking animals, milk production severely drops. Abortions may occur in infected cattle. Dehydration varies with the severity of disease. Temperatures typically rise 12 to 24 hours before other signs and may drop off again with the onset of diarrhea. Death rates vary depending on the serotype of Salmonella involved.

Clinical illness usually lasts 7–10 days, with recovery in 2 to 3 weeks. Some animals, however, never resume full production. Sick cows that recover may become carriers that shed Salmonella for varying periods of time (e.g., Salmonella Typhimurium is shed from 3 to 6+ months while Salmonella Dublin is shed for life).

In chronic cases, following an acute episode, fever (103°–104° F) is intermittent and watery diarrhea persists, resulting in progressive dehydration and weight loss. Recovery may be slow and death rates are difficult to predict because cattle are often culled due to unthriftiness and poor condition.

Supportive therapy, such as oral or intravenous electrolytes and fluids, are used to treat sick cows. While antibiotics are often used, they alone are seldom effective, especially if the disease has progressed to the diarrheal stage. One type of Salmonella, called Salmonella Typhimurium DT104, is often seen in sick cattle and is of particular concern to public health officials. Salmonella Typhimurium DT104 is highly virulent, and causes severe symptoms in animals and people. Outbreaks due to this type have resulted in cattle deaths and severe illness in farm families, because it is resistant to a wide range of antibiotics and is quite difficult to treat.

Poultry

Pullorum disease

Infections with Salmonella pullorum usually cause very high mortality in young chickens and turkeys within the first 2 - 3 weeks of age. In adult chickens, mortality may be high but frequently there are no clinical signs. The route of infection is oral (via feed or litter) or via the navel/yolk. Also transmission via the egg appears.

Clinical findings and lesions: Birds may die in the hatchery shortly after hatching. Affected birds huddle near a heat source, are anorectic, appear weak and have whitish diarrhoea around the vent. Survivors are small in size and frequently become carriers with localized infection of the ovary. These birds can continue to infect other birds. In mature chickens, chronic infections produce lesions indistinguishable from those of fowl typhoid.

Fowl typhoid

The causal agent of fowl typhoid is Salmonella gallinarum. Like S. pullorum, S. gallinarum is egg-transmitted and produces lesions in chicks and poults. However, there is a much bigger tendency to spread among growing or mature flocks. Mortality in young birds is similar to that seen in S. pullorum infection but may be higher in older birds.

Clinical findings and lesions: Clinical signs and lesions in young birds are similar to those seen with *S. pullorum* infection. Older birds may have a fever, be pale, dehydrated, and have diarrhoea. Also the liver is swollen, brittle and often bile-stained.

Diagnosis

As *Salmonella* often colonizes in the intestinal tract, this, and its contents, should be cultured. *Salmonellae* can spread to many tissues including ovary, spleen, liver, yolk sac, heart, heart blood, kidney, gall bladder, joint fluid/pus and the eyes. All of these can be cultured as part of a post mortem examination.

Prevention

Salmonella can be controlled with the successful combination of several strategies:

Hatching eggs should be clean and disinfected or fumigated.

Day old chicks should come from *Salmonella*-free breeder flocks.

Salmonella negative feed should be obtained. Feed should be heat-treated or treated with acids, to avoid recontamination after production. Also attention needs to be paid to the transport of feed (e.g. no open trucks to prevent contamination via pigeon-droppings).

Drinking water should come from a clean source and ideally should be chlorinated.

Only essential equipment should be allowed into the poultry house, after acknowledging that there are no *Salmonella* organisms present.

Bedding material is a potential risk of *Salmonella* infection. Floor feeding should be reflected on, as birds scratch through the bedding and litter, which increases the risk of infection.

6. Bird flu

Bird flu, also called avian influenza, is a viral infection that can infect not only birds, but also humans and other animals. Most forms of the virus are restricted to birds. H5N1 is the most common form of bird flu. It's deadly to birds and can easily affect humans and other animals that come in contact with a carrier. According to the World Health Organization Trusted Source, H5N1 was first discovered in humans in 1997 and has killed nearly 60 percent Trusted Source of those infected. Currently, the virus isn't known to spread via human-to-human contact. Still, some experts worry that H5N1 may pose a risk of becoming a pandemic threat to humans.

Causative agent

The virus causing avian influenza is an Influenzavirus A virus of the family Orthomyxoviridae. Several virus subtypes exist, which are divided on the bases of the antigenic relationships in the virus glycoproteins haemoagglutinin (H) and neuraminidase (N). At present 15 H subtypes have been recognised (H1-H15) and nine neuraminidase subtypes (N1- N9).

Influenza A viruses infecting poultry can also be divided on the basis of their pathogenicity (ability to cause disease).

The very virulent viruses cause highly pathogenic avian influenza (HPAI) with mortality in poultry as high as 100%. In the whole world there have been only 19 reported primary isolates of such viruses

from domestic poultry since 1959. A severe epidemic occurred in Italy in 1999/2000 causing 413 outbreaks with 16 Million birds affected.

Other AI viruses cause a much milder disease (low pathogenic avian influenza, LPAI). Clinical signs are much less evident or even absent and mortality is much lower.

Sometimes secondary infections or environmental conditions may cause exacerbation of LPAI infections leading to more serious disease. Evidence suggests that certain avian influenza virus subtypes of low pathogenicity may, after circulation for some time in a poultry population, mutate into highly pathogenic virus strains.

To date only viruses of H5 and H7 subtype have been shown to cause HPAI in susceptible species, but not all H5 and H7 viruses are highly pathogenic.

Clinical symptoms

The main symptoms of HPAI in poultry are depression, loss of appetite, cessation of egg laying, nervous signs, swelling and blue discoloration of combs and wattles due to disturbance of blood circulation, coughing, sneezing and diarrhoea. Sudden death can occur without any previous signs. The mortality rate may reach up to 100% depending on the species, their age, the virus type involved and environmental factors like concurrent bacterial infections.

Clinical signs of LPAI consist primarily of mild respiratory disease, depression and drop in egg production in laying birds.

The incubation periods for these viruses range from as short as a few hours to 3 days in individual birds and up to 14 days to spread throughout a flock.

Transmission and spread

All the available evidence suggests that the most common primary introduction of AI viruses into an area is by wild birds, usually waterfowl, but gulls and shorebirds have also been implicated. Direct contact between wild bird and poultry is not always necessary for introduction of virus into poultry farms, as infected waterfowl may spread AI viruses by infective faeces into an area and these may then be introduced to poultry farms by a variety of mechanisms that may transfer the virus mechanically. If contaminated with influenza viruses, surface water used as drinking water may also be a source of infection. Poultry kept in free range or poultry which have access to surface water are at specific risk. AI is transmitted within a farm by direct contact of infected animals with healthy animals, or indirect contacts with contaminated equipment or farm staff.

Spread of AI viruses from farm to farm is mainly by mechanical transfer of infective faeces, in which virus may be present at high concentrations and may survive for considerable periods. Shared water or food may also become contaminated.

However, man is a very important cause of secondary spread of AI for domestic poultry. Caretakers, farmers, workers, trucks and drivers visiting farms, moving birds or delivering food have caused the spread of AI virus both on to and within farms.

7. Fowl Pox

Nature of disease

Disease affects birds of all ages poor weight gain

Disease affects birds of all ages, poor weight gain

Fowl pox is a common, slow spreading, widely prevalent, contagious viral disease of poultry.

This is characterized by wart like growth over the non-feathered parts of skin and mucosa of the upper respiratory and digestive systems.

Economically very important as it can cause poor weight gain, drop in egg production in layers and mortality.

It has no zoonotic importance.

Causes

Avipoxvirus

Overcrowding

Through vaccination virus transfer from one to another

Disease is caused by an avipoxvirus, which is relatively resistant to common disinfecting procedures and can survive in dried scab for years.

Virus does not penetrate intact skin. Some break in the skin is required for the virus to enter the epithelial cells, replicate and cause disease.

Disease spreads mechanically through direct contact, where contaminated materials soil the abraded/lacerated skin, by mosquitoes and other biting insects.

In a contaminated environment, aerosols (droplets) generated from feather follicles and dried scabs carry the virus and spread the disease.

It can be transmitted by the respiratory tract.

During vaccination, the individuals may transfer the virus from affected to healthy birds.

Deposition of virus in eyes, through lachrymal duct it goes to larynx and causes upper respiratory tract infection.

Disease is frequently observed during rainy and winter seasons with persisting overcrowding and unhygienic conditions.

Disease remains a problem for a long time, in farms where multiple age birds are maintained, even after preventive vaccinations.

Clinical symptoms

Wart like growth in the non-feathered parts of the body

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Wart like growth in the non-feathered parts of the body

This disease is manifested clinically in one of the two forms, cutaneous or diphtheritic or both.

Poor weight gain and drop in egg production in layers is always significant.

In cutaneous form (dry pox), lesions in the form of scab appears on the comb, wattles, eyelids, external nares, corner of the beak and other non-feathered parts of the body.

Lesions on eye may affect the bird's vision or close both eyes affecting the ability of the bird to reach feed and water leading to starvation and deaths.

Papule → vesicles → pustules → crust/scab → scar formation.

In cutaneous form, flock mortality is usually low rarely exceeding 25%.

In diphtheritic form (wet pox or fowl diphtheria), initially small nodules are formed on the mucous membrane of mouth, oesophagus and trachea.

Later on, these nodules become yellow cheesy in nature, thereby, forming a diphtheritic membrane on these organs causing obstruction, interference with feeding and difficulty in breathing with mortality up to 50%.

Lesions initially start as nodular area with blanched appearance (papule)

It becomes enlarged and yellowish (pustules) terminating into thick, dark scab.

Prevention and control

Disinfection of premises

Sanitation

Two types of vaccines (pigeon pox and fowl pox vaccines) can be used for vaccination.

Pigeon pox vaccine is less pathogenic and can be used on chickens at any stage by wing web method and it produces immunity for 6 months therefore revaccination is required.

Fowl pox vaccine produces solid immunity, usually carried out at 6-8 weeks of age by intramuscular route or wing web method.

Successful or effective vaccination can be judged by takes, where examination of vaccinated birds after 7-10 days of vaccination, show swelling or a scab at the site of puncture or vaccine application.

Absence of takes indicates poor potency of the vaccine, presence of maternal antibodies and improper vaccination.

In such cases, revaccination with new batch/ lot of vaccine should be done.

During disease outbreak, affected birds if less than 30% should be segregated immediately and the remaining birds must be vaccinated at earliest possible.

Standard sanitation and strict biosecurity measures can control the fowl pox.

Disinfection of premises with sodium hydroxide (1:500), cresol (1:400) and phenol (3%) proved beneficial in control of fowl pox.

8. RD

Nature of disease

This is an acute viral disease of poultry characterized by involvement of respiratory system, drop in egg production and mortality as high as 100% in severe cases.

This virus has zoonotic effect and can causes human deaths.

Causes

Paramyxovirus type1 (PMV-1 belongs to the genus Avulavirus, family Paramyxoviridae). Based on the disease produced in chickens, NDVs have been classified into five pathotypes, namely Viscerotropic velogenic (most pathogenic), Neurotropic velogenic, Mesogenic (moderate pathogenic), Lentogenic (low pathogenic) and Asymptomatic.

Various physical (heat, irradiation and pH effects) and chemical compounds (potassium permanganate, formalin, ethanol, etc.) could destroy the virus. Fumigation of buildings and incubators can be used to kill the virus. Infected chickens are the primary source of virus. Virus is shed during incubation, during the clinical stage, and for a varying but limited period during convalescence.

Virus may also be present in eggs laid during clinical disease and in all parts of the carcass during acute virulent infections. Infected birds shed virus in exhaled air, respiratory discharges, and feces, etc. which contaminate feed and water. Infection in birds occurs through inhalation and ingestion of contaminated materials.

Wild and pet birds, movement of people and poultry equipment and even poultry products could aid in spread of infection. The virus has been found to survive for several days on the mucous membrane of the human respiratory tract and has been isolated from sputum.

Clinical symptoms

Twisting of neck and paralysis of wings and legs

Cyanosis of comb

Facial edema

Diarrhoea

Drop in egg production

Sudden death

Gross lesions

Haemorrhage in intestine

Petechial haemorrhage in proventriculus

Congestion and mucoid exudates seen in the respiratory tract, especially in trachea.

Prevention and control

Disease can be prevented effectively by an integrated approach of vaccination, proper management and strict biosecurity. Live virus vaccines both from lentogenic (La Sota, F, B1) and mesogenic (H, R2B, Mukteshwar) strains are used for induction of good immune response. Healthy chicks are vaccinated as early as day 1-4 of life.

Lentogenic strains are administered through ocular (eye) or nasal (nostril) route for primary vaccination of birds. Mesogenic strains are administered through subcutaneous or intramuscular route at 6-8 weeks of age as secondary vaccine for better protection of longer duration.

Killed vaccine with oil adjuvant are used in endemic areas and administered intramuscularly/ subcutaneously to maintain high and prolonged antibody titre in layers and breeders and better maternal antibodies in chicks.