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Anthrax

Anthrax, a highly infectious and fatal disease of cattle, is caused by a relatively large spore-forming rectangular shaped bacterium called *Bacillus anthracis*.

Symptoms:

- Sudden death (often within 2 or 3 hours of being apparently normal) is by far the most common sign;
- Very occasionally some animals may show trembling, a high temperature
- Difficulty breathing, collapse and convulsions before death. This usually occurs over a period of 24 hours;
- After death blood, may not clot, resulting in a small amount of bloody discharge from the nose, mouth and other openings

Treatment and control

- Due to the acute nature of the disease resulting in sudden death, treatment is usually not possible in animals even though Anthrax bacilli are clines. Treatment is of use in cases showing sub-acute form of the disease.
- In most cases, early treatment can cure anthrax. The cutaneous (skin) form of anthrax can be treated with common antibiotics.

Preventive measures:

- Regular annual vaccination of animals in endemic areas will prevent the disease from occurring.
- Vaccination may be carried out at least a month prior to expected disease occurrence in endemic areas.
- Never open a carcass of an animal suspected to have died from anthrax.

Contact a veterinarian immediately if the following symptoms are seen and seek advice on control measures to be adopted.

- Fever (106-108°F), loss of appetite, depression and dullness
- Suspended rumination3. Rapid pulse and heart rates
- Difficult breathing (dyspnoea)
- Lameness in affected leg
- Crepitation swelling over hip, back & shoulder

- Swelling is hot & painful in early stages whereas cold and painless inter.
- Recumbency (prostration) followed by death within 12-48 hrs.

Black quarter (black-leg)

It is an acute infectious and highly fatal, bacterial disease of cattle. Buffaloes, sheep and goats are also affected. Young cattle between 6-24 months of age, in good body condition are mostly affected. It is soil-borne infection which generally occurs during rainy season. In India, the disease is sporadic (1-2 animal) in nature.
Causal organism: it is a bacterial disease caused by *Clostridium chauvoei*
Symptoms:

- Fever (106-108°F), Loss of appetite, Depression and dullness
- Suspended rumination
- Rapid pulse and heart rates
- Difficult breathing (dyspnoea)
- Lameness in affected leg
- Crepitation swelling over hip, back & shoulder
- Swelling is hot & painful in early stages whereas cold and painless inter.
- Recumbency (prostration) followed by death within 12-48 hrs.

Treatment:

- Early treatment can be possible to complete cure of the animal.
- Consult with veterinarian immediately.

Foot and mouth disease

The foot-and-mouth disease is a highly communicable disease affecting cloven-footed animals. It is characterized by fever, formation of vesicles and blisters in the mouth, udder, teats and on the skin between the toes and above the hoofs. In India, the disease is widespread and assumes a position of importance in livestock industry. The disease spreads by direct contact or indirectly through infected water, manure, hay and pastures. It is also conveyed by cattle attendants. It is known to spread through recovered animals, field rats, porcupines and birds.
Symptoms

- fever with 104-105^{o} F
- profuse salivation - ropes of stringy saliva hangs from mouth
- vesicles appear in mouth and in the inter digital space

- lameness observed
- cross bred cattle are highly susceptible to it

Treatment

- the external application of antiseptics contributes to the healing of the ulcers and wards off attacks by flies.
- a common and inexpensive dressing for the lesions in the feet is a mixture of coal-tar and copper sulphate in the proportion of 5:1.

Precautions

- heavy milch animals and exotic breeds of cattle bred for milk should be protected regularly.
- it is advisable to carry out two vaccinations at an interval of six months followed by an annual vaccination programme.
- isolation and segregation of sick animals. It should be informed immediately to the veterinary doctor
- disinfection of animal sheds with bleaching powder or phenol
- attendants and equipment's for sick animals should be ideally separate
- the equipment's should be thoroughly sanitized
- proper disposal of left over feed by the animal
- proper disposal of carcasses
- control of flies

Rabies (Mad dog disease)

Rabies is a disease of dogs, foxes, wolves, hyaenas and in some places, it is a disease of bats which feed on blood.
The disease is passed to other animals or to people if they are bitten by an animal with rabies. The germs which cause rabies live in the saliva of the sick (rabid) animal. This is a killer disease but not every dog which bites is infected with rabies.
When the rabid animal bites another animal or human, the germs which live in its saliva pass into the body through the wound caused by the bite. The germs travel along the nerves to the brain. The time between the bite and the first appearance of signs that the bitten animal or human has been infected can take from 2 to 10 weeks or more. The time taken depends on the distance of the bite from the brain. If the bite is on the face or head, the bitten animal or human will quickly show signs, but if the bite is on the leg it will take much longer for signs to develop.

General signs of rabies

You should first look for the marks of the bite and discover where and when the animal was bitten. All rabid animals show similar signs in the beginning.

- they change their normal behaviour and behave very strangely.
- They stop eating or drinking.
- Male animal will try to mate (mount) other animals.
- there is no change in the body temperature.
- These signs will continue for 3 to 5 days. Then, before it dies, the animal will develop one or the other of two types of the disease:
    - the furious (mad) type of the disease makes the animal aggressive and it will bite anything.
    - The quiet (dumb) type when the animal is quiet and does not move.

Rabies in the dog

Dogs show either of the two types of rabies.

- a dog with the dumb or quiet type of the disease cannot move. It looks as if it has a bone stuck in the mouth and saliva drips from the mouth.
- rabies in the dog lasts about 10 days before the animal dies. If the animal does not die after this length of time then it may not be suffering from rabies.

Rabies in sheep, goats and cattle

Rabies is characterised by the animals becoming restless and excited. They may bite themselves and saliva drips from the mouth. The most important sign in cattle is that the animal bellows (calls) very frequently and with strange sound. The animals will become paralysed and die.

Rabies in the horse and camel

The horse will show the furious (mad) type of the disease. It will kick and bite and show signs similar to colic. The animal will die after paralysis of the back legs.

In the camel the signs of rabies are similar to those shown by an animal in the rut.

What to do with a biting dog

Remember that not every dog which bites has rabies. If the dog belongs to somebody ask the owner about its normal behaviour. If the dog is showing signs of rabies you must inform your veterinary officer immediately. The dog must be shot and if it has bitten anybody, they must be taken to a hospital immediately for vaccination.

Control of rabies

Dogs in your community can be vaccinated against rabies. You should ask your veterinary service about vaccination against rabies. If there is an outbreak of rabies, the livestock in your community can be vaccinated too.

Blue tongue

Bluetongue, a disease which is transmitted by midges, infects domestic and wild ruminants and also camelids, however sheep are particularly badly affected. Cattle, although infected more frequently than sheep, do not always show signs of disease. Virus spreads between animals occurs via the midges of Cullicoides species.
The likelihood of mechanical transmission between herds and flocks, or indeed within a herd or flock, by unhygienic practices (the use of contaminated surgical equipment or hypodermic needles) may be a possibility.
Clinical signs include:
Sheep : eye and nasal discharges, drooling, high body temperature, swelling in mouth, head and neck, lameness and wasting of muscles in hind legs, haemorrages into or under skin, inflammation of the coronary band, respiratory problems, fever, lethargy.
In cattle : nasal discharge, swelling of head and neck, conjunctivitis, swelling inside and ulceration of the mouth, swollen teats, tiredness, saliva drooling, fever.
Note: a blue tongue is rarely a clinical sign of infection
Control :
Inspect stock closely, particularly focusing on the lining of the mouth and nose and the coronary band (where the hoof stops and the skin starts). If an animal is suspected as having bluetongue, it must be reported as quickly as possible. Telephone your local animal health office immediately.

Listeriosis

Transmission : The organisms are excreted in the faeces, urine, aborted foetuses, uterine discharge and milk of infected animals. The organisms are sufficiently resistant to remain viable in animal and human faeces, sewage, soil, silage and dust foe several weeks and months. The blood sucking arthropods may spread infection since organisms have been isolated from cattle ticks and tabanid flies. Under natural conditions certain predisposing factors are related to clinical infection.
Symptoms : In farm animals the disease occurs towards the end of winter or early spring. The first signs of meningo- encephalitis are stiffness of neck, incoordinated movement of limbs and tendency to move in circles or to lean against a fence or

wall. There may be paralysis of muscles of jaw and pharynx. Incoordination becomes progressively more severe until the animal can no longer stand. The cattle which are not severely affected may survive. Abortions in cattle usually occur after 4-8 months of pregnancy and at a comparatively later stage in sheep. In pigs and horses, clinical signs are not common but may develop as encephalitis and septicaemia. In poultry, the disease usually causes sudden death, occasionally there are signs of torticollis, weakness and inco-ordination of the legs.

Mastitis

Mastitis, or inflammation of the mammary gland, is the most common and the most expensive disease of dairy cattle throughout most of the world. Although stress and physical injuries may cause inflammation of the gland, infection by invading bacteria or other microorganisms (fungi, yeasts and possibly viruses) is the primary cause of mastitis. Infections begin when microorganisms penetrate the teat canal and multiply in the mammary gland.

Treatment

- success depends on the nature of the aetiological agent involved, the severity of the disease and the extent of fibrosis.
- complete recovery with freedom from bacterial infection can be obtained in cases of recent infection and in those where fibrosis has taken place only to a small extent.
- such drugs as acriflavine, gramicidin and tyrothricin have now ceased to be in use, and have given place to the more effective drugs, such as sulphonamides, penicillin and streptomycin.

Footrot

Foot rot is a common cause of lameness in cattle and occurs most frequently when cattle on pasture are forced to walk through mud to obtain water and feed. However, it may occur among cattle in paddocks as well, under apparently excellent conditions. Foot rot is caused when a cut or scratch in the skin allows infection to penetrate between the claws or around the top of the hoof. Individual cases should be kept in a dry place and treated promptly with medication as directed by a veterinarian. If the disease becomes a herd problem a foot bath containing a 5% solution of copper sulphate placed where cattle are forced to walk though it once or twice a day will help to reduce the number of new infections. In addition, drain mud holes and cement areas around the water troughs where cattle

are likely to pick up the infection. Keep pens and areas where cattle gather as clean as possible. Proper nutrition regarding protein, minerals and vitamins will maximize hoof health.

Bovine rhinotracheitis

Infectious bovine rhinotracheitis (IBR) is a highly contagious, infectious respiratory disease that is caused by bovine herpesvirus-1 (bhv-1). It can affect young and older cattle. In addition to causing respiratory disease, this virus can cause conjunctivitis, abortions, encephalitis, and generalised systemic infections. Ibr is characterized by acute inflammation of the upper respiratory tract.

Treatment

There is no direct treatment for viral diseases. Infected animals should be isolated from the rest of the herd and treated with anti-inflammatory drugs and antibiotics for secondary infections if necessary. Carrier cattle should be identified and removed from the herd.

Prevention

Control of the disease is based on the use of vaccines.

Piglet diarrhea or scour

Of all the diseases in the sucking piglet, diarrhea is the most common and probably the most important. In some outbreaks, it is responsible for high morbidity and mortality. The main bacterial causes are E. Coli and Clostridia and the main parasite is Coccidia.

Clinical signs

Scour in the piglet can occur at any age during sucking but there are often two peak periods, before 5 days and between 7 and 14 days.

Acute disease

The only sign may be a perfectly good pig found dead. Post-mortem examinations show severe acute enteritis, so sudden that there may be no evidence of scour externally. Clinically affected piglets huddle together shivering or lie in a corner. The skin around the rectum and tail will be wet. Look around the pen for evidence of a watery to salad cream consistency scour. In many cases, there is a distinctive smell. As the diarrhea progresses the piglet becomes dehydrated, with sunken eyes and a thick leathery skin. The scour often sticks to the skin of other piglets giving them an orange to white color.

Prior to death piglets may be found on their sides paddling and frothing at the mouth.

Sub-acute disease:
The symptoms are similar but the effects on the piglet are less dramatic, more prolonged and mortality tends to be lower. This type of scour is often seen between 7 to 14 days of age manifest by a watery to thin salad cream consistency diarrhea, often white to yellow in color.

Management control and prevention

- adopt procedures to prevent the spread of the scour - disinfect boots between pens, use a disposable plastic apron when dosing piglets to prevent heavy contamination of clothing, wash hands after handling a scoured litter, disinfect brushes and shovels between pen.
- ensure that farrowing houses are only used on an all-in all-out basis with a pressure wash and disinfection between each batch.
- farrowing pens must be dry before the house is repopulated. Remember that moisture, warmth, waste food and faeces are ideal for bacterial multiplication.
- pen floors should be well maintained. Poor pen hygiene associated with bad drainage predisposes to scour.
- look carefully at the part of the pen floor where there are piglet faeces. Is this poorly drained? Do large wet patches develop? If so cover them with extra bedding daily and remove. This is a most important aspect of control.
- check nipple drinkers and feeding troughs for leakages.
- ensure that faeces are removed daily from behind the sow from the day she enters the farrowing crates until at least 7 days post-farrowing if the floors are slatted. Also remove faeces daily throughout lactation if they are solid concrete.
- maintain creep environments that are always warm and comfortable. Fluctuating temperatures are a major trigger factor to scour particularly from 7 to 14 days of age.
- assess the environment of all the farrowing house. Poor environments allow heavy bacterial multiplication and a much higher bacterial challenge is likely to break down the colostral immunity.
- check the sow's health. Animals affected with enteric or respiratory disease, lameness or mastitis predispose the litter to scour.
- where farrowing house floors are very poor, pitted and difficult to clean, brush them over with lime wash containing a phenolic disinfectant.
- Colostrum management : it is vital that the piglet receives the maximum amount of colostrum within the first 12 hours of birth. High levels of antibody are only absorbed during this period. Factors such as poor teat

access, poor crate design, and particularly the development of agalactia in the sow, associated with udder oedema, reduce intake.

PPR (goat plague)

PPR (Peste des petits ruminants) is a most important viral disease of goat capable of heavy mortality and commonly called as goat plague.

Etiology

The causative virus was first thought to be an aberrant strain of rinderpest virus that had lost its ability to infect cattle. Later molecular studies showed that it was distinct from, but closely related to, rinderpest virus.

Clinical signs:

The clinical sign of PPR in goats is often fulminating and fatal although apparent infection occurs in endemic areas. Incubation period may range from 2-6 days in field conditions. In acute form, there is sudden onset of fever with rectal temperature of at least 40°- 41°C. The affected goats show dullness, sneezing, serous discharge from the eyes and nostrils. During this stage farmers often think that the animal has developed cold exposure and may attempt to provide protection for cold. In the process goats, may be congregated and accentuate the process of transmission. After 2-3 days, discrete lesions develop in the mouth and extend over the entire oral mucosa, forming diphtheric plaques.

During this stage profound halitosis (foul smell) is easily appreciable and the animal is unable to eat due to sore mouth and swollen lips. Latter ocular discharge becomes mucopurulent and the exudate dries up, matting the eyelids and partially occluding the nostrils. Diarrhea develops 3-4 days after the fever and is profuse and faeces may be mucoid or bloody depending upon the damage. Dyspnea and coughing occur later due to secondary pneumonia. Death occurs within one week of the onset of the illness.

Treatment and control:

No specific treatment is recommended for PPR being viral disease. However, mortality rates can be reduced by the use of drugs that control the bacterial and parasitic complications. Lesions around the eyes, nostrils and mouth should be cleaned twice daily with sterile cotton swab. Immediate isolation of affected goats from clinically healthy goats is most importance measure in controlling the spread of infection. Nutritious soft, moist, palatable diet should be given to the affected goats. Provide parenteral energy infusion in anorectic goats along with appetizers.

Immediately measures should be taken for notification of disease to nearest government veterinary hospital.

Bovine babesiosis (tick fever)

Cause

Bovine babesiosis (bb) is a tick-borne disease of cattle. Transmission of b bovis takes place when engorging adult female ticks pick up the infection. They pass it on to their progeny via their eggs. Larvae (or seed ticks) then pass it on in turn when feeding on another animal. B bigemina is also passed from one generation of ticks to the next. Engorging adult ticks pick up the infection and nymphal and adult stages (not larval stages) of the next generation pass it on to other cattle. Morbidity and mortality vary greatly and are influenced by prevailing treatments employed in an area, previous exposure to a species/strain of parasite, and vaccination status. In endemic areas, cattle become infected at a young age and develop a long-term immunity. However, outbreaks can occur in these endemic areas if exposure to ticks by young animals is interrupted or immuno-naïve cattle are introduced. The introduction of babesia infected ticks into previously tick-free areas may also lead to outbreaks of disease.

Symptoms:

- high fever
- neurologic signs such as incoordination, teeth grinding and mania. Some cattle may be found on the ground with the involuntary movements of the legs. When the nervous symptoms of cerebral babesiosis develop, the outcome is almost always fatal.
- dark colored urine
- anorexia
- animals likely to separate from herd, be weak, depressed and reluctant to move
- n b. Bigemina parasitaemia often exceeds 10 per cent and may be as high as 30 per cent.

Subacute infections, with less apparent clinical signs, are also seen.

Prevention

Effective control of tick fevers has been achieved by a combination of measures directed at both the disease and the tick vector. Tick control by acaracide dipping is widely used in endemic areas. Dipping may be done as frequently as every 4-6 weeks in heavily infested areas. The occurrence of resistance of ticks, chemical residues in cattle and environmental concerns over the continued use of insecticides has led to use of integrated strategies for tick control. Babesiosis vaccines are readily available and are highly effective. Anti-tick vaccines are also

available in some countries and can be used as part of an integrated program for the control of ticks. Babesiosis can be eradicated by eliminating the host tick(s). In the us, this was accomplished by treating all cattle every two to three weeks with acaricides. In countries where eradication is not feasible, tick control can reduce the incidence of disease.

Milk fever

Milk fever, also known as Parturient hypocalcaemia and parturient paresis, is a disease which has assumed considerable importance with the development of heavy milking cows. Decrease in the levels of ionized calcium in tissue fluids is basically the cause of the disease. In all adult cows, there is a fall in serum-calcium level with the onset of lactation at calving. The disease usually occurs in 5 to 10-year-old cows, and is chiefly caused by a sudden decrease in blood-calcium level, generally within 48 hours after calving.

Symptoms

- in classical cases, hypocalcaemia is the cause of clinical symptoms. Hypophosphataemia and variations in the concentration of serum-magnesium may play some subsidiary role.
- the clinical symptoms develop usually in one to three days after calving. They are characterized by loss of appetite, constipation and restlessness, but there is no rise in temperature.

Calf scour

Calves may develop scours due to bacterial or virus infections. Scours is known as "calf scours" or neonatal calf diarrhea. The primary causes of scours include: Rota virus, Corona virus, *Cryptosporidium parvum,* Salmonella and *Escherichia coli*.

- **Determine if treatment is required.** Calves that are moving around in the pasture, with their tails up, probably do not need treatment. Check to see if the diarrhea is yellow or white. If this is the case, treatment is probably not needed.
- **Determine if the calf is looking listless**. Calves that are lethargic or not participating much in the playful activities with other calves are a red flag to pay attention to. Calves that are also losing condition are also cause for alarm.

- **Check to see if the calf is dehydrated.** You can check for dehydration by pulling on the calf's neck skin. If the skin "tents" this is a sign of dehydration.
- **Determine the calf's body temperature.** A normal body temperature ranges from 100.5 °f (38.1 °c) to 102.5 °f (39.2 °c). Anything outside of this range is a sign for treatment.
- **Separate the sick calf or calves from the healthy herd.** You'll want to do this to avoid spreading the disease further.
- **Administer fluids using your veterinarian-approved electrolyte solution.** You may need to inject the fluids via iv or orally.
- Follow appropriate nursing care protocol using your vet's guidelines. This may include providing shelter, feed and a warm place to sleep.
- A drawback from providing shelter is maintaining infectious control. You will have to work extra to get rid of soiled bedding and disinfect everything that a calf will touch, from the floor to the fence panels and even the feed bucket.
- **Enthnovet practice:** *Ingredients needed*: vasambu (*Acorus calamus*) leaves 2 numbers, dried ginger (*Zingiber officinale*) 50 gm, guava (*Psidium guajava*) tender leaves 200 gm. The above materials are ground and made into a bolus and administered orally one or two times.

African Swine Fever

This diseases can affect pigs of any age. High mortality rates. The key clinical signs include blue-purple cyanosis of snout, ears, tail and lower legs; high fever; and heavy discharge from eyes and nose. This disease is notifiable – contact your vet and local authorities if you believe this disease is affecting your herd.

Clinical signs

- High fever 40-42°C.
- Loss of appetite.
- Depression.
- Lethargic- sometimes refusal to stand or move.
- Very unsteady when stood up.
- Vomiting and/or diarrhoea with bloody discharge.
- White skinned pigs: extremities (nose, ears, tail and lower legs) become cyanotic (blue-purple colour).
- Discrete haemorrhages appear in the skin particularly on the ears and flanks.

- Group will huddle together and are usually shivering.
- Abnormal breathing.
- Heavy discharge from eyes and/or nose.
- Comatose state and death within a few days.
- Some pigs can show conjunctivitis with reddening of the conjunctival mucosa and ocular discharges.

Pregnant sows commonly undergo miscarriage or deliver stillborn piglets that are malformed – piglets can be tested for the virus.

Mortality rate in infected groups of pigs is high and there is no vaccination proven to prevent or cure infection, therefore, it is crucial that control begins on-farm. European, South American and Caribbean countries which have been infected have adopted a slaughter policy to eradicate the virus within the herd. Mild strains of the virus also occur which cause a milder but equally serious disease in domestic pig herds – individuals from these herds must also be slaughtered to prevent pathogenesis.

Diagnosis

Pigs that die early in an outbreak may not have any noticeable lesions but as the disease progresses the lesions then are striking. Bright red haemorrhages in the lymph nodes, kidneys, heart and linings of the body cavities are common findings. There may also be excess haemorrhagic fluid in the body cavities and gelatinous fluid in the lungs. The spleen may be enlarged, darkened and crumble on slight pressure.

Cause

African swine fever is caused by the *Asfarviridae* family of viruses which are distinct from the viruses associated with Classical swine fever. There are 22 known types of the ASF virus, allowing the epidemiological tracing of outbreaks to the source.

The infection can be introduced to uninfected herds in a number of ways:

- the feeding of contaminated feed and contaminated food waste used to supplement feed;
- through the bites of soft-bodied ticks, lice and flies;

- through inoculation with contaminated syringes and use of contaminated surgical equipment; and
- through the introduction of infected pigs to the herd.

Transmission of the virus within the herd is generally through direct contact with infected bodily discharges, faeces and vomit.

Prevention

There is no live or attenuated vaccine for the prevention of ASF therefore control of the virus is reliant on strict biosecurity.

- **Do not** feed domestic pigs food waste; this is illegal in the UK, other EU regions and some states within the US
- Where 'permitted garbage feeding' is legal in US states, pigs fed this way are prohibited from exportation.
- **Do not** leave food waste exposed for wild swine species to access. Dispose of food waste properly.
- **Abide** by strict biosecurity rules. Do not take pig meat onto farms, or restrict all food (and consumption of food) to a canteen. All staff on farm should be inducted onto a strict programme of hand and equipment sanitisation before and after contact with pigs.
- **Follow** rules and regulations on disposal of food waste at ferry ports and airports.
- **Provide** the means for staff and visitors to thoroughly sanitise their hands and equipment.
- **Ensure** that wild boar, warthogs and wild pigs, and materials potentially contaminated by such wild species do not come into contact with domestic pigs.
- **Check** infected regions before import of goods that could potentially be contaminated.
- **Advise** and educate people on the risks of bringing back pork products from infected regions.

Treatment

There is no treatment.

All infected animals must be isolated and culled immediately upon confirmation of presence of the virus.

Classical Swine Fever

This diseases can affect pigs of any age. High mortality rates. The key clinical signs include conjunctivitis; high fever; and heavy discharge from eyes and nose. This disease is notifiable – contact your vet and local authorities if you believe this disease is affecting your herd.

Clinical signs

The virus that causes CSF varies in virulence. Some strains are highly virulent and cause acute (i.e. rapid) serious disease. Some strains are of low virulence and cause chronic (i.e. long-lasting) disease, others are intermediate causing sub-acute disease.

Acute disease

- Clinical signs usually appear first in a small number of growing pigs which show non-specific signs of depression, sleepiness, and reluctance to get up or to eat. If you get them up they may wander to the feeder but eat very little or nothing and wander away again to lie down. They walk and stand with their heads down and tails limp. Over the following few days these signs get worse and more pigs become affected.
- Younger piglets may appear chilled, shiver and huddle together.
- Initially affected pigs may appear to be constipated but this generally changes to a yellow-grey diarrhoea as the disease progresses. Early on some of the pigs may develop conjunctivitis (inflammation of the eye surface) with thin discharges. This gets worse, the discharge getting thicker with time until some of the eyelids are completely closed and adhered.
- A constant early sign, which persists throughout the disease until just before death, is a high fever, over 42ºC (107ºF). Check the sick pigs' rectal temperatures. If they are all high suspect CSF.
- As the disease progresses the affected pigs become very thin and weak and develop a staggering walk. Initially this is probably through weakness but later it is due to infection of and damage to the spinal nerves. Partial paralysis of the hind end results in a drunken walk and a tendency to fall to a sitting or lying position. Diarrhoea worsens and some pigs vomit a yellowish bile. The pigs' skins go purple, first over the ears and tail, followed by the

snout, lower legs, belly and back. Affected pigs die in 10-20 days. Some pigs go into convulsions before death.

Sub-acute disease

The early signs in growing pigs are similar but they progress more slowly and are less severe. Affected pigs may be ill for up to 30 days before they die.

Chronic and aberrant disease and persistent infection

The virus can cross the placenta and infect the piglets in the sow's uterus. Sows that have been inadequately vaccinated that become infected, or sows which become infected with a virus of low virulence, may appear normal but give birth to shaking piglets many of which die. (Note: there are also other causes of shaking or trembling piglets).

If the virus crosses the placenta before the piglets' immune systems have developed they may be born apparently healthy although possibly weak and may grow on to be persistent carriers without at first showing clinical signs. They shed virus so they are a menace to other pigs. At several weeks or months of age they may develop typical clinical signs but these are likely to be milder, last longer and without the high temperatures.

Virus that infects the piglets in the uterus may cause other effects, namely, death, mummification, abortion or the birth of weak piglets some of which may be deformed. Vaccination of sows during pregnancy with some of the original attenuated virus vaccines resulted in trans-placental infection of unborn piglets with similar adverse results. The newer attenuated vaccines are claimed to be safer.

Low virulence strains of the virus may also multiply in the reproductive tracts of unvaccinated boars or boars which have been inadequately vaccinated. The vaccine virus itself in some of the older attenuated vaccines was thought to do this, resulting in returns to service and abortions.

Diagnosis

In acute or sub-acute outbreaks a presumptive diagnosis can be made on the typical clinical signs and post-mortem lesions but African swine fever and *Salmonella choleraesuis* infection produce some similar signs and lesions. *Salmonella choleraesuis* is frequently a concurrent pathological infection with CSF virus, triggered off from its latent state by the CSF virus infection.

In chronic or aberrant cases the clinical signs and lesions are less diagnostic and may only raise a suspicion of CSF.

In all suspected cases laboratory tests should be done to confirm the diagnosis. Investigations are usually carried out by the authorities.

Causes

Classical swine fever (CSF), otherwise known as hog cholera (HC) or just swine fever, is a specific viral disease of pigs. It affects no other species. It is a notifiable disease in most countries of the world.

Prevention

Vaccination

- Inactivated vaccines

On-farm precautions

- If you farm in a country where CSF is endemic or where there is a risk of CSF occurring consider routinely vaccinating your herd if vaccination is allowed. This will greatly reduce the possibility of contamination.
- CSF virus does not spread as readily as some other viral infections (e.g. TGE and FMD). Unlike FMD it is not windborne. Thus the conscientious application of simple biosecurity measures should keep it out of the herd.
- If CSF is in your country important precautions include reducing visitors to a minimum, taking precautions against contamination from vehicles, and not allowing pig meat products near any pigs.
- Any replacement pigs coming on to the premises should come from known safe sources and should be quarantined. In some areas the disease has become very mild and spread can go unrecognised.
- Pig buildings should be protected from stray animals, particularly wild pigs and boars.

Treatment

There is no treatment.

Canine Coronavirus

This is an intestinal infection that is highly contagious and affects both wild and domesticated dogs. The virus is limited to the upper two thirds of the small intestines. A lot of the time there may be no symptoms and the infection will clear up by itself. However this infection can be problematic for puppies and dogs that have other infections such as parvovirus.

Symptoms: It is common in older dogs for there to be no symptoms, but when there are symptoms these include vomiting, diarrhea, and dehydration. Treatment will involve administering extra fluids and electrolytes.

If you suspect your puppy may have contracted Coronavirus it is important to get him to the vets as it can be fatal for a puppy.

Canine Distemper

Canine distemper is a very serious viral disease. It can be transmitted through the air from dog to dog. It is highly contagious and there is no known cure, which is why it is very important that you get your dog vaccinated against it.

Symptoms: High temperature, coughing, lethargy, loss of appetite, heavy breathing, red eyes and a runny nose. Diarrhoea and vomiting can also be symptoms.

If you suspect that your dog has contracted canine distemper, contact your vet immediately. Do not take your dog into the vets until you have notified the receptionist, otherwise you may infect other dogs.

Canine Hepatitis

Canine Hepatitis, commonly known as Rubarth Disease is a viral disease that is often mistaken for canine distemper. It can be transmitted via faeces, urine, blood and saliva.

Symptoms: High temperature, coughing, loss of appetite, depression, vomiting, pale gums and abdominal pain. Symptoms of jaundice may occur as this disease infects the liver and kidneys.

If you suspect that your dog has contracted canine hepatitis, contact your vet immediately. Don't let him come into contact with any other dog and clear up his faeces.

Canine Influenza

Canine Influenza is a highly contagious respiratory viral infection that can cause kennel cough. It is easily transmitted between dogs in close proximity to each other.

Symptoms: Coughing, sneezing, nasal discharge, breathing difficulties, variable fever, loss of appetite, and lethargy.

If you think your dog may have influenza contact your vet immediately as failure to treat the virus could lead to secondary complications such as pneumonia.

Canine Parainfluenza

Canine parainfluenza is a contagious respiratory virus that can be transmitted via contact with infected dogs, shared bowls, and beddings. The virus can spread quickly between dogs that are kept in close contact such as dogs in kennels. Your vet will give you antibiotics for you to treat your dog.

Symptoms: A cough, a fever/temperature, nasal discharge, loss of appetite, and a lack of energy.

If you suspect your dog has contracted Canine Parainfluenza contact your vets immediately.

Kennel Cough

Also known as canine tracheobronchitis, kennel cough is a highly contagious and infectious disease. It is transmitted through the air, usually in areas where there is a dense population of dogs such as a kennel or a show. The treatment for kennel

cough is usually administered as a nasal spray, although there is an injection available for dogs who find it extremely uncomfortable.

Symptoms: A dry hacking or honking cough, retching and nasal discharge. Most dogs that contract kennel cough will show a lack of appetite, but some may not. The illness may progress into secondary pneumonia, and include a high temperature, lethargy, and in extreme cases death.

If your dog shows these symptoms contact your vet immediately. Do not take your dog into the vets until you have notified the receptionist, otherwise you may infect other dogs.

Leptospirosis

Leptospirosis is caused by a bacteria and is usually contracted from contaminated water, so try and prevent your dog from drinking out of any puddles, ponds, lakes, reservoirs or canals. The bacteria mainly comes from the urine of rats, pigs and cattle, which makes farms the most likely place for the disease to be transmitted.

Symptoms: High temperature, loss of appetite, vomiting, lethargy, depression, muscle pain, diarrhoea and blood in the urine. Leptospirosis affects the liver and the kidneys so watch out for excessive drinking and urinating, yellow eyes and persistent vomiting.

If you suspect that your dog has contracted leptospirosis, contact your vet immediately.

Parvovirus

Canine parvovirus is a life-threatening and highly contagious viral disease. It can be contracted from infected dog faeces, food and water bowls, clothes and carpets. It is very important that you have your dog vaccinated against parvovirus as it is highly contagious and can live on objects for months.

Symptoms: Loss of appetite, lethargy, persistent vomiting and bloody diarrhoea.

If you notice these symptoms get in contact with your vet immediately. Do not take your dog into the vets until you have notified the receptionist, otherwise you may infect other dogs.

Ranikhet Disease

Ranikhet disease, also known in the West as Newcastle disease is a contagious and highly fatal daises of flows. In spite of the notable work done towards its control, this disease still ranks as one of the most serious virus diseases of poultry. The disease occurs in almost all countries and usually assumes a server form affecting birds of all ages. Mortality in flows varies from 50 to 100 per cent.

Ranikhet disease is largely a disease of flows, but it also effects turkeys, pigenosn, grows, ducks, geese, koel pheasants, guinea-flows, partridges and doves. hedgehogs have been suspected as reservoirs of the disease. The disease is also suspected to cause conjunctivitis among laboratory workers and persons handling infected birds.

Symptoms

The symptoms vary according to the age of the affected birds. The first symptoms usually observed in young birds are sneezing, gasping and often droopiness. It is in this stage of the disease that the manifestations rather closely resemble those of infections bronchitis. Within a short time after appearance of respiratory symptoms, deaths occur in a flock in quick succession and in increasing numbers from day to day.

Among growing birds and in adult sudden deaths occur in a dew instances, and are followed by a number of birds showing respiratory symptoms. The affected birds are full and depressed with ruffled feathers. These symptoms are accompanied by diarrhea, characterize by the passing of a watery stool with an offensive smell. There is profuse salivation. The saliva often accumulates in the mouth and obstructs respiration, which results in the production of gurgling disused birds may be soft - shelled and deformed. In turkeys the disease runs a very mild course. In adults, in particular, it may pass unnoticed except for some dullness, loss of appetite and other minor symptoms.

Treatment and Prevention

At present there is no effective treatment of any value. Proper housing and general good care are indicated in an effort to shorten the duration and severity of the infection.

An early recognition of the disease and application of struck sanitary measures are of great value in the control of the disease. Some important measures for its prevention are ; slaughtering of all apparently ailing birds, segregating of in - contact in group of 10 to 15 each; removal of all infective materials such as droppings, residues of poultry cleanliness ; and provision of separate attendants for each group of birds.

The poultry farm should be at a distance from place of traffic. All newly purchased birds should be kept in segregation from not less than 10 days before taking them into the farm. The poultry runs should be ploughed from time to time and lime applied thereon as a general disinfectant. As far as possible the pens and runs should be made inaccessible to free - flying birds by providing a barrier of wire - netting.

Control

Control of Ranikhet diseases can be effect with judicious application of sanitary and vaccination measures.

IBD- Infectious Bursal Disease

Introduction

A viral disease, seen worldwide, which targets the bursal component of the immune system of chickens. In addition to the direct economic effects of the clinical disease, the damage caused to the immune system interacts with other pathogens to cause significant effects. The age up to which infection can cause serious immunosuppression varies between 14 and 28 days according to the antigen in question. Generally speaking the earlier the damage occurs the more severe the effects.

The infective agent is a Birnavirus (Birnaviridae), Sero-type 1 only, first identified in the USA in 1962. (Turkeys and ducks show infection only, especially with sero-type 2).

Morbidity is high with a mortality usually 0- 20% but sometimes up to 60%. Signs are most pronounced in birds of 4-6 weeks and White Leghorns are more susceptible than broilers and brown-egg layers.

The route of infection is usually oral, but may be via the conjunctiva or respiratory tract, with an incubation period of 2-3 days. The disease is highly contagious. Mealworms and litter mites may harbour the virus for 8 weeks, and affected birds excrete large amounts of virus for about 2 weeks post infection. There is no vertical transmission.

The virus is very resistant, persisting for months in houses, faeces etc. Subclinical infection in young chicks results in: deficient immunological response to Newcastle disease, Marek's disease and Infectious Bronchitis; susceptibility to Inclusion Body Hepatitis and gangrenous dermatitis and increased susceptibility to CRD.

Signs

- Depression.
- Inappetance.
- Unsteady gait.
- Huddling under equipment.
- Vent pecking.
- Diarrhoea with urates in mucus.

Post-mortem lesions

- Oedematous bursa (may be slightly enlarged, normal size or reduced in size depending on the stage), may have haemorrhages, rapidly proceeds to atrophy.
- Haemorrhages in skeletal muscle (especially on thighs).
- Dehydration.
- Swollen kidneys with urates.

Diagnosis

Clinical disease - History, lesions, histopathology.
Subclinical disease - A history of chicks with very low levels of maternal antibody (Fewer than 80% positive in the immunodifusion test at day old, Elisa vaccination date prediction < 7 days), subsequent diagnosis of 'immunosuppression diseases'

(especially inclusion body hepatitis and gangrenous dermatitis) is highly suggestive. This may be confirmed by demonstrating severe atrophy of the bursa, especially if present prior to 20 days of age.

The normal weight of the bursa in broilers is about 0.3% of bodyweight, weights below 0.1% are highly suggestive. Other possible causes of early immunosuppression are severe mycotoxicosis and managment problems leading to severe stress.

Variants: There have been serious problems with early Gumboro disease in chicks with maternal immunity, especially in the Delmarva Peninsula in the USA. IBD viruses have been isolated and shown to have significant but not complete cross-protection. They are all sero-type 1. Serology: antibodies can be detected as early as 4-7 days after infection and these last for life. Tests used are mainly Elisa, (previously SN and DID). Half-life of maternally derived antibodies is 3.5- 4 days. Vaccination date prediction uses sera taken at day old and a mathematical formula to estimate the age when a target titre appropriate to vaccination will occur.

Differentiate clinical disease from: Infectious bronchitis (renal); Cryptosporidiosis of the bursa (rare); Coccidiosis; Haemorrhagic syndrome.

Treatment

No specific treatment is available. Use of a multivitamin supplement and facilitating access to water may help. Antibiotic medication may be indicated if secondary bacterial infection occurs.

Prevention

Vaccination, including passive protection via breeders, vaccination of progeny depending on virulence and age of challenge. In most countries breeders are immunised with a live vaccine at 6-8 weeks of age and then re-vaccinated with an oil-based inactivated vaccine at 18 weeks. A strong immunity follows field challenge. Immunity after a live vaccine can be poor if maternal antibody was still high at the time of vaccination.

When outbreaks do occur, biosecurity measures may be helpful in limiting the spread between sites, and tracing of contacts may indicate sites on which a more rebust vaccination programme is indicated.

MAREKS DISEASE

Introduction

Marek's disease is a Herpes virus infection of chickens, and rarely turkeys in close association with chickens, seen worldwide. From the 1980s and 1990s highly virulent strains have become a problem in North America and Europe.

The disease has various manifestations: a) Neurological - Acute infiltration of the CNS and nerves resulting in 'floppy broiler syndrome' and transient paralysis, as well as more long-standing paralysis of legs or wings and eye lesions; b) Visceral - Tumours in heart, ovary, tests, muscles, lungs; c) Cutaneous - Tumours of feather follicles.

Morbidity is 10-50% and mortality up to 100%. Mortality in an affected flock typically continues at a moderate or high rate for quite a few weeks. In 'late' Marek's the mortality can extend to 40 weeks of age. Affected birds are more susceptible to other diseases, both parasitic and bacterial.

The route of infection is usually respiratory and the disease is highly contagious being spread by infective feather-follicle dander, fomites, etc. Infected birds remain viraemic for life. Vertical transmission is not considered to be important.

The virus survives at ambient temperature for a long time (65 weeks) when cell associated and is resistant to some disinfectants (quaternary ammonium and phenol). It is inactivated rapidly when frozen and thawed.

Signs

- Paralysis of legs, wings and neck.
- Loss of weight.
- Grey iris or irregular pupil.
- Vision impairment.
- Skin around feather follicles raised and roughened.

Post-mortem lesions

- Grey-white foci of neoplastic tissue in liver, spleen, kidney, lung, gonads, heart, and skeletal muscle.
- Thickening of nerve trunks and loss of striation.

- Microscopically - lymphoid infiltration is polymorphic.

Diagnosis

History, clinical signs, distribution of lesions, age affected, histopathology. Differentiate from Lymphoid leukosis, botulism, deficiency of thiamine, deficiency of Ca/Phosphorus/Vitamin D, especially at the start of lay.

Treatment

None.

Prevention

Hygiene, all-in/all-out production, resistant strains, vaccination generally with 1500 PFU of HVT at day old (but increasingly by in-ovo application at transfer), association with other strains (SB1 Sero-type 2) and Rispen's.

It is common practice to use combinations of the different vaccine types in an effort to broaden the protection achieved. Genetics can help by increasing the frequency of the B21 gene that confers increased resistance to Marek's disease challenge.

Latest information

In 2018, Aviagen published their comprehensive brief "Marek's disease control in broilers birds" on The Poultry Site. It includes details of transmission, diagnosis, vaccinations, vaccine administration and causes of the disease.

FOWL POX

Cause

Avian pox, caused by a poxvirus, is one of the oldest known infectious disease of birds. There are many different strains of the avian pox virus but most are species specific. However, some strains have the ability to infect birds across different families.

Significance

While avian pox infections in wild birds are often mild and self-limiting, some outbreaks cause high mortality rates. Birds native to isolated islands are particularly susceptible to this disease. In Hawaii, avian pox has negatively impacted many species of native birds. Avian pox can lead to significant losses when many captive birds are housed in close quarters. The virus can also spread between wild and captive birds (see Transmission).

Species Affected

Most bird species are believed to be susceptible to avian pox. About 230 species of wild and domestic birds have been reported with this disease; turkeys, finches, and doves are the most commonly affected wild bird species in New England. Upland game birds (such as mourning doves and wild turkeys), songbirds, and raptors can experience varying degrees of mortality due to avian pox. Wild waterfowl in North America have recently experienced an apparent increase in infection rate, though infections in these birds are still relatively uncommon. Domestic birds can also become infected with this virus but wild birds are not thought to be a major source of infection for domestic birds. Avian poxviruses are not known to infect humans.

Distribution

Avian pox is distributed worldwide except Antarctica, the Arctic, and some other remote regions. Infection rates appear to be greater in areas with temperate and warmer climates. The virus has been introduced to several remote islands such as Hawaii, the Galapagos, and the Canary Islands where it spreads rapidly among the native bird populations. Poxvirus outbreaks are common in aviaries, rehabilitation centers, and whenever captive birds are housed closely together.

Transmission

Biting insects such as mosquitoes, mites, fleas, midges, and flies are most frequently responsible for transmitting avian pox. These insects pick up virus particles when they take a blood meal from an infected bird and then transmit the virus when they do the same with another susceptible bird. Transmission rates are

highest when biting insects are abundant. The virus can also be acquired via direct contact with infected birds or contact with contaminated objects such as bird feeders. Avian poxvirus is highly resistant to drying and can remain infectious for months to years in dried scabs and on contaminated surfaces. The virus particles can become aerosolized and infect susceptible birds via inhalation, though this is rare. Birds can become infected by ingesting contaminated food or water. The virus can also enter the body through skin abrasions. Transmission rates are also influenced by bird population density, and higher infection rates occur when bird densities are greatest.

Clinical Signs

Avian pox develops slowly in affected birds. The most common clinical sign of avian pox is the formation of wart-like growths on the skin, particularly on unfeathered parts of the body such as the legs, feet, eyelids, base of the beak, and the comb and wattles. This is the cutaneous or “dry” form of avian pox. Birds with mild infections may only have a few growths that minimally affect their health status. These lesions can persist from 1 to 4 weeks but in most cases, the bird will survive and the nodules will heal potentially with some scarring. At times, nodules on the eyes can interfere with the bird’s ability to see food or predators and can lead to death. Mortality can also result from secondary bacterial infections that are common with this disease.

In more severe cases, birds may develop yellow-white, cheese-like lesions in their mouths and upper respiratory tract (this is known as the diphtheritic or “wet” form of the disease) which can lead to decreased food intake, difficulty breathing, and death.

Diagnosis

A presumptive diagnosis can be made based on clinical signs but laboratory tests (microscopic examination, virus isolation, serology, or PCR) must be conducted to confirm diagnosis.

Treatment

There is no known treatment for avian pox in wild birds. In captive birds, a variety of treatments have been used along with supportive cate to treat the pox lesions and prevent secondary infections. These methods will not eliminate the virus, and the disease will run its course with or without treatment.