Diseases in Pig

1– Porcine Teschovirus II

Pathogenesis

Natural infection is by ingestion and initial viral replication occurs in the tonsils and intestinal tract, especially the large intestine and the ileum. Virulent strains then establish a viraemia which leads to infection of the central nervous system and pregnancies.

Clinical signs

Most teschovirus infections are subclinical but different clinical syndromes have been associated with different viral strains.

The most severe polioencephalomyelitis is encountered in Teschen disease and this affects pigs of all ages. The early signs of Teschen disease include fever, anorexia and listlessness followed by ataxia. In severe cases nystagmus, convulsions, opisthotonus and death occurs within a few days.

When paralysis ensues the pig may sit like a dog or remain in lateral recumbency. Loud noises may trigger off rapid uncoordinated limb movements or lateral recumbency.

Sometimes milder teschovirus strains may induce a milder polioencephalomyelitis with low morbidity and mortality mainly in young pigs and this form can progress into a complete paralysis.

The term SMEDI was introduced to describe a range of reproductive conditions (Stillbirths, Mummies, Embryonic Deaths and Infertility) that were subsequently shown to be caused by teschovirus. However, it is now known that other viruses, such as parvovirus, might be involved.

Porcine teschoviruses have also been isolated from the male reproductive tract.

The role of porcine teschovirus in scour is uncertain because this virus can also be isolated from healthy animals.

The role of porcine teschoviruses as respiratory pathogens is also unclear although experimentally some strains of the virus have produced a pericarditis or pneumonic lesions.

Lesions

No specific changes have been associated with intestinal infections. No gross lesions are seen in encephalomyelitis although histological lesions are seen throughout the central nervous system.

Diagnosis

Teschen disease is designated as a notifiable disease by the OIE who detail the diagnostic protocols to be followed. Clinical signs coupled to an encephalomyelitis is suggestive of the disease but final diagnosis requires viral isolation from the central nervous system.

2 – Influenza I

Introduction

The first reports of influenza in swine occurred in Europe and the USA at the time of the human flu pandemic in 1918. The early H1N1 swine influenza viruses were the progenitors of the H1N1 lineage of swine influenza viruses. The early swine viruses and the 1918 humanisolates were closely related and both had an avian origin.

Influenza viruses are a major cause of respiratory disease in pigs and subclinical infections are often seen. The epidemiology of influenza viruses in swine can best be described a complex interplay of influenza viruses of human, porcine and avian origins.

Pigs play an important role as intermediate hosts in the reassortment and/or adaptation processes that produce new influenza viruses for man. This reassortment process also produces new swine viruses and these periodically change the epidemiology of swine influenza in different parts of the world. For example, in 2009 the then new H1N1 virus arose from a reassortment of European and North American lineages of the virus.

The Virus

Influenza viruses are members of the Orthomyxoviridae and reassortment occurs when two viral types infect the same host and exchange genetic RNA.

All swine influenza viruses belong to influenza A subtype viruses and these are sub-defined by the nature of their haemagglutinin (H) and neuraminidase (N) spike like glycoproteins on their surfaces. Currently, there are 16 known haemagglutinins and nine known neuraminidases and these define virus subtypes, for example, H1N1 and H3N2.

Public health

Animals, including pigs, are a potential source of human influenza viruses and the first proven transfer from pigs to farm workers occurred in 1976. Since then pig to human infections with influenza viruses have been reported around the world and serological studies show such infections to occur regularly.

For a pandemic of swine origin to occur in man the influenza virus must be sufficiently unique to be able to evade existing host immunity in man and it must be able to spread from person to person.

3 – Influenza II

Epidemiology

As well as infecting pigs and man, swine influenza viruses can infect wild boar, turkeys and, sometimes, wild waterfowl.Pigs can be infected by a wide range of influenza subtypes. For example, in recent years H1N1, H3N2, H3N3, H5N1, H9N2 and H4N6 have been isolated from pigs in Asia or Canada. In general terms avian influenza viruses find pigs to be dead end hosts. For an avian influenza virus to epidemiologically progress mutate or reassort with swine adapted influenza viruses to replicate efficiently in pigs.

Human influenza viruses have occasionally been isolated from pigs and transmission of human viruses between pigs requires them to adapt to their new host.

Antigenic drift is slower in porcine influenza virus than it is in human ones. For example, the current European swine H3N2 is related to the human virus of the 1970s and 1980s while the human form has significantly drifted away from it precursors.

Transmission

In the northern hemisphere outbreaks of porcine influenza often occurred in the autumn but as more and more pigs were housed this was less apparent.

Influenza viruses often are introduced into new herds with new stock and primary spreadis pig to pig by nasopharyngeal exposure and there can be over 10 million virus particles in a ml of nasal secretion. Aerosol transmission has been demonstrated for human and equine influenza viruses and is a likely route of transmission in highly populated pig areas.

In farrow to finish farms where there is a steady supply of young susceptible pigletswith waning maternal immunity the influenza virus seems to persistently cycle on the farm.

International geography

North America – Classical H1N1 dominated from 1930s to 1990s. Dramatic change in 1998 with emergence of two different strains of H3N2 of which one – a triple reassortant - spread widely across North America and evolved into four distinct phylogenetic clades.

Europe – In 1979 an H1N1 moved into pigs from wild ducks and has persisted there until today. European swine H3N2 viruses are derived from descendants of the 1968 ‘Hong Kong flu’. In the mid-1990s H1N2 became established.

Asia – More complicated as H3N2 influenza viruses have been regularly transmitted from man to pigs. Both North American and European influenza viruses have been introduced including H1N1. Big variations in dominant viral type between different parts of Asia. In the last 10 years H1N1, H3N2 and H9N2 have regularly spread from other species to pigs.

Pandemic – Following the 2009 H1N1 pandemic in man H1N1 virus was isolated from pigs in many parts of the world.

4 – Influenza III

The disease process

Viral replication is confined to the epithelial cells of the upper and lower respiratory tracts and viral excretion and transmission are by the respiratory route. In infected pigs virus can be isolated from the tissues of the respiratory tract as well as associated lymph nodes and the tonsils – usually from day one but for no longer than a week. The influenza virus prefers the lower respiratory tract in pigs.

Swine influenza virus rarely spreads from the respiratory tract although low amounts of virus can occasionally be found in the brain.

The actual cytokines produced by the influenza virus determine whether the disease is subclinical or clinical.

Clinical signs

The incubation period is 1-3 days. Typically infected pigs have high temperatures (40.5-41.5°C), inactivity, anorexia, huddling and a reluctance to stand up. Dyspnoea and laboured abdominal breathing are usually seen. Morbidity is up to 100%, but mortality is low if the infection is not complicated by secondary infections. Acute outbreaks of clinical respiratory disease are usually only seen in fully susceptible, seronegative stock.

Secondary pathogens such as Actinobacilluspleuropneumoniae, Pasteurellamultocida, Mycoplasma hyopneumoniae, Haemophilusparasuis and Streptococcus suis type 2 can become established as secondary infections and worsen the clinical picture. Other porcine viruses fre- quently infect pigs at a similar time to influenza infection and these include PRRSV and PRCV.

Following influenza infection reduced reproductive performance is sometimes seen, characterised by infertility, abortion, small weak litters and stillbirths.

Lesions

In uncomplicated infection the lesions are those of a viral pneumonia, which is often confined to the apical and cardiac lobes of the lungs. Sometimes interlobular oedema is present and the airways may be filled with blood tinged fibrinous exudates.

Lung lesions can also be very mild and unremarkable.

5 – Vesicular stomatitis

Introduction

Vesicular stomatitis occurs in cattle and pigs and resembles foot and mouth disease. For that reason this disease is important and it is classified by OIE as a notifiable disease. Reports on vesicular stomatitis first appeared in the 1800s but the virus was not isolated until 1925. Closely related viruses – Cocal virus and Alagoas virus – have caused livestock disease in South America.

Epidemiology

Vesicular stomatitis affects swine, cattle and horses and antibodies to this virus have been found in a wide variety of animals. In the Americas, vesicular stomatitis often occurred in cycles until the late 1970s, since when the virus has only been found in wildlife.

This disease can be transmitted by animal to animal contact and physically or biologically by insects. In affected animals the virus is localised to areas with lesions where the virus can remain active for up to 10 days post infection. It can also remain viable in saliva for up to four days.

The disease

Fever occurs for up to three days post infection and vesicle formation occurs on the lining of the mouth, the snout, teats and the coronary bands of the hooves 1-3 days post infection. These rupture 1-2 days later. Epidermal erosion and ulceration is followed by scabbing. Lesions are most common in the mouth and result in anorexia and weight loss. The foot lesions produce lameness. Morbidity can be high but mortality is low.

Diagnosis

Clinically this disease is indistinguishable from foot and mouth, swine vesicular disease or vesicular exanthema so samples must be taken for laboratory diagnosis. Differential diagnosis should also include porcine parvovirus, enterovirus, swine pox, trauma, chemical burns and photosensitisation.

Diagnosis can be made by testing paired sera when a fourfold increase in antibody titre is regarded as being diagnostic.

Prevention and control

When found in pigs immediate steps should be taken to stop the disease spreading. Treatment is largely ineffective. All areas occupied by infected animals should be thoroughly disinfected.

6 – Sarcoptic mange I

Introduction

Globally, sarcoptic mange or scabies is one of the most important ectoparasitic infestations of swine and its significance is often underestimated. This condition depresses performance in fatteners and decreases fertility in breeding sows.

Two forms are known – a chronic form, which is mainly seen in sows, and a pruritic (itchy) hypersensitive form, which is normally seen in growing pigs. Historically, sarcoptic mange was seen in up to 90% of herds but modern production systems and commercial breeders who have eliminated the condition from their herds have reduced this figure dramatically.

Aetiology

Sarcoptic mange is caused by the mite, Sarcoptesscabiei, which is a mite that burrows into the skin of the animal. Sarcoptesscabiei is a permanent skin parasite and its eggs, larvae, nymphs and adults grow and develop there. Adult mites mate on the skin's surface and then the females burrow through the skin laying a string of eggs behind them. After about a month these females die in the burrows.

After a few days the larvae hatch, moult into nymphs and then adults within the burrows in the skin. The adults then go to the surface and the next life cycle commences, each one of which lasts for 10-25 days.

In pigs up to 60kg bodyweight growth rate can be depressed by 4.5-12.0% and in breeding sows feed intake can be adversely affected.

Epidemiology

Sows usually hold the main reservoir of mites on the farm and mites are passed from animal to animal when these come into contact. Prior to the advent of AI boars played a key role in the transmission of mites between animals. Transmission from animal to animal is quite slow. Practices such as group housing of sows, continuous flow systems for growers and large groups of growing pigs favors the spread of mange.

Typically, hyperkeratosis ear lesions characterize infestation of adult pigs although lesions across the whole body can occur. Pigs can become infested with the mite if they are put into a pen immediately after infested pigs are removed from it.

Once mites leave the host they can survive for some time but their infest ability declines quickly. Infest ability for mites in straw are lost within 2-3 days, whereas survivability can be up to two weeks.

7 – Sarcoptic mange II

Clinical signs

Pruritus or itching is the most common clinical sign of sarcoptic mange or scabies and this occurs from 2-11 weeks after infestation. This has several phases starting with a non-responsive phase, followed in sequence by a delayed type hypersensitivity, hypersensitivity and a final immediate phase.
After infestation encrusted lesions develop which are rich in mites, especially on the inner surface of the ears. These can eventually cover three quarters of the ear's surface. Red papules associated with the hypersensitivity stage occur on the rump, flank and abdomen.
Hyperkeratotic mange is the most common form in adults and its lesions have been described as thick and asbestos like. Hyperkeratotic mange is often considered a disease of poor management and nutrition.

Diagnosis

Sarcoptic mange should be considered when growing pigs with red papules on their body are rubbing. Encrustations can be examined for mites.
Specific antibodies can be detected some 5-7 weeks after infestation. Herd testing is more sensitive than the testing of individual animals.

Differential diagnosis

It is essential to differentiate from other skin conditions such as parakeratosis, exudative epidermitis, dermatomycosis, niacin or biotin deficiencies, swinepox, insect bites and photosensitisation.

Treament

A key prerequisite to treatment is recognising the condition is present in the herd. Treatment is based on the correct application of acaricides, most of which work against sarcoptic mange providing they are successfully applied. The avermectins can be administered by injection or, in the case of ivermectin, in the feed.
Elimination of sarcoptic mange from a herd is centred around three key facts – piglets are born free of mites, the mites are highly host specific and have a poor survival away from the pig and modern acaricides are very effective. Herds that are free from mange can be established by hysterectomy or Caesarean derived pigs, depopulation and repopulation from mange free sources and by the use of modern acaricides. In the case of open herds incoming pigs must be closely scrutinised for mange.

Diseases In Poultry

1 – Staphylococcosis I

Introduction

Staphylococcal infections are relatively common in poultry and are usually caused by Staphylococcus aureus. Infections are found in joints, bones, tendons (especially of the lower leg) as well as in the skin, bursae, yolk sac, heart sac, liver and lungs. A staphylococcal septicaemia is frequently seen in layers and broilers where it causes sudden deaths.
In turkeys, Staphylococcus aureus is often isolated from birds condemned for 'green liver-osteomyelitis complex'.
MRSAs (Methicillin Resistant Staphylococcus Aureus) are of increasing interest in the human health field but the role of poultry in this problem is probably insignificant.

Epidemiology

This organism is found wherever poultry are reared. Staphylococci are widespread in nature and commonly found on the skin, in hatcheries, on farms and in processing plants. Some strains, but not all, of Staphylococcus aureus have the ability to cause disease in poultry following their entry through the skin following damage, for example, from beak trimming or vaccination by injection or mucous membranes or the open navel of young chicks.
If birds are immunosuppressed, for example by chicken anaemia agent, fatal septicaemias and/or gangrenous dermatitis can occur.

Clinical signs

The incubation period is short (2-3 days).
Early clinical signs include ruffled feathers, a reluctance to move and fever. These are followed by signs that reflect the part(s) of the bird's body where infection has become localised, such as swollen joints or sternal bursae.
Morbidity and mortality are usually low. Lameness from hock synovitis can result from birds not getting to feeders and becoming debilitated and dying.

Pathology

Gross lesions of osteomyelitis cause bones to become fragile and break and a good example of this is the condition known as femoral head necrosis. Arthritis and/or synovitis are commonly seen, for example in hock synovitis. Sometimes lesions are encountered in the spine.
In most soft tissue organs, such as liver, spleen and kidneys, lesions are characterised by congestion and tissue necrosis.
A mild gangrenous dermatitis of the wing tips (blue wing disease) is sometimes encountered.
Staphylococcal infections are often associated with foot abscesses (bumble foot) in older birds.
At processing staphylococcal infection is often associated with green livers or, occasionally, granulomata.

2 – Staphylococcosis II

Immunity

Immunity (active or passive) is not effective in countering or controlling Staphylococcus aureus infections in poultry.

Diagnosis

As similar lesions can be caused by several bacteria, diagnosis is based on isolating the causative bacterium, Staphylococcus aureus.

Differential diagnosis

Differential diagnosis needs to consider infections caused by bacteria such as E. coli, Pasteurella multocida and Salmonella gallinarum as well as by Mycoplasma synoviae and reoviruses.

Blood testing

Serological testing is not normally done for staphylococcal infections in poultry.

Treatment

Staphylococcus aureus infections can respond to medication but it is always sensible to undertake an antibiogram. Various drugs have been used with success but in many parts of the world amoxycillin is now often the preferred choice.
Management should look at what can be done to reduce the risk of damaging the skin which provides a route of entry for infection – for example using wood shavings that are splinter free and ensuring that there are galvanised spikes on male feeder exclusion grills on breeder farms.

As staphylococcosis often follows early Gumboro disease and chicken anaemia infections prevention of these by vaccination should be undertaken.
Prevention of staphylococcal infection in young chicks is all about hatching chicks with healed navels.

Vaccination

Use of live or dead Staphylococcus aureus vaccines does not appear to provide adequate protection.

3 – Hexamitiasis

Introduction

Hexamitiasis or infectious catarrhal enteritis of turkey poults is caused by a protozoan parasite that was known as Hexamita, but which is today known as Spironucleus meleagridis. The disease has been seen in several countries and S. meleagridis has also been found in pheasants, quail, partridge and peafowl.

The parasite

S. meleagridis is a flagellated protozoon and its eight prominent flagella include four anterior, two anterolateral and two posterior ones.

Clinical signs and pathology

Poults affected by hexamitiasis show no specific clinical signs but they succumb to a watery diarrhoea which can become yellowish in the later stages of the disease. General clinical signs include nervousness, listlessness and convulsions and coma late on in the disease.
Lesions include a catarrhal enteritis that is accompanied by atony which results in dilation of the intestine, especially in its upper reaches. Large numbers of S. meleagridis can be seen in the watery intestinal contents and the intestinal crypts. This parasite dies off quickly after bird death so it is best to look for it in birds which were in extremis and were euthanised immediately before sampling.
Diagnosis is made on the basis of a watery diarrhoea and the demonstration of S. meleagridis in the duodenal contents. However, it should be noted that survivors can become carriers and so the parasite can be encountered in apparently healthy birds.

Treatment

There is no vaccine. Treatment can be undertaken with chlortetracycline or butynorate. This should be supported by good hygiene, management and nutrition.

4 – Tuberculosis I

Introduction

In poultry tuberculosis, which is sometimes called mycobacteriosis and often shortened to just TB or avian TB, is caused by Mycobacterium avium. Avian tuberculosis is a chronic, contagious disease that persists in a flock.
In many countries avian tuberculosis is rarely seen in large commercial flocks but it is seen from time to time in backyard flocks. In some countries it is re-emerging in free range flocks.
Avian tuberculosis can occur in man and so this disease has a public health significance.

History

This disease was first described in chickens in the 1880s. This is a disease which declined with the housing and integration of poultry. The disease is an important one in wild bird collections (probably due in part to the age of many birds in such collections).

Clinical signs

Unthriftiness, depressed egg production and death are the commonly seen signs. Occasionally comb and wattles have a bluish colour. Signs of jaundice are seen when there is severe liver involvement.
Once the bird has become emaciated it is usually possible to feel nodular masses in the abdominal cavity along the intestines. Often there is a unilateral lameness due to a tuberculous arthritis.

Pathology

Tubercular lesions are commonly seen in the liver, spleen, intestines and bone marrow (in all granuloma formation is usually seen) and less frequently in heart, ovaries, testes and skin.
Livers and spleens are enlarged and hence more prone to rupture, which usually results in a fatal haemorrhage.
Ingestion of M. avium results in intestinal infection with eventual spread into the blood system.
Transmission of M. avium around the body via the blood results in lesions in a variety of organs.

5 – Botulism I

Introduction

Botulism is a 'poisoning' caused by the exotoxins of Clostridium botulinum. This disease has also been known as western duck sickness or limberneck. Most cases of avian botulism are caused by C. botulinum type C and a variety of avian species can be affected. The public health significance of avian botulism is minimal.

History

Botulism was first reported in chickens in 1917 following the ingestion of canned vegetables by chickens. Western duck disease was first seen in the USA in the early 1900s, but only later found to be caused by C. botulinum type C.

Distribution

This disease has affected waterfowl and poultry around the world. Originally thought to be a problem in free range poultry the problem has subsequently been seen in intensively reared poultry.

Aetiology

C. botulinum is a Gram positive, spore forming bacterium that can produce potent exotoxins. Although there are eight toxigenic subgroups, avian botulism is primarily caused by C. botulinum type C. Botulism toxins are amongst the most potent toxins known. Type C toxin is produced under anaerobic conditions and temperatures between 10-47°C.

The disease

In one study of 27 outbreaks of botulism in chickens birds aged from 2-8 weeks of age were affected. Older broilers are relatively resistant to botulism C1 toxin. Morbidity and mortality are high and high levels of toxin induce the disease within hours – with low doses the disease takes a day or two to appear. In chickens a flaccid paralysis progresses from the head to the legs. Initially birds are reluctant to move, if they are encouraged to walk they appear to be lame and their wings droop. Limberneck precisely describes the neck paralysis which is seen. Paralysis of the eyelids makes birds appear to be comatose or dead. Gasping is sometimes seen and death is from cardiac and respiratory failure.

Transmission

C. botulinum type C has a worldwide distribution and is found wherever there are large populations of waterfowl or poultry. The presence of C. botulinum type C in the intestinal tracts of wild and domestic birds and the production of resistant spores favours the spread of this bacterium.

6 – Enterococcus I

History

The advent of new microbiological identification technologies resulted in most streptococci, which had previously been called Lancefield group D streptococci, being reclassified as Enterococcus. Between the 1940s and 1970s there were various reports of faecal streptococci infections in poultry which, with the wisdom of hindsight, were probably enterococcal infections.

Aetiology

Enterococci are Gram positive spherical bacteria which, when viewed under the microscope, can be seen as single, paired or short chained spheres (cocci). 'Entero' means 'of the gut'.

There are various Enterococcus Spp that have been found in birds associated with disease including E. faecalis, E. faecium, E. durans, E. avium, E. hirae and E. cecorum.

E. faecalis, E. faecium, E. durans and E. hirae have all been associated with bacterial endocarditis. E. faecalis is the enterococcus most frequently associated with infections in poultry.

Epidemiology

E. faecalis affects poultry species of all ages and contaminated hatching eggs result in infected embryos and day old chicks. E. faecium can cause disease in ducklings.

Enterococci typically infect birds via the oral and aerosol routes; the latter often causing a severe septicaemia after experimental infection. In cage layers infection via skin wounds is seen. Concurrent enteritis often allows enterococci into the bird's body with septicaemia and/or endocarditis ensuing.

Incubation periods range from days to weeks.

Enterococcus Spp have been associated with brain necrosis and encephalomalacia in young chicks.

Clinical signs

There are two forms of enterococcal disease – acute and subacute, which often becomes chronic. In the former the signs are those of a septicaemia, namely depression, lethargy, pale

combs and wattles, diarrhoea, fine head tremors and loss of egg production. In the latter depression, loss of weight, lameness and head tremors may be seen.
Enterococcal egg transmission or faecal contamination of hatching eggs can result in late embryonic mortality and chicks failing to pip.

7 – Enterococcus II

Pathology

Gross lesions in the acute or septicaemic form of the disease include hepatomegaly, splenomegaly, enlarged kidneys and subcutaneous congestion. Omphalitis and yolk sac infection are seen in young chicks. Lesions of the chronic form of enterococcal disease include fibrinous perihepatitis and pericarditis, valvular endocarditis and necrotic myocarditis. Additional lesions arising from the valvular endocarditis include enlarged, pale, flaccid hearts, haemorrhagic areas in the myocardium and infarcts in the liver, spleen or heart.

Diagnosis

Diagnosis is based on post mortem findings and isolating the causal agent.
Differential diagnosis is basically the elimination of other bacterial septicaemias, such as colisepticaemia, pasteurellosis, staphylococcosis and erysipelas.

Treatment

Treatment can involve medication with antibiotics such as penicillin, the tetracyclines, erythromycin and novobiocin. Birds caught early in the disease usually respond well to medication but as the disease progresses the efficacy of treatment declines.
Environmental factors, feeding programmes, stress and housing, as well as genotype, all influence the pathogenesis of enterococcal infections.
There is no treatment for enterococcal endocarditis.

Other enterococci

Other enterococci and the conditions they are associated with are:
E. hirae – Septicaemia and endocarditis. Has been associated with osteomyelitis in young broilers.
E. durans – Bacteraemia and isolated from brains of birds showing nervous signs.
E. cecorum – Weakness, lameness, mortality. Enterococcal vertebral osteoarthritis in which organism isolated from spinal abscesses. Femoral osteomyelitis.

8 – Listeria

Introduction

Listeriosis, caused by Listeria monocytogenes, occurs very occasionally in chickens, turkeys, pigeons and waterfowl and very sporadically in some other avian species. L. monocytogenes can cause food poisoning in man following consumption of contaminated cooked poultry products. In most of these instances the L. monocytogenes comes from the environment of the cooking plant and not from the live birds the cooked product was derived from.
Contaminated dead birds or faeces from birds carrying L. monocytogenes can be a source of L. monocytogenes to ruminants, for example from litter spread or clamped in the field containing the ruminants.

The disease

Listeriosis can affect poultry as a septicaemic or encephalitic form. Septicaemic birds are emaciated and have diarrhoea, whereas birds afflicted by the encephalitic form show nervous signs including depression, incoordination, ataxia, opisthotonus and torticollis. The last of these signs is regularly seen.
L. monocytogenes is frequently found in faeces and the soil and infection can occur via ingestion, inhalation or via a wound. Outbreaks of listeriosis have been seen following beak trimming.

Pathology

In the septicaemic form enlarged spleens, multifocal necrotic hepatitis, myocardial necrosis and inflammation are usually seen. Ascites and haemorrhaging in the liver, heart, spleen, kidneys and brain are seen in broilers, while in hens salpingitis is a common sequel to septicaemia.
In birds affected by the encephalitic form Gram positive bacteria are seen in the mid brain, cerebellum and medulla oblongata.

Diagnosis

Diagnosis is made on the basis of clinical and post mortem findings coupled to the isolation of L. monocytogenes. There are 13 serotypes of L. monocytogenes and the majority of animal infections are caused by serotypes 1/2a, 1/2b and 4B.

Treatment

L. monocytogenes is usually resistant to the commonly used antibiotics.

9 – Haemorrhagic enteritis

Introduction

Haemorrhagic enteritis of turkeys is an acute viral disease that affects turkeys over one month of age. Typically bloody droppings are seen and birds die. This disease was first seen in Minnesota, USA and reached an epidemic level in some of the USA's turkey states in the 1960s.

Aetiology

Haemorrhagic enteritis virus is an adenovirus that is closely related to the marble spleen disease virus and the avian adenovirus, which causes splenomegaly in broiler breeders. This group of viruses is serologically distinct and are now referred to as siadenoviruses. The haemorrhagic enteritis virus remains very stable under a variety of conditions. The virus can remain viable in faeces for several weeks and the disease commonly recurs on infected premises.

The disease

Mortality varies from almost nothing to 60% and the disease has been seen in all the turkey raising areas of the world. Haemorrhagic enteritis is rarely seen in birds under six weeks of age and typically occurs between 6-11 weeks of age. In turkeys the incubation period is 5-6 days.

Clinically affected birds show depression and bloody faeces over a 24 hour period. Bloody faecal material is often seen around the vent. In those birds that survive, clinical signs subside in just over a week. Due to the heavy blood loss, birds that die in the acute stage often look pale. Birds recovering from this disease have a good protective immunity.

Post mortem findings

Birds are typically in a good bodily condition, but their small intestines are distended with blood. The intestinal wall is congested and may be covered by a fibrinonecrotic membrane. Spleens are enlarged and mottled.

Diagnosis

Diagnosis is by clinical and post mortem findings and by demonstration of the causal virus. The differential diagnosis includes lymphoid neoplasias, such as reticuloendotheliosis or lymphoproliferative disease, acute bacteraemias, such as colisepticaemia and erysipelas, highly pathogenic Newcastle disease, bacterial endotoxaemias, acute coccidiosis and various toxicities.

Control

Vaccination has been successfully used.