Ethiopian Veterinary Association

Poultry Health Management

Continuous Professional Development (CPD) Training Module

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1. Introduction

The poultry population of Ethiopia, composed solely of chickens, is estimated at 59.5 million (CSA, 2017). This number is very small in relation to the human population of the country estimated at about 105 million in 2017 (World Bank, 2017). In addition to its small size, the vast majority (about 91%) of the country's poultry is made up of the low productive indigenous chickens (CSA, 2017) managed under the low input-output extensive village backyard production system (Dessie and Ogle, 2001; Getu and Birhan, 2014) with high disease pressure (Dessie and Ogle, 2001). As a result the per-capita poultry meat (0.66 kg) and egg (0.36kg) consumption of the country is one of the lowest in the world (FAOSTAT, 2018).

The poultry production systems in Ethiopia may be classified broadly into family poultry production systems (i.e. the small-scale intensive, semi-intensive, extensive scavenging and small-extensive scavenging systems) and the industrial and integrated/medium- and large-scale intensive systems (FAO, 2019). Although the traditional backyard scavenging family poultry system is still the dominant one, the market-oriented improved family poultry systems using semi-scavenging crossbred chickens, and medium- and large-scale intensive poultry systems, are growing in importance. The medium- and large-scale intensive poultry systems include farms with >1000 broilers or >500 layers (FAO, 2019). These farms implement medium to high bio-security standards.

The demand for poultry meat and eggs has been growing and continues to grow in Ethiopia due to population growth, rising per capita income and urbanization (FAO, 2007; Shapiro et al., 2015). Moreover, as one of the most efficient animals in terms of feed conversion efficiency, poultry is expected to offset the considerable deficit the country is expected to face in red meat supply in the future. This entails a need to increase the number of exotic specialized birds, and intensification of production with appropriate feed supply and health care, among others (Shapiro et al., 2015).

These poultry development endeavors, however, are likely to be challenged mainly by shortage of supply and high cost of improved chickens, prevailing diseases and feed shortage. Diseases have already been very important constraints of poultry production in Ethiopia and affect both village and intensive poultry production (Dessie and Ogle, 2001; Abie *et al*., 2003; Dessie and Jobre, 2004; Lobago and Woldemeskel, 2004; Zeleke et al., 2005; Aragaw *et al*., 2010). Some diseases, however, can prove problematic when there is a transition from the extensive low input production with local chickens to semi-intensive and intensive high input production using exotic birds (Snoeyenbos, 1978).

Poultry diseases not only affect the production performance but also often lead to higher rates of mortality. Therefore, it is important for producers to respond to outbreaks of diseases as quickly as possible (Carpentier et al., 2019). The earlier a disease is recognized and defined, the more successful an intervention or treatment will be. Successful, early treatment of a disease will reduce the associated losses and costs.

The primary aim of poultry health management is to prevent the onset of disease, to recognize the presence of disease at an early stage, and to treat birds/flocks that are diseased as soon as possible and before they develop into a serious condition or spread to other flocks. To be able to do this it is necessary to recognize birds that are sick as early as possible. This requires close monitoring for signs of ill health.

2. Flock Health Monitoring

Learning outcomes

- Understand the meaning, importance and elements of poultry flock health monitoring
- Be able to plan and implement health monitoring in poultry flocks

A flock-health monitoring program can assist in reducing production costs by increasing the efficiency of feed utilization and decreasing the incidence of clinical and subclinical diseases. It allows producers to optimize conditions of production to obtain maximum efficiency. It permits the detection of subclinical problems, the early detection of outbreaks of disease, the identification of new or emerging problems, or the occurrence of otherwise unnoticed production losses. Flock-health monitoring can be of great value to the industry and to regulatory agencies. It is used by the poultry industry to optimize its vaccination, parasite control (e.g. coccidiosis), management and nutrition programs. Data acquired in a monitoring system can be used to make comparisons among production variables, between flocks, houses, complexes, and companies (Keirs et al., 1991).

A flock-health monitoring system has evolved over time and consists of regular, representative live-bird sampling, necropsy and scoring of gross observations; data storage and summary (Keirs et al., 1991). It should also include routine examination of the birds for signs of illness and suboptimal production.

Examination of chickens

It is important to observe chickens regularly to determine their health status. Observation is very important because if any disease situation is detected early it is possible to take precautionary measures in other flocks and in healthy chickens for the flock in which individual chickens are sick. Some poultry diseases are treatable if detected early. But early detection of disease problems in chickens needs a good

knowledge of their phenotypic, anatomical and physiological behaviors.

It is important to check chickens at least once daily- it is worth to spend some time watching how the flock behaves and interacts. Observe the flock from a distance, so that the birds will not feel threatened or excited. Many diseases cause changes in attitude or behavior including lethargy, inactivity and segregation.

Feed and water consumption should also be noted, a drop in appetite is often the first effect of illness in birds. With good records, any change in consumption will be quickly spotted. Other early indicators of disease include changes in egg production, weight gain, fertility, and egg hatchability.

Birds exhibiting abnormal behavior/signs should then undergo detailed examination. Detailed examination of chickens needs catching and restraining the bird. Examination involves careful observation of various parts of the body, including the natural openings (nostril, eye, vent etc.) the skeletal system, respiratory and gastrointestinal systems, skin and feathers, and the weight of the chicken (Habte et al., 2017).

The eyes of healthy chickens are bright and shiny. Some diseases cause discoloration, scars, accumulations and discharges in and around the eye, and sick birds often close or partially close their eyes. Birds with impaired vision will have problem feeding, and should be isolated from other birds to allow them to access feed more easily.

It is also important to examine the nostrils, beak and oral cavity for any kind of swelling, discharge, odor, discoloration and texture of the nostril and surrounding skin. When there is a problem in the respiratory tract, abnormal sounds such as rattles or wheezing may be heard as the bird breathes. Birds may also show open mouth breathing and tail bobbing (moving the tail feathers up and down).

Examination of birds should also include the skeletal system, especially if nutritional problems are suspected. Wings should be examined for swelling, fractures, discoloration of feathers or skin, bone deformation, plumage quality, wing paralysis and skin changes. The legs are examined similarly to the wings except that, in most birds, no feathers are present on the lower leg. Plumage also can be examined for damage, color changes, condition, evidence of soiling, frayed feathers, new feathers and parasite damage.

The vent should be examined last during examination of the bird. This is the area in which most evidence of gastrointestinal problems can be seen, including a vent heavily soiled by fecal material, which may indicate gastrointestinal disease. It is important to check for parasites, soiling of the feathers,

evidence of laying eggs, diarrhea, swelling, reddening, blood from the vent, prolapse, or other abnormalities. It is very important to take information on dropping color, its consistency and odor, as some infections cause distinctive changes in droppings, which can help in the diagnosis process.

Knowledge of the season at which certain diseases are more likely to occur can help to identify a disease in the early stages before it spreads throughout the flock.

Good records are an important part of good management which help to quickly identify health and production problems and their solution. Some of the basic observations/information that should be recorded include:

- Production records –including egg production and weight gain; loss of egg quality, including frequency of misshapen shells and frequency of cracks, are important because they can be an early signal of disease problems.
- Flock health records – document a disease event, its cause, circumstances of occurrence, attempted intervention and results obtained. Such a record is important for preventing a recurrence of the problem. Having ongoing records also prompts regular observation of the flock.

3. Common Major Diseases

(Adapted from: The Merck Veterinary Manual)

3.1. Viral diseases

Learning outcomes

- Sound knowledge (etiology, clinical manifestation, lesions, diagnosis, treatment, control/prevention) of the common viral diseases affecting poultry
- Plan and apply preventive/control measures for common viral diseases of poultry

3.1.1. Newcastle Disease

Newcastle disease (ND) is a highly contagious and often severe disease found worldwide that affects birds including domestic poultry. The disease appears in three forms: lentogenic or mild, mesogenic or moderate and velogenic or very virulent, also called Newcastle disease. The lentogenic strains are very widespread but cause few disease outbreaks. It usually presents as a respiratory disease, but depression, nervous manifestations, or diarrhea may be the predominant clinical form (from Oie).

Etiology

ND is caused by virulent strains of avian paramyxovirus type 1 (PMV 1), also known as Newcastle disease virus (NDV).

NDV is an RNA virus, classified into one of three virulence groups by chicken embryo and chicken inoculation as virulent (velogenic), moderately virulent (mesogenic), or of low virulence (lentogenic) group. Velogens and mesogens are now classified as virulent NDV (vNDV), the cause of Newcastle disease, whereas lentogens, widely used as live vaccines, as the low virulence NDV (loNDV).

Epidemiology and Transmission

ND is found throughout the world, the disease has been currently controlled in Canada, the United States and some western European countries. It continues in parts of Africa, Asia and South America. However, since wild birds can sometimes carry the virus without becoming ill, outbreaks can occur anywhere that poultry is raised.

ND is transmitted most often by direct contact with diseased or carrier birds. Infected birds may shed the virus in their feces, contaminating the environment. Transmission can then occur by direct contact with feces and respiratory discharges or by contaminated food, water, equipment, and human clothing. Newcastle disease viruses can survive for several weeks in the environment, especially in cool weather.

Generally, virus is shed during the incubation period and for a short time during recovery. Birds in the pigeon family can shed the virus intermittently for a year or more. Other wild birds such as cormorants have also been shown to have caused outbreaks in domestic poultry.

The virus is present in all parts of the carcass of an infected bird.

The disease is very contagious. When the virus is introduced into a susceptible flock, virtually all the birds will be infected within two to six days.

Clinical signs

Clinical manifestations vary from high morbidity and mortality to asymptomatic infections depending on factors such as: the strain of the virus, the species of bird infected, the age of the host, (young birds are the most susceptible), concurrent infection with other organisms, environmental stress and immune status. In some circumstances infection with the extremely virulent virus strains can result in high numbers of birds found dead with comparatively few clinical signs. The disease has a rapid onset with symptoms appearing between two and twelve days after exposure, and spreads rapidly through the flock.

Some virus strains attack the nervous system, others the respiratory, or digestive systems. Clinical signs include: respiratory signs – gasping, coughing, sneezing and rales; nervous signs – tremors, paralyzed wings and legs, twisted necks, circling, spasms, and paralysis; digestive signs; diarrhea; a partial or complete drop in egg production may occur. Eggs may be abnormal in color, shape, or surface, and have watery albumen; mortality is variable but can be as high as 100%.

Lesions

Remarkable gross lesions are usually seen only with viscerotropic velogenic Newcastle disease. Petechiae may be seen on the serous membranes; hemorrhages of the proventricular mucosa and intestinal serosa are accompanied by multifocal, necrotic hemorrhagic areas on the mucosal surface of the intestine, especially at lymphoid foci such as cecal tonsils. Splenic necrosis and hemorrhage and edema around the thymus may also be seen. Secondary bacterial infections increase the severity of respiratory lesions.

Diagnosis

NDV can be isolated from oropharyngeal or cloacal swabs or tissues from infected birds. Infection is confirmed by recovery of a hemagglutinating virus that is inhibited with NDV antiserum or by detection of NDV RNA by reverse transcriptase PCR. A rise in NDV antibody titer by hemagglutination-inhibition or ELISA of paired serum samples indicates NDV infection.

Prevention

Vaccines are available for chickens, turkeys, and pigeons. Unfortunately, ND vaccines do not provide sterile immunity, and in many areas of the world vaccines are used to prevent losses from sickness and death. Live lentogenic vaccines, chiefly B1 and LaSota strains, are widely used and typically administered to poultry by mass application in drinking water or by spray. Mucosal immunity induced in birds vaccinated by live vaccines applied by these routes decreases the amount of vNDV the vaccinated birds will shed if infected with vNDV, compared with the immune response induced by an inactivated vaccine. Alternatively, individual administration of live vaccines is via the nares or conjunctival sac. Healthy chicks are vaccinated as early as day 1–4 of life. However, delaying vaccination until the second or third week avoids maternal antibody interference with an active immune response.

Oil-adjuvanted inactivated vaccines are also used after live vaccine in breeders and layers. In countries where vNDV is endemic, a combination of live virus and inactivated vaccine can be used; or alternatively, if permitted by law, a live mesogenic strain vaccine may be used in older birds. The frequency of revaccination to protect chickens throughout life largely depends on the risk of exposure and virulence of the field virus challenge. Administering inactivated vaccines is more labor intensive,

because each bird has to be handled individually.

Fowlpox or turkey herpesvirus-vectored NDV vaccines are commercially available for chickens and have the advantage of being able to be administered in ovo at the hatchery.

3.1.2. Infectious Bronchitis

Infectious bronchitis (IB) is an acute, highly contagious upper respiratory tract disease in chickens.

Etiology

Avian infectious bronchitis (IB) is caused by the Gammacoronavirus infectious bronchitis virus (IBV) that only causes disease in chickens. The virus is worldwide in distribution, and there are many antigenic types that can cocirculate in a given region.

Transmission

IBV is shed by infected chickens in respiratory discharges and feces, and it can be spread by aerosol, ingestion of contaminated feed and water, and contact with contaminated equipment and clothing. Naturally infected chickens and those vaccinated with live IBV may shed virus intermittently for up to 20 weeks after infection. The incubation period is generally 24–48 hours, with the peak in excretion of virus from the respiratory tract lasting 3–5 days after infection.

Clinical Findings

The severity of disease and the body systems involved are influenced by: strain of the virus; age, strain, immune status, and diet of the chicken; and cold stress. In addition, coinfection with *Mycoplasma gallisepticum*, *M synoviae*, *Escherichia coli*, and/or *Avibacterium paragallinarum* can exacerbate disease.

Morbidity for flocks affected by infectious bronchitis is typically 100%. Chicks may cough, sneeze, and have tracheal rales for 10–14 days. Conjunctivitis and dyspnea may be seen, and sometimes facial swelling, particularly with concurrent bacterial infection of the sinuses. Chicks may appear depressed and huddle under heat lamps. Feed consumption and weight gain are reduced. Infection with nephropathogenic strains can cause initial respiratory signs, then later depression, ruffled feathers, wet droppings, greater water intake, and death.

In layers, egg production may drop by as much as 70%, and eggs are often misshapen, with thin, soft, wrinkled, rough, and/or pale shells, and can be smaller and have watery albumen. Egg production and egg quality can return to normal, but this may take up to 8 weeks. In most outbreaks, mortality is approximately 5%, although mortality rates can be as high as 60% when disease is complicated by

concurrent bacterial infection or when nephropathogenic strains induce interstitial nephritis in chicks. Infection of chicks may cause permanent damage to the oviduct, resulting in layers or breeders that never reach normal levels of production, so-called false layer syndrome.

Lesions

In the respiratory tract, the trachea, sinuses, and nasal passages may contain serous, catarrhal, or caseous exudates, and the air sacs a foamy exudate initially, progressing to cloudy thickening. If complicated by infection with *E coli*, there may be caseous airsacculitis, perihepatitis, and pericarditis. Birds infected when very young may have cystic oviducts, whereas those infected while in lay have an oviduct of reduced weight and length and regression of the ovaries. Infection with nephropathogenic strains results in swollen, pale kidneys, with the tubules and ureters distended with urates; in birds with urolithiasis, the ureters may be distended with urates and contain uroliths, and the kidneys may be atrophied.

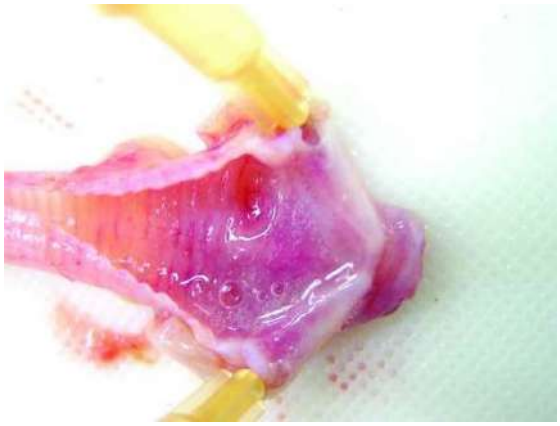

Figure 1. Trachea with excessive amount of mucus

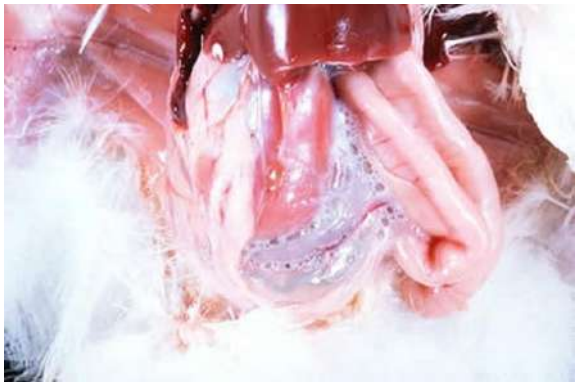

Figure 2. Airsacculitis

Diagnosis

Demonstration of seroconversion or a rise in antibody titer against IBV by ELISA, or hemagglutination inhibition or virus neutralization tests can be used for diagnosis when there is a history of respiratory disease or reduced egg production.

Definitive diagnosis is generally based on virus detection and identification. Virus can be isolated from of homogenates of tracheal, cecal tonsil, and/or kidney tissue. Diagnosis is commonly achieved using reverse transcriptase PCR assays to detect viral RNA in nucleic acid extracts of tracheal, cecal tonsil, or kidney tissue.

Treatment and Control

No medication alters the course of IBV infection, although antimicrobial therapy may reduce mortalities caused by complicating bacterial infections.

Attenuated live and killed vaccines are used to control the disease, but little or no cross reactivity between types requires the correct vaccine type be applied

The live-attenuated vaccines are initially given to 1- to 14-day-old chicks by spray, drinking water, or eye drop, and birds are commonly revaccinated approximately 2 weeks after the initial vaccination. Revaccination with a different serotype can induce broader protection. Attenuated or adjuvanted inactivated vaccines can be used in breeders and layers to prevent egg production losses as well as to pass protective maternal antibodies to progeny.

Selection of vaccines should be based on knowledge of the most prevalent virus type(s) in the area. There are a number of different IBV vaccine types licensed for use in various countries as well as live and killed autogenous vaccines specific for the variant virus in the region.

3.1.4. Infectious Laryngotracheitis

Infectious laryngotracheitis (ILT) is an acute, highly contagious, herpesvirus infection of mainly chickens characterized by severe dyspnea, coughing, and rales. It can also be a subacute disease with nasal and ocular discharge, tracheitis, conjunctivitis, and mild rales.

Etiology

The disease is caused by *Gallid herpesvirus 1*, commonly known as infectious laryngotracheitis virus (ILTV).

The virus can be easily transmitted by infected birds and fomites. Lax biosecurity, transportation of infected birds, and spread of contaminated litter facilitates spread of the virus. Infection is acquired via the upper respiratory tract and transmission occurs most readily from acutely infected birds.

Clinical Findings

Clinically, the disease may appear in three forms, namely peracute, subacute, and chronic or mild. In the peracute form, onset of disease is sudden with a rapid spread. The morbidity is high and mortality may exceed 50%. Some birds may die in good body condition before the appearance of signs, which are characteristic and comprise difficulty in breathing with extension of the neck and gasping in an attempt to inhale. There is also gurgling, rattling and coughing when birds try to expel obstructions in the

trachea. Conjunctivitis may also be observed. Clots of blood may be coughed up and can be found on the floor and walls of the house. In the subacute form, the onset of illness is slower and respiratory signs may extend over some days before deaths are seen. The morbidity is high but the mortality is lower than in the peracute form, between 10% and 30%. Chronic or mild ILT may be seen among survivors of either of the above forms of the disease, although some outbreaks themselves may be entirely mild. Signs include coughing, nasal, ocular and oral discharge, and reduced egg production.

Figure 3. Chickens coughing bloody exudate and gasping for air

Figure 4. Moderate conjunctivitis

Lesions

In peracute form, post-mortem changes are confined to the upper respiratory tract and are also characteristic, consisting of hemorrhagic tracheitis with blood clots, mucoid rhinitis, and blood-stained mucus along the length of the trachea. In the subacute form, post-mortem findings are less severe and consist of mucoid exudate with or without blood in the trachea. Yellow caseous diphtheritic membranes may be found adherent to the larynx and upper tracheal mucosa.

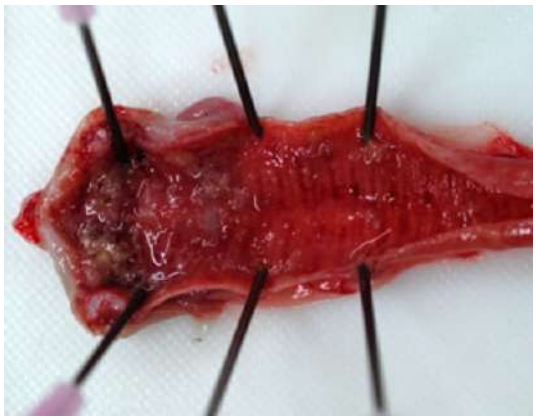

Figure 5. Severe hemorrhagic tracheitis with mucus

Diagnosis

Microscopically, the acute phase of the severe form of the disease is characterized by a desquamative, necrotizing tracheitis and conjunctivitis. A rapid diagnosis of the mild forms of the disease can be achieved by the detection of lesions that are pathognomonic of the infection, such as syncytial formation and intranuclear inclusion bodies in the trachea and conjunctiva mucosal epithelium. This diagnosis can be rapidly confirmed by detection of viral DNA using virus-specific PCR assays.

Laboratory diagnosis includes detection of microscopic lesions characteristic of ILTV replication, detection of viral DNA or viral antigen from upper respiratory tissues, and ultimately, virus isolation.

Control

In endemic areas and on farms where a specific diagnosis is made, ILTV is controlled by implementation of biosecurity measures and vaccination. Vaccination is done with live attenuated vaccines and viral vector recombinant vaccines. Live vaccines originated from virulent isolates that were attenuated by consecutive passages in embryos or tissue culture. These are applied via eye drop or through mass vaccination by water or spray. Viral vector recombinant vaccines in fowlpox and herpesvirus of turkeys have been designed to express ILTV immunogenic proteins and are administered to individual birds by in ovo, subcutaneous, or wing-web vaccination.

3.1.5. Avian Influenza

Avian influenza (AI) is a viral infection of domestic poultry, and pet, zoo, and wild birds. In domestic poultry, AI viruses are typically of low pathogenicity (LP), causing subclinical infections, respiratory disease, or drops in egg production, but a few AI viruses are highly pathogenic (HP), causing severe systemic disease with multiple organ failure and high mortality.

Etiology

Avian influenza viruses are type A orthomyxoviruses (*Alphainfluenzavirus* or *Influenzavirus A*) characterized by antigenically homologous nucleoprotein and matrix internal proteins, which are identified by serology in agar gel immunodiffusion (AGID) tests. AI viruses are further divided into 16 hemagglutinin (H1-16) and 9 neuraminidase (N1-9) subtypes.

Epidemiology and Transmission

Low pathogenicity avian influenza viruses are distributed worldwide. The viruses may be present in village or backyard flocks and other birds sold through live-poultry markets, but most commercially raised poultry in developed countries are free of AI viruses. The HPAI viruses arise from mutation of some H5 and H7 LPAI viruses and cause devastating epidemics.

The incubation period is highly variable and ranges from a few days in individual birds to 2 weeks in the flock. Transmission between individual birds is by ingestion or inhalation. Spread between farms is the result of breaches in biosecurity practices, principally by movement of infected poultry or contaminated feces and respiratory secretions on fomites such as equipment or clothing. Airborne dissemination between farms may be important over limited distances.

Sporadic natural and/or experimental infections have occurred in cats and dogs with H5 Eurasian HPAI viruses. Other mammals have been experimentally infected with H5 HPAI viruses, including pigs, ferrets, rats, rabbits, guinea pigs, mice, mink, and nonhuman primates.

Clinical Findings and Lesions

Most avian influenza viruses (H1-16 subtypes) are LPAI, but some of the H5 and H7 AI viruses are HPAI and highly lethal for chickens, turkeys, and related gallinaceous domestic poultry. This HPAI form of the disease has historically been called fowl plague. In most wild birds, AI viral infections are subclinical except for the recent H5 HPAI viruses of Eurasian lineage, which have been associated with mortality in wild and/or domestic waterfowl and other species of wild and domestic birds. Clinical signs, severity of disease, and mortality rates vary, depending on AI virus strain and host species.

Figure 6. Subcutaneous hemorrhage of the skin on feet and shanks

Figure 7. Ischemic necrosis of comb and wattles

Low Pathogenicity Avian Influenza Viruses

Low pathogenicity avian influenza viruses typically produce respiratory signs such as sneezing, coughing, ocular and nasal discharge, and swollen infraorbital sinuses in poultry. Sinusitis is common in domestic ducks, quail, and turkeys. Lesions in the respiratory tract typically include congestion and inflammation of the trachea and lungs. In layers and breeders, there may be decreased egg production or infertility, ova rupture (evident as yolk in the abdominal cavity) or involution, or mucosal edema and inflammatory exudates in the lumen of the oviduct. A few layer and breeder chickens may have acute renal failure and visceral urate deposition (visceral gout). The morbidity and mortality is usually low unless accompanied by secondary bacterial or viral infections or aggravated by environmental stressors.

Low Pathogenicity Avian Influenza Viruses

Even in the absence of secondary pathogens, HPAI viruses cause severe, systemic disease with high mortality in chickens, turkeys, and other gallinaceous poultry; mortality can be as high as 100% in a few days. In peracute cases, clinical signs or gross lesions may be lacking before death. However, in acute cases, lesions may include cyanosis and edema of the head, comb, wattle, and snood (turkey); ischemic necrosis of comb, wattles, or snood; edema and red discoloration of the shanks and feet due to subcutaneous ecchymotic hemorrhages; petechial hemorrhages on visceral organs and in muscles; and blood-tinged oral and nasal discharges. In severely affected birds, greenish diarrhea is common.

Birds that survive the peracute infection may develop CNS involvement evident as torticollis, opisthotonos, incoordination, paralysis, and drooping wings. The location and severity of microscopic lesions are highly variable and may consist of edema, hemorrhage, and necrosis in parenchymal cells of multiple visceral organs, skin, and CNS.

Diagnosis

- Avian influenza virus isolation
- Detection of AI viral RNA
- Detection of AI-specific antibodies
- Detection of PI antigens

The presence of clinical disease alone is not diagnostic. Low pathogenicity and high pathogenicity avian influenza viruses can be readily isolated from oropharyngeal and cloacal swabs, and HPAI viruses from many internal organs.

Differential Diagnosis

LPAI must be differentiated from other respiratory diseases or causes of decreased egg production, including:

- acute to subacute viral diseases such as infectious bronchitis, infectious laryngotracheitis, low virulent Newcastle disease, and infections by other paramyxoviruses
- bacterial diseases such as mycoplasmosis, infectious coryza, ornithobacteriosis, turkey coryza, and the respiratory form of fowl cholera
- fungal diseases such as aspergillosis

HPAI must be differentiated from other causes of high mortality such as virulent Newcastle disease, the peracute septicemic form of fowl cholera, heat exhaustion, and severe water deprivation.

Prevention and Treatment

Supportive Care

Treating LPAI-affected flocks with broad-spectrum antibiotics to control secondary pathogens may reduce morbidity and mortality.

Preventive Measures

Practice of exclusion biosecurity strategies to prevent introduction of AI into poultry is the best preventive measure. Suspected outbreaks should be reported to appropriate regulatory authorities.

Antigenically matched and properly administered vaccines can prevent clinical signs and death and greatly reduce virus replication and shedding from the respiratory and GI tracts. Specific protection is achieved through autogenous virus vaccines or from vaccines prepared from AI virus of the same hemagglutinin subtype. Antibodies to the homologous viral neuraminidase antigens may provide partial protection.

Zoonotic Risk

Avian influenza viruses exhibit host adaptation to birds. Human infections have occurred, with certain lineages, usually as isolated, rare, individual cases. The primary risk factor for human infection has been direct contact with live or dead infected poultry, but a few cases have resulted from consumption of uncooked poultry products, defeathering of infected wild swans, or close contact with human cases.

3.1.6. Infectious Bursal Disease (Gumboro disease)

Etiology

Infectious bursal disease (IBD) is caused by a birnavirus (infectious bursal disease virus; IBDV) that is most readily isolated from the bursa of Fabricius but may be isolated from other organs. Two serotypes of IBDV, designated serotypes 1 and 2, are recognized. Clinical disease has been associated only with serotype 1.

Epidemiology and Transmission

Infectious bursal disease (IBD) is seen in young domestic chickens worldwide. The causative virus IBDV is shed in the feces and transferred from house to house by fomites. It is very stable and difficult to eradicate from premises. IBD is highly contagious; results of infection depend on age and breed of chicken and virulence of the virus.

Clinical Manifestation

Infections may be subclinical or clinical. Infections before 3 weeks of age are usually subclinical. Chickens are most susceptible to clinical disease at 3-6 weeks of age when immature B cells populate the bursa and maternal immunity has waned.

Early subclinical infections are the most important form of the disease because of economic losses. They cause severe, long-lasting immunosuppression due to destruction of immature lymphocytes in the bursa of Fabricius, thymus, and spleen. The humoral (B cell) immune response is most severely affected; the cell-mediated (T cell) immune response is affected to a lesser extent. Chickens immunosuppressed by early IBDV infections do not respond well to vaccination and are predisposed to infections with normally nonpathogenic viruses and bacteria. Common diseases are usually exacerbated by IBDV infections. Some strains of IBDV can cause subclinical infections in older birds (3–6 weeks old), which leads to losses from poor feed efficiency and longer times to market. In these cases, the immunosuppression is usually transient, and convalescent birds may recover most or all of their humoral immune function. However, secondary infections that occur during the transient immunosuppression can cause significant economic losses.

In clinical infections, onset of the disease occurs after an incubation of 3-4 days. Chickens may exhibit severe prostration, depression, incoordination, watery diarrhea, ruffled feathers, soiled vent feathers, vent picking, and inflammation of the cloaca. Flock morbidity is typically 100%, and mortality can range from 5% to greater than 60% depending on the strain of virus and breed of chicken. Mortality is typically higher in layer breeds compared with broiler chickens. Recovery occurs in < 1 week, and broiler weight gain is delayed by 3–5 days. The presence of maternal antibody will modify the clinical course of the disease.

Virulence of field strains of the virus varies considerably. Viruses that range from naturally attenuated to very virulent (vv) have been observed. The vvIBDV strains can cause high (>20%) mortality.

Lesions

For both clinical and subclinical forms of the disease, all pathogenic IBDVs cause lesions in the bursa of Fabricious.

At necropsy, the lesions seen will depend on the strain of IBDV. For strains that cause a clinical disease, the cloacal bursa is swollen, edematous, yellowish, and occasionally hemorrhagic, especially in birds that died of the disease. Strains of vvIBDV cause similar cloacal bursa lesions, and congestion and hemorrhage of the pectoral and leg muscles can also occur. Some IBDV strains can cause atrophy of the cloacal bursa without the appearance of gross lesions in that organ. Chickens that have recovered from IBDV infections have small, atrophied, cloacal bursas due to the destruction and lack of regeneration of the bursal follicles.

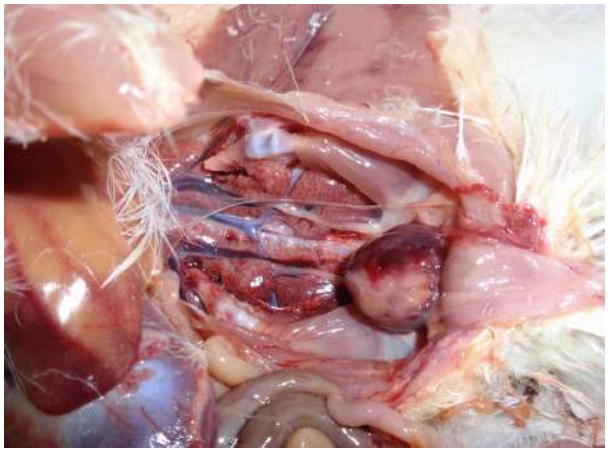

Figure 8. Enlarged hemorrhagic bursa of Fabricius in a chicken infected with very virulent infectious bursal disease virus

Diagnosis

- Diagnosis can be accomplished by clinical evaluation of the cloacal bursa for macroscopic and microscopic lesions followed by molecular detection of the viral VP2 gene using RT-PCR
- Sequence analysis of the VP2 gene is used to identify the IBDV genotype
- Virus isolation in chicken embryos or chicken embryo fibroblast cell cultures is possible but often not necessary

Initial diagnosis of IBD is accomplished by the observation of gross lesions in the cloacal bursa. This is followed by microscopic analysis of the bursa for lymphocyte depletion in the follicles. Molecular diagnostic assays are most often used to identify IBDV in diagnostic samples. The reverse-transcriptase-PCR assay is used to identify the viral genome in bursa tissue. Samples for molecular diagnostic testing are typically collected after maternal antibodies have waned.

IBDV may be isolated in 8- to 11-day-old, antibody-free chicken embryos with inocula from birds in the early stages of disease. Some strains of IBDV may also be isolated in cell cultures.

Serology can be used to detect the presence of antibodies to IBDV in convalescent chicks. The presence of IBDV antibodies in chicks is not always an indication of infection because most young chicks have maternal antibodies.

Control

Rigorous disinfection of contaminated farms after depopulation has achieved limited success. Live vaccines of varying low pathogenicity can be administered by eye drop, drinking water, or SC routes at 1–21 days of age. Replication of these vaccines and thus the immune response can be altered by maternal antibody, although the more virulent vaccine strains can override higher levels of maternal antibody. Vectored vaccines can be used *in ovo* or at hatch.

High levels of maternal antibody during early brooding of chicks in broiler flocks (and in some commercial layer operations) can minimize early infection, subsequent immunosuppression, or both. Breeder flocks should be vaccinated one or more times during the growing period, first with a live vaccine and again just before egg production with an oil-adjuvanted, inactivated vaccine. The inactivated vaccines induce higher, more uniform, and more persistent levels of antibody than do live vaccines. The immune status of breeder flocks should be monitored periodically with a quantitative serologic test. If antibody levels decrease, hens should be revaccinated to maintain adequate immunity in the progeny.

The goal of any vaccination program for IBD should be to use vaccines that most closely match the antigenic profile of the field viruses.

3.1.7. Fowlpox

Fowlpox is a slow-spreading viral infection of chickens and turkeys characterized by proliferative lesions in the skin that progress to thick scabs (cutaneous form) and by lesions in the upper GI and respiratory tracts (diphtheritic form). Virulent strains may cause lesions in the internal organs (systemic form). Fowlpox is seen worldwide.

Etiology

Fowlpox is caused by a large DNA virus of the genus Avipoxvirus of the family Poxviridae. It is resistant and may survive in the environment for extended periods in dried scabs.

Transmission

The virus is present in large numbers in the lesions and is usually transmitted by contact through abrasions of the skin. Skin lesions (scabs) shed from recovering birds in poultry houses can become a source of aerosol infection. Mosquitoes and other biting insects may serve as mechanical vectors. Transmission within a susceptible flock is rapid when mosquitoes are plentiful. The disease tends to persist for extended periods in multiple-age poultry complexes because of slow spread of the virus and availability of susceptible birds.

Clinical Findings

The cutaneous form of fowlpox is characterized by nodular lesions on various parts of the unfeathered skin of chickens. Generalized lesions of feathered skin may also be seen. In some cases, lesions are limited chiefly to the feet and legs. The lesion is initially a raised, blanched, nodular area that enlarges, becomes yellowish, and progresses to a thick, dark scab. Multiple lesions usually develop and often coalesce. Lesions in various stages of development may be found on the same bird. Localization around the nostrils may cause nasal discharge. Cutaneous lesions on the eyelids may cause complete closure of one or both eyes. Only a few birds develop cutaneous lesions at one time. Lesions are prominent in some birds and may significantly decrease flock performance.

In the diphtheritic form of fowlpox, lesions develop on the mucous membranes of the mouth, esophagus, pharynx, larynx, and trachea (wetpox or fowl diphtheria). Occasionally, lesions are seen almost exclusively in one or more of these sites. Caseous patches firmly adherent to the mucosa of the larynx and mouth or proliferative masses may develop. Mouth lesions interfere with feeding. Tracheal lesions cause difficulty in respiration. In cases of systemic infection caused by virulent fowlpox virus strains, lesions may be seen in internal organs. More than one form of the disease, i.e., cutaneous, diphtheritic, and/or systemic, may be seen in a single bird.

Often, the course of the disease in a flock is protracted. Extensive infection in a layer flock results in decreased egg production. Cutaneous infections cause low or moderate mortality, and these flocks generally return to normal production after recovery. Mortality is usually high in diphtheritic or systemic infections.

Figure 9. Scab-like lesion due to fowlpox

Diagnosis

Cutaneous infections usually produce characteristic gross and microscopic lesions. Microscopic examination of affected tissues stained with H&E reveals eosinophilic cytoplasmic inclusion bodies. Cytoplasmic inclusions are also detectable by fluorescent antibody and immunohistochemical methods. The elementary bodies in the inclusion bodies can be detected in smears from lesions stained by the Gimenez method. Viral particles with typical poxvirus morphology can be demonstrated by negative-staining electron microscopy as well as in ultrathin sections of the lesions. The virus can be isolated by inoculating chorioallantoic membrane of developing chicken embryos, susceptible birds, or cell cultures of avian origin.

Detailed genetic analysis reveals differences between vaccine strains and field strains responsible for outbreaks of fowlpox in previously vaccinated chicken flocks.

PCR can be used to amplify genomic DNA sequences of various sizes using specific primers. PCR has also been used effectively to differentiate field and vaccine strains of the virus.

Naturally infected or vaccinated birds develop both humoral and cell-mediated immune responses. Humoral immune response can be measured by ELISA, agar gel precipitation (AGP), or virus neutralization tests.

Prevention

Where fowlpox is prevalent, chickens should be vaccinated with a live virus vaccine. The most widely used vaccines are attenuated fowlpox virus and pigeonpox virus isolates of high immunogenicity and low pathogenicity. In high-risk areas, vaccination with an attenuated vaccine of cell-culture origin in the first few weeks of life and revaccination at 12–16 weeks is often sufficient. Because the infection spreads slowly, vaccination is often useful to limit spread in affected flocks if administered when < 20% of the birds have lesions. Passive immunity may interfere with multiplication of vaccine virus; progeny from recently vaccinated or recently infected flocks should be vaccinated only after passive immunity has declined. Revaccination with another serial lot of vaccine may be indicated.

3.1.8. Marek's Disease

Marek's disease (MD) is a highly contagious viral disease of poultry characterized by T-cell lymphomas and peripheral nerve enlargement. Marek's disease is one of the most ubiquitous avian infections; it is identified in chicken flocks worldwide. Every flock, except for those maintained under strict pathogen-free conditions, is presumed to be infected. Although clinical disease is not always apparent in infected flocks, a subclinical decrease in growth rate and egg production may be economically important.

Etiology

Marek's disease virus (MDV) is a member of the genus *Mardivirus* within the subfamily Alphaherpesvirinae. Within the genus *Mardivirus* are three closely related species previously designated as three serotypes of Marek's disease virus. *Gallid alphaherpesvirus* 2 (MDV serotype 1) represents all virulent Marek disease virus strains. *Gallid alphaherpesvirus* 3 (MDV serotype 2) and *Meleagrid alphaherpesvirus* 1 (turkey herpesvirus, MDV serotype 3) represent avirulent virus strains isolated from chickens and turkeys, respectively, and are commonly used as vaccines against Marek's disease.

Transmission

Marek's disease is highly contagious and readily transmitted among chickens. The virus matures into a fully infective, enveloped form in the epithelium of the feather follicle, from which it is released into the environment. It may survive for months in poultry house litter or dust. Dust or dander from infected chickens is particularly effective in transmission. Once the virus is introduced into a chicken flock, regardless of vaccination status, infection spreads quickly from bird to bird. Infected chickens continue to be carriers for long periods and act as sources of infectious virus. Shedding of infectious virus can be reduced, but not prevented, by prior vaccination. Marek's disease virus is not vertically transmitted.

Clinical Findings

MD can occur at any time, beginning at 3-4 weeks of age or older, sometimes even well after the onset of egg production. MD is associated with several distinct pathological syndromes, of which the lymphoproliferative syndromes are the most frequent.

In the classical form of the disease, characterized mainly by the involvement of nerves the most common clinical sign is partial or complete paralysis of the legs and wings; mortality rarely exceeds 10–15% and can occur over a few weeks or many months. A transient paralysis syndrome has been associated with Marek disease; in which chickens become ataxic for periods of several days and then recover.

In the acute form, which is usually characterized by visceral lymphomas in multiple organs, birds are often severely depressed and some may die without showing clinical signs. Disease incidence of 10–30% in the flock is not uncommon and outbreaks involving up to 70% can occur. Mortality may increase rapidly over a few weeks and then cease, or can continue at a steady or slowly falling rate for several months.

Lesions

In its classical form the characteristic finding is enlargement of one or more peripheral nerves. Those most commonly affected and easily seen at post-mortem are the vagus, brachial and sciatic plexuses. Affected nerves are often two or three times their normal thickness, the normal cross-striated and glistening appearance is absent, and the nerve may appear greyish or yellowish, and sometimes edematous. Lymphomas are sometimes present in the classical form of MD.

In the acute form, the typical finding is widespread, diffuse lymphomatous involvement of the liver, gonads, spleen, kidneys, lungs, proventriculus and heart. Sometimes lymphomas also arise in the skin around the feather follicles and in the skeletal muscles. Histologically, the lesions consist of a mixed population of small, medium, and large lymphoid cells plus plasma cells and large anaplastic lymphoblasts. These cell populations undoubtedly include tumor cells and reactive inflammatory cells. Affected birds usually have enlarged peripheral nerves, as is seen in the classical form. Nerve lesions are often absent in adult birds with MD.

Figure 10. Leg paresis, chicken

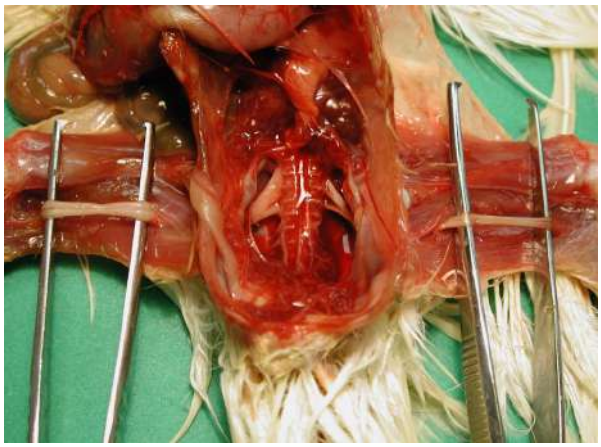

Figure 11. Unilateral enlargement of sciatic nerve and sacral plexus in a chicken

Diagnosis

For the diagnosis of Marek's disease, it is critical to diagnose the tumors and not the infection because Marek's disease is considered ubiquitous within commercial poultry flocks. Usually, diagnosis is based on enlarged nerves and lymphoid tumors in various viscera. The absence of bursal tumors helps distinguish this disease from lymphoid leukosis, although the presence of bursal tumors does not exclude Marek disease.

Immunohistochemistry can be used to confirm tumors are composed of predominant T-cell populations or expressing specific MDV antigens. There is a quantitative association between viral load and Marek's disease tumors; most tumor-bearing chickens have high viremia titers and are usually PCR positive. Thus, the demonstration of high quantities of virus, viral DNA, or viral antigens in tumor cells and the exclusion of other relevant tumor viruses should be sufficient for a specific diagnosis of Marek's disease.

Control

Vaccination is the central strategy for the prevention and control of Marek's disease, along with strict sanitation to reduce or delay exposure and by breeding for genetic resistance.

The most widely used vaccines include:

- Turkey herpesvirus (HVT, naturally avirulent *Meleagrid alphaherpesvirus* 1)
- SB-1 or 301B/1 (naturally avirulent *Gallid alphaherpesvirus* 3)
- CVI988/Rispens (attenuated *Gallid alphaherpesvirus* 2)

Vaccines are administered at hatch or *in ovo* to embryos at the 18th day of incubation. *In ovo* vaccination is now performed by automated technology and is widely used for vaccination of commercial broiler

chickens, mainly because of reduced labor costs and greater precision of vaccine administration.

Since the advent of vaccination, losses from Marek's disease have been reduced dramatically in broiler and layer flocks.

3.2. Bacterial diseases

Learning outcomes

- Sound knowledge (etiology, clinical manifestation, lesions, diagnosis, treatment, control/prevention) of the common bacterial diseases affecting poultry
- Plan and apply preventive/control measures for common bacterial diseases of poultry

3.2.1. Pullorum Disease

The historical name for this disease is bacillary white diarrhea (BWD). Pullorum disease is characterized by very high mortality in young chickens and turkeys.

Etiology

Pullorum disease is caused by *Salmonella enterica* Pullorum, usually written in short as *Salmonella* Pullorum.

Transmission

Transmission can be vertical (transovarian) but also occurs via direct or indirect contact with infected birds (respiratory or fecal) or contaminated feed, water, or litter. Infection transmitted via egg or hatchery contamination usually results in death during the first few days of life up to 2-3 weeks of age. Transmission between farms is due to poor biosecurity.

Clinical Findings

Pullorum disease usually causes very high mortality (potentially approaching 100%) in young chickens within the first 2-3 weeks of age. The disease may be seen in all age groups, but birds <4 weeks old are most commonly affected. Birds may die in the hatchery shortly after hatching..

Affected birds huddle near a heat source, are anorectic, appear weak, depressed and have whitish fecal pasting around the vent (diarrhea). In addition, the birds may have respiratory disease, blindness, or swollen joints.

Survivors are small in size and frequently become asymptomatic carriers with localized infection of the ovary. Some of the eggs laid by such hens hatch and produce infected progeny.

Lesions

There may be no lesions due to an acute septicemia and death. Lesions in young birds usually include unabsorbed yolk sacs and classic gray nodules in the liver, spleen, lungs, heart, gizzard, and intestine. Firm, cheesy material in the ceca (cecal cores) and raised plaques in the mucosa of the lower intestine are sometimes seen. Occasionally, synovitis is prominent. Adult carriers usually have no gross lesions but may have nodular pericarditis, fibrinous peritonitis, or hemorrhagic, atrophic, regressing ovarian follicles with caseous contents. In mature chickens, chronic infections produce lesions indistinguishable from those of fowl typhoid.

Figure 12. Granulomatous hepatitis in chicken liver due to *S*. Pullorum infection

Diagnosis

Lesions may be highly suggestive, but diagnosis should be confirmed by isolation, identification, and serotyping of *S.* Pullorum. Infections in mature birds can be identified by serologic tests, followed by necropsy evaluation complemented by microbiologic culture and typing for confirmation.

Control

Freedom from infection and elimination of positive birds and flocks is key to control. Treatment will not eliminate the carrier state and is therefore never recommended.

Control is based on routine serologic testing of breeding stock to assure freedom from infection. In addition, management and biosecurity measures should be taken to reduce the introduction of *S.* Pullorum from feed, water, wild birds, rodents, insects, or people. Birds should be purchased from sources free of *S.* Pullorum.

3.2.1. Fowl Typhoid

Etiology

The causal agent of fowl typhoid is *Salmonella* Gallinarum. Although *S.* Gallinarum is egg-transmitted and produces lesions in chicks and poults similar to those produced by *S.* Pullorum, there is a much greater 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

Fowl typhoid may be acute or chronic. Clinical signs and lesions in young birds are similar to those seen with *S.* Pullorum infection. Older birds may be pale, dehydrated, and have diarrhea.

Lesions in older birds may include: a swollen, friable, and often bile-stained liver, with or without necrotic foci; an enlarged spleen and kidneys; anemia; enteritis.

Diagnosis

Clinical signs and lesions of fowl typhoid are similar to *S.* Pullorum infection; therefore, diagnosis is confirmed by isolation, identification, and serotyping of *S*. Gallinarum.

Treatment and Control

Control measures are based on elimination of the disease, therefore treatment is never recommended.

Vaccines (killed or modified live) made from a rough strain of *S. enterica* Gallinarum (9R) had variable results in controlling mortality. The standard serologic tests for Pullorum disease also detect fowl typhoid.

3.2.3. Paratyphoid Infections in Poultry

Paratyphoid infections caused by non-host-adapted *Salmonella* are of public health significance because of contamination and mishandling of poultry products. *S. enterica* Enteritidis is a major food safety concern in the egg-laying industry. Fecal contamination of the eggshell is the main mode of transmission from breeders to progeny, although *S*. Enteritidis is also transovarially transmitted.

Etiology

Paratyphoid infections can be caused by any one of the many non-host-adapted salmonellae. These *Salmonella* infect many types of birds, mammals, reptiles, and insects. Paratyphoid infections are of public health significance via contamination and mishandling of poultry products. *S.* Typhimurium, *S.* Enteritidis, *S.* Kentucky, and *S.* Heidelberg are among the most common *Salmonella* infections in

poultry. Some serotypes are more pathogenic than others, and the prevalence of the serotypes varies widely by geographic location.

Transmission

Transmission usually occurs horizontally from infected birds, contaminated environments, or infected rodents. Except for *S.* Enteritidis, transmission of most serotypes to progeny from infected breeders is mainly through fecal contamination of the eggshell. *S.* Enteritidis can infect the interior of the egg through transovarial transmission. Infected birds remain carriers.

Clinical Findings and Lesions

Clinical signs of paratyphoid infection are seen in young birds, but clinical disease is not usually found in mature poultry. Mortality in young birds is most often limited to the first few weeks of age.

Although these clinical signs are not distinctive, hallmarks of the disease include: depression, poor growth, weakness, diarrhea and dehydration.

S. Enteritidis may cause decrease in feed consumption, diarrhea and a decrease in egg production in layers.

Lesions

Lesions in young birds may include an enlarged liver with focal necrosis, unabsorbed yolk sac, enteritis with necrotic lesions in the mucosa, and cecal cores. Infections occasionally localize in the eye or synovial tissues. Conversely, there may be no lesions due to acute death caused by septicemia.

Diagnosis

Isolation, identification, and serotyping of the causal agent are essential for diagnosis. Serology is not highly reliable.

Treatment and Control

Antibiotic use is not recommended. Several antibacterial agents help prevent mortality but cannot eliminate flock infection and may lead to drug resistance.

Control measures for paratyphoid *Salmonella* include sanitation in the hatchery and poultry houses; elimination of vectors such as wild birds, rodents, pets, and flies; feed management; vaccination; and use of competitive exclusion products. Due to transovarial transmission of *S.* Enteritidis, additional control measures include depopulation of infected breeder flocks and refrigeration of eggs. Complete protection is not afforded by vaccination, and it should be used in combination with other control measures to reduce the incidence of *Salmonella* infection.

3.2.4. Colibacillosis (Colisepticemia, *Escherichia coli* Infection)

Colibacillosis is a localized or systemic infection caused by avian pathogenic *Escherichia coli* (APEC). It manifests in diverse ways, including as acute fatal septicemia, subacute pericarditis, airsacculitis, salpingitis, peritonitis, and cellulitis. It is one of the most commonly occurring and economically devastating bacterial diseases of poultry worldwide.

Etiology and Pathogenesis

Colibacillosis is caused by avian pathogenic *Escherichia coli* (APEC). Though many *E. coli* are not pathogenic, some have acquired virulence factors, greatly increasing their pathogenicity. The majority of cases of colibacillosis appear to be due to *E. coli* that have acquired a number of virulence genes. Other cases are due to infection with commensal *E. coli* that gain access to birds weakened by some predisposing condition such as mycoplasmosis, infectious bronchitis, Newcastle disease, hemorrhagic enteritis, poor air quality, or other environmental stresses.

Whereas the majority of APEC were previously assigned to three main serogroups, O1, O2, and O78, more recent research has shown that there is great diversity in the serogroups of APEC causing colibacillosis.

APEC are generally nontoxigenic.

Large numbers of *E. coli* are maintained in the poultry house environment through fecal contamination. Initial exposure to APEC may occur in the hatchery from infected or contaminated eggs. The bacterial portal of entry into birds varies but can include the respiratory tract, skin trauma, cloaca, damaged intestinal mucosa, and navel. From these entry sites, *E. coli* can extend locally or gain access to the bloodstream to cause colisepticemia, which may progress from acute septicemia to death. Infection can also extend to serosal surfaces to cause subacute polyserositis and chronic granulomatous inflammation.

Clinical Findings and Lesions

Signs are nonspecific and vary with age, organs involved, and concurrent disease. Young birds dying of acute septicemia have few lesions except for an enlarged, hyperemic liver and spleen with increased fluid in body cavities. Birds that survive septicemia develop subacute fibrinopurulent airsacculitis, pericarditis, perihepatitis, and lymphocytic depletion of the bursa and thymus (unusually pathogenic salmonellae produce similar lesions in chicks). Although airsacculitis is a classic lesion of colibacillosis, it is unclear whether it results from primary respiratory exposure or from extension of serositis. Sporadic lesions include pneumonia, arthritis, osteomyelitis, peritonitis, and salpingitis.

Diagnosis

Isolation of a pure culture of *E. coli* from heart blood, liver, or typical visceral lesions in a fresh carcass indicates primary or secondary colibacillosis. Pathogenicity of isolates is established using multiplex PCR panels for plasmid-mediated virulence genes or when parenteral inoculation of young chicks or poults results in fatal septicemia or typical lesions within 3 days. Pathogenicity can also be detected by inoculation of chicken embryos.

Treatment and Control

Treatment of colibacillosis with antimicrobial agents is problematic due to widespread multidrug resistance among APEC and restrictions on antimicrobial use in poultry imposed by regulation and public concern. Most isolates are resistant to tetracyclines, streptomycin, and sulfa drugs, although therapeutic success can sometimes be achieved with tetracycline. Most APEC isolates are resistant to five or more antibiotics. APEC also show widespread resistance to disinfectants, further complicating control of colibacillosis.

Prevention of colibacillosis relies on good management to decrease exposure of birds to APEC and lessen the impact of stress and predisposing infections on the susceptibility of birds to APEC infection. In addition, experimental and commercial vaccines of various types have been used to prevent colibacillosis, to mixed effect.

3.2.5. Fowl Cholera

Fowl cholera is a contagious, bacterial disease of birds caused by *Pasteurella multocida*. Acutely, it causes elevated mortality. Chronically, it causes lameness, swollen wattles, and torticollis, but it can also be asymptomatic.

Fowl cholera is a contagious, bacterial disease that affects domestic and wild birds worldwide. It usually occurs as a septicemia of sudden onset with high morbidity and mortality, but chronic and asymptomatic infections also occur.

Etiology

Fowl cholera is caused by *Pasteurella multocida*. *P. multocida* is considered a single species although it includes three subspecies: *multocida*, *septica*, and *gallicida*. Subspecies *multocida* is the most common cause of disease, but *septica* and *gallicida* may also cause cholera-like disease.

In freshly isolated cultures or in tissues, the bacteria have a bipolar appearance when stained with Wright's stain. Although *P. multocida* may infect a wide variety of animals, strains isolated from

nonavian hosts generally do not produce fowl cholera. Strains that cause fowl cholera represent a number of immunotypes (or serotypes). *P. multocida* can be subgrouped by capsule serogroup antigens into five capsular types (A, B, C, D, and F) and into 16 somatic serotypes. Turkeys and waterfowl are more susceptible than chickens, older chickens are more susceptible than young ones, and some breeds of chickens are more susceptible than others.

Transmission

Chronically infected birds and asymptomatic carriers are considered to be major sources of infection. Wild birds may introduce the organism into a poultry flock, but mammals (including rodents, pigs, dogs, and cats) may also carry the infection. Dissemination of *P. multocida* within a flock and between houses is primarily by excretions from the mouth, nose, and conjunctiva of diseased birds that contaminate their environment. In addition, *P. multocida* survives long enough to be spread by contaminated crates, feed bags, shoes, and other equipment. The infection does not seem to be egg-transmitted.

Clinical Findings

Clinical findings from fowl cholera vary greatly depending on the course of disease. In acute fowl cholera, finding a large number of dead birds without previous signs is usually the first indication of disease. Mortality often increases rapidly. In more protracted cases, depression, anorexia, mucoid discharge from the mouth, ruffled feathers, diarrhea, and increased respiratory rate are usually seen. Pneumonia is particularly common in turkeys.

In chronic fowl cholera, signs and lesions are generally related to localized infections of the sternal bursae, wattles, joints, tendon sheaths, and footpads, which often are swollen because of accumulated fibrinosuppurative exudate. There may be lameness, as well as exudative conjunctivitis and pharyngitis. Torticollis may result when the meninges, middle ear, or cranial bones are infected.

Figure 13. Swollen wattles, chronic fowl cholera infection

Lesions

Lesions observed in peracute and acute forms of the disease are primarily vascular disturbances. These include general passive hyperemia and congestion throughout the carcass, accompanied by enlargement of the liver and spleen. Petechial and ecchymotic hemorrhages are common, particularly in subepicardial and subserosal locations. Increased amounts of peritoneal and pericardial fluids are frequently seen. In addition, acute oophoritis with hyperemic follicles may be observed. In subacute cases, multiple, small, necrotic foci may be disseminated throughout the liver and spleen.

In chronic forms of fowl cholera, suppurative lesions may be widely distributed, often involving the respiratory tract, the conjunctiva, and adjacent tissues of the head. Caseous arthritis and productive inflammation of the peritoneal cavity and the oviduct are common in chronic infections. A fibrinonecrotic dermatitis that includes caudal parts of the dorsum, abdomen, and breast and involves the cutis, subcutis, and underlying muscle has been observed in turkeys and broilers. Sequestered necrotic lung lesions in poultry should always raise suspicion of cholera.

Diagnosis

Although the history, signs, and lesions may aid field diagnosis, *P. multocida* should be isolated, characterized, and identified for confirmation. *P. multocida* can be readily isolated from viscera of birds dying from peracute/acute fowl cholera, whereas isolation from suppurative lesions of chronic cholera may be more difficult. At necropsy, bipolar microorganisms may be demonstrated by the use of Wright's or Giemsa stain of impression smears obtained from the liver in the case of acute cholera. In addition, immunofluorescent microscopy and in situ hybridization have been used to identify *P multocida* in infected tissues and exudates.

PCR has been used for the detection of *P. multocida* in pure and mixed cultures and clinical samples. A multiplex PCR has been developed that can differentiate between different somatic serotypes and may enable more efficient vaccine development.

Serology may be used to evaluate vaccine responses but has very limited value for diagnostic purposes.

Treatment

A number of drugs will lower mortality from fowl cholera; however, deaths may resume when treatment is discontinued, showing that treatment does not eliminate *P. multocida* from a flock. Eradication of infection requires depopulation and cleaning and disinfection of buildings and equipment. The premise should then be kept free of poultry for a few weeks.

When antibiotics are used, early treatment and adequate dosages are important. Sensitivity testing often

aids in drug selection and is important because of the emergence of multiresistant strains. Sulfamethazine or sulfadimethoxine in feed or water usually controls mortality. Sulfas should be used with caution in breeders because of potential toxicity and cannot be used in hens laying eggs for human consumption. High levels of tetracycline antibiotics in the feed (0.04%), drinking water, or administered parenterally may be useful.

Prevention

Good management practices, including a high level of biosecurity, are essential to prevention. Rodents, wild birds, pets, and other animals that may be carriers of *P. multocida* must be excluded from poultry houses. The organism is susceptible to ordinary disinfectants, sunlight, drying, and heat.

Both attenuated live vaccines and adjuvanted bacterins are available to aid in prevention. Adjuvant bacterins are widely used and generally effective. Because bacterins are only effective in preventing disease caused by the same serotypes included in the vaccine, somatic serotyping is important. Thus, it is important to know the most prevalent serotypes within an area. Autogenous bacterins are recommended when polyvalent bacterins are found to be ineffective.

Attenuated live vaccines are available for administration in drinking water to turkeys and by wing-web inoculation to chickens. These live vaccines can effectively induce immunity against different serotypes of *P. multocida*. They are recommended for use in healthy flocks only.

3.2.6. Infectious Coryza

Infectious coryza is an acute respiratory disease of chickens characterized by decreased activity, nasal discharge, sneezing, and facial swelling that occurs worldwide. The disease apparently affects only chickens.

Etiology

The causative bacterium of infectious coryza is *Avibacterium paragallinarum* (previously *Haemophilus paragallinarum*) that requires nicotinamide adenine dinucleotide (V-factor) for culture. The most commonly used serotyping scheme is the Page scheme, which groups *A. paragallinarum* isolates into three serovars (A, B, and C) that correlate with immunotype specificity.

Epidemiology and Transmission

Chronically ill or healthy carrier birds are the reservoir of infection for *A. paragallinarum*. Chickens of all ages are susceptible; however, susceptibility increases with age. The incubation period is 1-3 days with a typical disease duration of 2-3 weeks. Duration of illness may be longer in the presence of

concurrent diseases such as mycoplasmosis.

Infected flocks are a constant threat to uninfected flocks. Transmission is by direct contact, airborne droplets, and contamination of drinking water. Transmission does not occur via eggs.

Clinical Findings

In the mildest form of infectious coryza, the only signs may be listlessness, a serous nasal discharge and occasionally slight facial swelling. With increased severity extreme swelling of one or both infraorbital sinuses with edema of the surrounding tissues may prevent the eyes from fully opening. In adult birds, especially males, the edema may extend to the intermandibular space and wattles. The swelling usually abates in 10–14 days; however, if secondary infection occurs it can persist for months. There may be varying degrees of rales depending on the extent of infection. Egg production may be delayed in young pullets and severely reduced in producing hens. Affected birds may have diarrhea and feed and water consumption usually is decreased during acute stages of the disease.

Figure 14. Infectious coryza, facial swelling

Lesions

In acute cases, only the infraorbital sinuses may be involved and contain copious, grayish, semifluid exudate evident on gross inspection and during histopathologic examination. With chronicity this exudate may become consolidated. Histopathologic features include edema, hyperplasia and erosion of respiratory mucosal and glandular epithelia and edema with infiltration of heterophils, macrophages, and mast cells. Other lesions may include conjunctivitis, tracheitis, bronchitis, and airsacculitis, particularly if other pathogens are involved.

Diagnosis

Isolation of a gram-negative, satellitic, catalase-negative organism from chickens in a flock with a

history of a rapidly spreading disease is diagnostic for infectious coryza. Polymerase chain reaction (PCR) testing of live flocks assay has been reported to provide more accurate results versus to bacterial culture. A real-time version of the PCR assay is available. Production of typical signs after inoculation with nasal exudate from infected into susceptible chickens is also reliable diagnostically. No suitable serologic test exists.

Treatment

Because early treatment is important, immediate administration of medication via drinking water is recommended until medicated feed is available. Erythromycin and oxytetracycline are usually effective. Additionally, several newer-generation antimicrobials (eg, fluoroquinolones, macrolides) are active against infectious coryza. Various sulfonamides, including trimethoprim-sulfamethoxazole, and other drug combinations have been successful for treatment. Antimicrobial use in chickens is subject to national regulations that vary from country to country, and use and efficacy must be reviewed in light of relevant laws. In more severe outbreaks, although treatment may result in improvement, the disease may recur when medication is discontinued.

Preventive medication may be combined with a vaccination program if started pullets are to be reared or housed on infected premises.

Control

Prevention is the only sound method of control for infectious coryza. All-in/all-out flow of animals as part of sound farm management and biosecurity practices are important disease prevention measures. Replacement chickens should be raised on the same farm or obtained from clean flocks. If replacement pullets are to be placed on a farm that has a history of infectious coryza, bacterins/vaccines are available to help prevent and control the disease. Because serovars A, B, and C are not cross-protective, it is essential that bacterins contain the serovars present in the target population.

Vaccination on individual farms should be completed ~4 weeks before infectious coryza outbreaks typically occur. Controlled exposure to live organisms also has been used to produce protective immunity in layers in endemic areas.

3.2.7. *Mycoplasma gallisepticum* Infection in Poultry (Chronic Respiratory Disease, Infectious Sinusitis)

Mycoplasma gallisepticum causes respiratory infections in chickens, turkeys, and other avian species. Morbidity is typically high and mortality low in affected flocks, and signs are generally more severe in turkeys.

M. gallisepticum is commonly involved in the polymicrobial "chronic respiratory disease" in broiler chickens, leading to increased condemnations in the processing plant. In layers and breeders, it is usually subclinical, but causes a reduction in the number of eggs laid per hen over the production cycle. These diseases affect chickens and turkeys worldwide, causing the most significant economic losses in large commercial operations, and are commonly seen in noncommercial flocks.

M. gallisepticum is the most pathogenic avian mycoplasma. Integral membrane surface proteins (adhesins) that attach to receptors on host cells, allowing for colonization and infection, are important virulence factors involved in antigenic variation and immune evasion.

Epidemiology and Transmission

M. gallisepticum is transmitted vertically within some eggs (transovarian) from infected breeders to progeny, and horizontally via infectious aerosols and through contamination of feed, water, and the environment, and by human activity on fomites (shoes, equipment, etc). Infection may be latent in some birds for days to months, but when birds are stressed horizontal transmission may occur rapidly via aerosols and the respiratory route, after which infection and clinical disease spread through the flock. Once individuals or flocks are infected, they remain infected for life and act as carriers or reservoir for infection. Flock-to-flock transmission occurs readily by direct or indirect contact from the movement of birds, people, or fomites from infected to susceptible flocks.

Backyard flocks and multiple-age layer flocks may serve as potential reservoirs of *M.* gallisepticum. In many outbreaks, the source of infection is unknown. Cold weather, poor air quality or crowding, concurrent infections, and some live virus vaccinations may facilitate infection, disease, and transmission.

Epithelium of the conjunctiva, nasal passages, sinuses, and trachea are most susceptible to initial colonization and infection; however, in severe, acute disease, infection may also involve the bronchi, air sacs, and occasionally lungs. Once infected, birds may remain carriers for life. There is a marked interaction (polymicrobial disease) between respiratory viruses, *E. coli*, and *M. gallisepticum* in the pathogenesis and severity of chronic respiratory disease.

Clinical Findings and Lesions

In chickens, *M. gallisepticum* infection may be inapparent or result in varying degrees of respiratory distress, with slight to marked rales, difficulty breathing, coughing, and/or sneezing. Morbidity is high and mortality is low in uncomplicated cases. Nasal discharge and conjunctivitis with frothiness about the eyes may be present. Feed efficiency and weight gains are reduced. Commercial broiler chickens may

suffer high condemnations at processing due to airsacculitis. In laying flocks, there may be a chronic increase in mortality and a decrease in the overall production rate.

Uncomplicated *M. gallisepticum* infections in chickens result in relatively mild catarrhal sinusitis, tracheitis, and airsacculitis. *E. coli* infections are often concurrent and result in severe air sac thickening and turbidity, with exudative accumulations, adhesive pericarditis, and fibrinous perihepatitis. Microscopically, involved mucous membranes are thickened, hyperplastic, necrotic, and infiltrated with inflammatory cells. The mucosal lamina propria contains focal areas of lymphoid hypoplasia and germinal center formations.

Diagnosis

History, clinical signs, and typical gross lesions may be suggestive of *M. gallisepticum* infection. Serology by agglutination and ELISA methods are commonly used for surveillance. Hemagglutination-inhibition is used as a confirmatory test. *M. gallisepticum* should be confirmed by isolation from swab samples of infraorbital sinuses, nasal turbinates, choanal cleft, trachea, air sacs, lungs, or conjunctiva. Colonies on agar medium are used for species identification by immunofluorescence with species-specific antibodies.

Mycoplasma isolates must be identified by species, because birds may also be infected with nonpathogenic mycoplasmas.

Because of the fastidious nature of *Mycoplasma* and the difficulty of isolation, molecular diagnostic tests are becoming the most common method for detection and characterization of *Mycoplasma* infections in poultry. Real-time PCR is becoming the most common test used for diagnosis. It is a sensitive, specific, and fast detection test for *M. gallisepticum* and can be performed directly on clinical swabs taken from infected sites (eg, choana, sinuses, trachea, airsacs). Sequence typing by targeting and amplifying a specific sequence allows for differentiation between *M. gallisepticum* isolates and can be particularly useful for epidemiologic investigations and to identify the source of infection.

Treatment

Most strains of *M. gallisepticum* are sensitive to a number of broad-spectrum antibiotics, including tylosin, tetracyclines, and others but not to penicillins or those that act on the cell wall. Tylosin or tetracyclines have been commonly used to reduce egg transmission or as prophylactic treatment to prevent respiratory disease in broilers and turkeys. Antibiotics may alleviate the clinical signs and lesions but do not eliminate infection. Regulations on the use of antibiotics in food animals are rapidly evolving and should be consulted before use.

Control and Prevention

Control is achieved by good biosecurity and sourcing stock from *M. gallisepticum*-free breeder flocks. Prevention is based largely on obtaining chicks or poults from *M. gallisepticum*-free breeder flocks. The most effective control program is to establish *M. gallisepticum*-free breeder flocks, managed and maintained under good biosecurity to prevent introductions, and monitored regularly with serology to continually confirm infection-free status. In valuable breeding stock, treatment of eggs with antibiotics or heat has been used to eliminate egg transmission to progeny.

Laying chickens free of *M. gallisepticum* are desirable, but infection in commercial multiple-age egg farms where depopulation is not feasible is a problem. Inactivated, oil-emulsion bacterins are available and help prevent egg production losses but not infection. Some live vaccines are in use in certain countries. A commercial recombinant fowlpox-*M. gallisepticum* vaccine has been marketed.

3.3. Aspergillosis (Brooder Pneumonia, Mycotic Pneumonia, Pneumomycosis)

Aspergillosis is a disease, usually of the respiratory system, of chickens and turkeys. Severe outbreaks usually occur in birds 7–40 days old.

Etiology and Epidemiology

Aspergillus fumigatus is a common cause of aspergillosis. However, several other mold species may be incriminated, such as *A. flavus*, *A. niger*, *Rhizopus* spp, *Mucor* spp, and on rare occasions, *Penicillium* spp.

High mortality rates can be seen in chicks and poults that inhale large numbers of spores during hatching or when placed on bedding contaminated with mold spores. In older birds, infection is caused primarily by inhalation of spore-laden dust from contaminated litter, feed, or dusty range areas.

Clinical Findings and Lesions

The most common clinical signs of aspergillosis include: dyspnea, labored breathing, fever, inappetence and emaciation.

Less frequently, a neurologic form might present, with clinical signs that include torticollis and tremors. In chickens and turkeys, the lungs and airsacs are most frequently involved. Pulmonary lesions are commonly characterized by white to yellow plaques and nodules a few millimeters to several centimeters in diameter. In rare cases, birds may present with diffuse pulmonary congestion only. Occasionally, mycelial masses may be seen within the air passages on gross examination.

Common histopathologic lesions can include granulomatous pneumonia with intralesional fungal hyphae and heterophilic infiltrates. In addition, plaques and nodules also may be found in the trachea, syrinx, liver, intestines, and occasionally the brain.

Morbidity can be underestimated in finishing flocks until processing, when airsacculitis can be the cause of postmortem condemnation in poultry intended for the food supply.

An ocular form is seen in chickens and turkeys as mycotic keratitis, in which large plaques may be expressed from the medial canthus.

Figure 15. Severe granulomatous fungal pneumonia

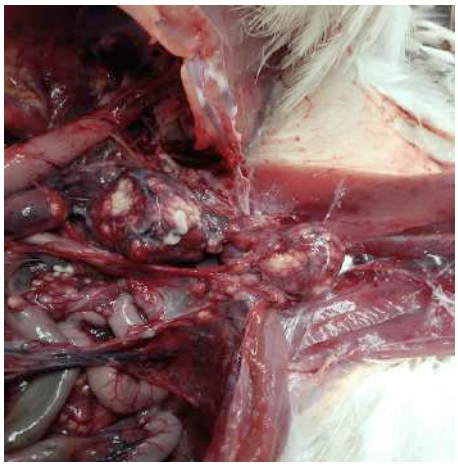

Figure 16. Diffuse granulomatous fungal airsacculitis

Diagnosis

Diagnosis of aspergillosis is most frequently based on the clinical presentation and gross lesions. For confirmation, the presence of mold in the affected organs can be demonstrated by culture or by microscopic examination of fixed tissue. There are several ways to obtain a sample of affected tissue: a piece of affected tissue may be excised, the lesion(s) may be sampled using a swab, or one of the plaques can be teased apart. Most commonly, the sample would then be placed on Sabouraud-Dextrose agar or some other medium specific for the growth of mold. Histopathologic examination using a special fungal stain reveals granulomas containing mycelia.

Treatment and Control

Treatment of affected birds for aspergillosis is generally ineffective. Spontaneous recovery can occur if reexposure to the mold is prevented.

Mitigation strategies can include: 1) removing the birds from the contaminated environment; 2) removal of contaminated material(s) to limit further exposure; 3) trying not to disturb the contaminated material(s) in order to limit further aerosolization of spores; and 4) increased ventilation or air exchange rates to possibly minimize the severity of the outbreak. Strict adherence to cleaning and disinfection procedures for any contaminated environment (eg, hatchery, barn, etc.) will minimize the risk of future outbreaks.

Grossly contaminated or cracked eggs should not be set for incubation because they enable bacterial and fungal growth. Affected eggs may explode and disseminate spores throughout the hatching machine. The use of moldy bedding or ranges should be avoided to prevent outbreaks. Contaminated surfaces may be sprayed or fumigated with enilconazole or other fungicidal disinfectant following the label directions.

3.4. Coccidiosis

Etiology and Epidemiology

Coccidiosis is caused by protozoa of the phylum Apicomplexa, family Eimeriidae. Most species affecting poultry belong to the genus *Eimeria* and infect various intestinal sites. The disease course is rapid (4–7 days) and is characterized by parasite replication in host cells with extensive damage to intestinal mucosa. Coccidia in poultry are generally host-specific, and the different species infect specific portions of the intestine. Coccidiosis occurs worldwide.

Clinical disease occurs only after ingestion of relatively large numbers of sporulated oocysts by susceptible birds (eg, those that are immunosuppressed and/or with concurrent disease). Both clinically infected and recovered birds shed oocysts in feces, which contaminate feed, dust, water, litter, and soil. Oocysts may be transmitted via equipment and personnel (eg, shoes) as well as the presence of insects (eg, flies) and rodents. Fresh oocysts are not infective until they sporulate; under optimal conditions (21°–32°C with adequate moisture and oxygen), this requires 1-2 days. The prepatent period is 4-7 days. Sporulated oocysts may survive for long periods, depending on environmental factors. Oocysts are resistant to some disinfectants commonly used around livestock but are killed by freezing or high environmental temperatures.

Pathogenesis

Pathogenicity of coccidiosis is influenced by host genetics, nutritional factors, concurrent diseases, age of the host, and species of the coccidium. *Eimeria necatrix* and *E. tenella* are the most pathogenic in chickens, because schizogony occurs in the lamina propria and crypts of Lieberkühn of the small intestine and ceca, respectively, and causes extensive hemorrhage. Most species develop in epithelial

cells lining the villi.

Protective immunity usually develops in response to moderate and continuing infection. True age-related immunity does not occur, but older birds are usually more resistant than young birds because of earlier exposure to infection.

Clinical Findings and Lesion

Signs of coccidiosis range from decreased growth rate to many sick birds, with severe diarrhea and high mortality. Decreased feed and water consumption, weight loss, and decreased egg production, may accompany outbreaks. Mild infections which would otherwise be classified as subclinical, may potentially lead to secondary infection, particularly *Clostridium* spp. infection. Birds that survive infections typically recover in 10–14 days but may never recover full growth and production. The lesions are almost entirely in the intestinal tract and often have a distinctive location and appearance that is useful in diagnosis.

E. tenella infections are found only in the ceca and can be recognized by accumulation of blood in the ceca. Cecal cores, which are accumulations of clotted blood, tissue debris, and oocysts, may be found at necropsy in birds surviving the acute stage.

E. necatrix produces major lesions in the proximal and mid portions of the small intestine. Small, white spots, usually intermingled with rounded, bright- or dull-red spots of various sizes, can be seen on the serosal surface. This appearance is sometimes described as "salt and pepper." The white spots are diagnostic for *E. necatrix* if clumps of large schizonts can be demonstrated microscopically. In severe cases, the intestinal wall is thickened, and the infected areas are dilated to 2–2.5 times the normal diameter. The lumen may be filled with blood, mucus, and fluid. Fluid loss may result in marked dehydration. Although the damage is in the small intestine, the sexual phase of the life cycle is completed in the ceca. Oocysts of *E. necatrix* are found only in the ceca. Because of concurrent infections, oocysts of other species may be found in the area of major lesions, complicating the diagnostic process.

E. acervulina is the most common cause of infection. Lesions include numerous whitish, oval or transverse patches in the upper half of the small intestine, which may be easily distinguished on gross examination. The clinical course in a flock is usually protracted and results in poor growth, an increase in culls, and slightly increased mortality.

E. brunetti is found in the lower small intestine, rectum, ceca, and cloaca. In moderate infections, the

mucosa is pale and disrupted but lacking in discrete foci, and it may be thickened. In severe infections, coagulative necrosis and sloughing of the mucosa occurs throughout most of the small intestine.

E. maxima develops in the small intestine, where it causes dilatation and thickening of the wall, petechial hemorrhage, and a reddish orange/pink viscous fluid exudate. The midgut serosa often has numerous whitish pinpoint foci, and may appear engorged. The oocysts and gametocytes (particularly macrogametocytes), present in the lesions, are distinctly large.

E. mitis affects the distal small intestine. Lesions are indistinct but may resemble moderate infections of *E. brunetti*. *E. mitis* can be distinguished from *E. brunetti* by finding small, round oocysts associated with the lesion.

E. praecox, which infects the proximal small intestine, does not cause distinct lesions but may impair growth. The oocysts are larger than those of *E. acervulina* and are numerous in affected areas. The intestinal contents may be watery. *E. praecox* is considered to be of less economic importance versus other species.

E. hagani and *E. mivati* develop in the proximal small intestine. The lesions of *E. hagani* are indistinct and difficult to characterize. However, *E. mivati* may cause severe lesions similar to those of *E. acervulina*. It is unclear whether *E. mivati* and *E. hagani* are separate species or variations in size of other known *Eimeria* spp.

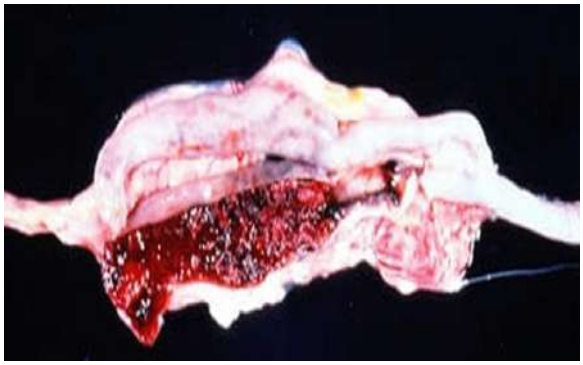

Figure 17. *Eimeria tenella*, gross lesions (hemorrhage), cecum

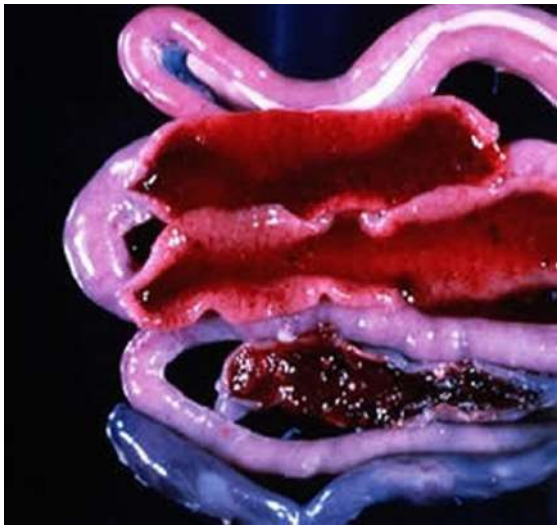

Figure 18. *Eimeria necatrix*, gross lesions (hemorrhage), midgut

Diagnosis

Diagnosis is based on the location of lesions in the host and their appearance; the size of oocysts present is used to determine the species. Coccidial infections are readily confirmed by demonstration of oocysts in feces or intestinal scrapings; however, the number of oocysts present has little relationship to the extent of clinical disease. Severity of lesions as well as knowledge of flock appearance, morbidity, daily mortality, feed intake, growth rate, and rate of lay are important for diagnosis. Necropsy of several fresh specimens is advisable. Classic lesions of *E. tenella* and *E. necatrix* are pathognomonic, but infections with other species are more difficult to diagnose. Comparison of lesions and other clinical signs allows a reasonably accurate differentiation of the coccidial species. Mixed coccidial infections are common.

A diagnosis of clinical coccidiosis is warranted if oocysts, merozoites, or schizonts are seen microscopically and if lesions are severe. Subclinical coccidial infections may be unimportant, and poor performance may be caused by other flock disorders.

Control

Practical methods of management cannot prevent coccidial infection. Poultry maintained at all times on wire floors to separate birds from feces have fewer infections; clinical coccidiosis is seen only rarely under such circumstances. Other methods of control are vaccination or prevention with anticoccidial drugs.

Vaccination

A species-specific immunity develops after natural infection, the extent largely depends on the severity of infection and the number of reinfections. Protective immunity is primarily a T-cell response.

Commercial vaccines consist of live, sporulated oocysts of the various coccidial species administered at low doses. Modern anticoccidial vaccines should be given to day-old chicks, either at the hatchery or on the farm. Because the vaccine serves only to introduce infection, chickens are reinfected by progeny of the vaccine strain on the farm. Most commercial vaccines contain live oocysts of coccidia that are not attenuated. The self-limiting nature of coccidiosis is used as a form of attenuation for some vaccines rather than biologic attenuation. Some vaccines sold in Europe and South America include attenuated lines of coccidia.

Layers and breeders maintained on floor litter must have protective immunity.

Anticoccidial Drugs

Many products are available for prevention or treatment of coccidiosis in chickens and turkeys.

Anticoccidials are given in the feed to prevent disease and the economic loss often associated with subacute infection. Prophylactic use is preferred, because most of the damage occurs before signs become apparent and because drugs cannot completely stop an outbreak. Therapeutic treatments are usually given by water. Antibiotics and increased levels of vitamins A and K are sometimes used in the ration to improve rate of recovery and prevent secondary infections.

Continuous use of anticoccidial drugs promotes the emergence of drug-resistant strains of coccidia. Various programs are used in attempts to slow or stop selection of resistance. For instance, producers may use one anticoccidial continuously through successive flocks, change to alternative anticoccidials every 4–6 months, or change anticoccidials during a single growout (ie, a shuttle program). Although there is little cross-resistance to anticoccidials with different modes of action, there is widespread resistance to most drugs.

Coccidia can be tested in the laboratory to determine which products are most effective. "Shuttle programs," in which one group of chickens is treated sequentially with different drugs (usually a change between the starter and grower rations), are common practice and offer some benefit in slowing the emergence of resistance.

Most anticoccidials currently used in poultry production are coccidiocidal.

The natural development of immunity to coccidiosis may proceed during the use of anticoccidials in the feed. However, in the production of broilers during a short growout of 37–44 days, this may be of little consequence. Natural immunity is important in replacement layer pullets, because they are likely to be exposed to coccidial infections for extended periods after termination of anticoccidial drugs. Anticoccidial programs for layer and breeder flocks are intended to allow immunizing infection while guarding against acute outbreaks.

Anticoccidials are commonly withdrawn from broilers 3–7 days before slaughter to meet regulatory requirements and to reduce production costs. Because broilers have varying susceptibility to infection at this point, the risk of coccidiosis outbreaks is increased with longer withdrawal.

Amprolium is an antagonist of thiamine (vitamin B_1). Rapidly dividing coccidia have a high requirement for thiamine. Because amprolium has poor activity against some *Eimeria* spp, its spectrum has been extended by using it in mixtures with the folic acid antagonists ethopabate and sulfadimethoxine. The primary use of amprolium currently is for water treatment during clinical outbreaks.

Clopidol (eg, decoquinate) is coccidiostatic against early development of *Eimeria* spp. by inhibiting mitochondrial energy production. Clopidol has a broad species spectrum.

Folic acid antagonists include the sulfonamides (not all legally approved) and ethopabate. These compounds are structural antagonists of folic acid or of para-aminobenzoic acid (PABA), which is a precursor of folic acid. (The host does not synthesize folic acid and has no requirement for PABA.) Coccidia rapidly synthesize nucleic acids, accounting for activity of PABA antagonists. Although resistance to antifolate compounds is widespread, they are commonly used for water treatment when clinical signs are already evident. Ormetoprim is active against the protozoan enzyme dihydrofolate reductase. It has synergistic activity with sulfonamides and often is used in mixtures with these compounds.

Halofuginone hydrobromide is related to the antimalarial drug febrifuginone and is effective against asexual stages of most species of *Eimeria*. It has both coccidiostatic and coccidiocidal effects, but coccidia may become resistant after extended exposure.

The ionophores (monensin, salinomycin, lasalocid, narasin, and maduramicin) form complexes with various ions, principally sodium, potassium, and calcium, and transport these into and through biologic membranes. The ionophores affect both extra- and intracellular stages of the parasite, especially during the early, asexual stages of parasite development. Drug tolerance was slow to emerge in chicken coccidia. Recent surveys suggest that drug tolerance is now widespread, but these products remain the most important class of anticoccidials.

Some ionophores may depress feed consumption when the dosage is above recommended levels. But the reduced feed consumption may be offset by improved feed conversion.

Nicarbazin was the first product to have truly broad-spectrum activity and has been in common use since 1955. Nicarbazin is toxic for layers, causing mottling of egg yolks, decreased egg production, and blanching of brown egg shells. A 4-day withdrawal period is required in broilers. Medicated birds are at increased risk of heat stress in hot weather.

Robenidine, a guanidine compound, allows initial intracellular development of coccidia but prevents formation of mature schizonts. It is coccidiostatic when given short term and coccidiocidal long term. Drug resistance may develop during use. A 5-day withdrawal period is needed to eliminate untoward flavor caused by residues in poultry meat.

Diclazuril is highly effective against a broad spectrum of coccidia. It is used mostly for prevention at 1

ppm in the feed.

4. Vaccination

Learning outcomes

- Able to select vaccination strategy suited for the flock
- Thorough understanding of potential causes of vaccine failure in poultry

Intensification of poultry production increases the challenge of diseases and disease control as the housing of this system places birds in proximity to each other and increases the risk of rapid spread of infectious diseases. Thus, poultry producers must constantly vaccinate birds to minimize the threat of disease outbreaks. Proper management of flock health by biosecurity and vaccination is one of the critical factors in profitable poultry production (Sharma, 1999).

A vaccination program for a given area should be tailored to local conditions including the type of production, flock history, endemic infectious agents, local pattern of disease, the level of biosecurity being practiced, vaccine availability, vaccine cost, the burden of disease, the presence of other infections that may interfere with vaccination, and the resources available to deliver the vaccine itself (Sharma, 1999; Marangon and Busani, 2006).

Certain viral and bacterial agents are endemic in poultry-producing areas and tend to cause recurring infections. Flocks must be routinely protected against these common pathogens. The most common method of protection is vaccination. Both live attenuated and inactivated or killed vaccines are available against poultry diseases. Most live vaccines are either mild isolates that induce a protective immune response against their pathogenic counterparts or pathogenic agents that have been attenuated. Both active and passive immunization are practiced to control diseases in poultry. Passive immunization is necessary when early post-hatch protection against an infection is critical, as with IBD. The protection is achieved by hyperimmunizing hens so that protective levels of maternal antibody are transmitted to progeny chickens (Sharma, 1999).

Vaccines are administered to eggs, that is, *in ovo* vaccination, or to chicks after they have hatched. Post-hatch delivery systems include aerosol, spray, drinking water, eye drop, and injection. In *in ovo* vaccination, the vaccine is injected in eggs during later stages of embryonation, usually at 17-18 days of incubation (Sharma and Burmester, 1982). *In ovo* vaccination is advantageous because the vaccinal protection is well established by the time the chick hatches and is first exposed to environmental pathogens. It also substantially reduces the labor cost associated with individual handling of chicks in the conventional vaccination procedure (Sharma, 1999).

There are three vaccination strategies that may be appropriate in different situations (Marangon and Busani, 2006):

- Routine vaccination can be the tool of choice in areas where the disease is endemic. It is effective in reducing mortality and production losses, and it could also contribute to eradication programmes.
- Emergency vaccination is an option whenever a new infectious disease is introduced in a previously unaffected area, and the epidemiological situation indicates that there could be massive and rapid spread of infection.
- Preventive vaccination is a measure that may be applied wherever a high risk of introduction and further spread of a contagious poultry disease, that has a clear impact on the industry, has been identified.

Table 1. Vaccination schedule practiced by Debre Zeit Agricultural Research Center (DCARC) (Habte et al., 2017)

Vaccine Type	**Age of bird**	**Route of administration**
Marek's disease	Day 1	Subcutaneous
Newcastle disease HB1 vaccine	Day 3	Eye drop
IBD Vaccine	Day 7 and 21	Eye drop/water
LaSota Newcastle disease vaccine	At day 27, 63, 112 and every 3 month	Eye drop/water
Fowl typhoid vaccine	At 6 and 12 weeks of age	Subcutaneous
Fowlpox vaccine	From day 70-90	Wing web

Table 2. Vaccination programs practiced in broiler and layer chicken flocks (Sharma et al., 1999)

Birds being vaccinated	Vaccine	Age when vaccine is administered
Broiler chickens	Marek's disease	*In ovo*
	Infectious bronchitis and Newcastle disease	Day 1, day 14
	Infectious bursal disease	Day 21
Layer chickens	Marek's disease	Day 1
	Infectious bursal disease	Week 2, Week 6, Week 12
	Newcastle disease and infectious bronchitis	Week 2, Week 6, Week 12
	Infectious laryngotracheitis and fowlpox	Week 12
	Mycoplasma gallisepticum	Week 15

4.1. Vaccine failure

(Adapted from: Sharif and Ahmad, 2018)

Even if vaccination is an effective means of disease prevention method, its failure may happen due to a range of different factors. Vaccination is said to be good if the immune response to the vaccine is protective and stable for the targeted period of time. Vaccine failure is the consequence of the inability of the chicken to develop adequate immunity after immunization or susceptibility of birds to field outbreak after administration of vaccine. High rates of vaccination failures have been recorded in vaccinated poultry flocks. The common breaches in transportation, handling, storage and administration of vaccines are responsible for high rates of vaccine failure in poultry flocks in developing countries.

The causes of vaccine failure can be categorized into two major factors: antigen factor and host response.

The protective vaccine antigen is of prime importance in the production of effective vaccine. The titer of antigen in the vaccine may be low to initiate protective immune response in the birds resulting in low immunity level. Association between virus concentration and immunogenicity of vaccines has been established. Aside from the titer of antigens in the vaccine, there are certain factors which may cause reduction in optimal vaccine dosage, such as use of chlorinated water for vaccination and use of vaccine for more birds than recommended.

The local disease causing agents in any area are of prime importance for vaccine manufacturing. The local serotypes/strains and locally isolated agents are considered the most suitable immunogens for

formulating vaccines. Failure to use local isolates may result in vaccine failure and disease outbreaks. Foreign vaccines may be made from serotypes that are different from field strain.

In the poultry sector, almost all the vaccines available are thermolabile in nature. The maintenance of proper cold chain during transportation and appropriate storage temperature is a prerequisite for optimal potency of vaccines. Apart from temperature, exposure of vaccines to direct sunlight should be avoided, as the UV radiation of the sunlight may degrade the antigens present in the vaccine and make it ineffective. The potency of vaccines is maintained to a certain period of time, provided that the transportation and storage temperature is properly maintained. The use of vaccines after the date of expiry may not result in optimal immune response.

Some of the viruses like the influenza virus are of a mutating nature, and as a result pose a serious threat regarding the effectiveness of vaccines in the control of diseases caused by such agents. A vaccine produced from a current strain/isolate may fail to protect a mutant arising in the future.

Birds vaccinated against diseases may not respond effectively due to various reasons resulting in vaccine failure. Stress of any origin can result in vaccine failure because it may reduce the chicken's ability to mount an effective immune response. Certain diseases such as mycotoxicosis, IBD, Marek's disease, etc. are immune-suppressive in poultry. These immune suppressive diseases may also lead to vaccine failure. Moreover, any other disease condition may also contribute to vaccine failure. Therefore, it is highly important that vaccination should be done in healthy birds. Vaccination of sick and diseased birds may not result in desired outcome. It may rather lead to extra stress and an increased susceptibility resulting in morbidity and mortality.

A vaccine may fail to fully protect against a disease if the birds were infected prior to, or soon after, vaccination because the antibodies produced against the pathogenic agent (due to the infection) will neutralize the antigen of the vaccine. If chickens were already incubating the disease at the time of vaccination they may still develop the disease because there may be inadequate time for antibody production to reach protective levels.

The previous exposure status of the bird to a pathogen, and passive protection, may affect the response to vaccination. Passive immunity results from passage of maternal immunity to chicks and this can influence the response to vaccination. If the breeder flock has high levels of circulating antibodies which pass to the progeny through the egg, this may interfere with the replication of live vaccines. This will decrease the immune response to the vaccine because it is not stimulating the immune system for the necessary duration or extent. Therefore, to induce higher protection levels it is necessary to accurately

follow vaccination program designed for each particular area (Habte et al., 2017). Vaccination at a very early age before the development of certain receptors may also result in vaccine failure.

Vaccination of chickens can fail to result in protective immunity if the vaccine is not administered correctly. Routes of vaccination affect the outcome of the vaccines. For example, sometimes when mass vaccination strategies using drinking water or feed are implemented, some birds may remain unvaccinated if they fail to consume adequate water/ food, and these unvaccinated individuals may cause of vaccination failure in the flock. Therefore, it is important to consider uniformity of the flock before starting a mass vaccination program. Live vaccines, which allow some lateral spread of the immunizing virus among birds, reduce the necessity for uniformity at time of application. The diluents used for live virus vaccines are very important to ensure that an adequate vaccine dose reaches the birds (Habte et al., 2017).

Some of the vaccines require a booster dose for successful immunization. The booster dose is required after 10–20 days of the initial dose. The initial dose is required for priming of vaccine while the booster is required for maximum protection against antigen. The lack of booster dose results in low antibody titers, resulting in vaccine failure.

5. Biosecurity

(Adapted from: Van Meirhaeghe et al., 2019)

Learning outcome

- Sound knowledge of biosecurity

In poultry production disease control is mainly based on prevention. Prophylactic use of antibiotics should be limited because of the risk of antimicrobial resistance. The mainstays of disease prevention are vaccination programmes, good management and biosecurity (Butcher and Miles, 2012).

5.1. General biosecurity principles

Biosecurity in poultry production is the most effective and economic way to control poultry diseases. Basic rules of biosecurity for any poultry unit include correct choice of geographical location of the farm, proper design of the buildings and positioning of equipment, well-planned operational protocols, focusing on potential sources of infection and the flow of people, materials, feed, eggs and flocks to and from the farm. When designing successful biosecurity programmes it is important to include education of all personnel involved in the operations of the unit.

The best strategy when building the new farm is to keep as far away from any other commercial (poultry) farming units and also processing plants as possible. This will limit natural transmission of pathogens. It is also essential to be aware of the prevailing wind currents, as many pathogens are air-borne and may be carried on the wind. Areas with high population of wild birds such as forests, around lakes, and rivers should be avoided because wild birds can be an important source of infection.

Ideally, the buildings should be as far away as possible from roads, along which poultry, feed or litter are transported. The whole farm should be fenced off; access should only be via the gates where everything entering and leaving the farm is recorded and where all vehicles can be disinfected.

Within the unit, there should be a clear separation of the dirty (outside world) and the clean areas, where the animals are kept. The immediate area around the poultry house should be kept clean and free of any vegetation.

The buildings should be secure from wild birds, rodents and insects that may be carriers of *Salmonella*, *Mycoplasma* and other diseases. The entrance to the buildings should be made of solid and cleanable surfaces, and a washbasin should be available at the entrance to wash and disinfect hands and clean clothes and footwear.

Litter and feed storage should be free of wild birds and vermin. Silos for feed storage must be clean and sealed. The whole building and equipment should be conceived in order to allow for easy and efficient cleaning and disinfection. Floors and walls must be smooth and without cracks, the floor should ideally be covered with concrete. Feeding and water lines should be easy to disinfect.

Identification of potential sources of infection and ensuring the proper control of all material and people entering and leaving the poultry unit are the fundamental aspects of biosecurity. A first rule is that diseases must be kept outside. The second important biosecurity rule is that diseases that have been brought inside, also should stay inside.

There are many ways in which micro-organisms can be introduced into farms. One of the potential vectors for the introduction of disease into farms are humans. People can carry pathogens into farms and within farms between houses, on footwear and clothes, hands, and equipment. In order to reduce the risk presented by humans, a general rule is to avoid unnecessary visitors at all times and to restrict access to the farm for any unauthorized people. If a visitor needs to enter the farm, he/she should be registered at the entrance. The visitors should be given clean clothing and boots; they should wash and disinfect their hands and if possible, take a shower. Farm personnel should also change clothes and footwear between houses.

Equipment and delivery vehicles can also be important sources of infection. Trucks that transport poultry, eggs or feed must be cleaned, washed and disinfected each time before loading. The vehicles should be disinfected at the entrance of the farm. Vaccination instruments and other equipment should not be taken inside the house without first being cleaned and disinfected.

Another way that pathogens can be introduced into poultry farms is via feed. It can be contaminated with pathogens (e.g. *Salmonella*) through ingredients, during production, delivery or storage. Trucks transporting feed must be cleaned and disinfected on their return from the farms before they enter feed storage areas in order to avoid cross-contamination. The drivers should not be allowed to enter poultry houses and should wear clean clothes and footwear when they enter the feed storage area on the farm.

Dead chickens are a perfect medium for bacterial growth. Chickens are curious and will pick on dead birds thus spreading more pathogens among the flock. Dead birds must be collected at least twice a day and must be stored in a container far from live birds until properly disposed. Sick birds should also be euthanized and removed to stop continuous infection.

Mice and rats are important transmitters of poultry diseases including *Salmonella* and *Campylobacter*. Feed, manure and rubbish should never be kept outside the house. Insects can also be an important source of infection. Flies and beetles and their larvae can spread pathogens. An efficient and continuous control programme should be set up and continuously monitored.

Once birds have left the house it should be cleaned and disinfected before new chicks arrive. The house must be mechanically cleaned. The litter should be taken out of the house and properly disposed. The surroundings of the building, the roof, outside walls, loading zones and personnel area, must be checked and cleaned to avoid recontamination of the house from outside. Special attention should be paid to the paved area in front of the entrance, as it becomes heavily contaminated when loading birds and removing litter. Water and feed lines must also be thoroughly cleaned. After thorough mechanical cleaning, all surfaces and equipment must be soaked with detergent for at least 20 minutes to remove all organic material. After soaking, the house must be rinsed down with water using high-pressure cleaning equipment.

Disinfectant should be applied by wet spray or foam application on dry surfaces but not later than 24 hours after cleaning. Sufficient time between flocks is needed in order to clean, disinfect and dry out poultry houses. In general, at least one week is recommended as a sanitary void between the flocks.

The drinking lines should be flushed before arrival of chicks and at least once a day during the first week. The water should also be treated starting from the day of chick arrival, if possible. During

vaccination it is important to stop the water treatment 1 day before the vaccination. Treatment can be resumed one day after vaccination. During the sanitary void and the cleaning and disinfection of the houses, the inside of the drinking lines should also be cleaned.

6. Necropsy (Post-Mortem Dissection)

(Adapted from: Morishita, 2019 and vetdiagnostix)

Learning outcomes

- Be able to proficiently conduct systematic dissection on dead or sacrificed birds in order to complement a diagnosis

The necropsy (post-mortem dissection) of poultry is a procedure that can be utilized by the veterinarian, the manager, or the grower to find reasons for the bird's death. Using a knife or scissors, a person can perform a basic necropsy to obtain diagnostic information; samples for further laboratory testing; or to ensure quality control of a flock.

A recently dead or currently ill bird can be chosen for a necropsy. Birds that have been dead for more than several hours are not recommended for diagnostic specimens since the natural decomposition process will create changes that may be confused with true pathological lesions. If a specimen cannot be necropsied immediately, it should be refrigerated until it can be performed within a day. If you choose to euthanize and necropsy a sick bird, first observe it for abnormal breathing patterns, abnormal posture, ruffling of feathers, and/or nasal or ocular discharge before euthanizing the bird.

Although the bird can be humanely euthanized by several approved methods cervical dislocation is the most common and realistic method under Ethiopian condition.

- Describe all lesions with brief notes according to the basic parameters of size, color, consistency, location and distribution.
- Consider submitting digital photographic images of lesions observed.

For post mortem examination on the farm utilize a stable flat surface set at a suitable height in a well-lit but shaded position. Have a basic instrument set to hand (scissors, plane forceps, shears, gloves) with water, soap and disinfectant to clean up afterwards and disposal bags for the carcass material.

Post Mortem Procedure

- Examine birds externally for evidence of trauma, soiling of feathers, vent damage, skin lesions, eye lesions, external parasites, foot pad lesions and joint swelling.

- Examine the oral cavity and conjunctiva and apply pressure to the infraorbital sinus for evidence of nasal discharge.
- Wet down the feathers with a disinfectant solution to limit the distribution of feathers during the dissection.
    - Dampening the plumage with a disinfectant solution is strongly recommended if a controlled disease (Avian Influenza, Newcastle Disease, *Salmonella Enteritidis* / *Gallinarum* / *Pullorum*) is suspected.
- Place the bird on its back with the head away from you. Grasp both legs and push down and away from the pelvis to loosen the joints (hips/ coxo-femoral joints).
- Incise the skin at the thigh-body wall junction, dislocate the coxo-femoral joints (the bird will now lie flat on its back)
- Tent the skin over the abdomen and make a small incision through the skin between the caudal end of the breast bone and the cloaca. Pull the breast skin away from you towards the neck and examine the subcutaneous tissue and breast musculature (Fig. 19).
- Examine the breast muscle for decreased muscle mass, paleness (anemia), or bruising. Examine the keel bone. A crooked keel bone can indicate rickets.
- Push strong scissors/shears through the incision and incise the right side (bird's left) coelomic (abdominal) wall, thoracic cage and coracoid bone. Repeat on the bird's right side.
- Grasp the keel near the abdomen and pull upwards or remove completely to expose the internal organs and chest cavity taking care not to damage the underlying terminal trachea/tracheal bifurcation (Fig. 20).
- Examine the liver for changes in size or discoloration, white or yellow spots, abscesses, and/or tumors. The normal liver should not extend beyond the tip of the keel.

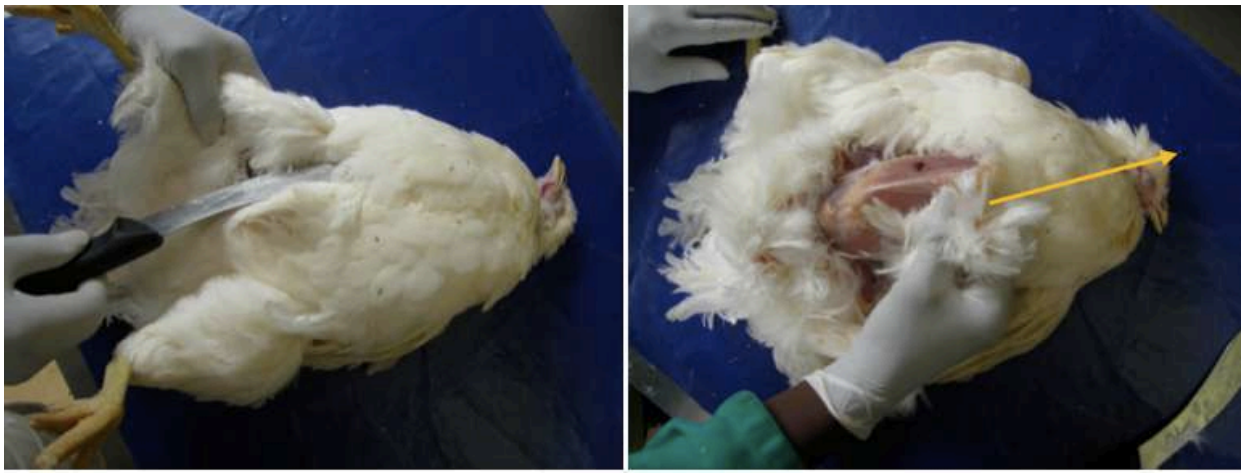

Figure 19. Starting the postmortem examination with skin incision and peeling the skin over the breast

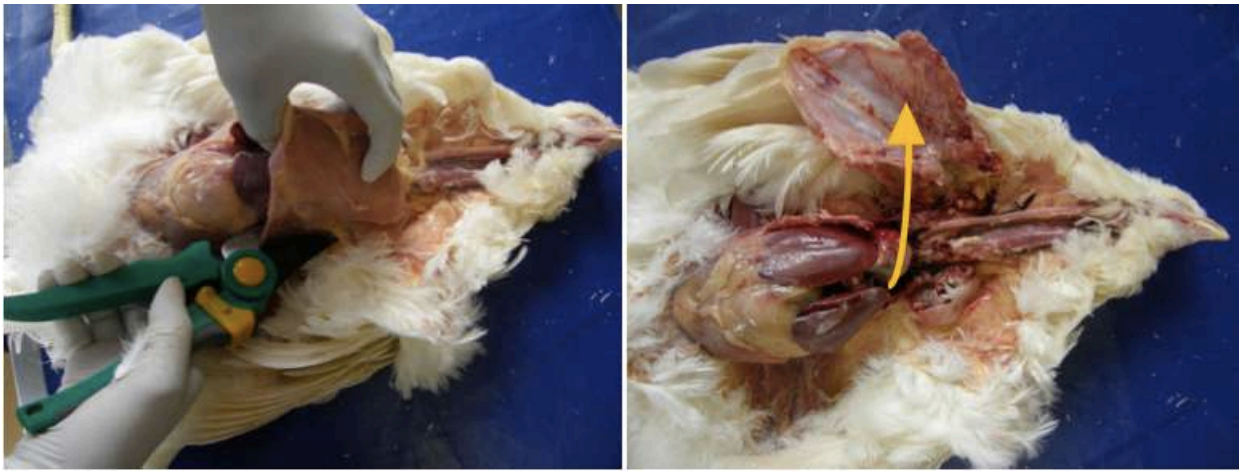

Figure 20. Removal of the breast

- Examine the air sacs for increased thickness and increased cloudiness. The normal air sac surfaces look like soap bubbles or clear cellophane wrap.
- Cut the gastrointestinal (GI) tract between the esophagus and proventriculus. Remove the proventriculus, ventriculus (gizzard), small intestines, large intestine, ceca, and cut off at the level of the cloaca. The pancreas will also be removed.
    - The GIT is gently extended and laid out on the left of the bird for further detailed examination (Fig. 21).
- Cut all attachments close to the intestines and set the GI tract aside. At the end of the necropsy, these organs can be opened up and examined for internal parasites.

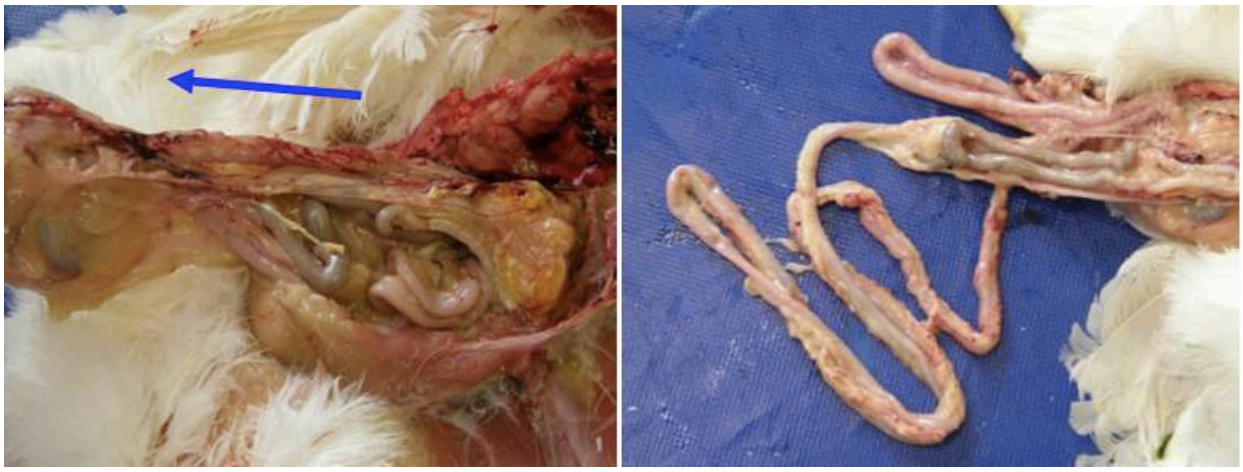

Figure 21. Removal and arrangement of the gastrointestinal tract for examination

- Next, remove the liver and spleen. A green discoloration of the liver near the gall bladder is a normal finding.
    - Carefully undermine and remove the bird's left and right liver lobes and gall bladder (Fig. 22). Rupture of the gallbladder results in release of bile into the coelomic cavity and bile has potent antibacterial properties which can significantly interfere with bacterial culture results.

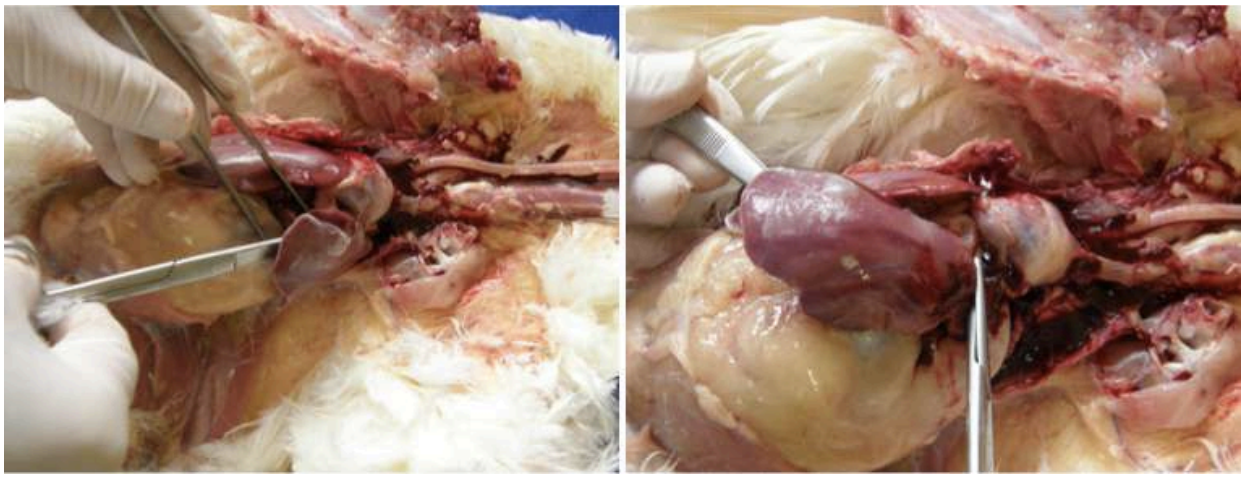

Figure 22. How to remove the liver for examination

- Now you can observe the organs located near the backbone of the carcass.
- Examine the kidneys and the left ovary/oviduct (or paired testes), which are positioned on top of the kidneys.
    - Examine the development and condition of the oviduct, ovary, ovarian follicles or testes with respect to age of the bird (Fig. 23).

- In layer birds, examination of the oviduct for abnormalities and any intra-coelomic eggs for deformities is routinely performed.

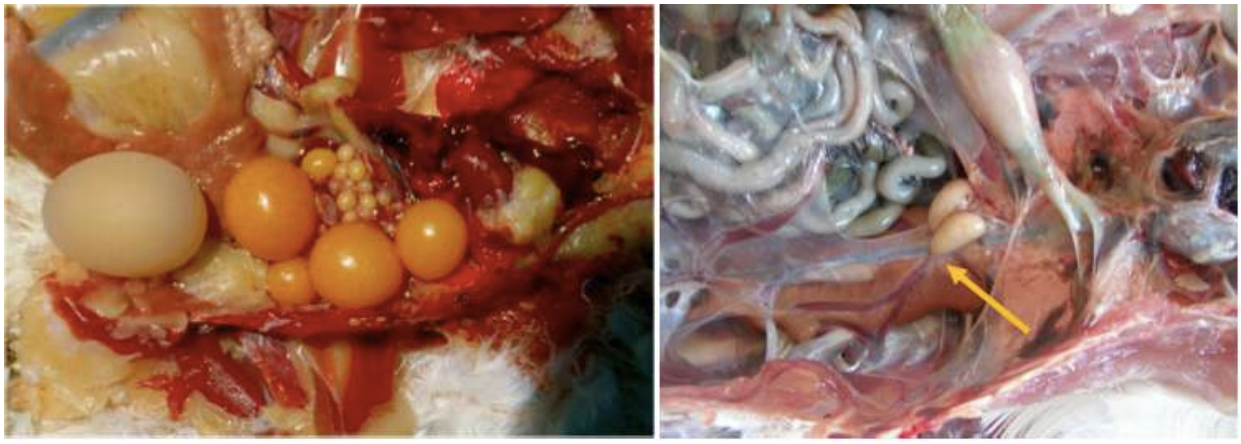

Figure 23. Anatomical locations of the ovary and testes

- The lungs, which are attached to the ribs, can be gently removed from the ribcage for further examination.
- The outer surface of the heart should be examined. A cloudy, thickened surface is suggestive of pericarditis. Also, note if there is excessive fluid located between the heart and the pericardium (membranous covering of the heart).
- Next, turn the bird around to face you and cut through the corner of the beak. Extend the cut through the throat and down towards the heart.
- Examine the interior surface of the esophagus and crop. Look for the presence of food and/or parasites (worms) in the crop. If the inside surface appears to resemble a towel, it may be an indication of a fungal infection called crop mycosis.
- Next cut through the larynx, trachea, and syrinx. The inside surface should be free of excess mucus.
    - A longitudinal section of the trachea from the syrinx (tracheal bifurcation) to the larynx will allow careful examination of the mucosa - with collection of samples by swabbing (for bacterial culture or for PCR) to evaluate for important respiratory pathogens such as avian influenza, Newcastle disease, infectious bronchitis virus, infectious laryngotracheitis virus, *Mycoplasma gallisepticum*, *Avibacterium paragallinarum*
- Turn the bird back to the previous positioning – feet in front of you.
- The sciatic nerve located on the interior upper thigh (located under muscle) should be exposed on both legs. The nerves should be the same size bilaterally with no swellings. Enlargement of this

nerve in one leg can be an indication of Marek's disease.

- This large nerve is positioned on the medial side (inside) of the femur which is covered by a group of three thigh muscles. The nerve lies medial to the femur and below two muscles caudal to the femur.
- Closed scissors, forceps or a finger may be used to separate the two caudal muscles to expose the ischiatic nerve (Fig. 24).
- Ischiatic nerve is the prime site for diagnostic lesions of Mareck's disease.

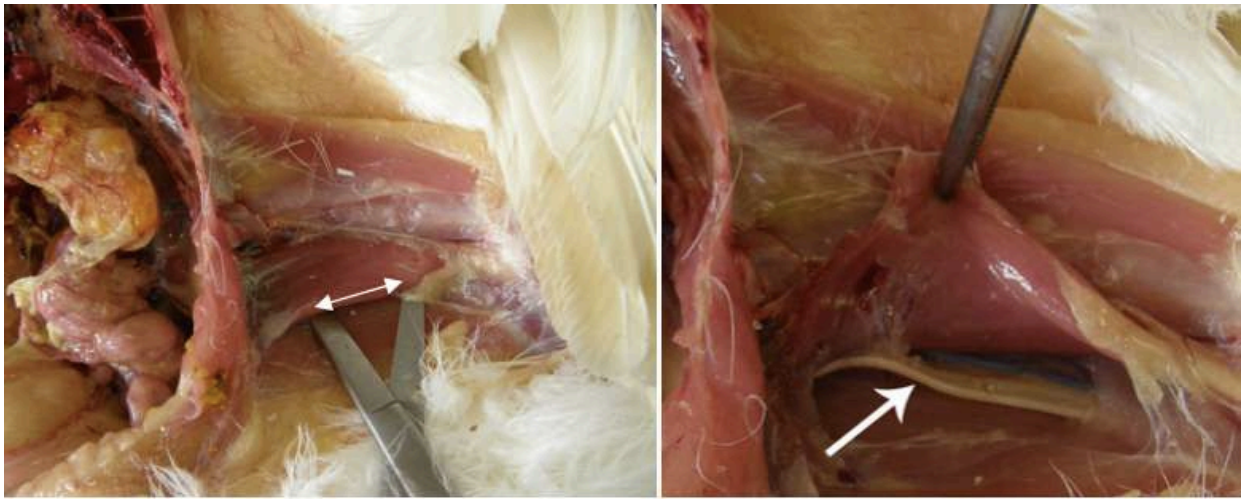

Figure 24. How to expose the ischiatic nerve for examination

- With a sharp knife, cut through the stifle and hock joints, looking for yellow or white pus-like material, blood, or excess fluid. Joints should appear shiny and white with just a small amount of clear, sticky fluid inside.
    - Incise the hock joint from the caudal aspect and the stifle joint from the anterior aspect to examine the joint cartilage and joint fluid (Fig. 25).

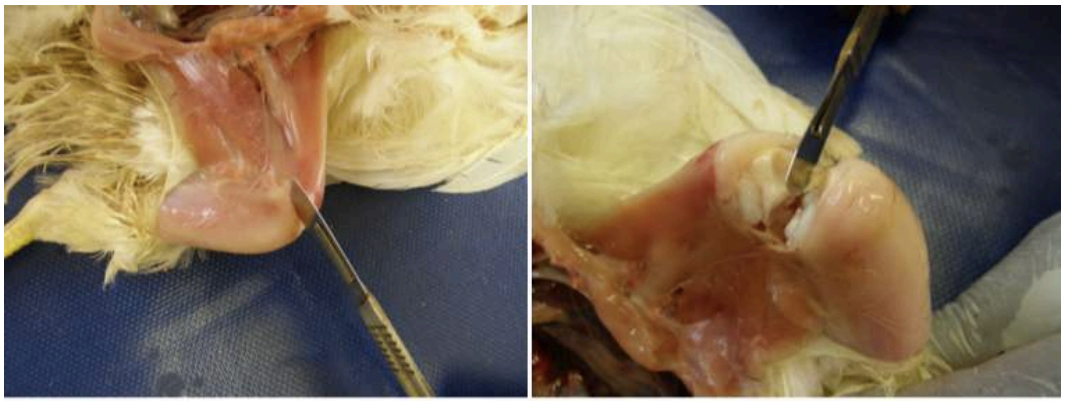

Figure 25. Incision into joints

- To examine the brain,
    - Remove the head at the atlanto-occipital joint, peel the skin towards the beak to expose the skull (Fig. 26).
    - Incise the skull from the occipital foramen forward in two tangential cuts to both orbits where the incisions join (Fig. 26).
    - Remove the excised cranium to expose the brain (27).
    - Gently undermine the brain from the anterior region levering gently upwards and place whole brain into a formalin bottle (Fig. 27).

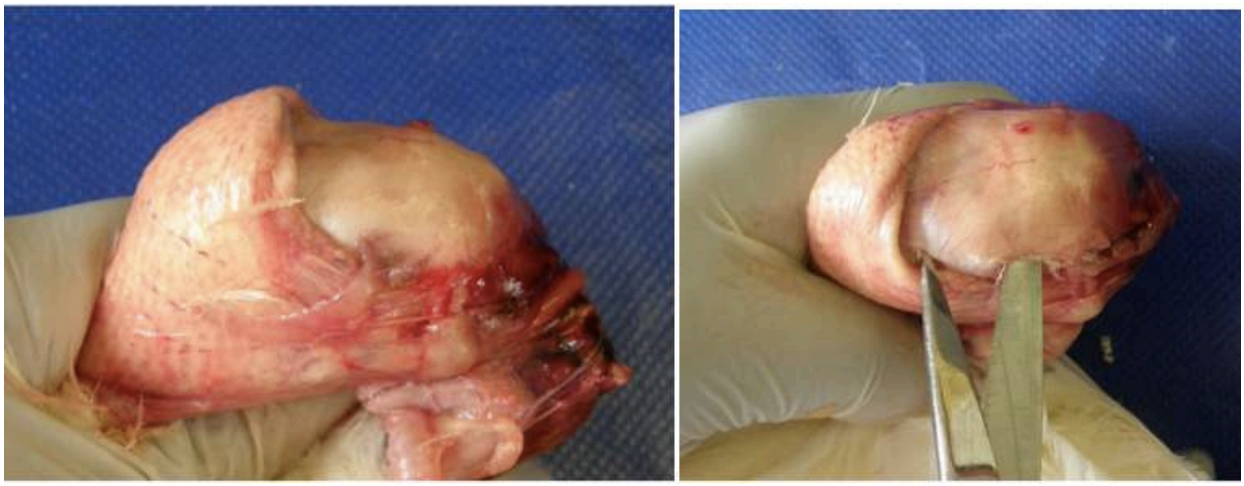

Figure 26. How to remove the head and incise the scull

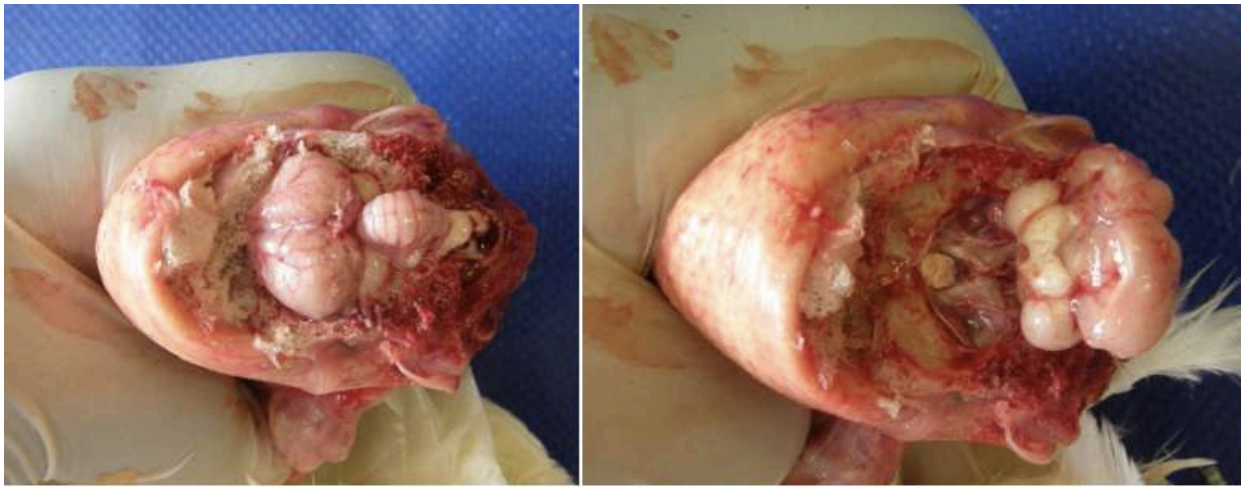

Figure 27. How to remove brain for examination

- To find the bursa of Fabricius, cut through the cloaca and look for a grape-like structure towards

the rear of the bird. The older the bird, the smaller the bursa. The bursa diminishes in size as the bird reaches sexual maturity.

- Cut the bursa in half. It should have wrinkles running parallel to each other on the surface and be cream colored in appearance. Note any discoloration or swelling
- Now return to the GI tract and starting with the proventriculus, cut length-wise. The inside wall is bumpy and this is normal as these are the digestive glands.
- Cut through the ventriculus, intestines, and ceca. Note the appearance of the inside walls (mucosa) and the presence of parasites (worms); blood; and/or a thickened or discolored surface.
- The whole intestine (including caecae) should be opened and the mucosal wall examined for any evidence of coccidia.
    - Upper small intestine (*Eimeria acervulina*): duodenum has transverse white bands to coalescing white plaques on duodenal mucosa. Moderately pathogenic occurring more commonly in older birds and often together with other *Eimerias*.
    - Mid small intestine (*Eimeria necatrix*): mid intestine distended with yellow / orange mucus. White spots (schizonts) and pinpoint hemorrhages. This is a severe pathogen causing high mortality.
    - Mid small intestine (*Eimeria maxima*): mid intestine and macroscopic lesions similar to *E.necatrix*. Very large oocysts are the distinguishing feature. Moderately pathogenic.
    - Lower small intestine and rectum (*Eimeria brunetti*): fibrinous to fibrinonecrotic enteritis of lower small intestine, rectum and proximal part of the cecum, with necrotic core formation. Severe pathogen inducing high mortality in young birds.
    - Cecum (*Eimeria tenella*): cecae filled with necrotic caseous cores. Highly pathogenic in young birds.
    - Examine cecal tonsils (Fig. 28) for hemorrhage- Cecal tonsillar hemorrhages are important indicator lesions for velogenic Newcastle disease and avian influenza.
- Dispose off the carcass properly and disinfect surfaces and tools.

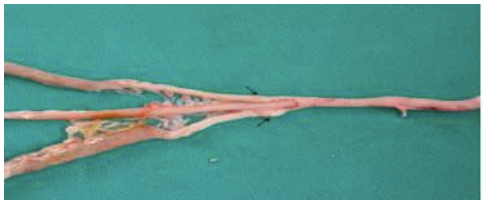

Figure 28. Ceca of chicken

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