

Major Viral Diseases of Poultry



1. *Newcastle Disease*
2. *Marek's Disease*
3. *Lymphoid Leukosis*
4. **Avian Influenza**
5. **Infectious Bursal Disease**
6. **Infectious Bronchitis**
7. **Infectious Laryngotracheitis**
8. **Fowl Pox**
9. **Avian Encephalomyelitis**
10. **Viral arthritis**

NEWCASTLE DISEASE (ND)

- ❖ It is a contagious & widespread disease affecting almost all species of birds. Chickens are the most sensitive, Companion birds, backyard flocks & game fowl serve as reservoirs.
- ❖ **Etiology- Avian paramyxovirus serotype 1 (APMV1)** causing ND outbreak in poultry. 3 strains/virulence:
 - Mild strains (lentogenic) - *used for live vaccines*
 - Medium strength strains (mesogenic) and
 - Virulent strains (velogenic)
- ✓ The pathogenicity depends on: strain & dose of the virus, route of infection and age of the bird.
- ✓ **Pre-existing infections and environmental factors** can exacerbate even the **lentogenic** stain

Transmission:

- ❖ **Aerosols** (inhalation of droplets) for **confined**
- ❖ **Oral** routes (feed & waters) for free ranging birds
- *Wind dispersal may occur over distances of 5 km*
- Spread between farms is by infected equipment, trucks, personnel, wild birds, *Wind*
- ND virus is highly contagious excreted through infected droppings and respiratory discharge 1 - 2 days after infection.
- The incubation period – usually 3 - 6 days (2 -15 days) depending on the virulence, route & dose of infection, immune status of the chicken

Four Major Manifestations/ Clinical Signs - viral stains, dose, route and viral tropism

- ❖ **Velogenic-viscerotropic virus** → Velogenic-viscerotropic ND an acute and highly lethal disease
- ❖ **Velogenic-neutropic virus** → Velogenic-Neurotropic ND which is an acute and fatal in chickens
- ❖ **Mesogenic virus** causes acute, moderately lethal disease, (Variable to high morbidity and moderate mortality), with both nervous and respiratory signs in adult
- ❖ **The lentogenic form** causes mild disease (subclinical disease)/encountered in most poultry-producing areas.

Clinical Signs

- ❖ ***Respiratory signs*** - coughing, marked gasping, dyspnea/labored breathing with wheezing and gurgling
- ❖ ***Nervous signs (Velogenic neurotropic)*** - Paralysis of leg/wing, twisted neck/torticollis are the main signs.
- ❖ ***GIT signs (Velogenic vescerotropic)***-violent greenish or bloody diarrhea
- ❖ Complete cessation/Drop in egg production 30 to 50 % or more, returning to normal levels in about 2-3 weeks is observed. Besides also egg shell quality will be affected (Shell-less, thin, loose color...).

- ✓ **Highly pathogenic strains (velogenic)** of ND cause high mortality with depression and death within 3 to 5 days. Affected chickens do not always exhibit respiratory or nervous signs.
- ✓ **Mesogenic strains** cause typical signs of respiratory distress but Nervous & respiratory signs similar to that of Velogenic strain maybe encountered
- ✓ **Lentogenic strains:** Mild to inapparent respiratory signs, Negligible mortality & ↓ in egg production
- **Lentogenic** NDV strains are mild, but sometimes still cause respiratory signs and death in day old infected chicks and drop in egg production in adults.
- *The NCD-virus can cause in humans only a light inflammation of the mucosa of the eyes and respiratory system.*

Pathology/lesion

Velogenic infection

- 1. Respiratory system:** lesions (Serous or catarrhal exudate) such as rhinitis, laryngitis, tracheitis, pneumonia, clouding of airsacs (may be yellowish or whitish) and petechial bleedings of heart fat
 - ❖ Mucous, marked congestion in the trachea, & lungs
- 2. Digestive system:** Prominent hemorrhages of **proventriculus**, gizzard and gut-associated lymphoid tissue, small intestine and liver
 - *These changes are not specific to vvND and may be observed with highly pathogenic strains of avian influenza and vvIBD*

- ✓ The presence of hemorrhagic lesions in the intestine is important to distinguish VVND from NVND

3. Nervous system: No gross lesion in CNS

- ❖ In laying hens infected with Velogenic viruses, haemorrhages in the ovary, flaccid and degenerated follicles, and egg yolk in the abdomen are commonly seen.
- ❖ **Mesogenic** - lesions are not specific, mostly limited to an inflammation of the respiratory tract
- ❖ **Lentogenic** - Mild conjunctivitis and tracheitis are observed. Recovered flocks show septicemia and airs acculitis due to secondary infection with *E. coli*.



Weakness (no lameness and no stiff neck)

Torticollis

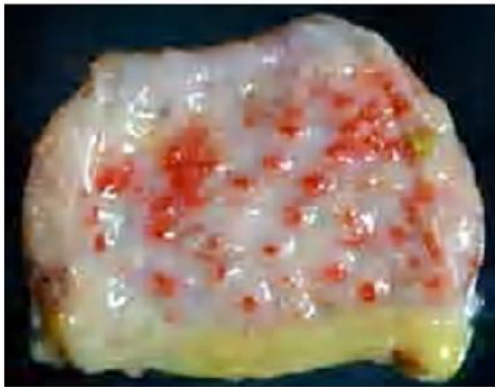


conjunctivitis



Foamy nasal discharge, accumulation of liquid in the lungs

Hemorrhage in proventriculus



Necrosis and hemorrhage in ceca



Proventriculus eroded and covered by a fibrinonecrotic exudate

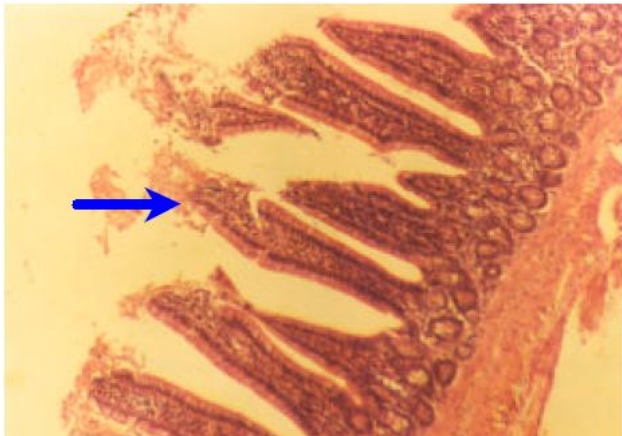
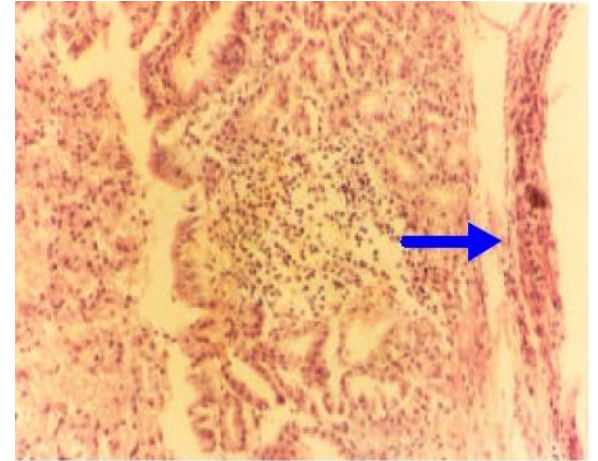
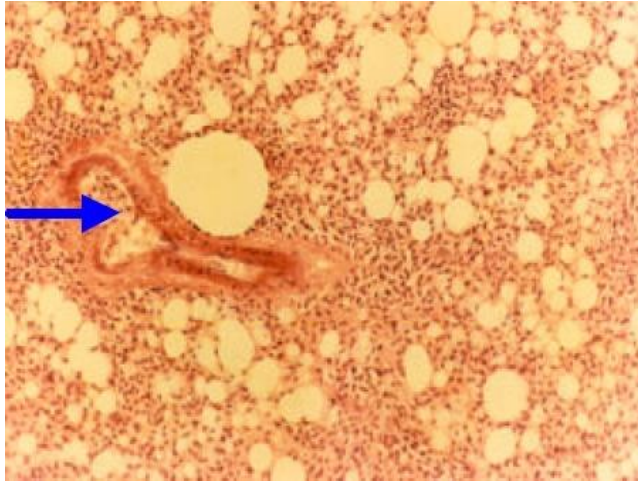
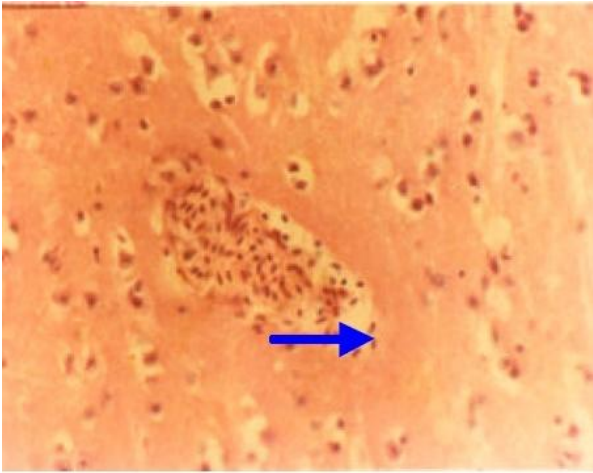


bleeding into the mucosa of the trachea



Bleeding throughout the intestine

Histopathology: *congestion, haemorrhage,*
oedema and lymphocytic infiltration in Brain,
Lungs, Proventriculus & Intestine



Diagnosis: based on history, Clinical signs and PME

✓ **DDX'S: Coryza, CRD, IB , Fowl cholera, ILT and AI**

- Confirmation can be obtained with:

- ❖ Virus isolation and identification or PCR,

- ❖ Serological test: HI or Elisa. HI test is used most widely

- ❖ Animal (4-7 weeks-old susceptible chickens) inoculation

Treatment: no specific treatment for ND; antibiotic treatment of secondary bacterial infections to reduce the losses.

Prevention and control – Eradication programme include:
Strict sanitation, appropriate management (biosecurity, other diseases control, all-in-all out...etc), killing of all infected flocks and a financial compensation to farmers, transport-bans, obligatory vaccination

✓ The best way is; **a very strict Bio-security** combined with a good immunization programme/**vaccination** and monitoring on basis of blood tests.

Vaccine type: 1. Low-virulence/**live-virus** vaccines (drinking water, intraocular , intranasal, spray).

2. Killed-virus oil emulsion vaccines (IM or SC)

3. The V4/I2-strain **live thermostable** mutant ND can be used (in areas with **defective cold-chain**).

NCD vaccine used in Ethiopia: **Live vaccine:** Hitchner B1/Lasota (lyophilized), Ocular route, Oral route and drinking water

Marek's Disease (MD): one of the most common diseases with worldwide distribution.

- ✓ Infection presumably exists in every flock except for those maintained under strict pathogen-free conditions. But, infected flocks do not always show clinical disease.

Etiology: A alphaherpesvirus. Three serotypes **MDV: 1, 2 and 3**, & all can be used for vaccine production.

- **Ocogenic (serotype-1)** - isolated from chicken & has transforming genes. It is the pathogenic one in poultry & has different strains (virulence & pathogenicity).
- **Non oncogenic MDV (serotype2)** - chicken isolate lack transforming genes
- **HVT (serotype3)**-turkey isolate lack transforming genes

Species affected - Poultry are the only important natural host. Also quail, turkeys and pheasants are susceptible.

- ✓ Turkeys are commonly infected with turkey herpes virus, an avirulent strain related to one causing the disease/infection

Source of infection and transmission

- Once infected, chickens continue to be carriers for long periods and act as a source of infectious virus.
- MD is highly contagious & readily transmitted among chickens
- ❖ *Air borne (direct/indirect) within the poultry house*
- ✓ Main transmission is by infected premises, where day-old chicks will become infected by the oral & respiratory routes.
- The virus matures into a fully infectious enveloped form in the epithelium of the feather follicle, from which it is released into the environment.

- It can remain infectious for months or years in litter or dust.
- The incubation period varies from three weeks to months.
- Dander from feather follicles of MD-infected chickens can remain infectious for more than a year and distributed by wind, equipment, and personnel
- Infected birds carry the virus in their blood for life (source of infection for susceptible birds)
- Young chicks are particularly susceptible to horizontal transmission. Susceptibility decreases rapidly after the first few days of age.

Clinical signs – MD affects chickens most between 12 - 24 weeks of age

- The disease has two forms
- **The classical form/neurolymphomatosis:** tumors in leg nerve cause lameness and paralysis; causes a bird to lie on its side with one leg stretched forward and the other backward
- ✓ Torticollis can be seen when the cervical nerves are affected.
- ✓ Respiratory symptoms may be observed too, if the vagus and intercostal nerves are involved.
- Blindness (tumors in the iris)
- Acute MD is an epidemic in susceptible or unvaccinated flocks causing depression paralysis, mortality and lymphomatous infiltrations/tumours in multiple organs.
- Subclinical infections result in impaired immune responses as MDV causes a lytic infection in lymphocytes.
- 10-50% morbidity and mortality up to 100%. Mortality usually occurs between 10 and 24 weeks of age and can reach up to 50% in unvaccinated flocks.

- *When the gizzard nerve is involved, the birds will have a very small gizzard and intestines and will waste away.*
- *Weight loss, paralysis of the wings and legs*

Pathology/Internal Lesions: Swollen nerves are the typical indication of MD

- **Neurological form:** enlargement and lose of striations of vagus, brachial & sciatic nerves
- ✓ No gross lesion in the brain
- **Visceral form-** tumours/nodules are in various organs: liver, spleen, heart, lung, kidney, muscle, proventriculus & gonad/ovaries
- ✓ Many organs may show nodules in the absence of gross nerve lesions. Affected organs show enlargement.
- **Cutaneous form:** Skin involvement often consists of tumors of feather follicles or in between follicles.
- The bursa is seldom tumorous and more frequently is atrophic.
- ❖ Nerve lesions are often unilateral, Note-always observe sciatic and brachial nerves for enlargement
- An irregular constriction of the iris can be seen if the eyes are involved.

Neurological form (progressive paralysis):



Paralysis (loss of muscle function) of wings



The picture shows an enlargement of a sciatic nerve

Twisted neck



Brachial plexus (nerve) is two or three times the normal thickness, swelling caused by fluid (oedema)

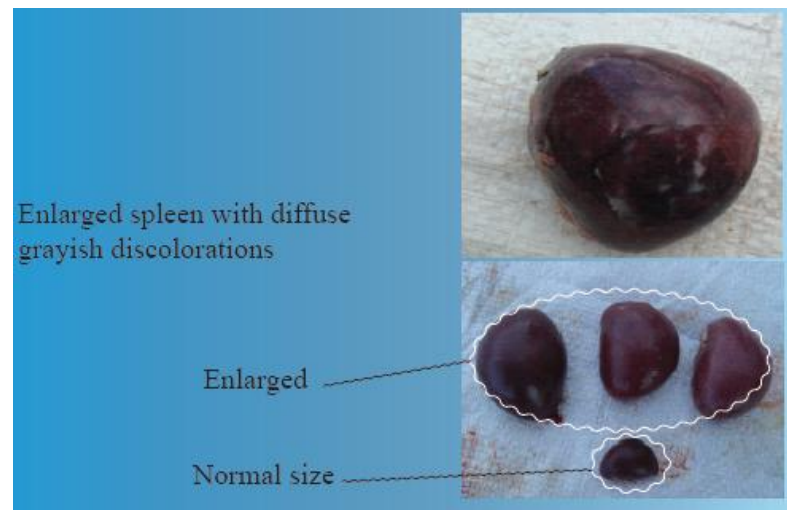
Skin (feather follicles) Lymphomas





Visceral form:

Enlarged liver with diffuse grayish nodules formed by abnormal growth of tissue.



Histopathology: *two main pathologies in the peripheral nerves*

- ***Degenerative /inflammatory type (also called as type B)***
 - ✓ Lymphocytes & plasma cells infiltration into tissue
 - ✓ **Demyelination and Schwann cell proliferation**
- ***Proliferative type (tumor cell infiltration) - type A***
 - ✓ Mass of proliferating lymphoblastic cells into tissue
 - ✓ MD cells (unusual cells with vacuolated cytoplasm and without nuclear details)
 - ✓ Demyelization and Schwann cell proliferation

CNS lesion- Focal perivascular cuffing in the brain

✓ In the spinal cord, infiltration and focal accumulation of lymphocytes in white matter

Eye lesions: Mononuclear cell infiltration into iris, eye muscle and conjunctiva may be involved

Lesions (proliferative lymphomatous) in the visceral organs

✓ Type a lesion is characterized by diffuse proliferation of small to medium size lymphocytes, lymphoblasts, MD cells and plasma cells

Bursal/thymus lesion - Cortical and medullary atrophy, necrosis and cystic cavity development in the medulla

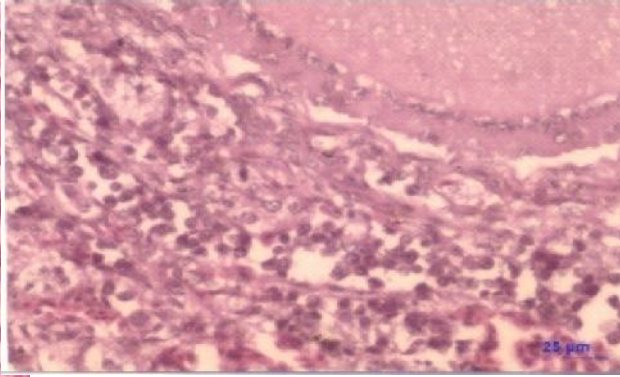
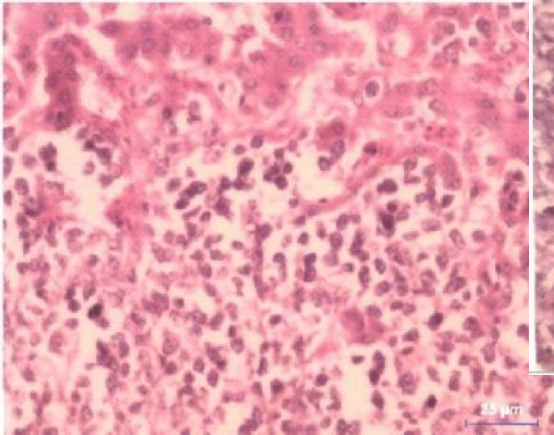
❖ Interfollicular lymphoid infiltration & atrophy of the thymus

Skin lesions - Lesions are largely inflammatory, may be also lymphomatous around feather follicle

❖ Infiltration of mononuclear cells around feather follicle

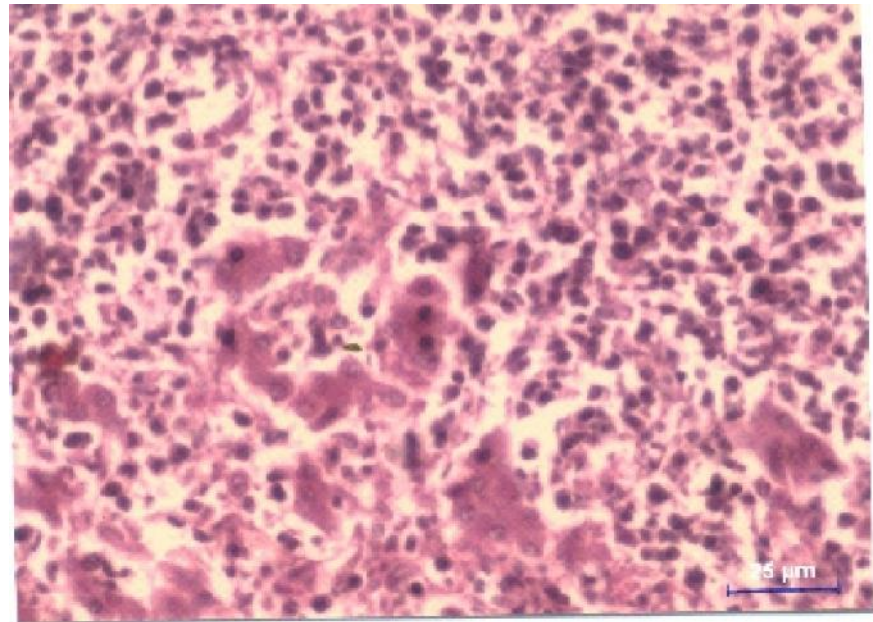
❖ In the dermis, there will be compact aggregation of proliferating cells often perivascular, few plasma cells, and histiocytes

Hematology: Leukocytosis (many T-cells) due to large lymphocytes and lymphoblast cells & Cell pleomorphism



Ovary - Pleomorphic lymphoid cell infiltration H&E

- Liver- Pleomorphic lymphoid cell infiltration



- Lymphoid leucosis - Liver- Multifocal uniform sized lymphoid cell infiltration H&E

Diagnosis: Chickens may be diagnosed positive for MD if at least one of the following is met

1. Lymphoid tumors in various tissues (liver, heart, skin, gonad) -----but they can also be indicative of lymphoid leucosis.
 2. However, Leukotic enlargement of peripheral nerves, either grossly or microscopically, is typical of MD.
 3. Eye involvement can be visible as an irregular constriction of the iris (ocular lymphomatosis) and discoloration
- Virus isolation/tissue culture or PCR from buffy coat (fresh blood samples) and/or affected organs can confirm the infection.

Treatment: no effective treatment for affected flocks.

Control: Hygiene, All-in-all-out, vaccination

- ❖ Vaccination of day-old chicks is an effective method of control
- Vaccination against Marek's disease is performed at hatchery/day old; there are two routes of application; In-ovo injection into the 18 days embryonated eggs or injection (IM or Subcutaneous) in day-old chickens.
- Different MD vaccines exist. Rispens (serotype-1), SB1 (serotype-2) and HVT vaccine strains (serotype-3).
- The choice on the strains for MD vaccination depend on the virulence of the strains present in the field.
- A new generation of vaccine is the polyvalent ones, also used to vaccinate broilers and to broaden the protection

- ✓ It has been demonstrated that MD vaccine only prevents the appearance of Marek's disease tumours and paralysis.
- ✓ It does not prevent the birds from becoming infected with MD-virus.
- It is therefore of major importance to maintain high hygienic and sanitary measures by good management to avoid early exposure of young chicken

Lymphoid Leukosis (Big liver disease/visceral leucosis)

- It is a bursal dependent neoplastic disease of birds.
- **Etiology:** Avian retro/leukosis viruses (subgroup A, B, C,D & J viral envelope glycoproteins).
- ✓ ***J-strains*** is more virulent strains.
- ✓ The virus is not stabile/can readily be inactivated by strict sanitation procedures.
- ✓ The virus causes severe proliferation of the white or red blood cells in lymphoid organs.
- ***Species affected:*** primarily a disease of chickens, lymphoid leukosis can infect turkeys, guinea fowl, pheasants...(14-16 weeks age and older).

Transmission: the virus is transmitted vertically via hatching eggs (Egg to offspring).

- ✓ Chicks infected in *ovo become immunotolerant and* are most likely to shed virus and develop tumors.
- Spread by bird-to-bird contact
- Horizontal infection by contact with contaminated environments----- The horizontally infected chickens produce antibodies and therefore have lesser chance of tumor formation.
- Blood sucking parasites.
- The incubation period is very long (30 - 500 days).
- ***Infected chicken are carriers for life.***

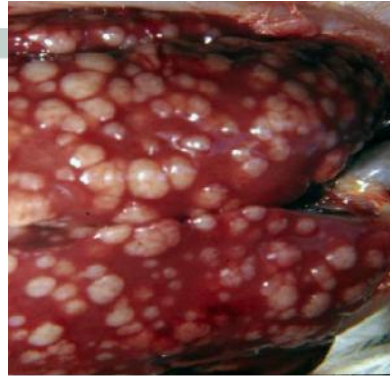
Clinical signs: The virus can affect various organs so naturally the symptoms are diverse.

- ✓ Emaciation/lower egg production
- ✓ Anemic/ Pale shriveled Comb and wattles
- ✓ Enlarged abdomen (**due to ascites**, enlarged liver, spleen, or kidney)
- ✓ Panting and Greenish diarrhea in terminal stages.
- ✓ Sudden death without showing any symptoms.
- ✓ Mortality rarely rises above the 20%

Internal Lesions: enlarged liver, spleen, kidneys, Bursa Fabricius and intestines can be found due to tumor formation (Lesions may be nodular, miliary or diffuse)



Nodular tumor on liver



Splenomegally with multiple foci of lymphoid tumor



Enlarged kidney with multiple gray nodules

Diagnosis: based on history (previous leukosis outbreaks, age, mgt.) and clinical signs

- Confirmed by - histopathology
- ✓ By testing blood samples (monitoring) and virus isolation from hatching eggs and cloacal swabs for the presence of the virus.

- **Differential diagnosis:** Because of the tumours, LL may be confused with Marek's disease, but in LL the nervous system is never involved (no paralysis).

- ✓ fatty liver disease

- ✓ pullorum, T.B.

Treatment and Control

- There is no treatment for Avian Leucosis and until now no vaccine is available.
- Reduction and Elimination of the virus from breeder hens.

- ✓ Test and slaughter

- ✓ By all in all out housing system.

Infectious Bursal Disease (Gumboro disease)

- ✓ **Other names** include infectious bursitis, avian nephrosis
- IBD is an acute, highly contagious and immunosuppressive disease of **young** chickens. IBD is an important viral disease in all major poultry producing areas of the world.
- Clinically it attacks young chickens, usually up to 6 to 8 weeks of age.
- ❖ **Etiology:** Birnavirus. The genome of IBDV consists of two segments of double-stranded RNA
- IBDV has two serotypes
 - ✓ Serotype 1 /Serotype 2

- ❖ **Serotype 1** is most pathogenic & further divided into
 - *Highly pathogenic*
 - *Intermediate plus*
 - *Intermediate*
 - *Low pathogenic and variant strains*
 - ✓ The serotype 1 IBDV isolates infect and destroys actively dividing **IgM-bearing B cells** in the bursa of Fabricius
 - ❖ **Serotype 2**: Turkey strains are apathogenic to chickens.
- Species affected** - Chickens and turkeys appear to be natural hosts.

Transmission: IBD virus is very infectious and spreads easily from bird to bird via

- *Direct contact* of young birds with **infected flocks/bird to bird**
- *Indirect infection* with IBDV (Contaminated equipment, feed, Water, feed, droppings, housing, and clothing of personnel)
- ❖ Houses in which outbreak has been occurred remain infectious for 54 to 122days & Contaminated food and water remain infectious for 52days (so ingestion is the main route)
- ✓ The virus is very stable, highly resistant to physical conditions and chemical agents; therefore it is difficult to eradicate from an infected farm.

Clinical Signs - Clinical IBD occurs usually between 3 and 8 weeks of age depending on maternal antibody levels.

- ❖ Affected birds are listless and depressed, pale,
- ❖ Watery white diarrhea and vent picking
- ❖ New cases of IBD have a mortality rate of about 5-10% but can be as high as 60% depending on the pathogenicity of the strain involved.
- ❖ Highly pathogenic strains are called “very virulent” IBD (vvIBD) resulting in high mortality.
- **Huddling**, Trembling, incoordination
- Subclinical IBD occurs with infections before 3 weeks of age.
- ✓ Early IBD infection result in permanent immunosuppression without mortality.
- ❖ Inflammation followed by atrophy of the bursa of Fabricius
- ❖ Variable degree of immunosuppression
- ✓ Immunosuppression is economically important due to increased susceptibility to secondary infections especially in the respiratory tract.
- In broilers it results in bad performance with lower weight gains and higher feed conversion ratios.
- ❖ Flocks are affected acutely and show variable morbidity (5-50%).
- ❖ Rapidly ascending mortality (5-50%), depending upon the *pathogenicity of the IBDV strain* and the *susceptibility of the flock*

Post mortem lesions/Pathology

- In acute cases, characteristic **enlarged bursa** with gelatinous bursal exudate, severely reddened haemorrhagic bursa
- Muscular (thigh/pectoral) hemorrhages and pale kidneys can be seen.
- Hemorrhages at the junction of the proventriculus & gizzard
- Visceral organs may show enlargement and hemorrhages
- Infection by variant strains is usually accompanied by a fast bursal atrophy (in 24 - 48 hours) without the typical signs of Gumboro disease.
- Also in chronic cases the bursa is smaller than normal (atrophy)
- Recovered birds show bursal atrophy



Normal chicken(left) and
gumboro chicken (right)



Bleeding into skeletal muscles, enlarged bursa of Fabricius



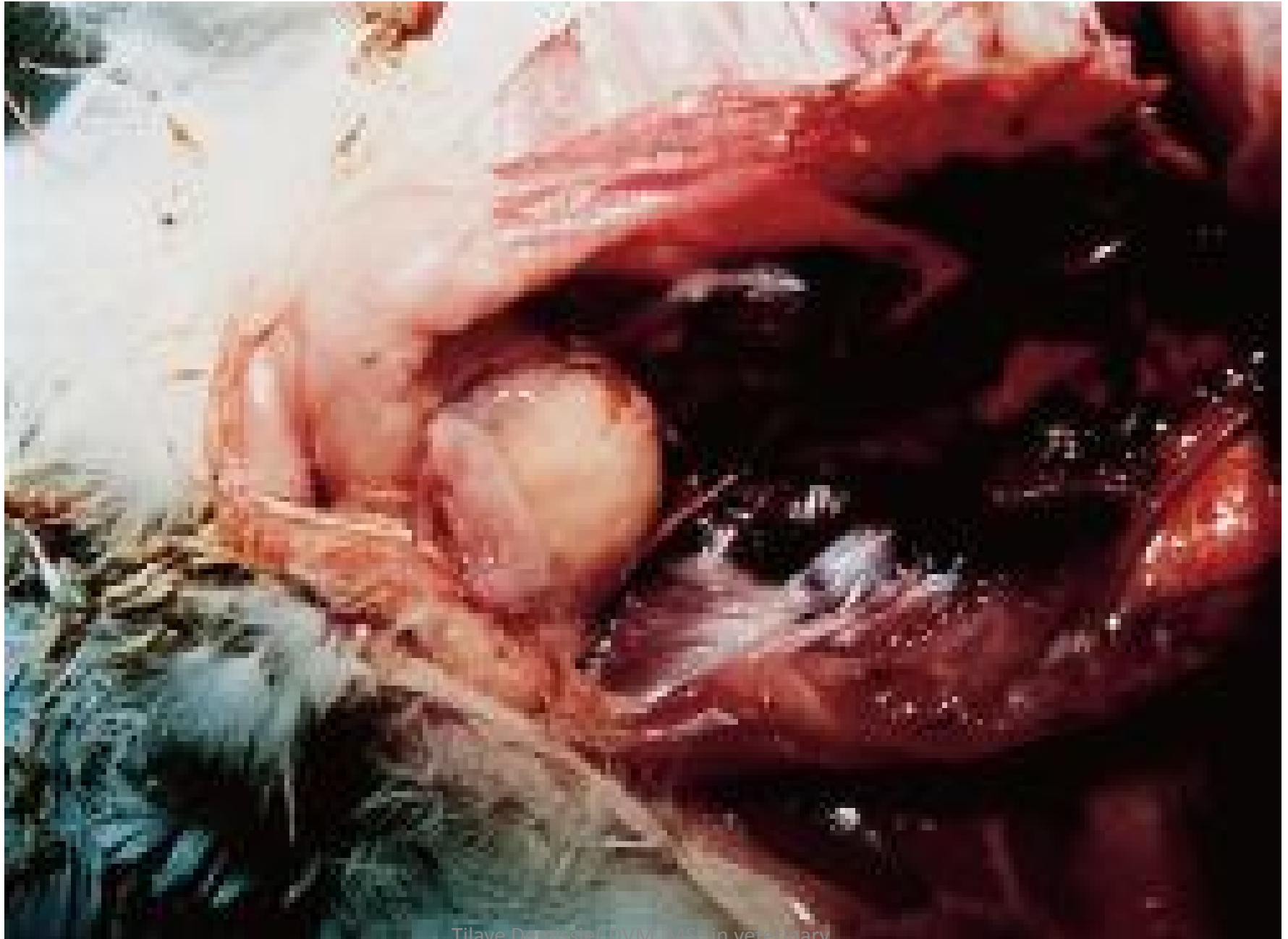
Bleeding and swollen bursa of Fabricius



Bleeding into skeletal muscle of leg



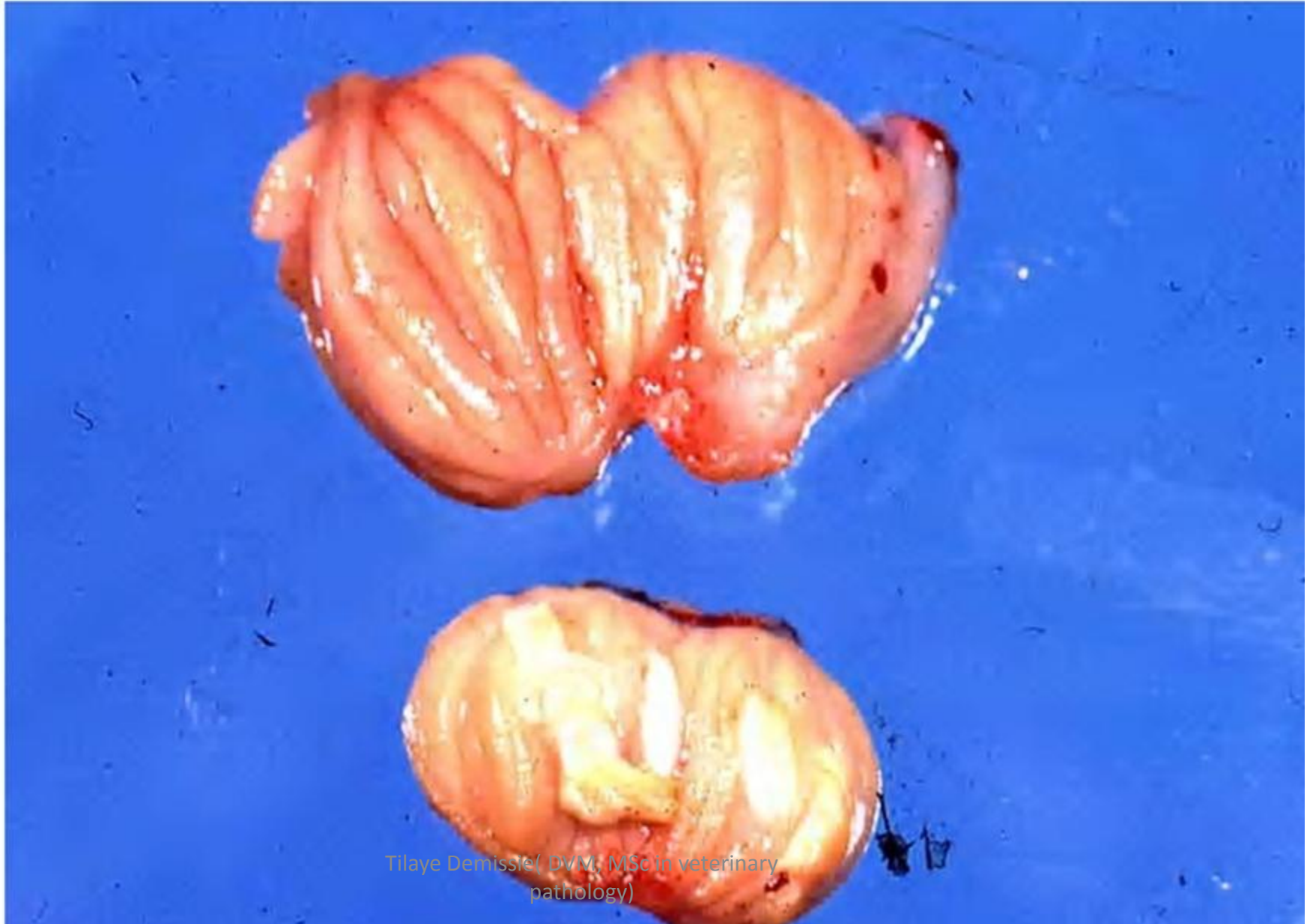
Tilaye Demissie(DVM, MSc in veterinary pathology)



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Petechial haemorrhage in bursa (sectioned)



Course of the disease!!

Tilaye Demissie(DVM, MSc in veterinary
pathology)

Petechial haemorrhage* on thigh muscle

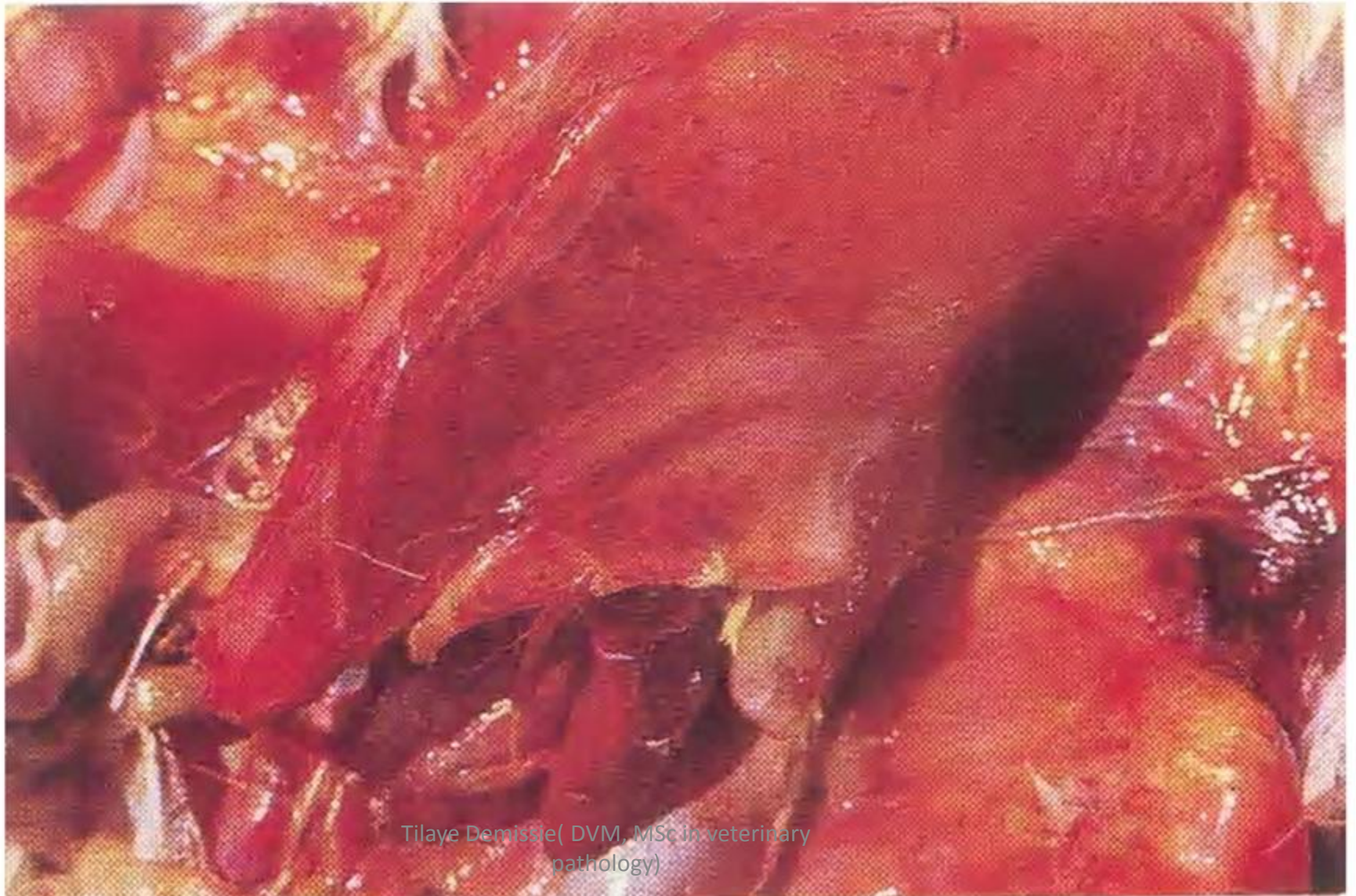


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Thigh muscle hemorrhage



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MICROSCOPIC LESION

Bursa show the following:

- ✓ Degeneration and necrosis (medullary portion of the lymphoid follicles become depleted of lymphocytes)
- ✓ Hetrophilic infiltration in to the interfollicular space (IFS)
- ✓ Interfollicular edema
- ✓ Cellular debris inside the cystic cavity
- ✓ Cystic cavity develop in medulla which is bounded by columinar epithelial cells
- ✓ Bursal epithelial layer proliferation produce a glandular appearabce of the bursa

Spleen- lymphoid necrosis around the periarteriolar sheaths (around germinal centres)

Thymus/cecal tonsils - mild lesions as there are very few susceptible B-cells

Kidney - Large casts of homogenous materials (urates) with heterophils in the kidney

✓ Enlarged kidneys with accumulation of urates in the renal tubules (due to dehydration or /and possibly with ICX formations in the glomerulus)

Diagnosis

- Typical clinical signs and post mortem lesions
- Histopathological examination, serology, virus isolation and PCR are confirming tools.
- ✓ IBD can be confused with sulfonamide poisoning, aflatoxicosis, and pale bird syndrome (Vitamin E deficiency).

Control

- Vaccination of breeders and young chicks is the best means of control.
- ✓ The induction of a high maternal immunity in the progeny of vaccinated breeders **to confer maternal immunity to progeny** , together with the vaccination of the offspring is the most effective approach to successful IBD control.

- Different kind of live and inactivated vaccines are available.
- Young chickens are vaccinated at an age of 1 to 3 weeks, depending on their maternal immunity and the risk of infection with (highly) pathogenic strains.
- Immunisation of breeder flocks to confer maternal immunity to progeny is practised by injection at the end of their rearing period with an inactivated vaccine.
- These re-vaccinated breeder hens will provide yolk-sac antibody, which protects chicks until about 3 weeks old.
- At this age chicks can be safely immunised with live vaccine, but if done at an earlier age the maternal immunity will interfere too much with the vaccine-virus.

IBD vaccine in Ethiopia: Lyophilized (intermediate standard)

- ✓ Route: eye drop for primary vaccination
- Drinking water method
- This vaccine is not recommended for clinically sick or stressed birds
- Schedule: Chicken with low level of maternal antibody at 1-2 weeks & second at 3-4 weeks
- Chicken with high level of maternal antibody at 3-4 weeks & second at 8-12 weeks of age.

Avian Influenza (Avian flu, influenza, fowl plague)

- ❖ **Etiology - Orthomyxo-viruses of type A:** there are several serotypes based on hemagglutinin (H) and neuraminidase (N) antigens occurring on the surface
 - ✓ H ag. is responsible for the attachment
 - ✓ N ag. is responsible for the release of the virions from host cell
- **Subtypes:** Currently, there are 16 H- types (H1 to H16) & 9 N-types (N1 to N9) and they can show up in all kinds of combinations and might cause disease in different species of birds including humans (flu).

Pathogenicity of Avian influenza varies with the strains can be categorized into three:

- **Highly pathogenic group** (HPAI) /“fowl plague” - For poultry the most important ones are the subtype H5,H7& H9. it causes catastrophic losses associated with viscerotropic & pansystemic infection
- **Moderately pathogenic and**
- **Low pathogenic type**

Antigenic variability

- ❖ **Antigenic drift-** point mutation in HA or NA gene
- ❖ **Antigenic shift-** exchange of genes between two closely related viruses infecting **the same cell**
- ❖ *All subtypes of Influenza type A can affect birds*

Species affected: Avian Influenza viruses have been shown to naturally infect a wide variety of wild and domestic birds and is world-wide in distribution. In poultry production main problems are in **chickens, turkeys and ducks.**

Source of infections: ***Migratory birds*** & **Secretion** containing the virus (droppings, nasal & ocular discharge)

Transmission: **Direct** contact with clinically ill bird

➤ **Indirect** (oral-faecal) - feed, water, vehicles

➤ **Wind dispersal:** the virus is relatively resistant to environmental exposure

❖ Rapid multiplication & ultimately affecting all poultry ***operations in a region***, unless appropriate controls are implemented

- ✓ Waterfowl and other Wild birds are considered as a reservoir of the pathogenic Influenza-strains, and plays great epidemiological role because they can spread the disease over great distances
- ✓ Carrier animals play an important role in persistent infections
- ✓ Import & export of exotic birds is also a major route of spread & quarantine (minimum of 5 wks) is necessary.
- ✓ Humans play an important role in transmitting the virus from pen to pen or farm to farm.

Clinical signs: vary depending on the pathogenicity (HPAI and LPAI) of AI virus involved and other factors as host species, sex, concurrent infections, acquired immunity and environmental factors.

- **LPAI** shows generally *mild symptoms*: respiratory signs like coughing sneezing, wet eyes, nasal discharge, depression, lethargy limited reduction of feed intake and limited drop in egg production; low mortality rate
- **HPAI** shows fast onset with increased mortality (50-90%) even Sudden death before clinical signs are seen
 - *Respiratory signs* - excess discharges around the nostrils and eyes, Swollen sinuses, sneezing
 - *GIT involvement*- Diarrhea
 - *Nervous signs* - twisted neck, incoordination
 - Cyanosis of the comb, swollen head (periorbital edema)
 - Drop in feed & water intake, severe drop in egg production

Pathology/Internal lesions

- ❖ **Highly pathogenic group:** Vesicles on the comb, wattle, Cyanosis & edema of the head. Skin lesions of the head including the wattle and comb comprise an important lesion in chickens for detection of highly pathogenic avian influenza viruses (H5N1)
- ✓ Severe haemorrhagic tracheitis, sinusitis, airsacculitis
- ✓ Subcutaneous hemorrhages (visible also on legs, comb, and wattles) - characteristic of H5N1
- ✓ Hemorrhages on serosa of all viscera and on the mucosa of the intestinal and respiratory tracts
- ❖ **Mild influenza:-** mild tracheitis, pulmonary edem, airsacculitis



Purple discoloration of wattles and combs with swelling caused by abnormal accumulation of fluid



Swollen head, accumulation of liquid in eyelids and comb



Pinpoint bleeding under the skin (mostly seen on feet and shanks)



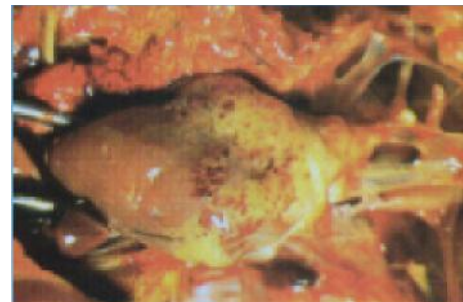
Bleeding into the ovaries



Bleeding into the gizzard



Bleeding in the mucosa of trachea



Bleeding in the muscle and in the fat around the heart



Birds can be found depressed; there may be nervous signs



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pathology)

Cyanosis, edema and swelling of the comb, wattle, and sinuses



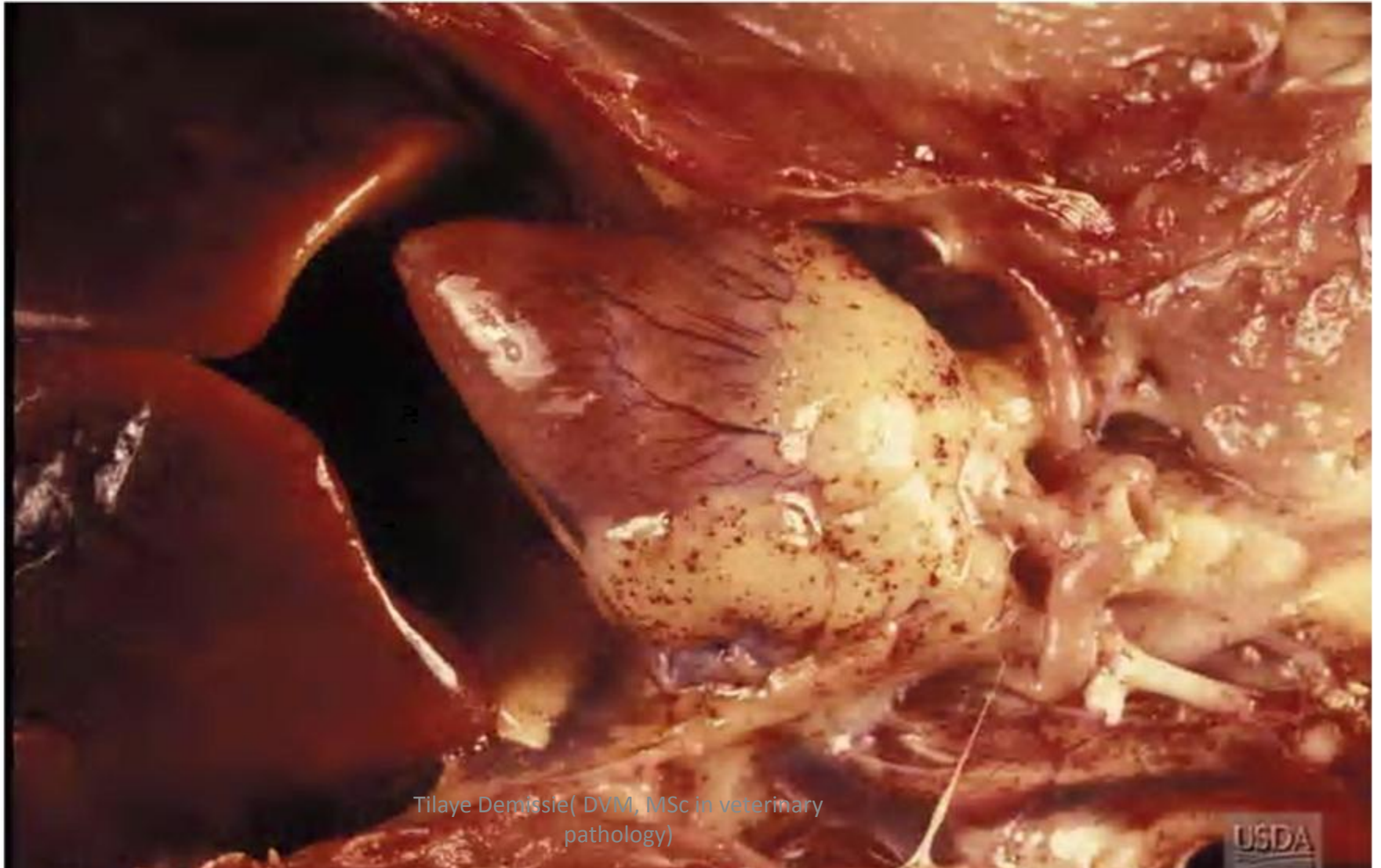
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Haemorrhagic breast muscles

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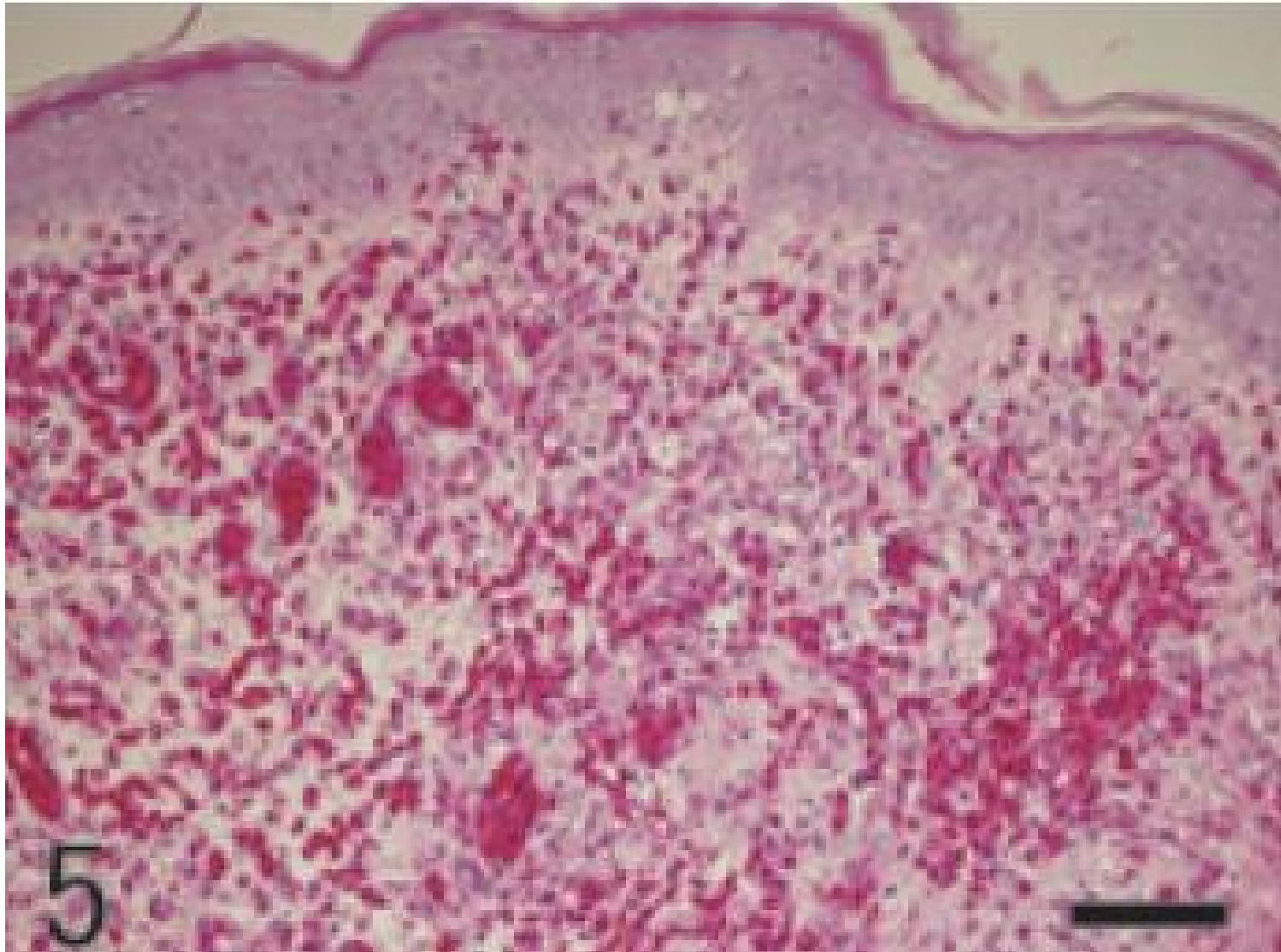


Pancreatitis with petechial haemorrhages

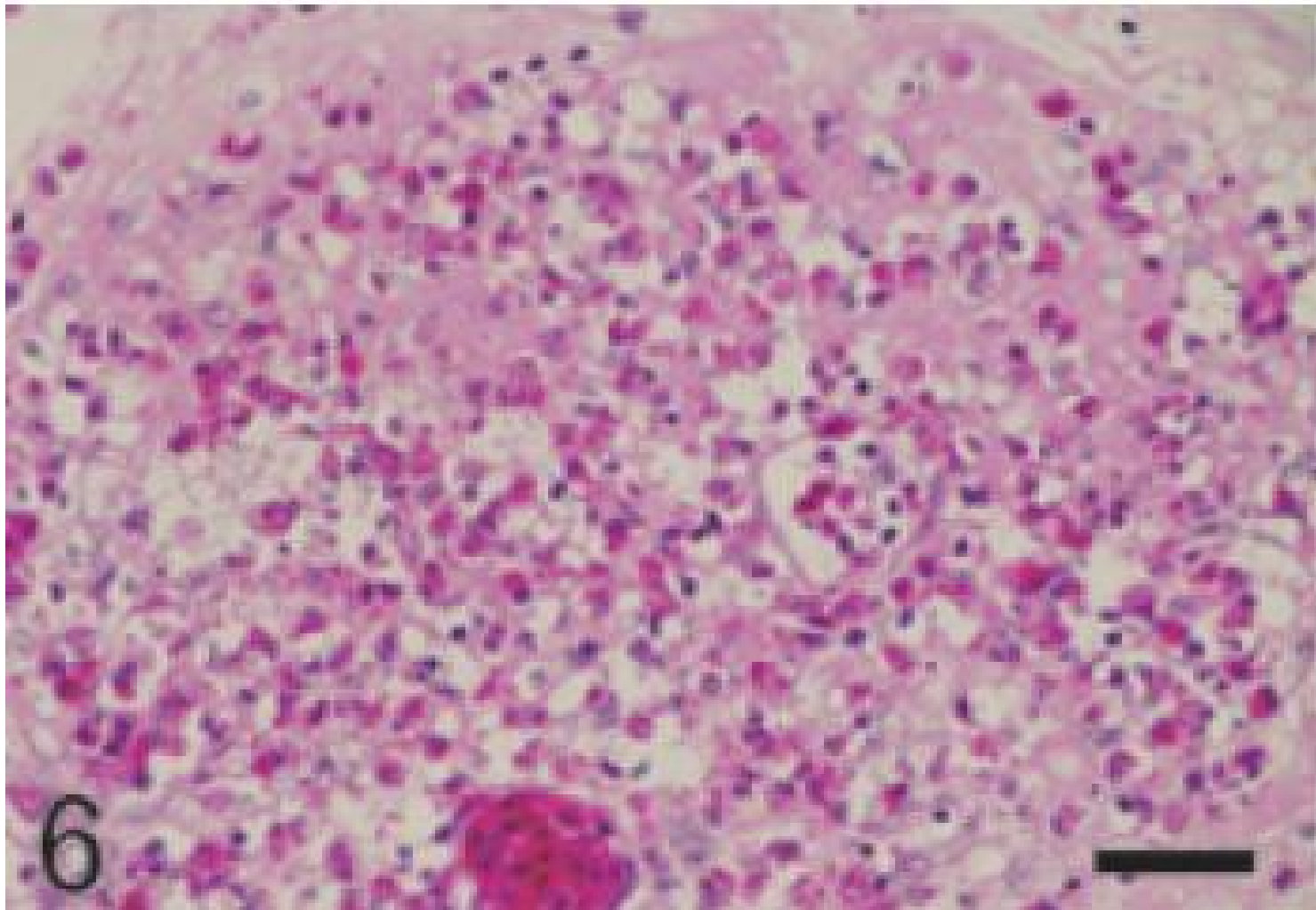
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Microscopic lesions

- ❖ **Comb, wattle:** Dermatitis (heterophilic infiltration, capillaritis, hemorrhage, edema, epidermal necrosis)
- ❖ **Lung:-** pneumonia, Heterophil and hemophagocytic macrophage infiltration at the blood capillary
- ❖ **Acinar cells Necrosis of the spleen**
- ❖ **Liver** - Lymphocytic inflammation/necrosis of hepatocytes at the portal areas
- ❖ **Non-suppurative meningoencephalitis** (neuronal necrosis, gliosis, perivascular cuffing)& Spinal cord Non-suppurative myelitis
- ❖ **Endothelial** cells necrosis with fragmented nuclei
- ❖ **Lymphoid tissue** depletion and **Myocarditis**
- ❖ **Necrosis** of parenchymal cells of the pancreas

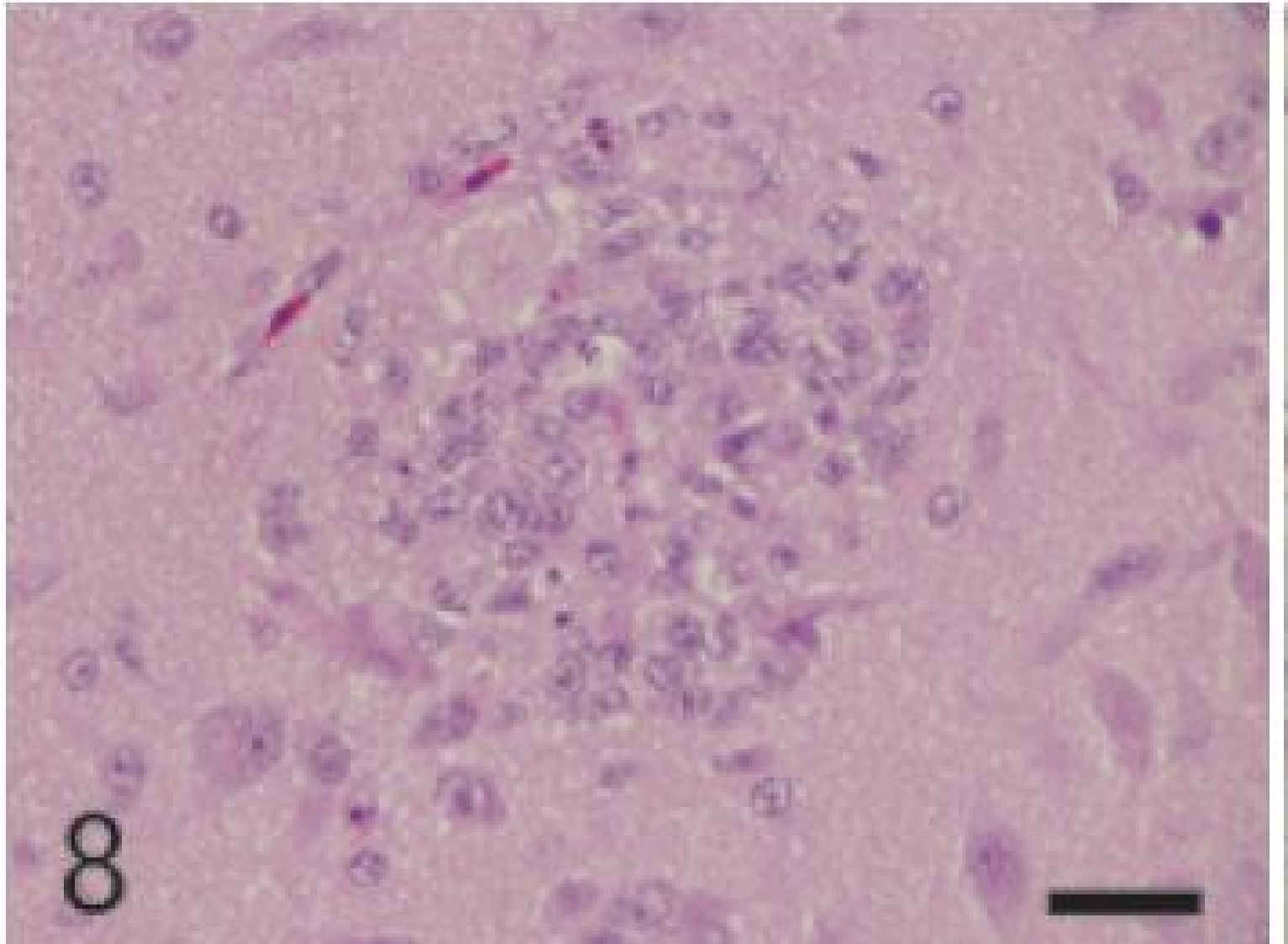


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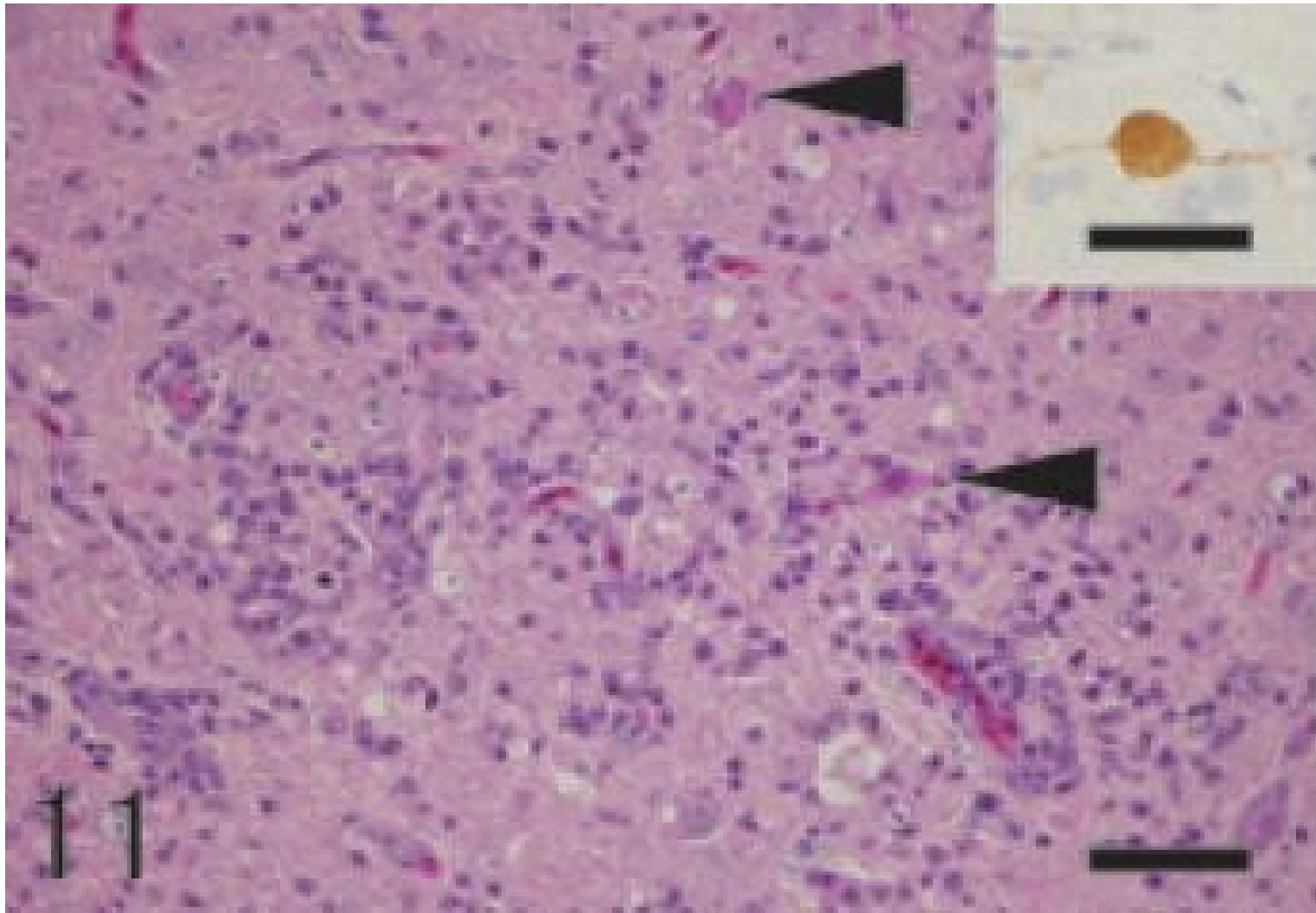
Heterophilic infiltration & capillaritis with edema at subcutaneous tissue of the wattle

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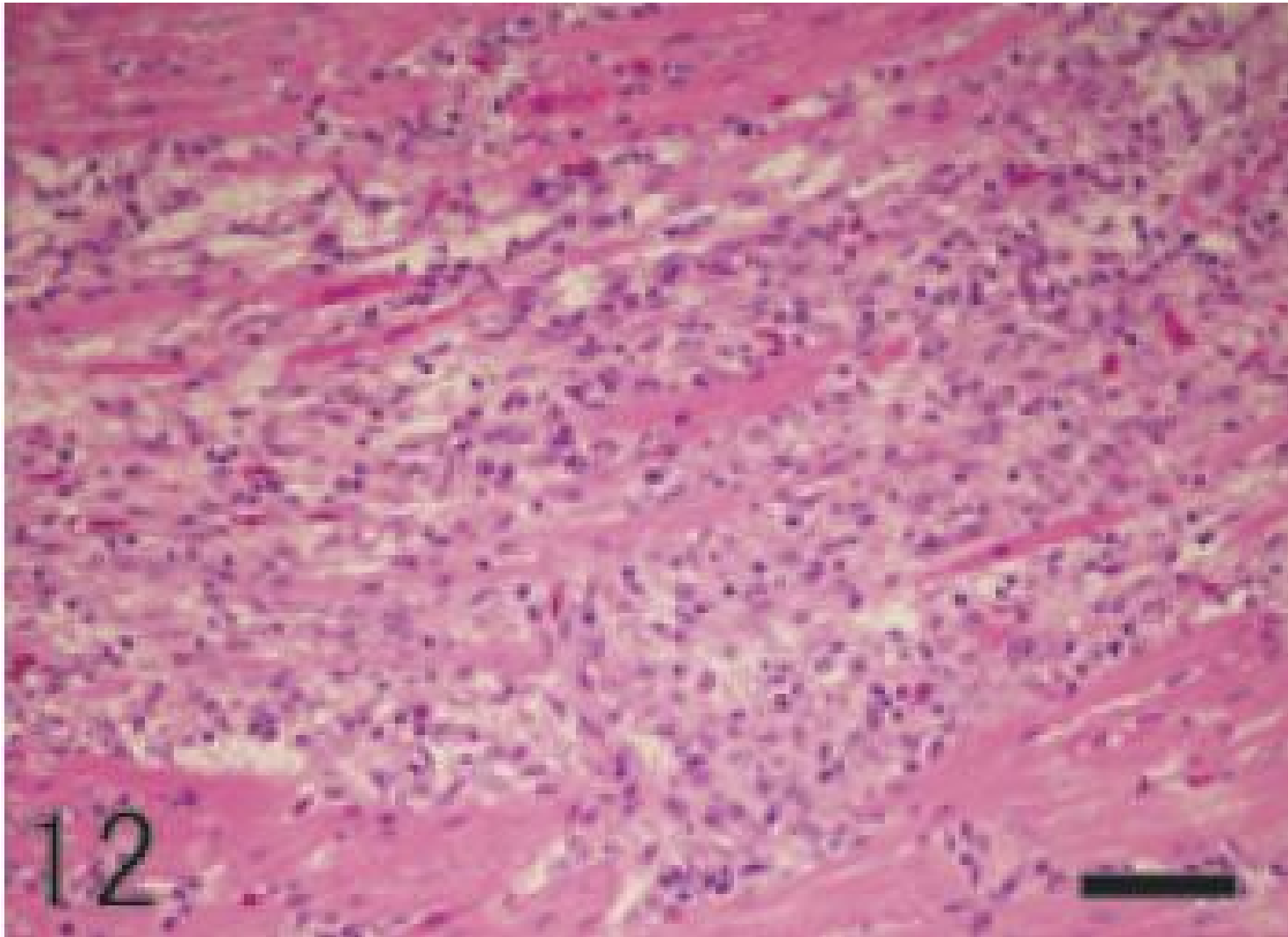
- small focus of gliosis of the brain Hematoxylin and eosin

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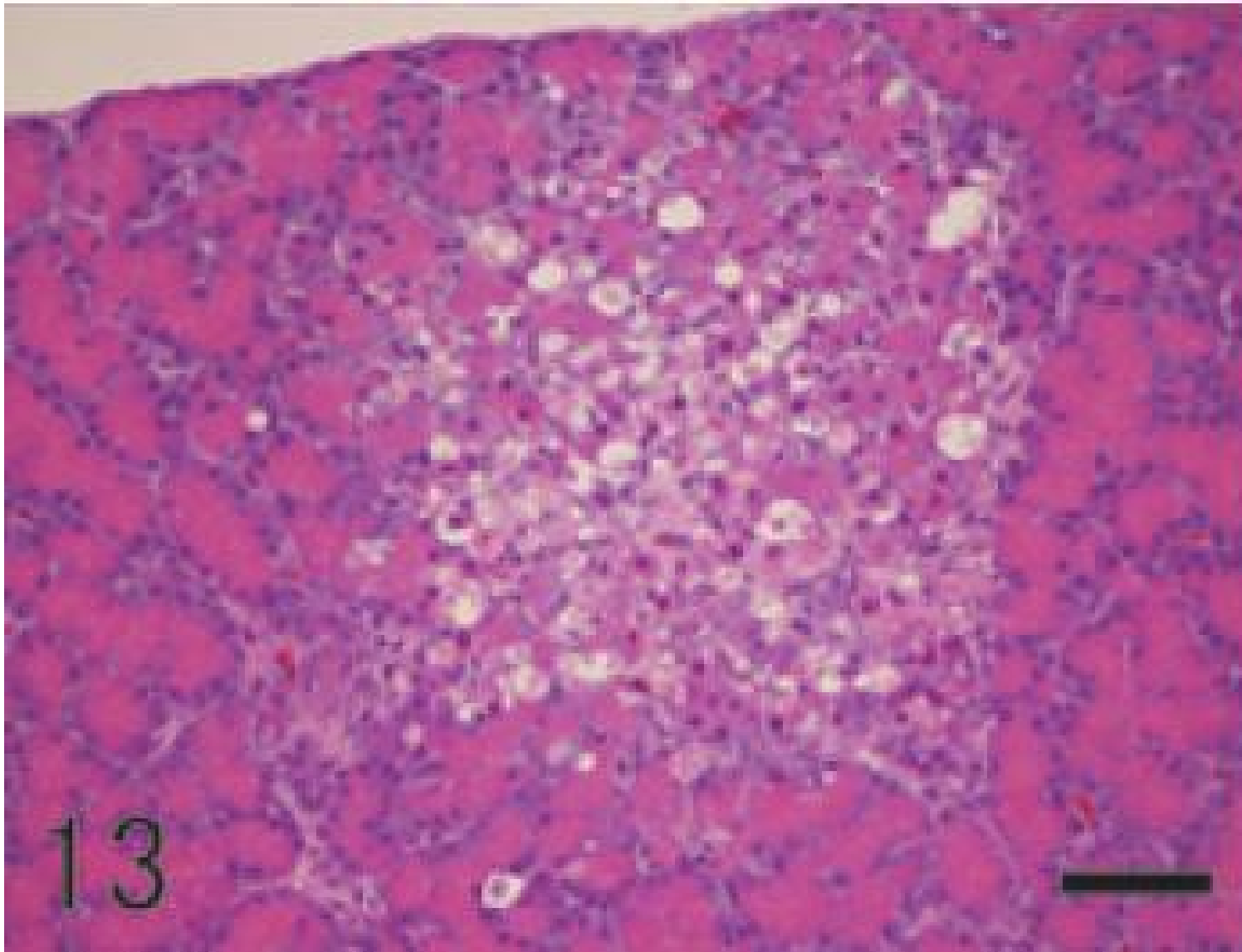
- **perivascular cuffing and gliosis with a few necrotic neurons (arrowheads) of the brain H&E**

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- **lymphohistiocytic myocarditis**

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- **focal necrosis and vacuolization of acinar cells of the pancreas**

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Diagnosis: Clinical signs are indicative for AI;

- Final confirmation by laboratory testing:
 - Direct detection of AI proteins or Nucleic acids (RNA) using PCR.
 - Virus isolation from infected organs, tracheal or cloacal swabs.
 - Serology from blood samples after infection and for routine monitoring showing specific AI antibodies.

Treatment: There is no treatment for Avian Influenza. Antibiotics will help to control secondary bacterial infections.

Prevention and control: Strict biosecurity

- Vaccination – Inactivated and recombinant vaccine (fowl pox virus expressing the H5 antigen of AI)
- ✓ *Live conventional vaccines are not recommended as this may favore genetic reassortment*
- **Public health-** *H5N1 has caused substantial human death in Asia and Africa*

Prevention and control: In many countries AI is a notifiable disease with specific local regulations on its control.

- In AI free areas the disease (LPAI and HPAI) is controlled by monitoring and stamping out: rapid diagnosis, slaughter, quarantine, no transport of animals and products, monitoring and financial compensation.
- In case of LPAI infected areas countries can decide to allow vaccination only for LPAI.
- In case of endemic HPAI and/or LPAI vaccination might be allowed.
- Vaccination is generally done with inactivated AI vaccines based on the strain H-type causing the outbreaks.

Infectious Bronchitis (IB) - a highly contagious, acute & economically important respiratory disease of chickens.

- **Etiology:** Corona virus - many strains; *E.g.* Massachusetts, D274, D1466, T-strain, S-strain, IB 4/91, QX, Arkansas and Connecticut.
- ✓ Each with great antigenic variation and with its own characteristics in symptoms (Different strains affect different organ systems: **respiratory, renal, reproductive**)
- ✓ Mass 41 & Conn 46 are used as vaccine
- ✓ Sensitive to heat, Common disinfectants, dryness and sunlight but may persist on contaminated premises for approximately 4 weeks
- ✓ The incubation period is only 1-3 days.

Species affected: Only chickens ---All ages are susceptible, but severe in young

- ✓ Particularly a problem in laying flocks/Hens
- variable production loss and affects egg quality.

Transmission: The virus is transmitted rapidly from bird to bird through the airborne route by inhalation of droplets containing virus excreted by infected chickens

- ✓ Direct contact with short time carriers
- ✓ Contaminated premises
- The virus can also be transmitted via the air between chicken houses & even from farm to farm
- **Contaminated premises (4 weeks) and Infected/carriers chickens (49 days)** become a source of infection

Clinical signs: Respiratory IB usually not significant – although tracheal plugs at the bifurcation cause asphyxiation, coughing, sneezing, rales, nasal and ocular discharge

- ✓ High morbidity In young chicken (<3 wks)
- ✓ Mortality in young chicks is usually high when the disease is complicated by other infectious agents such as mycoplasma and E. Coli.
- ✓ Nephrotropic strains may cause high mortality
- ✓ There is weakness, depression and huddling near the heat source.

- IB causes reduction of feed consumption & weight gain
- Common signs of IB in laying hens is that egg production drops markedly (3% - 60%) with many **soft shelled eggs** or **misshapen eggs** or ***rough surfaced shells***
- ✓ Low egg quality & shell irregularities may persist a long time after an outbreak of IB.



Post mortem lesions: Hyperemia & Serous/catarrhal exudate of trachea

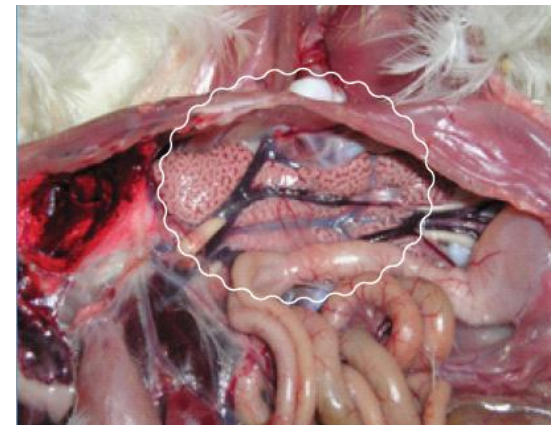
- ✓ A yellow cheesy plug at the tracheal bifurcation is indicative of IB infection
- ✓ Airsacculitis - redness and exudate in the air sacs
- In case of nephropathogenic infections - pale and swollen kidneys and distended ureters with urates
- Various changes in the oviduct depending on the time and severity of infection
- ✓ Salpingitis & permanently damaged oviduct
- ✓ An oviduct filled with albumin fluid (water belly).
- ✓ Even some animals might have free yolks in their abdomen
- Egg yolk peritonitis



Mild to moderate irritation of respiratory tract
with swelling of trachea



Cystic oviduct in an affected
(false) layer



Swollen and pale kidneys
with distended urinary tubes

Diagnosis: Clinical signs and post mortem lesions

➤ Laboratory confirmation based on virus isolation and identification with PCR.

- Serology using HI, Elisa or VN tests

Treatment: There is no treatment for IB.

- Antibiotics are used to control secondary bacterial infections.

Prevention: Vaccination with strain specific or cross protective live vaccines

- ✓ Complete prevention of IB is difficult because of variation of field strains and the ability of the virus to change. There is little cross protection between serotypes.

Infectious Laryngotracheitis (ILT)

- It is an acute viral disease of chicken & other birds.
- **Etiology:-** *Gallid herpesvirus1*. It can easily be destroyed by many disinfectants and is not resistant outside the host.
- Strain variation - Most strains are pathogenic
 - Mild LT - lowered growth rate and egg production
 - Moderate - severe strains -  morbidity/mortality
 - ✓  egg production (losses approaching 50%)
- ILT can look like Newcastle D. in an acute stage.
- ❖ *Mature or nearly mature* chickens are affected (all age group may be affected)

Species affected: Chickens are the primary natural host but other species (pheasants) can also be affected.

Transmission - occurs most readily from acutely infected birds. And also from permanent carriers

- ❖ Routes of entry: Mainly via **Inhalation** and **intraocular**
- Rarely **ingestion** (contaminated feed
- **Horizontal transmission** - via direct contact from bird to bird &/or transmission by contaminated egg trays, feed trucks & contaminated feed, poultry crates, dead birds, people, contaminated equipment, litter/manure.
- Wind dispersal over 3 km has been documented.
- ✓ But, the spread is often less rapid than other viral respiratory disease like IB and ND
- ✓ Incubation period varies from 4 -12 days.

Clinical signs: depends on the severity/strain of virus, immune status of birds and environmental conditions: ranges from ***Mild to severe cases***

Mild strain: Sneezing & gurgling , Conjunctivitis & swollen heads

- ✓ ↓ egg production, 10-50%, but can return to normal after 3-4 weeks
- ✓ Mortality can vary from 5 -70%.

In severe cases: An acute & marked respiratory distress with nasal discharge, moist rales, gasping

- ✓ Expectoration of bloody tracheal exudate/mucus
- ✓ Cyanosis of the head due to dyspnoea
- ✓ Wheezing noise/open mouth breathing

Post mortem lesions - found throughout the respiratory tract but most pronounced in the larynx and trachea.

- Depending on the severity of the infection:
 - Severe hyperemia/haemorrhagic of tracheal mucosa, with blood clots (pathogenic strain)
 - Tracheitis with diphtheric/necrosis changes: cheesy pseudomembranes plugs the trachea
 - ❖ Exudate & blood clots obstruct the glottis - asphyxiation
 - ❖ Bloody beak/ blood on face, head and feather

Diagnosis: Clinical picture with birds showing respiratory distress and expectoration of bloody mucus are indicative for ILT.

- Laboratory confirmation:
 - ✓ Histopathology showing intranuclear inclusion bodies in tracheal epithelial cells
 - ✓ Virus isolation from tracheal swabs on embryonated chicken eggs
 - ✓ Virus detection with PCR or IFT on tracheal samples.
 - ✓ Detecting antibodies from blood samples after infection.

Treatment: no treatment for ILT; emergency vaccination in the early stage of an infected flock may reduce the spread and limit the outbreak.

Prevention and control: In many countries **vaccination** is the preferred control method.

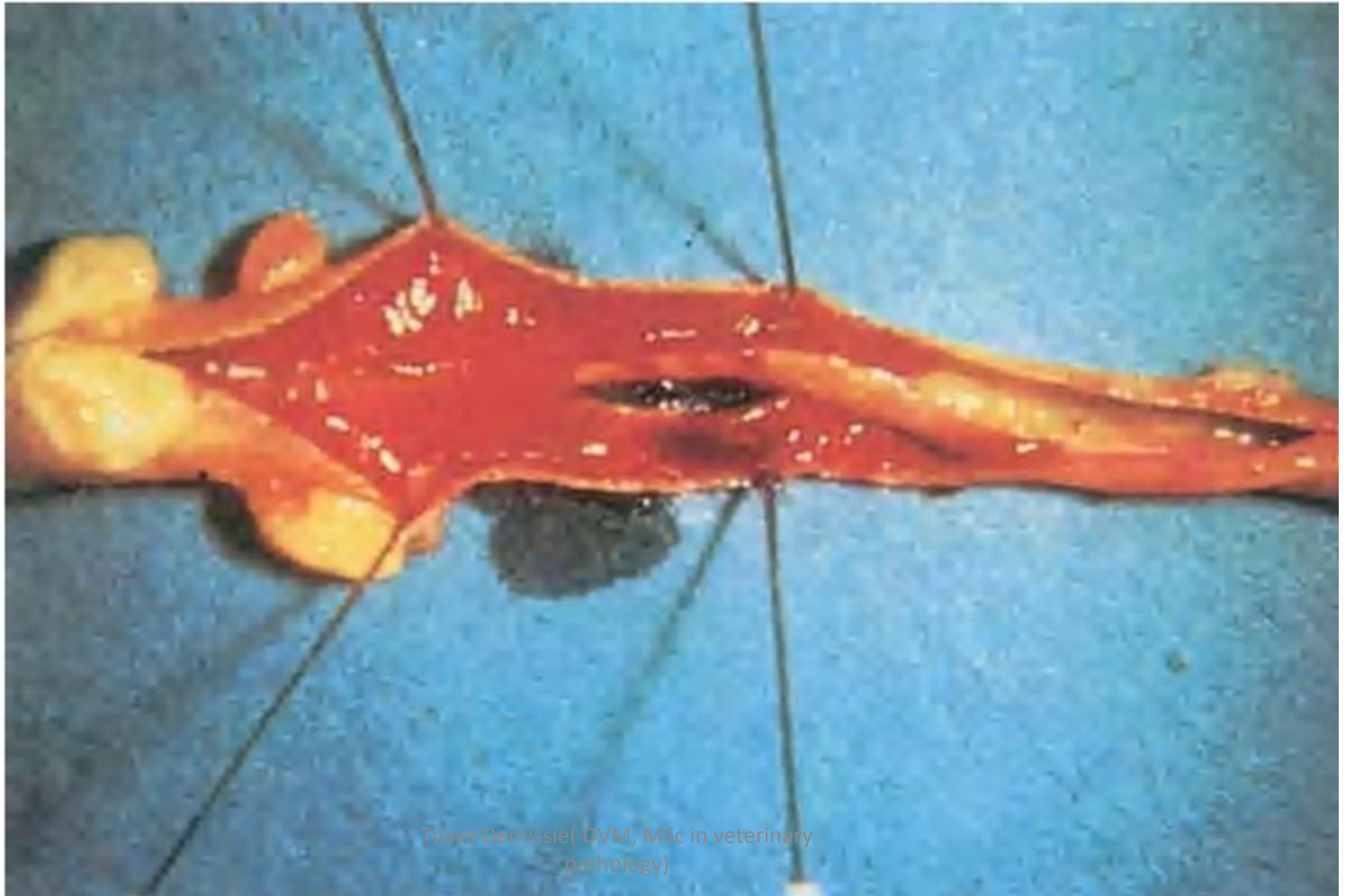
- In areas with a high risk of ILT-virus infection vaccination at a younger age by eye/nasal drop or coarse spray or drinking water.
- A disadvantage in the use of live vaccine is the potential spread of the vaccine-virus and the production of carrier-animals. So, it is recommended for use only in geographic areas where the disease is endemic.
- The new generation of recombinant vector vaccines are more suitable in the control and prevention of ILT.
- Recombinant vaccines based on HVT-vector carrying inserts of important immunogenic ILT proteins show good efficacy and do not spread and cannot revert to virulence because there is not a full ILT virus involved.



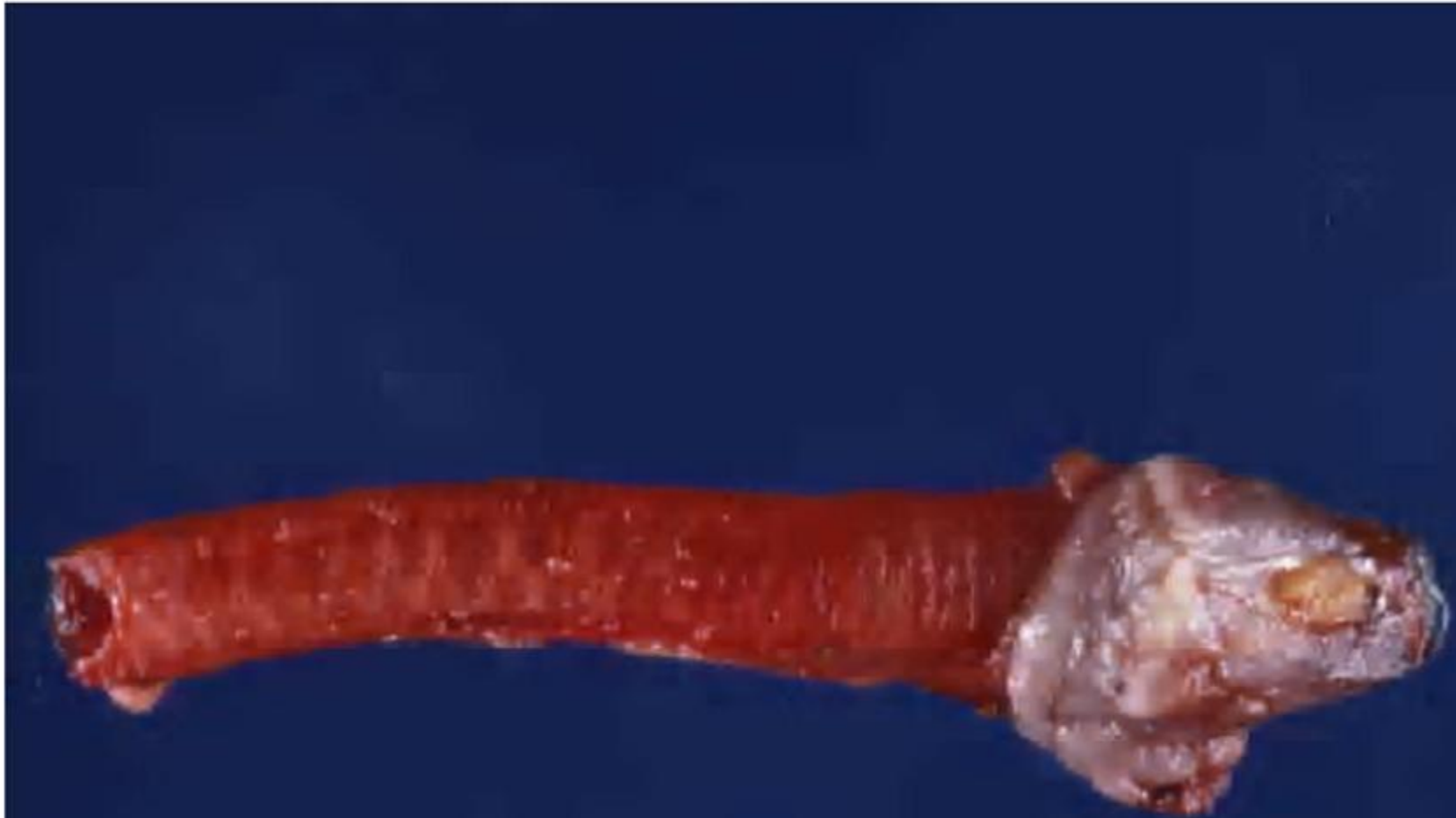
Gasping bird ILT infection



Gurgling with neck extended



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Fibrinonecrotic (cheesy) exudate



cm

1

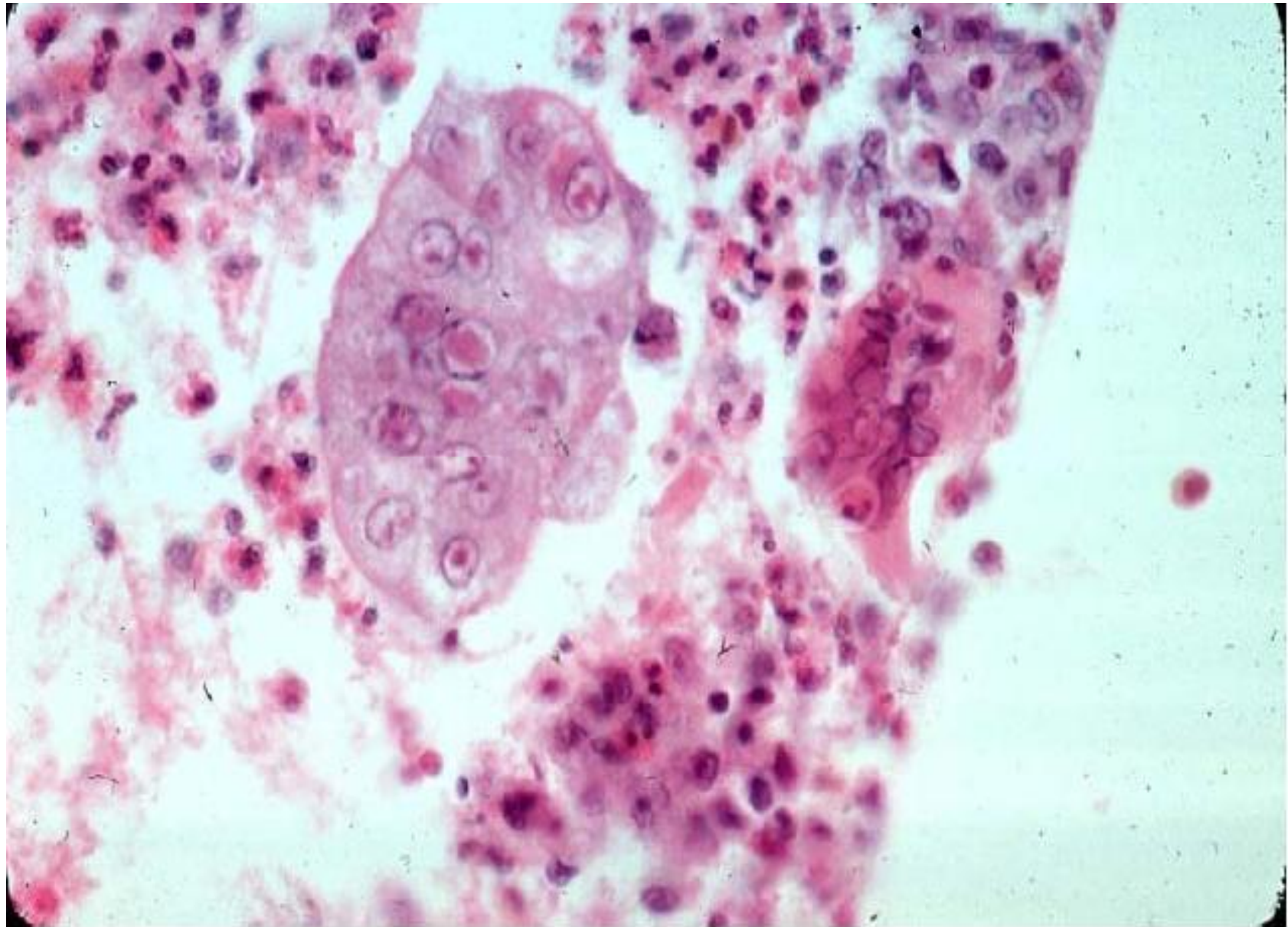
2

3

SPEC

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DATE



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IB



Gasping



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gasp



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Characteristic shell and content changes during an outbreak of IB



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Nephrotropic strain



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Abdominal airsac containing yellowish caseous exudate



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Fowl Pox (Avian Pox, Avian Diphtheria)

- It is a relatively slow-spreading disease characterized by pox-lesions in the skin (blisters, nodules, scabs....), and by diphtheria membranes (wet-pox) in the upper digestive and respiratory tracts.
- The disease is worldwide ...

Etiology: an **avian pox-virus**; not pathogenic to humans!

- The Pox-viruses are very stable and resistant to temperature change.
- They can survive for years in dust, in **dried scabs**

Species affected: Chickens, turkeys, pheasants and pigeons can be affected by different Fowl Poxvirus strains. **Any age** is susceptible

Transmission

- Direct contact - between infected and susceptible birds
- Ingestion of contaminated water or feed transmission
- Mosquitoes and other flying insects can also transmit the virus from bird to bird and also transmit the disease to near-by flocks.
- ✓ There is some evidence that the mosquito remains infective for life

Source of infection: infected or “carrier” birds - *Virus-containing scabs*

- The incubation period varies from 4 to 20 days.

Clinical signs: The disease occurs mainly in two forms:

➤ The skin/***dry form/ cutaneous form***

- Pox-lesions (blisters, papule, nodules, scabs, wart-like....) on featherless parts of the body/skin -- on the head, combs, wattles
- Eye involvement frequently seen

Diphtheritic Type/Wet Pox: diptherie or Pseudomembrane in the mouth, pharynx, oesophagus and trachea

- ✓ Yellow-white and cheesy in appearance.
- ✓ Breathe laboriously because the necrotic nodules, which may suffocate the birds.
- Depression, lack appetite, Reduced egg production, Mortality is variable (2%- 40%)



Gross lesion

Cutaneous form: Local epithelial hyperplasia (of epidermis, feather follicle) resulted in Pox-lesions like nodules, papules, vesicles, scabs and desquamation of degenerated epithelial cells.

✓ Seropurulent exudate & haemorrhagic granulating surfaces are visible when scabs are removed

Respiratory diphtheritic form: Opaque nodules or Fibrinonecrotic to pseudodiphtheritic and yellow-white and cheesy like diphtherie or Pseudomembrane on mucus membrane with bleeding erosions on removal

Diagnosis: history and clinical signs

- ✓ Confirm the presence of *intracytoplasmic inclusions* bodies in the respiratory mucosa and skin (histology) and/or virus isolation

Treatment: There is no effective treatment.

Prevention: preventive vaccination using a live vaccine is by far the most successful control method.

- Immunization is recommended in endemic areas (8 wks age)
- ✓ In areas where early exposure occurs, the age of vaccination can be advanced
- ✓ Even when an outbreak of Fowl Pox has been diagnosed, it is advisable to vaccinate the flock immediately (emergency vaccination) to stop further spreading of the infection.

Internal Lesions

- The internal lesions are similar to clinical signs.



Dry form: wart-like nodules on the skin (combs, face and wattles)





Diphtherie form



Wet form : Cankers are imbedded in the membranes of the mouth, larynx and trachea.

Cankers



Wet form: Brown nodular lesions in the mucosa membrane of larynx; when removed, an eroded area is left

Avian Encephalomyelitis /AE or Epidemic Tremor

- A Viral infection of young chicks and laying hens, characterized by ataxia, tremors of head and neck and paralysis of young chicks and a sudden drop in egg production for 4-5 days in laying hens.
- ✓ The disease is worldwide among poultry and economically losses are due to mortality in chicks, reduced egg production of a temporary nature in laying hens and lowered hatchability of fertile eggs.
- **Etiology:** Picornavirus - resistant to most chemicals, detergents & survives well in the environment
- **Species affected:** Primarily, chickens are susceptible to AE, but turkeys and pheasants have been reported as natural hosts.

Transmission: Horizontal/Lateral transmission among birds

- Egg transmission is the major route of spreading of AE--- eggs laid by infected hens for up to 1 month.
- ✓ The incubation period varies from 5 - 14 days depending on the route of infection
- ✓ The susceptibility depends on the age of the birds and their immune status

Clinical Signs: the disease is mainly seen in young chickens, between 1 and 3 weeks of age.

- The main symptoms of affected chicks are incoordination, paresis, paralysis, unsteadiness, sitting on their hocks, Clinical tremors and many fall on their sides
- A drop in egg production which returns to normal in about 2-3 weeks, increased mortality in young chickens



AE infected young chickens

Lesions: No gross lesions however, **Histology**, there is central chromatolysis & perivascular cuffing (brain)

Diagnosis: history and clinical signs

➤ Histological examination, Serology, virus Isolation...

Differential Diagnosis: CNS signs usually occurs in birds

- **Vitamin E/Selenium deficiency** – > 3 weeks of age.
- **Marek's** - >3wks of age
- **Newcastle** – CNS signs
- **Rickets** – inability to move around
- **Vitamin B1 /B2 (Thiamine)** deficiency – stargazing
- **Mycotic Encephalitis** – after 3 weeks of age

Treatment: There is no effective treatment for AE.

Prevention & Control

- **Preventive vaccination** of breeder pullets before egg production is the only effective means of AE control.
- ✓ Vaccination is usually given between **8 and 16 wks of age.**
- ✓ Vaccinate laying hens with only killed vaccines
- Don't hatch eggs from viremic hens
- Isolation of affected flocks

Viral arthritis/Tenosynovitis

- A widespread viral infection of poultry that affects the synovial membrane, tendon sheaths, tendons and myocardium of meat-type chickens, and occasionally turkeys.

Causes: Avian reoviruses

- Many antigenically different strains of reoviruses, like S1133, 1733, 2408, ERS, which are considered to be most important in meat-type chickens.
- The reovirus is a common inhabitant of the intestines of birds and not all strains are pathogenic

Species Affected: Chickens, turkeys and possibly pheasants are natural hosts.

- ✓ This disease can be seen in broilers, broiler-breeders and even some layer-breeds like White-Leghorns.

Transmission: The virus may be transmitted by droppings from bird to bird (Via respiratory or digestive routes)

- Transovarian/Egg transmission is also a factor when breeder flocks become infected during egg production.
- ✓ This occurs when the hens are viremic.

Clinical signs: The disease is more common in broiler breeders and broilers than in commercial egg-laying breeds of chickens, because the body-weight and quality of tendons are of influence as well.

- In young meat-type chickens: increased mortality, arthritis/ tenosynovitis, disuniform flocks resulting in loss of performance and economic losses.
- The first signs of reovirus infection can be observed in broiler type chickens between 2 and 10 weeks of age.

Affected birds have malpositioned feathers, especially on the wings and are called “helicopter chickens”.

Morbidity ranges from 5 - 50 % and mortality from 2 - 10 %.

Internal lesions: The hock joint may be somewhat swollen, but usually not as severely as with *Mycoplasma synoviae* (MS) or *Staphylococcus* infections.

The tendons usually appear discolored, brown or blood-tinged, with straw colored fluid between them.

Ruptured tendons may occur and, in older broiler breeders (29-30 weeks old), one may feel a hard knot in the tendon above the hock joint.

When the infection is complicated by *Mycoplasma synoviae* or



Infected birds may show malpositioned feathers (above) and swollen tendons (below)

- Swelling of the tendon sheaths of the digital flexor and metatarsal extensor. A knot is palpable in the tendon above the hock.
- Affected birds tend to sit on their hocks and are reluctant to move.
- Rupture of the gastrocnemius tendon. This occurs when birds start to jump on the slats. A large bruise may be visible above the hock.
- Reovirus induced enteritis will cause diarrhea, feed passage, and poor growth and feed conversion.
- The birds are reluctant to walk and when forced up have a painful, trembling gait.
- Staphylococcus the fluid may appear yellow and creamy
- A distinct swelling of the tendons of the shanks and also above the hock joint can be observed.

POSTMORTEM LESIONS: 1. Edema of tendons.

2. Excessive fluid in the hock joint.

3. Rupture of the gastrocnemius tendon and hemorrhaging dorsal to the hock.

4. Erosion of articular cartilage.

5. Staph will often invade afterwards causing purulent arthritis

DIFFERENTIAL DIAGNOSIS: 1. Mycoplasma synovitis.

2. Staphylococci, 3. Trauma

DIAGNOSIS

1. Serology - AGP & ELISA 2. Virus isolation

2. 3. Histopathology - no lesions associated with viral invasion

3. Best is to use history of company problem and clinical signs.

PREVENTION AND CONTROL

1. Depopulate farm and decontaminate.
 2. Breeder hens are vaccinated.
 3. Both attenuated and killed vaccine are available.
 4. Must use live vaccine primer before killed vaccine.
Problem breeder flocks may receive 2 live followed by 2 killed vaccines. This is very expensive.
 5. Under certain conditions broilers are vaccinated with live vaccines. Can be given with MDV at 1 day of age
- S.Q.

Diagnosis: Leg problems in broilers or broiler breeders associated with swelling of shank tendons or tendons above the hock joint sometimes accompanied by ruptured tendons, are indicative of reovirus infections.

- A positive blood test (Elisa/AGP test) is of some value as an indication of exposure to reovirus, but does not constitute proof of diagnosis.
- Histopathological examination of affected tissues and isolation of virus or PCR from such tissues are needed for a definite diagnosis.
- **Treatment:** none , but antibiotics to prevent 2nd bacterial infections/Staphylococcal infections. Also some painkillers like acetyl-salicylic acid, aspirins will release the pain in the tendons of affected broilers.

Control: Besides biosecurity, cleaning and disinfection between the cycles vaccination of broiler breeders is a must for reovirus control.

- Since egg transmission is the principal means of spread, a good reo vaccination program consists of live (priming) during the rearing period followed by one or two boosting injections with inactivated reo vaccines at the end of the rearing period is good for the maternal immunity to protect the broilers..
- The goal is to prevent vertical transmission and induce high and uniform “maternal derived antibodies” (MDA) in the breeders that will be passed, via the egg, to the progeny to protect the progeny during the most susceptible period of life.

Mycotic (fungal) diseases

- Fungi produces disease in two ways:
 - They invade and destroys body tissues
 - They can grow in feed and produce mycotoxins which cause disease when consumed by birds.
- Mycotic diseases in poultry are: **Aspergillosis, Mycotoxicosis, Dactylariosis, Candidiasis, Favus..**

Aspergillosis

(brooder pneumonia, mycotic pneumonia)

- It is a mycotic disease characterized mainly by respiratory signs, affect both domestic and wild birds
- **Etiology** - *Aspergillus fumigatus*, *A. Flavus*, *A. Niger*, *A. Glaucus*, *A. Nidulans*, *A. Terreus*
- **Toxins** - *A. fumigatus* has a toxin which is hemotoxic, neurotoxic and histotoxic.
- **Predisposing factors** – poor management practice, concurrent debilitating factors and immunosuppression increase the incidence and severity of infection.

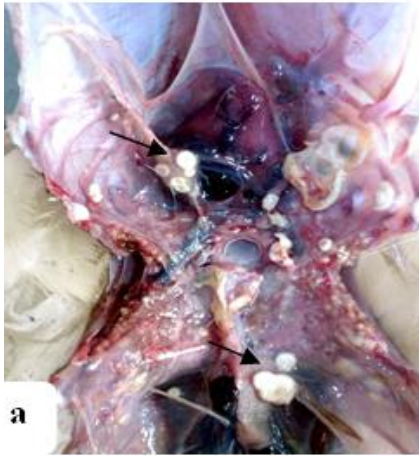
- **Susceptibility** - nearly all species of birds
 - ✓ Young chickens are very susceptible
- **Transmission** - Inhalation of fungus-spores
 - ✓ Egg borne disease (hatchery borne disease)
 - ✓ *Infection may occur through eyes when injuries present in it*
- **Clinical signs - Respiratory signs** - Dyspnea, gasping, accelerated breathing, serous excretion from nose and eyes, may suffer coughing , sneezing , gurgling or rattling noises only if complicated with I.B .or I.L .T (*in Aspergillosis usually there is no sound*)
 - ✓ **Eye infection and Nervous signs (encephalitis)**

General signs - depression, inappetance, increase thirst, pyrexia, dysphagia, dropping of wings, retardation of growth, emaciation, diarrhea in the later stage.

- ✓ Decreased hatchability, extensive embryo mortality
- ✓ Death usually results from suffocation.

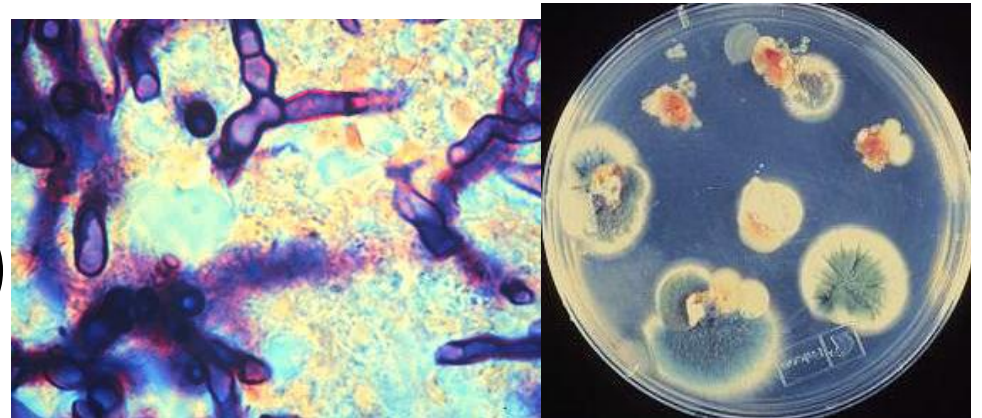
Gross lesions – acute -congestion of parenchymatous organs and mucosa with mucopurulent to gelatinous or caseous exudates

- ✓ Nodular formation, in the early infection the nodules is white then become greenish
- ✓ Caseous material under the eyelids, Ascites



Diagnosis - Case history, Clinical signs, PM lesions

- ✓ Isolation and identification of *Aspergillus spp.*
- ❖ Smear or imprints of nodules after addition of one or two drops of 10 % KOH and gentle heating to clear septated hyphae
- ❖ Staining by H&E and /or periodic acid schiff(PAS)
- ❖ Culture



Treatment - is generally ineffective but a few drugs used for treatment.

- ✓ Mycostatin - 200 mg / litre of H₂O or / kg feed.
- ✓ amphotericin B - 100 mg / liter or / kg feed

Aflatoxicosis

- **Mycotoxigenesis** – all diseases caused by fungal toxins
- **Aflatoxicosis** is the most prevalent
- ✓ **Aflatoxins** are highly toxic and carcinogenic mycotoxins produced by *A. flavus*, *A. parasiticus* and *Penicillium puberulum*.....
- ✓ There are 4 types of aflatoxins B1, B2 ,G1 and G2
- ✓ B1 is the most toxic and is the highest in contrn

Transmission - Ingestion of aflatoxin-contaminated animal feedstuffs and water

Epidemiology – worldwide, all species of birds

- ✓ Young birds are more susceptible than adults

Clinical signs– decreased production & hatchability

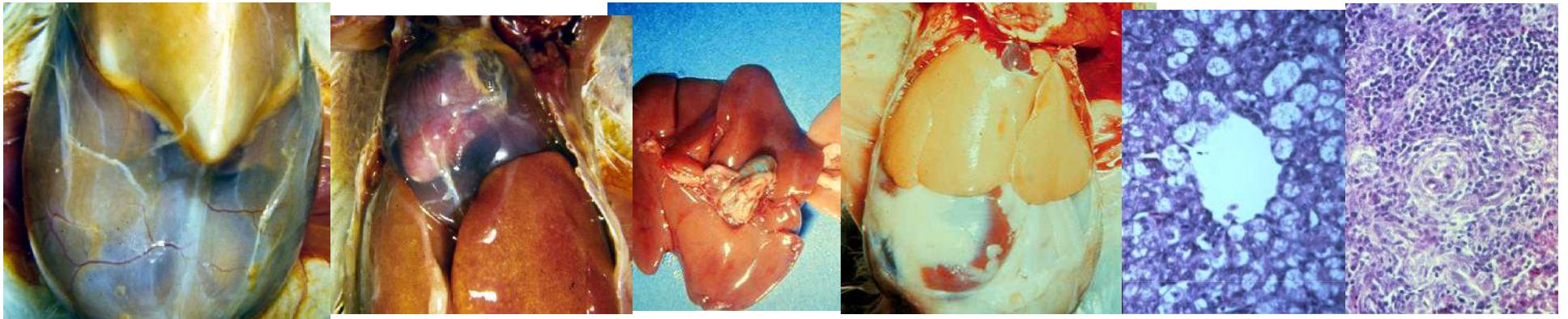
- ✓ Immunosuppressive effect
- ✓ Undigested food in the dropping, anemia, Lameness, Ataxia, convulsions, and opisthotonus

Gross lesions - Enlargement of the liver, kidneys , spleen and pancreas

- ✓ atrophy of bursa fabricci , thymus gland , spleen and testes.
- ✓ Hemorrhages present under the skin, on muscles and on the internal organs
- ✓ In chronic stage ascites, hydropericardium, cirrhosis

Histopathology - Necrosis of liver cells and presence of fat in hepatocytes, and Liver cirrhosis

- ✓ Vascular and degenerative lesions in pancreas and kidney.



Diagnosis - clinical history and signs.

- ✓ Clinical signs/ lesions are not pathognomonic
- ✓ A definitive diagnosis of mycotoxicosis involves identification and quantification of specific toxins.
- ❖ Analytic techniques for mycotoxins include, ELISA, chromatography and mass spectrometry

Prevention/Control - Using mycotoxin-free feed

- ✓ Proper storage, manufacturing and management practices that prevent mold growth and mycotoxin formation.
- ✓ Keeping the feed fresh, and maintaining clean feed handling equipment
- ✓ Pelleting feed destroys some fungal spores and generally decreases the fungal burden.
- ✓ Treat concurrent bacterial or parasitic infections.

Major Parasitic Diseases of Poultry

- Helminthes parasites, Ectoparasites
- Protozoal diseases - the most common disease of poultry caused by protozoa: ***Coccidiosis & Blackhead***

Coccidiosis

- The most prevalent infectious disease, protozoan disease, in poultry industry
- It is most injurious to young chicks and usually sub-acute or chronic in older
- In geese - highly fatal form in which the kidneys are the organ principally involved.
- Two importance genera: **Eimeria** and Isospora

Chicken coccidiosis – by 9 *Eimeria* species

1. *E. tenella* - "bloody coccidiosis"
2. *E. necatrix* - In **acute cases** the birds may die in 5-7 days after infection and In **chronic cases**, the birds may live for a long time with a wasting illness
3. *E. acervulina* - commonest and causes extensive morbidity but low mortality
- 4 *E. brunetti*, 5 *E. maxima*, 6 *E. mivati* and
7. *E. hagani* - moderate infection
8. *E. praecox* and - 9. *E. Mitis* - no appreciable tissue damage.

- **Symptoms:** (Acute –chronic) - dead birds --- droopiness and depression, Huddling , wings dropped, feathers ruffled and eyes closed

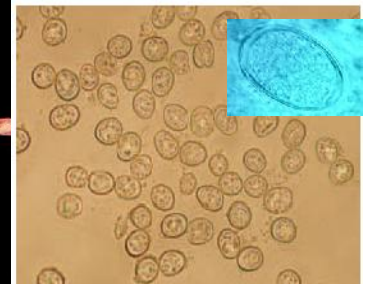
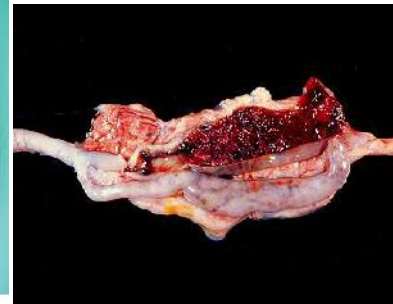


- Diarrhea is commonly present and frequently mixed with blood (paleness of the comb and wattles)
- In older – chronic ... weakness ...



- **Diagnosis based on:**

1. Symptom and autopsy lesions
2. A positive diagnosis - microscopic examination of a thin film of fecal material/intestinal mucosa



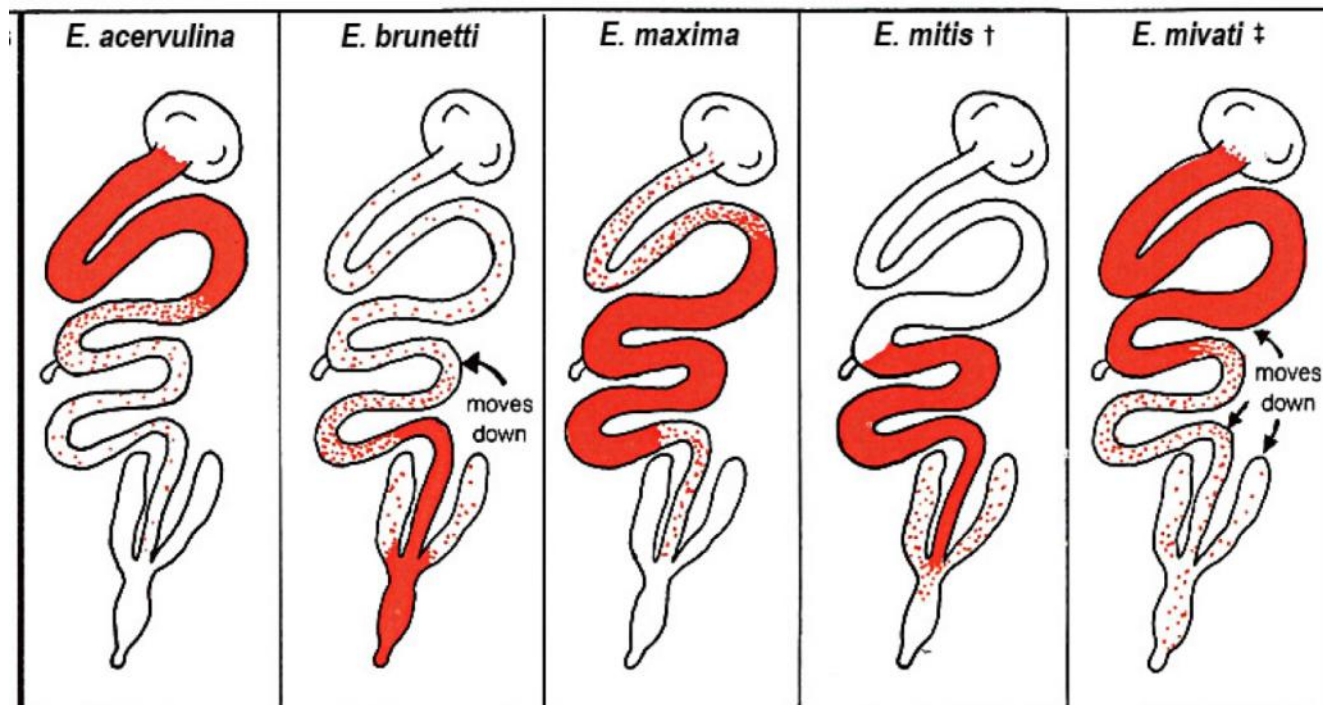
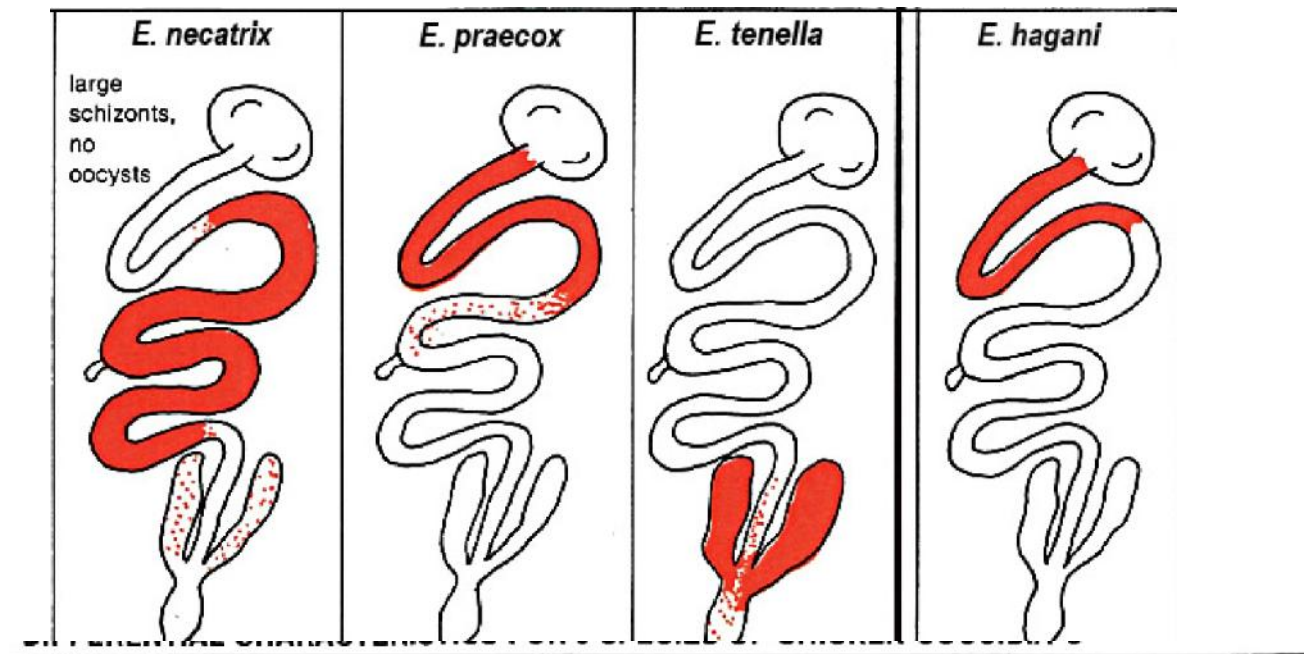
Treatment and Prevention,/Control:

1. Proper management - dry litter, clean and dry house, Avoid crowding of chicken.
 - Proper feeding of Vitamin A and K.
2. Treatment with drugs.
 - **sulfa** drugs Sulfaquinoxaline, Sulfamethazine, Sulfaquanidine, Sulfamerazine, Nitrofurazone, ureomycine, Terramycin, **Amprolium** etc

Coccidiostats - Long term continuation

administration of coccidiostats will induce resistance to a specific drug thus, changing of coccidiostats after using it for certain period of time is important

3. Commercial available vaccine (Coccivac)



Turkey Coccidiosis

- 1 ***E. meleagrimitis*** - huddle together with drooping wings, ruffled feathers, fluidy droppings (may contain blood with) severe weight loss and high mortality in young poults.
 - Necrotic enteritis in the duodenum & upper jejunum
2. ***E. Adenoeides*** - fluid droppings containing mucus and a small amount of blood.
 - A severe enteritis in the lower SI and ceca
3. ***E. gallopavonis*** – watery droppings and may contain tinges of blood. Highly pathogenic
 - In the ceca large intestine, and lower one third of the SI

Low pathogenic

- 4. *E. melagridis* - lower small intestine and ceca
- 5. *E. dispersa* - Upper one half SI
- 6. *E. subrothunda* - Upper two third SI
- 7. *E. innocua* – SI

Goose coccidiosis: - *E. truncata*

- In epithelium of the uriniferous tubules
- Very acute 2-3 days with high Mortality

HISTOMONIASIS (Infectious enterohepatitis/blackhead)

- The most destructive infectious disease to which turkeys are extremely susceptible
- Etiology - *Hiistomonas meleagridis*

Occurrence: widespread and young turkeys (1-3 months) are highly susceptible

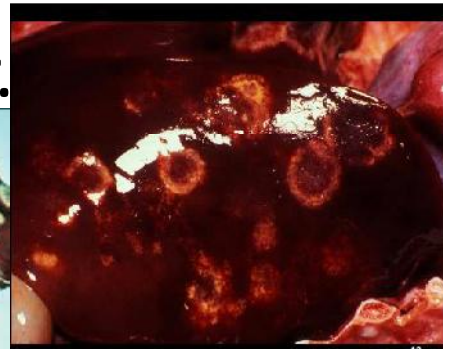
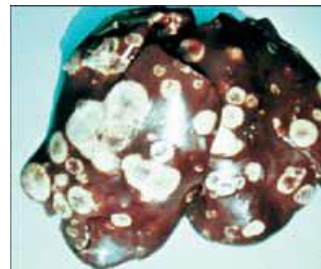
Transmission: 1. By carrier turkeys, chickens whose droppings contaminate the environment.

2. By ingestion of the cecal worm or its eggs and Earthworm also could transmit the disease.

Symptoms: Acute in young poults, more chronic in mature

- Inappetence, ruffled feather, droopy wings and drowsiness, emaciation
- a light green to a brownish/orange colored diarrhea
- The skin of the head becomes dark in color.

Lesions - Ulcerative enteritis,
Ulcerative hepatitis and Peritonitis



Diagnosis:- based on history and lesion

- Laboratory diagnosis: Direct microscopic examination of fresh fecal material

Prevention:

- Do not raise turkey near to chicken (isolation of rearing)
- Control cecal worms with medication, phenothiazine or hygromycin.

Treatment/Preventive for Blackhead,

- *Hepzide, Ipropan, Furox, Carb-o-Sep* etc

Helminths

Nematodes

1 ***Ascardia galli*** (large roundworm) – large (5-12 cm) , yellowish white worm... Small intestine

- most common nematode parasites of chickens

Life cycle (direct) - eggs are passed out in the droppings. The infective embryonated eggs are then picked up by other birds and hatch in the intestine – adult within 4 weeks

Symptoms - Emaciation, weakness .

Prevention: proper sanitation

Treatment: Piperzine hydrochloride



The Caecal Worm (*Heterakis gallinarum*)

- Small worm (0.7-1.5 cm), lives in the caecum
- Can harbour *Hiistomaonas meleagridis*
- **Life cycle:** similar with large roundworm
- Symptoms – unthriftiness, death.....
- Treatment - Phenothiazine or piperazine.

Hair Worms (*Capillaria spp.*)

These are long thread-like worms found in the crop esophagus small intestine and caecum.

- **Life Cycle:** indirect – IH such as an earthworm
- Transmission - the bird eats the earthworm and the parasite is released into the gut.

Syngamus trachea: Gape/red /forked worm -Male permanently attached in copula to female (Y)

Life cycle- direct/ingestion of eggs containing the embryos

Pathology - In young birds (< 8 weeks), the worms cause trachitis & accumulation of mucus and interference with respiration

- Sneezing, coughing and frequently shake their heads in an effort to expel the worms and mucus
- **Prevention** -frequent removal of litter to prevent accumulation of worm eggs and larvae.
- **Treatment** - unsatisfactory

Nematode of the eye.

- I) *Oxyspirura mansoni* and *Filaria mansoni*
 - found beneath the nictitating membrane, conjunctival sacs and **nasolacrimal** ducts.
 - Life cycle – indirect and
 - ingestion of Infected cockroach
- Pathology: Ophthalmia
- Treatment: Put a few drops of 5% creolin in the eye

Cestode Infestation

General morphology and characters:

- 1) Tapeworms are flat, white and segmented
 - 2) Adult body consists of: head or scolex, neck or growth zone and Strobila, Site - **digestive tract**
- ✓ Poultry became infected by swallowing infected IH (Invertebrate intermediate)

Symptoms and gross pathology - Catarrhal enteritis and diarrhea, emaciation and loss of appetite

Treatment: piperazine, chlorophene, niclosamide...

Control: proper disposal of poultry manure and keeping the IH out of the chicken house

Common poultry tapeworms.

1. *Amoebotaenia sphenoides*

- Intermediate host earthworms
- Region – Dudenum

2. *Choanotaenia infundibulum*

- Characteristics: rostellum armed with a single row of relatively few and very large hooks.
- IH: House flies, grasshoppers and beetles
- Region - Jejunum

3) ***Davainea proglottina***

- IH - Snails and slugs
- Region – Duodenum

4. ***Raillietina cesticellus***

- The most common species of cestode
- Rostellum with two rows of hooks
- IH - beetles (ground and dung)
- Region – Jejunum

5. ***Raillietina echinobothrida*** - IH - ants

- Region- Ileum

External Parasites

- **Lice, mites, ticks, fleas** and flies
- Deaths resulting from infestations of external parasites are rare, but production losses often occur because of the irritation caused to the birds. In addition, external parasites suck blood which often causes birds to become anaemic.

Ticks - The fowl tick, ***Argas persicus***

- The most important tick parasite of birds
- Loss of host blood, egg and meat production.
- Transmitters of other parasites, such as avian spirochaetosis, tularemia, babesiosis, anaplasmosis, encephalomyelitis and certain rickettsial diseases.

Lice -The lice affecting poultry are chewing lice; have cutting mouth parts and feed upon bits of feathers or scales from the skin.

- By their sharp claws and spiny structure, they cause considerable irritation and annoyance to the host

LC: - permanent parasites and host specific

- Lay eggs at the base of feathers, and hatch

Symptoms: lice in the vent, skin areas of a bird and reduced egg production during severe infestation

- **Common species of chicken lice.**

1) *Menacanthus stramineus* (body louse)

2) *Menopon gallinae* (chicken shaft louse)

3) *Gonicotes hologaster* (chicken pluff louse)

4) *Goniodes gigas* (large chicken body louse)

5) *Cuclotogaster heterographus* (chicken head louse)

6) *Liperurus caponis* (chicken wing louse)

7) *Mencanthus cosnutus* (chicken body louse)

- **Treatment:** sprayed, dusted or dipped with an appropriate insecticide. Because lice live only a few days off the host emptying a shed or yard for a week will clean it.

Mites - there are about twenty species of mites which are known to infest domestic poultry, but only a few of them are sufficiently injurious to be of economic importance

***Dermanyssus gallinae* – Chicken/red/rooster mite**

✓ They are intermittent parasites, hiding in cracks and crevices during the day and coming out to feed on the fowls at night - **In back yard chicken**
(Not common on commercial poultry farms)

Symptoms and diagnosis - because of the feeding habits, infestation may easily become severe. Their presence is easily determined by **examine the ends/undersides** of the **roosts** at the points of support

Northern fowl or Northern feather mite

- ***Ornithonyssus sylviarum***
- It occurs mostly in temperate as well as in subtropical areas.
- It lives and reproduces on the fowl
- The mites are most commonly found about the base of the tail and around the vent

Tropical feather mite (*Ornithonyssus bursa*)

- Common in tropical area
- Closely related to the northern fowl mite.

Scaly leg mites

- *Knemidocoptes mutans*
- The entire life cycle is spent upon the host.
- Severe irritation caused by the activities of the mites results in accumulation of grayish, dry debris under the scales - the shanks appear to be enlarged.
- Treatment: Soak the shank in soapy water and allow them to dry, then apply oil of caraway 1 part to petrolatum (Vaseline) 5 parts



The depluming mite

- *Knemidocoptes gallinae*.
- Resembles the scaly leg mite in general structure although it is smaller.
- They live at the base of the feathers, where they produce the condition known as "depluming scabies".
- The intense irritation induced by the mites causes the affected birds to pull out its own feathers
- Treatment similar to scaly leg mite.

The stick tight flea

- ***Echinophaga gallinacea*** – attached to the comb, face earlobes and wattles,
- They tend to be found in clusters and have brown to black color



Mosquitoes - common chicken house mosquitoes belong to ***Culex quinquefasciatus***.

- ***Aedes Stimulans*** – transmit fowl pox virus
- ***A. aegypti*, *A. vexans***

Flies - *Musca domestica* (IH for *Raillietina cesticillus* and *Choanotaenia infundibulum*)

NUTRITIONAL DISORDERS

- **Nutritional imbalances may arise from**
 - Deficiency of nutrients in the diet
 - Destruction or inactivation of nutrients during feed processing and manufacture,
 - When the components of the diet are not balanced
 - Impaired absorption and metabolism

Vitamins - required in minute amounts

Fat Soluble Vitamins
Vitamin A (Retinol)
Vitamin K
Vitamin E
Vitamin D

Water Soluble Vitamins	
Vitamin:	Name:
B1	Thiamine
B2	Riboflavin
B3	Niacin
B5	Pantothenic Acid
B6	Pyridoxine
B7	Biotin
B9	Folate
B12	Cobalamin
C	Ascorbic Acid

Vitamin A - involved in

- Vision through synthesis of **rhodopsin** (retinal purple) which is receptive to light.
- Synthesis of muco-polysaccharides and mucus secretion in the skin and mucosae

Deficiency Vit A – Ataxic, anorexia, growth retardation, drowsiness, weakness, emaciation, and ruffled feathers

- The yellow pigment in the shanks and beaks is usually lost,
- **Pale** comb and wattles,
- Ocular lesions, conjunctivitis, Nasocular discharge

Post-mortem lesions

- Eyelids inflamed and adhered with exudate
- Excessive urates in kidneys and ureters.
- Pustules in mouth and pharynx.

Microscopically - Atrophy and keratinisation (squamous metaplasia) of the epithelial cells

Diagnosis - Signs, lesions, feed formulation

Treatment - Vitamin A in drinking water

Prevention - Supplementation of diet with vit. A

Vitamin E Deficiency

- Vitamin E is essential for the normal embryonic development of birds. It is an **antioxidant** (protection of cellular membrane)
- Clinical conditions associated with its deficiency: muscular dystrophy, encephalomalacia and exudative diathesis and embryonic mortality
- In encephalomalacia - nervous signs may include *ataxia* (loss of balance and falling backward), *torticollis*, *myoclonus* (repeated muscle contractions of the legs), *Paralysis*.....
- In exudative diathesis
- *subcutaneous edema*



Post-mortem Lesions - Encephalomalacia form -

lesions in the CNS ---- Softened and swollen cerebellum with petichiae to ecchymotic Hemorrhages, necrosis

In the exudative diathesis form - subcutaneous edema under the ventral skin region.

In the muscular dystrophy form - muscle fibers can undergo degeneration

Diagnosis - Signs, lesions, Response to Rx
- analysis of the feed ration

Treatment and control - Treatment of affected birds with vitamin E preparations

- Adequate levels of vitamin E in the diet

vitamin K

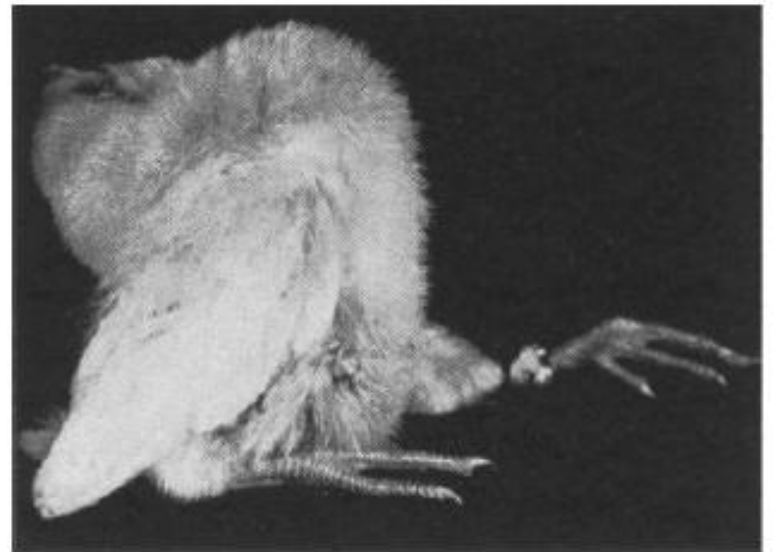
- Vitamin K is used as a cofactor and essential for the hepatic synthesis of coagulation factors, and osteocalcin
- Deficiency of vitamin K;
 - leads to a considerable reduction in blood levels of **prothrombin** and **increased clotting time**.
 - Hemorrhages may occur subcutaneously, intermuscularly
 - *Symptoms associated with the skeletal system are not as apparent*

Thiamin (Vitamin B1)

- ❖ Thiamin is an important cofactor in carbohydrates metabolism and for the synthesis of acetylcholine
- ❖ Deficiency of this vitamin - feed that contains thiaminase (such as fishmeal) or treatments with the anticoccidial amprolium
- ❖ Deficiency of thiamin leads to **extreme anorexia, polyneuritis, and death**

Clinical Signs and Pathology of Deficiency.

- **Anorexia** - loss of weight, ruffled feathers, apparent paralysis of muscles
- **Polyneuritis** - chicken characteristically sits on its flexed legs and draws back the head in a “**star-gazing**” position.
- A progressive decrease in body T° & RR
- Atrophy of organs (genital, heart, stomach and intestine), Death
- **No macroscopic lesions** in thiamine deficiency.
- **Treatment:** oral/ vitamin.



Vitamin D3 (Cholecalciferol)

- A deficiency in vitamin D3 will lead to **rickets** in immature flocks and **osteomalacia** in mature laying and breeding stock
 - depressed growth rate, poor feathering.
 - decreased skeletal density
 - costochondral (rib to spine) junctions are enlarged
 - the end plates of the long bones are irregular
 - enlargement of the parathyroid glands.
 - decrease in egg production and a marked deterioration in shell quality.

- **Osteomalacia/cage layer fatigue** - in laying hens,
 - Characterized by decreased mineral in bone resulting in soft, pliable bones including the beak, curved keel bone and bending of the ribs at the costochondral junction.
 - Vitamin D3 deficiency, Ca/P imbalance



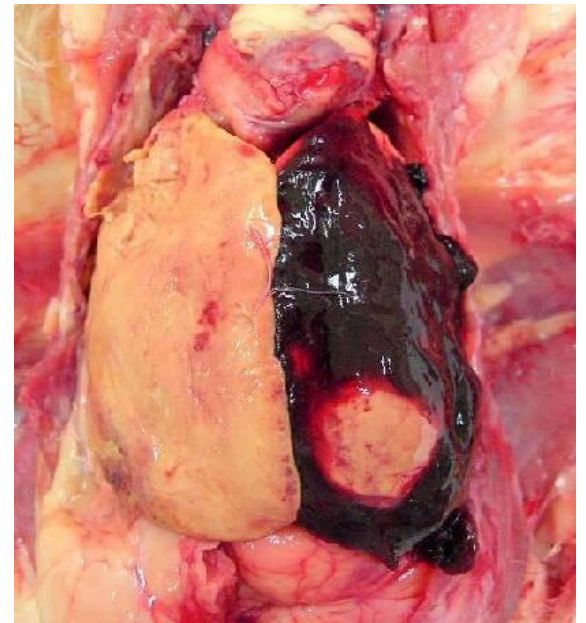
Miscellaneous/Metabolic Diseases

- **Gout** is a condition very commonly seen in older layer flocks and is related to kidney failure
 - Birds with gout usually show no clinical signs before death or may be extremely thin.
 - The lesions of gout are associated with the accumulation of urates/uric acid on the surfaces of the internal organs (**visceral gout**) as well as within joint spaces and along synovial membranes (**articular gout**).



- **Fatty liver syndrome** - caused by an imbalance of energy and protein intake.
- Most often in caged laying hens

Lesion- liver is enlarged, pale orange, soft, friable and is easily fractured - rupture of the fatty liver with hemorrhage into the abdominal cavity is a common cause of death in laying hens.



- **Ascites Syndrome/Right Ventricular Failure of Broiler Chickens**
 - Characterized by **pulmonary hypertension, right-sided heart failure** and **accumulation of excessive fluid** (transudate) in the abdomen.
 - Common in broiler chickens raised at high altitudes (decreased PO₂).
 - Respiratory infection can promote this problem
- **Clinical Signs:** Sudden death, reluctant to move, respiratory distress, distended abdome, Combs and wattles are cyanotic

- **Gross lesions:** Cyanotic combs and wattles, abdomen is distended by abundant clear, yellow fluid and fibrin, lungs edema, hydropericardium,

