

Here are the short-note answers for all "Q.4" questions from the 2024, 2023, 2022, 2019, and 2018 veterinary pathology papers you provided.

2024 Exam (Q.4)

4.1 Explain why? In lymphoid leucosis the transformed B cells have only IgM on their surface

In Lymphoid Leucosis (LL), the Avian Leukosis Virus (ALV) targets an immature B-lymphocyte precursor in the bursa of Fabricius. This viral-induced neoplastic transformation "freezes" or arrests the B-cell's maturation process.

Normally, B-cells mature and switch their surface antibody isotypes from IgM to other types (like IgG or IgA). Because the LL-transformed cells are "stuck" in this immature state, they proliferate as a clonal population that only expresses the early-stage **surface IgM**. They never mature to the point of isotype switching, which is why the resulting B-cell lymphoma is characterized by cells bearing only IgM.

4.2 Write down the Pathogenesis of Johne's disease

Etiology: *Mycobacterium avium* ssp. *paratuberculosis* (MAP).

Pathogenesis:

1. **Ingestion:** The primary route is fecal-oral, typically in young calves.
2. **Invasion:** MAP organisms are transported across the intestinal wall by M-cells, primarily in the Peyer's patches of the distal ileum.
3. **Macrophage Uptake:** The bacteria are phagocytosed by macrophages in the submucosa.
4. **Intracellular Survival:** MAP is a facultative intracellular pathogen. It resists intracellular killing and multiplies slowly within the macrophages.
5. **Chronic Inflammation:** This triggers a chronic, cell-mediated immune response, leading to a massive influx of macrophages and lymphocytes. The macrophages cluster and transform into epithelioid cells, forming a diffuse **granulomatous enteritis**.
6. **Thickening:** This cellular infiltration causes severe, corrugated thickening of the intestinal mucosa, which impairs nutrient and protein absorption, leading to chronic diarrhea, protein-losing enteropathy, and progressive emaciation (wasting).

4.3 Describe the important histopathological lesions for the diagnosis of poultry diseases like Avian Pox & Intestinal coccidiosis

- **Avian Pox:** The pathognomonic histopathological lesion is the presence of large, eosinophilic, **intracytoplasmic inclusion bodies** called **Bollinger bodies**. These are found in infected epithelial cells of the skin (cutaneous "pox" form) or mucous membranes ("wet" or diphtheritic form). These cells also show marked ballooning degeneration and hyperplasia.
- **Intestinal Coccidiosis:** The lesions vary by *Eimeria* species. The key findings are the developmental stages of the parasite within the intestinal epithelium or lamina propria.
 - For **cecal coccidiosis** (*E. tenella*), the most important diagnostic lesion is the presence of very large **second-generation schizonts** deep within the lamina

propria and submucosa of the ceca. These schizonts, along with gametocytes, cause massive destruction of the mucosa, severe hemorrhage, and necrosis.

4.4 Describe etiology and Pathogenesis of African horse sickness

Etiology: African Horse Sickness Virus (AHSV), an **Orbivirus** from the Reoviridae family. It is non-contagious and transmitted by biological vectors.

Pathogenesis:

1. **Transmission:** The virus is transmitted by biting midges (*Culicoides* species).
2. **Replication:** The virus initially replicates in regional lymph nodes and spleen, leading to viremia.
3. **Tropism:** The virus has a strong tropism for **endothelial cells** (vasculotropic) and, to a lesser extent, hematopoietic cells.
4. **Vascular Damage:** Widespread replication in endothelial cells causes severe damage to blood vessels, leading to increased vascular permeability.
5. **Edema & Hemorrhage:** This vascular damage results in the characteristic lesions:
 - **Pulmonary Form (Dunkop):** Severe pulmonary edema, leading to respiratory distress and death.
 - **Cardiac Form (Dikkop):** Subcutaneous edema of the head, neck, and chest, along with significant hydropericardium (fluid around the heart).
 - Hemorrhages are common, especially subendocardial and on serosal surfaces.

4.5 Describe the Pathogenesis of Dourine disease

Etiology: *Trypanosoma equiperdum*, a protozoan parasite.

Pathogenesis:

1. **Transmission:** Dourine is a venereal disease, transmitted almost exclusively during **coitus** (sexual contact).
2. **Local Stage:** The trypanosomes first multiply at the site of infection (vagina, urethra), causing inflammation, discharge, and edema of the external genitalia (e.g., "silver dollar" plaques).
3. **Systemic Invasion:** The parasites then invade the bloodstream, causing intermittent parasitemia and fever.
4. **Neuroinvasion:** The key pathogenic event is the parasite's invasion of the nervous system. It causes progressive **peripheral neuritis** (inflammation of nerves).
5. **Clinical Disease:** This nerve damage leads to the characteristic signs: muscle atrophy, depigmentation, loss of coordination, and progressive ascending paralysis, ultimately leading to recumbency and death.

4.6 Describe etiology and pathogenesis RP

Etiology: Rinderpest (RP) virus, a **Morbillivirus** in the Paramyxoviridae family. (Note: Rinderpest was declared globally eradicated in 2011).

Pathogenesis:

1. **Transmission:** Inhalation or ingestion of the virus.
2. **Initial Replication:** The virus replicates in the tonsils and regional lymph nodes (e.g., pharyngeal, mandibular).
3. **Viremia:** A massive viremia follows, spreading the virus throughout the body.

4. **Tropism:** The virus is both **lymphotropic** (attacks lymphocytes) and **epitheliotropic** (attacks epithelial cells).
5. **Lesions:**
 - **Lymphoid:** Severe necrosis of lymphoid tissues (lymph nodes, spleen, Peyer's patches) leads to profound leukopenia and immunosuppression.
 - **Epithelial:** The virus causes necrosis and erosion of the epithelial lining of the entire gastrointestinal tract, from the mouth (stomatitis) to the rectum. This results in severe, watery, bloody diarrhea and dehydration. The characteristic lesion is "**zebra stripe**" **hemorrhages** in the rectal folds.

4.7 Describe etiology and pathogenesis of Infectious canine Hepatitis

Etiology: Canine Adenovirus-1 (CAV-1).

Pathogenesis:

1. **Transmission:** Ingestion of virus (from urine, feces, or saliva).
2. **Initial Replication:** The virus replicates in the tonsils, leading to viremia.
3. **Tropism:** CAV-1 has a strong affinity for **hepatocytes** (liver cells) and **endothelial cells** (lining of blood vessels).
4. **Lesions:**
 - **Hepatitis:** Massive viral replication in hepatocytes causes acute **centrilobular to panlobular necrosis**. This leads to liver failure and jaundice.
 - **Vasculitis:** Replication in endothelial cells causes widespread vasculitis, leading to hemorrhages in multiple organs (e.g., petechiae on kidneys, "paintbrush" hemorrhages on gastric serosa) and edema.
5. **Inclusion Bodies:** The pathognomonic microscopic lesion is the presence of large, **intranuclear inclusion bodies** in hepatocytes and endothelial cells.
6. **"Blue Eye":** During recovery, antigen-antibody complexes can deposit in the cornea, causing a Type III hypersensitivity reaction that leads to corneal edema, giving the eye a cloudy, blue appearance.

4.8 Describe etiology and pathogenesis of coccidiosis in Rabbit

Etiology: *Eimeria* species. There are two forms in rabbits:

1. **Hepatic Coccidiosis:** Caused by *Eimeria stiedae*.
2. **Intestinal Coccidiosis:** Caused by several other species (*E. magna*, *E. intestinalis*, etc.).

Pathogenesis:

1. **Ingestion:** Rabbits ingest sporulated oocysts from contaminated feed, water, or bedding.
2. **Invasion:** Sporozoites are released and invade their target epithelial cells.
 - ***E. stiedae*:** Invades the **bile duct epithelium**.
 - **Intestinal species:** Invade the **intestinal epithelial cells**.
3. **Replication:** The parasites undergo multiple cycles of asexual (schizogony) and sexual (gametogony) replication, destroying the host cells in the process.
4. **Lesions:**
 - **Hepatic:** Cell destruction and host reaction cause massive **bile duct hyperplasia** (proliferation) and inflammation (cholangitis). This appears grossly as yellowish-white, nodular or cord-like streaks throughout the liver.
 - **Intestinal:** Cell destruction leads to villous atrophy, enteritis, malabsorption, and diarrhea.



2023 Exam (Q.4)

4.1 Aetiology, mode of transmission and gross lesions of blue tongue in sheep

- **Etiology:** Bluetongue Virus (BTV), an **Orbivirus** (family Reoviridae).
- **Mode of Transmission:** Transmitted biologically by biting midges of the *Culicoides* species. It is not contagious between animals.
- **Gross Lesions:** The lesions are due to viral-induced vasculitis (inflammation of blood vessels) and subsequent thrombosis.
 - Edema of the face, lips, and ears.
 - Hyperemia (redness) of the oral and nasal mucosa.
 - **Cyanosis** (blue discoloration) and edema of the tongue, which may protrude.
 - Ulcers and necrosis on the dental pad and tongue.
 - **Coronitis** (inflammation of the coronary band), leading to lameness.
 - Hemorrhages, especially at the **base of the pulmonary artery** (a pathognomonic lesion).
 - Skeletal and cardiac muscle necrosis.

4.2 Aetiology and lesions of Ranikhet disease

- **Etiology:** Ranikhet Disease (also known as Newcastle Disease) is caused by **Avian Paramyxovirus-1 (APMV-1)**. Strains are classified by pathogenicity:
 - **Velogenic:** Highly pathogenic.
 - **Mesogenic:** Moderately pathogenic.
 - **Lentogenic:** Low pathogenicity (often used for vaccines).
- **Lesions (Velogenic Viscerotropic form):**
 - **Gross Lesions:** Characterized by hemorrhages and necrosis.
 - **Proventriculus:** Hemorrhages at the junction with the gizzard.
 - **Intestine:** Severe, hemorrhagic, and necrotic foci in lymphoid tissue, especially the **cecal tonsils** and Peyer's patches.
 - **Other:** Splenic necrosis, cyanosis of the comb and wattles, and respiratory or nervous signs (torticollis, paralysis).
 - **Microscopic Lesions:** Necrosis of lymphoid tissue and epithelial cells, with hemorrhage and inflammation.

4.3 Gross and microscopic lesions of Black quarter

- **Etiology:** *Clostridium chauvoei*.
- **Gross Lesions:**
 - Affects large muscle groups (thigh, shoulder, hip) in cattle.
 - The affected muscle is dark red to **black**, dry, and spongy.
 - It is **crepitant** (crackles when pressed) due to the presence of gas bubbles produced by the bacteria.
 - The lesion has a characteristic "**rancid butter**" smell.
 - The overlying subcutaneous tissue is filled with yellowish, gelatinous, and

blood-stained edema.

- **Microscopic Lesions:**
 - Extensive **necrotizing, hemorrhagic, and emphysematous myositis**.
 - Muscle fibers are separated by gas bubbles, edema, and hemorrhage.
 - There is widespread coagulative necrosis of muscle fibers.
 - A striking feature is the **relative lack of neutrophils** (leukocytes are destroyed by bacterial toxins).
 - Large, rod-shaped, Gram-positive bacteria are abundant in the tissue.

4.4 Pathology of Glanders in horse

- **Etiology:** *Burkholderia mallei*.
- **Pathology:** Glanders is a zoonotic disease characterized by the formation of **pyogranulomatous nodules** in the lungs, skin, and upper respiratory tract.
- **Lesions (Three Forms):**
 1. **Nasal Form:** Nodules form in the nasal mucosa, especially on the septum. These nodules ulcerate, discharging a thick, sticky, purulent exudate. The ulcers often heal, leaving characteristic "**star-shaped**" scars.
 2. **Pulmonary Form:** Firm, greyish-white, "glanders nodules" are found throughout the lungs, resembling tuberculosis. These nodules have a caseous or purulent center and are surrounded by a fibrous capsule.
 3. **Cutaneous Form ("Farcy"):** Pyogranulomatous nodules ("**fancy buds**") develop along the lymphatic vessels of the limbs, head, and neck. These vessels become thickened and cord-like ("fancy pipes"). The nodules rupture, discharging a thick, oily, yellowish pus.
- **Diagnosis:** The **Strauss reaction** (severe, purulent orchitis 2-3 days after intraperitoneal injection in a male guinea pig) is a classic diagnostic test.

4.5 Pathology of Babesiosis in cattle

- **Etiology:** *Babesia bovis* or *Babesia bigemina*, tick-borne protozoan parasites.
- **Pathology:** The parasites invade and multiply within **erythrocytes (RBCs)**, causing massive **intravascular hemolysis** (destruction of RBCs in the bloodstream).
- **Gross Lesions:**
 - The cardinal sign is **hemoglobinuria** (hemoglobin in the urine), making the urine dark red or "port wine" colored.
 - Severe **anemia** (pale mucous membranes).
 - **Icterus** (jaundice) due to the breakdown of hemoglobin.
 - **Splenomegaly:** The spleen is severely enlarged, dark, and has a "pulpy" or "blackberry jam" consistency.
 - The liver is enlarged, yellowish-brown, and friable.
 - **Cerebral Babesiosis** (*B. bovis*): Sequestration of infected RBCs in brain capillaries causes nervous signs. The brain's grey matter may appear pink.

4.6 Pathology of Aflatoxicosis in poultry

- **Etiology:** Ingestion of aflatoxins, mycotoxins produced by fungi like *Aspergillus flavus* and *A. parasiticus*. Aflatoxin B1 is the most toxic.

- **Pathology:** Aflatoxin is primarily a potent **hepatotoxin**, and also carcinogenic and immunosuppressive. The **liver** is the primary target organ.
- **Lesions:**
 - **Acute:** High doses cause massive liver necrosis, hemorrhage, and rapid death.
 - **Chronic:** (More common) Low-level exposure leads to:
 - **Gross:** The liver is enlarged, firm, pale yellow, and fatty. Ascites (fluid in the abdomen) is common due to hepatic failure and portal hypertension.
 - **Microscopic:** The most characteristic lesions are **diffuse fatty change (steatosis)**, marked **bile duct hyperplasia** (proliferation of bile ductules), and progressive fibrosis leading to **cirrhosis**. This results in poor growth, reduced egg production, and increased susceptibility to other diseases.

4.7 PPR in goats

- **Etiology:** Peste des Petits Ruminants (PPR) virus, a **Morbillivirus** (family Paramyxoviridae).
- **Pathology:** The virus is lymphotropic and epitheliotropic, similar to Rinderpest. It causes high fever, severe diarrhea, and respiratory signs.
- **Lesions:**
 - **Oral:** Necrotic erosions on the gums, tongue, and dental pad.
 - **Oculonasal:** Severe purulent conjunctivitis and nasal discharge, often matting the eyes and nose.
 - **Intestinal:** The most characteristic lesion is "**zebra stripes**" or "zebra markings", which are linear hemorrhages in the mucosa of the colon and rectum, overlying inflamed Peyer's patches.
 - **Respiratory:** A severe **bronchopneumonia** is very common and is often the cause of death.
 - **Microscopic:** Syncytia (giant cell) formation and intracytoplasmic/intranuclear inclusion bodies are seen in affected epithelia.

4.8 KFD

- **Etiology:** Kyasanur Forest Disease (KFD) virus, a **Flavivirus** (family Flaviviridae).
- **Transmission:** Transmitted by ticks, primarily *Haemaphysalis spinigera*.
- **Pathology:** KFD is a zoonotic **hemorrhagic fever** that primarily affects monkeys (*presbytis*) and humans in forest areas.
- **Lesions:**
 - **In Monkeys:** It is highly fatal, causing high fever, tremors, and hemorrhagic disease. Gross lesions include widespread hemorrhages in the gastrointestinal tract, lungs, liver, and kidneys. There is also focal necrosis in the liver.
 - **In Humans:** The disease presents as a sudden-onset fever, headache, and severe muscle pain, which can progress to a hemorrhagic phase (bleeding from the nose, gums, GI tract) and/or neurological complications (meningoencephalitis).



2022 Exam (Q.4)

4.1 Tyzzer's disease

- **Etiology:** *Clostridium piliforme* (formerly *Bacillus piliformis*), a motile, spore-forming, Gram-negative rod.
- **Pathology:** It is an acute, fatal disease primarily affecting foals, rabbits, and rodents (mice, hamsters). It is often triggered by stress.
- **Lesions:** The disease is characterized by a "classic triad" of lesions:
 1. **Hepatitis:** The liver is enlarged and studded with numerous, multifocal, pale-yellow or white necrotic foci (miliary necrosis).
 2. **Myocarditis:** Similar necrotic foci may be seen in the heart muscle.
 3. **Enteritis:** Hemorrhagic and necrotic enteritis and colitis, especially in the lower ileum, cecum, and colon.
- **Microscopic:** The pathognomonic lesion, best seen with a silver stain (like Warthin-Starry), is the presence of long, filamentous bacilli arranged in "**haystack**" or "**pickup sticks**" patterns within hepatocytes at the margin of the necrotic foci.

4.2 Scrapie

- **Etiology:** A **prion**, which is an abnormal, protease-resistant isoform (PrPSc) of a normal host-coded protein (PrPC).
- **Pathology:** Scrapie is a fatal, transmissible spongiform encephalopathy (TSE) of sheep and goats. It causes progressive neurodegeneration.
- **Gross Lesions:** There are **no specific gross lesions**. The animal shows progressive wasting, intense pruritus (itching), and neurological signs (ataxia, tremors).
- **Microscopic Lesions:** The characteristic lesions are found in the CNS, particularly the brainstem (medulla oblongata). They include:
 1. **Spongiform change:** Bilaterally symmetrical vacuolation of the neuropil (grey matter).
 2. **Neuronal degeneration:** Vacuolation and degeneration of neurons.
 3. **Astrogliosis:** Proliferation of astrocytes.
 - There is **no inflammatory response**. The abnormal prion protein can be detected in the brain and lymphoid tissues (e.g., third eyelid, tonsils) by immunohistochemistry.

4.3 Inclusion body hepatitis

- **Etiology:** Fowl Adenovirus (**FAdV**) of various serotypes.
- **Pathology:** It is an acute, highly fatal disease of young chickens (especially broilers), causing severe hepatitis.
- **Gross Lesions:**
 - The **liver** is the main organ affected. It is enlarged, pale, friable, and may have petechial or ecchymotic hemorrhages.
 - The **kidneys** are often swollen, pale, and show a distinct tubular pattern.
 - **Bursa of Fabricius** and thymus may be atrophied, as some strains are immunosuppressive.
- **Microscopic Lesions:** The pathognomonic lesion is the presence of large, eosinophilic to basophilic **intracellular inclusion bodies** in hepatocytes. These inclusions fill the

nucleus, pushing the chromatin to the periphery (margination). There is also extensive multifocal to massive hepatocellular necrosis.

4.4 Kyasanur forest disease

(This is a repeat of question 4.8 from 2023).

- **Etiology:** Kyasanur Forest Disease (KFD) virus, a **Flavivirus**.
- **Transmission:** Transmitted by ticks, primarily *Haemaphysalis spinigera*.
- **Pathology:** KFD is a zoonotic **hemorrhagic fever** that primarily affects monkeys and humans.
- **Lesions (in Monkeys):** It is highly fatal, causing high fever and widespread hemorrhages, especially in the gastrointestinal tract, lungs, liver, and kidneys. Focal necrosis is also seen in the liver.
- **Lesions (in Humans):** Presents as an acute fever, myalgia, and headache, which can progress to a hemorrhagic phase (bleeding from nose, gums) and/or neurological complications (meningoencephalitis).

4.5 Black quarter

(This is a repeat of question 4.3 from 2023).

- **Etiology:** *Clostridium chauvoei*.
- **Gross Lesions:** Affects large muscles, which become dark red to **black**, dry, spongy, and **crepitant** (crackling) due to gas. The lesion has a "**rancid butter**" smell.
- **Microscopic Lesions:** Severe **necrotizing, hemorrhagic, and emphysematous myositis**. Muscle fibers are necrotic and separated by gas bubbles, with a notable **absence of inflammatory cells** (neutrophils). Large, rod-shaped bacteria are abundant.

4.6 Brooder pneumonia

- **Etiology:** This is another name for **Aspergillosis**. It is caused by the inhalation of spores from fungi, primarily **Aspergillus fumigatus**, but also *A. flavus*. It is common in young chicks ("brooders") from contaminated litter or hatchery environments.
- **Gross Lesions:**
 - Characterized by multiple, discrete, firm, yellow-to-grey **nodules** throughout the lungs.
 - The **air sacs** are also commonly affected, becoming thickened, cloudy, and sometimes covered in "beaded" plaques of fungal growth.
 - Nodules can also be found in the trachea, brain, and eyes.
- **Microscopic Lesions:** The nodules are **pyogranulomas**. They have a central core of fungal hyphae, necrotic debris, and inflammatory cells (heterophils, macrophages, and giant cells). The fungal hyphae are septate and show dichotomous (Y-shaped) branching, which can be visualized with GMS or PAS stains.

4.7 Johne's Disease [sic]

(This is a repeat of question 4.2 from 2024).

- **Etiology:** *Mycobacterium avium* ssp. *paratuberculosis* (MAP).
- **Pathogenesis:** MAP is an intracellular bacterium that is phagocytosed by macrophages

in the ileum. It survives and multiplies within them, triggering a chronic **granulomatous enteritis**.

- **Lesions:**

- **Gross:** The intestinal wall (especially the ileum) is severely thickened, with the mucosa thrown into deep, permanent, corrugated folds ("elephant skin" appearance). Associated lymph nodes are enlarged.
- **Microscopic:** The lamina propria and submucosa are massively distended by sheets of **epithelioid macrophages** and lymphocytes. Acid-fast stains (Ziehl-Neelsen) reveal large clumps of MAP organisms within the cytoplasm of these macrophages.

4.8 Caecal coccidiosis

- **Etiology:** *Eimeria tenella*. This is one of the most pathogenic *Eimeria* species in chickens.
- **Pathology:** The organism has a high affinity for the ceca. The most pathogenic stage is the **second-generation schizont**, which develops deep within the lamina propria, causing extensive damage to blood vessels and glands.
- **Gross Lesions:**
 - The **ceca** are severely distended, thickened, and filled with a mixture of **blood, necrotic debris, and fibrin**.
 - The cecal wall is dark red to purple due to intense hemorrhage.
 - In later stages, this material organizes into a hard, cheesy, laminated "**cecal core**".
- **Microscopic Lesions:** Massive destruction of the cecal mucosa with severe hemorrhage. Large schizonts and gametocytes are visible deep within the tissue, associated with sloughing of the epithelium.



2019 Exam (Q.4)

4.1 Tyzzer's disease

(This is a repeat of question 4.1 from 2022).

- **Etiology:** *Clostridium piliforme*.
- **Pathology:** An acute, fatal disease of foals, rabbits, and rodents, often stress-induced.
- **Lesions:** Characterized by the "triad" of:
 1. **Hepatitis:** Multifocal, pale necrotic foci throughout the liver.
 2. **Myocarditis:** Necrotic foci in the heart.
 3. **Enterocolitis:** Hemorrhagic and necrotic inflammation of the ileum, cecum, and colon.
- **Microscopic:** The pathognomonic lesion is "**haystack**" arrangements of long, filamentous bacilli (seen with silver stain) at the edge of necrotic liver foci.

4.2 Marek's disease

- **Etiology:** Marek's Disease Virus (MDV), a cell-associated **Herpesvirus**.
- **Pathology:** A highly contagious neoplastic disease of chickens. The virus transforms **T-lymphocytes**, causing them to proliferate and form lymphomas in various tissues.
- **Gross Lesions (Forms):**

1. **Neural Form:** Affected peripheral nerves (e.g., sciatic, brachial, vagus) are enlarged, greyish-yellow, and lose their normal cross-striations. This causes paralysis ("split leg").
2. **Visceral Form:** Diffuse or nodular **lymphomas** (tumors) in multiple organs, including the liver, spleen ("big liver disease"), kidney, ovary, and muscle.
3. **Ocular Form:** Lymphocytic infiltration of the iris causes a grey, irregular, and non-responsive pupil ("grey eye").
4. **Cutaneous Form:** Nodules in the skin and feather follicles.

4.3 Bacillary white diarrhoea [sic]

- **Etiology:** This is **Pullorum Disease**, caused by *Salmonella Pullorum*.
- **Pathology:** A bacterial disease primarily of young chicks, transmitted vertically (from hen to chick via the egg).
- **Gross Lesions:**
 - The name comes from the characteristic **white, chalky, "pasty" diarrhea** that mats the feathers around the vent.
 - **In Chicks:** Greyish-white necrotic nodules in the **lungs** (resembling brooder pneumonia), **liver**, and **heart**. The yolk sac is often unabsorbed and infected (omphalitis).
 - **In Adults:** Survivors become carriers. The main lesion is in the ovary, which contains **misshapen, discolored, shrunken, or cystic ova**.

4.4 Velogenic form of RD

- **Etiology:** This is the most severe form of Ranikhet Disease (Newcastle Disease), caused by highly pathogenic **Avian Paramyxovirus-1 (APMV-1)**.
- **Pathology:** There are two types of velogenic strains:
 1. **Viscerotropic Velogenic (VVND):** This form targets the digestive tract. It causes severe **hemorrhagic and necrotic lesions** in lymphoid-dependent tissues. Key lesions include hemorrhages in the **proventriculus** (especially at the tip), and severe necrosis and hemorrhage of the **cecal tonsils** and intestinal Peyer's patches. This results in bloody diarrhea and high, rapid mortality.
 2. **Neurotropic Velogenic (NVND):** This form targets the nervous system, causing primarily respiratory and neurological signs (torticollis, paralysis, tremors) with high mortality. Gut lesions are less common.

4.5 Exudative diathesis

- **Etiology:** A nutritional deficiency disease of young chicks caused by a deficiency of **Vitamin E** and/or **Selenium**.
- **Pathology:** The deficiency leads to a breakdown of antioxidants, resulting in increased **capillary fragility and permeability**.
- **Gross Lesions:**
 - Plasma leaks from the fragile capillaries and accumulates in the subcutaneous tissue.
 - This results in massive, "jelly-like" (**gelatinous**), **greenish-blue or reddish subcutaneous edema**.

- The edema is most prominent on the **breast, abdomen, and thighs**.
- The affected skin is often described as "weepy." In chronic cases, the exudate can become fibrinous. Birds are also anemic and may have muscular dystrophy.

4.6 Lockjaw

- **Etiology:** This is **Tetanus**. It is caused by the potent neurotoxin (**tetanospasmin**) produced by the anaerobic bacterium *Clostridium tetani*. Infection occurs when spores are introduced into deep, anaerobic puncture wounds.
- **Pathogenesis:**
 1. Toxin is produced in the wound and travels via retrograde axonal transport to the spinal cord.
 2. It acts on inhibitory interneurons (Renshaw cells), **blocking the release of inhibitory neurotransmitters** (glycine and GABA).
 3. This loss of inhibition leads to continuous, uncontrolled firing of motor neurons.
- **Lesions:** There are **no specific gross or microscopic lesions**. The animal exhibits clinical signs of **spastic paralysis**:
 - "**Lockjaw**" (trismus): Spasm of the masseter muscles.
 - "**Sawhorse stance**": Rigid extension of all four limbs.
 - Protrusion of the third eyelid.
 - Death occurs from **asphyxia** due to paralysis of the respiratory muscles.

4.7 Pulpy Kidney

- **Etiology:** This is **Enterotoxemia**, caused by *Clostridium perfringens* Type D in sheep.
- **Pathogenesis:** Usually affects lambs on a high-carbohydrate diet. The diet promotes rapid bacterial proliferation in the intestine. The bacteria produce **epsilon prototoxin**, which is activated by trypsin into the active **epsilon toxin**. This toxin is absorbed and increases the permeability of blood vessels, especially in the brain and kidneys.
- **Gross Lesions:**
 - The most characteristic finding is the **rapid post-mortem autolysis of the kidneys**, which become extremely soft, "pulpy," and hemorrhagic.
 - **Glycosuria** (sugar in the urine) is a very common finding.
 - Other lesions include hydropericardium (excess fluid in the pericardial sac), pulmonary edema, and **Focal Symmetrical Encephalomalacia (FSE)** (bilaterally symmetrical areas of necrosis in the brain).

4.8 Infectious bovine Rhinotracheitis

- **Etiology:** Bovine Herpesvirus-1 (BHV-1).
- **Pathology:** IBR is a multi-systemic disease. The most common form is respiratory.
- **Lesions:**
 - **Respiratory Form ("Red Nose"):** Causes acute inflammation of the upper respiratory tract. The nasal mucosa and trachea are intensely inflamed (hyperemic), edematous, and covered with a fibrino-purulent or diphtheritic membrane. This leads to nasal discharge, coughing, and severe respiratory distress.
 - **Genital Form (IPV/IPB):** Causes infectious pustular vulvovaginitis/balanoposthitis, characterized by pustules and ulcers on the genital mucosa.

- **Other Forms:** Can also cause abortion and encephalitis (in young calves).
- **Microscopic Lesion:** The pathognomonic lesion in all forms is **epithelial necrosis** with the presence of large, eosinophilic **intranuclear inclusion bodies**.

2018 Exam (Q.4)

4.1 Pathology of Paratuberculosis

(This is a repeat of question 4.2 from 2024 and 4.7 from 2022).

- **Etiology:** *Mycobacterium avium* ssp. *paratuberculosis* (MAP).
- **Pathology:** Known as Johne's Disease, it is a chronic, wasting disease of ruminants.
- **Gross Lesions:** The intestinal wall (especially the ileum and colon) is markedly **thickened**, and the mucosa is thrown into deep, permanent, corrugated folds that resemble the surface of the brain or "elephant skin." The associated mesenteric lymph nodes are enlarged and edematous.
- **Microscopic Lesions:** The lamina propria and submucosa are massively infiltrated by sheets of **epithelioid macrophages** and lymphocytes (diffuse granulomatous enteritis). The macrophages contain large numbers of acid-fast (Ziehl-Neelsen positive) bacteria.

4.2 Babesiosis

(This is a repeat of question 4.5 from 2023).

- **Etiology:** Tick-borne protozoa, *Babesia bovis* or *B. bigemina* in cattle.
- **Pathology:** The parasites cause massive **intravascular hemolysis** by multiplying within erythrocytes.
- **Gross Lesions:**
 - **Hemoglobinuria:** "Port wine" colored urine.
 - **Anemia:** Pale mucous membranes.
 - **Icterus (Jaundice):** Yellow discoloration of tissues.
 - **Splenomegaly:** The spleen is severely enlarged, dark, and has a "pulpy" consistency.
 - The liver is enlarged and yellow.
 - **Cerebral Babesiosis (*B. bovis*):** Sequestration of infected RBCs in brain capillaries.

4.3 Pathogenesis of Avian influenza

- **Etiology:** Avian Influenza Virus (AIV), an **Orthomyxovirus** (Type A).
- **Pathogenesis:**
 - **Low Pathogenic Avian Influenza (LPAI):** Virus replication is localized to the epithelial cells of the respiratory tract and/or the gastrointestinal tract. This causes mild respiratory signs, conjunctivitis, or decreased egg production.
 - **High Pathogenic Avian Influenza (HPAI):** HPAI strains (e.g., H5 and H7) arise from LPAI strains through mutation. The key pathogenic difference is that HPAI viruses cause a **systemic infection**. They are not restricted to the respiratory/gut tracts. They replicate in multiple organs, including vascular endothelium, hepatocytes, myocardium, and neurons. This widespread replication leads to

massive, multi-organ necrosis, vascular damage, edema, hemorrhage, and rapid death (often within 48 hours).

4.4 Pathology fowl typhoid

- **Etiology:** *Salmonella Gallinarum*.
- **Pathology:** A severe, acute or chronic systemic bacterial disease, primarily affecting adult poultry (in contrast to *S. Pullorum* in chicks).
- **Gross Lesions:**
 - The most characteristic lesion is an enlarged, friable **liver** with a distinctive "**coppery bronze**" or "**greenish-bronze**" sheen.
 - The **spleen** is also markedly enlarged (splenomegaly) and congested.
 - The bird is anemic and may be icteric.
 - The intestine may show ulceration.
 - In chronic cases, necrotic nodules ("typhoid nodules") may be seen in the liver and heart.
- **Microscopic Lesions:** The liver shows focal necrosis and a diffuse infiltration of macrophages (histiocytes), which contributes to the bronze color.

4.5 Tyzzer's disease of rabbits

(This is a repeat of question 4.1 from 2022 and 2019, but specified for rabbits).

- **Etiology:** *Clostridium piliforme*.
- **Pathology:** Rabbits are highly susceptible, especially young or stressed animals. The disease manifests as a highly fatal hepato-enteric disease.
- **Lesions:** The "classic triad" is present:
 1. **Hepatitis:** The liver is enlarged and filled with multifocal, pinpoint, pale necrotic foci.
 2. **Enterocolitis:** Severe hemorrhagic and necrotic inflammation of the cecum ("hemorrhagic typhlitis") and ileum.
 3. **Myocarditis:** Necrotic foci in the heart are also common.
- **Microscopic:** Silver stains (Warthin-Starry) reveal "**haystack**" clusters of long, filamentous bacilli at the border of necrotic zones in the liver.

4.6 Infectious ectromelia

- **Etiology:** **Mousepox** virus, an *Orthopoxvirus*.
- **Pathology:** A highly contagious viral disease of mice. It has two main forms:
 1. **Acute Form:** Rapidly fatal systemic disease. Characterized by massive **necrosis of the liver, spleen, and lymphoid tissues**. There are few or no skin lesions before death.
 2. **Chronic Form:** Occurs in more resistant mice. It is characterized by skin lesions, starting as a rash that progresses to ulceration. The hallmark is **necrosis and sloughing of the limbs, tail, and ears**, which gives the disease its name ("ectromelia" = absent limb).
- **Microscopic Lesions:** Eosinophilic **intracytoplasmic inclusion bodies** (pox inclusions) are found in affected epithelial cells.

4.7 Aspergillosis

(This is a repeat of question 4.6 from 2022, "Brooder Pneumonia").

- **Etiology:** Fungal disease caused by *Aspergillus fumigatus* or *A. flavus*, acquired by inhalation of spores.
- **Pathology:** It is a mycotic infection of the respiratory system, common in birds (brooder pneumonia).
- **Gross Lesions:**
 - **Lungs:** Multiple, firm, yellow-to-grey **nODULES** throughout the lung tissue.
 - **Air Sacs:** The air sacs become thickened, cloudy, and are often covered with velvety, greenish, or "beaded" plaques of fungal growth.
- **Microscopic Lesions:** The nodules are **pyogranulomas** containing a central core of necrotic debris, inflammatory cells (heterophils, macrophages), and branching, septate fungal hyphae (visualized with GMS or PAS stains).

4.8 Pathology of foot and mouth disease

- **Etiology:** Foot and Mouth Disease (FMD) Virus, an **Aphthovirus** (family Picornaviridae).
- **Pathology:** A highly contagious viral disease of cloven-hoofed animals. The virus is epitheliotropic (targets epithelium).
- **Gross Lesions:**
 - The characteristic lesion is the **vesicle (blister)**.
 - Vesicles are found in the oral cavity (tongue, dental pad, gums), on the skin of the **coronary band**, and in the **interdigital space**.
 - These vesicles rupture within 24-48 hours, leaving raw, painful erosions. This causes profuse,ropy salivation (drooling) and severe lameness.
 - **Myocarditis:** In young animals (calves, piglets), the virus is myocardiotropic, causing severe necrosis of the heart muscle. This appears as greyish-yellow streaks, known as "**tiger heart**" or "**tigroid heart**", and leads to sudden death.