ABORTIONS

- **Abortion** is defined as the expulsion of the fetus **before** it has reached the stage of development necessary for independent survival.
- **The expelled fetus** may be *alive* or *dead* at the time of abortion; however, it is usually dead.
- **The expulsion of a dead fetus** that has reached a developmental stage at which it could have been viable is referred to as **stillbirth**.
- If a live fetus is delivered before the expected term, this is termed **premature birth**.

Causes of Abortion:

- Non-infectious causes
- Infectious causes (approximately 95%)

NON-INFECTIOUS ABORTIONS

Their recognition is not easy, and there are also differences among animal species.

In Cattle:

- Nitrates
- Ergot (Claviceps spp.)
- Mycotoxins
- Pine needles (especially Pinus ponderosa) or lupines
- Selenium deficiency or excess
- Prolonged transportation (stress)
- Environmental temperature (heat stress)

INFECTIOUS ABORTIONS

Bacterial causes

Viral causes

Protozoal causes

Bacterial Infections of the Uterus and Fetus

BRUCELLOSIS

GENERAL CHARACTERISTICS

Zoonotic disease

Etiologic agent: *Brucella* spp.

Small, Gram-negative coccobacillus

Facultative intracellular pathogen

Tropism:

Genital organs

Placenta

B. abortus → cattle
B. melitensis → goats
B. suis → pigs
B. ovis → sheep
B. canis → dogs
Cross-species transmission (cross-infection) may occur

GENERAL PATHOGENESIS

The infection initially develops as a **systemic infection** following entry of the organism into the host.

Subsequently, the disease tends to become **chronic**, characterized by persistence of the organism within host cells.

During this chronic phase, **intermittent bacteremia** occurs, facilitating dissemination of the pathogen to target organs, particularly the reproductive system.

BRUCELLOSIS IN CATTLE

➞ *Brucella abortus*

TRANSMISSION

Main sources:

Aborted fetus

Placenta

Uterine discharge

Routes of transmission:

Alimentary route (**most important**)

Vaginal

Conjunctival

Through the skin (with or without lesions)

Sexual transmission: very rare

SUSCEPTIBILITY

Young animals → relatively resistant

Adults → susceptible

Females:

May remain lifelong carriers

ABORTION

Occurs after the **5th month of gestation**

Most commonly at **7–8 months**

Abortion rate:

Approximately **20–30%**

Involvement of the Pregnant Uterus

PATHOGENESIS AND OUTCOME

The agent spreads **hematogenously** and localizes primarily in the **endometrium, placenta, and fetus**, showing a marked *tropism for reproductive tissues*.

This tropism is associated with the presence of **erythritol** in the placenta, *which promotes the growth and multiplication of Brucella organisms*.

As a result of placental colonization and inflammation, fetal death commonly occurs, leading to **abortion or stillbirth**.

Rarely, live births may occur.

CELLULAR PATHOGENESIS

Agent Taken up by **erythrophagocytic trophoblasts**

Intracellular phase:

Replicates within the **rough endoplasmic reticulum (RER)**

Avoids lysosomal destruction and survives within host cells

Spread:

Cell-to-cell transmission
→ Dissemination to the fetus

👉 **Vasculitis:**

Occurs due to the effect of **endotoxins**

Placental Lesions

NOT PATHOGNOMONIC !

Appearance:

Yellow-gray, sticky exudate

Cotyledons:

Some appear normal

Some are necrotic

Intercotyledonary areas:

Thickened

Edematous, gelatinous

Fetal Lesions

GENERALLY:

Expelled within **20–72 hours**

Fetal fluids:

Usually normal

Occasionally dark

Umbilical cord:

Edematous

ORGAN LOCALIZATION

Females:

Spleen

Lymph nodes

Mammary gland

Males:

Testes

Accessory sex glands

Joints:

Arthritis

Bursitis

Tenosynovitis

EXCRETION

Urine/Feces:

✗ Not a significant route

Milk:

✓ Present

Especially:

High levels of shedding in **colostrum**

Important for transmission to offspring

Microscopic Findings

There is marked **mononuclear cell infiltration** within the affected tissues, particularly in the placenta.

The **chorionic epithelium** is heavily populated with the organism, with numerous bacteria present within trophoblastic cells.

The **chorionic villi** show lesions consistent with **placentitis**, including inflammatory infiltration and structural disruption.

In various organs, especially in the fetus, **granulomatous lesions** may develop, often accompanied by **multinucleated giant cells**, reflecting a chronic inflammatory response.

Trophoblasts are shrunken and hypereosinophilic (necrosis) or contain many intracytoplasmic basophilic coccobacilli.

CONTROL AND PREVENTION

- **Test and slaughter programs**
- **Vaccination** (e.g., *B. abortus* S19, RB51)
- **Strict biosecurity measures**
- Isolation of infected animals
- Proper disposal of aborted materials (fetus, placenta)
- Disinfection of contaminated areas

Milk hygiene:

- Pasteurization is essential

Zoonotic risk:

- Use of personal protective equipment (PPE)
- Avoid consumption of raw milk and dairy products

BRUCELLOSIS IN SHEEP AND GOATS

Brucella ovis

First described in **1950**

In rams:

- Causes **epididymitis**
- Transmitted via semen → **venereal route**

Brucella melitensis

Most important species !

Highest zoonotic potential

Endemic in:

- Mediterranean region
- Middle East
- South America

PLACENTA

Similar to lesions observed in cattle

Cotyledons:

- Necrosis
- Exudate

FETUS

Nonspecific edema

Body cavities:

Fluid accumulation (**ascites, hydrothorax**)

Notable finding:

Calcified plaques on the hooves
(significance remains unclear)

autolysis may obscure lesions

MICROSCOPIC FINDINGS

(Consistent with necrotizing placentitis)

Chorionic epithelium:

Contains abundant bacteria

Chorionic mesenchyme:

Bacteria are present free within the tissue

Blood vessels:

Vasculitis

FETAL ORGAN LESIONS

Pneumonia

Lymphadenitis

Interstitial nephritis

Liver:

Portal hypercellularity

AGE-RELATED DIFFERENCES

Young fetus:

Monocyte–macrophage hyperplasia

Early stage of **granulomatous inflammation**

Older fetus:

Well-organized lesions composed of:

- Macrophages
- Lymphocytes
- Plasma cells

→ Formation of **nodular lesions**

CLINICAL COURSE

Abortion occurs in **late gestation**

During the disease:

A **septicemic phase** is present

During this phase, the agent localizes in the **pregnant uterus**

SPECIAL FEATURES IN GOATS

More severe clinical course

Death may occur during early bacteremia

Frequently associated with **severe mastitis**

MAMMARY FINDINGS

May be the **initial clinical sign !**

Milk:

Watery

Clotted

The agent may be shed in milk for **weeks, months, or even years**

Important source of zoonotic transmission

BRUCELLOSIS IN DOGS

➞ *Brucella canis*

First described in **1966 (USA)**

Transmission:

Venereal (important)

Alimentary

Characterized by **persistent bacteremia**

CLINICAL COURSE

Abortion typically around **~50 days of gestation**

Fetus may be **dead or alive**

Live-born puppies usually **die shortly after birth**

PATHOLOGY

Placenta:

Chorionic epithelial cells contain **abundant bacteria**

Fetal lesions:

Bronchopneumonia

Myocarditis / Endocarditis

Placentitis

Renal hemorrhage

Subacute inflammation in **pelvic connective tissue**

Lymphadenitis (especially head lymph nodes)

Hepatitis

BRUCELLOSIS IN PIGS

Agent:
➞ *Brucella suis*

General features !

Produces a disease pattern **different from ruminants**

Lesions are more **widespread (systemic)**

Marked tendency for **abscess formation** (especially in pigs)

LESION CHARACTERISTICS

Granulomatous inflammation

Necrosis + caseification

UTERINE FINDINGS

Endometrium:

Miliary granulomas

Hyperplastic lymphoid nodules

Endometrial glands:

Dilated with **mucus and leukocytes**

Epithelium:

Desquamated / flattened

PREGNANT UTERUS

Mucopurulent exudate between placental areas

Contains **free and/or intracellular bacteria**

PLACENTA

Chorioallantoic membrane:

Edema

Congestion

Brucellosis: Causative Agents and Main Differences Among Animal Species

Brucella Species	Primary Host(s)	Main Transmission Route	Key Clinical Findings	Zoonotic Risk
Brucella abortus	Cattle	Ingestion of contaminated materials (placenta, fetal fluids), milk	Late-term abortion, retained placenta, metritis, decreased fertility	Moderate
Brucella melitensis	Sheep, Goats	Direct contact, ingestion, milk	Abortion (last trimester), weak offspring, mastitis	**High (most pathogenic for humans)**
Brucella suis	Pigs	Venereal, ingestion	Abortion, orchitis, infertility, lameness	High
Brucella canis	Dogs	Venereal, contact with aborted materials	Abortion (45–55 days), epididymitis, prostatitis	Low–Moderate
Brucella ovis	Sheep (rams)	Venereal	Epididymitis, reduced fertility (rare abortion)	Negligible

CAMPYLOBACTERIOSIS

Gram-negative, **spiral/comma-shaped** bacteria

Motile (**polar flagella**)

Microaerophilic

Subspecies

C. fetus subsp. venerealis

Cattle → genital tract pathogen

C. fetus subsp. intestinalis

Sheep → intestinal origin

TRANSMISSION (Venereal)

- Spread from **vagina** → **uterus**
- Localization in the **endometrium**

Outcome:

- **Early embryonic death**
- Repeated estrous cycles → **repeat breeding**

Persistence:

- Not persistent in the **uterus**
- May persist in the **vagina**

Reservoir:

- **Bulls act as asymptomatic carriers**

ENDOMETRIAL LESIONS

Mild to moderate

Lymphocytic endometritis

Glands:

Cystic dilation

PLACENTA

Edematous

Opaque

Leathery appearance

Cells:

Predominantly **macrophage infiltration**

COTYLEDONS

Yellow, soft

Peripheral necrotic villi

FETUS

Usually **autolyzed**

Lesions:

Purulent bronchopneumonia

Focal necrotic hepatitis

Fibrin on serosal surfaces

Stomach content:

Yellow, sticky, turbid

https://www.scielo.org.mx/scielo.php?script=sci_arttext&pid=S2007-11242019000401054

PATHOGENESIS IN SHEEP

The infection is acquired via the **oral route**, after which the organism colonizes the **intestine** and enters the bloodstream, leading to **bacteremia**.

Subsequently, the agent spreads **hematogenously** and localizes in the **pregnant uterus**, particularly in the placenta, resulting in placental infection and fetal involvement.

LIVER LESIONS

Size: **1–2 cm**

Brown center

Pale surrounding zone

Non-reactive focus

DIFFERENTIAL DIAGNOSIS

Should be differentiated from **necrobacillary lesions**

MICROSCOPIC FINDINGS

Necrosis

Histiocyte + neutrophil infiltration

Vasculitis

Placentitis

LEPTOSPIRAL AGENTS

Agent:

Spirochete

Aerobic

Thin, spiral-shaped organism

PATHOGENESIS

The organism enters the host via the **oral route or mucous membranes**, after which it disseminates through the bloodstream, leading to **septicemia**.

Subsequently, it localizes in the **kidneys**, where it establishes infection and results in **persistent carrier status**.

PREGNANCY

Abortion occurs **weeks after septicemia**

Typically in the **last trimester**

PLACENTA

Edematous

Organisms present in the **chorionic epithelium**

FETUS

Lesions are **mild**

May be **confused with autolysis**

KIDNEY ★

Interstitial nephritis

Mononuclear cell infiltration

Diagnosis:

Silver staining → *Leptospira* in **tubular lumen**

ADDITIONAL LESIONS

Ascites

Fibrinous peritonitis

Necrotic hepatitis

LISTERIOSIS

Agent:

L. monocytogenes

Gram-positive, **facultative intracellular**

Small rod-shaped bacterium

Transmission:

Silage

PATHOGENESIS

Following ingestion, the organism penetrates the **intestinal mucosa** and enters the bloodstream, leading to systemic dissemination.

From here, it follows two main pathways:

Spread to the **brain**, resulting in **encephalitis**

Localization in the **uterus**, leading to **abortion**

ABORTION

Typically occurs in the **last trimester**

GROSS FINDINGS

Liver & spleen:

Pinpoint (**miliary**) necrotic foci

Lung, kidney, brain:

Similar lesions

MICROSCOPIC FINDINGS

Necrosis + neutrophilic infiltration

Microabscess formation

Gram-positive bacteria easily visible

https://www.flockandherd.net.au/sheep/ireader/listeria-abortion.html

DIFFERENTIAL DIAGNOSIS

Should be differentiated from
Brucellosis and
Campylobacteriosis

CHLAMYDIAL INFECTIONS

Agent:

Chlamydia spp.

Especially:

- *C. psittaci* group
- *C. abortus*

MORPHOLOGY AND INTRACELLULAR LIFE

Small, **obligate intracellular bacteria**

Light microscopy:

Located within **cytoplasmic vacuoles** as **inclusion bodies**

These inclusions are **basophilic** and **granular**

➞ These **intracytoplasmic inclusion bodies** represent clusters of **replicating organisms** and constitute **the most critical diagnostic feature** in routine histopathological examination.

LIFE CYCLE

Elementary body (infectious form) attaches to the host cell and enters via **endocytosis**

Inside the cell, it transforms into the **reticulate body (replicative form)**

The reticulate bodies multiply within **membrane-bound vacuoles (inclusion bodies)**

After replication, they reorganize back into **elementary bodies**

Cell lysis → **release and spread** to adjacent cells

➞ As a result, a **high intracellular bacterial load** accumulates within infected cells

EPIDEMIOLOGY

In **sheep & goats**:

Major cause of abortion

In **cattle**:

Sporadic abortions

CLINICAL FINDINGS

➞ **Enzootic abortion of ewes (EAE)** *(classic term)*

Late-term abortion

Stillbirth

Weak offspring

Retention of fetal membranes

ZOONOTIC RISK !

High risk for **pregnant women**

May cause **severe systemic disease and abortion in humans**

TRANSMISSION

Oral route (most important)

Exposure to contaminated:

- Aborted materials
- Exudates
- Feces

✕ **Venereal transmission:**

Not present / not well established

IMMUNITY

Develops after infection

However:

- **Not fully protective**

PATHOGENESIS

Oral intake

Entry into the **intestinal epithelium**

Spread via **lymphoid tissues → bloodstream**

In pregnant animals:

Marked tropism for the **placenta**

PLACENTAL TROPISM

Agent:

Replicates within **trophoblast cells**

Outcome:

This leads to **cell destruction, necrosis, and inflammation**, resulting in placental damage and subsequent fetal compromise.

WHY DOES ABORTION OCCUR LATE?

After **placental development is complete**, the organism rapidly multiplies in placental tissues

➞ Abortion typically occurs in the **last one-third of gestation**

IMPORTANT CLINICAL POINT

Abortions are more common in **previously unexposed (naïve) animals**

Macroscopic findings

COTYLEDONS

Dull

Dark red

Necrotic

Covered with **dirty, blood-tinged exudate**

CHORIOALLANTOIS

Thickened

Edematous

Leathery appearance 

FETUS

•**Subcutaneous hemorrhage**

Thymus & lymph nodes:

•Hemorrhage

Liver:

•**1–2 cm necrotic foci**

MICROSCOPIC FINDINGS

Placenta

Chorionic epithelial cells:

Contain **inclusion bodies**

Chorionic membrane:

Severe vasculitis

Predominantly **mononuclear cell infiltration**

- Lymphocytes + macrophages

For differential diagnosis:

Giemsa → positive

Ziehl–Neelsen → may be visible

Most important finding:

➞ Inclusion bodies

FETAL ORGANS

Liver & spleen:

Necrosis

Mild mononuclear infiltration

Lung:

Mononuclear infiltration

Septal thickening

Brain:

Meningoencephalitis

Vasculitis

Hemorrhage

Lymphoid organs:

Lymphoreticular hyperplasia

COMPARISON OF ABORTIFACIENT AGENTS

Feature	Chlamydia	Brucella	Campylobacter
Intracellular	✓	✓	✗
Inclusion bodies	✓✓	✗	✗
Placenta	Thick, leathery	Necrotic cotyledons	Mild lesions
Vasculitis	✓	✓	±
Time of abortion	Late	Late	Mid-gestation

HAEMOPHILUS (*Histophilus somni*) INFECTIONS

Histophilus somni (formerly *Haemophilus somnus*)

Gram-negative, small **coccobacillus**

Facultative pathogen

Natural localization:

Normally found in:

- Vagina
- Prepuce
- Respiratory tract

➞ Acts as an **opportunistic pathogen**

Disease activation:

Triggered by **stress factors** (transport, crowding, environmental changes)

Often associated with **co-infections** (respiratory pathogens)

IN CATTLE

Associated with **infertility, abortion, endometritis, and vaginitis**

ABORTION

Typically **spontaneous**

Usually occurs during the **mid to late stages of gestation**

FETUS

Most fetuses are **autolyzed**,
which may **complicate diagnosis**

PATHOGENESIS

- The organism initially establishes **vaginal colonization**, after which it ascends to the **uterus**.
- This is followed by **bacteremia**, allowing systemic dissemination.
- Subsequently, the agent localizes in the **placenta**, where it establishes infection and contributes to fetal involvement.

MAIN PATHOGENETIC MECHANISM

VASCULITIS

The bacteria localize in the **vascular endothelium**, leading to endothelial damage.

As a result, **vascular injury**, **thrombosis**, and **ischemia** develop.

This process progresses from **vasculitis** to **fibrinoid necrosis**, resulting in **placental insufficiency**.

Ultimately, this leads to **fetal hypoxia and abortion**.

PLACENTAL LESIONS

Nonsuppurative placentitis

Cotyledons:

Mononuclear cell infiltration

Around trophoblasts:

- **Bacterial colonies**

Vessels:

In small and large arteries:

- **Fibrinoid necrosis**
- **Vasculitis**

FETAL LESIONS

Fibrinous bronchopneumonia

Lung:

Consolidation

Fibrin accumulation

Pleura:

Fibrinous exudate

SALMONELLOSIS

Agent

Family: **Enterobacteriaceae**

Gram-negative, rod-shaped

Motile (flagellated)

Non-spore forming

Host Specificity

Human → *S. typhi*

Pig → *S. choleraesuis*

Broad Host Range

S. typhimurium 

- cattle
- horse
- pig
- human

Abort Agents

Sheep → *S. abortus ovis*

Horse → *S. abortus equi*

Sheep → *S. montevideo*

EPIDEMIOLOGY

Type of Disease

→ **Paratyphoid infection**

Risk Factors (Stress-related)

Transport

Feed changes

Spoiled feed

Pathogenetic Mechanism

→ **Disruption of intestinal microbiota**

Pathogenesis

Oral ingestion

Invasion of intestinal mucosa

Peyer's patches → regional lymph nodes

Bacteremia / septicemia

→ Enteritis / septicemia
→ Bacteremia
→ Spread to the uterus
→ Placental infection
→ **Fetal death → abortion**

Mechanism of Abortion

In salmonellosis, abortion is **not a primary uterine disease**

It occurs as a **secondary consequence of septicemia**

Abortion

Usually occurs in **late gestation**

Newborns (if born alive) → **die within a few days**

Macroscopical Findings

Placenta

Lesions are **not specific**

Mild changes:

- edema

Occasionally:

- focal hemorrhages
- small necrotic foci

Fetus

Typically:

- edematous

No prominent or specific lesions

Ureaplasma spp. Infection

Agent

Ureaplasma spp.

Closely related to the **Mycoplasma group**

Lacks a cell wall 

Small, pleomorphic organisms

Difficult to culture

Normal Habitats

Nasal cavity

Vulva

Vagina

Prepuce

Semen

Embryo transfer fluids

→ **Commensal** → **opportunistic pathogen**

Transmission

•Venereal

•Embryo transfer

•Direct contact

Clinical Findings

•Vulvitis

•Endometritis

•Embryonic death

•Abortion (last trimester)

•Retention of fetal membranes

•Stillbirth

•Weak offspring

Pathogenesis

•Genital colonization

•Ascension to the uterus

•Colonization of placental membranes

Tropism

Especially for:

→ **Amniotic epithelium**

Mechanism of Injury

•Direct cellular damage

•Inflammation

•Exudation

Amnion

- **Most affected structure**
- Necrosis
- **Fibrinous exudate**

Chorioallantois

- Edema
- Inflammation

Fetus

- Generally:
  - weak
- No specific or characteristic lesions

Lungs

- Inflammation may be present

(due to aspiration + intrauterine exposure)

Additional Findings

- Erosive conjunctivitis
- Nonsuppurative inflammation

"Amnion is the primary target lesion in Ureaplasma infections"

RICKETTSIAL ABORTION (Sheep & Goats)

Agent

Coxiella burnetii

Rickettsia-like organism

Obligate intracellular

Key features

Highly resistant in the environment

Easily transmitted via aerosol

Zoonotic → Q fever (humans)

Epidemiology

Hosts: cattle, sheep, goats

Mostly: **subclinical infection**

Animals act as **reservoirs**

Transmission

Animal → animal

- Aerosol (**most important**)
- Milk (oral)
- Ticks (secondary role)

Animal → human

- **Inhalation (primary route)**

Pathogenesis

Entry: oral / inhalation

Replication in macrophages

Bacteremia → placental tropism

Target cells: **trophoblasts**

➞ Abortion:

Usually after **primary infection**

Last trimester

Gross & Microscopic Lesions

Chorioallantois

Thickened

Leathery appearance 

Covered with exudate

Cotyledons

May be necrotic

Covered with exudate

Placenta

Mononuclear cell infiltration

Numerous bacteria within trophoblasts

Fetus

Usually **no specific lesions**

May be weak at birth

☞ **NO vasculitis**

(main difference from Chlamydia)

Viral Infections of the Uterus and Fetus

HERPESVIRUS INFECTIONS

In domestic animals, many herpesviruses cause **infertility, abortion, stillbirth, and neonatal infections**.

These infections are commonly observed in **cattle, horses, and pigs**.

Latent infection

After primary infection, the virus **remains in the host for life**.

Under stress conditions, it can **reactivate and cause disease again**.

Host specificity

Most herpesviruses are **host-specific**, meaning they infect only particular animal species.

GENERAL FETAL LESIONS (Herpesvirus)

Gross findings

In parenchymal organs, there are **multiple small necrotic foci**.

These lesions are especially prominent in the **liver as pinpoint white spots** ★

Serous cavities may contain fluid (**transudate**).

Subcutaneous edema is commonly observed.

MICROSCOPIC FINDINGS (Herpesvirus)

The most characteristic feature is **intranuclear inclusion bodies**

Around necrotic areas, **eosinophilic inclusions** are commonly observed.

Vascular lesions

Mild **endothelial swelling** may be present.

However, **there is no prominent vasculitis**, which helps differentiate viral infections from bacterial causes.

EQUINE HERPESVIRUS (EHV-1, EHV-4)

EHV-1 causes **respiratory disease, abortion, and neurologic signs**.

EHV-4 is mainly associated with **late-term abortion (9–10 months)**.

PATHOGENESIS & CLINICAL FEATURES

The virus infects **maternal endothelial cells**.

This leads to **thrombosis, vascular occlusion, and endometrial necrosis**.

As a result, **placental separation occurs**, leading to fetal death.

Clinical feature (very important)

Abortion is **sudden**, and the mare usually appears **clinically normal**.

The fetus is typically **fresh (no autolysis)**

GROSS LESIONS

Lungs: heavy, edematous, and do not collapse

Liver: numerous necrotic foci

Spleen: enlarged

Generalized: petechiae and ecchymoses

BOVINE HERPESVIRUS (BHV-1)

Clinical

Causes **IBR (respiratory disease)** and **abortion**

Pathogenesis

Virus spreads from **respiratory tract** → **bloodstream** → **uterus**

Targets **trophoblasts and placental vessels**

Leads to **placental damage and fetal death**

Abortion

Can affect up to **25% of the herd**

Occurs at **5–8 months of gestation**

Fetus is usually autolyzed (unlike EHV)

Microscopic Findings

Multifocal necrosis in many organs

Especially **liver and adrenal glands**

Intranuclear inclusion bodies

Placenta

Necrotizing vasculitis in small vessels

CANINE HERPESVIRUS (CHV-1)

Clinical

Causes:

- **Neonatal death**
- **Abortion**
- **Premature birth**

High mortality in neonates (first 10 days of life)

Gross Findings

Pleural & peritoneal cavities: hemorrhagic fluid

Widespread petechiae

Liver & kidneys: multifocal necrosis

Microscopic Findings

Necrosis + intranuclear inclusion bodies

PARVOVIRUS INFECTIONS

General Features

Small DNA viruses

Target rapidly dividing cells **(mitotic cells)**

Pathogenesis

Infects **highly proliferative tissues**

Outcome depends on fetal age

Early infection → fetal death

Late infection → malformations

FELINE PANLEUKOPENIA VIRUS (FPV)

Most characteristic lesion

Cerebellar hypoplasia

Pathogenesis

Virus infects:

Dividing cells in external granular layer of cerebellum

These cells proliferate:

Late gestation + neonatal period

➞ Therefore:

Lesions can develop even after birth

Bunyaviruses (Akabane, Cache Valley)

•Target: **immature neurons**

→ neuron loss → denervation → malformation

☞ Key lesions:

•**Arthrogryposis**

•**Hydranencephaly**

•Muscle atrophy

Flaviviruses / Bluetongue

•Placental infection → **vascular damage**

→ **fetal hypoxia/anoxia** → **abortion**

•Lesions vary with **fetal age & immune status**

Equine Viral Arteritis (EVA)

•Transmission: respiratory + venereal

•Stallions = **carriers (key epidemiology)**

•Mechanism:

- **NOT direct fetal infection**
- Uterine vascular damage → **placental insufficiency** → **fetal anoxia**

Pestiviruses

Cattle (BVD)

•Cerebellar hypoplasia

•Ocular defects

Sheep (Border disease)

•CNS hypomyelination

•Hair/wool defects

Pig (Classical swine fever)

•Congenital tremor

PROTOZOAL INFECTIONS

TOXOPLASMA GONDII (Sheep & Goats)

General

Important **cause of abortion** in sheep and goats 

Source: **cat feces (oocysts)**

Transmission

Contaminated feed / water

Cat ↔ sheep cycle

Clinical

Dams are usually **asymptomatic**

Abortion

Occurs in **late gestation**

Often associated with:

- **recent infection**
- **spread within the flock** ★

Gross Findings

Fetus: usually normal

Placenta:

- Cotyledons → **1–2 mm white foci (very characteristic)**
- Chorioallantois → **edema**

Microscopic Findings

Cotyledons:

- **Necrosis**
- Trophoblast epithelium:
  - **hyperplasia + hypertrophy**

Fetus:

- Brain → **leukomalacia (secondary to hypoxia)**

NEOSPORA CANINUM (Cattle)

General

One of the **most important causes of abortion in cattle**

Abortion

Occurs at **3–9 months of gestation**

Gross Findings

Usually **no significant lesions** ★

Microscopic Findings

Brain (most important) ★

- **Multifocal necrosis**
- **Gliosis**

Lesions are:

- **perivascular (near capillaries)**

Parasite found in:

- **neurons**
- **endothelial cells**

Heart

- Subacute multifocal myocarditis

Liver

- Portal hepatitis
- Hepatocellular necrosis
- Fibrin thrombi

TRICHOMONAS FOETUS INFECTION (Cattle)

General

Infects both **pregnant and non-pregnant uterus**

Transmission via **coitus** from infected bulls

Reservoir (Bull)

Organism persists in:

- **glans penis**
- **preputial mucosa**

Initially:

- **purulent balanoposthitis**

Later:

- inflammation subsides but **carrier state persists**

Pathogenesis (Female)

After mating:

- **acute vaginitis** develops within a few days
  - vulvar swelling
  - vaginal discharge

Vaginal infection:

- **transient (self-limiting)**

Then:

- organism **ascends to cervix and uterus**
- causes:
  - **mucopurulent cervicitis**
  - **endometritis**

<u>Flagellated organism present in:</u>

- **uterine contents**
- **vaginal discharge**

Discharge:

- seen during **estrus**
- located in **vagina only (not vulva)**

SARCOCYSTIS INFECTIONS

General

Affects:

- **cattle, sheep, goats, pigs**

Clinical Importance

Causes:

- **abortion**
- **stillbirth**
- **neonatal death**

MYCOTIC ABORTIONS

General

Usually occur as:

- **sporadic cases (not outbreaks)**

Main Etiologic Agents

***Aspergillus spp*.** (most common in cattle & horses)

Others:

- **Absidia**
- **Mucor**
- **Rhizopus**

Inflammation of Cervix, Vagina and Vulva

INFECTIOUS PUSTULAR VULVOVAGINITIS (IPV)

Agent

Bovine herpesvirus-1 (BHV-1)

Clinical

Fever

Vulvar swelling & hyperemia

Sticky exudate

Lesions

Vestibule:

small white papules (~0.5 mm)

EQUINE COITAL EXANTHEMA

Equine herpesvirus-3 (EHV-3)

Key Feature

No systemic signs

Lesions

1–2 mm papules

Located in:

- **vagina & vestibule**

EPIVAG (Cervicovaginitis + Epididymitis)

Agent

Herpesvirus (uncertain type)

Clinical

Purulent discharge

Chronic course

Outcome

Up to **25% infertility** ★

Male

Epididymitis

Testicular lesions

OVINE POSTHITIS / VULVITIS

Cause

High protein diet

Corynebacterium renale

Pathogenesis

Urea → **ammonia formation**→ **chemical irritation** ★

Lesions

Redness

Ulceration

Clitoral enlargement

DOURINE (Trypanosoma equiperdum)

Trypanosoma equiperdum

Protozoon (trypanosome)

Only venereally transmitted trypanosome

Hosts

Horses, donkeys, mules

ransmission

Venereal (coitus)

Stallion ↔ mare

Pathogenesis

Entry via **genital mucosa**

Local multiplication → **blood/lymph spread**

Tropism for:

- **Genital tract**
- **Skin**
- **Nervous system**

Clinical Signs

Genital stage

- Edema of genitalia
- Mucopurulent discharge

Cutaneous stage

- **"Silver dollar plaques"** (urticarial skin lesions)

Neurologic stage

- Progressive weakness
- Paralysis

Lesions

- **Genital edema & inflammation**
- **Skin plaques**
- **Peripheral neuritis**

Outcome

- Chronic disease
- Progressive → **death if untreated**

SUMMARY

- **Only sexually transmitted trypanosome**
- **No insect vector !**
- **Skin plaques (silver dollar)**
- **Neuro signs in late stage**

TUMORS

Leiomyoma (Dog)

⭐ **Estrogen-dependent**

Origin: **smooth muscle (uterus/vagina)**

Polypoid mass

Usually **benign**

TVT (Transmissible Venereal Tumor)

⭐ **Sticker tumor**

Transmission: **coitus → cell transfer**

Not a true metastasis → **allograft tumor**

Fibropapilloma (Cattle)

Cause: **Papillomavirus**

Wart-like lesions

⭐ **Spontaneous regression common**

Squamous Cell Carcinoma (SCC)

⭐ **Sunlight-related (UV exposure)**

Occurs in **non-pigmented (light-colored) areas**

Common sites:

- Vulva
- Perineum

Malignant

Can be **locally invasive ± metastasis**

Malignant Melanoma (Horse)

⭐ Common in **grey mares**

Typical locations:

- **Perianal region**
- **Vulva**

Often multiple nodules

⭐ **High metastatic potential**