viral infectious diseases

Foot and mouth disease (FMD)

Synonyms :(apthous fever ;eczema contagious)

Definition :FMD disease is an acute febrile highly contagious disease of cloven footed animals . it is characterized by vesicular eruption in the epithelium of buccal cavity ,tongue ,muzzle ,feet ,teats, and udder .in calves there is focal degeneration of cardiac and skeletal muscles which often lead to death .

Etiology :

1-FMD virus belong to the family (picornaviridae)small RNA virus member of genus (apthovirus)

2- 7 serological types: ("O" "A" "C" Asia-1 and sat-1,sat-2 and sat -3)

3- The virus resistant to various external agent including common disinfectants ,the virus most stable in ph ranging from 7.4 to 7.6 .the virus can be activated after boiling over 15 minutes at 70 C^{o} .the virus is resistant to alcohol, ether and chloroform .

Mode of transmission :

1-Rapid spread by direct contact with infected animals .

2-Virus may gain entrance in a herd from infected materials ,food stuff ,feeding ,utensilsاواني water ect.

3-Spread through airway

4-In tropical area spread through the ingestion .

5-The infection impose a high spread during the **cooler season** .

6-All fomites like clothes ,harnessعدة الفرس ,mangers معلف ,bedding الاغطية ,straw ,hay….ect.

7-Free living birds may carrying the infection.

8-The human movement and entry of visitors in the farm .

9-Carrier may transfer the virus from animal to animal .

❖ **clinical Signs in Cattle:**

The disease is not fetal ,morbidity is 100% but mortality is less .and The incubation period (2-8) days.

1- Fever, pyrexia, hyperthermia.

2-slobbering and smacking lips. سيلان العاب ولحس الشفاه

3-shivering. يرتجف

4-tender and sore feet. تقرح القدم وانسلاخها

5-reduced milk yield and Mammary gland swelling, mass, hypertrophy udder,

6-sores and blisters on feet. تقرحات وفراقيس بالقدم

7-raised temperature.

8-sudden, severe lameness

9-lies down frequently and is very unwilling to rise. الاضطجاع وعدم الرغبه بالوقوف

10-blisters may be found on the hoof ,dental pad and sometimes the tongue.

11- oral and Tongue ulcers, vesicles, erosions, sores, blisters, cuts, tears.

12- Arrhythmia, irregular heart rate, pulse.

13- Abortion or weak newborns, stillbirth.

❖ **Pathogensis:-**

The primary site of infection and replication of FMD is in the mucosa of the pharynx. The virus may also enter through skin lesions or the GI tract. Once distributed throughout the lymphatic system, the virus replicates

in the epithelium of the mouth, muzzle, teats, feet, and areas of damaged skin (eg, knees and hocks of pigs). Vesicles then develop at the organs and rupture within 48 hr. More than 50% of ruminants that recover from illness and those that are vaccinated and have been exposed to virus can carry virus particles in the pharyngeal region–up to 3.5 yr in cattle, 9 mo in sheep, and >5 yr in African buffalo

❖ Lesion :

1-Microscopically lesion will show intracellular oedema and necrosis of stratum spinosum layer of the epithelial cell .vesicular lesion become apparent when sufficient amount of fluid accumulate inside .

2- Vesicles or blisters on the tongue, dental pad, gums, cheek, hard and soft palate, lips, nostrils, muzzle, coronary bands, teats, udder, snout of pigs, corium of dewclaws and interdigital spaces

3-Heart muscle show degenerative change .there is gray or yellow foci or stick in the myocardium which is defined" Tiger heart" .change may also noted in the pancrease and large intestine .there is typical eroded in the rumen pillar.

❖ Differential diagnosis:-

1-Vesicular stomatitis. 2-Swine vesicular disease . 3-Vesicular exanthema of swine. 4-Rinderpest. 5- Bovine viral diarrhoea and Mucosal disease.6-Infectious bovine rhinotracheitis. 7-Bluetongue 8- Epizootic haemorrhagic disease .9- Bovine mammillitis. 10- Bovine papular stomatitis (Contagious ecthyma). 11-Malignant catarrhal fever.

❖ Diagniosis :

Based on history ,clinical signs and laboratory diagnosis.

Lab exam. –

1-animal inoculation
2-complement fixation test (C.F.T.)
3-neutrilazation test (N.T.)
4-Agar gel diffusion precipitation test (A.G.P.T.)
5-flurescent antibody test (F.A.T.)
6-Tissue Culture Test(T.C.T)
7-ELISA.

Vesicular stomatitis

Synonyms: sore mouth , pseudo foot and mouth disease .

Definition:-It is an infectious viral disease of cattle ,horse , pigs ,characterized by vesicular lesions containing serous fluids in the mouth , foot , interdigital space ,udder and teats .

Etiology :

1-It is a RNA virus belong to **rhabdovirdiae** virus group . there are three serotype **vesiculo** virus named as ***Indiana*** strain ***,new jersey*** strain ,and ***Eocal*** (Trinidad) strain .

2-Temperature: Inactivated by 58°C for 30 minutes pH: Stable between pH 4.0 and 10.0.

3-Chemicals/Disinfectants: Sensitive to formaldehyde, ether and other organic solvents; chlorine dioxide, formalin (1%), 1% sodium hypochlorite, 70% ethanol, 2% glutaraldehyde, 2% sodium carbonate, 4% sodium hydroxide, and 2% iodophore disinfectants, all effective disinfectants.

4-Survival: Inactivated by sunlight; survives for long periods at low temperatures.

Susceptible hosts:vesicular stomatitis is found in all breeds and sexes of cattle ,horse ,pigs and donkeys. Sheep is resistant .outbreak is most common in cattle than pigs . amongst cattle ,calf under one year of age is generally resistant than adult .

Mode of transmission :-

1-Mechanism of transmission of VSV is unclear.
2-Contamination by transcutaneous or transmucosal route .
3-Arthropod transmission: sand flies, mosquitoes (Aedes spp.)and black flies
4-Experimental transmission: of VS has been demonstrated to occur from black flies (Simulium vittatum) to domestic swine and cattle.

pathogenesis

1-pathogenesis is very much similar to foot and mouth disease .

2-the virus through abrasion in oral pedal or teat skin the stratum spinosum and then invade the epithelium cell and multiply rapidaly . as a result of multiplication many cell are injured and the virus particles are released .

3-the virus then attach the cell of deeper layers . as a result of viral invasion there is intracellular edema which result in rupture of many intracellular bridges , spatial separation of cell and the formation of maculae . in about 30% of cattle ,cell turn necrosed and confluence with adjacent intracellular spaces producing fluids spaces or vesicles which rupture discharging virus to the surface and heal .

4-the course is short and healing is rapid in uncomplicated cases . secondary bacterial invation may produced deep ulcer and may retard the healing process .during the early stage , the virus may enter blood and produce viraemia which is followed by location of vesicular lesion in oral , pedal or teat skin . the vesicles are 2-3 cm in diameter .

Clinical Signs:-

1-Incubation period 3 to 5 days

2-Fever and vesicles that resemble FMD

3-Horses severely affected

1-Oral lesions:- Drooling, chomping, mouth rubbing, lameness

2-Coronary band lesions

4-Cattle, pigs

1-Vesicular lesions:-Oral, mammary gland, coronary band, interdigital region

2-Usually isolated to one body area

3-Salivation, lameness

5-Recover within 2 weeks.

Differential diagnosis:-

1- oral lesions in cattle include

FMD, rinderpest, infectious bovine rhinopneumonitis (IBR), bovine virus diarrhea (BVD), malignant catarrhal fever (MCF), bluetongue and chemical or thermal burns.

2-**In pigs, differentials include** FMD, swine vesicular disease, vesicular exanthema in swine, foot rot, chemical and thermal burns.

3-**For sheep**, be suspicious of FMD, bluetongue, contagious ecthyma, lip and leg ulceration, foot rot, and chemical or thermal burns.

Laboratory Diagnosis:-

1-Virus isolation

2-Viral antigen detection

1-Vesicular fluid or epithelium
2-ELISA, complement fixation,
3-virus neutralization

3-Antibody tests

1-Paired serum samples
2-ELISA, complement fixation,
3-virus neutralization

Rinder pest

Synonyms: cattle plague

Rinderpest: is a contagious viral disease affecting cloven hoofed animals (mainly cattle and buffalo). In 2011, rinder pest became the second disease to be declared officially eradicated, following eradication of the human disease smallpox in 1980.

ETIOLOGY

1-Rinderpest is associated with a morbillivirus (family Paramyxoviridae) and there are three distinct lineages (strains) with considerable variation in virulence between them but all are immunologically identical, lineages 1 and 2 being of African origin and lineage 3 of Asian origin.

2- **Temperature**: Small amounts of virus resist 56°C/60 minutes or 60°C/30 minutes.

pH: Stable between pH 4.0 and 10.0.

Disinfectants/chemicals: Susceptible to lipid solvents and most common disinfectants (phenol, cresol, β- propiolactone, sodium hydroxide 2%/24 hours used at a rate of 1 litre/m2).

Survival: Quickly inactivated in environment as RPV is sensitive to light, drying and ultraviolet radiation. Can remain viable for long periods in chilled or frozen tissues.

Transmission :-

1- By direct or close indirect contact between infected and susceptible animals .
2-Airborne transmission is limited and only possible under specific circumstances.
3- RPV is sensitive to direct sunlight thus fomites are not a viable means of transmission.
4- No evidence of vertical transmission.
5- Introduction of RPV into free areas is most commonly by means of infected animals.

Clinical signs:-

1-After an incubation period of 3–15 days.

2- fever, anorexia, depression, and oculonasal discharges developed, followed by necrotic lesions on the gums, buccal mucosa, and tongue. The hard and soft palates were often affected.

3-The oculonasal discharge became mucopurulent, and the muzzle appeared dry and cracked.

4-Diarrhea, the final clinical sign, could be watery and bloody.

5-Convalescence was prolonged and could be complicated by concurrent infections due to immunosuppression.

6- Morbidity was often 100% and mortality was up to 90% in epidemic areas, but in endemic areas morbidity was low and clinical signs were often mild.

Pathogensis:-

PPR virus penetrates the retropharyngeal mucosa, sets up a viremia and specifically damages the alimentary, respiratory and lymphoid systems. Infected cells undergo necrosis, and in the respiratory system, also proliferation. Death may occur from severe diarrhea and dehydration, before respiratory lesions become evere, or is hastened by concurrent diseases such as pneumonic pasteurellosis, coccidiosis or coliform enteritis. Ly mphoid necrosis is not as marked as in rinderpest and the possibility of immunosuppression. Most sheep and some adult goats recover.

Differential diagnosis

- Heart water
- Pneumonic pasteurellosis
- Contagious caprine pleuropneumonia in goats
- Contagious bovine pleuropneumonias
- Helminthosis
- Coccidiosis
- Contagious ecthyma.

Lesions:

Gross pathologic lesions occurred throughout the GI and upper respiratory tracts, either as areas of necrosis and erosion, or congestion and hemorrhage, the latter creating classic "zebra-striping" in the rectum. Lymph nodes could be enlarged and edematous, with white necrotic foci in the Peyer's patches. Histologic lesions included lymphoid and epithelial necrosis with viral-induced syncytia, and intracytoplasmic and intranuclear inclusions were often seen.

Laboratory diagnosis:-

1- Identification of the agent.

2-Antigen detection.
3-Agar gel immunodiffusion test
4-Direct and indirect immunoperoxidase tests
5-Counter immunoelectrophoresis
6- Immunohistopathology.
7-Virus isolation and identification.
8-Virus RNA detection.(Rinderpest-specific cDNA probes, Amplification by polymerase chain reaction (PCR)
9- Serological tests.(ELISA)

Malignant catarrhal fever (MCF)

Synonyms:-(malignant catarrh, malignant head catarrh, gangrenous coryza, and catarrhal fever).

Definition: it is an acute, sporadic, infectious disease of cattle and some other bovidae and cervidae characterized by low morbidity and extremely high mortality.

Etiology:

Family:-Herpesviridae

Genus:- *Rhadinovirus* multiple serotypes species and geographically dependent

1-Alcelaphine herpesvirus-1 (AHV-1) natural host: wildebeest in Africa

2-ovine herpesvirus-2 (OHV-2) natural host: domestic sheep and goats worldwide

3-Alcelaphineherpesvirus-2(AHV-2):-nonpathogenic but is latently carried by wildebeest.

4- caprine herpesvirus (CpHV-2) natural host: domestic goats.

5- Temperature: No data available, but virus is very labile.

6-pH: Mostly stable between pH 5.5–8.5

7-Disinfectants/chemicals: Inactivated by common disinfectants including sodium hypochlorite (3% solution if heavy organic debris present)

8-Survival: Inactivated rapidly by sunlight. Cell-associated virus survives 72 hours outside the host; cell-free virus is inactivated quickly in dry environments but may survive over 13 days in humid environments.

Transmission:-

1-AHV-1

A-Wildebeest calves

1-In utero.

2-Contact with nasal and ocular secretions.

3-Aerosols during close contact.

B-Adult wildebeest

1-Cell-associated form.

2-Rarely transmitted.

2-OHV-2

1-Respiratory (aerosol).

2-Transplacental rare.

3-Contact with nasal secretions.

4-Animal-to-animal rare.

Dead end hosts once infected with OHV-2 or AHV-1.

PATHOGENESIS

MCF is a fatal, multi systemic disease characterized **by lymphoid proliferation** and **infiltration**, and widespread vascular **epithelial and mesothelial lesions**, which are morphologically associated with lymphoid cells. **CD8+ T- lymphocytes** are the predominant cells associated with the vascular lesions. including the **epithelial erosions** and **keratoconjunctivitis.** The lymph node enlargement is due to atypical proliferation of sinusoidal cells and the cerebromeningeal changes usually referred to as **encephalitis.** There is commonly a **synovitis,** especially involving tibiotarsal joints and this also is associated with a **lymphoid vasculitis**. It is believed that the pathogenesis of this disease is the result of direct virus-cell interactions or perhaps immune-mediated responses directed against infected cells**.**

Clinical Signs :-

Experimental infections have an incubation period of 9 to 77 days.

1-Initial form:-

a-Depression, diarrhea, DIC, dyspnea, high fever, inappetence

2-Peracute form: sudden death

a-Head and eye form:-

characterized by Reddened eyelids, Bilateral corneal opacity, Crusty muzzle, nares, Nasal dischargeand Salivation. In the later stages of the head and eye form Erosions on the tongue and buccal mucosa, Joints, superficial lymph nodes swell, Horn, hoof coverings slough and Nervous signs.

b-Majority of cattle cases

3-Intestinal form

a-Initially like head and eye form, but death occurs from severe diarrhea.

4-Mild form:-

Inoculated animals; recovery expected.

Lesions:

1-Erosions on the tongue and soft and hard palate.
2-Necrotic areas in the omasal epithelium.
3-Multiple erosions of intestinal epithelium.
4-Greatly enlarged lymph node compared to normal
5-Necrotic areas in the larynx.
6-Urinary bladder mucosa hyperemic and edematous
7-Kidney often has raised white foci on the cortex

Differential Diagnosis:-

bovine viral diarrhea mucosal disease, bluetongue, rinderpest, foot and mouth disease, vesicular stomatitis, salmonellosis, pneumonia complex, oral exposure to caustic materials, mycotoxins, and some poisonous plants.

Laboratory Diagnosis:-

1-Histopathology
2-PCR
3-Virus isolation (AHV-1)
4-Serology
a-AHV-1 antibodies in wildebeest
1-Immunofluorescence, immunoblot, VN, ELISA, immunocytochemistry .
b-OHV-2 antibodies in sheep
1-Immunofluorescence, immunoblot .

Bovine virus diarrhea - Mucosal disease (BVD-MD)

Synonyms :- BOVINE Pestivirus disease complex.

Definition: is a contagious viral disease of cattle and other ruminants first seen in 1946 in New York as a gastro-enteritis with severe diarrhea, ulcerations of the muzzle and nasal and oral cavities, fever, reduction in milk secretion, cessation of rumination and abortions.

Etiology

Bovine Virus Diarrhoea (BVD) and Mucosal Disease (MD) are caused by a the viruses belong to family ***Flaviviridae*** and the genus ***pestivirus***. are RNA viruses, there are two biotypes designated depending on their effect on tissue culture cells

1-non-cytopathic (NCP): The non- cytopathic type is the most common and most important. the non-cytopathic type crosses the placenta, invades the fetus and establishes persistent infection in the fetus, which is crucial for spread of the virus. It is the cause of a wide range of congenital, enteric and reproductive diseases.

2-cytopathic (CP): the cytopathic biotype of the virus is usually associated with only mucosal disease in animals .

Methods of transmission

1-Direct contact

The virus is transmitted by **direct contact between animals**, by **transplacental transmission to the fetus**, **Discharges from the reproductive tract of an infected cow**, **Aborted fetuses and Nose-to-nose contact.**

2- Indirect contact

The virus is transmitted by **Airborne transmission**, **Flies and Fomites**.

Pathogenesis:-

1-Virus is shed in fluids: saliva, oculonasal discharge, urine, feces, semen, uterine secretions, amniotic fluid, fetal tissue, and blood

2-Primary replication occurs in tonsils and oropharyngeal lymphoid tissue (Waldeyer's ring)

3-BVDV targets lymphocytes and macrophages; enters circulating monocytes and is transported to lymphoid tissues and the subepithelial connective tissue of the dermis and GI tract, where it spreads locally to overlying epithelial cells and respiratory tract

4-Infection of pregnant cows leads to transplacental infection leading to various complications.

Clinical signs:-

1- subclinical infection (bovine virus diarrhea) The most frequent form of BVDV infection in cattle is non -clinical or a mild disease of high morbidity and low case fatality characterized by a mild fever, leukopenia, inappetence and mild diarrhea followed by rapid recovery in a few days and the production of virus neutralizing antibodies.

2- acute mucosal form of the disease (mucosal disease) is characterized by the sudden onset the morbidity is low and the casefatality rate is high (over 90%) Affected animals are depressed, anorexic and drool saliva, wetting hair around the mouth. Fever, tachycardia and polypnea are common. Ruminal contractions are usually absent and a profuse and watery diarrhea occurs 2-4 d after the onset of clinica1 illness. The feces are foul smelling and may contain mucus and variable quantities of blood**.** Lesions of the oral cavity mucosa consist of discrete, shallow erosions which become confluent, resulting in large areas of necrotic epithelium becoming separated from the mucosa of the mouth, the muzzle and entire oral cavity may have a 'cooked' appearance with the grayish colored necrotic epithelium with mucoperulant nasal discharge Dehydration and weakness are progressive and death occurs 5-7 d after the onset of signs**.**

3- Thrombocytopenia and hemorrhagic disease(non-cytopathic BVDV) Bloody diarrhea and ecchymotic hemorrhage of the visible mucosa**.**

4- Reproductive failure and neonatal disease includes increased embryonic mortality, fetal mummification, abortion, premature births, stillbirths, congenital defects and the birth of stunted weak calves**.**

Lesions

1-Acute BVD: In the mild form, mild erosions or shallow ulcerations of the oral cavity

2-Severe acute BVD: lesions closely resemble mucosal disease (below)

a-Linear esophageal ulcerations

b-Erosions and ulcerations of mouth, tongue, oral and ruminal papillae, abomasum, cecum/colon

c-Peyer's patches swollen, necrohemorrhagic, +/- diphtheritic membrane

d-Erosive-ulcerative interdigital dermatitis and coronitis

Differential Diagnosis:-

1-Infectious bovine rhinotracheitis (IBR)

2-Leptospirosis

3-Neospora caninum

4-Moldy feed exposure

5-Brucella abortus

6-Haemophilus somnus

7-Listeria monocytogenes

8-Congenital abnormalitie

DIAGNOSIS:

1-Virus isolation. 2- Immunofluorescent antibody assays. 3- Viral RNA detection using PCR. 4- Antigen detection using immunohistochemistry. 5- Serum virus neutralization. 6- Antigen capture ELISA.

Ephemeral fever

Synonyms: three day sickness ;stiff sickness; bovine epizootic fever

Definition :it is a benign arthropod transmitted viral disease of cattle characterize by high rise of temperature , stiffness , lameness , muscular tremor with spontaneous recovery with in few days . death may ensure in very limited cases with complication.

Etiology:-

1-Bovine ephemeral fever virus are caused by a the viruses belong to family Rhabdoviridae and the genus *Ephemerovirus*.

2- Ether sensitive and Inactivated at pH below 5 and above 10.

Transmission:-

1- The virus is transmitted by an insect vector.

Pathogenesis

Following entry of the virus in the host , the incubation period is usually 2-10 days .the virus multiply in the blood and viraemia sets in . the virus then localizes in the mesodermal tissues like joints , muscle , lymph nodes and thereby clinical manifestation like dyspnea . limb stiffness supervene . the virus grow in the reticulo endothelial cells , in the lungs spleen , lymph nodes rather than lymphoid cell or vascular endothelium .

Clinical sings:-

1-Fever usually lasting only 1-2 days

2-Stiffness, with a shifting lameness affecting one or more legs. Some animals become recumbent and may remain down for up to a week

3-Subcutaneous swelling in the sub-mandibular area or around limb joints

4-Elevated respiratory rate and sometimes dyspnoea

5-Nasal and ocular discharges

6-Drooling of saliva

7-Periorbital swelling

8-Increased excitability and agitation.

Severely affected animals lose condition, milking cows suffer a severe drop in production and cows in advanced pregnancy may abort. In most cases the infection has a short duration with a rapid recovery. However, occasional cases die and relapses can occur.

Post-mortem findings

Gross pathology of affected animals is usually unremarkable, with the main findings being:

1-Oedema of lymph nodes

2-Increased fluid in body cavities and joint cavities, often containing fibrin.

Differential diagnosis :-

1-Blue tongue

2-Babesiosis

3-Black leg.

Diagnosis:-

1-clinical signs in an epidemic.

2-Serology .

3- Virus isolation

4-The neutralization test and the ELISA are recommended for antibody detection and give similar results

SHEEP POX AND GOAT POX

Definition:- Sheep pox and goat pox (SGP) are highly contagious diseases of sheep and goats characterised by fever, ocular and nasal discharges. Pox lesions appear on the skin and on the respiratory and gastro-intestinal mucosa. Mortality can be high.

Etiology:-

1- Virus family *Poxviridae*, genus *Capripoxvirus*
2- temperature:- Susceptible to 56°C/2 hours; 65° C/30 minutes. Some isolates inactivated at 56°C/60 minutes.
3-Ph:- Susceptible to highly alkaline or acid pH (hydrochloric or sulphuric acid at 2% for 15 minutes).
4- Disinfectants/chemicals:- Inactivated by phenol (2%) in 15 minutes. Sensitive to detergents, e.g. sodium dodecyl sulphate. Sensitive to ether (20%), chloroform, formalin (1%) and sodium hypochlorite (2-3%), iodine compounds (1:33 dilution), 2%, quarternary ammonium compounds 0.5%.

Transmission

1-Transmission is usually by aerosol after close contact with severely affected animals containing ulcerated papules on the mucous membranes.
2-Infection may also occur through other mucous membranes or abraded skin
3-Chronically infected carriers do not occur .

Pathogenesis

Incubation period of sheep pox is 4-8 of that of goat pox is 4-15 days. After it inters, goat pox virus replicates locally in the tissues. Since the virus is epitheliotropic, it will infest the epithelium tissues of the organism. On the 7^{th} day post-inoculation, the virus

titer reached to its peak. The virus spread to the regional lymph nodes, after 3-4 days of primary viremia. The viremia spread in the body, and affected spleen, lungs and liver. The virus inhaled may also cause lungs lesions. five stages in the development of pox infection**. Roseola stage** is stage in which Skin lesions typically begin with small red spots with in three days of infection which is followed by papules. The affected animals are febrile at this stage. The second stage of pox lesion is **Papules** wich develops after 3 days of roseola stage. Nodular skin lesions that are developed from roseola stage (red spots) those are hard during palpation. Papules with in 5-6 days are changed to **vesicles** and known as vesicular stage**. Pustular stage** develops after 3 days of vesicular stage. The last stage of pox lesion is **scab**. Quantitative analysis using real-time PCR and isolation of the pathogenesis of Sheep pox virus and Goat pox virus in their respective hosts revealed high viral loads in skin .

Clinical signs

The diseases are more severe in lambs and kids than adults. Disease begins with:

1-Sudden onset of fever

2-Discharges from the nose and eyes, and salivation

3-Loss of appetite

4-Reluctance to move

5-Skin lesions appear in 1-2 days, extending all over the skin, but are most obvious on face, eyelids and ears, perineum and tail. Lesions may also be seen on the mucous membranes of the nostrils, mouth and vulva

6-Acute respiratory distress

7-Mortality peaks about two weeks after the onset of the skin lesions

Lesions begin as an area of reddening, progressing over two weeks to a papule, vesicle, pustule with exudation and scab formation. Healing is very slow.

A nodular form of the disease (‘stone pox’) can also occur this resembles lumpy skin disease in cattle.

Laboratory diagnosis:-

1-Transmission electron microscopy.
2-Virus isolation.
3-Agar Gel Immunodiffusion (AGID) Test.
4-Counter immunoelectrophoresis (CIE) test.
5-Latex agglutination test.
6-Reverse phase passive haemagglutination (RPHA) test.
7-Single radial haemolysis (SRH) test.

8-Enzyme-linked immunosorbent assays (ELISAs).
9-Virus serum neutralization test systems.
10-Polymerase chain reaction (PCR)
11-Hypersensitivity test

Rabies

Synonyms:- (Hydrophobia, Lyssa, Mad Dog, Madness, Lytta.)

Definition:- is a viral disease that causes inflammation of the brain in humans and other mammals. Early symptoms can include fever and tingling at the site of exposure. These symptoms are followed by one or more of the following symptoms: violent movements, uncontrolled excitement, fear of water, an inability to move parts of the body, confusion, and loss of consciousness.

Etiology :-

1-Rabies is caused by neurotropic RNA viruses belonging to the family rhabidoviridae and genus lyssa virus**.**

2-Temperature: Does not survive for more than 24 hours in dead animals when temperatures reach 21°C (70°F), but is highly resistant for extended periods at low or freezing temperatures.

3-pH: Sensitive to very low pH (below 3) or very high pH (greater than 11).

4-Chemicals/ Disinfectants: Inactivated by sodium hypochlorite, 45–75% ethanol, iodine preparations, quaternary ammonium compounds, formaldehyde, phenol, ether, trypsin, β-propiolactone, and some other detergents.

5-Survival: Does not survive well outside its host (in dried blood and secretions) as it is susceptible to sunlight and desiccation. It is also susceptible to ultraviolet radiation

Transmission

1-Rabies virus can be transmitted between mammals, whether they belong to the same or different species.

2- Rabies virus is primarily transmitted through the saliva of an infected animal. Saliva becomes infectious a few days prior to the onset of clinical signs.

3-Infection occurs primarily via bite wounds, or infected saliva entering an open cut or wound or mucous membrane, such as those in the mouth, nasal cavity or eyes.

4-Occasional, albeit rare, transmission by inhalation of infected aerosol has been described.

Pathogenesis:-

The commonest mode of transmission in man is by the bite of a rabid animal or the contamination of scratch wounds by virus- infected saliva. However, other routes have been implicated in the past, such as through mucous membranes of the mouth, conjunctiva, anus and genitalia. Infection by aerosol transmission had been demonstrated in experimental animals and has been implicated in human infection in rabies-infected bat caverns and in several laboratory accidents. Man to man transmission by transplantation of infected corneas were reported in 5 instances. Rabies is an acute infection of the CNS which is almost invariably fatal. The virus is similar to VSV of cattle. Following inoculation, the virus replicates in the striated or connective tissue at the site of inoculation and enters the peripheral nerves through the neuromuscular junction. It then spreads to the CNS in the endoneurium of the

Schwann cells. Terminally, there is widespread CNS involvement but few neurons infected with the virus show structural abnormalities. The nature of the profound disorder is still not understood.

Clinical sings:-

The incubation period is highly variable, ranging from 7 days to several years. It depends on several factors such as;

1. Dose of inoculum
2. The severity of the wound
3. The length of the neural path from the wound to the brain e.g. wounds on the face have a shorter incubation period than wounds in the leg

The clinical signs observed in rabies can usually be divided into two forms**.**

1- **furious form**:- The early signs of rabies typically include behavioral changes . the animal may appear anxious, aggressive or more friendly than normal. As the disease progresses, animals develop hypersensitivity to light and sound. They may also have seizures and/or become extremely vicious.

2-Paralytic Rabies:- The final stage of rabies is typified by paralysis of the nerves that control the head and throat . the animal will hypersalivate and lose the ability to swallow. As the paralysis progresses, the animal eventually goes into respiratory failure and dies.

Differential diagnosis

1- **Lead poisoning. 2- Lactation tetany**. **3-Vitamin A deficiency**. **4-Listeriosis.**

5-Enterotoxemia. **6-Pregnancy toxemia**.

Laboratory Diagnosis

1- Histopathology - Negri bodies are pathognomonic of rabies.
2- Virus cultivation.
3- Serology.
4- Rapid virus antigen detection**.**

Pseudo rabies

Synonyms:- Aujeszky's disease, Intentions bulbar paralysis, Mad itch.

Definition:- is a major viral disease manifested in swine by signs and lesions that vary among different age groups. The disease is characterized by three overlapping syndromes that reflect lesions in the central nervous system (CNS), respiratory system or reproductive system.

Etiology:-

1-The disease is caused by family Herpesviridae and genus *Varicellovirus*.

2-PRV is stable over a pH range of 4–12 and can remain infectious at cold temperatures for weeks. The virus is inactivated at high temperatures.

3-PRV is reportedly susceptible to disinfectants including orthophenolphenate compounds, peracetic acid, formalin, 2% sodium hydroxide, trisodium phosphate iodide disinfectants, 1–2% quaternary ammonium compounds, hypochlorite (bleach), and chlorhexidine

Mode of Transmission:-

1- Direct oronasal contact is the main route of transmission in domestic swine.
2- PRV is transmitted vertically. Venereal transmission is considered to be the main route in feral swine. The virus is also transmitted by air, water, and contaminated fomites.
3- Ingestion of infected meat or carcasses is linked to PRV transmission in dogs and cats, as well as free-ranging and captive wildlife.

4-contaminated drinking water and feed buckets may transmit the disease.

5-artificial insemination**.**

6-Aerosol**.**

Pathogenesis:-

After entrance of the virus -------- viraemia -------- localization of the virus in various organs ------- Multiplication in resp. epith. cells ------- The virus when pass from respiratory tract used to produce intense pruritus in the infected animal----- The virus on entry through local abrasions affect the peripheral nerve tract and pass centripetally causing damage of the nerve cells-------generalized systemic infection after involvement of central nervous system.

Clinical signs:-

1-Respiratory infection is usually asymptomatic in pigs more than 2 months old

2-can cause abortion, high mortality in piglets, and coughing, sneezing, fever, constipation, depression, seizures, ataxia, circling, and excess salivation in piglets and mature pigs. Mortality in piglets less than one month of age is close to 100%, but it is less than 10% in pigs between one and six months of age.

3-Pregnant swine can reabsorb their litters or deliver mummified, stillborn, or weakened piglets.

4- In cattle (see next section), symptoms include intense itching followed by neurological signs and death.

5-In dogs, symptoms include intense itching, jaw and pharyngeal paralysis, howling, and death Any infected secondary host generally only lives two to three days

Lesions:

Serous rhinitis, necrotic tonsillitis, or hemorrhagic pulmonary lymph nodes may be seen. Pulmonary edema, as well as pneumonic lesions of secondary bacterial pathogens, may be present. Necrotic foci (2–3 mm in diameter) may be scattered throughout the liver. Such lesions are typically found in young (<7 days old) piglets.

Microscopically, nonsuppurative meningoencephalitis is a characteristic lesion that can be present in gray and white matter. Mononuclear perivascular cuffing and neuronal necrosis may also be present. The meninges are thickened as a result of mononuclear cell infiltration. Necrotic tonsillitis with the presence of intranuclear inclusion bodies, as well as necrotic bronchitis, bronchiolitis, and alveolitis, are commonly seen. Focal areas of necrosis are often found in the liver, spleen, lymph nodes, and adrenal glands of macerated fetuses.

Differential diagnosis:-

1- Rabies. 2- Listeriosis**.** **3-** Polyencephalomalacia**.** **4-** Poisoning**.**

Diagnosis:-

1-gross and microscopic lesions,
2-virus isolation, fluorescent antibody testing, and serologic testing**.**
3-serum neutralization, ELISA, and latex agglutination**.**

Rift Valley Fever

Definition:- is a viral disease that can cause mild to severe symptoms. The mild symptoms may include: **fever, muscle pains, and headaches which often last for up to a week.** The severe symptoms may include: **loss of sight beginning three weeks after the infection, infections of the brain causing severe headaches and confusion, and bleeding together with liver problems** which may occur within the first few days.

Etiology

1-Rift Valley fever (RVF) virus is a negative-sense, single-stranded RNA virus of the family **Bunyaviridae** within the genus ***Phlebovirus***. Only one serotype is recognised but strains exist of variable virulence**.**

2-Temperature: Virus recoverable from serum after several months at 4°C or 120 minutes at 56°C.

3-pH: Resistant in alkaline environments but inactivated at pH <6.8.

4-Chemicals/Disinfectants: Inactivated by lipid solvents (i.e. ether, chloroform, sodium deoxycholate), low concentrations of formalin and by strong solutions of sodium or calcium hypochlorite (residual chlorine should exceed 5000 ppm).

5-Survival: Survives in freeze dried and Can survive contact with 0.5% phenol at 4°C for 6 months**.**

Transmission:

1-transcutaneous transmission, aerosol transmission, and mosquito or other insect bite.

2-contact with the blood, secretions, or excretions of infected animals.

3-Aerosol transmission of the disease**.**

Pathogensis:-

The pathogenesis of RVF results from the spread of virus from the site of introduction to the body and intitial replication sites to critical organs such **as the spleen, liver and brain**.

The liver is usually **enlarged, soft, friable and yellowish-brown to dark reddish-brown in colour. Irregular congested patches and haemorrhages** of varying size are often present in the substance of the liver together with pale foci. **Jaundice is** seen in only a relatively small proportion of lambs because of the short time to death**.**

Haemorrhages and oedema of the gall bladder are common and the **bile may contain blood.** Elsewhere, in newborn lambs, **petaechial and ecchymotic haemorrhages are found in the abomasal mucosa and the contents are often dark brown from the presence of partly-digested blood;** the contents of the small intestine may be similar. Most mature sheep have haemorrhages and oedema in the abomasal folds and sometimes free blood in the intestinal lumen. **the spleen is enlarged with haemorrhages in the capsule.**

Aborted cattle fetuses, calves and older cattle show lesions essentially similar to those in sheep fetuses, lambs and older sheep.

Clinical Findings:

The incubation period is 12–36 hr in lambs, and a biphasic fever of up to 108°F (42°C) may develop.

1- Mortality in **young lambs** is high (90%–100%), and animals usually die within 2–3 days. Adult sheep are less susceptible, with 10%–30% mortality; the incubation period is 24–72 hr, and animals show a generalized **depressen, hematemesis, hematochezia, and nasal discharge, although infection may also be inapparent.**

2- **Calves** are less susceptible than lambs, but mortality may still be as high as 70%; clinical signs are similar to those in sheep, Disease in adult cattle is often inapparent, but they may show anorexia, **lacrimation, salivation, nasal discharge, dysgalactia, and a bloody or fetid diarrhea, with a mortality of 5%–10%.** Sometimes, **abortion** may be the only sign of infection; the aborted fetus is usually autolyzed.

3- **In pregnant ewes**:abortion rates vary from 5% to almost 100% in different outbreaks and on different farms; abortion rates in cattle are usually <10%.**severe disease with ocular lesions, encephalitis, or severe hepatic lesions with hemorrhages**; in such cases, the fatality rate may be 10%–20%.

Lesions:

The **hepatic lesions** are similar in all species and vary mainly with the age of the affected individual. The most severe lesions, seen in **aborted fetuses and newborn** lambs, are moderately to **greatly enlarged, soft, friable livers with irregular congested patches.** Numerous grayish white necrotic foci are invariably present but may not be clearly visible. **Hemorrhage and edema of the wall of the gallbladder and mucosa of the abomasum are common. Intestinal contents are dark chocolate-brown.**

Differential diagnosis

1-Bluetongue. 2- Wesselsbron disease. 3- Enterotoxemia of sheep. 4- Ephemeral fever. 5- Brucellosis. 6- Vibriosis. 7- Trichomonosis. 8- Nairobi sheep disease. 9-Heartwater. 10- Ovine enzootic abortion. 11-Toxic plants. 12- Bacterial septicaemias 13- Rinderpest and Peste des petits ruminants 14- Anthrax**.**

Diagnosis

1-RVF antigen detection

- Agar gel diffusion test
- Virus isolation in mice/hamsters/tissue culture
- RT-PCR identification of RVF virus
- Capture ELISA test
- Immunochemical staining of fixed tissues

2- RVF antibody detection

- ELISA tests for IgM/IgG
- Indirect immunofluorescent/peroxidase tests
- Microtitre virus-serum neutralization tests
- Indirect haemagglutination tests
- Plaque reduction assays

Blue tongue

Definition: it is an infectious non – contagious borne virus disease of domestic and wild animals .this is predominantly disease of sheep but occasionally cattle and goat are affected .the disease is characterized by **high fever ,catarrhal inflammation of the buccal mucous membrane (stomatitis) and nasal mucous membrane (catarrhal rhinitis). Beside there is inflammation of the tongue , intestine and sensitive lamina of the foot.**

Etiology :

1- The disease is caused by a RNA containing virus 100-150 nm and belong to arthropod borne **orbivirus** of **reoviridae** family

2-the virus is fairly resistant against commonly used disinfectants . but it is destroyed by 3%sodium hydroxide .

Mode of transmission :

1-The disease is not contagious but spread through blood sucking **midges** of the, **mosquitoes, biting flies.**

2- transmission through semen and placental route is possible .

Pathogenesis:

Following transmission through the bite of the vector the virus **multiply in the blood stream and leads to a primary viraemic stage**, the virus get **attached with the red**

blood cells . the characteristic lesion appear due to damage of blood vessels . it produces hyperaemia . hemorrhages and oedema of various tissue .

fatal infection may be noted in white tailed deer**. There is viraemia followed by high concentration of virus in spleen , lymph nodes characteristic by high rise of temperature**

Clinical finding :

In this infection , the morbidity is usually 50% and the **mortility rate range from 10-90% in different geographical locations . incubation period varies from 1-10 days.**

The disease is characterized by high temperature for 5-6 days with progressive reddening of the buccal mucous membrane . the disease divided in to three forms :

1-acute form: is characterized by **high rise temperature .nasal discharge , salivation and lacrymation .there is drooling of saliva and swelling of gum and lips . these changes may be followed by ulceration of the dental pad . muzzle turns dry and show burnt appearance , skin of other areas like flank , neck , back . there is cyanotic and bluish appearance of the tongue .**

Udder may be swollen and teat may show signs of ulceration **. coronary band may become swollen and it may crack .** hoof may separated leading to **lameness** . diarrhea and dysentery may supervene . rapid shallow respiration is not uncommon and pneumonia may be a feature . there is appreciable reduction of body weight . animal may show torticollis . signs of lameness due to coronitis is not uncommon .

2-Abortive form is characterized by abortion and deformities .

3-Sub acute form or subclinical form : is noted in cattle and generally passed unnoticed .

Sheep is the most susceptible animal. High morbidity rate in them is noted . stress factors like lower plane of nutrition , worm ,burden , inclement weather , fatigue due to transportation .

Lesion :

Characteristic lesion are observed in the mouth and tongue

Mouth : there is hyperaemia
Tongue :it become blue and gangrenous
Leg :coronary band show congestion and hemorrhage
Skeletal and cardiac muscle: singes of hyperaemia.
lunge :pneumonia .

Placenta: hemorrhage**.**

Differential diagnosis:-

1- Contagious ecthyma. 2- Foot and mouth disease. 3- Vesicular stomatitis. 4- Malignant catarrhal fever. 5- Bovine virus diarrhea. 6- Infectious bovine rhinotracheitis. 7- Parainfluenza-3 infection. 8-Sheep pox .

Diagnosis :

1-Animal inoculation
2-Isolation of the virus .
3-Complement fixation test
4-Neutralization test .
5-Plaque inhibition test .
6-Micro gel diffusion test.
7-Flurescence antibody test .
8-Agar gel immune diffusion test
9-ELISA.

Equine infectious anemia

Synonyms:- horse malaria or swamp fever and Slow Fever.

Definition:- (EIA) is infectious viral disease can affect horses, donkeys, asses and other equine species.This virus destroys red blood cells and is spread through blood-to-blood contact, not through close proximity or casual contactthe disease characterized by fever, anemia ,edema, weakness, and death.

Etiology:-

1-Equine infectious anemia is caused by equine infectious anemia virus (EIAV), a **lentivirus** in the family **Retroviridae**.

2- the virus can be cultivated in tissue culture and the virus can be killed by exposing them to heat and commonly available house detergents .

Transmission:-

1-Equine infectious anemia virus is transmitted mechanically on the mouthparts of biting insects.

2-This virus can also be transmitted in blood transfusions or on contaminated needles, surgical instruments and teeth floats.

3-Possible transmission through milk has been reported in some nursing foals.

4-transmitted the virus to a mare with a vaginal tear during breeding.

5-The possibility of aerosol transmission by infectious material during close contact was raised .

Pathogenesis

Virus on entry localizes in the splanchnic organs mostly in spleen , kidney ,liver and lymph node and multiply there . after a period of around 30 days of viraemia virus attached the erythrocyte and cause their destruction . they also cause the damage of the endothelial lining of the blood vessels.

Periphereal nerves ,meninges and the brain tissue are affected and cause nervous manifestation . there is haemolytic anaemia due to haemolysis or phagocytosis by reticuloendothelial system . profound anaemia sets in due to haemolysis and defective erythropoiesis renal glomeruli causing glomerulitis.

Clinical signs

The incubation period is a week to **45 days** or longer. Some horses remain asymptomatic until they are stressed.

the disease can appear in three forms: **acute** and **chronic**.

1- **Acute EIA** is most often associated with the first exposure to the virus, with fever and hemorrhages evident from 7 to 30 days after exposure. Acute EIA is thought to be associated with massive virus replication in and destruction of infected macrophages. In the acute form, animals are extremely sick and may die.

2-Chronic cases exhibit the more classic clinical signs such as anemia, fever, depression, hemorrhages, progressive weakness, loss of weight and swelling of the legs, brisket and low abdomen. Equids that have the chronic form or recover from the acute form, usually have intermittent attacks that may vary considerably in time intervals and severity of symptoms. Some horses die during these recurrent episodes.

Post Mortem Lesions

1-The spleen, liver and abdominal lymph nodes may be enlarged, and the mucous membranes can be pale.

2- In chronic cases, emaciation may also be noted. Edema is often found in the limbs and along the ventral abdominal wall.

3- Petechiae may be observed on internal organs, including the spleen and kidney. Mucosal and visceral hemorrhages and blood vessel thrombosis have also been reported.

4-Chronically infected horses that die between clinical episodes usually have no gross lesions, but some animals may have proliferative glomerulonephritis or ocular lesions.

Defferential diagnosis

Acute disease

- Babesiosis
- Equine granulocytic ehrlichiosis
- Equine viral a rteritis
- Autoimmune hemolytic anemia
- Leptospirosis
- Parasitism
- Idiopathic thrombocytopenia,

Chronic disease

- Internal abscessation (metastatic

Streptococcus equi infection

- C hronic inflammatory disease, neoplasia and chronic hepatitis.

Diagnosis

1-agar gel immunodiffusion .

2-enzyme-linked immunosorbent assays (ELISAs).

3- Reverse-transcriptase polymerase chain reaction (RT-PCR) assays.

Equine Viral Arteritis

Equine viral arteritis (EVA):- is an acute, contagious, viral disease of equids characterized by fever, depression, anorexia, leukopenia, dependent edema conjunctivitis, , nasal discharge, respiratory distress, skin rash, temporary subfertility in affected stallions, abortion, and infrequently, illness and death in young foals.

Etiology

1-Equine viral arteritis is caused by equine arteritis virus (EAV), an RNA virus in the genus *Arterivirus*, family Arteriviridae Isolates vary in their virulence and potential to induce abortions.

transmission

Infection spreads through transmission of the virus between horses in 4 main ways:

1-venereal infection of mares by stallions during mating تزاوج

2-artificially inseminating mares with semen from infectious stallions

3-contact with aborted fetuses and other products of parturition

4-direct contact in droplets from the respiratory tract (e.g. through coughing and snorting).

PATHOGENESIS

1-After inhalation of the virus it binds to the respiratory epithelium and infects alveolar macrophages.

2-Three days after infection the virus detectable in circulating monocytes with subsequent systemic distribution of infection.

3-The virus localized in vascular endothelium .

4-there is significant damage to blood vessels by day 10. The virus infects renal tubular epithelium and can persist there for up to 2 weeks. Medial necrosis of blood vessels might cause anoxia of associated tissues. Virus is not detectable in any tissue by 28 days after infection, with the exception of accessory sex glands in intact male horses. Abortion is caused by a severe necrotizing myometritis and presumed consequent reduction in fetal blood flow.

Clinical Signs

The incubation period varies from 2 days to 2 weeks. Most EAV infections, especially those that occur in mares bred to long-term carriers, are asymptomatic.

1-Fulminant infections with severe interstitial pneumonia and/ or enteritis can be seen in foals up to a few months of age.

2-Systemic illness also occurs in some adults. In adult horses, the clinical signs may include fever, depression, anorexia, limb edema (particularly in the hind limbs), and dependent edema of the prepuce, scrotum, mammary gland and/or ventral body wall.

3-Conjunctivitis, photophobia, periorbital or supraorbital edema and rhinitis can also be seen. Some horses develop urticarial شروي; the hives شرى may be localized to the head or neck, but are sometimes generalized.

4-Abortions or stillbirths can occur in mares that are pregnant when they are exposed.

5-Temporary decreases in fertility, including reduced quality sperm and decreased libido, may be seen in stallions during the acute stage of the disease. The decrease in sperm quality has been attributed to increased scrotal temperature and edema, and can persist for up to 4 months.

6-Outbreaks have not been reported among donkeys or mules, and although antibodies have been found, there is little information on the clinical signs in these species. Fever, sometimes accompanied by mild depression, mild conjunctivitis, a slight oculonasal discharge and/or mild dependent edema were reported in donkeys inoculated with an EAV strain that is moderately virulent for horses.

Post Mortem Lesions

1-In acute cases, the lesions are characterized by edema, congestion and hemorrhages of the subcutaneous tissues, visceral organs and lymph nodes.

2-These changes are often found in the subcutaneous tissues of the limbs and abdomen, the thoracic and abdominal lymph nodes, and the small and large intestines (especially the colon and cecum), but may occur throughout the body.

3-Accumulations of clear, yellowish fluid may be found in the peritoneal cavity, pleura and pericardium.

4-Foals may also have pulmonary edema, interstitial pneumonia, emphysema, splenic infarcts and enteritis.

In mares that abort, the endometrium may be swollen and congested, and can contain hemorrhages. Aborted fetuses are often partially autolyzed, but may be well preserved. In some fetuses, the only gross lesions may be excess fluid in the body cavities and signs of interlobular interstitial pneumonia.

Differential diagnosis:-

1-equine influenza 2- strangles .3-equine infectious anemia. 4-African horse sickness, 5-purpura hemorrhagica. 6-Abortion 7-Salmonella abortus equi. 8-leptospirosis.

Diagnosis:-

1-Virus isolation can be attempted from swabs of the nose, throat, or eyes; semen, placentas, or fetal tissue; and blood samples.

2- common method of diagnosis is testing blood for the virus' neutralizing antibodies that cause EVA.