Chapter 10

Exotic Diseases

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This presentation will focus on the Office International des Epizooties (OIE) List A diseases of particular interest to ruminant practitioners. OIE List A diseases are transmissible diseases that have the potential for very serious and rapid spread, irrespective of national borders, that are of serious socio-economic or public health consequence and that are of major importance in the international trade of animals and animal products.

Reports are submitted to the OIE as often as necessary to comply with Articles 1.1.3.2 and 1.1.3.3 of the *International Animal Health Code*.

Reference should also be made to AUSVETPLAN (http://www.aahc.com.au/ausvetplan/)

- Foot and mouth disease
- Vesicular stomatitis
- Rinderpest
- Peste des petits ruminants
- Sheep pox and goat pox
- Lumpy skin disease
- Bluetongue
- Contagious bovine pleuropneumonia

Vesicular and erosive diseases

Foot and mouth disease

FMD is a highly contagious viral vesicular disease of cloven-hoofed animals. Although not highly lethal in adult animals, it causes serious production losses and is a major constraint to international trade in livestock and livestock products. There are seven distinct serotypes of FMD that do not cross-protect. Serotypes O and A are found globally, with serotype O responsible for the recent outbreaks in both Japan and UK; Asia 1 occurs in Asia; C has most recently been reported in Europe and South America; and SAT 1, 2 and 3 are confined to Africa with some incursion into the Middle East.

FMD virus is relatively stable in cool, humid environments but is rapidly inactivated at pH<5 and is susceptible to formalin, glutaraldehyde and hypochlorite disinfection.

Pathology

Pathogenesis
Infection of susceptible animals occurs primarily through inhalation of airborne droplets but also through breaks in the skin or oral mucosa. Initial replication occurs in the mucosa or lymphoid tissues of the oropharynx with localisation to the stratum spinosum of selected areas of the epidermis and proliferating myocardial cells of young animals. Development of vesicles may be exacerbated in frictional areas of the skin.

Clinical signs and gross pathology
The first case of FMD in Australia is likely to be in a pig as pigs may eat contaminated offal and they are an efficient amplifying host. However, infected cattle usually develop obvious clinical signs of FMD and so are also considered a good indicator species. Cattle are most infectious when they have early acute signs of disease, whereas sheep excrete a large proportion of the total virus excreted over a 1 to 2 day period before the occurrence of clinical signs.

- Incubation period of 2 to 5 days
- Fever, cessation of milk production, depression
- Inappetance, loss of condition
- Ropey saliva and lip smacking
- Vesicles in mouth and on feet (interdigital spaces, coronary bands, bulb) and on teats of lactating animals. Early coronary band lesions may simply appear as blanching of the area.
- Lameness, reluctance to move and shuffling of feet
- Death in young animals due to myocarditis
- Rapid healing of ruptured vesicles – usually within two weeks

In sheep, one third of infected animals may not show clinical signs. Where present, FMD lesions in this species are often small, rupture easily and heal quickly. Minor erosions following vesicular rupture are no longer identifiable as FMD, and the duration of lesion presence in an individual sheep averages only two days. Most lesions in sheep occur on the feet, usually in the interdigital cleft. Non- specific signs such as lameness, pyrexia and nasal discharger may also be identified. Where oral lesions occur, they are usually on the dental pad.

Some cattle, sheep and goats carry virus persistently in the pharynx in the face of a humoral immune response. This state may persist in cattle for up to 3 yrs and in sheep for up to 8mths. Experimental transmission of disease from carriers has not been established but epidemiological evidence suggests that cattle at least may be responsible for initiating field outbreaks of disease.

Specimens for laboratory diagnosis

- For virus isolation - Vesicular fluid; nasal swabs; epithelial covering of vesicles, or epithelial tags from freshly ruptured vesicles; whole blood; esophageal /pharyngeal fluid (probang). From dead animals tissue samples including heart, spleen, lymph node, adrenals, kidney and thyroid should be included. Foot lesions tend to have higher concentrations of virus for longer periods than lesions at other sites.
- For serology - serum from affected animals and unaffected herdmates
- For histopathology (to assist with later differential diagnosis) – lesional tissue including gastrointestinal tract, together with the range of tissues submitted for virus isolation.

Unfixed tissues and blood should be forwarded chilled in an AAHL-approved transport container. If a transit time of more than 48hrs is envisaged then ship the specimens on dry ice. Esophageal/pharyngeal fluid samples should be shipped frozen on dry ice unless they will be received at AAHL on the same day.

Differential diagnosis

Don't – all cases of vesicular disease are FMD until proved otherwise.

Vesicular stomatitis does not occur in Australia. Although responsible for considerable production losses, its primary importance is as a differential diagnosis for FMD. Vesicles may also occur following exposure to chemical irritants or scalding. More advanced cases of FMD, that is past the initial vesicular stage, may be mistaken for bovine papular stomatitis, mucosal disease, bovine malignant catarrh, IBR, rinderpest/PPR, bluetongue, photosensitization, foot rot and trauma.

Vesicular stomatitis

See Foot and Mouth Disease. Unlike FMD, vesicular stomatitis also affects horses and it is a zoonosis. Infection and disease of sheep is rare.

Rinderpest

Rinderpest is an ancient disease that probably originated in Central Asia. It is of considerable historic interest as the threat of its occurrence was the catalyst for the foundation of modern veterinary schools and state veterinary services in Europe.

The host range of rinderpest comprises all cloven-footed animals including kudu, eland, warthogs, giraffe and yaks but it causes disease principally in cattle and buffaloes. Current areas of endemicity include East Africa (Sudan, Uganda, Kenya, Ethiopia) and Asia where epizootics still occur in spite of vaccination programs. In these areas yearlings in

particular are affected as adults are protected by vaccines or natural immunity and calves by colostral antibody. Introduction of infected animals into susceptible populations is generally followed by high morbidity and mortality. There has been a single outbreak of rinderpest in Australian cattle near Fremantle in 1923. These animals had been transported from Derby following contact with infected pigs loaded onto the source ship in Asia and subsequent eradication was by rigorous application of quarantine procedures and slaughter of infected herds.

FAO (Food and Agriculture Organisation) has a Global Rinderpest Eradication Program the aim of which has been to eliminate the disease by 2010 using targeted vaccination and enhanced disease surveillance followed by active surveillance without vaccination. Technically, the ingredients for eradication are present and include excellent heat stable vaccines, good diagnostic tests, absence of carrier state in infected animals, no true reservoir in wildlife or insects and poor virus survival outside host. However, as in the past, such eradication programs continue to be blighted by breakdown of infrastructure services with global eradication of a major pandemic disease so far only achieved with smallpox.

The rinderpest virus is very unstable in the environment and survives a few days only in secretions, excretions or carcases. Infection by large droplets occurs following close contact between animals, with the virus present in expired air, tears, nasal secretions, saliva, urine, feces and milk.

There is a short incubation period of 2-6 days thus disease outbreaks can be explosive. Virus excretion commences at the end of the incubation period and may continue for the next 14 days or so.

Very few sequence changes are required to alter virus virulence and some strains cause extremely mild disease. The molecular basis of pathogenicity is not understood. In areas of endemicity the disease tends to be a milder syndrome with lower incidence and lower mortality rates than the classical highly lethal disease seen on first introduction to a susceptible population.

Pathology

Penetration of upper respiratory tract (URT) mucous membranes is followed by viral replication in URT lymphoid tissue, followed by viremia with virus attachment to mononuclear cells and proliferation in systemic lymphoid tissues, gastrointestinal tract and respiratory tract. Lesions observed at gross post mortem examination and histopathology naturally reflect the pathogenesis of the infection.

Clinical signs

- febrile illness with nasal and ocular discharge, constipation 1-2d
- necrotic plaques (no vesicles) on oral mucous membranes, nose, tips of cheek papillae, urogenital tract; dry flaky muzzle

  1-2 d
- fluid, dark foul smelling feces containing mucus, blood, necrotic mucosa

  2-3d
- death

  6-12d

Gross pathology

- Mucosal erosions – mouth, pharynx, esophagus, urogenital tract, nasal turbinates
- Erosions – abomasum especially pylorus, Peyer's patches of small intestine
- Hemorrhages with erosions in cecum and colon especially on the crests of the longitudinal mucosal folds ("tiger" or "zebra" striping)
- Swollen and congested lymph nodes

Specimens for laboratory diagnosis

Samples should include

i) 20ml of clotted blood from several animals (affected and unaffected) for serology

ii) Early clinical cases (fever, mucosal lesions, no diarrhea)
   - anticoagulated blood (20ml)
   - prescapular lymph node aspiration biopsy (by 14g sterile needle and syringe)
   - tears (cotton wool swab) in sterile saline
   - necrotic plaques

   Transport fresh and chilled. If transit time is expected to be greater than 72hrs then freeze to –80°C and ship on dry ice.

iii) Freshly killed acute cases (at least 2 animals)
   - 20g spleen, tonsil, lymph nodes Transport fresh and chilled
   - tonsil, liver, spleen, lymph nodes, kidney, brain, lesional gut fixed in formalin

Where possible avoid collecting samples from carcases as they rapidly become non-infectious following the pH changes occurring in autolysis and putrefaction.

Differential diagnosis

- Mucosal disease – usually affects a small number of animals in a herd. Note that the presence of pestivirus does not invalidate a provisional diagnosis of rinderpest

- Malignant catarrhal fever – corneal opacity reflecting uveitis, sporadic occurrence, lack of in contact transmission, association with pregnant sheep
- IBR – oral lesions fairly uncommon, no severe gut signs
- FMD (healing lesions) – feet lesions, no severe gut signs, no mortalities in adults

Peste des petits ruminants

PPR is a disease of sheep and goats endemic in the Middle East, sub-Saharan Africa and South Asia and is very similar clinically and pathologically to rinderpest (from which it probably evolved) that also occurs in sheep and goats. PPR may also be difficult to recognise on first introduction as respiratory signs may dominate due to the secondary complication of pneumonic pasteurellosis. Mortalities may be especially severe in goats compared to sheep.

Differential diagnosis

- Rinderpest/ PPR
- Bluetongue – feet lesions, hemorrhage at base of pulmonary artery
- **FMD (healing lesions)**
- Capripox – skin lesions

Capripox

Capripox causes sheep and goat pox and lumpy skin disease in cattle. Goat pox may in fact be identical to sheep pox, but host adaptation has occurred in endemic areas.

The threat of introduction of capripox to Australia is quite low, although it might occur through persistence of virus on livestock vessels, peoples' clothing and equipment, and unprocessed animal products. It is theoretically possible for infected insects to introduce lumpy skin disease.

Capripox infection is established via the subcutaneous route by biting insects (cattle) or the respiratory route via aerosols e.g. dried scabs (sheep). Local multiplication is followed 4-7 days later by viremia with organ localisation after 10-14 days. The sites of viral replication account for the nature of lesions that develop, namely

- Epithelium – vesicular skin lesions with both epithelial degeneration and hyperplasia
- Endothelium – dermal and subcutaneous vasculitis leading to edema, ischemia and infarction

Evolution of capripox lesions
This occurs over 2 to 3 days with progression through erythematous macules, papules, and vesicles (especially in sheep pox) through to depressed pustules with a grey necrotic centre ("sit fast"), crusts and slowly healing ulcers. The full thickness of the hide may be involved.

Sheep pox

The disease is endemic in sheep in Africa, Asia, and the Middle East and outbreaks have occurred more recently in Italy, Bulgaria and Greece. Mortalities of 80-100% can be expected in susceptible populations especially in young animals. Lower mortalities may be seen in adults, with fewer lesions confined to the areas under the tail and between the legs.

The virus is resistant to dessication and viable for up to 3mths on the wool of recovered animals or 6mths in dried crusts, particularly if these are protected from sunlight. In considering the trade implications of an outbreak of sheep pox it is worth remembering that the major markets of Australian wool are to sheep pox free countries. There is a much smaller market of live sheep export to sheep pox free countries as the main market is to the Middle East where sheep pox is endemic. Entry of sheep pox into Australia would be followed by major economic loss due to severe systemic disease with high mortality, decreased wool and meat yields, banning of exports of sheep products, and cost of disease prevention programs. Fine wool Merinos are especially susceptible to infection.

The occurrence of classical acute sheep pox in Australia would in all likelihood be readily diagnosed but low virulence isolates may prove a diagnostic challenge. Immunity to sheep-pox is predominantly cell mediated and infected animals and vaccinates may develop only low levels of neutralising antibody.

Control measures including slaughter, ring vaccination and the imposition of quarantine depend entirely upon confirmatory diagnosis in the laboratory.

Pathology

Clinical signs

- Fever, lacrimation, serous nasal discharge, salivation, hyperesthesia
- 2 to 3 days later, skin lesions develop especially in sparsely woolled areas and mucous membranes; respiratory distress
- survival is followed by prolonged convalescence

Gross pathology

- skin lesions – macules; wart-like oozing wheals; vesicles; necrotic crusts; ulcers
- pulmonary nodules – grey/white, small, generally subpleural

Specimens for laboratory diagnosis

- a range of skin lesions, lymph nodes and lung – fresh (chilled) and fixed in formalin
- 10ml of clotted blood from several animals (affected and unaffected) for serology

Lumpy skin disease

In cattle the disease occurs in South-East Africa, the Middle East and Madagascar. There is variable but often prolonged morbidity and variable mortality, with calves more likely to die than adults. The disease is not highly contagious, and mechanical transmission by biting insects is believed to be of more epidemiological importance than in sheep pox. Viral replication in the dermis occurs at the site of inoculation with the formation of a primary skin nodule. Viremia and fever ensue, followed by the development of generalised skin lesions in which virus persists for several weeks. Secondary lesions may also develop in upper respiratory tract, lung, esophagus, rumen, abomasum, kidney, testis and uterus.

Pathology

Clinical signs

- fever, salivation, oculonasal discharge, ventral edema, lymphadenopathy
- sudden appearance of a few to hundreds of skin lesions over most of the body including the heavily haired skin and mucous membranes
    - firm, flat, round nodules 0.5 to 5cm diameter involving the entire thickness of the skin
    - skin lesions may resolve or persist as intradermal masses; most undergo necrosis and sequestration to form a cone-shaped flat topped core of pink/grey necrotic tissue ("sit-fast")
    - sloughing of the necrotic tissue is followed by slow healing of the ulcer and permanent scarring of the hide
- respiratory distress

Specimens for laboratory diagnosis

- a range of skin lesions, lymph nodes and lung – fresh (chilled) and fixed in formalin
- 20ml of clotted blood from several animals (affected and unaffected) for serology

Differential diagnosis-sheep

- scabby mouth – transient vesicular stage with heavy crust formation; primarily on the mouth and feet of lambs and kids and the udder of dam; high morbidity, low mortality
- bluetongue – feet lesions, hemorrhage at base of pulmonary artery

- photosensitization
- dermatophilosis
- parasitic skin disease including Psoroptes ovis

Differential diagnosis-cattle

- Bovine herpesvirus-2 – superficial skin only involved with sloughing of scabs in about 3 weeks, usually without scarring; no lymphadenopathy, no internal lesions, no mortality
- Papular stomatitis – muzzle of calves and udder of dam
- Photosensitization
- Insect and tick bites
- Dermatophilosis
- Urticaria

Bluetongue

The major arboviral groups of veterinary significance present in the Asia-Western Pacific region include *Orbiviruses* of the bluetongue serogroups. The life cycle of arboviruses includes replication in both a vertebrate host and an arthropod vector. In the case of bluetongue the vector is a *Culicoides* midge and the virus is transmitted between hosts by the vector. The most common and widely distributed vector in Australia is *Culicoides brevitarsis* and its distribution currently delineates that of bluetongue viruses in the mainly cattle-raising areas of Australia.

Historically, bluetongue has been considered to be an African virus, possibly originally involving an antelope/midge cycle. All ruminant species including sheep, goats, cattle, buffaloes, antelope and deer are susceptible to BT infection. Ecologically and epidemiologically speaking, bluetongue is now best considered an infection of cattle as a higher % of exposed animals will seroconvert, it is easier to get an isolate from cattle, and there is a long viremia (~ 60 days). This new role for cattle may have followed agricultural expansion of cattle in African countries and thence elsewhere.

However, BT is regarded as a *disease* of sheep as this is the species in which the clinical expression is most severe with mortalities up to 70%, although there is variation in breed susceptibility and in the virulence of different strains.

Pathology

Clinical signs sheep

- may be somewhat protracted (into several weeks) and are exacerbated by exposure to sunlight
- fever: four to eight days after infection lasting for about a week

- face: swollen muzzle, hyperemic and congested tongue (deeply cyanotic tongue is rare) and mucous membranes; salivation; serous nasal discharge
- feet: erythema, congestion, and ultimately hemorrhage of coronary band due to acute coronitis; lameness.
- Necrotic lesions in oral cavity, hemorrhagic diarrhea, respiratory difficulties, loss of skeletal muscle mass

The clinical signs and gross pathology reflect the underlying pathogenetic process, namely vasculitis with endothelial cell injury.

Gross pathology

- Petechial or ecchymotic hemorrhages on oral mucosa, intestinal mucosa, base of pulmonary artery in tunica media
- Erosions with diphtheritic membranes on oral mucosa
- Edema and hemorrhages in subcutaneous tissues
- Pale foci of skeletal and myocardial muscle necrosis
- pulmonary edema and hydrothorax

Specimens for laboratory diagnosis
The best specimen for isolation attempts and PCR is clotted blood from febrile sheep – the virus is in the clot and is stable at refrigeration temperatures (do *not* freeze). The serum from these specimens can be decanted and, together with serum samples from apparently healthy herdmates, used for antibody determinations. In the case of dead animals, lymphoid tissues including spleen may also be submitted.

Differential diagnosis

- **FMD (healing lesions) –** no mortalities in adults, generally mild disease in sheep
- Peste des petits ruminants/ rinderpest
- Sheep pox – skin lesions
- Photosensitization
- Footrot
- Pneumonia
- Acute hemonchosis

Contagious bovine pleuropneumonia

Contagious bovine pleuropneumonia is caused by *Mycoplasma mycoides mycoides* and infects cattle, water buffalo and yaks. The disease is currently a serious problem in Africa.

CBPP is one of the three great cattle plagues of history and is the most devastating disease Australian livestock has seen. CBPP was introduced in 1858 in Melbourne, at a time when half the Australian population lived in Victoria. Millions of cattle suffered a slow and agonising death from

pleurisy and pneumonia and many more suffered prolonged convalescence and disability. By 1864 it had reached the Gulf of Carpentaria and became endemic in the great cattle herds of Northern Australia. Infection repeatedly returned to southern herds through cattle movements to major markets in the south.

In 1958 the Standing Committee on Agriculture initiated the process of developing a control and eradication program. Two key techniques that could be applied cheaply and simply to animals, many of which were wild and in inhospitable landscapes, underpinned the successful eradication of the disease. These were a diagnostic test that would reliably detect infected animals and a durable vaccine. The field program began in 1961, the last disease was found in 1967 and Australia was declared free in 1973.

CBPP organism is fragile outside the host and sensitive to dessication and to disinfectants. Infection of susceptible animals occurs following close contact between animals, probably via infected droplets. The incubation period varies from 3wks to 4mths and introduction of disease is followed by slow spread within the herd. Chronically infected animals that may have recovered from the acute disease or be subclinically infected are important vehicles for disease persistence in the herd and for spread to new areas.

Clinical signs

- Fever
- Rapid respiratory rate and dry cough
- Signs persist for 2 to 8 wks after which the animal dies (50%) or undergoes a slow recovery (50%).

Gross pathology

- Fibrinonecrotic lobar or bronchopneumonia with distension of interlobular septa by fibrinous exudate and alternation of normal lobules with red or grey consolidation or necrosis – "marbling"
- Serofibrinous pleuritis
- Formation of sequestra following vasculitis and thrombosis leading to infarction. Organisms remain viable in sequestra for years.
- Tendency to diaphragmatic lobe involvement

Specimens for laboratory diagnosis

- Lung, bronchial lymph node, pleural fluid (10ml) – fresh chilled and fixed in formalin
- Joint fluid (calves)– fresh chilled and fixed in formalin
- 20ml blood for serology from clinically affected animals plus healthy herdmates

Differential diagnosis

- Bacterial pneumonia
- Aspiration pneumonia

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