CHAPTER 5
SPECIFIC DISEASES OF SHEEP AND GOATS

Diseases caused by viruses

Rift valley fever (RVF)

RVF is an acute viral disease of sheep, cattle, goats and humans. It is manifested with hepatitis and high mortality in young lambs and calves, and abortion in adult animals. Rift valley fever resembles influenza in humans. The disease is of significant importance in Africa.

Transmission : Biting insects and mosquitoes. Possible direct contact via cornea. Human infection occur by handling diseased tissues, and strict precautions should be instituted to prevent infection with this virus, such as wearing goggles and gloves.

Antemortem findings :

Sheep

1. Incubation 12 – 48 hours in young animals
2. High morbidity and mortality in lambs and calves
3. Fever
4. Lambs refuse to eat, have abdominal pain and are recumbent.
5. Animals seek a shaded area because of photophobia (squinting and blinking)
6. Photosensitization characterized with a thickened head and ears.
7. Encrustation around the muzzle (Fig. 149)
8. Vomiting in adult animals
9. Congenital malformation of the brain and muscles
10. Abortion in ewes during the illness or convalescence

Cattle

1. Edematous unpigmented skin showing cracking and sloughing due to photosensitization
2. Salivation and inflammation in the mouth
3. Abdominal pain
4. Diarrhoea associated with haemorrhagic inflammation of stomachs and intestine
5. Lameness
6. Cessation of milk production
7. Abortion

Postmortem findings :

1. Cyanotic visible mucosae
2. Necrosis of the liver in lambs (liver may be mottled grey, or reddish-brown to bright yellow in colour)
3. Edematous and haemorrhagic gall bladder

4. Haemorrhage of the gastrointestinal tract, serosae, internal organs and lymph nodes
5. Partial erosions may be seen in the ileum, caecum and colon
6. Udder is purple but inflammation is not observed
7. Haemorrhages in the fetus and haemothorax (Fig. 150)

Judgement : Carcass of an animal showing clinical signs of Rift Valley fever is *condemned*. Reactors and recovered animals are *approved*. Affected parts of the carcass, liver and the blood must be condemned.

Differential diagnosis : Defect in porphyrin metabolism, fungal conditions, acute viremias/toxaemias including enterotoxaemia, bluetongue, bovine ephemeral fever, Wesselbron disease, rinderpest, heartwater, East Coast fever; abortions caused by Brucella, Vibrio, Trichomonas, Nairobi sheep disease and ovine enzootic abortion

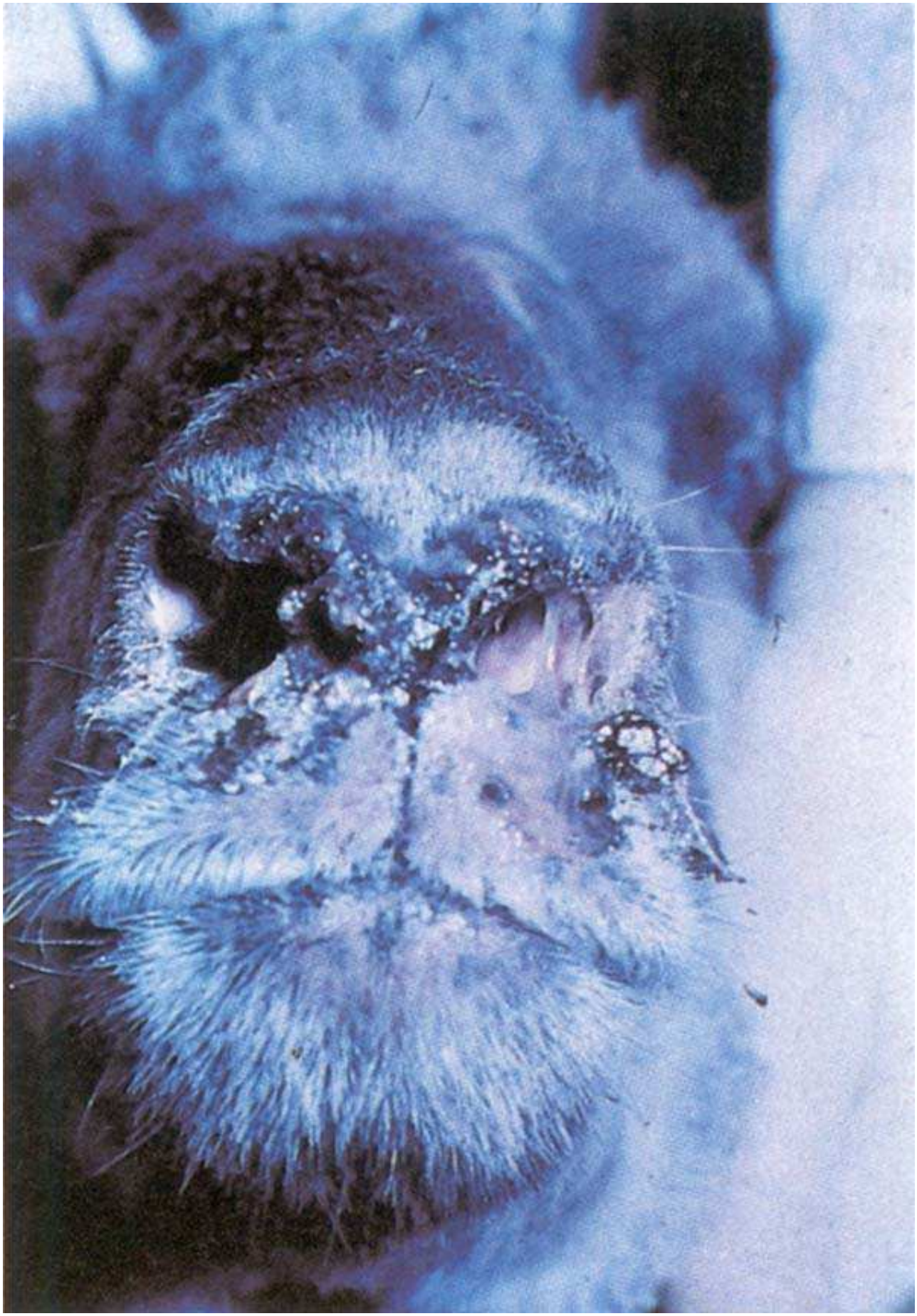


Fig. 149: RVE. Encrustation around the muzzle.

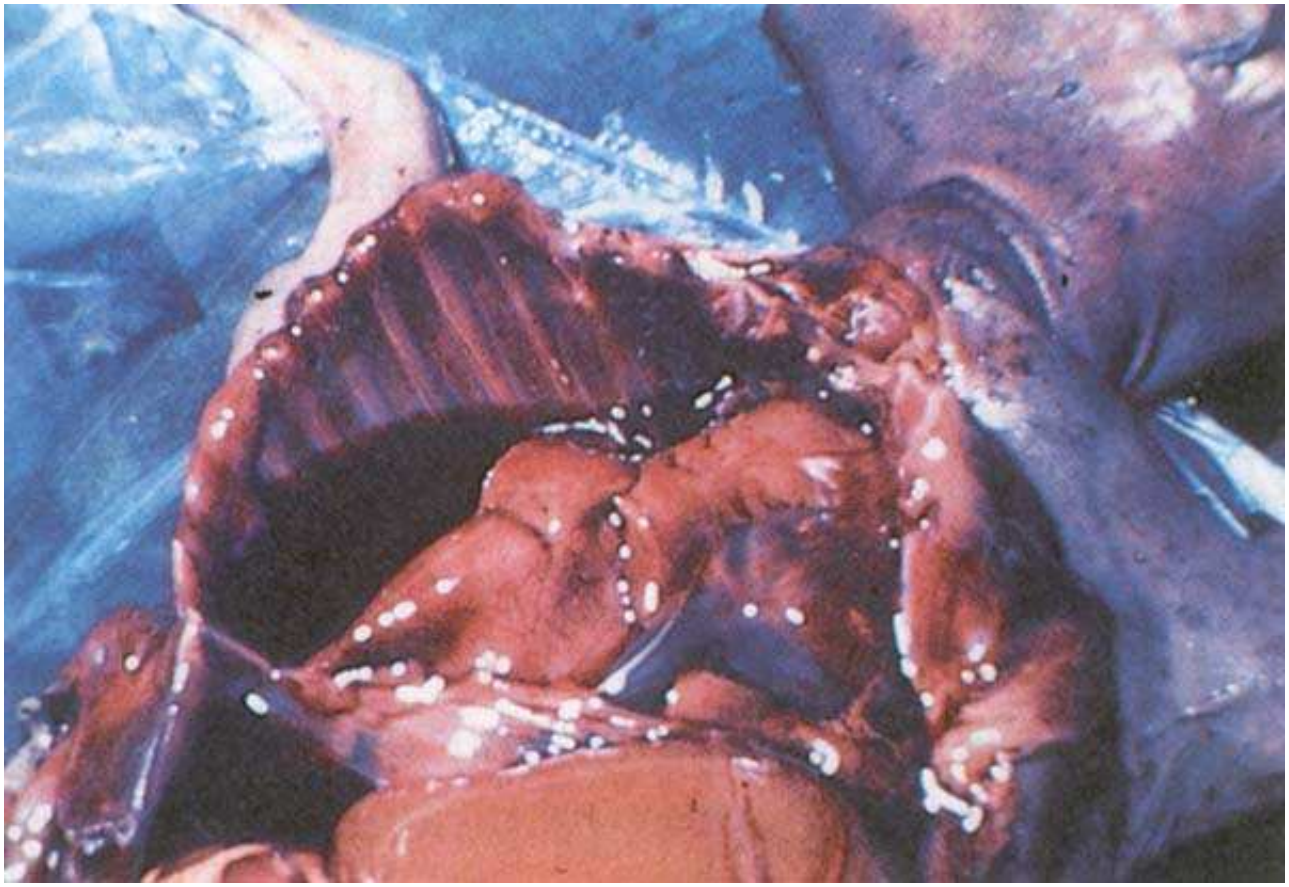


Fig. 150: RVF. Haemorrhages in the fetus and haemothorax.

Contagious ecthyma (contagious pustular dermatitis, orf)

A highly infectious pox virus disease of sheep and goats manifested by the occurrence of the pustular and scabby lesions on the lips, muzzle and udder.

Transmission : Direct contact between animals. Indirect contact with dry scabs in pens. The virus is resistant to drying and may be viable in scabs for months and years in empty feedlots and pens. Farm workers may disseminate the virus among animals of different pens with contaminated equipment, feed and farm vehicles.

Antemortem findings :

1. Incubation: 2 – 3 days
2. Pustular and scabby lesions on the muzzle (Fig. 151), lips and eyes.
3. Lesions on the udder and teats and the coronary band
4. The invasion of lesions by larvae of the screw worm fly and secondary bacterial ection with Fusobacterium necrophorum
5. Lambs and kids are unable to suckle or graze due to lip lesions.
6. Uncomplicated cases may heal within one month.
7. Emaciation
8. Pneumonia in feeder lambs

Postmortem findings :

1. Pustular and scabby lesions on the head, udder and feet
2. Ulcerative lesions in the nasal cavity and erosions in the mucosa of the oesophagus and upper respiratory tract.
3. Inflammation of the reticulum, omasum and intestine
4. Necrotic lesions in the lungs, pleura and liver

Judgement : The carcass is *condemned* if the disease is accompanied with inflammation of the stomachs and intestines, and with bronchopneumonia. Otherwise, it is *approved*.

Differential diagnosis : Bluetongue, sheep and goat pox, ulcerative dermatosis, cutaneous anthrax and vesicular diseases

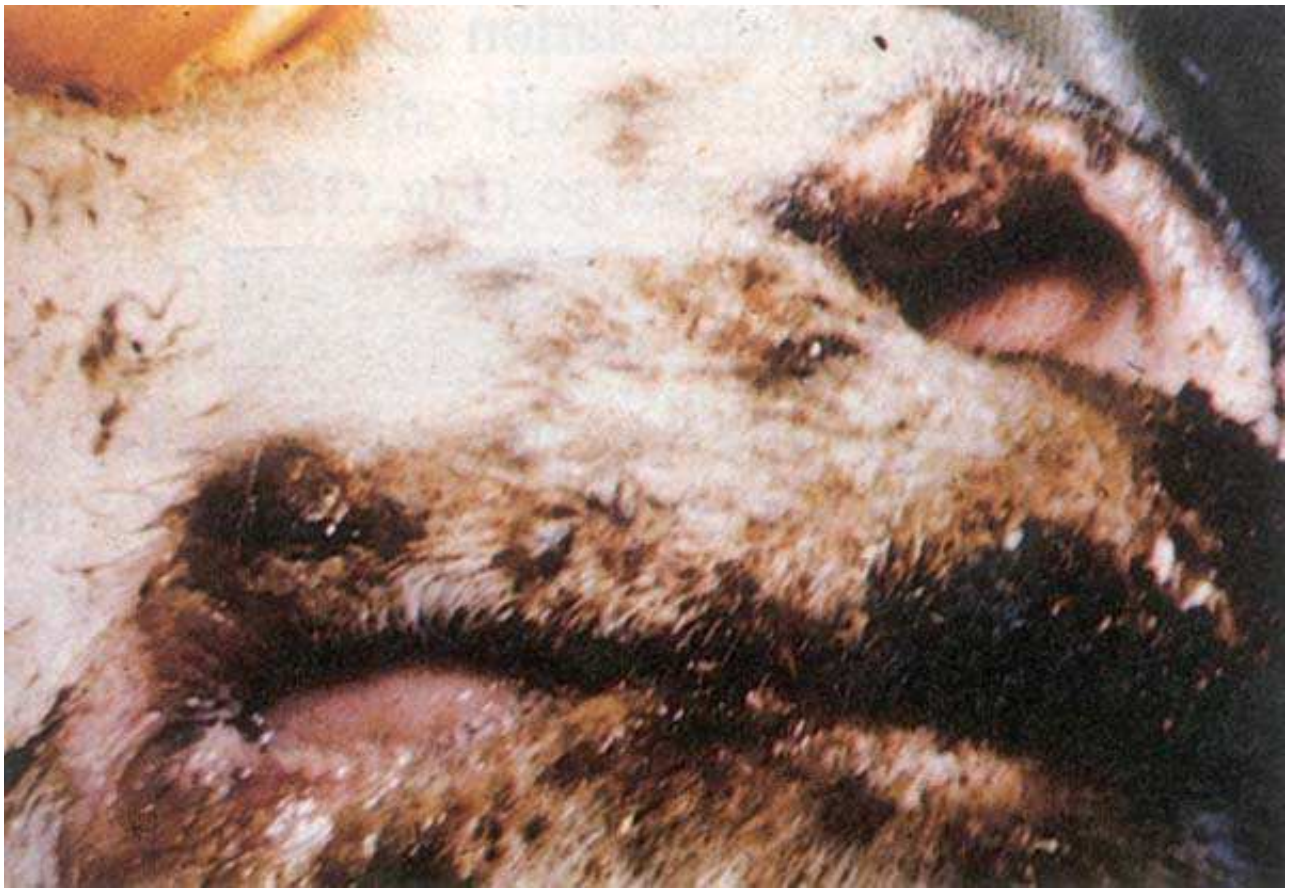


Fig. 151: Contagious ecthyma. Close up view of a proliferative muzzle lesion.

Bluetongue (BT, catarrhal fever of sheep, "soremuzzle disease")

Bluetongue is a highly contagious viral disease of sheep, manifested by fever, oral lesions, lameness and emaciation. The disease occurs mostly in the African region, but also in Asia and the Pacific and in the Western hemisphere, but can be well controlled by vaccination.

Transmission : Biting insects, especially Culicoides gnats and mosquitoes. Vertical transmission occurs in utero. Semen of infected bulls and mechanical transfer of infected blood by needles.

Antemortem findings :

In sheep:

1. Incubation 6 – 8 days
2. Fever
3. Difficult breathing
4. Excessive salivation
5. Loss of appetite, weakness and emaciation
6. Reluctance to move
7. Mucopurulent to bloody nasal discharge (Fig. 152)
8. Edema of the face, lips and jaw
9. Cyanosis of the tongue and mucous membranes (bluetongue) with erosion and sloughing of the oral mucosa (Fig. 153)

10. Lameness associated with sore feet caused by the inflammation of the coronary band (Fig. 154)
11. Abortion and deformed lambs

In cattle, the disease resembles the infection in sheep and the clinical signs are from unapparent to mild.

Postmortem findings :

1. Vesicles or ulcers in the mouth
2. Generalized edema and haemorrhage of subcutaneous tissue and musculature
3. Excessive mucus in the trachea
4. Congestion of lungs
5. Generalized lymphadenitis
6. Enlarged spleen
7. Necrosis of the heart and skeletal muscles

Judgement : Carcass of an animal affected with bluetongue is *condemned* when the clinical signs of an acute disease are associated with generalized postmortem lesions. The reactor animals are *approved*.

Differential diagnosis :

Sheep: Photosensitization, contagious ecthyma, sheep pox, polyarthritis, footrot, foot abscesses, laminitis, vesicular stomatitis, white muscle disease, muscular dystrophy in lambs, lungworm infestation and pneumonia.

Bovine: Bovine viral diarrhoea, malignant catarrhal fever, infectious bovine rhinotracheitis, stomatitis, laminitis and Ibaraki disease, FMD.

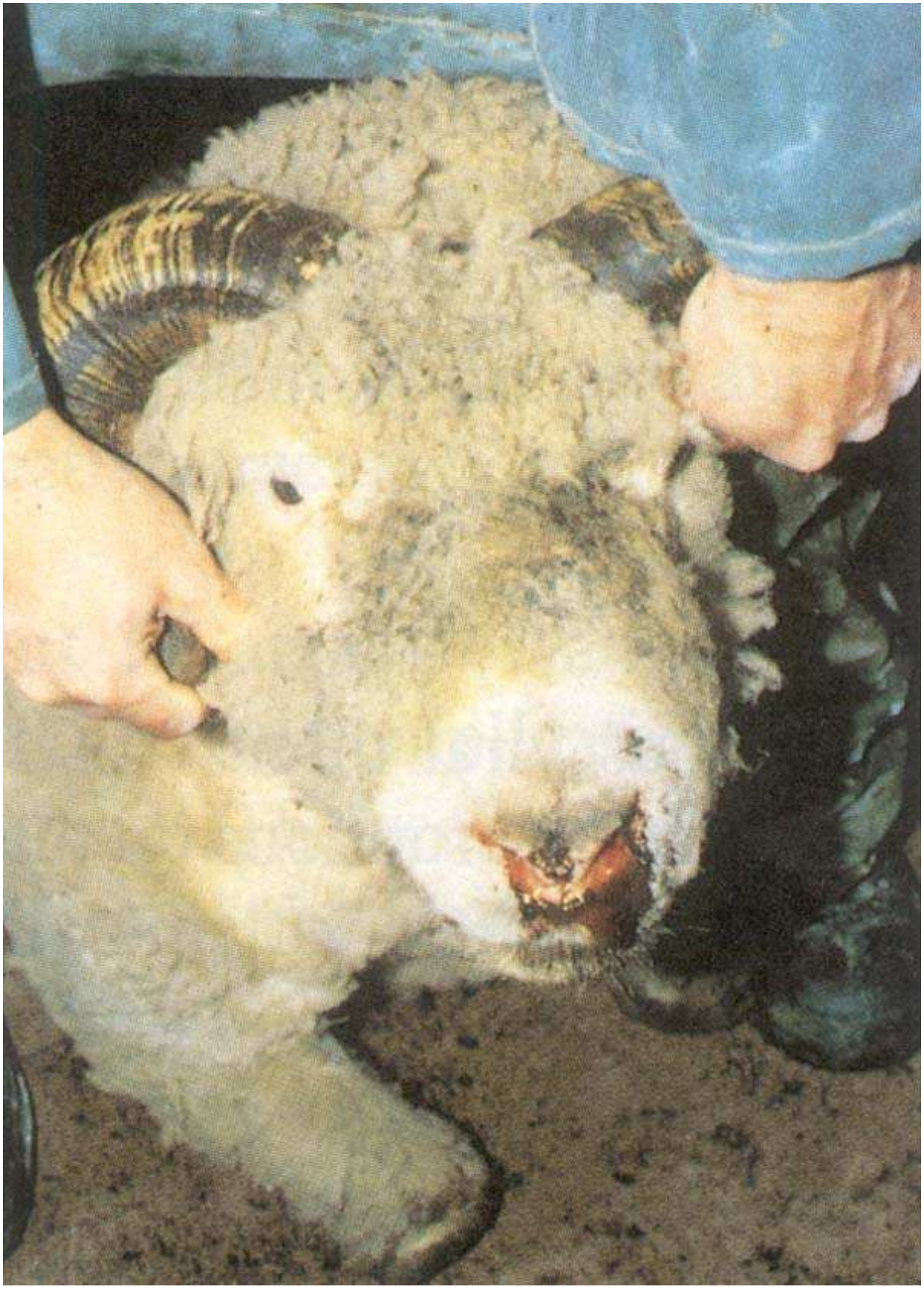


Fig. 152 : Blue tongue. Mucopurulent to bloody nasal discharge.

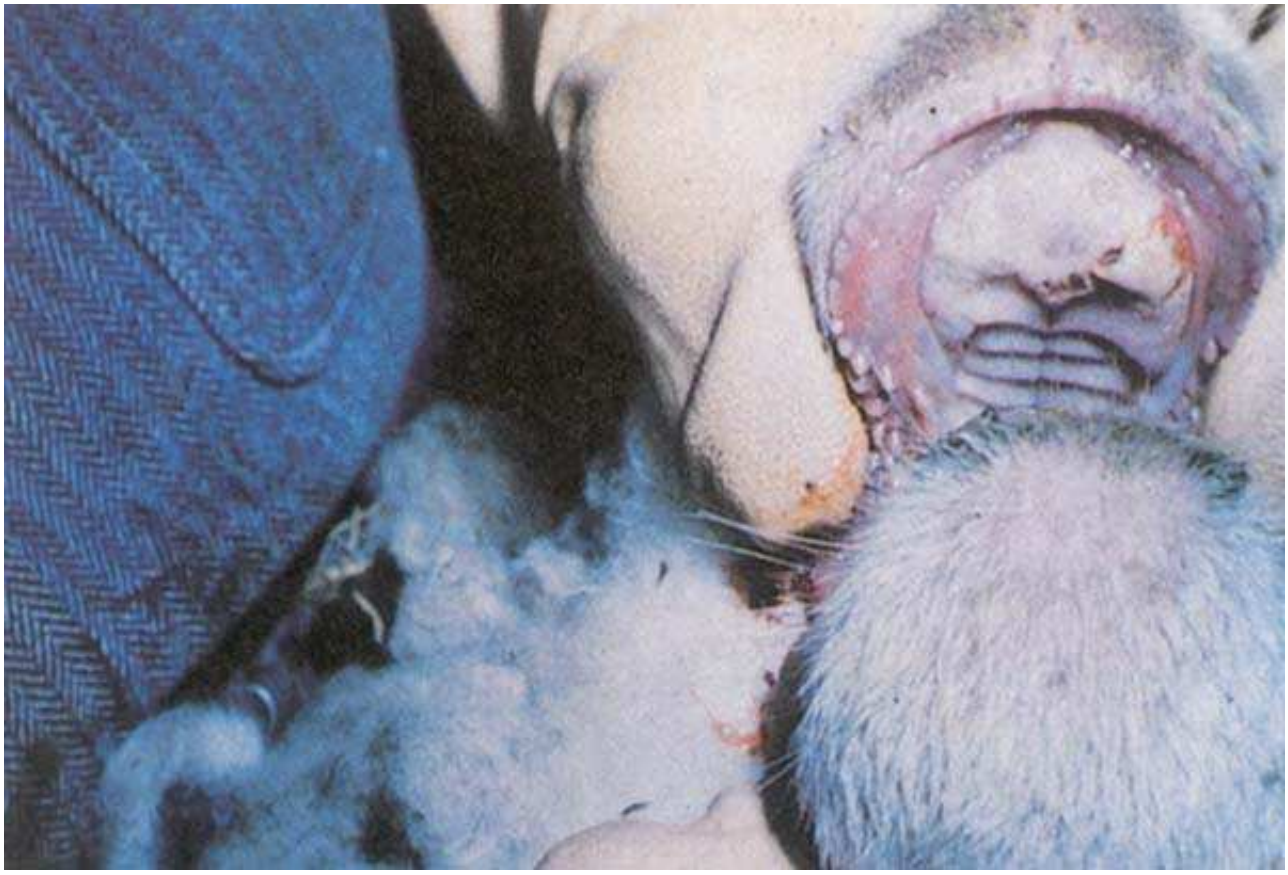


Fig. 153 : Blue tongue. Intense congestion and swelling of lips and gums and sloughing of the dental pad mucosa.

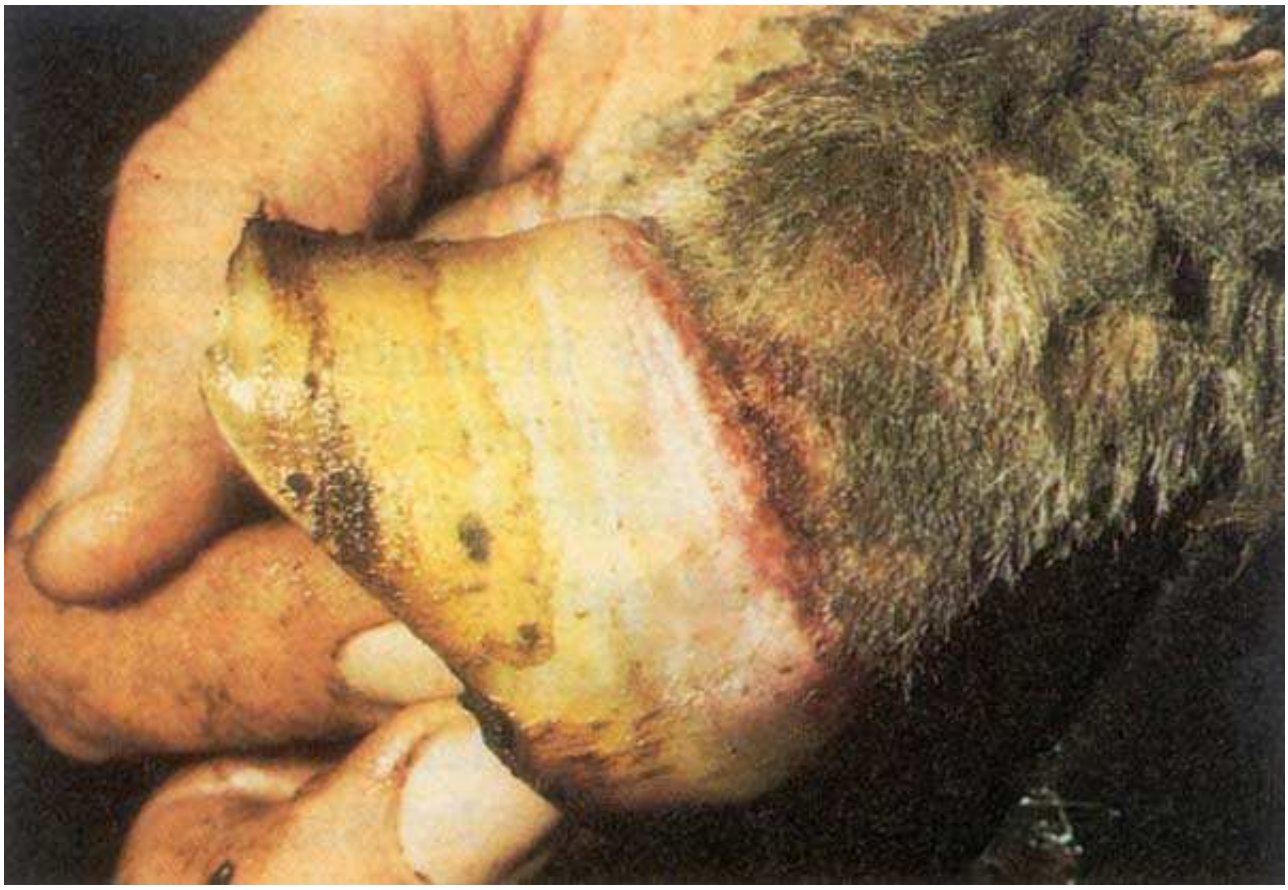


Fig. 154 : Blue tongue. Close-up view of a lesion on the coronary band of a sheep.

Sheep and goat pox

Sheep and goat pox is a contagious viral disease of sheep and goats manifested by papular and pustular eruptions on the skin and in generalized conditions with haemorrhagic inflammation of the respiratory tract.

Transmission : Direct contact with infected animals, aerosols of nasal secretions and saliva and dried scabs. Indirectly by fomites and transportation vehicles.

Antemortem findings :

1. Incubation 6 – 8 days
2. Fever
3. Laboured breathing
4. Depression
5. Lacrimation and salivation
6. Lesions on the muzzle and lips (Fig. 155)
7. Skin lesions may vary from macules, papules, vesicles, pustules to pocks and scabs.
8. Necrosis and coalescing of the lesions and loss of wool (Fig. 156)
9. Clinical signs of goat pox are less severe than in sheep pox. The benign form of sheep pox is commonly found in adult sheep and the malignant form in lambs.

Postmortem findings :

1. Reddish to whitish firm nodules in the mucosa of the pharynx and trachea
2. Reddish to whitish nodules in the lungs (Fig. 157). Rarely pneumonia
3. In malignant form: inflammation of the respiratory and digestive tract

Judgement : Carcass of an animal showing the clinical disease without secondary complications is conditionally *approved* pending heat treatment. The recovered animals are *approved*. The carcass is *condemned* if the acute febrile or pustular stage of the disease is associated with secondary bacterial infections or if the carcass is inadequately bled. If bacteriological examination showed negative results, this carcass may be *conditionally approved* pending heat treatment.

Differential diagnosis : Contagious ecthyma, scabies, eczema. ulcerative dermatitis and peste des petits ruminants.

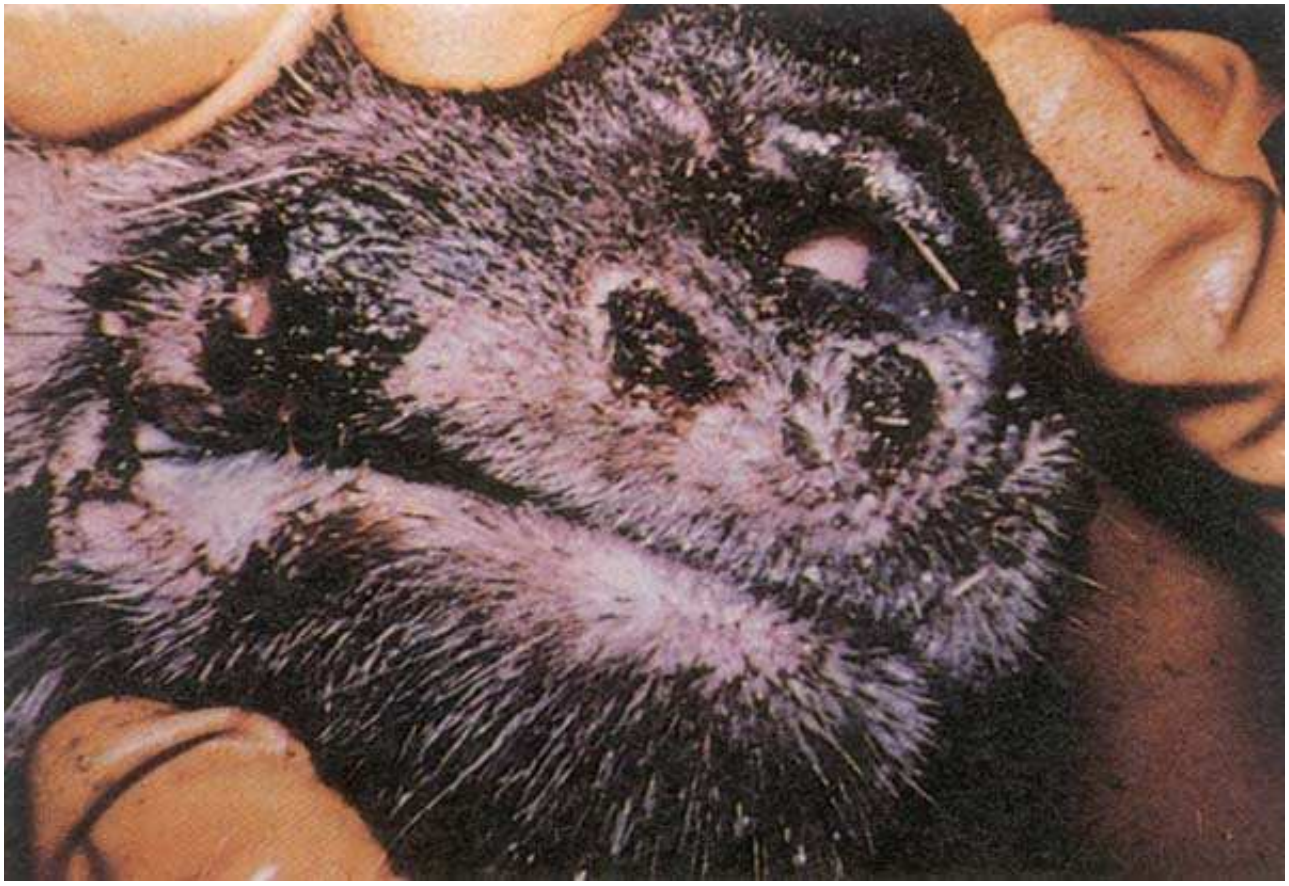


Fig. 155: Sheep pox. Lesions on the muzzle and lips.

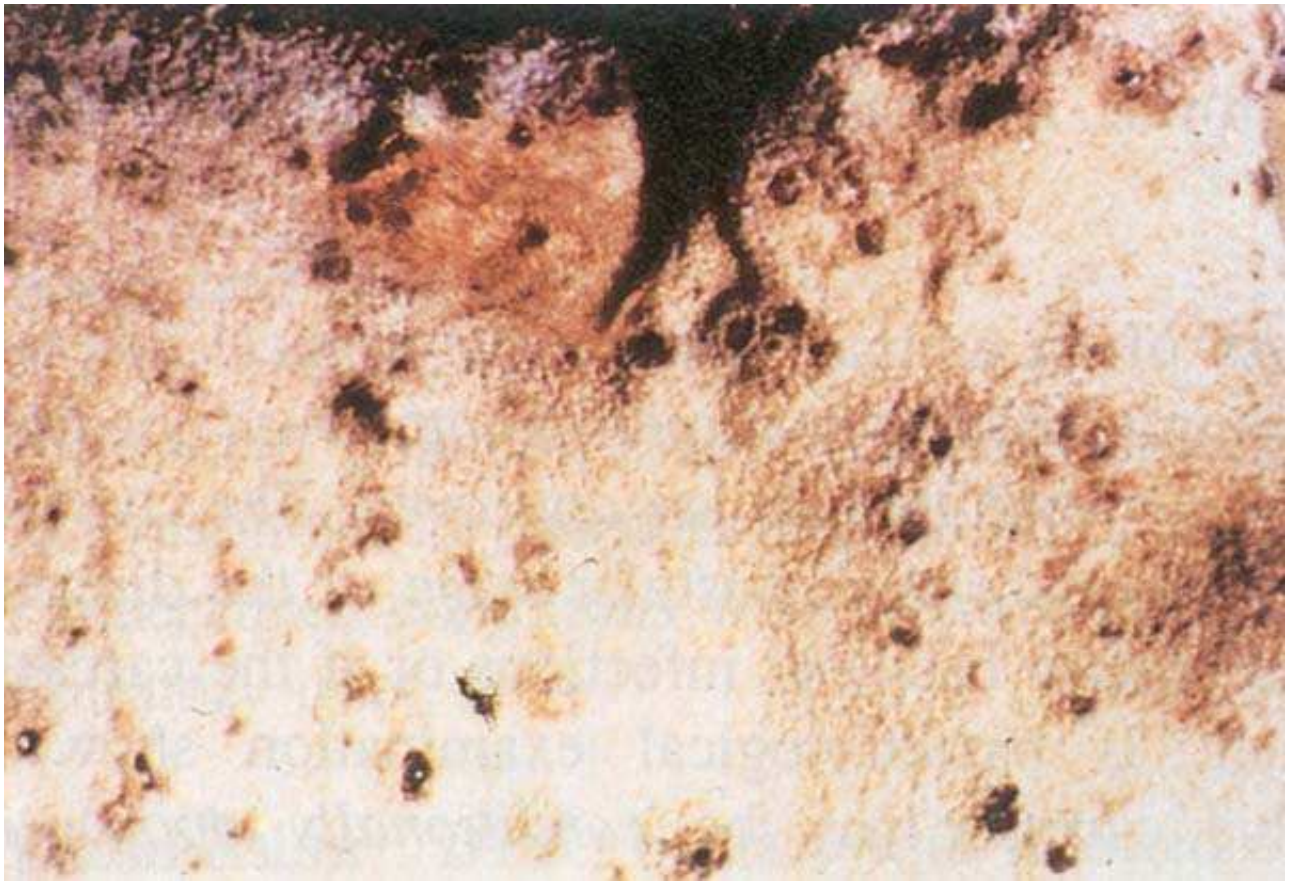


Fig. 156: Sheep pox. Necrosis and coalescing of the lesions and loss of wool.

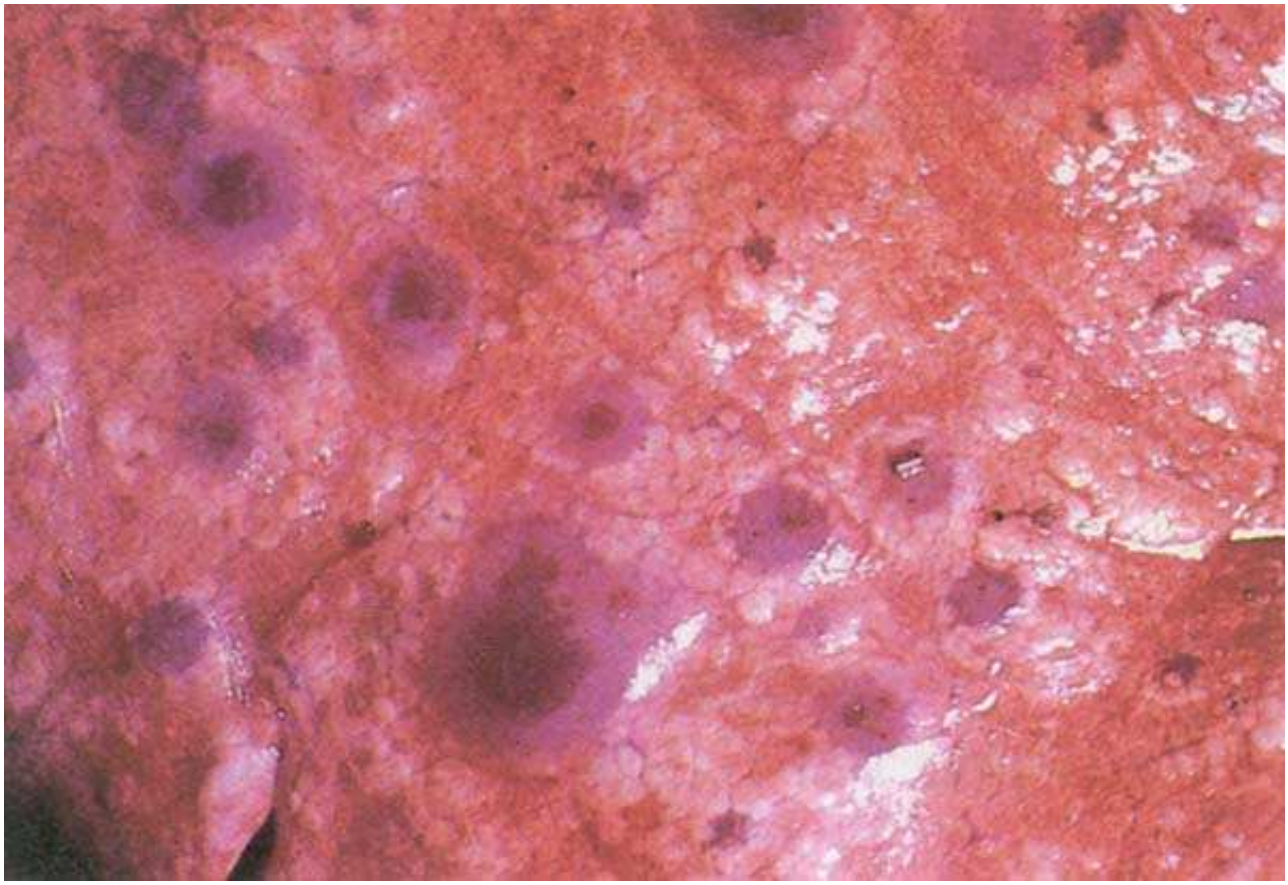


Fig. 157: Sheep pox. Reddish to whitish nodules in the lungs.

Scrapie

Scrapie is a chronic disease of the central nervous system in sheep and occasionally goats characterized by itching, nervous signs and a long incubation period. It is caused by a viral agent called "*viroid*" or "*prion*", which has some of the characteristics of the virus, a "slow" virus like BSE and Maedi.

Transmission : Most likely, the organism enters through breaks in the skin and mucous membranes of susceptible sheep. The agent is present in the lymph nodes, spleen, spinal cord and brain of infected sheep. It is transmitted from sick animals to healthy animals through pasture, where it may be infective for over 3 years. Vertical transmission from the dam and possibly the sire in sheep may also occur. The disease may be transmitted by inoculation of infective material. The agent is resistant to rapid freezing, thawing, boiling for 30 minutes and even to a 20 % formalin solution. At temperatures of 0 – 4°C, the prion is still active after two years. Oscillation of the temperature from 37– 70°C does not affect its infectivity. At temperatures of 94–98°C, the prion is still resistant for 24 hours.

Antemortem findings :

1. Dry wool and rough skin
2. Loss of wool from the head down over the side of the face, rump, thigh, tail base and abdomen
3. Changes of behaviour. Charging of fences, dogs etc.
4. Biting of legs, flanks and belly because of severe itchiness (pruritus)
5. Smacking and rarely curling of the lips and wagging of the tail during rubbing of the skin over the back and sacrum
6. Grinding the teeth
7. Twitching of muscles, excitability and wild expression of the eyes
8. Restless animal, continuously laying down and getting up
9. Incoordinated gait, tendency to run and fall down.

10. Convulsions

Postmortem findings :

1. No gross lesions observed
2. Microscopy reveals the presence of large vacuoles in the cytoplasm of neurons; this is considered a diagnostic lesion.

Judgement : Carcass and viscera affected with the clinical disease are *condemned*. Carcass of contact animals, offspring and ancestors may have a limited distribution or it may be condemned if economically feasible.

Differential diagnosis : Pseudorabies, scabies, thallium poisoning, cobalt deficiency, louping ill, pregnancy toxaemia, external parasitism and photosensitive dermatitis

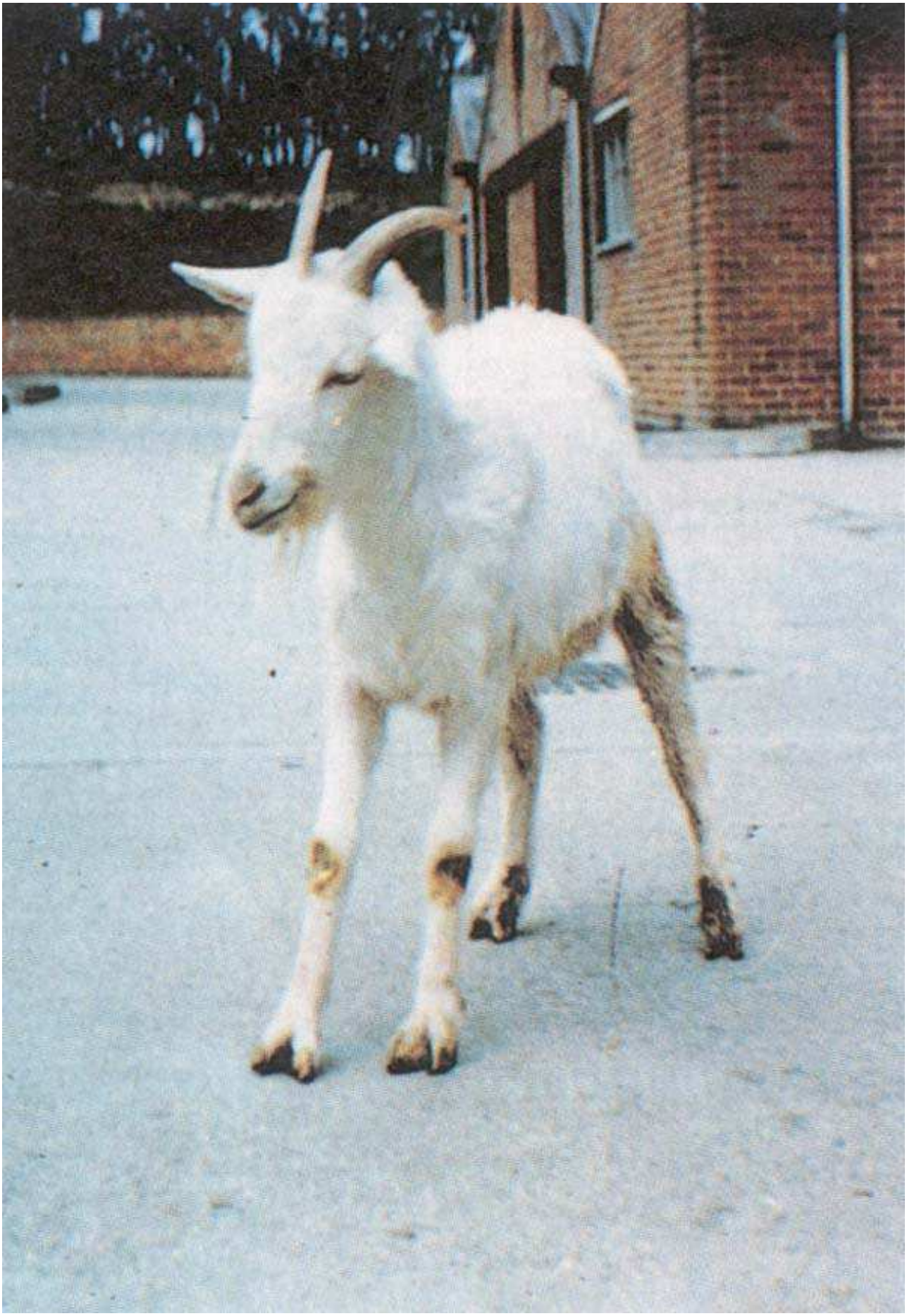


Fig. 158: Scrapie. Incoordinated gait, twitching of muscles and wild expression in the eyes.

Pulmonary adenomatosis (Jaagsiekte, Driving sickness)

Pulmonary adenomatosis is a chronic progressive pneumonia of sheep with the development of

a primary lung neoplasm. This neoplasm is carcinomatous and infrequently metastatic to regional lymph nodes. A *retrovirus* causes the disease and a herpesvirus acts in a secondary role. This is a disease of old ewes, more then 4 years of age. Lambs and yearling are rarely affected.

Transmission : The disease is experimentally transmitted by inhalation of infected droplets by sheep that are kept in close contact. Vertical transmission from pregnant ewes to fetus has also been demonstrated.

Antemortem findings :

1. Incubation 2 months to 2 years
2. Difficult breathing and lacrimation
3. Loss of weight and emaciation
4. When the rear of a sheep is lifted, excess fluid will run from the nose (wheel barrow test).
5. Emaciation and lacrimation

Postmortem findings :

1. The lungs are increased in size and weight (as much as triple their normal size) and do not collapse when the thoracic cavity is opened (Fig. 159).
2. Bluish grey consolidation of the ventral part of the lung
3. Secondary bacterial infections in the lungs
4. Focal lung lesions are interspersed with areas of emphysema.
5. Metastasis of the neoplasm into the bronchial and mediastinal lymph nodes may occur infrequently.

Judgement : Carcass judgement depends on the extent of lung involvement, condition of the carcass and secondary bacterial infection. Extensive lung lesions with metastasis and loss of musculature *would necessitate the condemnation* of the carcass.

Differential diagnosis : Verminous pneumonia, Maedi/Visna, caseous lymphadenitis and other debilitating diseases

Fig. 159: Pulmonary adenomatosis. Lung lesions showing light grey, enlarged apical and cardiac lobes consisting of numerous greyish coalescing nodules (1 mm to 1 cm in diameter).

Ovine progressive interstitial pneumonia (Maedi, Maedi-visna)

Maedi/visna is a highly fatal viral disease of sheep and goats caused by a *lentivirus*.

Transmission : Through colostrum to newborn lambs and less often by contact with respiratory route.

Antemortem findings :

1. Listlessness
2. Difficult breathing and frequent coughing
3. Nasal discharge
4. Emaciation
5. Lameness
6. In chronic cases, anaemia and secondary bacterial infections

Postmortem findings :

1. Enlarged grey-yellow non collapsible lungs of rubbery consistency (Fig. 160)
2. Cross section of lung parenchyma showing a meaty appearance
3. Enlarged and firm mediastinal lymph nodes

Judgement : Carcass in good flesh with slight to moderate pulmonary involvement is approved. An emaciated carcass with extensive pulmonary lesions or secondary bacterial infection is condemned.

Differential diagnosis : Parasitic pneumonia, pulmonary adenomatosis (Jaagsiekte) and pseudoglanders (Melioidosis)

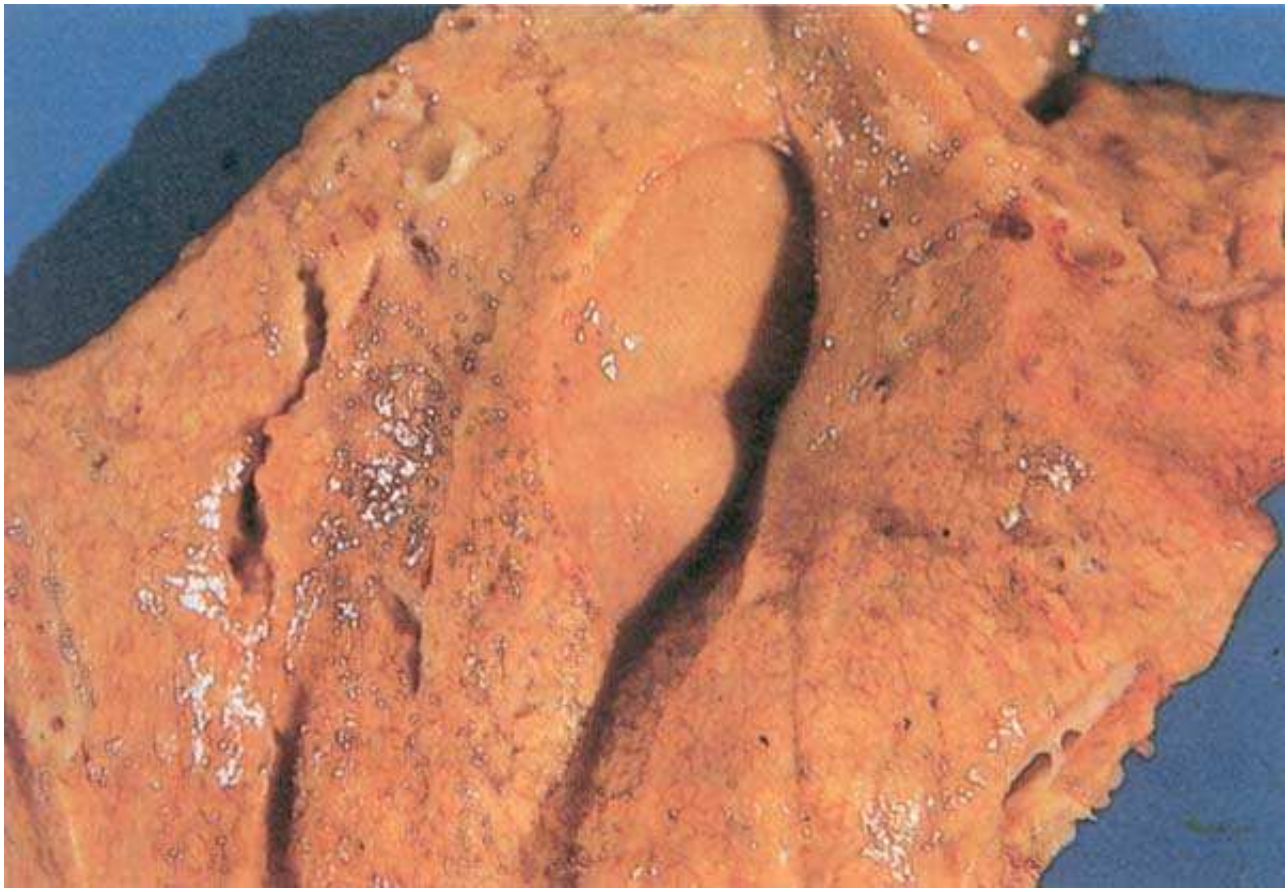


Fig. 160: Ovine progressive interstitial pneumonia. Cross section of the lung parenchyma. The lungs are enlarged, non collapsible and have a meaty appearance.

Nairobi sheep disease

Nairobi sheep disease is a non contagious, *tick borne viral disease* in sheep manifested by acute haemorrhagic inflammation of the stomach and intestine and by respiratory signs.

Transmission : Adult forms of a tick Rhipicephalus appendiculatus which attach themselves inside the ear of an animal. Unfed adult ticks are infective for one year. Faeces does not contain the virus.

Antemortem findings :

1. Incubation 4 – 15 days
2. Fever; during fever the blood, urine and tissue are infective
3. Rapid painful breathing
4. Dullness and depression
5. Mucopurulent nasal discharge
6. Pain and grunting with defecation
7. Acute haemorrhagic gastroenteritis
8. Bright to dark green faeces (is important in the differential diagnosis.)
9. Abortion in pregnant ewes
10. Swollen vulva and external genitalia
11. Collapse and death

Postmortem findings :

1. Excess fluid in the pericardium
2. Ecchymotic and petechial haemorrhage in the heart muscle
3. Acute haemorrhagic inflammation of the stomachs (Fig. 161) and intestine
4. Distended gall bladder contains thick syrupy bile
5. Enlarged and edematous lymph nodes
6. Hyperaemic genital tract

Judgement : Carcass of an animal affected with the acute disease accompanied with fever and acute gastrointestinal lesions is *condemned*. Carcass of recovered animals and of animals with non systemic or generalized lesions is *approved*. The affected organs are *condemned*.

Differential diagnosis : Rift Valley fever in sheep. Diarrhoea in RVF may show blood tinged watery faeces, but is not green in colour as in NSD. In rinderpest ulcerative lesions are noted with bloody (and not green) faeces. Heartwater, anthrax and plant poisoning should also be considered in differential diagnosis.

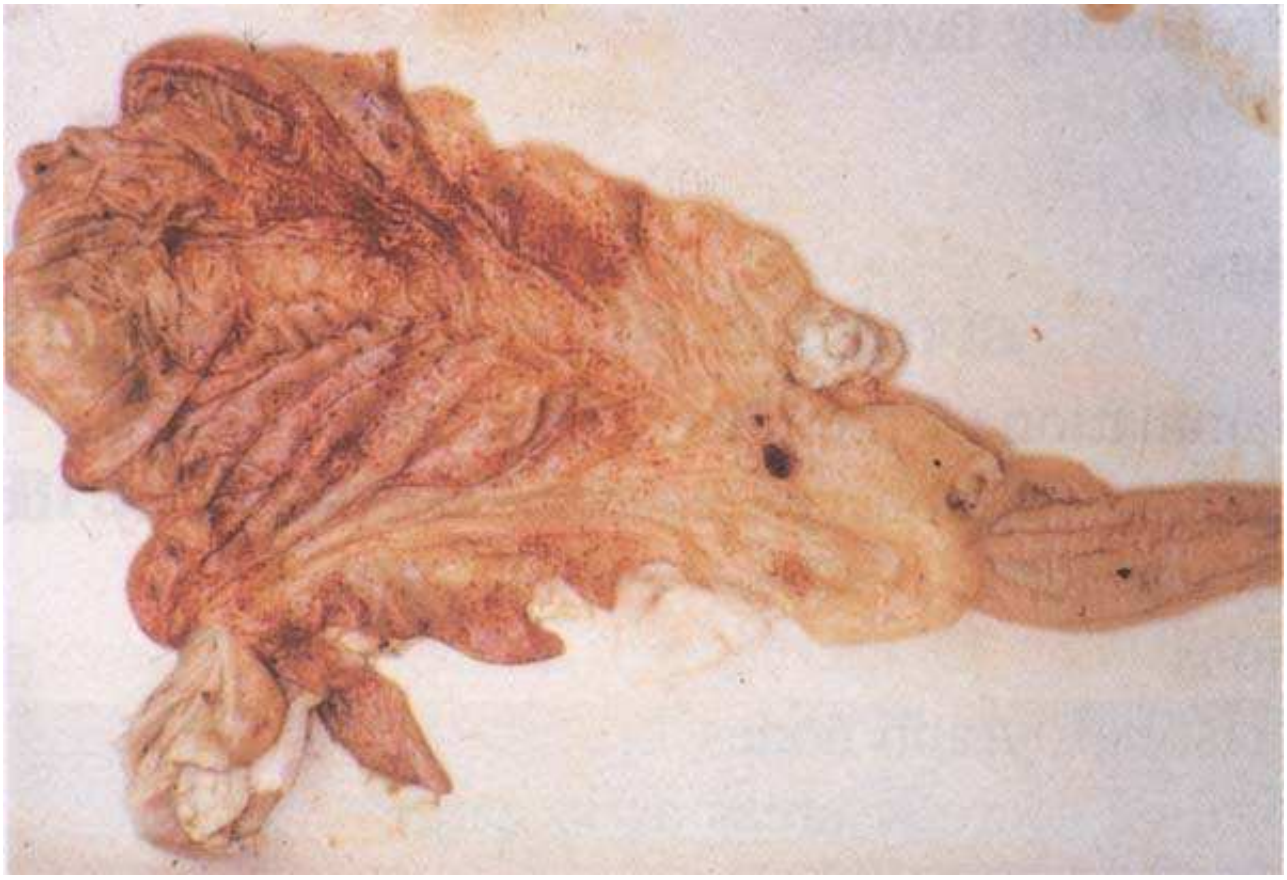


Fig. 161: Nairobi sheep disease. Acute haemorrhagic inflammation of the stomachs.

Diseases caused by Mycoplasma spp.

Contagious caprine pleuropneumonia

Contagious caprine pleuropneumonia is a contagious disease of goats caused by *Mycoplasma mycoides subs. capri* (mycoplasma biotype F 38). The disease resembles bovine pleuropneumonia, however it is not transmissible to cattle.

Transmission : By inhalation; carrier or infected animals may also bring the infection into the flock.

Antemortem findings :

1. Incubation: 6 – 10 days
2. Extremely infective with morbidity of 100 %
3. Acute disease with mortality of 60 – 70 %
4. Fever
5. Cough
6. Tongue sticking out and frothy salivation
7. Mouth breathing in terminal stage
8. Lagging and frequently laying
9. Death in few days

Postmortem findings :

1. Fibrinous inflammation of the pleura (Fig. 162)
2. Slight interlobular pulmonary reaction. Lesion may be present in only one lung.
3. Pleural adhesions
4. Enlarged mediastinal lymph nodes
5. No sequestration of necrotic areas as in cattle

Judgement : Carcass of an animal affected with contagious caprine pleuropneumonia which shows no systemic involvement is *approved*. The affected organs are *condemned*. The septicemic form of the disease calls for carcass *condemnation*.

Differential diagnosis : Foot and mouth disease, vesicular stomatitis, shipping fever (pasteurellosis), East Coast fever, foreign body pneumonia, infectious bovine rhinotracheitis, tuberculosis, chlamidial infections and lungworms

Fig. 162: Contagious caprine pleuropneumoni a. Fibrinous inflammation of the pleura.

Diseases caused by bacteria

Brucellosis (see Chapter 3)

Fig. 163 A: Brucellosis. Edema and swelling of scrotum.

Black quarter (Black leg)

Black quarter is an acute infectious disease of sheep and cattle manifested by inflammation of the muscles, toxaemia and high mortality. It is caused by Clostridium chauvoei.

Transmission : Contaminated soil. The organisms enter into the digestive tract with feed and through cuts which occur during the shearing, docking, and castration, and via naval infection during birth. Infection of the vulva and vagina of the ewes during lambing may cause serious outbreak of the disease. Black leg is worldwide in distribution. Well nourished and grass fed animals are more often affected.

Antemortem findings :

1. Fever
2. Loss of appetite
3. Depression
4. Stiff gait and reluctance to move due to lameness
5. Subcutaneous edema is not common.
6. Gaseous crepitation occurs before death.
7. Head lesions associated with edema and nose bleeding

Postmortem findings :

1. Subcutaneous edema particularly noted around head.
2. Affected muscle is dark brown, dry and sponge like or moist. A pungent odour is noted. Less gas is formed than in cattle.
3. Tongue, heart muscle and/or diaphragm may be blackish red. Marked abdominal extension if fetus is infected.
4. Genital tract lesions in the walls of the vagina and occasionally uterus
5. Serosanguineous and haemorrhagic fluid in body cavities and pericardial sac

6. Edema of lungs

Judgement : *Total condemnation* of the carcass and viscera of an animal affected with black leg. It is prohibited to slaughter and dress the animal diagnosed with this disease on antemortem examination.

Differential diagnosis : Other acute Clostridial infections, lightning strike, anthrax, bacillary haemoglobinuria, malignant edema, extensive haemorrhage, acute lead poisoning and lactation tetany

Enterotoxaemia (Pulpy kidney)

This disease is a fatal toxaemia in lambs, sheep, goats, calves and seldom in adult cattle. The disease is manifested by diarrhoea, involuntary contraction of muscles, paralysis and sudden death. It occurs after a sudden change to a better, more nutritious diet. The disease is often noted in sheep that have been fed heavy grain, and in animals which graze on lush growing pastures. *Clostridium perfringens* multiplies in abomasum and intestine and produces toxin which paralyses the vital centres in brain and damages endothelium of blood vessels. The disease occurs extensively in particular in Southern Africa but is well controlled by vaccination.

Antemortem findings :

1. Short course of the illness (2 – 12 hours) in lambs and longer course (24 hours) in sheep
2. Animal found dead without previous sign of the disease
3. Dullness and depression
4. Rapid shallow respiration
5. Loss of appetite and frothing
6. Muscular contractions
7. Green pasty diarrhoea
8. Grinding of the teeth and muscular tremor
9. Logging behind the flock
10. Staggering and recumbency

Postmortem findings :

1. No lesions in peracute cases
2. Large amount of clear, straw coloured pericardial fluid
3. Petechial haemorrhages of the heart muscle
4. Congestion of the abomasal and intestinal mucosa (Fig. 163) and liver
5. Soft pulpy kidneys a few hours after death is characteristic of this disease
6. Overload of the rumen and abomasum with concentrate
7. Haemorrhage and edema in sheep brain
8. Rapid decomposition of the carcass

Judgement : Carcass of an animal affected with enterotoxaemia is *condemned*.

Differential diagnosis : Sudden death in lambs: pasteurellosis, hypocalcemia and hypomagnesemia (reduced blood calcium and magnesium), polioencephalomalatia (less acute form), acute rumen impaction (no convulsions are present and the course is longer) and other septicemias. Adult sheep: rabies, acute lead poisoning, pregnancy toxaemia and louping-ill

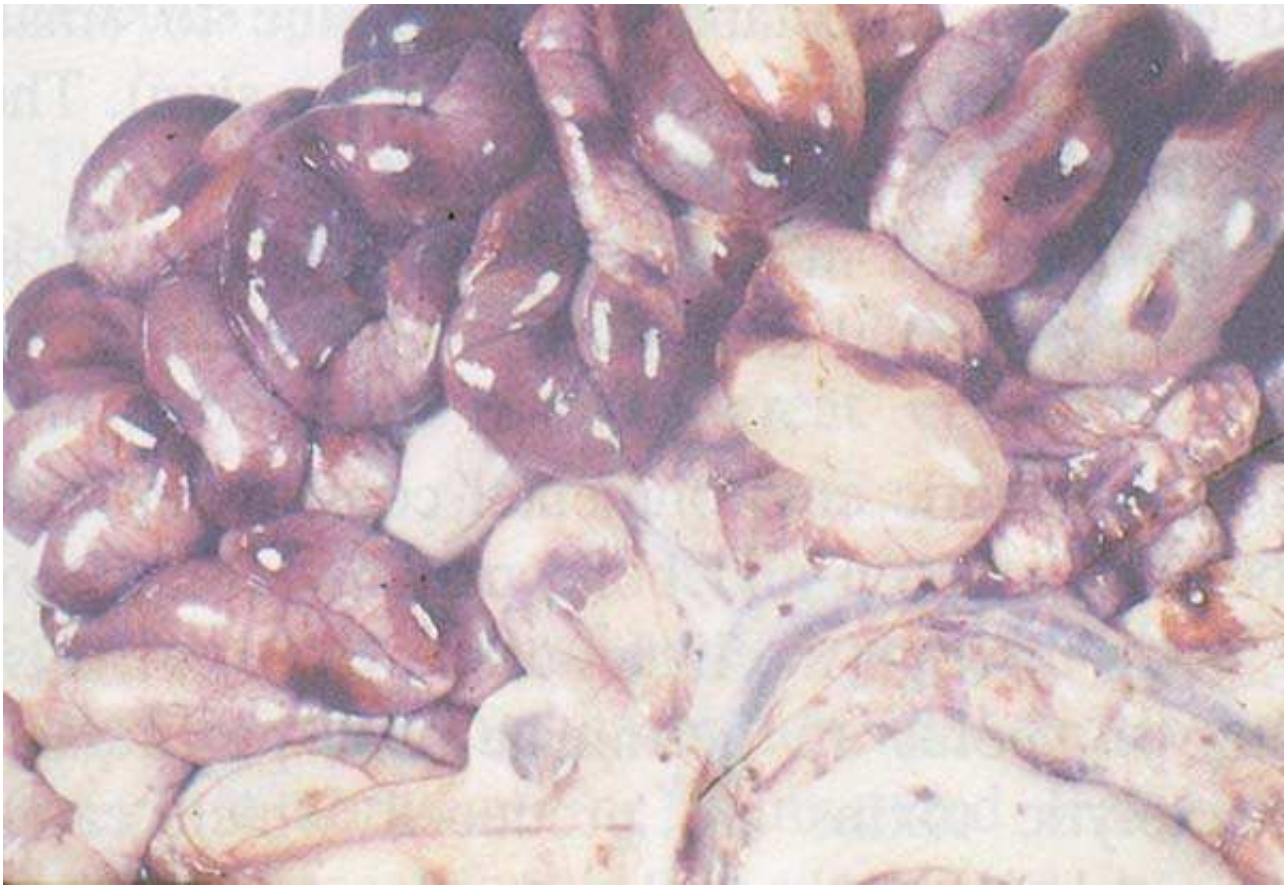


Fig. 163: Enterotoxaemia (pulpy kidney). Dilated intestine showing a patchy congestion. Note also congestion of mesenteric lymph nodes.

Infectious necrotic hepatitis (Black disease)

Black disease causes acute necrotic hepatitis in sheep and cattle and rarely in pigs. It is caused by bacterium *Clostridium novyi* in association with immature fluke invasion of the liver.

Antemortem findings :

1. Fever (40 – 42°C)
2. Rapid and shallow respiration
3. Sheep may be found dead without clinical signs.
4. Sick animal usually segregates from the rest of the flock.
5. Depression and incoordination
6. Recumbency

Postmortem findings :

1. Dark brown swollen liver showing necrotic areas surrounded by a zone of hyperaemia (Fig. 164)
2. Evidence of recent infestation of liver flukes
3. Darkened and cyanotic subcutaneous tissue due to small blood vessel engorgement (dark appearance of the skin). The name “Black disease” was derived from this.
4. Clear straw coloured fluid in the abdominal and thoracic cavities and in the pericardial sac

Clostridium novyi is an endemic environmental contaminant and remains latent in the liver, spleen and bone marrow. Immature liver flukes, by migrating through the liver, cause liver necrosis. This initiates Cl. novyi spores to germinate and proliferate. Necrotizing and haemolytic toxins are produced which cause generalized toxaemia and haemolysis of the blood.

Judgement : Carcass and viscera affected with black disease are *condemned*.

Differential diagnosis : Fascioliasis, enterotoxaemia, blackleg, malignant edema anthrax

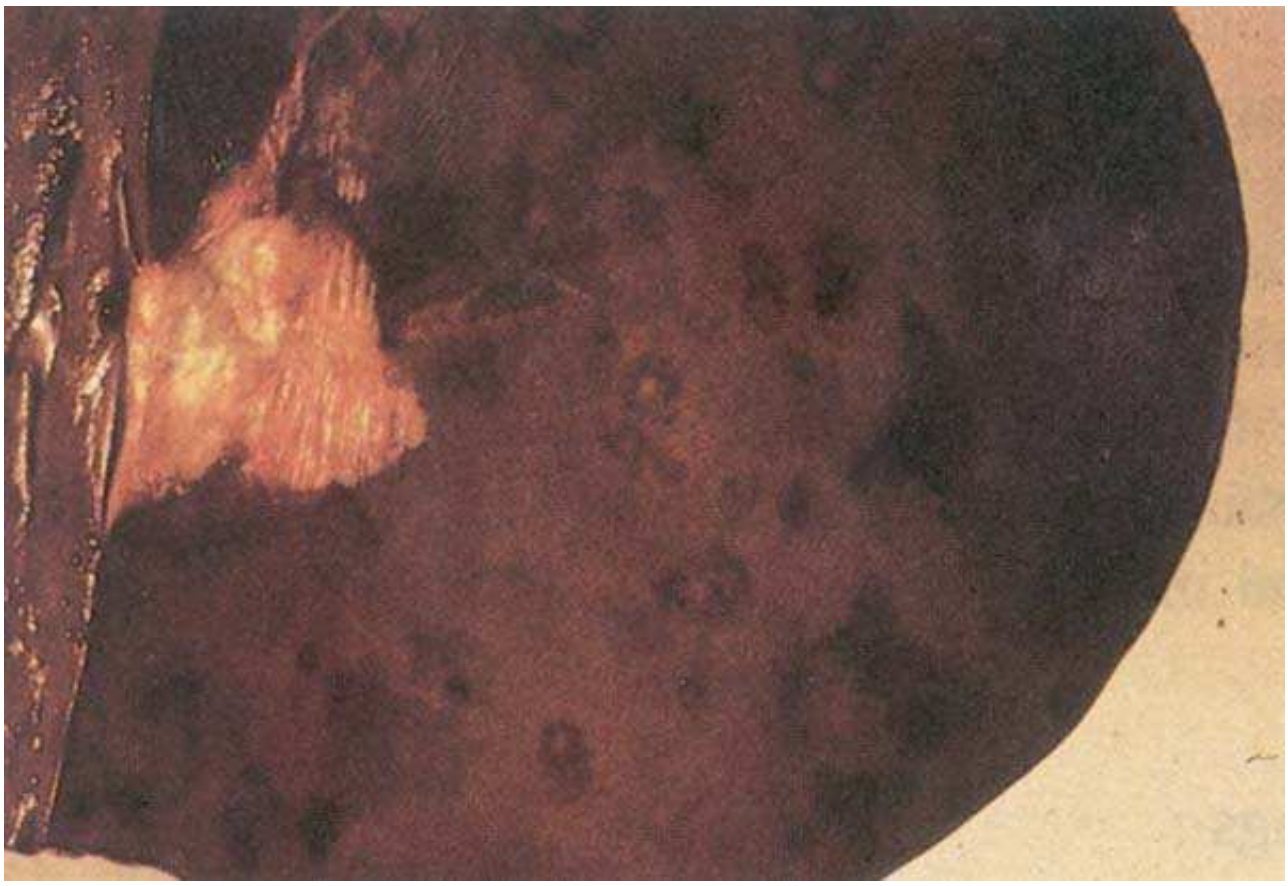


Fig. 164: Black disease. Dark brown swollen liver showing necrotic areas (1–2 cm) in diameter surrounded by a zone of hyperaemia.

Caseous lymphadenitis

This is a chronic disease of sheep and goats manifested by abscesses in the lymph nodes. It is caused by *Corynebacterium pseudotuberculosis*. Caseous lymphadenitis has a worldwide distribution and causes great economic losses to the sheep industry.

Transmission : Discharge from the lymph nodes, via wounds caused by shearing, castration and docking, contaminated sheep dips, skin abrasions or traumatized oral mucosa. Animals with open abscesses should be segregated in order to prevent the spread of the disease.

Antemortem findings :

1. Animal is lagging behind the flock.
2. Dyspnea
3. Purulent ocular and nasal discharge
4. Enlarged superficial body lymph nodes
5. Generalized disease is associated with weight loss, depression and loss of appetite.

Postmortem findings :

1. Caseous abscesses in the superficial lymph nodes and carcass musculature (Fig. 165)
2. Firm and dry abscess in the kidney (Fig. 166) and other organs Soft pasty abscess in the early stages changes to firm and dry with a characteristic laminated appearance in the later stages of disease.
3. Abscess content is creamy and pasty in goats
4. Pneumonia

Differential diagnosis : Abscesses in the organs and viscera, neoplasm, echinococcosis and

other parasitic lesions

Judgement : If this condition is associated with extensive involvement of many lymph nodes and tissues, suggesting a haematogenous spread, the carcass is *condemned*. Otherwise it is *approved*. The affected tissue is *condemned*.

Remarks : An abscess in a body lymph node is a sequel to the organism gaining entrance into the body via skin wounds etc. The drained area of the lymph node should be examined. If no other lesions are observed, it may be an indication that the lymph node has sequestered the agent. It is not necessary to condemn a quarter or a carcass due to a lesion in one lymph node or in several lymph nodes.

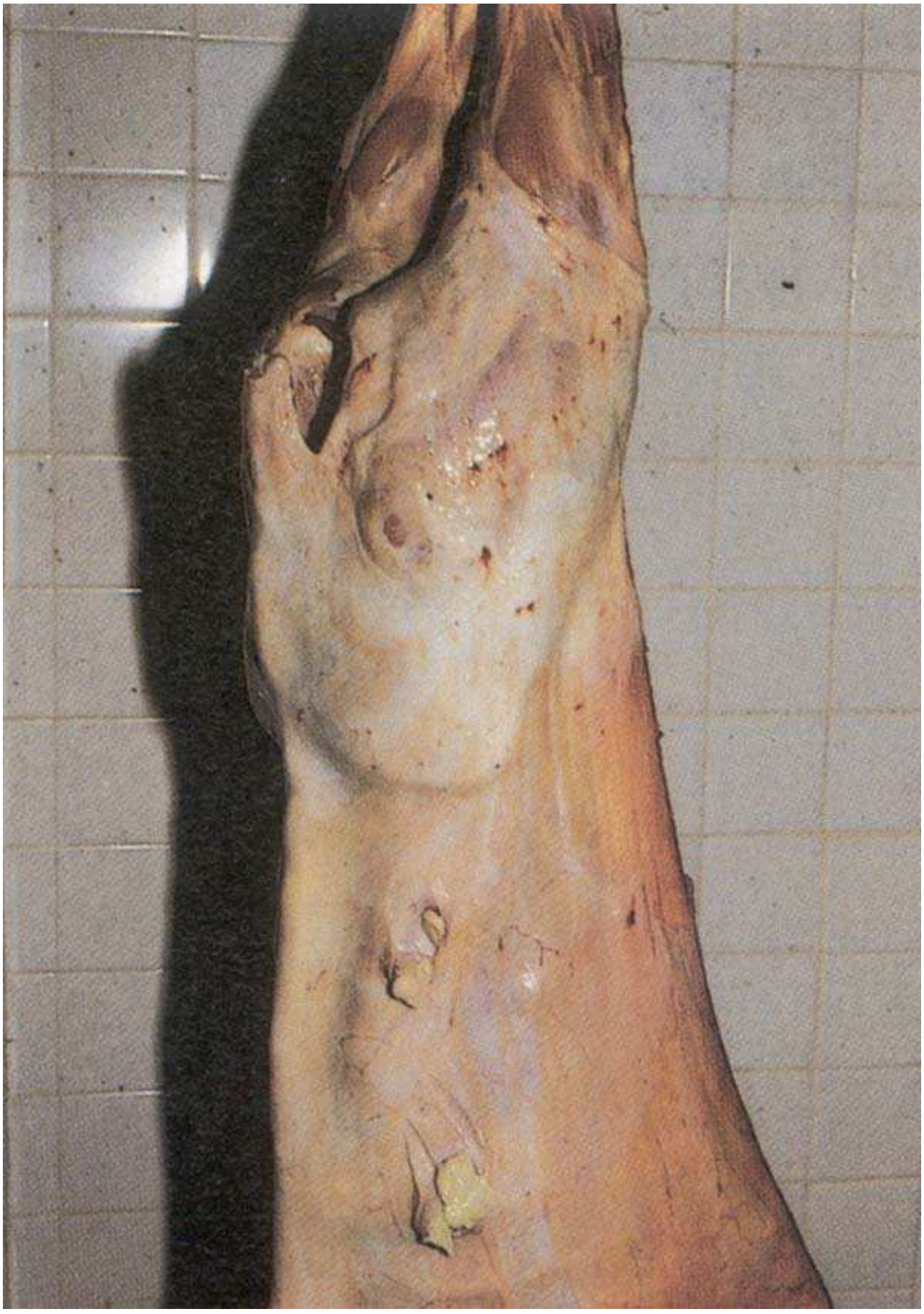


Fig. 165: Caseous lymphadenitis. Caseous abscess filled with greenish-yellow pus in the abdominal muscles.

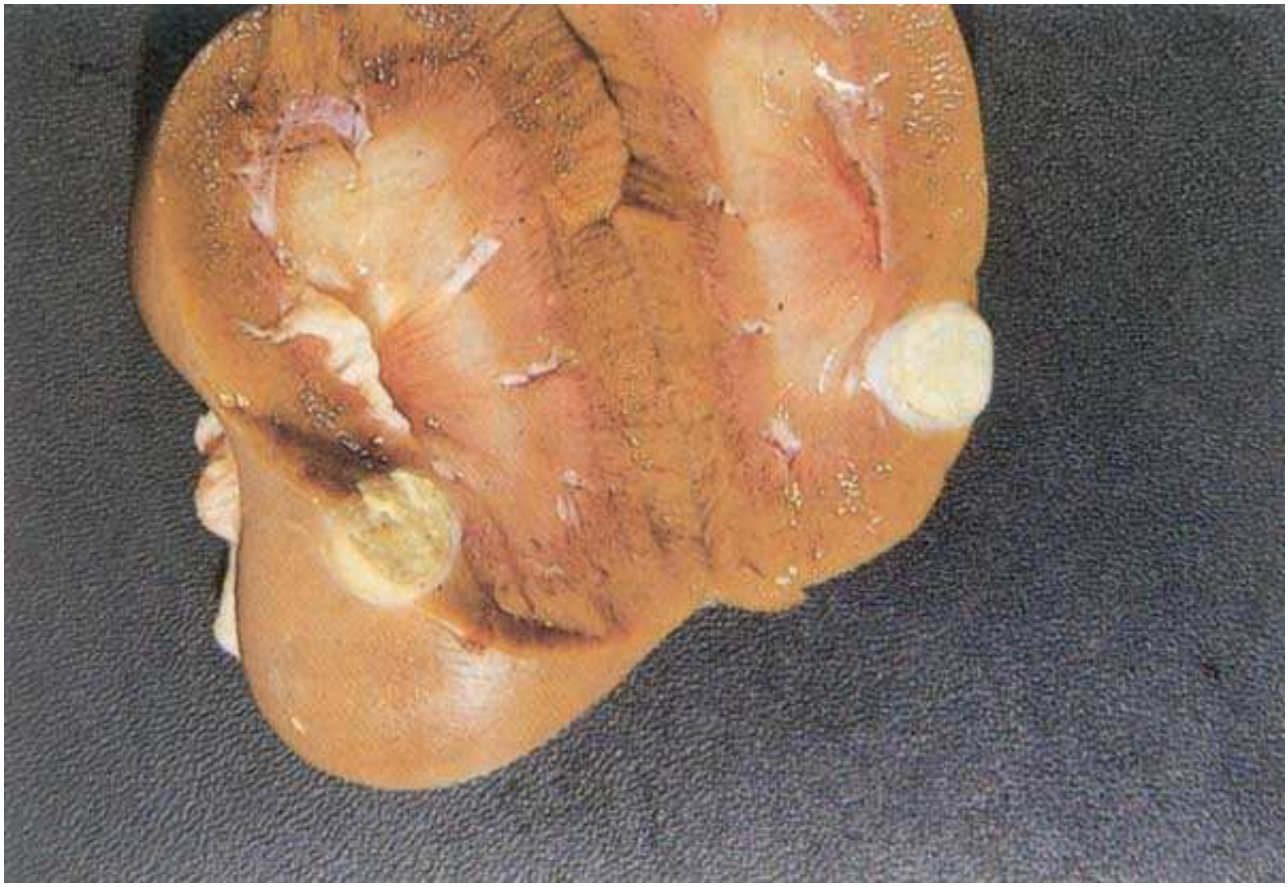


Fig. 166: Caseous lymphadenitis. Firm and dry abscess in the kidney.

Parasitic diseases

Diseases caused by helminths

Coenurus cerebralis infection (Gid, Sturdy)

Coenurosis is a disease of the brain and spinal cord caused by the intermediate stage of *Taenia multiceps* which inhabits the intestine of dogs, cats and wild carnivores. The clinical disease occurs in sheep and rarely in cattle.

Life cycle : *Eggs* expelled with dog faeces are ingested by the intermediate host (sheep). The *larvae* hatch in the intestine and pass with the blood stream towards different organs. The larvae which reach the brain and spinal cord grow to the *coenurid* stage. Coenurus cerebralis will further mature in the brain and spinal cord.

Antemortem findings:

During migration of larval stage

1. Blindness
2. Muscular tremor and incoordination
3. Excitability and collapse

Infection with the fully developed larval stage

4. Salivation
5. Wild expressions
6. Frenzied running and convulsion
7. Deviation of eye and head
8. Loss of function

9. Dullness
10. Incomplete mastication
11. Head pressing
12. Incomplete paralysis and, in spinal cord involvement, inability to rise

Postmortem findings :

1. Thin walled cyst in the brain (Fig. 167)
2. Lesion in the lumbar region and rarely, in the cervical area of the spine

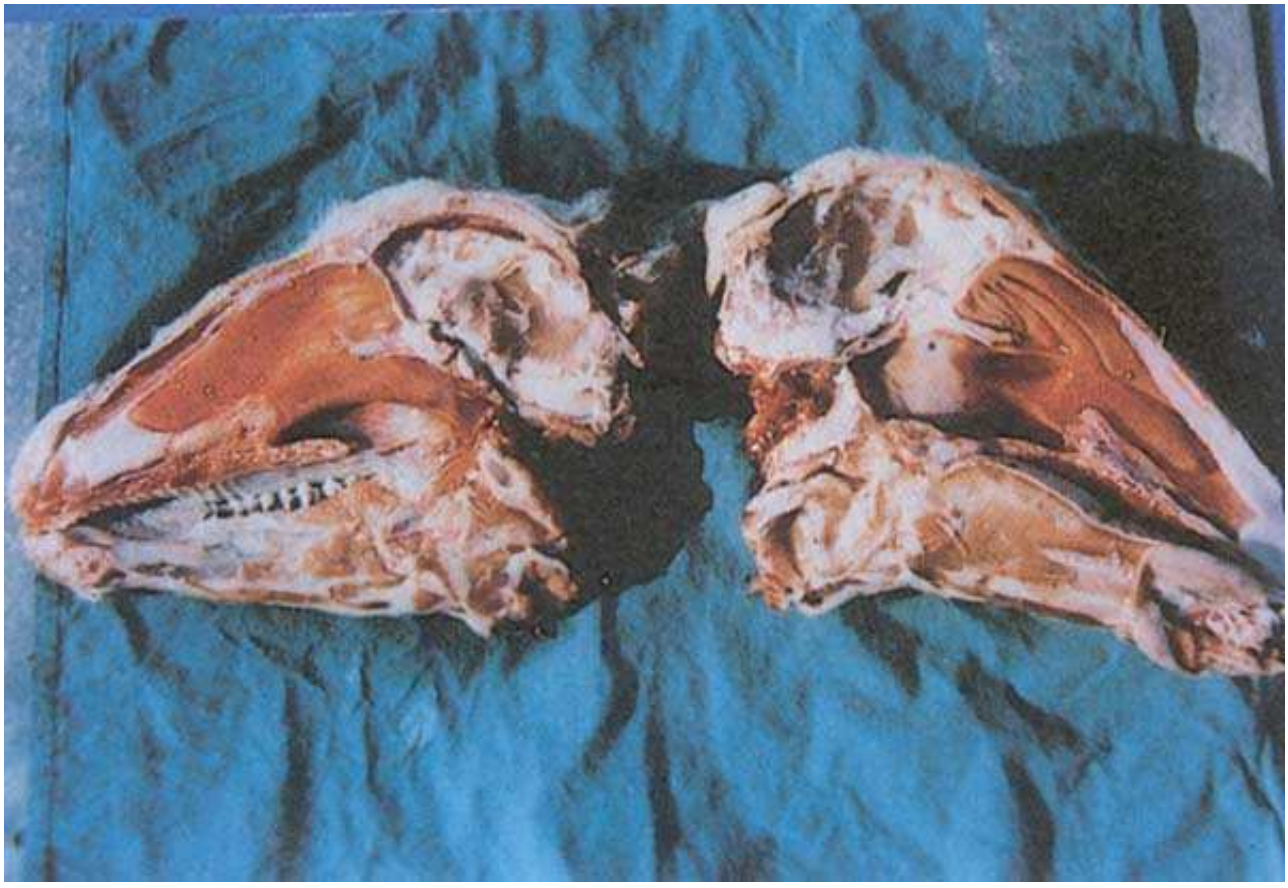


Fig. 167: Coenurus cerebralis. Thin walled cyst in the brain.

Judgement : Carcass affected with coenurosis is *approved*. Affected brain and organs are *condemned*.

Differential diagnosis : Abscess, haemorrhage, brain tumours and in early stages, inflammation of the brain and rabies

Echinococcosis (Hydatid disease)

Hydatid disease occurs in sheep, cattle, swine, horses and humans. Echinococcosis is a disease which occurs when the larval stage of *Echinococcus granulosus* and *Echinococcus multilocularis* are ingested by an intermediate host (sheep, cattle). These larvae then develop into hydatid cysts in various tissues. The adult tape worms are found in dogs, cats and other carnivores. They may ingest the hydatid cysts by eating infected organs of the intermediate hosts. The scolex attaches to the intestinal wall. Adult tapeworms develop in approximately seven weeks and *eggs* are shed in the faeces and are ingested by sheep and cattle. The ova hatch to liberate the *onchospheres* which penetrate the intestinal wall and through the portal venous supply to the liver where they become arrested. In older sheep and cattle the larvae may reach the lungs and various other organs through the systemic circulation. The most common sites of cysts are the liver and lungs. The *cysts* are different sizes and shapes and they contain a clear fluid. Due to the growth of the cyst, pressure atrophy is noted in the surrounding tissue.

Daughter cysts are found outside the mother cyst and are formed due to trauma or external pressure on the mother cyst. They may or may not be attached to the mother cyst. Daughter-cyst formation may have neoplastic characteristics when there is penetration to the blood and lymph vessels and metastases to various distant organs.

Humans gets infected with hydatid disease via the ingestion of ova from Echinococcus tapeworm in the dog. This usually occurs by touching dog hair that has been contaminated by ova from faeces. It also may occur by the dog transferring ova from the anus to its mouth and then by licking humans.

Postmortem findings : Multiple Echinococcus granulosus cysts in the liver, lungs (Fig. 168) and other organs.

Judgement : The animal carcass affected with echinococcosis is *approved* if edema and emaciation are not found. Otherwise the carcass is *condemned*. The affected organs are also *condemned* and must be destroyed. The lungs are most commonly affected and these should be carefully checked because lesions are often missed on routine inspection.

Differential diagnosis : C. tenuicollis, C. cellulosae, calcified TB lesions and congenital cysts

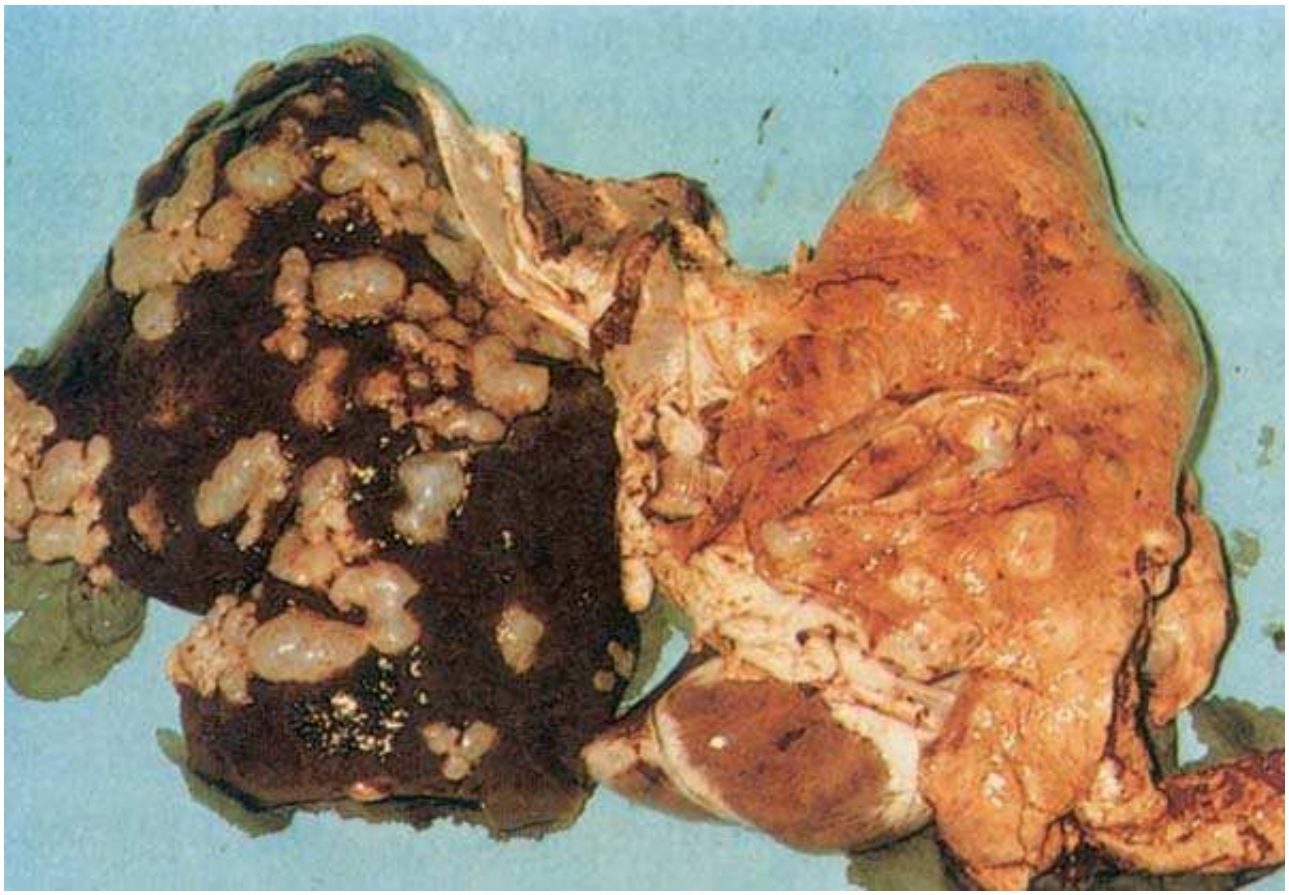


Fig. 168 : Echinococcosis. E. granulosus cysts in the liver.

Lung worms

Dictyocaulus filaria is the common sheep lung worm which cause verminous pneumonia or bronchitis.

Life cycle : (Fig. 169) Adult worms live in the bronchi where they lay *eggs* which are coughed up to pharynx and swallowed by the host. The eggs are hatched in the digestive tract and the *larvae* are then expelled in the faeces. In a moist environment and moderate temperature, the larve will become infective in 3 – 7 days. Larvae are rsistant to cold, althourhg it will cause their maturation to be delayed. Upon digestion by sheep (primary host), larvae penetrate the intestinal wall and reach the meenteric lymph nodes. From the mesenteric lymph nodes via the blood

stream, larvae migrate to the lung alveoli and further to the bronchi. They *mature in the bronchi* and lay eggs. The cycle is then repeated.

Muellerius capillaris parasitises in the alveoli and pulmonary parenchyma. Intermediate hosts are snails and slugs which sheep inget during grazing. Larvae reach the lungs and produce small greyish nodules on the back of the lungs.

antemortem findings :

1. Difficult breathing
2. Cough and nasal discharge
3. Fever if secondary infection present

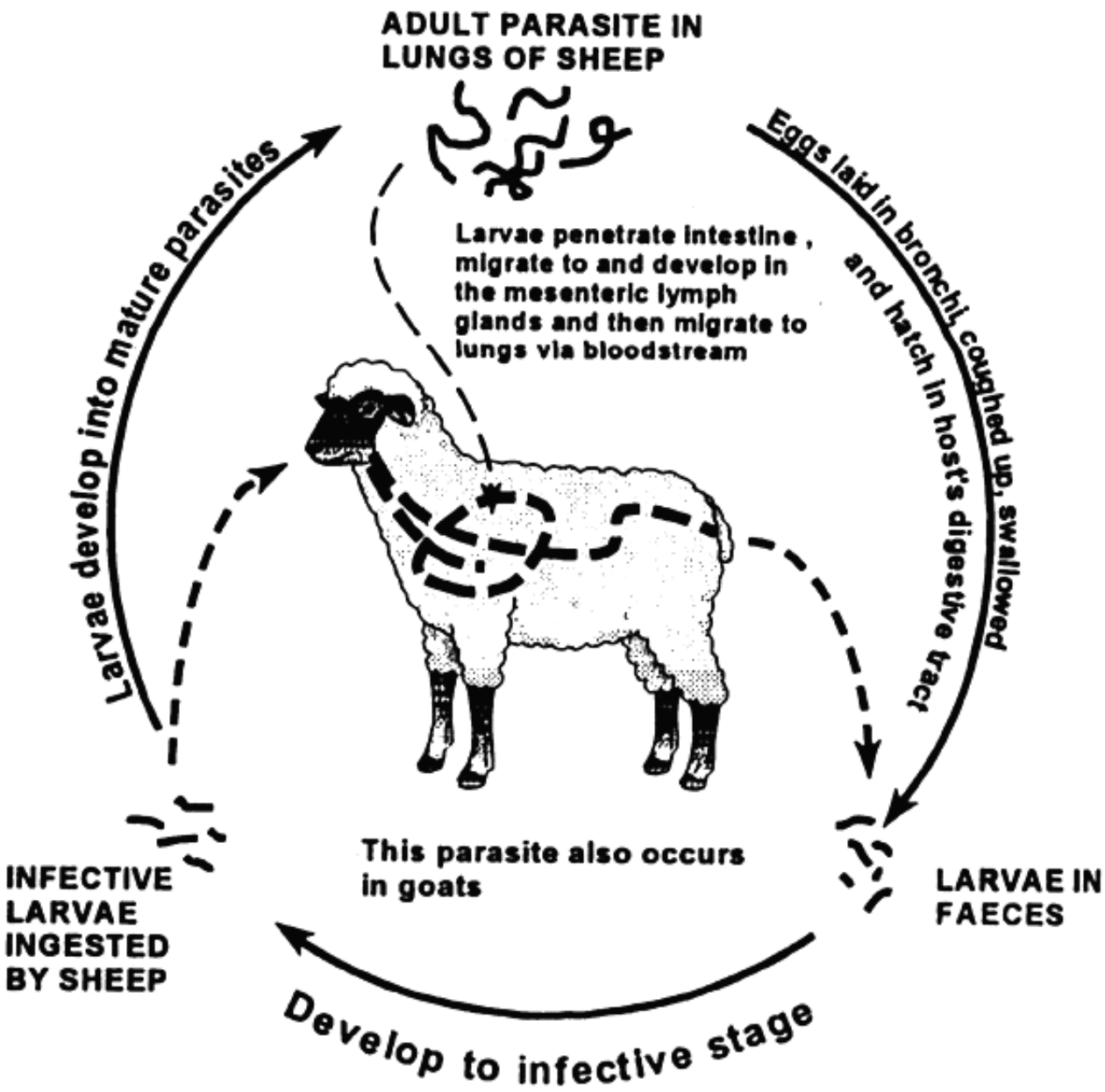
Post mortem findings :

1. Exudate in bronchioles and resulting collapse of long portion
2. Verminous pneumonia with consolidation of lung parenchyma
3. Enlarged lung lymph nodes
4. Greyish-green nodules encysted or clacified with Muellerius capillaris infestation (Fig. 170)

Judgement : Carcass is *approved* in lung worm infestation if no secondary changes are observed. The lungs are *condemned*. If lung worm infestation has caused pneumonia, emaciation or anaemia, the carcass is *condemned*.

Diferential diagnosis : bacterial bronchoppeumonia, abscess, necrobacillosis, tuberculosis, actinobacillosis, hydatid disease and atelectasis

Fig. 169 : Life history of lungworm of sheep


ADULT PARASITE IN LUNGS OF SHEEP
Larvae penetrate intestine , migrate to and develop in the mesenteric lymph glands and then migrate to lungs via bloodstream
Eggs laid in bronchi, coughed up, swallowed and hatch in host's digestive tract
LARVAE IN FAECES
Develop to infective stage
INFECTIVE LARVAE INGESTED BY SHEEP
Larvae develop into mature parasites
This parasite also occurs in goats

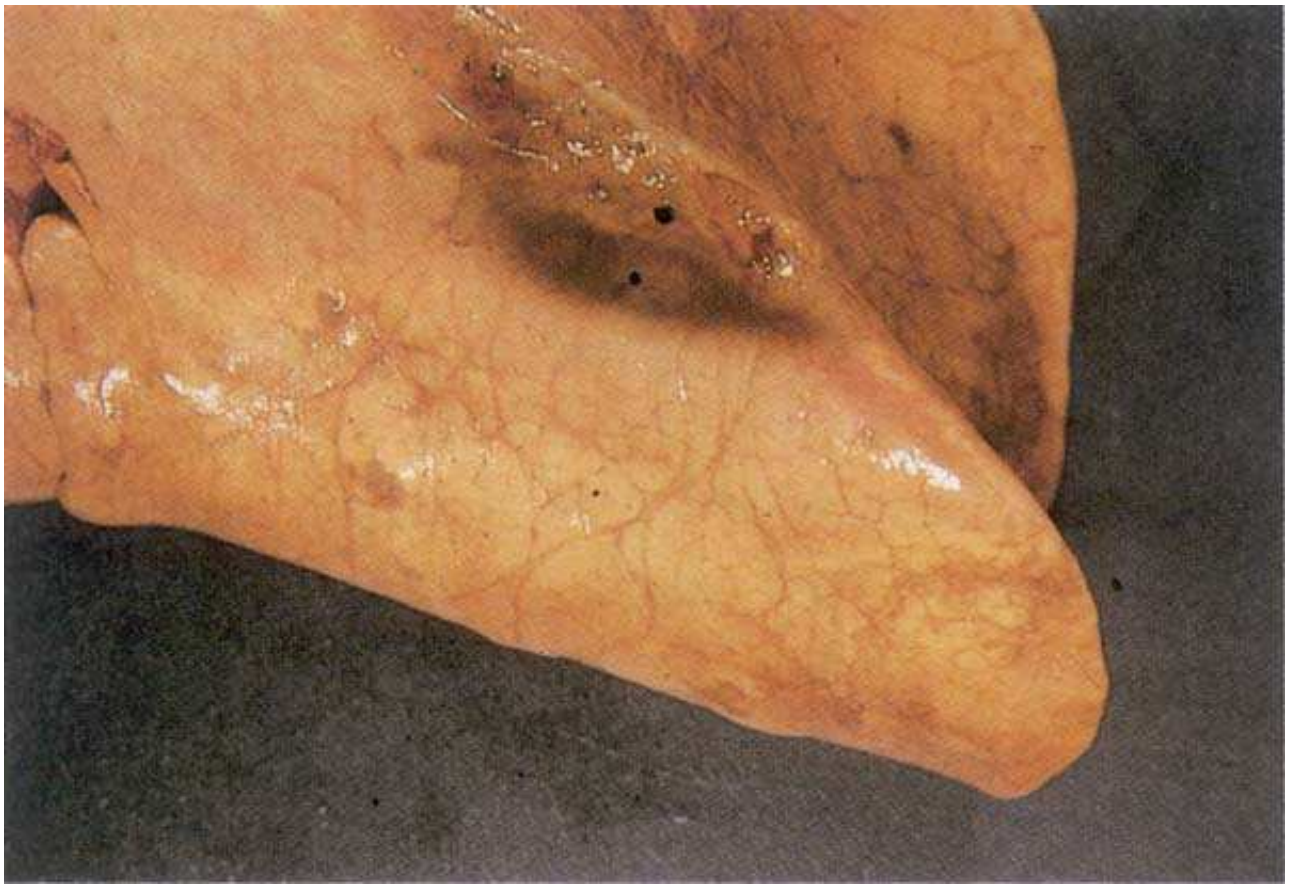


Fig. 170: Lung worms. Numerous greyish nodules of M. cpaillaris in the lung parenchyma.

Fascioliasis

The fluke *Fasciola hepatica* is most frequently found in sheep and cattle and less often in goats and swine. Acute fascioliasis occurs almost entirely in sheep. In sheep and cattle, wandering flukes damage liver tissue and bile ducts which then become thickened and fibrous.

Life cycle: Thadult flukes of Fasciola hepatica are found in the bile ducts and gall baladder. The eggs are shed into the bile duct from which they pass to the intestine. With animal faeces, the eggs are expelled out on the pasture.

The larve *(miracidia)* enter aquatic snails (Limnea truncatula) which are the intermediate hosts and develop into *sporocysts* and later into *rediae*. The rediae will further develop into the final larval stage *(cercaria)*. Cercaria will transform in the external environment to *metacercaria*. If ingested by herbivorous animals, metacercaria will penetrate the small intestinal wall, cross the peritoneal space and reach the liver. In the bile ducts, metacercaria will mature into an *adult fluke*. The metacercariae which do not reach the bile ducts will encapsulate in the liver parenchyma.

Fasciloides magna is a large liver fluke which is prevalent in elk, deer and moose. Sheep and goats are susceptible to infection if they share the pasture with those wild animals. F. magna in sheep continuously migrate through the liver parenchyma and may cause death in less than six months.

Lancet flukes (*Dicrocoelium dendriticum*) in sheep cause little damage to the liver parenchyma except for a moderate to marked thickening of the bile ducts.

Antemortem findings :

1. Weight loss
2. Anaemia and edema
3. Chronic diarrhoea

Postmortem findings :

1. Black parasitic debris in the liver (Fig. 171), lungs, diaphragm and peritoneum
2. Black lymph nodes of the lungs and liver due to fluke excrement

Judgement : Carcass of an animal affected with fascioliasis is *approved* if in good flesh and emaciation and edema are not observed. A heavily infested parasitic liver is *condemned*.

Differential diagnosis : Nutritional deficiencies of copper and cobalt, infectious necrotic hepatitis, black disease, anthrax, enterotoxaemia, melanosis, melanoma

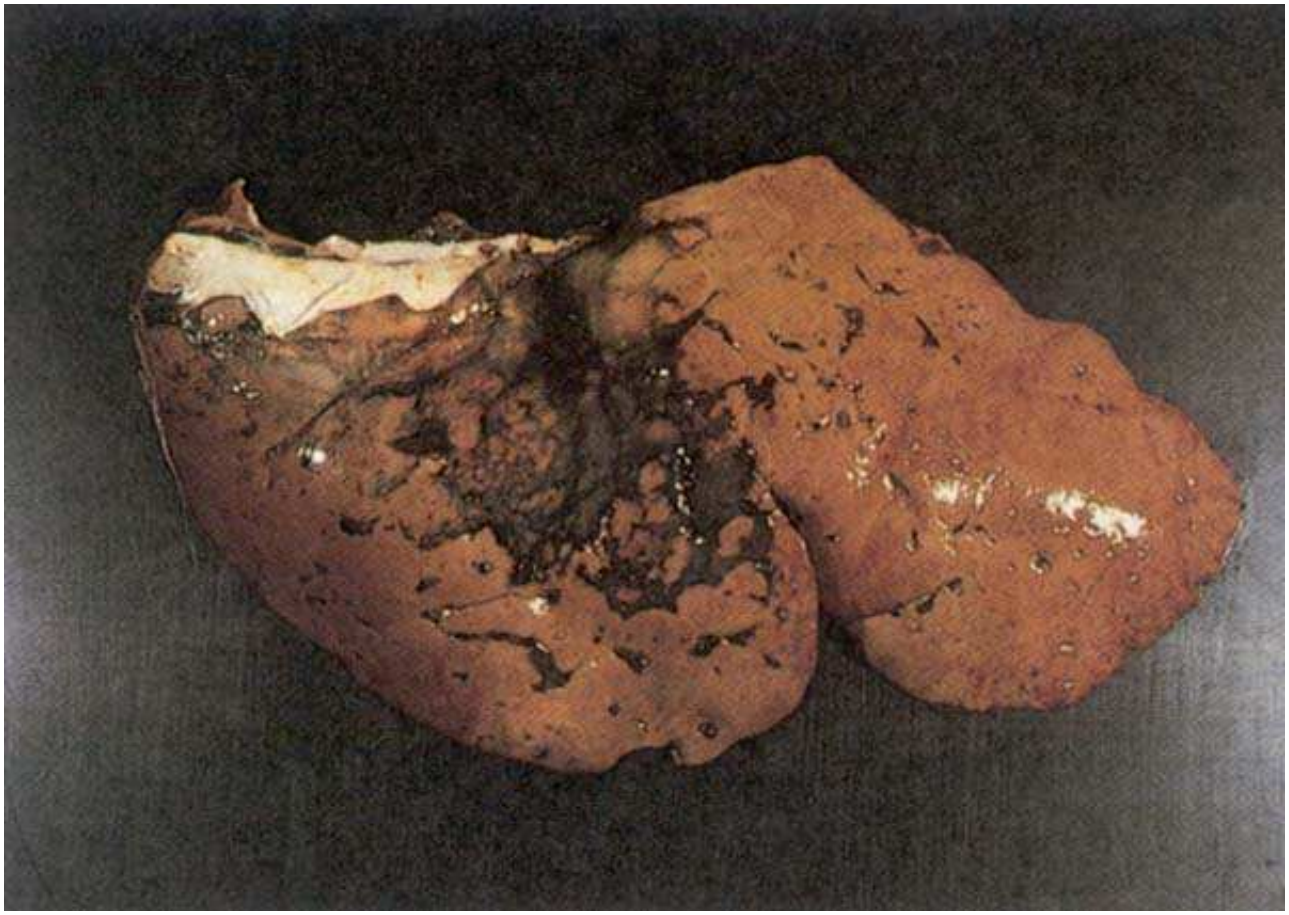


Fig. 171: Fascioliasis. Black parasitic debris in the liver.

Cysticercus tenuicollis infestation

Cysticercus tenuicollis is the cystic stage of tape worm *Taenia hydatigena* which is found in dogs and cats. *Ova* pass with dog faeces on the pasture and may get ingested by intermediate hosts sheep and pigs. *Larvae* which develop from ova penetrate the intestine and pass by portal vein to various tissues especially the omentum, mesentery, peritoneum and liver. Migration through the liver leaves greyish-white tortuous tracts. If larvae reach the liver surface they develop into thin-walled fluid filled bladders and if they fail they degenerate and become calcified.

Heavy infestation with Cysticercus tenuicollis in young animals causing liver damage and haemorrhages or peritonitis, rarely results in the death of the animal.

Antemortem findings :

Moderate to heavy infections produce:-

1. Loss of appetite
2. Depression

3. Weakness

Postmortem findings :

1. Cysts of different diameters on the liver, diaphragm and peritoneum
2. Subserosal cysts on the liver (Fig. 172)

Judgement : The carcass affected with cysticercus tenuicollis is *approved*. The organs are *condemned* and affected serous membranes should be stripped.

Differential diagnosis : C. bovis, C. cellulosae, hydatid cysts and calcified TB lesions

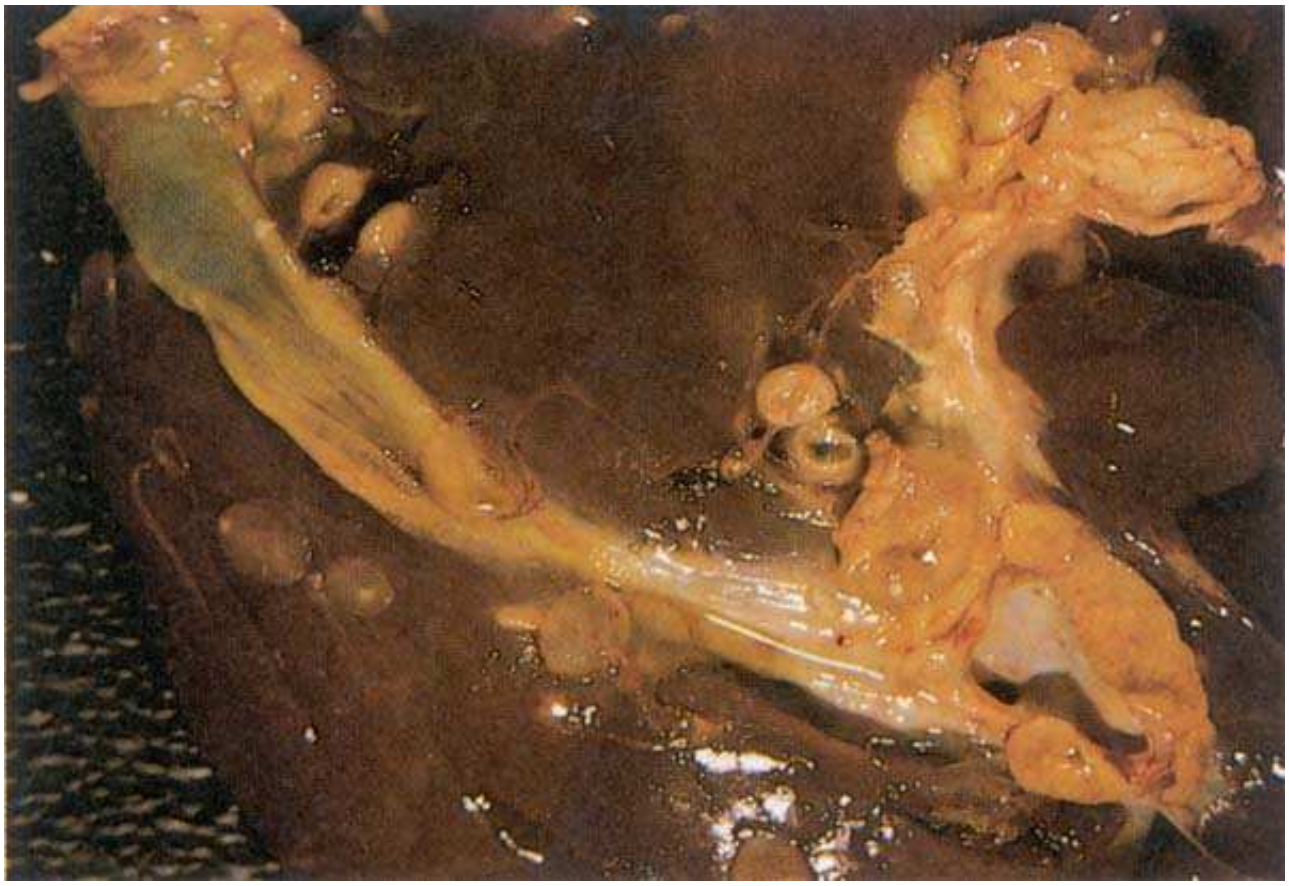


Fig. 172: Cysticercus tenuicollis. Numerous subserosal cysts in the liver.

Cysticercus ovis infestation (sheep measles, sheep bladder worm)

Cysticercus ovis is the larval stage of *Taenia ovis*, a tapeworm found in the intestines of dogs and wild carnivores. Its development is similar to that of *Taenia saginata*. However, in the case of *Taenia ovis*, the definitive hosts are *sheep*. The cysts are found in the heart, diaphragm, masseters and the skeletal musculature of sheep. They are fully developed from 7 to 10 weeks after the ingestion of the ova. The rapid degeneration of cysts commence almost immediately after the cysts reach maximum development. When degenerated, the cysts appears as a caseous nodule in the musculature.

Antemortem findings : Usually no clinical signs are recognized.

Postmortem findings :

1. The cysts are oval, measure 9 mm × 5 mm when fully developed and are most common in the heart (Fig. 173), the masseters, the diaphragm and the skeletal musculature (Fig. 174).
2. In older animals the cysts degenerate and calcify
3. The degenerated cysts appears as greenish yellow caseous nodules with calcification

often present.

Judgement : In moderate or light infestation consisting of a small number of dead or degenerated cysticerci, the carcass can be *boned out* under supervision, the *cysts removed* and the meat passed after being held for 10 days at -10°C. If the *freezing treatment* is not possible, the *heating* of the carcass at 56°C is suggested.

In heavy infestations the carcass is *condemned*. It is commonly considered that an animal is heavily infested if lesions are discovered in two of the usual inspection sites including the masseter muscle, tongue, oesophagus, heart, diaphragm or exposed musculature and in two sites during incision into the shoulder and the rounds. Carcasses with C. ovis infestations may not be acceptable for export.

Differential diagnosis : Sarcocystosis, eosinophilic myositis, neurofibromatosis, abscesses, C. tenuicollis, caseous lymphadenitis

Fig. 173: Cysticercus ovis. The heart of an old ewe showing heavy infestation with C. ovis. The cysts have degenerated and undergone calcification. (Courtesy Dr. D. Baucks)

Fig. 174: Skeletal muscles of a sheep with several cysts of C. ovis undergoing calcification. (Courtesy Dr. D. Baucks)

Stilesia hepatica

This is a tape worm which occurs in the bile duct of sheep, goats and wild ruminants. The life cycle is not completely known but oribatid mites are suspected of transmitting the parasites. The parasite affects animals of all ages and is considered non pathogenic. Heavy infections are frequently seen in apparently healthy sheep. With almost complete occlusion of the bile ducts, icterus and the other clinical signs are not observed. There are areas where approximately 80 % of sheep and goat livers are affected.

Judgement : The carcass is *approved* unless associated with emaciation. The affected liver is *condemned*. In some parts of the world, all sheep livers are condemned on postmortem inspection, because of high rate of liver infections.

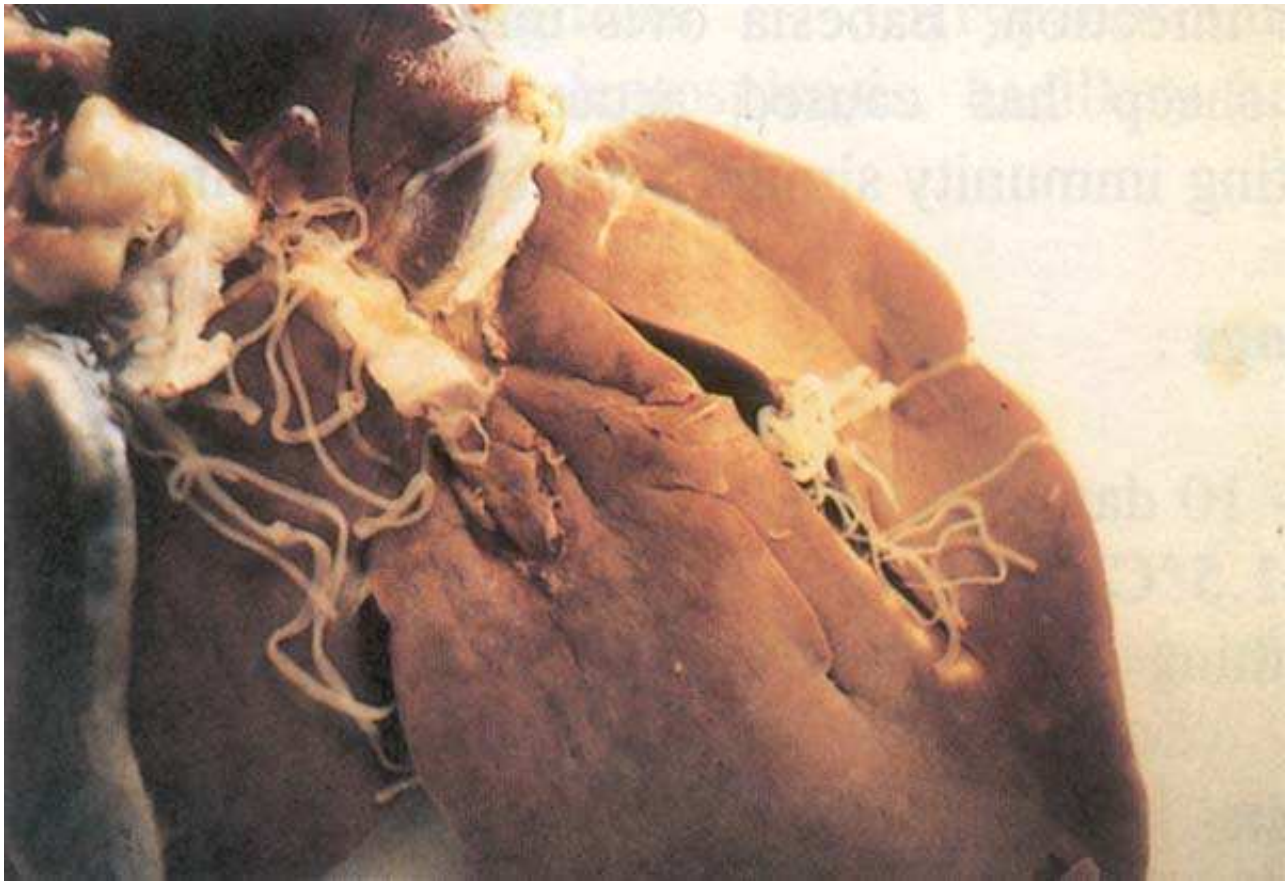


Fig. 175: Stilesia hepatica. Long, threadlike parasite (20-50 cm long and up to 3 mm wide) in the sheep liver.

Diseases caused by protozoa

Babesiosis (Piroplasmosis, Texas fever, Red water, Tick fever)

Babesiosis is a protozoan parasitic febrile disease of cattle, horses, sheep and swine caused by Babesia spp..

In sheep and goats, babesiosis is caused by *Babesia motasi* and *Babesia ovis*. Acute signs of the disease are characterized with fever, anaemia, parasitemia and haemoglobinuria. B. ovis usually causes a milder form of the disease than does B. motasi. The parasite grows and multiplies in the blood corpuscles (erythrocytes) of sheep and goats and causes haemoglobin (constituent of erythrocytes) elimination in urine (haemoglobinuria).

Transmission : Different species of ticks in the family Ixodidae serve as vectors of infection. Babesia ovis infection transmitted experimentally in sheep has caused acute signs of disease, parasitemia and lasting immunity similar with babesiosis in cattle.

Antemortem findings :

1. Incubation 7 – 10 days
2. High fever (41.5°C)
3. Difficult breathing
4. Anaemia
5. Loss of appetite
6. Dark reddish brown urine
7. Recovered animals may be emaciated, have reduced milk production, and some may also abort.

There are no characteristic signs in the chronic disease.

Postmortem findings :

1. Enlarged, yellow liver and distended gall bladder containing thick dark bile. The bladder mucosa is edematous and yellow.
2. Subcutaneous tissue and connective tissue in the muscles are edematous and jaundiced.
3. Thin watery blood and red urine in the bladder
4. Enlarged spleen
5. Edematous and haemorrhagic lymph nodes

Judgement : Carcass of an animal in the subclinical form of the disease or in the chronic stage may have a *favourable judgement* providing the carcass is adequately set and icterus is not present. An animal carcass showing acute form of the disease accompanied with fever, marked anaemia and haemoglobinuria and/or emaciation is *condemned*.

Differential diagnosis : Trypanosomiasis, theileriosis, haemobartenellosis, leptospirosis, bacillary haemoglobinuria and anaplasmosis

Toxoplasmosis

Toxoplasmosis is a contagious disease of animals and man caused by protozoon *Toxoplasma gondii*. It is found most frequently in pigs and sheep. Toxoplasma in sheep is manifested with abortion and stillbirths in ewes.

Life cycle : see Fig. 147

Antemortem findings:

1. Abortion and stillbirths in ewes
2. Fever
3. Generalized tremor
4. Difficult breathing

The systemic disease is seldom found in sheep.

Postmortem findings:

1. Multiple granulomatous lesion in the lungs
2. Hydrothorax
3. Ascites
4. Intestinal ulceration
5. Necrosis in the liver, spleen and kidneys
6. Necrosis of placenta
7. Brain haemorrhage, edema and ventricular dilatation (Fig. 176)
8. Inflammation of the brain (Fig. 177)

Judgement: Carcass of an animal showing clinical signs of acute disease is *condemned*. Recovered and reactor animals are *approved*.

Differential diagnosis:

Abortion in ewes: brucellosis, campylobacteriosis, listeriosis, salmonellosis and Rift Valley fever
Brain lesions: salt poisoning, chlorinated hydrocarbons, lead, mercury, Vitamin A deficiency, hypoglycaemia, encephalomalacia, meningitis, rabies and scrapie

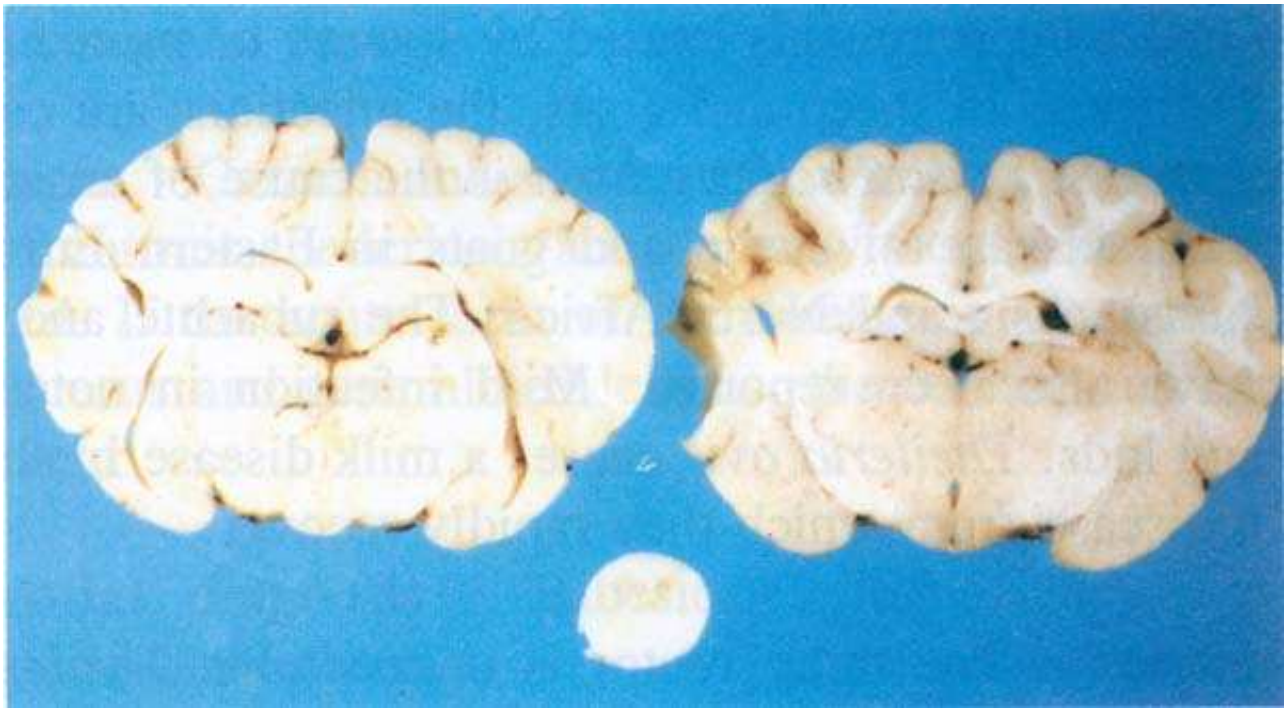


Fig. 176: Toxoplasmosis. Brain haemorrhage, edema and ventricular dilatation. The specimen was fixed in 10% formalin solution.

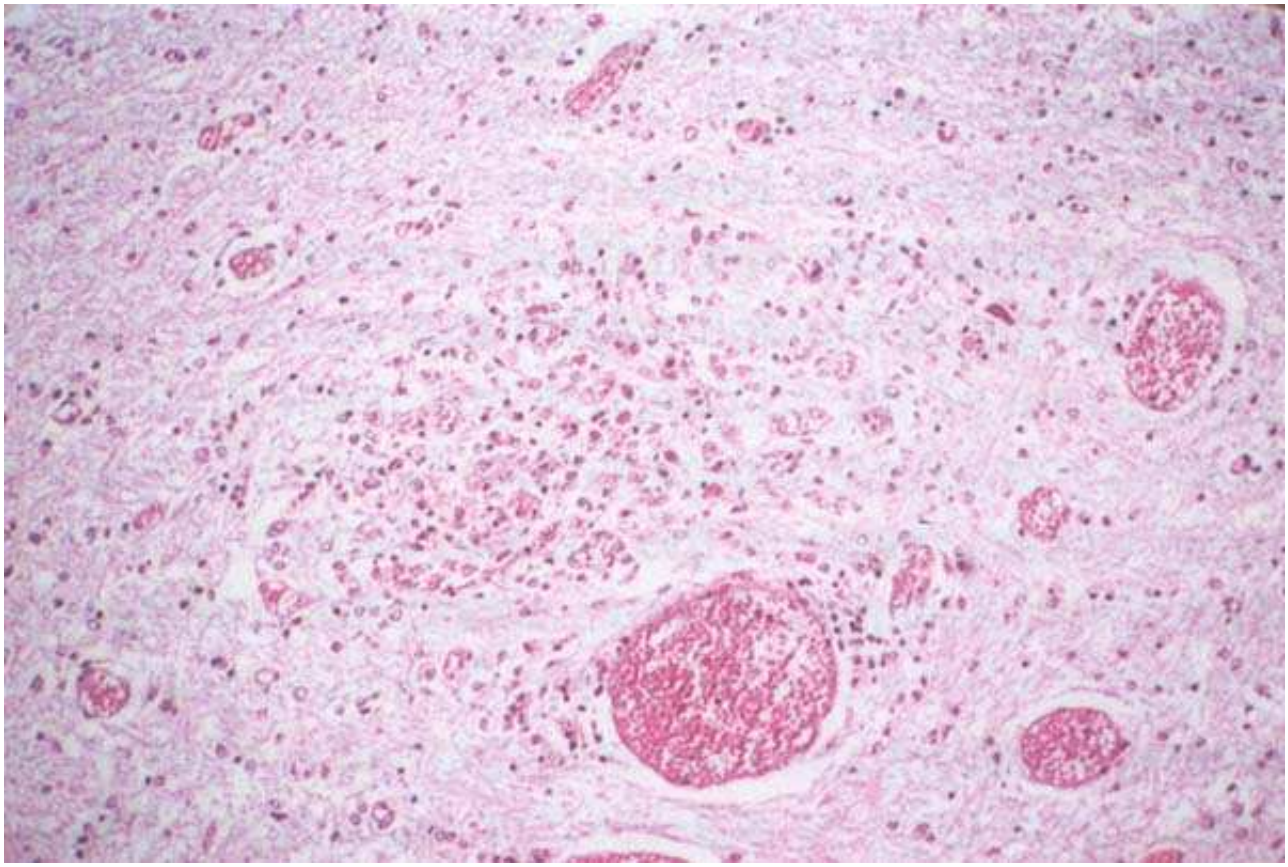


Fig. 177: Toxoplasmosis. Inflammation of the brain (encephalitis). Tachyzoites are distributed throughout the brain where they encysts and produce bradyzoites.

Theileriosis (Malignant ovine or caprine)

Theileriosis is thick borne disease of sheep and goats, cattle, buffalo and wild ruminants caused by species of protozoa in the genus Theileria. In sheep and goats, the infections are caused by T. hirci and T. ovis. *Theileria hirci* is the cause of an acute and highly fatal disease of sheep and goats in Eastern Europe, the Middle East, Asia and North Africa. The subacute and chronic forms have also been reported. Mild infection in noted young lambs and kids. *Theileria*

ovis causes a milk disease in sheep and goats; a disease from which they rapidly recover.

Transmission : The thick vector is unknown in *Theileria hirci* infection, although Hyalomma spp. are suspected.

Antemortem findings :

In acute form

1. Morbidity rate of 100 % and mortality of 46 – 100 %
2. Fever (40°C - 41°C)
3. Loss of appetite and listlessness
4. Increased heart rate and difficult breathing
5. Edema of the throat and subsequent death
6. Hyperaemia of the conjunctiva and nasal discharge
7. Swollen superficial lymph nodes
8. Atony of the rumen in the chronic form
9. Mild fever, anaemia, icterus, weakness and emaciation

Postmortem findings :

1. The lesions are basically similar as those observed in bovine Theileriosis (T. parva).
2. Edema of the lungs
3. The yellowish-brown liver may be increased in size and shows soft and friable consistency.
4. Enlarged haemorrhagic lymph nodes and enlarged spleen
5. Kidney infarcts
6. Petechial haemorrhage in subcutaneous, subserosal and submucosal tissue

Judgement : Carcass and viscera of an animal showing clinical signs of chronic theileriosis and being without gross lesions, are *approved*. If the acute form of the disease is accompanied with fever, icterus and generalized lesions, the carcass and organs are *condemned*.

Differential diagnosis : Babesiosis, Rift Valley Fever and catarrhal fever of sheep

Sarcocystosis in sheep (Sarcosporidiosis)

Sarcocystosis of sheep is a widespread infestation caused by four species of *Sarcocystis* (Table 2). Nearly all adult sheep in most parts of the world are infested. Three other species of Sarcocystis have been described from goats. Their prevalence and importance in meat inspection are not fully known.

The general pattern of the life-cycle is similar to that described for Sarcocystis cruzi in cattle except that each species uses its own definitive hosts. S. tenella and S. gigantea cause the most widespread infestations. S tenella produces microcysts and are the most pathogenic. S. gigantea produces macrocysts and are generally not pathogenic but because of their large size they are important in meat inspection.

Table 2: Sarcocystis spp. in sheep

Species	Distribution	Definitive Hosts	Size and Shape of Cyst	Pathogenicity
Sarcocystis tenella	World-wide	Dog, coyote and red fox	Microscopic, up to 0.7 mm long, may be found in the central nervous system	Pathogenic. Causes anorexia, weight loss, anaemia, fever, abortion and even death. It is the most pathogenic sheep Sarcocystis sp.
Sarcocystis gigantea	World-wide	The domestic cat	Macroscopic, oval or elongated and measures up to 1 cm long, More	Only mildly pathogenic.

			common in order sheep.	
Sarcocystis arieticanis	Europe, Australia, New Zealand and the USA	Dog	Microscopic, up to 0.9mm long.	They are less pathogenic than S. tenella
Sarcocystis medusiformis	Australia and New Zealand	Cat	Macroscopic, filiform and elongated up to 8mm long and 0.2mm wide.	Pathogenicity not known.

Antemortem findings (in S. tenella infection):

1. Fever
2. Anaemia
3. Loss of appetite and weight loss
4. Retarded growth
5. Enlarged lymph nodes
6. Abortion
7. Nervous signs

Postmortem findings (in S. gigantea infestations) :

1. Oval, elongated or fusiform cysts up to 1 cm long and 0.5 cm wide in the oesophagus (Fig. 178), pharynx, diaphragm, skeletal musculature, tongue and heart
2. In S. tenella infestations haemorrhages in the serous surface of the viscera, cardiac and skeletal muscles
3. Serous atrophy of pericardial and perirenal fat

Judgement : In heavy infestations the carcass is *condemned*. In moderate to light infestations the lesions are removed and the carcass is *passed*.

Differential diagnosis : Myositis, cysticercosis, grass seeds, necrotic lesions

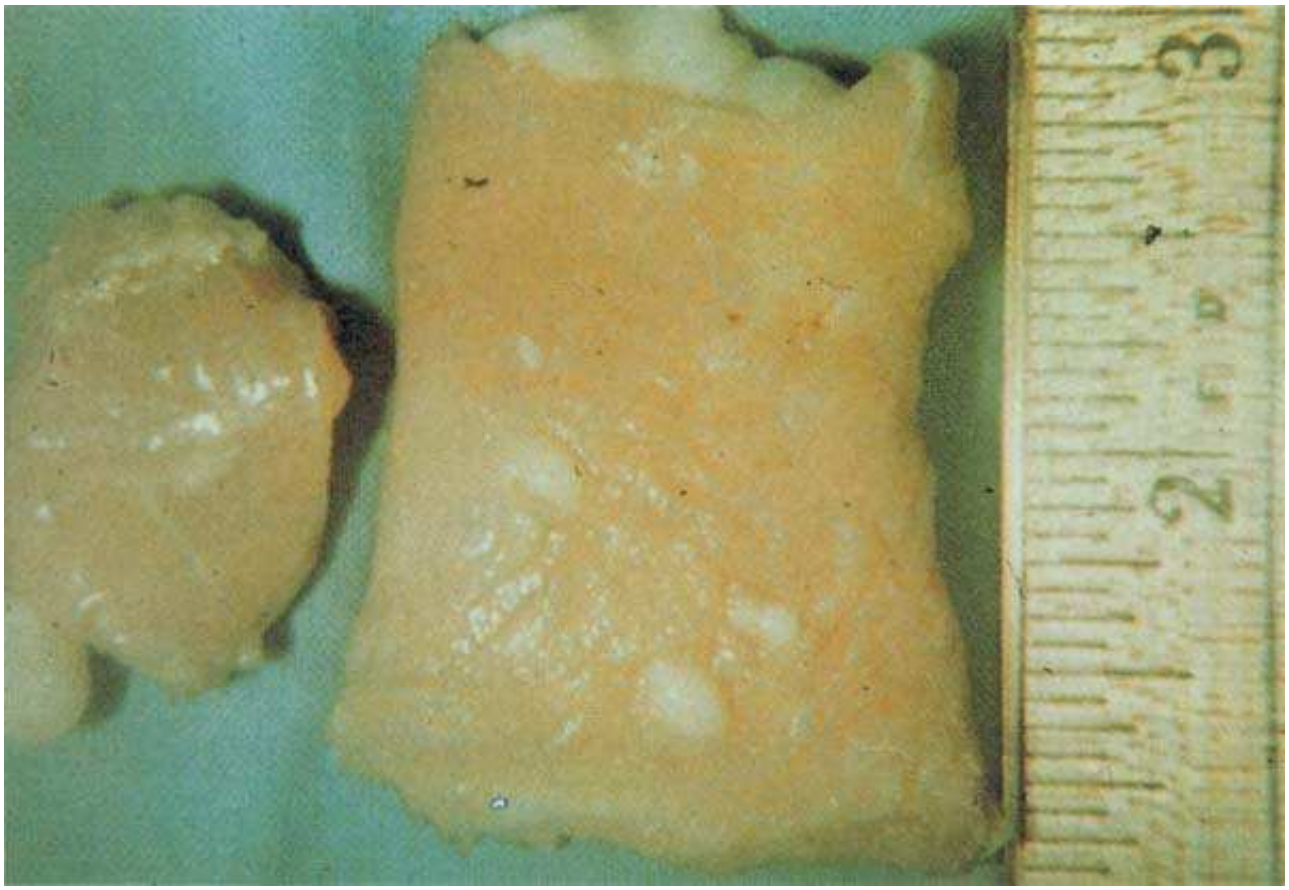


Fig. 178: Sarcocystis gigantea in the oesophagus of a sheep. They resemble cooked rice grains.