MJF COLLEGE OF VETERINARY AND ANIMAL SCIENCE, CHOMU, JAIPUR

DEPARTMENT OF VETERINARY PATHOLOGY

RINDERPEST

AETIOLOGY

- Rinderpest is caused by a negative-strand RNA virus of the Morbillivirus genus within the family Paramyxoviridae.

- Three genetically distinct lineages (1–3) of the rinderpest virus (RPV) have been recognised as causal agents of disease in Africa and Asia.

Resistance to physical and chemical action

- **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.

Hosts

- Highly fatal among domestic cattle, water buffalo (*Bubalus bubalis) and yak (Bos grunniens); European cattle (Bos primigenius taurus) more susceptible than zebu breeds* (*Bos primigenius indicus)*
- Highly susceptible wild animals: African buffalo (*Syncerus caffer), giraffe (Giraffa cameloparadalis), eland (Taurotragus oryx), kudu (Tragelaphus strepsiceros and T. imberbis), wildebeest (Connochaetes sp.) and various antelopes*
- Sheep and goats are susceptible but epidemiologically unimportant
- Asian pigs seem more susceptible than African and European pigs
- Dogs can seroconvert upon consuming infected meat and become resistant to infection with canine distemper virus
- Rinderpest is rare among Camelidae; especially in endemic areas. They are dead-end hosts and do not transmit the virus

Transmission

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

Sources of virus

- Shedding of virus begins 1–2 days before pyrexia in tears, nasal secretions, saliva, urine and faeces
- Blood and all tissues are infectious before the appearance of clinical signs
- During periods of clinical disease, high levels of RPV can be found in expired air, nasal and ocular discharges, saliva, faeces, semen, vaginal discharges, urine and milk
- Infection is via the epithelium of the upper or lower respiratory tract
- No carrier state

Clinical Signs

- Four forms
  - Classical or Acute form
  - Per acute form
  - Mild Sub acute or Endemic form
  - Atypical form

Classical or Acute form

Clinical disease is characterised by an acute febrile attack within which prodromal and erosive phases can be distinguished

Prodromal period lasts approximately 3 days

- Affected animals develop a pyrexia of between 40 and 41.5°C together with partial anorexia, depression, reduction of rumination, constipation, lowered milk production, increase of respiratory and cardiac rate, congestion of visible mucosae, serous to mucopurulent ocular and nasal discharges, and drying of the muzzle

Erosive phase with development of necrotic mouth lesions

- At height of fever: flecks of necrotic epithelium appear on the lower lip and gum and in rapid succession may appear on the upper gum and dental pad, on the underside of the tongue, on the cheeks and cheek papillae and on the hard palate; erosions or blunting of the cheek papillae
- necrotic material works loose giving rise to shallow, nonhaemorrhagic mucosal erosions

Classical or Acute form- contd

Gastrointestinal signs appear when the fever drops or about 1–2 days after the onset of mouth lesions

- diarrhoea is usually copious and watery at first; later may contain mucus, blood and shreds of epithelium; accompanied, in severe cases, by tenesmus

Diarrhoea or dysentery leads to dehydration, abdominal pain, abdominal respiration, and weakness

Terminal stages of the illness, animals may become recumbent for 24–48 hours prior to death and possible death within 8–12 days

Deaths will occur but mortality rate will be variable; may be expected to rise as the virus gains progressive access to large numbers of susceptible animals

- Initial mortality rates may be in the order of 10–20%

Some animals die while showing severe necrotic lesions, high fever and diarrhoea, others after a sharp fall in body temperature, often to subnormal values

In rare cases, clinical signs regress by day 10 and recovery occurs by day 20–25

Peracute form

- No prodromal signs except high fever (>40–42°C), sometimes congested mucous membranes, and death within 2–3 days

- This form occurs in highly susceptible young and newborn animals

Mild subacute or endemic form

Clinical signs limited to one or more of the classic signs

- Usually no associated diarrhoea
- May show a slight, serous, ocular or nasal secretion
- Fever: variable, short-lived (3–4 days) and not very high (38–40°C)
- No actual depression; animals may continue to graze, water and trek
- Low or no mortality; except in highly susceptible species (buffalo, giraffe, eland, and lesser kudu) in these wild species: fever, nasal discharge, typical erosive stomatitis, gastroenteritis, and death

Atypical form

- Irregular pyrexia and mild or no diarrhoea

➢ fever may remit slightly in the middle of the erosive period, and

➢ 2–3 days later, return rapidly to normal accompanied by a quick resolution of the mouth

- Lesions, a halt to the diarrhoea and an uncomplicated convalescence

➢ The lymphotropic nature of RPV leads to immunosuppression and favours recrudescence of

- Latent infections and/or increased susceptibility to other infectious agents

Nasal discharge due to Rinderpest

Occular discharge due to Rinderpest

Lethargic calf with Rinderpest

Dehydrated and dead calf with Rinderpest

Inflamed vaginal lining with Rinderpest

Cow in field with diarrhea

Oral erosion in Rinderpest

Dead carcass with occular and nasal discharge

Convalescence after profuse diarrhea in Rinderpest

Stamping out

Lesions

- Either areas of necrosis and erosions, or congestion and haemorrhage in the mouth, intestines and upper respiratory tracts
- Highly engorged or grey discolouration of abomasum (epithelial necrosis of mucous membrane);
- pyloric region severely affected and shows congestion, petaechiation and oedema of the submucosa
- Rumen, reticulum and omasum usually unaffected; necrotic plaques are occasionally encountered on the pillars of the rumen
- Enlarged and oedematous lymph nodes

Lesions - contd

- White necrotic foci in Peyer’'s patches; lymphoid necrosis and sloughing leaves the supporting rchitecture engorged or blackened
- Linear engorgement and blackening of the crests of the folds of the caecum, colon and rectum (‘Zebra striping’)
- Typically the carcass of the dead animal is dehydrated, emaciated and soiled
- Histologically, evidence of lymphoid and epithelial necrosis accompanied by viral associated syncytia and intracytoplasmic inclusions
- In milder form of rinderpest: most domestic animals escape development of erosions

➢some may develop slight congestion of mucous membranes and small, focal areas of raised, whitish epithelial necrosis may be found on the lower gum (no larger than a pin head); possibly a few eroded cheek papillae

Lesions - contd

- In milder form of rinderpest in wild animals

➢ African buffaloes infected with milder RPV lineage 2 have demonstrated enlarged peripheral lymph nodes, plaque-like keratinised skin lesions and keratoconjunctivitis

➢ Lesser kudus were similarly affected with blindness due to severe keratoconjunctivitis but no diarrhoea

➢ Necrosis and erosions of the buccal mucosa together with dehydration and emaciation

Red swollen payer patches or zebra stripping

Swallen mucosal fold or zebra sripping

Congestion and haemorrhage of abomasum

Differential diagnosis

Cattle

- Bovine viral diarrhoea/mucosal disease
- Malignant catarrhal fever
- Infectious bovine rhinotracheitis
- Foot and mouth disease
- Papular stomatitis
- Jembrana disease
- Vesicular stomatitis
- Contagious bovine pleuropneumonia
- Theileriosis (East Coast fever)
- Salmonellosis
- Necrobacillosis
- Paratuberculosis
- Arsenic poisoning

Differential diagnosis - contd

Small ruminants

- Peste des petits ruminants
- Nairobi sheep disease
- Contagious caprine pleuropneumonia
- Pasteurellosis

Swine

- *Campylobacter spp.*
- *Brachyspira hyodyesntereiae*
- Salmonellosis

Laboratory diagnosis

- Virus isolation
- Antigen detection by Agar Gel Immunodiffusion Test
- Histopathology and Immunohistochemistry
- Lineage Identification by using RT-PCR
- Differential Immunocapture ELISA
- Chromatographic strip test
- Serological test
  - Virus neutralization test
  - Competitive ELISA

Prevention and Control

- Sanitary Prophylaxis
  - Quarantine and Isolation
  - Cleaning of premices

- Medical Prophylaxis
  - Vaccination

Malignant Catarrhal Fever

Malignant Catarrh, Malignant Head Catarrh, Gangrenous Coryza, Catarrhal Fever, Snotsiekte

Overview

- • Organism
- • Economic Impact
- • Epidemiology
- • Transmission
- • Clinical Signs
- • Diagnosis and Treatment
- • Prevention and Control
- • Actions to Take

Center for Food Security and Public Health, Iowa State University, 2011

The Organism

The Organism

- Herpesviridae

–Genus *Rhadinovirus*

- Multiple serotypes

– Species and geographically dependent

- AHV-1 natural host: wildebeest in Africa
- OHV-2 natural host: domestic sheep and goats worldwide
- AHV-2 nonpathogenic
- CpHV-2 natural host: domestic goats

Importance

History

- Cases occur worldwide each year

- MCF in wildebeests in Africa for centuries

- U.S.
  - In cattle since 1920s
  - First bison case in 1973 (South Dakota)
  - Problem in zoo animals

- New Jersey exotic theme park, 2002

- AHV-1 diagnosed in Ankoli cattle

Economic Impact

- Variable given the carrier status
- Zoologic parks affected through losses of expensive animals
- Not reportable in all 50 states
  - Tracking true losses difficult
- Concern for bison breeders, cattle producers, elk and deer farmers

Epidemiology

Geographic Distribution

- AHV-1 primarily in Africa
  – Carried by wildebeest, hartebeest, topi
  – Also in zoologic and wild animal parks

- OHV-2 worldwide
  – Carried by domestic and wild sheep and goats
  –Major cause of MCF worldwide

Geographic Distribution

- Positive bison have been found in U.S. and Canada, Utah, Wyoming, Colorado, Montana, California, Oregon, Ohio, Kansas, Nebraska, North Dakota, South Dakota– Saskatchewan, Ontario,and Alberta

- Often misdiagnosed in bison

Morbidity/Mortality

- Carrier species asymptomatic
  – Wildebeest, hartebeest, topi, sheep, goats

- Low morbidity in other species
  – U.S. outbreaks 30 to 40%
  – < 1% in water buffalo, deer

- Mortality 100%
  – Domestic cattle, white-tailed, axis, Pere David's deer

Transmission

Animal Transmission

- AHV-1

–Wildebeest calves

- In utero

- Contact with nasal and ocular secretions

- Aerosols during close contact

– Adult wildebeest

- Cell-associated form

- Rarely transmitted

Animal Transmission

OHV-2

– Respiratory (aerosol)

– Transplacental rare

– Contact with nasal

secretions

– Animal-to-animal rare

Dead end hosts

Human Transmission

- MCF has not been documented as causing disease in humans

- Caution at lambing time
  – Equipment used could spread infection to susceptible animals

- Virus quickly inactivated by sunlight
  –Minimizes risk of fomite spread

Animals and Malignant Catarrhal Fever

Species Affected

- Carrier species
  - Sheep, goats, wildebeest, hartebeest, topi

- Susceptible species
  - Cattle, bison, elk, reindeer, moose, domestic pigs, giraffe, antelope, wapiti, red and white-tailed deer, Pere David's deer, white-tailed & white-bearded gnu, gaur, greater kudo, Formosan sika deer, axis deer, nilgai, banteng

Clinical Signs

- Incubation period 9 to 77 days experimentally

–Unknown in natural infections

– Subclinical infections develop under stress

- Initial clinical signs

– Depression, diarrhea, DIC, dyspnea, high fever, inappetence

– Sudden death

Clinical Signs

- Peracute form: sudden death

- Head and eye form

–Majority of cattle cases

- Intestinal form

– Initially like head and eye form, but death occurs from severe diarrhea

- Mild form

– Inoculated animals; recovery expected

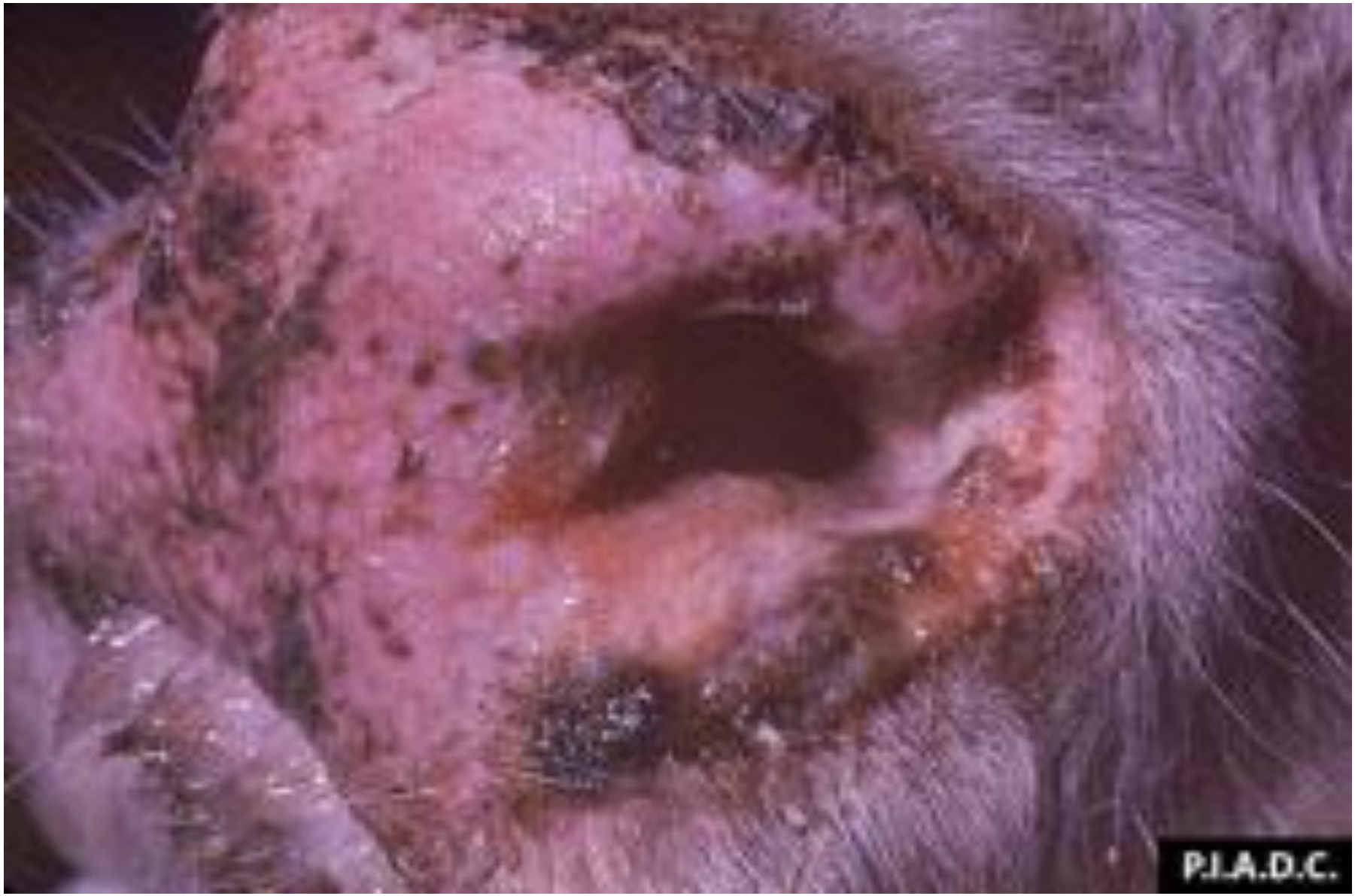
Post Mortem Lesions

- Erosions on the tongue and soft and hard palate
- Necrotic areas in the omasal epithelium
- Multiple erosions of intestinal epithelium
- Greatly enlarged lymph node compared to normal
- Necrotic areas in the larynx
  - Diptheritic membrane often present

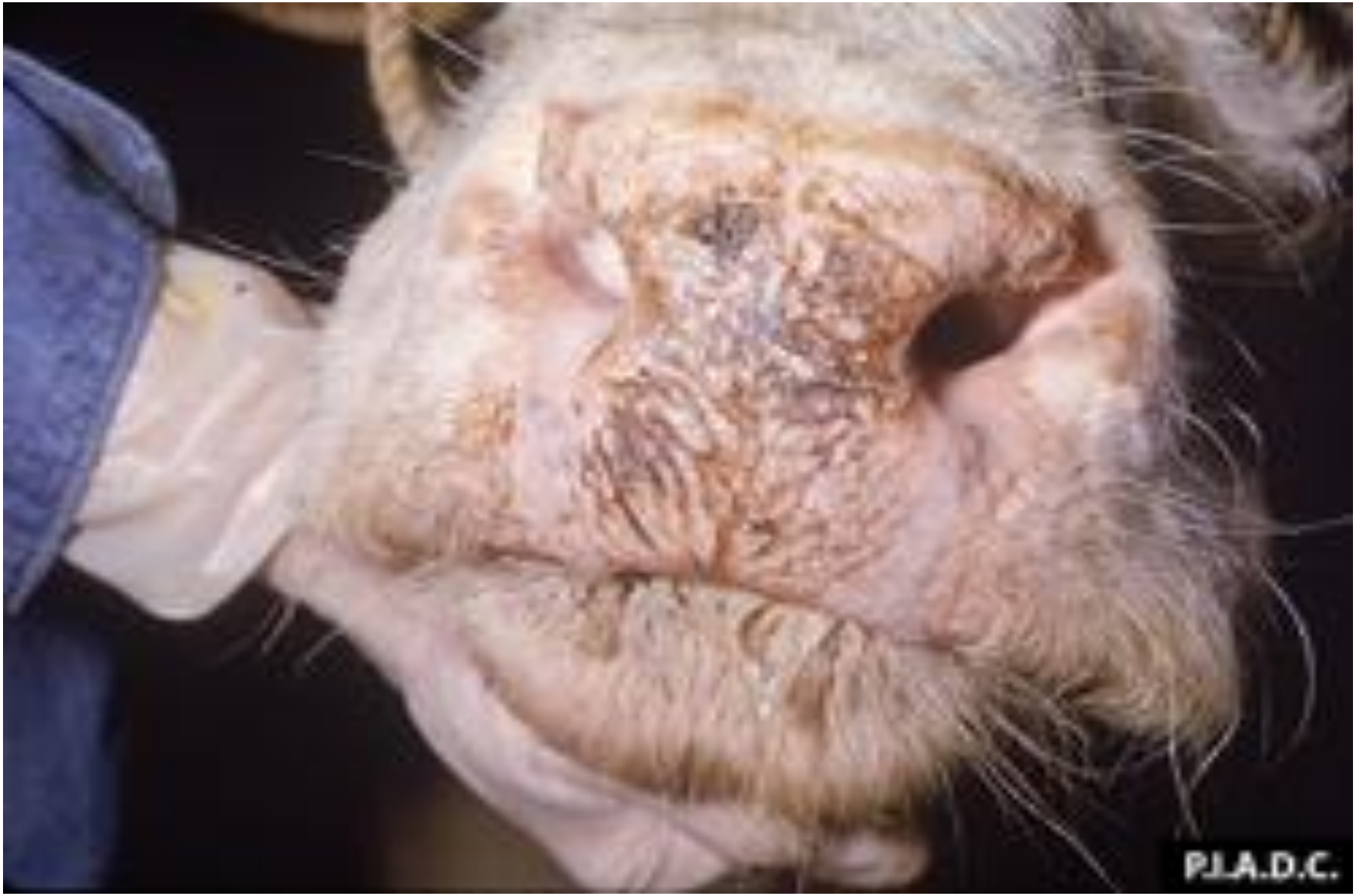
Post Mortem Lesions

- Urinary bladder mucosa hyperemic and edematous

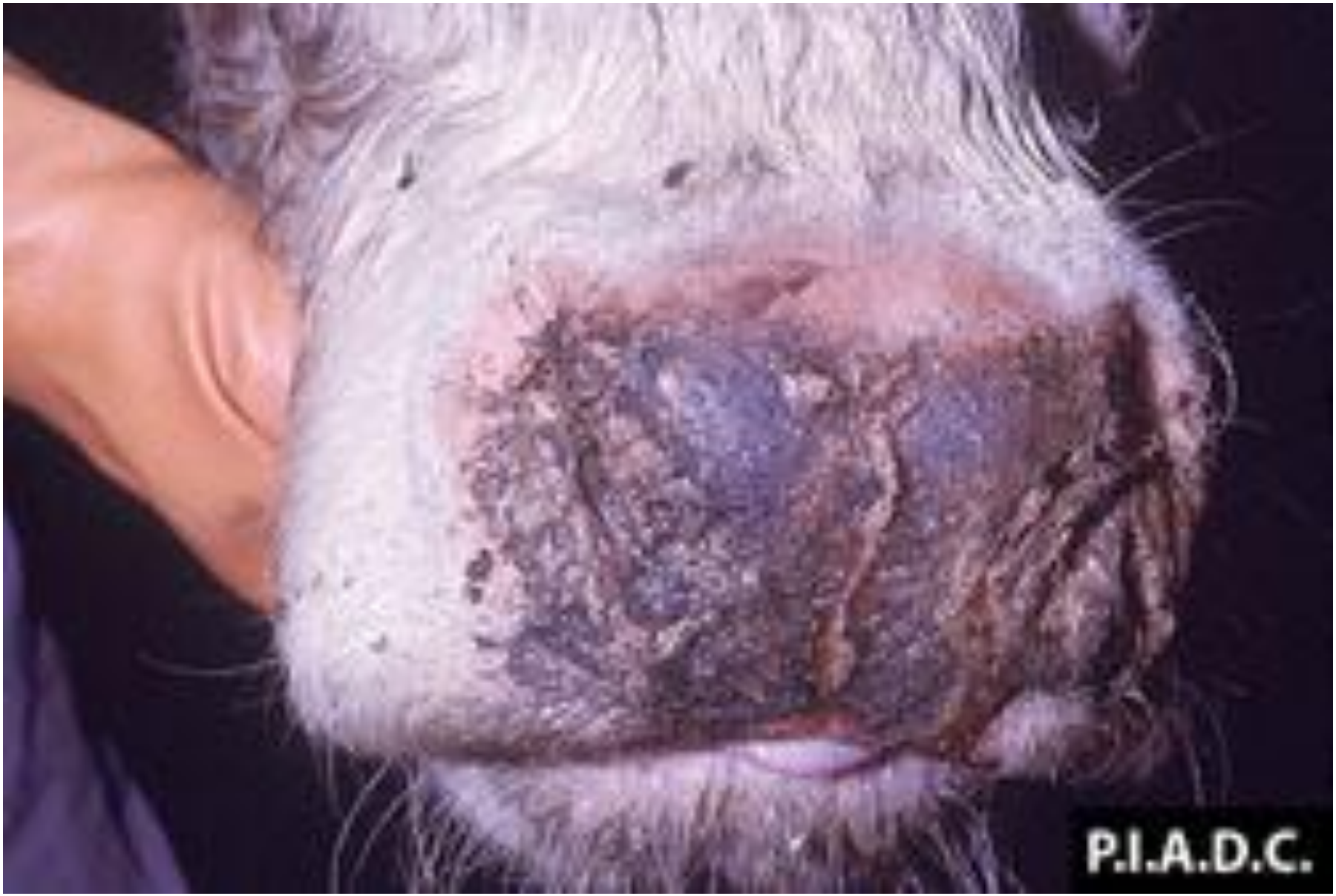
- Kidney often has raised white foci on the cortex



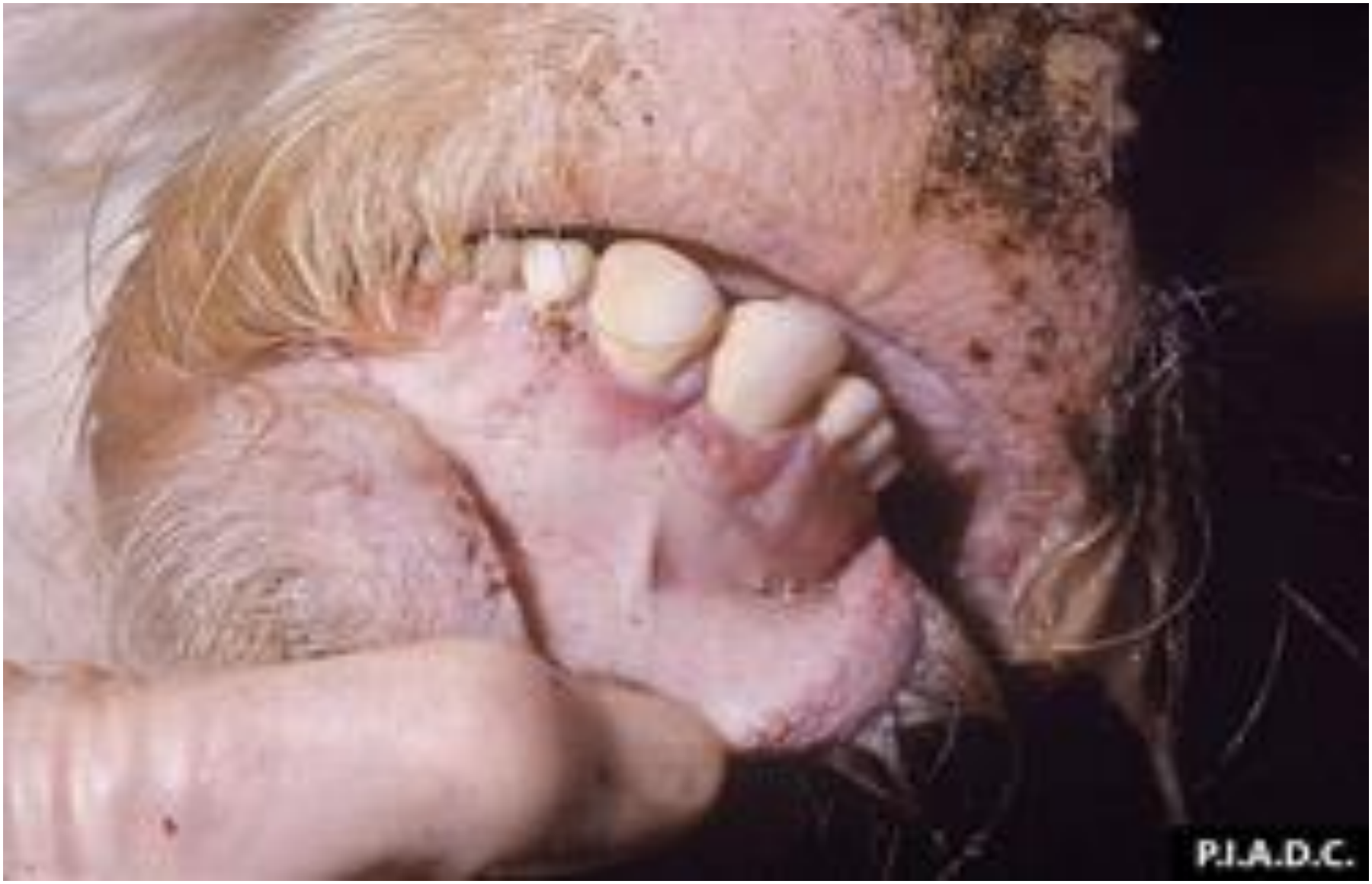
The muzzle is hyperemic, multifocally covered by adherent mucopurulent exudate, and contains many shallow erosions.



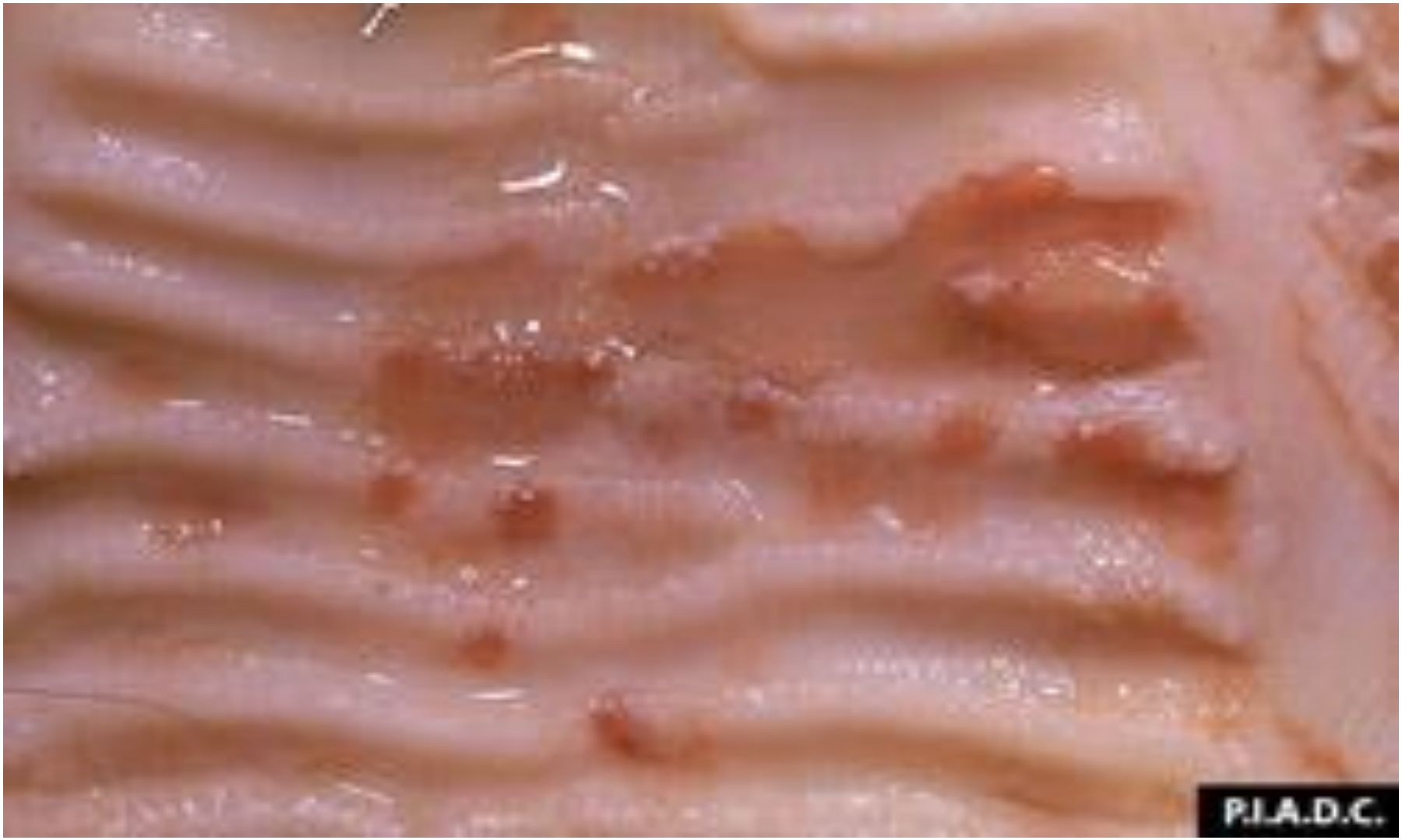
Bovine muzzle showing multiple shallow erosions filled with nasal exudate



Bovine, muzzle. There is diffuse superficial necrosis of the muzzle.

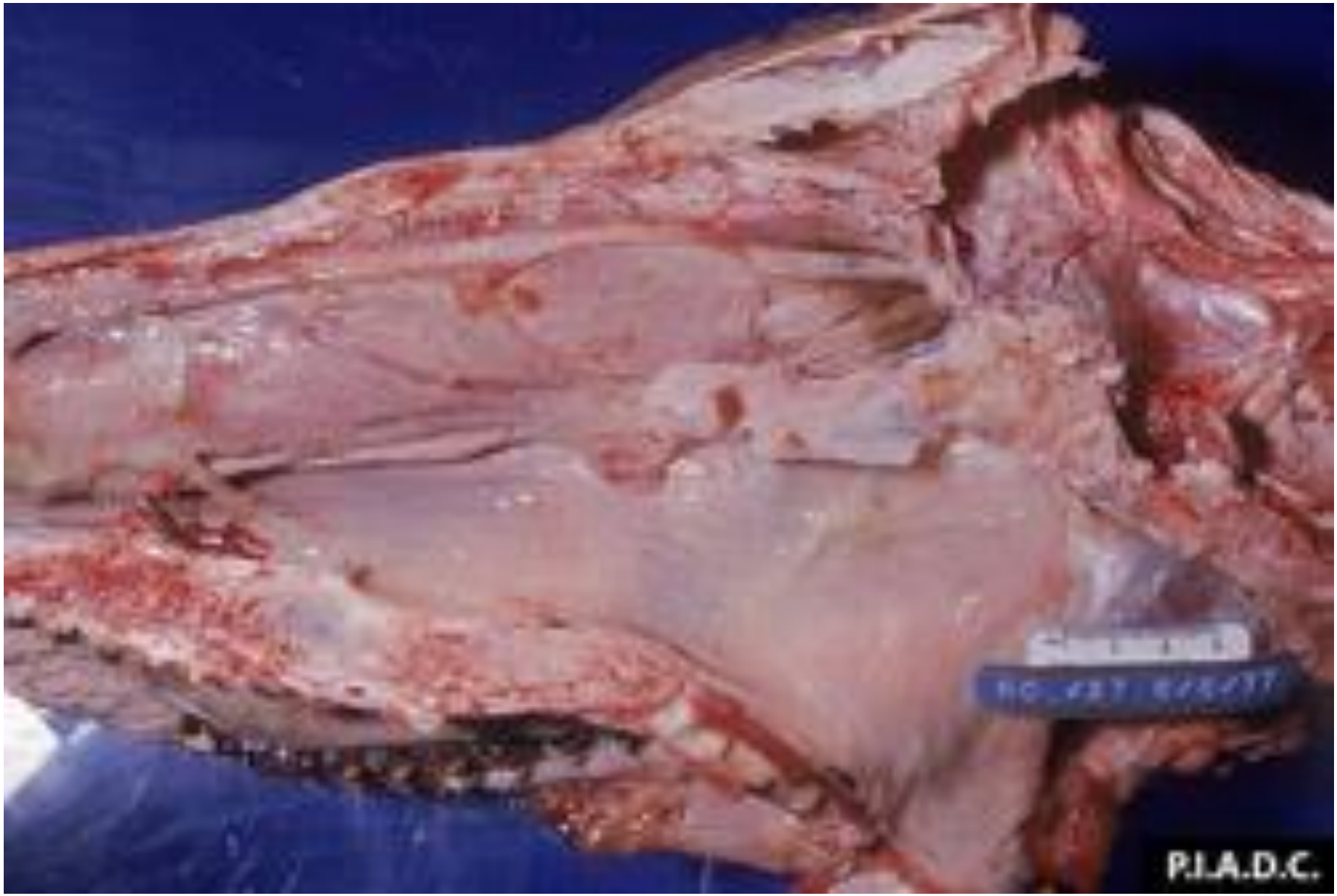


Bovine, oral mucosa. There is a gingival hyperemia and focal erosion.

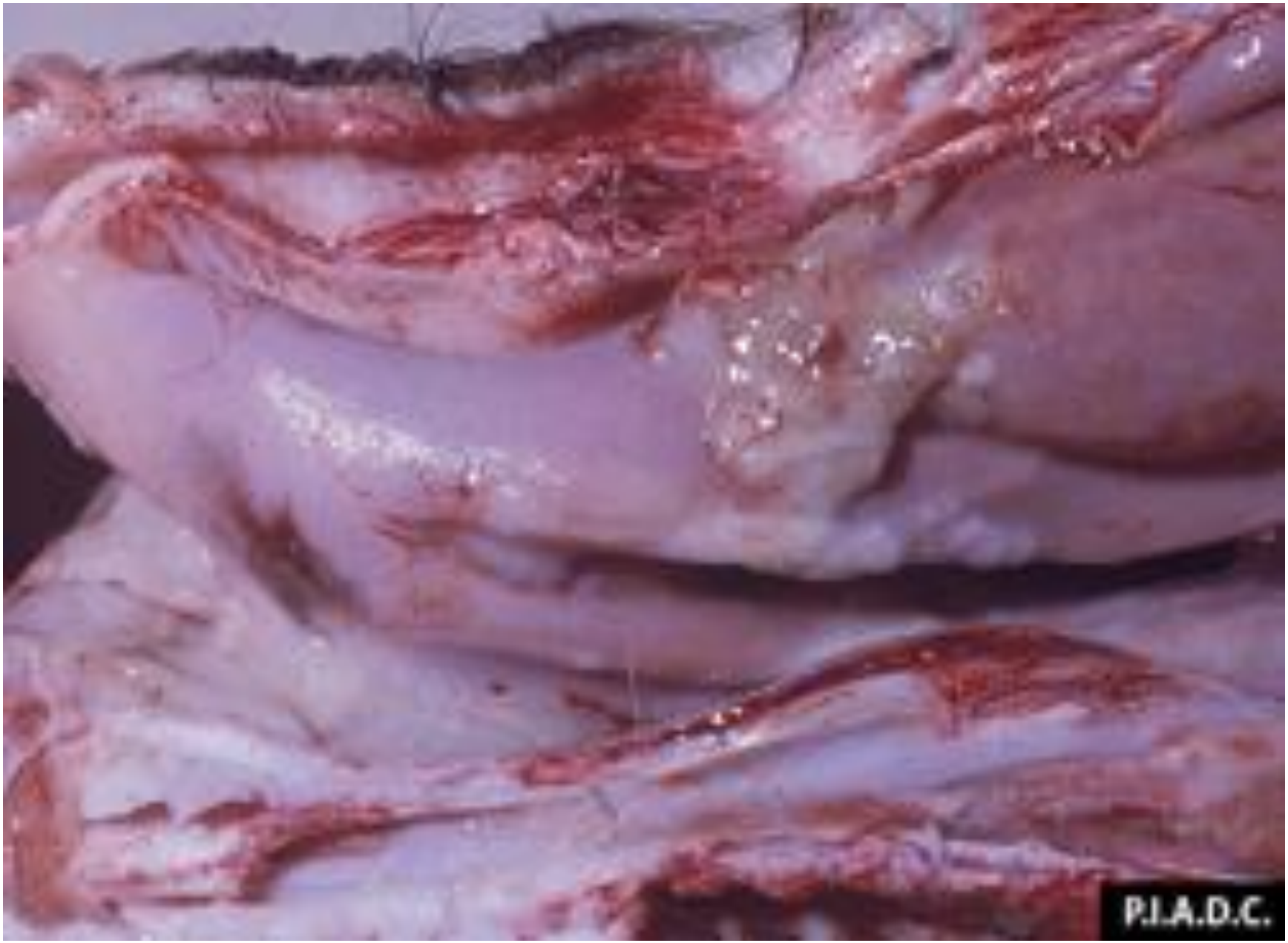


Bovine, hard palate. There are multiple coalescing mucosal erosions.

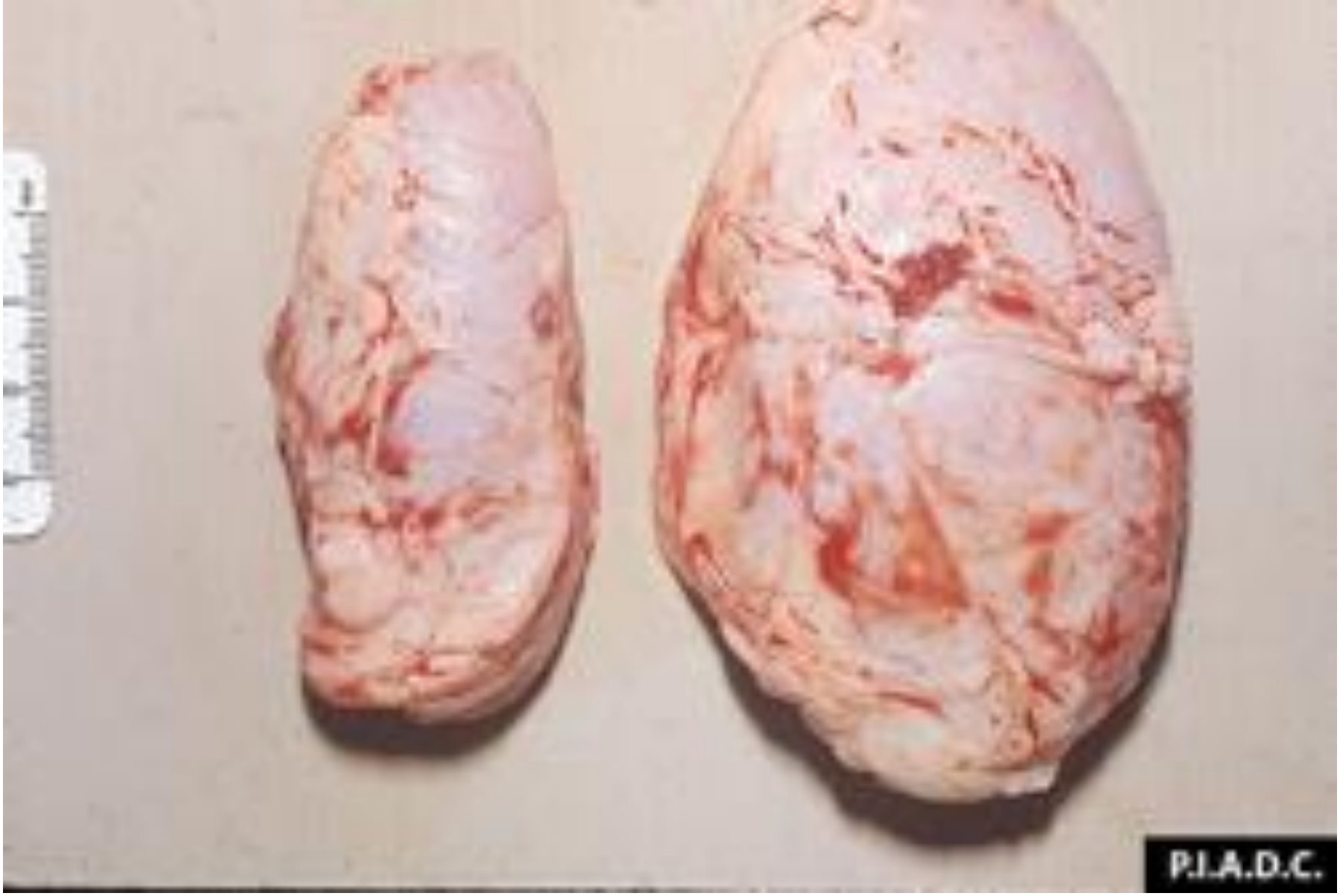
Bovine, skin. There are numerous raised plaques (multifocal dermatitis).



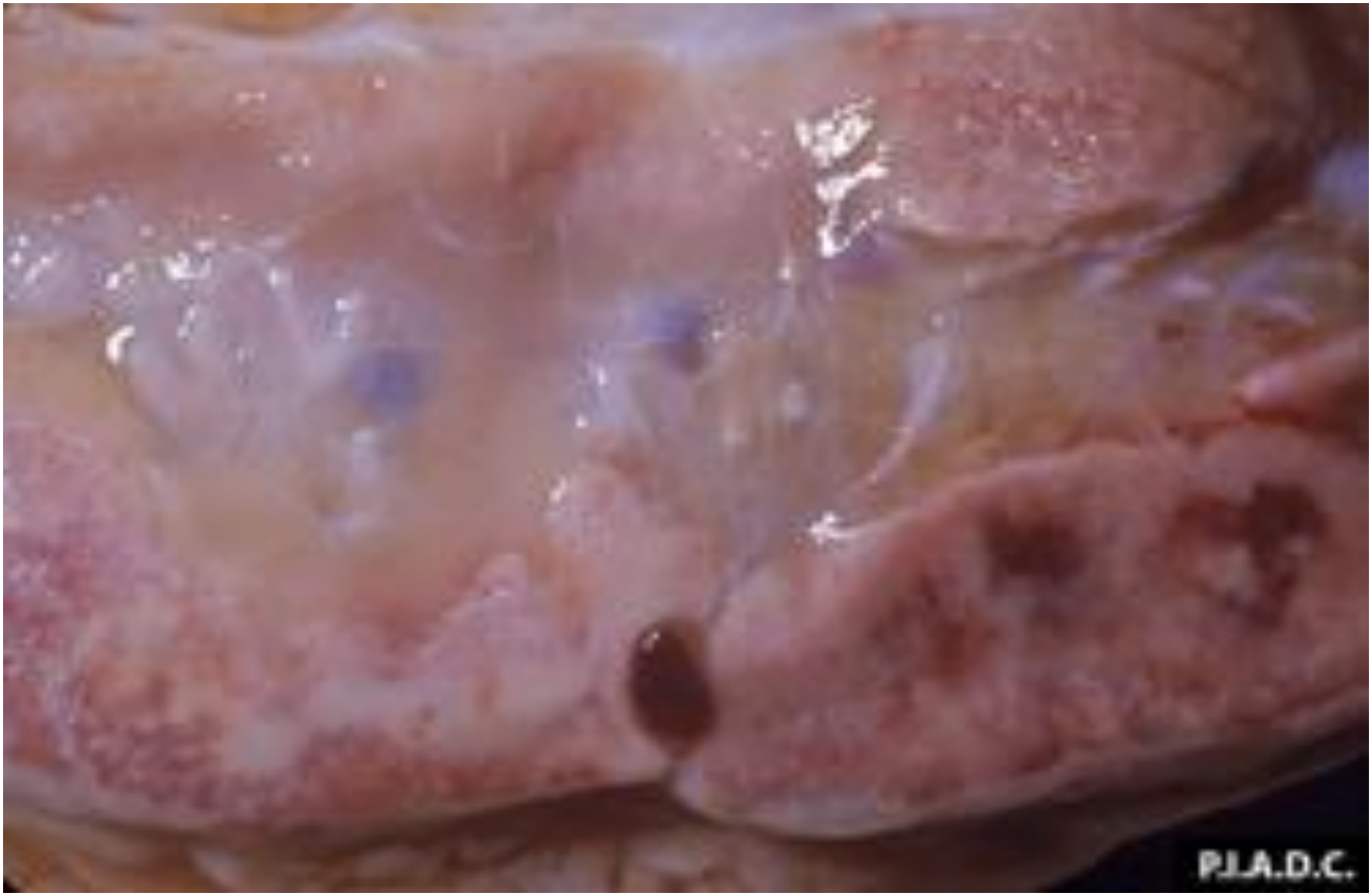
Bovine, head, sagittal section. Mucoid exudate multifocally covers the nasal and pharyngeal mucosa.



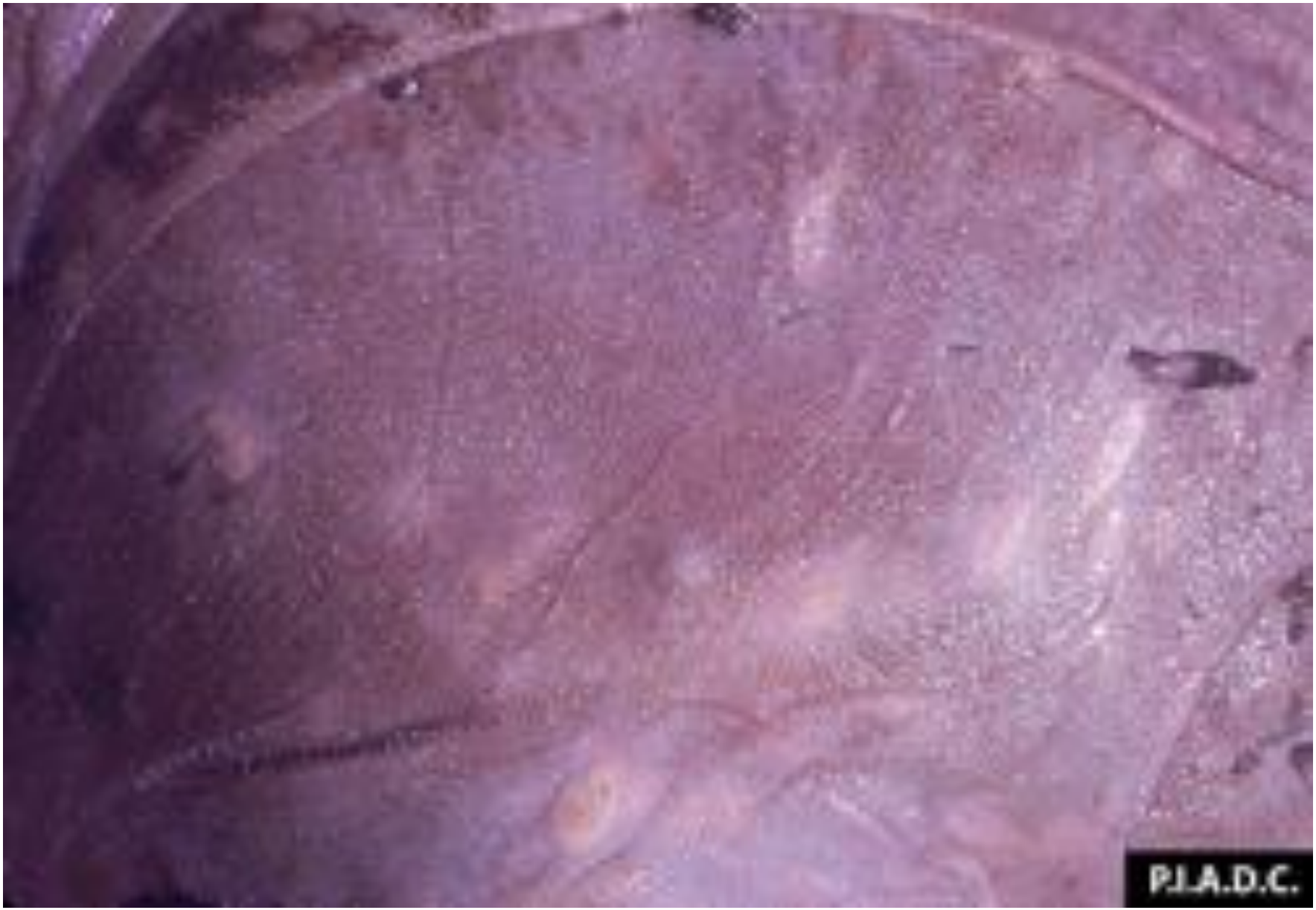
Bovine, nasal turbinate. There is a small amount of mucoid exudate.



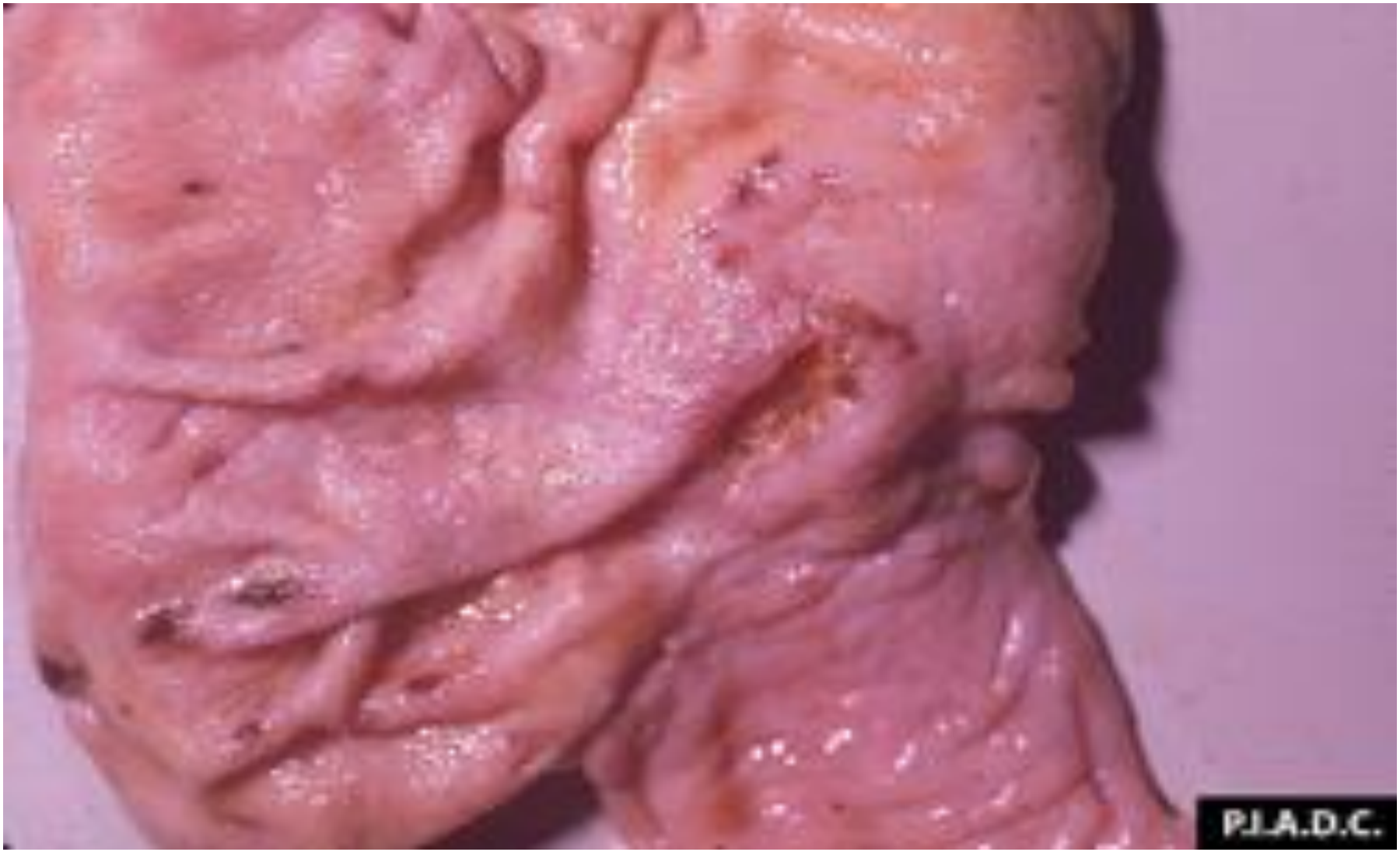
Bovine, prescapular lymph nodes: Moderately (left) to markedly enlarged (right) due to MCF.



Bovine, prescapular lymph node. There are foci of hemorrhage (and necrosis) in the cortex, and the medulla is edematous.

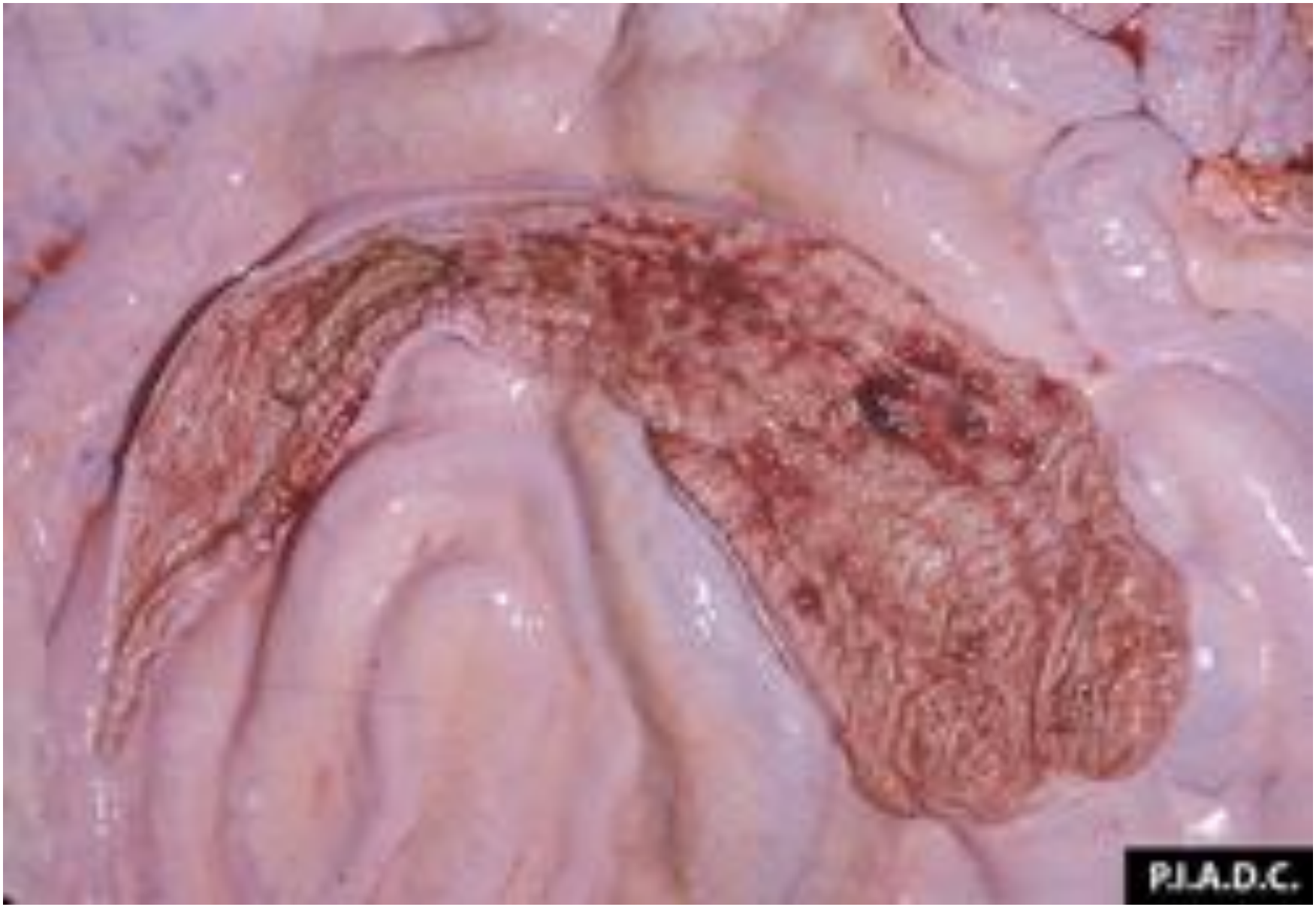


Bovine, omasum. Omasal leaves contain multiple pale foci of necrosis; on the rigth there are several ulcers.



Bovine, cecum and ileum. There are scattered small foci of mucosal hemorrhage and erosion.

Bovine, colon. There is severe longitudinal linear congestion of the mucosa.



Bovine, spiral colon. There are multiple mucosal hemorrhages.

Differential Diagnosis

- BVD mucosal disease
- Bluetongue
- Rinderpest
- FMD
- Vesicular stomatitis
- Salmonellosis
- Pneumonia complex
- Oral exposure to caustic materials
- Mycotoxins
- Poisonous plants

Sampling

- Before collecting or sending any samples, the proper authorities should be contacted

- Samples should only be sent under secure conditions and to authorized laboratories to prevent the spread of the disease

Clinical Diagnosis

- Any susceptible animal with sudden death, fever, erosions of the mucosa, nasal/lacrimal discharge, or bilateral corneal opacity should be tested for MCF– Particularly with a history of exposure to sheep, goats, antelope, or wildebeest during parturition

Laboratory Diagnosis

- Histopathology
- PCR
- Virus isolation (AHV-1)
- Serology

–AHV-1 antibodies in wildebeest

- Immunofluorescence, immunoblot, VN, ELISA, immunocytochemistry–OHV-2 antibodies in sheep
- Immunofluorescence, immunoblot

Treatment

- Survival is rare if clinically ill
- Mortality reaches 100%
- Supportive therapy, antibiotics for secondary bacterial infection– Recovered animals will remain virus carriers

BLUE TONGUE

Synonyms:-

- Sore Muzzle
- Pseudo Foot-and-Mouth Disease
- Muzzle Disease

Etiology

- Bluetongue results from infection by the bluetongue virus, a member of the genus Orbivirus and family Reoviridae.

- Twenty-four serotypes have been identified worldwide; six serotypes (1, 2, 10, 11, 13, and 17) have been found in domesticated or wild ruminants in the United States.

- Bluetongue viruses are closely related to the viruses in the epizootic hemorrhagic disease (EHD) serogroup.

Species Affected

- Bluetongue virus infects many domesticated and wild ruminants including sheep, goats, cattle, buffalo, deer, antelope, bighorn sheep and North American elk.

- Clinical disease is seen often in sheep, occasionally in goats, and rarely in cattle.

- Severe disease can also occur in some wild ruminants including white-tailed deer (*Odocoileus virginianus), pronghorn (Antilocapra americana) and desert bighorn sheep (Ovis canadensis).*

- In Africa, some large carnivores have antibodies to bluetongue, and, in the United States, a contaminated vaccine resulted in some abortions and deaths in pregnant dogs.

Geographic Distribution

- The bluetongue virus has been found in many parts of the world including Africa, Europe, the Middle East, Australia, the South Pacific, North and South America, and parts of Asia.

- The virus is present in some regions without associated clinical disease.

- In the United States, the distribution of the vector limits infections to the southern and western states.

Transmission

- Bluetongue virus is transmitted by biting midges in the genus Culicoides. Culicoides varipennis var sonorensis is the principal vector in the United States, C. brevitarsis in Australia, and C. imicola in Africa and the Middle East.
- C. imicola is also the major vector in southern Europe, but C. dewulfi has been identified as the vector in the 2006 northern European outbreaks.
- Other Culicoides species can also transmit the virus and may be important locally.
- Ticks or sheep keds can be mechanical vectors but are probably of minor importance in disease transmission.
- Cattle are the major amplifying host due to their prolonged viremia and the feeding preferences of many Culicoides species.

Incubation Period

- In sheep, the incubation period is usually 5 to 10 days.
- Cattle can become viraemic starting at four days post-infection, but rarely develop symptoms.
- Animals are usually infectious to the insect vector for several weeks.

Clinical Signs

- The vast majority of infections with bluetongue are clinically inapparent.
- In sheep, the clinical signs may include fever, excessive salivation, depression, dyspnea and panting. Initially, animals have a clear nasal discharge; later, the discharge becomes mucopurulent and dries to a crust around the nostrils.
- The muzzle, lips and ears are hyperemic, and the lips and tongue may be very swollen. The tongue is occasionally cyanotic and protrudes from the mouth.
- The head and ears may also be edematous. Erosions and ulcerations are often found in the mouth; these lesions may become extensive and the mucous membranes may become necrotic and slough.

Clinical Signs-contd

- The coronary bands on the hooves are often hyperemic and the hooves painful; lameness is common and animals may slough their hooves if they are driven.

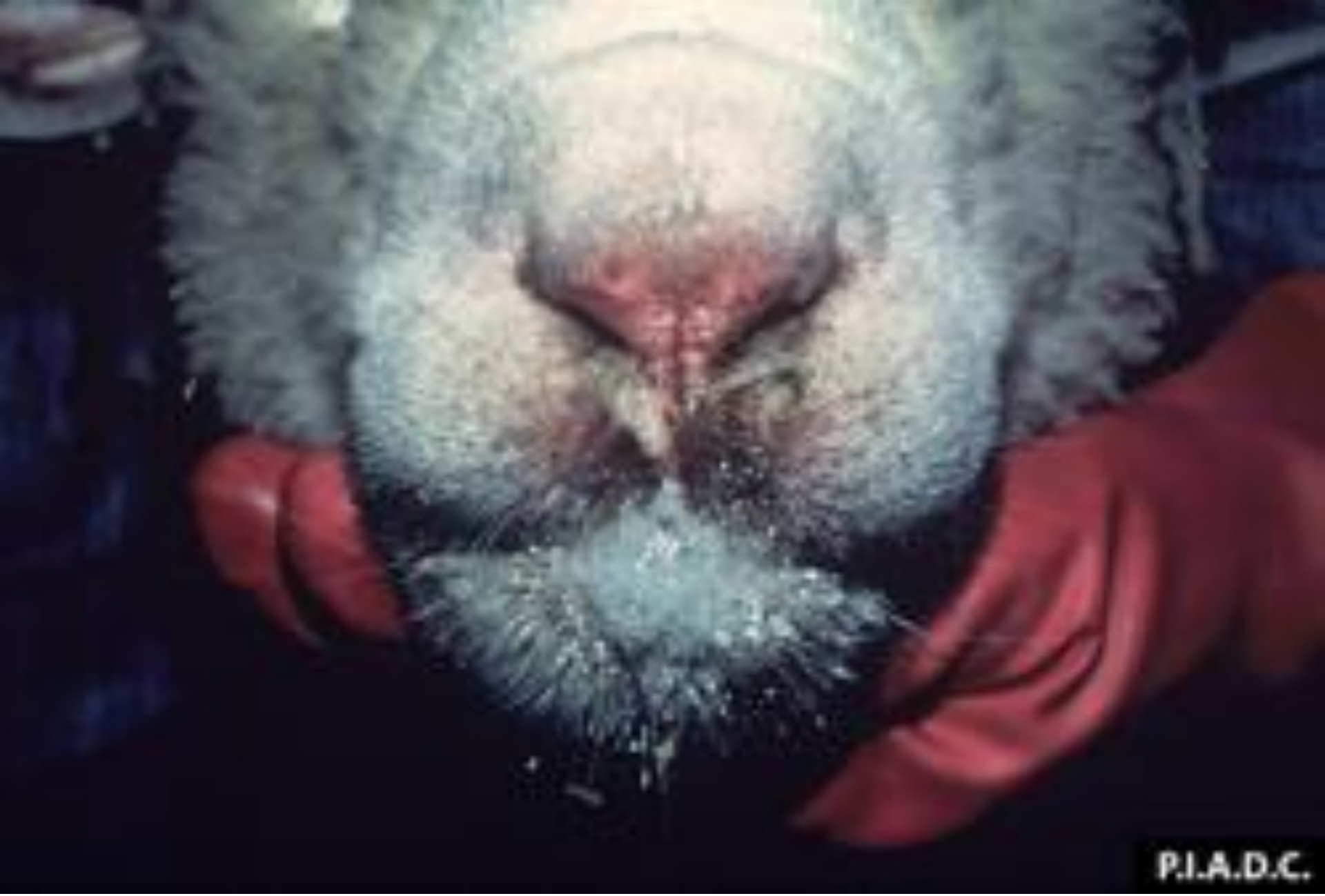
- Pregnant ewes may abort their fetuses, or give birth to “dummy” lambs. Additional clinical signs can include torticollis, vomiting, pneumonia or conjunctivitis.

- The death rate varies with the strain of virus. Three or four weeks after recovery, some surviving sheep can lose some or all of their wool.

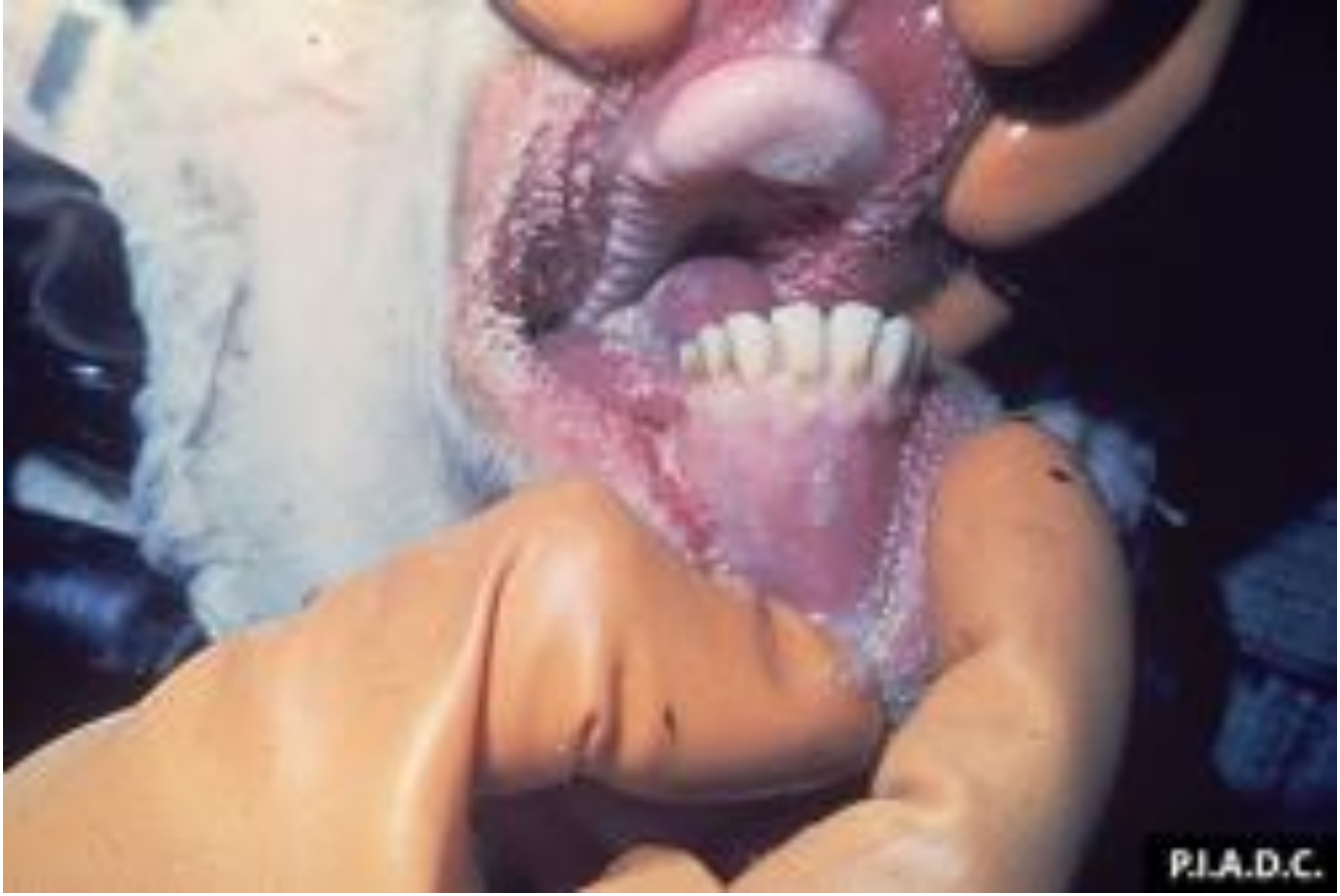
- Recrudescence of clinical disease has been reported in sheep, possibly as the result of persistent infections in ovine T-lymphocytes.

Clinical Signs-contd

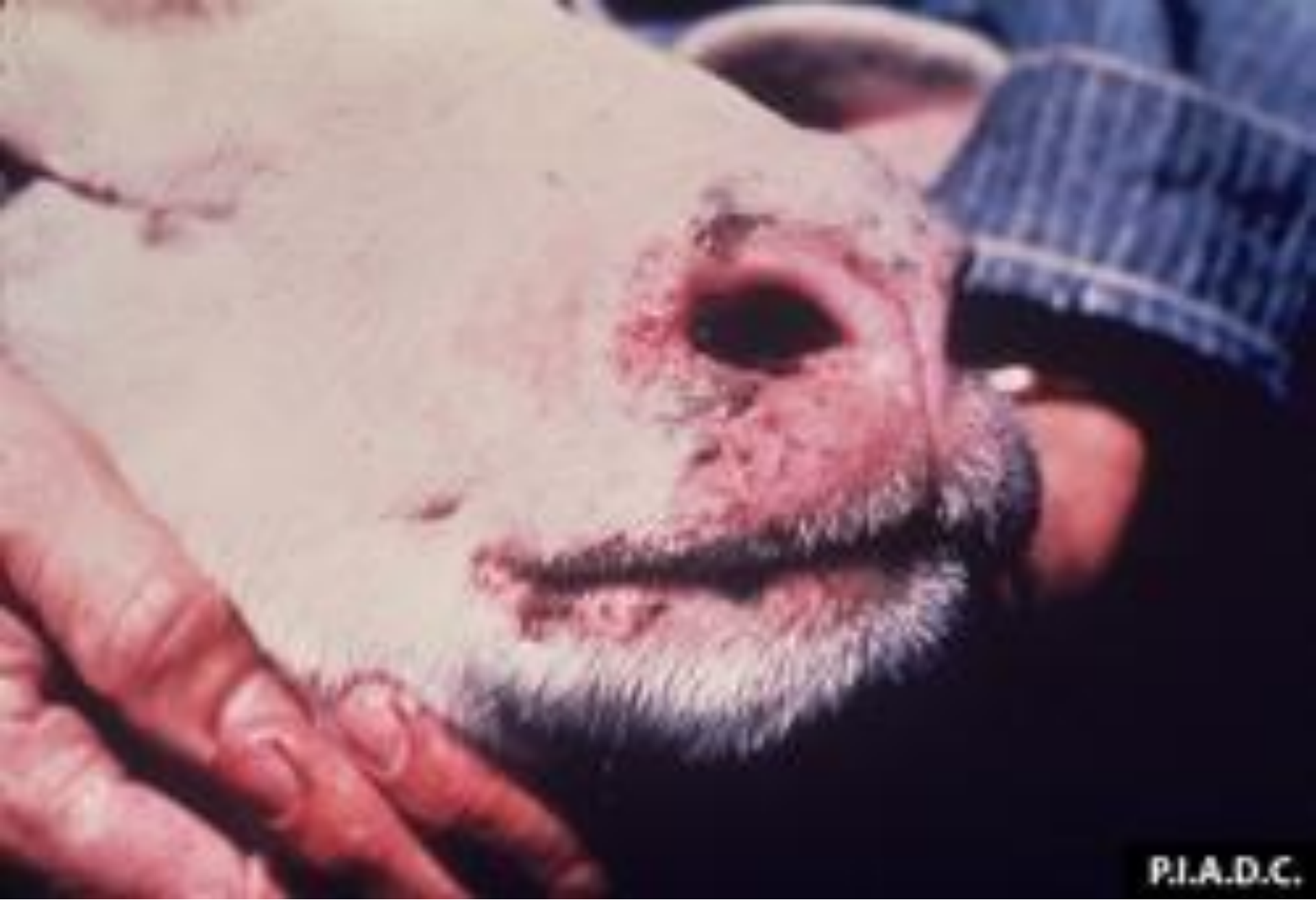
- Infections in cattle are usually subclinical; often, the only signs of disease are changes in the leukocyte count and a fluctuation in rectal temperature.
- Rarely, cattle have mild hyperemia, vesicles or ulcers in the mouth; hyperemia around the coronary band; hyperesthesia; or a vesicular and ulcerative dermatitis.
- The skin may develop thick folds, particularly in the cervical region. The external nares may contain erosions and a crusty exudate.
- Temporary sterility may be seen in bulls. Infected cows can give birth to calves with hydranencephaly or cerebral cysts.
- Cattle that have clinically apparent disease may develop severe breaks in the hooves several weeks after infection; such breaks are usually followed by foot rot. Infections in goats are usually subclinical, and similar to disease in cattle.



Sheep, Bilateral nasal discharge, Excessive salivation, erosion of nasal planum



Sheep, mouth. There is linear erosion and reddening of the right buccal mucosa.



Sheep. There are multiple erosions and crusts on the muzzle and lips.

Post mortem Lesions

- In sheep, the face and ears are often edematous.

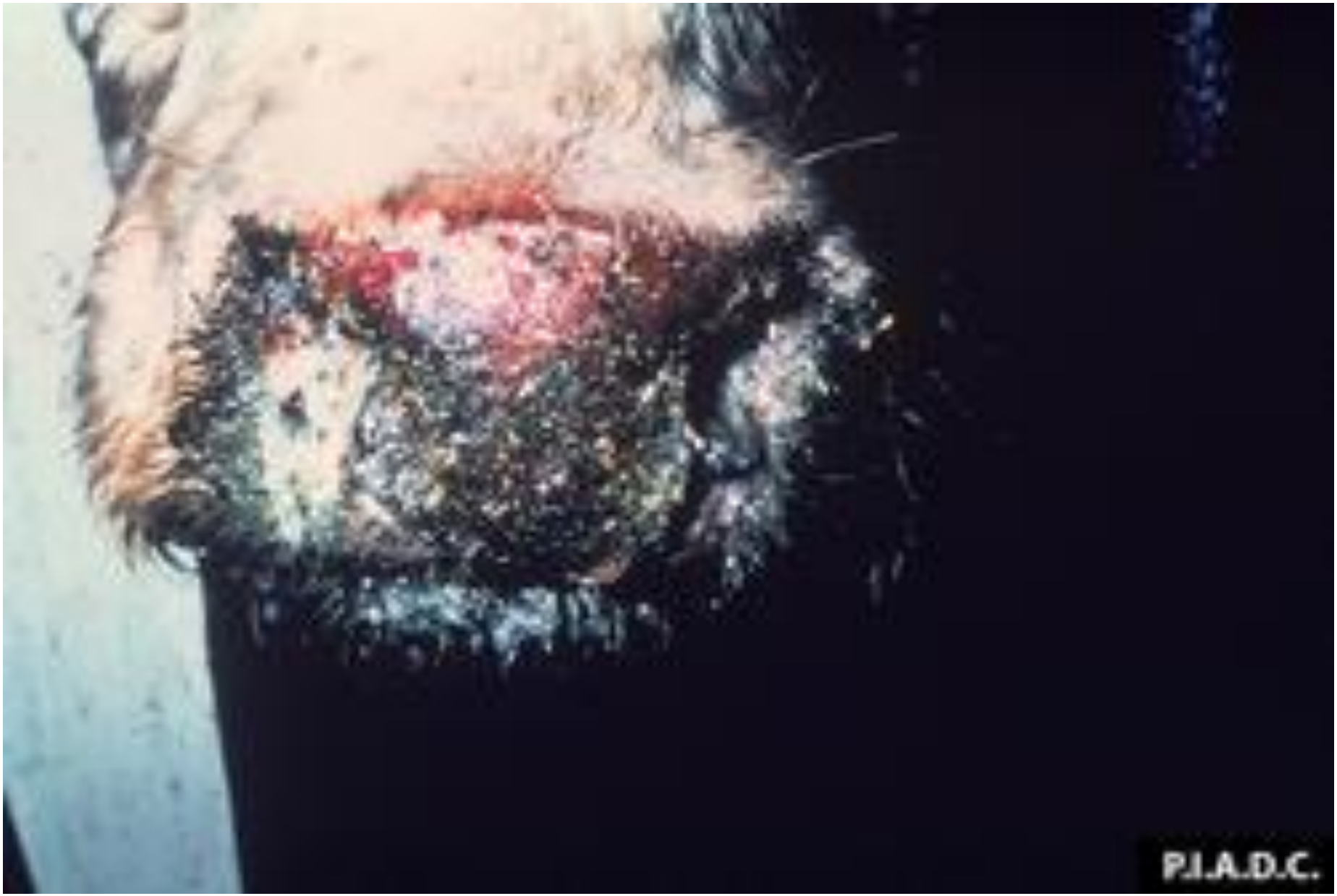
- A dry, crusty exudate may be seen on the nostrils. The coronary bands of the hooves are often hyperemic; petechial or ecchymotic hemorrhages may be present and extend down the horn.

- Petechiae, ulcers and erosions are common in the oral cavity, particularly on the tongue and dental pad, and the oral mucous membranes may be necrotic or cyanotic.

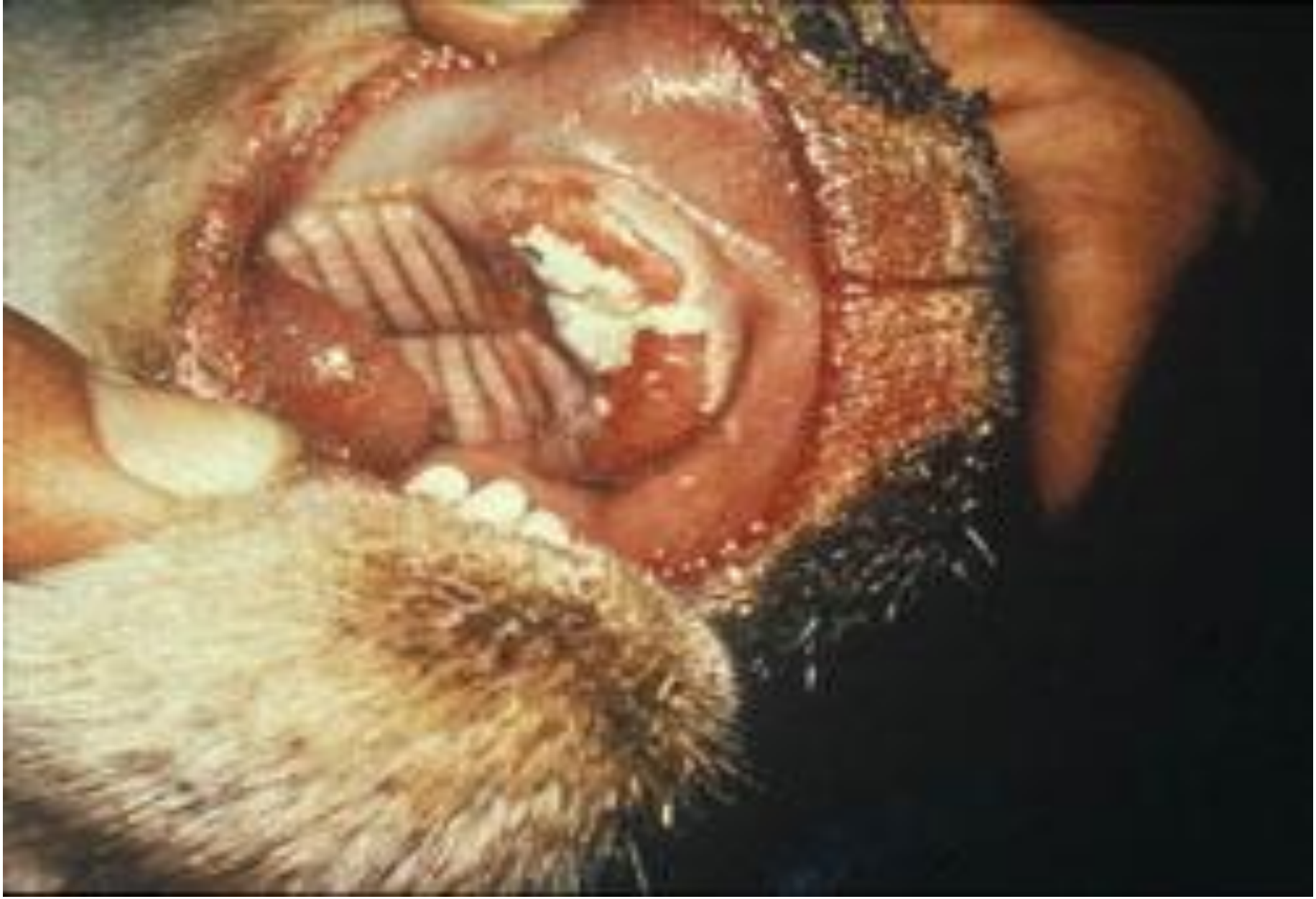
- The nasal mucosa and pharynx may be edematous or cyanotic, and the trachea hyperemic and congested. Froth is sometimes seen in the trachea, and fluid may be found in the thoracic cavity.

Post mortem Lesions-contd

- Hyperemia and occasional erosions may be seen in the reticulum and omasum. Petechiae, ecchymoses and necrotic foci may be found in the heart.
- In some cases, hyperemia, hemorrhages and edema are found throughout the internal organs. Hemorrhage at the base of the pulmonary artery is particularly characteristic of this disease.
- In addition, the skeletal muscles may have focal hemorrhages or necrosis, and the intermuscular fascial planes may be expanded by edema fluid.
- In deer, the most prominent lesions are widespread petechial to ecchymotic hemorrhages.
- More chronically infected deer may have ulcers and necrotic debris in the oral cavity.
- They may also have lesions on the hooves, including severe fissures or sloughing.

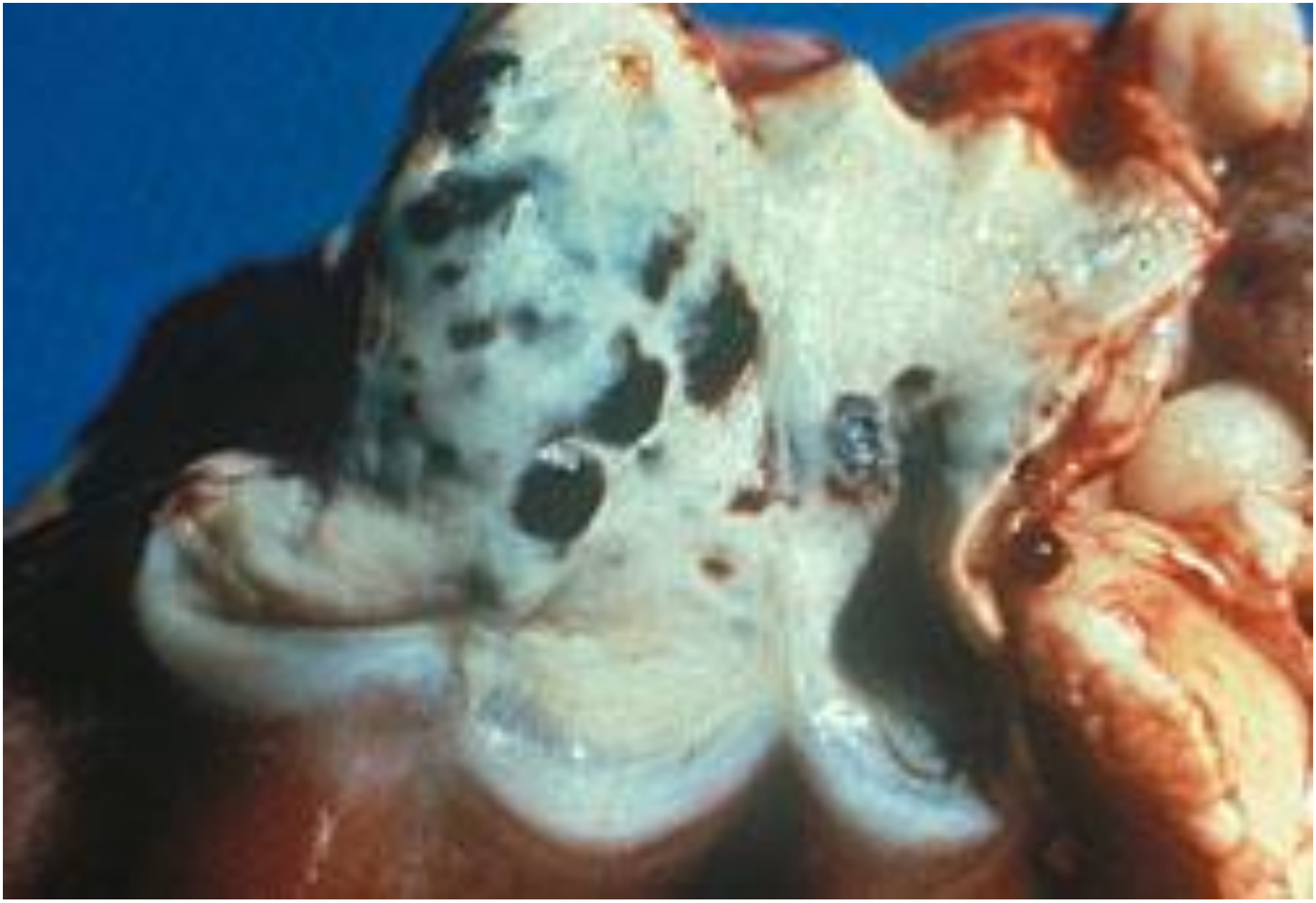


Bovine. The muzzle is covered by an adherent crust, and the underlying (eroded) tissue is hyperemic.



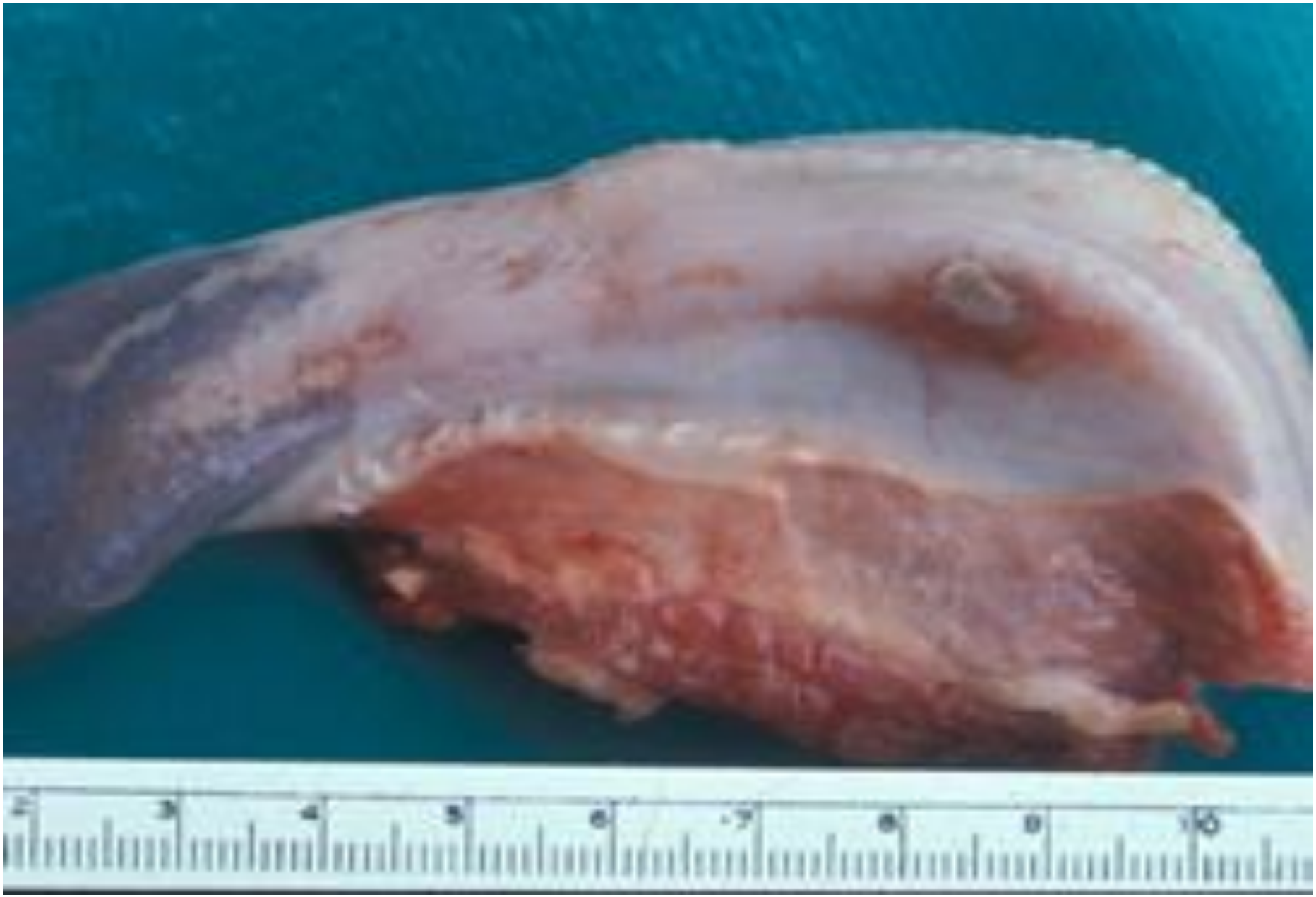
Sheep, mouth. Most of the dental pad is eroded; the remaining pale mucosa is necrotic.

Bovine, mammary gland. There is extensive coalescing ulceration of the teat skin.

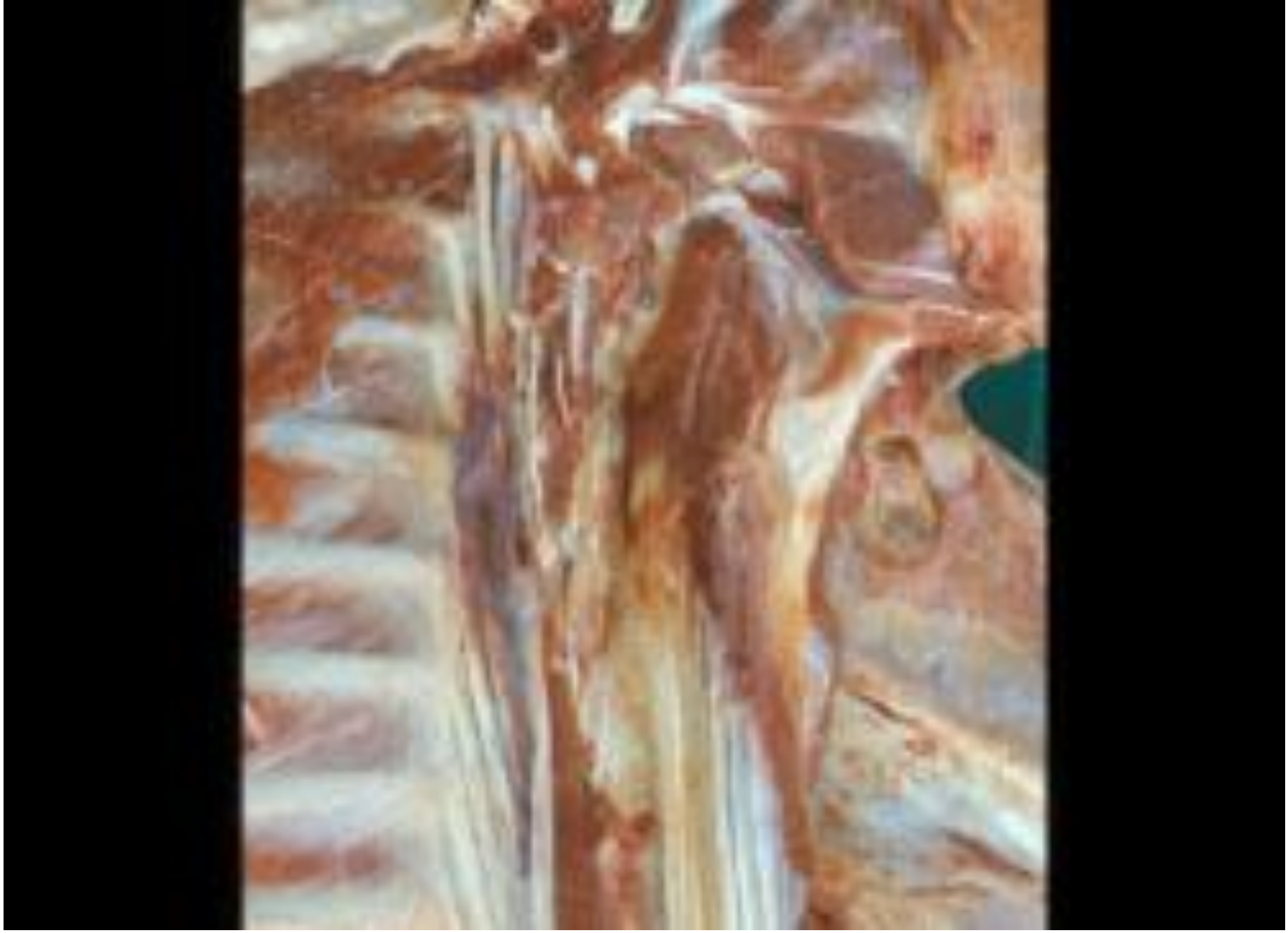


Sheep, pulmonary artery. There are multiple ecchymoses on the intimal surface.

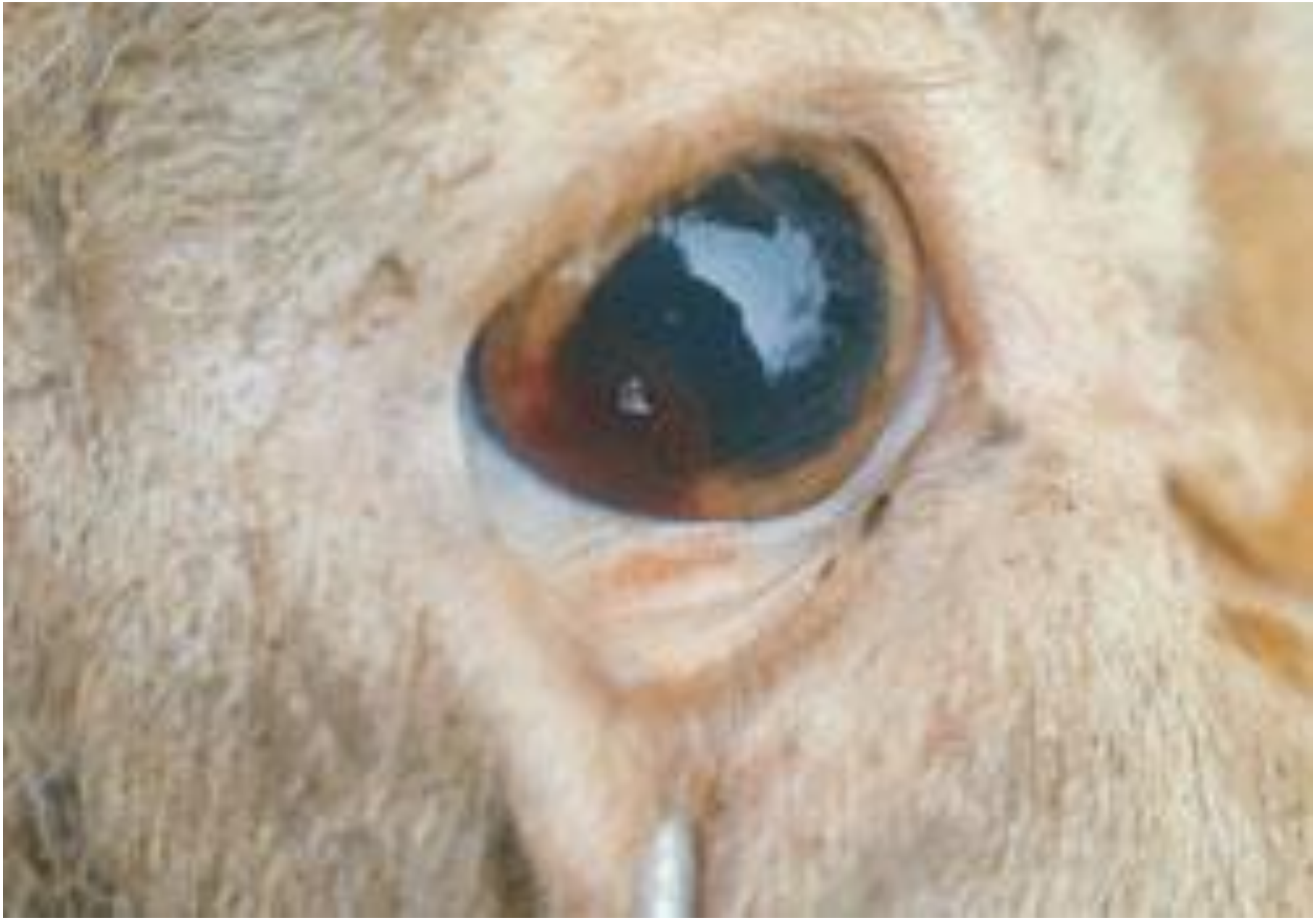
Sheep, foot. There are multiple petechiae in the hoof wall, and there is marked hyperemia of the coronary band.



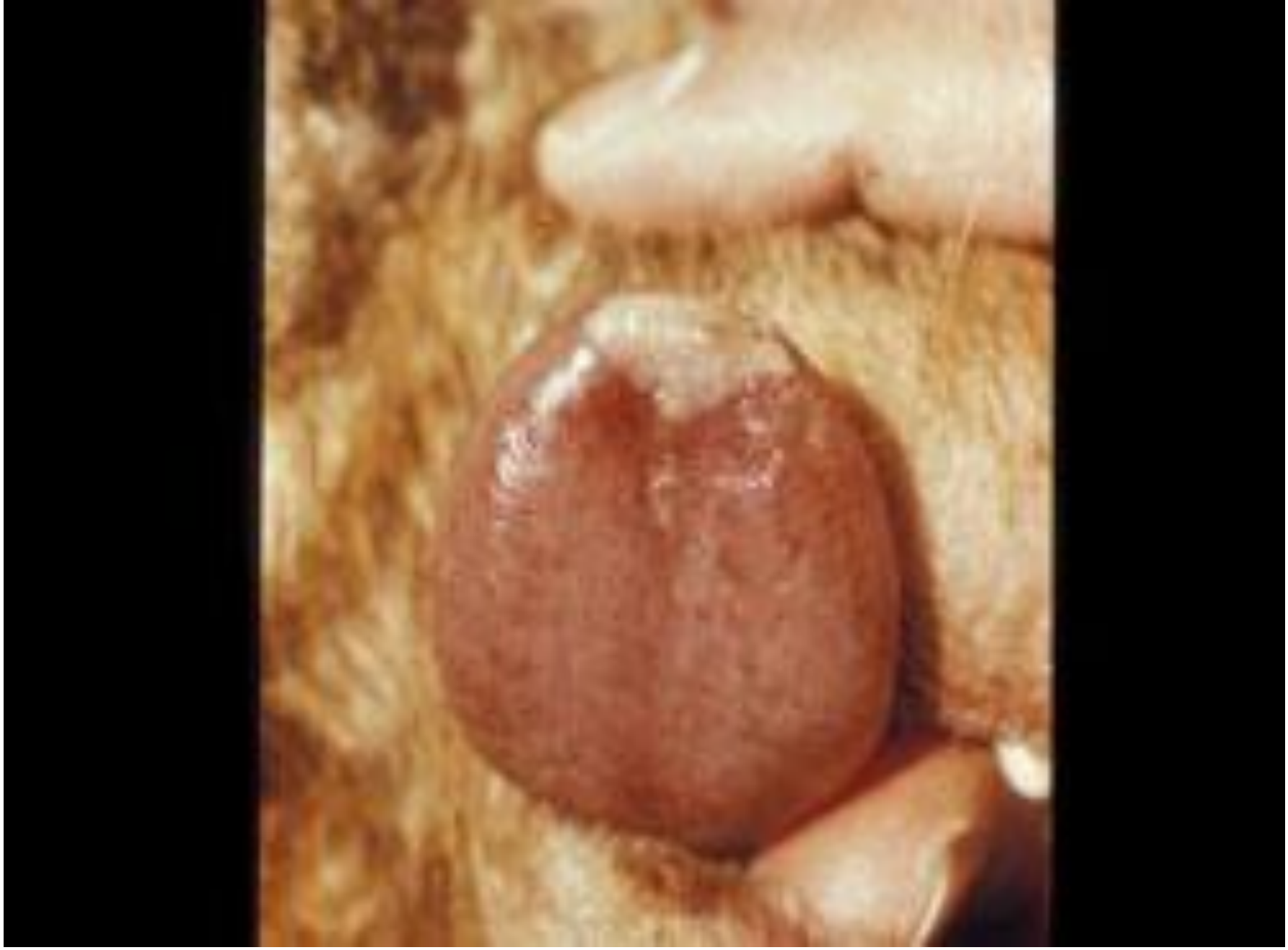
Sheep, tongue. The lateral mucosa contains several ulcers that are covered by exudate and surrounded by zones of hyperemia.



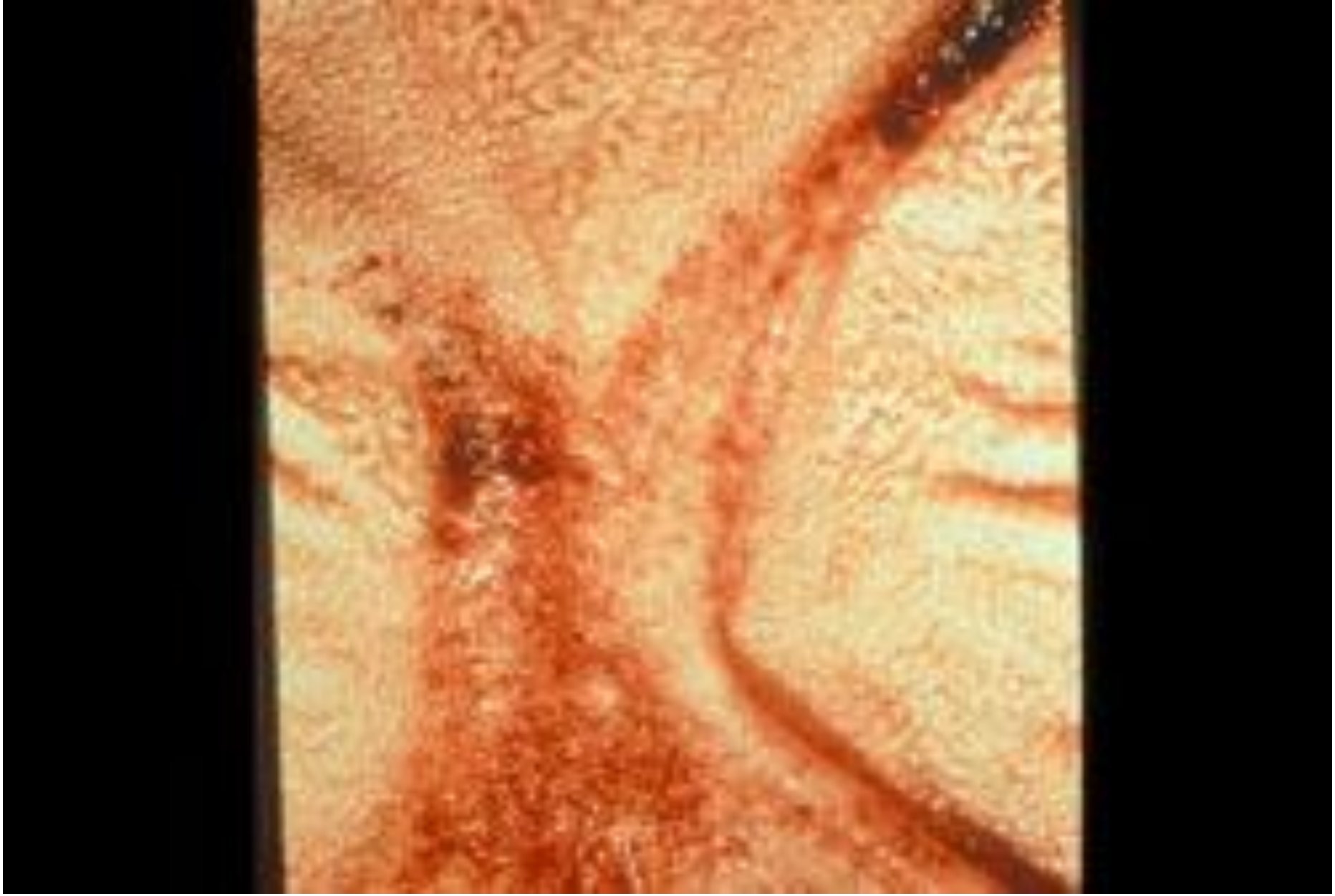
Sheep, skeletal muscle. There is a focus of hemorrhage on the left; pale areas are consistent with myodegeneration.



Sheep, eye. There are foci of bulbar and palpebral conjunctival hemorrhage.



Sheep, tongue. There are disseminated mucosal petechiae, and a single large vesicle on the tip.



Sheep, rumen. There are multiple mucosal hemorrhages centered on the pillars.

Sheep, fetuses. The larger of these aborted macerated fetuses exhibits torticollis.

Morbidity and Mortality

- In sheep, the severity of disease varies with the breed of sheep, virus strain and environmental stresses.
- The morbidity rate can be as high as 100% in this species. The mortality rate is usually 0-30%, but can be up to 70% in highly susceptible sheep.
- Similar morbidity and mortality rates are seen in bighorn sheep.
- Bluetongue is usually severe in whitetail deer and pronghorn antelope, with a morbidity rate as high as 100% and a mortality rate of 80-90%.
- Most infections in cattle, goats and North American elk are asymptomatic.
- In cattle, up to 5% of the animals may become ill, but deaths are rare. In some animals, lameness and poor condition can persist for some time.

Diagnosis

- Clinical:

  Clinical Signs

  lesions

- Differential Diagnosis

  foot-and-mouth disease, vesicular stomatitis, peste des petits ruminants, plant photosensitization, malignant catarrhal fever, bovine virus diarrhea, infectious bovine rhinotracheitis, parainfluenza-3 infection, contagious ecthyma (contagious pustular dermatitis), sheep pox, foot rot and *Oestrus ovis infestation.*

Diagnosis-contd

- Laboratory Tests

  Isolation via inoculation in Chicken egg embryo

  AGID

  Antigen capture ELISA

  Immunofluorescence

Recommendation Action

- Blue tongue is reportable disease in many states.
- State authorities should be consulted for more specific information.

Federal area Veterinary incharge


Thank You.


Thank you ...