# 6_INTESTINAL PATHOLOGIES

**ENTEROPATHIES: 4th grade course2025-2026**

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# Enteropathies in ruminants

- Enteropathies in ruminants refer to a set of pathologies affecting the intestine, characterized by damage to the intestinal mucosa, often leading to inflammation, malabsorption and leakage of proteins.

The causes of enteropathies in ruminants are varied and include:

Infectious agents:

Bacteria: Salmonella spp., Clostridium spp. (responsible for enterotoxemia).

Virus: Mucosal disease virus (BVDV).

Parasites: Various intestinal parasites.

Dietary factors: Sudden changes in diet can upset the rumen and gut balance.

Chronic inflammatory diseases: Similar to inflammatory bowel disease (IBD) in other species, sometimes of autoimmune origin.

Genetic factors: Some predispositions may exist.

# I-DIARRHEES

Definition:

evacuation too quickly of stools that are too liquid".

results in the emission of soft faeces,

Diarrhea is caused by a sudden alteration in the physiological mechanisms that control the secretion of water and electrolytes.

# Mechanism of diarrhea

- Increased intestinal osmotic activity:
- which comes from malabsorption:
- sugars, (especially carbohydrates)
- proteins and
- Lipids
- which promotes the movement of water towards the intestinal lumen by osmotic effect.

# Disturbances in water and electrolyte absorption

The disorders observed during infectious and parasitic diarrhoea act in two ways:

1) a very high secretion of water and electrolytes by the jejunal mucosa induced by the action of microbial enterotoxins,

2) bacteria or parasites cause mucosal lesions, which lead to a local vascular reaction with capillary dilations and mucosal ulcerations, which facilitates plasma exudation.

# Dysfonctionnement moteur

Accelerated transit causes diarrhea

This acceleration can be caused by;

prostaglandins,

Serotonin (An amino substance produced by certain cells of the intestine and brain, playing an important role as a vasoconstrictor and neurotransmitter).

fatty acids and bile acids

Intestinal stasis promotes:

microbial proliferation in the small intestine.

Fat malabsorption,

diarrhea,

The action of bile acids increases diarrhea.

# II-FUNCTIONAL ENTEROPATHIES

## 1. SPASMS

The appearance is brutal and

The evolution is rapid towards recovery.

These spasms are due to the ingestion of a large quantity of cold water (or young fodder during frosts)

A sudden change in the outside temperature.

## 2. DILATATION

follows intestinal paresis in older animals.

It is a paralysis of peripheral nerve (submucosal plexus) or central nerve origin.

The obstacles will result in hypertrophy upstream and then dilation.

Symptoms:

slowing down of transit,

constipation and more or less colic.

Difficult diagnosis.

Treatment:

Administration of flax seeds,

Na or Mg sulphate,

# III-MECHANICAL ENTEROPATHIES

## 1. OBSTRUCTIONS

rare in cattle, (the natural receptacle of foreign bodies is the network).

Clogging agents are/

egagropiles or trichobezoars, phytobezoars,

The obstruction can be/

and results in occlusion, or

partial and manifests as constipation


*Égagropiles, ægagropile (poile de coloration foncée et en laine de coloration claire)photos Dahmani Ali)*

# 2. OCCLUSIONS

- Définition
- Normal discontinuation of the flow of gases and matter from the intestine.
- Causes serious disorders including dehydration and/or acidosis.
- The prognosis is bleak

**Etiology of occlusions**

Functional causes :(ileus)

1. Paralytic (cessation of peristaltic movements) in lesions of the central or peripheral nervous system (rabies), (intoxication by datura)

2. Spasmodic in certain helminthoses and in lead poisoning.

3. Mechanical causes: They are due to either narrowings or topographical anomalies

- **3. SHRINKS:**

- Found in scars (from parasites or surgery),

- in hematomas or in steato-necrosis imprisoning an intestinal loop.

# 4-INCARCERATION :

- is a strangulation by a vicious anatomical disposition.
- A loop of the small intestine is imprisoned in a fibrous flange,
- either by adhesion (traumatic reticuloperitonitis) or by hernia ring (in umbilical, inguinal, omental hernias).

## 5. VOLVULUS:

Rotation of the intestine on itself or around a pedicle.

## 6. ELBOW:

It can be found on a remnant of an umbilical artery

# 7. INVAGINATION :(intussusception)

introduction

Diseases of digestive origin are very common in cattle.

Intussusception (intussusception) is among the major obstructive bowel diseases in ruminants.

Found in adults and calves.

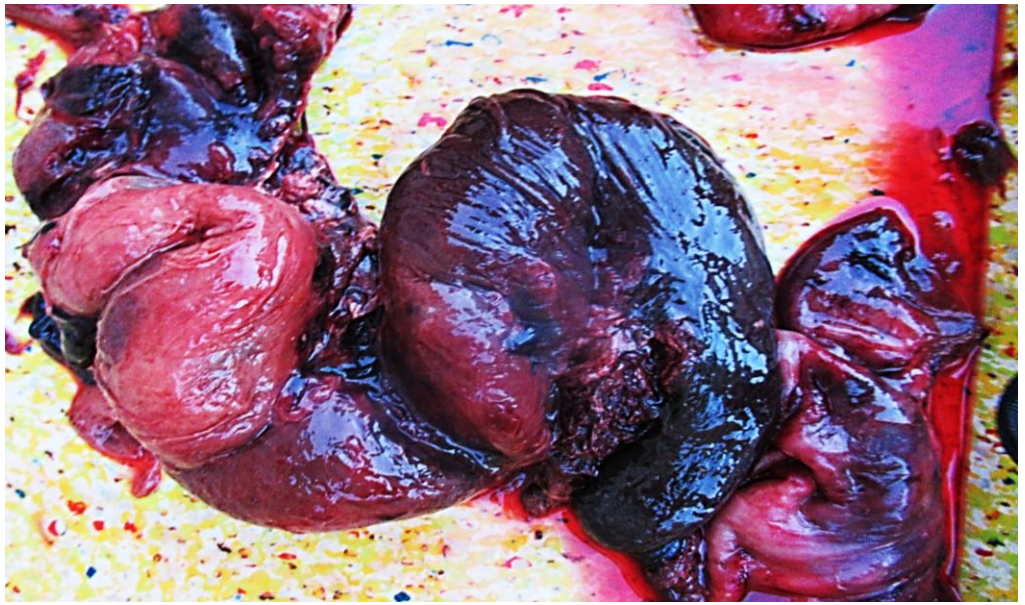
Is considered an invagination of one portion of the intestine into another adjacent portion.

very common in bovine pathology

Pathogenesis:

The decrease in the diameter of the intestine and the obstruction of the veins and arteries of the mesentery lead to oedema and intraluminal and intra-peritoneal exudation.

There are permeability disorders with spasms and anoxia which will lead to necrosis of the intestinal walls (serous, muscular and mucous membranes)



Invagination tube with necrosis of the intestinal walls by anoxia (photosDahmani A,)

- The pain confirms the spasm.
- colic and then occlusive shock.
- Humoral syndrome results in:
- absorption disorders
- increased discharge in the injured area and in the abdominal cavity,
- (loss of water, electrolytes and proteins leading to a decrease in blood pressure and the appearance of tachycardia, anuria and hyperuremia),

- **Symptoms:**
- **Beginning phase:**
- **The syndrome is more rapid if the site of the lesion is more anterior.**
- **Presence of colic that stops as soon as there is necrosis of the intussususception tube**

at first:

Drop in milk production.

Ocular mucous membranes more or less injected.

Slowing down the frequency of defecations.

Atony of the rumen.

Anorexia.

Later:

cow emits bloody dung that soils the perianal

Small amounts of dung

blackish in appearance,

Anorexia is constant.

Colic is less obvious.

Dehydration and enophthalmia.

*cow emits bloody dung that soils the perianal( photo Dahmani A.*

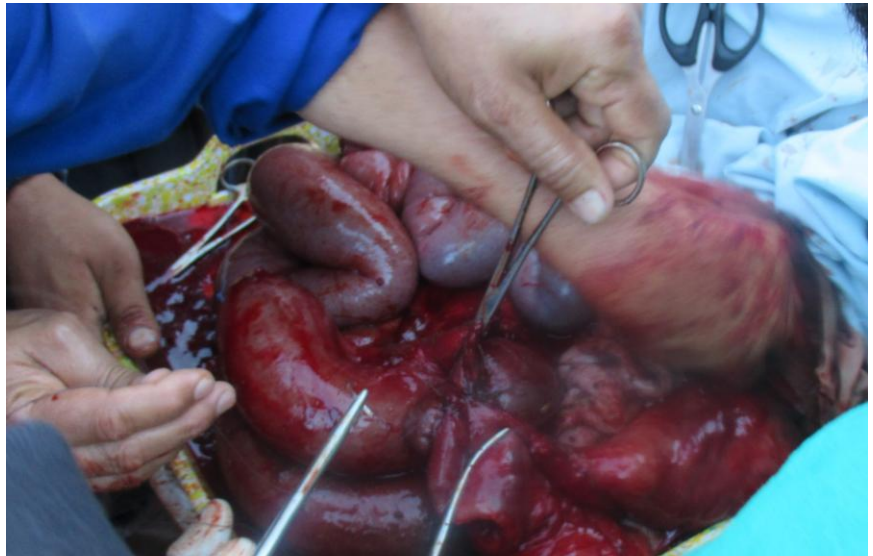
- Status Phase:
- the colic ceases. Anorexia.
- Transit is stopped early if the lesion is posterior.
- After 48 hours, defecation becomes weak, at first, then replaced by the evacuation of a mixture of mucus and blood and then a complete cessation of defecation. We can see a slight tenesmus.

Status Phase:

- The temperature is normal or slightly higher;
- increased heart rate (90 to 120 b/min).
- the muzzle is dry with the presence of scabs,
- head is carried low,
- sunken eyes (enophthalmia)/Dehydration
- Breathing is irregular with expiratory complaints. anorexia, a weak defecation with a bloody appearance.
- Cessation of milk production.

- **The treatment is purely surgical, it consists of a laparotomy and an enteroectomy of the invaginated portion (invagination pudding).**
- **infuse isotonic glucose solutions and isotonic saline solutions intravenously. The total volume can range from 5 to 20 liters**
- **a surgical operation by the right flank has 2 hand crosses behind the hypochondra on the animal standing**

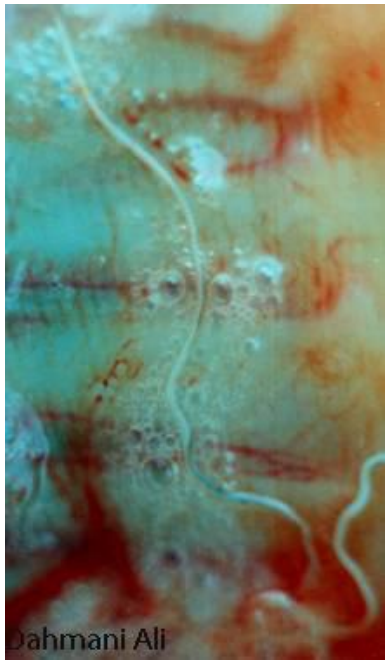
*entéro-ectomie du boudin d'invagination (photos Dahmani A).*



*une entéro-ectomie de la portion invaginée (boudin d'invagination)( Photos Dahmani Ali)*

# IV-ENTEROPATHIES

- **1-Parasitics:**
- **They are caused by nematodes**
- **( dictyocaulosis ,**
- **Gastrointestinal strongylosis**
- **Haemonchos**
- **and**
- **protozoa (coccidioses)**




## • 2-Toxic:

The toxins that induce intestinal symptoms are:

- nitrates and nitrites (consumption of cruciferous vegetables or legumes, cereals that have been spread on fields with phosphate fertilizer (PCN).

- molybdenum, which causes copper deficiency (diarrhoea, discolouration of the hair,...),

NaCl, beet collars and pulp (K+OH-),

acorns (tannins),

annual mercurial (haemoglobinuria due to the presence of coumarin in the plant),

apples (acidosis),

organochlorines and organophosphorus.

# 3-Infectious: (infectious diseases)

- 3.1 Non-specific
- These conditions appear:
- in newborns (colibacillosis, rotavirosis, coronavirus,...)
- in adults where infection may be:
- primary or
- secondary.

### 3.1.1. primary :

- appears in rationing errors (sudden change of diet) which result in indigestion, an imbalance in the intestinal flora leading to enterotoxemia.

- 3.2.2.secondaire :

- to very widespread hepatitis, which results in portal hypertension. The latter leads to internal congestion which manifests itself in chronic diarrhea and kidney damage that will give rise to a uremic syndrome.

- 3.2/ Specific
  - Observed at:
  - mucous membrane disease (viruses),
  - paratuberculosis,
  - salmonellosis, and
  - winter dysentery.

# V-DIARRHOEAL COLIBACILLOSIS IN ADULT CATTLE

can cover two syndromes:

1. Diarrheal: not serious

2. Enterotoxic syndrome:

manifests itself in animals aged 18 months to 6 years, beyond that, the disease is rare.

Etiology

E. coli in general is subdivided into:

"Invasive" E. coli

Have several properties: virulence gene, resistance to the bactericidal power of serum, siderophore (siderophores are iron chelators synthesized and secreted by microorganisms to enable them to draw the iron essential for their development), surface antigens) which allow them to resist the body's defense mechanisms and to multiply there (colibacillary septicemia);

Enterotoxigenic E. coli:

Carry fine protein filaments (K99) to attach to enterocytes, produce a toxin responsible for water and electrolyte losses (colibacillary enterotoxicosis).

- **Symptoms:-**
- A phase of hyperthermia
- which lasts 48 hours, the temperature is 41°C or more, with despondency, inappetence and inrumination.-
- A phase of diarrhoea:
- after 48 hours, the temperature drops to around 39°3 - 39°5,
- the animal has a profuse, greyish and foul-smelling diarrhoea.
- are often accompanied by slight spurs,
- rarely colic.
- In 50% of cases, inflammation of the intestine with clearly hemorrhagic feces.
- There are threads of reddish, undigested blood on the faeces.

## Pre-comatose or comatose phase:

after 1 to 2 days during which this diarrhoea persists, the animal becomes exhausted, its breathing accelerates and it falls into a pre-comatose state in hypothermia (temperature around 37°C). Fatal evolution is then the rule.

## **Pathogenesis** :

The symptoms of the disease are attributed to a direct action of E. coli bacteria on the intestine that causes exogenous and endogenous fluid leakage, and to an action of E. colibacillary toxins that leads to acute hypotension, transsudation in the serous cavities and a state of gastroplegia accompanying diarrhoea

## Diagnostic :

Easy if there is concomitant mastitis or arthritis.

-The speed of evolution.

- By laboratory examination: by seeding from feces.

# Treatment:

- 1. Antibiotic therapy (streptomycin, colistin, neomycin, ampicillin),
- 2. Rehydration
- (isotonic solutes of NaCl, glucose or bicarbonate).
- IV or per Os :(potassium, sodium, magnesium).

Thank you