Abomasal ulcers and displacement

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Introduction

- Diseases of abomasum is common in high yielding dairy cattle
- Hypomotility and accumulation of gas, ingesta, fluid in viscus leading to dehydration, metabolic alkalosis, hypochloremia, hypokalemia
- High grain diet and confinement predispose for abomasal involvement
- Hypomotility of abomasum – associated with hypocalcaemia, endotoxaemia, acidosis, alkalosis, hyperinsulinaemia, hyperglycemia
- Abomasal pH – 1.4 to 4.5

Abomasal ulcers and displacement

Lesson I Introduction Abomasal ulcers and displacement

Lesson II Abomasal ulcers – Clinical signs, Diagnosis & treatment

Lesson III Left displacement of abomasum

Lesson IV Right displacement of abomasum

Lesson V Abomasal bloat & impaction

Unit : Abomasal ulcers and displacement
Lesson : 1

Introduction Abomasal ulcers and displacement

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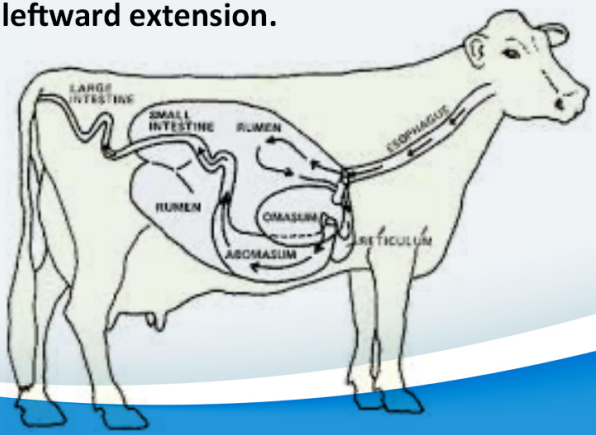
Anatomy of abomasum

- Non pregnant animal – positioned below rumen in the ventral part of abdomen, predominantly on right side
- During pregnancy – gravid uterus forces the abomasum more cranially with leftward extension.



Etiology

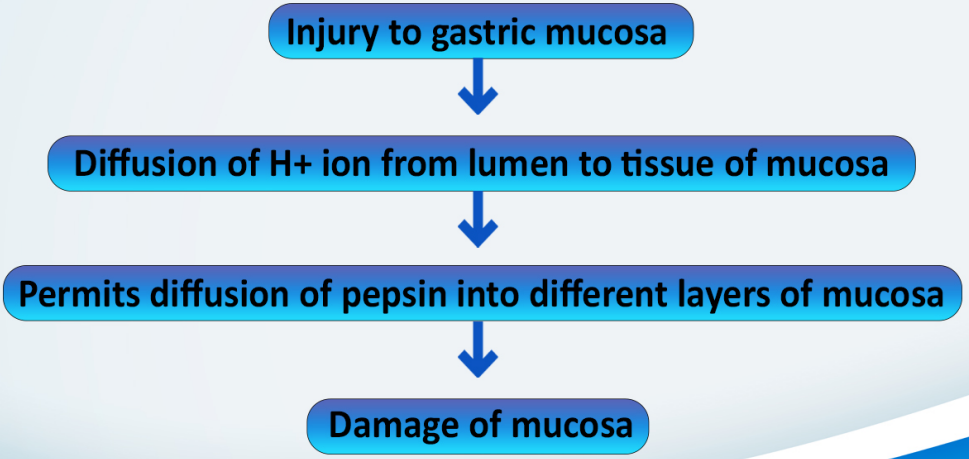
Primary ulceration

- **Abomasal hyper acidity**
- **Mechanical abrasion of pyloric antrum – coarse roughage, trichobezoars**
- **Bacterial infections** – *Clostridium perfringens type A or Fungai – Aspergillus fumigatus, Mucor spp.*
- **Copper deficiency**
- **Concurrent stress – parturition, lactation, transport, severe inflammatory process, severe pain (result in hypercortisolemia)**

Secondary ulceration - **secondary to other diseases**

- **Erosions due to viral diseases - BVD, Rinderpest, BMC**
- **Reflux of bile into abomasum (Bile acids – gastric injury)**
- **Displacement of abomasum(LDA, RDA), volvulus**
- **Lymphosarcoma/ Lymphoma of abomasum**
- **NSAIDS**
- **Theileriosis**

Pathogenesis



Classification

- Type I – Non perforating ulcers
- Type II – Ulcers with severe blood loss
- Type III – Perforating ulcer with local peritonitis
- Type IV – Perforating ulcer with diffuse peritonitis

Type I – Non perforating ulcers

- Incomplete penetration – not penetrating muscularis mucosa
- Minimal intraluminal injury and haemorrhage
- Focal abomasal thickening

Type II – Ulcers with severe blood loss

- Penetration of wall of major abomasal vessels usually submucosa
- Intraluminal haemorrhage and anaemia
- Accumulation of fluid in abomasum
- Metabolic alkalosis, hypochloremia, hypokalemia
- Rumen chloride content increase
- Increased plasma - gastrin

Type III – Perforating ulcer with local peritonitis

- Penetration of wall of major abomasal vessels usually submucosa
- Intraluminal haemorrhage and anaemia
- Accumulation of fluid in abomasum
- Metabolic alkalosis, hypochloremia, hypokalemia
- Rumen chloride content increase
- Increased plasma - gastrin

Type IV – Perforating ulcer with diffuse peritonitis

- Penetration and leakage of abomasal content – diffuse peritonitis

Thank you