Pathology 3rd stage

#### **Digestive system**

### \*Anomalies of mouth

**1- cleft palate:** Failure of fusion of lateral palate.

\*Causes: 1-genetic 2-Toxic agents 3-Steroid treatment in the late stage of pregnancy.

**2-Hare lip**:- Failure to infusion of the upper lip in middle line. The animal will die because the starvation & aspiration.

**\*Stomatitis:-** Inflammation of mucosa-membrane of oral cavity.

## (1)Vesicular stomatitis:-

By viral infection such as F.M.D characterized by vesicle or blister of the oral mucosa.

\*Grossly:-

Small clear, fluid filled vesicle in the lip, tongue, esophagus and mammary gland.

### (2) Erosive & ulcerative stomatitis:-

Which causes by viral infection. Ulcers: are characterized by segmental or more extensive loss of the epidermis, including the basement membrane, with exposure of the underlying dermis.

Erosion: is characterized by the partial loss of the epithelium, with the basement membrane left intact.

This condition occur in bovine diarrhea and equine viral rhinitis.

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## (3) Popular stomatitis:-

Its caused by para pox virus. Characterized by popular in muzzle & in oral cavity & mouth & characterized by eosinophilic cytoplasmic inclusion body.

(4)Necrotizing stomatitis:- in cattle & sheep.

In cattle called calf diphtheria. Caused by <u>fusibacterium</u> <u>necrophorous</u>.

# \*Grossly:-

Yellowish-gray, brown bronchus and surrounded by hyperemia in the mucosa of oral cavity, lung& pharynx.

## \*Microscopically:-

There is coagulative necrosis surrounded by granulation tissue & hyperemia.

### \*Dental Caries:-

Its decalcification of harm dental substance due to lactic acid produce from carbohydrate which accumulate between teeth and also bacteria enzyme.

**\*Bloat = Ruminal tympany:-** Over distension due to gas production from fermintation of carbohydrate (CHO) & this gases include (CO2 and methane).

## \*Effect of the bloat.

(1)gases push the diaphragm which interfere with respiration.

(2)Pressure on the abdominal vein which interfere with blood circulation , anoxia lead to death.

**\*Ruminitis:-** Inflammation of rumen.

The rumen contain normally G -ve bacteria, which one to cellulytic of cellulose, when changes to the grain lead to fermintation  $\rightarrow$  lactic acid formation  $\rightarrow$  decrease PH in rumen  $\rightarrow$  increase growth of G +ve such as lacto bacillus spp  $\rightarrow$  decrease growth of G-. Lactic acid lead to distended the ruminal wall.

Decrease  $PH \rightarrow$  increase osmatic pressure in the lumen of the rumen  $\rightarrow$  transport of tissue fluid to the lumen of the rumen. Lead to ruminitis.

#### \*Stomach:

Local defence mechanism like

(1)Motility

(2)IgA prevent attachment of bacteria to the mucosa.

(3) Acidity of stomach.

#### \*Gastric dilation & rapture

(1)Acute gastric dilation :- Occur in the horse due to intestinal obstruction and displacement this occur due to take large amount of grain.

(2)Chronic gastric dilation:- It's a secondary, this condition occur due to gastric lymphoma in the dog, cattle, horse.

\*Gastritis:- Inflammation of stomach or Abomasitis (inflammation of abomasum).

\*general clinical symptoms:-Pain, anoroxia, vomiting.

Pathology 3rd stage -6- Assist. Prof. Dr. Aamir Al-Ghareebawi \*Types of gastritis:-

## (1)Acute catarrhal gastritis:- characterized by

a-redness and thickness of surface mucosa.

b-Increase of the mucosal secretion.

c-congestion, desquamation of epithelia.

d-Inflammatory cell infiltration in mucosa + submucosa.

# (2)Acute hemorrhagic gastritis:-

Grossly:-a-deeper reddening of mucosa.

b-Free R.B.C in the lumen.

Causes:-a-Destructive poisons.

b-Point of attachement of helminth (Trichostrongylus).

c-Anthrax.

(3)**Diphtheritic gastritis:-** Characterized by fibrin exudate on the mucosa. Under the layer of fibrin, there is necrotic cell. This condition seen in malignant catarrhal fever in cattle.

# (4)Chronic gastritis:-

In this type of inflammation there is proliferation of epithelial mucosa lead to

(a)Thickness of mucosa also hyperplasia of goblet cells  $\rightarrow$  increase secretion of mucin.

(b)chronic inflammatory cells infiltration in mucosa and submucosa .

Pathology 3rd stage -6- Assist. Prof. Dr. Aamir Al-Ghareebawi (5)Chronic atrophic gastritis:- Characterized by atrophy of gastric gland and chief cells with lymphocyte, plasma cell, eosinophilic cell infiltration.

(6)Eosinophilic gastritis:- Either local due to parasitic infection or diffuse due to allergy and eosinoplilic cell infiltration in the mucosa and submucosa.

\*Microscopically:-

1-There is eosinophiles infiltration in the submucosa.

2-fibrosis in the lamina properia.

3-Lymphoid hyperplasia.

### \*Gastric ulcerative & erosion:-

Its destruction of epithelial mucosa & basal membrane and glandular tissue, some time reach to muscular layer & serosa which called ( perforating gastric ulcer) in the dog and human but in the animal its only reach to muscular layer.

\*Microscopically:- There is focal ulceration in the mucosa with or without fibrosis.

### \*Classification of gastritis

1-Uremic gastritis:- due to chronic renal disease urea precipitation in the gastric tissue .

2-Parasitic gastritis:-

3-Tumor of stomach

Pathology 3rd stage-6-Assist. Prof. Dr. Aamir Al-Ghareebawi(1)leiomyoma(2)malignantlymphoma(3)sq.cellcarcinoma(4)adenocarcinoma.

# Intestine

\*Defence mechanism of intestine:-

1-secretion of oral cavity & intestine lead to prevent bacterial attachment

to the mucosa.

2-normal acidity of the intestine or stomach .

3-Non pathogenic bacterial flora.

4-Intestinal peristaltic movement of the intestine mucosa.

5-bile salt.

6-IgA, IgM..

7-Bacterial growth inhibition by pancreatic solution.

# \*Diarrhea:-

Pathogenic diarrhea.

There are (4) mechanism of diarrhea.

1-Malabsorption:- With or with out bacterial infection.

2-Hyper secretion by intact mucosal.

3-Exudation due to increase permeability of capillaries.

4-Hypermotility of intestine.

Micro organism invade enterocyte  $\rightarrow$  enterotoxin  $\rightarrow$  enterocyte release

cytokine  $\rightarrow$  interleukin  $\rightarrow$  increase macrophages  $\rightarrow$  adenosin  $\rightarrow$ 

histamine  $\rightarrow$  increase fluid + decrease absorption  $\rightarrow$  diarrhea.

### \*Anomalies of intestine:-

(1)Atresia:- Part of digestive system occluded such as atresia ani or atresia coli.

(2)Meckels diverticulum:- Its reminant of omphalo mesenteric duct near the terminal part of ileum & represent stalk of yolk sac.

(3)Mega colon or fecal filled colon:- In dog & humen & fowal caused by congental defected

(4)enterolithis:- Occur in the Arabian horse from Amonium magnesium phosphate precipitate around foreign body in the intestine. Occur in colon which lead to infection of colon.

#### **\*Intestinal Impaction:-**

This condition occur in the horse due to infection of parasite and treatment of this parasite lead to rapid dead large number of parasite also in the horse occur due to accumulation large amount of sands.

**\*Peritonitis:-** Inflammation of periton.

(1) its occur due to inflammation, either generalized or localized rout of infection.

(2)Rupture of intestine & uterus.

(3)Direct extension from abdomenal organ.

Peritonitis either fibrinous, serous, or granulomatous, acute or chronic

Pathology 3rd stage-6-Assist. Prof. Dr. Aamir Al-Ghareebawi\*Enteritis:-inflammation any part of the intestine:-

# **Type of enteritis:-**

- 1-Catarrhal enteritis.
- 2-Purulent enteritis.
- 3-Fibrinous enteritis.
- 4-Necrotize enteritis.
- 5-hemorrhagic enteritis.
- 6-Chronic proliferative enteritis.
- 7-Regional cicatrizing enterocolitis.

### \* Causes

- (1)Toxic agent (chemical or poison plant).
- (2)infectious agent. Such as anthrax, salmonella.