Rinderpest and MCF

- Gross lesions are similar to those of BVD-MD
- Rinderpest is highly contagious and usually affects many animals, unlike MD or MCF.
- Nervous signs & eye lesions are common with MCF.
- MCF is associated with sheep (world-wide) or with wildebeest (Africa)
- Rinderpest is now almost controlled world-wide
MCF gross lesions, cattle

Corneal opacity

Ulcerative stomatitis

Ulcerative stomatitis

Rhinitis. Serocellular crusts and exudate covering the nostrils
MCF gross and microscopic lesions, cattle

**Ulcerative glossitis**

**Ulcerative abomasitis**

**Cryptitis.** Necrosis and loss of cryptal epithelium. Crypts are filled with cell debris. Intestine.

**Vasculitis** and lymphoplasmacytic perivascular infiltrates, brain.
Enteric viral diseases - porcine

- **Transmissible gastro-enteritis (TGE)**
  - Coronavirus infection
  - Highly contagious & with high mortality in pigs > 14 days old
  - Vomiting & diarrhea
  - Severe villous atrophy

- **Others**
  - Porcine epidemic diarrhea
  - Porcine rotavirus

**TGE:** Thin walled intestine dilated with gas & fluid also contains undigested milk.
TGE, small intestine, piglet. Moderate villous atrophy, the villi are covered with flattened epithelial cells (top & right). Marked villous atrophy & fusion (bottom).
Enteric viral disease - feline

- Feline panleukopenia
  - Syn: feline parvovirus enteritis
  - Affects rapidly dividing cells in intestine, bone marrow & lymphoid organs
  - Young cats most susceptible
  - Fecal-oral route of infection
  - Clinical signs
    - Vomiting, diarrhea, dehydration & panleukopenia

- Feline infectious peritonitis
  - Coronavirus infection
  - Multisystemic disease (peritonitis/pyogranulomas)
Feline panleukopenia - lesions

- **Intestinal lesions**
  - Dilated hemorrhagic intestines grossly
  - Severe crypt necrosis & villous atrophy
  - Intranuclear inclusions (acute disease only)
  - Squamous metaplasia & hyperplasia of crypts

- **Other lesions**
  - Lymphoid necrosis
  - Bone marrow hypoplasia
  - Fetal cerebellar hypoplasia

Hyperemia of intestinal wall, diphtheritic appearance of necrotic mucosa (bottom)

Cerebellar hypoplasia, kitten
Panleukopenia virus enteritis, small intestine, cat (left). Villi are short and denuded of epithelium. Several crypts are dilated and lined by a squamoid epithelium, other crypts are hyperplastic. Note chronic inflammatory cells in lamina propria. Similar lesions in canine parvovirus (CPV-2, right)
Enteric viral diseases - canine

- Canine parvovirus enteritis (CPV-2)
  - Similar to feline disease
  - Interstitial myocarditis in puppies (rare)
- Minute virus of canine (MVC, CPV-1)
  - Only mildly pathogenic
- Canine distemper
  - Pantropic morbilivirus

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*PV-1, inclusion bodies, small intestine, puppy*
Canine parvovirus enteritis, small intestine, dog. Segmental hyperemia (left) and the typical “ground-glass” appearance of the serosa (right)

Canine parvovirus enteritis, small intestine, dog. Mucosal hemorrhage, shortening of villi and dilation of crypts (left), few regenerating crypts and some inflammatory cells (right)
Bacterial enteritides

- Colibacillosis
- Salmonellosis
- Clostridial diseases (enterotoxemia, etc)
- Johne’s disease (paratuberculosis)
- Lawsonia enteritis
- Swine dysentery (& colonic spirochetosis)
- Others
Colibacillosis (*E. coli*)

- Common disease of newborn
- *E. coli* is part of normal gut flora
- Disease depends on
  - Number of organisms ingested
  - Ability to adhere to enterocyte surface
  - Ability to proliferate
  - Ability to produce toxins or invade tissues
Predisposing factors

- Heavily contaminated environment
- Failure to receive colostrum
- Milk substitutes
- Cold stress
- Overcrowding
- Concurrent infections
Forms of colibacillosis

- Enterotoxic *E. coli* (ETEC)
  - Enterotoxin → secretory diarrhea
- Postweaning colibacillosis
- Septicemic colibacillosis
  - Fibrinous polyserositis
- Edema disease (verotoxigenic *E. coli*)
- Entero-invasive *E. coli* (EIEC)
- Attaching and effacing *E. coli* (AEEC)
  - Effacement of microvilli
- Mucoid enteritis of rabbits
Enterotoxic colibacillosis. Toxins induce hypersecretion & reduce absorption leading to increased fluid in gut lumen (top, right) pig. The bacteria first attach to the microvillous border & proliferate, forming mats (arrows, bottom,) pig.
Edema disease

- Synonym: Gut edema, enterotoxemic colibacillosis
- Seen in pigs 6-14 wks old, dietary change
- 35% morbidity, 100% case mortality
- Angiotoxin absorbed from intestine
  - Fibrinoid necrosis of arteries/arterioles
  - Generalized edema (stomach, intestine & gall bladder, skin, eyelids)
  - Multifocal neuronal degeneration
- Neurologic signs, usually no diarrhea
Edema disease, pig. Note edema of the eyelids, snout (top), stomach wall (top right) and mesentery of spiral colon (right) as a result of an angiotoxin produced by *E. coli.*
Salmonellosis
An important zoonosis world-wide

- Many spp of salmonellae, but only few cause disease in animals & humans
- Feco-oral route of transmission
  - organisms invade enterocytes & macrophages
  - enteritis, septicemia & endotoxemia
- Most animals are symptomless carriers
- Human infections mostly from poultry & swine products
Lesions of salmonellosis

▪ **Septicemic** form
  - Petechial hemorrhages (generalized)
  - Multifocal hepatitis (paratyphoid nodules)

▪ **Acute** form
  - Fibrino-necrotic enterocolitis, necrosis of Peyer’s patches & mesenteric lymphadenopathy

▪ **Chronic** form
  - Ulcerative entero-colitis & proctitis
  - Rectal strictures & obstruction

▪ **Differential diagnosis**

Paratyphoid nodule with intralesional bacteria (*Salmonella typhimurium*), liver, cow
**Salmonellosis.** Septicemic form with blue-red discoloration of extremities (pig, top left) & hemorrhagic colitis (horse, top right). Subacute to chronic form with fibrinous cast in intestine (bovine, bottom left) or with button ulcers in ileocecal junction (pig, bottom right)
**Chronic salmonellosis** in pigs. Histo of colon with button ulcer (arrow, above). Marked dilation of the colon (bottom left) is due to rectal stricture (bottom right) secondary to ulcerative proctitis and fibrosis.
Clostridial enteritis

- *Clostridium perfringens*: enterotoxemia
  - Five exotoxins (A, B, C, D, E) which act through cAMP/cGMP to cause diarrhea
  - Result in sudden death in well-nourished animals

- *C. piliforme*: Tyzzer’s disease
  - Enterocolitis & hepatitis in rodents, etc

- *C. difficile*: zoonosis
  - Associated with prolonged antibiotic use

*Cl. piliforme*, liver, foal
Clostridial enterotoxemias

- *C. perfringens* type A
  - Hemorrhagic enteritis & abomasitis
- Type B
  - Mild dysentery
- Type C
  - Severe lesions in young animals
  - Struck in adults
- Type D
  - Overeating disease
  - Pulpy kidney in sheep
- Type E
  - Peracute disease in dogs?
Pathogenesis of pulpy kidney disease

- Older well-fed ruminants
- Associated with sudden change in feed (high CHO) → rapid multiplication of clostridial organisms → production of angiotoxin → absorption → multisystemic hemorrhages, encephalomalacia & nephrosis → rapid death
- Glycosuria in sheep (from hepatic glycogen)
Lesions of enterotoxemia

- **Gross**
  - Marked hemorrhagic discoloration of intestines
  - Hemorrhages on serosal surfaces
  - Rumen/stomach full of feed
  - Soft, dark-red kidneys (severe nephrosis)
  - Pericardial effusion (fibrinous pericarditis)
  - Focal symmetrical encephalomalacia
Enterotoxemia, focal symmetrical encephalomalacia, brain, lamb. *Clostridium perfringens* type D infection
Pulpy kidney disease lesions

- Microscopic
  - Hemorrhagic enteritis
  - Abundant Gram positive bacilli (monomorphic) in intestinal mucosa

- Other diagnostic features
  - Terminal glycosuria
  - Detectable toxin in intestinal content
Enterotoxemia, smear of intestinal mucosa. Note uniform population of Gram positive thick, straight-sided rods, either singly or in pairs, seldom in chains. Toxin can also be detected in feces or in serous exudates.