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Post-mortal Approach to the Alimentary System – Part 2

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Fore-stomachs and rumen

External examination of the fore-stomachs and rumen should include observations for distentions/change in shape, displacements, adhesions and discolouration (red/black areas of infarction associated with mycotic rumenitis). All compartments of the fore-stomachs and rumen should be opened and examined for foreign bodies and parasites, and the content should be analysed for composition, consistency, pH and odour.

Content should then be thoroughly washed out to expose the mucosa of each of the compartments for examination. Attention should be paid to the lining epithelium of the rumen for presence or absence of papillae, length of papillae and integrity of the epithelium. The fore-stomach and rumen lining should then be stripped and subepithelial mucosa evaluated for evidence of subepithelial erythema/congestion indicative of chemical rumenitis.

Bloat

Primary/frothy bloat occurs within three days of starting a new diet. Certain feed components, such as legumes and grain concentrates, promote the formation of a stable foam, with foam production being highest when the rumen pH is between 5 and 6. Primary frothy bloat needs to be differentiated from post-mortal bloat, especially if the animal has been dead for a few hours in situations with high environmental temperatures.

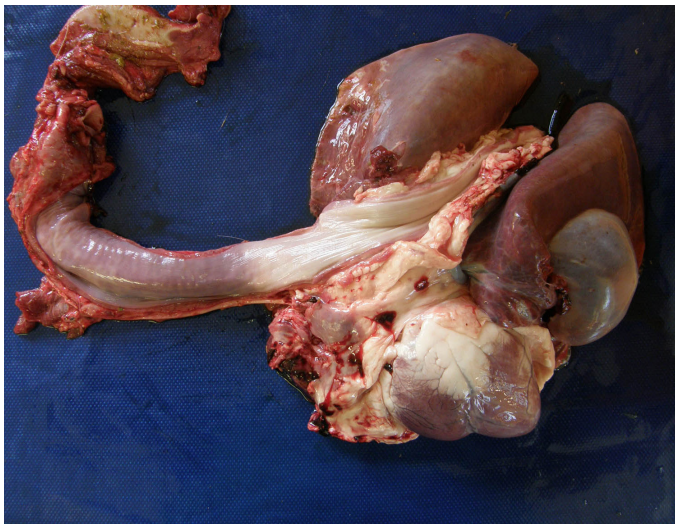

Figure 1. Goat: opened oesophagus to reveal the classic bloat line.

Post-mortal features which indicate a primary ante-mortal bloat include generalised subcutaneous congestion cranial to the thoracic inlet, with the most reliable post-mortal indicator being the bloat line which is a sharp line of demarcation in the oesophageal mucosa between the bloodless oesophagus distal to the thoracic inlet and the congested proximal oesophagus cranially (figure 1). Secondary/free gas bloat is the result of physical or functional obstruction or stenosis of the oesophagus resulting in failure to eructate.

Foreign bodies

These include trichobezoars (hairballs) and phytobezoars (plants balls). Trichobezoars are usually seen in young bucket-fed calves which suckle on one another and in foetuses where hair circulating in the amniotic fluid is swallowed by the foetus. Phytobezoars on the other hand are observed in adult ruminants associated with ingestion of indigestible roughage.

Rumenitis

Figure 2. Bovine rumen: opened with ingesta washed out to reveal shortening and denuding of papillae of the ventral sac (arrows), when compared to the dorsal sac (star). A chronic puckered scar, devoid of papillae, with ulceration evident on the anterior rumen pillar (oval).

Three forms of rumenitis are described, namely rumen acidosis, bacterial rumenitis and mycotic rumenitis.

1. Rumen acidosis: post-mortal indicators of

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acidosis include watery rumenal and intestinal contents with an acidic smell. Rumen papillae on normally discoloured brown/black, are friable and detach easily especially from the ventral rumen sac (figure 2). Caution should be exercised as easy detachment of the rumen epithelium is a common post-mortal change particularly in animals that have been dead for a few hours in high environmental temperatures.

In many feedlot operations, rumen lesion scoring is practiced at the abattoir as a management tool to monitor feed management at the lot.

- Normal: Gross appearance of healthy epithelium with thick, lush papillae with no signs of inflammation, ulceration, or other insult.
- Mild: Consolidated portions of the ruminal mucosal surface displaying short (relative to normal) or denuded papillae.
- Severe: Active rumenitis lesions; focal or multifocal ulcerations characterised by demarcated, irregularly circular, depressed, red, foci or healed ulcerations (scars) characterised by focal or multifocal puckered scars (star shaped) devoid of papillae.

Rumen pH should be measured as soon as possible after death, as increase in pH occurs post-mortally with pH starting to rise within ±40 minutes after death. Therefore, rumen pH measured some time after death would likely have been significantly lower at the time of death.

2. Bacterial rumenitis usually occurs secondary to rumen acidosis or mechanical injury to the mucosa. Bacteria most commonly involved include *Trueperella pyogenies* and *Fusobacterium necrophorum*.

3. Mycotic rumenitis is observed as a complication of rumen acidosis, secondary to mechanical injury, excessive administration of antibiotics which reduces the number of normal flora and allows overgrowth of fungi. Lesions of mycotic rumenitis are associated with the angio-invasive nature of the fungi and are characterised by varying sized full thickness (transmural), infarctive lesions which are generally circular and well delineated (figure 3). Fungi most commonly involved include *Aspergillus*, *Mucor*, *Rhizopus*, *Absida* and *Mortierella*.

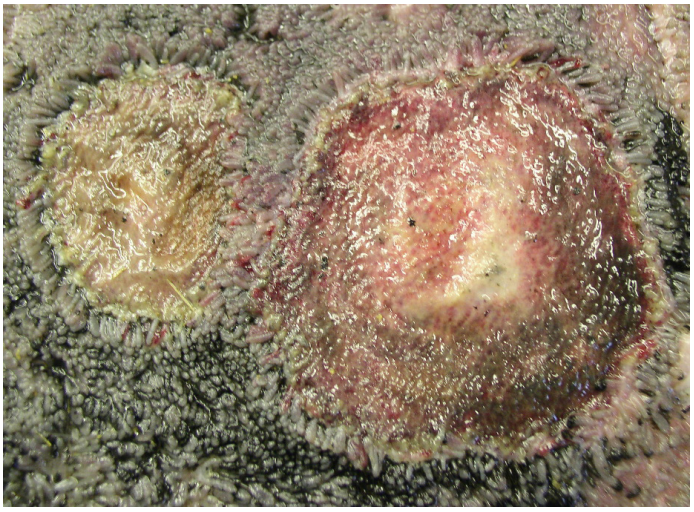

Figure 3. Bovine rumen: opened to reveal well-circumscribed, circular lesions of mycotic rumenitis associated with transmural infarction.

Miscellaneous conditions

Vagal indigestion results in a functional outflow problem with massive rumenal distention. Massive rumen distention with fluid content is also well documented in cases of kikuyu poisoning in cattle and sheep.

Parasites

Paramphistomum spp (conical fluke) are trematode parasites, which are fairly commonly encountered on farms with vlei areas in South Africa. They have been reported in cattle, sheep, other ruminants and camelids. The adult parasites reside in the rumen with eggs being passed in the faeces of the host. Eggs hatch in water liberating miracidia which infect snails. Following development in the snail, cercariae are released which encyst on vegetation. Definitive hosts (ruminants, camelids) are infected by ingesting fluke metacercariae while grazing. Larval paramphistomes then develop in the initial portion of the small intestine (duodenum) and they move retrograde up the small intestine to the rumen where they develop into adults.

It is the immature stages of the parasite, found in the initial small intestine, which are responsible for the major pathological and clinical effects. These immature stages are so-called “plug feeders” resulting in severe erosion of the duodenal mucosa (figure 4). Heavy infestation with immature parasites is characterised by oedema, haemorrhage and ulceration of the initial small intestine, with anaemia, dehydration and subcutaneous oedema “bottle jaw”. Adult parasites in the rumen on the other hand are well tolerated. Mortality in acute outbreaks can approach 90%.

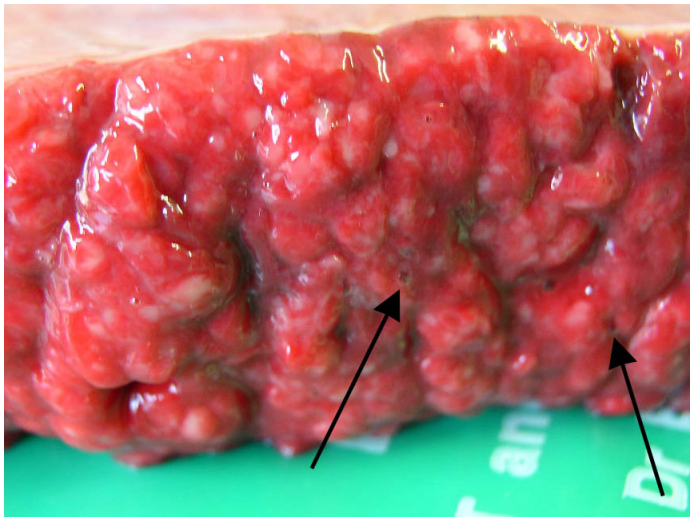

Figure 4. Bovine duodenum: severe mucosal proliferation with multifocal pinpoint ulcerations (arrows) associated with feeding sites of the immature stages of Paramphistomum sp.

Stomach and Abomasum

Gross examination of the stomach/abomasum should include external examination for evidence of distention, adhesions, discolouration (infarcts) and perforations. Thereafter the organ is opened to facilitate examination of the content and mucosal surface. Any parasites observed should be collected and submitted to a helminthology laboratory for identification. Washing of the mucosa enables examination for areas of inflammation, erosion, ulceration and possible neoplasia.

Gastric dilation and volvulus

Simple gastric dilatation occurs in a variety of animal species and is well-documented in man. In the canine species acute gastric dilatation usually results in gastric rotation with development of the acute, often life-threatening gastritis dilatation-volvulus syndrome. This syndrome is a common condition in the dog and most frequently encountered in the deep-chested, large breeds. In the true anatomical sense, the stomach rotates around the ventro-dorsal axis, hence the term volvulus is more correct than torsion (figure 5).

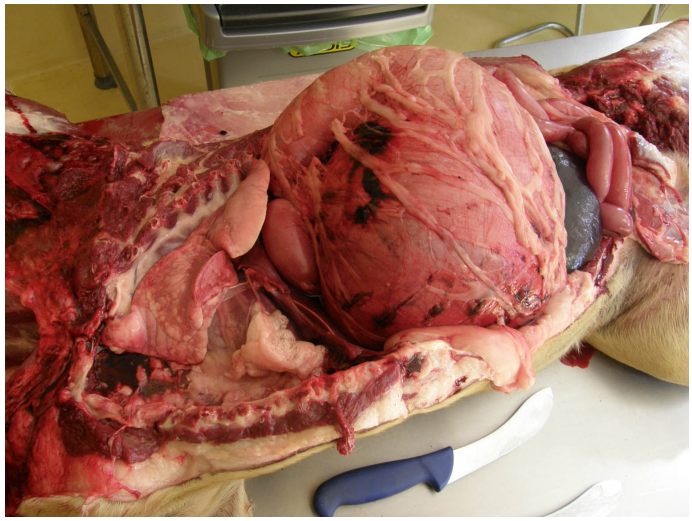

Figure 5. Dog: opened abdomen with distended stomach which has undergone volvulus.

Rotation of the stomach usually follows repeated episodes of gastric dilatation with stretching and relaxation of the gastro-hepatic ligament. These repeated episodes in conjunction with overfeeding, post-prandial exercise and possible hereditary predisposition, result in rotation of the stomach. In the dog, gastric rotation more frequently occurs alone without concurrent splenic displacement. However, in some instances the spleen accompanies the stomach and is displaced to the right side of the abdomen, becoming severely engorged and distended due to vascular compression.

Gastric dilation and rupture

Primary gastric dilatation in horses is a sequel to consumption of excessive fermentable carbohydrate, sudden access to lush pasture or excessive intake of water. Ingesta may swell through absorption of saliva and gastric secretion. This results in increased distention of the stomach with systemic dehydration. Acute gastric dilatation and rupture in horses frequently occurs as a terminal event due to intestinal obstruction and displacement. The presence of haemorrhage and evidence of inflammation, such as fibrin strands along the margins of the rupture, which usually involves the greater curvature are pathologies that indicate ante-mortal gastric rupture.

Abomasal displacement

- Left-sided displacement is usually a non-fatal condition of high producing dairy cattle, usually occurring in the 6 weeks after calving.
- Right-sided displacement account for only ± 15% of abdominal displacements and 20% of these develop volvulus. They have been described in post-parturient dairy cows and in calves.

Abomasal dilation and tympany

Abomasal tympany with inflammation/ulceration is an emerging syndrome in calves. Severe acute bloat with high mortality are the primary features of the condition. *Clostridium perfringens* type A and *Sarcina* alone or in combination are most frequently involved, and with

Clostridium septicum (braxy) are the only proven bacterial agents capable of inducing abomasal pathology. It is believed that natural occurrence of the disease requires a quantity of highly fermentable substrate (starch, glucose, lactose etc) and a bacterial flora capable of rapidly fermenting the substrate.

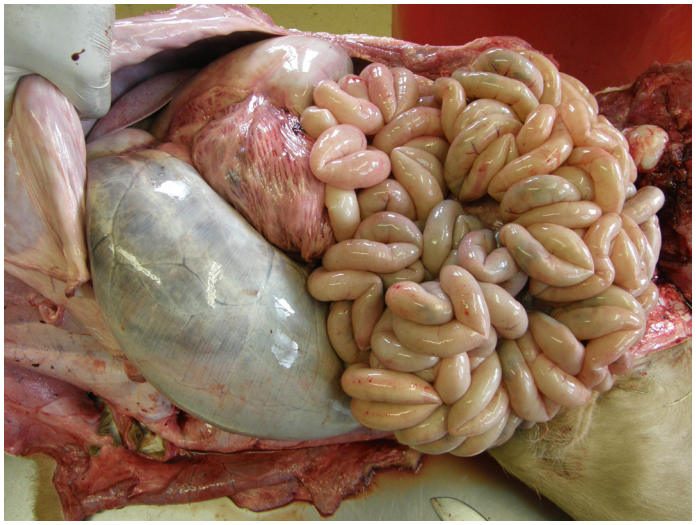

Figure 6. Bovine calf: abdomen opened with dilated abomasum with tympany.

Gastritis

In many septicaemias in pigs (salmonellosis, swine dysentery, Glassers' disease, colibacillosis), bacterial emboli occlude vessels in the submucosa with associated thrombosis, haemorrhage, infarction and ulceration. Similar pathology can be induced by vomitoxin, a mycotoxin produced by *Fusarium* species.

Genuine gastritis is usually reported in dogs and cats with more generalised gastrointestinal inflammatory diseases such as food allergy or idiopathic inflammatory bowel disease. Infectious agents in small animals appear to be minor significance when compared with chemical, mechanical, or idiopathic insults.

The role of gastric *Helicobacter*-like organisms in gastritis in dogs and cats remains controversial and confirming a causal relationship between *Helicobacter* and gastric disease has proved very difficult.

Gastric/abomasal ulcerations

Gastric ulcers are associated with the administration of nonsteroidal anti-inflammatory drugs in horses and dogs. Equine gastric ulcer syndrome is observed in 40-90% of competitive and performance horses.

Gastroduodenal ulcer disease is a well-documented syndrome in suckling and early weanling foals. This condition is seen most commonly in intensive mare/foal operations. The most severe lesions are usually observed in the anterior duodenum and ulceration is frequently associated with duodenal and/or gastric perforation. Experimentally, the disorder has been reproduced by excessive dosing with nonsteroidal anti-inflammatory drugs and there has been some association with rotavirus diarrhoea. Nonsteroidal anti-inflammatory drugs are excreted via the milk and so treatment of the lactating mare with such drugs increases the risk of exposure to the suckling foal. It is the submucosal vascular thrombosis observed in this condition that is believed to be the central pathogenic mechanism in the development of ulceration due to mucosal ischemia.

In ruminants, abomasal ulceration has been described with rumen acidosis, abomasal displacement, impaction, torsion, BVD, heavy metal poisonings and lymphoma.

Most cases in dogs and cats are idiopathic although some cases in dogs have been associated with the use of nonsteroidal anti-inflammatory drugs and gastric ulceration in both species has been associated with mast cell neoplasia. Gastric ulceration is sometimes observed with uremic gastritis in dogs and cats, but this is usually secondary to the mineralisation of the glands and vessels in the lamina propria with associated ischaemic necrosis.

In pig's gastric ulceration involving the *pars oesophagea* of the stomach is a condition with high prevalence under modern pig husbandry conditions. On the other hand,

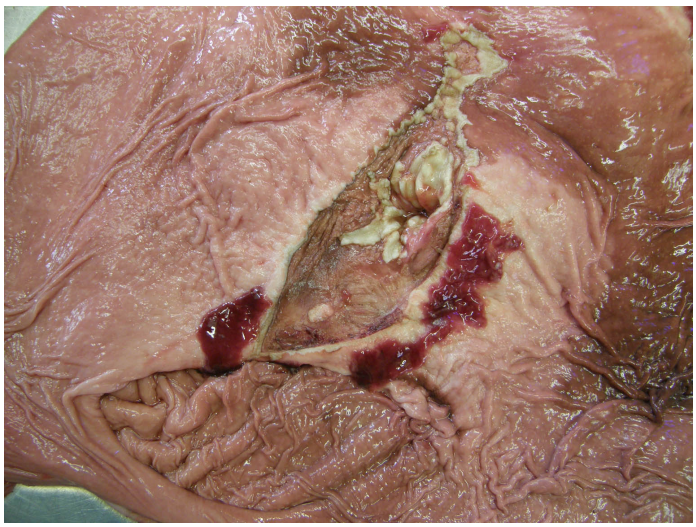

Figure 7. Porcine: mucosal surface of the stomach of the sow with gastric ulceration involving the pars oesophagea.

significant ulceration of the cardiac, pyloric or fundic mucosa is rarely reported in swine. An important predisposing factor is the feeding of finely ground rations, which have proven to be ulcerogenic through their stimulation of acid secretion. Weaners, growers and lactating sows are most commonly affected, mortalities reported are due to perforation and bleed out.

Neoplasia

May arise from the surface epithelium, within the mucosa, tunica muscularis or serosa. Detailed histopathology is required for classification.

Small and Large Intestine

On opening of the abdominal cavity, the intestinal tract is evaluated *in situ* to check for any obvious displacements and to determine that all components are in their expected anatomical locations. The volume and nature of any fluid accumulations within the peritoneal cavity are noted. After examination of the cranial mesenteric root in equines the entire gastrointestinal tract is removed for inspection as regards colour, consistency, symmetry and integrity. Examination of the serosal and mucosal aspects of all areas of the intestinal tract including duodenum, jejunum, ileum, cecum and colon, should be carried out. Evaluation of the intestinal content for the presence of internal parasites should also be performed. In addition, examination of the regional mesenteric lymph nodes forms part of the procedure.

Developmental abnormalities

Occlusion of the intestinal lumen as the result of anomalous development results in atresia, which is then used to name the part of the bowel affected such as *atresia ani* or *atresia coli*.

Megacolon.

A large distended, faeces-filled large intestine characterises this condition and it may be congenital or acquired. Congenital forms are described in pigs, dogs, cats and foals.

Megacolon is not that uncommon in the cat. Most cases are idiopathic and seem to be a result of colonic inertia. Pelvic fracture mal-unions are the next most common cause. Rare cases of feline dysautonomia and congenital hypoganglionosis of myenteric ganglia have also been associated with the condition.

Intestinal displacements

- **Intussusception:** when one segment of the intestine becomes telescoped into the immediately distal segment of the intestine. Development of this pathology is believed to be due to altered motility as a consequence of irritation and or inflammation.

- **Volvulus and torsion:** volvulus involves twisting of the intestine on its vertical mesenteric axis, while torsion involves rotation of the intestine on its longitudinal mesenteric axis. Both volvulus and torsion result in compression of mesenteric veins and arteries resulting in ischaemia of the affected portion of intestine. At post mortem, the affected segment of the intestine is distended with gas and discoloured, either dark red or black. Torsion of the long axis of the mesentery occurs commonly in swine, uncommonly in horses and rarely in dogs and cats. In dogs, mesenteric torsion has been associated with ingestion of large quantities of food and with exocrine pancreatic insufficiency in German shepherd dogs.

- **Renosplenic entrapment:** this is unique to horses with entrapment of the large colon due to left dorsal displacement of the left dorsal colon or left ventral colon between the spleen and left body wall.

- **Right dorsal displacement:** is also an equine condition associated with right dorsal displacement of the left dorsal and ventral colons to the right of the caecum and may result in torsion.

Enteritis/colitis

Various aetiological agents have the capability of inducing specific enteritis and/or colitis pathologies in the various species, including infectious agents (viruses, bacteria, fungi, algae, protozoa), internal parasites, immune mediated diseases, various toxins and idiopathic disorders.

Peritoneum

Ascites

Is defined as excessive accumulation of fluid within the peritoneal cavity and includes transudates, modified transudates and exudates. This condition is non-specific

and can result from any cause of hypoproteinaemia such as heart, liver or kidney failure, protein-losing enteropathies, lymphangiectasia, lymphatic blockage, ruptured lymph ducts, bladder rupture and hypertension.

Fat necrosis

The four main categories of fat necrosis are nutritional, pancreatic, traumatic and idiopathic.

- Nutritional – pansteatitis ("yellow fat disease"), is a nutritional disorder which affects cats, swine, equines, mink, Nile crocodiles and fish-eating birds and which has been experimentally induced in rats and chickens by feeding a diet high in unsaturated fatty acids and deficient in vitamin E. This is a generalised disorder of fat deposits characterised by extensive adipocyte degeneration and inflammation with progressive peroxidation of unsaturated fatty-acids (which imparts the yellow discoloration).

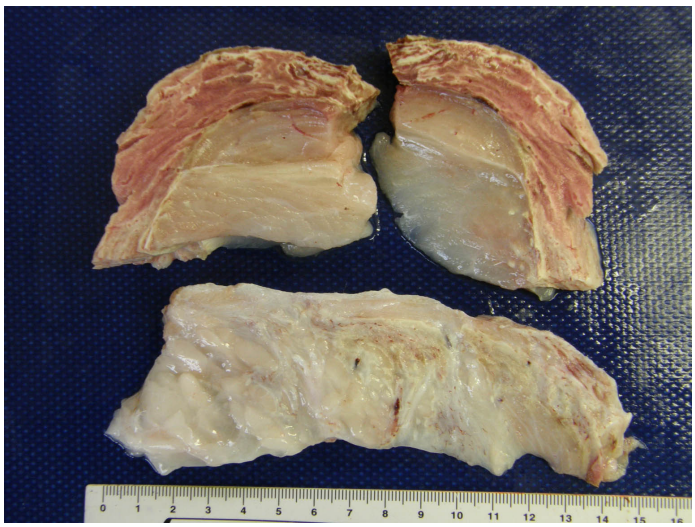

Figure 8. Nile crocodile: yellow fat disease with yellow discoloration of the necrotic subcutaneous fat.

- Pancreatic – Pancreatic tumours and pancreatitis release pancreatic enzymes into the bloodstream. In adipose tissues, these enzymes hydrolyse the stored lipids and release fatty acids and glycerol, which results in panniculitis. Trypsin release results in a hypercoagulable state that damages blood vessels. Resultant ischaemia causes further adipocyte damage and necrosis. The released fatty acids combined with calcium, magnesium and sodium lines forming soaps resulting in chalky white deposits within the affected fat.
- Traumatic - is most commonly observed in dogs and cats, and results from direct, usually blunt trauma to adipose tissue, chronic pressure or decreased blood supply. This latter finding is more common in obese cats in which excessive fat deposition is accompanied by reduced vascularity, predisposing to ischaemia from the slightest of insults.
- Idiopathic – can be focal or massive and occurs in all species but observed most commonly in large animals, especially sheep, obese dairy cows and horses. Massive necrosis of intra-abdominal fat can envelop intestinal loops resulting in stricture and functional blockage. The exact aetiology remains unknown but suspected to be nutritional and origin.

Peritonitis

Various aetiological agents are capable of inducing inflammation of the peritoneum, including viral, bacterial, parasitic, mechanical, as well as bile peritonitis as a result of organ rupture. Most infections of the peritoneal cavity are traumatic and origin. Vasculopathies/ vasculitis resulting in impaired vascular permeability are observed with various infectious aetiologies in particular endotheliotrophic viruses.

Neoplasia

Intra-abdominal/omental lipomas and mesotheliomas are the most common primary neoplasms of the abdominal cavity in animals.

Sample Collection Procedures

Histopathology

Tongue

The organ should be bread sliced into 1-cm-thick slices for examination and a slice is then placed in 10% buffered formalin for histopathology.

Oesophagus

Intact tubes of oesophagus, 1 to 2 cm in length, should be placed in 10% buffered formalin for histopathology. Retention of intact tubes facilitates thorough examination of the oesophagus as orientation of the mucosa, submucosa and tunica muscularis are retained and so all the critical structures can be easily located and evaluated on the histological sections.

Fore-stomachs (ruminants)

All three compartments should be sampled (reticulum, rumen and omasum). Histological evaluation enables a critical insight into the digestive status of the individual,

particularly evidence of chemical rumenitis (rumen acidosis, rumen alkalosis, kikuyu poisoning). Samples must be fresh with mucosa still attached, if mucosa has started to slough away it is unlikely that any useful histological interpretation will be obtained.

Stomach/abomasum

All three areas of the stomach namely cardiac (asterisk), fundus (squares) and pyloric (circle) need to be sampled into 10% buffered formalin for histological examination (figure 9). Rapid fixation is a prerequisite to avoid post-mortal artefacts of acid auto-digestion.

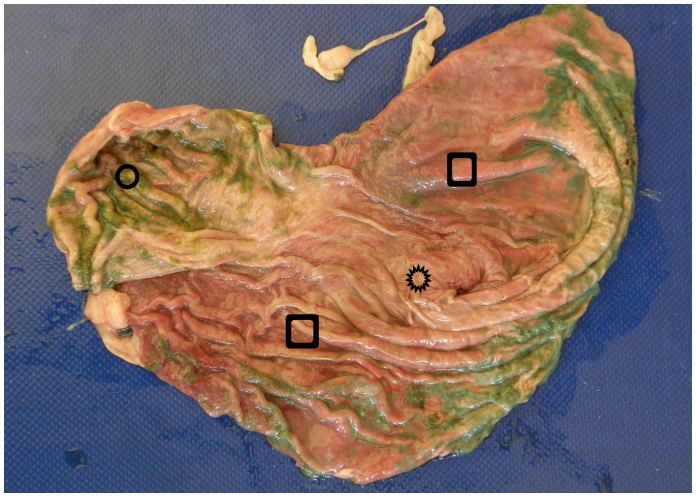

Figure 9. Canine stomach mucosal surface: demonstrating cardiac (asterisk), fundus (squares) and pyloric (circle) areas that need to be sampled for histopathological examination.

Small and large intestine

The intestine is an extremely large organ and therefore it is essential that multiple sites be collected for evaluation i.e. duodenum, jejunum, ileum, caecum and colon. Where possible, it is always extremely useful to include a mesenteric lymph node. To examine the intestinal mucosa at necropsy, the intestine is opened along the anti-mesenteric border. Intestinal tissue collected for histopathology should be retained as a tube, open at each end, so that all layers (mucosa, submucosa, muscularis and serosa) are included in their normal architectural pattern.

Rapid fixation is important for maintenance of villous and microvillous morphology, which is essential for the histological diagnosis of many agents such as *Cryptosporidium*, attaching and effacing *Escherishia coli*, *Giardia* etc., and is achieved post-mortally, by gently washing out the ingesta from the tube of intestinal tissue with 10% buffered formalin. As demonstrated in figure 10, the one end of the tube of intestine is grasped with forceps; formalin aspirated into a syringe is then gently run through the intestinal segment, which is then allowed to sink in the formalin container.

Figure 10. Formalin fixation of the intestine demonstrating gentle flushing out of the ingesta from the intact tube of intestine with formalin.

Microbiology

The preferred sample type for microbiological cultures are charcoal medium swabs as these enable culturing of both anaerobic and aerobic bacteria as well as fungi. The charcoal medium also enables bacteria and fungi to survive for prolonged periods, should there be any delays in transport to the laboratory. Sterile 3 cm intact sections of fresh intestine can also be used for culture purposes but should only be considered if charcoal medium swabs are not available. Where anaerobic cultures are to be performed on fresh intestinal sections, the segments submitted should be tied off at both ends and then placed in a sample envelope which is folded to exclude all air.

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MULTIPLE-CHOICE QUESTIONS

QUESTION 1

At what rumen pH range is production of stable foam at its highest?

a. 3 – 4
b. 4 – 5
c. 5 – 6
d. 6 – 7
e. 7 – 8

QUESTION 2

Which of the following are not considered post-mortal indicators of ante-mortal rumen acidosis?

a. Watery rumen content with acidic smell
b. Watery intestinal content
c. Rumen papillae discoloured brown/black
d. Rumen epithelium is friable
e. Rumen epithelium is tightly adherent especially on the ventral sac

QUESTION 3

Which of the following pathologies are considered characteristic of a mycotic rumenitis?

a. Clumping of rumen papillae
b. Large areas of denuding of rumen papillae
c. Excessive adherent exudate on the surface of the rumen papillae
d. Well circumscribed, transmural circular infarcts
e. Extensive sloughing of the rumen mucosa

QUESTION 4

Which of the following factors are not thought to contribute to volvulus of the stomach in the dog?

a. Repeated gastric dilation
b. Overfeeding
c. Aerophagia
d. Post-prandial exercise
e. Pre-prandial exercise

QUESTION 5

Which of the following bacteria are capable of inducing primary abomasal pathology?

a. *Sarcina* species
b. *Clostridium novyi*
c. *Clostridium sordelli*
d. *Clostridium perfringens* type D
e. *Clostridium perfringens* type C

QUESTION 6

Which of the following factors is thought to be a major causative factor in the gastroduodenal ulcer disease syndrome of foals?

a. Coronavirus
b. Nonsteroidal anti-inflammatory drugs
c. Strongyle verminosus
d. *Trichothecene* mycotoxins
e. *Clostridium perfringens* type A

QUESTION 7

In which region of the stomach are gastric ulcers most commonly encountered in pigs?

a. Greater curvature
b. Lesser curvature
c. *Pars oesophagea*
d. Fundus
e. Pylorus

QUESTION 8

Which of the following aetiologies are not known to cause fat necrosis?

a. Diets deficient in copper
b. Diets high in unsaturated fatty acids
c. Diets deficient in vitamin E
d. Pancreatitis
e. Trauma

QUESTION 9

In which species is traumatic fat necrosis commonly observed?

a. Pigs
b. Sheep
c. Cattle
d. Horses
e. Dogs

QUESTION 10

Which are the preferred samples for performing microbiological cultures on the intestinal tract?

a. Ames medium swabs
b. Charcoal medium swabs
c. Intestinal content in a sterile container
d. 3 cm section of intestine
e. Intestinal segment tied off at both ends

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