★ MEDICINE

★ Liver.

Pathophysiology of:

★ Photosensitivity.

- hypersensitivity to sunlight due to exo/endogenous photodynamic substances.
- can ingest them or from hepatic cell damage get accumulation of phylloerythrin = inflam ^ & necrosis of unpigmented skin.

★ Petechiae

- Liver disease causing ↓ in or lack of production of clotting factors
- pinpoint haemorrhage.

★ Hepatic Encephalopathy

- Neurobehavioural signs that may fluctuate due to severe hepatic insufficiency or failure.
- caused by abnormal neurotransmitters (ammonia toxicity?)

★ Melaena or acholic faeces

- Melaena may be due to gastro-intestinal haemorrhage due to gastro duodenal ulcers

Acholic faeces → if have obstruction of extra-hepatic bile duct - no bile in faeces - no faecal pigments + fat malabsorption.

★ Jaundice

- Bilirubin in blood (elevated levels)
- Pre/hepatic/post - if hepatobiliary disease could be:
  - reduced clearance
  - impaired conjugation or bile flow

★ Ascites

- Transudate/mod transudate in abdomen due to hypoalbuminaemia, portal hypertension, Na + fluid retention

* Vomiting + Diarrhoea
- Vomiting not assoc. w/ feed - hepatic failure - ↑ of toxins in blood - trigger chemoreceptor in 4th ventricle.
- abnorm stimulat° of autonomic Syster
DIARRHOEA → bile acid abnormalities + fat malabsorption.

* Polydipsia + Polyuria
- often see w/ portosystemic shunts - portal circulation bypasses liver - straight to circulat°.

* Clinical Pathology → Dog → Acute hepatopathy
→ elevated Alanine Aminotransferase (ALT) (2-3x normal)
└ use in conjunction w/ (CK = muscle)
→ elevated Aspartate Aminotransferase (AST)
- sensitive / not specific, ↑ in liver = >> sever than ALT.
→ elevated Alkaline Phosphatase (ALP)
→ sig if 2x normal
→ elevated Gamma-Glutamyltransferase (GGT)
- more liver specific than ALP (dogs)
- also indicates cholestasis
→ Hyperbilirubinaemia - impaired uptake / conjugation
→ (may cause Bilirubinuria
→ May get increased serum Bile Acids → Prob more chronic.

* Chronic Liver Insufficiency. (Clin path'd)
- In chronic processes N2 levels may be w/in normal ranges + indicators of function are abnormal.
- ∴ MAY get ↑'s in ALT, AST, ALP + GGT.
- Hyperbilirubinaemia + Bilirubinuria
- ↑ in serum Bile Acids - may be markedly ↑ ↑ post-prandially.
- Elevated ammonia in blood (terminal hepatic insufficiency + sever, diffuse necrosis)

* Chronic Liver cont.

- decreased Urea Synthesis
- decreased Globulin Synthesis
- decreased Albumin Synthesis
- low Fibrinogen levels (if no inflammation)
- Hypoglycaemia
- Clotting factor deficiencies
- ↓ Cholesterol levels

* ALT + ALP.

ALT - Alanine Aminotransferase
- Sig. liver damage if 2-3 x normal levels
- reasonably liver specific - dogs & cats - elevated w/ muscle damage ∴ do CK as well.
- ↑ levels due to damage or leakage.
- ↓↓'s in 2-3 d if insult resolves & is norm in 2-3wks
- not used → Horses, cattle, sheep, goats, pigs.

ALP - Alkaline Phosphatase
- Not specific for cholestasis - usually elevated from this cause though.
- elevation significant if 2x normal
- any elevation in cat = sig.
- not used - ruminants or horses.
- Liver, bone, intestine, placenta (dogs - corticosteroid induced)

* ID & GGT

ID - Iditol Dehydrogenase
- used for - Horses, Cattle, Sheep, Goats
- short ½ life - returns to norm in 4-5 d (acute)
- ↑ in horses w/ strangulating bowel lesions & acute enterocolitis

GGT - GammaGlutamyl Transferase
- More specific for cholestasis than ALP

GGT
- More liver specific - dogs - than ALP
- detects cholestasis → Horses, cattle, sheep, pigs, dogs
- ↑ levels urine = tubular damage

★ Measurement of Bile acids
- Detect dysfunction of liver
- increased serum levels if
  - hepatocellular damage
  - Cholestasis
  - ↓ functional mass
  - Abnorm's of portal circulation
- low if hepatic uptake norm. 725 μmol/L = histol/gross lesion
- Post-prandial - ↓ in ileal disease
- Markedly ↑↑ - post prandial = Vascular shunting & cirrhosis.

★ Jaundice
Pre-hepatic
- over-prod⁻ of Bilirubin - saturates liver
- haemolysis, auto-agglut⁻, +Baemia, reticulocytosis ....

Hepatic Jaundice
- Due due hepatocellular disease
- acute or chronic → Impaired uptake or conjugation of Bilirubin.

Post-hepatic
→ Obstruction to bile flow → reflux BR → blood
- Intrahepatic obstruction - swelling of cells, or
  Extrahepatic " "
- lesions of bile duct, gall bladder, pancreas or bowel

*Non-hepatic causes of jaundice:
- mild - septic patients
- hyperthyroid cats
- starvation, fever,
- artefact due to lipaemia, haemolysis

* Clinical Pathol → Porto-Systemic Shunt.
- Decreased serum Albumin and/or Globulin (dogs)
- Mild → Mod increase - ALT & ALP (can be norm)
- Increased pre and/or post-prandial serum bile acids
- Increased (fasting) blood NH_3 (NH_3 tol test)
- Ammonium biurate crystalluria
- RBC microcytosis - no anaemia
- Low urea, glucose, cholesterol

* 4 Processes lead to Hepatobiliary disease?
- Clinical pathol tests can differentiate.
1. Inflammation
2. Fibrosis
3. Infection
4. Toxins.

* CBC, Biopsy, NZ's, ultrasonography, radiography, C&S, Bilirubin, bile acids, cholesterol, Albumin, urea, glucose, PCV.

✱ Pancreatitis - Clinical Present° - Dogs & Cats.

Hxx = Well nourished, high fat diet.
- scraps, garbage
- abdominal trauma, shock, glucocorticoids, Morphine, hyperlipidaemia, organophosphates .....

Clinical signs: ⇒ often obese, middle aged, female dogs.
- Acute abdominal pain
- Febrile, dehydrated
- Vomiting, anorexia, dehydrated
- increased thirst, often drinking small amounts
- shock may develop.

Cats - difficult to dx.
- anorexia, lethargy, wt loss
- fever, hypothermia
- tachycardia
- abdom. pain

✱✱ Icterus (20%) + vomiting (40%)
- dehydrat°, pallor
- inflam bowel disease
- cholangiohepatitis

* Interpret° of Clinical Pathol. pancreatitis - Dogs/Cats/Horses

- AMYLASE
- increased in dog 3-4x in pancreatitis
- may remain normal though
- decreased or norm. levels in cats
- increased (not always) in horses

LIPASE
- More specific - often raised in dog (can be norm)
- 3x increase usually = acute pancreatitis
- increased levels in some cats w/ " "
- May be increased or norm. in horses - abdom fluids useful

* TRYPSIN-LIKE IMMUNOREACTIVITY
- increased maybe in dogs
- May be BEST to detect pancreatitis in CATS.

- May have Neutrophilic leucocytosis w/ left shift, may have toxic neutrophils.
- fasting hyperlipidaemia & hypercholesterolaemia
- high PCV & plasma protein

* Horses - may have hyperfibrinogenaemia - acute

May:
- ↓ albumin
- transient hypocalcaemia
- ↑ ALT & ALP - 2° liver damage
- hyperBLaemia
- exudate - peritoneal cavity w/ ↑ amylase & lipase
- hyperglycaemia

* How Evaluate Dog/Cat
- Ultrasound, radiographs, Abdom fluid
- determine severity - Septic ? Shock? Diabetes M? renal failure? Hypocalcaemic?

* Treatment Options
- Fluids / Nil per os
- Δ diet permanently
- anti-emetics
- Ab's parenterally
- Pain relief
- Corticosteroids: Not if contribute to problem
- Plasma / Neurotect
- Abdom. lavage + surgery if unresponsive

* Pathogenesis + Clinical Present= - Dog w/ EPI
- develops due to loss of exocrine panc acinar cells
- leads → Maldigestion - loss of digestive NZ's + $NaHCO_3$
- acidification of gut contents
- affects mucosal function + =precipitat= of bile salts
- commonly due → Panc. Acinar Atrophy
also (Autosom recessive → GSD's)
- caused by chronic pancreatitis + destruct= - tissue

- Δ in SI = Mucosal NZ abnormalities, impaired transport of f.acids, sugars, a.a's
- Malabsorpt= - Vit B_{12}
- ↑ serum levels of folate

*Clinical Signs:
- wt loss
- ravenous appetite
- ↑ faecal bulk - grey, greasy, fatty
- Diarrhoea which improves w/ fasting or low fat diet
- pups + young adults (GSD) panc. acinar atrophy.

* How Dx EPI? (Dog/cat)
- Clinical signs + Hx.
* Decreased → Trypsin-like Immunoreactivity
- Dogs in Australia (cats - US)
* Plasma turbidity test
- feed corn or peanut oil to dog - collect heparin blood pre + post prandially
- should see clear then turbid post. If don't - do again w/ panc. NZ. If turbid post - then know it is EPI.
* Faeces w/ undigested meat fibre, neutral fat, starch
* Faecal proteolytic activity

* Treatment
- low fat, highly digestible, low insoluble fibre diet 2-3 times /day.
- allow what NZ's have to work in small amts - don't stress too much w/ lots fat/protein etc
- Pancreatic enzymes (as have deficiency)
- Vit E + B12 - not absorbing properly - antioxidants
- Cimetidine? H2 blocker - ↓ acidity - less mucosal damage
- Al/Mg hydroxide gel - ↓ steatorrhoea
- Neomycin - non-absorb Ab → ↑ fat digestion - prevent bact. overgrowth.

✱ Clinical signs w/ Oral / Pharyngeal disorders

Oral cavity disease
- difficulty in food prehension
- dysphagia
- ptyalism
- difficulty w/ mastication
- halitosis, inappetance, wt loss
- anorexia
- rubbing mouth, excess salivation
- food dribbles out of mouth

Pharyngeal
- Gagging or regurgitation (not vomiting)
- dysphagia
- Snoring or inspiratory stridor
- anorexia
- ejection of food frm pharynx or nasopharynx
- ptyalism

✱ Dx tests for oral / pharyngeal

a Oral
- Physical exam - watch eat, drink - manual exam
- Radiography + imaging techniques
- Histopath / FNA / impression smears / aspirat⁼ of fluid
- LN
- Microbiol
- clinical path - CBC, serum BC, USG, UA

Pharyngeal
- Systemic exam
- Radiography, histopath, FNA, imp⁼ smears, FNA, LN, clinical path
- endoscopy, dental mirror, CT, contrast videofluorometry, Electromyogr