Pathology of the Liver and Biliary Tract – 2
Developmental, Circulatory and Metabolic Disorders

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Congenital cysts most likely originate from capsule or the embryonic bile ducts (ductal plate malformations).
DEVELOPMENTAL ANOMALIES

Congenital Cysts

- Differentiate from parasitic cysts
DEVELOPMENTAL ANOMOLIES

Congenital Cysts

- Polycystic liver disease can be incidental or significant!
Rupture and Displacements

- Hepatic Displacements
  - Ventral hernia
  - Diaphragmatic hernia

- Rupture
  - Trauma
  - Enlarged liver is predisposed
  - Rapidly fatal (blood loss)
**MISCELLANEOUS CHANGES**

**Tension Lipidosis**
- Focal areas of lipidosis near mesenteric attachments
  - Cattle and horses

**Capsular Fibrosis**
- Common in horses
  - Resolution of peritonitis
  - Parasitic migration?
Grouped according to whether blood flow into, through, or from the liver is impaired

- Impairment of blood flow from the liver
- Impairment of blood flow through the liver
- Impairment of blood flow to the liver
CIRCULATORY DISTURBANCES

Impaired blood flow into the liver

- Impairment of blood flow through the portal vein (>hepatic artery) before it enters the liver
- Possible sequelae:
  - Portal hypertension (prehepatic)
  - Liver infarcts

Possible sequelae:
- Portal hypertension (prehepatic)
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Portal Vein Hypoplasia (Microvascular dysplasia)

- Congenital malformation → portal vein hypoplasia
- Histologic diagnosis – US is often normal
- Small and toy breed dogs: Yorkies
- Secondary histologic findings: arteriolar hyperplasia, lobular atrophy
- Clinical findings are similar to PSS
- +/- portal hypertension and ascites
Impaired blood flow through the liver

- Due to increased resistance of blood flow within the sinusoids
  - Cirrhosis*
  - Amyloidosis
  - Intrahepatic arteriovenous shunts
- Sequela:
  - Portal hypertension (intrahepatic)
  - Acquired PS shunts

* Cirrhosis, cat
Conditions that lead to ↑ resistance to venous outflow in the hepatic vein or vena cava
  • Passive congestion*
  • Right heart failure*
  • Venal caval thrombosis
  • Thrombosis of the hepatic vein
  • Veno-occlusive disease

Sequela:
  • Portal hypertension (posthepatic)
Acute Passive Congestion

- Slight enlargement of liver
- Prominent reticular pattern

Chronic Passive Congestion

- Hepatomegaly with rounded margins
- Nutmeg appearance
  - Centrilobular congestion
  - Midzonal fatty change
  - Centrilobular fibrosis & hemosiderosis

Impaired blood flow from the liver

Acute passive congestion

Chronic passive congestion
Impaired blood flow from the liver

Histology – Chronic passive hepatic congestion

Centrilobular fibrosis and siderophages

CIRCULATORY DISTURBANCES

Congenital portosystemic shunts

- Allows blood from the portal venous system to bypass the liver and enter systemic circulation
  - Usually a single large-caliber vessel (unlike acquired shunts → multiple small vessels)
- Decreased vascular flow and trophic substances to the liver → hepatic atrophy
- No portal hypertension or ascites (unlike portal vein hypoplasia/microvascular dysplasia)
- Toxic substances (ammonia) are not removed from the blood
- Ultrasound +/- contrast imaging are crucial for clinical diagnosis!

Other Vascular / Circulatory Disorders

Clinical signs:
- Stunted growth
- Hepatic encephalopathy

Clinical pathology findings:
- Hyperammonemia
- Decreased urea
- Increased bile acids
- Ammonium biurate crystals in urine
CIRCULATORY DISTURBANCES

Congenital portosystemic shunts

Extrahepatic PSS:
- Portocaval or Portoazygous
- Small breed dogs and cats

Intrahepatic PSS
- Patent ductus venosus
- Large breed dogs

Secondary changes:
- Portal regions lack a portal vein and have multiple arterioles
- Looks identical to primary portal vein hypoplasia (microvascular dysplasia) on histology

CIRCULATORY DISTURBANCES

Telangiectasia

- Focal areas with dilated sinusoids filled with blood
- Gross: Multiple 1 – 5 mm dark red foci
- Histo: Dilated sinusoids and loss of hepatocytes
- Incidental in cattle and cats

Anemia

- Centrilobular degeneration / necrosis
  - This zone receives the least $O_2$
Hemorrhagic infarcts secondary to mycotic rumenitisavo.

CIRCULATORY DISTURBANCES

Other Vascular / Circulatory Disorders

• Uncommon
  • Dual blood supply
• Thrombosis of hepatic artery
• Torsion of hepatic lobe
  • Infarction, shock and death may occur
• Secondary to mycotic rumenitis
• Hepatic vein thrombosis

Hemorrhagic infarcts secondary to mycotic rumenitis, cow
### ACCUMULATIONS OF:

- Fat
- Glycogen
- Amyloid
- Copper
- Pigment
**Sources of FFA:**
- Adipose tissue
- Chylomicrons from gut
- Hepatic production from AA and glucose (not in ruminants)

**FFA in liver:**
- Esterified to triglycerides
- Used as energy (oxidized)
- Production of phospholipids & cholesterol
- Complexed with apoproteins
  - Released as lipoproteins

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*Pathologic Basis of Veterinary Disease (2006), 4th ed., Mosby-Elsevier, chapter 8*
Fatty Liver (Hepatic Lipidosis)

Fatty liver occurs when the rate of triglyceride accumulation in liver exceeds rate of degradation or secretion into circulation

Potential mechanisms of hepatic lipidosis:
• Excess dietary intake of fat / carbohydrates
• Excess mobilization of fat from stores
  • Due to ↑ demand (lactation, starvation)
• Abnormal hepatic function(↓ oxidation FA)
• ↑ esterification of FA
• ↓ apoprotein synthesis or impaired secretion (hepatoxins / drugs)
Fatty Liver (Hepatic Lipidosis)

- Rounded margins (hepatomegaly)
- Soft – friable greasy texture
- Enhanced reticular pattern or diffuse yellow to orange discolouration
- May float in fluid
METABOLIC DISTURBANCES

• Macrovesicular lipidosis
  • Single large distinct clear vacuole in the hepatocellular cytoplasm – displaces the nucleus to the periphery

• Microvesicular lipidosis
  • Multiple small distinct clear vacuoles within the hepatocellular cytoplasm
Fatty Liver (Hepatic Lipidosis)

Significance of Hepatic Lipidosis

- Depends on:
  - Cause
  - Severity
  - Duration
- Reversible in mild cases
- May cause:
  - Hepatocellular necrosis
  - Fat embolism
  - Liver rupture → hemoabdomen
  - Increased susceptibility to toxic damage
Fatty Liver (Hepatic Lipidosis)

Dietary Cause
- Dietary excess
- Fasting in obese animals
- Cobalt / Vitamin E deficiency

Toxic / Anoxic Injury
- Decreased oxidation of fatty acids
- Decreased formation/secretion of lipoproteins

Causes/syndromes of lipidosis

Ketosis*

- Excess fat metabolism due ↑ energy demand
- Pregnant ewes (= pregnancy toxemia)
- Lactating dairy cow

Bovine fatty liver syndrome*

- Obese animals
- Anorexia
  - Retained placenta, metritis, milk fever, abomasal displacement

Causes/syndromes of lipidosis

Fatty Liver (Hepatic Lipidosis)
METABOLIC DISTURBANCES

Fatty Liver (Hepatic Lipidosis)

Feline fatty liver syndrome*
- Idiopathic
- Obesity + anorexia
- Progresses to liver failure with icterus and hepatic encephalopathy

Equine hepatic lipidosis
- Obesity, pregnancy, lactation
- Ponies, mini horses, donkeys
- Hyperlipemia, hepatic encephalopathy, liver rupture, DIC

Endocrine disorders
- Diabetes
- Hypothyroidism

Causes/syndromes of lipidosis
• Glycogen is normal in hepatocytes
• Excess storage of glycogen occurs with:
  – Diabetes mellitus
  – Hyperadrenocorticism*
    • “Steroid hepatopathy”
      – Cushing’s Disease
      – Iatrogenic
      – Prolonged stress
  – Glycogen storage diseases
Glycogen Accumulation

- Steroids induce glycogen synthetase → promotes hepatic storage
- Enlarged, pale liver due to swollen hepatocytes (midzonal areas)
- PAS stain distinguishes it from fat

Often have ↑ Alkaline Phosphatase (ALP)
Questions?