Post Mortem Approach to the Liver

Gross Examination

Examine the liver in situ – after opening the abdominal cavity examine the liver in context with other changes in the abdominal cavity. Gently wash out any excessive fluid accumulation or haemorrhage in the abdomen to provide a clear in-situ picture of pathology (Figure 1). Run water as a gentle soak and **not under pressure** as this may remove and destroy critical lesions such as hematoma, adhesions etc. Figure 2 is a case of a ruptured liver in a calf with hematoma and adhesions associated with the rupture site clearly indicating antemortal pathology. If this material had been removed by high pressure washing of the abdomen, it then becomes extremely difficult to distinguish between ante-mortem and post mortal rupture.

Examine both the diaphragmatic and visceral aspects – evaluate size, colour, shape, lesion distribution and consistency. Since the liver capsule is unable to expand any, enlargement of the liver results in rounding of the liver margins (Figure 3). Consistency is evaluated by compressing a 1 cm thick slice of liver between the thumb and index finger.
Examination of vascular supply to liver – observe the entrance of the portal vasculature into the hepatic parenchyma for any evidence of shunts or other anomalies. In this example of hepatic arterio-venous fistulae, note the aneurismal dilatation of portal veins and tortuous hepatic arteries.

Importance of “Bologna Slicing” – slicing the organ into 1 cm thickness slices not only enables assessment of consistency but also allows for evaluation of the cut surface and assists in determining the distribution of lesions and exact location of focal lesions. Slicing also enables assessment of the consistency throughout the organ.
Common Gross Liver Lesions

**Figure 8**

**Diffusely enlarged liver (hepatomegaly)**
May be seen in a variety of conditions where there is either diffuse swelling of hepatocytes, blood pooling or diffuse infiltration by neoplastic cells. Hepatomegaly with “congestion” is a common post mortal finding due to terminal hypostasis with rigor mortis and intestinal gas pushing mobile blood to the liver from the muscle and bowel (Figure 8). It is also noted with degenerative hepatopathies (toxic, metabolic) and fatty liver syndromes where there is significant swelling of hepatocytes. Diffuse neoplastic infiltrates as might be expected with lymphoid, myeloid and mast cell neoplasia.

**Figure 9**

**Chronic passive congestion (nutmeg) liver**
Liver may be slightly enlarged but retains its smooth surface. Parenchyma has a mottled dark and light parenchyma due to the blood pooling in centrizonal areas. As the lesion progresses colour becomes more uniform with arborizing pale areas in a dark background. The cause of this pathology is increased resistance to forward blood flow and is seen with chronic heart
disease, lung diseases, heart abnormalities and caudal vena caval obstruction (thrombosis, neoplasia). This lesion can be seen in animals of all ages including the fetus.

![Image](image1.jpg)

**Figure 10**

**Figure 11**

**Fatty Liver**

Characterised by a diffusely swollen yellow liver with rounded edges (Figure 10). There is decreased consistency and affected liver biopsies float in water (Figure 11). This condition may be seen in all species with acute starvation when there is body fat to mobilize to the liver. In chronic starvation, fatty liver is not observed as there is no fat to mobilize. It is also observed with metabolic diseases (negative energy balance, diabetes mellitus, ketosis in cattle, pregnancy toxaemia in sheep), toxic hepatosis (aflatoxins, *Cycad* sp) and hyperlipidaemic syndromes (horses, cats).

![Image](image2.jpg)

**Figure 12**

**Extensive (massive / sub-massive) hepatic necrosis**

Associated with extensive hepatocyte necrosis across all lobules with parenchymal discolouration (necrotic liver tissue paler in colour). Consistency is markedly decreased. Most commonly seen with acute toxic hepatosis or viral hepatitis.
Miliary hepatic necrosis
This is characterised by small pale foci of necrosis evenly distributed through all lobules of the liver. It is most commonly observed with embolic bacterial infections (Figure 13), as observed in this case of Tyzzer's disease (*Clostridium piliforme*) in a cat, certain protozoal infections (Figure 14) as in this case of toxoplasmosis from a Dassie and some instances of viral hepatitis (Bovine herpes vius abortion).

Multifocal hepatic necrosis
Multifocal areas of necrosis with some lobules affected by a greater or lesser degree. This a common feature of necrobacillosis (*Fusobacterium necrophorum*) in cattle (Figure 15) and a feature of many mycotic infections.
Decomposition (putrefaction) vs Infectious necrotic hepatitis
Decomposition often characterised by multifocal to miliary discrete pale foci scattered through the liver. Many are associated with the portal areas creating a zonal pattern than can be mis-interpreted as miliary necrosis as evidence by the canine liver in Figure 16. What is important about putrefactive foci is that they are not rimmed by inflammation (reaction zone). Putrefactive liver usually floats in liver due to the post mortal gas production. Infectious necrotic hepatitis is also associated with Clostridial bacteria but in the ante-mortem setting. Dormant clostridia within the liver undergo proliferation in areas of hepatic necrosis which provide the required anaerobic environment. As noted in this case of infectious necrotic hepatitis in a wildebeest (Figure 17) necrotic areas are irregular in shape, are variable in size and rimmed by a red reaction zone of inflammation. In both instances these necrotic / putrefied areas may be associated gas bubbles.

Peliosis hepatis /Telangiectasia
This characterised by dilated blood filled sinusoidal spaces (Figure 18). This condition has been reported in cattle, dogs, cats and humans, although the pathogenesis remains unknown. Two forms of the condition are described either from local obstruction of small branches of the portal vein with subsequent hepatic atrophy and sinusoidal dilatation (phlebectatic / telangiectasis) or from random focal hepatic necrosis (parenchymal type).
Hepatic cirrhosis vs Post necrotic scarring
Hepatic cirrhosis is defined as a uniform increase of connective tissue at the same location and severity equally in all lobules due to chronic repetitive damage (cardiogenic, pyrrhizidine alkaloid etc). Grossly the liver is essentially of normal size or slightly enlarged with normal shape but with increased consistency. Post necrotic scarring on the other hand is a condition that develops following acute hepatic necrosis due to a single insult. Areas of necrosis then undergo nodular regeneration. Grossly the liver has distorted architecture with a multinodular cobblestoned appearance and has increased consistency.

Hepatic cysts
Frequently documented in aborted bovine foetuses as demonstrated in Figure 21. Parasitic cysts, particularly tapeworm cysts are common particularly in young animals. If observed in older animals one must consider biliary tumours particularly if the fluid is green. Hepatic cysts are very common in cats and include obstructive biliary dilatation, biliary cystadenomas and cystadenocarcinomas.
Hepatic tumours
Primary liver tumours hepatocellular and cholangiocellular are well documented (Figure 22). The liver is also a common metastatic location for tumours at other primary abdominal locations (pancreatic, gastrointestinal, splenic).

Sample collection

a. Histopathology

Liver tissue slices no thicker than 1cm to enable good fixation should be collected from the left and right lobes of the liver, as well as from any site with grossly visible lesions. Liver sections should include tissues with capsular tissue as well as tissue from at least 1cm below the capsule (Figures 23 and 24).

Where possible avoid collecting biopsies from liver margins, as they are furthest from the main blood supply and prone to fibrosis. This fibrosis can mask underlying pathology and can be over-interpreted as excessive fibrosis. Large focal lesions should be sampled at the periphery to avoid necrotic centres and evaluate the interaction of the lesion with the normal adjacent tissue.
Tissue slices are then placed in 10% buffered formalin. Ensure that the formalin jar has adequate formalin and is not overfilled with tissue - work on a ratio of 1 part tissue to 9 parts formalin.

b. Microbiology (Bacterial / Fungal culture, PCR, virus isolation)

At post mortem, the liver is considered the most important “catch all” organ of choice for culture, as drainage from all abdominal visceral sites passes through the liver. Liver tissue collected for microbiological purposes should be collected immediately after opening the abdominal cavity while all the organs are still in situ, to avoid any possible contamination.

A ± 1cm block of tissue (maintains central anaerobic conditions), should be collected aseptically and placed in a sterile container. Do not under any circumstances place multiple tissue types in a single container to create a “tissue pool”, as this results in cross contamination and significantly decreases the chances of isolating the primary bacterial pathogen. Alternatively, a charcoal swab can be inserted into the liver tissue and placed in the charcoal gel. Specimens are then transported on ice at 4°C to the laboratory.

c. Mineral Analysis

The objectives of mineral analysis are
- To determine if mineral deficiency or excess exists.
- Access the prevalence of deficiency.
- Estimate endogenous reserves of trace minerals.

There are a group of micronutrients which are best described in liver tissue and these include selenium, copper, zinc, manganese, iron and cobalt. Calcium, phosphorus, magnaesium and electrolytes are better measured in serum.
Formalin-fixed liver tissue is the preferred sample for mineral analysis, as analyses are validated on fixed liver and formalin-fixation provides significant practical advantages when shipping to the laboratory, as there are no cold chain issues. In addition, archived liver tissue stored in formalin over many years can still be analyse enabling retrospective mineral analysis. A minimum of ± 20 g of liver is required for analysis (Figure 26). For most micronutrients normal physiological levels vary with the age of the animal.

Some complications associated with mineral analysis in serum / plasma include

- Plasma / serum concentrations of selenium, copper and zinc are affected by infection, stress, pregnancy and erythrocyte hemolysis.
- Metalloenzyme activities (eg: glutathione peroxidase used to measure selenium) in plasma are depleted during shipment to the laboratory.
- Zinc contamination by rubber stoppers of blood tubes.

Prior to submission to the laboratory, formalin fixed liver is removed from the fixative, covered with tissue paper and placed in a sealed sample envelope. This facilitates sample transport, as without the risk of formalin leakage, absorbent packing material is not required.

Further Reading