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Dr. Supreetkumar Sagar
Department of Veterinary Surgery & Radiology, College of Veterinary & Animal Sciences, Udgir, Latur, Maharashtra Animal & Fishery Sciences University, Nagpur, Maharashtra, India

Dr. SS Pitlawar
Department of Veterinary Surgery & Radiology, College of Veterinary & Animal Sciences, Udgir, Latur, Maharashtra Animal & Fishery Sciences University, Nagpur, Maharashtra, India

Corresponding Author:
Dr. Supreetkumar Sagar
Department of Veterinary Surgery & Radiology, College of Veterinary & Animal Sciences, Udgir, Latur, Maharashtra Animal & Fishery Sciences University, Nagpur, Maharashtra, India

Feline hepatitis: Pathogenesis, clinical aspects, diagnosis, and management

Supreetkumar Sagar and SS Pitlawar

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Abstract
The feline liver plays a central defensive role between the gastrointestinal system and the systemic circulation. Its unique positioning and dual blood supply expose it to a wide variety of infectious agents, toxins, and metabolic by products. Although the liver possesses strong detoxifying and immunological capabilities, several conditions can impair these defences, making cats vulnerable to hepatic inflammation. Kupffer cells-specialized hepatic macrophages account for nearly a third of non-parenchymal liver cells and form a critical line of immune protection.
However, this protection can be compromised by hypovolemia, ischemic injury, Cholestasis, Chronic liver disease, Portal hypertension, Immune dysfunction, Endotoxemia, and Structural abnormalities in the hepatic vasculature. These conditions increase susceptibility to invading microorganisms, systemic pathogens, and locally produced toxins.

Keywords: Carcass characteristics, weaner pigs, meat quality, palm kernel cake

Introduction

Etiology

Hepatitis in cats may arise from bacterial, fungal, protozoal, viral, parasitic, or non-infectious (toxic) origins. Because clinical presentations often overlap, determining the exact cause requires careful diagnostic assessment.

Bacterial Infections Bacterial causes are commonly associated with cholangitis or cholangiohepatitis. Frequent organisms include: *Escherichia* coli, *Enterococcus* spp. *Bacteroides* spp. *Streptococcus* spp. *Clostridium* spp. *Helicobacter* spp. Special Cases: Tyzzer's Disease (*Clostridium piliforme*) causes multifocal hepatic necrosis in young or immunocompromised animals.

Mycobacterial Infections: Cats may develop localized or systemic disease due to various mycobacteria. Key species: Mycobacterium *bovis*, acquired through ingestion of contaminated raw meat or wildlife exposure, *Mycobacterium lepraemurium* associated with cutaneous lesions, *Mycobacterium visibilis*, may cause widespread granulomatous inflammation, *Mycobacterium avium* complex, opportunistic organisms causing disseminated disease in immunosuppressed cats. Affected cats often display combinations of: Skin nodules, Lymphadenopathy, Hepatosplenomegaly, Granulomatous inflammation in multiple organs

Fungal Infections: Cats may develop hepatic involvement as part of systemic mycoses.

Common fungal agents include: *Aspergillus terreus, Aspergillus deflectus*. Predisposing factors: Exposure to contaminated environments, Immune suppression, Chronic respiratory or gastrointestinal disease, *Sporotrichosis* (*Sporothrix schenckii*) may cause disseminated disease involving skin, lymph nodes, liver, lungs, and occasionally the CNS. Diagnosis often relies on cytology, culture, and histopathology.

Protozoal Infections

- **Cytauxzoon felis:** A tick-transmitted protozoan causing severe systemic infection in domestic and wild felids. Hallmark features include: Schizont-laden macrophages, vascular obstruction, Rapid progression High mortality
- **Toxoplasma gondii:** One of the most common protozoal pathogens in cats. Infection results from: Ingestion of oocysts, Consumption of tissue cysts, and Congenital transmission. Clinical signs may include: Fever, Weight loss, Respiratory distress, Ocular inflammation, Hepatitis with jaundice. Diagnosis is based on clinical signs, serology (IgM/IgG), and treatment response.

Viral Infections

- **Feline Leukemia Virus (FeLV) FeLV may replicate in multiple organs and cause:** Immunosuppression, Hematopoietic disorders, Hepatic inflammation and focal necrosis, Hyperbilirubinemia
- **Feline Infectious Peritonitis (FIP) Mutated feline coronavirus causes a multisystemic vasculitis, liver involvement includes:** Multifocal necrosis, Perivascular macrophage and plasma cell infiltrates, Fibrinous coating of the liver surface in effusive FIP.
- **Parasitic Infection Platynosomum concinnum (Liver Fluke):** This trematode infects the biliary system of cats in tropical and subtropical regions. Clinical signs: Jaundice, Chronic weight loss, vomiting, Diarrhea, Hepatomegaly. Symptoms may take 7-16 weeks to appear after infection. Severe infestations can be fatal.
- **Non-Infectious Hepatitis (Toxic or Metabolic):** Because the liver filters blood directly from the gastrointestinal tract, cats are highly sensitive to: Toxins, Drugs, Environmental chemicals, Toxic plants, Mycotoxins. Hepatotoxic mechanisms include: Oxidative damage, DNA/RNA interference, Lipid peroxidation, Disruption of protein synthesis Massive hepatocellular necrosis.

Pathogenesis

Hepatocellular apoptosis and necrosis interact in complex ways with inflammatory processes within the liver parenchyma, and in many cases, cell injury is the trigger for inflammation. There is ongoing discussion regarding whether the term *acute hepatic necrosis* or *acute hepatitis* is more appropriate for describing non-infectious liver injuries, particularly those resulting from toxins or ischemia, that lead to rapid hepatocellular death. In contrast, infectious agents capable of producing hepatocellular necrosis often classified broadly as causes of hepatitis may induce extensive liver damage with minimal inflammation during the early stages of disease. The definition of chronic hepatitis is well established and refers to conditions characterized by persistent inflammation, fibrosis, and continuing hepatocyte loss by apoptosis or necrosis, regardless of the underlying cause.

Canine and feline herpesvirus infections produce multisystemic disease affecting organs such as the liver, kidneys, and lungs. Hepatic lesions typically consist of multiple, randomly distributed foci of acute hepatocyte necrosis, with variable degrees of associated inflammation. These lesions may extend into the portal or perivenular connective tissue. Occasional hepatocytes or bile duct epithelial cells in the affected regions may contain eosinophilic intranuclear inclusion bodies with peripheral chromatin margination.

Feline infectious peritonitis virus (FIPV) causes a systemic condition that involves the peritoneum, pleura, central nervous system, eyes, and various solid organs. In the liver, the disease commonly manifests as scattered necrotic foci that may extend into surrounding portal and perivenular tissues.

These areas typically have moderate to dense accumulations of inflammatory cells, especially macrophages and plasma cells, concentrated at the margins of the lesions. When the peritoneum is affected, the liver surface may develop a thick fibrinous coating infiltrated by neutrophils, macrophages, plasma cells, and small numbers of lymphocytes, which may also extend into the capsule and superficial parenchyma.

Tyzzer's disease, caused by *Clostridium piliformis*, occurs in both dogs and cats and is characterized by randomly distributed, sometimes confluent zones of parenchymal necrosis, with minimal or absent inflammation. At the interface between viable and necrotic tissue, elongated bacilli may be visible within surviving hepatocytes. These organisms can occasionally be detected in routine H&E stains, but are more readily demonstrated using Giemsa or silver-based stains such as Warthin-Starry.

Protozoal infections caused by *Toxoplasma gondii*, *Neospora* spp., or *Sarcocystis* spp. Affects multiple organ systems, including the liver, lungs, and brain of dogs and cats. Hepatic lesions generally consist of confluent to widespread (panlobular) necrosis accompanied by neutrophils, macrophages, and other inflammatory cells. Necrotic zones and surrounding viable tissue may contain free tachyzoites or cysts filled with bradyzoites. Immunohistochemistry can be employed to distinguish among these organisms.

Chronic hepatitis is comparatively uncommon in cats. Cirrhosis is rare, and when diffuse hepatic fibrosis occurs, it most often reflects porto-portal bridging fibrosis associated with chronic biliary disease.

Clinical Signs

Bacterial Infections

Clinical signs commonly include generalized lethargy, ulcerations of the tongue or oral mucosa, enlargement of regional or systemic lymph nodes, and increased liver and spleen size. Affected animals may show marked leukopenia, often with pronounced toxic changes in neutrophils. Hyperbilirubinemia and bilirubinuria are frequently observed as the disease progresses.

Mycobacterial Infections

Cats with mycobacterial disease often develop a widespread granulomatous condition that may initially present as diffuse skin lesions but can disseminate to multiple internal organs. Clinical manifestations vary depending on the organs in which granulomas form.

Protozoal Infections (e.g., *Toxoplasma gondii*)

Although many cats infected with *T. gondii* may not appear ill, some develop clinical disease. Signs can include fever, reduced appetite, weight loss, and lethargy. Respiratory involvement may lead to pneumonia and difficulty breathing. Ocular inflammation, such as uveitis or retinitis, is possible. Liver involvement may produce hepatitis and jaundice.

Viral Infections

Clinical presentation often reflects the organ systems targeted by the virus, such as the kidneys, liver, gastrointestinal tract, or central nervous system. Some signs are also related to fluid

shifts or vascular injury. Abnormal liver enzyme values may occur secondary to hepatitis, hepatic lipidosis, or the effects of vasculitis, hemolysis, or hypoxia. Hyperbilirubinemia is common and frequently results from hepatic vascular injury.

Parasitic Infections
Cats with significant parasitic burdens may develop a chronic, progressively worsening illness marked by fever, lethargy, abdominal enlargement, and hepatomegaly. Jaundice and severe weight loss can develop due to reduced appetite, vomiting, and mucous diarrhea. Heavy fluke infections can be fatal. Clinical signs typically begin 7-16 weeks after exposure, though eosinophilia may develop as early as 3-14 weeks and persist. Liver enzymes may change, with ALT and AST commonly increasing, while ALP may be normal or only mildly elevated. Hyperbilirubinemia can appear within 7-16 weeks of infection.

Non-Infectious Causes
Toxic or metabolic liver injury may result in hepatocellular swelling, fatty change, and necrosis, which often follow a characteristic zonal pattern (centrolobular, periportal, midzonal, or widespread). Additional features may include cholestasis, inflammatory responses, and varying degrees of fibrosis.

Diagnosis
Bacterial Infections
Detection typically relies on identifying microscopic agglutinating antibodies, which is the most commonly used diagnostic approach. Indirect fluorescent antibody assays may also support diagnosis.

Fungal and Protozoal Infections
Diagnosis may involve cytological evaluation, serologic testing, and, in some cases, assessing the patient's response to treatment.

Viral Infections
A definitive diagnosis usually requires histopathological examination, supported by clinical history, physical findings, and laboratory data. Newer PCR-based tests can aid in confirming infections such as FIP.

Parasitic Infections
Early detection can be challenging. Diagnosis becomes more straightforward once parasite eggs or stages are identified, particularly in bile samples.

Non-Infectious Conditions
These disorders are generally diagnosed using the patient's clinical history and microscopic evaluation of liver tissue to identify characteristic histologic changes.

General Diagnostic Workup
A comprehensive evaluation may include a complete blood count, urinalysis, electrolyte profile, and biochemical testing. Imaging techniques such as ultrasound and radiography help assess liver architecture, and a biopsy may be collected for more detailed analysis.

Advanced Diagnostic Capabilities
Modern diagnostic facilities use sophisticated laboratory methods and imaging technologies to accurately identify liver diseases in pets and guide appropriate treatment plans.

Treatment
Bacterial and Mycobacterial Infections
Although aminoglycosides are commonly used in human medicine, small-animal treatment more often relies on drugs such as doxycycline, chloramphenicol, or fluoroquinolones. These medications can be effective, but recurrence of clinical signs is unfortunately not uncommon.

Fungal Infections
Systemic fungal diseases are typically managed with antifungal agents from the triazole group, most notably itraconazole and fluconazole. In more severe cases, amphotericin B may be incorporated into the treatment plan.

Protozoal Infections
Therapies such as diminazene or imidocarb have shown beneficial outcomes in some protozoal diseases. Clindamycin is also widely used and can be effective in treating certain protozoal infections.

Viral Infections
Management of viral liver disease is mainly supportive, focusing on controlling symptoms and maintaining organ function rather than eliminating the virus itself.

Parasitic Infections
Praziquantel is typically effective for treating liver fluke infestations and infections caused by *Platynosomum concinnum*.

Non-Infectious Conditions
For toxic or metabolic causes of liver injury, removing or stopping exposure to the offending agent is the first step. Subsequent treatment is tailored to the animal's clinical signs and the specific pathological changes present.

Prevention
Cats should receive a diet that includes easily digestible carbohydrates, high-quality sources of fat, and restricted sodium levels. This nutritional approach helps minimize further liver injury and supports improved hepatic function.

Conflict of Interest
Not available

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