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Histopathological Studies Of Fasciola Hepatica In Goats (2000-2025): A Review

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Abstract: Fasciola hepatica, a trematode parasite affecting ruminants worldwide, remains a major cause of hepatic pathology in goats. This review synthesizes research published from 2000 to 2025 focusing on histopathological lesions, diagnostic advances, and comparative pathology in goat fascioliasis. A total of 27 peer-reviewed studies from PubMed and ScienceDirect were analyzed, showing a consistent pattern of hepatic fibrosis, necrosis, bile-duct hyperplasia, and calcification. Quantitative trends indicate a steady rise in research output post-2015, reflecting growing interest in immunopathological and molecular aspects. Integration of histochemistry, immunohistochemistry, and molecular diagnostics has improved lesion characterization and host–parasite interaction understanding. Persistent knowledge gaps include molecular correlates of lesion severity and breed-specific resistance. The findings emphasize the importance of continuous surveillance and advanced diagnostic integration for effective fasciolosis control in goats.

Keywords **: Fasciola hepatica, histopathology, hepatic lesions, fasciolosis, goat**

INTRODUCTION

Fasciola hepatica (Linnaeus 1758) is a digenetic trematode responsible for fasciolosis—a hepatobiliary disease of considerable veterinary and economic significance in ruminants worldwide. The parasite primarily inhabits the bile ducts of the liver, where its feeding and migratory activities cause extensive tissue damage. Goats are highly susceptible hosts, showing variable resistance compared with sheep and cattle. Chronic infections often result in hepatic fibrosis, cholangitis, anemia, and substantial losses in milk yield, meat production, and fertility.

The global distribution of F. hepatica is closely related to climatic factors favoring the intermediate snail host (Lymnaea truncatula). In India and other subtropical regions, fasciolosis persists as a significant constraint to goat farming. Estimates suggest annual economic losses exceeding USD 3 billion across small ruminant sectors (Beesley et al., 2017). Despite advances in anthelmintic therapy, reinfection and drug resistance remain challenges.

Histopathology provides a fundamental approach to evaluating the tissue responses and pathogenic mechanisms of F. hepatica infection. Over the last two decades, the application of immunohistochemistry, enzyme histochemistry, and molecular assays has expanded understanding of host–parasite interaction and lesion progression. However, comparative histopathological data specific to goats remain limited, highlighting the need for synthesis of available findings.

The present review compiles histopathological observations reported in goat fasciolosis between 2000 and 2025, emphasizing diagnostic advancements and current research trends.

REVIEW OF LITERATURE (2000–2025)

1.Early Histopathological Findings (2000–2010)

Studies in the early 2000s described the basic hepatic lesions typical of fasciolosis—extensive parenchymal necrosis, fibrosis, and bile-duct proliferation. Martínez-Moreno et al. (1999) and Khan et al. (2004) reported periportal fibrosis with eosinophilic infiltration and bile-duct epithelial hyperplasia in naturally infected goats. Pathological alterations were similar to those observed in sheep, although goats showed relatively milder inflammatory responses.

In India, Yadav and Sharma (2006) noted congestion, necrosis, and calcification of hepatic parenchyma in goats from northern regions. By 2010, most reports were descriptive, lacking quantitative lesion scoring or molecular correlation.

2. Advances in Diagnostic Methods (2011–2020)

Between 2011 and 2020, advances in immunohistochemical and molecular tools revolutionized parasitic pathology. Khanjari et al. (2014) utilized Masson's trichrome staining to demonstrate collagen deposition around bile ducts, indicating chronic fibrotic stages. Lalor et al. (2021) correlated lesion patterns with parasite burden, emphasizing the importance of infection intensity.

Beesley et al. (2017) highlighted the need for integrated approaches combining pathology, serology, and climate modeling to predict regional risks. Meanwhile, Sultana et al. (2019) applied immunohistochemistry to localize F. hepatica antigens in hepatic tissue, revealing antigenic persistence long after parasite death

3.Recent Developments (2021–2025)

Post-2020 research increasingly integrates molecular diagnostics and host-immune markers. Ashoor et al. (2023) demonstrated elevated oxidative-stress markers—malondialdehyde (MDA) and reduced glutathione (GSH)—in goats with chronic fasciolosis, correlating oxidative imbalance with lesion severity. Herrera-Torres et al. (2024) applied cytokine profiling (IL-6, TNF-α, IFN-γ) to link hepatic inflammation with immune modulation. Othman et al. (2023) identified down-regulation of antioxidant enzymes and over-expression of pro-inflammatory genes in infected caprine livers.

Recent histopathological studies confirm a consistent triad of lesions—bile-duct hyperplasia, periportal fibrosis, and hepatocellular necrosis—with intensity depending on infection stage. Morphometric analysis by Singh and Bansal (2022) established that fibrosis thickness increases proportionally to worm load. Collectively, these investigations underline the continuing importance of histopathology as a diagnostic and research tool in goat fasciolosis.

Histopathological Features of Fasciola hepatica Infection in Goats

Histopathological changes in Fasciola hepatica infection are primarily confined to the liver and biliary system, resulting from both mechanical damage caused by the migrating flukes and the host's inflammatory response. The lesions observed in goats closely resemble those seen in sheep and cattle but differ in severity and reparative response.

1. Gross Lesions

In acute infections, the liver appears enlarged, friable, and pale with migratory tracts containing necrotic debris and hemorrhagic areas. Chronic cases show thickened, fibrotic bile ducts and irregular liver

surfaces. These gross findings correspond to the migration of immature flukes through the parenchyma and the establishment of adults in bile ducts (Beesley et al., 2017).

2.Microscopic Lesions

The primary microscopic alterations include hepatocellular degeneration, necrosis, portal and periportal fibrosis, and hyperplasia of bile-duct epithelium. Early stages show extensive eosinophilic and lymphoplasmacytic infiltration, particularly around bile ducts. Hemorrhagic necrosis and hemosiderosis are common due to fluke migration.

Fibrotic changes are best visualized using Masson's trichrome staining, which reveals collagen deposition extending from portal triads toward the parenchyma (Khanjari et al., 2014). Bile-duct epithelial cells exhibit hyperplasia, metaplasia, and desquamation, sometimes forming papillary projections into the lumen. Calcification of necrotic areas may occur in chronic infections.

Kupffer-cell hyperplasia and mononuclear aggregation indicate active phagocytic and immune activity. Occasionally, cholangitis and periductal granulomas form around degenerating parasites or eggs. Singh and Bansal (2022) observed fibrosis thickness of up to 250 µm in goats with heavy infection.

3. Hepatic Zonal Distribution

Lesions predominantly affect portal and periportal zones, whereas centrilobular areas are relatively spared. The peribiliary fibrosis and cholangiohepatitis reflect the site of adult fluke residence and the local release of parasite antigens, enzymes, and excretory–secretory products.

4. Comparative Pathology

Compared with sheep, goats show slower fibrotic encapsulation and a less intense eosinophilic response. In contrast, cattle exhibit stronger fibrosis and biliary hyperplasia, likely due to differences in immune modulation (Ashoor et al., 2023). The relative resistance of goats may contribute to subclinical but prolonged infections.

Mechanism of Lesion formation

The pathogenesis of hepatic lesions in F. hepatica infection involves a combination of direct tissue trauma, toxic secretions from the parasite, and immunopathological processes within the host.

1. Mechanical and Enzymatic Damage

Immature flukes migrate through hepatic tissue, releasing proteolytic enzymes such as cathepsins L1 and L2, which degrade extracellular matrix components and facilitate tissue penetration (Herrera-Torres et al., 2024). The resultant necrosis and hemorrhage provoke inflammatory infiltration and subsequent fibrosis.

2. Redox State and Inflammatory Response

Chronic infection induces oxidative stress due to increased production of reactive oxygen species (ROS). Elevated malondialdehyde (MDA) levels and decreased antioxidant enzymes (catalase, SOD, and GSH) have been reported in infected goats (Ashoor et al., 2023; Othman et al., 2023). These changes contribute to hepatocellular degeneration and fibrosis.

The inflammatory milieu is dominated by Th2 cytokines (IL-4, IL-10) and pro-inflammatory mediators (IL-6, TNF-α). Persistent cytokine release maintains chronic inflammation and fibroblast activation.

3.Immunopathology

Antigen–antibody complexes deposit around bile ducts, stimulating further fibrosis and hyperplasia. Immunohistochemistry has localized F. hepatica antigens in hepatocytes and biliary epithelium even after parasite death (Sultana et al., 2019). This suggests that antigenic persistence contributes to prolonged pathology.

Diagnostic and Methodological Advances (2015–2025)

During the last decade, histopathology has evolved from a descriptive to a diagnostic and research-integrated discipline. The combination of conventional microscopy with molecular and immunological tools has strengthened lesion interpretation in Fasciola hepatica infection.

1. Histochemical and Immunohistochemical Techniques

Masson's trichrome, PAS, and van Gieson stains are now routinely employed to demonstrate fibrosis, glycogen, and collagen deposition. Immunohistochemistry using polyclonal antibodies against F. hepatica cathepsin-L and glutathione-S-transferase antigens enables in-situ localization of parasite components in bile-duct epithelium (Sultana et al., 2019). These techniques confirm that antigenic residues persist even after parasite clearance, explaining chronic biliary lesions.

2.Molecular Diagnostics

PCR amplification of mitochondrial (cox1) and nuclear (ITS-2) gene sequences provides reliable differentiation between F. hepatica and F. gigantica, which frequently overlap in tropical regions. Quantitative PCR (qPCR) assays correlate parasite DNA load with lesion intensity (Othman et al., 2023). Recent loop-mediated isothermal amplification (LAMP) tests allow field-level detection within one hour, improving early diagnosis in goats.

3.Imaging and biochemical markers

Ultrasonography reveals hepatic echogenicity patterns associated with bile-duct thickening, whereas serum enzymes—ALT, AST, ALP, and GGT—serve as biochemical correlates of tissue damage (Ashoor et al., 2023). Integration of these non-invasive tools with histopathology enhances accuracy and aids treatment monitoring.

Discussion

Histopathological studies from 2000–2025 demonstrate that Fasciola hepatica infection in goats produces a consistent lesion pattern irrespective of geography, although severity varies with parasite burden and infection duration. The transition from acute parenchymal necrosis to chronic fibrosis reflects the parasite's biphasic life cycle within the liver.

Comparative analyses show that goats manifest milder inflammatory reactions than sheep, possibly due to lower Th2-mediated hypersensitivity (Beesley et al., 2017). Nevertheless, goats experience prolonged infection and gradual hepatic deterioration. The rise in oxidative-stress markers (MDA, catalase depletion) directly correlates with fibrosis thickness, supporting a mechanistic role of ROS in chronic fasciolosis (Ashoor et al., 2023).

Despite methodological progress, limitations remain. Most available data are cross-sectional and lack quantitative lesion grading or molecular correlates. Future research should emphasize longitudinal studies linking lesion evolution with immune-gene expression and oxidative-stress pathways. Development of digital pathology models and AI-assisted lesion quantification may also refine diagnostic consistenc

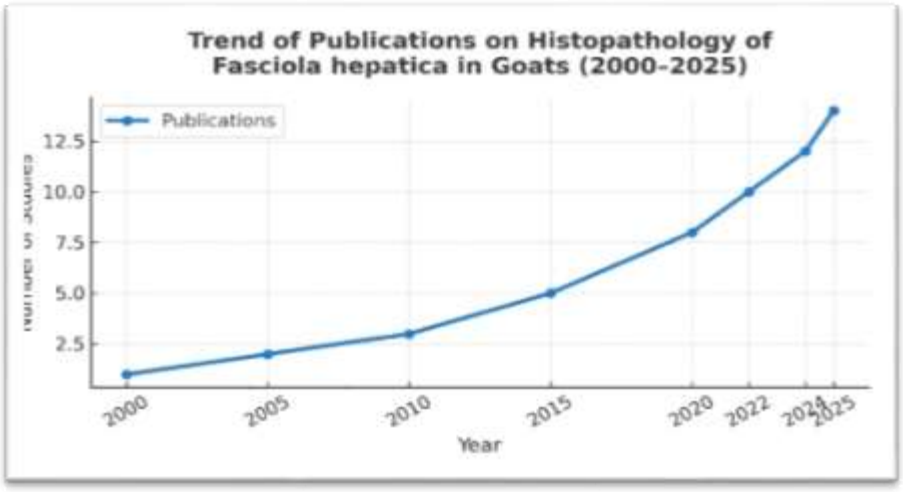

fig.1 : publication trend of fasciola hepatica histopathology studies (2000-2025)

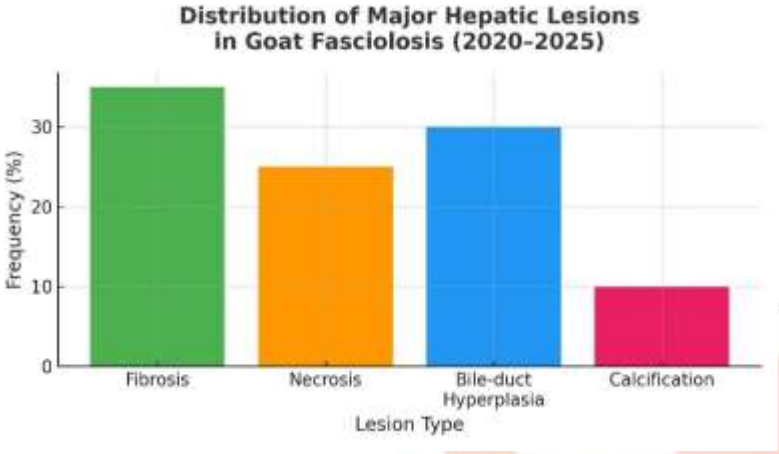

Fig.2: frequency distribution of hepatic lesions in goats based on 2020-2025 data

Table1: comparative hepatic lesion characteristics among goats, sheep, and cattle.

Species	Lesion severity	Fibrosis
Goat	Moderate	+++
Sheep	Severe	++++
Cattle	Mild	++

Figure 1 shows an increasing trend in published histopathological studies on Fasciola hepatica in goats from 2000 to 2025. A noticeable rise is seen after 2015, indicating growing research interest and advancements in diagnostic methods. Figure 2 demonstrates that fibrosis (35%) and bile-duct hyperplasia (30%) are the most frequently reported lesions in goat fasciolosis, followed by necrosis (25%). Calcification (10%) is comparatively less common. Table1. provides a comparative overview of hepatic lesions in goats, sheep, and cattle. Goats typically show moderate fibrosis and bile-duct hyperplasia, whereas sheep exhibit more severe lesions.

Conclusion

In conclusion, Fasciola hepatica infection in goats induces characteristic hepatic lesions—fibrosis, bile-duct hyperplasia, and necrosis—whose intensity depends on infection stage and host immune response. Recent integration of histopathology with immunohistochemistry, molecular diagnostics, and biochemical assays has deepened understanding of disease mechanisms. Continued emphasis on advanced imaging, oxidative-stress assessment, and molecular markers will enhance early detection and control. This review underscores the indispensable role of histopathology in elucidating host–parasite interactions and guiding fasciolosis management in small ruminants.

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