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Pathomorphological and biochemical studies of hepatic lesions in slaughtered pigs

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Abstract

The present study was conducted to investigate gross, histopathological and biochemical alterations associated with hepatic lesions in slaughtered pigs. A total of 200 non-descript pigs were examined at an organized abattoir in Mumbai. Blood and tissue samples were collected for biochemical and histopathological analyses. Out of 200 pigs, 111 (55.5%) showed lesions in various organs, with liver involvement in 20 cases (10%). Among these, enlargement and congestion (5.5%) were most common, followed by hydatid cysts (1.5%), milk spots (1.5%), diffuse changes (2%) and focal microscopic alterations (12.5%). Histopathology revealed hepatocellular degeneration, necrosis, fibrosis and inflammatory infiltration. Serum ALT and ALP levels were significantly elevated, while albumin levels were reduced. The findings indicate that hepatic lesions are common and correlate well with biochemical alterations. The predominance of focal lesions suggests that many hepatic affections remain subclinical.

Keywords: ALT, ALP, hepatic lesions, histopathology, hydatidosis, pigs

1. Introduction

Pig production is a cornerstone of global livestock growth, providing vital food security and livelihood support, particularly for rural and tribal communities in North-Eastern India (Roy *et al.*, 2025) ^[1]. However, productivity remains limited by poor management, high parasitic burdens and infectious diseases that degrade growth and carcass quality. The liver is exceptionally vulnerable to pathological insults as the primary site for metabolism, detoxification and immune regulation. Various degenerative lesions like cloudy swelling and fatty changes were reported in liver (Deepak Kumar *et al.*, 2022) ^[2, 3]. These lesions represent significant economic losses (Purohit *et al.*, 2021) ^[4] and pose substantial zoonotic risks to public health. While gross pathology and histopathology identify physical damage, correlating these findings with serum biochemical markers allows for a deeper understanding of subclinical liver dysfunction (Kumar *et al.*, 2023) ^[5]. This study investigates the prevalence and nature of hepatic lesions in slaughtered pigs. We aim to enhance diagnostic accuracy and underscore the impact of liver health on porcine welfare and consumer safety by analyzing pathological lesion and biochemical alterations.

2. Materials and methods

2.1 Study Area and Animals

The study was conducted on 200 non-descript pigs slaughtered at an organized abattoir in Mumbai during 2009. Animals were randomly selected and originated from different regions of Maharashtra, Goa and Gujarat.

2.2 Sample Collection: Blood samples were collected before slaughter in plain vials for serum separation. Liver collected from the animals were studied properly and samples were collected and fixed in 10% neutral buffered formalin.

2.3 Laboratory Analysis

2.3.1 Serum Biochemistry: The serum was separated by centrifugation for 10 min at 1500 rpm and parameters as mentioned below were estimated by using serum biochemistry semi-autoanalyser (Erba- Chem.7), using commercial reagent kits for Total protein by

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Biuret method (Tietz, 1986) ^[6]. Albumin, by BCG Dye method (Doumas *et al.*, 1972) ^[7]. Alanine aminotransferase (ALT) as per IFCC method (International Federation of Clinical Chemistry) suggested by Tietz, (1995) ^[8], Alkaline phosphatase (ALP) (Tietz *et al.*, 1983) ^[9], Total and direct bilirubin. (Pearlman and Lee, 1974) ^[10].

2.3.2 Histopathology

Tissues were processed and stained using hematoxylin and eosin staining and examined microscopically. (Luna, 1968) ^[11].

2.4 Statistical Analysis

Data were analyzed using completely randomized design (CRD) Snedecor and Cochran (1994) ^[12].

3. Results and discussion

Out of 200 pigs examined, 111 (55.5%) showed lesions in various organs, of which 20 (10%) animals exhibited hepatic affections (Fig. No.1, 4,5,9,10 & 13). Among these, 5.5% showed enlargement and congestion, 1.5% hydatid cysts, 0.5% *Cysticercus cellulosae*, 0.5% *Cysticercus tenuicollis*, 1.5% milk spots, 2% diffuse changes, and 12.5% focal microscopic alterations. The higher proportion of focal lesions indicates that many hepatic abnormalities remain subclinical and are detectable only on microscopic examination.

The incidence of hydatidosis (Fig. No.5 & 9) in the present study was comparable to earlier findings. Varma (1990) ^[13] reported 1.3% prevalence, while Vijaysmitha *et al.* (1993) ^[14] observed 3.02% incidence, with the liver as the primary site. Similar observations were made by Gatne (2001) ^[15] and Singh *et al.* (1988) ^[16]. The higher occurrence of hydatid cysts in the liver may be attributed to its role as the primary filter for migrating oncospheres, where they become lodged and develop into cysts (Gaurat, 2002) ^[17]. All cysts observed in the present study were sterile, unlike earlier reports indicating higher fertility rates (Gatne, 2001; Pednekar *et al.*, 2009) ^[15, 18]. Although the reason for sterility could not be established, sterile cysts do not contribute to further transmission.



Fig 1: Severe degree of fatty degeneration

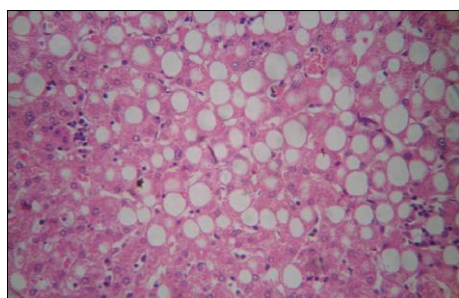


Fig 2: Diffuse fatty degeneration in liver. H& E stain, 400X

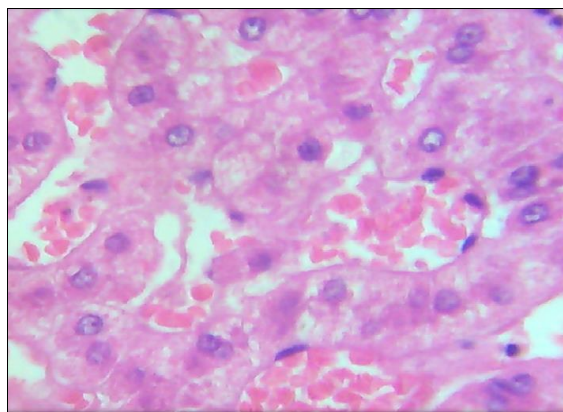


Fig 3: Congestion in liver. H& E stain, 1000X

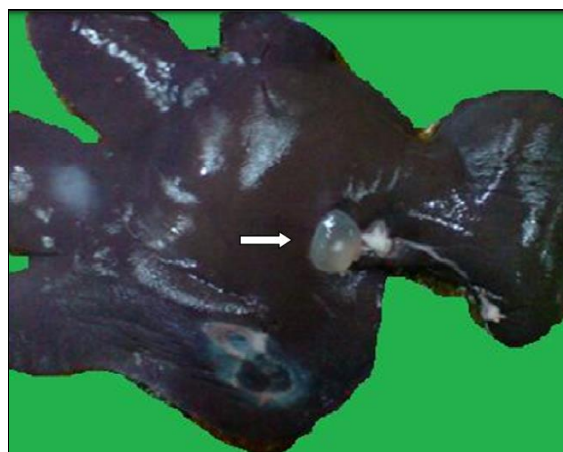


Fig 4: *Cysticercus tenuicollis* in dorsal surface of highly congested liver

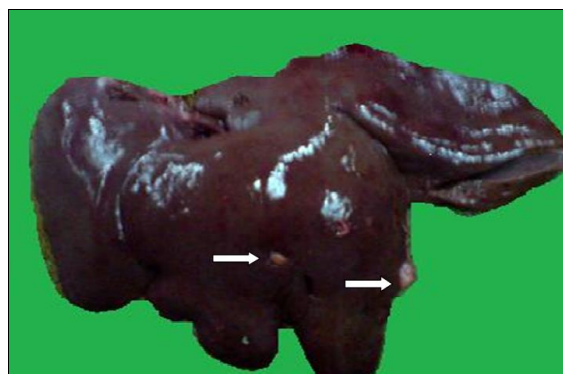


Fig 5: Presence of 2 (two) visible hydatid cyst (white arrow)

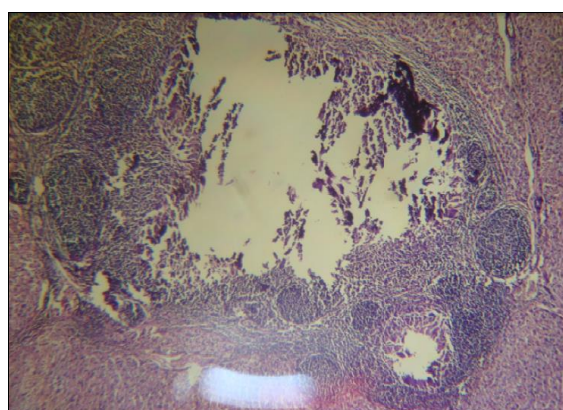


Fig 6: Section showing a hydatid cyst surrounded by fibrous tissue, inflammatory cells and germinal follicles. H& E stain, 400X

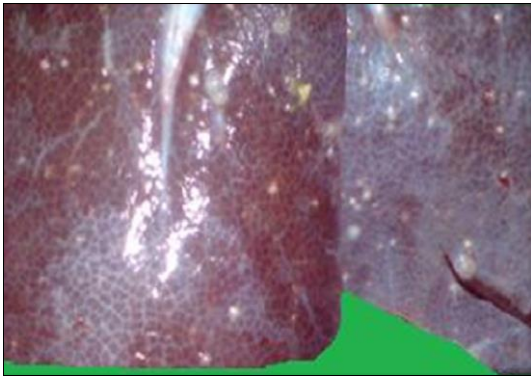


Fig 7: Numerous *Cysticercous cellulosae* scattered across the liver parenchyma

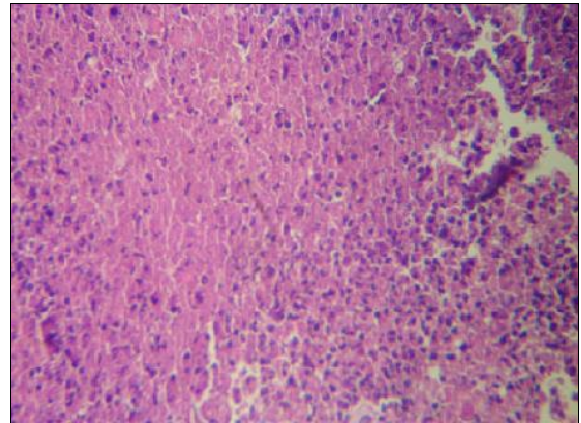


Fig 11: Necrosis with infiltration of inflammatory cells. H and E stain, 100X

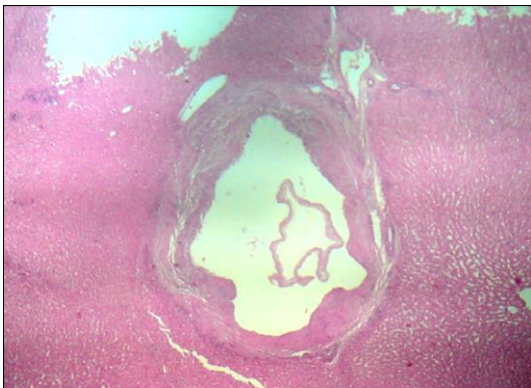


Fig 8: A Cystic structure with a luminal content in the liver parenchyma forming a thick fibrous tissue surrounding it. H and E stain, 40X

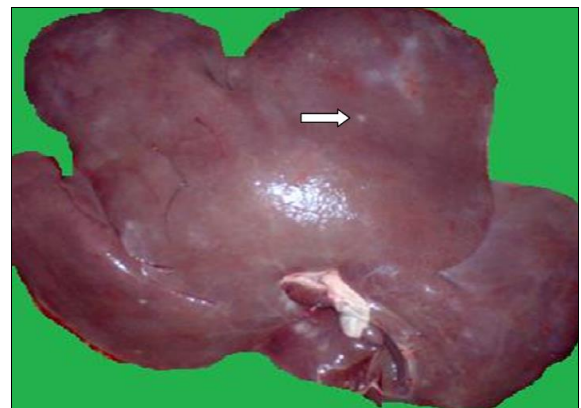


Fig 12: Milk spot (white arrow) in liver parenchyma



Fig 9: Hydatid cyst in right and left lateral lobe and focal necrosis in the medial lobe

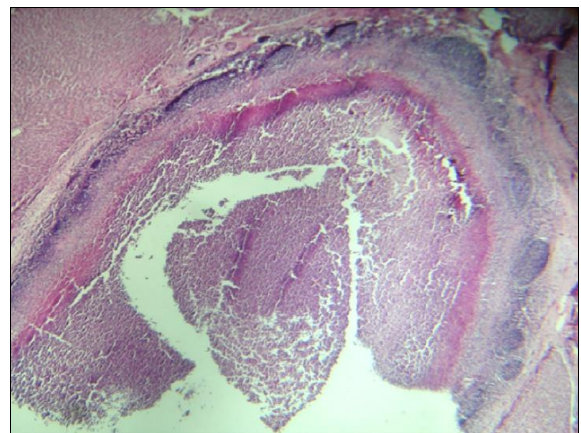


Fig 13: Cross section of a milk spot. H and E stain, 40X

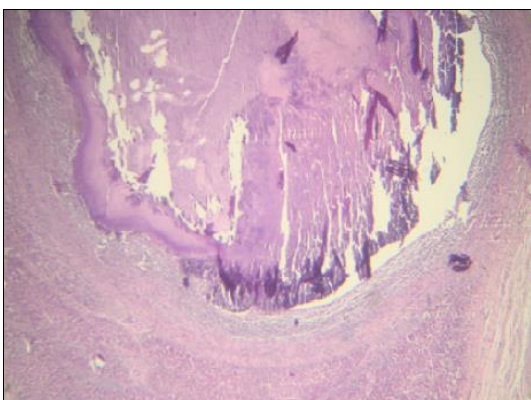


Fig 10: Cross section of a hydatid cyst. H and E stain, 40X

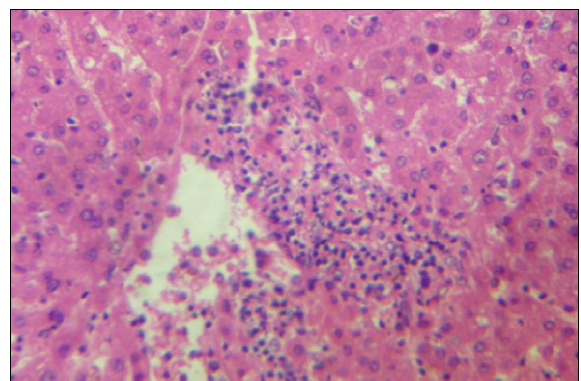


Fig 14: Focal necrosis with inflammatory cell infiltration. H and E stain, 400X



Fig 15: Diffuse fibrosis of liver

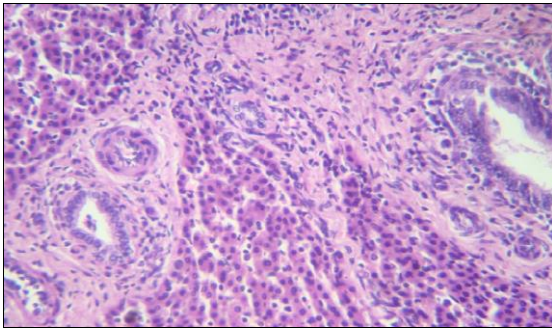


Fig 16: Proliferation of fibrous connective tissue with bile duct proliferation. H and E stain, 400X

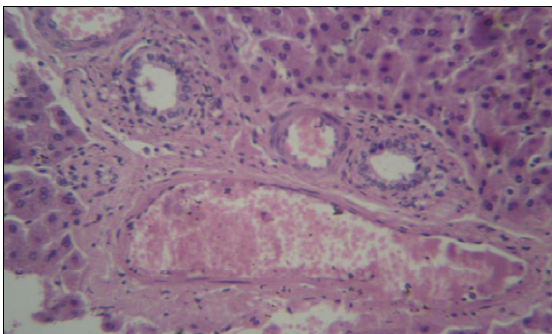


Fig 17: Congestion and dilatation of blood vessels in portal triad. H and E stain, 400X

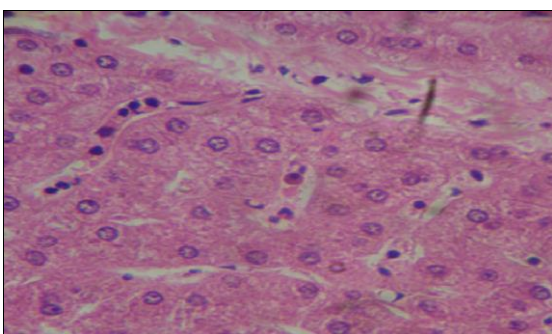


Fig 18: Focal hepatocellular swelling. H and E stain, 400X

Parasitic infections, including milk spots (Fig. No. 10), were recorded in 3.5% of animals, which is comparatively lower than previous reports, possibly due to environmental or management differences. Milk spots are associated with migration of *Ascaris suum* larvae, leading to hepatocellular damage followed by fibrous tissue proliferation (Soulsby, 1982) ^[19]. Histopathologically, these lesions showed marked fibrosis, eosinophilic infiltration and bile duct proliferation and consistent with earlier reports (Corwin and Stewart, 1992) ^[20].

Based on gross and histopathological findings, hepatic lesions were classified into six groups (A-F) to facilitate clinicopathological correlation.

Group A (congestion and enlargement): Showed congestion, dilation of portal triads, increased sinusoidal spaces, hepatocellular degeneration, necrosis and mononuclear cell (MNC) infiltration. *Salmonella* spp. were isolated from two animals in this group. These findings are in agreement with Wilcock and Schwartz (1992) ^[21], who described focal necrosis and inflammatory infiltration in salmonellosis. Biochemically, significantly elevated ALT levels were observed, indicating hepatocellular damage due to increased membrane permeability. ALP levels were comparatively low, suggesting minimal biliary involvement. Total protein and albumin levels were comparable to normal animals indicating acute hepatic injury with mild functional impairment.

Group B (parasitic cysts)

Showed necrotic masses surrounded by inflammatory cells, fibrosis and hemorrhages. In some cases, fibrous tissue proliferation resulted in widening of septa. Both ALT and ALP levels were elevated, indicating hepatocellular damage along with biliary involvement. Reduction in total protein and albumin levels suggested moderate hepatic dysfunction. Hemoglobin and PCV values were slightly reduced but remained within normal limits (Coles, 1980 ^[22] and Benjamin, 2005) ^[23]. Degenerated hydatid cysts lacked clear structural demarcation, supporting earlier findings that such lesions may resemble caseous or mineralized masses (Margaret and Hayes, 2006) ^[24].

Group C (milk spots)

Exhibited excessive fibrous tissue proliferation, particularly in portal areas, along with bile duct hyperplasia. These findings are consistent with repeated larval migration of *Ascaris suum* (Corwin and Stewart, 1992) ^[20]. Serum ALP levels were elevated, indicating biliary involvement, while reduced albumin levels suggested chronic hepatic damage although total protein remained comparable to normal animals.

Group D (diffuse lesions)

Showed generalized hepatocellular degeneration and inflammatory infiltration. This group exhibited reduced total protein and albumin levels, indicating impaired liver function.

Group E (focal microscopic lesions): Showed focal hepatic degeneration, lymphoid proliferation in portal tracts, and centrilobular hemorrhages. However, biochemical parameters remained within normal limits, suggesting that focal lesions do not significantly affect overall hepatic function.

Group F (normal)

Animals showed no gross or microscopic abnormalities

Serum bilirubin levels, both total and direct, did not differ significantly among groups, indicating that hepatic lesions were not severe enough to impair bilirubin metabolism. This finding is consistent with earlier reports that bilirubin levels remain only mildly elevated even in severe hepatic diseases in domestic animals (Coles, 1980) ^[22]. Furthermore, bilirubin is considered a less specific indicator of hepatic dysfunction compared to enzymes such as ALP (David, 2008) ^[25].

Table 1: Mean of serum biochemical values according to lesions found on liver

Group	A	B	C	D	E	F
SGPT (IU/L)	77.91± 14.472 ^a	67.45±23.322 ^{ab}	44.80±5.233 ^{bcd}	43.65±2.365 ^{cd}	51.50±2.834 ^{bc}	43.450±0.760 ^d
ALP (IU/L)	79.81±13.242 ^c	179.50±31.003 ^a	172.19±13.580 ^a	87.58±22.874 ^{bc}	107.15±9.342 ^b	81.52±2.851 ^c
TP (gm/dL)	6.60±0.323 ^a	5.30±0.625 ^b	6.19±0.061 ^{ab}	5.59±0.524 ^b	5.81±0.310 ^b	6.83±0.077 ^a
Alb (gm/dL)	2.48± 0.179 ^{ab}	1.94± 0.288 ^{bc}	2.05± 0.043 ^{bc}	1.77± 0.397 ^c	2.11±0.140 ^{bc}	2.50± 0.047 ^a
Glob (gm/dL)	4.13± 0.276 ^{ab}	3.36±0.555 ^b	4.14± 0.092 ^{ab}	3.82± 0.170 ^{ab}	3.70± 0.239 ^b	4.33± 0.066 ^a
TB (mg/dL)	0.39±0.134	0.40±0.090	0.35±0.033	0.26± 0.026	0.35± 0.034	0.33± 0.013
DB (mg/dL)	0.13± 0.017	0.07±0.013	0.11±0.042	0.15±0.026	0.11±0.021	0.11±0.007

Values (n₁=11, n₂=4, n₃=3, n₄=4, n₅=25 and n₆=153; mean±SE) in the same row bearing at least one common superscript do not vary significantly (P ≤ 0.05).

Table 2: Analysis of variance

TEST	Source of variation	S.S.	d.f.	M.S.	F cal.
SGPT	Treatment (between group)	52420	5	10484	21.80843**
	Error (within group)	93263	194	480.7	
	Total	145683	199		
ALP	Treatment (between group)	71088.37	5	14217.67	9.819106**
	Error (within group)	280904.3	194	1447.96	
	Total	351992.6	199		
TP	Treatment (between column)	35.2747	5	7.054941	6.296478**
	Error (within group)	217.3689	194	1.120458	
	Total	252.6436	199		
Alb	Treatment (between column)	6.554384	5	1.310877	3.59452**
	Error (within group)	70.74938	194	0.364688	
	Total	77.30377	199		
Glob	Treatment (between column)	12.33042	5	2.466083	3.25035*
	Error (within group)	147.1903	194	0.758713	
	Total	159.5207	199		
TB	Treatment (between group)	0.07658	5	0.015316	0.458992 ^{NS}
	Error (within group)	6.473551	194	0.033369	
	Total	6.550131	199		
DB	Treatment (between group)	0.014129	5	0.002826	0.345214 ^{NS}
	Error (within group)	1.588013	194	0.008186	
	Total	1.602142	199		

NS: Non significant at P ≤ 0.05 ** Highly Significant at P ≤ 0.01

Overall, the study demonstrates a clear correlation between histopathological findings and serum biochemical alterations (Table 1 & 2). Elevated ALT levels were associated with hepatocellular damage, while increased ALP levels indicated biliary involvement, particularly in parasitic conditions. Reduction in albumin levels reflected compromised liver function in chronic and diffuse lesions. These findings highlight the importance of integrating pathological and biochemical assessments for accurate diagnosis of hepatic lesions in pigs.

4. Conclusion

Hepatic lesions in slaughtered pigs were mainly associated with parasitic infections and degenerative changes.

Significant alterations in serum ALT, ALP and albumin levels correlated with pathological findings. Histopathological examination remains essential for definitive diagnosis. Control of parasitic infections is necessary to reduce hepatic pathology and improve animal health.

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