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Research Article

PATHOLOGY OF HEPATIC DISORDERS IN BUFFALOES

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ABSTRACT

Present study was undertaken to elucidate the pathology of hepatic disorders in thirty buffaloes/buffalo calves brought for post-mortem to the department. The gross lesions in liver revealed maximum mortality in female buffalo calves of age group up to one month. Gross pathological changes in liver were congestion and haemorrhages, necrotic foci on liver surface, induration and hepatomegaly. Histopathologically, liver revealed congestion, haemorrhages, hydropic degeneration, fatty changes and necrosis accompanied with infiltration of leucocytes, predominantly neutrophils, proliferation of fibroblasts, bile duct hyperplasia and atrophy of hepatocytes. Pathological changes were also observed in lungs, heart, intestines, spleen, kidneys and lymph nodes. It is concluded that hepatopathology in buffaloes/buffalo calves is invariably associated with pathological changes in other organs.

Key words: Pathology, hepatic disorders, buffalo calves

The liver gets first exposure to inimical agents because of the highly specialized functions of the hepatic parenchymal cells and dual blood circulation. Diseased liver adversely affects the health and growth of animals and also causes economic losses due to condemnation of such livers at the time of meat inspection (Purushotaman and Rajan, 1985). Buffalo calves suffer from higher mortality than cow calves (Tomar and Tripathi, 1991) mainly due to managemental problems and diseases like hepatitis, pneumonia and scours. A few studies had been conducted in past regarding incidence of hepatic diseases in buffaloes (Nair *et al.*, 2006). Therefore, proposed work was aimed to study pathological changes in hepatic disorders in buffaloes/buffalo calves.

MATERIALS AND METHODS

Proposed study was conducted on 30 carcasses of buffaloes (7) and buffalo calves (23) brought for post mortem to the Department of Veterinary Pathology, CCS Haryana Agricultural University, Hisar. All the thirty carcasses selected were showing gross lesions in the liver. Thorough post mortem examination was conducted as early as possible and the gross lesions were recorded. Small pieces of liver, heart, lungs, spleen, mesenteric lymph nodes, intestines, kidneys were collected in 10 per cent neutral buffered formalin for histopathological studies. The fixed tissues were processed for paraffin embedding technique. The paraffin sections were cut at the thickness of 4-5 μ and stained with routine haematoxylin and eosin (Luna, 1968).

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RESULTS AND DISCUSSION

Age and sex wise mortality among buffaloes/ buffalo calves is depicted in Table 1. Age wise distribution of mortality in buffalo/buffalo calves indicated that maximum mortality was in age groups of day one to one month followed by >1 month to 3 months, >3 years, >6-12 months, >3-6 months and least in >1-3 years. The results of buffalo calf mortality were similar to Umoh (1982), Kinjavdekar *et al.* (1994), and Khan and Khan (1996). Sex wise distribution of mortality revealed higher female mortality than males. Regarding month and season wise distribution of mortality, it was higher in August followed by November, September and October in study of six months i.e. July to December, 2005 (Table 2).

Gross pathology : Gross pathological changes noticed on post mortem examination are depicted

Table 1
Age and sexwise distribution of mortality among buffalo/buffalo calves

Age group	No. of cases	Sex	
		Male	Female
0 day - 1 month	8/30 (26.6)*	3/30 (10)	5/30 (16.8)
>1 - 3 months	7/30 (23.3)	3/30 (10)	4/30 (13.3)
>3 - 6 months	2/30 (6.6)	1/30 (3.3)	1/30 (3.3)
>6 - 12 months	5/30 (16.8)	1/30 (3.3)	4/30 (13.3)
>1 - 3 yrs.	1/30 (3.3)	-	1/30 (3.3)
>3 yrs.	7/30 (23.3)	-	7/30 (23.3)
Total	30	8 (26.6)	22 (73.6)

* Figures in bracket indicate percentage of mortality

in Table 3. Congestion of liver was found (Fig 1) as the most prominent change and other changes included petechial to ecchymotic haemorrhages, adhesions, necrotic foci, paleness and hepatomegaly. Similar lesions had been reported earlier by Sengupta *et al*. (1968), Motto *et al.* (1989), Sridhar *et al.* (1995), Yadav *et al.* (1999), Sinha *et al.* (2001), Carlson *et al.* (2002) and Khatoon *et al*. (2003)

Histopathological findings: In the liver, microscopic lesions were congestion of blood vessels, dilation of sinusoids leading to atrophy of hepatocytes, haemorrhages, hydropic degeneration fatty changes (Fig 2) and centrilobular necrosis. There was mild leucocytic infiltration in portal triad area and initiation of lymphoid follicle formation in parenchyma (Fig 3), fibrinous perihepatitis leading to thickening of capsule (Fig 4) and bile duct hyperplasia. In one case, there was presence of clostridial like rod shaped organisms in sinusoids leading to necrosis of hepatocytes. Similar findings had been earlier reported by Barri *et al.* (1981), Motto *et al.* (1989), Singh *et al.* (2000), Sinha *et al.* (2001), Khatoon *et al.* (2003) and Laven *et al.* (2004).

Table 2
Month wise distribution of mortality among carcasses of buffaloes/buffalo calves

Months	Mortality
July	2
August	10
September	4
October	4
November	9
December	1

In lungs, lesions observed were congestion, oedema in alveoli and interstitial space and fibrinous exudate in few cases leading to thickening of pleura. Some cases were showing congestion in submucosa and marked mononuclear cells infiltration predominantly lymphocytes in mucosa of bronchiole. There was peribronchiolar lymphoid cell accumulation and necrosis of parenchymal tissue and replacement by lymphoid aggregations. Focal area of necrosis along with leucocytic infiltration leading to replacement of alveoli of lung was also seen. There was thickening of interlobular septa due to fibrinous exudate. Similar infiltrative and proliferative changes in lungs had been reported by Jubb *et al.* (1993), Singh *et al.* (1996) and Libby *et al.* (1997).

The heart revealed oedema, congestion and haemorrhages in myocardium. Some cases were showing areas of necrosis and severe leucocytic infiltration predominantly by mononuclear cells. Sarcocystosis was observed in few cases. In few cases, fibrinous pericarditis with fibrinous exudate extended into myocardium along with necrosis of muscle fibres were evidenced. These changes were also observed by Singh *et al.* (1996) and Khan and Khan (1997).

In intestine, there was severe congestion in mucosa, submucosa and serosa along with mononuclear cells infiltration. Other changes were goblet cell hyperplasia, formation of ulcer leading to break in continuity of mucosa along with desqumation of mucosal epithelium and leucocytic infiltration in mucosa and depletion

Fig 1. Congested and swollen liver with round edges in buffalo calf

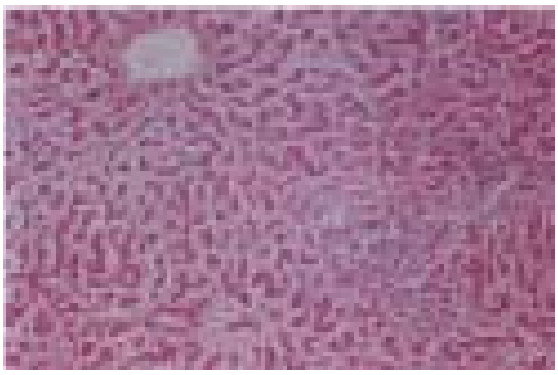

Fig 3. Necrosis of hepatocytes and initiation of lymphoid follicle formation in parenchyma of liver in buffalo. (H. & E. x 33)

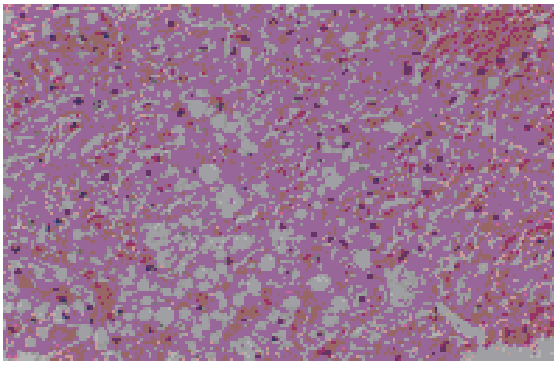

Fig 2. Fatty changes giving signet ring appearance of hepatocytes in liver of buffalo calf. (H. & E. x 33)

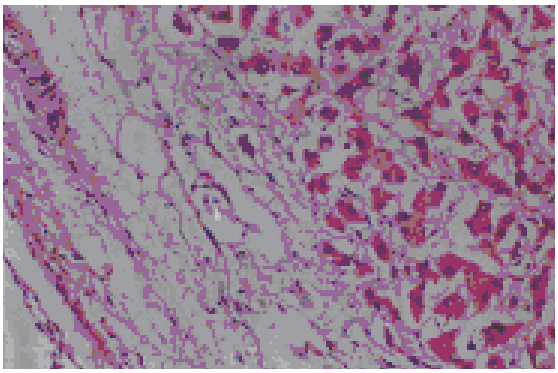

Fig 4. Fibrinous perihepatitis and thickening of capsule of liver of buffalo calf. (H. & E. x 33)

Table 3
Gross pathological changes observed at post mortem examination of buffaloes/buffalo calve

Gross changes	Liver	Lung	Heart	Spleen	Kidney	Lymph node	Intestine
Congestion	16	15	4	2	5	-	11
Hemorrhages	3	2	3	1	1	-	4
Paleness	2	-	-	-	-	-	-
Necrotic foci	3	-	1	-	-	-	-
Adhesions	2	2	5	2	-	-	-
Firmness and indurations	2	-	-	-	-	2	-
Nodular formation	-	1	-	-	-	-	-
Hydropericardium	-	-	1	-	-	-	-
Enlargement	2	-	-	1	-	-	-

of lymphocytes in Peyer's patches of large intestine. Similar findings had been reported by Libby *et al.* (1997) and Tsolis *et al.* (1999).

In kidneys, there was congestion, coagulative necrosis and mild leucocytic infiltration in interstitial tissue. Areas of focal necrosis and lymphoid aggregates were seen. Atrophy of glomerular tuft and fibroblast proliferation in the interstitial tissue and also around blood vessel were recorded. Similar vascular and degenerative changes had been reported earlier by Khan and Khan (1997). Spleen was showing depletion of lymphoctyes in white pulp and haemosiderosis as reported earlier (Pearson *et al.*, 1978, Jubb *et al.*, 1993).

Mesenteric lymph nodes revealed oedema and congestion in capsule. There were areas of medullary congestion in the lymph node. Depletion of lymphocytes in germinal centre of lymphoid follicle in cortex of lymph gland was

also evidenced. In some cases, there was necrosis and cystic formation along with fibroblast proliferation in cortex of lymph node. Severe fibroblast proliferation in the cortex of lymph node was also seen. Congestion in cortex and medulla and depletion of lymphocytes in germinal centre of lymphoid follicle in cortex of lymph gland had been evidenced earlier by Maity *et al.* (2000).

On the basis of the studies it is concluded that liver lesions contribute predominantly to mortality of buffaloes/buffalo calves along with lesions in other organs especially heart, intestine, spleen, kidneys and mesenteric lymph nodes. Gross and histopathological lesions observed will help in revealing pathogensis of hepatic diseases and in correlation of etiological agents in buffaloes/buffalo calves.

REFERENCES

Barri, M.E.S. and Adam, S.E.I. (1981). The toxicity of *Crotolaria saltiana* to calves. *J. Comp. Path.* **91**: 621-627.

Carlson, S.A., Stoffregan, W.C. and Bolin, S.R. (2002). Abomasitis associated with multiple antibiotic resistant *Salmonella enterica Serotype typhimurium* phagetype DT 104. *Vet. Microbiol.* **85**: 233-240.

Jubb, K.V., Kennedy, P.C. and Palmer, N. (1993). Pathology of Domestic Animals, Vol. II (4th edn.). Academic Press Inc., Sen Diego, California. pp. 277

Khan, A. and Khan, M.Z. (1996). Neonatal calf mortality in Pakistan I. Prevalence and factors influencing mortality in buffalo and cow neonates. *Buffalo J.* **12**: 219-229.

Khan, A. and Khan, M.Z. (1997). Gross and histopathological findings in buffalo and bovine diarrhoeic neonates. *Veterinarski Archiv.* **67**: 203-213.

Khatoon, N., Bilqes, F.M. and Mirza, S. (2003). Histopathological changes in the liver of buffaloes by digenic trematode *Paramphistomum cervi*. *J. Biol. Sci.* **6**: 486-489.

Kinjavdekar, P., Parai, T.P., Srivastava, B.B. and Nautiyal, L.P. (1994). Mortality pattern in calves. *Indian J. Anim. Sci.* **64**: 811-812.

Kundu, S.S., Mishra, A.K. and Pathak, P.S. (2004). Buffalo production under different climatic regions (1st edn.) International Book Distributing Co., Lucknow.

Laven, R.A., Livesey, C.T., Offer, N.W. and Fountain, D. (2004). Apparent subclinic hepatopathy due to excess copper intake in lactating Holstein cattle. *Vet. Rec.* **5**: 120-121.

Libby, S.J., Adams, L.G., Ficht, T.A., Allen, C., Howard, A., Whitford, A.A., Buchmeier, N.A., Bossie, S. and Guiney, G.D. (1997). *SPV* genes on the *Salmonella dublin* virulence plasmid are required for severe enteritis and systemic infection in natural host. *Infect. Imm.* **65**: 1786-1792.

Luna, L.G. (1968). Manual of Histologic Staining Methods of the Armed Foreces Institute of Pathology. (3rd edn.). McGraw Hill Book Co., New York.

Maity, S.B., Deb, P., Das, R. and Som, T.L. (2000). Pathology of lymph nodes in cattle. *Indian J. Vet. Path.* **24**: 32-34.

Motto, M.R., Bali, H.S. and Gupta, P.P. (1989). Pathological changes in the liver of buffaloes in fascioliasis and schistomiasis. *Indian J. Comp. Microbiol. Immunol. Infect. Dis.* **10**: 177-179.

Nair, M.G., Kumar, R. Lakkawar, A.W. and Varshney, K.C. (2006). A slaughter house and necropsy based study of lesions in bovines. *Indian Vet. J.* **83**: 490-493.

Pearson, G.R., Mcnulty, M.S. and Logan, E.F. (1978). Pathological changes in the small intestine of neonatal calves with enteric colibacillosis. *Vet. Path.* **15**: 92-101.

Purushothaman, A.V. and Rajan, A. (1985). Incidence and pathology of hepatic disorders in cattle. *Kerala J. Vet. Sci.* **16**: 85-94.

Sengupta, U. and Iyer, P.K.R. (1968). Studies on pathology of haemosiderosis in Indian buffaloes. *Indian Vet. J.* **45**: 818-822.

Singh, D.V., Sodh, S.P.S., Brar, A.P.S. and Grewal, J.J. (1996). Certain pathological profiles of endotoxic shock in buffalo calves. *Buffalo J.* **12**: 283-288.

Singh, R., Chandra, D., Rathore, B.S., Singh, K.P. and Mehrotra, M.L. (2000). Investigation of mortality in cattle and buffaloes with particular reference to hepatic schistosomiasis in cattle. *Indian J. Vet. Path.* **24**: 8-11.

Sinha, B., Ranjan. K.S., Pandey, T.N., Sinha, B.K., Roy, M.K. and Prasad, L.N. (2001). Aflatoxin induced hepatotoxicity in buffalo calves. *Indian J. Vet. Pathol.* **25**: 73.

Sridhar, R., Hemalatha, S. and Sundararaj. (1995). A case of aberrant location of *Toxocara vitulorum* in the gall bladder of a buffalo calf. *Cheiron* **24**: 43-45.

Tomar, S. and Tripathi, V.M. (1991). Quantification of mortality in Murrah buffalo female calves at different age. *Indian J. Dairy Sci.* **44**: 6-8.

Tsolis, R.M., Adams, L.G., Ficht, T.A. and Baumler, A.J. (1999). Contribution of *Salmonella typhimurium* virulence factors to diarrhoeal disease in calves. *Infect. Imm.* **67**: 4879-4885.

Umoh, J.U. (1982). Relative survival of calves in a university herd in Zaria, Nigeria. *Brt. Vet. J.* **138**: 507-514.

Verma, G.S., Sadana, D.K. and Busu, S. B. (1980). Studies on mortality in buffalo calves. *Indian J. Dairy Sci.* **33**: 87-90.

Yadav, S.C., Sharma, R.L., Kalicharan, A., Mehra, O.R., Dass, D.S. and Verma, A.R. (1999). Primary experimental infection of riverine buffaloes with *Fasciola gigantica*. *Vet. Parasitol.* **82**: 285-296.