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.

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.
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)

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

<table>
<thead>
<tr>
<th>Age group</th>
<th>No. of cases</th>
<th>Sex</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>Male</td>
<td>Female</td>
</tr>
<tr>
<td>0 day - 1 month</td>
<td>3/30 (10)</td>
<td>5/30 (16.8)</td>
</tr>
<tr>
<td>&gt;1 - 3 months</td>
<td>3/30 (10)</td>
<td>4/30 (13.3)</td>
</tr>
<tr>
<td>&gt;3 - 6 months</td>
<td>1/30 (3.3)</td>
<td>1/30 (3.3)</td>
</tr>
<tr>
<td>&gt;6 - 12 months</td>
<td>1/30 (3.3)</td>
<td>4/30 (13.3)</td>
</tr>
<tr>
<td>&gt;1 - 3 yrs.</td>
<td>-</td>
<td>1/30 (3.3)</td>
</tr>
<tr>
<td>&gt;3 yrs.</td>
<td>-</td>
<td>7/30 (23.3)</td>
</tr>
<tr>
<td>Total</td>
<td>8 (26.6)</td>
<td>22 (73.6)</td>
</tr>
</tbody>
</table>

* Figures in bracket indicate percentage of mortality

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), fibrous 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).

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 bronchile. 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 desquamation of mucosal epithelium and leucocytic infiltration in mucosa and depletion of lymphocytes in lamina propria.

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

<table>
<thead>
<tr>
<th>Months</th>
<th>Mortality</th>
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<tbody>
<tr>
<td>July</td>
<td>2</td>
</tr>
<tr>
<td>August</td>
<td>10</td>
</tr>
<tr>
<td>September</td>
<td>4</td>
</tr>
<tr>
<td>October</td>
<td>4</td>
</tr>
<tr>
<td>November</td>
<td>9</td>
</tr>
<tr>
<td>December</td>
<td>1</td>
</tr>
</tbody>
</table>
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 pathogenesis of hepatic diseases and in correlation of etiological agents in buffaloes/buffalo calves.

REFERENCES


