ABSTRACT

The present investigation was carried out to study the various pathological conditions occurring spontaneously in the livers of buffaloes. A total number of 2,119 buffalo liver samples was collected irrespective of sex, breed and age of the animal from slaughterhouses located in and around Bikaner, Rajasthan. Among the various pathological conditions of the livers, fatty change was found to be the highest followed by cirrhosis, congestion, cell swelling, abscess, RES response, haemorrhage, pigmentation, necrosis, apoptosis and telangiectasis.

Keywords: buffalo, fatty changes, liver, prevalence, spontaneous lesions

INTRODUCTION

The buffalo is one of the key animals in the agricultural economy, contributing substantially to the gross national income (GNP) by way of good quality milk, meat, export quality leather and physical power. The buffalo is a part of the property, possession and profession of rural farmers. Not only that, it is an easily ‘convertible currency’ and a reliable ‘living bank’ to serve the immediate needs of the rural masses in several communities. Of all domestic animals, the Asian buffalo holds the greatest promise and potential for production (Cockrill, 1994). India has world’s best buffalo dairy breeds and provides superior buffalo germplasm to several countries of the world (Kaikini, 1992). The liver is one of the vital organs of the body, susceptible to various affections which influence the total health status of the animal. Further, considerable liver damage may be present before clinical signs are apparent largely due to the high degree of reserve functional capacity. The present investigation was carried out on the livers of buffaloes to investigate the various spontaneous lesions of the liver.

MATERIALS AND METHODS

The materials for the present study comprised livers obtained from buffaloes slaughtered at slaughterhouses located in and around Bikaner, Rajasthan. A total of 2,119 livers of slaughtered buffaloes were examined irrespective of age, breed and sex for spontaneous liver lesions. Gross study was performed during collection of samples from the slaughterhouses primarily and then during trimming of the samples for histopathology. Out of these, 476 livers exhibiting...
gross alterations were cleaned with normal neutral saline and the changes were recorded. The lesion containing tissue samples were collected and preserved in 10% neutral formalin solution for histopathological study. After 48-72 h, these tissue pieces were washed overnight in running tap water, dehydrated in ascending grades of alcohol, cleared in benzene and embedded in paraffin wax (60-62°C melting point). Sections of 4-5 micron thickness were cut and stained with haematoxylin and eosin as per the standard procedure recommended by Lille (1954).

RESULTS AND DISCUSSION

The various spontaneous liver lesions observed during the present study were as shown in Table 1. Examination of the livers of 476 buffaloes revealed various types of lesions in 180 animals, amounting to 37.82%.

Among the various pathological lesions of the livers, fatty changes were found to be the highest (6.09%), akin to the observation of Gupta (1983). However, Kulkarni (1992) and Dhote et al. (1992) reported much lower incidences, being 2.45% and 1.5%, respectively. While Purushotam and Rajan (1985) noticed the condition in a much higher number of cases (21.7%). Grossly these livers appeared pale, soft, and greasy with rounded edges. Histopathologically, the hepatic cells showed presence of fat droplets as clear round spaces and granular cytoplasm. Large clear cavities were seen indicating that the scattered cells with fatty changes coalescence (Figure 1). Sinusoids were often reduced by swollen fatty cell cords (Figure 2). Focal, diffuse, pericentral and periportal types of fatty changes noticed presently were comparable to those described by Jubb et al. (1993) and Dhote et al. (1992). Ketosis may be one of the possible causes of fatty changes.

A total of 5.25% cases showed partial or complete cirrhosis. The liver was constricted in many cases. Thickened capsule, finally granular surface, hard to cut and numerous newly formed bile ducts in cirrhotic liver were observed in the present study.

Table 1. Spontaneous liver lesions in slaughtered buffaloes (n = 476).

<table>
<thead>
<tr>
<th>S. No.</th>
<th>Spontaneous lesions</th>
<th>No. of Cases (180)</th>
<th>Percentage of affected animals (37.82)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Fatty change</td>
<td>29</td>
<td>6.09%</td>
</tr>
<tr>
<td>2.</td>
<td>Cirrhosis</td>
<td>25</td>
<td>5.25%</td>
</tr>
<tr>
<td>3.</td>
<td>Congestion</td>
<td>24</td>
<td>5.04%</td>
</tr>
<tr>
<td>4.</td>
<td>Cell swelling</td>
<td>19</td>
<td>4.00%</td>
</tr>
<tr>
<td>5.</td>
<td>Abscess</td>
<td>18</td>
<td>3.78%</td>
</tr>
<tr>
<td>6.</td>
<td>RES response</td>
<td>18</td>
<td>3.78%</td>
</tr>
<tr>
<td>7.</td>
<td>Haemorrhage</td>
<td>14</td>
<td>2.94%</td>
</tr>
<tr>
<td>8.</td>
<td>Pigmentation</td>
<td>13</td>
<td>2.73%</td>
</tr>
<tr>
<td>9.</td>
<td>Necrosis</td>
<td>9</td>
<td>1.90%</td>
</tr>
<tr>
<td>10.</td>
<td>Apoptosis</td>
<td>7</td>
<td>1.47%</td>
</tr>
<tr>
<td>11.</td>
<td>Telangiectasis</td>
<td>4</td>
<td>0.84%</td>
</tr>
</tbody>
</table>
study as described by previous researchers (Gupta, 1983; Moorty et al., 1984; Dhote et al., 1992; Vegad and Katiyar, 1998). Microscopically considerable proliferation of fibrous connective tissue was marked mainly in the portal areas replacing hepatic cells. There was little lymphocytic infiltration of fibrous strands. Portal veins and sinusoids were dilated and irregular in shape. Cirrhosis was frequently noticed concomitant with parasitic condition; however, in many cases contributory factors could not be incriminated.

Congested liver 24 (5.04%) showed the character of “nutmeg” liver and mottled appearance. Microscopically, sinusoids were dilated and central veins were engorged with erythrocytes. Slight periductal infiltration of round cells was noticed. Hepatic cells around the central veins were degenerated and atrophied. Moderately proliferation of fibrous tissue was also seen in portal spaces, corroborating the descriptions of Cohrs (1967) and Gupta (1983).

Cell swelling were found in 19 (4.00%) of the cases. Grossly, the affected livers were enlarged and of lighter color with rounded edges. Microscopically, the enlarged rounded hepatocytes containing eosinophilic granular cytoplasm which obliterated sinusoidal space.

Abscess was found in only 2.94% cases. Grossly, whitish foci on the surface of the liver were found (Figure 3). Their size varied from 0.5 to 1.5 cm in diameter. Polymorphonuclear leukocytes at the center surrounded by a thin fibrous capsule were seen microscopically (Figure 4). Similar histopathological changes have been reported in earlier studies (Gupta, 1983 and Dhote et al., 1992).

RES response was observed in 3.78% cases. Grossly and microscopically the livers with RES response showed variable extent of diffusely distributed small foci in the form of Kuffer’s cell hypertrophy and hyperplasia, degeneration, necrosis and granulomas being comparable to the descriptions of Chors (1967). These apparently were consequent to the body defense against underlying subtle infections.

In the 14 cases of haemorrhage, the liver showed a dark brown petechial and/or ecchymotic nature and superficial or deeper location microscopically variable extents of degeneration, necrosis and neutrophilic and lymphocytic infiltrations in the involved area in conformity with Dhote et al. (1992).

Pigmentation was seen in 13 (2.73%) cases. The darker colored liver showing yellowish brown granules of haemosiderin in the Kuffer’s cells macrophages and free in the sinusoids in H&E stain (Figure 5) being blue in Perls’ (Figure 6) were similar to the reports of Gupta (1983) and Tripathi and Chattopadhyay (1989).

In hepatic necrosis (1.90%), pale patches were found on the surface of liver presenting a mosaic appearance. White, opaque necrotic foci were uniformly distributed throughout the liver. Microscopically, the hepatic cells were very much swollen and granular with pyknotic or lytic nuclei, infiltration with neutrophils and lymphocytes, engorged veins, fibroplasias and Kuffer’s cell proliferation were observed. This presumably may be due to involvement of some toxins in feed (Jubb et al., 1993) possibility fungal, bacterial or plant toxins or chemicals playing some role in causing the condition.

Apoptosis was seen in seven (1.47%) cases. Occurrence of apoptic cells and apoptic bodies in the hepatocytes were similar to Kumar et al. (1992) and Bhel and Tripathi (1999). These apparently pointed to be the result of the mechanism regulating the number of cells since these were...
Figure 1. Photomicrograph of liver section showed presence of fat droplets as clear round space and granular cytoplasm of hepatic cells. H&E X 200.

Figure 2. Photomicrograph of liver section showing fatty changes with coalescence forming large sized cavities, sinusoidal congestion and edema is also seen. H&E X 400.

Figure 3. Photomicrograph of centrilobular hemosiderosis showing golden brown pigment in the Kuffer cells and in the sinusoids. H&E X 200.
Figure 4. Photomicrograph showing blue haemosiderin pigment in the Kuffer cells. Pearls’ prussian blue staining X 200.

Figure 5. Surface and cut surfaces of liver pieces showing abscesses (a,b) and congestion (d).

Figure 6. Photomicrograph showing within outside area of calcification, detritus and pyogenenic membrane. H&E X 200.
neither frequent and nor abundant.

Telangiectasis was observed in 0.84% cases. This was comparable to Gupta (1983) and Hassib et al. (1995), who reported 1% and 0.58%, respectively. The gross and microscopic character of liver showed as dark reddish depressed single or multiple areas of variable size and dilatation of group of sinusoids and/or larger cavernous spaces lined by endothelium and containing erythrocytes with or without connections were in line with the descriptions of Runnels (1976).

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REFERENCES


