GIANT CELL PNEUMONIA IN BUFFALO CALVES
(BUBALUS BUBALIS)

R. G. ARORA AND D. S. KALRA
Department of Veterinary Pathology, Haryana Agricultural University, Hisar

SUMMARY

Two outbreaks of 'giant cell pneumonia' in buffalo calves, accompanied by respiratory distress and mild catarrh of the intestines, recorded for the first time in India, are described. The possibility of PI3 virus, playing a role in the causation of the disease, is suggested on the basis of characteristic histopathological findings.

INTRODUCTION

The respiratory infections either alone or associated with enteritis are incriminated as one of the major causes of mortality amongst young buffaloes. Of these infections the giant cell pneumonia, which has recently been recognised in bovine calves (Omar, 1966), is assuming increased pathological significance. The condition, as the name indicates, is characterised histologically, by the appearance of "syncytial giant cells" in lung sections and occurs alone or as a complex of pneumoenteritis. In buffaloes, there appears to be no report of mortality from giant cell pneumonia though Balani and Iyer (1968) recorded a few cases in apparently healthy buffaloes, slaughtered at Bareilly (India) for food purposes. The present study pertains to the investigation of two disease outbreaks that occurred in the years 1970 and 1971 among buffalo-calves of the buffalo breeding herd located at the Government Livestock Farm Hisar (India).

MATERIAL AND METHODS

At the time of investigation, information in regard to population at risk, number of animals affected and died, age of affected animals, etc. was obtained. The affected calves were examined clinically, and their temperature recorded. Microscopic examination of blood smears and faecal samples, from the affected cases as well as from a few of the incontact and apparently healthy animals was carried out. Necropsy examination was conducted, on six calves within 1-4 hours after death and appropriate material collected for laboratory studies. Cultures were attempted from heart blood, lung tissue, intestine and associated lymph nodes on blood and MacConkey agar. Pieces of affected lung, collected in 50 per cent glycerine saline, were used for virological examination. Lung, trachea, intestine and associated lymph nodes, collected in 10 per cent formal saline, were processed for histopathological examination using conventional techniques.

OBSERVATIONS

During the last three years (1969-71), the disease broke out on two occasions, the first in December 1970, and the second in March 1971. It involved buffalo calves of 3-8 months of age and of both sexes. The morbidity and mortality data are given below:

<table>
<thead>
<tr>
<th>Period</th>
<th>Population at risk</th>
<th>Number affected</th>
<th>CSMR*</th>
<th>Number died</th>
<th>CFR**</th>
</tr>
</thead>
<tbody>
<tr>
<td>December, 1970</td>
<td>126</td>
<td>30</td>
<td>23.81%</td>
<td>12</td>
<td>40.0%</td>
</tr>
<tr>
<td>March, 1971</td>
<td>100</td>
<td>15</td>
<td>15.0%</td>
<td>7</td>
<td>46.67%</td>
</tr>
</tbody>
</table>

*C.S.M.R. = Cause-specific morbidity rate.
**C.F.R. = Case fatality ratio.
GIANT CELL PNEUMONIA IN BUFFALO CALVES

The clinical signs manifested by the sick calves were anorexia, moderate amount of bilateral nasal discharge of mucopurulent nature, mild diarrhoea, dyspnoea and a harsh hacking cough. The rectal temperature was 103-105°F. The affected calves had a tendency to isolate themselves and were reluctant to suckle their dams. Deaths usually occurred within 24 to 72 hours of the onset of symptoms. Treatment of the ailing animals with sulphamezathine, antibiotics and anthelmintics did not yield any encouraging results, but some animals which were sick for about a week, made an eventual recovery. At the farm, the management and other husbandry practices were not satisfactory. The calves were housed in open sheds and were liable to be exposed to cold.

LABORATORY STUDIES

Microscopic examination of wet and stained blood smears failed to reveal any evidence of protozoan infection. Faecal samples were found negative for helminth and coccidial infection. Cultures attempted from heart blood and affected tissues (lung, intestine and associated lymphnodes) did not yield any organism of pathological significance.

Cell free filtrate of the lung tissue, when passaged in the embryonating hen egg, did not haemagglutinate the chicken red blood cells.

GROSS PATHOLOGY

Except for mild catarrh of the intestines, the lesions were essentially confined to the respiratory tract. Both the lungs revealed areas of consolidation of the apical and cardiac lobes together with anterio-ventral portions of diaphragmatic lobes. In one case the anterior halves of the diaphragmatic lobes were hepatised in addition to complete consolidation of apical and cardiac lobes. The hepatised areas were purplish red in colour and firm in consistency. In some cases the areas revealed a number of small pea-sized, greyish white nodules, raised above the pleural surface. Interlobular septae were slightly thickened in most cases. A dirty white to greyish thick exudate was seen on cutting open the bronchi. Sometimes similar exudate was present in the trachea, the mucosa of which was severely congested. The mucosae of nasal passages were also hyperaemic, and covered with mucpurulent exudate. Intestines, particularly ileum and caecum, showed the presence of slimy mucus exudate admixed with intestinal contents. The mucosa was slightly hyperaemic and swollen.

HISTOPATHOLOGY

Lungs. Microscopically, the lung sections revealed the infiltration of mononuclear cells and presence of cellular debris in the bronchiolar and alveolar luminae. Varying degree of polymorphs also constituted the formation of cellular exudate present in the alveoli and bronchi. The most characteristic histological feature was the appearance of syncytial giant cells which were either lying free in the lumen or still attached to the alveolar walls (Fig. 1). Another significant feature was the septal cell proliferation at some places, so as to cause a discontinuous lining of the alveolar septa giving rise to the formation of the pseudoepithelialisation. In addition, a moderate to heavy peribronchial and perivascular lymphoidal cell infiltration was observed in some areas.