BRONCHOPNEUMONIA OF CALVES CAUSED BY PARAINFLUENZA VIRUS TYPE 3

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THERE HAVE BEEN MANY ISOLATIONS of Parainfluenza virus type 3 (PI3) from cattle in recent years. Various serological surveys have indicated that this virus is widespread in the cattle population. The virus has been associated with upper respiratory tract infection and the shipping fever complex, though many subclinical infections also occur (5, 8). There are, however, comparatively few reports of lower respiratory disease associated with this virus without the complication of secondary bacterial infection. Bögel (3) recorded pneumonia in one calf but found that tracheo-bronchitis was the more usual result of experimental infection. Saunders and Berman (9) noted bronchopneumonia in several experimentally infected calves including one fatal case unassociated with secondary *Pasteurella s.p.p.* infection. Pneumonitis in two experimental calves was recorded by Woods *et al.* (10). Well documented evidence that this virus can cause lower respiratory tract disease is presented by Betts *et al.* (2) who described both natural and experimentally produced disease. Alveoli were filled with proteinaceous material, mononuclear cells and cellular debris. There was bronchiolar epithelial hyperplasia with necrosis and desquamation in the later stages and there was epithelialisation and some epithelial giant cell formation in the alveoli. Intracytoplasmic acidophilic inclusions were noted in bronchiolar and alveolar epithelium and in alveolar macrophages. Dawson *et al.* (4) described essentially the same findings in experimentally infected calves although pneumonia was not clinically apparent.

Recently, several animals from outbreaks of pneumonia in young cattle with high morbidity but low mortality have come to necropsy. The type of pneumonia found did not readily fit any of the recognized pathological entities (7) although in some cases there was bacteriological and histopathological evidence of *Pasteurella spp.* infection.

This report concerns two dead calves from two separate outbreaks of respiratory disease in housed calves during January, 1965. On the first farm there had been evidence of respiratory disease affecting various calves for a period of four or five months. The calf submitted for necropsy was four months old and had been treated with chloramphenical the night previously. The outbreak on the second farm was explosive with all 18 calves ranging in age from ten days to six months being affected within two days. Many of these developed clinical signs of pneumonia while all showed coughing and reduced food intake. The younger calves were more severely affected than the older ones and some were still coughing five weeks later. Mortality was limited to two three-week-old, and one four-month-old animals. This latter, which had been extensively treated with chloramphenical, was submitted for necropsy.

GROSS PATHOLOGY

Findings in each calf were similar. The apical and cardiac lobes and antero-ventral margins of the diaphragmatic lobes showed light grey-red consolidation with moderate distension of the interlobular septa with gelatinous fluid. In the deeper parts of the diaphragmatic lobes consolidation was more extensive, occurring in the vicinity of the larger bronchi. A lobular pattern was evident. In both cases the dorsal parts of the diaphragmatic lobes showed extensive interstitial emphysema. Calves appeared to have asphyxiated. The cut surface of the lung showed light red

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consolidation which was not particularly moist. From the bronchi a small amount of slightly blood-tinged mucoid pus could be expressed.

Histopathology

Representative paraffin sections from different parts of the lung of each calf were stained with hematoxylin and eosin. Examination revealed an acute catarrhal exudation of alveoli and bronchioles which appeared to be preceded or accompanied by flooding of alveolar spaces with proteinaceous fluid. A catarrhal bronchiolitis chiefly affecting the terminal bronchioles extended proximally to include milder changes in the smaller bronchi. The epithelial cells appeared to round up and lift off the basement membrane prior to undergoing necrosis. In the affected bronchi some edema appeared about the basement membranes and a mild infiltration with leucocytes. Interalveolar capillaries were greatly dilated and congested though there was no local increase in cellularity. The alveoli and bronchioles contained varying amounts of proteinaceous fluid and cells which were mainly alveolar macrophages but included varying numbers of polymorphs. A number of multinucleate cells were noted although these were never numerous. The characteristic feature of this condition was the finding of large numbers of discrete acidophilic intracytoplasmic inclusion bodies in alveolar macrophages and bronchiolar and bronchial epithelial cells. In a few small areas nearly every cell was affected although in other parts inclusions were less numerous.

Distension of interlobular septa by proteinaceous fluid which had been noted grossly was confirmed. Septa were also infiltrated with a moderate number of neutrophils.

Bacteriological and Virological Examinations

Pasteurella multocida was isolated from the lung of the first calf. Lung tissue from the second animal proved to be bacteriologically sterile.

Unfortunately fresh tissue was not kept for virology. Formalised tissue was washed, however, and sections treated with fluorescent antibody to PI3 virus. Extensive fluorescence indicated the presence of large amounts of specific virus in the lung. In addition, an antigen prepared from extracts of lungs fixed complement to high titers in the presence of PI3 guinea-pig antiserum, but not in the presence of PI1 or PI2 antiserum or normal guinea-pig serum.

Discussion

The reports of Betts *et al.* (2) and Dawson *et al.* (4) provide well documented evidence that PI3 virus can initiate bronchiolitis and pneumonia in calves. The present report confirms these findings. The pathology of a catarrhal bronchiolitis associated with many intracytoplasmic acidophilic inclusion bodies and apparently unassociated with a heavy bacterial infection is highly suggestive of a viral etiology. Fluorescent antibody studies and serological studies indicate PI3 virus infection.

Reports by Jarret (6) and Baxter (1) of inclusion body pneumonias of calves probably described the same condition. Betts *et al.* (2) found that inclusion bodies were more numerous at five days than at seven days in experimentally infected calves. Disappearance of inclusions with time, a low mortality rate, or secondary bacterial infection may be reasons why this disease has not been noted more frequently. Several other cases of calf pneumonia examined at this laboratory[1] have shown somewhat similar histopathology although without inclusion bodies in the lower respiratory tract. From one of these cases which had been treated extensively with antibiotics, PI3 virus was isolated from a lung which was bacteriologically sterile. Acidophilic intracytoplasmic inclusions were demonstrated in the nasal epithelium.

Whereas it is probable that the majority of PI3 virus infections of calves are either inapparent, associated with influenza-like disease or the shipping fever complex, it is evident that bronchiolitis and pneumonia may also occur. The experimental transmission of PI3 to calves by Dawson *et al.* (4) gave rise to pneumonia although this

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was clinically inapparent. They suggested that it might only be important under conditions of stress and secondary bacterial involvement. The present report and that of Betts *et al.* (2) indicate that pneumonia caused by PI3 virus is probably important *per se*.

Summary

Two separate cases of pneumonia characterized by bronchiolitis with a serocellular catarrhal exudate are described. The finding of many acidophilic intracytoplasmic inclusion bodies in lower respiratory tract epithelium and alveolar macrophages, together with a marked fluorescence with specific fluorescent antisera and a positive complement fixation test using lung extract as antigen, indicated that Parainfluenza virus type 3 was the etiological agent. Pertinent literature supporting the diagnosis is discussed.

Résumé

Il est question ici de deux cas séparés de pneumonie caractérisée par une bronchiolite accompagnée d'exudation catarrhale séro-cellulaire. La présence de plusieurs corps d'inclusion acidophiliques intracytoplasmiques dans l'épithélium et les macrophages alvéolaires de la partie inférieure des voies respiratoires, ainsi qu'une fluorescence marquée avec des antiserums fluorescents spécifiques et une épreuve complémentaire positive de fixation où on a utilisé comme antigène des extraits de poumon, indiquent que l'agent étiologique était le "virus parainfluenza" du type numéro 3. On analyse aussi des études pertinentes qui appuient ce diagnostic.

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ABSTRACT

Bar-Moshe, B. (1964). The isolation of Mycoplasma from an outbreak of bovine mastitis in Israel. Refuah Vet. 21, 99.

Organisms morphologically and culturally indistinguishable from Mycoplasma were isolated from udder secretions of 19 out of 34 cows with mastitis in a herd with a high incidence of this disease. It was characterized by sudden dysgalactia and absence of general symptoms. Routine bacteriological tests were usually negative, though five isolations of Staphylococcus aureus and one of Streptococcus dysgalactiae were made.

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