Atypical Interstitial Pneumonia in Cattle

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SUMMARY

Atypical interstitial pneumonia is described as two clinical syndromes in young cattle. One syndrome occurs in animals which have clinical evidence of pneumonic pasteurellosis, responds initially to treatment for one to two days and then develops acute signs of atypical interstitial pneumonia. The second syndrome involves acute respiratory distress in young calves due to atypical interstitial pneumonia with antecedents of enzootic pneumonia. The postmortem lesions are described along with discussion of the possible pathogenesis of the condition and treatment.

RÉSUMÉ

Pneumonie interstitielle atypique des bovins

Les auteurs décrivent la pneumonie interstitielle atypique des jeunes bovins, comme deux syndromes cliniques. L’un d’eux affecte des sujets qui manifestent une évidence clinique de pasteurellose pulmonaire; il répond initialement au traitement, pour un ou deux jours, et évolue ensuite vers l’apparition de signes aigus de pneumonie interstitielle atypique. L’auteur se manifeste sous la forme de troubles respiratoires aigus, attribuables à la pneumonie interstitielle atypique, chez des sujets ayant déjà souffert de pneumonie enzootique. Les auteurs décrit aussi les lésions pulmonaires macroscopiques et commentent la pathogénèse probable, ainsi que le traitement de cette condition.

Atypical interstitial pneumonia (AIP) has been defined as an acute, noncontagious respiratory disease of cattle characterized by sudden onset of expiratory dyspnoea and poor response to treatment (1). Initial description of the disease divided it into acute and chronic forms (1). The exact cause is not known, but investigators have conjectured that dietary tryptophan and a hypersensitivity are two possible causal factors (7). The purpose of this report is to describe AIP as it has been observed in two distinct clinical syndromes in young cattle under intensive management systems.

CLINICAL FINDINGS

The first syndrome occurred in cattle between six and 24 months of age with a common history of recent introduction to the feedlot. Affected cattle became anorexic with a temperature of 40°C to 42°C and have had all the clinical features described for pneumonic pasteurellosis (1, 3). When treated with antibiotics, they initially showed a favourable response for a day or two: temperatures returned to normal and the animals started to eat again. During this apparent recovery phase, animals developed a severe expiratory dyspnoea over a six to 12 hour period with a marked increase in respiratory rate. Cattle remained bright and alert but anorexic. On auscultation of the lung, there were increased bronchial tones over the entire lung field and in animals with the most severe respiratory distress there was at times crepitant raies and friction rubs of interstitial emphysema. Many cases had an increased area of auscultation often amounting to a 2-4 cm posterior displacement of the boundary of the lung field. Most animals did not respond and died within 24 to 96 hours of developing the expiratory dyspnoea.

The second clinical syndrome has been observed in calves between three and six months of age. It was usually seen in dairy calves housed under intensive management systems. Affected calves often had a history of enzootic pneumonia as previously described (1, 3). They had apparently recovered from infection and were doing well when the owner reported the sudden onset of expiratory dyspnoea. These calves had the clinical features previously described for older cattle. Most calves did not respond to any treatment and died in 24 to 96 hours from the onset of expiratory dyspnoea.

POSTMORTEM FINDINGS

The gross lesions in the lungs of the first type of syndrome occurred in feeder cattle and fitted the classical description of acute atypical interstitial pneumonia (6). The lungs did not collapse, were heavy and in some, but not all instances, had extensive interstitial emphysema. All lobes were affected but some normal lobules remained in each lobe. Affected lobules were red to tan in colour, rubbery, wet and distinctly separated by edema and/or emphysema from neighbouring lobules. Seventy-five percent or more of the lung parenchyma was affected. Bronchial lymph nodes were moderately enlarged, wet and firm. Large air passages contained much froth.

The main microscopic features of the lesions were massive flooding of terminal air passages with proteinaceous edema fluid and marked congestion of alveolar walls. The dense proteinaceous edema

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fluid sometimes filled alveoli or formed hyaline membranes along alveolar walls and ducts. Considerable variation in the extent of the latter lesions occurred, in particular, the amount of protein and the extent of involvement in individual lobules. Alveolar walls were thickened by edema and had a marked hyperplasia of type II cells which gave an acinar appearance to the alveoli. Inflammatory cells in the lumen of the alveoli and ducts were generally present in moderate numbers. A mixture of neutrophils and macrophages were mostly seen with the occasional eosinophil and giant cell. The epithelium of small bronchioles appeared normal in some areas of the affected lung, but in other areas it was distinctly abnormal. Necrosis and absence of epithelium, evident in parts of the lining, were associated with disorientation of the remaining epithelium. Epithelial cells varied markedly in size and shape and some appeared stretched in an attempt to maintain continuity. Exudate in the lumen was similar to that in alveolar ducts. Large air passages and blood vessels appeared normal except for dilated, fluid filled lymphatics beside their walls. Interlobular tissue was very edematous and lymphatic vessels were very distended with proteinaceous fluid. Sometimes emphysema was prominent in these areas.

In the second type, which occurred in younger animals, there was evidence of chronic bronchopneumonia in the anterior ventral portions of the lungs. The lesions occasionally involved only a small portion of lung or in some cases most of the apical and cardiac lobes. Affected lobules were usually partially collapsed, reddish and firm with bronchiolar mucopurulent exudate in varying quantities. The lesions in the remainder of the lung were very similar, in both gross and microscopic features, as those described for the first type except for the fact that interstitial emphysema was much reduced or absent. The acute lesions in the posterior portions of the lung accounted for the acute clinical signs. Viral and bacterial culture of the lung in the areas involved with the severe interstitial pneumonia have been essentially negative with no clear indication of microbiological etiology.

TREATMENT AND PREVENTION
Many different antibiotics and anti-inflammatory drugs have been used to treat this condition with little success. Occasional animals in acute distress have apparently responded to dexamethasone (0.25 mg.kg⁻¹) administered by the intravenous route with repeated doses if required over two to three days. Aspirin has been reported to be useful in the treatment of experimental acute interstitial pneumonia of calves (4). In our experience, no improvement was noted in the cases treated with aspirin therapy.

Methods to prevent the condition are not known at the present time but steps can be taken to try to prevent both pneumonic pasteurellosis and enzootic pneumonia (3).

DISCUSSION
The clinical and postmortem findings have many aspects in common with both dietary, and allergic types of atypical interstitial pneumonia. No evidence to implicate dietary factors has been found. It is possible that autoimmune phenomena are occurring in previously damaged lung (2). Atypical interstitial pneumonia has been described in yearling feedlot cattle but there is no report of any previous illness in the cases described (5).

These cases have been observed for several years by many clinicians and pathologists. We have heard that the same is true at other institutions. The cases tend often not be be discussed at veterinary meetings because of the failure of clinicians, pathologists and microbiologists to shed light on the etiology, treatment or prevention of the problems. Also cases tend to occur singly and do not receive great emphasis as a major problem.

The purpose of this report is to encourage discussion and exchange of information about the condition and to encourage investigation of the pathogenesis and etiology.

REFERENCES