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Atypical Interstitial Pneumonia in the Bovine

by Martin D. Ficken*
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Case Report

On August 5, 1980, a dead 950 pound steer was submitted to the Iowa Veterinary Diagnostic Laboratory for necropsy (IVDL Case 18274). The history included acute dyspnea, raspy cough, a temperature of 40.5-41.7°C (105-107°F) in 20 out of 125 animals and two deaths. These cattle had been purchased on July 17, 1980 and first noticed sick on August 4, 1980. The tentative diagnoses were Hemophilus pneumonia and viral pneumonia. Treatment included intravenous tetracycline and intramuscular penicillin and resulted in a "fairly good" response.

At necropsy the lungs failed to collapse, filled the thoracic cavity, were emphysematous with large bullae, and had an obvious, lobular, meaty pattern on section. No other significant gross lesions were found.

Microscopic changes in the lungs included vesicular and bullous emphysema, proteinaceous edema, and atelectasis. A few foci of neutrophils were found in and around the bronchioles. Proliferation of type II pneumocytes was a dominant feature with cuboidal epithelium lining the alveoli. No bacteria or virus could be isolated or demonstrated. The history and the subsequent laboratory findings resulted in a diagnosis of atypical interstitial pneumonia (AIP).

Lungs failed to collapse and were meaty and lobular

Cuboidal epithelium and proteinaceous edema

Discussion

The term atypical interstitial pneumonia was introduced to describe bovine respiratory disorders in which some or all of the following lesions are found: congestion, edema, hyaline membranes, interstitial emphysema, alveolar epithelial hyperplasia, fibrosis, and cellular infiltration of interalveolar septa. Grossly, the frequently observed lesions are; the lungs do not collapse upon opening the thorax, interstitial and interlobular emphysema which accentuates lobular demarcation, and pulmonary edema.

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Interstitial and interlobular emphysema accentuating lobular demarcation

The clinical signs usually associated with AIP are dyspnea, loud expiratory grunts, frothing at the mouth, mouth-breathing, tachypnea, and coughing.

Treatment of AIP usually involves removing the animals from the source of the problem which many times is the feed or pasture. Many animals will recover if only this is done. Other forms of symptomatic treatment which have been suggested include atropine, corticosteroids, antibiotics, antihistamines, diuretics, and epinephrine.

In the last decade, much more information has been provided about the diseases grouped under AIP and it is now apparent that there are important differences between the various forms in terms of clinical signs, epidemiology, etiology, and pathogenesis which justify division of AIP into several entities. The following divisions have been proposed by Breeze et al. (Table 1) and were found very useful for this presentation.

**TABLE 1**

| An Index of Syndromes Included in Atypical Interstitial Pneumonia of Cattle |
|-------------------------------|----------------------------------|
| A. Hypersensitivity diseases  | 1. Extrinsic allergic alveolitis (farmer's lung) |
|                               | 2. Milk allergy                   |
| B. Diseases of unknown etiology | 1. Fog fever and acute bovine pulmonary emphysema |
|                               | 2. Diffuse fibrosing alveolitis   |
|                               | 3. Atypical interstitial pneumonia of calves |
| C. Parasitic diseases        | 1. Dictyocaulus viviparus         |
|                               | 2. Ascaris lumbricoides           |
| D. Plant poisoning           | 1. Ipomoea batatas (sweet potato) |
|                               | 2. Zieria arborescens (stinkwood) |
|                               | 3. Perilla frutescens (purple mint) |
|                               | 4. Brassicae                      |
|                               | 5. Other plants                   |
| E. Exposure to irritant gases and fumes | 1. Nitrogen dioxide |
|                               | 2. Smog                           |
|                               | 3. Zinc oxide                     |
|                               | 4. Chlorine and hydrogen sulfide  |
| F. Experimentally-induced syndromes | 1. Systemic anaphylaxis |
|                               | 2. Bordetella pertussis infection |
|                               | 3. Administration of indolic compounds |

A. Hypersensitivity diseases

1. Extrinsic allergic alveolitis (farmer's lung). Farmer's lung is an allergic respiratory disease of man. The pathogenesis is considered to be a type III (Arthus) hypersensitivity reaction in the peripheral parts of the lung. It develops after exposure to spores of *Micropolyspora faeni* and other thermophilic Actinomycetes which are normally found in the dust of moldy hay. Precipitating antibodies are associated with vascular damage by immune complexes at the alveolar level and the mechanism has been described as "extrinsic allergic alveolitis".

A disease similar to farmer's lung has been described in cattle which are fed moldy hay and the lung lesions resemble those seen in man. Precipitating antibodies against *M. faeni* have been found in cattle with respiratory disease. The disease has been reported in western England and is frequently found in regions where hay is baled at a high moisture content (greater than 30%) due to climatic conditions. Overheating occurs and a thermophilic microflora takes over. It has been postulated that *M. faeni* could grow and sporulate in moist manure and silage. These spores could become so overwhelming that they become a major portion of "pen dust" and be inhaled causing the hypersensitivity reaction. This is postulated because of the incidence of AIP in yearling feedlot cattle. The greatest incidence of cases being in the summer and fall, the seasons of most "pen dust", supports this possibility.

Farmer's lung may develop suddenly or be a chronic problem. The acute form is rarely seen in more than one animal in a herd at a time, however successive cases over a period of time may result. The clinical signs of the acute form are sudden dullness, decreased appetite, and a fall in milk production. Coughing occurs but may be overlooked. Crackles may be heard cranioventrally upon auscultation. Chronic cases have a history of excessive weight loss and coughing for several years in the winter months with remission during the grazing season. It is assumed that remission occurs because the animals are removed from the source of the antigen during the grazing season. Tachypnea and hyperpnea are present with coughing producing green mucus.

At necropsy, in acute cases, many small grey spots are present on the pleural surface. The peripheral acini of some lobules are over-
inflated and this produces a pale-pink, raised edge around a darker red-central portion. Microscopically there are infiltration of interalveolar septa by lymphocytes, plasma cells, and interstitial cells, intraseptal lymphocytic aggregates without germinal centers, characteristic epitheloid granulomata with multinucleated giant cells, bronchiolitis, and bronchiolitis obliterans. In chronic cases the macroscopic appearance is similar to the acute cases. However, in some lobules, focal fibrosis of the interalveolar septa with localized alveolar epithelial hyperplasia is present. This fibrotic change only affects a minority of the lung segments. There may even be focal replacement of the alveolar epithelium by tall columnar ciliated or mucus-secreting cells.

Treatment of this disease entails removing the offending feed-stuff, adequate ventilation, and water-spraying to reduce the dust, in conjunction with the before-mentioned symptomatic treatment for those acutely ill. Prevention and control of this disease is the same as the treatment.

2. Milk allergy. Milk allergy is a term used to describe a sudden onset of urticaria which sometimes is accompanied by respiratory distress. This condition usually goes unnoticed since it only features a transient urticaria. Clinically there is dyspnea just before milking during the end of lactation or after a milking routine change. Lesions include pulmonary congestion, edema, interstitial emphysema, hyaline membranes, and intraalveolar hemorrhage. The disease is an autoallergy, in which the animal becomes allergic to its own milk, principally alpha-casein.

B. Diseases of unknown etiology

1. Fog fever and acute bovine pulmonary emphysema. Fog fever in England is an acute respiratory syndrome with minimal coughing which occurs in beef cattle greater than two years of age. It usually occurs in the fall months (August to November) following a change to a better, often lush pasture. This disease is probably the same disease seen in the United States and Canada which is termed acute bovine pulmonary emphysema (ABPE). Almost all outbreaks of fog fever/ABPE occur within two weeks after moving cattle from dry pastures to lush pastures such as hay or silage aftermath (Foggage), hence the term fog fever. Fog fever has nothing to do with environmental conditions such as fog. This condition has been mainly limited to fat, single-suckler females of the Hereford breed. The morbidity rate is variable but may reach 50%. Many of the severely affected animals may die. Severe cases have clinical signs of gross dyspnea, loud expiratory grunts, frothing at the mouth, mouth-breathing, and tachypnea. Auscultation sounds are soft with crackles only heard occasionally. Less severe cases may have only tachypnea and hypopnea. Usually the whole herd becomes more tranquil during these outbreaks. Coughing is not a dominant feature in any of the animals.

In animals that die there are ecchymotic and petechial hemorrhages in the larynx, trachea, and bronchi and the airways are filled with frothy fluid. Cranial lung lobes are deep-red and the cut surface has a smooth glistening glass-like appearance. Intersitial emphysema with large bullae is present in all of the lung and may extend along the back. Pulmonary edema is present especially in the ventral part of the lung and gelatinous edema fluid can be found in the perivascular connective tissue and interlobular septa. Microscopically, hyaline membranes line the alveoli and alveolar ducts; the air spaces are filled with edema fluid, eosinophils, neutrophils, alveolar macrophages, and multinucleated giant cells. In most animals there is some alveolar epithelial hyperplasia. The alveoli are lined by a single layer of cuboidal type II pneumocytes containing frequent mitotic figures. If animals live three or four days and then are slaughtered the interstitial emphysema and pulmonary edema have usually regressed, but all of the lung tissue is rubbery as a result of severe, diffuse alveolar epithelial hyperplasia.

The pathogenesis of ABPE has been studied and evidence supports the view that ABPE is caused by ruminal production of 3-methylindole (3MI) from ingested L-tryptophan (TRP) found in herbage. The 3MI is absorbed into the bloodstream from the rumen and metabolized by a mixed function oxidase (MFO) system to produce pneumotoxicity (Figure 1). Much investigation has gone into the prevention of this disease. Two sites of intervention are possible: 1) inhibiting 3MI metabolism by
L-tryptophan ingestion in pasture \(\rightarrow\) metabolism in rumen to indoleacetic acid (IAA) \(\rightarrow\) decarboxylation of IAA by *Lactobacillus* spp. to produce \(\rightarrow\) 3-methylindole (3MI)

| Pulmonary lesions, congestion, edema, hyaline membranes, interstitial emphysema, alveolar epithelial hyperplasia, bronchiolar epithelial hyperplasia | \(\rightarrow\) destruction of Type I pneumocytes and nonciliated bronchial secretory cells | 3MI in blood \(\rightarrow\) metabolism by MFO system | Urinary metabolites largely oxindoles |

Figure 1

The MFO system and 2) manipulating the rumen flora to prevent 3MI production (Figure 1).

Pretreatment with piperonyl butoxide, an inhibitor of the MFO system, prevents 3MI-induced respiratory disease in goats\(^{10}\) and in cattle (unpublished results). However, general understanding of the total animal effects of MFO inhibitors is limited and at the present time it seems unlikely that dosing breeding stock on a regular basis with the necessary large amounts of inhibitor would prove to be practical or readily gain approval from regulatory agencies.

The more practical area for prevention of ABPE appears to be in altering the ruminal metabolism to lower or inhibit 3MI production. According to Hammond et al.\(^{10}\), 27 compounds were tested for their ability to decrease the conversion of TRP to 3MI. These compounds included deamination and methane inhibitors, decarboxylase inhibitors, ionophores, and antibiotics. In *in vitro* studies, desoxysalaminomycin, X-206-Na, chloral hydrate, nigericin, lasalocid, monensin, narasin, and salinomycin all reduced 3MI production greater than 80% at five micrograms per milliliter without reducing total volatile fatty acid production\(^{10}\).

Further studies were done *in vivo* to determine if monensin and lasalocid would effectively reduce ruminal conversion of TRP to 3MI\(^{10}\). Results of this experiment concluded that monensin effectively inhibited TRP-induced ABPE. Lasalocid was questionable. It seems that monensin is an effective preventative of ABPE but Food and Drug Administration regulations prohibit its use in breeding stock (except on an experimental basis). Treatment of ABPE consists of removing the animals from the offending pasture and symptomatic treatment with the drugs discussed previously for treatment of AIP.

2. Diffuse fibrosing alveolitis. Diffuse fibrosing alveolitis (DFA) is a disease which usually affects cattle over six years of age and is encountered in both beef and dairy breeds. The history is usually of chronic progressive pulmonary disease ranging from a few weeks to two years in duration. Typically the animals are thin but bright with a good appetite. Coughing is always present. Tachypnea and marked hyperpnea are seen even at rest. On auscultation, loud whistling sounds are frequently heard all over both lung fields and crackles may be heard cranioventrally\(^{21}\).

The lungs are very pale, almost white, very firm to cut, and extremely heavy. There is diffuse involvement of all lobes of both lungs. Most lobules are white and fleshy but some may be grey-red and collapsed. Very firm white or yellow lobules are often seen due to severe fibrosis, cellular infiltration of interalveolar septa, and alveolar epithelial hyperplasia\(^{21}\).

Microscopically, the most striking changes are in the respiratory acini where interalveolar septa are greatly thickened by fibrosis and cellular infiltration of mature plasma cells, lymphocytes, mast cells, and interstitial cells. There is widespread distortion and obliteration of alveolar spaces. Alveolar spaces are filled by large mononuclear cells and there is diffuse alveolar epithelial hyperplasia, the alveoli being lined by cuboidal type II pneumocytes. Occasionally the alveolar epithelium is replaced by tall columnar ciliated cells or by a mixture of ciliated and mucus-secreting cells and in the latter instance the alveoli are often filled with mucus. Bronchitis, bronchiolitis, and hypertrophy of the tracheobronchial mucus-secreting apparatus are apparent in many animals and leukocytes are common in the tracheobronchial epithelium\(^{21}\).
The etiology of DFA is unknown. At least 50% of the animals have precipitins to *M. faeni* so DFA may be a result of chronic farmer's lung. In support of this is the fact that many animals with DFA are from farms which have a farmer's lung problem. However, many animals which have clinical and pathologic features of DFA have no detectable precipitins to *M. faeni* indicating other etiologic factors may be involved.

3. Atypical interstitial pneumonia of immature cattle. AIP has been reported in immature cattle. In most of the documented cases, the calves have been on corn silage, moldy hay, or some other forage. The calves have usually been weaned four to five weeks before they develop signs of the disease. The affected calves are alert and there is little or no ocular or nasal discharge. Temperatures usually range from 38.9–40.0°C (102–104°F). Morbidity is high and almost all calves in the herd have dyspnea, particularly after exercise.

Grossly, the lungs do not collapse upon opening the thoracic cavity and they are edematous and emphysematous. Lobular demarcation is accentuated due to generalized edema. Froth is present in the trachea and bronchi. The lungs are firmer than normal. If complicating bacterial pneumonia is present it is usually cranioventrally with *Pasteurella* spp. most often isolated.

Microscopically there is edema and emphysema. The edema may be intraalveolar or interstitial. Eosinophils are generally present. Neutrophils often occur in focal aggregations in alveolar spaces or they may be diffusely scattered. Atelectasis is commonly seen adjacent to emphysematous areas. Hemorrhage may be present. If the disease has progressed, there is proliferation and swelling of alveolar epithelium. Hyalin membranes are commonly observed.

Treatment includes removal from the offending feed for 10 to 14 days followed by gradually bringing the calves back on feed. There is usually noticeable improvement in four to seven days. Recurrence usually does not occur. Symptomatic treatment mentioned before has been advocated.

Prevention of this disease has not been explored but monensin may be helpful since the pathogenesis of this syndrome may involve tryptophan.

C. Parasitic diseases

1. *Dictyocaulus viviparus*. Parasitic bronchitis is a familiar and common problem of immature grazing cattle. Any age of animal can be affected especially if kept free of the disease when younger. This situation may arise when groups of cattle are moved from regions where parasitic bronchitis is not a problem to areas where the disease is endemic, or when animals that have only grazed clean pastures in an endemic area are suddenly exposed to pastures contaminated by other cattle. The main signs are dramatic drop in milk yield and widespread and frequent coughing. In individuals, the chief signs are frequent coughing, marked tachypnea, and dullness together with crackles which are often audible over much of the caudal lung fields. *Dictyocaulus viviparus* larvae are present in the feces.

At necropsy, many adult lungworms are found in the airways and there is extensive consolidation, particularly of segments in the ventral parts of the caudal lung lobes. There is severe bronchitis and bronchiolitis and aspirated eggs and larvae may be found in the alveolar spaces. Interstitial emphysema, pulmonary edema, hyaline membranes, alveolar epithelial hyperplasia, and pulmonary eosinophilia may also be present.

Treatment is worming the animals plus any symptomatic treatment deemed necessary. Prevention and control is by periodic worming and vaccines.

2. *Ascaris lumbricoide*. Acute AIP caused by aberrant *A. lumbricide* suus larvae has been diagnosed in cattle. Usually it is associated with the movement of cattle into a lot or onto a pasture heavily contaminated with *A. lumbricide* larvae.

Clinical signs of this disease are depression, anorexia, coughing, elevated temperature, increased heart and respiratory rates, dyspnea, and forced expiratory grunts. Auscultation reveals increased vesicular murmurs and depressed rumen function.

Macroscopically, increased firmness is found in the apical, cardiac, and cranioventral one-third of the diaphragmatic lobes. Alveolar and interstitial emphysema are present in the remaining parts of the lung. The pleural surface and cut surface are mottled and yellow exudate oozes from the cut surface. The trachea and bronchi are filled with

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a red frothy exudate. Moderate generalized hyperemia is also present. Nematode larvae should also be observed on the cut surface.

Microscopically, *A. lumbricoides* larvae are sometimes found in large numbers in the bronchioles. On other occasions it is necessary to place ground-up lung in a cloth bag, place the bag in a funnel of warm water, and let the larvae migrate out of the bag to the bottom of the funnel. In acute cases extensive hemorrhages, neutrophils, other leukocytes, along with necrosis of bronchiolar epithelium and of alveoli are seen. Chronically, lymphocytes, plasmocytes, eosinophils, destruction of bronchial epithelium, and localized bronchial proliferations are seen.

Treatment of this disease is removal of the animals from the contaminated pasture and symptomatic drug therapy.

D. Plant poisoning

1. *Ipomoea batatas* (sweet potato) AIP has been reported in cattle fed moldy sweet potatoes. The condition is believed to be caused by several abnormal metabolites produced by the fungus *Fusarium solani* when it infects sweet potatoes. Among these metabolites are ipomeamarone, ipomeamaranol, and 4-ipomeanol. Sweet potato broth medium infected with *F. solani* has been used to produce respiratory disease in cattle.

Macroscopically, the lungs are wet, firm, and do not collapse upon opening the thorax. Small hemorrhages are found throughout the lung and there is severe interstitial and interlobular emphysema.

Microscopically, edema, alveolar and interstitial emphysema, alveolar epithelial proliferation, and hyaline membranes are seen.

Supportive treatment as mentioned previously is indicated as well as removing the contaminated sweet potatoes. Prevention is, of course, not feeding moldy sweet potatoes.

2. *Zieria arborescens* (stinkwood). In Tasmania, stinkwood has been thought to cause a fatal acute respiratory syndrome in cattle. The cattle eat the leaves of the stinkwood plant and in about two weeks they die of AIP. The toxic compound has not been identified but some reports indicate an oil in the leaves may be the cause.

Lesions seen are interstitial emphysema, pulmonary edema, hyaline membranes, and alveolar hyperplasia.

Supportive treatment can be used with removal of the cattle from the stinkwood.

Treatment and prevention is the same as for *Ipomoea batatas*.

4. *Bassicae*. Acute respiratory disease has been reported in Canada and England in cattle feeding on rape or kale. It is thought that these incidents are identical to fog fever/ABPE but it is possible that specific toxic factors are involved and not yet identified.

5. Other plants. Hyslop has reported AIP in cattle feeding on the sedge *Carex rostrata* and red clover contaminated by the fungus *Rhizoctonia leguminicola*, however little information is available.

E. Exposure to irritant gases and fumes

1. Nitrogen dioxide. “Silo-filler’s” disease of man is caused by inhalation of nitrogen dioxide. Nitrogen dioxide has also been thought to cause AIP in cattle. From work done by Cutlip, it seems that nitrogen dioxide is probably not a significant etiology of AIP. It has been suggested that nitrates in plant feedstuffs could release nitrogen dioxide gas during fermentation in the rumen and that eructation of this gas and subsequent inhalation would cause the lesions of AIP. Cattle forced to inhale nitrogen dioxide developed clinical signs of severe dyspnea and lesions of pulmonary edema and emphysema, bronchial, bronchiolar, and alveolar epithelial hyperplasia, and general hyperemia. However, other changes not characteristic of AIP developed. Infarcts and massive fibrin deposition were found in the lungs, necrosis of the proximal convoluted tubules and infarcts were found in the kidneys, as well as other vascular changes which have not been reported in field cases of AIP. These differences in lesions plus an unsuccessful attempt to induce AIP in cattle by ruminal insufflation with nitrogen dioxide.

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suggest that this gas is probably not involved in the pathogenesis of AIP. Furthermore, nitrogen dioxide gas could not be demonstrated in the rumen gas of cattle subjected to change from dry to succulent pastures or in animals affected by or recovering from AIP.

2. Smog. Acute respiratory distress and deaths were noted in cattle in London in 1952 and 1963 at the Smithfield Fatstock Show. At the time of the exhibitions there was dense atmospheric smog. It received considerable newspaper coverage but few facts were recorded or found. At this time there appears to be no correlation between smog and AIP.

3. Zinc oxide. AIP can be seen in cattle exposed to fumes of zinc oxide derived from oxyacetylene cutting and arc welding of galvanized pipe. The problem is most likely to occur in poorly ventilated buildings during remodeling of the building or in an attached shop to the livestock building where welding is being done.

Clinical signs reported are head extension, mouth-breathing, marked expiratory grunts, and subcutaneous emphysema. Congestion and hemorrhage in the trachea, interstitial emphysema, and interlobular and pulmonary edema are found grossly. Microscopically there are hemorrhages in the tracheal submucosa, diffuse congestion in all of the lung, and emphysema in the subpleural and interlobular septae. Alveolar changes vary from complete atelectasis to emphysema. Proteinaceous fluid, macrophages, and hemorrhage are present in some of the atelectatic areas as well as in some bronchioles. Numerous leukocytes may be found throughout the alveoli with eosinophils being very prominent in the interlobular edema.

4. Chlorine and hydrogen sulfide. MacDonald et al. reported a case of accidental chlorine gas poisoning of cattle. Emphysema, edema, and hyaline membranes were noted at necropsy but the evidence was not conclusive that the chlorine gas was the cause.

Hydrogen sulfide has been found in rumen gases of cattle placed on succulent pastures but any correlation between that and AIP has not been proven.

F. Experimentally induced syndromes

1. Systemic anaphylaxis. Acute systemic anaphylaxis begins two minutes after antigen injection in experimentally sensitized cattle. There are some well-known anaphylactic reactions to vaccines, drugs, and to larvae of Hypoderma lineatum and H. bovis.

Experimentally-induced anaphylaxis produces clinical signs of dyspnea, apnea, pulmonary hypertension, and systemic hypotension. At necropsy there is intraalveolar hemorrhage, pulmonary congestion, edema, and interstitial emphysema.

Over the years, there have been repeated suggestions that anaphylaxis or hypersensitivity could be the underlying cause of fog fever/ABPE, but there is little evidence to support the view that these factors are involved in the pasture-associated syndrome.

2. Bordetella pertussis infection. Intravenous infusion of B. pertussis suspension into five calves followed by marked hematomological changes and an acute respiratory distress syndrome. Severe diffuse alveolar epithelial hyperplasia with pulmonary edema, hyaline membranes, and interstitial emphysema was found in one of the five animals at necropsy. The pathogenesis of these lesions is unknown.

3. Administration of indolic compounds. AIP has been produced in cattle with the oral administration of D- or L-tryptophan and 5-methylindole. This is the apparent cause of fog fever/ABPE and is illustrated in Figure 1. Other information is included in the section of fog fever and acute bovine pulmonary emphysema.

As has been mentioned previously, there are many syndromes included under the term atypical interstitial pneumonia. Diagnosis is usually made at necropsy and to differentiate the syndromes, a complete history (feed, environment, clinical signs) is necessary. Treatment usually involves removal of the offending substance (usually feed or pasture) and symptomatic treatment with atropine, corticosteroids, antibiotics, antihistamines, diuretics, and epinephrine. Prevention of the various syndromes includes avoiding the various substances and in the case of TRP-induced ABPE, monensin may someday be used routinely for prophylaxis.

References


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