Pulmonary Emphysema in Newly Weaned Calves*

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Bovine pulmonary emphysema has been recognized in many countries for a number of years. There is confusion as to the cause and the nomenclature of the various diseases of cattle which have pulmonary emphysema as a major lesion. The term "pulmonary emphysema" is used as the name of a disease and as lesion identification, which adds to the confusion. The lung can respond to an irritant in only a few ways, which allows for lesions to appear similar even though there are different causes.

Perhaps all of the following terms are separate entities. It is also possible that more than one of the suspected etiologic agents could be involved in a specific case. We believe the etiology of pulmonary emphysema in newly weaned calves is different from others previously described.

Some of the names that have been given are: bovine pulmonary emphysema; acute bovine pulmonary emphysema, or ABPE; St. George disease; fog fever; atypical interstitial pneumonia, which is currently the most popular; pulmonary adenomatosis; silo filler's disease; cow asthma; hypersensitive pneumonitis; extrinsic allergic alveolitis; "Urner pneumonie"; bovine farmer's lung; acute alveolar emphysema and edema; panters; and lungers (1,2,6,8,10,11,13).

Suspected causes of this disease syndrome are almost as diverse as the terms that are used in its identification. *Micropolyspora faeni* has been considered to be the cause by researchers in Canada and Europe (13). Lungworms, *Dictyocaulus viviparus,* have been incriminated as causing pulmonary emphysema (2). Sclerotium, which was isolated from sweet potatoes being fed cattle by Peckham, et al., was shown to cause pulmonary emphysema (9). Seaton suggested that nitrogen dioxide could cause this disease (11). Later, Clark indicated it could be caused by pimelea (6). Dickinson, Carlson, and others at Washington State University, using more basic research, were able to reproduce the disease by oral or intraruminal doses of D,L-tryptophan or L-tryptophan (3,4,5). Intraperitoneal or intravenous injections did not produce pulmonary emphysema (4). Rumen microorganisms converted L-tryptophan and indole acetic acid to 3-methylindole *in vitro.* When 3-methylindole was given intravenously to cattle, pulmonary emphysema developed (5). Recently, Wellemans of the Netherlands has suggested the cause as maybe being due to respiratory syncytial virus (12).

Records from 1971 through 1977 show the number of cases of pulmonary emphysema in newly weaned calves (see Table 1). The laboratory cases are only a rough indicator of the total number of cases in the area. We think it is much more prevalent than these numbers indicate.

The gradual decline of cases, as demonstrated in Table 1, after 1972 is probably because many of the practicing veterinarians in the area have become proficient in the field diagnosis of the disease and specimens are not sent to the laboratory. The herd size of affected calves is in the general range of 150 to 4700 animals. The economic importance is great, but no dollar-per-head value has been established.

Table 1
North Platte Veterinary Science
Laboratory Cases of Pulmonary
Emphysema in Newly Weaned Calves

Year	Number of Cases
1971	12
1972	36
1973	34
1974	29
1975	11
1976	8
1977	27

Clinical Signs

Affected calves are alert. There is little or no ocular or nasal discharge. Their temperatures range from 102-104°F (39-40°C). If concurrent infectious pneumonia is present, the temperatures are higher. Morbidity is high and practically all of the calves in the herd have evidence of dyspnea, particularly after exercise. Some may die after forced exertion. Most of the cases that come to our laboratory are presented with a history of having been fed corn silage, but occasionally they have been fed moldy hay or some other type of forage. The calves have usually been weaned 4 to 5 weeks, occasionally longer, before they start to show signs of the disease. This type of

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pulmonary emphysema is often misdiagnosed as infectious shipping fever complex. Since both conditions usually occur in late fall and early winter, it is easy to understand why the entities are confused.

Gross Lesions

The lungs do not collapse upon opening the thoracic cavity. They are usually edematous and emphysematous. The edema is generalized and lobular demarcation is accentuated. Considerable froth is often present in the trachea and bronchi. The affected lungs are firmer than normal. Consolidating pneumonia may be associated with this, and if it is, it generally occurs in the antero-ventral lobes. *Pasteurella hemolytica* or *P. multocida* can often be isolated from lungs that have concurrent pneumonia. Lungs that are not pneumonic are generally bacteriologically negative. The mediastinal lymph nodes may be enlarged. Other organs are usually normal.

Microscopic Appearance

The most pronounced microscopic changes are edema and emphysema. (For an excellent review of the current concepts on the mechanisms of pulmonary edema, readers are referred to J.V. Hurley's article in the *Journal of Pathology,* Vol. 125, pps. 59-79.) The edema may be intra-alveolar and/or interstitial. Eosinophils are generally present in some part of the affected lung. Usually, they are scattered throughout all sections. They are often dispersed through the interstitial edema. On occasion, they are concentrated in perivascular tissues. Neutrophils often occur in focal aggregations in the alveolar spaces, or they may be diffusely scattered. It is believed that these are secondary to the earlier lung changes. Atelectasis is commonly observed adjacent to emphysematous areas. Hemorrhages may or may not be present. Theoretically, hemorrhage may occur after anoxia or rupture of the alveolar capillaries. The more of the latter, naturally, the more hemorrhage that is observed. If the disease has progressed, there is also proliferation and swelling of alveolar epithelium. Hyalin membranes on alveolar linings are commonly observed.

Suggested Treatment

We routinely recommend removal from silage or moldy hay for 10 days to two weeks and then suggest bringing the calves back on the original feed gradually. Experience has demonstrated that cattle which are removed from the associated ensilage or hay will have noticeable improvement within four to seven days after change of feed (sometimes as soon as two days). Recurrence of the condition has not occurred to our knowledge. Area veterinarians suggest the treatment of affected animals should include corticosteroids and antihistamines, b.i.d. When possible, it is advisable to provide treatment with a minimum of restraint during the first two or three days. We recommend crowding them with gates or some similar manner of restraint. If there is secondary infection, and there usually is, antibiotics can be used, either injectable or in the feed. Animals that are extremely dehydrated can usually survive probing and rehydration by the third day. Some veterinarians recommend the use of electrolytes in the drinking water as a part of the recovery program.

Prevention

Several methods of prevention have been suggested, including such practices as limited feeding of ensilage and mixing with other forage. Proponents of the nitrous oxide theory have suggested cooling the ensilage to ambient temperature so that cattle eating the ensilage are less likely to inspire gas from the forage.

Recently, Hammond, et al., reported ***in vitro*** **evidence that monensin would prevent bacteriologic conversion of tryptophan to 3-methylindole. Likewise, tryptophan-induced pulmonary emphysema in mature Hereford cows was inhibited by giving the monensin in gelatin capsules orally twice daily. Treatment was started one day before and continued through four days after the tryptophan dose (7).**

A few practitioners in our area report some success using monensin in the feed of a limited number of calves. Usage has not been extensive enough to expound on the field efficacy.

The following brief case histories are incorporated to provide a concept of the natural disease as observed in our area, the response to alteration of the feed, and treatments.

Case Histories

Case No. 1. Four dead, weaned steers and heifers were presented to the laboratory. The owner gave a history that he had been "backgrounding" these calves. Two of the animals came from a pen of 400+ being fed hay and a small amount of grain. These calves did not have pulmonary emphysema but did have infectious bovine rhinotracheitis. The other two calves were from a pen of approximately 400 that were being fed ensilage and grain. These had gross lesions of pulmonary emphysema and infectious bovine rhinotracheitis.

The affected cattle were vaccinated with infectious bovine rhinotracheitis vaccine, and the cattle being fed ensilage were changed to dry hay and grain. The owner reported a few months later that he believed that if he had made the feed change and vaccinated for IBR earlier, he could have saved 40 head of calves.

Case No. 2. Nearly 5,000 calves were weaned and placed in approximately 20 different pens, all receiving primarily corn ensilage as their diet. After approximately a month on this diet, at least 90% of these calves developed a dry cough and evidence of dyspnea but were generally alert. The approximately 20 calves that died had pulmonary emphysema. The diet of all cattle was changed to dry hay for 10 days. The veterinarian in charge reported that there was

marked improvement in these cattle four days after being removed from the ensilage diet.

Case No. 3. One hundred eighty weaned calves were fed a mixture of one-third corn and two-thirds oats with some molasses, protein, and aureomycin crumbles. This was fed for approximately two weeks after weaning, after which the calves were placed on ensilage. Approximately three weeks after being on ensilage, the owner noticed sick calves. The third day after this, he treated the entire herd with injectable antibiotics but most of the calves did not respond. The veterinarian referred one dead calf to the laboratory. We diagnosed pulmonary emphysema and recommended they remove them from the silage diet. The owner was extremely reluctant to do so, but because of the severe condition and long illness of the calves, he did. Their diet was changed to rolled corn and oats ration along with prairie hay and some alfalfa. There was dramatic response but there were a few cases of infectious pneumonia. The owner reported losing 28 out of 180, most of which were due to pulmonary emphysema and/or pneumonia.

Case No. 4. Approximately 800 steers were being backgrounded. Four hundred of these came from one ranch while 400 came from another. Dead animals were brought to the laboratory for laboratory diagnosis, whereupon pulmonary emphysema was diagnosed. The owner reported they were being fed primarily silage and that the involvement seemed to be calves from one herd. He stated that he could not take them off ensilage because of the lack of other feed. Deaths continued to occur periodically during the following few weeks, and there was not the dramatic response that we have seen in other cases where ensilage had been removed from the diet for a few days.

Discussion

When there is an overlap of a term used as the name of a disease and a gross lesion, there is confusion. Pulmonary emphysema is a prime example of this situation. The condition described herein does not conform totally to similar conditions previously described. For example, these calves are not confined in a barn as in the reports of *Micropolyspora faeni;* neither were they on pasture as was the case with lungworm infection. Also, lungworm infection is nearly nonexistent in central Nebraska.

Acute pulmonary emphysema occurs in central Nebraska when adult cattle are moved from dry grass pasture to new-growth grass in the lower areas. Calves are rarely affected at this time. Cases of what we believe to be atypical bovine interstitial pneumonia are occasionally observed in our laboratory. Morbidity is quite low in contrast to that found in pulmonary emphysema in newly weaned calves.

The cause of pulmonary emphysema in newly weaned calves can only be postulated at this time. Circumstantial evidence suggests a relationship to consumption of silage or moldy hay by the calves. Corn silage is more often a major part of the diet in our area. Calves on silage do not have the clinical signs until approximately 4 weeks after weaning and change of diet, so there may be some change in bacterial flora which could change the biochemical constituents of the rumen liquor. On the other hand, there may be some infectious cause such as syncytial virus. Thirdly, the cause may be a combination of biochemical toxin and infectious agent. Only controlled research will resolve the question of etiology. This paper is not to answer the question of etiology but to emphasize the clinical signs, lesions, relationship to diet and weaning, as well as the economic importance.

Addendum

Since this article was completed, an article entitled "A Reappraisal of Atypical Interstitial Pneumonia in Cattle" by Breeze, et al., was published in the November 1978 *Bovine Practitioner.* The following table taken from this article clarifies some of the misunderstanding of atypical interstitial pneumonia in cattle.

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* infusion
   3. Administration of indolic compounds

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Discussion

Question: Dr. Hibbs, have you noticed what type of temperature affects this condition? We found that when we had our first frost, and our first cold days and cold nights, this all quit. If we had warm days and cold nights, we really had a mess. Have you associated any of that?

Dr. Hibbs: I would have to say "no." As a matter of fact this case occurred on a really cold day. I don't remember what the temperature was but I know that I put on a hood and some things, being an indoors person. In the fall when we start bringing the cattle in and we have those warm days and cold nights and it warms up the next day, we have a mess and then if we have constant cold, it is all over. There are probably more of you in here that work more with dairy calves than I do that could offer better advice because I have not worked with dairy calves for 13 years. Is there a dairy practitioner in here?

Question: Do you have a program of vaccination for dairy calves?

Answer: That is what you call really being put on the spot. I guess I can bluff like the rest of you. I think the situation with dairy calves varies just a little bit but in those herds where we feel we have a problem with BVD we very often will use BVD vaccine as early as 30 days of age and then come back and do it again when we calfhood vaccinate for bangs, which we like to do at around 6 months. I think you have to key it to your own program and you may want to do some serologies and several other things to get things worked around. Many of those calves that are vaccinated around 30 days don't produce any immunity and I think that in those that do it is probably fairly short lived but that's a seat-of-the-pants, barnyard-type observation and it might not hold water in your situation.

Question: Can you vaccinate dairy cattle in the face of an outbreak?

Answer: As far as your calves are concerned, I think if you are in a dairy herd and you have a continuous on-going calf raising situation and IBR running through them, you have to go ahead. I think you have to break that thing off somewhere. I'm convinced that there are a few of them for which BVD vaccine is probably going to be pretty rough, but you just have to go through the sweat. As far as your cows are concerned, I would not be caught vaccinating pregnant cows with modified live vaccine. For that reason we like to vaccinate heifers before they are bred at least once and in problem herds maybe twice. If it is really tough we will vaccinate cows during the time they are open.

Question: You mentioned that you had a 50% response with BVD vaccine, was that IM or oral vaccination?

Answer: That was with the IM. We were unable to demonstrate any response with the oral method.

Question: What is a probiotic?

Dr. Davis: The probiotics that we used in our trials were composed primarily of lactobacillus acidophilus organisms. These were used in the bolus form and also in feed supplements. There were some other organisms in there in lesser numbers but the work that has been done has been primarily with lactobacillus acidophilus organisms.

What is a probiotic? Compared to antibiotics, probiotics are just the opposite. That is, the probiotic is intended to change the microbial population in the intestine so that you'll have more beneficial microorganisms such as the lactobacillus organisms and less harmful bacteria. You are trying to establish a better microrganism population in the intestine.

Question: Is there a contraindication for a vaccination program for BVD in the face of an outbreak?

Dr. Caley: We have concluded that, working with our own, if these animals come in where they are showing signs of BVD and we go ahead and accept them, we vaccinate them within 24 hours. Frankly, we'd admit that you take a little hide with some of the hair, but that is better than dragging it out for another 60 days, so we go ahead and vaccinate, yes.

Question: Do you have any experience with high dosages of vitamin C intravenously in the treatment of pulmonary adenomatosis?

Dr. Hibbs: I don't personally have any experience. I know of some people who have used it. The people in our area have just about quit.

Question: Do you use expectorants in the drinking water?

Dr. Hibbs: You may not like what I am going to tell you about expectorants in the water for a condition such as this and that is that they are probably a waste of time! They probably make the owner feel better and you feel better, but as far as the animal is concerned I would question the efficacy of them. I just came back not too long ago from a respiratory symposium at the University of Illinois and they had some pretty high-powered people there. But, anyway, there were a number of British, Scottish, Canadians and M.D.'s there. I gathered, particularly from visiting with an M.D. clinician who does research with respiratory conditions in man that inhalants, for example, are not of very much value. Expectorants sometimes may be a little bit supportive, but with the whole process, the cough process, the discharge process, you want to be very careful that you are not interfering with the natural processes that should be taking place.

Question: What is the dosage for rumensin?

Answer: You must remember that I am not a practitioner so I'm not giving dosages out. The dosage I think that was used on the adult, as a matter of fact, I am not openly recommending. I'm saying that there might be a place for it and that time will tell. It's like some other new things that come along and we hedge, so I'm hedging, but the dosages that I recall on the cow in the literature was 100 milligrams twice a day.

Question: What about diuretics?

Answer: I don't know of anybody that uses them. I suppose they would be indicated on an individual basis. You also have to recognize that when a clinician starts to work with these animals, he has to be a little careful, because they will drop on you. I assume that some people have tried it but again I'd have to back off.

Question: Do they respond if you see them early? I seem to think so.

Answer: There is no pneumonia to speak of there, very slight pneumonia and we'll see that in a lot of animals that die from some other cause. The fact that they seem to respond that well, there must be a lot of those that have a temporary emphysema reaction because if one would base the prognosis on the lesions that we see, he would have been wrong because you would think that they wouldn't respond to any type of treatment, but they do.

The question was perhaps that edema may be the primary initial lesion followed by emphysema and I can't answer that. It very well could be. Incidentally, if you are interested in the physiology of edema, I have a reference for you that is about 6-8 pages. It's an excellent reference, too. It is by J.V. Hurley in General Pathology, Vol. 125, pages 59-79, and it is an excellent article. I think probably you are correct. If you look closely at the histology you would say there is a lot of non-functional lung even though it may appear grossly not to be too badly affected. We don't see the early case. There just has not been anybody at this point that has been willing to give us a calf to post that is initially starting, and you would still have to sample a large number of them. I'd say that there is very little functional lung. I'm convinced that they actually die from asphyxiation, or lack of oxygen.

Question: You mentioned that a viral agent is involved?

Answer: Oh, yes, I mentioned that a viral agent may be involved. There are several people working with this right now and hopefully it will resolve that question.

Question: What about testing calves?

Answer: I can tell you what our experience has been on testing calves to start and indications that they are negative and then intermingle those with vaccinated ones. In some instances the titer established in the non-vaccinates exceeds our best titer that we vaccinated and we've not checked out every vaccine, but I can assure you that in most of them that we have had anything to do with, the incidence will eventually establish a titer.

Question: But can you say that you don't maybe get an abortion once in a while? And secondly, we perhaps then are talking about one that gives us a titer that does not result in an abortion in a dairy cow. But I would not completely eliminate the possibility of what I've seen.

Answer: BVD abortion is very difficult to diagnose. The time of exposure has a bearing on whether the cow will abort. A lot of cows are carrying a protective titer that have not aborted. We have two herds on record; one was vaccinated in the face of an outbreak and they continued to die. Maybe they had something else.

Question: Have you explored the possibility of tryptophan-indole mechanism in your silage?

Dr. Hibbs: No, we have not, but we did apply for some funds to support a study but we did not get any–but we should study this.