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Presumptive hypersensitivity pneumonitis in dairy cattle: a case report

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Abstract
Hypersensitivity pneumonitis is a pathological condition caused by the continuous inhalation of various antigenic compounds, leading to an exacerbated immune response and subsequent lung injury. Although this condition has been reported in several animal species, it has not yet been documented in cattle in South America. This report describes a case that occurred in 2020 on a dairy farm operating under a dry-lot system with soil bedding, located in Buenos Aires province, Argentina. An increased frequency of respiratory cases was observed among lactating cows, occasionally resulting in death. Affected animals exhibited dyspnea, adynamia, orthopneic position, ptyalism, and normal rectal temperature. Postmortem examination of one affected cow revealed severe generalized pulmonary and mediastinal emphysema. Microscopically, mild to moderate thickening of the alveolar septa was observed, accompanied by increased infiltration of lymphocytes, macrophages, and neutrophils. The alveoli contained abundant multinucleated giant cells distributed multifocally, along with occasional macrophages and neutrophils, and multifocal areas of intra-alveolar edema. The interlobular septa were expanded due to lymphatic distension, edema, and a mild diffuse infiltration of macrophages and lymphocytes. Bacteriological cultures were negative. Although parainfluenza-3 virus was isolated from the lung tissue, immunohistochemistry (IHC) results were negative. The epidemiological context, pathological findings, and clinical signs were consistent with hypersensitivity pneumonitis, although its specific etiology could not be determined. Dairy production systems that utilize soil bedding may predispose animals to this condition, even though potential allergens were not assessed in this case. Nonetheless, this clinical and pathological presentation should be considered as a differential diagnosis for respiratory disease under similar production conditions.

Key words: pneumonia, bovine, hypersensitivity, multinucleated giant cells.

Presunta neumonía por hipersensibilidad en ganado lechero

Resumen. La neumonitis por hipersensibilidad es una condición patológica que ocurre debido a la inhalación continua de diferentes compuestos antigénicos y una respuesta inmune exacerbada causando lesión pulmonar. Aunque esta condición ha sido reportada en diferentes especies animales, no ha sido reportada en bovinos de Sudamérica. Este trabajo describe un caso registrado en un tambo con corral seco con un sistema de cama de suelo en la provincia de Buenos Aires en 2020. Un aumento del número de casos de enfermedad respiratoria fue detectado en vacas lecheras con ocasional muerte. Los animales afectados mostraron disnea, adinamia, posición ortopneica, ptialismo y temperatura rectal normal. Durante el examen post mortem de una de las vacas afectadas, se observó un enfisema generalizado pulmonar y mediastínico severo. Histopatológicamente, se observó un engrosamiento leve a moderado de los septos alveolares con aumento de la celularidad (linfocitos, macrófagos y neutrófilos). Los alvéolos estaban llenos de abundantes células gigantes multinucleadas distribuidas multifocalmente, con macrófagos y neutrófilos ocasionales y parches multifocales de edema intraalveolar. Los septos interlobulillares se expandieron por distensión linfática, edema y leve infiltrado difuso de macrófagos y linfocitos. Los cultivos bacteriológicos fueron negativos. Aunque se aisló el virus de la parainfluenza-3 del pulmón, la inmunohistoquímica fue negativa. Las condiciones epidemiológicas, los hallazgos patológicos y los signos clínicos fueron compatibles con neumonitis por hipersensibilidad, aunque lamentablemente no se pudo determinar su origen. Los sistemas lecheros con la inclusión de camas de tierra probablemente estén predispuestos a esta condición, aunque no se evaluaron alérgenos probables. No obstante, esta presentación clínica y patológica debe incluirse como un diagnóstico diferencial de la enfermedad respiratoria en condiciones de producción similares.

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Palabras clave: neumonía, bovino, hipersensibilidad, células gigantes multinucleadas.

INTRODUCTION

Bovine respiratory disease (BRD) has a diverse etiopathogenesis, with variable response to treatment and prevention tools. BRD is responsible of severe economic losses in both beef and dairy cattle (Panciera and Confer 2010). BDRs are often closely related to the environment, as the respiratory system is continuously exposed to the inhalation of potentially pathogenic material (Wiseman 1978). In general, allergic or hypersensitivity responses require immunologic sensitization to one or more specific antigens or allergens, whereas pathologic responses occur after a variable number of repeated encounters with the allergens (Van Metre 1997). Bovine hypersensitivity pneumonitis is rarely diagnosed and its etiopathogenesis has not been completely elucidated, although it is usually associated with the inhalation of organic dusts (Breeze 1985). This disease shares many similarities with its human counterpart known as "farmer's lung" and is associated with exposure of dust from moldy hay, grain or other plant products containing spores of thermophilic actinomycetes such as *Saccharopolyspora rectivirgula* and *Thermoactinomyces vulgaris* (Wilkie 1978, Wiseman 1978, Breeze 1985, Van Metre 1997, Bourke et al. 2001, Carvallo and Stevenson 2022). However, cases have been reported mainly in adult cattle with mold-contaminated bedding materials, due to continuous and prolonged contact with contaminated material causing an acute or chronic clinical disease resembling "farmer's lung" disease in humans (Kawai et al. 1973, Sharma 1989, Cebollero et al. 2005, Stöber 2005, Spagnolo et al. 2015). In addition, environmental dust (less than 4 μm) carrying fine particles of manure containing bacterial endotoxins can reach the alveolar spaces and generate a hypersensitivity reaction (McVean et al. 1986).

The disease is clinically characterized by anorexia, dyspnea of variable intensity, cough, and decreased production (Van Metre 1997, Stöber 2005, Carvallo and Stevenson 2022). The pathological findings are characterized as a type III or type IV hypersensitivity process (Kawai et al. 1973, Breeze 1985, Stöber 2005, Caswell et al. 2015, Carvallo and Stevenson 2022). Gross findings include acute cases are small subpleural gray areas, pulmonary edema, emphysema, subserous petechial hemorrhages, among others (Wilkie 1978, Breeze 1985, Van Metre 1997, Carvallo and Stevenson 2022). Microscopic findings are characterized by multinucleated cells, lymphoplasmacytic and macrophage interalveolar infiltration and/or obliterating bronchiolitis (Breeze 1985, Van Metre 1997, Stöber 2005, Carvallo and Stevenson 2022). Although respiratory disease is usually associated with infectious causes in livestock farming, little is known about the potential impact of non-infectious pneumonias, such as hypersensitivity pneumonitis. This paper describes an increased prevalence of respiratory disease in a dairy herd in Argentina, possibly associated with hypersensitivity pneumonitis.

MATERIALS AND METHODS

Sporadic respiratory disease occurred during 2020 in a dairy farm with 1060 Holstein cows, located in Rivadavia, Buenos Aires province, Argentina (35°29'02.9"S 63°13'44.1"W). The animals were divided into seven pens according to their productive stage, kept in dry pens with beds made with soil from the same pen. The cows were vaccinated with a commercial vaccine against BRD [bovine herpesvirus type 1 and 5 (BoHV-1, BoHV-5), bovine viral diarrhea virus (BVDV) type 1 and 2, bovine parainfluenza virus-3 (*Respirovirus*; BPIV-3), *Pasteurella multocida*, *Mannheimia haemolytica* and *Histophilus somni*] and neonatal diarrhea (bovine rotavirus serotypes 6 and 10 and *Escherichia coli* J5) 15 and 45 days after drying. At the time of calving, they receive another dose of neonatal diarrhea vaccine. Cows received a total mixed ration (TMR), which varied in its composition according to lactation stage (fresh, high, medium, and low production cows) composed of corn silage, wheat silage, alfalfa silage, alfalfa hay, soybean meal, whole cottonseed, and soybean hulls. The high-performance cows also received pass-through protein feed (Aminmax®, Gepsa, Argentina), protected fat (Megalac®, Church & Dwight Co., USA), bicarbonate and protected methionine (Smartamine®, Adisseo, Brazil). Cows were given and intraruminal bolus of monensin at the beginning of the close-up period, 21 days before the expected date of calving, regardless of the cow type (production group, days in milk, milk yield, parity, or reproductive status).

The presence of diseases diagnosed in milking cows with different days in milk, milk yield and parity, of different pens during the year was recorded. During the previous year, a new program (DeLaval DelPro® and the partner program DeLaval Cow Health index, Tumba, Sweden) was implemented in the dairy farm, which allowed the early individual identification of cows with a sudden individual reduction in daily individual milk yield. Each identified cow was then clinically evaluated and treated by in-house practitioners with oxytetracycline.

A 5-year-old cow in the second stage of lactation with sudden milk loss was clinically analyzed confirming similar signs to the previous affected cows. Cow was treated with flunixin meglumine and no clinical improvement was noted. Therefore, the affected cow was euthanized 24 hours after clinical recognition. Sampling procedures and euthanized were performed according with the standard operational procedures recommended by the Institutional Committee for the Care and Use of Experimental Animals at the National Institute of Agricultural Technology (INTA CeRBAS), Argentina. Blood samples were collected in pairs by jugular vein puncture with EDTA. Blood count was performed using a hematology analyzer (Mindray BC-2300) to measure hematocrit, erythrocytes, leukocytes, hemoglobin, and relative and absolute leukocyte formula. Tissue samples were collected from the lung, heart, liver, kidney, adrenal gland, tongue, mediastinal lymph node, brain, cerebellum, spinal cord, rumen, reticulum, abomasum, spleen, thymus, small intestine and large intestine, and fixed in a 10% buffered formalin solution. Samples were subsequently processed and stained with hematoxylin and eosin (H&E) for histological examination. Fixed lung tissue was processed by immunohistochemistry (IHC) for the detection of BPIV-3 and bovine respiratory syncytial virus (BRSV), according to the standard operational protocols of the Histopathology Lab of INTA Balcarce.

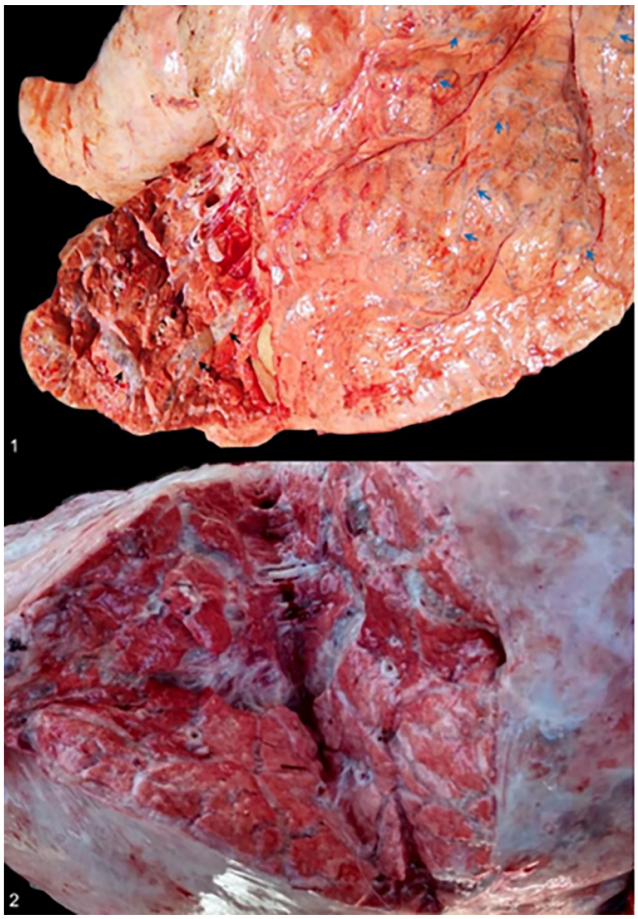
Bacteriological and virological tests were performed on lung samples. For bacteriological diagnosis, lung samples were cultured in Columbia Blood Agar medium with 7% bovine blood added and incubated at 37 °C for 24-48 hours in an atmosphere of 5-10% CO_2. For virus isolation, lung samples were inoculated into MDBK cell cultures, performed 3 blind passages, and observed microscopically to detect the presence of cytopathic effect. Direct immunofluorescence (DIF) was used with monoclonal antibodies against BPIV-3, BoHV-1, BVDV and (BRSV), following the standard operational protocols of the Virology Lab of INTA Balcarce.

RESULTS

The previous historical cumulative annual clinical prevalence of respiratory disease in dairy cows was 5%. However, the new implementation of detection programs on the dairy farm allowed the early identification of individual cows with a sudden decrease in daily milk production. As a result, an increase in the prevalence of respiratory disease was observed during this year, reaching a morbidity of 12.7% (135/1060) with no clear seasonal pattern associated with climatic conditions. Affected cows showed dyspnea or abnormal breath sounds upon auscultation, and some pyrexia. Most of the affected animals (105 out of 135) were treated with antibiotics (oxytetracycline) and nonsteroidal anti-inflammatory drugs (NSAIDs; flunixin meglumine or meloxicam). Twenty-five out of the 105 cows responded favorably. Fifty out of the 105 treated cows were culled due to lack of recovery after treatment. The other 30 treated cows (out of the 105) did not return to their initial milk production (an approximate loss of 10 liters per cow) and body condition. Another 30 affected cows (out of the 135) cows were treated only with NSAIDs only, and a milk yield recovery was observed. Cases occurred throughout the year, similarly in both primiparous and multiparous cows. Clinical disease was diagnosed in cows at different stages of lactation: 0 to 30 (13%), 31 to 120 (39%), 121 to 150 (31%) and >251 (17%) days of lactation.

A sudden decrease in milk yield (50% of her previous milk yield) was detected in a 5 years-old cow in the second stage of lactation (21 to 120 days of lactation). Clinical examination revealed dyspnea, orthopneic position, tachypnoea (40 respiratory movements per minute), ptyalism, tachycardia (96 beats per minute), and normal rectal temperature (38.6 °C) was registered. The blood count of the affected cow was normal.

On necropsy, the lungs were markedly distended occupying almost the entire thoracic cavity (they did not collapse on opening the chest). Generalized multifocal emphysema was observed in both lungs (figures 1 and 2), more evident in the interlobular septa. A small area of consolidation (5%) was found in the right pulmonary cranioventral lobe. Emphysema was also observed in the mediastinum and perinephric fat.



Figures 1-2. Gross pathological findings in the lung of a necropsied Holstein cow described in the manuscript. (1) Generalized interlobular pulmonary emphysema affecting right basal lobe. Emphysema was evident from the surface (blue arrows) and in the lung parenchyma (black arrows). (2) Interlobular septal emphysema in the middle pulmonary lobe.

Microscopically, the main lesions were in lungs characterized by diffuse, mild to moderate, alveolar septal thickening mainly peribronchiolar with increased cellularity (lymphocytes, macrophages and neutrophils). Interlobular septa were slightly distended by lymphatic dilatation, edema, and diffuse infiltrate of macrophages and fewer lymphocytes. Occasionally in the bronchi, subepithelial infiltration by macrophages and few neutrophils were observed, with extravasation into the lumen intermixed with edema and abundant neutrophils. Rarely, scarce peribronchiolar macrophages are seen in bronchioles. Multifocally, extensive areas of alveoli were filled with abundant multinucleated giant cells (Figure 3), occasional macrophages, neutrophils, and rarely eosinophils. Occasional pneumocyte type II proliferation was observed covering alveoli. Also, multifocal alveolar edema and rarely fibrin was noted. Multiple areas of alveolar, interlobular and subpleural emphysema, surrounded by atelectatic areas were present. Small and medium-sized blood vessels had perivascular macrophages and fewer lymphocytes. Other minor findings were histiocytic perivascular nephritis and diffuse splenic hemosiderosis.

No pathogenic bacteria have been isolated. Viral detection of BoHV-1, BVDV and BRSV was negative. BPIV-3 was isolated in the lung sample. Fixed lung tissue was processed by immunohistochemistry (IHC) for the detection of BPIV-3 and BRSV antigen and was negative.

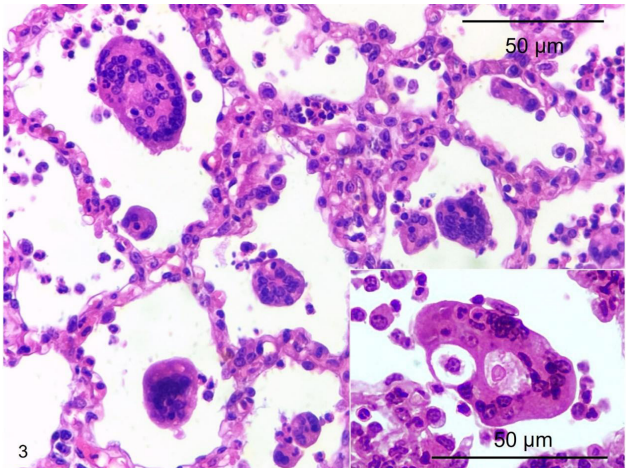


Figure 3. Histopathological findings in the lung of a necropsied Holstein cow described in the manuscript. Mild thickening of alveolar septa by lymphocytes, macrophages and neutrophils with abundant multinucleated giant cells, occasional neutrophils, and fewer macrophages in alveoli of the right basal lobe. Inset: detailed image of multinucleated giant cell. H&E.

DISCUSSION

This work describes an increase in the frequency of respiratory cases in milking cows occurred in 2020 on a dairy farm operating under a dry-lot system in Buenos Aires province. Respiratory disease was characterized by dyspnea, adynamia, orthopneic position, ptyalism, and normal rectal temperature. During postmortem examination severe generalized pulmonary and mediastinal emphysema was observed. Microscopically, pulmonary lesions were characterized by alveolar septal thickening, and abundant intra alveolar multinucleated giant cells.

Although a higher morbidity rate was detected in 2020 compared to previous years (12.73% vs. 5%), the overall mortality rate of the dairy farm did not increase compared to the previous year (≈ 7.5%). The increased detection of affected cows was assumed to be related to the inclusion of the dairy farm management platform. The initial decrease in milk yield reduction would allow an early detection of an affected cow, followed by a clinical examination and recognition of respiratory signs. This clinical condition did not always result in the death of the affected animals, as early detection and the treatment resulted in a good prognosis. Compared to previous years, there were no changes in the management, husbandry, and nutrition of dairy cows. However, climatic conditions were generally drier and windier than previous years, according to the farmer (data not showed). The clinical, gross, and histopathologic findings in the affected cow were compatible with previous reports of hypersensitivity pneumonitis in cattle (Wilkie 1976, 1978, Fink 1984, Breeze 1985, Bourke et al. 2001, Stöber 2005, Caswell et al. 2015).

Although the BPIV-3 is a common respiratory pathogen and it was isolated in the lung of the autopsied cow, no intralesional detection was observed by IHC. This aspect is especially important since the viral agent would not be associated with the lesions observed. Furthermore, the negative IHC result for PI3 and BRSV highlights the finding of multinucleated giant cells, since the formation of syncytia associated with these viral agents would be ruled out. Adult cows can be carriers of BPIV-3 (Ellis 2010). In addition, BPIV-3 may have acted as a predisposing factor for respiratory disease. BPIV-3 preferentially affects naïve calves or fetuses (Ellis 2010, Macías-Rioseco et al. 2019). Although BPIV-3 could form syncytia in cell cultures (Ellis 2010) the most common histopathological finding during infection is necrosis of bronchial and bronchiolar ciliated and non-ciliated epithelial cells (Ellis 2010, Macías-Rioseco et al. 2019), not observed in the histopathological examination of the pulmonary tissues of this cow. Furthermore, the presence of interlobular emphysema and bullae has not been reported in experimental BPIV-3 infections (Ellis 2010). Although multinucleated giant cells could be confused with these sloughing and syncytial formations, their intra alveolar presence could indicate a subacute process of allergen exposure (Fink 1984). Therefore, the presence of multinucleated giant cells and septa expanded by mononuclear inflammatory infiltrate observed in the affected cow, would suggest a process of hypersensitivity pneumonitis (Fink 1984, Breeze 1985, Weber et al. 1993, Van Metre 1997, Stöber 2005, Caswell et al. 2015).

Hypersensitivity pneumonitis has a wide variety of possible causes, often predisposed by repeated inhalation of moldy hay dust, and can be acute or chronic affecting a housed herd (Stöber 2005). Type III hypersensitivity usually occurs due to the presence of *S. rectivirgula* spores or other small spore fungi that reach the alveoli and cause immune complex deposition (Wilkie 1976, Van Metre 1997, Stöber 2005). Exposure to the allergen of this bacterium during the winter months can lead to an increase in the number of cattle with antibodies to *S. rectivirgula*, which can identify herds that have suffered intense exposure (Wiseman et al. 1973). However, the presence of these antibodies is not diagnostic because clinically normal animals may have similar titers (Van Metre 1997). It mainly affects older cows (more than 5 years old) (Stöber 2005), although, in this study, primiparous and multiparous cows were similarly affected.

Another predisposing factor for the development of the disease is environmental dust, which reaches the lower parts of the respiratory tract dragging dry manure carrying bacterial endotoxins, which could trigger a serious hypersensitive respiratory condition (Miller and Woodbury 2003). This dust is usually produced by the trampling of the animals in the pens fragmenting the dry manure on the floor. Therefore, these housing conditions may be the cause of this dust exposure (Sweeten et al. 1988, Auvermann et al. 2000). Particles suspended in the dust and floating in a range between 2 and 4 microns tend to increase the incidence of pneumonia in the herd, since their small size allows them to reach the lower respiratory tract producing damage at this level (McVean et al. 1986). When soil moisture exceeds 30%, the amount of environmental dust is reduced (Sweeten et al. 1988). Therefore, the recent climatic conditions could probably predispose to the presentation of hypersensitivity pneumonitis.

It is not possible to confirm the etiology of the respiratory disease in all the affected dairy cows. However, the pathologic findings allow a strong suspicion of hypersensitivity pneumonia in the necropsied cow. The presence of an environmental factor, such as the presence of dust could have influenced this presentation. Therefore, the type of soil, climate and husbandry conditions should be

evaluated to identify predisposing factors to this condition. In this sense, despite the observed histopathological findings, there are serious diagnostic limitations, making an environmental study necessary to evaluate the possible presence of thermophilic actinomycetes or other factors that may be involved in the triggering of these pathologies.

This study provides a complete description of a presumed case of hypersensitivity pneumonitis in a dairy farm in Argentina, with clinical and pathological findings compatible with previous descriptions. Although there are some limitations in the etiological confirmation of this respiratory disease, histopathological findings are suggestive of such hypersensitivity processes. This work also highlights the importance of incorporating these new dairy farm management platforms for the early detection of health events, likely improving their prognosis.

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REFERENCES

1. Auvermann B, Parker D, Sweeten J. Manure harvesting frequency: the key to feedyard dust control in a summer drought. *Agri. Life Extension.* 2000; E-52. https://tammi.tamu.edu/files/2014/04/manureharvesting.pdf
2. Bourke S, Dalphin J, Boyd G, McSharry C, Baldwin C, Calvert J. Hypersensitivity pneumonitis: current concepts. *Eur. Respir. J.* 2001; 18: 81-92.
3. Breeze R. Respiratory disease in adult cattle. *Vet. Clin. North Am. Food. Anim. Pract*. 1985; 1: 311-346.
4. Carvallo F, Stevenson V. Interstitial pneumonia and diffuse alveolar damage in domestic animals. *Vet Path*. 2022; 59: 586-601.
5. Caswell J, Williams K. Chapter 5. Respiratory system. In: Grant Maxie M. editor. Jubb, Kennedy, and Palmer´s Pathology of Domestic Animals: Volume 2. 6th ed. UK; 2015. p. 512-513.
6. Cebollero P, Echechipía S, Echegoyen A, Lorente M, Fanlo P. Neumonitis por hipersensibilidad (alveolitis alérgica extrínseca). *Anales Sis. San. Navarra.* 2005; 28: 91-99.
7. Ellis J. Bovine parainfluenza-3 virus. *Vet. Clin. North Am. Food Anim. Pract*. 2010; 26: 575-593.
8. Fink J. Hipersensitivity pneumonitis. *J. Allergy Clin. Immunol*. 1984; 74: 1-9.
9. Kawai T, Salvaggio J, Harris J, Arquembourg P. Alveolar macrophage migration inhibition in animals immunized with thermophilic actinomycete antigen. *Clin. Exp. Immunol*. 1973; 15: 123-130.
10. Macías-Rioseco M, Mirazo S, Uzal F, Fraga M, Silveira C, Maya L, Riet-Correa F, Arbiza J, Colina R, Anderson ML, Giannitti F. Fetal pathology in an aborted Holstein fetus infected with bovine parainfluenza virus-3 genotype A. *Vet Pathol*. 2019; 56: 277-281.
11. McVean D, Franzen D, Keefe T, Bennett B. Airborne particle concentration and meteorologic conditions associated with pneumonia incidence in feedlot cattle. *Am. J. Vet. Res*. 1986; 47: 2676-2682.
12. Miller D, Woodbury B. Simple protocols to determine dust potentials from cattle Feedlot soil and surface samples. *J. Environ. Qual.* 2003; 32: 1634-1640.
13. Panciera R, Confer A. Pathogenesis and pathology of bovine pneumonia. *Vet. Clin. Food Anim. Pract*. 2010; 26: 191-214.
14. Sharma O. Hypersensitivity pneumonitis: a clinical approach. In: Herzog H, ed. Progress in respiration research. Basel, Karger, 1989. p. 1-10.
15. Spagnolo P, Rossi G, Cavazza A, Bonifazi M, Paladini I, Bonella F, Sverzellati N, Costabel U. Hypersensitivity pneumonitis: a comprehensive review. *J. Invest. Allergol. Clin. Immunol*. 2015; 25: 237-250.
16. Stöber M. Reacciones de sensibilidad de bronquios y pulmones. Medicina Interna y Cirugía del Bovino. Editorial Intermédica. 4° ed. 2005. P. 313-314.
17. Sweeten J, Parnell C, Etheredge R, Osborne D. Dust emissions in cattle feedlots. *Vet. Clin. North Am. Food Anim. Pract.* 1988; 4: 557-578.
18. Van Metre D. Allergic respiratory disease. *Vet. Clin. North Am. Food Anim. Pract*. 1997; 13: 495-514.
19. Weber S, Kullman G, Petsonk E, Jones W, Olenchock S, Sorenson W, Parker J, Marcelo-Baciu R, Frazer D, Castranova V. Organic dust exposures from compost handling: case presentation and respiratory exposure assessment. *Am. J. Ind. Med.* 1993; 24: 365-374.
20. Wilkie B. Hypersensitivity pneumonitis: experimental production in calves with antigens of *Micropolyspora faeni*. *Can. J. Comp. Med*. 1976; 40: 221-227.
21. Wilkie B. Bovine allergic pneumonitis: an acute outbreak associated with mouldy hay. *Can. J. Comp. Med.* 1978; 42: 10-15.
22. Wiseman A, Dawson C, Pirie H, Breeze R, Selman I. The incidence of precipitins to *Micropolyspora faeni* in cattle fed hay treated with an additive to suppress bacterial and mould growth. *J. Agric. Sci.* 1973; 81: 61-64.
23. Wiseman A. Influence of environment on respiratory disease. In: Respiratory diseases in cattle: a Seminar in the EEC Programme of Coordination of Research on Beef Production held at Edinburgh. 1978; 149-157.