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**Case Report**

# Bilateral Giant Pulmonary Emphysematous Bullae in a Calf

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**Abstract**

Bilateral giant pulmonary emphysematous bullae were diagnosed in a calf. The necropsy revealed two gas-filled bullae completely involving the right and left diaphragmatic pulmonary lobes. Histologically, the outer surface of the giant bullae was made of visceral pleura while the inner layer consisted of the adjacent lung, which presented signs of atelectasis or emphysema. Aspiration bronchopneumonia was observed in the right and left apical lobes. The animal died as the result of acute respiratory failure.

**Key Words:** cattle diseases, pulmonary emphysema, respiratory system

## Introduction

Several pulmonary diseases can be characterized by the formation of circumscribed air- or fluid-containing cavities with variably distinct walls. The main cavitary pulmonary lesions are bullae, cysts, abscesses and cystic bronchiectasis (12).

Although bullous emphysema with simple or multiple bullae is a common lung pathology, few reports of giant emphysematous bullae have been described in the veterinary literature (8, 14). The majority of the reports have been in humans in association with chronic inhalation of tobacco smoke, with Marfan syndrome or with lung cancer (2, 13, 15, 16).

Bullous emphysema is histologically referred to as the presence of emphysematous areas with a complete destruction of lung tissue producing an airspace greater than 1 cm in diameter. Lung bullae are air-filled spaces within the lung parenchyma resulting from the destruction of the alveolar spaces (11). Giant bullae consume more than one third of a lung and can completely occupy a given segment or lobe (6). The majority of giant bullae increase gradually in size and result in worsening respiratory function (13). Bullae must be clearly differentiated from other disorders, such as congenital cystic adenomatoid malformation, bronchogenic cysts and congenital lobar emphysema (1).

Bullae are air-filled spaces lined by visceral pleura, connective septa or the adjacent lung (6). In humans, they may present either as isolated spaces in an otherwise normal lung or as part of a diffuse disease, such as emphysema. The association of bullae with neoplasia and inflammatory diseases of the lung has also been reported (11).

In emphysema, the structural modifications (alveolar distension with destruction of the walls and of the pulmonary capillary bed) that contribute to the understanding of pathophysiological disorders include the increase of static pulmonary volumes, the loss of elastic recoil and the impairment of gas transfer. The effects of voluminous bullae associated with diffuse emphysema are difficult to study and to demonstrate. The bullae accentuate the altered elastic properties of the emphysematous lung and, therefore, contribute to the expiratory flow limitation (17). There are different

 

modes of pathogenesis to explain the origin of the emphysematous changes; on the one hand, the changes can be caused by an inherited defect in connective tissue synthesis, such as in Marfan syndrome, or on the other hand, there can be an imbalance between proteases and antiproteases (7). It seems likely that emphysematous bullous cysts arise as a result of some overexertion such as coughing, shouting or digging in the presence of an inflamed or ulcerated bronchus with the escape of air into the interstitial tissue of the lung (2).

The emphysematous bullae are usually of pathological interest. The patient can be asymptomatic. However, the bullae can rupture into the pleural cavity and cause a tension pneumothorax (2, 8). If the cyst formation is extensive, the patient may die of asphyxia because there is not enough normal lung tissue left to meet his respiratory needs (2). Complete spontaneous resolution of a giant bulla is a rare occurrence (13).

To our knowledge, there are few reports of giant emphysematous bullae in animals. The purpose of this study is to report a case of bilateral giant emphysematous bullae in a calf**.**

## Case Report

A 27-day-old Dutch calf was admitted to the veterinary hospital with a history of dysphagia, regurgitation, lethargy, cough, respiratory effort and cyanosis. According to the veterinarian of the farm, the calf had always presented regurgitation after being fed with milk in the feeding bottle, and the breathing symptoms appeared later. The animal was treated for pneumonia with penicillin but died several hours later.

The necropsy revealed two gas-filled bullae that measured about 11 x 9 x 6 cm in size and completely involved the right and left lower diaphragmatic pulmonary lobes (Figs. 1 and 2). The adjacent pulmonary tissue showed signs of atelectasis (Fig. 2) and areas of bullous emphysema. The cranioventral portion of the right and left pulmonary apical lobes was pinkish-red and consolidated. Trace amounts of milk and vegetable fibers were observed in the pulmonary parenchyma, suggesting aspiration bronchopneumonia. The tracheal mucosa was congested, and a minimal amount of milk was observed inside the tracheal lumen.

In the esophagus, a sharp line of demarcation between the pale, ischemic distal esophagus and the congested proximal esophagus at the thoracic inlet was observed similar to the "bloat line" (Fig. 3). Hemorrhages were observed in the epicardium of the right ventricle of the heart. The liver and kidneys were moderately congested.

Histologically, pockets of gas were present in the alveolar space. The outer surface of the bullae was made of visceral pleura while the inner layer consisted of the adjacent lung. The adjacent tissue showed the collapsed air spaces that characterize atelectasis and areas of bullous emphysema characterized by alveolar distension with destruction of the walls and formation of small bullae (Fig. 4). There was no inflammation in the diaphragmatic lobes. The cranioventral portion of the right and left pulmonary apical lobes showed abundant neutrophils, macrophages, and cellular debris within the lumen of the bronchi, bronchioles, and alveoli. Inside the lumen of the bronchi, bacteria, vegetable fibers and protein material suggestive of milk were also observed. The bronchi and bronchioles did not present developmental anomalies such as hypoplasia.

## Discussion

Based on the macroscopic and microscopic features, a diagnosis of bilateral giant emphysematous bullae associated with bullous emphysema was made. Bullae are characterized as air spaces within the lung parenchyma (11). These pathologic characteristics are different from those of pleural air cysts, bronchogenic cysts and congenital cystic adenomatoid malformations.

A pleural air cyst is defined as interstitial emphysema within the pleura that causes a thin-walled prominence on the surface of the lung (3). Histologically, the pockets of gas were present in the visceral subpleural space (between the mesothelial cells and the basal lamina of the pleural connective tissue), where the pleura was elevated. The main consequences of this disorder are rupture and pneumothorax (9).

Bronchogenic cysts are lesions of congenital origin filled with mucus or serous fluid microscopically characterized by a cystic wall lined with respiratory epithelium and containing cartilage, smooth muscle and seromucinous glands (5).

A congenital cystic adenomatoid malformation consists of disorganized pulmonary tissue that can exist with or without gross cyst formation. A type I lesion is characterized by formation of large cysts lined by ciliated, cuboidal or columnar epithelium. A type II lesion is characterized by formation of small cysts lacking mucus-producing cells and cartilage. A type III lesion consists of a solid mass with small cysts; the lesion is lined with nonciliated, cuboidal epithelium (4, 10).

There are two possible explanations for the pathogenesis of the emphysematous giant bullae in this case. Based on the information that the calf had always presented regurgitation, the bullae could have been congenital, and the aspiration bronchopneumonia could have been caused by regurgitation due to the compression of the cysts on the esophagus because of the sharp line observed in the esophagus. Alternatively, it could have been that the emphysema and the giant bullae were caused by the bronchopneumonia.

The greatly impaired lung parenchyma caused the death of the animal through acute respiratory failure.

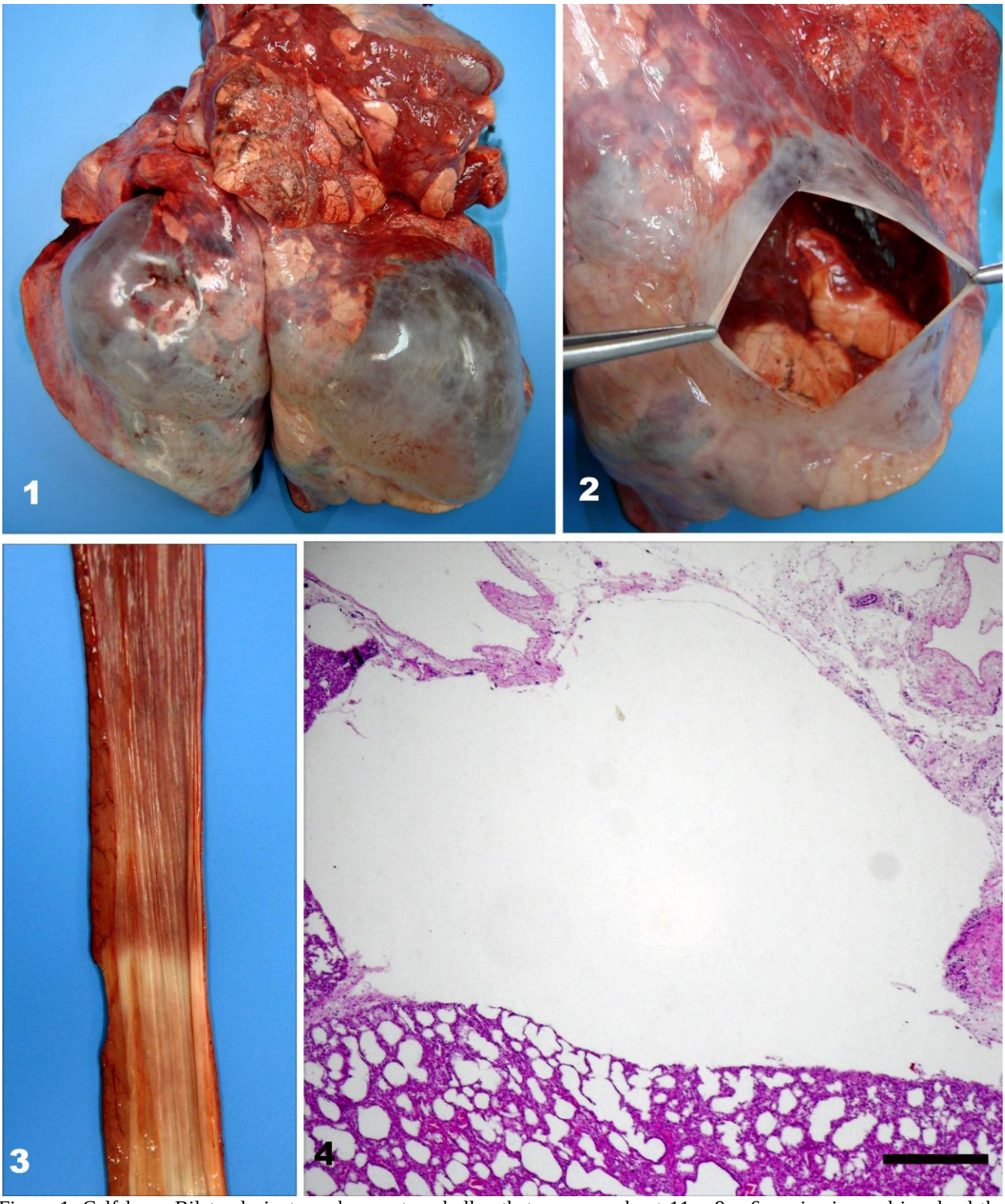


Figure 1. Calf lung. Bilateral giant emphysematous bullae that measure about 11 x 9 x 6 cm in size and involved the right and left diaphragmatic pulmonary lobes.
Figure 2. Giant emphysematous bullae without liquid and with atelectasis of the adjacent pulmonary tissue.
Figure 3. Esophagus with a sharp line of demarcation between the pale, ischemic distal esophagus and the congested, proximal esophagus at the thoracic inlet.
Figure 4. Bullae with the outer surface consisting of visceral pleura and the inner layer consisting of the adjacent lung with atelectasis. Haematoxylin-eosin. Bar = 533 µm.

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