

Acute pulmonary emphysema cum pulmonary edema apparently associated with feeding of *Brassica juncea* in a dairy buffalo

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Received: 06.06.2008

Abstract: This preliminary report describes the occurrence of acute pulmonary emphysema cum pulmonary edema ensuing in extensive subcutaneous emphysematous swellings in a dairy buffalo (*Bubalus bubalis*) apparently associated with a sudden shift from berseem (*Trifolium alexandrinum*) to *Brassica juncea* fodder. Tachypnea, expiratory dyspnea, open-mouth breathing, loud expiratory grunt with abdominal lift, and crackles in ventral aspects of the lungs with normal rectal temperature characterized the condition clinically. A substantial improvement was noted 3 h after treatment with parenteral administration of prednisolone plus dexamethasone, diclofenac sodium, and furosemide. Per os administration of chlortetracycline was undertaken after dyspnea had abated. Extensive subcutaneous swellings developed as sequel on withers, back, axillae, and thorax, was deflated using a hypodermic needle. The buffalo completely recovered in 10 days. As far as could be ascertained, this is the first report dwelling on acute pulmonary emphysema cum pulmonary edema.

Key words: Pulmonary emphysema, pulmonary edema, *Brassica juncea*, dairy buffalo

Introduction

Pulmonary emphysema, variously known as fog fever, bovine asthma, acute respiratory distress syndrome, pulmonary adenomatosis, and bovine panting, is an acute non-infectious disease of cattle clinically characterized by severe respiratory distress (1,2). In some cases, pulmonary edema also accompanies pulmonary emphysema as a complication. Although a wealth of literature is

available on this condition in cattle, there is no report on the occurrence of a similar syndrome in dairy buffalo (*Bubalus bubalis*). The present report describes a case of pulmonary emphysema cum pulmonary edema in a Nili-Ravi dairy buffalo.

Case history

Professional assistance was sent for to treat a 6-year-old, 600 kg Nili-Ravi buffalo suffering from a

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severe respiratory distress for the last 30 min. The subject was in the 2nd month of her 3rd lactation. It was in excellent body condition (body condition score 5) as per the scoring guidelines for Holstein dairy cows (3). Besides the affected buffalo, the client also maintained another lactating adult buffalo, a 3-year-old buffalo heifer and a 2-month-old buffalo calf. All were stall-fed with zero grazing. They were kept tethered to their posts in the village street during the daytime and in a shed at night. Anamnesis revealed that a sudden switch to chopped *Brassica juncea* (sarsoon) fodder with very little wheat chaff (1 kg per 30 kg green fodder) had been made 2 days before. Prior to this change, animals were maintained on chopped berseem (*Trifolium alexandrinum*) fodder with an ample (7 kg per 30 kg green fodder) addition of wheat chaff. *Brassica juncea* fodder field had been fertilized with a nitrogenous fertilizer (urea) 10 days before.

Clinical examination of the affected buffalo revealed a normal rectal temperature (37.8 °C), tachypnea (44 per min), expiratory dyspnea accompanied by open-mouth breathing, flaring of nostrils, loud expiratory grunt, and abdominal lift. The head was extended and the animal presented a lethargic, tranquil look. A moderate degree of frothing was present at the mouth. Loud breathing sounds and crackles were audible on auscultation of ventral lung field. A diagnosis of acute pulmonary emphysema cum pulmonary edema was deduced on the basis of characteristic clinical signs of this disease (2,4) and consideration of relevant epidemiological circumstances (i.e. sudden switch to *Brassica juncea* fodder with very little wheat chaff, application of fertilizer to the fodder field). The following treatment was instituted:

Prednisolone 7.5mg + dexamethasone 2.5 mg/mL, 12 mL i.m.

Diclofenic Sodium 50 mg/mL, 20 mL i.m.

Furosemide 10 mg/mL, 20 mL i.m.

Results and discussion

An appreciable improvement in the respiratory distress of the buffalo was noted after 3 h use of the therapy detailed above and the patient was then dosed

per os with 50 g of chlortetracycline 200 mg/g. Feeding of *Brassica* fodder was stopped and animals were maintained on sugar cane fodder plus concentrate. Respiratory distress had abated in the following day. However, on day 2, extensive soft emphysematous swellings developed on the withers and back. A 16-gauge, 2.5 cm hypodermic needle was inserted into the centers of swellings and trapped air discharged by propelling it towards the needle. On day 3, emphysematous swellings extended to the axillae and thorax, which were similarly deflated by the hypodermic needle. Further treatment beyond day 3 was limited to per os administration of 6 tablets of mebhydrolin, 50 mg/tab per day for 4 days. The animal completely recovered on day 10 of initiation of treatment.

Acute bovine pulmonary emphysema and edema (ABPEE) occurs following recent abrupt and fairly dramatic changes from marginal to better quality pastures (2,5). ABPEE has been reported to occur in cattle grazing on a wide variety of meadow grasses (purple mint, stinkwood), alfalfa, rapeseed, kale (*Brassica* species), and turnip-tops (4,6). There is a marked tendency for the condition to arise sooner in cattle grazing on fertilized pastures than in those on unfertilized fields (6). The disease is usually apparent within 4-10 days after the introduction of the herd to a new pasture (1,4,7) and clinical signs are generally limited to animals older than 2 years (6). Clinical signs and epidemiological features (e.g. sudden change in fodder, application of nitrogenous fertilizer to *Brassica* fodder field, age of the affected buffalo) of the condition and natural history described in the present report correspond fairly well to those reported previously (8,9). A lag phase of 2 days between switching to *Brassica* fodder is, however, shorter than that reported for bovine (4,8), which may be ascribed to species differences between cattle and buffalo.

ABPEE results from excessive conversion of L-tryptophan to 3-methylindole (3-Mi; a tryptamine analogue) and this conversion is brought about by certain species of ruminal microbes, notably *Lactobacillus* (10). Certain pasture management practices are believed to create a ruminal environment that favors the conversion of L-tryptophan to 3-Mi. Following its production, 3-Mi is absorbed and transported to the lungs where it is converted to pneumotoxic metabolite (3-methyleneindolenine) by

mixed function oxidase system (cytochrome P450 enzymes) in type-I pneumocytes or Clara cells and prostaglandin-H synthetase, which results in necrosis of bronchio-alveolar epithelium and subsequent ABPEE (10-12). It may be that pathogenesis of Brassica-associated-pulmonary emphysema cum edema in buffalo involves prior digestive or metabolite activation of an ingested factor that is completed in 2 days.

A variety of therapeutic agents, including corticosteroids, antihistaminics, anticholinergics, antibiotics, nonsteroidal anti-inflammatory drugs, diuretics, etc., have been used by various researchers (1,8). The use of antibiotics appears unnecessary (13). To what extent per os administration of chlortetracycline aided in the recovery in the subject of the present report is open to conjecture since corticosteroid, diuretics, and NSAIDs were all used.

Another lactating buffalo, a 3-year-old buffalo heifer, and a 2-month-old buffalo calf fed the same fodder remained unaffected. Individual variability in the conversion of L-tryptophan to the premier emphysemagenic factor (3-methylindole) in the rumen (4,14) may explain in part at least the sparing of these animals fed the same fodder. Exaggerated respiratory efforts may break alveoli, allowing escape of air into subcutaneous tissue (15). This may account for development of emphysematous swellings on withers, back, axillae, and thorax in the wake of acute pulmonary emphysema cum edema in the subject of the present report. As far as could be ascertained from the available literature, this is the first documentation of acute pulmonary emphysema cum edema in dairy buffalo.

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