Journal of Equine Veterinary Science 34 (2014) 1234–1239

Contents lists available at ScienceDirect

Journal of Equine Veterinary Science

journal homepage: www.j-evs.com

Case Report

Extensive Dystrophic Pulmonary Calcification in a Welsh Pony Mare

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ARTICLE INFO

Article history:
Received 20 December 2013
Received in revised form 23 June 2014
Accepted 9 July 2014
Available online 16 July 2014

Keywords:
Lung calcification
Mineralized lung

ABSTRACT

A 12-year-old Welsh pony mare was presented to the Ontario Veterinary College Teaching Hospital for signs of intermittent lethargy and increased abdominal breathing effort of 6 months duration. After physical examination, blood work, bronchoscopy, bronchoalveolar lavage, and diagnostic imaging of the thorax and attempted lung biopsy, pulmonary mineralization of unknown origin was suspected. The pony was treated palliative for 7 months with nonsteroidal anti-inflammatories and inhaled corticosteroids to treat accompanying airway inflammation before being euthanized because of poor prognosis and deterioration of clinical signs. On postmortem examination, the pulmonary architecture of the right and left cranioventral lung lobes, accessory lobe, and cranial potions of the left caudal lung lobe was replaced by hard mineralized tissue. No other organs other than a mediastinal lymph node and the lung were affected by mineralization. After decalcification, thick sheets of fibrous connective tissue organized into layers and lamellae replaced the normal architecture of the pulmonary parenchyma in more than 90% of the lung lobe examined on histopathology. The findings were consistent with generalized severe pulmonary fibrosis and dystrophic calcification.

1. Introduction

Dystrophic pulmonary calcification is a rare clinical syndrome associated with varying underlying disorders in humans, dogs, and horses. This case report describes a case of extensive generalized dystrophic pulmonary calcification involving both cranioventral lung lobes to an extent not reported before. No signs of neoplasia, previous respiratory disease, or metabolic disease were present.

2. Case Presentation

A 12-year-old Welsh pony mare was presented for a history of lethargy and increased respiratory effort. The pony was used as a show hunter up until the time of presentation. For 6 months before presentation, the pony began tiring easily when being ridden and intermittent increased abdominal effort during breathing was observed. No coughing or other signs were observed. Appetite and fecal output were normal. The referring veterinarian reported no abnormalities on repeated physical examination. Serial blood hematology and biochemistry over the next 6 months showed persistent anemia (red blood cell count, 5.4×10^{12} to 6.4×10^{12} cells/L; reference range, 8.8×10^{12} to 12.5×10^{12} cells/L; hematocrit, 0.27–0.31 L/L; reference range, 0.32–0.52 L/L; hemoglobin, 102–105 g/L; reference

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http://dx.doi.org/10.1016/j.jevs.2014.07.002

range, 110–190 g/L) and hyperglobulinemia (64–69 g/L; reference range, 22–40 g/L). Transient mild mature neutrophilia (8.3×10^9 cells/L; reference range, $2.7–6.7 \times 10^9$ cells/L) was also noted. A course of antibiotic treatment with trimethoprim sulfonamide (24 mg/kg PO, every 12 hours for 20 days) was administered, and mild improvement was noted; however, hematological abnormalities and exercise intolerance were unchanged. The pony was regularly dewormed and vaccinated against influenza and tetanus and had been in current ownership for 9 years. The current owners reported no prior medical problems; however, no history from the previous owner was available.

On presentation to the Ontario Veterinary College, University of Guelph, the mare was bright and in good body condition (body condition score [BCS], 3/5; 313 kg). Mild tachypnea (respiratory rate, 36 breaths/min) with increased abdominal effort was present. Increased bronchial sounds were auscultated diffusely over the caudodorsal lung fields bilaterally. Reduced lung sounds, most evident on rebreathing, were noted cranioventral. No crackles or wheezes were heard. All other findings on physical examination were within normal limits.

Hemogram and serum biochemical abnormalities included anemia, hyperfibrinogenemia, hypoalbuminemia, and hyperglobulinemia. No others significant abnormalities were present (Table 1). Results for a coagulation profile including plasma prothrombin time and partial thromboplastin time and arterial blood gas analysis, including pH, partial oxygen pressure, partial carbon dioxide pressure, base excess, and bicarbonate, were normal.

On thoracic radiographs, a mineralized opacity in the caudoventral region of the lungs, immediately caudal to the heart was present (Fig. 1). A second mineralized opacity was present cranial to the heart. The caudodorsal aspect of the lungs had a mild interstitial pattern. Dorsoventral radiographs confirmed that the mineral opacities were present bilaterally (Fig. 2). There were normal contours of the cardiomediastinum.

On thoracic ultrasound imaging, an irregular surface of the right ventral lung, multiple comet-tail or ring-down artifacts, and additionally several 3.5-cm round focal areas of consolidation were present. On the left side, the visceral pleura was thickened (up to 1 cm) creating a “rind-like” appearance. In addition, the pleural margin was irregular with multifocal ring-down artifacts and several lesions extending into the pulmonary parenchyma (Fig. 3). Abdominal ultrasound imaging result was normal. Attempt at ultrasound-guided lung biopsy failed because of the abnormal consistency of the lung tissue preventing penetration of the visceral pleura bilaterally.

Bronchoscopy illustrated severe bronchoconstriction and a blunted carina. Cytologic evaluation of bronchoalveolar lavage fluid supported severe inflammatory airway disease: nucleated cell count, 2.1×10^9 cells/L (reference range, $0.2–0.4 \times 10^9$ cells/L); lymphocytes, 49% (reference range,15%–30%); macrophages, 35% (reference range, 35%–70%); mast cells, 3% (reference range, 0%–2%); and segmented neutrophils, 14% (reference range, 0%–4%); macrophages were activated and Curschmann spirals were present. No bacteria or crystalline material was seen.

Table 1
Hematology and serum biochemistry of a horse affected with extensive generalized pulmonary calcification.

Parameter	Reference Range	Initial	2 mo	7 mo
Hematocrit, L/L	0.28–0.44	0.30	0.37	0.32
RBC, $\times 10^9$	6.9–10.7	5.9	7.2	6.4
Hemoglobin, g/L	112–169	111	136	114
MCV, fL	36–45	50	51	50
MCH, pg	14–18	19	19	18
MCHC, g/L	369–426	376	374	352
Red cell distribution width, %	18–21	17.5	17.2	16.4
White blood cells, $\times 10^9$	5.1–11.0	8.8	13.7	10.9
Band neutrophils, $\times 10^9$	0–0.2	0	0	0
Seg. neutrophils, $\times 10^9$	2.8–7.7	6.95	12.06	8.94
Lymphocytes, $\times 10^9$	1.3–4.7	1.50	0.69	1.31
Monocytes, $\times 10^9$	0.1–0.8	0.26	0.82	0.55
Eosinophils, $\times 10^9$	0–0.7	0.09	0	0
Basophils, $\times 10^9$	0–0.2	0	0.14	0.11
Platelets, $\times 10^9$	83–270	193	336	302
Mean platelet volume, fL	6–11	6.1	7.1	6.8
BUN, mmol/L	4.2–8.9	7.9	11.2	2.7
Creatinine, mmol/L	80–130	65	177	61
Alk. Phos., IU/L	119–329	64	123	177
GGT, IU/L	7–54	22	67	130
GLDH, IU/L	1–7	2	9	22
SGOT (AST), IU/L	259–595	183	314	224
CK, IU/L	108–430	98	144	153
Total bilirubin, μmol/L	21–57	10	13	5
Direct bilirubin, μmol/L	2–3	3	3	1
Glucose, mmol/L	3.7–6.7	6.4	6.4	6.0
Sodium, mmol/L	136–144	136	133	131
Potassium, mmol/L	3.1–4.3	4.5	4.4	3.6
Chloride, mmol/L	95–104	102	88	96
Calcium, mmol/L	2.75–3.35	2.98	3.77	2.77
Phosphorus, mmol/L	0.73–1.71	0.82	0.80	0.85
Calcium × phosphorus (mg^2/dL^2)	Not available	30.2	37.4	29.1
Magnesium, mmol/L	0.6–1.0	0.7	0.6	0.8
Anion gap, mmol/L	6–21	13	17	13
Total protein, g/L	58–75	96	80	101
Albumin, g/L	30–37	27	28	21
Globulin, g/L	26–41	69	52	80
Cholesterol, mmol/L	1.7–2.70	1.91	2.73	1.63
Haptoglobin, mmol/L	0.1–1.7	1.74	1.69	1.5
Fibrinogen, g/L	1.2–2.3	3.3	3.7	

Alk. Phos., alkaline phosphatase; BUN, blood urea nitrogen; CK, creatinine kinase; GGT, gamma-glutamyl transferase; GLDH, glutamate dehydrogenase; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; MCV, mean corpuscular volume; RBC, red blood cells; Seg. neutrophils, segmented neutrophils; SGOT (AST), aspartate transaminase.

Aerobic culture of bronchoalveolar fluid was negative. An echocardiogram was performed and showed normal functional and anatomic indices of the heart. No signs of pulmonary hypertension were present (pulmonary artery diameter and tricuspid regurgitation flow velocity and/or pressure differential). Testing for rheumatoid factor was negative, and antinuclear antibody was positive. Protein electrophoresis showed a polyclonal gammopathy (total protein, 94 g/L; reference range, 58–75 g/L; albumin, 28 g/L; reference range, 25–36 g/L; globulin, 66 g/L; reference range, 26–41 g/L; alpha$_1$-globulin, 2 g/L; reference range, 5–14 g/L; beta$_1$-globulin, 8 g/L; reference range, 9–18 g/L; gamma globulins, 42 g/L; reference range, 6–14 g/L). Immunoelectrophoresis showed increased levels of immunoglobulin G. No M-protein was detected.