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Case Report

Acquired Pulmonic Stenosis Due to a Compressing Intrathoracic Abscess in a Thoroughbred Yearling

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ABSTRACT

A 12-month-old Thoroughbred filly was admitted for poor weight gain, intermittent fever, and distal limb edema. Initial clinical examination revealed tachycardia, tachypnea, and pyrexia. A loud holosystolic murmur was identified by cardiac auscultation, and thoracic auscultation identified bilateral diffuse crackles. Echocardiography demonstrated severe extrinsic pulmonic stenosis, enlargement of the right ventricle and compression of the left ventricle. Mediastinal ultrasound revealed a large, encapsulated, cavernous mass in the cranial thorax, compressing the heart. Bacterial culture of tracheal aspirates and aspirations of the intrathoracic mass yielded a pure culture of *Streptococcus equi* spp. *zooepidemicus*. Acquired pulmonic stenosis has not previously been reported in horses, whereas congenital pulmonic stenosis has. This case report demonstrates that in line with human medicine, pulmonic stenosis can be acquired because of thoracic masses compressing the pulmonic artery.

1. Introduction

Pulmonic stenosis is a rare finding in the horse, characterized by an increased resistance emptying the right ventricle that is caused by either intrinsic obstruction or extrinsic compression of the right ventricular outflow tract. This in turn causes hypertrophy of and increased pressure within the right ventricle and atrium. Severe stenosis will decrease the stroke volume of the heart and cause compensatory increased heart rates [1]. The condition in turn will thus lead to systemic circulatory changes causing exercise intolerance, weight loss, peripheral edema, and pulmonary edema [2].

Pulmonic stenosis has been described as a congenital disorder causing severe hypoxia in affected foals [3,4]. These congenital abnormalities are usually diagnosed early in life because of their severe clinical impact [4]. Only a single report describes an assumed congenital malformation of the pulmonic valves causing pulmonic stenosis in an adult horse [1].

Theoretically, pulmonic stenosis can also be acquired in the horse because of external compression. Extrinsic pulmonic stenosis due to cranial thoracic neoplasia has been described in the human literature [5–9]. Cranial thoracic abscesses may arise as a sequel to infections such as pneumonia, pulmonary abscesses, pericarditis, or pleuropneumonia and can result in cardiac compression depending on localization [10,11]. Multiple reports describe cranial thoracic masses including neoplasia and abscess formation in the horse, but to the authors' knowledge, there are no reports of masses causing acquired pulmonic stenosis in the horse [12,13].

2. Case History

A 12-month-old Thoroughbred filly was referred to the Large Animal Teaching Hospital at the University of

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Copenhagen for evaluation of ill thrift and intermittent fever of 3- to 4-week duration. Before referral, a loud holosystolic murmur was noted as well as distal limb edema of the front limbs. Four weeks before admission, several yearlings in the same herd were noted to have respiratory symptoms including nasal discharge, fever, and cough. No prior diagnostic tests were performed on the facility to determine the cause of the infection. The horse never showed overt symptoms of airway disease and had not received antibiotic treatment before referral.

2.1. Initial Clinical Findings

At the time of admission, the filly was bright and alert with a good appetite. The horse appeared unthrifty, with matted hair coat and a body condition score of 2/9. The initial clinical examination revealed an elevated heart rate (60 bpm) and rectal temperature (38.7°C). The filly presented with moderately elevated respiratory rate (20 rpm), a mild abdominal respiratory pattern, and bilateral mucopurulent nasal discharge. An intermittent productive cough was also noted, and coughing was easily provoked by compression of the larynx and trachea. The submandibular lymph nodes were normal on palpation. Thoracic auscultation identified diffuse crackles bilaterally. No rattling sounds were evident on tracheal auscultation.

Moderate, pitting distal limb edema was noted on both front limbs. The filly appeared well hydrated, mucous membranes were pink and moist, and capillary refill time was within 2 seconds. Normal stasis and emptying of the jugular veins was noted, and on palpation of the facial artery, a strong pulse with normal rhythm was identified.

Cardiac auscultation revealed a holosystolic murmur, grade 4/6 with point of maximal intensity over the pulmonic ostium. Persistent tachycardia was noted, but no arrhythmias could be identified on auscultation or subsequent base-apex electrocardiogram examination.

2.2. Initial Diagnostics

Blood samples at the time of admission revealed mild anemia (packed cell volume: 27% [20%–41%]), severe neutrophilic leukocytosis (white blood cells: 20,980 cells/μL [5,450–12,650 cells/μL], neutrophils: 14,920 cells/μL [2,260–7,220 cells/μL], lymphocytes: 4,240 cells/μL [1,260–5,740 cells/μL], monocytes: 1,280 cells/μL [0–1,000 cells/μL], eosinophils: 100 cells/μL [0–1,000 cells/μL], basophils: 120 cells/μL [0–290 cells/μL], hyperfibrinogenemia [fibrinogen: 7.76 g/L; 1–4 g/L]), and increased serum amyloid A (3265.4 mg/L [0–30 mg/L]), and decreased iron levels (5.99 μmol/L [13.1–25.1 μmol/L]). All these blood parameters indicate severe inflammation.

Cranial thoracic ultrasound, through the triceps muscle, revealed a large, oval-shaped, cavernous, multiloculated mass measuring approximately 15 by 20 cm. The mass was surrounded by a hyperechoic capsule approximately 3 cm in thickness and was filled with slightly hyperechoic fluid material. The mass appeared to occupy the majority of the cranial thoracic space, exerting significant compression of the heart and in particular the pulmonary artery (Fig. 1). Thoracic ultrasound was suggestive of mild bilateral pleural inflammation (comet tails) and mild pneumonia.

Echocardiography revealed severe enlargement of the right ventricle (luminal diameter) and compression of the left ventricle, with marked bulging of the interventricular septum into the left ventricle. Echocardiographic measurements are presented in Table 1. The right ventricular internal diameter at end diastole was increased, and the left ventricular internal diameter at end diastole demonstrated severe extrinsic volume restriction (Fig. 2). The pulmonary artery was severely compressed by the external mass. The diameter of the pulmonary artery obtained from the right parasternal angled view was severely reduced to 3.53 cm in systole and 1.74 cm in diastole (Fig. 3). Color Doppler examination identified no valvular regurgitation at the pulmonic, tricuspid, mitral, or aortic valves. Maximum pulmonary velocity was 2.3 m/s (reference values, 0.78–1.04 m/s [15]; Fig. 3). Minimal pericardial fluid was observed (Fig. 2).

Endoscopy of the upper airways demonstrated moderate pharyngeal lymphoid hyperplasia (grade III/IV) and mucopurulent exudate in the trachea (grade III/IV) [16]. A subsequent transendoscopic tracheal aspirate through a nonguarded catheter using 30 mL of sterile saline demonstrated macroscopically visible mucopurulent secretions and contained 82% neutrophils, 7.5% macrophages, 1.5% lymphocytes, 0.5% eosinophils, and 8.5% epithelial cells, suggestive of pulmonary inflammation.

Bacterial culture of tracheal aspirates and a direct ultrasound-guided aspirate of the intrathoracic mass yielded a pure culture of *Streptococcus equi* spp. *zooepidemicus* sensitive to penicillin.

Thoracic radiographs demonstrated increased craniodorsal radio density and diffusely increased bronchointerstitial pattern, suggestive of chronic bronchopneumonia. On radiographs, no mass could be visualized and they were not diagnostic in terms of distribution of the cranial thoracic mass.

A blood culture obtained by strict aseptic technique via venipuncture of the left jugular vein yielded no bacterial growth.

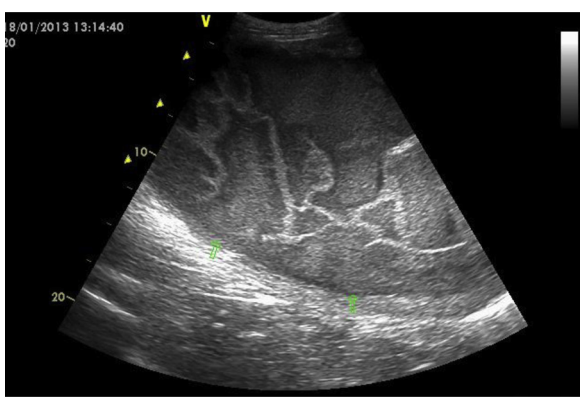

Fig. 1. Cranial thoracic ultrasound: Left parasternal ultrasound view obtained at the third intercostal space, showing a large cavernous fluid-filled structure with a thick capsule occupying almost the entire cranial thorax.