The echocardiography diagnosis of cor pulmonale in a horse

Lightowler, C.[1]; Pidal, G.[2]; Chiaramonte, P.[3]; Maidana, H.R.[4]; Pérez Valega, E.[4]; Kaenel, R.M.[4]

[1]Head, Cardiologic Unit; [2]Equine Area, Cardiologic Unit; [3]Fellow, Cardiologic Unit, Veterinary Teaching Hospital, School of Veterinary Sciences, Buenos Aires University, Chorroarñin 280, Buenos Aires (1427), Argentina. [4]Semiology Cathedra, School of Veterinary Sciences, Northeast University (Corrientes, Argentina). E-mail:olmo@fvet.uba.ar

Abstract

Lightowler, C.; Pidal, G.; Chiaramonte, P.; Maidana, H.R.; Pérez Valega, E. Kaenel, R.M.: ***The echocardiography diagnosis of cor pulmonale in a horse.*** *Rev. vet. 18: 1, 46–49, 2007.* The authors describe the two-dimensional and Doppler signs that characterize the pulmonary hypertension caused by chronic obstructive pulmonary disease (COPD) in a horse.

Keywords: horse, echocardiography, cor pulmonale.

Resumen

Lightowler, C.; Pidal, G.; Chiaramonte, P.; Maidana, H.R.; Pérez Valega, E. Kaenel, R.M.: ***Diagnostico ultrasonografico del "corazón pulmonar" en el caballo.*** *Rev. vet. 18: 1, 46–49, 2007.* Los autores describen los signos ecocardiográficos bidimensionales y Doppler característicos de la hipertensión pulmonar que desarrolla en el "corazón pulmonar" producido por enfermedad pulmonar obstructiva crónica (EPOC) en un caballo.

Palabras Clave: caballo, ecocardiografía, corazón pulmonar.

INTRODUCTION

The two-dimensional echocardiography and cardiac Doppler are important tools in the diagnosis of heart disease in horses [2, 14, 18, 20]. A lot of information exist referred to the anatomical characteristics, quantitative aspects and values of the different indexes referred to the left ventricle, but little about the right ventricle in this specie, especially referred to the echocardiography signs for diagnosis of cor pulmonale [2].

CASE HISTORY

The diseased horse was an 8-year-old pony, 198 kg body weight and 1.20 m height. The horse was presented to consult with a pour general state, expiratory dyspnea with countershock of flanks and sporadic cough. The reference indicated that the process was chronic (more of 6 months of duration).

Physical examination. The clinical evaluation put in evidence tachypnea (26 breath/min) and the presence of pulmonary wheeziness and crackles. At cardiac level we detected tachycardia (78 beat/min) and the presence of a loud tricuspid murmur (4/6). A presumptive diagnosis of chronic obstructive pulmonary disease (COPD) was made and a bronchoalveolar lavage, ventigraphy, thorax radiography, electrocardiogram and an echocardiography study were indicated. The follow up of the patient was of 75 days and in this moment the owner request its euthanasia.

Bronchoalveolar lavage (BAL). Total volume of BAL recovered = 107 ml. Total cell recovery = 2.02 x 10^6 ml. Cell count (%) = neutrophils: 13.1; lymphocytes: 37.2; macrophages: 39.2; mast cells: 5.4; eosinophils: 0.7 and epithelial cells: 4.04.

Ventigraphy. At five minutes of introduced the probe develops a delta wave of basal partial pleural pressure of 35 cm of water (normal value: < 4 cm of water). Ten ml of clembuterol IV were injected. At 10 minutes a value of partial pleural pressure of 21 cm of water was obtained. Result: severe bronchospasm at rest and partial remission with clembuterol IV.

Thoracic radiography. Increase of the pulmonary density with diffuse interstitial and peribronchial pattern. Air-bronchograms and bronchial calcifications. Bronchopneumonia (Figure 1).

Electrocardiography. Electrocardiographic study revealed a resting heart rate of 78 beat per minute with a sinus rhythm. Waves, segments and intervals were normal.

Echocardiography (two-dimensional and Doppler). The most significant findings were a marked enlargement of right ventricle with evident flattening of the interventricular septum (Figure 2) and paradoxical

Recibido: 7 mayo 2007 / Aceptado: 4 junio 2007

movement of interventricular septum (Figure 3). The diastolic diameter of the right ventricle was the double of the left ventricle. The aortic diameter at annulus level was 5.2 cm and the pulmonary artery diameter at valve level was 6.2 cm (Figure 4). The color flow mapping showed an important tricuspid regurgitation (Figure 5).

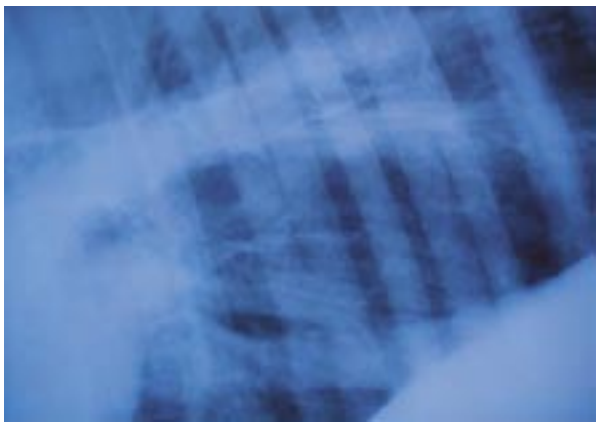

Figura 1. Thorax X-ray. Increase of the pulmonary density with diffuse interstitial and peribronchial pattern.

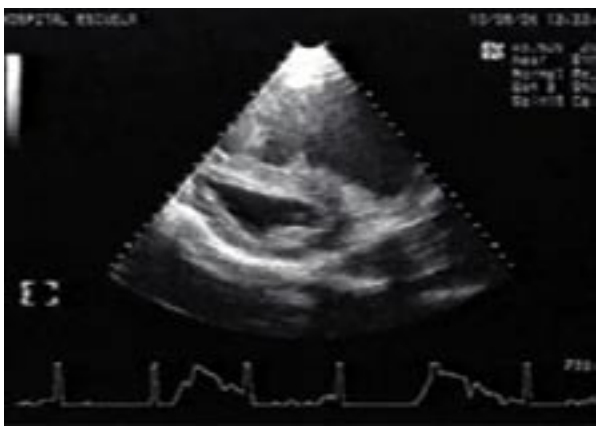

Figura 2. Short axis right paraesternal window. Cordae tendinae level view. The right ventricular cavity is enlarged and the interventricular septum is flattened due the right side hyperten-sion.

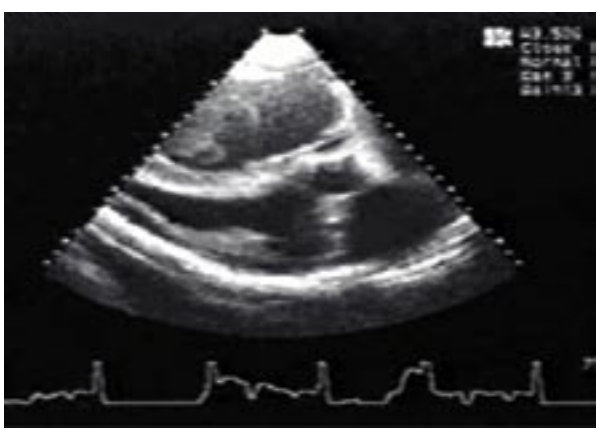

Figura 3. Long axis right paraesternal window. Fourth chambers view. The frame show ventricular systole. The interventricular septum bow toward left ventricle (paradoxical movement).

The peak velocity of the tricuspid flow wave (measured through spectral continuous Doppler) was 4.45 m/sec (pressure gradient of 79.21 mmHg). The evaluation of the transpulmonary flow through pulsated Doppler showed a peak velocity of 0.62 m/sec with an evident shortening of the time to peak velocity (0.06 sec), right ventricular pre-ejective periods prolongation (0.98 sec) and a right ventricular ejection time reduced (0.24 sec). The ascending branch of the transpulmonary flow wave showed a midsystolic notching due to the midsystolic closure of pulmonary valve typical of pulmonary hypertension (Figure 6).

Necropsy. Abdominal cavity and abdominal organs without particularities. Thoracic cavity: scarce pleural effusion. Lungs: congestive with adherent mucus in bronchus and bronchioles. Atelectatics areas and enfisematous bullae. Heart: right ventricle enlargement (Figure 7). Cardiac weight: 3.27 kg (1.65% body weight).

DISCUSSION

Cor pulmonale is a constellation of right heart morphologic and functional changes, most of which are echocardiographically detectable, occurring in conjunction with pulmonary hypertension secondary to chronic

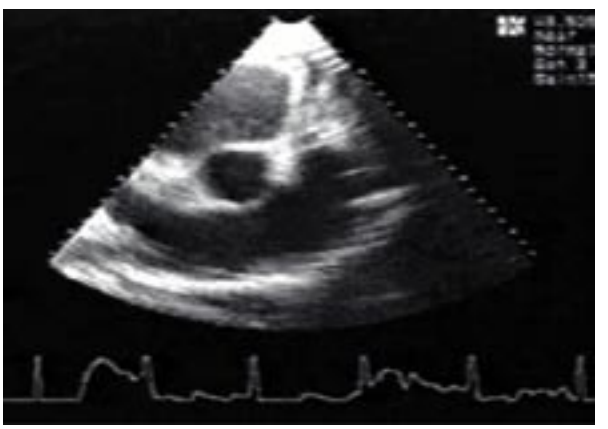

Figura 4. Short axis right paraesternal window. Cardiac base view. The diameter of the pulmonary artery is enlarged and the aortic / pulmonary ratio is altered.

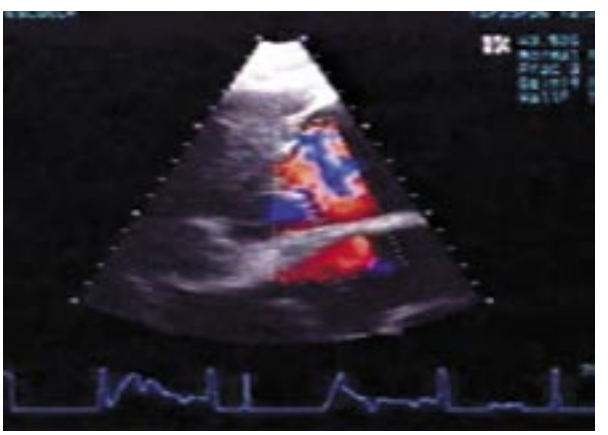

Figura 5. Color flow mapping at tricuspid level. The picture show the regurgitante flow.

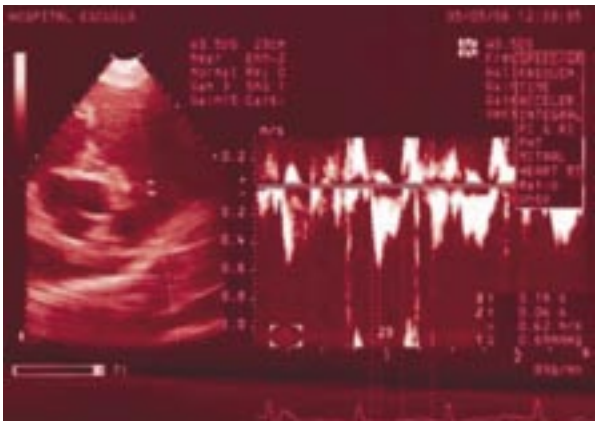

Figura 6. The picture shows the transpulmonary flow wave, interrogated with pulsated Doppler. The gate is positioned at pulmonary valve level. The right ventricular ejection time was 0.24 sec; the time to peak velocity (acceleration time) was 0.06 sec; the deceleration time was 0.18 sec and the acceleration time / ejection time ratio was 0.25. The ascendant branch of the flow wave show a notch due to the midsystolic closure of the pul-monary valve promote by the increase of pressure in the right side of the heart.

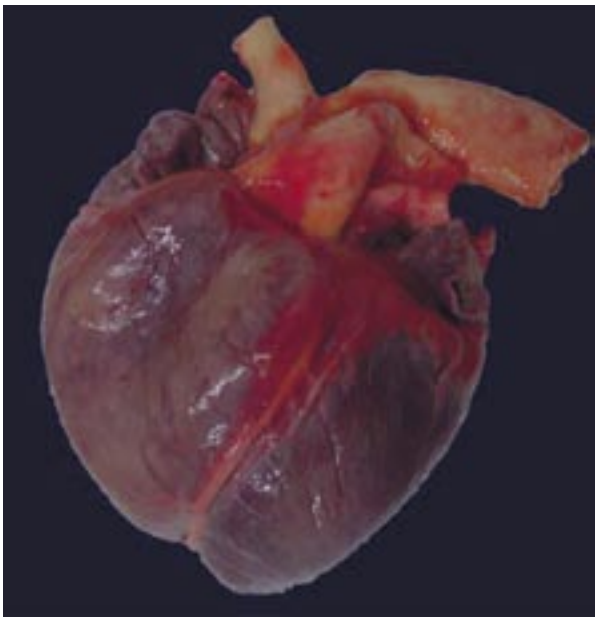

Figura 7. Is evident the right ventricle enlargement.

lung disease. The physiologic stimulus is exposure to chronically increased afterload in the pulmonary circuit. The syndrome manifest as hypertrophy of the right ventricular wall with diastolic compliance abnormalities in conjunction with chamber enlargement, systolic dysfunction and ultimate clinical right heart failure [4, 17] . The cor pulmonale has several etiologies [21] . In horses the most common is the chronic obstructive pulmonary disease [21, 22] . The literature describes a very low incidence of cor pulmonale in horses affected with COPD although it is unclear if this is due to lack of observation or a truly low incidence [7, 8] . Horses severely affected with COPD consistently develop pulmonary hypertension, but do not always develop right ventricular hypertrophy [5] .

References about echocardiographic two-dimensional signs of pulmonary hypertension exist in dogs [13] and equine [2] , but the references about Doppler ultrasound signs referred to cor pulmonale (and pulmonary hypertension) in horses are scarce. The right ventricular enlargement with a flattened interventricular septum and paradoxical septal motion secondary to right ventricular volume overload and dilated pulmonary artery are seen with severe pulmonary hypertension [6, 9, 10, 19] .

The Doppler ultrasound facilitates the cuali and quantitative evaluation of the pulmonary artery pressure [16] . Agreement exists that the quantification of the pressure in the pulmonary artery (ruled out the existence of valvular stenosis) can be carried out applying the Bernoulli equation to the velocity of the regurgitant flow of the tricuspid valve (when exists) [1, 11, 12, 15, 25] .

Other characteristics of the Doppler ultrasound evaluation permit to confirm the existence of pulmonary hypertension (although not quantify it) [3] . The measurement of the right ventricular pre-ejective period, the ejection time of the same ventricle, the time since the beginning of the flow to the point of the peak velocity (time to peak velocity) and the study of the profile of the ascending branch of the pulmonary flow wave permit to infer the presence and estimate the degree of the pulmonary hypertension [2, 9, 15, 16] .

When the right ventricle should increase its end-diastolic pressure to eject against an increased resistance (pulmonary bed hypertension) the pre-ejective period (isovolumetric contraction time) is prolonged, the ejection time is shortened and the peak velocity is reached prematurely due to premature decay of the pressure gradient which shortens the time of acceleration of the transpulmonary flow wave.

Other qualitative assessment of the pulmonary artery flow is also useful in identifying pulmonary hypertension as the mid-systolic decrease in flow or notch, due to the midsystolic closure of pulmonary valve [3, 23, 24] . This alteration in Doppler flow probably relates to reflected wave as a result of elevated pulmonary vascular resistance and systolic pressures. This sign indicate the presence, but not the exact severity of pulmonary hypertension.

In conclusion, we consider that the evaluation of the pulmonary pressure through the study of the flow velocity of the tricuspid regurgitation and the measurement of the time to peak velocity of the pulmonary flow and the evaluation of the profile of the ascending branch of the transpulmonary flow wave searching the corresponding notch due to mesosystolic closure of the pulmonary valve through the Doppler ultrasound, are sufficient for diagnosis of pulmonary hypertension in the horse.

REFERENCES

1. **Berger M, Haimowitz A, Van Tosh A**. 1985. Quantitative assessment of pulmonary hypertension in patients with

tricuspid regurgitation using continuous wave Doppler ultrasound. *J Am Coll Cardiol* 6: 359-365.

2. **Boon JA**. 1998. *Manual of Veterinary Echocardiography*, William & Wilkins, Baltimore, 447 p.
3. **Chan KL, Currie PJ, Seward JB, Hagler DJ, Mair DD, Tajik AJ**. 1987. Comparison of three Doppler ultrasound methods in the prediction of pulmonary artery pressure. *J Am Coll Cardiol* 9: 549-554.
4. **Commes KA**. 1995. Echocardiographic findings in acute and chronic pulmonary disease: differentiation from primary cardiac disease. In: *The Practice of Clinical Echocardiography* (Otto CM Ed.), Saunders, Philadelphia, p. 565-581.
5. **Davis JL, Gardner SY, Brooke IM, Schwabenton BS, Breuhaus BA**. 2002. Congestive heart failure in horses: 14 cases (1984–2001) *JAVMA* 220: 1512-1515.
6. **Demadron E, Bonagura JD, O´Grady MR**. 1985. Normal and paradoxical ventricular septal motion in the dog. *Am J Vet Res* 46: 1832-1841.
7. **Dixon P, Nicholls J, McPherson EA, Lawson GH, Thomson JR, Pirie HW, Breeze RG**. 1982. Chronic obstructive pulmonary disease anatomical cardiac studies. *Equine Vet J* 14: 80-82.
8. **Dixon PM, Railton DI, McGorum BC**. 1995. Equine pulmonary disease: a case control study of 300 referred cases. Part 3: Ancillary diagnostic findings. *Equine Vet J* 27: 428-435.
9. **Feigenbaum H**. 1994. *Echocardiography*, Lea & Febiger, Pennsylvania, 695 p.
10. **Fenely M, Gavaghan T**. 1986. Paradoxical and pseudoparadoxical interventricular septal motion in patients with right ventricular volume overload. *Circulation* 74: 230-238.
11. **Hatle L, Angelsen BA, Tromsdal A**. 1981. Noninvasive estimation of pulmonary artery systolic pressure with Doppler ultrasound. *Br Heart J* 45: 157-162.
12. **Kitabatake A, Inoue M, Asao M, Masuyama T, Tanouchi J, Morita T, Mishima M, Uematsu M, Shimazu T, Hori M, Abe H**. 1983. Noninvasive evaluation of pulmonary artery flow Doppler echocardiography. *Circulation* 68: 302-309.
13. **Lombard CW, Buergelt CD**. 1983. Echocardiography and clinical findings in dogs with heartworm-induced cor pulmonale. *Comp Cont Ed* 5: 971-980.
14. **Marr C**. 1999. *Cardiology of the Horse*, Saunders, London, 342 p.
15. **Miguere M, Escamill R, Coca F**. 1990. Pulsed Doppler echocardiography in the diagnosis of pulmonary hypertension in COPD. *Chest* 98: 280-285.
16. **Nanda NC**. 1993. *Doppler Echocardiography*, Lea & Febiger, Pennsylvania, 464 p.
17. **Otto CM**. 2000. *Textbook of Clinical Echocardiography*, Saunders, St. Luis, 528 p.
18. **Patteson M**. 1996. *Equine Cardiology*, Blackwell Science, Oxford, 264 p.
19. **Perlman AS, Clark CE, Henry WL**. 1976. Determinant of ventricular septal motion: influence of relative right and left ventricular size. *Circulation* 54: 83-91.
20. **Reef V**. 2000. *Equine Diagnostic Ultrasound*, Saunders, Philadelphia, 560 p.
21. **Robinson NE**. 1997. Pathogenesis and management of airway disease. *Proceedings of Am Ass Eq Pract* 43: 106-114.
22. **Sage AM, Valberg S, Hayen DW, Firshman AM, Jacob C**. 2006. Echocardiography in a horse with cor pulmonale from recurrent airway obstruction. *J Vet Intern Med* 20: 694–696.
23. **Turkevich D, Groves BM, Micco A, Trapp JA, Reeves JT**. 1988. Early partial systolic closure of pulmonic valve relates to severity of pulmonary hypertension. *Am Heart J* 115: 409-418.
24. **Weyman AE, Dillon JC, Feigenbaun H, Chang S**. 1974. Echocardiography patterns of pulmonary valve motion with pulmonary hypertension. *Circulation* 50: 905-909.
25. **Yock PG, Popp RL**. 1984. Noninvasive estimation of right ventricular systolic pressure by Doppler ultrasound in patients with tricuspid regurgitation. *Circulation* 70: 657-662.