Rupture of chorda tendineae of the tricuspid valve in a horse: a case report

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Abstract:

A 2-year-old cachectic cross-breed gelding was admitted to Veterinary Teaching Hospital of University of Tehran following the onset of a marked respiratory distress, coughing and ventral edema. Clinical examinations indicated harsh respiratory and expiratory sounds as well as jugular vein distention. The respiratory and heart rates were 35/min and 60 bpm, respectively. A grade III/IV pansystolic murmur with the PMI on the tricuspid valve, which could be heard on the left side, was detected. Sinus tachycardia was revealed by electrocardiography. Rupture of the chorda tendineae of the tricuspid valve, pulmonary artery regurgitation, pulmonary artery hypertension, tricuspid valve regurgitation, pulmonary artery, and right ventricular dilation were also found in echocardiography. These findings were confirmed at post mortem examinations. On the basis of the findings, chorda tendineae rapture of the tricuspid valve and right side heart failure due to primary pulmonary hypertension were diagnosed.

Case History

To control the blood flow, cardiac valves open and let the blood rush to the ventricles, and they close to prevent the blood from flowing backward (Bonagura and Reef, 2004; Sacs and Yaganathan, 2007). Involvement of the chorda tendineae of the atrio-ventricular valve apparatus can compromise proper closure of the entire valve (Kittleson and Kienle, 1998; Sisson et al., 1999). Both of the Valvular disorders, insufficiency and stenosis, can reduce cardiac output and increase cardiac work load (Radostis, et al., 2007). The degenerative type of valve disease has been reported as the most common cause of cardiac valves involvement in horses (Bonagura, 1995, Bonagura et al, 1985; Brown, 1985; Dedrick et al., 1988; Reef, 1987 and Reef et al., 1998). Endocarditis and degenerative valve diseases are the most common causes of tricuspid chordal rupture. In comparison with mitral condition, rupture of the tricuspid valve chorda tendineae is rare in horses (Bonagura and Reef, 2004; Else and Holmes, 1972; Holms and Miller, 1984; Reef et al., 1991 and Reimer and Reef, 1991). As far as literature shows, the present study is the first documented case of rupture of the chorda tendineae of the tricuspid valve secondary to chronic respiratory disease in horses.

Clinical Presentation

A 2-year-old cachectic crossbreed gelding with clinical signs of lassitude, movement disorder, respiratory distress, coughing, jugular vein distention, and ventral edema was presented to Veterinary Teaching Hospital, University of Tehran. Having no cardiovascular background, the veterinarian and the owner discussed the possibility of chronic respiratory disease. The veterinarian used Pantrisole (20 mg/kg), Flunexin meglumine (1.1 mg/kg), and Forusemide (1 mg/kg) to improve clinical sings. Body temperature was within the normal limit (37.9). On auscultation, the intensity of respiratory sounds was found to be

higher than normal. Also, respiratory movement was detected (35/min). The heart rate ranged from 50 to 60 bpm, and auscultation showed a grade III/IV pansystolic murmur with the point of maximal intensity (PMI) on the tricuspid valve area. This murmur could also be heard on the left cardiac area. A base-apex electrocardiogram was recorded on a single channel electrocardiogram machine (Fukuda 501-b-III) with a calibration of 10 mm equal to 1 millivolt and the paper speed of 25mm/sec. On the basis of the animal's age, clinical examination, and signs, ventricular septal defect (VSD), endocarditis, bronchopneumonia, and insufficiency of the right side of the heart were taken into consideration for differential diagnosis. M- Mode, B-Mode, and color Doppler echocardiograms were recorded on an echocardiograph machine (Micromax-Sonosit USA) using a phase array transducer with a frequency of 1 to 5 MH. Medical treatment on the basis of clinical signs was carried out with Penicillin (20000 IU/kg), Gentamicin (4.4 mg/kg), and Furosemide (2 mg/kg) for two days. Hematological and histhopathological examinations were also carried out.

Diagnostic Testing

Electrocardiography revealed sinus tachycardia (Figure 1). Hematological examinations showed leukocytosis (15300 per/µL), hyperfibrinogenemia (5.3 g/L), hypoalbominemia (21g/L), and hyperglobulinemia (45.4 g/L). The left ventricular internal diameters at the end of systole and at the end of diastole in M mode were 7.5 Cm and 9.3 Cm, respectively. Also, M mode echocardiography showed right ventricular internal diameter at the end of systole and at the end of diastole 3.8 Cm and 8.7 Cm, respectively. The prolapse and asynchronous movements of the chorda tendineae of the tricuspid valve at all phases of cardiac cycle were also found (Figure 2A). In B mode echocardiography, the diameter of the pulmonary artery at the pulmonic valve and aortic root diameter at the sinus of valsalva were 6.83 Cm and 4.75 Cm, respectively. Color Doppler echocardiography revealed pulmonary and tricuspid valves regurgitation (Figures 2B). The horse showed no improvement and was euthanized. Rupture of the septal cusp of the chorda tendineae of the tricuspid valve (Figure 3), bronchitis, bronchopneumonia, hepatic congestion, right ventricular, and right atrial dilation were confirmed at necropsy. Histopathological finding of the tricuspid valve was not remarkable. Neither echocardiography nor post-mortem examination showed mitral insufficiency or left side heart failure. On the basis of the findings, chorda tendineae rapture of the tricuspid valve and right side heart failure, due to primary pulmonary hypertension, were diagnosed.

Assessment

A case of ruptured chorda tendineae of the tricuspid valve in a horse is reported in the present study. Pulmonary regurgitation can be found in horses with congestive heart failure and pulmonary hypertension. Pulmonary artery dilation and right ventricular dilation are the echocardiographic findings in horses with moderate to severe pulmonic regurgitation (Bonagura and Reef, 2004; Reef, 1995 and Reimer and Reef, 1991). In this case, similar echocardiographic findings were diagnosed. Right side heart failure in horses has mostly been caused by pulmonary hypertension secondary to left side heart failure (Brown et al., 1983; Holms and Miller, 1984 and Miller and Holmes, 1985). Respiratory diseases are an uncommon cause of right side heart failure in horses (Dixon, 1978; Reimer and Reef, 1991). Increasing right ventricular pressure and tricuspid regurgitation has been reported in newborn infants with respiratory distress (Reller et al., 1987). Right side heart failure and pulmonary hypertension, due to pulmonic valve rapture, have also been reported in a horse (Reimer and Reef, 1991). Accordingly, pulmonary hypertension can result from a variety of causes such as left side heart failure, chronic pneumonia, or pulmonic valve rupture, which in turn overtaxes the right ventricle, and consequently results in right ventricle volume overload (Detweiler, 1996; Reimer and Reef, 1991). Dilation of the pulmonary artery, as well as the comparison of the diameter of the aortic root and the pulmonary artery, is used clinically as a noninvasive indicator of pulmonary hypertension in horses (Bonagura and Reef, 2004; Bonagura et al, 1985; Dedrick et al., 1988; Reef, 1987; Reef, 1995; Reef, 1998; Reef et al., 1998 and Reimer and Reef, 1991). Although pulmonary artery pressure was not accurately

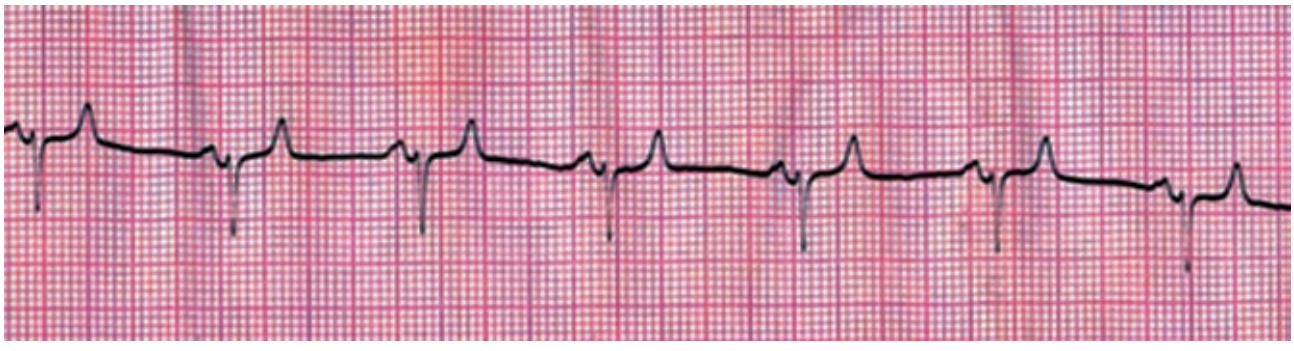

Figure 1. Base-apex electerocardiogram in the horse shows sinus tachycardia.

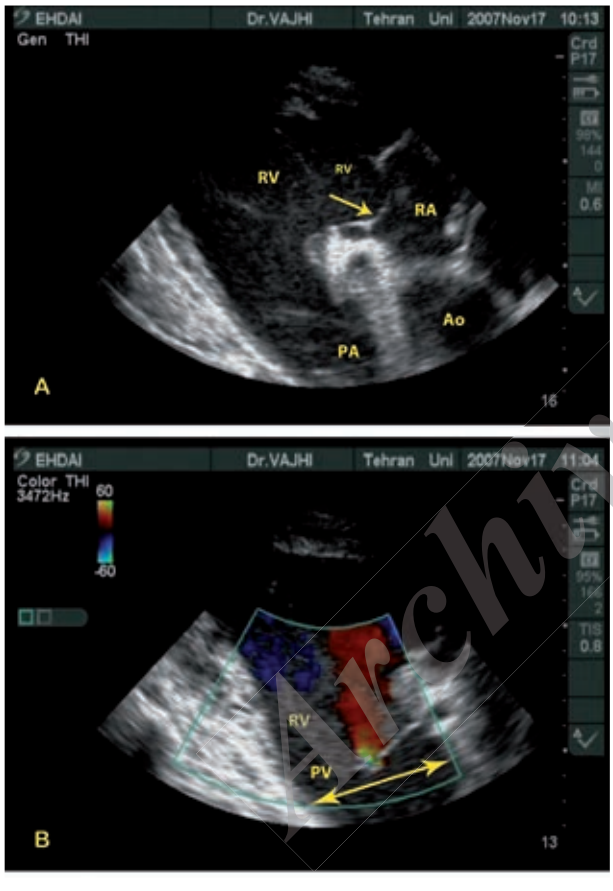

Figure 2(A). long-axis echocardiogram of the right ventricular inflow and outflow tracts from the right cardiac window shows right ventricular dilation and chorda tendineae rapture. The leaflet of the tricuspid valve (TV) prolapses (arrow) into the right atrium (RA). RV: right ventricle; AO: aorta, PA: pulmonary artery, RV: right ventricle. (B) Long-axis color-flow echocardiogram (right parasternal angled view). The yellow to red jet is deflected into the right ventricle (RV) outflow tract and shows pulmonary insufficiency and dilation (double arrow). PV: pulmonary valve.

determined in this report, comparing the large diameter of the pulmonary artery with the aortic root, which was detected in this report, supports the assumption of pulmonary hypertension. This may be due to chronic respiratory disease. In line with our study, pulmonary hypertension was diagnosed in horses with chronic respiratory disease (Dixon, 1978; Johansson et al., 2007). Ruptured chorda tendineae and flail tricuspid valve leaflet are rare in horses (Else and Holmes, 1972; Reef, 1995 and Reef et al., 1991). Conversely, chorda tendineae rupture of the mitral valve has been described in horses by several reports (Brown et al., 1983; Holms and Miller, 1984 and Reef, 1987). Endocarditis and degenerative diseases of the tricuspid valve are less common causes of the right side heart failure; however, they are considered to be common causes of tricuspid chordal rupture in horses (Bonagura et al, 1985; Else and Holmes, 1972; Reef, 1995 and Reef et al., 1991). Ruptured tricuspid chorda tendineae can be diagnosed in echocardiography by asynchronous or chaotic movement of the tricuspid leaflet from the right atrium to the right ventricle during all phases of the cardiac cycle (Bonagura et al, 1985; Reef, 1995 and Reef, 1998). Rupture of the chorda tendineae in human occurs spontaneously or due to congenital defects. Papillary muscle fibrosis is commonly considered as a cause of spontaneous rupture of the chorda tendineae (Braunwald, 1988). Tricuspid insufficiency and rupture of the chorda tendineae of the tricuspid valve have been previously reported in human (José dos Santos et al., 2001). In the present report, the histopathological results of the tricuspid valve were not remarkable; medical history, clinical sings,

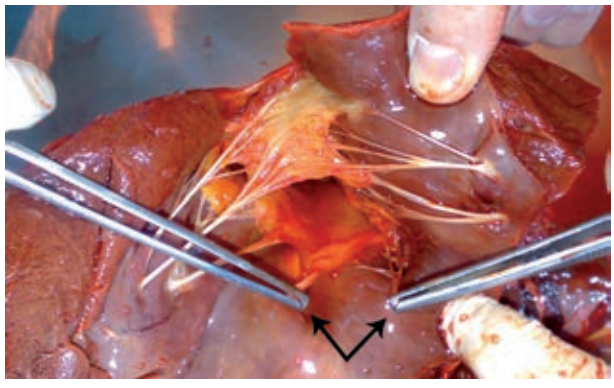

Figure 3. Postmortem photograph of the case shows a septal cusp ruptured chorda tendineae of the tricuspid valve (arrows).

echocardiographic, and necropsy findings strongly indicated that this might have occurred due to a primary pulmonary hypertension, secondary to chronic respiratory involvement. It is believed that this phenomenon might lead to right ventricular volume overload and increased right atrial pressure. These caused dilation of the tricuspid annulus, as well as altered papillary muscle support, and finally rupture of the chorda tendineae of the tricuspid valve. In this situation, the blood pressure of the right atrium increases suddenly, but there is not enough time for compensation. Therefore, ventral edema, hepatic congestion, and other clinical signs can occur. In this respect, in a study of 28 dogs with spontaneous chorda tendineae rupture, the authors described that one- third of the dogs had a history of acute pulmonary edema without any known pre-existing heart disease (Ettingerand and Buergelt, 1969). Pulmonary hypertension, cardiomyopathy, and myocarditis can also lead to the secondary dilation of tricuspid annulus or alteration of papillary muscle support (Bonagura and Reef, 2004). The effects of pulmonary hypertension on the left and right ventricles have been properly described (Chin et al., 2005; Juang et al., 2009, and Marcus et al., 2000).

In conclusion, Chorda tendineae rupture of the tricuspid valve is an uncommon cause of heart failure in horses, which might be mistaken with other cardiovascular disorders based on the age, medical history, and clinical sings. Therefore, echocardiography may be useful as a noninvasive method for differential diagnosis. In a horse with severe pulmonic regurgitation and right ventricle dilation_ and with no evidence of left side heart failure_ addition of pulmonic valve rupture and chorda tendineae rupture of tricuspid valve should also be considered in echocardiography. Based on literature review, the present study could be the first published case of rupture of the chorda tendineae caused by primary pulmonary hypertension, secondary to chronic respiratory disease in a horse.

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رخداد پارگی طناب‌های وتری دریچهٔ سه‌لتی در اثر افزایش فشار خون اولیهٔ دریچهٔ ریوی در یک رأس اسب: گزارش موردی

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چکیده

یک اسب نر دوساله با تاریخچهٔ دیسترس تنفسی، سرفه و ادم ناحیهٔ شکم به بیمارستان آموزش و پژوهشی دانشکدهٔ دامپزشکی دانشگاه تهران ارجاع داده شد. در معاینات بالینی رگ‌های وداجی به صورت دوطرفه متسع و افزایش شدت صدای تنفسی دم و بازدمی در همهٔ قسمت‌های منطقهٔ ریه به گوش می‌رسید. تعداد تنفس و ضربان قلب به ترتیب در محدودهٔ ۳۵ و ۶۰ عدد در دقیقه بود. سمع قلبی یک سوفل پانسیستولیک درجهٔ III/IV با شدت بیشینه روی دریچهٔ سه‌لتی را برملا کرد که در سمت چپ هم شنیده می‌شد. نوار الکتروکاردیوگرام این اسب تاکی‌کاردی سینوسی را نشان داد و اکوکاردیوگرافی پارگی طناب‌های وتری دریچهٔ سه‌لتی، برگشتی جریان خون از دریچهٔ ریوی و سه‌لتی، اتساع و افزایش فشار خون ریوی و اتساع بطن راست را نشان داد. کالبدشایی حیوان یافته‌های بالینی گفته‌شده را تأیید کرد. بر اساس یافته‌های بالینی پاره‌شدن طناب‌های وتری دریچهٔ سه‌لتی و نارسایی قلب راست در اثر فشار خون اولیه دریچهٔ ریوی تشخیص داده شد.

واژه‌های کلیدی: طناب‌های وتری، اکوکاردیوگرافی، اسب، افزایش فشار خون ریوی، نارسایی دریچه ریوی

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