

TRICUSPID VALVE DYSPLASIA AND ITS THERAPEUTIC MANAGEMENT IN A DOG

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[Received:24.7.2014; Accepted: 25.5.2015]

Three year male Labrador dog weighing about 32 kg was presented with primary complaints of anorexia, severe ascites, exercise intolerance and weakness. Right side heart enlargement was evident on thoracic radiography, while electrocardiogram revealed sinus tachycardia, increased duration of P wave and QRS complex, right electrical axis shift and suppression of R wave amplitude. Echocardiography confirmed the enlargement of the right atrium and right ventricle along with tricuspid dysplasia. Based on the findings of diagnostic investigations, the case was diagnosed as tricuspid valve dysplasia, regurgitation. The dog was medically managed with furosemide, enalapril, spironolactone and pimobendan.

Key words: Tricuspid valve, dysplasia, dog, management.

Introduction

Tricuspid valve dysplasia (TVD) is a congenital malformation of the tricuspid valve, chordae tendineae, or papillary muscles, resulting in tricuspid insufficiency (Friedman, 1999), causing valvular regurgitation (Hyun, 2005). Most common etiology of TVD in dogs is underdevelopment of chordae tendineae, which causes the direct attachment of papillary muscles to the valve leaflets (Kittleson, 1998). Dogs with tricuspid dysplasia commonly have absence of or very short fused chordae tendineae and long thick septal leaflets adherent to the septum (Liu and Tilley, 1976). Tricuspid leaflets may have little movement which appear to be very large, or be adhered to the inter-ventricular septum or to the papillary muscles (Hirschklau *et al.*, 1977). Right-sided congestive heart failure is the end-result and is characterized by ascites, hepatomegaly and/or jugular vein distension (Nam *et al.*, 2008). Reported occurrence of this abnormality ranges from 7.0 to 7.5 percent (Baumgartner and Glaus, 2003) and 3.1 percent (Oliveira, *et al.*, 2011) of all congenital heart defects. Up to 25.8 percent breed incidence was reported for Labrador retrievers (Kittleson, 1998), which was thought to be a heritable disorder (Famula *et*

Indian Journal of Canine Practice

al., 2002). The diagnosis is usually based on clinical signs and ancillary exams (Sousa *et al.*, 2006) like radiography, electrocardiography and echocardiography. Diagnostic features of TVD include cardiomegaly with massive right atrium enlargement on the thoracic radiography (Hoffmann *et al.*, 2000), right apical systolic murmur (Hyun, 2005), high P waves, deep S waves in leads I, II, III, and aVF, and splintered QRS complexes on the ECG (Kornreich and Moïse, 1997), markedly enlarged right atrium and right ventricle, often smaller size left atrium and left ventricle than normal, and abnormal tricuspid leaflet adherence to the inter-ventricular septum or to the papillary muscles (Hirschklau *et al.*, 1997) on the echocardiography, and a turbulent regurgitant flow into right atrium during systole on the color spectral Doppler studies (Kittleson, 1998). Present case describes tricuspid valve dysplasia and its therapeutic management in a dog.

Case history and clinical observation

Three year old male Labrador dog weighing about 32 kg was referred to Chandrika Chimanlal Doshi Cardiovascular Unit of Department of Veterinary Clinical

Volume 7 Issue 1, June, 2015

Medicine, Bombay Veterinary College, Mumbai for evaluation of signs of anorexia, weakness, ascites and exercise intolerance. Physical examination revealed lethargy, tachycardia (155 beat/min) along with pale mucous membranes. Thoracic auscultation over the right apex revealed a loud 4/6 holosystolic heart murmur. Abdominal palpation did not permit delimitating internal structures due to severe ascites. Interpretation of routine hemato-biochemical data revealed anemia (2.5×10^6 erythrocytes/ μ l), thrombocytopenia (85×10^3 platelets/ μ l), hypoproteinemia (4.6 g/dl), hypoalbuminemia (2.2 g/dl), and increase in serum alanine aminotransferase (203U/l), aspartate aminotransferase (243U/l), alkaline phosphatase (745 U/l), blood urea nitrogen (25.8 mg/dl) and serum creatinine (1.9 mg/dl). Congenital tricuspid dysplasia was suspected due to age, severe ascites, exercise intolerance and the loud heart murmur over the tricuspid area that dog underwent for further evaluation by radiography, ECG and echocardiography.

Radiographic studies of the thoracic and abdominal cavities revealed a globoid cardiac shadow, right atrial enlargement (Fig. 1 and 2) in lateral and ventro-dorsal views

along with increased cardiac sternal contact and ascites, suggesting right-sided congestive heart failure. Electrocardiogram revealed sinus tachycardia (190 bpm), increased duration of P wave (0.06 ms) and splintered QRS complex (0.08 ms), right electrical axis shift, and suppression of R wave amplitude (0.47mV). Echocardiography showed very large and extensively dilated right atrium with very small displaced left atrium and left ventricle. There was paradoxical movement of inter-ventricular septum and tricuspid valve along with displacement of leaflets below normal atrioventricular ring with partial adhesion of septal cusp with atrioventricular ring (Fig. 3,4,5 and 6) There was no evidence of any atrial and ventricular septal defect. Large size RA changed the normal anatomy of heart in the thoracic cavity which posed difficulty in echocardiographic imaging. Dog was given furosemide (2mg/kg, im, tid), enalapril (0.5mg/kg, po, bid), and spironolactone (1.0 mg/kg sid) and pimobendan (0.25mg/kg, po, bid). After one week when ascites was reduced, the dose of furosemide was reduced to 1.5 mg/kg, im, sid., for next two weeks, dog was kept on enalapril (0.25 mg/kg) and pimobendan (0.25mg/kg, po, bid).

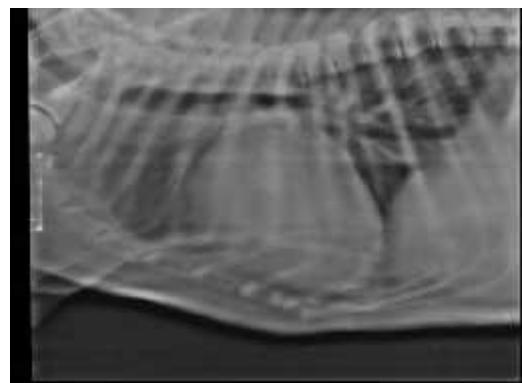


Fig 1-2 Dorso-ventral and lateral radiograph showing right side cardiac enlargement

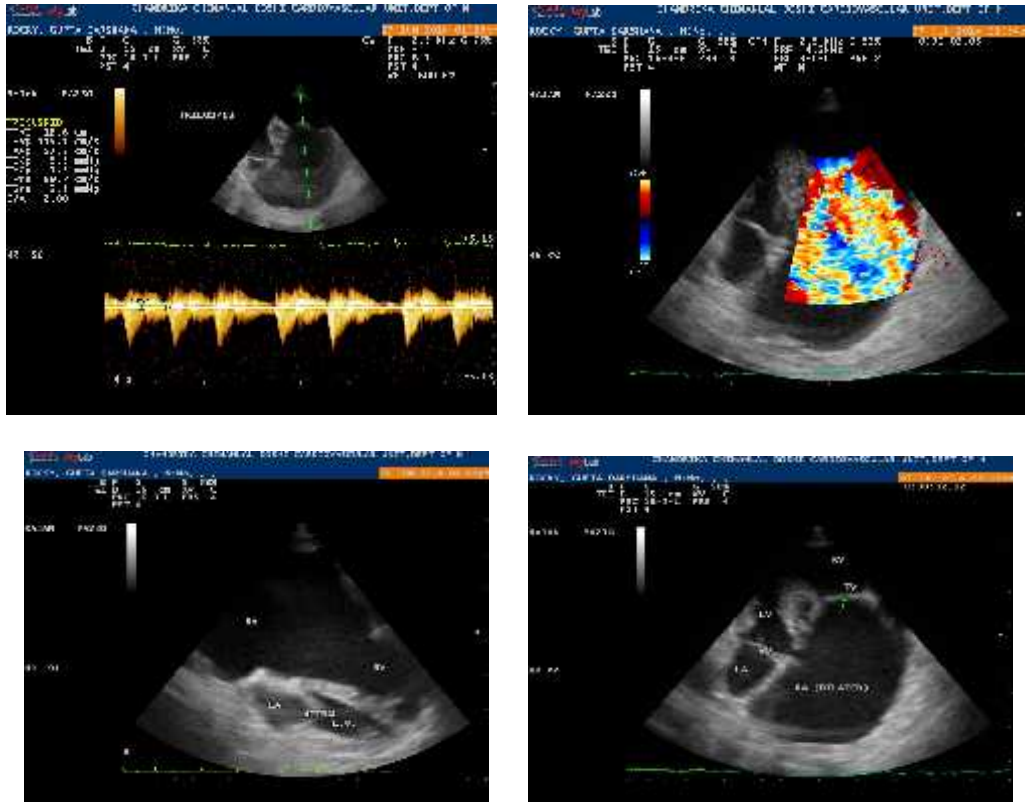


Fig-3,4,5 and 6 echocardiograph showing right side enlargement, tricuspid dysplasia and regurgitation

Discussion

The dog was presented with symptoms of ascites, lethargy, exercise intolerance and weakness which are classical features related to right congestive heart failure, due to tricuspid valve insufficiency (Sisson *et al.*, 2000) along with holosystolic heart murmurs over the right apex. (Chetboul *et al.*, 2004). Thoracic radiography showed right atrium enlargement as reported previously (Hoffmann *et al.*, 2000). Kittleson (1998) reported that a massive right atrium enlargement in a young animal with right apical systolic murmur is generally pathognomonic for severe tricuspid dysplasia. Besides, right-axis deviation and increased duration of P wave (0.06 ms) and splintered QRS complex (0.08 ms), there were no other electrocardiographic evidences of right heart enlargement. Sinus tachycardia was probably related to the activation of neuro-humoral responses to overwhelm the leakage of blood into the right atrium and the decrease in pulmonary stroke volume (Kittleson, 1998).

The ECG proved to be not too sensitive and specific diagnosis of this condition (Kornreich and Moise, 1997). Most striking finding on echocardiography is right atrial enlargement (Chetboul *et al.*, 2004). Alterations of the tricuspid valve seen in this case were similar to reports by previous workers that included thickened and notched leaflets with little movement (Cave, 2001). The diagnosis of tricuspid dysplasia was confirmed by Doppler interrogation of tricuspid valve, in which a large turbulent jet into the right atrium during ventricular systole was observed (Friedman, 1999).

Medical treatment is usually directed to improve quality of life by medically reducing the amount of abdominal fluid, for which diuretics and angiotensin converting enzyme inhibitors (ACE) are commonly employed (Tilley *et al.*, 2011). The dog was put on furosemide, enalapril and pimobendan to overcome compensatory mechanisms. Spironolactone was also given to competitively inhibit the action of aldosterone

on distal tubular cells, thereby, increasing fractional excretion of sodium and water (Sousa *et al.*, 2006). Complete stopping of fluid accumulation is often not effective and necessary on the view of long-term management (Kittleson, 1998), therefore most of the TVD cases required periodic abdominocentesis and fluid removal. However, periodic removal of abdominal fluid causes substantial loss of plasma albumin, although, it rarely results in significant complication. However, other medications (e.g. inotropes, venous dilators, spironolactone) may be helpful to slow down the fluid accumulation, as seen in this case. Due the severity of the present case, the combination therapy using furosemide and ACE was not enough for the retardation of fluid accumulation. After the addition of inotropes, it may delay the further accumulation of ascitic fluid. Therefore, on the basis of therapeutic management, it was suggested that addition of inotropes might be the good option for slowing fluid accumulation frequently seen in the right sided heart failure.

References

- Baumgartner, C. and Glaus, T. (2003). Congenital cardiac diseases in dogs : A retrospective analysis. *Schweiz. Arch. Tierheilkd.*, **145**(11):527-33, 535-36.
- Cave, T. (2001). What was your diagnosis? Congenital tricuspid dysplasia with concurrent Ebstein's anomaly. *J. Small Anim. Pract.*, **42**: 311-312.
- Chetboul, V., Tran, D. and Carlos, C. (2004). Congenital malformations of the tricuspid valve in domestic carnivores: a retrospective study of 50 cases. *Schweiz. Arch. Tierheilkd.*, **146**: 265-275.
- Famula, T.R., Siemens, L.M., Davidson, A.P. (2002). Evaluation of the genetic basis of tricuspid valve dysplasia in Labrador Retrievers. *Am. J. Vet. Res.*, **63**: 816-820.
- Friedman, W.F. (1999). Cardiopatia congênita na lactância e infância. In: Braunwald, E. Tratado de medicina cardiovascular. *São Paulo: Roca*, **29**: 935-1027.
- Hirschklau, M.J., Sahn, D.J., Hagan, A.D., Williams, D.E. and Friedman, W.F. (1997). Cross-sectional echocardiographic features of Ebstein's anomaly of the tricuspid valve. *Am. J. Cardiol.*, **40**: 400-404.
- Hoffmann, G., Amberger, C.N., Seiler, G. and Lombard, C.W. (2000). Tricuspid valve dysplasia in fifteen dogs. *Schweiz. Arch. Tierheilkd.*, **142**: 268-277.
- Hyun, C. (2005). Small Animal Cardiology. Korean Edition, Panmun Book, Seoul., Pp. 186-191.
- Kittleson, M.D. (1998). Congenital abnormalities of the atrioventricular valves. In: Kittleson MD, Kienle RD (eds.). Small Animal Cardiovascular Medicine. Mosby, St. Louis. Pp. 273-281.
- Kornreich, B.G. and Moise, N.S. (1997). Right atrioventricular valve malformation in dogs and cats: an electrocardiographic survey with emphasis on splintered QRS complexes. *J. Vet. Intern. Med.*, **11**:226-230.
- Liu, S.K. and Tilley, L.P. (1976). Dysplasia of the tricuspid valve in dog and cat. *J. Am. Vet. Med. Assoc.*, **169**:623.
- Oliveira., P., Domenech, O., Silva, J., Vannini, S., R. Bussadori, R. and Bussadori, C. (2011). Retrospective Review of Congenital Heart Disease in 976 Dogs. *Vet. Int. Med.*, **25**(3): 477-483.
- Sisson, D.D., Thomas, W.P. and Bonagura, J.D. (2000). Congenital heart disease. In: Ettinger, S.J.; Feldman, E.C. Textbook of Veterinary Internal Medicine: Diseases of the dog and cat. Philadelphia: Saunders, Pp. 737- 787.
- Sousa, M.G., Gerardi, R.O. and Alves, A.A. (2006). Camacho Tricuspid valve dysplasia and Ebstein's anomaly in dogs: case report. *Arq. Bras. Med. Vet. Zootec.*, **58**(5):762-767.

Tidholam, A. (1997). Retrospective study of congenital heart defects in 151 dogs. *J. Small Anim. Pract.*, **38**: 94-98.
 Tilley, L.P., Francis W.K. and Smith, Jr.

(2011). Blackwell's Five-Minute Veterinary Consult: Canine and Feline, Fifth Edition, John Wiley & Sons, Pp. 522-523.

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