CASE REPORT
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CONGENITAL SUB-CRISTAL VENTRICULAR SEPTAL DEFECT IN A PUP

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ABSTRACT

A three-month-old male pug was presented with the complaint of syncopal episodes. Clinical examination revealed cyanotic mucosa, systolic murmur, weak pulse, with sinus tachycardia, p-pulmonale, increased R amplitude and ST elevation on electrocardiogram. On detailed cardiologic evaluation, altered cardiac silhouette, sluggish ventricular contractility, enlarged atria and ventricles, severe turbulent flow on the interventricular septum, increased CPK, hyponatremia and hypoproteinemia were found. Therapeutic management with enalapril, digoxin, diltiazem and lasilactone were attempted. The animal succumbed to death following episode of syncope at home. The post-mortem disclosed cardiomegaly and a defect in the membranous portion of the interventricular septum. Based on the post-mortem findings, the case was diagnosed as ventricular septal defect.

Keywords: Cardiac disorder, Syncope, Ventricular septal defect

INTRODUCTION

Congenital heart defects refer to the abnormalities in the morphology and functionality of the heart and the adjacent great vessels that are present at the time of birth, usually occurring as a result of defective embryonic development. Most of the defects are detected soon after the birth. But, some animals appear normal initially, which may be attributed to the developing hemodynamic changes during the first few months after birth (Ettinger and Feldman, 2010). The defective hole being located at the upper interventricular membranous septum involving aortic root and septal tricuspid valve root is known as Sub-cristal ventricular septal defect (VSD).

Tilley *et al.* (2008), reported that Patent ductus arteriosus (PDA), aortic stenosis, pulmonary stenosis. Tetralogy of fallot and ventricular septal defect as the most common congenital defects in dogs. This paper reports a congenital occurrence of Ventricular septal defect with associated

cardiac changes in three months old male pug.

CASE HISTORY AND OBSERVATIONS

A three month old, male dog (pug) with a body weight of 4.2 kg was reported with an episode of syncope just after heavy exercise every day for the past one month. The animal was usually fed on rice, pedigree and chicken and housed in a cage. On detailed clinical examination, cyanosis of the visible mucous membrane and weak pulse were recorded. Temperature and respiration rate were noted within the normal range. On cardiac auscultation, a to and fro murmur could be heard on both right and left thoracic region over the heart area and bronchoalveolar sounds were noted on lung auscultation. In hospital electrocardiogram, sinus tachycardia, p-pulmonale, increased R-amplitude, deep Q-wave in lead I, III and ST elevation which points towards right atrial and bi-ventricular enlargement and myocardial hypoxia were observed (Fig. 1). No arrhythmia could be detected during the five minutes routine ECG. According to Tilley *et al.* (2008), cardiomyopathy does not get ruled out on normal ECG which may be because of uniformed enlargement of each chamber of the heart. Chest radiography revealed changes in the cardiac silhouette, increase sternal contact with a VHS of 11.2 indicative of cardiomegaly. B mode echocardiogram showed sluggish left ventricular contractility, enlarged left and right ventricles and right and left atria. Doppler echocardiogram conveyed severe tricuspid regurgitation, and turbulent flow was noted on the interventricular septum close to the left ventricular outflow tract, connecting both the ventricles (Fig. 2). Left ventricular functional indices were recorded and is given in the **Table 1.**

All the values got markedly decreased beyond the normal limits especially the stroke volume, ejection fraction and fractional shortening. Sub normal values of SV, EF and FS may be the leading cause of syncopal episode when the animal exhibits heavy activity. Complete blood count depicted no abnormalities (Table 2). Serum biochemistry revealed increased creatine phosphokinase values that may be owed to muscular damage of cardiac origin, and reduced total protein level and sodium level (Table 2). Hypoproteinemia might be which might be due to decreased absorption or loss owing to bowel edema (Wotton, 2010) and sodium influx into the cardiac muscle for depolarization might be the cause for hyponatremia (Sesh *et al*., 2013).

TREATMENT AND DISCUSSION

Based on the clinical signs, electrocardiogram, echocardiogram and radiographic findings a clinical tentative

Table 1. Echocardiogram findings

Sl. No	Indices	Values	Normal Values
01	IVSd	1.10 cm	0.5 cm
02	LVIDd	1.16 cm	2.0 cm
03	EDV	3.03 mL	--
04	IVSs	1.21 cm	1.0 to 1.7 cm
05	LVIDs	1.04 cm	1.3 cm
06	ESV	2.29 mL	--
07	SV	0.75 mL	1.15 – 1.82 ml
08	EF	24.65 %	50-65 %
09	FS	10.00 %	27-48 %

(Ettinger and Feldman, 2010 and Rourke and Bishop, 1971)

diagnosis was made as congenital ventricular septal defect. The owner was informed about the poor prognosis of the condition. The animal was given the following medications to therapeutically manage the condition; tablet Enalapril - 0.25 mg per kg BID, tablet Lasilactone (furosemide and spironolactone) - 2 mg per kg BID, tablet carvedilol - 0.1 mg per kg OD and tablet digoxin - 0.001 mg per kg OD were prescribed. For the next few days (after 1.5 week), owner reported increased activity level which progressed to four to five times of syncopal episodes per day and developed ascites within a week. After two weeks, the animal succumbed to death at home after showing a few syncopal episodes.

The post mortem report revealed globular heart (cardiomegaly), non-uniformity of the mitral valve and a 6 mm hole in the membranous portion of the interventricular septum. The hole formed a bridge between the aortic root and sub tricuspid valve connecting both the ventricles (Fig. 3.). Based on the PM report, diagnosis was confirmed as interventricular septal defect at the membranous portion of interventricular septum.

Table 2. Hematology and Serum Biochemistry findings

Sl. No	Parameters	Values	Normal values
01	WBC	13.9 x 10^3/ uL	5-14.1 x 10^3/ uL
02	RBC	7.31 x 10^6/uL	4.95-7.87 x 10^6/uL
03	HBG	17.0 g/dL	11.9-18.9 g/dL
04	PCV	52.9 %	35-57 %
05	PLT	226 x 10^3/uL	211-621 x 10^3/uL
06	Creatinine	0.89 mg/dL	0.5-1.7 mg/dL
07	ALT	72.0 IU/L	10-109 IU/L
08	Total protein	4.2 g/dL	5.4-7.5 g/dL
09	Albumin	2.5 g/dL	2.3-3.1 g/dL
10	CPK	840.6 IU/L	52-368 IU/L
11	Sodium	135.5 mEq/L	142-152 mEq/L
12	Potassium	5.15 mEq/L	3.9-5.1 mEq/L

(Merk, 2016)

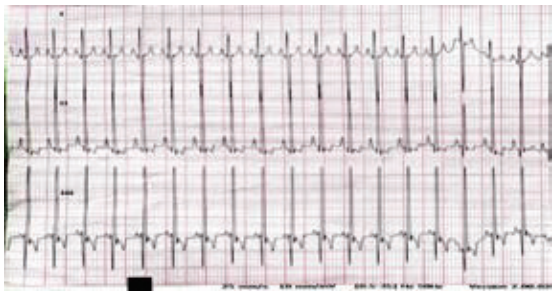

Fig 1. Electrocardiogram: P-pulmonale, increased R amplitude, deep Q wave in lead I and III, ST elevation indicative of bi-atrial and bi-ventricular enlargements and myocardial hypoxia.

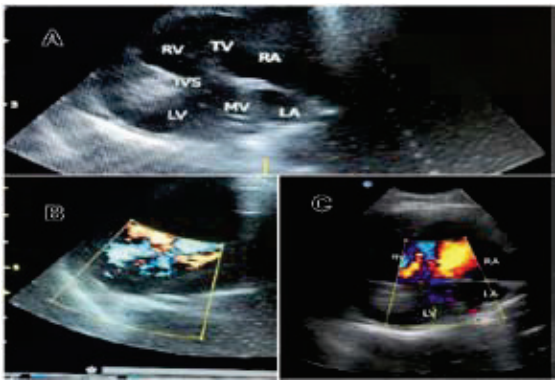

Fig.2. Echocardiographic picture showing enlarged left ventricle (LV), left atrium (LA), right ventricle (RV) and right atrium (RA). B mode echocardiography displaying intraventricular septal defect (IVSD) (A). Colour doppler echocardiogram shows IVSD (B) and severe tricuspid regurgitation (C).

In the present case study, the size of the defect was measured to be 6mm (0.6 cm) which is quite large to affect the pressure equilibrium among the heart chambers. Severely dilated right atrium and ventricle was shown on echocardiogram with evidence of severe tricuspid regurgitation (Fig.1). In accordance to the study of Bomassi *et al*. (2015) mild aortic regurgitation was noticed in the present

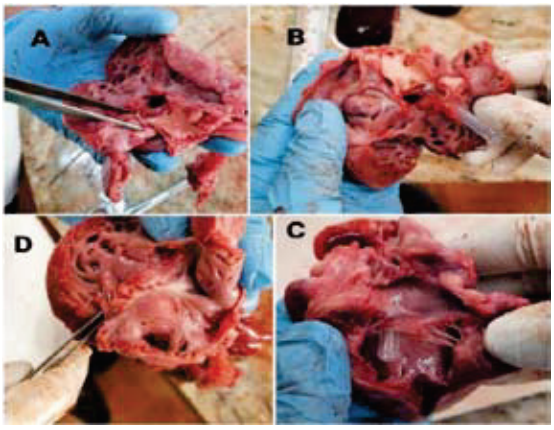

Fig 3. Postmortem pictures of Heart: A. VSD on the left side; B. Connection of VSD with the aortic root; C. VSD on the right side cranial to the septal leaflet of tricuspid valve; D. Irregular or non-uniform borders of mitral valve.

case. Left to right shunting might have predisposed to pulmonic valve insufficiency which in turn may be a leading cause of tricuspid regurgitation. All these system failures headed to reduce the inflow of oxygenated blood from the lungs resulted in reduce stroke volume and subnormal systemic circulation. Syncope is a transient loss of consciousness due to inadequate blood supply to the brain. Syncopal episodes during heavy activity, weak pulse and cyanotic mucous membrane may be a reflection of less blood supply owing to weak contractility of left ventricle and less stroke volume. Breeds predisposed or prone to VSD were reportedly Beagle, Chow Chow, Husky, English Bulldog, Terrier and Poodle and the most common location for VSD is at the membranous portion of the septum (Tilley *et al*., 2008).

CONCLUSION

Ventricular septal defect may be defined as the anatomical discontinuation of the interventricular septum which separates the left from the right ventricle. Ventricular septum is muscular at the apex and tappers as membranous at the base of the heart. VSD is more commonly formed at the membranous portion. The direction of the blood flow depends on the ventricular pressure and the size of the defect. Clinical diagnosis could be achieved by doppler echocardiography which could be evident by turbulent blood flow at the interventricular region. Inefficient cardiac function and hemodynamic changes due to VSD may be managed therapeutically. The Prognosis of the condition is usually grave. Studies regarding early disease diagnosis based on mild signs of the disease and its management would require. Surgical correction of VSD may have better prognosis which will be available in high facility or well-equipped hospitals.

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