International Journal of Science, Environment and Technology, Vol. 8, No 1, 2019, 70 – 75

ISSN 2278-3687 (O)
2277-663X (P)

MEDICAL HISTORY AND CLINICAL FEATURES OF ACQUIRED HEART DISEASES IN DOGS: 106 CASES

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Abstract: Acquired heart diseases (AHD) are common and often fatal when it leads to CHF in dogs and it occurs most often secondary to degenerative Mitral Valve Disease (MVD), Dilated Cardio Myopathy (DCM), Pericardial diseases and Hypertrophic Cardio Myopathy (HCM). Animals with acquired heart diseases were selected from the animals that were brought to MVC teaching hospital and they were grouped as Dilated Cardiomyopathy (DCM), Mitral Valve Disease (MVD), Pericardial diseases, Hypertrophic Cardiomyopathy (HCM). 106 animals with acquired heart diseases were selected and they were grouped as Dilated Cardiomyopathy (DCM), Mitral Valve Disease (MVD), Pericardial diseases, Hypertrophic Cardiomyopathy (HCM). The observed chief complaints included inappetance, exercise intolerance, abdominal enlargement, syncope and weakness. Tachycardia, ascites and murmurs were the common clinical signs in all the groups of AHDs.
Keywords: Canine, DCM, MVD, HCM, History, Clinical Signs.

INTRODUCTION

Acquired heart diseases are the major silent killers in dogs, similar to human beings. Acquired heart diseases (AHD) are common and often fatal when it leads to CHF in dogs characterized by cardiac dysfunction, neuro-hormonal activation, sodium and water retention and increase in left ventricular (LV) filling pressures (LVFP). It occurs most often secondary to degenerative mitral valve disease (MVD), dilated cardiomyopathy (DCM) and pericardial diseases. Hypertrophic cardiomyopathy (HCM) is another AHD which is a rare form of heart muscle disease in dogs.

MATERIALS AND METHODS

All the selected animals were subjected to routine clinical examination comprising of physical examination as suggested by McCurin and Poffenbarger (1991) and detailed cardiovascular assessment as suggested by Tilley (1992) and Ware (2007).

Clinical Presentation: Chief complaints, age at onset, management practices, medication history and chronology of events were assessed. Appetite, physical activity, dyspnoea, abdominal enlargement, pulsation and additional clinical findings were assessed. Lethargy,

*Received Dec 12, 2018 * Published Feb 2, 2019 * www.ijset.net*

weakness, exercise intolerance, syncope, weak femoral pulse with pulse deficit, tachycardia, concurrent progressive or refractory congestive heart failure were assessed through clinical examination

RESULTS AND DISCUSSION

The nature of presenting complaints is presented in Table-I.

In the present study the major history and clinical signs in DCM were abdominal distension, exercise intolerance, weight loss, persistent cough, weakness, dyspnoea and syncope. The above findings are in agreement with Fisher, (1972); Tidholm *et al.,* (1997); Bulmer, (2006); Erling and MazzaFerro, (2008); and Fox, (1988).

In MVD persistent cough might be due to compression of left main stem brochi the major finding in our study, which is similar to the findings of Bonagura and Frank (1983), Kittleson and Kienle (1998) and Kvart and Häggström (2000).

In HCM group, syncope alone was the predominant complaint. This concurs with Thomas, (1987). In pericardial effusion group, signs were similar to DCM group. History reported in the study may be attributed to systolic and/or diastolic failure, dilated left atrium might be due to regurgitation; pulmonary oedema might be due to poor cardiac performance and circulatory collapse might be due to reduced cardiac output. Major study findings are in agreement with many authors (Meurs *et al.,* 2001; Martin *et al.,* 2009; and Martin *et al.,* 2010; Darke, 1985; Tidholm and Jonsson, 2005; and Martin *et al.*, 2009).

Table-I: Medical History in Dogs with Acquired Heart Diseases (AHD)

Chief Complaints	Group – II Dilated Cardiomyopathy (n=58)	Group – III Mitral Valve Disease (n=39)	Group – IV Pericardial Effusion (n=6)	Group – V Hypertrophic Cardiomyopathy (n=3)
Inappetance (%)	72.41	30.77	33.33	66.67
Exercise intolerance (%)	68.97	51.28	33.33	66.67
Dyspnoea (%)	43.10	25.64	16.67	66.67
Abdominal enlargement (%)	86.21	12.82	83.33	-
Weakness (%)	62.07	46.15	66.67	66.67
Syncope (%)	10.34	5.13	-	100.00

The clinical findings in different groups of AHDs are presented in **Table-II**.

The predominant physical examination findings in the present study were tachycardia, murmur, pulmonary oedema and ascites followed by other findings like pedal oedema and pulse deficit might be due to atrial fibrillation in DCM group. Murmur might be due to regurgitation secondary to dilatation; pulmonary oedema might be due to poor cardiac performance; and ascites might be due to increased sodium and water retention in cardiac cases. These findings concur with several authors (Häggström *et al.*1995; Swenson *et al.*, 1996; Tidholm and Jonsson, 1996; Tidholm *et al.* 1997; Ristic, 2004; Martin *et al.,* 2009; and Martin *et al.,* 2010). Systolic murmur and low pitched pro diastolic (S3) gallop sound were auscultated as an evidence of severe ventricular diastolic impairment as reported by Sisson and Thomas, (1995).

In MVD group systolic murmur in different grades, honking cough, tachycardia, pulmonary oedema were the major clinical findings. Regurgitation might be the reason for murmurs; cough might be due to the compression of left main stem bronchi; and pulmonary oedema might be due to overworked left ventricle weren't able to pump out enough of the blood it received from lungs. These findings are similar to findings of Bonagura and Frank (1983); Häggström *et al.* (1995); and Häggström, (1996).

In pericardial effusion group the major findings were tachycardia, pulsus paradoxus, ascites and coughing. In HCM group tachycardia and pulse deficit due to very low cardiac output were major signs. In pericardial effusion and HCM the signs are mainly because of impairment in filling of ventricle and very low cardiac output. Majority of the signs were also noticed in various studies by Moise *et al.*, (1986); Häggström *et al.* (1995); Swenson *et al.* *(*1996); French *et al.* (1998); Kittleson and Kienle, (1998); and Kovacic and Muller, (2003) and Shaw and Rush, (2007).

Table-II: Clinical Presentation in Dogs with Acquired Heart Diseases (AHD)

Chief Complaints	Group - II Dilated Cardiomyopathy (n=58)	Group – III Mitral Valve Disease (n=39)	Group – IV Pericardial Effusion (n=6)	Group – V Hypertrophic Cardiomyopathy (n=3)
Tachycardia (%)	**86.21**	**66.67**	**83.33**	**100.00**
Murmur (%)	**89.66**	**89.74**	-	-
Pulmonary oedema (%)	**60.34**	38.46	-	-
Pulse deficit (%)	36.21	5.13	**83.33**	-

Ascites (%)	**82.76**	15.38	**66.67**	-
Coughing (%)	20.69	**89.74**	**50.00**	-
Pedal oedema (%)	**77.59**	20.51	33.33	-
Vomiting (%)	5.17	5.13	-	-
Weight loss (%)	17.24	20.51	-	-

CONCLUSION

The observed chief complaints included inappetance, exercise intolerance, abdominal enlargement, syncope and weakness. Tachycardia, ascites and murmurs were the common physical examination findings in all the groups of AHDs. Presence of signs such as Tachycardia (86.21 per cent) and ascites (82.76 per cent) in DCM; and coughing (89.74 per cent) and murmur (89.74 per cent) in MVD showed the presence of acquired heart disease in dogs.

Acknowledgments: Authors are thankful to the Tamil Nadu Veterinary and Animal Sciences University (TANUVAS), Chennai, India.

Conflict of Interest: None declared.

References

[1] Bonagura, J.D and S.P. Frank. 1983. Echocardiographic features of aortic valve endocarditis in a dog, a cow and a horse. *J. Am. Vet. Med. Assoc.*, **182**: 595-599.

[2] Bulmer, B.J. 2006. Performing a cardiovascular physical examination. *Vet. Med.*, **101:** 37-47.

[3] Darke, P.G.G. 1985. Myocardial disease in small animals. *Br. Vet. J.*, **141**:342–348.

[4] Erling, P. and E.M. Mazzaferro. 2008. Left-sided congestive heart failure in dogs: treatment and monitoring of emergency patients. *Compend. Contin. Educ. Vet*. **30**:94-104.

[5] Fisher, E.W. 1972. Heart disease in the dog. *J. Small Anim. Pract.,* **13:**553-60.

[6] Fox, P. 1988. Canine myocardial disease. In: *Canine and feline cardiology*. Ed P.R. Fox. Churchill Livingstone, New York. Pp. 467–87.

[7] French, C.I., R.S. Irwin, F.J. Curley and C.J. Krikorian. 1998. The impact of chronic cough on quality of life. *Arch. Int. Med.,* **158:**1657- 1661.

[8] Häggström, J. 1996. Chronic valvular disease in Cavalier King Charles spaniels-epidemiology, inheritance and pathophysiology (Ph.D Thesis). Swedish University of Agricultural Sciences, Uppsala, Sweden.

[9] Häggström, J., K. Hansson and C. Kvart. 1995. Heart sounds and murmurs: changes related to severity of chronic valvular disease in the Cavalier King Charles spaniel. *J. Vet. Int. Med.,* **9**: 75-85.

[10] Kittleson, M.D. and R.D. Kienle. 1998. Primary myocardial disease leading to chronic myocardial failure. In: *Small animal cardiovascular medicine,* 1st Ed. M. Msoby. Philadelphia, pp.319-346.

[11] Kittleson, M.D. and R.D. Kienle. 1998. Primary myocardial disease leading to chronic myocardial failure. In: *Small animal cardiovascular medicine,* 1st Ed. M. Msoby. Philadelphia, pp.319-346.

[12] Kovacic, J.C. and D.Muller. 2003. Hypertrophic cardiomyopathy: state-of-the-art review, with focus on the management of outflow obstruction. *Intern. Med. J.,***33**(11):521-9.

[13] Kvart, C. and J. Häggström. 2000. Acquired valvular heart disease. In: *Textbook of Veterinary Internal Medicine. Diseases of the dog and cat*. Edn. S.J. Ettinger and E.C. Feldman, 5th edn. W.B. Saunders, Philadelphia. pp. 787-800.

[14] Martin, M.W.S, M.J. Stafford and B. Celona. 2009. Canine dilated cardiomyopathy: a retrospective study of signalment, presentation and clinical findings in 369 cases. *J. Small Anim. Pract.,* **50:**23-29.

[15] Martin, M.W.S, M.J. Stafford and B. Celona. 2009. Canine dilated cardiomyopathy: a retrospective study of signalment, presentation and clinical findings in 369 cases. *J. Small Anim. Pract.,* **50:**23-29.

[16] Martin, M.W.S, M.J. Stafford, G. Strehlau and J.N. King. 2010. Canine dilated cardiomyopathy: a retrospective study of prognostic findings in 367 clinical cases. *J. Small Anim. Pract.,* **51:**428–436.

[17] Martin, M.W.S, M.J. Stafford, G. Strehlau and J.N. King. 2010. Canine dilated cardiomyopathy: a retrospective study of prognostic findings in 367 clinical cases. *J. Small Anim. Pract.,* **51:**428–436.

[18] McCurnin, D.M. and Poffenbarger. 1991. In: *Small animal Physical diagnosis and Clinical procedures*. Saunders (Philadelphia).

[19] Meurs, K.M., M.W. Miller and N.A. Wright. 2001. Clinical features of dilated cardiomyopathy in Great Danes and results of a pedigree analysis: 17 cases (1990-2000). *J. Am. Vet. Med. Assoc.,* **218**:729-732.

[20] Moise, N.S., A.E. Dietze, L.E. Mezza, D. Strickland, H.N. Erb and N.J. Edwards. 1986. Echocardiography, electrocardiography, and radiography of cats with dilatation

cardiomyopathy, hypertrophic cardiomyopathy, and hyperthyroidism. *Am. J. Vet. Res.* **47**(7): 1476-86.

[21] Ristic, J. 2004. Companion Animal Practice: Clinical assessment of the dog with suspected cardiac disease. *Practice,* **26:**192-199.

[22] Shaw, S.P. and J.E. Rush. 2007. Canine pericardial effusion: diagnosis, treatment, and prognosis. *Compend. Contin. Educ. Vet.,* **29**:405-411.

[23] Sisson, D.D. and W.P. Thomas. 1995. Myocardial diseases. In: *Textbook of Veterinary Internal Medicine*, 4th eds. S.J. Ettinger and E.C. Feldman, W.B. Saunders, Philadelphia, pp 995-1032.

[24] Swenson, L., J. Häggström, C. Kvart and R. Kumar Junea. 1996. Relationship between parental cardiac status in Cavalier King Charles Spaniels and prevalence and severity of chronic valvular disease in offspring. *J. Am. Vet. Med. Assoc*., **208**:2009- 2012.

[25] Tidholm, A. and L. Jönsson. 1996. Dilated cardiomyopathy in the Newfoundland: A study of 37 cases (1983–1994). *J. Am. Anim. Hosp. Assoc*., **32:**465–470.

[26] Tidholm, A. and L. Jönsson. 2005. Histologic characterization of canine dilated cardiomyopathy. *Vet. Pathol*., **42**:1-8.

[27] Tidholm, A., H. Svensson and C. Sylven. 1997. Survival and prognostic factors in 189 dogs with dilated cardiomyopathy. *J. Am. Anim. Hos. Assoc.,* **33:**364–368.

[28] Tidholm, A., H. Svensson and C. Sylven. 1997. Survival and prognostic factors in 189 dogs with dilated cardiomyopathy. *J. Am. Anim. Hos. Assoc.,* **33:**364–368.

[29] Tilley, L.P. 1992. *Essentials of Canine and Feline Electrocardiography*. 3rd edn. Lea and Febiger, Philadelphia. Pp 567-576.

[30] Ware, W. 2007. Overview of electrocardiography. In: *Cardiovascular Disease in Small Animal Medicine*, Manson Publishing, pp: 51-64.