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,
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 Mazza Ferro, (2008); and Fox, (1988).

In MVD persistent cough might be due to compression of left main stem bronchi 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)

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

<table>
<thead>
<tr>
<th>Chief Complaints</th>
<th>Group - II Dilated Cardiomyopathy (n=58)</th>
<th>Group – III Mitral Valve Disease (n=39)</th>
<th>Group – IV Pericardial Effusion (n=6)</th>
<th>Group – V Hypertrophic Cardiomyopathy (n=3)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tachycardia (%)</td>
<td>86.21</td>
<td>66.67</td>
<td>83.33</td>
<td>100.00</td>
</tr>
<tr>
<td>Murmur (%)</td>
<td>89.66</td>
<td>89.74</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Pulmonary oedema (%)</td>
<td>60.34</td>
<td>38.46</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Pulse deficit (%)</td>
<td>36.21</td>
<td>5.13</td>
<td>83.33</td>
<td>-</td>
</tr>
<tr>
<td>Ascites (%)</td>
<td>82.76</td>
<td>15.38</td>
<td>66.67</td>
<td>-</td>
</tr>
<tr>
<td>Coughing (%)</td>
<td>20.69</td>
<td>89.74</td>
<td>50.00</td>
<td>-</td>
</tr>
<tr>
<td>Pedal oedema (%)</td>
<td>77.59</td>
<td>20.51</td>
<td>33.33</td>
<td>-</td>
</tr>
<tr>
<td>Vomiting (%)</td>
<td>5.17</td>
<td>5.13</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Weight loss (%)</td>
<td>17.24</td>
<td>20.51</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

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.

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Conflict of Interest: None declared.

References


