CLINICAL EVALUATION OF DOGS WITH ASCITES IN JOS – METROPOLIS, PLATEAU STATE

MOH’D, J.G; OGBE, A.O; *HAJARA, A.Y; RASHIDAT, O.Y; ELISHA, D.G, AND JONAH, A.C.

Vet Clinic, FCAHPT, NVRI Vom, Plateau State
*Biochemistry Dept; NVRI, Vom, Plateau State

ABSTRACT

Samples were taken from ascitic dogs presented in some selected Veterinary Clinics in Jos metropolis for Clinical evaluation. The result showed a high rate of hookworm infestation in the faeces of dogs with ascites. The result further showed that out of 26 dogs tested for blood parasites, 14 (53.8%) were positive for Babesia canis, and 6 (23.1%) are positive for microfilaria. The result of urinalysis showed a high level (100%) of protein in the urine of dogs with ascites. The liver function test result shows alanine amino transferase (ALT) levels in the range of 20 – 102 IU/L, while aspartate amino transferase (AST) was 16 – 36 IU/L. The post mortem findings of the dogs that died of ascites showed granular or nodular contracted kidneys and the liver appears cirrhotic with rough and granular lesion.

KEY WORDS: Ascites, Proteinuria, Liver cirrhosis, Granular kidneys.

INTRODUCTION

Many types of disease problems have been associated with liver, or kidney damage, the consequences of which may result in ascites. However, the reticuloendothelial systems are capable of regeneration when injured or damaged. The regenerative capability of the liver presents difficulty in determining the extent of liver damage in disease (Sirois, 1995). Reduced hepatic perfusion and hepatic congestion may increase the serum alanine aminotransferase (ALT) also known as glutamic pyruvate transaminase (SGPT) and aspartate aminotransferase (AST) also known as glutamic oxalo-acetic transaminase (SGOT). However, these elevations tend to be mild to moderate, and persistently elevated liver enzymes, especially when >400 i.u/L usually indicates a primary disorder of the liver (John, 2001).

The presence of infectious and parasitic disease (babesia, microfilaria, hook worms etc.) in the body can damage the organ system, and malnourished animals are known to develop lower resistance due to effect on the systemic status. Circulating microfilariae can form urine complexes notably in the kidneys (Merck, 1991). Thrombosis of smaller arteries may occur as a result of death of worms, the effect of the lesions, in conjunction with obstructing fibrosis can lead to pulmonary hypertension and secondary right heart problem. The kidneys may show evidence of
glomerulonephritis (Merck, 1991). Extreme protein loss through the kidney can lead to severe hypoproteinaemia (Ritchie et al., 1994), which can result in ascites. Chronic liver disease, nephritic syndrome, protein-losing enteropathy, neoplasia and congestive heart failure can cause ascites (Ritchie et al., 1994), and this may affect the overall central role of the liver in the metabolism of proteins, fats, carbohydrates, certain vitamins and minerals as well as the regulatory role of the kidneys in plasma protein concentration and blood pressure.

THE AIMS AND OBJECTIVES

1. To clinically evaluate dogs presented with ascites.
2. To also carry out analysis of blood (serum), faeces, and urine samples of dogs with ascites for presence of infecting agents or parasites and organ/ tissue damage or dysfunction.
3. To highlight possible areas of management (control) and further research.

MATERIALS AND METHODS

Data and Dogs

Data and dogs used in this study are those presented with a history of ascites in some selected Vet Clinics in Jos metropolis. The diagnostic methods include clinical examination, palpation, auscultation and abdominocentesis for evidence of ascites.

Sample Analysis

Blood sera, faeces and urine samples were used for laboratory analysis. Thin blood film, smear, liver function test, simple flotation and urine analysis were also carried out.
RESULTS AND DISCUSSION

The histopathological analysis of liver and kidney is as shown in Figure 1.

Fig. 1 Liver and Kidney Lesions characterized by granular or nodular contracted kidneys with cirrhotic liver.

The result of faecal analysis is as shown in table 1

TABLE 1: Prevalence of blood parasites in dogs with ascites in Jos metropolis

<table>
<thead>
<tr>
<th>PARASITES (Seen)</th>
<th>MALE DOGS</th>
<th>FEMALE DOGS</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number Tested</td>
<td>Number Positive (%)</td>
</tr>
<tr>
<td>Babesia canis</td>
<td>13</td>
<td>7 (53.8)</td>
</tr>
<tr>
<td>Microfilaria</td>
<td>13</td>
<td>4 (30.8)</td>
</tr>
<tr>
<td>Total</td>
<td>26</td>
<td>11 (42.3)</td>
</tr>
</tbody>
</table>

The results of faecal parasites analysis showed a high rate of hookworm infestation. *Ancylostoma caninum* is the principal cause of canine hookworm disease in most tropical areas of the world (Merck, 1991). Transmission occurs from ingestion of infective larvae from the environment, colostrum or milk of infected bitches (dogs) and skin penetration where they get to the blood to the lungs and are coughed out, swallowed to mature in the small intestine. However, in young puppies the larvae on migration get arrested in somatic tissues but later activated during pregnancy and
accumulates in the mammary glands (Merck, 1991). This may explain the low
detection of ova (eggs) in some faecal sample.

Debilitated and malnourished hookworm-infested animals are known to suffer
chronic anaemia from the blood sucking parasites. Serum seepage around site of
attachment in the intestine may lead to loss of blood protein (hypoproteinemia). The
liver and other organs of the affected dog may appear ischaemic with some fatty
infiltration of the liver (Merck, 1991).

The result further showed that out of 26 dogs tested for blood parasites, 14
(53.8%) were positive for Babesia canis, and 6 (23.1%) are positive for microfilaria
parasites as shown on table 1. Babesiosis and microfilariosis are tropical disease
problems particularly areas wherever suitable tick – vectors and mosquitoes occur.
During tick feeding babesia parasites in the tick saliva are known to get into
bloodstream and into hosts red blood cells (RBC) damaging the cells resulting in
hemoglobinemia, anemia, anoxia and thus damage to the highly vascularised organs
dependent on oxygen – carrying capacity of blood like the liver, kidney and spleen
(Merck, 1991). Microfilariae on the other hand are small microscopic parasites
released into the blood stream by heartworms found in the pulmonary arteries and
right ventricle of the dog’s heart. A mosquito ingests several microfilariae when it
bites a dog. The mosquito serves as an intermediate host as well as a vector. Most
affected dogs are known to develop congestive heart failure & ascites. However, it
appears there is no sex preference in infection rate by the blood parasites as both male
and female dogs are infected as shown in table 1.

The result of the Urinalysis showed a high level (100%) of protein in the urine
of dogs with ascites. The primary protein found in abnormal urine is albumen.
Normally, all protein is reabsorbed in the kidney tubules and none is excreted (Sirois.
1995). The result of urinalysis is as shown in table 2.

<table>
<thead>
<tr>
<th>TABLE 2: urine composition of dogs with ascites in Jos metropolis</th>
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<tbody>
<tr>
<td>Urine composition</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Protein</td>
</tr>
<tr>
<td>Ketone bodies</td>
</tr>
<tr>
<td>Bilirubin</td>
</tr>
<tr>
<td>Bile Salt</td>
</tr>
<tr>
<td>Urobilinogen</td>
</tr>
<tr>
<td>Reducing Subs.</td>
</tr>
<tr>
<td>Pus Cells</td>
</tr>
<tr>
<td>Red blood Cells</td>
</tr>
<tr>
<td>Epithelial Cells</td>
</tr>
<tr>
<td>Yeast Cells</td>
</tr>
<tr>
<td>Crystals</td>
</tr>
<tr>
<td>Casts</td>
</tr>
<tr>
<td>Parasites</td>
</tr>
<tr>
<td>Bacteria</td>
</tr>
</tbody>
</table>
Abnormal presence of protein in urine samples (proteinuria) may be associated with nephritis, glomerulonephritis, cystitis, ureteritis, urethritis, urolithiasis and haemoglobinuria (Ugochukwu, 2001). If proteinuria is severe and persistent, edema and or ascites may occur. The presence of significant proteinuria, hypoaalbuminemia, hypercholesterolemia and ascites is the nephrotic syndrome. There was also presence of pus cells 4 out of 5 dogs (80%), and epithelial cells (20%). Apparently healthy (normal) dogs also tested positive for pus cells, 3 out of 5 (60%) and epithelial cells, 2 (40%) while yeast cells was found only in 3 (60%) of the dogs with ascites. Bacteria was found in 5 (100%) of the dogs with ascites and 3 (60%) in normal dogs tested as shown in table 2.

Increased number of leukocytes in urine samples (referred to as pyuria) may be indicative of pyogenic processes taking place in the urogenital apparatus. Increase number of leukocytes are associated with urethritis, cystitis, pyelonephritis and nephritis (Ugochukwu, 2001). Different types of epithelial cells ranging from squamous to transitional may occur in small number in a normal urine sample, but increased number are indicative of inflammatory reaction in the Urogenital tract. Normally, no bacteria should be seen in urine. However, when large number of bacteria is seen in Urine may be associated with catheterization with unsterilised instruments, paracentesis and cystocentesis, otherwise cystitis, pyelonephritis, metritis, vaginitis, prostatitis, urethritis, ureteritis may be responsible (Ugochukwu, 2001).

The result of the liver function test is as shown in table 3.

**TABLE 3: Values of liver function test (LFT) and blood parameters of dogs with ascites in Jos-Metropolis**

<table>
<thead>
<tr>
<th>VALUES</th>
<th>ASCITES DOGS</th>
<th>NORMAL DOGS</th>
</tr>
</thead>
<tbody>
<tr>
<td>ALT (SGPT) i.u/L</td>
<td>20 – 102</td>
<td>14 – 29</td>
</tr>
<tr>
<td>AST (SGOT) i.u/L</td>
<td>16 – 36</td>
<td>12 – 18</td>
</tr>
<tr>
<td>Total Protein (g/dl)</td>
<td>5.6 – 8.8</td>
<td>5.0 – 8.0</td>
</tr>
<tr>
<td>PCV (%)</td>
<td>21 – 27</td>
<td>35 – 50</td>
</tr>
</tbody>
</table>

Reference (Normal) Values

- ALT (SGPT) i.u/L = 5 – 24
- AST (SGOT) i.u/L = 6 – 43
- Total Protein (g/dl) = 5 – 8
- PCV (%) = 38 – 55

The liver function test result shows alanine aminotransferase (ALT) otherwise known as Serum glutamic pyruvate transaminase (SGPT) levels in the range of 20 – 102 i.u.L (normal range 5 – 24) for all samples tested from dogs with ascites, while
aspartate amino transferase (AST) formerly known as Serum glutamic oxalo-acetic transaminase (SGOT) was 16 - 36 i.u/L (normal range = 6 - 43iu / L) as shown on table III.

In dogs, cats and primate; ALT is found in large amount within the hepatocytes. Damage to hepatocytes will result in release of large amount of this enzyme. In other species serum ALT levels are of little diagnostic value (Sirois, 1995). Elevated amount of ALT is indicative of liver cell damage hepatotoxicosis, leptospirosis, infectious canine hepatitis, fatty liver, diabetes mellitus and hepatic cancer (Ugochukwu, 2001).

AST (16-36 i.u/L) and total protein (5.6 – 8.8 g/dL) appears to be within normal range values (5.0 – 8.0g/dL) as shown in table III.

The postmortem findings of dogs (carcasses) that died with ascites showed granular or nodular contracted kidneys i.e chronic nephritis and the liver appears cirrhotic with rough and granular or nodular lesion. About 1000 milliliter (1 litre) of clear fluid was aspirated from the peritoneal cavity of three Alsatian dogs (live) and two (dogs) that die of ascitic syndrome.

The principal effect of cirrhosis is the interference with the flow of portal blood through the many hepatic ramifications on its way to the heart. The result is chronic passive congestion of the spleen and of the digestive organs. Mild digestive disturbances and discomfort follow, but the chief effect of the retarded venous flow is ascites, a collection of edema fluid in the peritoneal cavity (Jones and Hunt, 1983).

CONCLUSION

The liver and the kidneys are very important organs of metabolism and excretion in the body of animals. Disease problems affecting these vital organs therefore can lead to problems with the metabolism of nutrients and the removal of their potentially harmful by-products.

Most important considerations are the nutritional care and health management of the animals with ascites.

Nutritional care management is the cornerstone for the treatment of dogs with liver disease and will reduce the need for some of the more costly and potentially hazardous medical therapies (Tim, 2003).

REFERENCES


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