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Canine Ascites

by W. Michael Peden, DVM
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INTRODUCTION

Canine ascites is an infrequently seen clinical sign which is often the primary complaint for presentation of an animal to a veterinarian. As in all cases a good history is a priority as further questions may reveal behavioral changes, vomiting, anorexia, and other clinical signs which may be important in defining the primary problem. Ascites itself can physically interfere with respiration, cause general discomfort, and disturb fluid and electrolyte metabolism. The underlying cause must be determined and treated. Simply removing the ascitic fluid will only give short term relief.

A diagnostic evaluation of an animal presented with ascites may include a complete blood count (CBC), biochemical evaluation, abdominal paracentesis and biochemical and cytologic analysis of the fluid obtained, radiographs, biopsy, and organ function tests. A physical exam always precedes any further diagnostic aids.

Ascites is a sign that something serious has gone wrong with the animal and the client should be appraised that diagnosis could be expensive, the prognosis may be poor, and treatment difficult or unrewarding. In general the two main causes of ascites are cardiac problems and liver disease, with the greater percentage caused by cardiac problems.

Common cardiac problems associated with ascites are heartworms, congestive cardiomyopathy, right heart failure, and congenital pulmonic stenosis. Hepatic diseases which cause ascites and/or hypoalbuminemia are liver insufficiency, chronic-active hepatitis,

cirrhosis, and cholangitis. Most of the cardiac and hepatic diseases are associated with portal hypertension as the direct cause of ascites.

Another cause of fluid accumulation in the peritoneal cavity is hypoproteinemia due to renal loss of protein. Diseases such as amyloidosis and glomerulonephritis may cause massive proteinuria. Other causes of abdominal fluid accumulation include neoplasia of an abdominal organ, ruptured urinary bladder, and hemorrhage from trauma or neoplasia.

The following steps need not necessarily be performed in any certain sequence, but a logical procedure should be followed in order to arrive at a diagnosis. For example, blood work and radiographs or analysis of abdominal fluid may be done in a different order than presented here, but all work should be done with the idea of ruling out the easiest and most obvious diseases first. Ascites is most often due to cardiac disease which can be ruled out relatively easily by a physical exam and thoracic radiographs. If these findings are normal, more involved diagnostic aids may be required to rule out liver disease or neoplasia. A urinalysis may reveal the causes of hypoalbuminemia to be via the kidneys. In short, a logical pattern should be kept in mind in order to arrive at a reasonable diagnosis with a reasonable amount of time and effort.

PHYSICAL EXAM

The animal is often presented with a complaint of abdominal enlargement. The causes of distension (gas, liquid, organomegaly) should be determined. Intraabdominal gas will give a sharp rebound to percussion and a higher pitched resonance when auscultated than fluid. The ascitic abdomen has a charac-

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teristic pear shape and percussion of the abdomen will result in a sharp fluid rebound on the opposite side.

Fluid distensions may occur within the gastrointestinal tract or any hollow organ. Palpation is used to rule out a palpable obstruction such as an intussusception. However, the great volume of fluid may limit a good physical examination. Ballotment through the abdominal wall may indicate a mass or enlargement of an organ.

Since cardiac disease is a major cause of ascites, particular care should be paid to auscultating the heart. Murmurs may be indicative of a number of problems and the location of the murmur may be a clue to the particular problem. A murmur on the right side, third to fifth intercostal space may indicate a tricuspid insufficiency due to cardiomyopathy or valvular fibrosis. In congestive heart failure both mitral and tricuspid insufficiency may be noted. Severe heartworm disease can cause right sided heart failure and a positive Knott's test and characteristic thoracic radiographs will support the diagnosis. Delayed heart sounds such as late closure of the pulmonary valve may also indicate heartworms.

Congenital pulmonic stenosis may be indicated by a murmur on the left side near the cranial sternum. This condition may lead to right ventricular enlargement and right heart failure.

Idiopathic congestive cardiomyopathy may be suspected when a large breed dog with ascites is showing a deficit between the heart and pulse rate. If atrial fibrillation is present (>80%) the heart rate can be greater than 200 beats per minute.

Hepatomegaly or splenomegaly are often noted with cardiac problems due to venous congestion. These signs may also indicate neoplasia or hyperplasia. It is important not to be led astray by signs which may indicate more than one disease. Icterus is another clinical sign which could be confusing as it is usually associated with hepatic disease or hemolytic disease.

RADIOGRAPHS

The most important ancillary test used in diagnosing the cause of ascites is radiography. Cardiac causes of ascites are easy to rule out with a thoracic radiograph. In most instances, cardiomegaly will be seen which

alerts the clinician to consider the heart more carefully. Specific examples include: 1) heartworm disease with enlarged right ventricle and enlarged pulmonary arteries, 2) biventricular enlargement with mitral and tricuspid insufficiency, 3) enlarged right ventricle and poststenotic dilation of pulmonary artery with congenital pulmonic stenosis, 4) marked generalized cardiomegaly with idiopathic congestive cardiomyopathy of large breed dogs. Physical findings along with radiographic findings may add support to cardiac cause of ascites.

If there is no indication for cardiac disease as the cause of ascites, abdominal radiographs may be helpful. Abdominocentesis prior to radiographing the abdomen is beneficial as the amount of fluid in the abdomen will obscure any detail in most cases. Removal of the fluid will give better definition and possibly enable visualization of the abdominal organs and relative positions. For example, caudal displacement of the stomach gas shadow may indicate hepatomegaly. Dorsal displacement of the intestines may indicate a splenic tumor or mass. Pneumoperitoneography may help in visualizing the abdominal organs. Cystography may be used to evaluate the bladder if paracentesis shows urine in the fluid.

CBC and BLOOD CHEMISTRY

A CBC should be done on all animals presented with ascites even though the information will not give a definitive diagnosis. An eosinophilia may be a further indication of heartworm disease. An inflammatory leukogram may indicate an inflammatory process such as peritonitis.

Protein levels should be determined and a specific check for albumin levels should be made. Hypoalbuminemia rarely is the primary cause of ascites but definitely contributes to the continuation of the problem. In order for hypoalbuminemia to cause ascites the albumin level must be less than 1.5 g/dl. Decreased hepatic synthesis will contribute to hypoalbuminemia but a more likely cause is expansion of the plasma volume and subsequent dilution of albumin^{2,7}

A Knott's test for *Dirofilaria immitis* should also be performed. It should be remembered that a significant percentage of animals suffering from heartworms are negative with the Knott's test. If all other

signs indicate heartworm disease, a negative Knott's test should not be the basis for discarding that diagnosis.

Evaluation of the serum may reveal abnormal chemical values which may be extremely helpful in diagnosing the primary problem after cardiac disease has been ruled out. A urinalysis may yield information on bilirubin levels, urobilinogen, and renal loss of protein.

In general, biochemical analysis of serum is primarily aimed at determining the status of the liver by 1) measuring substances excreted or produced in the normal liver, 2) measuring enzymes which are associated with abnormal liver function, and 3) measuring the rate of removal of certain dyes such as BSP (sulfabromophthalein).⁸

Bilirubin is a breakdown product of hemoglobin and measurements of total bilirubin, conjugated bilirubin, and unconjugated bilirubin are helpful in assessing hepatic function, cholestasis, or hemolytic disease. A marked rise in conjugated bilirubin as opposed to unconjugated bilirubin is a strong indication of cholestasis. If the increase is proportionately greater in the unconjugated bilirubin, a hemolytic cause should be suspected for the increase in bilirubin.²

Bilirubinuria is often noted before bilirubinemia and may be the first indication of cholestasis. Urinary urobilinogen may help differentiate a partial or complete biliary obstruction. A positive test for urobilinogen indicates that the bile duct is patent to some degree.²

Serum alkaline phosphatase (SAP) is also a good indicator of cholestasis. Cholestasis stimulates *de novo* synthesis of SAP and these increased levels are reflected in the circulation. Increased SAP levels often precede hyperbilirubinemia or bilirubinuria and any increase in the latter two should be accompanied by increased SAP levels if they are due to cholestasis.^{2,8}

Serum glutamic-pyruvate transaminase (SGPT) and serum glutamic-oxalacetic transaminase (SGOT) are leakage enzymes indicative of hepatocellular damage. SGPT is the only enzyme specific for hepatic damage. A number of factors may induce hepatocellular leakage including hypoxia, toxins, drugs, hepatitis, fatty change and degeneration due to metabolic disorders. Lesions accompanying leakage of enzymes may be fatty change,

hepatocellular necrosis, fibrosis, or biochemical lesions. Levels of significance for these enzymes can be found in most clinical pathology references. It is important to remember that the magnitude of the increase in enzyme levels is proportionate to the damage, but no evaluation of the reversibility of the damage can be made from these levels. The persistence of high levels of these enzymes is a poor prognostic sign, as the half-life is 2-4 days, and persistent high levels mean continuing damage. Other enzymes which can be used to detect hepatocellular disease include isocitrate dehydrogenase, glutamate dehydrogenase, and arginase.²

Reduced functional hepatic mass can be measured by a BSP excretion test or an ammonia tolerance test. BSP is rapidly removed from the blood and conjugated in the liver and ammonia is converted to urea in the liver. Both of these processes depend on an adequate hepatic mass and blood flow. The results for both tests may be affected by cardiac disease (inadequate blood flow) which could lead to a false diagnosis of a primary liver problem if the results were used alone for a diagnosis. In addition BSP clearance is hastened by hypoalbuminemia and delayed by hyperbilirubinemia. For these reasons the results of such tests may be confusing and the tests should be far down on the list of diagnostic aids. In general, increased ammonia concentration has been documented most frequently in animals with portocaval venous shunting and liver atrophy.^{2,8}

ABDOMINAL PARACENTESIS

Abdominal paracentesis is accomplished by advancing a needle into the most pendulous area of the abdomen while keeping a slight negative pressure in the syringe. This insures that fluid will be withdrawn as soon as the needle enters the peritoneal cavity. Fluid should be drawn into a container containing anticoagulant.⁶

The fluid should be evaluated for cell counts and cytology, biomechanical parameters, and cultured. All degrees of transudates and exudates may be found in ascites, and examination of the fluid is a valuable diagnostic test.

Cytologic examination of the fluid (direct smear or sediment) may indicate lymphosarcoma or other neoplasia as the origin of the fluid. Bloody or cloudy fluid often indicates

an inflammatory or neoplastic process, and the number and type of cells should be evaluated.

Specific gravity, the presence of bile or urine, and the protein level should all be checked. A specific gravity of greater than 1.030 with high numbers of red blood cells and white blood cells may indicate bleeding into the abdominal cavity. Urine in the fluid may indicate a ruptured bladder. Bile indicates a direct communication of the biliary tree and peritoneal cavity.⁵

A low protein level (less than 2.0 g/dl) is indicative of a pure transudate, possibly due to hypoproteinemia caused by renal disease or intestinal malabsorption. Most ascitic fluid is a modified transudate with a protein level of 2.5 to 5.0 g/dl. Modified transudates are seen in many diseases such as congestive heart failure, portal venous obstruction, cirrhosis, hepatoma, and abdominal neoplasia.^{2,6}

LIVER BIOPSY AND LAPAROTOMY

The results of various tests and physical findings may suggest or rule out hepatic disease as the cause of ascites. A liver biopsy is one method which can be used to confirm other findings and establish a specific diagnosis. A liver biopsy should only be attempted when the information obtained will be beneficial to the patient, as the possibility of serious iatrogenic complications is an obvious concern. Therefore if other findings can establish a specific diagnosis there is little necessity for a liver biopsy. A liver biopsy may be the only way to diagnose neoplasia, toxic hepatitis, or cirrhosis and in these instances histology is necessary to establish a specific diagnosis, prognosis, and treatment regimen.

Before attempting a liver biopsy the animal's condition in regard to PCV, clotting time, prothrombin time and platelet number should be evaluated. If these values are questionable the biopsy is contraindicated as massive hepatic bleeding may result.

There are many techniques for obtaining a specimen of liver. These include a keyhole technique, percutaneous transthoracic or transabdominal approach, and exploratory laparotomy.^{3,5} The keyhole technique is the safer of the two as it permits digital exam of the liver and surrounding organs, the hepatic lobes can be immobilized, precise selection of the biopsy site can be obtained, and any hemorrhage can be easily ascertained. The

key hole technique will be the only technique described here.

The animal should be fasted and fat should be given per os to contract the gall bladder. Ascitic fluid should be removed before attempting a biopsy. The animal is placed in dorsal recumbency and general anesthesia or local anesthesia and sedation is employed. The area to be incised is on the ventral midline, directly posterior to the xiphoid. A small incision large enough to insert a finger is made into the peritoneal cavity. The area is palpated and the left hepatic lobe is immobilized. This is a larger lobe in the dog and biopsy of this lobe has less risk for gall bladder penetration as it lies on the right side of the animal. A separate skin incision is made to the left of the first incision to permit entry of the biopsy needle into the peritoneal cavity. The index finger is used to guide the biopsy needle to the biopsy site. The liver lobe is immobilized by pinning it against other lobes or the diaphragm with the index finger. The needle is positioned so that it will not penetrate vital structures such as the large vessels and gall bladder. If a Menghini needle is used for the biopsy a slight negative pressure is maintained in the syringe and needle as the needle is moved rapidly into and out of the liver in one smooth, continuous motion. The biopsy needle is placed in fixative and the negative pressure is released. The liver should be examined for bleeding and the incision closed.⁵

If neither neoplasia or hepatic disease can be ruled out a combined exploratory laparotomy and biopsy can be considered to arrive at a diagnosis.

PATHOGENESIS OF HEPATIC AND CARDIAC ORIGIN ASCITES

Ascites due to cardiac problems is brought about by a failing right heart leading to splanchnic pooling and congestion. Hepatic disease and fibrosis also lead to splanchnic pooling of blood. Both of these problems lead to portal hypertension. In ascites due to portal hypertension there is a derangement of fluid and electrolyte metabolism of the animal. With portal hypertension plasma proteins readily escape from the sinusoidal capillaries into the interstitial space of Disse. From there only the hepatic capsule separates the proteins from the abdominal space. The increased hydrostatic pressure causes the

movement of large amounts of fluid and protein into the abdominal cavity. The peritoneal lymphatics are unable to return the volume of fluid to the normal circulation. The loss of fluid into the abdominal cavity leads to a decrease in effective plasma volume and in cardiac output. This causes a decrease in glomerular filtration, which leads to the release of renin and the conversion of angiotensinogen to angiotensin II, and secretion of aldosterone by the adrenal gland. The kidney saves more sodium and water to try and increase effective plasma volume and does increase total plasma volume.⁷

Only 20% of the blood is in the high pressure arterial side of the circulation and all conservation mechanisms operate on the low pressure side. Increasing total volume may not necessarily increase effective plasma volume and return the arterial side to normal function. An animal with ascites has its circulatory system working at maximum compensation and any disruption of fluid or electrolyte beyond what has already happened may throw the animal into circulatory failure or collapse.

In normal animals expansion of the extracellular fluid compartment will result in diuresis due to a poorly understood phenomenon known as Third Factor. This is believed to be a hormone mediated action on renal microcirculation but is not well understood. The ascitic animal will not respond to volume expansion as a normal animal will, thus making Third Factor very important in the mechanism of fluid retention in animals with ascites. In addition, aldosterone is metabolized by the liver, and in hepatic insufficiency the effect of aldosterone may be prolonged.⁷

TREATMENT

Treatment of ascites will depend upon the underlying cause. If cardiac failure is the primary cause the standard treatment of diuretics and digitalis may help resolve the ascites as cardiac function is improved. Paracentesis and removal of fluid may be beneficial, but the loss of protein in the fluid is undesirable. In addition the sudden removal of large quantities of fluid may further compromise the animal.

Surgical treatment and removal of fluid will be the obvious choice for treating operable neoplasia and traumatic injuries leading to accumulation of bile, blood, and

urine in the peritoneal cavity.

Hepatic origin ascites requires the most definitive diagnosis and treatment in order to be adequately resolved. If chronic-active hepatitis is diagnosed the treatment will revolve around arresting the inflammation, correcting nutritional derangements, resolving fibrosis and resolving any complications. Corticosteroids will decrease inflammation but may increase protein catabolism and increase ammonia production which is undesirable. A high quality, low protein diet in numerous small feedings is desirable to diminish bacterial conversion of excess protein to ammonia in the colon. Adequate energy should be included to minimize catabolism of proteins. Eggs, milk, lean meat, glucose, and B complex vitamins are examples of high quality foods to be included in such a diet. Corticosteroids and colchicine may help resolve fibrosis. Antibiotics should be used to minimize bacterial infection via the portal circulation. Confining the animal is beneficial as blood flow to the liver is increased and the work load of the liver is decreased.

Ascites itself is usually managed with diuretics and a low sodium diet. A loop diuretic such as furosemide is the diuretic of choice unless there is already an electrolyte imbalance. If potassium values are low the potassium sparing diuretics such as triamterene may be indicated. Removal of large quantities of fluid at any one time is not generally recommended because of the albumin loss and possibility of circulatory collapse. The removal of fluid over several days is a better plan, and even that may not benefit the animal as intraabdominal pressure may have a role in the rate of ascites formation, such that decreasing pressure may only increase the rate of formation of ascites.

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Porcine Intestinal Adenomatosis: A Clinical Review

by Craig Rowles*
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INTRODUCTION

Porcine Intestinal Adenomatosis (PIA) is a disease affecting the alimentary tract of pigs.¹ Previous literature has described several syndromes relating to PIA, including Regional Ileitis²⁻⁴ (RI), Necrotic Enteritis⁵⁻⁷ (NE), and Proliferative Hemorrhagic Enteropathy⁸⁻¹⁰ (PHE). However, it wasn't until recently that morphologic evidence was found, linking all four syndromes with a single etiologic agent.¹¹⁻¹²

A clinical review of PIA, including incidence and prevalence, etiology, lesions, diagnosis, treatment and control, follows.

INCIDENCE AND PREVALANCE

PIA is reported to be worldwide in distribution. Cases have been documented in Australia, Canada, Denmark, Finland, India, Sweden, the United Kingdom, and the United States.¹³ Two substantial slaughterhouse studies have been conducted to determine the percentage of swine showing lesions at slaughter.^{2,14} Each indicated a low but appreciable level of lesions, .25% and < 1% respectively.

No literature exists relating the number or percentage of herds infected with PIA. However, it is generally agreed that this disease does pose a significant economic loss for swine producers.

Any age group may show clinical signs. However, it appears that at two different times a pig may be more likely to show clinical signs. During the post weaning period, pigs seem to show clinical signs relating to PIA, RI, and NE.¹⁵ PHE, on the other hand, seems

to strike 6 mo. - 1 yr. old gilts and boars as they enter the breeding herd.

ETIOLOGY

Early observations of the disease were often hampered by other concurrent infections. Hence, an etiologic agent was difficult to identify. In 1972, however, a minimal disease herd broke with several cases of PIA.⁸ Through the use of special culturing techniques³ and immunofluorescence,⁸ *Campylobacter sputorum* var *mucosalis* was identified. To date, this organism has not satisfied Koch's postulates. Yet, it is universally agreed that the disease would not occur without this vibrioid organism.^{8,18}

Campylobacter sputorum var *mucosalis* is a gram negative, short, irregularly curved rod, 0.25 um wide and 0.95-2.8 um long. Sea gull, spiral and comma forms occur. Coccal forms also occur but are less prominent.¹³

Colonies grow on many solid nutrient media with 5-7% added blood. Microaerophilic conditions are necessary for growth. Colonies are 1.5-2.0 um in diameter after 48 hrs. and can be differentiated from other vibriones by the production of a yellow pigment and by tube agglutination tests.^{12,13}

Because this is an intracellular organism affecting the intestinal crypt cells, special culturing techniques must be used to isolate the organism from intestinal samples.¹² Most practitioners are not equipped to use this method of diagnosis.

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