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Ascites In Dogs

V V V Amruth Kumar*

Associate Professor, Department of Veterinary Medicine, College Of Veterinary Science, Mamnoor; Warangal;Telangana.

*Corresponding Author: Dr.V.V.V.Amruth Kumar; Associate Professor, Department of Veterinary Medicine, College Of Veterinary Science, Mamnoor; Warangal;Telangana.

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Abstract

Ascites is a significant complication observed in pets with various underlying health conditions. It is characterized by abdominal distension due to the accumulation of fluid, which can vary in color and consistency depending on the cause. Common causes of ascites in pets include hypoproteinemia, left-sided heart failure, congestive heart failure, cirrhosis, hepatic and renal diseases, ancylostomiasis, and bacterial infections such as tuberculosis. The pathogenesis of ascites is often linked to conditions such as portal hypertension, cirrhosis, and hepatorenal syndrome. Diagnosing ascites can be challenging due to the wide range of diseases that may cause it. Standard diagnostic methods include physical and clinical examinations, ultrasonography, computed tomography, serum ascitic albumin gradient (SAAG), and biochemical analyses (e.g., triglycerides, urea, creatinine, total protein). Emerging diagnostic techniques with potential clinical relevance include the use of leucocyte esterase reagent strips, platelet indices, and tumor markers. While diagnosis is typically made through standard procedures, idiopathic cases may require diagnostic laparotomy. The application of novel diagnostic tools, such as platelet indices, leucocyte esterase reagent strips, and tumor markers, can help streamline the diagnosis of ascites. Treatment is based on identifying the underlying cause of the ascites.

Keywords: ascites, hypoproteinemia, left-sided heart failurecirrhosis, hepatic diseases, renal diseases, ancylostomiasis, portal hypertension, hepatorenal syndrome, serum ascitic albumin gradient (SAAG), abdominal distension, abdominal effusion.

1. Introduction

Ascites refers to the accumulation of fluid in the peritoneal cavity and is a clinical manifestation rather than a standalone disease. It can occur as a result of various underlying conditions in animals. This means that ascites is not a condition that can be treated in isolation; instead, the root cause must be identified and addressed for effective management. Ascites can develop from a range of diseases, including hepatic disorders, various types of neoplasms, portal hypertension, hypoproteinemia (low serum protein levels), right-sided heart failure, reduced plasma oncotic pressure, and increased capillary permeability due to inflammation or infection (e.g., tuberculosis). Other causes include kidney dysfunction, pre- and post-hepatic portal hypertension, trauma (e.g., rupture of blood vessels, lymphatic vessels, or the urinary bladder), peritonitis (such as from ancylostomosis), bleeding disorders, and malnutrition. In pets, hepatic and cardiac diseases are among the most common causes of ascites.Other signs that often accompany ascites in pets, indicating an underlying condition, may include syncope, vomiting, lethargy (obtundation), seizures, anemia (evidenced by pale mucous membranes), weakness, and rapid panting. Diagnosing the cause of ascites can be complex due to the range of potential underlying diseases. The diagnostic process should begin with a thorough

physical and clinical examination, which can provide important clues about the root cause. A complete blood count (CBC) may show evidence of bacterial infection, while abdominal ultrasound or sonography helps distinguish between fluid buildup and abdominal masses or organ enlargement. Blood tests, including those for total protein, albumin, creatinine, urea, liver enzymes, and coagulation status, can help identify hypoalbuminemia, hypoproteinemia, and liver or kidney issues. Cardiac conditions may be diagnosed with an electrocardiogram (ECG), Echocardiogram and cardiac auscultation can detect murmurs or arrhythmias. Abdominal paracentesis is both a diagnostic and therapeutic procedure, often performed to reduce fluid accumulation and relieve symptoms like dyspnea (difficulty breathing). This procedure, combined with treatment of the underlying cause, plays a crucial role in managing ascites. Once the cause is accurately diagnosed, treatment focuses on addressing life-threatening symptoms, such as dyspnea, and providing the appropriate therapy based on the specific condition.

1. Classification of Ascites

Ascites can be classified based on its origin, with particular emphasis on hepatic causes. It is generally divided into three categories: pre-hepatic, post-hepatic, and hepatic.

1. **Pre-hepatic Causes**: These arise from conditions that occur before blood reaches the liver, such as portal vein thrombosis, bacterial infections like tuberculosis, malnutrition, hypoalbuminemia, and parasitic infections like strongyloidosis and entamoeba. Other causes include trauma leading to rupture of lymphatic vessels, blood vessels, or the urinary bladder, renal failure, and various types of neoplasms, including those affecting the breast, lungs, ovaries, stomach, pancreas, or colon. Around 20% of neoplastic ascites result from tumors of unknown origin.
2. **Post-hepatic Causes**: These occur when there is obstruction or impaired blood flow after the liver. Conditions include congestive heart failure, often associated with pulmonary hypertension, left-sided heart failure, right-sided heart failure, constrictive pericarditis, Budd-Chiari syndrome, and the formation of strictures or webs in the inferior vena cava.
3. **Hepatic Causes**: Ascites of hepatic origin is typically associated with liver diseases such as cirrhosis, portal hypertension, and hepatitis. About 85% of cases of portal hypertension are linked to cirrhosis.

Historically, ascites was classified into two main categories—**transudates** and **exudates**—based on the total protein concentration in the ascitic fluid. **Exudates** were defined by a high total protein concentration (>2.5 g/L), while **transudates** had a low protein concentration (<2.5 g/L). Both categories were further subcategorized into **modified transudates** and **modified exudates** depending on the exact protein levels. Transudates with less than 2.5 g/L of protein were typically associated with conditions like **portal hypertension** or **hypoalbuminemia**, whereas exudates with protein levels above 2.5 g/L were often linked to inflammatory or neoplastic conditions such as **bacterial infections, tuberculosis, malignancies**, **pancreatitis**, or **myxedema**.

However, relying solely on total protein concentration presented challenges in accurately classifying ascites, leading to the introduction of the **serum-ascites albumin gradient (SAAG)** as a more reliable diagnostic tool. With **SAAG**, ascites is classified as either **transudate** or **exudate** based on the gradient value. A **SAAG >1.1 g/dL** indicates ascites due to **portal hypertension**, while a **SAAG <1.1 g/dL** suggests ascites from other causes, such as malignancy or infection. This method has demonstrated a sensitivity of 94% and specificity of 90% for detecting **portal hypertension**, offering a more accurate approach than total protein concentration alone.

The SAAG is calculated by subtracting the ascitic fluid albumin level from the serum albumin level, both measured on the same day. A gradient greater than 1.1 g/dL is highly accurate (97-100%) in identifying ascites caused by portal hypertension, while a gradient less than 1.1 g/dL points to non-portal hypertension causes, such as **neoplasm** or other non-hypertensive conditions. This shift in diagnostic criteria has made SAAG the preferred method for classifying ascites in clinical practice today.

High gradient (>1.1 g/l) SAAG	Low gradient (< 1.1 g/l) SAAG
Portal hypertension	Bacterial infection
Cardiac diseases	Peritoneal tuberculosis
Liver cirrhosis	Pancreatic ascites
Myxoedema	Parasitic disease
Hepatitis	Ancylostomosis
Hypoalbuminaemia	Nephrotic syndrome
Portal vein thrombosis	Rupture of the urinary bladder, left-sided heart failure, right-sided heart failure, congestive heart failure

Classification of ascites based on SAAG.

2. Pathophysiology of ascites in cirrhosis

Ascites is a common and significant complication in liver cirrhosis, often indicating decompensated liver disease and a poor prognosis with reduced life expectancy. The development of cirrhosis can be attributed to various factors, with the heart playing a crucial role through a complex interaction with the liver. The precise mechanisms by which the heart and liver influence each other in the development of ascites are not fully understood. Circulatory abnormalities, which are common in cirrhotic patients, led to the peripheral arterial vasodilation hypothesis proposed in the last century. These abnormalities include increased cardiac output, portal hypertension, reduced peripheral vascular resistance, arterial hypotension, and splanchnic vasodilation. Cardiac-related circulatory disturbances affect blood volume and tissue perfusion, particularly impairing kidney function. In the early stages of the disease, renal dysfunction is minimal, but as cirrhosis progresses, the kidneys may struggle to excrete sodium, leading to sodium and water retention. Cirrhotic ascites primarily result from impaired renal sodium excretion. Several mechanisms contribute to ascites formation in cirrhosis, with intrinsic factors like arterial vasodilation impacting blood pressure-regulating hormones such as the **renin-angiotensin-aldosterone system (RAAS)**, which promotes sodium reabsorption in the distal nephron. The **sympathetic nervous system (SNS)** also stimulates renal vasoconstriction and further sodium retention. The heart, sympathetic nervous system (SNS), and renin-angiotensin-aldosterone system (RAAS) work together in a synergistic manner to promote sodium retention and contribute to the development of ascites in cirrhotic patients.

2.1 Pathogenesis of portal hypertension in ascites

The portal vein is a key vessel formed by a group of veins that supply blood to the visceral organs, including the abdomen, pancreas, and intestines and which carries deoxygenated blood to liver. These veins branch into smaller vessels within the liver tissue. Conditions like cirrhosis, along with intrinsic factors, can cause blockages in these smaller vessels, leading to increased blood pressure in the veins and resulting in portal hypertension. Ascites occurs when the post-sinusoidal pressure gradient exceeds 12 mmHg. In portal hypertension, the elevated hydrostatic pressure within the liver sinusoids allows fluid to leak into the peritoneal cavity. The severity of ascites is mainly influenced by the level of hydrostatic pressure rather than plasma albumin levels. Symptoms of portal hypertension include hematuria, dysentery, bloody vomiting due to spontaneous variceal rupture and hemorrhage, encephalopathy from impaired liver function, and thrombocytopenia. Factors such as an abnormal increase in nitric oxide production and the circulation of vasoconstrictors like catecholamines, leukotrienes, and angiotensin II contribute to higher hepatic vascular resistance, further worsening portal hypertension.

3. Diagnosis of ascites

There is typically no fixed procedure for diagnosing ascites; however, using a systematic approach tailored to different disease conditions can be helpful in reaching an accurate diagnosis. A step-by-step method usually begins with a thorough physical examination.

3.1 The diagnostic algorithm for ascites.

Standard diagnostic procedures

History
Physical examination
Clinical examination
Complete blood count
Abdominal paracentesis: For ascetic fluid analysis: visual inspection, SAAG, total protein, bacteria isolation
Abdominal radiography, Ultrasound, Computed tomography
Biochemical analysis: Urea and Creatinine, SAAG, total protein, Triglycerides
Novel diagnostic markers in ascites: Leukocyte esterase reagent strip, platelet indices, tumor markers, Laparatomy

Suspected diseases

Renal disease: Complete blood count, urea/creatinine, electrolyte balance
Cirrhosis: Ultrasound, Platelet indices, Polymophonuclear neutrophil count
Cardiac disease: ECG, Echocardiography
Hepatic diseases-hepatitis: Liver enzymes, ALT, AST ALP, Lactate dehydrogenase.
Peritoneal carcinoma
Bacterial infection: Bacterial culture and sensitivity, urinalysis, complete blood count

Physical examination:

During a physical examination, the veterinarian checks for the presence of ticks and fleas, which can cause discomfort for the pet. Tick infestations lead to blood volume depletion and anemia, which can be detected by examining the pale mucous membranes of the eyes and gums. Ectoparasitism from ticks can also contribute to malnutrition, as the ticks compete with the host for essential nutrients needed for protein and albumin synthesis. This may result in hypoalbuminemia or hypoproteinemia, leading to a decrease in plasma oncotic pressure, increased vascular permeability, and fluid leakage into the abdominal cavity. A distended or pulsating jugular vein may indicate a cardiovascular abnormality, while yellowing of the eyes suggests jaundice and potential liver disease. Generalized lymphadenopathy could indicate lymphosarcoma or other inflammatory conditions. Observing the pet from a distance can help identify signs of dyspnea due to abdominal displacement of the diaphragm, which compromises respiration. Respiratory distress may manifest as rapid panting and a shift in the normal respiratory pattern from a coastal to a costo-abdominal or abdominal pattern. Black tarry feces may indicate gastrointestinal bleeding, which could be associated with conditions like ancylostomosis or portal hypertension.

Upon examining the abdomen, signs of distension may be present, and the cause can range from pregnancy or abdominal masses to fluid accumulation or organ enlargement. To diagnose ascites, it is important to differentiate between these possibilities. One initial method is abdominal ballottement, where a clenched fist is used to assess the presence of fluid versus masses. This can be complemented with other tests, such as a pregnancy test, to rule out pregnancy. However, ballottement has limitations when it comes to distinguishing between abdominal masses and organ enlargement.

Abdominal ultrasound is a more effective tool for differentiating between abdominal contents. It is especially useful for detecting ascitic fluid, identifying its site of production, and distinguishing between transudates and exudates. Recent research has also shown that examining the echotexture of ascitic fluid can help determine the underlying cause of ascites.. However, abdominal paracentesis remains an important technique for further differentiating the nature of ascitic fluid.

Diagnostic fluid markers in ascites

The color of ascitic fluid is a crucial indicator in diagnosing the cause of ascites. It can vary from clear to yellowish, reddish, or opaque, often containing fibrin flakes and debris, depending on the underlying etiology. The color of ascitic fluid can provide important clues to its underlying cause, as outlined below.

1. **Pinkish discoloration**:
   This is typically seen in cases of bacterial infection, where the fluid may become purulent. The exudate is often turbid, containing more than 2.0 gm of protein and over 6,000 cells/μL, predominantly neutrophils, with evidence of significant bacterial infection. This type of ascites is considered a medical emergency due to the risk of sepsis.
2. **Clear straw-colored fluid**:
   This is usually a modified transudate, characterized by the presence of fibrin cells and white blood cells like neutrophils and lymphocytes. It is commonly observed in long-standing ascites due to conditions such as right-sided heart failure, cancer, or liver disease, which may allow fibrinogen to invade the fluid.
3. **Clear opaque fluid**:
   This type of fluid is a pure transudate, typically free from contamination except for a few mesothelial cells and tissue macrophages. It is often seen in conditions like portal hypertension, liver diseases, hypoalbuminemia, protein-losing enteropathy, kidney impairment, and albuminuria.
4. **Reddish discoloration**:
   This is indicative of hemorrhage and the presence of frank blood in the peritoneal cavity, often due to trauma, coagulopathies, or blood vessel neoplasms. The fluid usually contains a high number of red blood

cells, with a packed cell volume (PCV) greater than 20%.

5. **Greenish discoloration**:
   This is seen when bile leaks into the peritoneal cavity, often due to bile duct rupture or gallbladder issues.

Milky or slightly yellowish discoloration:

This suggests a collection of lymph in the peritoneal cavity, often resulting from trauma, infection, cancer, or right-sided heart failure. The fluid is turbid and opaque, often referred to as chyle. It tests positive for lipids with Sudan III staining due to its high lipid content.

Clinical examination

Clinical examination plays a crucial role in identifying the cause of ascites. An elevated body temperature may indicate an underlying infectious or inflammatory condition, such as bacterial tuberculosis. Prolonged capillary refill time can suggest decreased circulatory volume, often due to cancer or an infectious process. Auscultation of the heart can reveal various cardiovascular issues, such as muffled heart sounds, which are indicative of pericardial effusion or cardiac tamponade. Heart murmurs or irregular rhythms may point to right-sided heart failure. An increased heart rate or tachypnea could be a response to dyspnea, often caused by the cranial displacement of the diaphragm into the thoracic cavity. Cardiovascular abnormalities can be further confirmed with electrocardiography and echocardiography.

Biochemical investigation in ascites

The assessment of biochemical markers, including liver enzymes, total protein levels, serum-ascitic albumin gradient (SAAG), albumin concentration, total bilirubin, as well as kidney function markers like creatinine and urea levels, is essential in diagnosing the underlying cause of ascites.

Serum ascites albumin gradient (SAAG)

The serum-ascitic albumin gradient (SAAG) is currently considered the most reliable method for diagnosing the cause of ascites, particularly when it is related to portal hypertension. A SAAG value of 1.1 g/dl (or 11 g/l) is indicative of ascites caused by portal hypertension.

Total protein

While the traditional classification of ascites into transudates and exudates has become less prominent with the introduction of SAAG, it remains relevant in clinical practice for comparative analysis and prognostic assessment. Protein concentrations below 15 g/l are often associated with an increased risk of spontaneous bacterial peritonitis in cirrhosis.

Triglycerides

An elevated triglyceride concentration in ascetic fluid above 2.2 mmol/l indicates chylous ascites.Chylous ascites is common in neoplastic cases although it may occur in 6% of cirrhosis **.**

Urea and creatinine

Elevated levels in urea and creatinine concentrations in ascetic fluid indicate prerenal failure due to peritoneal absorption of urea.Urinary ascites is often associated with bladder changes and urethra obstruction.

Cytology

Cytology of ascetic fluid is often indicated in suspected malignancy and idio pathic cases. Positive cytology is highly indicated in suspected cases of peritoneal carcinomatosis. The sensitivity of cytology can be enhanced by examination of three samples from separate paracenteses. The sensitivity is also enhanced by prompt analysis of ascetic fluid and obtaining large volume of up to 50–1000 ml in patients with initial negative result.

Diagnostic laparotomy

Diagnostic laparotomy is recommended when the cause of ascites is difficult to identify. It allows for direct visual inspection of the peritoneal cavity and provides an opportunity for biopsy collection for histological and microbiological analysis. This procedure is particularly useful in diagnosing conditions such as peritoneal carcinomatosis and tuberculous peritonitis.

Leucocyte esterase reagent strip

Several studies have demonstrated the effectiveness of leucocyte esterase reagent strips in diagnosing spontaneous bacterial peritonitis (SBP) and in urinary analysis, with sensitivity ranging from 80% to 93% and specificity from 93% to 98%. The negative predictive value is notably high, ranging from 97% to 99%, making it a reliable tool for ruling out SBP. Recent developments have introduced an ascites-specific reagent strip with a cutoff value of 250 cells/mm³, further improving diagnostic accuracy.

Platelet Indices

Increased platelet indices, such as mean platelet volume and platelet distribution width, have been observed in cirrhosis. Although the full clinical value of these indices is yet to be determined, they show potential as diagnostic tools.

Tumor Markers

The use of tumor markers such as alpha-fetoprotein, des-gamma-carboxyprothrombin, and cancer antigen 125 in diagnosing cancer in ascitic fluid is still debated. While elevated levels of these markers are associated with malignancies, they can also be seen in conditions like pancreatitis and gastritis.

Imaging Tools

Radiographic imaging is valuable for detecting small amounts of ascitic fluid and for identifying its underlying cause. Abdominal ultrasonography can detect as little as 100 ml of intraperitoneal fluid.while computed tomography (CT) enhances sensitivity by identifying minute fluid quantities. CT imaging also aids in evaluating internal organ conditions, such as cirrhosis, intra-abdominal tumors, and organ enlargement. It can identify thickening of the mesentery and bowel walls, matting of bowel loops, and enlarged mesenteric lymph nodes, which may suggest tuberculosis peritonitis. Contrast CT may show enhancement of the peritoneal lining, and scintigraphy with technetium sulfur colloid or radiolabeled albumin can assist in diagnosing cirrhosis and large hydrothorax.

Bacterial Culture

Spontaneous bacterial peritonitis (SBP) can develop due to decreased complement levels in ascitic fluid, which serve as antibacterial factors. Suspicion of SBP warrants culturing ascitic fluid in both aerobic and anaerobic blood media to isolate the causative organisms. Additionally, Mycobacterium tuberculosis DNA can be detected in ascitic fluid via polymerase chain reaction (PCR), offering a sensitivity of 94%, which is superior to the acid-fast smear (0%) and mycobacterial culture (50%).

General Treatment Options in Ascites

Ascites is primarily treated by addressing the underlying cause and alleviating the associated symptoms. The goal is to relieve symptoms and prevent the progression of ascites. In cases of congestive heart failure, the primary focus is to enhance cardiac contractility, normalize arrhythmias, and improve cardiac output. Medications such as dopamine and digoxin can be administered according to recommended dosages for dogs with congestive heart failure. Dogs with right-sided heart failure should be kept on cage rest and placed on a sodium-restricted diet

Paracentesis is performed to relieve abdominal pressure on the diaphragm and improve respiration. Repeated paracentesis is typically not necessary unless the initial treatment is ineffective. The amount of fluid removed during paracentesis should not exceed 1.0 kg per day for dogs with both ascites and peripheral edema, or 0.5 kg per day for those with ascites alone. Serum albumin levels may decrease during paracentesis, so it should be monitored and replenished intravenously if needed, at the same volume of fluid removed. Administering albumin at a dose of 1.5 g/kg on the first day and 1.0 g/kg on the third day has been shown to preserve renal function and reduce mortality.

For cases of syncope, isotonic crystalloid fluid replacement, such as Plasmayte A, Normosol R, or 0.9% saline, can be used for resuscitation and to treat conditions like hypernatremia, hyponatremia, hypercalcemia, metabolic alkalosis, or oliguria-related renal failure. Diuretics are often used alongside paracentesis to relieve ascites. These diuretics are typically administered once daily. Spironolactone, with a 24-hour half-life, is given at a dose of 100 mg/day, with a maximum dose of 400 mg/day for a response. In hospitalized dogs, spironolactone may be administered as 2 mg/kg intramuscularly every 2 hours, and 3 mg/kg orally at night. Alternatively, spironolactone can be replaced by triamterene or amiloride, both of which effectively counteract aldosterone's action on the collecting tubules. Furosemide, commonly used as the first-line treatment for ascites, has a half-life of 1.5 hours and is typically dosed at 40 mg/day, with a maximum dose of 160 mg/day for patients who do not respond to initial treatment. The dose can be divided into 3 mg/kg intravenous doses every 2 hours and 4 mg/kg orally at night. To minimize the risk of furosemide resistance, bumetanide and spironolactone can be combined with furosemide in a ratio of 100:40. This combination enhances natriuresis, improves fluid balance, and reduces the likelihood of potassium deficiency due to furosemide use.

Other diuretics, such as torsemide and bumetanide, have demonstrated superior efficacy compared to many other diuretics. Torsemide, in particular, has a longer half-life than both furosemide and bumetanide. Patients with cirrhotic ascites often experience complications such as spontaneous bacterial peritonitis (SBP), portal hypertension, and

hepatorenal syndrome (HRS). Cases without these complications are referred to as "uncomplicated ascites."

The standard treatment for SBP in humans typically involves the immediate administration of a third-generation cephalosporin, such as intravenous ceftriaxone (1 to 2 g daily for 5 days), with a dose of 1 g daily recommended for dogs. Oral fluoroquinolones are also effective for treating SBP, with piperacillin and tazobactam as alternative options. The choice of antibiotic depends on the results of culture and sensitivity tests to minimize issues with drug resistance. Antibiotics are usually given over an extended period to ensure the complete resolution of the infection.

Portal hypertension is managed with antihypertensive medications. For example, metolazone (Mykron, Zaroxolyn) helps eliminate edema in congestive heart failure by inhibiting sodium reabsorption in the distal tubules, which is beneficial for renal conditions. Mannitol (Osmitrol) works by increasing the osmotic pressure of the glomerular filtrate, thereby enhancing urine output and preventing electrolyte reabsorption. In cases of recurrent ascites due to portal hypertension, the use of transjugular intrahepatic portosystemic shunt (TIPS) may be required. TIPS creates a side-to-side anastomosis between the high-pressure portal system and the low-pressure hepatic vein, effectively reducing portal pressure and aiding in the management of ascites. This reduction in portal hypertension leads to decreased activation of the renin-angiotensin-aldosterone system (RAAS) and an increase in sodium excretion.

Persistent ascites caused by cirrhosis may be treated with a liver transplant, which is considered only after all other treatments have been exhausted. The liver has a remarkable regenerative capacity, and hepatocytes can function even when few viable cells remain, making liver transplants a last resort.

Renal failure is addressed by controlling blood pressure with appropriate medications, avoiding hepatotoxic drugs, and refraining from the use of non-steroidal anti-inflammatory drugs (NSAIDs) such as acetaminophen. In severe cases of kidney damage, continuous venovenous hemodialysis (CVVHD) is preferred over intermittent dialysis.

Encephalopathy resulting from hepatic failure is best managed in an intensive care unit (ICU). Early signs of encephalopathy may be treated on an outpatient basis, but patients should be monitored closely for progression to grade II encephalopathy, which requires ICU admission. Such patients should be routinely assessed for mental status changes, with sedatives like low-dose short-acting benzodiazepines used to manage restlessness. Sedated patients should be kept at rest to avoid movements that could increase intracranial pressure. In cases of late-stage encephalopathy, dyspnea can be managed with intratracheal intubation to prevent aspiration pneumonia. Cerebral edema and intracranial hypertension, common in late encephalopathy, are prevented by regularly monitoring the patient's renal function, liver enzymes, protein levels, glucose, electrolytes, and acid-base balance, along with neurological assessments to detect any elevated levels.

Severe bleeding associated with coagulopathies can be treated by addressing the underlying coagulopathy with transfusions of coagulation products like fresh frozen plasma and platelets, along with vitamin K administration. In extreme cases, transfusions of packed red blood cells may be necessary. Persistent bleeding following significant transfusions may indicate retroperitoneal bleeding. Several herbal and antioxidant medications have shown potential benefits in treating ascites of hepatic origin. Although the use of these drugs remains controversial, N-acetylcysteine and Silybum marianum continue to be the preferred treatments for acetaminophen-induced hepatic damage and hepatic dysfunction, respectively

Conclusion

Ascites is a common condition in pets, especially middle-aged dogs, and can result from various causes, including cirrhosis and cardiac diseases. Accurate diagnosis involves physical and clinical examinations, blood tests, cytology, and biochemical analyses. Advances in diagnostic tools, such as platelet indices, leucocyte esterase reagent strips, tumor markers, and bacterial DNA detection, enhance the accuracy of ascites diagnosis. Treatment focuses on addressing the underlying cause and

relieving symptoms, with various therapeutic options available depending on the specific etiology.

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