

HEMORRHAGIC GASTROENTERITIS

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Hemorrhagic Gastroenteritis, renamed Acute Hemorrhagic Diarrhea Syndrome (AHDS) is a disease often without a known cause. More prevalent in young, small breed dogs, it can strike any dog at any age of any breed at any time. It is thought to possibly be caused by exposure to a novel food item or perhaps an atypical immune response to bacteria. Clostridial species of bacteria may play a role but its significance is not yet understood. Dogs suffering from AHDS are often previously healthy before acutely developing vomiting and hematochezia. Quick recognition of the problem and aggressive treatment by the veterinary team is needed, and with treatment, the prognosis for these patients is good.

History and Clinical Signs:

Acute Hemorrhagic Diarrhea Syndrome is often readily recognized by the owner. The disease starts with the dog exhibiting depressed mentation and the dog may start vomiting but signs rapidly progress to explosive and voluminous hematochezia. Due to large numbers of clotted red blood cells in the diarrhea, it is often described as strawberry jam in appearance. Owners may note anorexia prior to the diarrhea. It is important to collect a complete history on these patients, as medication (such as NSAID) ingestion can lead to hematochezia as well as parasitic infection, viral infection, and systemic disease.

Examination:

When these patients arrive at the hospital, they should be immediately triaged and their perfusion status noted. Heart rate, mucous membrane color, capillary refill time, pulse quality and blood pressure should be measured and used to determine perfusion.

Large volumes of liquid diarrhea can quickly lead to hypovolemia and AHDS dogs often present to the hospital already exhibiting the clinical signs of shock. The cause of shock in small animal patients is the result of a deficit of oxygen supply to tissues and the oxygen demand of those tissues. The body has baroreceptors to warn the body of decreased blood flow in an attempt to avoid cell death. These receptors are located in the aorta and kidneys and in times of hypovolemia sense when blood flow is low and signal back to the body that blood flow is lacking. When low blood flow in the kidneys is detected, the Renin-Angiotensin-Aldosterone System (RAAS) is kicked off. When the kidneys sense that blood flow through the organ is less than normal (like in hypotension and hypovolemia) renin is released. Renin acts on circulating angiotensinogen and changes it to angiotensin I. Angiotensin I is changed to angiotensin II with the aid of angiotensin converting enzyme (ACE) which is produced by the lungs. Angiotensin II will cause vasoconstriction and increased sodium retention by the kidneys. The increase in sodium will lead to an increase in circulating volume which, when combined with vasoconstriction, will increase the blood pressure and perfusion to the kidneys. Angiotensin II will signal the adrenal glands to secrete aldosterone. Aldosterone will also signal the kidneys to retain sodium and excrete potassium. This will further contribute to increased circulating volume and increased blood pressure for the patient. The body performs this adaptive response to stimulus and keeps cells oxygenated and blood pressure maintained. These compensatory efforts will display as subtle clinical signs in early shock and are often missed by pet owners.

As shock progresses without any intervention, these compensatory mechanisms are soon overwhelmed and can no longer keep up the "business as usual" blood flow. Vasoconstriction begins to preferentially decrease blood flow to major organ systems (beginning with the periphery, then the gastrointestinal tract, progressing to the liver, then kidneys, and eventually brain) in order to maintain as much perfusion as possible to the heart, lungs, and brain. This whole body decompensation exhibits in the clinical signs of shock: pale mucous membranes, poor pulse quality, decreased blood pressure, and depressed mentation. If these signs are ignored and treatment not administered, organ systems will fail due to lack of oxygen and the animal will die.

Stabilization of AHDS:

Once the possibility of AHDS and more importantly, hypovolemia is recognized, immediate treatment should begin. Diagnostics are important, but should wait until the patient is more clinically stable. The most important goal is volume replacement, as these dogs are often suffering an acute loss of salt and water through vomit and diarrhea. A large bore short length IV catheter will deliver a large amount of fluids quickly, and should be placed in a cephalic or jugular vein. Crystalloid fluids are electrolyte and water replacement (LRS, Plasmalyte-A, 0.9%NaCl for

example) and are administered quickly in an attempt to restore circulating volume and therefore oxygen delivery to cells. Given intravenously, these fluids will remain in the intravascular space for about 45 minutes, improving perfusion and oxygen delivery, before shifting into other areas of the body. These other areas include the interstitial space but in some patients can also include the GI tract, the lungs, or can contribute to peripheral edema.

Treatment starts with a bolus of 20ml/kg ($\frac{1}{4}$ of the dog's blood volume of 90ml/kg) and vitals closely monitored for improvement. Once the bolus is complete, the vital signs are once again assessed. The bolus amount is repeated until vital signs begin to improve. It is important for veterinary technicians and nurses to closely monitor patients during fluid administration for signs of improvement, or for signs of fluid shifts and further losses. Heart rate, mucus membrane color and CRT, and blood pressure should be recorded after each bolus. Urine production, diarrhea, vomiting, and respiratory status should also be monitored and noted as a sign that fluids are quickly shifting from the intravascular space.

The stabilization period for these dogs can be lengthy; it may take hours to stabilize them to where their heart rate and blood pressure normalize. Because these dogs are often small breeds, monitoring them closely for fluid overload should occur, as well as monitoring their temperature closely. As their perfusion parameters return to normal, ECG changes are common and should be watched. Reperfusion injury and ischemia can cause VPCs and patients should be monitored and treated as needed for arrhythmias.

Diagnosis:

Once the dog is stable, or during the stabilization process, diagnostics can begin. AHDS is a diagnosis based on ruling out other diseases and situations that can cause hematochezia. Blood should be drawn at the time of IV catheter placement and a CBC and blood chemistry panel run. A classic indicator of AHDS is hemoconcentration (elevated PCV above 60%) with a normal or slightly low total protein reading. These patients are often hypoalbuminemic as they are losing large amounts of protein through their diarrhea. Dogs should be tested for parvovirus, intestinal parasites, bacterial infections, GI foreign body, and toxin exposure to rule out those as the cause for hematochezia. Radiographs and abdominal ultrasound are often utilized in the diagnostic process to rule out systemic reasons for diarrhea.

Hospitalization and Monitoring:

Treatment of AHDS is aimed at maintaining perfusion while replacing and maintaining fluid losses. Intravenous crystalloid therapy is continued and closely monitored until losses decrease and the patient can eat and drink reliably. Monitoring is focused on heart rate, mucous membrane color and capillary refill time, pulse quality, and blood pressure. Body weight should be measured every 8-12 hours to ensure appropriate fluid therapy which can be adjusted for weight loss or gain.

Gastric motility may be a concern in these dogs, as hypoperfusion can cause decreased motility. A metoclopramide CRI can help stimulate motility. Metoclopramide is also an anti-emetic, helpful in these patients. Ondansetron and maropitant are also commonly used medications to decrease nausea and vomiting. Aspiration pneumonia can be a complication in more severe cases and keeping their gastric residual volumes low and preventing vomiting/regurgitation is important. Gastroprotectants can be considered in the treatment of AHDS, especially if hematemesis is present. The use of probiotics is increasing in veterinary medicine and especially in cases of acute diarrhea. More prospective studies are required to know their exact benefit, but anecdotal use is promising.

While hematochezia and decreased perfusion due to hypovolemic shock brings concern for a breakdown in gut mucosal barrier and the potential for gut-derived sepsis, AHDS patients typically do not experience these complications, perhaps due to their immune system remaining normal. Systemic antibiotic use is generally not recommended in these cases. Metronidazole, long used in cases of acute diarrhea, may shorten the duration of diarrhea but most dogs will recover without the use of metronidazole. However, regardless of diagnosis, any patients that experiences leukopenia, prolonged hypotension, and is hemodynamically unstable may require antibiotic therapy.

Nutrition may need to be supplemented in patients that do not quickly respond to fluid therapy or those that suffer from profound hypoalbuminemia. As long as the patient is not vomiting, placing a NG or NE tube is relatively simple and can provide immediate nutrition to the ailing enterocytes. The decision to place the tube all of the way

into the stomach should be made based on volume of fluid in the stomach, as having a way to remove volume is ideal in order to decrease regurgitation (and the risk for aspiration pneumonia) and increase patient comfort.

Pain management should not be overlooked, as there is cramping and gut pain associated with hemorrhagic diarrhea. While opioids are known to cause ileus they are still commonly used in the early treatment stages of these cases, but monitoring the patient and switching them to a less potent medication should happen as soon as possible.

Nursing care is intensive with these dogs. Keeping them clean is the most time-consuming care. Diarrhea left on hair and skin can irritate and burn the skin, leaving the dog with a painful rash that can last for days. In long-haired dogs the desire is to shave the hair away to keep them clean, but hair can be a barrier and help keep skin safe. Clipping feathers away is fine, but shaving them to the skin will lead to more intense irritation. Frequent bathing and caring for the skin in the perianal region is necessary, sometimes with a diaper rash product. Monitor these patients closely for blood pressure and perfusion deficits, monitor their pain levels, and offer them food and water as soon as they are no longer vomiting. Once they can eat and their diarrhea has ceased, they can return home. AHDS patients, when treated promptly and aggressively, can make a full recovery.

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