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Acute Haemorrhagic Diarrhoea Syndrome in Dogs: Current Understanding of Aetiology, Diagnosis and Management

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Acute haemorrhagic diarrhoea syndrome (AHDS), formerly referred to as haemorrhagic gastroenteritis (HGE), is a common emergency condition in dogs characterized by the sudden onset of haemorrhagic diarrhoea, severe haemoconcentration, and rapid dehydration. Although the exact cause remains uncertain, growing evidence suggests a multifactorial pathogenesis involving intestinal dysbiosis and toxigenic strains of *Clostridium perfringens*. Prompt recognition and aggressive supportive care are essential, as affected dogs can deteriorate rapidly due to hypovolemic and distributive shock. This review summarizes current knowledge regarding the etiology, clinical features, diagnostic approach, therapeutic management, and prognosis of AHDS in dogs.

Introduction

Acute haemorrhagic diarrhea syndrome (AHDS) is an acute gastrointestinal disorder characterized by the sudden development of bloody diarrhea, often accompanied by vomiting and marked hemoconcentration. The term AHDS has largely replaced the historical designation haemorrhagic gastroenteritis (HGE) because affected dogs typically exhibit minimal or no gastric inflammation. The condition can occur in dogs of any breed and age; however, small and toy breeds are disproportionately represented in clinical reports. The disease is considered a medical emergency because substantial fluid losses into the intestinal lumen may rapidly result in severe dehydration, electrolyte disturbances, and circulatory shock. Early intervention is therefore critical for successful outcomes.

Etiology and Pathogenesis

Despite extensive investigation, the precise cause of AHDS remains incompletely understood. Several mechanisms have been proposed, including dietary indiscretion, bacterial toxins, intestinal dysbiosis, and abnormal host immune responses. A consistent finding in many affected dogs is the presence of large numbers of *Clostridium perfringens* within the small intestine. Whether bacterial proliferation represents a primary cause of disease or develops secondary to intestinal injury remains unclear. Traditional enterotoxins produced by *C. perfringens* and toxins A and B produced by *Clostridioides difficile* do not appear to play a central role in disease development. Recent studies have identified the pore-forming toxin NetF, produced by type A *C. perfringens*, in association with severe necrotizing intestinal lesions. This discovery has renewed interest in the potential contribution of bacterial virulence factors to disease pathogenesis. Furthermore, acute enteritis causes profound alterations in the intestinal microbiome, reducing bacterial diversity and resulting in significant dysbiosis that may contribute to disease severity.

Epidemiology and Clinical Presentation

Although AHDS can affect any dog, middle-aged small-breed dogs appear particularly susceptible. Breeds reported to be overrepresented include Yorkshire Terriers, Miniature Schnauzers, Miniature Pinschers, and Maltese dogs. The average age of affected dogs is approximately five years. Clinical signs usually develop abruptly. The hallmark feature is profuse haemorrhagic diarrhea, often described as containing large amounts of fresh blood. Vomiting, anorexia, lethargy, abdominal discomfort, and varying degrees of dehydration are frequently observed. In severe cases, rapid fluid loss may result in hypovolemic shock characterized by weak pulses, prolonged capillary refill time, tachycardia, and altered mentation. Without timely treatment, circulatory collapse and life-threatening complications may occur.

Diagnostic Approach

Diagnosis is primarily based on clinical presentation combined with characteristic laboratory findings. One of the most notable abnormalities is marked hemoconcentration, with packed cell volume (PCV) commonly exceeding 60–65%, despite normal or decreased plasma protein concentrations. Hematologic abnormalities are variable and may include leukocytosis, leukopenia, or mild thrombocytopenia. Serum biochemical analysis frequently reveals prerenal azotemia, electrolyte imbalances, hypoglycemia, and increased hepatic enzyme activities. Metabolic acidosis may develop secondary to poor tissue perfusion and gastrointestinal fluid losses. Abdominal radiography generally reveals nonspecific evidence of enteritis, including fluid- or gas-distended intestinal loops, and is primarily useful for excluding surgical gastrointestinal disease. Because no single diagnostic test confirms AHDS, clinicians must rely on a combination of historical information, physical examination findings, laboratory abnormalities, and exclusion of other causes of acute haemorrhagic diarrhea.

Differential Diagnoses

A thorough diagnostic investigation is essential to distinguish AHDS from other conditions that may present with acute haemorrhagic diarrhea and vomiting. Important differential diagnoses include:

- Canine parvoviral enteritis
- Salmonellosis and campylobacteriosis
- Clostridial enteropathies
- Severe intestinal parasitism
- Dietary indiscretion
- Toxicity, including mushroom intoxication and anticoagulant rodenticide exposure
- Intestinal intussusception or volvulus
- Acute necrotizing pancreatitis
- Acute hepatic disease
- Hypoadrenocorticism
- Sepsis
- Immune-mediated thrombocytopenia

Treatment and Clinical Management

The cornerstone of AHDS therapy is aggressive fluid resuscitation. Restoration of intravascular volume is essential to correct dehydration, improve tissue perfusion, and reverse shock. Initial treatment typically consists of intravenous isotonic crystalloid boluses of 10–20 mL/kg administered according to the patient's cardiovascular status. Perfusion parameters should be reassessed frequently, and additional boluses provided as required. Once stabilization is achieved, remaining fluid deficits should be corrected over the subsequent 6–12 hours while accounting for maintenance requirements and ongoing gastrointestinal losses. In patients with refractory distributive shock, vasopressor support may be necessary. Electrolyte abnormalities, particularly hypokalemia, should be identified and corrected promptly.

Supportive medications are frequently indicated. Maropitant (1 mg/kg IV or SC every 24 hours) is commonly used to control vomiting. Gastroprotective therapy may be considered in dogs with suspected gastric mucosal compromise secondary to hypoperfusion. Common options include pantoprazole (1 mg/kg IV every 12 hours) or omeprazole (1 mg/kg PO every 12 hours). The routine use of antimicrobial therapy remains controversial. Current evidence suggests that dogs with uncomplicated mild-to-moderate AHDS derive little benefit from empirical antibiotic administration. Consequently, antimicrobial treatment is generally reserved for patients with suspected sepsis, severe systemic inflammatory response syndrome, or evidence of intestinal mucosal sloughing. Nutritional support should be introduced as early as clinically feasible. Following a brief fasting period of no more than 12–24 hours, small and frequent meals consisting of highly digestible diets are recommended. Early enteral nutrition helps maintain intestinal barrier integrity and may facilitate recovery. Probiotic supplementation has gained increasing attention as an adjunctive therapy. Probiotics may promote restoration of a healthy intestinal microbiome, support mucosal repair, and reduce the duration of diarrhoea. Veterinary-specific probiotic formulations produced by reputable manufacturers are preferred because product quality and microbial viability are generally more reliable.

Prognosis

The prognosis for dogs with AHDS is generally favourable when diagnosis and treatment occur early. Most patients respond rapidly to aggressive fluid therapy and supportive care, with clinical improvement occurring within 24–48 hours. Hospitalization typically ranges from one to seven days, with a median duration of approximately three days. Nevertheless, severe complications can occur, including disseminated intravascular coagulation (DIC), bacterial sepsis, and aspiration pneumonia in vomiting patients. Delayed presentation or inadequate treatment significantly increases the risk of mortality.

Conclusion

Acute haemorrhagic diarrhea syndrome remains one of the most common gastrointestinal emergencies encountered in canine practice. Although its exact pathogenesis has yet to be fully elucidated, accumulating evidence supports a complex interaction between intestinal dysbiosis, bacterial virulence factors, and host responses. Early recognition, prompt fluid resuscitation, correction of electrolyte abnormalities, and appropriate supportive care are the primary determinants of successful outcomes. Future research aimed at clarifying the underlying mechanisms of disease may facilitate the development of more targeted therapeutic strategies.