Focus on acute haemorrhagic diarrhoea syndrome – Part 2

In this the second part of a two-part series *Bryn Jones*, from Willows Veterinary Centre and Referral Service, focuses on testing, treatment and the aftermath of AHDS.

Acute haemorrhagic diarrhoea syndrome (AHDS) is encountered frequently in practice, particularly in the emergency setting. It has a peracute/acute onset and manifests as voluminous haemorrhagic diarrhoea, often preceded by vomiting, without an immediately identifiable cause. The first part of this article (BSAVA *Companion*, March 2025, pp. 10–14) discussed the potential presentations of this illness, alongside the pathogenesis. Possible underlying causes of AHDS were also outlined, with a specific focus on the likely role of clostridial toxins. This second part will focus on an optimal diagnostic pathway, therapy and likely outcomes of this syndrome.

Diagnosis

Presentation

AHDS is diagnosed based on presenting signs alongside exclusion of differential diagnoses. The most common differential for a dog presented with acute haematochezia is likely to be acute colitis. Differentiation between AHDS and acute colitis is largely subjective, although dogs with acute colitis should have minimal systemic signs, no vomiting, and a lower volume of diarrhoea, with mucus and tenesmus frequently observed. Acute colitis can be caused by dietary indiscretion/intolerance, infectious pathogens (e.g. *Campylobacter* sp., *E. coli*) or even environmental stress. More often, an underlying trigger is not identified. In most dogs with acute colitis empirical, supportive, outpatient treatment is appropriate (e.g. probiotics, dietary modification) and the episode is self-limiting. Acute colitis and AHDS can both be associated with acute decompensation of chronic enteropathy; therefore, interrogation of the history is still advisable in case further investigation or follow-up care are indicated.[1]

Other differentials for gastrointestinal haemorrhage should be considered, including coagulopathy (particularly disorders of primary haemostasis i.e. thrombocytopenia or thrombocytopathy) or structural diseases (e.g., ulcers, neoplasia). Dogs with these causes of melaena are often anaemic rather than haemoconcentrated and are perhaps more likely to have reticulocytosis by the time of presentation. Faeces is more typically dark brown/black and can be tarry. These dogs are typically presented as emergencies due to anaemia or perforation rather than hypovolaemia. A careful consideration of the history should be undertaken for any suggestion of underlying causes e.g., NSAID administration, or unexplained weight loss. The physical examination should include a thorough assessment for any other signs of mucosal or cavitary haemorrhage. Pyrexia is not common in AHDS, and should prompt consideration of infectious disease, sepsis or involvement of other organs.[1]

Blood tests

Alongside or following stabilization, complete blood count and serum biochemistry (including electrolytes) is advised to exclude extra-GI causes of haemorrhage (e.g., hepatopathy, uraemia, thrombocytopenia) and identify any complications (e.g. neutropenia, acute kidney injury (AKI), hypoalbuminaemia, electrolyte derangements).[2] Manual PCV measurement is considered more accurate than automated. Ongoing re-evaluation of PCV can be used as a marker of hydration status. Serum albumin concentration should be assessed after rehydration. Increased urea concentration with a normal creatinine concentration can lend a further clue to the presence of gastric haemorrhage.

Hypoadrenocorticism has been associated with an acute haemorrhagic diarrhoea presentation,[3,4] so basal cortisol measurement should be considered in all patients, especially with any suggestive signalment (e.g., middle-aged, female, predisposed breed such as Poodle or Bearded Collie) or history. Dogs can have glucocorticoid-deficient ('atypical') hypoadrenocorticism in the absence of mineralocorticoid deficiency, without classic electrolyte derangements.

Although serum CRP concentration does variably correlate with clinical severity, there is no indication currently that its measurement is clinically useful – the value at time of admission was not associated with length of hospitalization.[5,6]

Imaging

Occasionally, abdominal imaging should be considered in AHDS if there is a concern for structural disease. Ultrasonography is likely to be more sensitive than radiography for detecting neoplasia, ulcers or perforation. Another benefit of ultrasonography is that intestinal motility and fluid loss can be subjectively assessed. The diagnostic utility of ultrasonography for canine diarrhoea has been questioned, with no utility or counterproductive findings in 60% of dogs.[7] As acute pancreatitis rarely results in a haemorrhagic diarrhoea presentation,[8]

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this differential may be considered but presents a diagnostic challenge. Some dogs with primary intestinal disease have changes to lipase activities and the pancreas ultrasonographically. Most dogs with 'idiopathic' AHDS do not appear to have marked changes to either. In younger dogs with an acute history who stabilize rapidly, it seems reasonable to reserve imaging for patients who do not progress as expected or develop complications.

Faecal testing

The most important differential for AHDS is parvoviral enteritis (CPV-2). Some form of CPV testing is likely indicated for dogs with acute haemorrhagic diarrhoea, especially if young, unvaccinated or neutropenic. Faecal point-of-care tests are most commonly used and based on detection of CPV-2 antigen. In the UK the IDEXX SNAP Parvo Test (IDEXX, Hoofddorp NL) has a reported sensitivity and specificity of 100%. Recent vaccination is not reported to induce false positivity.[9,10] These observations are based on relatively small studies. True sensitivity closer to 80% has been reported when compared to PCR.[11] Faecal antigen is typically detectable 3–12 days post-exposure (waning by day 8), which usually correlates with clinical onset, but may be extended in some dogs.[12] Whether differentiating between parvoviral enteritis and AHDS affects treatment is debatable, although the former carries a less favourable prognosis and identification is important for infection control purposes.

Faecal analysis, culture and PCR can identify specific pathogens present. For many dogs where a bacterial or viral pathogen is identified it is unlikely the sole cause of AHDS due to significant prevalence in non-diarrhoeic dogs too. Giardiasis does not typically result in profuse haemorrhage or systemic illness, so co-infection or subclinical carriers should be considered where this is identified. There does not appear to be any benefit in testing for *Clostridia perfringens* or CPE/*cpe*, and this is no longer recommended.[13] The author generally considers faecal analysis/culture only for dogs that fail to improve as expected, those fed raw-food diets, or where there is concern for an outbreak, e.g. if signs develop during hospitalization or in multiple dogs from the same household/facility.

Treatment and complications

Fluids and electrolytes

Often, dogs with AHDS are presented dehydrated or with hypovolaemic shock. Fluid losses can be rapid, and faecal volume observed externally is not always representative of the degree of intestinal losses. Dogs can be hypovolaemic without external signs of dehydration due to the delay in interstitial fluid shift. For dogs who are collapsed, very lethargic or tachycardic, blood pressure should be measured.

Most dogs with shock are responsive to fluid resuscitation, with fluid boluses often utilized initially. A common protocol is to administer 10–15 mg/kg over 5–10 minutes, followed by reassessment of clinical parameters. Multiple boluses can be administered, especially if there is a clinical response each time, (e.g. reduced heart rate, increased blood pressure, improved mentation). Vasopressors are rarely required unless sepsis is present. It should be appreciated that an estimated 80% of intravenous isotonic crystalloid fluids are absorbed into the interstitial space within the hour, necessitating further fluid administration even without external loss.[14]

Estimated dehydration	Physical examination findings
<5% (subclinical)	Normal
5–6% (mild)	Tacky or dry mucous membranes
6–8% (moderate)	Dry mucous membranes Skin tenting Tachycardia Normal pulses and blood pressure
8–10% (severe)	Dry mucous membranes Further skin tenting Weaker (or sometimes hyperkinetic) pulses
>10–12% (hypovolaemia)	Dry mucous membranes Increased capillary refill time Skin tenting (severe) Tachycardia or bradycardia Weak/absent pulses Altered mentation Cold extremities Hypothermia Hypotension
Fluid deficit (ml) = % dehydration x bodyweight (kg) x 1000 Maintenance requirements (ml) per 24 hours = (30 x bodyweight [kg]) + 70	

TABLE 1: Estimation of hydration status and fluid requirements. Reproduced/adapted from multiple sources.[15,16]

Once clinical signs of shock (e.g. hypotension, tachycardia, recumbency) have dissipated, fluid therapy is continued that accounts for dehydration correction (minus any volume of fluid already administered during stabilization), maintenance requirements, and further losses (Table 1).

An estimated 73% of dogs with AHDS have dehydration >5% (i.e. detectable on clinical examination) on presentation.[5] A reasonable target is to replace deficits within the first 24 hours, although in retrospective studies replacement is usually faster.[5] Administering the calculated volume too quickly can theoretically result in diuresis and exacerbate fluid loss.[14] Vomiting and diarrhoea generally result in isotonic dehydration and eunatraemia. Simpler calculations than that presented in Table 1 can be utilized for maintenance requirements, e.g. 50–60 ml/kg/day or 2 ml/kg/h.[16] A crystalloid such as 0.9% saline is appropriate for most situations (especially resuscitation), but buffered isotonic solutions (e.g.

Lactated Ringer's/Hartmann's) are often preferred to address/prevent acidosis and hypokalaemia. The practicalities and controversies of hypertonic or colloid solutions are not discussed here. Regular reassessment of cardiovascular parameters, bodyweight and ideally haematocrit/total solids are essential, especially if significant fluid losses continue. Estimation of losses by all members of the care team is encouraged, as veterinary surgeons may not be present when faeces are passed (Figure 1). Dehydration assessment gives only an estimation. Overhydration can also occur, especially in dogs with hypoproteinaemia or systemic inflammation. AHDS dogs can rapidly improve, and fluid losses can therefore decrease sharply.

In less severe cases (e.g., those with mild dehydration and no electrolyte derangements), oral rehydration therapy may be effective and associated with lower treatment costs.[17] Close monitoring is crucial as these patients could rapidly deteriorate. Volume-responsive AKI can be seen in some hypovolaemic dogs. This will often resolve with appropriate fluid resuscitation within 24 hours, though a small number may develop persistent azotaemia due to intrinsic renal AKI secondary to renal hypotension.

Electrolyte derangements are common with vomiting and diarrhoea. Faeces have high potassium content so profound diarrhoea can cause hypokalaemia that requires intravenous supplementation. Hypokalaemia can exacerbate carbohydrate intolerance, anorexia, lethargy, and GI hypomotility.[14] GI signs and hypoperfusion can lead to metabolic acidosis, but this is typically corrected by appropriate fluid therapy without specific attention.

Supportive medications

Anti-emetic medications are often administered due to nausea/vomiting and to improve food intake. Commonly used medications are maropitant (1 mg/kg i.v. q24h), metoclopramide (1–2 mg/kg/day CRI) and ondansetron for refractory cases (0.5–1 mg/kg i.v. q12h). Omeprazole and other gastroprotectants are not indicated and, in fact, some can occasionally cause vomiting and diarrhoea. The gastric mucosa appears to be spared in AHDS, and the primary indication for proton pump inhibitors is gastric ulceration.[18,19] Analgesia can be required due to abdominal pain (uncommon in AHDS), presumably related to ileus, or potentially dermatitis. Opioids (e.g. methadone 0.1–0.2 mg/kg i.v. every 4–6 hours) and/or paracetamol (15 mg/kg i.v. q8h) are most frequently used. Although opioids can be associated with worsening of ileus in humans, this does not appear to be as prevalent in small animal patients.

FIGURE 1: Demonstration of difficulties of subjective estimation of fluid loss. Fluid volume left to right: 200 ml, 500 ml, 1000 ml.

Dysmotility

Ileus is a complication of AHDS that can prolong recovery. Reduced/altered motility in patients with acute GI disease is likely multifactorial in aetiology. Stress and pain may cause neurogenic inhibition of the enteric nervous system via sympathetic activation. Hospitalization itself appears to delay gastric emptying.[20] Inflammation and bacterial translocation can interfere with the local enteric nervous system. Opioids, hypokalaemia and acidosis may contribute further. Ileus can often be identified ultrasonographically or inferred from regurgitation/vomiting or abdominal pain. First-line therapy for ileus is usually metoclopramide given as CRI (1–2 mg/kg/day). For severe gastric ileus/atony, a nasogastric feeding tube can be used to drain gastric residual fluid prior to and between meals to reduce regurgitation. Loperamide has been described by some to limit diarrhoea, but this has the capacity to exacerbate ileus and is therefore not favoured by the author. Anti-spasmodics such as hycosine butylbormide (Buscopan®) may be more appropriate, but experience in dogs is limited. Reduction of stress and pain are crucial considerations, through ongoing assessment of analgesia and any measures to possibly improve mental health (e.g. reducing noise levels, owner visits). Cisapride, ranitidine or erythromycin are occasionally employed as prokinetics in refractory cases – caution should be exercised with the latter particularly, due to antimicrobial stewardship considerations and limited evidence for efficacy. Enteral nutrition is a key intervention to combat ileus and dysmotility. Fat ingestion retards gastric emptying, so low-fat diets should be beneficial.[21]

Nutrition

Theory and evidence support early enteral nutrition in dogs with severe enteritis to reduce dysmotility/ileus (in turn reducing pain), ensure nourishment of enterocytes, and address systemic caloric requirements.[22] For dogs not eating voluntarily, feeding tube placement should be considered in the first 24 hours of hospitalization. As prolonged anorexia is not anticipated in most AHDS patients, nasoenteric tubes are often adequate. There was no difference in complication rates between nasoesophageal or nasogastric positioning – the latter allows for suctioning of residual fluid.[23] When dogs do not tolerate tube placement, where large volumes are required, or where prolonged use is anticipated, an oesophagostomy tube may be indicated. However, this requires general anaesthesia and has financial implications too. The volume of food administered is typically small initially to assess tolerance, then steadily increased. A common protocol is to administer 33% of the resting energy requirements (RER) for the first 24 hours, divided over 4-6 meals, followed by 66% RER on day 2 and moving up to 100% RER on day 3 if well tolerated. For dogs that cannot tolerate higher volumes, every attempt should be made to administer at least some enteral nutrition. Sometimes this may require administration of very small volumes (e.g. 5–10% of RER per day) or very low-rate CRI. A highly digestible, high calorie diet is likely appropriate for most situations.

Glutamine is the main metabolite supporting normal enterocyte function and mucosal integrity and

is principally absorbed directly from the lumen. Stores are typically exhausted after 24–48 hours of anorexia.[24] Most recovery diets contain glutamine, but it can also be added to water (0.5 g/kg/d) or rehydration fluids. Glutamine supplementation improves canine GI motility.[25]

Antibiotics

Historically, antibiotics have been frequently utilized in AHDS, most frequently metronidazole and/or penicillins.[17,26,27] A decade ago, 63% of dogs with diarrhoea in the UK were prescribed antibiotics, increasing when diarrhoea was haemorrhagic (to over 80%!).[28] More recent evidence from Demark has shown a lower use of 31%.[5] These findings go against guidelines advising that most bacterial pathogens cause self-limiting diarrhoea, and use of antimicrobials could be more harmful than beneficial and induce resistance.[2,29,30] As AHDS signs often may be attributable to bacteria, the use of antibiotics can seem intuitively logical. Another oft-cited justification is the prevention of secondary bacterial translocation due to compromised mucosal integrity. However, as the causative agent is often unknown in AHDS, responsible antimicrobial stewardship would dictate use only when there is evidence of at least probable benefit. Many AHDS cases may be caused by viruses or clostridial toxins which will not be eliminated by antimicrobials. As well as stewardship concerns, antimicrobials may also have direct adverse effects e.g. immune-mediated disease, gastrointestinal irritation, neurological signs (with metronidazole) and dysbiosis (which can persist for months), though one study did not identify any short-term adverse effects.[31–37] Diarrhoea was reported in 56% of healthy dogs following metronidazole administration.[38] *C. perfringens* and *C. difficile* appear to be developing resistance to metronidazole, particularly concerning in the latter species given how debilitating human infections can be.[39–41]

Most importantly, numerous studies have confirmed that an overwhelming majority of dogs with AHDS have self-limiting disease within a few days. Prospective, placebo-controlled, blinded studies have shown no benefit to antibiotic use in AHDS regarding mortality, *C. perfringens* numbers, hospitalization duration or severity.[31,37,42] Other retrospective or non-randomized studies corroborate this.[5,37] Dogs with AHDS do not have higher incidence of bacteraemia than healthy dogs, and those that do have bacteraemia do not appear to be at increased mortality risk.[43]

Antibiotics are thus recommended only when sepsis is suspected and should be used promptly in those cases.[29] Sepsis should be suspected in any dog with hypotension or shock that is not responsive to fluid resuscitation, especially if at least two SIRS criteria are present (Table 2).[2] The SIRS criteria as a proxy for sepsis probably represents an overly simplistic approach that is not sensitive or specific.[44–46] Assessment of SIRS criteria should be performed after fluid resuscitation attempts, criteria improve rapidly and markedly following rehydration, and the number of truly septic patients is low.[5] CRP measurement did not correlate significantly with the need for antimicrobials.[6] Marked neutropenia (<0.5–0.75 x 10^9/l) is an indication for antibiotic use, due to the risk of sepsis especially with a compromised gastrointestinal barrier. Broad-spectrum antibiotics (e.g. amoxicillin-clavulanate intravenously 20 mg/kg q8h, or trimethoprim/sulphonamide) are generally appropriate.[2] Short courses of 3–5 days or until neutrophil count normalizes are likely adequate.[2] Metronidazole has frequently been employed due to perceived importance of anaerobic cover and/or immunomodulatory effects.[27] Evidence for the latter is sparse, and potentiated amoxicillin should have activity against anaerobes typically encountered in the gastrointestinal tract.[41] In AHDS, when metronidazole was added to amoxicillin-clavulanate, no benefit was noted.[47]

Clinical criteria	Result
Hypothermia	<37.5°C
Hyperthermia	>39.3°C for large dogs >39.4°C for small dogs
Tachycardia	>140 bpm
Tachypnoea	>40 bpm
Laboratory criteria	
Leukocytosis	>25 x10^9/l
Leukopenia	< 6 x10^9/l
Band neutrophilia	>3%
Hypoglycaemia	<3.9 mmol/l

TABLE 2: SIRS criteria for dogs. Reproduced/adapted from multiple sources.[5,37,43,46]

Microbiome manipulation

Efforts focussed on promoting a healthier GI microbiome without the use of antibiotics is preferred. In dogs with AHDS administered a probiotic (Vivomixx®, Gastropharm Ltd, St Albans), the return of normal, 'healthy' bacteria was hastened compared to a control group, with slightly faster clinical recovery.[48] The abundance of *C. perfringens*, specifically *cpe*- and *netF*- positive organisms, were reduced at an earlier timepoint. Recently published guidelines did not advocate in favour or against probiotic use in acute diarrhoea due to the lack of convincingly relevant benefits and the potential costs and stress of administration regardless of low risk.[2,42]

Faecal microbiome transplant (FMT) is an emerging treatment in veterinary medicine with the aim of introducing the microbiome from a healthy donor to a patient with suspected or proven dysbiosis (usually via the rectum). FMT use has been well-described for *C. difficile* infection in people with cure rates of 95–98%.[49,50] It follows logically to be considered in AHDS, where dysbiosis is often present/assumed alongside possible involvement of clostridial organisms. FMT has shown some promise in puppies with parvoviral enteritis, other canine acute diarrhoea, and a dog with *C. difficile*-related diarrhoea.[51–53] In a small AHDS pilot study, treated dogs had much improved bacterial diversity (closer to their donors) compared to control dogs at time of discharge, but not at 30 days, characterized by increased SCFA-producing bacteria likely beneficial for enteric health.[54] Despite this, there was no clinical benefit observed. Other studies found no benefit of FMT over probiotics,

supportive treatment or antibiotics.[37,55] However, these were all small studies, and treatment was administered to all dogs at the time of diagnosis. We know that most dogs recover quickly with supportive treatment, but there may be a sub-population that could benefit from FMT. Given the seemingly negligible risks, the author considers FMT in patients that are not showing significant improvement within 2–3 days, or those where diarrhoea persists for longer than 1–2 weeks.[56]

Practical management of diarrhoea

A potentially severe complication in canine diarrhoea is contact dermatitis induced by faecal scald (Figure 2). This most commonly affects the perianal region, but can involve the tail, scrotum and hindlimbs. Dermatitis can be severe enough to necessitate extended wound management/debridement, amputation, scrotal ablation or even play a role in euthanasia decisions. Regular assessment of the region, as well as barrier creams, tail bandages and gentle bathing +/- topical antimicrobials are likely helpful. A faecal foley catheter can reduce the risk by eliminating leakage and reduces nursing requirements (Figure 3). Faecal catheters should be re-positioned frequently (every 4 hours) as necrosis and stricture are potential complications, and if pain is noted at the time of placement continued attempts are discouraged. Rectal strictures can also form in AHDS patients secondary to persistent diarrhoea and straining.

Albumin

In prolonged cases, hypoalbuminaemia can develop, and adequate nutritional support is the most important step to address this. Hypoalbuminaemia can interfere with wound healing (specifically perineal), drug metabolism and fluid balance.[57,58] Albumin or fresh frozen plasma transfusion can potentially improve oncotic pressure, but are usually only mildly effective, and the risks/benefits (not discussed here) should be carefully considered.

Monitoring response

An 'AHDS activity index' (Table 3) was created to guide clinical assessment and track progress of patients, but likely is more useful for studies than clinically relevant individualized information.[59] Judging by current trends, it is likely to be a target for machine/AI learning soon, and this may identify patients with a more guarded prognosis or needing further treatment or monitoring.

Prognosis and outcomes

Dogs are generally discharged once eating, stools are improving, and they are no longer reliant on intravenous fluids to maintain hydration. Oral anti-emetics, probiotics and analgesia can continue at home. Average hospitalization time has been reported as 3–4 days, with marked clinical improvement usually in the first 1–2 days.[5,19,31,54,59,60] Presence of bacteraemia, CPE or *netF* did not affect outcome.[13,31,43,61] Soft stools may persist for several days into recovery, unsurprising given the severity of mucosal damage. Decreased faecal consistency is still present on day 5 in more than a third of dogs.[59]

Mortality

A survival rate of 93–100% has been reported, with most studies towards the higher end of this range.[5,6,17,19,37,43,48,59,62] True outcomes may be even better, which are biased towards referral populations which have failed to rapidly improve. Deaths due to severe AHDS have been reported, with evidence of mucosal necrosis.[63–65] The cause in such cases has been suggested as

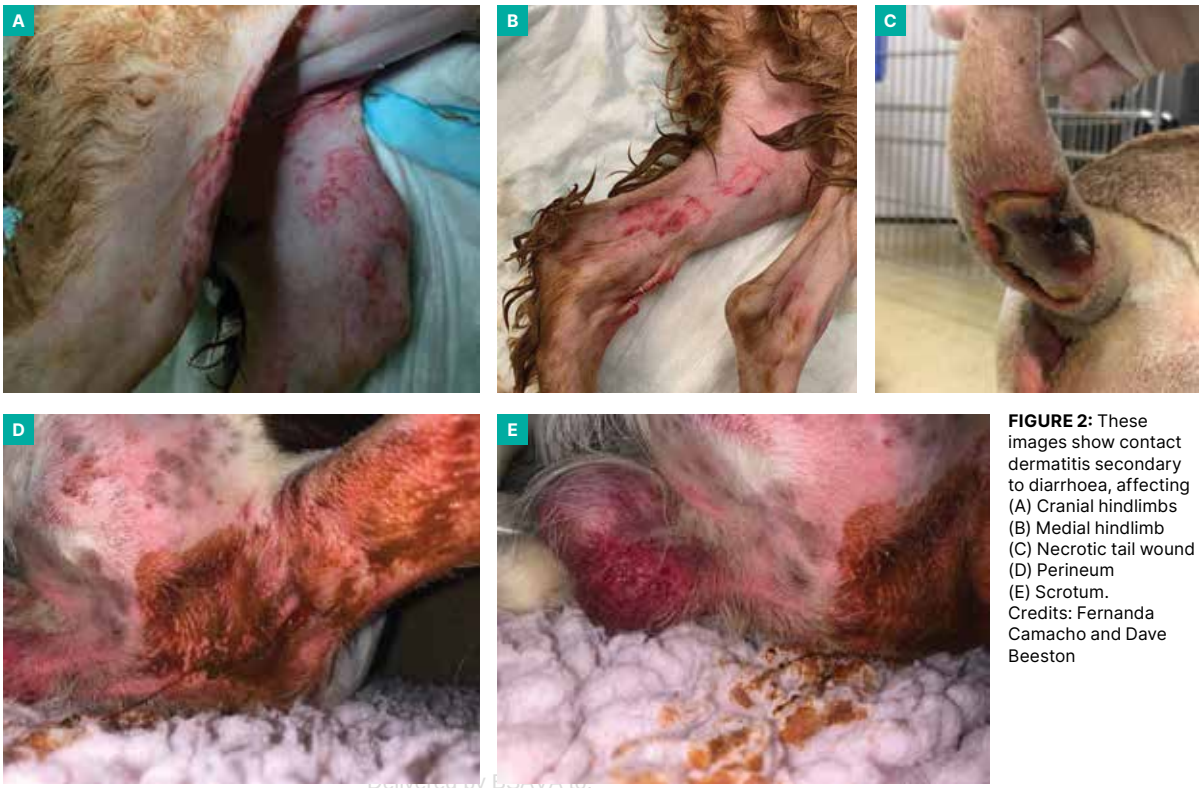

FIGURE 2: These images show contact dermatitis secondary to diarrhoea, affecting
(A) Cranial hindlimbs
(B) Medial hindlimb
(C) Necrotic tail wound
(D) Perineum
(E) Scrotum.
Credits: Fernanda Camacho and Dave Beeston

FIGURE 3: A dog with haemorrhagic diarrhoea and faecal foley catheter in place. Credit: Fernanda Camacho

endotoxaemia or sepsis, but euthanasia due to financial considerations also appears to account for some, suggesting the true survival rate with effective treatment may be better.[5,62] Older dogs may be more at risk of death, but one study reported the age of the patient as a specific reason for euthanasia, which may confound true interpretation.[5,59] In some retrospective studies, dogs receiving multiple antibiotics had lower survival, but this more likely simply reflects a higher disease severity prior to treatment than a causative effect.[5,66]

Long-term outcomes

Similarly to parvoviral enteritis, 28% of previously healthy dogs with AHDS go on to develop chronic GI disease, 2.57 times more likely than healthy controls.[67,68] Whether this represents long-term dysbiosis, mucosal injury leading to loss of tolerance, or whether acute disease is simply the first indicator of a chronic process is unknown. Antibiotic administration does not appear to affect this risk.[67]

About the author

Bryn graduated from the University of Nottingham in 2014 before spending some years in a small animal hospital in South-West Wales. After completing an internship at the RVC and a 3-year Small Animal Medicine residency in AMVS in Hampshire, Bryn settled at Willows at the start of 2023. Bryn has a keen interest in nephrology, hepatology and particularly gastroenterology.

Score	0	1	2	3
Activity	Normal	Mildly reduced	Moderately reduced	Severely reduced
Appetite	Normal	Mildly reduced	Moderately reduced	Severely reduced
Vomiting (times/day)	0	1	2–3	>3
Faecal consistency	Normal	Slightly soft	Very soft	Watery
Defecation (times/day)	1	2–3	4–5	>5
Dehydration (%)	0	<5	5–10	>10
Total score	0–3	4–5	6–8	9 or more
Clinical significance	Insignificant	Mild	Moderate	Severe

TABLE 3: Criteria for categorization of 'AHDS index'. Reproduced from Mortier *et al*. 2015[59]

Reflect on your reading

1. Which of the following tests is probably least useful in most patients with AHDS?
   a) PCV/total solids
   b) Electrolyte measurement
   c) Haematology
   d) Faecal culture
2. Which of the following gastrointestinal medications is probably not useful in AHDS?
   a) Maropitant
   b) Omeprazole
   c) Metoclopramide
   d) Ondansetron
3. Which of the following two scenarios are potential indications for antibiotic treatment in AHDS?
   a) Shock that is poorly responsive to fluid resuscitation
   b) Presence of haematochezia
   c) Suspicion of clostridial disease
   d) Severe neutropenia
4. True or false – it is not possible for dogs to be hypovolaemic without showing clinical signs of dehydration first (e.g. skin tenting, tacky membranes)?
5. Which of the following amino acids is the most essential to enterocyte nutrition?
   a) Lysine
   b) Glutamine
   c) Tryptophan
   d) Glycine

Answers available online in the BSAVA Library.

References and further reading are available at **www.bsavalibrary.com**.

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