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Veterinary TECHNICIAN

Canine Parvovirus

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A very sick 4-month-old puppy presents at your veterinary clinic. The owner reports acute onset of vomiting and bloody, foul-smelling diarrhea. When the owner mentions that the puppy has not been vaccinated, an "alarm sounds" in your mind. You hope that the puppy is not infected with parvovirus.

Canine parvovirus (CPV) is one of the most common infectious diseases in unvaccinated dogs younger than 6 months.[1] Without treatment of CPV infection, the mortality rate can be as high as 91%; with aggressive treatment, the mortality rate drops to 4% to 48%.[2] Understanding the pathophysiology of CPV infection is important for enabling veterinary technicians to provide prompt treatment and adequate patient care.

Etiology, Epidemiology, and Transmission

Most viruses create disease by causing degeneration of the cells in which they replicate.[3] Viruses contain genetic material in the form of RNA or DNA, which produces viral material such as enzymes and proteins.[3] During viral replication, virions accumulate, causing cells to swell.[3] The integrity of the cell membrane is disrupted, causing the cell to lyse or become necrotic, both of which result in the release of new virions into the lymph and blood.[3] The death of infected cells, in addition to inflammatory and other responses provoked by the virus, causes clinical signs.[3]

Parvoviruses (Parvoviridae) are small, single-stranded, non-enveloped, DNA viruses.[1,2] It is thought that CPV evolved from feline panleukopenia virus (FPV) or from a form of parvovirus that affected wildlife and caused hemorrhagic diarrhea.[2] There are two types of CPV: type 1 (CPV-1) and type 2 (CPV-2).[1,4,5] In the 1970s, CPV-1 was more common; currently, CPV-2 is the dominant strain in the United States and causes classic parvoviral enteritis.[6] This article focuses on CPV-2 because it is more common and pathogenic.

CPV-2 primarily affects puppies aged 6 weeks to 6 months.[1,2] Parvovirus is less likely to affect older dogs because of their immunity through natural infection or immunization.[2] Neonatal puppies are likely to be protected by the maternal antibodies for the first few weeks of life.[2] These antibodies are transferred to puppies through colostrum; the antibody titer of a puppy depends on the serum titer of the bitch at whelping, the size of the litter, and the amount of colostrum absorbed by the puppy.[2] The half-life of the maternal antibodies is approximately 10 days.[2] When a puppy is weaned and the antibody titer begins to decrease, immunization is essential for continued protection.[2] Depending on when a puppy is first vaccinated, the bitch's antibody titer may still be high enough to interfere with the immune response but too low for protection.[2] This leaves the puppy susceptible for a period of time because neither the bitch's antibodies nor the immune response to the CPV-2 vaccine is enough to prevent infection.[2]

CPV-2 is most commonly spread via the fecal–oral route, although it can also be spread through the fecal–nasal route.[1] After the virus is ingested, it replicates in the oropharynx, thymus, and mesenteric lymph nodes for 2 days.[1,2] The virus then moves into the blood, and marked viremia develops within 1 to 5 days after exposure.[1] The virus reaches the intestinal crypts via the blood.[1,6] Viral shedding begins on day 3 and continues for 7 to 10 days.[1]

Normal Physiology and the Effects of Infection

Parvovirus attacks rapidly dividing cells such as those in the lymphoid tissue, thymus, intestinal epithelium, bone marrow, and heart. Reviewing the normal physiology of the systems affected by CPV-2 infection is helpful for understanding how the infection affects patients.

Thymus, Bone Marrow, Lymph Nodes, Liver, and Spleen

The thymus is a central lymphoid tissue located between the lungs in the precardial mediastinal space.[7] However, in older animals, small fragments of active thymus can be found in bone marrow.[8] The thymus is well developed and very active during late prenatal and early postnatal life, making it susceptible to CPV-2. Lymphoid stem cells are produced and predestined to become B or T lymphocytes.[9] In young animals, predetermined T lymphocytes move via the blood to the thymus, where they multiply and become

Key Points

- Canine parvovirus is a significant viral pathogen that can affect dogs of any age but more commonly affects puppies.
- To fully understand the effects (pathology and pathophysiology), clinical signs, and treatment of canine parvovirus 2 (CPV-2), veterinary technicians need to understand the normal anatomy and physiology of the body systems that CPV-2 affects.
- Because of the high morbidity and mortality associated with CPV-2 infection and the brief opportunity for treatment, early recognition of infected dogs and immediate, aggressive treatment are critical.

Glossary

Antithrombin—inhibits clotting by binding to clotting factors and blocking their activity

Enterocytes—epithelial cells covering the intestinal villi and crypts

Granulopoiesis—granulocyte production

Hematopoietic stem cells—bone marrow cells capable of producing all types of blood cells

Hypovolemic shock—a decrease in cardiac output caused by an inadequate amount of fluid in the vascular system, which leads to insufficient tissue perfusion

Immunoglobulins—antibodies

Interstitial fluid—extracellular fluid that is outside the vascular system and bathes the cells

Intraosseous—within the bone

Intussusception—enfolding of one intestinal segment into another

Lymphopenia—a decrease in the number of lymphocytes in the blood

Oncotic pressure—the osmotic pressure due to plasma colloids that helps control movement across the capillary wall

Phlebitis—inflammation of a vein

Thrombocytopenia—a decrease in the number of platelets circulating in the blood

Thrombosis—formation of a clot inside a blood vessel

Virion—a virus particle composed of a nucleus, capsule, and DNA or RNA

mature T lymphocytes.[9] Once mature, T lymphocytes move to other peripheral lymphoid organs, such as the spleen and lymph nodes.[9] T lymphocytes have many functions that are collectively referred to as *cell-mediated immune responses*.[9]

When CPV-2 is ingested or inhaled, it moves into lymphoid tissues such as the mesenteric lymph nodes and the thymus.[1,2] CPV-2 replicates in these lymphoid tissues for 2 days before dissemination to the crypt cells of the small intestine.

Small and Large Intestines

The small intestine has a very large surface area that allows efficient absorption of nutrients.[10] The functional unit of the small intestine is the crypt–villus axis, which is composed of the intestinal crypts, a maturation zone, and the villi.[10] The intestinal crypts contain epithelial cells that rapidly divide and secrete most of the intestinal fluid.[10] As cells approach the maturation zone, they undergo final division and become mature enterocytes.[10] Once mature, enterocytes lose the ability to secrete fluid; instead, they develop a plasma membrane with a specialized region containing cylindrical protrusions called *microvilli*.[10] Enterocytes migrate up the villi to compose the intestinal epithelium.[10]

Marked viremia develops 1 to 5 days after exposure to CPV-2.[1] The virus travels through the blood to the intestinal germinal epithelium, where the virus replicates, impairing the ability of the enterocytes to mature.[1,2] Enterocytes normally migrate to the tips of the villi to replace sloughed enterocytes; however, as the virus replicates, enterocytes die, causing the villi to shorten.[1,2,11] As this happens, the villi lose the ability to secrete fluid and absorb nutrients.[2] In addition, the "barrier" function (nonspecific immunity) of the epithelium is compromised or destroyed, allowing (1) potential pathogens to enter the blood and (2) massive losses of blood, water, and electrolytes.

Fluid that enters the intestinal lumen comes from endogenous secretion within the gastrointestinal (GI) tract and from ingestion of food and liquids.[10] Most of the fluid is absorbed from the lumen, but a small amount is excreted into the feces.[10] A small decrease in absorption from the lumen can result in malabsorptive diarrhea.[10,12] When secretion into the lumen increases, secretory diarrhea develops.[12]

CPV-2 infection can cause abdominal pain, which may be elicited on palpation. This can be secondary to acute gastroenterocolitis, enteritis, or intussusception.[2] The cause of intussusception is not completely understood: it is thought that the damaged intestinal wall and the changes in motility are contributing factors.[10] As a wave of peristalsis travels down the intestine, the damaged segment of intestine does not move and is enveloped by the intestine proximal to it.[10] Intussusception can cause obstruction, signs of which include vomiting and diarrhea.[2] These may be inappropriately disregarded as deteriorating clinical signs due to the infection.[2]

Bone Marrow

Bone marrow is found in the medullary cavity of long bones and the interstices of spongy bone.[13] In young animals, all bone marrow is red; as animals age, most red marrow becomes adipose tissue called *yellow marrow*.[13] Red bone marrow contains hematopoietic stem cells, which can produce all types of blood cells.[14,15] Hematopoietic stem cells differentiate into rapidly dividing, committed stem cells called *progenitor cells*, which differentiate into specific blood cells.[15] CPV-2 attacks red bone marrow and progenitor cells, affecting erythrocyte, leukocyte, and platelet counts.

The Heart

Heart-wall thickness is mainly determined by the myocardium—the muscle layer between the endocardium and the epicardium.[16] Myocytes—the cells of the myocardium—rapidly divide in utero and during the first 2 weeks of life.[2]

Myocarditis due to CPV-2 infection affects puppies in utero or before 8 weeks of age.[1,17] Clinical signs of myocarditis due to CPV-2 infection can vary. Cardiac signs may be seen without previous cardiac disease, or the puppy may have acute diarrhea or may die without signs of cardiac disease.[1] Some puppies may have diarrhea and then die weeks or months later due to congenital heart failure.[1] On necropsy, examination of the heart shows enlargement, a pale myocardium, loss of myofiber, and lysis of myocytes.[17] Older dogs that survive neonatal infection may have scarring and thinning of the myocardium.[17] Currently, myocarditis due to CPV-2 infection is rare, although it may be seen in puppies of an unvaccinated bitch.[1]

Clinical Signs

The most common presenting clinical signs associated with CPV-2 infection include anorexia, vomiting, bloody diarrhea,

Box 1. Criteria[a] for SIRS in Dogs[19]

- Hypothermia (<100.4˚F; <38˚C) or hyperthermia (>104.0˚F; >40˚C)
- Leukopenia (>18,000 cells/mm3) or leukocytosis (<5000 cells/mm^3)
- Tachycardia (>120 bpm)
- Tachypnea (>20 breaths/min)

[a]The presence of two or more criteria is required for a diagnosis.

and lethargy. Clinical signs of CPV-2 infection can vary, depending on how long the dog has been infected and the severity of the infection.[5] The severity depends on the amount of viral inoculum received, the virulence of the strain, and the host's immune response.[5]

Clinical signs of CPV-2 infection do not present simultaneously.[1] When first infected, most dogs become lethargic, anorexic, and febrile without signs of GI involvement.[2] Vomiting and small bowel diarrhea develop in 24 to 48 hours.[2,4] Small bowel diarrhea is characterized by melena and a large amount of loose or watery stool.[18] As the disease progresses, the stool becomes bloody, which is a sign that the mucosal barrier has been compromised, resulting in massive losses of fluid (water), electrolytes (especially potassium), plasma proteins (albumin, globulins), and blood. Severe vomiting can lead to esophagitis.[4]

Nausea almost always precedes vomiting, and drooling is a fairly consistent sign of nausea early in the course of the disease. However, drooling may not be a presenting clinical sign, and owners may not notice drooling or know that it is associated with nausea.

Vomiting and diarrhea can cause severe dehydration and hypovolemic shock, which can decrease tissue perfusion.[2] The presence of vomiting and anorexia leads to malnourishment and dehydration. Signs of hypovolemic shock include pale mucous membranes with a delayed capillary refill time (>2 seconds), tachycardia, weak femoral or dorsal pedal pulses, hypotension, and cool extremities.[2]

Laboratory Findings

On a hemogram, leukopenia is the most common finding of CPV-2 infection.[2] If leukopenia is not seen on presentation, the patient often develops severe leukopenia due to viral leukocytolysis within the next 72 hours.[3,5] It is not uncommon for a patient's leukocyte count to be as low as 500 to 2000 cells/µL (normal: 5400 to 15,300 cells/µL).[10] Destruction of progenitor cells in bone marrow, combined with loss of neutrophils through the GI wall, leads to neutropenia.[5,10] Other findings on a hemogram may include anemia (due to blood loss through the GI tract) and thrombocytopenia (due to [1] the loss of progenitor cells in bone marrow and [2] increased platelet use in the GI tract).[5] In addition, the viral attack on the lymphoid tissue causes lymphopenia, which contributes to leukopenia, and platelet consumption leads to thrombocytopenia.

Abnormalities on the serum chemistry profile can include prerenal azotemia due to decreased tissue perfusion and dehydration, hypokalemia secondary to anorexia and losses from vomiting and diarrhea, hypoalbuminemia due to GI losses, and hypoglycemia due to anorexia and/or underlying sepsis.[2]

Damage to the GI tract allows bacteria to translocate and cause bacteremia.[2] The bacteria release endotoxin (lipopolysaccharide [LPS])[19] when they multiply, when their cell walls break down, and when they die.[20] LPS targets various cells, including mononuclear cells, neutrophils, vascular endothelial cells, and platelets.[20] Of these cells, mononuclear phagocytes are key to the inflammatory response.[20] Binding of LPS to mononuclear phagocytes causes the release of cytokines, including tumor necrosis factor, interleukins, and platelet-activating factor,[20,21] all of which have important roles in sepsis and in systemic inflammatory response syndrome (SIRS; **BOX 1**). The release of endotoxins and the ensuing chain reaction cause SIRS. Sepsis is a systemic response to infection, whereas SIRS is the clinical manifestation of this response.[19]

The "attack" on the lymphoid tissue results in immunosuppression. The lymphopenia and neutropenia associated with bacteremia and sepsis can result in overwhelming septic shock and in death.

Diagnosis

Although clinical signs including fever, nausea, abdominal pain, vomiting, and bloody diarrhea in a young, unvaccinated dog are often highly suggestive of CPV-2, testing should be conducted to confirm the infection.[1] In-hospital testing can be performed using a fecal antigen ELISA,[1,2,4,5] which is specific and somewhat sensitive for CPV-2.[1] Viral shedding in the feces usually begins on day 3 after infection and continues until day 10, with peak shedding occurring on days 4 to 7.[2] The virus is rarely evident in the feces after day 12.[1] Five to 12 days after a modified live CPV-2 vaccine is given, shedding of the vaccine virus in the feces may produce a false-positive result on an ELISA.[2] The vaccination usually produces a weak positive result.[1] A false-negative result may be obtained if testing is performed after viral shedding has stopped or if serum neutralizing antibodies bind with antigen in diarrhea.[1]

Treatment

No specific antiviral agents are approved for treating CPV-2 infection. Therefore, treatment of CPV-2 infection mainly consists of supportive care.[1] The restoration of fluid volume and electrolyte balance is very important, especially in puppies that have had severe vomiting and diarrhea and that present in hypovolemic shock.[2]

On presentation, the largest possible intravenous (IV) catheter should be placed.[2] If the patient has circulatory collapse or is too young for placement of an IV catheter, an intraosseous catheter can be placed[2] (**BOX 2**). IV fluids should be started immediately; if the patient is in hypovolemic shock, fluid deficits may be replaced in the first 1 to 2 hours to help prevent further deterioration of the patient's condition as well as death.[2] The fluids can be given as "quickly" as 90 mL/kg/h; however, the patient's degree of dehydration and physiologic end points should be considered when choosing a fluid rate.[2] Although quick replacement of the fluid

Box 2. Placing an Intraosseous Catheter[a]

Materials

- Lidocaine (1%) for local anesthesia
- Scalpel blade
- 18- to 22-gauge spinal needle (for small dogs); 18- to 25-gauge hypodermic needle (for neonates)
- 12-mL syringe
- 3 mL of heparinized saline
- Antiseptic ointment

Procedure

Common sites for placement include the wing of the ilium, the intertrochanteric fossa of the femur, the tibial tuberosity, the medial surface of the proximal tibia, and the greater tubercule of the humerus.

1. Prepare the area as you would for any surgery by clipping and scrubbing the site.
2. Inject lidocaine into the skin for local anesthesia.
3. Using the scalpel blade, make a stab incision down to the periosteum.
4. Pass the needle through the incision and into the cortex of the bone, seating the needle by twisting it in 30˚ turns and applying light pressure. When the needle has entered bone marrow, resistance will suddenly decrease. If possible, advance the needle to the hub.
5. Needle placement can be confirmed by moving the limb. If placed correctly, the needle will move with the limb without trembling. If the needle trembles, it is outside the bone. Needle placement can also be confirmed by using the 12-mL syringe for aspiration. If bone marrow is aspirated, the placement is correct.
6. Flush the needle with heparinized saline. You should not feel resistance; if you do, turn the needle to move the bevel away from the inner cortex.
7. Place a tape strip where the needle enters the skin, and then suture the tape in place by placing the suture though the tape and skin.
8. Place antiseptic ointment over the insertion site, and then place a bandage over the needle to prevent contamination as well as movement of the joint.

- Contraindications for placement include diseases of the bone, infection at the site, and sepsis.
- Complications include infection, extravasation of fluids into the surrounding tissue, and bone fractures.
- Blood products, saline, plasma, and colloids can be safely administered through the catheter.
- Medication dosages do not need to be changed: they are the same as if they were given through an IV catheter.
- An intraosseous catheter can be in place for up to 72 hours if the site is kept clean.

[a]Macintire DK, Drobatz KJ, Haskins SC, Saxon WD. Nutritional support of critical patients. In: *Manual of Small Animal Emergency and Critical Care Medicine.* Philadelphia: Williams & Wilkins; 2005:91-94.

deficit is not ideal, it is important to weigh the benefits with the risks. It is also important to remember that rapid infusion expands the intravascular space for only a short period.[11] Within 1 hour, 75% to 85% of administered crystalloids move from the vasculature into the interstitial fluid compartment.[11] The patient's temperature should be monitored closely during administration of a fluid bolus. A bolus of crystalloids at room temperature can cause the rectal temperature to drop 0.5˚C (0.9˚F).[22]

Vomiting and diarrhea can cause a large loss of GI protein.[11] This can lead to a decrease in plasma colloid oncotic pressure (COP), and fluid resuscitation reduces the COP even more.[11] COP is important for holding fluid in the vasculature. When the COP decreases, fluid leaks into the interstitial space, causing edema.

When perfusion improves, the fluid rate can be decreased to a maintenance rate and, if necessary, a colloid (e.g., synthetic colloid, fresh frozen plasma [FFP], whole blood) can be added to the fluid regimen.[2] Administration of a colloid should be considered if the albumin level decreases to <2 g/dL (normal: 2.6 to 4.0 g/dL) or the total protein level decreases to <4 g/dL (normal: 5.8 to 7.2 g/dL).[2]

By remaining in the vasculature and attracting water and sodium from the interstitial space, synthetic colloids provide volume expansion and oncotic pressure.[23] Hydroxyethyl starch (hetastarch) has a plasma half-life of 25.5 hours and can achieve a volume expansion that lasts 12 to 48 hours, depending on the dose.[23] A bolus of 5 mL/kg can be given, followed by a maintenance rate of 20 mL/kg/d.[23]

FFP can also be a beneficial treatment.[2,11] FFP can provide amino acids, albumin, and anticoagulants in the form of protein C and antithrombin III.[11] FFP also provides immunoglobulins, which may help neutralize circulating virus, and serum protease inhibitors, which can decrease the systemic inflammatory response that may accompany CPV-2 infection.[2] Although FFP provides albumin, 22.5 mL/kg of FFP is required to raise the serum albumin by 0.5 mg/dL.[2] Synthetic colloids are preferred to FFP because the former provide more vascular support, are less expensive, and are easier to store.[12] However, combination fluid therapy is the most effective: a crystalloid can provide volume expansion, FFP can provide immunoglobulins and serum protease inhibitors, and a synthetic colloid can reduce the total crystalloid volume required and provide vital COP.[23]

Vomiting and diarrhea can cause a patient to lose not only fluids and proteins but also electrolytes. Hypokalemia (secondary to vomiting and diarrhea) is a common complication in puppies with CPV-2 infection.[2] Hypokalemia can cause muscle weakness

Table 1. Drugs for Treating CPV-2 Infection[a]

Drug Class	Drug	Pharmacology	Dosage
Gastrointestinal protectants	Sucralfate	Reacts with hydrochloric acid to form a paste that binds to ulcers and protects from acid, bile, and pepsin	• Small dogs: 0.25 g PO tid • Medium dogs: 0.5 g PO tid • Large dogs: 1 g PO tid
	Ranitidine	Inhibits histamine at the H_2 receptors of parietal cells, reducing gastric acid output and treating or preventing gastric ulceration	0.5–2.0 mg/kg PO, IV, or IM bid–tid
	Cimetidine	Same as ranitidine	5–10 mg/kg PO, IV, or IM tid–qid
	Famotidine	Same as ranitidine	0.5 mg/kg PO, IV, or IM sid–bid when used with other gastrointestinal protectants
Antibiotics	Metronidazole	Bactericidal, but exact mechanism of action is not completely understood; it is thought to disrupt DNA and nucleic acid synthesis	15 mg/kg IV bid
	Ampicillin	Bactericidal aminopenicillin	22 mg/kg IV tid
	Cefazolin	First-generation cephalosporin; inhibits mucopeptide synthesis in the cell wall, causing the wall to become an ineffective barrier	22 mg/kg IV tid
	Enrofloxacin	Bactericidal fluoroquinolone that inhibits bacterial DNA	5–20 mg/kg IV, IM, or PO sid or divided bid
Antiemetics	Metoclopramide	Stimulates motility of the upper GI tract; increases lower esophageal sphincter pressure to prevent or reduce gastroesophageal reflux; antagonizes dopamine at receptor sites in the central nervous system to act as an antiemetic	0.2–0.4 mg/kg PO, IM, or SC qid; 1–2 mg/kg CRI sid
	Chlorpromazine	Blocks the chemoreceptor trigger zone and the vomiting center in the brain	0.5 mg/kg IM or SC tid–qid
	Ondansetron	5-HT3 receptor antagonist	0.11–0.176 mg/kg IV bid–tid

[a]Plumb DC. *Veterinary Drug Handbook*. 4th ed. White Bear Lake, Minnesota: PharmVet Publishing; 2002.

and paralysis, ileus, cardiac arrhythmias, and polyuria.[2] Potassium chloride can be added to fluid therapy to treat or prevent hypokalemia.[2] Potassium chloride should not be administered at a rate >0.5 mEq/kg/h because higher amounts can cause cardiac arrhythmias, resulting in death.[2]

Vomiting and diarrhea can also cause hypoglycemia,[2] which should be prevented, especially in young puppies, by adding 2.5% to 5% dextrose to fluid therapy.[2]

CPV-2 infection disrupts the intestinal mucosal barrier, possibly allowing translocation of bacteria and leading to endotoxemia and sepsis.[2] Therefore, administration of a broad-spectrum antibiotic should be considered.[2] Excellent antibiotic coverage of gram-negative and anaerobic bacteria can be provided by using a β-lactam antibiotic in combination with an aminoglycoside.[2] Because aminoglycosides can be nephrotoxic, they should be used with caution.[6] Cartilage abnormalities in young, growing dogs have been linked to administration of enrofloxacin.[2]

It is difficult to keep a patient hydrated and maintain the electrolyte balance if the patient continues to vomit; therefore, the use of antiemetics is valuable for treating CPV-2 infection.[6] Phenothiazine derivative antiemetics, such as chlorpromazine, work by blocking the chemoreceptor trigger zone and the vomiting center in the brain.[2] Chlorpromazine causes arteriolar vasodilation and can cause hypotension, so it should not be used in severely dehydrated patients.[2]

Dopaminergic antagonist antiemetics, such as metoclopramide, work by blocking the chemoreceptor trigger zone, increasing pressure in the lower esophageal sphincter, and coordinating motility of the upper intestinal tract.[2] If chlorpromazine and metoclopramide are ineffective, ondansetron HCl may be added because it works as a 5-HT_3 receptor antagonist and is usually effective against intractable vomiting.[2] Maropitant citrate can also be used.

It is equally important to prevent vomiting to allow the patient to rest. Continuous vomiting and diarrhea are very tiring, so the ability to sleep can improve patient recovery.

Fluid Therapy

Fluid therapy is administered by a veterinarian and consists of maintenance, correction of dehydration, and replacement of ongoing

losses. Maintenance fluids should be administered at a rate of 66 mL/kg/d. The patient's percentage of dehydration should be estimated by the veterinarian and corrected over the next 24 hours by adding the deficit to the maintenance fluid rate. Replacement of ongoing fluid losses is the technician's responsibility. Until vomiting and diarrhea have been resolved, this fluid loss can be replaced by doubling the estimated amount of vomit and/or diarrhea to compensate for underestimation. An alternative to estimation is to place an absorbent pad or towel in the patient's cage and weigh the vomit and/or diarrhea. Fluid losses can be replaced by administering a second bag of the chosen crystalloid at the same time as the maintenance fluids. The replacement fluids should be administered at a rate that does not cause the patient to become fluid overloaded. Clinical signs of fluid overload include nasal or ocular discharge, an increase in respiratory rate or effort, an increase in lung sounds on auscultation, peripheral edema, and weight gain (beyond what is expected after dehydration is corrected). The replacement fluid rate can be calculated by recording fluid losses for 4 hours and adding them together; the total amount should be replaced over the next 4 hours. Fluid losses should be calculated every 4 hours and the replacement rate adjusted accordingly.

Because of the associated damage to the GI tract, administration of GI protectants (e.g., sucralfate, ranitidine, cimetidine, famotidine) is important in treatment. Because sucralfate can only be given orally, the clinician may choose to avoid this drug until the patient is no longer vomiting. The mechanisms of action of these drugs are listed in **TABLE 1**.

Nutritional Support

It is important to remember that the addition of dextrose to IV fluids does not qualify as nutritional support, which must be provided[2] (**BOX 3**). It is common to withhold food and water until the patient is not vomiting.[1] However, it has been shown that enteral feeding may help decrease the risk of bacterial translocation by helping to maintain the integrity of the GI wall.[2] This is achieved by improving blood flow, decreasing ulceration, and increasing mucus production.[24] Puppies fed via a nasoesophageal tube (**BOX 4**) from the first day of hospitalization had a shorter recovery time and maintained body weight compared with puppies that were not fed until they had no clinical signs for 12 hours.[1] Enteral feeding may also be accomplished by syringe or nasogastric tube[2] (**BOX 4**). A nasogastric tube also allows evaluation of gastric motility through measurement of the residual gastric volume.[2] Diets provided via a nasoesophageal or nasogastric tube can be administered as boluses or a constant-rate infusion.[2] Before each bolus feeding, the feeding tube should be aspirated: if the residual volume is 50% or more of the last amount fed, gastric emptying is delayed and feeding should be withheld for a short period.[2] When feeding is provided via constant rate infusion, the feeding tube should be aspirated every 6 hours: if the residual volume is twice the amount that was fed in the past hour, gastroparesis exists, and medications to increase motility may be indicated.[2]

The feeding tube must be maintained. Clogging of the tube can be prevented by never allowing the diet to sit in the tube and by flushing the tube with warm water four times a day or after every bolus feeding.[25] It may be necessary to place an Elizabethan collar on the patient to prevent removal of the tube.[25] To monitor movement of the feeding tube, it can be marked with a pen where the tube enters the naris.[25]

If a patient does not tolerate enteral feeding, partial parenteral nutrition may be used.[2] This requires placement and aseptic handling of a dedicated central catheter.[2] This IV catheter cannot be used for anything else, and the insertion area must be checked for phlebitis daily.

Box 3. Calculating Feeding Requirements[a]

- Resting energy requirement (RER) of dogs: (30 × body weight [kg]) + 70
- RER of dogs <2 kg: 70 kcal × body weight ($kg^{0.75}$)
- It is not necessary to multiply the RER by an illness factor to prevent overfeeding.
- Start feeding 25% of the requirement. If the patient tolerates the feedings, the clinician may choose to increase the feedings by 25% of the requirement every 12 to 24 hours.

[a]Macintire DK, Drobatz KJ, Haskins SC, Saxon WD. Nutritional support of critical patients. In: *Manual of Small Animal Emergency and Critical Care Medicine.* Philadelphia: Williams & Wilkins; 2005:91-94.

Inpatient Versus Outpatient Care

While inpatient care is best, it can be cost prohibitive; therefore, outpatient care may be the only option for some owners. Owners can withhold food for 12 to 24 hours to allow their pet's GI tract to recover; if the dog is vomiting, water may also be withheld.[2] It is extremely important to ensure that outpatients receive sufficient subcutaneous (SC) fluids to keep them well hydrated. However, if the patient is severely dehydrated, SC fluids will be ineffective.[2] This is due to peripheral vasoconstriction associated with severe dehydration, which may cause poor distribution of SC fluids, preventing restoration of the circulating volume.[1] SC fluids can cause SC infection and sloughing of the skin at the injection site.[2] Food and water can be slowly reintroduced when there has been no vomiting for 24 hours. However, if the vomiting persists or begins again once food and water is reintroduced, the patient should be hospitalized. Hospitalization should also be recommended if the patient does not eat or drink, if diarrhea worsens, or if the patient's attitude declines at all. The owner should be encouraged to call the clinic with any concerns.

Monitoring

CPV-2 infection can be painful because it damages the GI wall. Therefore, it is important to recognize the signs of pain early and initiate pain medication. It is easier to prevent pain than to relieve and control it. The latter can require a higher dose of analgesia compared with a preemptive or proactive approach.[26] Signs of pain include tachycardia, tachypnea, vocalization, restlessness, a hunched posture, trembling, and crying when handled.[26] If the veterinary technician is concerned that a patient is in pain, a clinician

Box 4. Placing a Nasogastric or Nasoesophageal Tube[a]

Materials

- Local anesthetic (0.5%–1%)
- 3.5- to 8-French feeding tube
- Lidocaine gel
- Tissue glue
- Tape
- Suture and needle holders
- Skin staples

Nasogastric Tube Placement

- Place a small amount of local anesthetic in the naris. Hold the head up for a few seconds so the anesthetic can move into the naris.
- Measure the feeding tube from the nasal meatus to the last rib (where the stomach is located).
- Mark the feeding tube at the nasal meatus. This marks the length of tube that you will insert.
- Most patients tolerate placement well without sedation. It is important to brace your hand on the patient's head so that your hand moves with the patient, preventing inadvertent removal of the tube.
- Insert the tube into the naris using short, intermittent advances, and direct it ventromedially. After each advance, release the tube and allow the patient to move and adjust to the tube.
- You may feel resistance when the tube reaches the midnasal region; you might need to use firm pressure to pass the tube. If you cannot advance the tube, pull out a short length of it and try again. If the tube will not pass with another attempt, the tube may be too large for the patient and will need to be removed and replaced with a smaller tube.
- As you advance the tube into the esophagus, the patient will swallow. If it begins to cough or becomes dyspneic, the tube is probably in the trachea, so you should remove the tube and start over.
- When you have advanced the tube to the premeasured length, stop and aspirate the tube with a syringe.
- If the tube is in the stomach, you may aspirate gastric juices.
- If the tube is in the esophagus, you may feel negative pressure when you pull the syringe plunger. This is due to the esophagus collapsing onto the tube.
- If the tube is in the trachea or coiled in the pharynx, you may aspirate air.
- To confirm placement of the tube, inject 3–6 mL of sterile water through the tube. If the patient coughs, the tube is in the trachea and should be removed.
- It is important to know that lack of a cough does not mean that the tube is not in the trachea. Severely ill patients may not cough with this stimulus.
- Always confirm placement of the tube with a lateral radiograph.
- When you are satisfied with the placement of the tube, place a small amount of tissue glue in the alar notch and position the tube so it goes along the side of the head or between the eyes. The tube should not obstruct the patient's visual field. You can fasten the tube with skin staples or place a tape tag around the tube and suture it in place.
- An Elizabethan collar must be placed on the patient to prevent removal of the tube.

Nasoesophageal Tube Placement

- A nasoesophageal feeding tube can be placed the same way, with one exception: when measuring for correct placement, measure to the last rib and then subtract one-third of the length. This should place the tube in the esophagus instead of the stomach.
- Placement of the tube in the esophagus is sometimes preferred to placement in the stomach because the latter can prevent closure of the pyloric sphincter, causing the patient to vomit, especially if it is already nauseated.
- When placing an esophageal tube, ensure that the tip of the tube does not sit directly over the heart because this can cause irritation. Always confirm placement of the tube with a lateral radiograph.

[a]Macintire DK, Drobatz KJ, Haskins SC, Saxon WD. Nutritional support of critical patients. In: *Manual of Small Animal Emergency and Critical Care Medicine.* Philadelphia: Williams & Wilkins; 2005:91-94.

should be notified. As the main caregiver, the veterinary technician is most likely to notice changes in a patient's attitude or condition.

The patient's IV catheter should be evaluated every day, with special attention to the insertion site. The veterinary technician should look for signs of thrombosis, infection, or phlebitis.[26] If these occur, the catheter should be removed and a new one placed in a different vein. Even if the insertion site looks normal, the catheter should be replaced every 72 hours.[26] If the bandage becomes wet or soiled, it should be changed as soon as possible.

It is important for the veterinary technician to monitor the patient's attitude. Changes such as increased lethargy or obtundation should be brought to a clinician's attention immediately. Signs of nausea, including drooling, lip licking, frequent swallowing, and "smiling," should be watched for closely.

Changes in the vital signs should also be watched for closely. The vital signs should be checked at least every 6 hours but more frequently if there is concern that the patient is declining. The following should be recorded: respiratory rate and effort, heart rate, change in mucous membrane color (pale, cyanotic, hyperemic), capillary refill time, and temperature.

As the primary caregiver, you are the patient's advocate. If you have concerns, be sure to take them to a clinician. Just because the vital signs are scheduled to be checked every 6 hours does not mean that you have to wait to check the patient's temperature or heart rate if you notice a change in the patient's attitude.

Most puppies are very social and benefit from some kind words and a few minutes of attention that are unassociated with examination, treatment, and monitoring.[26] An effort should be made to consolidate monitoring tasks and treatments so the patient has time to rest in between, which is important for recovery.[26] However, giving the patient time to rest is difficult when diarrhea and vomiting are severe. If the patient vomits, defecates, or urinates on one side of the cage and then lies down on the other side, it may be possible to remove the soiled bedding without disturbing the patient.

It is very important to keep the puppy clean. Daily bathing may be required to keep the patient free of urine and diarrhea and to avoid urine scalding, which can break down the skin.[26] If the patient urinates or defecates on itself, it should be washed with a mild soap, thoroughly rinsed, and dried.[26] The cage should be kept as clean as possible. This can be labor intensive if the patient has frequent vomiting and diarrhea. However, the patient's well-being is just as important as the medical treatments.[26] The patient will feel better if it is clean and dry.

Infection Control and Isolation

Another important role of the veterinary technician is to isolate and control the infection. Ideally, the patient should be placed in an isolation ward. If this is not available, a cage that is as far away as possible from other patients can be used. An isolation perimeter around the cage can be designated with tape on the floor. One veterinary technician should be assigned to the patient's care to decrease the risk of contaminating the hospital. This person should be sure not to handle other puppies or immunocompromised patients. If this cannot be avoided, the caregiver should take extra precautions, such as thorough hand washing with soap and water as well as gloving.

There should be a separate area for trash and laundry associated with the affected patient. The designated caregiver should dispose of the trash and wash the laundry at the end of every shift. All supplies used during the shift should be replenished so that the next caregiver does not have to replenish stock during treatments. The laundry should be washed with household bleach (sodium hypochlorite). In addition, food and water bowls should be cleaned separately and with bleach or another effective disinfectant of CPV-2 (i.e., Parvosol II RTU Disinfectant [Agi-Labs], Kennel Kare SC [Health Technology Professional Products]).

When the patient has been discharged, the isolation area should be cleaned. Like other Parvoviridae, CPV-2 is resistant to the environment and very hard to inactivate.[1] CPV-2 can live for 5 months or more in the environment and on inanimate objects.[1] Household bleach should be diluted to one part per 30 parts water.[1,6] For bleach to be effective, the virus must be exposed to it for at least 10 minutes.[1]

Outcome

The mortality rate associated with CPV-2 infection can be high. This is partly due to the ability of the virus to damage the GI tract and initiate a systemic inflammatory response.[2] Early initiation of treatment improves the chance of survival.[2] Patients that survive the first 4 days that they show clinical signs usually recover swiftly, whereas complications such as sepsis or intussusception can lengthen the time in the hospital.[2]

Prevention

The most effective prevention of CPV-2 infection is immunization. The age at which immunization is most effective depends on several factors, including the antibody titer of the bitch and the immunogenicity and antigen titer of the vaccine.[1] Interference by maternal antibodies is the main reason for vaccine failure.[1] Puppies from a bitch with a low antibody titer can be effectively immunized at 6 weeks of age, whereas vaccination of puppies from a bitch with a high titer should be delayed because the maternal antibodies may persist.[1] If the immune status of the puppies is unknown, they can be vaccinated with a high-titer, attenuated, live CPV-2 vaccine at 6, 9, and 12 weeks of age, followed by annual revaccination.[1] Antibody titers can be checked before revaccination.[2] The titer level verifies immunologic response to infection but does not guarantee protection against infection.[2] However, measuring the titer can be useful because overvaccination can increase the risk of immune-mediated disease.[2]

Client Education

Client education is a crucial part of preventing CPV-2 infection. Clients should be given a recommended vaccination schedule for their puppies. Clients should also be told about the period when the maternal antibody level is no longer high enough to provide protection but the vaccine has not started to provide immunity.[5] The half-life of the maternal antibodies is approximately 10 days.[2] When the puppy is weaned, the antibody titer begins to decline.[2] When the puppy is given its first vaccination, neither the vaccine nor the bitch's antibodies can provide protection.[2] The puppy is at risk until it receives its third CPV-2 vaccination. Until the puppy is protected, it is important for the client to take preventive measures, including staying away from places with many dogs, such as canine parks and daycare centers.

If a new puppy is being introduced into a household where CPV-2 has recently infected other dogs, it may be best to keep the puppy somewhere else until it has completed its immunizations.[4] Puppies that recover from CPV-2 infection have natural immunity for at least 20 months and possibly for life.[1]

References

1. Mccaw DL, Hoskins JD. Canine viral enteritis. In: Greene CE, ed. *Infectious Diseases of the Dog and Cat.* 3rd ed. St Louis: Elsevier; 2006:63-71.
2. Prittie J. Canine parvoviral enteritis: a review of diagnosis, management, and prevention. *J Vet Emerg Crit Care* 2004:14(3):167-176.
3. Cheville NF. Cytopathology of viral diseases. *Ultrastructural Pathology: An Introduction to Interpretation.* Ames: Iowa State University Press; 1994:491-615.
4. Willard MD. Disorders of the intestinal tract. In: Nelson RW, Couto CG, eds. *Small Animal Internal Medicine.* 3rd ed. St Louis: Mosby; 2005:433-435.
5. Rewerts JM, Cohn LA. CVT update: diagnosis and treatment of parvovirus. In: Bonagura JD, ed. *Kirk's Current Veterinary Therapy XIII: Small Animal Practice.* Philadelphia: WB Saunders; 2000:629-632.
6. Wingfield WE, Macy DW. Canine parvovirus. In: Wingfield WE, ed. *Veterinary Emergency*

Medicine Secrets. 2nd ed. Philadelphia: Hanley & Belfus; 2001:353-357.
7. Venzke WG. Thymus. In: Getty R, ed. *Sisson and Grossman's The Anatomy of the Domestic Animals.* Vol 2. 5th ed. Philadelphia: WB Saunders; 1975:1670.
8. Venzke WG. Thymus. In: Getty R, ed. *Sisson and Grossman's The Anatomy of the Domestic Animals.* Vol 1. 5th ed. Philadelphia: WB Saunders; 1975:181.
9. Latimer KS. Leukocytes in health and disease. In: Ettinger SJ, Feldman EC, eds. *Textbook of Veterinary Internal Medicine.* Vol 2. 4th ed. Philadelphia: WB Saunders; 1995:1916-1919.
10. Burrows CF, Batt RM, Sherding RG. Diseases of the small intestine. In: Ettinger SJ, Feldman EC, eds. *Textbook of Veterinary Internal Medicine.* 4th ed. Philadelphia: WB Saunders; 1995;2:1169-1233.
11. Hackett TB. Hemorrhagic diarrhea. In: Wingfield WE, Raffe MR, eds. *The Veterinary ICU Book.* Jackson, Wyoming: Teton; 2002:763-770.
12. Cunningham JG. Movements of the gastrointestinal tract. In: *Textbook of Veterinary Physiology.* 3rd ed. Philadelphia: WB Saunders; 2002:238-239.
13. Getty R. General osteology. In: Getty R, ed. *Sisson and Grossman's The Anatomy of the Domestic Animals.* Vol 1. 5th ed. Philadelphia: WB Saunders; 1975:21.
14. Weiser MG. Erythrocyte responses and disorders. In: Ettinger SJ, Feldman EC, eds. *Textbook of Veterinary Internal Medicine.* Vol 2. 4th ed. Philadelphia: WB Saunders; 1995:1897.
15. Ganong WF. Circulating body fluids. In: *Review of Medical Physiology.* 22nd ed. New York: McGraw-Hill; 2005:515-516.
16. Pasquini C, Spurgeon T, Pasquini S. *Anatomy of Domestic Animals.* 9th ed. Pilot Point, Texas: Sudz Publishing; 1997:383.
17. Kittleson MD, Kienle RD. Primary myocardial disease leading to chronic myocardial failure (dilated cardiomyopathy and related diseases). In: *Small Animal Cardiovascular Medicine.* St. Louis: Elsevier; 1998:338-339.
18. Jergens AE. Diarrhea. In: Ettinger SJ, Feldman EC, eds. *Textbook of Veterinary Internal Medicine.* Vol 1. 4th ed. Philadelphia: WB Saunders; 1995:113.
19. Otto CM. Sepsis. In: Wingfield WE, Raffe MR, eds. *The Veterinary ICU Book.* Jackson, Wyoming: Teton; 2002:695-709.
20. Kirby R. Pathophysiology of systemic inflammatory response syndrome: septic shock. *Proc IVECCS* 2005:725-730.
21. Morresey PR. Synthesis of proinflammatory mediators in endotoxemia. *Compend Contin Educ Pract Vet* 2001:23(9):829-852.
22. Gostelow RF. The effect of room temperature crystalloid fluid boluses on the rectal temperature of hypovolemic small animal emergency patients. *Proc IVECCS* 2006. Accessed April 2011 at www.vin.com.
23. Day TK. Shock syndrome in veterinary medicine: pathophysiology, clinical recognition, and treatment. In: DiBartola SP, ed. *Fluid Therapy in Small Animal Practice.* 2nd ed. Philadelphia: WB Saunders; 2000:436-438.
24. Powell LL. ID and treatment of bacterial translocation: literature review & evidence-based strategies. *Proc IVECCS* 2006:349-353.
25. Michel KE. Monitoring the enterally fed patient to maximize benefits and minimize complications. *Proc IVECCS* 2006:495-498.
26. Davis H. Nursing care of the critically ill patient. *Proc ACVC* 2001. Accessed April 2011 at www.vin.com/VINDBPub/SearchPB/Proceedings/PR05000/PR00461.htm.

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1. CPV-2 infection can affect the
- **a.** bone marrow.
- **b.** GI tract.
- **c.** heart.
- **d.** all of the above

2. An effective disinfectant of CPV-2 is
- **a.** chlorhexidine.
- **b.** diluted bleach.
- **c.** alcohol.
- **d.** formaldehyde.

3. The most common abnormality on a hemogram of a dog with CPV infection is
- **a.** thrombocytopenia.
- **b.** neutropenia.
- **c.** leukopenia.
- **d.** anemia.

4. Fresh frozen plasma can be used to
- **a.** decrease the amount of crystalloid required.
- **b.** decrease oncotic pressure.
- **c.** increase vascular volume.
- **d.** provide immunoglobulins.

5. When a feeding tube is used, feeding should be withheld if the residual volume is ____ or more of the last volume fed.
- **a.** 30%
- **b.** 40%
- **c.** 50%
- **d.** 60%

6. When potassium chloride is added to fluids, the rate of administration should not exceed
- **a.** 0.25 mEq/kg/h.
- **b.** 0.5 mEq/kg/h.
- **c.** 0.5 mEq/lb/h.
- **d.** 1.0 mEq/kg/h.

7. The half-life of maternal antibodies is approximately ___ days.
- **a.** 8
- **b.** 9
- **c.** 10
- **d.** 12

8. CPV-2 is shed in the feces from day ___ to day___ after exposure.
- **a.** 3; 10
- **b.** 5; 8
- **c.** 3; 15
- **d.** 2; 9

9. CPV-2 infection most commonly affects dogs aged ____ weeks to ____ months.
- **a.** 4; 4
- **b.** 6; 6
- **c.** 5; 5
- **d.** 12; 12

10. Vaccine failure is most commonly due to
- **a.** overvaccination.
- **b.** interference by maternal antibodies.
- **c.** interference by the patient's antibodies.
- **d.** early vaccination.