JP, a 6-week-old, 3.5-kg intact male pit bull mix, presented for anorexia, vomiting, and bloody diarrhea. He had been acquired from a flea market and seemed healthy but thin.

**HISTORY**
The day after the owners acquired JP, the puppy began vomiting and, the following day, he had diarrhea. On day 4, JP was presented to the primary care veterinarian, who evaluated him for progressive listlessness, anorexia, continued vomiting, and bloody diarrhea.

The owners stated that JP had 2 littermates at the flea market that were not as playful as JP. They also confirmed the puppies had not received any vaccinations.

SNAP Parvo Test (idexx.com) results were positive, and JP was referred to another facility that had an isolation unit and provided 24-hour care.

**PHYSICAL EXAMINATION**
The physical examination findings of JP upon presentation to the referral facility are listed in Table 1. JP also had bloody diarrhea staining his ventral abdomen and hindlimbs. He vomited just after palpation. While JP was able to stand with support, he was extremely weak.

**DIAGNOSTIC APPROACH**
Peripheral IV access was attempted but, due to JP’s volume-depleted state, multiple attempts at cephalic catheterization failed. An 18-gauge, 6-cm catheter was placed into the left jugular vein, allowing a minimal volume of blood to be obtained for analysis of packed cell volume (PCV), total solids (TS), blood urea nitrogen (BUN) (Azostix, usa.healthcare.siemens.com), blood glucose (BG), and blood smear. Fecal flotation was performed on voided stool.

Depending on available resources and the financial limitations of the owners, diagnostics can follow 3 tiers of diagnostic evaluation (see Levels of Diagnostic Evaluation).

**Initial Analysis**
- Initial laboratory tests revealed:
  » BG concentration of 36 mg/dL (pediatric [6- to 8-weeks old] reference interval, 134–272 mg/dL)
  » PCV of 40% (pediatric [6- to 8-weeks old] reference interval, 27%–36%)
  » TS of 4.8 g/dL (pediatric [6- to 8-weeks old] reference interval, 3.9–4.2 g/dL)
  » BUN level of 30 to 40 mg/dL (pediatric [6- to 8-weeks old] reference interval, 14–15.5 mg/dL).
- Blood smear revealed fewer neutrophils than expected, with mild toxic change; this was grossly estimated to be a white blood cell count of 2000 to 3000 (cells/mL) on 100×

**TABLE 1. Physical Examination Findings at Referral Facility**

<table>
<thead>
<tr>
<th>BEHAVIOR</th>
<th>Listlessness</th>
</tr>
</thead>
<tbody>
<tr>
<td>CARDIAC/RESPIRATORY</td>
<td>Tachycardia (heart rate, 180 beats/min) Tachypnea (respiratory rate, 40 breaths/min), but not dyspneic Poor pulse quality Pale pink mucous membranes</td>
</tr>
<tr>
<td>BODY CONDITION</td>
<td>Hypothermia (96°F [35.6°C]) 12% dehydration (marked skin tenting and sunken eyes) Cachexia, with a 3/9 body condition score</td>
</tr>
<tr>
<td>PALPATION</td>
<td>Painful abdominal palpation Fluid-filled intestinal loops</td>
</tr>
</tbody>
</table>
magnification. Approximately 10 platelets per high power field were seen on 1000×; this was estimated to be a platelet count of 100,000 to 150,000 (cells/mcL).

- High numbers of *Toxocara canis* eggs were identified in the stool.

- Doppler blood pressure measurement was attempted, but proved difficult due to the small size of the puppy (pediatric [6- to 8-weeks old] reference range, approximately 112 mm Hg).¹

### Further Analysis

After initial treatment, blood was collected in low-volume tubes for routine complete blood count (CBC) and serum biochemical profile (*Table 2*, page 34).

### CBC Results

- Marked lymphopenia and neutropenia were seen, typical of parvoviral enteritis.
- Relative hemoconcentration was likely due to severe dehydration. Low PCV due to age and/or anemia due to gastrointestinal (GI) bleeding may become evident after fluid therapy.

### Biochemistry Results

- JP’s hypoglycemia was more severe than could be attributed to age, cachexia, and decreased food intake.
- Cholesterol is often lower in puppies than adult dogs.
- Electrolyte depletion was attributed to vomiting and diarrhea.
- Low albumin and total protein levels were most likely caused by protein loss through the GI tract, including via GI bleeding.
- Hypocalcemia was at least partially related to severe hypoalbuminemia.
- Increased alkaline phosphatase, bilirubin, and phosphorus were most likely related to a combination of age and illness.

### DIAGNOSIS

#### Cormorbidities

In this patient, canine parvovirus infection had been diagnosed before referral. Additional comorbid conditions may include infection with another virus (eg, coronavirus), intestinal parasitism, intestinal bacterial infection or overgrowth (eg, *Escherichia coli*, *Salmonella* species, *Campylobacter* species, *Clostridia* species), intussusception, or foreign body.

#### Sepsis & Hypovolemic Shock

The presence of hypovolemic shock and sepsis—caused by dehydration, leukopenia, and intestinal bacterial translocation—is the most life-threatening condition present in JP. Associated hypoglycemia, hypoalbuminemia, and electrolyte or acid-base disturbances must be addressed.
### TABLE 2.
Clinicopathologic Results²⁻⁵

<table>
<thead>
<tr>
<th>VARIABLE</th>
<th>RESULT</th>
<th>ADULT REFERENCE INTERVAL</th>
<th>PEDIATRIC (6⁻8 WEEKS OF AGE) INTERVAL</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Complete Blood Count</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Red blood cell count (× 10⁶/L)</td>
<td>5.06</td>
<td>5.34-8.5</td>
<td>4.3-5.1</td>
</tr>
<tr>
<td>Hemoglobin (g/dL)</td>
<td>13</td>
<td>12.3-19.7</td>
<td>8.5-11.3</td>
</tr>
<tr>
<td>Hematocrit (%)</td>
<td>40</td>
<td>37-57</td>
<td>26.5-35.5</td>
</tr>
<tr>
<td>Mean corpuscular volume (fL)</td>
<td>60</td>
<td>59-76</td>
<td>63.2-74.3*</td>
</tr>
<tr>
<td>Mean corpuscular hemoglobin (pg)</td>
<td>23</td>
<td>20.7-25.6</td>
<td>23-25.5*</td>
</tr>
<tr>
<td>Mean corpuscular hemoglobin concentration (g/dL)</td>
<td>33.2</td>
<td>32-36.4</td>
<td>31.4-35.1</td>
</tr>
<tr>
<td>Platelet count (× 10³/L)</td>
<td>136,000</td>
<td>200-500</td>
<td>&gt; 150,000</td>
</tr>
<tr>
<td>White blood cell count (× 10³/L)</td>
<td>2.6</td>
<td>4.53-14.99</td>
<td>12.6-26.7</td>
</tr>
<tr>
<td>Segmented neutrophils (× 10³/L)</td>
<td>1.82</td>
<td>2.27-10.14</td>
<td>4.2-17.6</td>
</tr>
<tr>
<td>Band neutrophils (× 10³/L)</td>
<td>0.26</td>
<td>0-0.26</td>
<td>0-0.3</td>
</tr>
<tr>
<td>Lymphocytes (× 10³/L)</td>
<td>0.52</td>
<td>0.76-4.23</td>
<td>2.8-16.6</td>
</tr>
<tr>
<td>Monocytes (× 10³/L)</td>
<td>0.15</td>
<td>0.15-1.35</td>
<td>0.5-2.7</td>
</tr>
<tr>
<td>Eosinophils (× 10³/L)</td>
<td>0</td>
<td>0.08-1.1</td>
<td>0.1-1.9</td>
</tr>
<tr>
<td>Basophils (× 10³/L)</td>
<td>0</td>
<td>0-0.15</td>
<td>0</td>
</tr>
<tr>
<td>Reticulocytes (%)</td>
<td>0.4</td>
<td>n/a</td>
<td>2.6-6.2</td>
</tr>
<tr>
<td><strong>Serum Biochemical Profile</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Glucose (mg/dL)</td>
<td>86ᵇ</td>
<td>81-133</td>
<td>134-272</td>
</tr>
<tr>
<td>Blood urea nitrogen (mg/dL)</td>
<td>33</td>
<td>8-28</td>
<td>14-15.5</td>
</tr>
<tr>
<td>Creatinine (mg/dL)</td>
<td>1</td>
<td>0.6-1.6</td>
<td>0.6-1.6</td>
</tr>
<tr>
<td>Sodium (mEq/L)</td>
<td>136</td>
<td>143-152</td>
<td>143-152</td>
</tr>
<tr>
<td>Potassium (mEq/L)</td>
<td>3.1</td>
<td>3.4-4.9</td>
<td>3.4-4.9</td>
</tr>
<tr>
<td>Chloride (mEq/L)</td>
<td>106</td>
<td>108-117</td>
<td>108-117</td>
</tr>
<tr>
<td>Bicarbonate (mEq/L)</td>
<td>18</td>
<td>18-26</td>
<td>18-26</td>
</tr>
<tr>
<td>Anion gap (mEq/L)</td>
<td>3</td>
<td>13-22</td>
<td>n/aᵃ</td>
</tr>
<tr>
<td>Albumin (g/dL)</td>
<td>1.9</td>
<td>2.9-4</td>
<td>2.1-2.7</td>
</tr>
<tr>
<td>Plasma protein (g/dL)</td>
<td>4.8</td>
<td>6-8</td>
<td>6-8</td>
</tr>
<tr>
<td>Total protein (g/dL)</td>
<td>3.2</td>
<td>5.2-7.4</td>
<td>3.9-4.8</td>
</tr>
<tr>
<td>Calcium (mg/dL)</td>
<td>8</td>
<td>9.2-11.3</td>
<td>9.2-11.3</td>
</tr>
<tr>
<td>Phosphorus (mg/dL)</td>
<td>5.7</td>
<td>2-5</td>
<td>8.7-11.5</td>
</tr>
<tr>
<td>Cholesterol (mg/dL)</td>
<td>137</td>
<td>133-338</td>
<td>111-258</td>
</tr>
<tr>
<td>Total bilirubin (mg/dL)</td>
<td>0.4</td>
<td>0.1-0.4</td>
<td>0.1-0.2</td>
</tr>
<tr>
<td>Alanine aminotransferase (U/L)</td>
<td>48</td>
<td>9-58</td>
<td>9-24</td>
</tr>
<tr>
<td>Alkaline phosphatase (U/L)</td>
<td>178</td>
<td>5-129</td>
<td>144-177</td>
</tr>
<tr>
<td>Gamma-glutamyltransferase (U/L)</td>
<td>&lt; 3</td>
<td>0-5</td>
<td>0-7</td>
</tr>
<tr>
<td>Creatine kinase (U/L)</td>
<td>267</td>
<td>10-274</td>
<td>10-274</td>
</tr>
<tr>
<td>Colloid osmotic pressure (mm Hg)</td>
<td>12</td>
<td>21-25</td>
<td>&lt; 18</td>
</tr>
</tbody>
</table>

---

*a Values are for 4-week-old puppies rather than 6- to 8-week-old puppies
*b After glucose bolus upon admission
*c No published data available on reference interval for this variable in pediatric patients

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**Should Oxygen Be Given During Initial Resuscitation?**

While JP was tachypneic—likely secondary to hypovolemia—he was not dyspneic and his lung sounds were clear. Therefore, supplemental oxygen was probably not necessary but could have been provided until normoxemia was confirmed. However, for a neonate (< 2 weeks of age), oxygen therapy would be recommended.
TREATMENT APPROACH

Therapy for Hypovolemic Shock
- Due to the life-threatening hypoglycemia, JP received an initial IV bolus of 0.5 g/kg dextrose (3 mL of 50% dextrose diluted in 7 mL of 0.9% saline over 1 minute).
- Aggressive fluid therapy was indicated due to the extreme dehydration and hypovolemic shock (see Fluid Therapy Plan).
- A bolus of 120 mL (34 mL/kg) of warmed lactated Ringer’s solution was administered over 15 minutes, followed by 33 mL/H (10 mL/kg/H) of lactated Ringer’s solution with 5% dextrose and potassium chloride supplementation.
- A colloid was administered in addition to the crystalloid.
- BG was measured every 6 H, with glucose supplementation adjusted as necessary to maintain normoglycemia.

Therapy for Parvovirus & Sepsis
JP was treated with symptomatic and supportive care (see Typical Supportive Therapies for Parvoviral Enteritis), including provision of warmth and zinc oxide barrier therapy to prevent moist dermatitis.

Due to sepsis secondary to leukopenia, antibiotic therapy was initiated (IV initially; then PO once per day).

Crystalloid fluids, along with dextrose and potassium chloride supplementation as needed
Colloidal support, such as VetStarch, hetastarch, or plasma
Antiemetics, such as maropitant, dolasetron, or metoclopramide
Gastric protectants, such as famotidine or pantoprazole
Parenteral antibiotic therapy while hospitalized (eg, ampicillin/sulbactam, cefoxitin)
Antidiarrheals, such as probiotics or metronidazole
Analgesics, such as buprenorphine and lidocaine
Nutritional support, such as nasogastric tube feeding (if oral feeding refused) or mirtazapine

Fluid Therapy Plan

1. Expand intravascular volume and treat shock with IV bolus of fluids; JP received a 120-mL bolus (30 mL/kg). Volume resuscitate the patient appropriately based on clinical signs of improved:
   - Pulse quality
   - Heart rate
   - Mentation
   - General perfusion parameters.

2. Correct estimated dehydration: JP’s estimated dehydration was 12%. The remaining deficit was calculated as follows:
   - 3.5 kg (body weight) × 0.12 (dehydration) = 420 mL fluid deficit
   - 420 mL (original deficit before fluid bolus) – 120-mL bolus fluid volume = 300 mL
   - 300 mL (remaining deficit)/12 H = 25 mL/H

3. Provide for estimated ongoing loss: JP had frequent diarrhea and vomiting, with an estimated loss of 75 mL Q 24 H; therefore, 75 mL/24 H = 3.1 mL/H

4. Provide maintenance fluids; fluid requirements for puppies vary with age (Table 3). For JP, fluid requirements were:
   - 3.5 kg (body weight) × 80 mL Q 24 H = 280 mL Q 24 H
   - 280 mL/24 H = 11.7 mL/H

5. Consider colloidal support (see Table 2):
   - VetStarch (abbotanimalhealth.com): 2 mL/kg/H
   - 3.5 kg (body weight) × 2 mL/kg/H = 7 mL/H

Crystalloid fluid rate for the first 12 hours—after the initial fluid bolus—was calculated by considering:

Replacement (25 mL/H) + ongoing losses (3.1 mL/H) + maintenance (11.7 mL/H) = 39.8 mL/H
Colloid volume was subtracted (39.8 mL/H – 7 mL/H), which equaled a crystalloid fluid rate of 33 mL/H.

During the remaining period of hospitalization, and after the dehydration deficit had been replaced, fluid rate was decreased to reflect only maintenance needs and ongoing losses.
vomiting abated). A potent antiemetic was initiated to treat nausea and minimize risk for aspiration pneumonia. The supportive medications and nutrition JP received are outlined in Table 4; daily in-hospital monitoring for parvovirus patients is described in Table 5.

### TABLE 4. Supportive Therapy for JP

<table>
<thead>
<tr>
<th>THERAPY TYPE</th>
<th>MEDICATION/DIET</th>
<th>DOSAGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antiemetic</td>
<td>Maropitant 1 mg/kg SC or IV Q 24 H</td>
<td></td>
</tr>
<tr>
<td>Gastroprotectant</td>
<td>Famotidine 0.5 mg/kg IV Q 12 H</td>
<td></td>
</tr>
<tr>
<td>Antibiotic</td>
<td>Ampicillin/subactam 25 mg/kg IV Q 8 H</td>
<td></td>
</tr>
<tr>
<td>Antidiarrheal</td>
<td>Probiotics, dependent on probiotic chosen</td>
<td></td>
</tr>
<tr>
<td>Analgesics</td>
<td>Buprenorphine 0.02 mg/kg IV Q 8 H PRN</td>
<td></td>
</tr>
<tr>
<td>Nutrition</td>
<td>Clinicare (abbottanimalhealth.com) RER/24 H = mLs of Clinicare per H via nasogastric tube</td>
<td></td>
</tr>
</tbody>
</table>

CRI = constant rate infusion; PRN = as needed; RER = resting energy requirement

### TABLE 5. Daily Monitoring Recommended for Parvovirus Patients

<table>
<thead>
<tr>
<th>THERAPY SPECIFICS</th>
<th>FREQUENCYa</th>
</tr>
</thead>
<tbody>
<tr>
<td>Physical examination</td>
<td>Temperature, heart rate, respiratory rate, lung auscultation, pulse quality, mucous membrane color, capillary refill time, abdominal pain, urine output</td>
</tr>
<tr>
<td>Monitoring</td>
<td>Packed cell volume, total solids, blood glucose</td>
</tr>
<tr>
<td>Blood monitoring</td>
<td>Electrolytes, especially potassium</td>
</tr>
<tr>
<td>Blood pressure</td>
<td>Q 6 to 8 Hb</td>
</tr>
<tr>
<td>Pulse oximetry</td>
<td>Dependent on clinical signs</td>
</tr>
<tr>
<td>Nursing care &amp; barrier treatment</td>
<td>For example, zinc oxide</td>
</tr>
<tr>
<td>Nutritional evaluation</td>
<td>Gastric residual volume, tolerance of nasoesophageal or nasogastric tube feeding</td>
</tr>
</tbody>
</table>

a. Dependent on severity of clinical signs
b. These parameters may need to be monitored more frequently, even Q 1 H, in some very critical or dynamic patients.

Ongoing Supportive Care

JP’s vital parameters improved markedly over the first few hours of therapy. By the following day, he subjectively looked less nauseated and had less abdominal pain. A temporary nasogastric feeding tube was placed at that time for nutritional support, with sporadic gastric suctioning to measure.

Other Therapeutic Options: Worth It?

In the past, fresh or fresh frozen plasma from recovered dogs had been suggested to provide antiparvoviral antibodies. Recent studies, however, have demonstrated no beneficial effect of this method and shown that even recently recovered animals have minimal anti-canine parvovirus antibody concentrations. Moreover, such treatment may prime the dog for future transfusion reactions later in life.

Equine endotoxin antiserum, recombinant human granulocyte-stimulating factor, and antiviral agents (eg, oseltamivir) have not been shown to improve survival or outcome. In small studies, use of feline interferon has been weakly associated with improved survival; however, this agent is not readily available in veterinary hospitals in the U.S.
residual gastric volume. By day 4, JP was ingesting small amounts of meat-based baby food, and the nasogastric tube was removed. JP was dewormed with fenbendazole and discharged later that day.

**PROGNOSIS**

JP was discharged on day 4, and the owner was taught how to administer medications and encourage JP to eat. The primary care veterinarian performed a recheck examination 3 days later, and reported that JP was acting and eating normally at that time.

The prognosis for canine parvovirus infection is fair to good. Perhaps surprisingly, severity of neutropenia is not a negative prognostic factor; rather, severity of dehydration and lymphopenia may be instead. Recently, several studies have evaluated other measures that may affect prognosis.

A study from Colorado State University compared standard in-hospital treatment versus a modified outpatient treatment (using volume resuscitation followed by SC fluid therapy and supportive care), with recent survival rates of 80% to 90% reported with treatment. Both protocols can be successful, with a slightly lower survival rate in outpatients.

Hospitalization with intensive therapy was initially indicated for JP due to his severe hypoglycemia, dehydration, and shock, but a modified outpatient protocol (SC fluids, antiemetics, antibiotics) may be a good alternative for less severely affected patients or clients with financial limitations.

**IN SUMMARY**

Although there are differences between young and adult animals, pediatric patients can still be treated aggressively and respond well to therapy. However, clinicians must be aware of their normal physiologic and hemodynamic measures. The small size of these patients should not limit our ability to appropriately treat them.

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**TEMPERATURE**

- Normal rectal temperature in neonates is 96°F ± 1.5°F (35.6°C ± 0.7°C) in the first week of life; then 98.6°F to 100°F (37°C to 38.2°C) in the second and third weeks of life.
- Adult temperatures are reached by 7 weeks of age.
- Careful warming should be initiated to prevent overheating.

**HYPOGLYCEMIA**

- Neonates and pediatric patients are prone to hypoglycemia due to decreased glycogen stores, inefficient hepatic gluconeogenesis, and an immature glucose feedback mechanism.
- Hypoglycemia is worsened by anorexia, ongoing losses (eg, vomiting, diarrhea), dehydration, and sepsis.
- Frequent BG monitoring is warranted in these patients; however, the minimum amount of blood should be drawn to prevent iatrogenic anemia.

**IMMUNE SYSTEM**

- In neonate and pediatric patients, the immune system is not fully mature until 3 to 6 months.
- Poor husbandry (eg, lack of vaccination, lack of parasite prevention) often worsens clinical disease.

---

**Key Points:**
Treating the Pediatric Patient

In critically ill neonate and pediatric patients, goals of treatment should be prioritized by the four H’s:

- Hypovolemia/hydration
- Hypothermia
- Hypoglycemia
- Hypoxemia

**FLUID THERAPY**

- Dehydration can rapidly progress to hypovolemia in neonates and pediatric patients; therefore, aggressive fluid therapy is warranted because these small patients can deteriorate quickly.
- Fluid requirements for neonates and pediatric patients are much higher than those for adults.
- In critically ill pediatric patients, fluid therapy for shock must initially be given by IV or intraosseous routes. Intraperitoneal or SC routes are not adequate due to slower absorption and, ideally, should not be used in the critically ill, dehydrated, or hypovolemic patient.
- Colloids can be used in pediatric patients; however, keep in mind that puppies have a lower colloid osmotic pressure than adult dogs. If necessary, a colloid (eg, hetastarch, 1 mL/kg/H; VetStarch, 2 mL/kg/H) can be used to keep colloid osmotic pressure above 15 mm Hg. No published data are available on colloid use in neonates.
Pathophysiology of Parvovirus

Canine parvovirus (CPV) is a common and severe pathogen that affects young dogs that are unvaccinated, under-vaccinated, or immunosuppressed. The virus first emerged in dogs in the mid 1970s and has since mutated into 3 different forms: CPV-2a, CPV-2b, and, most recently, CPV-2c.

All 3 forms of CPV are environmentally stable, nonenveloped viruses transmitted via the fecal–oral route.

- The virus initially replicates in oropharyngeal lymphoid tissues, leading to viremia; rapidly dividing cells of the GI tract, thymus, lymph nodes, and bone marrow are most affected.
- Loss of both intestinal epithelial villous and crypt cells leads to malabsorption and increased intestinal permeability, accompanied by vomiting, diarrhea, and GI bleeding.
- Destruction of bone marrow cells results in neutropenia and, to a lesser degree, thrombocytopenia.
- Translocation of intestinal bacteria, complicated by neutropenia, often leads to bacteremia, endotoxemia, and sepsis.

Without treatment, CPV can be life threatening due to sepsis, severe fluid losses and electrolyte derangements secondary to anorexia, vomiting, and diarrhea. In order to ensure the best outcome, treatment should be aimed toward symptomatic supportive care, aggressive fluid therapy, antiemetics, antibiotic therapy, and nutritional support.

References


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Justine A. Lee, DVM, Diplomate ACVECC & ABT, is the CEO and founder of VETgirl (vetgirlontherun.com), a subscription-based podcast and webinar service that offers RACE-approved veterinary continuing education. Dr. Lee recently received the 2015 NAVC Speaker of the Year Award, and is the author and editor of several veterinary textbooks, book chapters, and scientific publications. She completed her veterinary training at Cornell University, Angell Animal Medical Center (Boston), and University of Pennsylvania.

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