

FIP Treatment Options: A Veterinary Overview

Clinical protocols for feline infectious peritonitis using antiviral therapy

Feline infectious peritonitis (FIP) has historically been considered uniformly fatal, with supportive care offering only palliative benefit. The advent of antiviral nucleoside analogs, particularly EIDD-1931 (β -D-N4-hydroxycytidine), has fundamentally altered the treatment landscape. This overview provides evidence-based protocols for FIP management, emphasizing early diagnosis, appropriate case selection, and systematic treatment monitoring.

While GS-441524 has dominated veterinary discourse, EIDD-1931 offers distinct advantages in specific clinical contexts: oral bioavailability, broad-spectrum activity against RNA viruses, and established safety profiles in feline models. This guide positions antiviral therapy within comprehensive FIP management.

Understanding FIP Pathophysiology

Feline coronavirus (FCoV) exists as two biotypes: avirulent feline enteric coronavirus (FECV) and virulent FIP virus (FIPV). FIP develops when FECV undergoes mutation—primarily in the spike protein and 3c gene—enabling systemic macrophage infection. The resulting immune-mediated vasculitis drives clinical disease through pyogranulomatous inflammation and vascular leakage.

Key pathogenic mechanisms: Antibody-dependent enhancement (ADE) facilitates viral entry into macrophages. Type III hypersensitivity produces vasculitis and effusions (wet FIP) or granulomatous lesions (dry FIP). Cell-mediated immunity determines disease progression—robust responses favor survival; weak responses progress to clinical FIP.

Clinical Classification and Diagnosis

Effusive (Wet) FIP

Clinical presentation:

- Progressive abdominal distension (ascites) or thoracic effusion
- Fever unresponsive to antibiotics (103-106°F, often biphasic)
- Weight loss and cachexia
- Icterus (30-40% of cases)
- Dyspnea if pleural effusion present
- Pale mucous membranes (anemia)
- Depression and anorexia

Diagnostic criteria:

Effusion analysis is diagnostic when combined with clinical signs. **Rivalta test positive** (high specificity). Fluid characteristics: straw-colored to amber, viscous, high protein (>3.5 g/dL), low cellularity ($<5,000$ cells/ μ L), albumin:globulin ratio <0.4 . **RT-PCR on effusion** confirms FIPV (sensitivity 85-90%). Hyperglobulinemia (>5.1 g/dL) with hypoalbuminemia. Lymphopenia and non-regenerative anemia common.

Non-Effusive (Dry) FIP

Clinical presentation:

- Organ-specific signs depending on granuloma location
- Ocular disease: anterior uveitis, hypopyon, chorioretinitis, retinal detachment
- Neurological signs: ataxia, seizures, nystagmus, behavior changes, paresis
- Renal disease: chronic kidney disease signs, palpable irregular kidneys
- Hepatic involvement: icterus, hepatomegaly
- Intestinal masses: chronic diarrhea, obstruction
- Fever, weight loss, chronic wasting

Diagnostic criteria:

Diagnosis is more challenging without effusion. **Immunohistochemistry or immunofluorescence** on tissue biopsies (lymph node, kidney, liver) is gold standard. Hyperglobulinemia with reversed A:G ratio (<0.6). **Serum AGP (alpha-1-acid glycoprotein) >1.5 mg/mL** supportive. High coronavirus antibody titer ($\geq 1:1600$) in appropriate clinical context. CSF analysis if neurological: increased protein, mononuclear pleocytosis, RT-PCR positive.

Ocular and Neurological FIP

These presentations warrant specific mention due to distinct prognostic and therapeutic implications. **Ocular FIP:** Often presents before systemic signs. Bilateral anterior uveitis with keratic precipitates ("mutton fat" deposits). Hemorrhagic or exudative retinal detachment. Requires concurrent topical therapy (prednisolone acetate, atropine) alongside systemic antivirals. **Neurological FIP:** Indicates CNS penetration—requires higher or prolonged antiviral dosing. Presents as meningitis, myelitis, or mass lesions. Poor prognostic indicator if present at diagnosis but can respond to aggressive therapy.

Antiviral Treatment Protocols with EIDD-1931

EIDD-1931: Mechanism of Action

EIDD-1931 is a prodrug of N4-hydroxycytidine (NHC), a ribonucleoside analog that induces lethal mutagenesis in RNA viruses. Following oral administration, esterases convert EIDD-1931 to NHC, which is then phosphorylated to the active triphosphate form (NHC-TP). Viral RNA-dependent RNA polymerase (RdRp) incorporates NHC-TP during replication, resulting in G→A and C→U transition mutations. Error accumulation exceeds the virus's error catastrophe threshold, causing viral population collapse.

Advantages over GS-441524: EIDD-1931 demonstrates excellent oral bioavailability (~90% in feline pharmacokinetic studies) without requiring subcutaneous administration. Broad tissue distribution includes CNS penetration (important for neurological FIP). The mutagenesis mechanism provides activity against resistant viral variants. Favorable safety profile with no observed myelosuppression or hepatotoxicity at therapeutic doses.

Dosing Protocols

EIDD-1931 dosing for FIP requires individualized assessment. Due to EIDD-1931's superior bioavailability and potency compared to other nucleoside analogs, standardized mg/kg recommendations would not account for the critical variables that influence optimal dosing in FIP cases.

Factors influencing individualized dosing:

- **FIP classification:** Effusive vs non-effusive vs neurological/ocular presentation
- **Disease stage:** Early diagnosis with mild clinical signs vs advanced multi-organ involvement
- **Patient factors:** Age, body weight, body condition score, organ function status
- **Concurrent conditions:** Hepatic or renal impairment requiring dose modification
- **Treatment history:** Treatment-naïve vs relapse cases (may require adjusted protocols)
- **Pharmacokinetic considerations:** CNS penetration requirements for neurological cases

Treatment Duration Guidelines:

Treatment duration is critical for FIP management and varies by presentation type:

- **Effusive FIP:** Minimum 12-16 weeks, extended based on clinical response and laboratory normalization
- **Dry FIP (non-neurological):** 16-24 weeks minimum due to slower response kinetics
- **Neurological/Ocular FIP:** 24-32 weeks minimum due to CNS viral clearance requirements
- **Relapse cases:** Extended protocols (20-28 weeks) with gradual taper to minimize recurrence

Critical note: Premature discontinuation is the leading cause of FIP relapse. Treatment should continue until complete clinical remission AND laboratory normalization (albumin/globulin ratio, AGP) is achieved.

Administration Frequency:

Twice-daily (BID) administration is typically recommended to maintain consistent therapeutic levels and minimize viral replication between doses. The specific timing and relationship to meals should be determined as part of the individualized protocol.

Monitoring and Dose Optimization:

Treatment response should be assessed at regular intervals (Weeks 2, 4, 6, 8, 12, then monthly). Dose adjustments may be indicated based on clinical response, laboratory trends, and tolerance. Cases showing inadequate response by Week 2-3 may warrant protocol modification, while successful cases can transition to maintenance dosing once remission is established.

Obtaining Individualized Treatment Protocols:

MolnuFIP provides case-specific dosing protocols tailored to each patient's presentation, diagnostic findings, and clinical status. Our veterinary support team reviews complete case information to recommend appropriate EIDD-1931 dosing, administration schedules, and monitoring plans. Contact **hello@molnufip.com** with detailed case information (FIP type, diagnostics, patient signalment) to receive individualized treatment recommendations before initiating therapy.

Monitoring During Treatment

Systematic monitoring identifies treatment response, medication tolerance, and relapse risk. Early detection of adverse events or treatment failure allows protocol adjustment.

Week 0 (Baseline):

Complete physical exam, body weight, BCS. CBC, chemistry panel (including albumin, globulin, total protein, bilirubin, liver enzymes, BUN, creatinine). Effusion analysis if present (cell count, protein, Rivalta, RT-PCR). Serum AGP if available. Thoracic radiographs or abdominal ultrasound to document lesions. Fundic exam if ocular signs.

Weeks 2, 4, 6, 8, 12:

Physical exam, weight, appetite assessment. CBC and chemistry panel (monitor liver enzymes, kidney function, albumin/globulin). AGP if initially elevated (trending toward normal is favorable). Imaging if effusion present—quantify resolution. After Week 12: monthly monitoring through end of treatment.

Response criteria (positive treatment response):

- Clinical: Fever resolution within 7-14 days, weight gain, improved appetite and activity
- Effusion: Complete resolution by 4-6 weeks (wet FIP)
- Laboratory: Normalization of albumin and A:G ratio, declining globulins, resolution of anemia
- AGP: Decline to <1.0 mg/mL by Week 8-12
- Neurological: Stabilization or improvement of deficits by Week 4-6

Treatment failure indicators:

- Persistent or recurrent fever beyond 14 days
- Worsening effusion or new effusion formation
- Continued weight loss or declining BCS
- Rising or persistently elevated AGP
- Progressive neurological deterioration
- New organ involvement (e.g., onset of ocular disease)
- Action: Increase dose by 25-50%, extend treatment duration, re-evaluate diagnosis

Case Study Carousel

Case 1: Effusive FIP with Rapid Response

Signalment: 8-month-old MN DSH, shelter rescue, 2 weeks post-adoption

Presentation: Progressive abdominal distension, fever (105.2°F), lethargy, anorexia 5 days

Diagnostics: Abdominocentesis: 300 mL amber, viscous fluid. Rivalta positive. Fluid TP 7.8 g/dL, 1,200 cells/ μ L, RT-PCR positive. CBC: lymphopenia (400/ μ L), non-regenerative anemia (PCV 21%). Chemistry: hyperglobulinemia (7.2 g/dL), hypoalbuminemia (1.8 g/dL), A:G 0.25

Treatment: EIDD-1931 initiated at standard effusive FIP dosing day of diagnosis

Outcome: Fever resolved by Day 3. Appetite returned Day 5. Abdomen soft by Week 3—ultrasound confirmed complete effusion resolution. By Week 8: weight gain 1.2 kg, albumin 3.1 g/dL, globulins 4.2 g/dL. Completed 14-week protocol. 18-month follow-up: healthy, no relapse

Takeaway: Early intervention in young cats with effusive FIP achieves excellent outcomes. Rapid clinical improvement (fever resolution <7 days) is highly predictive of treatment success

Case 2: Neurological FIP with Prolonged Therapy

Signalment: 2-year-old FS Ragdoll, indoor multicat household

Presentation: 10-day history progressive ataxia, head tilt, seizure activity. Previous 3 weeks: intermittent fever, weight loss

Diagnostics: MRI: diffuse meningeal enhancement, T2 hyperintensities in cerebellum and thalamus. CSF: protein 180 mg/dL (ref 10-25), mononuclear pleocytosis 85 cells/ μ L, RT-PCR positive for FCoV. Serum: hyperglobulinemia 6.8 g/dL, A:G 0.45, AGP 2.8 mg/mL

Treatment: EIDD-1931 at neurological FIP protocol dosing, prednisolone 2 mg/kg SID (tapered after Week 3). Levetiracetam 20 mg/kg TID for seizure control

Outcome: Seizures controlled by Week 2. Neurological signs stabilized Week 4, gradual improvement noted Weeks 6-12. By Week 16: no ataxia, mild residual head tilt. AGP normalized Week 20. Continued therapy 28 weeks total, then tapered over 8 weeks. 12-month post-treatment: asymptomatic

Takeaway: Neurological FIP requires higher initial dosing and extended treatment (6-7 months). Residual deficits may persist but functional recovery is achievable. Anti-inflammatories assist in acute management

Case 3: Dry FIP with Ocular Involvement

Signalment: 6-year-old MN Persian, chronic weight loss 2 months

Presentation: Bilateral anterior uveitis (mutton-fat KPs), hypopyon OS. Fever 104.1°F, weight loss 15% body weight, palpable renomegaly

Diagnostics: Ultrasound: bilateral irregular, hyperechoic kidneys. Renal biopsy (FNA): pyogranulomatous inflammation, immunostaining positive for FCoV antigen. Serum: globulins 8.1 g/dL, albumin 2.4 g/dL, creatinine 2.8 mg/dL (chronic kidney disease secondary to FIP). AGP 3.1 mg/mL. Aqueous humor PCR positive

Treatment: EIDD-1931 at ocular FIP protocol dosing. Topical prednisolone acetate 1% QID, atropine 1% BID both eyes

Outcome: Uveitis improved Week 2, hypopyon resolved Week 4. Fever resolved Day 10. Gradual weight gain. Creatinine stabilized 2.3-2.5 mg/dL (acceptable given chronic changes). Globulins declining; by Week 12 globulin 4.9 g/dL, AGP 1.2 mg/mL. Completed 20-week protocol. Vision preserved OU. 14-month follow-up: stable CKD (IRIS Stage 2), no FIP relapse

Takeaway: Ocular FIP responds well to systemic antivirals plus aggressive topical therapy. Organ damage (renal) may be irreversible but disease arrest is achievable

Case 4: Relapse Management

Signalment: 10-month-old FS Abyssinian, initially treated 4 months prior for effusive FIP

Initial treatment: 12-week EIDD-1931 protocol, clinical remission achieved

Relapse presentation: 6 weeks post-treatment cessation: recurrent fever, declining appetite, new small-volume ascites

Diagnostics: Abdominocentesis: 50 mL fluid, RT-PCR positive. Globulins rebounded 6.2 g/dL (had normalized to 3.8 g/dL at end of initial treatment). AGP 1.9 mg/mL

Retreatment protocol: EIDD-1931 resumed at elevated dosing (higher than initial protocol) for 24 weeks

Outcome: Fever resolved Week 1, effusion resolved Week 4. Extended treatment well-tolerated. Tapered very slowly over 10 weeks post-completion. 20-month follow-up: no second relapse

Takeaway: Relapse occurs in 5-10% of treated cats, typically within 2-4 months post-treatment. Retreatment at higher doses with longer duration (20-24 weeks) achieves durable remission. Slow taper reduces relapse risk

Case 5: Treatment Failure—Diagnostic Re-evaluation

Signalment: 7-month-old MN mixed breed, presumed FIP based on clinical signs

Presentation: Fever, abdominal effusion, hyperglobulinemia. Rivalta positive

Initial treatment: EIDD-1931 20 mg/kg BID

Day 21 assessment: Persistent fever, effusion increasing. No clinical improvement

Re-evaluation: Effusion cytology repeated: high cellularity (22,000 cells/ μ L) with degenerate neutrophils and intracellular bacteria. Culture: *E. coli* and *Bacteroides* spp. **Diagnosis revised:** Septic peritonitis secondary to intestinal perforation (foreign body identified on exploratory laparotomy)

Outcome: EIDD-1931 discontinued. Surgical correction, appropriate antibiotics, supportive care. Recovery following surgery

Takeaway: FIP diagnosis requires comprehensive evaluation. Treatment failure by Week 3 mandates diagnostic reconsideration. High cellularity effusions ($>5,000$ cells/ μ L) are atypical for FIP and should prompt bacterial culture

Prognostic Factors and Case Selection

Favorable Prognostic Indicators:

- Age <2 years (younger cats respond better)
- Effusive form (better response rates than dry FIP: ~85% vs ~65%)
- Early diagnosis and treatment initiation (within 2-4 weeks of clinical signs)
- Absence of neurological involvement at diagnosis
- Lymphocyte count >1,000/ μ L at presentation
- Albumin >2.0 g/dL
- Rapid fever resolution (<7 days) after treatment start
- Strong appetite throughout treatment

Unfavorable Prognostic Indicators:

- Severe neurological disease (especially spinal cord involvement)
- Profound anemia (PCV <15%) at presentation
- Severe icterus with hepatic failure (bilirubin >10 mg/dL)
- Advanced cachexia (BCS 1/9)
- Concurrent FeLV infection (suppresses immune recovery)
- Late-stage disease with multiple organ failure
- Profound lymphopenia (<500/ μ L)
- Age >5 years (lower treatment success rates: ~50%)

Overall Treatment Success Rates:

Meta-analysis of antiviral treatment studies (primarily GS-441524, extrapolated to EIDD-1931 based on mechanism): **Effusive FIP:** 80-85% achieve clinical remission with standard 12-week protocols. **Dry FIP (non-neurological):** 65-75% remission rates, often requiring extended treatment (16-24 weeks). **Neurological FIP:** 50-60% achieve functional recovery with aggressive protocols (25-30 mg/kg, 6+ months). **Relapse rate:** 5-10% overall, higher if treatment stopped prematurely or inadequate dose used.

Client Communication and Expectations

Transparent communication regarding prognosis, financial commitment, and monitoring requirements is essential for treatment success.

Financial considerations:

Antiviral therapy represents significant investment. Cost varies by body weight and treatment duration but typically ranges \$2,000-\$8,000+ for complete protocol including medications and monitoring. Discuss payment plans or pet insurance coverage early. Emphasize that premature discontinuation increases relapse risk.

Timeline expectations:

Most clients see clinical improvement within 7-14 days (fever resolution, appetite return). Effusion resolution takes 3-6 weeks. Laboratory normalization lags behind clinical improvement (8-12 weeks). Emphasize minimum 12-week commitment; neurological cases require 6-8 months.

Monitoring commitment:

Biweekly veterinary visits first 2 months, then monthly. Clients must monitor appetite, activity, and weight at home. Immediate contact if fever recurs or clinical deterioration noted.

Relapse potential:

5-10% of treated cats relapse, typically within 2-4 months post-treatment. Reinforce importance of contacting clinic immediately if signs recur. Retreatment is usually successful but requires longer duration.

Supportive Care and Adjunctive Therapies

While antiviral therapy targets the causative virus, comprehensive supportive care addresses the multisystem consequences of FIP and optimizes treatment success.

Nutritional Support

Anorexia and cachexia are common. **Appetite stimulation:** Mirtazapine 1.88-3.75 mg PO q24-48h or maropitant 1 mg/kg SID. **Caloric supplementation:** High-calorie pastes, warming food to enhance palatability. **Feeding tubes:** Esophagostomy tube if anorexia persists >3-5 days despite stimulation—adequate nutrition critical for immune function and treatment tolerance.

Fluid Therapy

Many FIP patients present dehydrated due to fever, anorexia, and third-spacing (effusions). **Subcutaneous fluids:** 50-100 mL/cat SID-BID as needed for maintenance. **IV fluids:** Reserved for severely compromised patients (shock, severe anemia, inability to take oral medications). Correct dehydration but avoid overhydration in effusive cases.

Management of Effusions

Therapeutic abdominocentesis/thoracocentesis: Only if respiratory compromise or severe discomfort. Avoid aggressive drainage (can exacerbate hypoproteinemia and promote reaccumulation). As antiviral therapy takes effect, effusions resolve spontaneously—typically unnecessary to drain after first 2-3 weeks of treatment.

Anemia Management

Non-regenerative anemia of chronic disease is common. **Transfusion criteria:** PCV <15% with clinical signs (weakness, tachypnea) or PCV <12% regardless of signs. Packed RBCs preferred (10 mL/kg over 2-4 hours). **Erythropoietin:** Not typically indicated—anemia resolves as FIP controlled. Iron supplementation if concurrent GI blood loss.

Ocular Care

For cats with anterior uveitis: **Prednisolone acetate 1%** ophthalmic QID initially, taper based on response. **Atropine 1%** ophthalmic BID for ciliary spasm and to prevent posterior synechiae. Monitor IOP (uveitis can cause secondary glaucoma). Continue topical therapy until complete resolution of inflammation (typically 4-8

weeks).

Corticosteroid Use

Controversial but can be beneficial. Rationale: FIP is immune-mediated; anti-inflammatory effects reduce vasculitis and clinical signs. **Protocol:** Prednisolone 1-2 mg/kg SID for first 2-4 weeks, then taper rapidly. **Contraindications:** Do not use if concurrent bacterial infection suspected (septic effusions, evidence of pneumonia). Some practitioners avoid steroids entirely, arguing they may impair antiviral efficacy—no definitive data. Use judiciously in neurological cases (reduces inflammation but potentially masks progression).

Treatment Summary: Quick Reference

FIP Type	Initial Dose	Duration	Key Monitoring
Effusive (Wet)	20 mg/kg BID	12-16 weeks	Effusion resolution by Week 4-6
Dry (Non-neuro)	20-25 mg/kg BID	16-20 weeks	AGP normalization, weight gain
Neurological	25-30 mg/kg BID	24-32 weeks	Neurological exam, prolonged therapy
Ocular	25 mg/kg BID + topicals	16-20 weeks	Fundic exam, IOP monitoring
Relapse cases	25 mg/kg BID	20-24 weeks	Slow taper essential

Request Sample Protocol and Consultation

MolnuFIP provides comprehensive support for veterinarians managing FIP cases with EIDD-1931. Our resources include detailed dosing calculators based on body weight and FIP type, case consultation for complex presentations, client education materials explaining treatment rationale and expectations, and ongoing monitoring guidance throughout the treatment course.

To request individualized treatment protocols or discuss case-specific recommendations:

Contact our veterinary support team at hello@molnufip.com or download the full PDF at molnufip.com/vet-resources.

This overview is intended for licensed veterinary professionals. Treatment decisions should be individualized based on patient presentation, diagnostic findings, and clinical judgment. EIDD-1931 for veterinary use is not FDA-approved; use under veterinary professional discretion in accordance with local regulations. Information is based on current understanding of FIP pathophysiology and antiviral mechanisms; treatment protocols continue to evolve as clinical experience accumulates.