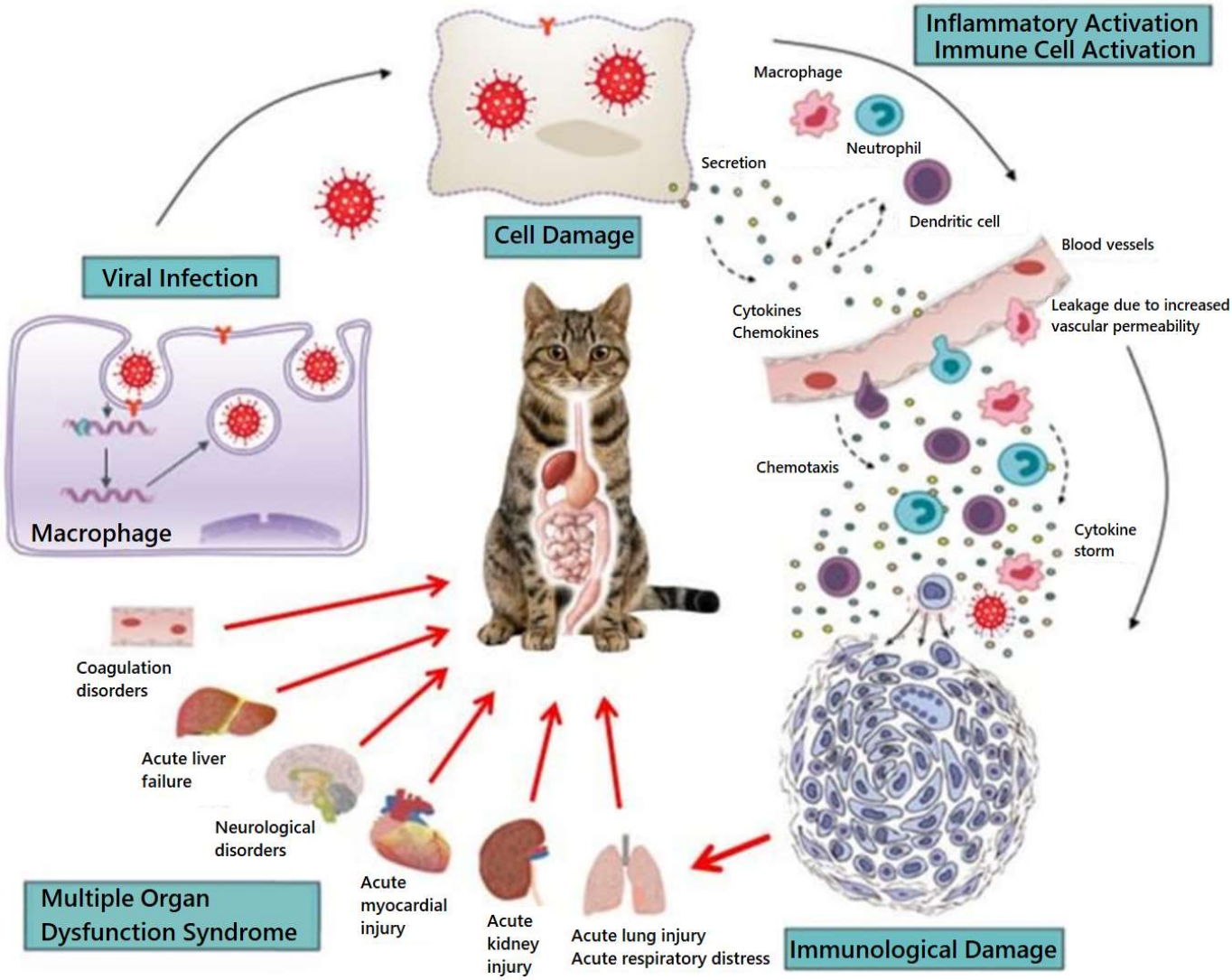
Supportive and Adjunctive Therapy in FIP

Organ–System Comorbidities in FIP

Feline infectious peritonitis (FIP) is not only a viral replication disease, but a multilayered immune-vascular destruction syndrome; therefore, the clinical picture does not always remain limited to a single organ. In FIP, as a result of macrophage-tropic viral replication, cytokine storm, and immune complex accumulation, endothelial dysfunction, increased capillary permeability, microthrombosis, and widespread tissue edema develop. This process creates a broad clinical spectrum, ranging from tachycardia, capillary leakage, and effusions in the cardiovascular system; to cholestasis and hepatocellular damage in the liver; functional perfusion loss in the kidney; enzyme dysregulation in the pancreas; vomiting, diarrhea, and malabsorption in the gastrointestinal system; and meningeal inflammation, neuronal edema, and seizures in the nervous system. Thus, what determines the severity of the disease is often not the viral load, but rather this immunopathological destructive effect affecting multiple organ systems. For this reason, antiviral therapy alone is not sufficient in the treatment of FIP; the correct recognition and timely support of accompanying organ-system comorbidities are critical factors determining both treatment response and survival.



Interpretation of Clinical Presentations and Diagnostic Delay in FIP

Most owners of cats with FIP present to the clinic not with suspicion of FIP, but after observing **symptoms related to pleural/abdominal effusion mimicking primary cardiomyopathy, vomiting and abdominal pain resembling acute pancreatitis, azotemia and polyuria/polydipsia suggestive of chronic kidney failure, or jaundice and elevated liver enzymes with the appearance of cholestatic hepatopathy.** These organ-focused and seemingly specific clinical presentations may direct clinicians toward heart-, pancreas-, kidney-, or liver-centered differential diagnosis and treatment algorithms. However, the underlying process may be immune complex–mediated vasculitis resulting from persistent FCoV replication in macrophages, endothelial dysfunction at the level of the microcirculation, and cytokine-dominant systemic inflammation. **Continuous release of macrophage-derived IL-1β, IL-6, TNF-α, and similar proinflammatory mediators constitutes the main inflammatory axis that leads to progression of clinical deterioration unless antiviral treatment is initiated.** Therefore, transient clinical improvements achieved through organ-specific approaches alone (for example, protocols addressing only heart failure, only pancreatitis, or only acute kidney injury) create misleading windows of improvement, thereby delaying the true diagnosis. Since these temporary improvements do not alter the biological dynamics of the disease, they negatively affect the patient's prognosis. Indeed, large case series and review studies emphasize that, particularly **in the non-effusive form of FIP, no pathognomonic clinical or laboratory finding exists, that most cases follow a course with chronic, nonspecific findings giving the impression of a single-organ disease, and that a definitive diagnosis is often considered only when multi-organ involvement** develops or when the expected response to organ-specific treatments is not achieved (Pedersen, 2014; Kipar & Meli, 2014; Riemer et al., 2016; Tasker, 2018; Felten et al., 2019; Solikhah et al., 2024).

For this reason, the diagnostic approach in FIP requires raising the possibility of FIP at an early stage when unexplained effusion (pleural, peritoneal), fluctuating fever, weight loss, hypoproteinemia, increased acute-phase response, and laboratory abnormalities indicating involvement of multiple organ axes are present in a young or middle-aged cat. **This systemic process centered on infected macrophages cannot be halted by organ-specific treatment alone; unless antiviral therapy is initiated, the disease does not biologically slow down.** This awareness is decisive for the clinician in order to increase diagnostic speed and avoid missing the cat's critical time window. However, the effectiveness of antiviral therapy truly emerges only if organ-system damage is stabilized.

Pathophysiology, Comorbidities, and Treatment Protocols in Feline Infectious Peritonitis

1) Cardiovascular System – Myocardial, Endothelial, and Microvascular Dysfunction

Clinical Condition	Treatment	Dose	Route	Duration	Notes
Endothelial dysfunction, increased capillary permeability	MPFF (Diosmin + Hesperidin)	10–15 mg/kg	PO	2–6 weeks	First-line vascular support agent
Oxidative stress, microthrombosis, cold extremities	N-acetylcysteine (NAC)	50–70 mg/kg	PO	2–4 weeks	Orally safe
Systemic inflammation with increased cardiac workload	Omega-3 fatty acids	30–50 mg/kg	PO	4–12 weeks	EPA-dominant
Pleural, pericardial and/or abdominal effusion	Furosemide	1–2 mg/kg q8–12h	SC / IM / PO	3–10 days	SC/IM administration provides faster onset in acute cases
Refractory effusion	Torsemide	0.1–0.2 mg/kg	PO	5–14 days	Potent diuretic
Increased risk of thrombosis	Clopidogrel	18.75 mg/cat q24h; low-dose maintenance: 5 mg/cat q72h	PO	4–8 weeks	ISCAID-approved

📌 **Note:** In feline infectious peritonitis (FIP), immune complex deposition causes injury to the vascular endothelium, resulting in vasculitis and microthrombosis. During the first 2–3 weeks of antiviral therapy, capillary leakage may occur, leading to tachycardia and subsequent reduced peripheral perfusion. In prolonged or advanced FIP cases, secondary myocarditis, pleural effusion, and reduced venous return may develop. Key diagnostic findings may include mild to moderate elevation of NT-proBNP, mild hypokinetic regions on echocardiography, pericardial effusion, persistent tachycardia, and cold extremities.

2) Vascular Circulation – Microthrombosis, Endothelial Injury, Capillary Leak

Clinical Condition	Treatment	Dose	Route	Duration
Vasculitis, endothelial activation	MPFF (Diosmin + Hesperidin)	10–15 mg/kg	PO	2–6 weeks
Reduced RBC deformability, microthrombosis	N-acetylcysteine (NAC)	50–70 mg/kg	PO	2–4 weeks
Chronic inflammation	Omega-3 fatty acids	30–50 mg/kg	PO	4–12 weeks
Pleural, pericardial and/or abdominal effusion	Furosemide	1–2 mg/kg q8–12h	SC / IM / PO	3–10 days

3) Pancreas – Pancreatitis, Enzymatic Dysregulation

Clinical Condition	Treatment	Dose	Route	Duration
Dehydration / pancreatitis	Lactated Ringer's solution / 0.9% sodium chloride	4–6 ml/kg/h	IV	24–72 hours
Vomiting	Maropitant	1 mg/kg	SC / PO	3–5 days
Severe vomiting	Ondansetron	0.1–0.2 mg/kg q8	IV / PO	3–5 days
Gastrointestinal protection	Sucralfate	0.5–1 g	PO	5–10 days

4) Liver – Elevated ALT/AST, Jaundice, Cholestasis

Clinical Condition	Treatment	Dose	Route	Duration
Hepatic oxidative stress	N-acetylcysteine (NAC)	50–70 mg/kg	PO	2–4 weeks
Hepatocellular stress	SAMe	20 mg/kg	PO	4–6 weeks
Antioxidant therapy	Silymarin	20 mg/kg	PO	4–8 weeks
In the presence of cholestasis	Ursodeoxycholic acid (UDCA)	10–15 mg/kg	PO	4–8 weeks
Elevated ammonia levels	Hepa-Merz (LOLA)	¼–½ sachet/day (50–200 mg/kg)	PO	2–4 weeks
Anti-inflammatory phytochemicals	Polyphenolic combination (curcumin, EGCG, quercetin, resveratrol)	Curcumin 10–15 mg/kg, EGCG 5–10 mg/kg, quercetin 5–10 mg/kg, resveratrol 1–3 mg/kg	PO	4–12 weeks

📌 **Note:** Reduced hepatic perfusion, sinusoidal endothelial injury, and cholestasis may adversely affect the absorption, first-pass metabolism, and conversion to the active form of orally administered antiviral agents.

5) Kidney – Creatinine, BUN, Proteinuria

Clinical Condition	Treatment	Dose	Route	Duration
Dehydration / prerenal azotemia	%0.9 NaCl / LR	3–5 ml/kg/saat	IV	24–72 hours
Proteinuria	Benazepril	0.5 mg/kg	PO	4–6 weeks
Hypertension	Amlodipine	0.1–0.2 mg/kg	PO	Until clinical resolution
Acute hypokalemia	Potassium-supplemented fluids (KCl-added fluids)	20–40 mEq/L	IV	24–72 hours
Mild to moderate hypokalemia	Potassium citrate / potassium gluconate	2–4 mEq/kedi/gün	PO	
Impaired oral antiviral absorption	Ondansetron + maropitant	Ondansetron 0.1–0.2 mg/kg q8–12h + Maropitant 1 mg/kg q24h	PO / SC	3–7 days

📌 **Note:** Reduced renal perfusion may alter the pharmacokinetics of antiviral therapy.

6) Neurological System – Cerebral Edema, Epilepsy, Ataxia, Nystagmus

Clinical Condition	Treatment	Dose	Route	Duration
Cerebral edema	Mannitol	0.5–1 g/kg	IV 20–30 min	1–3 days
Alternative to cerebral edema management	3% hypertonic saline	4 ml/kg	IV	1–3 days
Seizures	Levetiracetam	20 mg/kg q8h	PO	2–4 weeks
Acute seizure	Diazepam	0.2–0.5 mg/kg	IV / PR	Repeat if necessary
Neuroinflammation	Prednisolone	1–2 mg/kg	PO	3–7 days

7) Serosal Cavity Effusions – Peritoneal, Pleural, and Pericardial Effusions

Clinical Condition	Treatment	Dose	Route	Duration
Peritoneal / pleural / pericardial effusion	Furosemide	1 mg/kg q8–12h	SC / IM / PO	3–10 days
Refractory effusion	Torsemide	0.1–0.2 mg/kg q24h	PO	5–14 days
Respiratory compromise (pleural effusion)	Partial thoracocentesis	—	Thoracic	Single administration / as needed
Hemodynamic compromise (pericardial effusion)	Pericardiocentesis	—	Pericardial	Emergency / single administration
Abdominal compression (ascites)	Partial abdominal paracentesis	—	Abdominal	Single administration
Albumin < 2.0 g/dL	Fresh frozen plasma (FFP)	10–15 ml/kg	IV	If necessary

📌 Note:

1. **Complete drainage of peritoneal effusion should NOT be performed.**
   If complete drainage is performed, **acute intravascular volume depletion, hypotension, rebound effusion,** and **significant protein loss** may occur.
2. Diuretics alone are not a definitive solution.
   In FIP, the primary problem is **vascular leakage rather than sodium–water retention.**
   Therefore, **MPFF, NAC, and omega-3 fatty acids must be used as adjunctive therapies.**
3. **Pericardial effusion requires IMMEDIATE pericardiocentesis without delay.**
   If emergency intervention is not performed, **tachycardia, weak pulse, hypotension, and pulsus paradoxus** may develop.

8) Gastrointestinal System – Constipation, Diarrhea, Vomiting, Anorexia

Clinical Condition	Treatment	Dose	Route	Duration
Constipation	Lactulose	0.5 ml/kg	PO	If necessary
Diarrhea	Metronidazole	10–15 mg/kg	PO	5–7 days
Chronic diarrhea	B12	250–500 µg	SC	Weekly × 4
Vomiting	Maropitant	1 mg/kg	SC / PO	3–5 days
Severe vomiting	Ondansetron	0.1–0.2 mg/kg	IV / PO	3–5 days
Gastrointestinal protection	Sucralfate	0.5–1 g	PO	5–10 days
	Famotidine	0.5 mg/kg	PO	5–10 days
Inappetence	Mirtazapine	1.88 mg/kg q48–72	PO	Until appetite returns (usually 3–7 doses)
	Cyproheptadine	1–2 mg/kg	PO	5–10 days

📌 Note:

Famotidine should not be used long-term (tachyphylaxis). Mirtazapine is not a long-term appetite stimulant. Sucralfate has milder effects and can be continued longer if needed, but it may cause sedation; if prolonged, the dose should be reduced.

9) Secondary Diabetes Following Pancreatic Damage

Clinical Condition	Treatment	Dose	Route	Duration
Hyperglycemia	Lantus (glargin)	0.25–0.5 U/kg	SC	Daily (until glucose regulation is achieved)

📌 Note:

Blood glucose should be monitored frequently
Dietary adjustments should be made
If pancreatitis is present, fat intake should be restricted
If corticosteroids are necessary, they should be used only under antiviral therapy and at low doses

10) Uveitis, Retinal Vasculitis, Secondary Ocular Infections

Clinical Condition	Treatment	Dose	Route	Duration
Purulent conjunctivitis, redness	Moxifloxacin 0.5% ophthalmic solution	3–4 times daily	Topical	7–10 days
Purulent conjunctivitis (alternative)	Ofloxacin ophthalmic solution	3 times daily	Topical	7–10 days
Gram-negative–dominant secondary infection	Tobramycin ophthalmic solution	3 times daily	Topical	7–10 days
Anterior uveitis (cornea intact)	Prednisolone acetate 1% ophthalmic suspension	2–4 times daily	Topical	7–14 days
Uveitis + pain / risk of synechiae	Atropine sulfate 1% ophthalmic solution	1 times daily	Topical	3–5 days
Keratitis / corneal defect	Tobramycin ophthalmic ointment	2 times daily	Topical	7–10 days
Keratitis / epithelial damage	Ofloxacin ophthalmic solution	3 times daily	Topical	7–10 days
Dry eye / epithelial support	Artificial tears / ocular lubricant gel	2–4 times daily	Topical	Until clinical recovery
Ocular vasculitis / inflammation	Prednisolone (low-dose systemic therapy)	1–2 mg/kg	PO	3–7 days
Ocular microcirculation disorder	MPFF (diosmin+hesperidin)	10–15 mg/kg	PO	4–8 weeks
Oxidative stress / retinal protection	N-Acetylcysteine (NAC)	50–70 mg/kg	PO	2–4 weeks
Retina / endothelial support	Omega-3 (EPA-dominant)	30–50 mg/kg	PO	8–12 weeks

📌 Note: If there is a corneal ulcer topical steroids must never be used. In ocular FIP, topical treatments are supportive; the main determinants of therapy are systemic antivirals and endothelial stabilization.

11) Other Comorbid Conditions and Secondary Infections Associated with FIP

Clinical Condition	Treatment	Dose	Route	Duration
Upper respiratory tract infection (Chlamydia / Mycoplasma)	Doxycycline	5 mg/kg q12h	PO	7–14 days
Rhinotracheitis with secondary bacterial infection	Amoxicillin–clavulanate (amoxiclav)	20 mg/kg q12h	PO	7–14 days
Pneumonia	Amoxicillin–clavulanate + marbofloxacin	20 mg/kg q12h + 2 mg/kg q24h	PO	14–21 days
Bacterial gastrointestinal infection (diarrhea, bacterial translocation)	Metronidazole	10–15 mg/kg q12h	PO	5–7 days
Mixed gastrointestinal flora infection	Amoxicillin–clavulanate (amoxiclav)	20 mg/kg q12h	PO	7–10 days
Lower urinary tract infection (LUTI)	Amoxicillin–clavulanate (amoxiclav)	20 mg/kg q12h	PO	7–14 days
Complicated urinary tract infection	Marbofloxacin	2 mg/kg q24h	PO	14 days
Suspected sepsis	Amoxicillin–clavulanate + marbofloxacin ± metronidazole	Based on clinical status	IV / PO	14–28 days
Suspected or concurrent toxoplasmosis	Clindamycin	5–11 mg/kg q12h	PO / IV	14–28 days
Oral, dental, and periodontal infection	Clindamycin or amoxicillin–clavulanate	5–11 mg/kg q12h veya 20 mg/kg q12h	PO	7–14 days
Bacterial otitis externa	Amoxicillin–clavulanate (amoxiclav)	20 mg/kg q12h	PO	7–14 days
Skin and soft tissue infection	Cephalexin or amoxicillin–clavulanate	20–30 mg/kg q12h	PO	7–14 days
Concurrent intestinal parasitic infection and suspected Giardia	Fenbendazole ± metronidazole	50 mg/kg + 10 mg/kg	PO	5–7 days

📌 **Note:** In cats with FIP, when the integrity of the tympanic membrane is unknown, ototoxic aminoglycosides should be avoided in the topical treatment of otitis externa; ear drops containing florfenicol or fluoroquinolones are preferred. Metronidazole should be used in FIP only for short durations and in selected cases due to the risk of neurological adverse effects; similarly, marbofloxacin should be administered at low doses and for limited periods, taking into account potential retinal and neurological risks.

12) Pain Management

Clinical Condition	Treatment	Dose	Route	Duration
Visceral / abdominal pain	Hyoscine butylbromide	0.2–0.5 mg/kg	SC / IM	As needed
Ascites-related discomfort	Hyoscine butylbromide + metamizole (dipyrone)	0.1–0.2 ml/kg	SC / IM	1–3 days
Severe pain (case-dependent)	Buprenorphine (opioid analgesic)	0.01–0.02 mg/kg	IM / IV	1–3 days
Musculoskeletal pain (only if clinically necessary)	Meloxicam (NSAID)	0.05 mg/kg	PO	1–2 days max

13) Prednisolone Protocol in Cats with FIP

Prednisolone should be administered only under concurrent antiviral therapy. When used as monotherapy, it may worsen FIP by promoting macrophage-associated viral replication.

Low-dose prednisolone use:

- Reduces NF-κB and IL-6 activity
- Decreases vasculitic burden
- Alleviates cerebral edema and meningeal inflammation
- Restores appetite
- Accelerates reduction of abdominal and pulmonary effusions
- Relieves pain associated with excessive cytokine production

Clinical Condition	Treatment	Dose	Route	Duration
Systemic inflammation (fever, lethargy, vasculitis)	Prednisolone	0.25 mg/kg/day	PO	7–14 days, followed by gradual taper based on clinical response
Vascular inflammation associated with ascites / pleural effusion	Prednisolone	0.5 mg/kg/day	PO	5–10 days, then taper once effusion stabilizes
Severe neuroinflammation (under antiviral therapy)	Prednisolone	0.5 mg/kg/day	PO	10–14 days, then slow taper according to neurological improvement
Neurological FIP (meningoencephalitis, ataxia, nystagmus)	Prednisolone	0,5 mg/kg/day	PO	5–10 days (induction), then stepwise taper over 2–4 weeks
Ocular FIP (uveitis, retinal vasculitis)	Prednisolone	0,5 mg/kg/ day	PO	7–14 days, followed by taper; total duration guided by ocular response
ADE-related flare, rebound inflammation	Prednisolone	0.25–0.5 mg/kg/ day	PO	5–10 days, taper once inflammatory markers and clinical signs regress
Inappetence	Prednisolone	0.25 mg/kg/gün	PO	Short-term only, until appetite improves (typically 3–7 doses), then discontinue

📌 Note: Principles of Administration

- Antiviral therapy is the primary treatment. If corticosteroids are to be used, antiviral therapy must already be initiated (or no more than 1–2 days prior to antiviral therapy) and the goal should be short-term control of inflammation. Duration should be individualized based on clinical response rather than fixed time frames. Steroid tapering should begin as soon as clinical stabilization is achieved.
- Dose and Duration: In neurological cases, 1 mg/kg/day (short-term—usually during the initial days), followed by rapid and gradual tapering as soon as clinical improvement begins. In IMHA, 1–2 mg/kg/day, with gradual tapering once hematological targets are achieved.
- Topical ocular steroids: Prednisolone acetate eye drops may be initiated concurrently with antiviral therapy and tapered slowly; intraocular pressure (IOP) monitoring is mandatory due to the risk of secondary glaucoma.
- If systemic corticosteroids are not required, NSAIDs may be preferred as anti-inflammatory, analgesic, and antipyretic agents in appropriately selected patients (adequate blood pressure, renal perfusion, and appetite).

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