

27 to 25.8%, while dyspnea, abdominal distension, and icterus resolved completely ($p < 0.05$). The
28 mean clinical severity score demonstrated a marked reduction, decreasing from 6.40 ± 1.20 at
29 baseline to 1.55 ± 0.90 at Day 84 ($p < 0.001$), indicating pronounced overall clinical
30 improvement. Hematologic abnormalities characteristic of FIP also improved significantly.
31 Hemoglobin increased from 7.72 ± 1.57 to 9.75 ± 1.18 g/dL, hematocrit from $28.28 \pm 5.91\%$
32 to $36.70 \pm 4.57\%$, and total leukocytes decreased from $13.77 \pm 5.09 \times 10^3/\mu\text{L}$ to 7.31 ± 2.18
33 $\times 10^3/\mu\text{L}$ ($p < 0.05$). Lymphocyte count increased (1.38 ± 0.96 to $2.56 \pm 0.88 \times 10^3/\mu\text{L}$) and
34 neutrophils decreased (10.18 ± 2.90 to $4.02 \pm 1.31 \times 10^3/\mu\text{L}$), lowering the neutrophil-to-
35 lymphocyte ratio from 10.53 ± 6.82 to 1.98 ± 1.46 ($p < 0.001$). Mid-treatment data (Day 42)
36 demonstrated early hematologic recovery.

37 **Conclusion:** These findings demonstrate that remdesivir induces marked and consistent
38 clinical improvement and substantial hematologic normalization in naturally occurring FIP in
39 Iran, providing region-specific evidence supporting its efficacy.

40 **Keywords**

41 Clinical improvement, Feline coronavirus, Hematology, Molecular diagnosis, Remdesivir.

42

43 **1. Introduction**

44 Feline infectious peritonitis (FIP) is a highly fatal, immune-mediated disease that develops
45 following mutations in the widely circulating feline coronavirus (FCoV)[1,2]. While most
46 FCoV infections cause mild or subclinical enteric disease, a subset of viral mutations enhances
47 replication within monocytes and macrophages, leading to the emergence of the virulent feline
48 infectious peritonitis virus (FIPV)[1,3]. This altered cellular tropism allows systemic
49 dissemination and induces pyogranulomatous inflammation, immune dysregulation, vasculitis,

50 and cytokine-mediated tissue injury[3–5]. Consequently, affected cats may develop either the
51 effusive form, characterized by protein-rich serous effusions, or the non-effusive form, in
52 which granulomatous lesions involve multiple organs[3,6]. Without antiviral intervention, FIP
53 is considered almost uniformly fatal[7,8].

54 The clinical manifestations of FIP are diverse and often non-specific, complicating diagnosis
55 and necessitating integration of physical examination findings, hematologic abnormalities, and
56 molecular testing[4,9]. Frequently reported signs include persistent fever, progressive weight
57 loss, anorexia, lethargy, dyspnea, abdominal distension related to effusion, lymphadenopathy,
58 and mucous membrane pallor or discoloration. Neurologic and ocular abnormalities are
59 common in non-effusive disease[4,7]. Hematologic disturbances such as non-regenerative
60 anemia, leukocytosis with neutrophilia and lymphopenia, thrombocytosis or thrombocytopenia
61 are recognized markers that support clinical suspicion and assist in evaluating treatment
62 response[5,6].

63 Advances in molecular diagnostics have markedly improved diagnostic accuracy. Reverse-
64 transcription polymerase chain reaction (RT-PCR) assays targeting conserved regions of the
65 FCoV genome, particularly the spike (S) gene, are now routinely used in clinically compatible
66 cases[4,10]. Although PCR positivity alone cannot distinguish benign enteric biotypes from
67 pathogenic FIPV, detection of viral RNA in blood or effusion substantially increases diagnostic
68 confidence when interpreted within the proper clinical and hematologic context[9,10].

69 The therapeutic landscape of FIP has changed significantly with the introduction of antiviral
70 agents that inhibit viral RNA-dependent RNA polymerase. Remdesivir, an adenosine
71 nucleotide analogue with proven activity against multiple coronaviruses, has shown promising
72 clinical efficacy in naturally occurring FIP[7,8]. Reported outcomes include rapid
73 improvement in systemic signs, resolution of effusion, restoration of appetite and activity, and

74 gradual normalization of hematologic parameters[5,8]. Despite these encouraging findings,
75 detailed prospective evaluations—including structured clinical scoring and serial complete
76 blood count (CBC) monitoring—remain limited, particularly in studies employing
77 standardized treatment protocols[7,8].

78 To date, no prospective investigation from Iran has documented the clinical and hematologic
79 trajectories of PCR-confirmed FIP cases treated with remdesivir. Such evidence is essential for
80 establishing region-specific treatment expectations, guiding therapeutic decisions, and
81 supporting broader clinical adoption. The objective of the present study was to characterize the
82 clinical manifestations and hematologic alterations of 31 PCR-confirmed cats with FIP treated
83 with an 84-day remdesivir protocol. By systematically evaluating key clinical signs and CBC
84 parameters at baseline, mid-treatment, and at the completion of therapy, this study provides a
85 comprehensive description of treatment-associated recovery in naturally occurring FIP.

86

87 **2. Materials and Methods**

88

89 **2.1. Study Design and Animals**

90 This prospective observational study enrolled 31 domestic cats that were examined in privately
91 owned veterinary clinics in Tehran for clinical signs suggestive of feline infectious peritonitis
92 (FIP). Clinical abnormalities prompting evaluation included persistent fever, weight loss,
93 anorexia, lethargy, dyspnea, abdominal distension, dehydration, jaundice (icterus), mucous
94 membrane pallor or discoloration, lymphadenopathy, and neurologic or ocular abnormalities.
95 Enrollment was permitted only after molecular confirmation of feline coronavirus (FCoV)
96 infection using blood-based PCR.

97 All cats were evaluated before initiation of therapy and were monitored for a total of 84 days.
98 Eligibility criteria included: (1) clinical signs compatible with FIP and (2) a positive blood PCR
99 result for FCoV. Cats were excluded if they had received antiviral medications,
100 glucocorticoids, or other immunosuppressive drugs within the preceding 30 days. All
101 diagnostic and therapeutic procedures were performed in accordance with accepted veterinary
102 clinical practice.

103

104 **2.2. Clinical Evaluation**

105 A structured physical examination was performed at three time points: prior to treatment (Day
106 0), mid-treatment (Day 42 ± 2), and completion of therapy (Day 84 ± 3). At each visit, the
107 attending veterinarian recorded body temperature, appetite, demeanor, hydration status,
108 mucous membrane coloration, respiratory pattern, abdominal contour, peripheral lymph node
109 size, and the presence or absence of pallor, dyspnea, effusion-associated abdominal distension,
110 ocular lesions, or neurologic deficits. Baseline history regarding the onset and progression of
111 clinical signs was documented. All examinations were performed by the same veterinarian to
112 minimize inter-observer variability and were conducted using a standardized checklist to
113 ensure consistency and enable paired comparisons across time points.

114

115 **2.3. Clinical Severity Score**

116 A clinical severity score was assigned to each cat on Days 0, 42, and 84 to quantify overall
117 clinical disease burden. The scoring system was adapted from previously published FIP
118 assessment methods [8]. Each abnormal clinical finding—including weight loss, anorexia,
119 lethargy, fever, dyspnea, abdominal distension, pallor, icterus, dehydration, lymphadenopathy,
120 vomiting, and diarrhea—was given a value of 1 if present. The total score represented the sum
121 of abnormalities for each cat, with higher scores indicating more severe clinical involvement.

122

123 2.4. Molecular Confirmation of FCoV Infection

124

125 2.4.1. Sample Collection and RNA Extraction

126 For molecular confirmation, 1–2 mL of peripheral blood was collected from each cat at
127 presentation. RNA extraction was performed using a commercial viral RNA extraction kit
128 (Total RNA Extraction Mini Kit Plus with YTZOL reagent, Yekta Tajhiz Azma, Iran)
129 according to the manufacturer's instructions. Extracted RNA was stored at -20°C until
130 analysis.

131

132 2.4.2. RT-PCR for FCoV Detection

133 RT-PCR was performed using primer sets targeting a conserved region of the FCoV spike (S)
134 gene, following the protocol of Wang et al. (2022) [11]. Primer sequences and amplicon size
135 are presented in Table 1. Amplification conditions included: reverse transcription at 50°C for
136 30 minutes, initial denaturation at 95°C for 10 minutes, followed by 40 cycles of 95°C for 15
137 seconds and 60°C for 60 seconds. Positive and negative controls were included in each PCR
138 run. Samples were considered positive when the amplification curve crossed the threshold
139 before cycle 35. Only cats exhibiting compatible clinical signs and testing positive by RT-
140 PCR were included in the study.

141

142 **Table 1. Primer sequences used for RT-PCR detection of feline coronavirus (FCoV)**

Target gene	Primer name	Sequence (5'→3')	Amplicon size (bp)
Spike (S) gene	FCoV-S-F	AGATCCAGTTGAGGTAGAAGTT	218
	FCoV-S-R	ATCATCTTGTCTGCGTTCTTC	

143

144 **2.5. Hematologic Evaluation**

145 Blood samples were collected from the cephalic vein on Days 0, 42, and 84. At each time point,
146 3–5 mL of blood was obtained, and 1–2 mL was placed into EDTA tubes for complete blood
147 count (CBC) analysis. All samples were analyzed within 2 hours to minimize cellular
148 degradation. CBCs were performed using a veterinary automated hematology analyzer
149 (NIHON KOHDEN Celltac Alpha VET, model MEK-6550K, Japan), which provided red
150 blood cell indices, hemoglobin concentration, hematocrit, total leukocyte count, absolute
151 differential leukocyte counts, platelet counts, and erythrocyte indices and analyzer-specific
152 reference intervals were applied. Wright–Giemsa–stained smears were reviewed when
153 necessary to verify automated differential results.

154

155 **2.6. Treatment Protocol**

156 All cats received remdesivir according to a standardized 84-day regimen adapted from
157 established therapeutic protocols for FIP [8]. The protocol consisted of a 4-day induction phase
158 followed by a maintenance phase. During induction, cats with effusive FIP received 10 mg/kg
159 once daily intravenously or subcutaneously, while cats with non-effusive or neurologic/ocular
160 forms received 15 mg/kg once daily. Beginning on Day 5 and continuing through Day 84, all
161 cats received once-daily subcutaneous injections of remdesivir.

162 Maintenance dosages were adjusted according to clinical form: 8–10 mg/kg for effusive cases,
163 10–12 mg/kg for non-effusive cases, and 12–15 mg/kg for cats exhibiting neurologic or ocular
164 involvement. Injection sites (lateral thorax, flank, dorsal cervical region) were rotated routinely
165 to minimize discomfort and reduce the risk of tissue irritation. All injections were administered
166 slowly, and cats were monitored for adverse reactions including injection-site swelling, pain,
167 lethargy, and gastrointestinal disturbances.

168

169 **2.7. Statistical Analysis**

170 Paired categorical clinical variables (Day 0 vs. Day 84) were analyzed using McNemar’s test.
171 Hematologic parameters were assessed for normality using the Shapiro–Wilk test. Normally
172 distributed variables were compared using paired t-tests; non-normally distributed variables
173 were analyzed using the Wilcoxon signed-rank test. Mean values, standard deviations (SD),
174 and p-values were calculated to assess changes across time points. A p-value < 0.05 was
175 considered statistically significant. Statistical analyses were performed using SPSS (IBM SPSS
176 Statistics, Version 26.0).

177

178 **3. RESULTS**

179 **3.1. Description of the Study Population**

180 Thirty-one domestic cats diagnosed with feline infectious peritonitis (FIP) were enrolled after
181 confirmation of feline coronavirus RNA by blood-based reverse-transcription polymerase
182 chain reaction (RT-PCR). The cats ranged in age from 5 to 24 months, with a median age of
183 13 months, and body weights between 1.9 and 4.4 kg (mean: 3.2 ± 0.7 kg). The study
184 population consisted of 16 males and 15 females. Based on clinical form, 16 cats (51.6%)
185 exhibited the effusive type, 9 cats (29.0%) presented with the non-effusive form, and 6 cats
186 (19.4%) showed ocular or neurologic involvement. All 31 cats successfully completed the
187 standardized 84-day remdesivir protocol and were included in the final clinical and
188 hematologic analyses (Table 2).

189

190 **Table 2. Baseline characteristics of the 31 PCR-confirmed FIP cats**

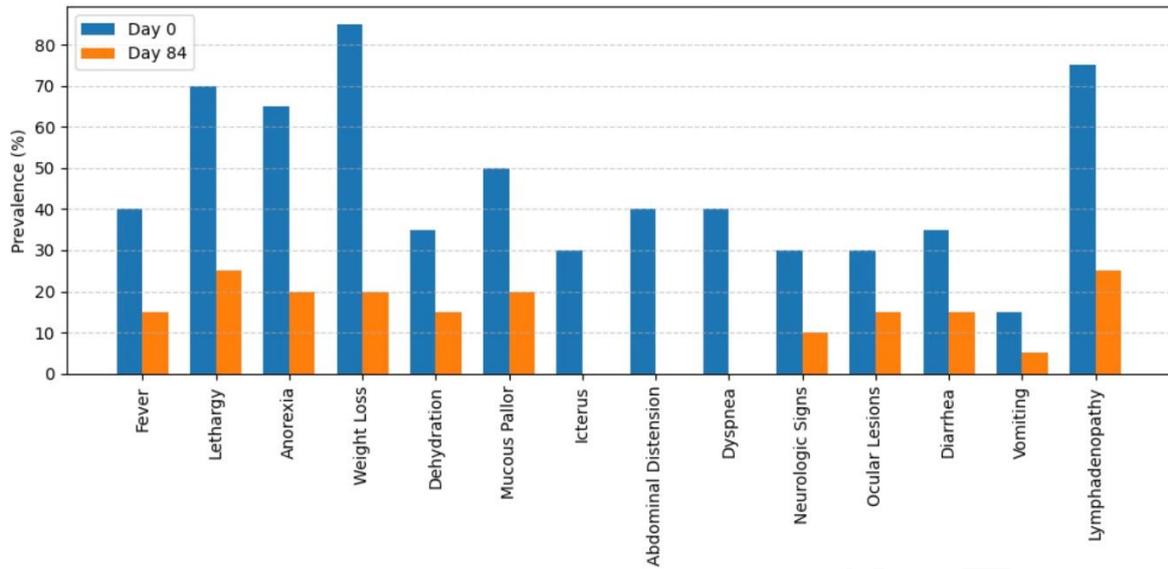
Signalment	Category	Number (n)	Percentage (%)	Range within Group
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Sex	Male	16	51.6%	–
	Female	15	48.4%	–
Age (months)	≤ 12 months	14	45.2%	5 – 12 months
	> 12 months	17	54.8%	13 – 24 months
Body Weight (kg)	≤ 3.0 kg	12	38.7%	1.9 – 3.0 kg
	> 3.0 kg	19	61.3%	3.1 – 4.4 kg
Clinical Form	Effusive	16	51.6%	–
	Non-effusive	9	29.0%	–
	Ocular/Neurologic	6	19.4%	–

191

192 **3.2. Clinical Findings**

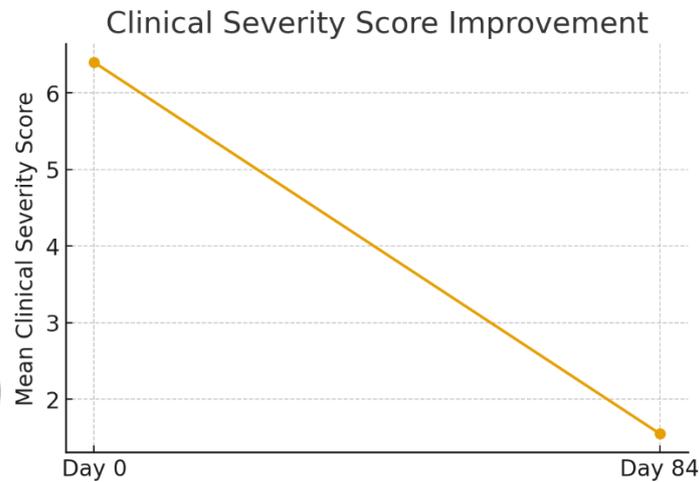
193 At baseline (Day 0), the most frequently observed abnormalities included weight loss (83.9%),
 194 anorexia (64.5%), and lethargy (67.7%). Other commonly noted signs were fever, dyspnea,
 195 abdominal distension, and mucous membrane pallor, each affecting approximately 38.7–48.4%
 196 of the cats. Following completion of the 84-day remdesivir regimen, substantial improvement
 197 was recorded across all parameters. The prevalence of weight loss declined to 19.4%, anorexia
 198 to 6.5%, and lethargy to 25.8%. Fever decreased from 38.7% to 16.1%, while dyspnea and
 199 abdominal distension resolved completely in all affected cats. Mucous membrane pallor also
 200 improved from 48.4% at baseline to 19.4% at Day 84. McNemar’s test confirmed significant
 201 reductions in weight loss, anorexia, lethargy, dyspnea, and abdominal distension ($p < 0.05$). No
 202 new clinical abnormalities developed during the treatment period. The mean clinical severity
 203 score demonstrated a marked reduction, decreasing from 6.40 ± 1.20 at baseline to 1.55 ± 0.90
 204 at Day 84 ($p < 0.001$), indicating pronounced overall clinical improvement (Table 3)(Fig 1 &
 205 2).



206

207 **Figure 1.** Prevalence of major clinical signs at baseline (Day 0) and after treatment (Day 84)
 208 in 31 PCR-confirmed cats with feline infectious peritonitis (FIP). Bars represent mean values
 209 with standard error.

210



211

212 **Figure 2.** Clinical severity score at baseline (Day 0) and after completion of the 84-day
 213 remdesivir protocol (Day 84) in 31 PCR-confirmed cats with feline infectious peritonitis. The
 214 score was calculated as the total number of abnormal clinical signs present in each cat. A
 215 marked reduction in the mean score was observed, indicating substantial clinical improvement.

216

217 **Table 3. Prevalence of principal clinical signs at baseline (Day 0) and after treatment**
 218 **(Day 84) in 31 cats with FIP**

Clinical sign	Day 0 n (%)	Day 84 n (%)	p-value*
Weight loss	26 (83.9%)	6 (19.4%)	<0.001

Anorexia	20 (64.5%)	6 (19.4%)	0.004
Lethargy	21 (67.7%)	8 (25.8%)	0.004
Fever	12 (38.7%)	5 (16.1%)	0.125
Dyspnea	12 (38.7%)	0 (0.0%)	0.008
Abdominal distension	12 (38.7%)	0 (0.0%)	0.008
Pallor	15 (48.4%)	6 (19.4%)	0.031
Icterus	9 (29.0%)	0 (0.0%)	0.031
Dehydration	11 (35.5%)	5 (16.1%)	0.219
Diarrhea	11 (35.5%)	5 (16.1%)	0.344
Vomiting	5 (16.1%)	2 (6.5%)	0.625
Lymphadenopathy	23 (74.2%)	8 (25.8%)	0.006
Clinical Severity Score (mean ± SD)	6.40 ± 1.20	1.55 ± 0.90	<0.001

219

220

221 3.3. Hematologic Results

222 Serial hematologic assessments revealed progressive improvement throughout the 84-day
 223 treatment period. At baseline, red cell indices were reduced, with an RBC count of 5.66 ± 1.18
 224 $\times 10^6/\mu\text{L}$, hemoglobin of 7.72 ± 1.57 g/dL, and hematocrit of $28.28\% \pm 5.91\%$. By Day 84,
 225 these values had increased significantly, reaching $7.55 \pm 1.10 \times 10^6/\mu\text{L}$ for RBC, 9.75 ± 1.18
 226 g/dL for hemoglobin, and $36.70\% \pm 4.57\%$ for hematocrit ($p < 0.001$). Total leukocyte count
 227 demonstrated a marked decline, decreasing from $13.77 \pm 5.09 \times 10^3/\mu\text{L}$ at baseline to $7.31 \pm$
 228 $2.18 \times 10^3/\mu\text{L}$ by Day 84 ($p < 0.05$). (Table 4)

229 Absolute differential leukocyte counts also improved. Neutrophils decreased from 10.18 ± 2.90
 230 $\times 10^3/\mu\text{L}$ at baseline to $4.02 \pm 1.31 \times 10^3/\mu\text{L}$ at Day 84, while lymphocytes increased from 1.38
 231 $\pm 0.96 \times 10^3/\mu\text{L}$ to $2.56 \pm 0.88 \times 10^3/\mu\text{L}$ ($p < 0.001$). These changes produced a substantial
 232 reduction in the neutrophil-to-lymphocyte ratio, falling from 10.53 ± 6.82 at baseline to $1.98 \pm$
 233 1.46 at Day 84. Monocytes and eosinophils showed mild but statistically significant changes
 234 over the treatment period, falling from $0.81 \pm 0.58 \times 10^3/\mu\text{L}$ to $0.21 \pm 0.16 \times 10^3/\mu\text{L}$ and rising
 235 from $0.19 \pm 0.09 \times 10^3/\mu\text{L}$ to $0.49 \pm 0.34 \times 10^3/\mu\text{L}$, respectively ($p < 0.05$). Band neutrophils,
 236 present at low levels at baseline ($0.28 \pm 0.14 \times 10^3/\mu\text{L}$), declined to near zero by Day 84. Platelet
 237 counts decreased from an initially elevated value of $462.74 \pm 229.61 \times 10^3/\mu\text{L}$ to 280.68 ± 73.50
 238 $\times 10^3/\mu\text{L}$ at Day 42 and subsequently stabilized within the normal range at 298.42 ± 78.15
 239 $\times 10^3/\mu\text{L}$ at Day 84 ($p < 0.05$).

240

241 **Table 4. Hematologic parameters as mean \pm SD at Day 0, Day 42, and Day 84 in 31 cats**
 242 **with FIP treated with remdesivir**

Parameter	Day 0 (Mean \pm SD)	Day 42 (Mean \pm SD)	Day 84 (Mean \pm SD)	p-value*
RBC ($10^6/\mu\text{L}$)	5.66 ± 1.18	6.84 ± 1.01	7.55 ± 1.10	<0.001
Hemoglobin (g/dL)	7.72 ± 1.57	9.03 ± 1.06	9.75 ± 1.18	<0.001
Hematocrit (L/L)	28.28 ± 5.91	32.58 ± 3.45	36.70 ± 4.57	<0.001
MCV (fL)	42.12 ± 4.62	43.58 ± 2.58	44.27 ± 1.90	0.044
MCH (pg)	11.31 ± 2.87	15.10 ± 1.02	15.12 ± 0.68	<0.001
MCHC (g/dL)	29.18 ± 2.65	33.28 ± 1.45	33.54 ± 1.01	<0.001
WBC ($10^3/\mu\text{L}$)	13.77 ± 5.09	11.08 ± 4.79	7.31 ± 2.18	<0.001
Neutrophils ($10^3/\mu\text{L}$)	10.18 ± 2.90	6.98 ± 1.96	4.02 ± 1.31	<0.001
Lymphocytes ($10^3/\mu\text{L}$)	1.38 ± 0.96	2.66 ± 0.67	2.56 ± 0.88	<0.001
Monocytes ($10^3/\mu\text{L}$)	0.81 ± 0.58	0.32 ± 0.23	0.21 ± 0.16	0.005

Eosinophils (10³/μL)	0.19 ± 0.09	0.54 ± 0.39	0.49 ± 0.34	0.018
Band cells (10³/μL)	0.28 ± 0.14	0.11 ± 0.06	0.00 ± 0.00	<0.001
Platelets (10³/μL)	462.74 ± 229.61	280.68 ± 73.50	298.42 ± 78.15	0.006
Neutrophil-to-Lymphocyte Ratio	10.53 ± 6.82	2.80 ± 0.97	1.98 ± 1.46	<0.001

243 **Note.** Absolute differential counts are expressed as ×10³ cells/μL. p-values derived from
244 paired t-tests or Wilcoxon signed-rank tests after assessment of distribution normality.

245

246 4. Discussion

247 The objective of this prospective observational study was to characterize the clinical and
248 hematologic responses of 31 PCR-confirmed cats with feline infectious peritonitis (FIP) treated
249 with a standardized 84-day remdesivir protocol. Across all evaluated parameters, the results
250 demonstrated substantial clinical improvement accompanied by marked normalization of key
251 hematologic abnormalities. Core clinical signs—including weight loss, anorexia, lethargy,
252 fever, dyspnea, abdominal distension, mucous membrane pallor, icterus, and dehydration—
253 declined steadily throughout the treatment period, with complete resolution of dyspnea and
254 abdominal distension by Day 84. Hematologic disturbances typically associated with active
255 FIP, such as anemia, leukocytosis with neutrophilia, lymphopenia, and thrombocytosis, also
256 improved significantly by Days 42 and 84. These combined findings indicate a strong
257 therapeutic response and provide evidence supporting the effectiveness of remdesivir in
258 naturally occurring FIP.

259 The clinical outcomes in this cohort are broadly consistent with findings from larger antiviral
260 studies. In a retrospective analysis of 307 cats treated with legally sourced remdesivir or
261 **consistent** 441524, Taylor et al. (2023) reported survival rates approaching 90% [8]. While the
262 present study reported a 100% completion rate among the 31 cats included in the final dataset,

263 this value does not represent the entire initially assessed population. Additional cats were
264 screened at presentation but were excluded due to severe clinical deterioration, loss to follow-
265 up, or death prior to Day 84. Thus, completion data must be interpreted cautiously and
266 considered within the context of this attrition. A major distinction from retrospective
267 investigations is that all cats retained in the present study were required to have blood-based
268 PCR confirmation. Although blood PCR has lower sensitivity than effusion PCR, its high
269 specificity in clinically compatible cases reduces diagnostic heterogeneity and strengthens
270 confidence that the improvements observed reflect genuine FIP treatment responses.

271 Comparable patterns of clinical improvement have also been documented in controlled
272 antiviral trials. Cosaro et al. (2023) demonstrated non-inferiority between oral remdesivir and
273 GS-441524 in 18 cats with effusive FIP, reporting rapid reduction of effusion and resolution
274 of systemic illness[12]. The present study included effusive, non-effusive, and
275 ocular/neurologic cases, yet the magnitude and speed of improvement—particularly the
276 complete disappearance of dyspnea and abdominal distension—parallel the rapid stabilization
277 described in effusive-only cohorts. By incorporating structured hematologic evaluation at fixed
278 time points, the present study expands upon previous findings and provides more detailed
279 insight into the physiological trajectory of recovery during remdesivir therapy.

280 The hematologic trends observed here align closely with findings from GS-441524-based
281 research. Larson et al. (2025) reported increases of approximately 25–30% in hemoglobin and
282 hematocrit during antiviral therapy; the present study demonstrated similar improvements of
283 26% and 32%, respectively[13]. Total leukocyte count decreased by nearly half between
284 baseline and Day 84, mirroring reductions in systemic inflammation noted by Larson et al.
285 (2025)[13]. Tršar et al. (2025) also documented improved neutrophil and lymphocyte
286 distributions following GS-441524 treatment; the current cohort exhibited a comparable

287 transition from neutrophilia toward a balanced leukocyte profile, reflected in a marked
288 reduction in the neutrophil-to-lymphocyte ratio[14].

289 The structured clinical severity score used in this study proved to be a practical tool for
290 quantifying treatment response. The decline in mean score from 6.40 at baseline to 1.55 at Day
291 84 reflects a substantial reduction in overall disease burden. Because this scoring system relies
292 solely on readily accessible clinical observations, it is well suited for general veterinary
293 practice, where advanced diagnostic biomarkers such as alpha-1 acid glycoprotein, serum
294 amyloid A, or cytokine profiling may be unavailable. When combined with serial complete
295 blood count evaluation, this scoring approach provides a comprehensive and practical method
296 for monitoring clinical progression and therapeutic efficacy.

297 Despite these strengths, several limitations must be acknowledged. The sample size, although
298 larger than many prospective FIP studies, remains insufficient for detailed subgroup
299 comparisons among effusive, non-effusive, and neurologic/ocular cases. The absence of a GS-
300 441524 control group limits direct evaluation of comparative antiviral efficacy; however,
301 numerical comparisons with existing literature offer meaningful context. Biochemical
302 parameters and acute-phase proteins were not assessed, restricting evaluation of systemic
303 inflammation to hematologic indices alone. Additionally, all cases originated from a single
304 geographic region, which may reduce generalizability. Finally, follow-up was limited to the
305 duration of the 84-day protocol; longer-term monitoring, such as that performed by Larson et
306 al. (2025), would be necessary to assess relapse risk and long-term remission durability [13].

307

308 **5. Conclusion**

309 This prospective study demonstrates that an 84-day remdesivir regimen produces substantial
310 clinical recovery and marked hematologic normalization in PCR-confirmed feline infectious
311 peritonitis (FIP). Major clinical abnormalities—including respiratory compromise,
312 gastrointestinal signs, and systemic lethargy—improved rapidly, while red cell indices,
313 leukocyte profiles, and platelet values showed marked normalization throughout the treatment
314 period. Notably, this study represents the first prospective clinical evaluation of remdesivir for
315 naturally occurring FIP in Iran, providing novel, region-specific data on its clinical and
316 hematologic effects. The close alignment between these findings and those reported in
317 international antiviral studies further supports the therapeutic efficacy of remdesivir. Based on
318 the clinical and hematologic responses, remdesivir can be recommended as an effective and
319 accessible antiviral option for the treatment of FIP in clinical practice. Remdesivir is currently
320 available in Iran and is manufactured by several domestic pharmaceutical companies.
321 However, to date, no published studies have evaluated its efficacy against indigenous feline
322 coronavirus (FCoV) strains in Iran. The main limitations of this study include a relatively
323 limited sample size, the lack of biochemical and inflammatory biomarker assessment, and
324 restricted long-term post-treatment follow-up. Future investigations should include larger
325 controlled trials, integration of biochemical and inflammatory markers, and molecular
326 characterization of regional FCoV strains. Despite these limitations, the present study provides
327 strong, region-specific evidence that remdesivir represents an effective and accessible antiviral
328 option for managing FIP in clinical practice.

329 **Ethical Considerations**

330 **Compliance with ethical guidelines**

331 The study involved client-owned animals undergoing routine clinical care and no additional
332 experimental procedures were performed. Therefore, formal institutional ethical approval was

333 not required. Informed consent was obtained from all owners prior to inclusion of their animals
334 in the study.

335 **Data availability**

336 All data analyzed during this study are included in this article.

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342 **Authors' contributions**

343 Conceptualization, study design, project administration, and supervision: Seyedeh Missagh
344 Jalali, Mohammad Razi Jalali and Bahman Mosallanejad; Data acquisition: Erfaneh Khavari;
345 Data interpretation and analysis: Seyedeh Missagh Jalali and Erfaneh Khavari; Statistical
346 analysis: Seyedeh Missagh Jalali and Erfaneh Khavari; Writing the original draft: Erfaneh
347 Khavari and Seyedeh Missagh Jalali; Review and editing: Seyedeh Missagh Jalali and
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349 **Conflict of interest**

350 The authors declared no conflict of interest.

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355 **Declaration of Generative AI and AI-assisted technologies in the writing process**

356 The authors declare that they have used AI-based writing assistance tools available in
357 Grammarly (Grammar Check, Clarity, and Writing Enhancement features) to improve the
358 readability, and language of the manuscript.

359

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