Case Study: Acute Medical Pain in a Cat

Introduction

This case report describes a 6-year-old domestic shorthair cat presenting with severe acute abdominal pain secondary to pancreatitis and illustrates the application of a validated feline pain scale, evaluation of hospitalisation stress, contextualised decision-making regarding acupuncture, escalation from intermittent opioid therapy to a fentanyl–ketamine CRI when initial analgesia proved insufficient, and the practical implementation of WSAVA-aligned cat-friendly hospital care. A discussion of pathophysiology, analgesic rationale, hospitalization decisions, and critique of management is provided.

Clinical Report

Merlin was a 6-year-old male, domestic shorthair (European Shorthair) cat presented on an urgent basis for acute vomiting, marked vocalisation, and apparent abdominal pain. According to the owner, Merlin had been normal the previous day. Overnight he had become increasingly restless, intermittently hiding and then emerging to emit sharp cries when picked up or touched. He had vomited several times and had refused food since the morning. There was no reported access to toxins, medications, or foreign bodies, and no history of trauma. Merlin was an indoor cat with no known chronic illnesses and up-to-date core vaccinations.

On presentation, Merlin was quiet but responsive. He showed a crouched, hunched posture on the examination table, with his abdomen tucked and his head slightly lowered. His heart rate was elevated with 160 bpm (considered tachycardic for species and context), mucous membranes were slightly tacky, and skin tenting suggested moderate dehydration (approximately 6–7%). Respiratory rate was mildly increased with 44/min, although he did

not exhibit obvious respiratory distress. A firm, non-fluctuant swelling of the rostral mandible and chin region was noted. Additionally, both mandibular canine teeth showed markedly exposed tooth necks, raising suspicion of concurrent dental disease. At this stage, however, the oral findings appeared unlikely to be the primary cause of Merlin's acute presentation; given the severity of his abdominal pain and systemic signs, the mandibular swelling and dental abnormalities were recorded as relevant but initially considered secondary findings.

Palpation of the cranial abdomen elicited a pronounced pain response: Merlin tensed his abdominal musculature, vocalised sharply, and attempted to escape. He resisted further palpation and showed clear avoidance behaviour. His facial expression was consistent with acute pain: narrowed eyes, flattened ears, and a tense muzzle. Overall, he appeared highly uncomfortable. Pain assessment was performed using the Feline Grimace Scale (FGS), in accordance with WSAVA recommendations for acute pain in cats and with the need to minimise handling in a stressed, painful feline patient. Given Merlin's discomfort and the priority to reduce stress, a handling-based multidimensional scale such as the CMPS-Feline was deliberately not used at this stage. His FGS findings were consistent with severe acute visceral pain and guided early escalation of analgesia. On admission, Merlin showed multiple facial action units consistent with a high Feline Grimace Scale (FGS) score: Laterally positioned ears, marked orbital tightening, tense muzzle, horizontally oriented whiskers, and a lowered head posture. When scored according to the validated FGS criteria, these findings corresponded to an estimated FGS score of 6–7/10, placing him clearly in the severe acute pain range. This score, combined with the clinical findings of tense abdominal guarding, vocalisation on palpation, and marked postural changes, supported the classification of his condition as a severe acute visceral pain state requiring prompt and aggressive analgesia.

Given the acute onset of abdominal pain and vomiting, a diagnostic workup was initiated. Blood samples were collected for complete blood count, serum biochemistry, and feline pancreatic lipase immunoreactivity (fPLI). An abdominal ultrasound examination was performed. A mild stress leukogram was present, characterised by neutrophilia, consistent with acute abdominal pain and stress. Serum biochemistry showed a mildly increased creatinine, most consistent with pre-renal azotaemia due to dehydration. Total protein was mildly elevated, with globulins slightly above reference range, also compatible with dehydration and systemic inflammation. The feline pancreatic lipase (fPLI) concentration was markedly increased, strongly supporting a diagnosis of acute pancreatitis. Abdominal ultrasonography demonstrated a mildly enlarged, heterogeneous pancreas, with hyperechogenic peripancreatic fat consistent with peripancreatic inflammation. No evidence of foreign body obstruction, biliary rupture, or other acute surgical pathology was identified. Collectively, the findings were consistent with acute pancreatitis and severe associated abdominal pain. Given the severity of Merlin's visceral abdominal pain, his moderate dehydration, and the need for injectable medications and close monitoring, hospitalisation was deemed medically necessary. He was admitted for IV fluid therapy, analgesia, further diagnostics, and repeated pain assessments. Merlin was placed in a quiet ward away from canine patients. His cage was set up with a cardboard hiding box, soft bedding, dim lighting, and minimal stimuli. Handling was kept as low-frequency as possible and clustered so that catheter checks, pain scoring, and nursing procedures occurred together rather than intermittently.

An intravenous catheter was placed, and Merlin was started on isotonic crystalloid fluid therapy (Ringer's lactate) to correct dehydration, support pancreatic perfusion, and maintain

electrolyte balance. The rate was adjusted based on perfusion parameters, urine output, and reassessment of intravascular volume. Maropitant (Cerenia) was administered as an antiemetic and anti-nausea agent to support comfort and the early return to feeding.

For analgesia, buprenorphine was selected as the first-line opioid, consistent with WSAVA acute pain recommendations for medically induced visceral pain. Merlin received buprenorphine at 0.04 mg/kg IV to provide an adequate starting dose for severe visceral pain. Pain was reassessed four hours later using the Feline Grimace Scale (FGS), which showed no meaningful improvement in facial expression, posture, guarding, or interaction. Although a second dose of buprenorphine could have been administered at this time, the complete lack of response to the initial dose indicated that repeating the same opioid was unlikely to provide adequate analgesia. Given the severity of Merlin's visceral abdominal pain, prompt escalation was required, and a fentanyl–ketamine continuous rate infusion (CRI) was initiated to achieve continuous and titratable analgesia. Both selected CRI doses (fentanyl 0,003 mg/kg/hr and ketamine 0,005 mg/kg/min) fall within current recommendations for severe visceral abdominal pain in cats.

A structured Feline Grimace Scale (FGS) reassessment was performed one hour after the CRI was initiated. Merlin showed a clear clinical improvement: His facial expression relaxed (ears more neutral, reduced orbital tightening, relaxed muzzle), his posture became less hunched, abdominal guarding was markedly decreased, he changed position more willingly within the cage. His FGS score decreased from the severe range to moderate pain.

By the evening of the first hospital day, Merlin voluntarily accepted small amounts of food, which is a key indicator of improved comfort in cats with pancreatitis. Grooming behaviour

also returned, further supporting that analgesia was finally adequate. The CRIs were continued overnight with ongoing supportive care and environmental optimisation.

Merlin tolerated the hospital environment well. He remained alert, settled between examinations, and did not show persistent fear behaviours. He accepted brief, calm interaction without distress. However, during longer periods of sustained handling- such as those required for procedures - he showed mild tension and signs of stress.

Although acupuncture was considered as part of a multimodal pain plan, it was withheld in this acute phase. Merlin showed subtle stress indicators during prolonged handling, and avoiding additional procedures was prioritised to maintain a calm, low-arousal environment.

Clinical Outcome

On the following morning, Merlin's visceral abdominal pain remained improved and stable. His abdomen could be palpated without eliciting guarding, and his Feline Grimace Scale (FGS) score remained within the moderate range. However, at this time he displayed marked and unexpected sensitivity to palpation of the chin and rostral mandible. This focal pain had likely been less apparent the previous day due to the dominance of his severe visceral abdominal pain, and it became clinically evident only after adequate abdominal analgesia had been achieved. The firm swelling noted over the rostral mandible during initial examination had not appeared to be the primary issue at that time, but the newly pronounced focal pain prompted further evaluation. Given the combination of persistent facial tension despite well-controlled abdominal pain, pronounced chin sensitivity, and the presence of a mandibular swelling, diagnostic imaging was indicated. Merlin was sedated for dental radiographs, which

revealed an aggressive osteolytic lesion of the rostral mandible consistent with neoplastic disease. Following review of the radiographic findings and discussion of the prognosis, the owners elected humane euthanasia. Merlin remained under sedation, and euthanasia was performed in this sedated state with the owners present.

Discussion & Critique

Acute pain in small animals is defined as pain of sudden onset in response to a specific injury, surgical procedure, or medical condition. It is typically of short duration and resolving as healing occurs[1]. The WSAVA Pain Guidelines describe medical pain as pain arising from non-surgical, non-traumatic conditions such as pancreatitis, acute kidney injury, pneumonia/ pleuritis, gastrointestinal distension, urinary obstruction, ischemia, or acute enlargement of solid organs[2]. These conditions often produce visceral pain, which is typically diffuse, difficult to localise, and severe due to distension, inflammation, organ capsule stretch, and activation of visceral nociceptors[3].

Acute pancreatitis in cats results in significant abdominal visceral pain through inflammatory mediator release, pancreatic edema, peripancreatic fat necrosis, and activation of visceral afferents[4]. In addition, visceral pain pathways readily trigger early central sensitisation if inadequately managed[3]. For this reason, WSAVA emphasises that analgesics should be provided early, even before a definitive diagnosis is confirmed, whenever medical pain is strongly suspected[2]. This principle guided the initial approach in this case, where early analgesia was prioritised alongside diagnostics.

Pain recognition in cats is uniquely challenging. Physiological variables such as heart rate, respiratory rate, temperature, and blood pressure are not reliable indicators of pain, as they are strongly influenced by stress, fear, anxiety, and environment[6]. Neuroendocrine biomarkers (beta-endorphins, catecholamines, cortisol) correlate with pain experimentally, but are not clinically practical[7]. Accurate feline pain assessment therefore depends on behavioural evaluation, focusing on posture, facial expression, activity, interaction, appetite, and response to palpation[8]. Following WSAVA recommendations, validated tools such as the Feline Grimace Scale (FGS)[9], the Glasgow Composite Measure Pain Scale–Feline (CMPS-Feline)[10], and multidimensional scales such as UFEPS-SF[11] are recommended. FGS is particularly valuable when minimal handling is required, whereas multidimensional composite scales require interaction and are not always feasible in fearful or unsocialised cats[9]. In this case, the FGS was selected because it permits evaluation with minimal handling - an important consideration in a painful, stressed feline patient. Merlin's FGS score of 6–7/10 clearly reflected severe acute visceral pain and justified the decision to initiate opioid analgesia promptly and to escalate treatment when buprenorphine alone proved insufficient.

Environmental stress is a major modulator of feline pain. Fear, noise, unfamiliar smells, bright lighting, lack of hiding options, and excessive handling can increase pain perception, suppress normal behaviours (e.g. grooming, eating), and reduce the effectiveness of analgesics[2]. WSAVA and ISFM Cat Friendly Clinic guidelines therefore encourage environmental optimisation - pheromones, hiding boxes, soft bedding, dim lighting, and clustered handling - to reduce stress and improve analgesic outcomes[12]. Although hospitalisation inherently exposes cats to unfamiliar stimuli, it was medically necessary in this case due to severe visceral pain, dehydration, vomiting, and the need for IV fluids,

injectable medications, and continuous analgesia. All feasible cat-friendly modifications were implemented, and Merlin adapted well, settling between examinations and showing no persistent fear behaviours. Because prolonged physical contact modestly increased stress, acupuncture - despite its potential benefit for visceral discomfort[2] - was withheld to avoid exacerbating stress and interfering with analgesic progress.

The analgesic decision-making in this case reflects the importance of timely escalation when first-line therapy is inadequate. Buprenorphine at 0.04 mg/kg IV was an appropriate initial opioid for severe visceral abdominal pain according to WSAVA recommendations[2]. After four hours, FGS reassessment demonstrated no meaningful improvement. Although a second dose of buprenorphine could have been administered at this time, the complete lack of response to the initial dose indicated that repeating the same opioid was unlikely to provide sufficient analgesia. To avoid prolonging severe visceral pain and to prevent ongoing nociceptive input from driving central sensitisation, escalation to a fentanyl–ketamine continuous rate infusion was selected as the most appropriate next step. The chosen doses - fentanyl at 0,003 mg/kg/hr and ketamine at 0,005 mg/kg/min - fall within contemporary recommendations for severe visceral pain in cats[2,13]. Fentanyl provides rapid and potent μ-opioid analgesia, while low-dose ketamine contributes NMDA-receptor antagonism, reduces wind-up, and enhances multimodal analgesia without inducing anaesthesia. Merlin’s rapid improvement - relaxed facial expression, reduced guarding, return of grooming, and voluntary food intake - validated this escalation strategy.

A notable aspect of this case is the emergence of a second, previously masked source of pain. Once visceral abdominal pain was controlled, Merlin exhibited marked focal sensitivity of the rostral mandible. Radiographs revealed an aggressive osteolytic lesion of the mandible

consistent with neoplastic disease. Although earlier sedation for oral imaging might theoretically have revealed the lesion sooner, it would have required prolonged handling and was not feasible during the initial period of severe abdominal pain and physiological instability. Furthermore, the lesion's aggressive nature made it highly unlikely that earlier recognition would have altered the ultimate prognosis.

A further reflection point concerns the initiation of the fentanyl–ketamine CRI without a preceding loading dose. In some cases, a small fentanyl bolus (0,002-0,005 mg/kg IV) or a low-dose ketamine bolus (0.2-0.5 mg/kg IV) can accelerate onset of analgesia[2]. However, bolus administration - particularly of μ-opioids or ketamine - carries a higher risk of dysphoria, agitation, or stress-induced amplification of pain in sensitive feline patients. Given Merlin's marked stress on presentation and the presence of buprenorphine already occupying μ-receptors, initiating the CRI without a loading dose was a reasonable and defensible choice. Nevertheless, a carefully titrated loading dose remains a valid alternative approach for future cases, depending on the individual cat's stress susceptibility, cardiovascular status, and behavioural tolerance.

Overall, while the management of this case aligned closely with contemporary evidence-based recommendations for feline medical pain, it illustrates key principles of feline pain medicine: the necessity of structured and repeated pain assessment, the impact of environmental stressors, the importance of timely analgesic escalation, the risk of masked concurrent pain sources, and the value of contextualised, patient-centred decision-making. These reflections reinforce a multimodal approach that integrates behavioural assessment, environmental optimisation, multimodal pharmacology, and ongoing clinical reassessment.

Summary

This case report describes a 6-year-old male domestic shorthair cat presenting with acute vomiting, vocalisation, and severe abdominal pain. On admission, Merlin showed a crouched posture, abdominal guarding, tachycardia, moderate dehydration, and a high Feline Grimace Scale (FGS) score consistent with severe acute visceral pain. Laboratory testing revealed markedly increased feline pancreatic lipase, and ultrasound findings supported a diagnosis of acute pancreatitis. Hospitalisation was required for IV fluid therapy, antiemetics, analgesia, and repeated pain assessments. A cat-friendly environment was implemented to minimise stress. Initial opioid analgesia with buprenorphine produced insufficient improvement on reassessment. Because pain remained severe, analgesia was escalated to a guideline-aligned fentanyl–ketamine CRI, resulting in rapid and clinically meaningful improvement, including reduced guarding, improved facial expression, grooming, and voluntary food intake. On the following day pronounced focal pain of the rostral mandible became evident. Sedated dental radiography revealed an aggressive osteolytic mandibular lesion consistent with neoplastic disease. In light of the grave prognosis, euthanasia was performed under sedation. This case highlights key principles of feline acute pain management: structured pain scoring, early analgesic escalation, stress reduction during hospitalisation, and the importance of repeated reassessment to identify concurrent pain sources. Areas for improvement include considering a fentanyl loading dose to accelerate onset of CRI analgesia, performing earlier sedation for oral imaging once visceral pain was stabilised, and increasing the frequency of structured pain reassessments during the initial hours of hospitalisation.

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