Case Study: Chronic Musculoskeletal Pain in a Dog

Introduction

This case report describes an 11-year-old Golden Retriever with advanced DLSS and Cauda Equina Syndrome presenting with severe chronic maladaptive pain, restlessness, impaired mobility, and declining quality of life. A multimodal analgesic plan was implemented, incorporating NSAIDs, amantadine, paracetamol, acupuncture, manual therapy, lifestyle adaptation, and environmental modification. The discussion reflects on the pain mechanisms involved and the practical challenges of managing long-standing pain in a geriatric, neurologically impaired dog.

Clinical Report

Sally, an 11-year-old, 29-kg spayed female Golden Retriever, was presented in May for evaluation of severe chronic pain and marked deterioration in mobility and daily functioning. According to the owners, the dog had become increasingly restless over several weeks, pacing extensively at night, panting, and showing inability to settle or rest. Sleep had become severely fragmented, with constant repositioning and wandering. The owners, both elderly and physically limited, felt overwhelmed and had already scheduled euthanasia for the following day due to the perceived severity of Sally's distress.

Sally had a history of hypothyroidism, managed effectively with levothyroxine, and cognitive decline, treated with propentofylline (Karsivan). Three years earlier, she had undergone bilateral gold bead implantation around the hip joints for hip dysplasia, which had resulted in only temporary improvement. Over the previous months, her mobility and comfort had steadily worsened.

Radiographs revealed advanced, multi-level lumbar degeneration with severe spondylosis, vertebral bridging, and chronic intervertebral disc disease at several levels (most notably L3–4 and L4–5). Additional marked degeneration at L7–S1 was consistent with lumbosacral stenosis and explained the patient's clinical cauda equina signs. The findings represented a disseminated, chronic degenerative process affecting multiple lumbar regions. At the time of initial presentation, Sally was not receiving any analgesic medications. NSAIDs had previously been administered intermittently but without consistent use. Gabapentin had been tried at another clinic but discontinued from the owners after two days because of severe ataxia; the medical record indicated an initial dosing of 10 mg/kg twice daily, which likely contributed significantly to the adverse effects.

On physical examination, Sally exhibited a markedly short-strided gait, with pronounced stiffness and evident difficulty rising from recumbency. Her pelvic limbs intermittently buckled during ambulation, consistent with lower motor neuron involvement. Palpation of the lumbosacral region elicited a strong pain response, particularly with tail-lift testing. There was pronounced paraspinal hypertonicity between L3 and S3 and focal hyperesthesia on palpation over the lumbosacral junction.

Neurologic evaluation identified moderately reduced proprioception in both pelvic limbs, significantly reduced patellar reflexes, and mildly reduced cranial tibial reflexes. Superficial and deep nociception remained intact. These findings were consistent with chronic nerve root compression and lower motor neuron deficits of the L4–L6 segments.

Pain scoring was performed using the Canine Osteoarthritis Staging Tool (COAST), the Liverpool Osteoarthritis in Dogs questionnaire (LOAD), and a Health-Related Quality of Life

(HRQoL) assessment. At presentation, Sally's COAST score corresponded to Stage 4, indicating severe functional impairment with marked difficulty rising, pronounced gait abnormalities, frequent hindlimb collapsing, and loss of endurance. The LOAD score was in the high-severity range, reflecting substantial mobility restriction, exercise intolerance, and reduced willingness to move. HRQoL assessment revealed severe compromise across multiple domains, including sleep disruption, restlessness, reduced comfort, and markedly diminished engagement with her environment. These tools were used consistently at each evaluation and were essential in monitoring her response to therapy, guiding clinical decision-making, and communicating disease progression to the owners.

Collectively, the findings supported the presence of advanced chronic nociceptive pain, neuropathic pain from nerve root compression, and significant central sensitisation.

Given the severity of Sally's pain, initiation of NSAID therapy was indicated, in line with international osteoarthritis and chronic pain guidelines, which recommend NSAIDs as first-line pharmacologic treatment. Because the owners had a full supply of robenacoxib at home and were strongly motivated to use it, NSAID therapy was re-initiated at 2 mg/kg once daily with robenacoxib to provide rapid anti-inflammatory coverage and ensure adherence. Although alternative NSAIDs might have been preferred based on clinician preference and pharmacokinetic profile, timely initiation of NSAID therapy was essential, and robenacoxib offered a practical and immediately available option within a broader multimodal plan. The option of continuing disease-modifying monoclonal antibody therapy with bedinvetmab (Librela) was discussed; however, the owners had previously observed no meaningful improvement after two injections and were strongly opposed to further use. Their reluctance was respected, and alternative multimodal strategies were pursued. Amantadine, at 4 mg/kg

once daily, was included to target NMDA receptor–mediated central sensitisation and chronic wind-up mechanisms. Paracetamol (10 mg/kg twice daily) was added as a centrally acting analgesic to enhance multimodal pain control. Although dosing every 8–12 hours is possible, a twice-daily schedule was initially selected to ensure tolerability and to preserve room for later escalation if required. Propentofylline was continued to support cognitive function and arousal in the context of concurrent cognitive decline.

Acupuncture point selection was based on a full TCVM assessment, including tongue and pulse evaluation. Sally's pattern was consistent with a chronic Bi-Syndrome with underlying Kidney Jing deficiency and mild Shen disturbance associated with cognitive dysfunction. Points were therefore selected to address spinal pain, support Kidney Jing, and calm the Shen. In the first treatment session, BL20, BL23, BL60 and Bai-Hui were used to modulate lumbosacral discomfort, nourish Kidney energy, and reduce paraspinal tension. Myofascial release techniques and treatment of the paraspinal trigger points were applied concurrently. Sally relaxed visibly during treatment and showed improved comfort over the following 48 hours.

During the second session one week later, electroacupuncture was introduced to enhance segmental neuromodulation. GB34 was added as an influential point for tendons and ligaments, addressing compensatory strain in the hindlimbs due to chronic LS pain. ST36 and KI3 were included to support Qi and Blood, strengthen the hindlimbs, and address underlying Kidney deficiency. Sally again tolerated treatment well and showed improved ease rising and settling at night.

Approximately two weeks after the second treatment, a third acupuncture session was performed. At this visit, palpation again revealed focal hyperesthesia over the L4–S3 region, prompting a segmental electroacupuncture approach. Back-Shu points corresponding to these segments (BL25, BL26, BL27, BL28) were selected to modulate nerve root irritation, reduce myofascial spasm, and strengthen local Qi and Blood flow. GB30 was added to address gluteal tension and hindlimb weakness. Improvement in comfort and mobility was noted for more than 10 days following this session.

Two weeks after the third session, a fourth booster treatment was performed to maintain neuromodulatory effect and support ongoing comfort during daily activity.

Pain scoring using COAST, LOAD, and HRQoL assessments was performed at every visit and used to guide ongoing decisions regarding the treatment plan. Detailed changes in these scores are summarised in the Clinical Outcome section.

As part of the initial management plan, the home environment was modified to reduce slipping, improve stability, and support safe mobility. The owners placed non-slip rugs throughout frequently used areas of the house, and Sally was provided with anti-slip socks, which significantly improved her confidence and traction on hard floors. She lived in a ground-floor apartment without stairs, eliminating the need for climbing or descent and reducing risk of falls. More advanced mobility aids, such as a Help-’em-Up harness, were discussed, but the owners were physically unable to use such equipment safely and consistently due to their own mobility limitations. Within these constraints, the implemented environmental modifications were realistic, safe, and immediately beneficial.

Clinical Outcome

Sally showed substantial improvement within the first two weeks of treatment. According to the owners, the previously relentless nighttime pacing ceased almost entirely, and she was able to sleep through the night, typically requiring only one brief outdoor trip. Continuous panting diminished markedly. Her demeanor became calmer and more content, and she appeared more engaged with her surroundings. Although neurologic deficits persisted, she walked more willingly and at a slightly brisker pace during her short daily walks. The owners described her as “like having our dog back again.”

Follow-up evaluations demonstrated corresponding improvement in objective pain and quality-of-life scores. COAST improved from Stage 4 to Stage 3, LOAD scores showed a clear reduction in severity, and HRQoL shifted from severely impaired to moderately impaired, particularly reflecting improved sleep and reduced restlessness. These changes indicated meaningful and sustained improvement in comfort and daily function.

Sally remained stable for several months without requiring modifications to her analgesic regimen. The owners preferred not to change NSAIDs or retry gabapentin, and given her consistent comfort, the established multimodal protocol was continued.

In October, during a period when the primary clinician was unavailable, Sally experienced a marked decline. Her neurologic deficits progressed, making rising increasingly difficult, and she began exhibiting episodes of disorientation consistent with worsening cognitive decline. Although pain remained reasonably controlled, her functional deterioration exceeded the caregiving capacity of her elderly owners. After discussion of her overall prognosis and

quality of life, humane euthanasia was elected. The procedure was carried out by a colleague, with the owners present.

Discussion & Critique

Degenerative lumbosacral stenosis (DLSS) produces a complex and multifaceted pain profile involving nociceptive, neuropathic, and central sensitisation mechanisms[1]. Chronic cauda equina compression likely contributed to ectopic nerve root activity through demyelination and altered ion-channel expression[2], reinforcing the rationale for including amantadine to target NMDA-mediated sensitisation[3].

The multimodal analgesic plan addressed these layered mechanisms of chronic maladaptive pain. Although NSAIDs represent first-line therapy for osteoarthritis and chronic pain syndromes, Robenacoxib was not the pharmacologic first choice for a patient with advanced DLSS and mixed nociceptive–neuropathic pain. NSAIDs such as meloxicam or carprofen may offer more predictable long-term tissue effects[4]. However, the owners had a full supply of Robenacoxib at home, were strongly inclined to continue its use, and timely initiation of NSAID therapy was essential. Continuing Robenacoxib therefore reflected a pragmatic balance between ideal pharmacologic choice and real-world adherence, consistent with contextualised care principles. Similarly, although re-initiation of bedinvetmab (Librela) could have been considered, the owners perceived no benefit from previous doses and bedinvetmab was therefore not re-initiated.

Amantadine was included to target NMDA-mediated central sensitisation and chronic wind-up, mechanisms strongly suspected given the chronicity and severity of Sally's discomfort[3].

Paracetamol was added as a centrally acting analgesic[5], initially administered twice daily to ensure tolerability and preserve the option for later escalation to an every-8-hour schedule if required. This staged dosing strategy balanced safety, efficacy, and owner compliance. While adjunctive use of pregabalin or a carefully titrated retrial of gabapentin might have provided additional neuropathic pain control, owner reluctance following prior adverse effects represented a valid limiting factor and was respected in the treatment plan.

Acupuncture and electroacupuncture were incorporated based on both TCVM pattern diagnosis and western evidence for neuromodulation and endogenous analgesia. Sally's TCVM findings were consistent with a chronic Bi-Syndrome underpinned by Kidney Jing deficiency and mild Shen disturbance associated with cognitive decline. Acupuncture points-including BL23, ST36, KI3, Bai-Hui, GB34, GB30, and segmental Back-Shu points (BL25–BL28) - were selected to modulate nociceptive processing, reduce paraspinal hypertonicity, support Kidney Jing, and enhance hindlimb strength[6]. Electroacupuncture facilitated segmental neuromodulation, particularly over the caudal lumbar and lumbosacral regions[7]. Manual therapy further assisted in relieving myofascial tension and improving mobility. Lifestyle adaptations, especially short and frequent walks, helped prevent fatigue, reduce post-exercise flare-ups, and support functional stability.

Serial pain assessment using COAST[8], LOAD[9], and HRQoL[10] tools proved essential throughout the treatment course. These repeatable, validated instruments align with WSAVA Global Pain Council recommendations[4] for monitoring chronic pain and its impact on daily functioning. In this case, owner-reported metrics were particularly valuable in assessing parameters such as sleep quality, restlessness, engagement, and emotional wellbeing - domains not reliably captured during brief clinic examinations.

Advanced imaging with MRI would have yielded more precise characterisation of the degree of nerve root compression and soft-tissue involvement. According to principles emphasised in the WSAVA Global Pain Guidelines[4], diagnostic decisions should be individualised and consider patient comorbidities, caregiver capacity, expected therapeutic impact, and overall welfare. In this case, declining MRI was medically sound: general anaesthesia in a geriatric dog with cognitive decline carried a heightened risk of postoperative confusion and dysphoria, and MRI findings would not have altered the therapeutic plan.

Several aspects of the case present opportunities for reflection. Incorporation of an additional validated pain instrument such as the Canine Brief Pain Inventory (CBPI) might have yielded more granular longitudinal data[11], though it likely would not have altered clinical decision-making. Additional cognitive-supportive medication was considered but not pursued, as the dog's cognitive decline seemed to be rather multifactorial (pain-related, neurologic, and age-related) rather than a primary canine cognitive dysfunction syndrome. No pharmacologic cognitive therapy was expected to meaningfully alter the trajectory of decline. Environmental optimisation - non-slip flooring, anti-slip socks, and a stair-free layout - was effective in improving safety and mobility. More advanced mobility aids, such as a Help-'em-Up harness, could theoretically have further supported functional mobility, but the owners' own physical limitations made consistent use of these measures unrealistic. This reflects a core principle of contextualised decision-making: treatment plans must align not only with the patient's medical needs but also with caregiver capacity[4]. The adaptations that were feasible provided meaningful benefit without imposing unsustainable demands on the owners.

Overall, this case illustrates the complexity of chronic maladaptive pain in a geriatric dog with DLSS and cognitive dysfunction. It highlights the importance of a multimodal, context-

responsive treatment plan supported by serial pain assessment, owner partnership, and balanced clinical judgement.

Summary

This case describes an 11-year-old Golden Retriever with severe chronic pain due to advanced degenerative lumbosacral stenosis and Cauda Equina Syndrome, compounded by central sensitization, myofascial tension, and concurrent cognitive dysfunction. At presentation, the dog exhibited significant restlessness, sleep disruption, and impaired mobility. Pain assessment using COAST, LOAD, and HRQoL was performed consistently and aligned with WSAVA guidelines. A multimodal analgesic plan incorporating NSAIDs, amantadine, paracetamol, acupuncture, electroacupuncture, manual therapy, and supportive lifestyle modifications resulted in substantial improvement within two weeks, restoring sleep, reducing pacing and panting, and improving engagement and mobility. Sally's eventual decline was predominantly driven by progressive neurologic deficits and worsening cognitive decline that ultimately exceeded caregiver capacity, leading to humane euthanasia. This case highlights the effectiveness of multimodal analgesia and integrative therapies in managing chronic maladaptive pain and illustrates the importance of structured owner-reported outcome measures. Areas for improvement include earlier neuropathic analgesic trials, enhanced caregiver support, and closer follow-up during periods of decline.

References

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