LONG-TERM MULTIMODAL CONTROL OF SPINAL PAIN AND EARLY SIGNS OF SENILE DEMENTIA WITH MICRO-PEA, LOW-DOSE GABAPENTIN AND PHYSIOTHERAPY. A CASE REPORT IN A GERIATRIC DOG

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Introduction

Chronic pain may increase dementia risk and accelerate cognitive decline [1]. Palmitoylethanolamide (PEA) is a safe pro-homeostatic lipid mediator with pain-relieving and neuroprotective properties [2-5]. Micro-PEA (i.e., ultra- or co-micronised PEA) offers superior oral absorption and efficacy over other formulations [6], making it a valuable dietary aid for pain relief and neurocognitive support [4-6]. This case report presents the long-term clinical course of a geriatric dog with chronic pain and early signs of cognitive decline, managed through a multimodal protocol including micro-PEA supplementation, low-dose painkillers, and physical therapies.

Methods

A 14-year-old, 25.6 kg (BCS: 3/5), spayed female Lupo Italiano dog was referred for progressive hind limb weakness over the past 6 months, with slowed gait, difficulty climbing stairs and rising (mainly in the morning), and fecal incontinence. The neurologic exam revealed ataxia, paraparesis, diminished reflexes (flexor, patellar, cranial tibial, perianal). Pain on thoracolumbar palpation and positive lordosis test were recorded. Biomechanical evaluation showed hypotonia and muscle atrophy (paraspinal, pelvic limb). Low-held tail, hind limb abduction during stance and gait, short-strided gait with dragging, forward weight shifting and compensatory shoulder muscle hypertonia were also noted. Findings were consistent with thoracolumbar disc degeneration and lumbosacral stenosis, with suspected degenerative myelopathy. The owner declined further diagnostics, opting for a conservative treatment plan. Since NSAIDs had been ineffective, and gabapentin at standard initial dose (10 mg/kg BID) caused adverse effects (diarrhea and lethargy), a complementary feed with PEA-q (PEA co-ultramicronised with quercetin, 300+60 mg SID) was added to low-dose gabapentin (4 mg/kg BID). Biweekly physiotherapy sessions also begun, combining pain management (dry needling, myofascial release, laser therapy) with underwater treadmill sessions to support motor function.

Results

Six months after starting the treatment, the dog's motor function remained stable, with no signs of spinal pain, despite the progressive nature of the neurodegenerative condition. However, early signs of cognitive decline were reported, including nighttime restlessness and increased episodes of fecal incontinence. A complementary feed for brain aging containing ultramicronised-PEA and phosphatidylserine (300+75 mg SID) was therefore added to the therapeutic plan. Twelve months later, the owner reported improved cognitive responsiveness and motor confidence, with a return

to normal defecation. Maintenance physiotherapy was reduced to monthly sessions, oral therapies continued, and the clinical condition remained stable.

Conclusions

This case illustrates how a multimodal conservative treatment combining micro-PEA, tailored physiotherapy, and low-dose pharmacological support can effectively control chronic pain and mitigate discomfort in a geriatric dog. It also supported cognitive well-being, providing a safe and sustainable option for long-term care in frail, aging patients affected by degenerative diseases.

References

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