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Adjuvant cannabidiol in the management of canine cutaneous squamous cell carcinoma: a long-term survival case and translational review

Abstract

Cutaneous squamous cell carcinoma (SCC) is a common epithelial malignancy in dogs and shares many molecular and pathological features with human cutaneous SCC. Standard management relies on surgical excision and radiotherapy; however, recurrence remains frequent when margins are narrow or anatomic constraints limit complete tumor removal. Cannabidiol (CBD), a non-psychoactive phytocannabinoid, exhibits anti-inflammatory, anti-proliferative, pro-apoptotic, and anti-angiogenic activity in several cancer models. This manuscript presents a clinical case of canine SCC treated with adjuvant oral CBD following marginal surgical excision and integrates a translational literature review. The patient received full-spectrum 1% CBD (>0.3% THC) during the day and 3% CBD (<0.3% THC) at night for 60 days. No early recurrence was observed, biochemical parameters remained within normal limits, and the dog remains alive 14 months after diagnosis. Mechanistic evidence from preclinical and comparative oncology studies suggests that CBD modulates apoptosis, autophagy, angiogenesis, and immune pathways relevant to SCC progression. These findings support further investigation of CBD as an adjunct therapy in veterinary oncology.

Keywords: cannabidiol, inhibition, oncology, radiation

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Introduction

Cutaneous squamous cell carcinoma (SCC) accounts for approximately 20–30% of canine epithelial skin tumors and is strongly correlated with chronic ultraviolet exposure, sustained inflammation, and cumulative DNA damage. Comparable mechanisms operate in human cutaneous SCC, positioning canine patients as valuable translational models. Despite improvements in veterinary surgical oncology, local recurrence remains a significant clinical challenge—particularly when complete excision is limited by tumor size, anatomical location, or owner preference regarding adjuvant radiation therapy. These challenges highlight the need for adjunctive therapies that may modulate oncogenic pathways and reduce relapse risk. The endocannabinoid system (ECS) regulates cutaneous homeostasis and inflammation. Cannabidiol (CBD), though only weakly interacting with CB1 and CB2 receptors, modulates a broad range of molecular targets, including TRPV1, PPAR-γ, adenosine signaling, and FAAH inhibition. Through these pathways, CBD exerts anti-inflammatory, anti-oxidative, anti-proliferative, pro-apoptotic, and anti-angiogenic effects. Preclinical research demonstrates reduced keratinocyte proliferation, apoptosis induction, DNA damage, and angiogenesis suppression.[1,2,5] This manuscript integrates a clinical case report with a translational review of mechanistic CBD research, providing insights for veterinary and comparative oncology.

Case description

A 10-year-old female mixed-breed dog presented with a 10-cm ulcerated, keratinized abdominal mass exhibiting raised, erythematous, irregular borders. Histopathology confirmed a well-differentiated squamous cell carcinoma characterized by keratin pearls, atypical polygonal cells, and stromal invasion. Surgical excision was performed; however, margins were narrow due to anatomical constraints. Radiation therapy was declined by the guardian, who elected adjuvant CBD therapy based on emerging evidence of anti-inflammatory and anti-tumor properties.

CBD Treatment Protocol

I. Daytime: 1% full-spectrum CBD oil (>0.3% THC)
II. Nighttime: 3% CBD oil (<0.3% THC)
III. Dose: 5 mg/kg/day, orally, twice daily
IV. Duration: 60 consecutive days post-surgery

Clinical examinations and serum biochemistry (ALT, ALP, urea, creatinine) were assessed at baseline, day 30, and day 60.

Results

The dog demonstrated normal wound healing, progressive reduction of erythema, and no signs of early recurrence throughout the 60-day monitoring period. Appetite, behavior, and activity levels remained stable. Serum ALT, ALP, urea, and creatinine remained within reference intervals. As of the most recent follow-up, the patient has survived 14 months post-diagnosis with no visible recurrence, suggesting a favorable clinical course.

Discussion

Mechanistic basis for CBD as an adjunct therapy

Apoptosis and Autophagy

CBD induces mitochondrial dysfunction, ROS formation, and caspase-mediated apoptosis.[2] In canine neoplastic cell lines, CBD activates ERK and JNK, triggering autophagy followed by apoptosis.[4]

Anti-proliferative pathways

CBD modulates TRPV1, PPAR-γ, and MAPK pathways, contributing to cell cycle arrest and reduced proliferation.[2,4]

Anti-angiogenic effects

CBD suppresses angiogenesis in vitro and in vivo, reducing endothelial migration, tube formation, VEGF, MMP-2, MMP-9, and uPA.[5]

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DNA damage and cell-cycle arrest

In human oral SCC (HSC-3) cells, CBD induces γ-H2AX DNA damage and G0/G1 cell-cycle arrest, consistent with tumor-suppressive activity.[1]

Immune modulation

CBD enhances anti-tumor immunity, increasing T-cell and NK-cell infiltration and activating MAPK pathways in HPV-associated squamous cell carcinoma models.[3]

Additionally, CBD reduces IL-6 and TNF-α and promotes pro-resolving lipid mediator production.[6]

Endocannabinoid system modulation

CBD's inhibition of FAAH increases endogenous endocannabinoids, augmenting anti-proliferative and immunomodulatory effects.[2]

Comparative oncology perspective

CBD inhibits proliferation and migration and induces apoptosis across multiple human cancer cell types.[2]

In canine neoplastic cells, CBD reduces proliferation and acts synergistically with chemotherapeutic agents.[4]

CBD also modulates PI3K/AKT/mTOR signaling, sensitizing tumor cells to chemotherapy 6

Collectively, these findings highlight CBD's potential as an adjunct to veterinary SCC management.

Limitations

I. The report describes a single case, limiting generalizability.

II. No imaging (CT/MRI) or molecular tumor profiling was available for longitudinal comparison.

III. CBD formulations vary widely between producers, potentially affecting reproducibility.

IV. Translational relevance remains limited by the scarcity of controlled in vivo veterinary cancer trials.

Conclusion

Adjuvant CBD therapy was well tolerated following marginal excision of canine cutaneous SCC and may have contributed to favorable wound healing, reduced inflammation, and absence of early recurrence. Mechanistic evidence strongly supports CBD's potential anti-tumor effects via apoptosis induction, proliferation reduction, angiogenesis inhibition, and immune modulation. The dog's 14-month survival without clinical recurrence further supports CBD's promise as an integrative adjunctive therapy. Controlled clinical trials in veterinary oncology are warranted.

Acknowledgments

None.

Conflicts of interest

None.

References

1. Khurana SK, Sehrawat A, Tiwari R, et al. Bovine brucellosis - a comprehensive review. *Vet Q*. 2021;41(1):61–88.
2. Shakir R. Brucellosis. *J Neurol Sci*. 2021;420:117280.
3. Wyatt HV. Lessons from the history of brucellosis. *Rev Sci Tech*. 2013;32(1):17–25.
4. Wang XH, Jiang H. Global prevalence of human brucellosis. *Zhonghua Liu Xing Bing Xue Za Zhi*. 2020;41(10):1717–1722.
5. Franco MP, Mulder M, Gilman RH, et al. Human brucellosis. *Lancet Infect Dis*. 2007;7(12):775–786.
6. Ulu-Kilic A, Metan G, Alp E. Clinical presentations and diagnosis of brucellosis. *Recent Pat Antiinfect Drug Discov*. 2013;8(1):34–41.
7. Bouomrani S, Belgacem N, Ben Hamad M, et al. Bilateral Panuveitis Revealing Acute Septicemic Brucellosis. *EC Ophthalmology*. 2018;9.6:437–441.
8. Bouomrani S, Dey M, Ahmed A. Acute Renal Failure Complicating Septicemic Brucellosis. *J Pathol Infect Dis*. 2019;2(2):1–2.
9. Bouomrani S, Mrad H, Ben Teber S. Cutaneous leuckocytoclastic vasculitis revealing acute brucellosis. *American Journal of Medical Case Reports*. 2021;9(6):335–338.
10. Mrad H, Besleï W, Mahdhaoui W, et al. Isolated Acute Pericarditis Revealing Brucellosis. *AJMCR*. 2021;9(12):675–677.
11. Trabelsi S, Khfifi S, Ben Amor R, et al. Isolated subacromial-deltoid bursitis as first manifestation of Brucellosis. *MOJ Orthop Rheumatol*. 2022;14(4):97–99.
12. Shoaei SD, Bidi N. Serologic evaluation of brucellosis in patients with psychiatric disorders. *Caspian J Intern Med*. 2012;3(4):557–558.
13. Pourmontaseri H, Rismani M, Karami B, et al. A rare case report of neuro-brucellosis with concurrence of depression, visual impairment, bilateral sensorineural hearing loss, and paraplegia. *PLoS Negl Trop Dis*. 2025;19(7):e0012824.
14. *Ince LU, Kavuran NA, Ozcan A, et al. Severe major depression: A case of neurobrucellosis. Med Science*. 2017;6(1):178–179.
15. Naderi H, Sheybani F, Parsa A, et al. Neurobrucellosis: report of 54 cases. *Trop Med Health*. 2022;50(1):77.
16. Raina S, Sharma A, Sharma R, et al. Neurobrucellosis: A Case Report from Himachal Pradesh, India, and Review of the Literature. *Case Rep Infect Dis*. 2016;2016:2019535.
17. Eini P, Keramat F, Hasanzadehhoseinabadi M. Epidemiologic, clinical and laboratory findings of patients with brucellosis in Hamadan, west of Iran. *J Res Health Sci*. 2012;12(2):105–108.
18. Feki I, Abbes W, Sakka S, et al. A psychiatric disorder reveals a neurobrucellosis. *Presse Med*. 2016;45(12 Pt 1):1197–1200.
19. Kanjo MA, Ahmed HM, Alnahari EA. Unusual presentation of neurobrucellosis in Jeddah, Kingdom of Saudi Arabia. *Neurosciences (Riyadh)*. 2021;26(4):385–388.
20. Obuaya CC, Gangatharan GT, Karra E. Brucella-Induced Acute Psychosis: A Novel Cause of Acute Psychosis. *Case Rep Infect Dis*. 2021;2021:6649717.
21. Gul HC, Erdem H, Bek S. Overview of neurobrucellosis: a pooled analysis of 187 cases. *Int J Infect Dis*. 2009;13(6):e339–43.
22. Tekin-Koruk S, Duygu F, Gursoy B, et al. A rare case of seronegative neurobrucellosis. *Ann Saudi Med*. 2010;30(5):412–414.
23. Moogahi S, Rostami H, Salmanzadeh S, et al. Undiagnosed Brucellosis in Psychiatric Patients: A Cross-Sectional Study. *Arch Clin Infect Dis*. 2023;18(2):e136729.
24. Eren S, Bayam G, Ergönül O, et al. Cognitive and emotional changes in neurobrucellosis. *J Infect*. 2006;53(3):184–189.
25. Ates MA, Algül A, Geçc Ö, et al. Acute psychosis due to neurobrucellosis: a case report. *Anatolian J psychiatry*. 2008;9(3):188–190.