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THE ROLE OF INFLAMMATION IN THE PATHOPHYSIOLOGY OF CRANIAL CRUCIATE LIGAMENT RUPTURE IN DOGS - A REVIEW



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Abstract: *Cranial cruciate ligament rupture in canine patients is driven by early synovial inflammation and immune responses that weaken the ligament prior to clinical rupture. The resulting mechanical instability fuels a secondary inflammatory cascade leading to osteoarthritis, meniscus degradation, pain sensitisation, and muscle atrophy, ultimately deteriorating quality of life.*

• Introduction

Cranial cruciate ligament (CCL) rupture in dogs is a chronic, degenerative pathology driven by early synovial inflammation and immune responses that degrade collagen before clinical rupture. Subsequent joint instability triggers secondary meniscal tears, subchondral bone remodeling, and accelerated osteoarthritis. Associated pain sensitization and muscle atrophy impair proprioception, neuromuscular control, and overall quality of life.

• Material and method

A comprehensive literature search was conducted across major biomedical databases (PubMed, ScienceDirect, MDPI, AVMA, Wiley, Thieme, BMJ) and veterinary textbooks.

• Results and discussions

The canine stifle is critical for propulsion and load distribution. Disruption of the extra-synovial CCL exposes hidden collagen self-antigens, triggering humoral and cell-mediated immune responses. Pro-inflammatory cytokines (IL-1, IL-6, TNF-α, IL-8, KC, MCP-1) and proteolytic enzymes (cathepsins, MMPs) shift collagen turnover toward matrix degradation.

Synovial biomarkers (IL-8, KC, MCP-1, lubricin, acrolein) rise prior to clinical instability, marking active synovitis and oxidative stress. Subsequent joint instability and chronic inflammation accelerate secondary osteoarthritis, meniscal damage, and subchondral bone remodeling. Pain-induced disuse atrophy and loss of joint proprioception ultimately compromise motor control and quality of life.

• Conclusions

- CCL rupture is primarily a chronic degenerative and inflammatory condition rather than purely traumatic, driven by altered cellular metabolism.
- High MMP concentrations catalyze collagen weakening prior to macroscopic rupture, while pro-inflammatory conditions impair fibroblast collagen synthesis, perpetuating tissue degradation.
- Mononuclear cell infiltration in degenerated ligaments points to an ongoing immune response.
- Mapping synovial cytokine profiles enables early diagnosis and targeted therapies to limit osteoarthritis before severe instability occurs.

References
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