CORTICOSTEROID-INDUCED LAMINITIS: FACT OR FICTION?

Empirical evidence suggests that corticosteroid administration can trigger laminitis. Although the incidence is very low, it has been a matter of debate in equine veterinary medicine for many years. Experimental studies have not been able to show the dose or dose regimen required to reliably induce laminitis. Moreover, the exact mechanism of cause and effect between corticosteroids and laminitis has not been elucidated, and could involve the induction of insulin resistance, vascular dysfunction, and/or interference with keratinocyte proliferation, differentiation and matrix integrity. Single or multiple cases of iatrogenic laminitis have been reported after intramuscular, intra-articular or oral administration of various products including triamcinolone, dexamethasone, methylprednisolone and betamethasone, with a majority occurring after multiple or large doses and particularly with the more potent formulations like triamcinolone. As laminitis is commonly associated with endocrine disease, horses with (subclinical) predisposing factors for laminitis (e.g. EMS, PPID), or with a previous history of laminitis may be more sensitive to exogenous steroids, while normal horses seem to have very low risk. Intriguingly, many horses tolerate multiple or higher doses, whereas other horses develop problems despite receiving very small doses. The reported time of onset of laminitis has been reported to vary from 0 (onset during course of treatment) to more than 3 weeks days after cessation of corticosteroid treatment. Most frequently, all four feet were affected, and it has been associated with a guarded prognosis.

References
6. McGowan C., Cooper D., Ireland J. (2016). No evidence that therapeutic systemic corticosteroid administration is associated with laminitis in adult horses without underlying endocrine or severe systemic disease. RCVS Veterinary Evidence online, doi: 10.18849/ve.v1i1.12.g19