CALCINOSIS CUTIS AND IATROGENIC HYPERADRENOCORTICISM ASSOCIATED WITH TOPICAL STEROID TREATMENT IN A DOG

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INTRODUCTION
Calcinitis cutis is an uncommon disorder in which inorganic insoluble mineral salts are deposited in the dermis, subcutis or epidermis. It is most commonly seen in dogs with iatrogenic or endogenous hyperadrenocorticism (HAC). In the authors’ knowledge, this is the first case reported of calcinitis cutis caused by iatrogenic HAC due to chronic steroid topical treatment in a dog.

CLINICAL CASE
8 years old male entire French Bulldog presented for a 24 months history of non-pruritic ulcerative cutaneous lesions of the right elbow treated with a topical local treatment based on fluocinolone acetonide, neomycin and gramicidin (Midacina®) twice a day. During the treatment period, the patient started to show a progressive bilateral alopecia and thin skin. The previous serum biochemistry profile revealed an increased alkaline phosphatase, urine specific gravity of 1.022 and normal total thyroxine. The owners had stopped the topical treatment two weeks before first consultation.

TREATMENT
Due to the abrupt withdrawal of topical glucocorticoids and risk of iatrogenic hyperadrenocorticism, prednisone was initiated (0.3 mg/kg SID) with a reduction plan of 25% every week for 6 weeks. Dimethyl sulfoxide (DMSO) was prescribed to reduce mineralization (applied at the lesion SID) and fusidic acid 2% (applied at the ulcerative lesion BID).

FOLLOW-UP
After 10 days of the initiation of the oral prednisone, the hair started to grow and the calcinitis cutis lesions decreased in size. Two weeks after the prednisone was stopped, basal cortisol was 3 µg/dl and post-stimulation with ACTH 4.9 µg/dl. Total calcium was within normal limits.

DISCUSSION
There has been significant variability in the reported prevalence of calcinitis cutis in dogs with HAC, ranging from 1.7 to 40%. In our case, topical glucocorticoid medication induced generalized skin lesions, predominantly involving lateral trunk and underarm areas, rather than mild and localized lesions as seen in previous studies. Although it was not possible to perform a skin biopsy to confirm the diagnosis, the features and consistency of the papulonodular lesions were highly consistent with calcinitis cutis. The lack of skin biopsy is a limitation on this clinical case.

It remains unclear whether high doses of corticosteroids increase the risk of mineral deposition in the skin. It is thought that excess of cortisol alter the structure of proteins of collagen and elastin fibers, predisposing them to calcification. Calcinitis cutis has been also reported secondary to infections (blastomycosis, leptospirosis or paecilomycosis) and percutaneous absorption or subcutaneous injections of calcium containing solutions.

Adrenocorticotrophic hormone (ACTH) release is easily suppressed by exogenous corticosteroids due to the negative feedback mechanism of the hypothalamic-pituitary-adrenocortical (HPA) axis. The ACTH stimulation test has been proven to be a sensitive indicator of adrenocortical suppression. Tests results showed HPA axis suppression due to prolonged use of topical glucocorticoids. Due to the abrupt withdrawal of topical treatment and ACTH stimulation test results, the patient was at risk for iatrogenic adrenal insufficiency. Although no signs of glucocorticoid deficiency were observed, prednisone supplementation was given.

It has been reported that the resolution of dermatologic lesions may take up to 6 months or even longer in some cases. Studies have shown that adjunctive topical treatment with DMSO gel once a day may help to reduce calcinitis cutis lesions. During DMSO therapy, serum calcium levels should be monitored periodically since hypercalcemia is a potential adverse effect of the treatment. In this case, total calcium was within normal range after 6 weeks with DMSO therapy.

CONCLUSION
This is the first case reported of calcinitis cutis caused by iatrogenic HAC due to chronic steroid topical treatment in a dog. Withdrawal of topical steroid treatment, in addition with the administration of local DMSO and fusidic acid plus a slow reduction of oral prednisone resulted in a gradual recovery of HPA axis improvement and improvement of cutaneous lesions.