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

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INTRODUCTION

Calcinosis cutis is an uncommon disorder in which inorganic insoluble mineral salts are deposited in the dermis, subcutis or epidermis.[1] It is most commonly seen in dogs with iatrogenic or endogenous hyperadrenocorticism (HAC).[1,2] In the authors' knowledge, this is the first case reported of calcinosis 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 topic 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.

DERMATOLOGICAL EXAM

- Non pruritic symmetric alopecia of the trunk
- Papular and nodular dermatosis on the right side (Fig. 1 and 3)
- Fibrotic lesion on the right elbow
- Severe atrophy of abdominal zone (Fig. 2)
- Epidermal collarettes

Papulonodular dermatitis was clinically compatible with calcinosis cutis.

DIAGNOSTIC TESTS

- Wood's lamp examination: Negative
- Skin scraping and microscopic examination of plucked hairs: negative.
- Cytology: Neutrophilic inflammation with intracellular cocci.
- Basal cortisol: < 0.5 µg/dl and a cortisol post-stimulation with tetracosactide hexa-acetate (Synacthen Depot®) of 1,6 µg/dl.

TREATMENT

Due to the abrupt withdrawal of topical glucocorticoids and risk of iatrogenic hypoadrenocorticism, 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 calcinosis 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.

Fig .1 Papulonodular dermatitis on the right side

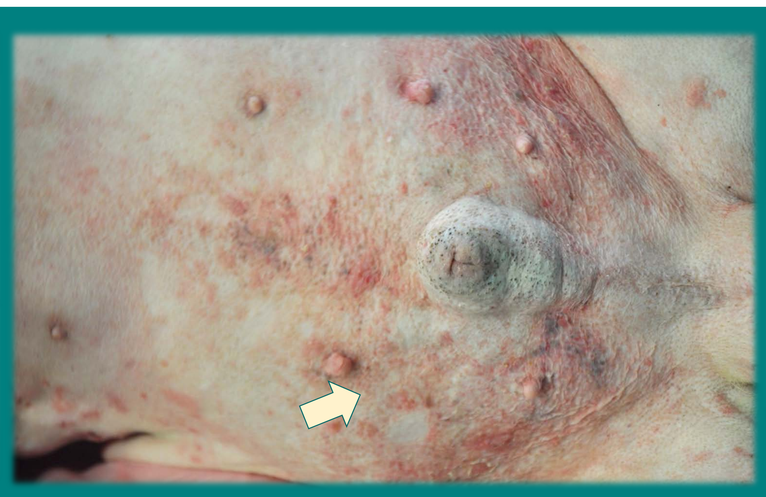

Fig. 2 Abdominal atrophy

Fig. 3 Papulonodular and ulcerative lesion

DISCUSSION

There has been significant variability in the reported prevalence of calcinosis cutis in dogs with HAC, ranging from 1.7 to 40%.[3] 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.[4] Although it was not possible to perform a skin biopsy to confirm the diagnosis, the features and consistence of the papulonodular lesions were highly consistent with calcinosis 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.[1] It is thought that excess of cortisol alter the structure of proteins of collagen and elastin fibers, predisposing them to calcification.[1] Calcinosis cutis has been also reported secondary to infections (blastomycosis, leptospirosis or paecilomycosis) and percutaneous absorption or subcutaneous injections of calcium containing solutions.[5-8]

Adrenocorticotropic 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.[4] 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.[9] Studies have shown that adjunctive topical treatment with DMSO gel once a day may help to reduce calcinosis cutis lesions.[10] During DMSO therapy, serum calcium levels should be monitored periodically since hypercalcemia is a potential adverse effect of the treatment.[10] In this case, total calcium was within normal range after 6 weeks with DMSO therapy.

CONCLUSION

This is the first case reported of calcinosis 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 and improvement of cutaneous lesions.

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