ABSTRACT

This case report describes a practical approach for diagnosing and treating a 17-month-old Syrian hamster with hyperadrenocorticism based on: history, systemic signs, dermatological lesions, and therapeutic trial. The patient was monitored for 16 weeks while he was treated with trilostane and achieved hair regrowth and the resolution of systemic and demeanour signs.

Keywords: alopecia; Cushing disease; hamster; hyperadrenocorticism; trilostane

INTRODUCTION

Hyperadrenocorticism (HAC), also known as Cushing disease, is a disease of the endocrine system resulting primarily from chronic excess production of glucocorticoids, particularly cortisol. Cortisol is an important hormone that plays an essential role in the regulation of the metabolism of carbohydrates, proteins and lipids. Of all pet animals, Cushing syndrome with similar clinical signs is most common in dogs (mostly in older patients), in cats it is rarely present [2, 4, 9].

Hyperadrenocorticism is rarely diagnosed in domestic hamsters in a clinical practice [7, 13], however, in laboratory hamsters the disease is found in higher numbers [1]. Primary HAC due to neoplasia of adrenal cortex is most common in males and older animals, with adrenocortical adenoma being one of the most commonly reported benign neoplasms in the Syrian hamster [12]. Secondary HAC can also develop due to pituitary neoplasia causing excess adrenocorticotropic hormone (ACTH) secretion [1]. Iatrogenic HAC may also occur when the patient receives excess glucocorticoid treatment [11]. All three types of HAC lead to hypercortisolemia and show similar systemic signs such as polydipsia (PD) and polyuria (PU), polyphagia and cutaneous lesions such as bilateral symmetrical flank alopecia and lateral thigh area, hyperpigmentation, skin thinning, lack of skin elasticity and comedones [5, 10, 11].

CASE PRESENTATION

Signalment and history

A 17-month-old male Syrian hamster (Mesocricetus auratus) was presented for a second opinion due to five months of non-pruritic progressive alopecia. The patient was previously treated with topical ivermectin, systemic
dexamethasone (5 months prior to the second opinion and a month after the clinical signs appeared) and systemic enrofloxacin, which did not improve the clinical signs. The owner also described signs of PD and PU, changes in demeanour by hiding in his bedding, lethargy, weight loss and a strong urine odour of the bedding despite being replaced frequently.

CASE MANAGEMENT

General physical examination

The patient appeared bright, alert and responsive. The body weight was 0.150 kg which appeared to be slightly below the normal body condition. The mucous membranes were pink and moist. On abdominal palpation the patient was found to be comfortable and no masses were palpated.

Dermatological examination

There was bilateral, non-symmetrical, multifocal, regional alopecia on the flanks and lumbosacral area and diffuse hypotrichosis on the ventral abdomen and medial hind limbs (Figure 1). The remaining hair was not easily epilated. Additionally, there was dermal thinning and an area of comedones.

Differential diagnosis

Given the history, the clinical signs and the dermatological lesions, top on the list of differential diagnoses was hyperadrenocorticism (HAC). Other conditions were also considered including parasitic (demodicosis, sarcoptic mange, notoedrosis), bacterial (staphylococcal folliculitis), fungal (dermatophytosis), systemic (nephrosclerosis, amyloidosis), endocrinopathies (diabetes mellitus, hypothyroidism), neoplastic (epitheliotropic lymphoma), nutritional (pantothenic acid deficiency), and hypersensitivity (allergic or contact dermatitis) disorders, however these were not highly suggestive given the whole clinical presentation.

TREATMENT AND OUTCOME

Diagnostic approach

The author and the owner agreed to proceed with a therapeutic trial as the remaining differential diagnoses were unlikely and there was a high index of suspicion for HAC.

Treatment

Week 1: Trilostane (Trilostane 10 mg.ml⁻¹; Summit) at a dose of 2.5 mg.kg⁻¹ once daily (sid) per os (p.o.) was prescribed. For the urine odour and the PU, the patient was prescribed enrofloxacin (Baytril 2.5% oral solution; Elanco) 5 mg.kg⁻¹ twice daily (bid) p.o. for seven days as a urinary tract infection (UTI) was suspected.
Follow up

Week 2: the patient was reviewed and some of the previously described clinical signs improved. The urine odour was resolved, the PD and PU improved. The patient’s body weight increased 20 g during the two-week treatment period. The patient also showed signs of demeanour improvement where he was more active and playful. Lastly, new hair started to grow on the alopecic areas and there was no further hair loss (Figure 2). It was agreed to continue the treatment with trilostane at the same dose.

Week 6: the owner reported that the patient was better in himself, active and lively, and the PU reduced compared to when he was initially presented. The owner also reported that there was one incident where a creamy brown liquid was found in his housing and that was put down as diarrhoea as the trilostane is known to cause gastrointestinal signs as an adverse reaction [3]. It was also noted that the patient’s weight had reduced by 35 g. It was advised to increase the trilostane dose to 3.75 mg kg⁻¹ sid/p.o. Lastly, more hair grew and the alopecic areas were reduced (Figure 3).

Week 7: the owner reported that the patient had erythematous genitalia, anorexia, more episodes of creamy brown liquid was found in his housing and was also PD. The owner ceased the treatment for seven days and the reported clinical signs were resolved.

Week 8: the patient was presented for a review and there was a further 10 g weight loss. The genitalia had crusting, however there was no erythema. PU and urine odour were also reported. More hair grew and there was no further hair loss. It was advised to reintroduce trilostane at the original dose, 2.5 mg kg⁻¹ sid/p.o., as the adverse reactions were suspected to be due to the higher dose. In view of the PU, enrofloxacin at the dose of 5 mg kg⁻¹ bid/p.o. for seven days was prescribed for a suspected UTI.

Week 11: the patient gained 10 g, the owner reported that there was significant improvement with the demeanour, the PU resolved and the urine odour reduced. The alopecic areas improved even more with the exception of some small areas on the right side of the flank and lumbar area (Figure 4). It was suspected that the hair follicles may have been irreversibly damaged.

Week 16: the patient was presented with altered demeanour and neurological signs with the suspicion of pituitary neoplasia. Although there was a further weight gain of 35 g and the patient was still having a good appetite, it was agreed to humanely euthanize as the condition was affecting the quality of life of the patient. No necropsy was performed.
Final diagnosis

Pituitary dependent HAC diagnosis was reached based on the history, systemic signs, dermatological lesions and therapeutic trial.

Ethical considerations

Consent for permission to publish this case report was obtained from the owner of the patient. The author declares no known conflicts of interest.

DISCUSSION

Although in dogs and cats the diagnostic process of HAC can be challenging in some cases, it is certainly much more practical to diagnose compared to hamsters [8]. In laboratory hamsters, the diagnosis of HAC is usually done using biochemistry for serum alkaline phosphatase and cortisol, and also urine cortisol creatinine ratio. Dynamic function tests such as ACTH stimulation test and dexamethasone suppression test are commonly used for the diagnosis of canine HAC, however in hamsters those tests are currency empiric [10]. Those tests were not considered, as the majority of them include high cost, lack of practicability and risks. Sampling may require several blood samples for the diagnosis and treatment monitoring which may need to be taken under general anaesthesia (GA). Furthermore, due to the size of the hamsters and blood volume restrictions, clinicians may fail to obtain the right amount in order to run the laboratory tests [6].

In terms of treatment, in literature searches, only a few case reports were found and no controlled clinical trials, involving treatment with metyrapone and 1,1-dichloro-2-2bis (p-chlorophenyl) ethane for HAC [1]. Mitotane is another drug of choice, with limited results [8, 10, 11]. An adapted canine HAC protocol with ketoconazole treatment was mentioned in a suspected case of HAC in a hamster with limited results too [7]. Trilostane was the only available drug to source in order to block the adrenal synthesis of glucocorticoids for this patient. When the dose of trilostane was at 3.75 mg.kg⁻¹ sid/p. o. the patient lost some body weight, was anorexic and also developed diarrhoea. When the dose was reduced back to 2.5 mg.kg⁻¹ sid/p. o. the side effects were resolved. In the study that described treatment of two Syrian hamsters [13], a trilostane dose of 5 mg.kg⁻¹ sid/p. o. was used and anorexia and lack of activity was reported. Subsequently, the treatment was ceased for two days and then trilostane was introduced at a dose of 2.5 mg.kg⁻¹ sid/p. o. and the adverse reactions improved [13]. On the dermatological note, the alopecia and hypotrichosis improved significantly and the therapeutic trial also confirmed the diagnosis of HAC even though no diagnostic techniques were performed.

During the treatment and monitoring, the patient had two occasions of suspected UTI. The patient was treated with enrofloxacin with a good response resolving the clinical signs [3]. UTIs are commonly reported in canine patients with HAC [4, 8], however in the exotics literature UTIs are not reported as a secondary complication of HAC.

In conclusion, this case report presented a 16-week treatment with trilostane with minimal side effects and had significantly improved hair regrowth. Even though no laboratory tests were performed, the therapeutic trial confirmed the diagnosis of HAC due to the high index of suspicion. This practical clinical approach for diagnosing and treating HAC could be established in domestic hamsters in order to avoid the risks of a GA and sampling but also to minimise the costs involved during the process.
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