



Liliane Simpson-Louredo^{1,2*}, Andrés Araújo Gutierrez¹ and Ana Luíza Mattos-Guaraldi¹

¹School of Medical Sciences, University of the State of Rio de Janeiro - UERJ, Brazil

²National Institute of Quality and Control in Health (INCQS), Oswaldo Cruz Foundation (Fiocruz), Brazil

Dates: Received: 23 January, 2017; Accepted: 22 February, 2017; Published: 23 February, 2017

*Corresponding author: Liliane Simpson-Lourêdo, MS DVM, Faculty of Medical Sciences, State University of Rio de Janeiro - UERJ - Av. 28 de Setembro, 87 - Funds, 3rd floor, Brazil, CEP: 20 551-030; Tel: +55 (21)2868-8280; Fax: +55(21)2868-8376; E-mail: liliane.uerj@gmail.com

Keywords: ACTH; Adrenal insufficiency; Dog; Na: K ratio

<https://www.peertechz.com>

Case Report

ACTH Level and Sodium-Potassium Ratio in Screening of Primary Canine Hypoadrenocorticism

Abstract

This article describes a primary hypoadrenocorticism (HA) case report in a spayed mix-breed German shepherd ten years old female dog that had unspecific symptoms during a week. The initial suspicious were poisoning and after, renal insufficiency, since laboratorial exams demonstrated azotemia. In both cases, the treatment were unsuccessfully. After laboratory exams and blood sodium: potassium ratio (Na: K) measure (23:1), the presumptive diagnosis was primary HA, that it was confirmed by plasma adrenocorticotrophic hormone (ACTH) level (280 pg/mL). Although the gold standard test to confirm HA is ACTH stimulation test, in many situations this exam is not feasible, once synthetic ACTH is expensive and often unavailable, especially in developing countries. In the case related here, only Na: K and plasma ACTH level were used as an alternative method to identify primary HA properly, associated to clinical signs, which was confirmed by full recovery of the patient after indicated therapy.

Introduction

Naturally occurring primary hypoadrenocorticism (Addison's disease) is a uncommon canine disease, frequently disregarded and misidentified at clinical routine [1,2]. In Brazil, only one case was currently found described in the available literature [3]. The main cause of canine HA is atrophy or destruction of adrenal cortices. This malfunction can be resultant from several causes; however, an immune-mediated or idiopathic disorder seem to be the principal factor [1,2]. This endocrinopathy causes a wide range of symptoms that are common to other diseases [2,4]. The lack of pathognomonic clinical signs makes it has been referred to as "the great pretender," due to its ability to mimic other common diseases in the dog and thereby represent a diagnostic challenge [5]. The primary HA clinical signs occur when at least 80-90% of adrenal gland tissue is destroyed, resulting in mineral and glucocorticoids deficiencies [5]. The absence of negative feedback results in a high serum level of ACTH [6]. The owner's complaints are related to nonspecific signs, as vomiting, lethargy, weakness and anorexia that are associated to multiple systems and illnesses, as gastrointestinal, renal or neurologic disorders. HA can affect dogs of any age, but normally it occur in young to middle-aged dogs, especially females [2-4].

The ACTH stimulation test is the gold standard for diagnosing HA in dogs and consists to measure cortisol levels before and after administration of synthetic or commercial

gel preparations of purified porcine pituitary extract [6,7]. However, problems with the availability of synthetic ACTH and increased costs have prompted the need for alternative methods, especially in developing countries [6].

Secondary HA is a rare condition characterized by deficient pituitary secretion of ACTH; iatrogenic HA is more common than the naturally occurring form and usually results from exogenous glucocorticoid administration. In this circumstance, aldosterone secretion is preserved and serum electrolytes persists normal [1,5]. Electrolytes abnormalities may be used to distinguish primary to secondary HA, as they hardly change in secondary HA [2,3]. Therefore, calculation of the sodium-potassium ratio and plasma ACTH level is a useful screening test for diagnosing primary HA [6].

Case Report and Diagnosis Procedures

A spayed mix-breed German shepherd ten years female dog showed increasing clinical signs, as vomiting, weakness and anorexia for a week. The animal had a complete schedule of vaccination and ecto/endoparasites prevention, including heartworms. The initial diagnosis was a simple poisoning case, being medicated. As there was not improvement in the general symptoms, a treatment with fluid therapy and supportive care was started in a veterinary hospital. Laboratory exams indicated thrombocytopenia and azotemia. The enzyme-linked immunosorbent assay (ELISA) SNAP test was performed

in order to investigate Erlichiosis, resulting negative. After medication, the clinical signs were controlled and exams were normal and the bitch was released. After four days, the animal got worse, stopping to eat and showing ataxia. Complementary exams (head/thoracic radiographs and abdominal ultrasound) were performed, without significant results, except a reduction in spleen volume for hypovolemia. That time, hematologic evaluation revealed normochromic normocytic anemia, anisocytosis, rouleaux erythrocyte, polychromasia, leukocytosis (neutrophilia and monocytosis) and normal count of platelets. The Na: K was decreased (23:1), so the suspicion of diagnosis was HA, confirmed by the high plasma ACTH level (248 pg/mL). Again, fluid therapy and supportive care were given to patient and proper therapy (prednisolone and fludrocortisone) was started; a marked progress of general clinical signs was noted. The stabilization of primary HA is defined for absence of symptoms with a sodium: potassium ratio >27:1 and both electrolyte concentrations within a laboratory reference levels [8]. After three years, the HA is controlled and the patient is in good health, with normal appetite and activity; secondary diabetes or other concurrent abnormalities were not noted until the present date.

Discussion

Primary HA is normally ignored in veterinary practice, especially for its capability to pretend clinical signs shared to other disorders. In this particular case, initial suspicious was poisoning and renal insufficiency; only after failed therapies and unsuccessful diagnosis an endocrinopathy was supposed. Although gold standard definitive test to identify HA is ACTH stimulation test, the conclusion was achieved by a combination of clinical signs, Na: K and plasma ACTH level, since availability of synthetic ACTH for the test and increased costs make this exam particularly hard to get in developing countries, showing the need of alternative methods.

Conclusion

This case report describes a chronic HA case in a patient that still remains healthy after three years under appropriated treatment. Interestingly, the definitive diagnosis was based on clinical signs, Na: K and only ACTH blood measure, without ACTH stimulation test. This article is important to remind clinicians about this often neglected disease in the clinical routine once the HA is an uncommon disease in dogs and the difficult for veterinarian to identify. Moreover, it emphasizes an alternative scheme to improve and make easier HA diagnosis.

References

1. Feldman EC (1992) Moléstias das glândulas adrenais. In: Ettinger, S.J., 3ª Ed., Tratado de Medicina Interna Veterinária, Ed. Manole, São Paulo, 3, 1839.
2. Kintzer PP, Peterson ME (1997) Primary and Secondary canine Hypoadrenocorticism. *Vet Clin North Am Small Anim Pract* 27: 349-357. [Link: https://goo.gl/QGX2pV](https://goo.gl/QGX2pV)
3. Emanuelli MP, Lopes STA, Schmidt C, Maciel RM, Godoy CLB (2007) Hipoadrenocorticism primário em um cão. *Ciência Rural, Santa Maria*, 37: 1484-1487. [Link: https://goo.gl/IU8CF5](https://goo.gl/IU8CF5)
4. Hanson JM, Tengvall K, Bonnett BN, Hedhammar Å (2016) Naturally Occurring Adrenocortical Insufficiency – An Epidemiological Study Based on a Swedish-Insured Dog Population of 525,028 Dogs. *J Vet Intern Med* 30: 76–84. [Link: https://goo.gl/BIWUiR](https://goo.gl/BIWUiR)
5. Susan C Klein, Mark E Peterson (2010) Canine Hypoadrenocorticism: Part I. *Can Vet J* 51: 63–69. [Link: https://goo.gl/djkU5o](https://goo.gl/djkU5o)
6. Boretti FS, Meyer F, Burkhardt WA, Riond B, Hofmann-Lehmann R, et al. (2015) Evaluation of the Cortisol-to-ACTH Ratio in Dogs with Hypoadrenocorticism, Dogs with Diseases Mimicking Hypoadrenocorticism and in Healthy Dogs. *J Vet Intern Med* 29: 1335–1341. [Link: https://goo.gl/zxhsZg](https://goo.gl/zxhsZg)
7. Susan C Klein, Mark E Peterson (2010) Canine Hypoadrenocorticism: Part II. *Can Vet J* 51: 179–184. [Link: https://goo.gl/zaIXK9](https://goo.gl/zaIXK9)
8. Roberts E, Boden LA, Ramsey IK (2016) Factors that affect stabilisation times of canine spontaneous hypoadrenocorticism. *Vet Rec* 179: 98. [Link: https://goo.gl/1PWrbk](https://goo.gl/1PWrbk)