Proceedings of the 34th World Small Animal Veterinary Congress
WSAVA 2009

São Paulo, Brazil - 2009

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Hyperadrenocorticism is one of the most common endocrinopathies in mid-age to older dogs. Historically, therapy using mitotane—a chemotherapeutic which promotes necrosis and adrenal cortex atrophy—has been standard in managing this disease. However, due to its many side-effects, such as diarrhea, emetic episodes, nausea, and acute Addisonian crisis, its use must be made with much care. A therapeutic alternative is trilostane, a competitive inhibitor of 3'hydroxysteroid dehydrogenase—and its efficacy has been shown to be similar to that of mitotane. Current research is focused on comparing efficacy and safety of these medications. Thus, this report is aimed towards describing histopathological alterations in adrenal glands of a dog submitted to therapy with trilostane. One female, mixed-breed canine, 6 years old, diagnosed with hyperadrenocorticism, presented symptoms compatible with hypoadrenocorticism after 17 days of a 6mg/kg per day treatment with trilostane. Serum sodium and potassium levels were measured, and the patient was subjected to the ACTH stimulation test. Results confirmed the clinical suspicion, and treatment with trilostane was discontinued. The patient received intensive treatment and presented full recovery. After 6 months, the animal presented neurological alterations, and after an unfavorable clinical evolution, the owner opted for euthanasia. During necroscopy, an increase in volume was observed both in the hypophyseal and adrenal glands. Histopathological evaluation showed hypophyseal adenoma and diffuse atrophy of adrenal glands, with parenchymal fibrosis. The use of trilostane also requires medical attention, because it may promote adrenal necrosis, and thus lead to Addisonian crisis, which, in turn, if not immediately diagnosed and treated, may lead to death. New researches must be conducted in order to better assess the alterations which may be caused by the use of trilostane.