What's new in the monitoring of hyperadrenocorticism?

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Trilostane is the most common medical treatment for canine hyperadrenocorticism. There are challenges in achieving stability whilst maintaining safety. Monitoring of a patient's condition should move towards a comprehensive approach, placing more emphasis on at-home monitoring. This should help to highlight patients that are unwell, and also allow the optimal dose of trilostane to be achieved. Whilst it would be desirable to rely solely on an owners' observations, an objective tool is required to ensure that treatment remains safe.

Traditionally the post-ACTH stimulation cortisol has been used to guide dose adjustments and to detect dogs at risk of iatrogenic hypoadrenocorticism. There is consistent evidence that the correlation between the post-ACTH stimulation cortisol and clinical control is poor. In addition, newer evidence supports the continued use of trilostane in a small subset of dogs with low post-ACTH stimulation cortisol, that are clinically well.

Alternative methods of monitoring dogs receiving trilostane have been investigated with varying success. These methods include baseline (or pre-ACTH) cortisol, endogenous ACTH, haptoglobin and the urine corticoid: creatinine ratio (UCCR) before and after trilostane. In most of these studies, control was ultimately defined by the results of the post-ACTH stimulation cortisol and the owners' perception of clinical control was not well described. Haptoglobin has recently shown more promise when compared to clinical control at home.

The pre-trilostane cortisol is an alternative tool which has been shown to have much better correlation with clinical control reported by owners. In addition, there are some instances when this result may be better than the post-ACTH stimulation cortisol at detecting iatrogenic hypoadrenocorticism. This test is easier, cheaper and has no side effects, offering a more practical solution for busy practice life. New evidence also suggests that the pre-trilostane cortisol is more repeatable than post-ACTH stimulation cortisol. The suggested target range of 40–138 nmol/l should be used alongside consistent owner observations, physical examination findings and the results of additional appropriate tests, such as biochemical and electrolyte results.

A holistic approach is required when monitoring hyperadrenocorticism. More emphasis should be placed on owner's observations combined with biochemical, electrolyte and cortisol measurements to guide dose

adjustments to achieve stability and avoid complications of treatment.

KEY LEARNING OBJECTIVES

- Clinical response as determined by owners' observations of signs at home should be a key component of monitoring dogs receiving trilostane
- Traditional monitoring using the post-adrenocorticotrophic hormone (ACTH) stimulation cortisol does not accurately reflect clinical control
- Pre-trilostane cortisol and other novel monitoring techniques may be useful adjunctive tools alongside clinical signs

MULTIPLE CHOICE QUESTIONS

1. Which of the following is true regarding haptoglobin?
 - (A) It is a negative acute phase protein
 - (B) IL-10 is thought to be the main stimulator of its synthesis
 - (C) Dogs with hyperadrenocorticism have been shown to have equivalent increases in haptoglobin when compared to dogs receiving exogenous corticosteroids
 - (D) Haptoglobin is produced primarily but not exclusively by the liver in response to exogenous and endogenous steroids
2. Regarding pre-trilostane cortisol, which of the following statements is correct?
 - (A) It can be measured 11, 12 or 13 hours after the previous dose, without it affecting the result
 - (B) It should always be interpreted together with the post-ACTH stimulation cortisol result
 - (C) It is more repeatable than the post-ACTH stimulation cortisol, meaning that a dog on a stable dose of trilostane is more likely to have a similar pre-trilostane cortisol compared to a post-ACTH cortisol irrespective of the day the test is performed
 - (D) It is a good stand-alone test, owing to its excellent ability to differentiate controlled and uncontrolled dogs, with minimal overlap
3. When does the cortisol nadir after trilostane administration occur?
 - (A) 0–2 hours after administration
 - (B) 2–4 hours after administration
 - (C) 4–6 hours after administration
 - (D) 6–8 hours after administration