

Managing Acute Hypoadrenocorticism



Hypoadrenocorticism may result from a lack of adrenocorticotrophic hormone (ACTH) but is primarily caused by adrenal gland dysfunction. This retrospective study aimed to describe the efficacy, outcome, and adverse events associated with IV hydrocortisone and fluid therapy for managing acute hypoadrenocorticism.

Thirty dogs met the inclusion criteria with laboratory data and pre- and post-ACTH stimulation test results compatible with hypoadrenocorticism. All dogs received IV fluid therapy pending diagnostic testing. All remained hyperkalemic, hyponatremic, or both immediately before starting IV hydrocortisone treatment at either 0.5 mg/kg/hr ($n=9$) or 0.625 mg/kg/hr ($n=21$). In the 23 dogs for which complete electrolyte data were available, the mean rate of change of sodium concentration over 24 hours was 0.48 (± 0.28) mmol/L/hr; the mean rate of change of potassium was -0.12 (± 0.06) mmol/L/hr. Plasma sodium concentration increased by >12 mmol/L in 24 hours in 7 dogs. Within 24 hours, all dogs had normal potassium concentrations.

All dogs survived to discharge; the median time from the start of hydrocortisone treatment to discharge was 2 days. Only 1 dog had adverse events noted, with temporary neurologic signs presumably caused by an increase in sodium concentration. The authors concluded that the use of IV hydrocortisone and fluid therapy is an effective treatment for dogs with hypoadrenocorticism.

Global Commentary

If the findings are validated in future studies, this alternative therapeutic protocol could become the most appropriate for hypoadrenocorticism. The choice of hydrocortisone is rational and scientifically sound because its equipotent mineralocorticoid and glucocorticoid effects make it well-tailored for addressing the hormonal deficits that constitute the hallmark of Addison disease. Additionally, the glucocorticoid potency of hydrocortisone is limited, thereby decreasing incidence of side effects attributed to excessive glucocorticoid activity.

This therapeutic approach seemed more effective than those using other glucocorticoids, as the correction of acid-base and electrolyte abnormalities was solely achieved with fluid therapy and hydrocortisone. Remarkably, therapeutic measures such as bicarbonate and insulin were unnecessary—an important finding, as their administration can be associated with potentially

serious side effects. Although fluid therapy was administered at a conservative rate, the authors found it enough to correct azotemia when hydrocortisone therapy was given concurrently. This is good news because excessive fluid administration has been increasingly recognized as deleterious in critically ill patients. The study, however, was unclear in describing the degree of dehydration at initial patient presentation. This raises the question of whether a different fluid therapy approach might be required for patients with serious dehydration. Future studies should address this question. Still, this approach was more effective, simpler, and less demanding in terms of medical resources and resulted in fewer side effects—what we hope for every time a new therapeutic approach comes along.—*Nuno Felix, DVM, MD, MS*

Source

Gunn E, Shiel RE, Mooney CT. Hydrocortisone in the management of acute hypoadrenocorticism in dogs: a retrospective series of 30 cases. *J Small Anim Pract.* 2016;57(5):227-233.

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