УДК 636.7:619:616.43

PRIMARY HYPOADRENOCORTICISM IN A DOG — CASE REPORT

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Hypoadrenocorticism is a rare but serious disorder of the endocrine system. Because of the variety of clinical signs, hypoadrenocorticism is often difficult to diagnose. Ultimately diagnosis is made based on the combination of clinical signs, suggestive of routine blood tests, and the results of a specific test for this disorder (1). Dogs in collapse with acute adrenocortical insufficiency must be treated immediately with intravenous glucocorticoids and large volumes of intravenous fluids to correct dehydration and electrolyte imbalances. Long-term treatment involves the administration of hormones (2, 3). Because dogs with Addison's disease cannot produce more cortisol in response to stress, stress should be minimized whenever possible. It may be necessary to increase the amount of hormones given during periods of stress (e.g. boarding, surgery, travel) (4).

Introduction. Early anatomists had been aware of the adrenal glands for years, however, it wasn't until 1849 that Thomas Addison described a syndrome in humans that he associated with the dysfunction of the adrenal glands. At that time, no therapy was available, and patients with the disorder died from their illness. The first reported canine case of Addison's disease was described 104 years later, in 1953 [1, 2].

Hypoadrenocorticism called Addison disease is caused by deficient secretion of mineralocorticoids (aldoseron) and glucocorticoids by adrenal gland. The adrenal gland is a small gland located near the kidney. The adrenal is composed of two layers, the cortex and the medulla. The outer area of the kidney, or cortex, secretes corticosteroid hormones such as cortisol and aldosterone [1–4]. The medulla part of the kidney, part of the sympathetic nervous system, secretes adrenaline or epinephrine, which is generally not affected by Addison's disease. Hypoadrenocorticism is tends to occur in young to middle-age female dogs [3]. Although it has been diagnosed in very young dogs (5 weeks) and very old dogs (15 years), the average age at diagnosis is 4,3 to 5,4 years. Females constitute 70 percent to 75 percent of all reported cases (4).

There are three forms of Addison's disease in dog: primary, secondary and atypical [1–4]. Primary and atypical Addison's disease is usually the reason behind immune mediated damage to the glands. Secondary hypoadrenocorticism is from failure of the pituitary hormones to stimulate the adrenals with adrenocorticotropic hormone (ACTH). Dogs with Addison's disease typically show clinical signs of lethargy, inappetence, vomiting, diarrhea, weakness, and weight loss — which often suggest more common disease such as renal, gastrointestinal, or various toxicological disorders [1]. Characteristic laboratory abnormalities include hyperkalemia, hyponatremia, azotemia, mild metabolic acidosis, and a normal leukogram with or without lymphocytosis or eosinophilia. In some dogs, however, these typical laboratory abnormalities are not present; particularly, electrolytes may be normal or show changes in only one parameter [2].

Addison's disease is a disorder with symptoms that are common to many other physical ailments, making diagnosis difficult and sometimes a process of elimination. But once Addison's disease is correctly diagnosed, a properly treated dog can live a normal, active life.

Task, the aim of the article. The aim of the study was an analysis of morphological and biochemical changes in the blood of the dogs with Addison disease.

Material and methods. This study is the case presentation of the mixed breed, female, marked aged 7 year. The examinations were performed as follows: the history and clinical examination, abdominal USG, echocardiography, electrocardiography, blood examination (erythrocyte count, hematocrit value, hemaglobin concentration, leucocyte count with leucogram, thrombocyte count and erythrocyte indices, urea and creatinine concentration, activity of alanine aminotransferase [ALP], asparagine aminotransferase [AST], alkaline phosphatase [AP] and concentration of total protein, albumin, cholesterol, glucose, Na+, K+, Cl-). Next dog underwent the endocrine testing. After test blood was collect. After that was IV administrated synthetic ACTH

(cosyntropin 0,5 mg/kg IV). After 1 hour was taken serum sample. In bought samples was examinated cortisol concentration.

Results of researches. The dog presented with weakness, lethargy, vomiting, shaking, weight loss, and loss of appetite. Symptoms of Addison's disease are very similar to other diseases and they seem rather vague. Symptoms may either include but are not limited too; diarrhea, drinking more, urinating more, dehydration, and a fast heart rate in some patients. Many times these symptoms may go unnoticed or correctly diagnosed and the dog finally presents in a Addisonian crisis. During an Addisonian crisis the dog may present in acute collapse and in shock. The result of morphological and biochemical tests are showed in table 1.

Tab. 1

Test results of chosen morphological and biochemical parameters in blood

Parametr	
Erythrocyte [T/l]	4,57
Hematocrit [l/l]	0,295
Hemaglobin [mmol/l]	6,4
MCV [f/l]	65
MCH [f/mol]	1,40
MCHC [mmol/l]	21,7
Leukocyte [g/l]	17,4
Limfocyte [%]	22
Monocyte [%]	3,9
Granulocyte [%]	74,1
Thrombocyte [g/l]	18
Urea [mmol/l]	32,08
Creatinine [μmol/l]	284
AspAT [U/l]	68
AlAT [U/l]	147
ALP [U/l]	157
Total protein [g/l]	65
Albumin [g/l]	33
Cholesterol [mmol/l]	4,0
Glucose [mmol/l]	8,58
Na+	109
K+	5,37
Cl-	77
Ca++	2,42
P++	2,67

The dog did not manifest any changes of the abdominal cavity organs in ultra-sound examination. Other tests, such as an electrocardiogram (EKG) and heart USG either did not show any abnormalities. These tests would be performed if a dog's potassium levels were so high that it caused the heart rate to become dangerously slow or erratic. In our study potassium level was high, but in reference range. A definitive diagnosis was made by a blood ACTH stimulation test. Base value of cortisol in serum was 3,80 ng/ml. One hour after administration of exogenous adrenocorticotrophic hormone (ACTH) — 4,71 ng/ml.

C O N C L U S I O N

Addison disease was diagnosed by a series of blood tests. A chemistry panel show a low sodium level, and the resulting Na:K ratio was low. The kidney enzymes was high. The Adrenocorticotropic hormone (ACTH) stimulation test gave a definitive diagnosis for Addison's disease.

ПЕРВИННИЙ ГІПОАДРЕНОКОРТИЦИЗМ У СОБАКИ

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Р Е З Ю М Е

Гіпоадренокортицизм — рідкісне і важке захворювання ендокринної системи, яке важко діагностувати внаслідок різноманітних клінічних симптомів. Діагноз ставлять на основі клінічних ознак, результатів аналізу крові та специфічних тестів. Собакам з тяжким перебігом захворювання внутрішньовенно вводять глюкокортикоїди та велику кількість рідини з метою корекції зневоднення і дисбалансу електролітів. Довготривале лікування негативно впливає на баланс гормонів. Оскільки собаки при хворобі Аддісона не можуть продукувати у великій кількості кортизол, то необхідно мінімізувати стресові ситуації. Під час стресу (транспортування, хірургічного втручання, подорожі) кількість препарату можна збільшувати.

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А Н Н О Т А Ц И Я

Гипоадренокортицизм — редкое и тяжелое заболевание эндокринной системы. Из-за разнообразия клинических признаков болезнь трудно диагностировать. В конечном результате диагноз может быть поставлен на основании клинических симптомов, результатов исследования крови, а также использовании специфических тестов. Больным собаками с тяжелым течением болезни внутривенно немедленно вводят глюкокортикоиды и в больших количествах жидкость с целью коррекции обезвоживания и дисбаланса электролитов. Длительное лечение отрицательно влияет на баланс гормонов. Поскольку собаки с болезнью Аддисона не могут производить большое количество кортизола в ответ на стресс, стрессы должны быть минимизированы. Количество препарата можно увеличить, в стрессовых ситуациях (транспортировка, хирургические вмешательства, путешествия).

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Рецензент: провідний науковий співробітник лабораторії живлення овець та вовно утворення, с. н. с. В. В. Гавриляк.