Hypoadrenocorticism Associated with Sepsis in Dogs – Case Report

https://doi.org/10.56238/uniknowindevolp-104

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ABSTRACT
Hypoadrenocorticism is an uncommon condition in dogs and is most often caused by immune-mediated destruction of the adrenal glands resulting in decreased mineralocorticoid and glucocorticoid production. Definitive diagnosis requires adrenocorticotropic hormone (ACTH) stimulation testing to demonstrate low basal and post-ACTH cortisol levels. The symptoms are varying and usually not specific. An acute hypoadrenocortical crisis in a dog represents a true medical emergency due to the resulting severe hypovolemia, dehydration, hypotension, electrolyte derangements, and acid-base abnormalities. The prognosis for hypoadrenocorticism is excellent with appropriate mineralocorticoid and glucocorticoid supplementation. This report describes the case of a non-defined breed female dog, 8 years old, 8.9 kg diagnosed with primary hypoadrenocorticism. Treatment was based on replacement of hydroelectrolyte, glucose and corticosteroid deficits, promoting rapid evolution in the patient's clinical condition, with disappearance of symptoms and normalization of laboratory parameters by the end of the treatment.

Keywords: Hypoadrenocorticism, Addison's disease, Mineralocorticoids, Glucocorticoids, Dog.

1 INTRODUCTION

Hypoadrenocorticism (AH) or "Addison's disease" is an infrequent endocrinopathy in the clinical routine of small animals, mimics cynical manifestations of other diseases, is difficult to diagnose and is characterized by adrenocortical hormonal secretory insufficiency. It affects adult dogs, usually females between 4 and 5 years of age and is uncommon in cats. (FELDMAN, *et al*. 2015)

It is a systemic disease of chronic evolution with varied implications. It affects different organs and presents great variability of nonspecific clinical signs, which makes the diagnosis difficult since it can be confused with other conditions such as chronic kidney disease and chronic gastroenteritis. Most of the time, the animals end up dying even before the definitive diagnosis due to the evolution of clinical manifestations, which can cause sepsis and shock (hypothermia, hypotension, bradycardia, hypoglycemia, hyponatremia, and hypokalemia) (FELDMAN, PETERSON, 1984; FELDMAN *et al*., 2015).

The definitive diagnosis is made through the ACTH stimulation test, where serum cortisol is measured before and after stimulation with the hormone. Patients positive for hypoadrenocorticism do not respond to the test, the test remains unchanged after stimulation. The treatment is based on the

replacement of glucocorticoids and/or mineralocorticoids, depending on which classification of AH the patient presents (GRECO, 2006).

In this sense, the present report aims to describe the case of a dog, female, SRD, 8 years weighing 8.9kg complaining of intermittent gastroenteritis for 3 months that evolved to hematochezia, with evident and progressive weight loss, already diagnosed with Hypoadrenocorticism 2 years ago, treated with fludrocortisone (continuous use) and the difficulties that permeated the case.

2 LITERATURE REVIEW

Hypoadrenocorticism (AH) is an uncommon endocrine disease in dogs, with a reported prevalence of 0.09% and rare in cats (KIMURA et al, 2019). Occurs due to deficiency of mineralocorticoids and/or glucocorticoids and can be characterized as primary, secondary, or iatrogenic (MORAIS; DIBARTOLA, 2008). The primary form of hypoadrenocorticism or "Addison's disease" is the one that most affects dogs, being first described by Thomas Addison in 1855 (BAUMSTARK et al., 2014).

The typical primary form, caused by the destruction or atrophy of the three layers of the adrenal cortex, is glucocorticoid and mineralocorticoid deficiency, especially aldosterone. Other conditions such as fungal diseases (coccidiosis and cryptococcosis), adrenal amyloidosis and neoplasms may also be responsible for adrenal insufficiency. (KLEIN; PETERSON, 2010).

The secondary form presents as glucocorticoid deficiency caused by decreased secretion of ACTH. Pituitary dysfunction occurs causing atrophy of the reticular and fasciculate zones and its main cause is associated with abrupt cessation of prolonged corticosteroid treatments, causing the hypothalamic-pituitary axis to be unable to respond to the sudden lack of glucocorticoids (GRECO, 2007). The iatrogenic cause can occur in cases of adrenalectomy and prolonged treatments with mitotane or trilostan (CHURCH, 2004; MORAL; DIBARTOLA, 2008).

Dogs that present primary hypoadrenocorticism and arrive for care have a history of depression, lethargy, anorexia, weight loss, vomiting, diarrhea, muscle weakness, tremors, polyuria (PU) and polydipsia (PD) and abdominal pain, and the most common clinical manifestations are related to the gastrointestinal tract and mental status. For the most evident clinical signs to develop, it is believed that the adrenal corticals need to be destroyed by 90% (NELSON; COUTO, 2006; GRECO, 2007; KLEIN; PETERSON, 2010).

Polydipsia occurs as a compensatory mechanism to polyuria. In patients with AH who experience inefficient aldosterone production, loss of water and sodium, and reabsorption of potassium and hydrogen can lead to compensatory PU/PD syndrome. Low aldosterone production can also lead to common clinical manifestations such as weight loss, decreased appetite, vomiting, weakness,

malaise, and other changes that mimic gastrointestinal diseases and kidney diseases (FELDMAN *et al*. 2015; TILLEY; Smith, 2015).

Changes in the gastrointestinal tract occur due to insufficient cortisol production that affects the gastric mucosa, motility and tissue perfusion leading to the formation of ulcers and consequently vomiting, diarrhea, hematochezia, hematemesis, and melena. Most often these symptoms respond to supportive treatment with fluid therapy and antiemetics. Seizures and ataxia are neurological signs that usually occur in patients with severe hyponatremia (WAKAYAMA *et al*., 2017). If hyponatremia and hyperkalemia become severe, hypovolemia, prerenal azotemia, and the resulting arrhythmias may trigger Addisonian crisis. Clinical manifestations intensify and become more severe. In severe cases, the animal may be in shock and agonizing. The Addisonian crisis must be distinguished from other disorders that life-threatening, such as diabetic ketoacidosis, necrotizing pancreatitis, and septic peritonitis (NELSON; COUTO, 2006 – Table 1).

The severity of organ dysfunction in patients in crisis, such as sepsis and circulatory shock, carries a high risk of mortality. Most often, the mechanisms involved in shock are complex and involve more than one of the three main hemodynamic changes, namely hypovolemia, myocardial dysfunction and altered vascular tone (SINGER *et al*., 2016)

Diagnosis is based on the patient's history, clinical signs, electrolyte changes, and confirmation with the ACTH stimulation test. The most common changes in primary AH are azotemia, metabolic acidosis, hypochloremia, hyperkalemia, and hyponatremia, but not all electrolyte changes may not be present (BORIN- CRIVELLENTI, 2015).

Table 1 – Laboratory Changes Associated with Hypoadrenocorticism in Dogs and Cats (NELSON; COUTO, 2006; Vargas, 2015).

CBCPANEL	BiochemicalUrinalysis
Anaemia non-regenerativeHyperkalemiaIsostenuria	
Leukocytosis by neutrophiliaHyponatremia	
	hyperstenuria Neutropenia mildHypochloremia
EosinophiliaAzotemia	pre-renal
LymphocytosisHyperphosphatemia	Hypercalcemia
	Hypoglycemia
	Metabolic acidosis

Routine laboratory tests are not specific. On ultrasound it is possible to find the adrenals reduced in size, suggesting adrenocortical atrophy (ADLER, *et al*. 2007). On radiography, it is possible to find changes suggestive of megaesophagus and signs of hypovolemia such as narrowing of the vena cava and pulmonary artery, which, although uncommon, associated with other findings, can help direct the diagnosis. (MELIÁN; PETERSON, 1996; GRECO, 2007; Vargas, 2015). Some dogs may also present microhepathy on abdominal radiography (MELIÁN *et al*., 1999).

In the electrocardiogram (ECG) we can identify increase in the amplitude of the T wave and decrease in the amplitude of the P wave, as well as shortening of the QT interval and increase of the PR interval (BORIN-CRIEVENTTI, 2015; Vargas, 2015). 2nd degree heart block and atrial fibrillation may also be present (MELIÁN; PETERSON, 1996; POMROME; ANTUNES, 2012).

Treatment should be instituted even before confirmation of the diagnosis by ACTH stimulation tests and/or cortisol dosage, and should be based on clinical signs, biochemical dosages, and ECG. Therapy for AH aims to reverse hypovolemia, shock, hyperkalemia and hypoglycemia (KOENIG, 2013).

The main cause of death of patients with AH is hypovolemic shock. Treatment with aggressive fluid therapy is imperative and should be directed to the control and correction of hypovolemia, hypotension, hyperkalemia, hypoglycemia, metabolic acidosis, and cardiac arrhythmias associated with the condition. A bolus of 20-30ml/kg, IV of crystalloid solution is indicated. Many studies suggest the use of 0.9% physiological saline solution (with 154 mEq of Na+/L) as the fluid of choice, however, multiple cases of myelinolysis have already occurred with the rapid correction of hyponatremia. The manifestation of myelinolysis, which can take weeks to months to resolve, manifests with neurological signs that include lethargy, ataxia, weakness, hypermetry, and difficulty swallowing. Therefore, Lactate Ringer's solution is preferred by some clinicians since they are more alkalizing than saline solutions, correcting acidosis more efficiently. Hyperkalemia is the greatest threat to the patient's life and should be addressed immediately, especially if accompanied by cardiac arrhythmias. Hyperkalemia is reduced with rehydration and by the improvement of renal perfusion, however, patients with manifestations of bradycardia or arrhythmias, should be treated with calcium gluconate (considered cardioprotective agent), or insulin, glucose, bicarbonate (KOENIG, 2013; Thompson, 2018).

If the patient has mild or moderate hypoglycemia, glucose should be added to the fluid, obtaining a 2.5% to 5% glucose solution. In cases of severe hypoglycemia, in addition to glucose in fluid therapy, 1 to 2 ml/kg of 25% glucose should be administered in bolus (VARGAS, 2015).

In conjunction with fluid therapy, glucocorticoids that can be used are dexamethasone, prednisolone, and hydrocortisone. Hydrocortisone has the advantage of being a mineralocorticoid of short duration, and quickly assists in the correction of hyperkalemia (SPENCE.; GUNN.; RAMSEY, 2018). Dexamethasone is the glucocorticoid of choice for the treatment of patients in acute crisis, and can be administered intravenously at the initial dose of 0.5 to 1 mg/kg, being repeated every 12 hours at a dose of 0.05 to 0.1 mg/kg/IV until the oral form can be safely administered, being then the choice for the acute treatment of patient. Or use of prednisolone, prednisone and hydrocortisone will interfere in the ACTH stimulation test, therefore, its use is indicated after the confirmation of the diagnosis of AH (VARGAS, 2015). Dexamethasone has no mineralocorticoid effect, but will provide a source of

rapid glucocorticoid absorption (SPENCE; GUNN; RAMSEY, 2018). Mineralocorticoid supplementation begins as soon as the animal presents sodium and potassium alterations and is done with fludrocortisone acetate 0.02mg/kg/day, VO, every 12 hours. To this protocol, prednisone 0.25-0.5mg/kg, VO, is added every 12 hours, after the reestablishment of feeding and water intake by the patient (BORIN-CRIVELENTTI, 2015).

Gastrointestinal changes can be treated with supportive therapy and symptomatic therapy that may include omeprazole, sucralfate, ondansentron, and maropitant citrate. For patients with bleeding in the gastrointestinal tract, with the aim of avoiding bacterial translocation, the use of antibiotic is indicated (ampicillin at a dose of 22mg/kg/IV every 8 hours) (KOENIG, 2013). Most patients in Addisonian crisis respond to treatment within a few hours, but those who are more debilitated take 2-3 days to see significant improvement (SHIEL; Mooney, 2019).

The prognosis in canines with adrenal insufficiency is excellent. The most important factor in determining the response to long-term treatment of an animal is the education of the owner. If frequent controls are carried out and the owner is aware of the treatment and its continuity, the animals may have a "normal" life expectancy (NELSON; COUTO, 2006).

3 CASE REPORT

A patient of the canine species, SRD, female, 8 years old, 8.9 kg, complaining of intermittent gastroenteritis for 3 months that evolved to hematochezia with progressive and evident weight loss, was treated at the veterinary hospital in the west zone of São Paulo. It was reported by the tutor that there was worsening of the fecal score. The feces were in large volume with liquid diarrhea and high frequency, as well as episodes of tremors, emesis and anorexia. The patient had already been diagnosed with hypoadrenocorticism 2 years ago and was continuously using fludrocortisone.

In the emergency room, the patient was prostrate, with slightly pale mucous membranes, capillary filling time of 3', dehydration of 5%, mild diffuse abnormal pain on abdominal palpation, eupneic with RF 24 mrpm, HR 120 bpm, cardiopulmonary auscultation (PCA) with normophonetic heart sounds, without arrhythmia and clean pulmonary fields, temperature 37.4ºC, Systolic blood pressure (SBP) 100 mmHg, blood glucose 116 mg/dL (reference value in dogs 60 to 120mg/dL), ECC 3/9 and IMM 1/3. Follow-up was suggested during the patient's hospitalization.

During the period in which she remained hospitalized, the patient varied her level of consciousness between prostrate and alert. At the beginning of hospitalization, the parameters were measured every 2 hours and were always around: HR: 84 - 160 bpm, RF: 16 - 40 mrpm, PCA: with normophonetic sounds, without arrhythmia, clean lung fields, SBP: 80 to 130 mmHg, the mucous membranes ranged from pale to pink, dehydration from 5% to normal and rectal temperature between 37.1 - 38.3ºC.

Dry, wet and natural food were offered 8 hours after admission and the patient accepted spontaneously. She urinated throughout the hospitalization period without macroscopic changes and presented an emetic episode with food content at the beginning of the second day of hospitalization.

In the initial two days of stay, the patient presented two episodes of large volume of liquid diarrhea (score 0), which associated with the degree of dehydration, caused hypotension with SBP of 80 mmHg. The two episodes of hypotension were responsive to the volume where a water challenge of 10 mL/kg was performed in 20 minutes, and with the SBP normalized, the fluid therapy was adequate for a rate of 3 mL/kg/h.

During the first three days of hospitalization, the patient presented several fecal leaks with gradual and progressive improvement in frequency and aspect. In conjunction with the gradual improvement, she also showed improvement in the fecal score: she did not defecate on the first and second day (score 0), evolving to score 2 on the fourth and score 4 on the fifth day. With the favorable evolution, she did not present other episodes of hematochezia or melena until discharge.

In the abdominal ultrasound examination performed at the admission of the patient was visualized moderate degree of biliary mud in the gallbladder, spleen with slightly reduced dimensions, stomach with thick walls and submucosal layer in greater evidence, thickening of all intestinal segments with moderate distention and predominantly gaseous and mucoid contents, characterizing enteritis, right lobe of the pancreas with coarse parenchyma, but with maintained dimensions and echogenicity, suggesting chronic pancreatopathy (APPENDIX 1).

In the control ultrasound examination, performed on the fourth day of hospitalization, biliary mud, spleen with maintained dimensions, improvement in the thickening of the gastric walls, intestinal loops with thickening only in the jejunum were also observed, characterizing jejunitis. The pancreas still had a coarse parenchyma showing signs of a possible chronic process (APPENDIX 2).

The blood count performed at the time of admission showed several alterations: hematocrit 39%; protein 6.8g/dL; leukocytes 3080 thousand/mm³; metamyelocytes 1%; rods 19%; toxic and hyposegmented neutrophils and platelets 278 thousand/mm³ (ANNEX 3). Other serial examinations were performed every 48 hours and a decrease in hematocrit was observed, ranging from 39% to 25%. Leukocytes rose with each test. In the second leukogram they were 3530 thousand/mm³, with 86% of segmented, toxic and hyposegmented neutrophils. In the third and fourth tests, leukocytes were 13,870 thousand/mm³ with mild presence of reactive lymphocytes and 10210 thousand/mm³ with normal cell morphology, respectively. The patient had hypoalbuminemia of 2.1g/dL at the first dosage and the others had no noteworthy alterations (ANNEXES 4, 5, 6, and 7).

During hospitalization, to analyze blood pH and metabolic disorders that cause acid-base imbalance, serial blood gas analyses of the blood were made. Hypoadrenocorticism causes important

changes that are characteristic of the endocrine pathogenesis in question, such as changes in the sodium/potassium ratio. Hyponatremia can be seen in all dosages (APPENDIX 8).

The drug protocol was instituted with hydrocortisone 5mg/kg/IV every 8 hours, omeprazole 1mg/kg/IV every 12 hours, dipyrone 25mg/kg/IV initially every 8 hours and every 12 hours 5 days later, enrofloxacin 5mg/kg/IV every 12 hours, ceftriaxone 30mg/kg/IV every 12 hours, tramadol 3mg/kg/IV initially every 8 hours and reduced to every 12 hours 3 days after and metoclopramide 0.3 mg/kg/SC every 8 hours.

Four days after initiation, fludrocortisone was restarted at a dose of 0.025 mg/kg/VO every 12 hours and prednisolone 0.2 mg/kg/VO every 12 hours. Fluid therapy with 0.9% saline solution was maintained at a dose of 3ml/kg/hour.

During the hospitalization, the patient underwent evaluation with a gastroenterologist, and trypsinogen (not authorized by the tutor initially), cobalamin and folate were requested, since the tutors reported feces with the presence of previously undigested food. Due to the complaint of numerous feeding changes, initially without medical guidance and after with the follow-up of a nutritionist, the tutors were advised about the possibility of the patient presenting food hypersensitivity and inflammatory bowel disease associated with AH, given the constant episodes of gastroenteritis.

It was indicated 24 hours of observation after the return of oral medications and control blood gases. The patient remained clinically stable with parameters within the normal range, feeding spontaneously, without emetic or diarrheal episodes. She was released with medical discharge and will continue to be followed up essentially with an endocrinologist.

4 DISCUSSION

The animals diagnosed with hypoadrenocorticism are usually canines, young females between 4 and 6 years of age of different breeds or SRD, as the patient of the case reported. In the anamnesis, the main complaints of the tutors are tremors, dark diarrhea, anorexia, prostration with weight loss; all these signs being nonspecific (KLEIN, 2010).

The present report describes a case of AH in an 8-year-old female SRD dog attended with initial complaints of prostration, hematochezia, sporadic emesis, apathy, diarrhea, tremors and hyporexia. The patient had already been diagnosed with hypoadrenocorticism 2 years ago and was continuously using fludrocortisone. Clinical signs, even if nonspecific, are associated with renal and gastrointestinal disorders, which occur due to a lack of glucocorticoids, and polyuria secondary to renal sodium loss with compensatory polydipsia occurs due to mineralocorticoid deficiency (MELIÁN; PETERSON, 1996). All these symptoms were observed in the patient in this report.

The hypoglycemia that normally occurs in almost all patients with hypoadrenocorticism can be explained by the decrease in gluconeogenesis and hepatic glycogenolysis, due to the absence of

glucocorticoids, however, the glycemia of the patient in question remained stable throughout the hospitalization. Another important change caused by mineralocorticoid deficiency is hyperkalemia. Mineralocorticoids are responsible for maintaining the electrolyte balance of sodium, chloride, potassium and water in the renal tubules. Aldosterone is responsible for sodium retention and renal excretion of potassium. Poor production of this corticosteroid causes retention of this ion, leading to hyperkalemia. High serum potassium levels can alter the cardiac conduction system and cause changes, such as bradycardia (NELSON; COUTO, 2006; GUNN *et al*., 2016; Thompson, 2018). Although these alterations are common in AH, in the serial examinations of blood gases of the patient (ANNEX 8), these alterations were not observed. Potassium levels ranged from 3.1 to 4.7 mmol/L and blood glucose levels ranged from 83 to 130mg/dL.

The fluid of choice for volume replacement is the 0.9% saline solution, since it contains sodium and chloride in its composition. The most decompensated dogs have a tendency to be hyponatremic. However, Ringer's lactate solution is preferred by some clinicians, since they are more alkalizing than saline, correcting acidosis more efficiently (GUNN *et al*., 2016). Correction of serum sodium concentration should be slow. Rapid elevation of sodium can cause osmotic demyelination, resulting in permanent damage or neurological death and should be avoided. (NELSON; COUTO, 2015). The choice of fluid therapy for volume replacement of the patient was physiological 0.9%.

According to Mooney 2012, the presence of non-regenerative anemia with hematocrit between 20 and 35% caused by gastrointestinal hemorrhages. As observed in ANNEXES 3, 4, 6 and 7, the patient started treatment with hematocrit of 39% that decreased in all control exams reaching 25%.

Ultrasound examination is a noninvasive complementary examination that describes ultrasound findings of the adrenal glands. They may be atrophied or reduced in size, making them difficult to see. Animals carrying Addison's disease usually present with a diminished adrenal gland (KONIG, 2013). On abdominal ultrasound performed on the patient, the images revealed adrenal glands partially visible with regular contours and dimensions of 1.51cm long x 0.28 cm thick in the left gland. The right adrenal gland was not characterized. Another alteration observed on ultrasound was the thickening of the intestinal loops, mainly duodenum and jejunum (APPENDIX 1), leading to consider the hypothesis of inflammatory bowel disease and/or food hypersensitivity associated with hypoadrenocorticism.

The ACTH stimulation test is the gold standard test for the diagnosis of hypoadrenocorticism. Basal cortisol and cortisol should be measured after ACTH administration, which is lower than 2ug/dL, AH is confirmed, since there was no adrenal response to stimulation. Despite being a test with 100% sensitivity for hypoadrenocorticism, it does not allow to differentiate primary from secondary HA, however, it is not enough to differentiate between primary and secondary hypoadrenocorticism and, for this, sodium and potassium concentrations should be evaluated respectively (GUNN *et al*., 2016).

Most of the time the treatment should be instituted even before the confirmation of the diagnosis by ACTH stimulation tests and / or cortisol dosage, and should be based on clinical signs, biochemical dosages and ECG (VARGAS, 2015). A single initial dose of dexamethasone may be performed prior to the ACTH stimulation test. For nosocomial treatment after diagnosis, fludrocortisone acetate is the most commonly used mineralocorticoid supplement. The initial dose is 0.02 mg/kg/day, being divided into two doses and performed orally. The dose in the first 6 to 18 months of therapy should be the ceiling dose, and after this period, the dose reaches a plateau and stabilizes (NELSON; COUTO, 2015). Although the single daily dose is effective for some patients, others have persistent hyperkalaemia and/or hyponatremia until treatment is changed to twice-daily administration. However, initially, all patients should receive glucocorticoid plus mineralocorticoid until electrolytes and clinical signs stabilize. After the stabilization of the patient close to the clinical signs, only chronic therapy can be instituted (THOMPSON, 2018).

The drug protocol was instituted with hydrocortisone 5mg/kg/IV every 8 hours, omeprazole 1mg/kg/IV every 12 hours, dipyrone 25mg/kg/IV initially every 8 hours and every 12 hours 5 days later, enrofloxacin 5mg/kg/IV every 12 hours, ceftriaxone 30mg/kg/IV every 12 hours, tramadol 3mg/kg/IV initially every 8 hours and reduced to every 12 hours 3 days after and metoclopramide 0.3 mg/kg/SC every 8 hours.

Four days after initiation, fludrocortisone was restarted at a dose of 0.025 mg/kg/VO every 12 hours and prednisolone 0.2 mg/kg/VO every 12 hours. Fluid therapy with 0.9% saline solution was maintained at a dose of 3ml/kg/hour.

It can be stated that AH has a good prognosis if diagnosed and treated correctly. It is necessary that the person responsible for the patient is aware that the disease has no cure and that the patient needs constant clinical monitoring, and it is necessary to adjust doses and medications frequently. However, it is a controllable disease and patients have good quality of life after diagnosis, if treated rigorously and correctly.

5 FINAL CONSIDERATIONS

Hypoadrenocorticism is a difficult condition to diagnose depending on the moment of crisis/decompensation due to the use of corticosteroids in the emergency room and hospitalization environment. Its clinical manifestations are nonspecific and can often be confused with renal or gastroenteric disorders. This report reinforces the importance of considering hypoadrenocorticism as a differential diagnosis of patients with prerenal azotemia and gastroenteric signs and reiterates that the evaluation of patients with suspected AH can be confirmed with the measurement of the sodium:potassium ratio, however, the test of choice is the cortisol dosage in the ACTH stimulation test.

REFERENCES

BORIN-CRIVELLENTI, S. Endocrinologia. In: CRIVELLENTI, L.Z.; BORIN-CRIVELLENTI, S. Casos de Rotina em Medicina Veterinária de Pequenos Animais. 2. ed. MedVet: São Paulo, 2015.

FELDMAN, E.C.; PETERSON, M.E. Hypoadrenocorticism. Vet Clin North Am Small AnimPract, v. 14(4), p. 751-766, 1984.

FELDMAN, E. C., NELSON, R. W., Hypoadrenocorticism (Addison's disease) Canine andFeline Endocrinology and Reproduction, 3ª ed. p. 394-439. EUA: Elsevier. 2004.

FELDMAN, E. C.; NELSON, R. W.; REUSCH, C. E.; SCOTT-MONCRIEFF. J. C.; BEHREND, E. N. Canine and Feline Endocrinology. 4ed, p.492, St. Louis, 2015.

GALAC, S., REUSCH, C.E., KOOISTRA, H.S., RIJNBERK, A. ADRENALS. IN: RIJNBERK, A., KOOISTRA, H.S. (Ed.), Clinical Endcrinology of Dogs and Cats, 2ª ed., pp. 93-154 - Hannover: Springer Ilustrated, 2010.

GRECO, D. S. Hypoadrenocorticism in small animals. Clinical techniques in small animal practice, v. 22, n. 1, p. 32-35, 2007.GUYTON, A. C.; HALL, J. E. Hormônios Adrenocorticais.In: Tratado de fisiologia médica. 11 Ed. Saunders Elsevier, 2006.

GUNN E.; SHIEL R.E., MOONEY C.T. Hydrocortisone in the management of acute hypoadrenocorticism in dogs: a retrospective series of 30 cases Journal of Small Animal Practice, v. 57, p. 227–233, 2016 DOI: 10.1111/jsap.12473

KIMURA, Y., IWAKI, S., KAMESHIMA, S., ITOH, N. Um caso de hipoadrenocorticismo canino que necessita de transfusão de sangue para anemia aguda grave devido a hemorragia gastrointestinal. Journal of Veterinary Medical Science, 2019.

KLEIN, S. C.; PETERSON, M. E. Canine hypoadrenocorticism: part I. The canadian veterinaryjournal, v. 51, n. 1, p. 63, 2010. Klein, S.C., Peterson, M.E.,2010.

KLEIN, S. C.; PETERSON, M. E. Canine hypoadrenocorticism: part II. The Canadian Veterinary Journal, v. 51, n. 2, p. 179, 2010.

KOENIG, A. Endocrine Emergencies in Dogs and Cats Vet Clin Small Anim., v. 43 p. 869– 897, 2013

MELIAN, C., STEFANACCI, J., PETERSON, M.; KINTZER, P. Achados radiográficos em cães com hipoadrenocorticismo primário de ocorrência natural. Journal of the American Animal Hospital Association, 35 (3), 208-212, 1999.

MOONEY, C. T.; PETERSON, M. E. BSAVA Manual of Canine and Feline Endocrinology. 4rd Ed. Gloucester: British Small Animal Veterinary Medical Association, 2012:116-132.

MORAIS, H. A.; DIBARTOLA, S. P. Advances in Fluid, Electrolyte and Acid-Base Disorders,Vet Clin Small Anim, p 423-754, 2008.

NELSON, R.; COUTO, C. G. Medicina interna de pequenos animais. 5. Ed., Elsevier Brasil, 2015.

SINGER, M., DEUTSCHMAN, C., SEYMOUR, W.C., et al., The Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis – 3), 2016.

SHIEL, RE. E MOONEY, CT. Redefinindo o paradigma do hipoadrenocorticismo atípico em cães. Companion Animal, 24(3), 132-140, 2019.

REUSCH CE. Hipoadrenocorticismo. In: Ettinger SJ, Feldman EC, editores. Textbook of Veterinary Internal Medicine. 5ª ed. Filadélfia: WB Saunders. pp. 1488–1499, 2000.

SAITO, M., Olby, N. J., Obledo, L., Gookin, J. L. Muscle Cramps in Two Standard Poodles with Hypoadrenocorticism, Journal of the American Animal Hospital Association, v. 38, p 437-443, 2002.

THOMPSON, A. L. et al. Comparison of classic hypoadrenocorticism with glucocorticoid- deficient hypoadrenocorticism in dogs: 46 cases (1985–2005). Journal of the American Veterinary Medical Association, v. 230, n. 8, p. 1190-1194, 2007.

VARGAS, M. A. Hipoadrenocorticismo in: in: JERICÓ, M.; M. ANDRADE NETO, J. P. D; KOGIKA, M. M. Tratado de medicina interna de cães e gatos. 1ed, Rocca, Rio de Janeiro, 2015.

WAKAYAMA, JA, FURROW, E., MERKEL, LK,; Armstrong, PJ. Um estudo retrospectivo de cães com hipoadrenocorticismo atípico: um corte diagnóstico ou continuum? Journal of Small Animal Practice, 58 (7), 365-371, 2017.

ATTACHMENTS

ANNEX 1 - ULTRASONOGRAPHY PERFORMED AT ADMISSION

WeVets
Centro Veterinário 24H

NOME DO TUTOR	DADOS DO ANIMAL	
JULIA THEODORO DUARTE	Nº CADASTRO: 101452	RAÇA: SRD
VETERINÁRIO SOLICITANTE	NOME: PANTUFA	SEXO: FÊMEA
ISABELLY UTSONOMIA	ESPECÍE: CANINA	IDADE: 08ANOS

LAUDO ULTRASSONOGRÁFICO

FÍGADO: com dimensões mantidas, contornos regulares, bordas finas, ecogenicidade elevada (hepatopatia/alteração endócrina) e ecotextura homogênea. Vasos hepáticos apresentando trajeto e calibre de aspectos mantidos, em áreas passíveis de avaliação.
VESICULA BILIAR: em topografia habitual, repleção moderada, conteúdo anecogênico e moderada quantidade de material ecogênico (lama biliar), paredes normoespessas, mantidas e regulares.

BAÇO: em topografia habitual, dimensões discretamente reduzido, contornos definidos, margens regulares, ecogenicidade mantida e ecotextura homogênea. Nota-se áreas de mielolipoma adjacente a região hilar.

ESTOMAGO: em topografia habitual, distensão moderada, conteúdo gasoso, paredes espessas medindo aproximadamente 0,41 cm até 0,52 cm de espessura, mantidas e regulares, camada submucosa em maior evidência. (sugestivo de gastrite/processo inflamatório)

ALÇAS INTESTINAIS: em topografia habitual, distensão moderada, conteúdo predominantemente gasoso e mucoide, paredes espessas medindo aproximadamente 0,49 cm de espessura (segmento duodenal), 0,35 cm (segmento íleo) e 0,30 cm até 0,43 cm (segmento jejunal), mantidas e regulares, com camada submucosa em maior evidência e peristaltismo reduzido.
Cólon de aspecto regular e com espessamentos (mediu cerca de 0,29 cm – cólon descendente; 0,30 cm – cólon transverso e 0,34 cm – cólon ascendente de espessura), distendido por conteúdo líquido, medindo cerca de 1,58 cm de diâmetro. Na suspeita de corpo estranho recomenda-se controle. (sugestivo de processo inflamatório).

Nota-se as vezes em região de parede intraluminal do cólon descendente presença de estrutura tendendo a peduncular, hiperecogênica, homogênea, medindo cerca de 0,54 cm x 0,34 cm (A esclarecer)

RINS: em topografia habitual, simétricos (RE: 5,20 cm e RD: 5,45 cm), contornos regulares, cortical com ecogenicidade mantida, predominantemente homogêneos bilateralmente, mineralizações de recesso pélvico. Relação corticomedular de espessura preservadas. Sem sinais de litíase e ou processo hidronefrótico até o presente momento.

PANCREAS: lobo direito de pâncreas com ecogenicidade mantida, parênquima finamente grosseiro (sugestivo de pancreatopatia crônica) e dimensões mantidas, com cerca de 0,98 cm de espessura.

ADRENAIS: não caracterizadas (paciente com grande quantidade de conteúdo gasoso em alças intestinais).

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ANNEX 2 – CONTROL ULTRASONOGRAPHY PERFORMED 4 DAYS AFTER ADMISSION

WeVets
Centro Veterinário 24H

NOME DO TUTOR	DADOS DO ANIMAL	
JULIA THEODORO DUARTE	Nº CADASTRO: 101452	RAÇA: SRD
VETERINÁRIO SOLICITANTE	NOME: PANTUFA	SEXO: FÊMEA
JULIANA PETTI	ESPECÍE: CANINA	IDADE: 08ANOS

LAUDO ULTRASSONOGRÁFICO

FÍGADO: com dimensões mantidas, contornos regulares, bordas finas, ecogenicidade elevada (hepatopatia/alteração endócrina) e ecotextura homogênea. Vasos hepáticos apresentando trajeto e calibre de aspectos mantidos, em áreas passíveis de avaliação.
VESICULA BILIAR: em topografia habitual, repleção moderada, conteúdo anecogênico e moderada quantidade de material ecogênico (lama biliar), paredes normoespessas, mantidas e regulares.

BAÇO: dimensões mantidas, contornos definidos, margens regulares, ecogenicidade mantida e ecotextura homogênea. Nota-se áreas de mielolipoma adjacente a região hilar.

ESTOMAGO: em topografia habitual, distensão moderada, conteúdo heterogêneo (alimento), paredes normoespessas medindo aproximadamente 0,24 cm de espessura, mantidas e regulares.

ALÇAS INTESTINAIS: em topografia habitual, distensão moderada, conteúdo gasoso e mucoide, paredes espessas em segmento jejunal, medindo aproximadamente 0,38 cm até 0,45 cm, mantidas e regulares. (sugestivo de processo inflamatório).
Demais segmento com paredes normoespessas, medindo cerca de 0,43 cm de espessura (segmento duodenal), mantidas e regulares e peristaltismo reduzido.
Cólon de aspecto regular e sem espessamentos (mediu cerca de 0,16 cm – cólon descendente de espessura), distendido por conteúdo mucoide, com estruturas amorfas em permeio (alimento não digerido?). Na suspeita de corpo estranho recomenda-se controle.

RINS: em topografia habitual, simétricos (RE: 5,53 cm e RD: 5,61 cm), contornos regulares, cortical com ecogenicidade mantida, predominantemente homogêneos bilateralmente, mineralizações de recesso pélvico. Relação corticomedular de espessura preservadas. Sem sinais de litíase e ou processo hidronefrótico até o presente momento.

PANCREAS: lobo direito de pâncreas com ecogenicidade mantida, parênquima finamente grosseiro (sugestivo de pancreatopatia crônica) e dimensões mantidas, com cerca de 0,97 cm de espessura.

ADRENAIS: parcialmente visibilizada, apresentando contornos regulares, ecogenicidade mantida e dimensões com cerca de 1,51 cm (comprimento) x 0,28 cm (espessura de polo caudal) a esquerdo. Adrenal direita não caracterizada.

BEXIGA: em topografia habitual, distensão moderada, conteúdo anecogênico, paredes normoespessas medindo aproximadamente 0,16 cm de espessura, mantidas e regulares.

Sem sinais de linfonodomegalia. Ínfima quantidade de líquido livre adjacente aos segmentos intestinais e discreto aumento da ecogenicidade do mesentério.

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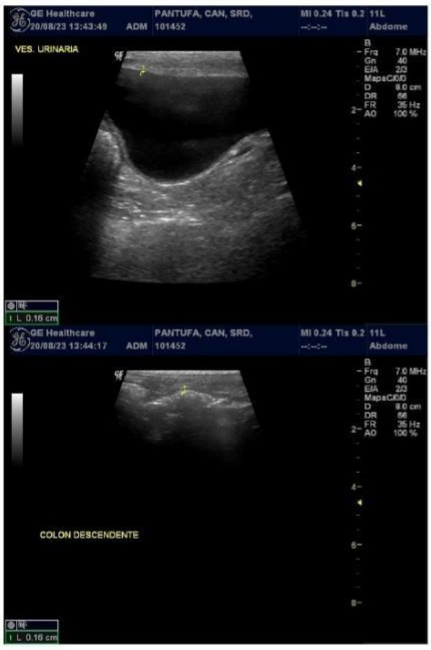
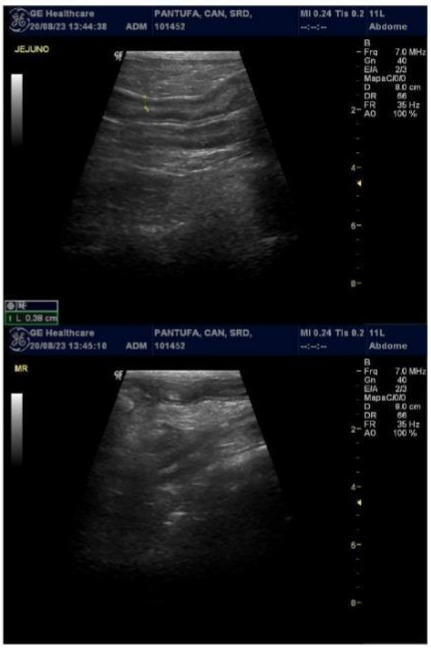
O estudo ultrassonográfico do sistema gastrintestinal é um sistema dinâmico, em constante movimentação, sendo assim, qualquer decisão sobre a terapêutica, pode indicar a realização de novo estudo ultrassonográfico e/ou exames complementares.

Em comparação com exame anterior do dia 15/08/23 houve melhora em relação a inflamação em segmento cólico e duodenal, com conteúdo mucoide. Com persistente inflamação em segmento jejunal e peristaltismo reduzido, com aumento da ecogenicidade do mesentério e ínfima quantidade de líquido livre.

Assinatura digital: Thais do Valle Araujo - CRMV/SP: 42377





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ANNEX 3 - BLOOD COUNT PERFORMED AT ADMISSION

HEMOGRAMA

MATERIAL:SANGUE TOTAL
MÉTODO..: AUTOMATIZADO/MICROSCOPIA
EQUIPAMENTO: MINDRAY BC 5000 VET

Eritrograma

	RESULTADOS		VALORES DE REFERÊNCIA
Eritrócitos:	6,22	milhões/mm³	5,70 a 7,40 milhões/mm³
Hemoglobina:	12,60	g/dl	14,00 a 18,00 g/dl
Hematócrito:	39	%	38,0 a 47,0 %
V.C.M.:	57,2	u³	63,00 a 77,00 u³
H.C.M.:	20,3	pg	21,00 a 26,00 pg
C.H.C.M.:	35,4	g/dl	31,00 a 36,00 g/dl
R.D.W:	14,2	%	12,00 a 16,00 %
Proteína total:	6.8	g/dl	6,0 a 8,0 g/dl
Eritroblastos:	2	%	
Observações série vermelha:	Discreta anisocitose por macrocitose e Policromasia		

Leucograma

	RESULTADOS				VLR. REF. ABSOLUTO	VLR. REF. RELATIVO
Leucócitos:	3.080	/mm³			6,0 a 16,0 mil/mm³	
Mielócitos:	0	%		/mm³	0 /mm³	0%
Metamielócitos:	1	%	31	/mm³	0 /mm³	0%
Bastonetes:	19	%	585	/mm³	0 a 200 /mm³	0 a 1 %
Segmentados:	71	%	2.187	/mm³	3300 a 12800 /mm³	55 a 80 %
Eosinófilos:	1	%	31	/mm³	100 a 1450 /mm³	1 a 9 %
Basófilos:	0	%	0	/mm³	0 /mm³	0 a 0 %
Linfócitos típicos:	5	%	154	/mm³	780 a 6400 /mm³	13 a 40 %
Linfócitos atípicos:	0,0	%	0	/mm³	0 /mm³	0 %
Monócitos:	3	%	92	/mm³	100 a 960 /mm³	1 a 6 %
Outros (*)		%		/mm³		
Observações série branca:	Moderada toxicidade dos neutrófilos; Moderada presença de neutrófilos hiposegmentados					

Contagem plaquetária:	278	mil/mm³	200 a 500 mil/mm³
Avaliação plaquetária:	Discreta presença de agregados plaquetários; Presença de macroplaquetas; Amostra com presença de fibrina		
Pesquisa de Hematozoário:	NÃO SOLICITADO		

Liberado por: Dr(a): Priscilla Alves Ribeiro - CRMV : CRMV-SP:50683 / SP

HEMOGRAMA

MATERIAL:SANGUE TOTAL
MÉTODO..: AUTOMATIZADO/MICROSCOPIA
EQUIPAMENTO: MINDRAY BC 5000 VET

DATA DA COLETA: 17/08/2023

Eritrograma

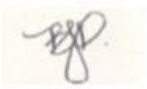
	RESULTADOS		VALORES DE REFERÊNCIA
Eritrócitos:	5,37	milhões/mm³	5,70 a 7,40 milhões/mm³
Hemoglobina:	10,40	g/dl	14,00 a 18,00 g/dl
Hematócrito:	34,0	%	38,0 a 47,0 %
V.C.M.:	57,9	u³	63,00 a 77,00 u³
H.C.M.:	19,4	pg	21,00 a 26,00 pg
C.H.C.M.:	33,4	g/dl	31,00 a 36,00 g/dl
R.D.W:	14,1	%	12,00 a 16,00 %
Proteína total:	5.0	g/dl	6,0 a 8,0 g/dl
Eritroblastos:	0	%	
Observações série vermelha:	Discreta anisocitose por macrocitose e Policromasia		

Leucograma

	RESULTADOS				VLR. REF. ABSOLUTO	VLR. REF. RELATIVO
Leucócitos:	3.530	/mm³			6,0 a 16,0 mil/mm³	
Mielócitos:	0	%		/mm³	0 /mm³	0%
Metamielócitos:	0	%	0	/mm³	0 /mm³	0%
Bastonetes:	0	%	0	/mm³	0 a 200 /mm³	0 a 1 %
Segmentados:	86	%	3.036	/mm³	3300 a 12800 /mm³	55 a 80 %
Eosinófilos:	0	%	0	/mm³	100 a 1450 /mm³	1 a 9 %
Basófilos:	0	%	0	/mm³	0 /mm³	0 a 0 %
Linfócitos típicos:	11	%	388	/mm³	780 a 6400 /mm³	13 a 40 %
Linfócitos atípicos:	0,0	%	0	/mm³	0 /mm³	0 %
Monócitos:	3	%	106	/mm³	100 a 960 /mm³	1 a 6 %
Outros (*)		%		/mm³		
Observações série branca:	Moderadapresença de neutrófilos hiposegmentados ; Moderada toxicidade dos neutrófilos					

Contagem plaquetária:	484	mil/mm³	200 a 500 mil/mm³
Avaliação plaquetária:	Discreta presença de agregados plaquetários; Plaquetas normais em morfologia		
Pesquisa de Hematozoário:	NÃO SOLICITADO		

Liberado por: Dr(a): Beatriz Jorge Paixão - CRMV : CRMV-SP:60127 / SP
Data da liberação: 17/08/2023 11:05:02



UREIA

MÉTODO..: CINÉTICO/COLORIMÉTRICO
MATERIAL:SORO SANGUÍNEO

RESULTADO: 73,5 mg/dL

Valor(es) de Referência: 10 - 60 mg/dL

Observações:

Liberado por: Dr(a): Beatriz Jorge Paixão - CRMV : CRMV-SP:60127 / SP

ALT (TGP)

MÉTODO..: CINÉTICO/COLORIMÉTRICO
MATERIAL:SORO SANGUÍNEO

RESULTADO: 68,4 U/L

Valor(es) de Referência: 7,0 - 92 U/L

Observações:

Liberado por: Dr(a): Beatriz Jorge Paixão - CRMV : CRMV-SP:60127 / SP

FOSFATASE ALCALINA

MÉTODO..: CINÉTICO/COLORIMÉTRICO
MATERIAL:SORO SANGUÍNEO

RESULTADO: 165,8 U/L

Valor(es) de Referência: 10 - 96 U/L

Observações:Resultado repetido e confirmado

Liberado por: Dr(a): Beatriz Jorge Paixão - CRMV : CRMV-SP:60127 / SP

CREATININA

MÉTODO..: CINÉTICO/COLORIMÉTRICO
MATERIAL:SORO SANGUÍNEO

RESULTADO: 0,70 mg/dL

Valor(es) de Referência: 0,5 a 1,6 mg/dL

Observações:

Liberado por: Dr(a): Beatriz Jorge Paixão - CRMV : CRMV-SP:60127 / SP

ALBUMINA

MÉTODO..: CINÉTICO/COLORIMÉTRICO
MATERIAL:SORO SANGUÍNEO

RESULTADO: 2,1 g/dL

Valor(es) de Referência: 2,3 - 3,8 g/dL

Observações:

Liberado por: Dr(a): Beatriz Jorge Paixão - CRMV : CRMV-SP:60127 / SP

ANNEX 6 - 2ND SERIAL BLOOD COUNT

HEMOGRAMA

MATERIAL:SANGUE TOTAL
MÉTODO..: AUTOMATIZADO/MICROSCOPIA
EQUIPAMENTO: MINDRAY BC 5000 VET

Eritrograma

	RESULTADOS		VALORES DE REFERÊNCIA
Eritrócitos:	4,40	milhões/mm³	5,70 a 7,40 milhões/mm³
Hemoglobina:	9,90	g/dl	14,00 a 18,00 g/dl
Hematócrito:	30,0	%	38,0 a 47,0 %
V.C.M.:	68,1	u³	63,00 a 77,00 u³
H.C.M.:	22,5	pg	21,00 a 26,00 pg
C.H.C.M.:	33,0	g/dl	31,00 a 36,00 g/dl
R.D.W:	13,9	%	12,00 a 16,00 %
Proteína total:	4.4	g/dl	6,0 a 8,0 g/dl
Eritroblastos:	0	%	
Observações série vermelha:	Discreta anisocitose por macrocitose; Discreta policromasia		

Leucograma

	RESULTADOS				VLR. REF. ABSOLUTO	VLR. REF. RELATIVO
Leucócitos:	13.870	/mm³			6,0 a 16,0 mil/mm³	
Mielócitos:	0	%		/mm³	0 /mm³	0%
Metamielócitos:	0	%	0	/mm³	0 /mm³	0%
Bastonetes:	0	%	0	/mm³	0 a 200 /mm³	0 a 1 %
Segmentados:	93	%	12.899	/mm³	3300 a 12800 /mm³	55 a 80 %
Eosinófilos:	0	%	0	/mm³	100 a 1450 /mm³	1 a 9 %
Basófilos:	0	%	0	/mm³	0 /mm³	0 a 0 %
Linfócitos típicos:	6	%	832	/mm³	780 a 6400 /mm³	13 a 40 %
Linfócitos atípicos:	0	%	0	/mm³	0 /mm³	0 %
Monócitos:	2	%	277	/mm³	100 a 960 /mm³	1 a 6 %
Outros (*)	0,0	%	0,0	/mm³		
Observações série branca:	Discreta presença de linfócitos reativos					

Contagem plaquetária:	556	mil/mm³	200 a 500 mil/mm³
Avaliação plaquetária:	Discreta presença de agregados plaquetários; Plaquetas normais em morfologia		
Pesquisa de Hematozoário:	NÃO SOLICITADO		

Liberado por: Dr(a): Ligia Pinho Cuccato - CRMV : CRMV-SP:35085 / SP

Ligia Pinho Cuccato

HEMOGRAMA

MATERIAL:SANGUE TOTAL
MÉTODO..: AUTOMATIZADO/MICROSCOPIA
EQUIPAMENTO: MINDRAY BC 5000 VET

Eritrograma

	RESULTADOS		VALORES DE REFERÊNCIA
Eritrócitos:	4,24	milhões/mm³	5,70 a 7,40 milhões/mm³
Hemoglobina:	8,40	g/dl	14,00 a 18,00 g/dl
Hematócrito:	25	%	38,0 a 47,0 %
V.C.M.:	58,5	u³	63,00 a 77,00 u³
H.C.M.:	19,8	pg	21,00 a 26,00 pg
C.H.C.M.:	33,9	g/dl	31,00 a 36,00 g/dl
R.D.W:	14,2	%	12,00 a 16,00 %
Proteína total:	4,0	g/dl	6,0 a 8,0 g/dl
Eritroblastos:	0	%	
Observações série vermelha:	Discreta anisocitose por macrocitose.		

Leucograma

	RESULTADOS				VLR. REF. ABSOLUTO	VLR. REF. RELATIVO
Leucócitos:	10.210	/mm³			6,0 a 16,0 mil/mm³	
Mielócitos:	0	%		/mm³	0 /mm³	0%
Metamielócitos:	0	%	0	/mm³	0 /mm³	0%
Bastonetes:	0	%	0	/mm³	0 a 200 /mm³	0 a 1 %
Segmentados:	87	%	8.883	/mm³	3300 a 12800 /mm³	55 a 80 %
Eosinófilos:	6	%	613	/mm³	100 a 1450 /mm³	1 a 9 %
Basófilos:	0	%	0	/mm³	0 /mm³	0 a 0 %
Linfócitos típicos:	4	%	408	/mm³	780 a 6400 /mm³	13 a 40 %
Linfócitos atípicos:	0,0	%	0	/mm³	0 /mm³	0 %
Monócitos:	3	%	306	/mm³	100 a 960 /mm³	1 a 6 %
Outros (*)		%		/mm³		
Observações série branca:	Morfologia celular normal					

Contagem plaquetária:	540	mil/mm³	200 a 500 mil/mm³
Avaliação plaquetária:	Discreta presença de agregados plaquetários; Plaquetas normais em morfologia		
Pesquisa de Hematozoário:	NÃO SOLICITADO		

Liberado por: Dr(a): Beatriz Correa dos Reis - CRMV : CRMV-SP:56188 / SP

Hemogasometria

Sangue Venoso Sangue Arterial

	Resultado	Valores de referência	
pH sanguíneo	7,449	7,34 a 7,46	7,36 a 7,44
PCO2	33,9mmHg	32 a 49 mmHg	36 a 44 mmHg
PO2	40mmHg	24 a 48 mmHg	90 a 100 mmHg
HCO3	23,5mEq/l	23 a 28 mEq/L	23 a 28mEq/L
TCO2	25mmol/L	24 a 29 mmol/L	16 a 25mmol/L
sO2	78%	93 a 100 %	
Na	109mmol/L	146 a 156 mmol/L	
K	4,7mmol/L	3,5 a 5,5 mmol/L	
iCa	1,13mmol/L	1,12 a 1,40mmol/L	
Glu	130mg/dl	60 a 115mg/dl	
Hct	36%	38 a 51%	
Hb	12,2g/dl	12 a 18g/dl	

Hemogasometria

Sangue Venoso Sangue Arterial

	Resultado	Valores de referência	
pH sanguíneo	7,443	7,34 a 7,46	7,36 a 7,44
PCO2	28,9mmHg	32 a 49 mmHg	36 a 44 mmHg
PO2	84mmHg	24 a 48 mmHg	90 a 100 mmHg
HCO3	19,8mEq/l	23 a 28 mEq/L	23 a 28mEq/L
TCO2	21mmol/L	24 a 29 mmol/L	16 a 25mmol/L
sO2	97%	93 a 100 %	
Na	121mmol/L	146 a 156 mmol/L	
K	3,6mmol/L	3,5 a 5,5 mmol/L	
iCa	1,09mmol/L	1,12 a 1,40mmol/L	
Glu	83mg/dl	60 a 115mg/dl	
Hct	27%	38 a 51%	
Hb	9,2g/dl	12 a 18g/dl	

Hemogasometria

Sangue Venoso Sangue Arterial

Sangue Venoso Sangue Arterial

	Resultado	Valores de referência	
		Sangue Venoso	Sangue arterial
pH sanguineo	7,4	7,34 a 7,46	7,36 a 7,44
PCO2	37,1mmHg	32 a 49 mmHg	36 a 44 mmHg
PO2	47mmHg	24 a 48 mmHg	90 a 100 mmHg
HCO3	23,4mEq/l	23 a 28 mEq/L	23 a 28mEq/L
TCO2	25mmol/L	24 a 29 mmol/L	16 a 25mmol/L
sO2	83%	93 a 100 %	
Na	124mmol/L	146 a 156 mmol/L	
K	4,1mmol/L	3,5 a 5,5 mmol/L	
iCa	1,15mmol/L	1,12 a 1,40mmol/L	
Glu	96mg/dl	60 a 115mg/dl	
Hct	21%	38 a 51%	
Hb	7,1g/dl	12 a 18g/dl	

Hemogasometria

Sangue Venoso Sangue Arteria

	Resultado	Valores de referência	
pH sanguineo	7,456	7,34 a 7,46	7,36 a 7,44
PCO2	34mmHg	32 a 49 mmHg	36 a 44 mmHg
PO2	50mmHg	24 a 48 mmHg	90 a 100 mmHg
HCO3	24mEq/l	23 a 28 mEq/L	23 a 28mEq/L
TCO2	25mmol/L	24 a 29 mmol/L	16 a 25mmol/L
sO2	87%	93 a 100 %	
Na	129mmol/L	146 a 156 mmol/L	
K	3,5mmol/L	3,5 a 5,5 mmol/L	
iCa	1,13mmol/L	1,12 a 1,40mmol/L	
Glu	101mg/dl	60 a 115mg/dl	
Hct	23%	38 a 51%	
Hb	7,8g/dl	12 a 18g/dl	

Hemogasometria

Sangue Venoso Sangue Arterial

	Resultado	Valores de referência	
pH sanguíneo	7,558	7,34 a 7,46	7,36 a 7,44
PCO2	26,8mmHg	32 a 49 mmHg	36 a 44 mmHg
PO2	53mmHg	24 a 48 mmHg	90 a 100 mmHg
HCO3	23,9mEq/l	23 a 28 mEq/L	23 a 28mEq/L
TCO2	25mmol/L	24 a 29 mmol/L	16 a 25mmol/L
sO2	92%	93 a 100 %	
Na	132mmol/L	146 a 156 mmol/L	
K	3,1mmol/L	3,5 a 5,5 mmol/L	
iCa	1,06mmol/L	1,12 a 1,40mmol/L	
Glu	110mg/dl	60 a 115mg/dl	
Hct	20%	38 a 51%	
Hb	6,8g/dl	12 a 18g/dl	

Hemogasometria

Sangue Venoso Sangue Arterial

	Resultado	Valores de referência	
pH sanguíneo	7,539	7,34 a 7,46	7,36 a 7,44
PCO2	24,2mmHg	32 a 49 mmHg	36 a 44 mmHg
PO2	160mmHg	24 a 48 mmHg	90 a 100 mmHg
HCO3	20,6mEq/l	23 a 28 mEq/L	23 a 28mEq/L
TCO2	21mmol/L	24 a 29 mmol/L	16 a 25mmol/L
sO2	100%	93 a 100 %	
Na	126mmol/L	146 a 156 mmol/L	
K	4,9mmol/L	3,5 a 5,5 mmol/L	
iCa	1,16mmol/L	1,12 a 1,40mmol/L	
Glu	100mg/dl	60 a 115mg/dl	
Hct	21%	38 a 51%	
Hb	7,1g/dl	12 a 18g/dl	