

Adrenocortical insufficiency syndrome in endurance horse

Síndrome de Insuficiencia Adrenocortical en un caballo de resistencia

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Abstract

At last 5Km in the endurance competition a gelding (Australian Stock Horse) with 10-years-old showed poor performance and exhausted. On clinical examination the horse presented exhausted, depression, lethargy and anorexia. After start the treatment the horse death. On necropsy showed multiples hemorrhages in the adrenal cortex and renal cortical with papillary necrosis, acute tubular necrosis. Histopathology showed: hemorrhages in adrenal cortex, capsule thickening and atrophy cortical with coagulation necrosis of zona glomerulosa, coagulation necrosis focal zona fasciculata, only congestion of the zona reticularis. In conclusion, we report a case of adrenocortical insufficiency syndrome in a resistance horse with rapid development of clinical signs, without response to emergency treatment, confirmed with laboratory, necropsy and histopathology results.

Keywords: Adrenocortical, Endurance, Horse, Let down

Resumen

En los últimos 5Km en la competencia de resistencia, un caballo castrado (Australian Stock Horse) con 10 años de edad mostró un rendimiento pobre y agotado. En el examen clínico el caballo presentaba agotamiento, depresión, letargo y anorexia. Luego de iniciar el tratamiento el caballo murió. En la necropsia se evidenciaron múltiples hemorragias en la corteza adrenal y zona cortical renal con necrosis papilar y necrosis tubular aguda. La histopatología mostró: hemorragias en la corteza adrenal, engrosamiento y atrofia de la cápsula cortical con necrosis por coagulación de la zona glomerulosa, necrosis por coagulación focal de la zona fasciculata y congestión de la zona reticularis. En conclusión, reportamos un caso de síndrome de insuficiencia adrenocortical en un caballo de resistencia con rápido desarrollo de signos clínicos, sin respuesta a tratamiento de emergencia, confirmado con resultados de laboratorio, necropsia e histopatología.

Palabras claves: Adrenocortical, Resistencia, Caballos, Colapso

Introduction

Endurance horses cope well with the stress and physical demands of endurance racing. Many of the changes in blood biochemistry seem to be grounded in a dehydration, increased energy expenditure, and exercise related muscular damage (Larsson et al., 2013). Veterinary inspection of horses at endurance rides evaluates the clinical status of horses to determine their fitness to start and continue in the event, assessing heart rate and his recovery, mucous membrane colour and capillary refill, skin recoil, gastrointestinal sounds and muscle pliability, along with physical signs of injury or lameness (Barnes et al., 2010). In equine long-distance endurance competitions, nearly 40% of the starters are eliminated at a veterinary control, and the majority for metabolic reasons 15% (Younes et al., 2016). Elimination for metabolic reasons was related to signs of dehydration and electrolyte imbalance; findings consistently recorded for those VOM (Vetted Out Metabolic) were slowed capillary refill, slowed skin recoil time (2–3 s for both) and gastrointestinal sounds scored as very reduced or absent (Barnes, et al., 2010). Adrenal exhaustion or “let down” syndrome are much discussed, poorly documented syndromes ascribed to adrenal insufficiency in the horse (Smith, 2009). Iatrogenic Equine Metabolic Syndrome in Thoroughbred associated with overdose and chronic dexamethasone and triamcinolone (Morales et al., 2009) has been described in horses. Chronic administration of corticoids can also lead to adrenocortical insufficiency (Smith, 2009). A syndrome of adrenal exhaustion resulting in lethargy, anorexia, and poor performance is also anecdotally described in race horses, and has been attributed to adrenal insufficiency associated with long-term steroid administration or chronic stress (Hart & Barton, 2011). Adrenal insufficiency should be considered in the differential diagnoses of horses with depression, anorexia, weight loss, hyponatremia, hypochloremia, hyperkalemia, or hypoglycemia, particularly if the horse has recently come off the track or some other form of intensive training or corticosteroids have been administered (Smith, 2009). However, measurement of basal cortisol concentrations and adrenocorticotrophic hormone (ACTH) stimulation testing in race and endurance horses has not provided convincing evidence for adrenal insufficiency in these equine athletes (Hart & Barton, 2011). Low cortisol levels have not been found in racehorses that turn in poor performances blamed on adrenal exhaustion, abnormal response to ACTH challenge has not been noted in endurance horses after 22-4-Km (36-mile) rides, low circulation cortisol levels have been noted after 160-Km (100-mile) rides (Smith, 2009). An ACTH stimulation test can be used to confirm the diagnosis; cortisol should increase two fold to threefold 2 to 4 hours after stimulation (Smith, 2009). The aim of this study is to report a case of adrenocortical insufficiency syndrome in endurance horse.

Report of case

At last 5Km in the endurance competition (80Km, Qualifier Dubai International Endurance City-FEI), a gelding (Australian Stock Horse) with 10-years-old showed poor performance and exhausted and a sustained heart rate of 82bpm at Veterinary inspection, and the horse was stopped and disqualified of the race. No medication was administered before the race, the horse has not presented previous episodes of fatigue or exhausted during training. Clinical signs: On clinical examination the horse presented exhausted, depression, lethargy and anorexia; the following values: temperature: 99.5-102.1°F (39.9°C), pulse: 82 bpm, respiration (breathing rate): 45 bpm, mucous membranes (gums): Moist congested, capillary refill time: three seconds or less. Hematology and biochemistry: evidence hyperproteinemia (418 g/dL or g/L), Na: 142mmol/L (Normal 138 mmol/L, mean 134-142), hypopotassemia (2.6mmol/L), hyperchloremic (105mmol/L), iCa: 1.69mmol/L (Normal 1.29 mmol/L (mean of 1.19-1.35), TC02: 25mmol/L, hyperglycemia 8.05 mmol/L., (normal 4.9,mean of 4.3-5.5 mmol/L), BUN: 23mg/dL (mean 8 -27 mg/dL), Crea: 150,31µmol/L.,(normal is 125, mean of 85-165, Hct: 46% PCV, HB15.6g/dL (normal 13.5g/dL., (mean of 11.1-15.9) and Anion Gap: 16mmol/L (normal 19,3 mmol/L (mean 15,4-23,4). Treatment: The horse was treated with emergency hydration intravenous (Lactated Ringer’s solution) 20liters, injectable solution: Commercial name: Duphalyte 500ml (vitamins: thiamine hydrochloride (vit B1), riboflavin (as riboflavin sodium phosphate) (vit B2), pyridoxine hydrochloride (B6), cyanocobalamin (vit B12), nicotinamide, dextranthenol, electrolytes: calcium chloride hexahydrate, magnesium sulphate heptahydrate, potassium chloride, amino acids: arginine, cysteine, anhydrous dextrose), Vitamin B complex, electrolytes, amino acids and dextrose 500ml, glucose 5% 100ml. After 4 hours of treatment the horse showed greater degree of depression, lethargy, let down and collapse. The following values: temperature: 97.52°F (36.4°C), pulse: 67 bpm, respiration (breathing rate): 36 breaths per minute, mucous membranes (gums): Moist congested, capillary refill time: four seconds or less. Biochemistry: selected evidence: BUN: 25mg/dL, Crea: 1.8mg/dL and Cortisol (as 115,03 mmol/L., normal 136 (mean 71-240mmol/L). The treatment was maintained, additionally, was administered via intravenous flunixin meglumine (1.1mg/ml), but after two hours of the last biochemical test the horse died. Necropsy: On necropsy showed Zenker degeneration was observed (rhabdomyolysis in the specific muscle groups affected included the caudal aspect of the neck and shoulder: trapezius, brachialis, rhomboideus, subscapularis, triceps, and pectoralis muscles, hindquarters: psoas major, gluteal, rectus femoris, gracilis, semimembranosus, and semitendinosus muscles), myocardium with diffuse or multifocal pale areas with ecchymoses, full-thickness white streaks extending through the myocardium of the ventricles, and focal areas of hemorrhage, multiples hemorrhages in the adrenal cortex (ecchymosis and petechiae) and renal cortical and papillary necrosis, acute tubular necrosis. Histopathology: Histologically,

Zenker's degeneration, characterized by loss of cross-striations, fiber swelling, hypereosinophilia of the sarcoplasm, hyaline fragmentation of the sarcoplasm of individual muscle fibers, and pyknotic nuclei were seen. Myocardium: moderate to severe myocardial degeneration and necrosis without inflammatory infiltrates and acute, focally extensive subendocardial hematomas without evidence of myocardial degeneration. Hemorrhages in adrenal cortex, capsule thickening and atrophy cortical with coagulation necrosis of zona glomerulosa, coagulation necrosis focal zona fasciculata, only congestion of the zona reticularis. Renal cortical and medullary necrosis, acute tubular necrosis, degeneration vacuolar and glycogen nephrosis, glomerulonephritis membranous by myoglobinuria. The decreased blood volume (dehydration) is primarily the result of sweat loss and a low fluid intake. Significant changes in muscle parameters (Creatine kinase –CK, aspartate aminotransferase –AST and Creatinine) were found in endurance races (Larsson et al., 2013). Cortisol insufficiency can result from Hypothalamic-pituitary-adrenal axis (HPA) impairment at one or several levels, and may be transient or permanent (Hart & Barton, 2011). Severe adrenocortical hemorrhage and necrosis resulting in adrenal insufficiency – Waterhouse-Friderichsen syndrome – is also described in both people and horses with septic and endotoxic shock, and is believed to result from vascular derangements and ischemia associated with the primary disease (Hart & Barton, 2011). In general, horses with adrenal insufficiency have a history of depression, anorexia, exercise intolerance, weight loss, bad hair or lameness (Satué-Ambrojo et al., 2017). At necropsy it is more common to find enlarged adrenal gland in racehorses than it is to find atrophic glands (Smith, 2009). Multiple hemorrhages in the adrenal cortex and renal cortical and papillary necrosis, acute tubular necrosis was report in Iatrogenic Equine Metabolic Syndrome in Thoroughbred horses (Morales et al., 2009). Histologically, renal cortical and medullary necrosis, acute tubular necrosis, degeneration vacuolar and glycogen nephrosis, glomerulonephritis membranous and hemorrhages in adrenal cortex, capsule thickening and atrophy cortical with coagulation necrosis of zona glomerulosa, coagulation necrosis focal zona fasciculata and coagulation necrosis, congestion of the zona reticularis (Morales, et al., 2009), the same characteristics showed in this case. However, this case of adrenocortical insufficiency syndrome in endurance horse was presented acutely, possibly associated with the stress of the exercise, duration and hydroelectrolytic imbalance. The hormonal response during exercise is also influenced by hemodilution or hemoconcentration actions related to the displacement of plasma fluids inside and outside the vascular beds, a greater secretion of CORT can be expected during and after exercise on horses during resistance competitions (Satué-Ambrojo et al., 2017). Unfortunately hematological and seric levels were not evaluated before the race, the cortisol level was only taken at one time, however the macroscopic lesions observed during the necropsy and the histological observations of the adrenal glands are suggestive of a adrenal insufficiency acute. Cortisol (CORT) is frequently used to assess stress levels induced by exercise, different studies have been carried out in relation to stress in horses such as the load

stress in tow, participation in equestrian dressage competition, competition of resistance, jumping, tourist driving and education, It has been shown that moderate exercise in horse increases CORT by up to 29% compared to baseline levels through the stress response, also, the plasma concentration of CORT was more than double the normal value 60 minutes after exercise (Satué-Ambrojo et al., 2017). Additionally, the compartmentalization syndrome (rhabdomyolysis-myoglobinuria), contributed to the hydroelectrolytic imbalance and the severe damage of the adrenal cortex with hemorrhage and dysfunction. Cumulative doses of dexamethasone and triamcinolone induce EMS, possibly associated with training, and career management practices (Morales et al., 2009). This hypertrophy of may be the result of repeated administration of exogenous ACTH, which in turn could lead to “let down” when injections are discontinued, or it may be caused by chronic stress (Smith, 2009). However, the horse was not previously medicated with corticosteroids or any other medication, according to its clinical history. The multiple organ failures complicating the clinical and compromises the horses life (Morales et al., 2009).

Conclusion

In conclusion, we report a case of adrenocortical insufficiency syndrome in a resistance horse with rapid development of clinical signs, without response to emergency treatment, confirmed with laboratory, necropsy and histopathology results. This case represents an important precedent for the need for veterinary inspection during endurance horse racing, not only at veterinary checkpoints.

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