Equine metabolic syndrome and atheroesclerosis in a mare American Quarter Horse

Abelardo Morales[1]; Francisco García; Diana Villoria[1]; David Fernández[2]; Gerardo Campos[2]; Hector Zerpa[2],
[1]Department of Veterinary Pathology, [2]Veterinary Hospital Dr. Daniel Cabello Section Large Animal
Faculty of Veterinary Science Central University of Venezuela Maracay, Aragua State Venezuela.
Autor para correspondencia: Abelardo Morales Briceño Email: aamorales13@gmail.com
Department of Veterinary Pathology, Faculty of Veterinary Science
Central University of Venezuela Maracay, Aragua State Venezuela

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SUMMARY

The aim of this study was to describe the presence of atherosclerosis associated with Equine Metabolic Syndrome in a mare American Quarter Horse. Were studied a mare American Quarter Horse of 8 years old, with history of Equine Metabolic Syndrome, collapse, shock and sudden death. On necropsy, were observed dermatitis alopecic with hyperpigmentation and xantomathosis of subcutaneous tissue. In the abdominal cavity was observed hemoperitoneum severe by rupture of the aorta flow. In the wall of the aorta was observed fat deposits and plates type atheroma. Histopathology revealed severed fatty degeneration and hepatic necrosis. The aorta showed lipid-filled foam cell stage (fatty streak) into an advanced, complicated lesion that contains abundant extracellular cholesterol ester in the atheromatous gruel within the arterial intima. In conclusion were reported atherosclerosis associated the equine metabolic syndrome a mare American Quarter horses.

Key words: AQH, atherosclerosis, EMS, equine.

RESUMEN

El objetivo de este estudio fue describir la presencia de aterosclerosis asociada con el síndrome metabólico equino en una yegua Cuarto de Milla Americano. Se estudió una yegua Cuarto de Milla, de 8 años de edad, con antecedentes de síndrome metabólico equino, colapso, shock y muerte súbita. En la necropsia, se observó alopecia dermatitis con hiperpigmentación y xantomatosis del tejido subcutáneo. En la cavidad abdominal se observó hemoperitoneo masivo severo por la ruptura de la arteria aorta. En la pared de la aorta se observó depósitos de grasa y placas de ateromas. La histopatología reveló degeneración grasa y necrosis hepática. La aorta evidenció las paredes con depósitos de lípidos, células espumosas (estría grasa) en una lesión avanzada y complicada que contienen ésteres de colesterol y abundante placas ateroma en la íntima arterial. En conclusión se informó de la aterosclerosis asociada al síndrome metabólico equino un caballo yegua Cuarto de Milla Americana.

Palabras clave: AQH, ateroesclerosis, EMS, equino.

INTRODUCTION

Equine Metabolic Syndrome (EMS) is a severed disease that includes obesity, insulin resistance and laminitis (Eustace, 2008; Donald, 1996). In EMS peripheral adipocytes synthesize adipokines which are analogous to cortisol, resulting in Cushing syndrome like-signs that include obesity, laminitis and insulin resistance (Eustace, 2008, Radin, et al., 2009; Rosenfeld, et al., 2002). The principal components of EMS are increased adiposity, insulin resistance but the production of adipokines by adipocytes and the ability to convert circulating cortisone to cortisol results in clinical manifestations that are similar to horses with Cushing's syndrome additional present laminitis. Obesity is observed in the majority of cases, insulin resistance, hyperinsulinemia and hypertriglyceridemia are components of an equine metabolic syndrome phenotype associated with increased laminitis risk in horses (Eustace, 2008; Geor, Frank, 2009). Atherosclerosis can be defined as the development of abnormal fat deposits in the artery wall. Atherosclerotic lesions are believed to progress through a focal, lipid-filled foam cell stage into an advanced, complicated lesion that contains abundant extracellular cholesterol ester in the atheromatous gruel within the arterial intima (Radin, et al., 2009). There are few reports in the literature of the development of atherosclerosis associ-

ated with EMS. The aim of this study was to describe the presence of atherosclerosis associated with Equine Metabolic syndrome in a mare American Quarter Horse.

MATERIALS AND METHODS

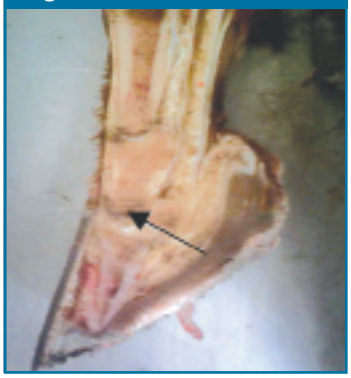
Were studied a mare American Quarter Horse of 8 years old, with history of Equine Metabolic Syndrome (dermatitis alopecic with hyperpigmentation, obesity and chronic laminitis), collapse, shock and sudden death. Bloods sample were collected previous at mortem. Were examined by necropsy and samples of tissue were recollected (Aluja y Constantino, 2002; Banks, 1996). The tissue samples of tissue were fixed in formalin buffered 10% and processed by conventional H&E techniques (Banks, 1996). The concentrate diet was evaluated.

RESULTS

On necropsy, were observed dermatitis alopecic with hyperpigmentation and xantomathosis of subcutaneous tissue. In the abdominal cavity was observed hemoperitoneum severe by rupture of the aorta flow. In the wall of the aorta (intima) was observed fat deposits of plates type atheroma (Figure 2). Multiples hemorrhages in the adrenal cortex. Liver presented fibrosis chronic. Multifocal necrotic areas were present in the other lobes and telangiectasy. Renal cortical and papillary necrosis. Spleen presented severed congestion and hemorrhage. The stomach presented Gastric ulcer syndrome severed, Grade 3 (Merrit, 2003), petechiae epicardial hemorrhage. Histopathology study revealed severed fatty degeneration and hepatic necrosis. Hemorrhage 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. Acute tubular necrosis, vacuolar degeneration and glycogen nephrosis, glomerulonephritis membranous. Spleen germinal center development within the lymphoid follicles should be noted as decreased. Reactive extramedullary hematopoiesis may be seen in conjunction with conditions that target the destruction of lymphocytes. Decreased cellularity of the lymphoid follicles, marginal zone and red pulp region were presented. Chronic gastritis surface, erosion focal and hyperkeratosis infiltrated of lymphocytes in the lamina propia. The aorta showed lipid-filled foam cell stage (fatty streak) into an advanced, complicated lesion that contains abundant

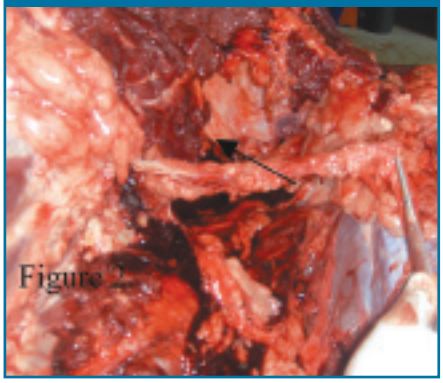
extracellular cholesterol ester in the atheromatous gruel within the arterial intima (Figure 3 & 4). Laminitis was observed cause failure of the hoof-distal phalanx bond the epidermal lamellae are stretched beyond their normal limits and significant epidermal lamellar necrosis (Figure 1). Hemoglobin 52 g/L, Hematocrit 28 %, Protein 25 g/L, Leucocytes 5,3 x 10^9/L. Urea Nitrogen 21 mmol/L, Creatinine 130 µmol/L, Glucosa 8,8 mmol/L, cholesterol 5,4 mmol/L, Cortisol 450 nmol/L, Bilirrubin Total 25,2-29,5 µmol/L, Unconj 18,2 µmol/L, Conj 11,3 µmol/L. Alkaline phosphatase 61,0 U/L, Lactate dehydrogenase 17,7 U/L, Sorbitol dehydrogenase 2,2 U/L, Creatin Phosphokinase 48,1 U/L, Transaminases Aspartate amino 167,2 U/L, Alanine amino 3,8 UL. Albumin 22 g/L, Globulin 21 g/L. Concentrate diet (commercial) was based on 15% crude protein, crude fat 3.5%, crude fiber 12% and EIN 46%.

Figure 1



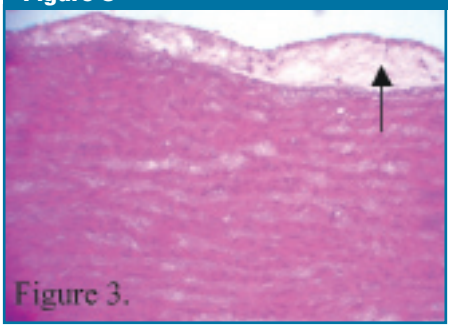
Laminitis of equine with Equine Metabolic Syndrome failure of the hoof-distal phalanx bond the epidermal lamellae are stretched beyond their normal limits and significant epidermal lamellar necrosis.

Figure 2



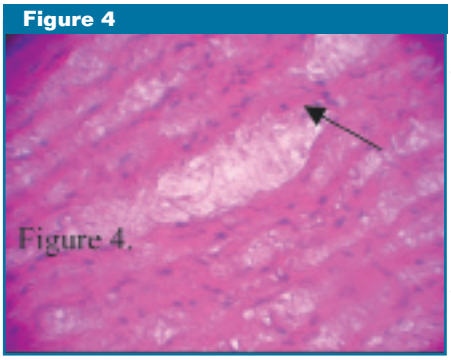
Rupture of the aorta flow.

Figure 3



The aorta showed lipid-filled foam cell stage (fatty streak) into an advanced, complicated lesion that contains abundant extracellular cholesterol ester in the atheromatous gruel within the arterial intima. (hematoxylin and eosin method 10X).

Figure 4



The aorta showed lipid-filled foam cell stage (fatty streak) into an advanced, complicated lesion that contains abundant extracellular cholesterol ester in the atheromatous gruel within the arterial intima. (hematoxylin and eosin method 20X).

DISCUSSION

The equine metabolic syndrome (EMS) refers to several ailments are similar to horses with Cushing's syndrome. Obesity, insulin resistance, hyperinsulinemia and hypertriglyceridemia, laminitis of common clinical sign. Atherosclerosis is known to occur in many arteries throughout the body, although the association between atherosclerosis extent in different arteries is variable (Eustace, 2008; Donald, 1996; Jubb, et al., 1984). In human subjects, atherosclerosis in the coronary arteries is the underlying cause of CHD (coronary heart disease) with its associated mortality from myocardial infarction (Radin, et al., 2009). For many decades that have passed, there are still few animal models that reproducibly exhibit the high frequency of spontaneous atherosclerotic plaque rupture and occlusive thrombosis that occur in humans (Lawrence, 1999). Not one of the titles of the thousands of citations retrieved includes any reference to plaque rupture or intra-plaque hemorrhage (Lawrence, 1999). This doesn't mean that plaque rupture never occurs in nonhuman primates or rabbits, but the absence of references to plaque rupture reflects how atherosclerotic lesions develop and the limitations imposed on modeling this disease process (Lawrence, 1999). Atherosclerosis progresses episodically and may involve multiple stresses, including responses to infection and inflammation both within the artery and at other locations (Lawrence, 1999). Abnormal production or regulation of adipokines occurs in obese individuals and is implicated in the development of a variety of associated co-morbidities including metabolic syndrome, type 2 diabetes, atherosclerosis, heart disease, and cancer in people, although evaluation in domestic species is just beginning (Radin, et al., 2009). Adipokines are now being examined as potential biomarkers for risk assessment for development of complications related to obesity (Radin, et al., 2009). Slowly growing plaques gradually accumulate lipid within foam cells; proliferation of smooth muscle cells and elaboration of intracellular matrix produce the definitive fibrous plaque. In general, such plaques tend to have adherent endothelial layers that are not prone to sudden disruption with associated activation of coagulation. The plasma level of cholesterol is determined by genetic factors, by the type and amount of fat in the diet, and by other factors such as obesity, physical activity, and disease states. Based on the results of animal studies, epidemiologic data, and interventional studies, there is good evidence for an association between hypercholesterolemia and atherosclerosis. The main causes of reduced HDL cholesterol include: obesity, physical inactivity, androgenic and related steroids (including anabolic steroids), beta-blocking agents, hypertriglyceridemia, and genetic factors. In contrast, weight reduction, exercise and some medications elevate HDL cholesterol levels. Nutrition and physical activity play a primary role in the development of equine metabolic syndrome. It is necessary to reduce the percentage of protein and carbohydrates in the diet of horses with predisposition, with little physical activity. Kidney lesions are suggestive of previous episodes of rhabdomyolysis, myoglobinuria, as well as formation and deposition of immune complexes in the renal glomerulus, possibly associated with post vaccination response (VEE, EEE, WEE, HVE1, HVE4). With respect to glands adrenal lesions are suggestive of chronic stress and do not rule out a possible iatrogenic origin. Generating a systemically hypertensive disorders affecting the vascular system, with emphasis on the palmary digital arteries which are susceptible to changes in blood pressure as well as large vessels like the aorta. Susceptibility to EMS may be established from before birth and obesity develops in some horses as soon as they reach maturity. In conclusion were reported atherosclerosis associated an equine metabolic syndrome an a mare American Quarter horses. This is the first report of the development of atherosclerosis in a horse associated with equine metabolic syndrome.

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