A Retrospective Study of Mortality Causes in Arabian Horses

Abelardo Morales-Briceño

SHS Veterinary Center, Al Wathba, Abu Dhabi, United Arab Emirates, aamorales13@gmail.com

Follow this and additional works at: https://ciencia.lasalle.edu.co/mv

Citación recomendada


This Artículo is brought to you for free and open access by the Revistas científicas at Ciencia Unisalle. It has been accepted for inclusion in Revista de Medicina Veterinaria by an authorized editor of Ciencia Unisalle. For more information, please contact ciencia@lasalle.edu.co.
A Retrospective Study of Mortality Causes in Arabian Horses

Abelardo Morales-Briceño

Received: 24 May 2020, Approved: 28 June 2020, Versión Online First: 18 August 2020

How to cite this article: Morales-Briceño A. A Retrospective Study of Mortality Causes in Arabian Horses. Rev Med Vet. 2020;(41). https://doi.org/10.19052/mv.vol1.iss41.3

Abstract

This study aims to describe the causes of death in Arabian horses at Al Wathba, in Abu Dhabi (United Arab Emirates) during 2018-2019. This study was conducted in a cohort of all Arabian horses that died. Data was collected retrospectively and only those horses with a full necropsy report available were included in the study. The carcass and all internal organs of each horse were examined and representative samples of tissues with abnormalities were collected in 10% neutral buffered formalin and processed for histopathological examination. A descriptive statistical analysis was performed. A total of 25 Arabian horses were examined post-mortem. Out of them, 35% were females. In general, colic occurred specifically at a higher frequency (80%), cases of collapse and sudden death during exercise and races occurred in 8%, cases of perinatal death in 8% and one case of laminitis (4%). The annual mortality in relation to the estimated population was 1% approximately. In conclusion, we identified and described the causes of death in Arabian horses in Al Wathba, Abu Dhabi, UAE during the period 2018-2019. In summary, a pathology of the digestive system — specifically acute abdominal crisis (colic) — is the main cause of death in Arabian horses, with 80%; other causes of mortality with a low incidence were in order collapse and sudden death during exercise and races, perinatal death and laminitis. These results allow establishing risk prevention measures of mortality in horses for specific activities like handling, feeding, training and races.

Keywords: Arabian, horses, colic, mortality, sudden death.

Introduction

The Arabian horse has been part of the history and culture of the Middle-East countries, especially the United Arab Emirates. Al Wathba has had a great development of horse stables in recent years with an estimated population of approximately 2500 horses in the endurance disciplines (85%), Other activities include show and breeding (8%), flat races and breeding (3%), enjoyment (2%) and jumping / 2%.

---

1 Research article.
2 SHS Veterinary Center, Al Wathba, Abu Dhabi, United Arab Emirates. aamorales13@gmail.com https://orcid.org/0000-0002-9122-9251
Anatomical considerations for the Arabian horse: some Arabians, though not all, have 5 lumbar vertebrae instead of the usual 6, and 17 pairs of ribs rather than 18. Specifically, in Arabian horses, some pathologies associated with hereditary disorders have been described, which are briefly mentioned below. Genetic disorders considerations for the Arabian horse (1): Severe Combined Immunodeficiency (SCID): recessive disorder, fatal when homozygous, carriers (heterozygotes) show no signs. Affected foal is born with a complete lack of an immune system and, thus, generally dies after an opportunistic infection, usually before getting 3 months old. There is a DNA test available to detect healthy horses who are carriers of the gene causing SCID, thus testing and carefully planned matings can now eliminate the possibility of an affected foal ever being born. Lavender Foal Syndrome (LFS): also called Coat Color Dilution Lethal (CCDL). Recessive disorder, fatal when homozygous, carriers show no signs. The condition was named this way because most affected foals are born with a coat color dilution that lightens the tips of the coat hairs, or even the entire hair shaft. Foals with LFS are unable to stand at birth, often have seizures, and are usually euthanized within a few days of birth.

In November 2009, Cornell University announced that a DNA test has been developed to detect carriers of LFS (1). Simultaneously, the University of Pretoria also announced that they had also developed a DNA test. Cerebellar abiotrophy (CA or CCA): recessive disorder, homozygous horses are affected, carriers show no signs. An affected foal is usually born without clinical signs, but at some stage, usually after six weeks of age, develops severe incoordination, a head tremor, wide-legged stance and other symptoms related to the death of the purkinje cells in the cerebellum. Such foals are frequently diagnosed only after they have crashed into a fence or fallen over backwards, and often are misdiagnosed as suffering from a head injury caused by an accident. Severity varies, with some foals having a fast onset of severe coordination problems, others showing milder signs. Mildly affected horses can live a full lifespan, but most are euthanized before adulthood because they are so accident-prone and deemed as dangerous. As of 2008, there is a genetic test using DNA markers associated with CA to detect both carriers and affected animals.
Clinical signs are distinguishable from other neurological conditions, and a diagnosis of CA can be verified by examining the brain after euthanasia (1). Occipital Atlanto-Axial Malformation (OAAM): this is a condition where the occipital, atlas and axis vertebrae in the neck and at the base of the skull are fused or malformed. Symptoms range from mild incoordination to the paralysis of both front and rear legs. Some affected foals cannot stand to nurse; in others the symptoms may not be seen for several weeks. This is the only cervical spinal cord disease seen in horses less than 1 month of age, and a radiograph can diagnose the condition. There is not a genetic test for OAAM, and the hereditary component of this condition has not been well researched so far.

Equine juvenile epilepsy or Juvenile Idiopathic Epilepsy: sometimes referred to as “benign” epilepsy, is not usually fatal. Foals appear normal between epileptic seizures, and seizures usually stop occurring between 12 and 18 months. Affected foals may show signs of epilepsy anywhere from two days to six months from birth. Seizures can be treated with traditional anti-seizure medications, which may reduce their severity (1).

Though this condition has been studied since 1985 at the University of California, Davis, the genetic mode of inheritance is unclear, notwithstanding the cases studied were all of one general bloodline group. Recent research updates suggest that a dominant mode of inheritance is involved in the transmission of this trait. One researcher hypothesized that epilepsy may be linked in some way to Lavender Foal Syndrome due to the fact that it occurs in similar bloodlines and some horses have produced foals with both conditions (1).

Guttural Pouch Tympany (GPT): occurs in horses ranging from birth to one year old and is more common in fillies than in colts. It is thought to be genetic in Arabians, possibly polygenic in inheritance, but more research is needed. Foals are born with a defect that causes the pharyngeal opening of the Eustachian tube to act like a one-way valve where air can get in but cannot get out. The affected guttural pouch is distended with air and forms a characteristic nonpainful swelling. Breathing is noisy in severely affected animals (1). Diagnosis is based on clinical signs and radiographic examination of the skull. Medical management with NSAIDs and antimicrobial therapy can treat upper respiratory tract inflammation (1).
Surgical intervention is needed to correct the malformation of the guttural pouch opening, thus providing a route for air in the abnormal guttural pouch to pass to the normal side and be expelled into the pharynx. Foals that are successfully treated may grow up to have fully useful lives. Juvenile idiopathic epilepsy of Egyptian Arabian foals and LFS appear to be phenotypically and genetically distinct disorders. There was no apparent association between JIE and LFS, CA, SCID, and OAAM1 (2). A 9-year-old Arabian mare was evaluated for a 7-day history of malaise; it was treated with antimicrobials and the malaise quickly resolved. However, in a recheck CBC on day 13, the apparent degenerative left shift remained.

Further evaluation of the blood smear revealed many hyposegmented granulocytes with coarse mature chromatin and normal cytoplasmic features. Based on the microscopic examination, the horse was diagnosed with Pelger-Huët anomaly, this is the second known report of equine Pelger-Huët anomaly, both of which affected Arabian horses (3). A genome wide association study (GWAS) using Arabian horses with a history of severe laminitis secondary to EMS revealed significant genetic markers near a single candidate gene that may play a role in cholesterol homeostasis, being the best marker, BIEC2-263524 (chr14:69276814 T > C), correlated with elevated insulin values and increased frequency of laminitis ( = 0.0024 and = 9.663 × 10, respectively). In a second population of Arabian horses, the BIEC2-263524 marker maintained its associations with higher modified insulin-to-glucose ratio (MIRG) values ( = 0.0056) and BCS ( = 0.0063). Screening of the predicted coding regions by sequencing identified a polymorphic guanine homopolymer and 5 haplotypes in the 3' untranslated region (UTR), an 11 guanine (11-G) allele was correlated to elevated insulin values in the GWAS population ( = 0.0008) and, in the second population, to elevated MIRG and increased BCS > 6.5 (= 0.0055 and = 0.0162, respectively). The BIEC2-263524-C and the 3' UTR -11(G) polymorphisms were correlated at a 98% frequency, indicating strong linkage disequilibrium across this 150-kb haplotype (4).

Although heritability estimates provide valuable insights into the genetic contribution to a trait, they do not provide information on the number of contributing genes, specific genes involved, or where in the genome these genes are located. Identification of the coding and non-coding variants contributing to a complex trait are important for understanding its complete pathophysiology and to gain a better
understanding of how genes interact or are influenced by the environment (5). The causes of death in horses of different breeds and disciplines and their risk factors have been described in the international literature, where they mention: colic, catastrophic musculoskeletal injuries, systematic bacterial or viral infections, parasitic infestations, respiratory problems, sudden death, perinatal death, among others. Colic is the main cause for morbidity, mortality and premature deaths, and is the number one health concern in horses (6). Colic is the most common reason for emergency veterinary treatment, and a major reason for death or euthanasia across a range of international studies (7).

A postmortem diagnosis in mature and aged equids, death or euthanasia was attributed to disease of the digestive system (41.5%), pituitary gland (12.9%), locomotor system (10.0%), nervous system (7.9%), cardiovascular system (4.6%), urinary system (4.6%), reproductive system (4.2%), respiratory system (4.2%), integumentary system (4.2%), lymphoid system (2.5%), liver (2.5%), or systemic neoplasia (1.2%). Nervous system disease was more common in the 15- to 19-year-old group. Urinary tract disease was more common in the 20-year-old group. Neoplastic disease, regardless of the systemic location, was the basis for death or euthanasia in 18.7% of all equids. Squamous cell carcinoma, lymphoma, and melanoma were the most common malignant neoplasms, PPID (Pituitary Pars Intermedia Dysfunction) was the most common specific diagnosis, based on the postmortem presence of hyperplasia or adenoma, and was the reason for euthanasia in 47.7% of 65 equids with PPID (8). To the author’s knowledge, there is a lack of scientific information on the causes of death in Arabian horses in highly populated regions of the Middle East. This study aimed to describe the causes of death in Arabian horses at Al Wathba, in Abu Dhabi (United Arab Emirates) during 2018-2019.

**Materials and Methods**

This study was conducted in a cohort of 25 Arabian horses that died between September 14, 2018 and December 19, 2019. The average annual population of horses breeding, training and competing at the
racetrack Al Whatba, Abu Dhabi-UAE during the study period was 2,500 horses. Data was collected by necropsy with authorization by the owners. Age, sex and activity of each horse at the time of death were reviewed and recorded. Only those horses with a full necropsy report available were included in the study. The carcass and all internal organs of each horse were examined and representative samples of tissues with abnormalities were collected in 10% neutral buffered formalin and processed for histopathological examination (9). Necropsies were performed in the field considering all the recommendations for field necropsy and the carcasses were collected by the state collection system. This technique has been described by AAEP (9, 10, 11, 12).

The technique is as follows: the horse is placed in left lateral recumbency and examined for overall body condition. Any wounds or external abnormalities should be noted. Necropsy begins by making a curve paracostal incision through the paralumbar fossa and ending at the xyphoid. Abdominal cavity is accessed but avoiding puncturing any underlying bowel (12). This can be particularly difficult when substantial gas distention is present, and careful incision through each separate layer of abdominal musculature can help prevent any inadvertent puncture. Incision runs cranially by coursing between the front limbs and up the ventral neck, ending at the mandibular symphysis. A second vertical incision is made behind the shoulder, just caudal to the triceps. Starting ventrally, the skin and Latissimus dorsi are sharply dissected from the ribcage but carefully avoiding puncturing the diaphragm (12). The resulting muscular flap can be reflected dorsally to expose the abdominal cavity and ribcage to examine the cardiopulmonary system. A stab incision is made into the diaphragm near the sternum. Suction of air into the incision confirms negative pressure within the thorax. The diaphragm is cut away along its attachment to the thoracic wall. Using a rib cutter, each rib is transected at its ventral and dorsal attachment and the ribcage is reflected cranially. Maintaining muscular attachment cranially will aid in the replacement of the ribcage back into position for later closure (12). The lungs can be examined in situ or removed from the chest for evaluation (9). The heart may be removed and opened for inspection of all chambers and valves. Dissecting through the ventral neck incision helps in exposing the trachea and esophagus. Reflection of the limb provides a better view of the cranial thorax and thoracic inlet, but it may also make difficult the incision closure and replacement.
of limbs back into normal anatomic position after necropsy. The entire “pluck” (tongue, larynx, trachea, esophagus, heart, and lungs) may also be removed en masse for a complete evaluation.

To examine the gastrointestinal tract, abdominal viscera are evaluated for abnormalities in location and appearance before removal from the abdomen (9, 12). The pelvic flexure is located and the large colon is exteriorized, laying it alongside the carcass. Removal of the large colon from the abdomen makes evaluation easier and allows better visualization of the remaining abdominal organs. The entire length of the small intestine and small colon should be examined as well as the liver, spleen, both kidneys, and adrenals. The urogenital tract can also be evaluated at this time. To extract the brain, the skin is firstly and then the muscles from the dorsal skull. A hack saw is used to make a transverse cut through the frontal bone just dorsal to the eyes, and then forming a triangle by connecting the first cut with points just medial to the occipital condyles. The section of calvarium are lifted and removed to expose the brain. The primary cause of death was categorized by organ system, i.e., cardiovascular, musculoskeletal, digestive, integumentary, nervous, respiratory, urogenital and multi-system.

**RESULTS**

A total of 25 Arabian horses were examined post-mortem, 35% (9/25) of them were females and 65% (16/25) males. The age average was 8.24, SD: 3.688, V: 13.60. In general, colic occurred specifically at a higher frequency of 80% (20/25). Out of them, 65% (13/20, 8 mares, 4 geldings and one stallion) showed gastric impaction by sand (2 cases had gastric tumors associated, one case had a gastrointestinal stromal tumor and one case had a cecum carcinoma); 25% (5/20, 5 gelding) showed cecum impaction by sand; and 10% (2/20, 2 geldings) showed intestinal torsions, 2 cases of collapse and sudden death during exercise and races (8%, 2 geldings), 2 cases of perinatal death (8%, one mare and one stallion), and one case of laminitis (4%, 1 stallion). The clinical records of the horses with abdominal crisis (colic) reported common and eventual eating of sand, as well as changes in handling, training, diet and water availability at libitum in 48 hours before starting the clinical signs associated with colic.
Only 2% had a history of previous medication with Imidocar sulfate (Imizol) and coincided with the 2 cases of torsion in a segment of the small intestine. Gastric ulceration associated with Equine Gastric Ulcer Syndrome (EGUS) was found in 98% of the studied horses. The first case of sudden death occurred after a 40 Km training resulting in a collapse and sudden death. We observed diffuse petechial to equymotic hemorrhage in the caudo-dorsal lobes as well as rupture of segmental bronchial arteries, associated with exercise-induced pulmonary hemorrhage (EIPH). The second case of sudden death occurred in an 80 Km race with a collapse in the last 5 Km and sudden death. In addition to the observed lesions, compatible with EIPH, an acute renal failure associated with the rhabdomyolysis-myoglobinuria-azotemia syndrome was observed. The cases of perinatal death are described below.

The first one had a neonatal adjustment syndrome. Necropsy revealed meconium aspiration pneumonia and eponychia with meconial dye. The second case was associated with failure to ingest colostrum. A case of chronic laminitis with a 2-year history showed poor body conditions, skin excoriations and chronic tetralaminitis with rotation of the third phalanx of the forelimbs, perforation of the sole and exposure of the third phalanx with osteomyelitis, bacteremia and septicemia. The annual mortality in relation to the estimated population was 1%. It is possible that other deceased horses did not undergo a necropsy and were not considered in the study.

**DISCUSSION**

The main cause of death in Arabian horses was associated with colic. The first cause of colic in horses was the low availability of water, the second cause the ingestion of sand, the third cause the management conditions including factors such as abrupt changes in the diet (quantity, food and hay), horses under stress conditions, stable moves, transportation, changing the groomer, owner, trainer and medications.

According to the literature, the factors increasing the risk of colic include the feeding practices (type and quality of food, type and changes in feeding habits), the horse intrinsic factors (sex, age and breed),
management (type and changes of housing and activity), medical history (a previous colic, administration of a medical treatment) and parasite control (the presence of worms and type of deworming program) (13). A prospective study to describe clinical epidemiology of equine colic in the Society for Protection of Animal Abroad and Donkey Sanctuary Project Clinic, at Debre Zeit, Ethiopia, from November 2014 to April 2015, reported that the incidence of colic was 10.3% (14). The incidence of colic in our study was 80% in Arabian horses. The proportion of case incidence were 63.1 (41/65), 33.8 (22/65), and 3.1% (2/65), in donkeys, horses, and mules, respectively, and the case fatality rate of equine colic was 15.38% (10/65). Statistically, the differences were highly significant (p < 0.01) for the impaction colic in relation to the species (14). Nutrition, and particularly the changes in feeding, is often associated with colic (15).

Recent change in diet, recent change in type of hay, history of previous episode of colic, history of abdominal surgery for colic, recent change in weather conditions, recent change in housing, Arabian breed, administration of an anthelmintic during the 7-day period prior to examination, failure to receive regular deworming, age > 10 years, and regular exercise (vs pastured all the time) were associated with increased risk of colic (16). These risk factors coincide with the results from our study; however, additionally in countries of the Middle East, it is necessary to consider horses that eat sand due to mineral deficits or aberrant behaviors. This behavior undoubtedly predisposes them to develop gastric and cecal impactions, with fatal consequences in most cases. Sometimes horses eat sand because they are bored, or because they do not get enough hay or grass. In rare cases a horse will eat sand because he has a mineral deficiency, possibly due to a lower immune system or because he is shedding his winter coat. Sometimes the horse eats too much sand because the grass in the pasture is so short that the horse unintentionally eats the roots or maybe the horse imitates the behavior of other horses that are in the same pasture (17).

Eating manure is normal for foals and has a legitimate function, by eating manure they digest important microorganisms and bacteria that are necessary for a healthy intestinal flora. On the hard, there are various reasons for an adult horse eating manure, but often is due to a lack of roughage (there is nothing else to
eat) or out of boredom. This is not normal: the reason for this behavior needs to be solved (17). In these cases, it is recommended to assess the amount of food and grass available, including the quality and availability of minerals, as well as consider the paddock environment.

In the Middle East, sandboxes are common, which can predispose them to the ingestion of sand. Interview with 183 equine owners revealed incidence of equine colic as the sixth major disease condition affecting equine in Ethiopia (14). Sudden death (SD) associated with exercise in racehorses is a sporadic event, but when it occurs, poses a serious health risk to jockeys, and adversely affects racehorse welfare and the public perception of horse racing (18). Sudden death in horses may be associated with Exercise-Induced Pulmonary Hemorrhage (EIPH), ruptured aorta, traumatic brain injury, cervical trauma and sudden cardiac death. The cases of collapse and sudden death observed in our study are associated with EIPH, with only one case developing rhabdomyolysis-myoglobinuria-azotemia syndrome. This may be associated with overtraining. Every year veterinary clinical evaluations of horses are increasing in official FEI (Federation Equestrian International) competitions, however in some cases it is possible to observe a collapse due to overtraining (exhausted horse syndrome) and EIPH. This is observed in the number of horses withdrawn from competition due to metabolic conditions and lameness during the official FEI endurance competitions.

Neonatal encephalopathy (NE) and neonatal maladjustment syndrome (NMS) are terms used for newborn foals that develop noninfectious neurologic signs in the immediate postpartum period. Cerebral ischemia, hypoxia, and inflammation leading to neuronal and glial dysfunction and excitotoxicity are considered key mechanisms behind the NE/NMS. Attention has been paid to endocrine and paracrine factors that alter brain cell function (19). Neonatal Maladjustment Syndrome (NMS)/Hypoxic ischemic encephalopathy (HIE) was observed in a neonatal. The syndrome is caused by central nervous system asphyxia (lack of oxygen) before, during or after delivery. Hypoxia is thought to initiate metabolic cascades that result in decreased energy production, ion dysregulation, increased concentrations of excitatory neurotransmitters (especially glutamate and aspartate), and impaired protein synthesis, an increase in
intracellular calcium concentration appears to play a prominent role in neuronal injury. Oxygen-free radicals, nitric oxide production, and pro-inflammatory cytokines are also implicated when a hypoxic episode is not evident. Exposure to inflammatory cytokines is likely to initiate a similar cascade of events. All lesions observed at necropsy are compatible with the neonatal maladjustment syndrome.

Other genetic disorders described in the literature (Severe Combined Immunodeficiency, Lavender Foal Syndrome or Coat Color Dilution Lethal, Cerebellar Abiotrophy, Occipital Atlanto-Axial Malformation, Equine Juvenile Epilepsy, or Juvenile Idiopathic Epilepsy, Guttural Pouch Tympany, Pelger-Huët Anomaly) as associated with mortality in the Arabian horses were not observed. The importance of colostrum for the passive transfer of maternal immunity to foals is well recognized (20). The second case of perinatal death was associated with failure to ingest colostrum during the first 24 hours after birth, and failure of passive transfer of colostral immunity. Laminitis has been a recognized disease since early Greek and Roman times, but it is still bothering both practitioners and scientists (21).

Laminitis, one of the most debilitating conditions of all equids, is now known to be the result of several systemic disease entities. Firstly, laminitis is now considered to be a clinical syndrome associated with systemic disease (endocrine disease, sepsis or Systemic Inflammatory Response Syndrome, SIRS) or altered weight bearing rather than being a discrete disease entity (22). Secondly, laminitis associated with endocrine disease (endocrinopathic laminitis) is now believed to be the predominant form in animals showing (primarily) lameness. Thirdly, the designation of laminitis as a primary and severe basement membrane pathology now requires revision. Instead, current data now proposes a variable subclinical stage associated with gross changes in the hoof capsule, with stretching and elongation of the lamellar cells, which is an early and key event in the pathophysiology (22). Endocrinopathic laminitis is common in horses and ponies, but the recurrence rate of the disease is poorly defined. The recurrence rate for endocrinopathic laminitis was 34.1% (23).
Only one case of chronic endocrine tetra-laminitis characterized by immunosuppression and secondary bacterial complications was diagnosed. In conclusion, we identified and described the causes of death in Arabian horses in Al Wathba, Abu Dhabi, UAE, during the period 2018-2019. In summary, pathologies of the digestive system, specifically acute abdominal crisis (colic), are the main causes of death in Arabian horses (80%). Other causes of mortality with a low incidence were, in order, collapse and sudden death during exercise and races, perinatal death, and laminitis. The necropsy performed by the Equine Veterinary Pathologist can objectively elucidate the cause of death and promote preventive measures in cases of population medicine in the racetrack (infectious diseases: AIE, EEE, EEV, among others), but can also determine previous lesions not detected in the clinical examination, even in cases of sudden death (EIPH, aorta rupture). Additionally, from a legal point of view, it can be determinant for sanctions by the authorities to those involved in the death of race horses (veterinarians, trainers and owners). These results allow establishing risk prevention measures of mortality in horses, specifically in handling, feeding, training and race issues.

REFERENCES


