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Hereditary equine regional dermal asthenia in two Quarter horses in Austria

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Summary

Hereditary equine regional dermal asthenia (HERDA) is a skin disease with changes of the collagen fibres in Quarter horses. The mode of inheritance is autosomal recessive. Affected horses suffer from hyperextensible skin that is easily torn and results in badly healing wounds and atrophic scars. The skin lesions are usually located on the dorsum and rarely on the neck or the extremities. The disease typically starts in the first 2 years of life and has been reported in North America, Brazil and England. The diagnosis is based on the clinical skin lesions and the histopathologic changes of skin biopsies. Furthermore ancestors known to be HERDA-carriers increase the suspicion. A genetic test has recently been developed which allows the detection of heterozygous carriers and homozygous affected horses easily by analysis of DNA in the hair bulb. This case report describes HERDA diagnosed in 2 Quarter horses in Austria for the first time.

Abbreviations: EDS = Ehlers-Danlos Syndrome; H&E = haematoxylin and eosin; HERDA = hereditary equine regional dermal asthenia; VMU = Veterinary Medicine University

Schlüsselwörter: hereditäre equine regionale dermale Asthenie, HERDA, Quarter Horse, Kollagen.

Zusammenfassung

Hereditäre equine regionale dermale Asthenie bei zwei Quarter Horses in Österreich

Hereditäre equine regionale dermale Asthenie (HERDA) ist eine Hauterkrankung mit Veränderungen der Kollagenfasern beim Quarter Horse. Sie wird autosomal rezessiv vererbt. Betroffene Pferde leiden unter einer überaus dehnbaren und leicht reißenden Haut, woraus schlecht heilende Wunden und atrophische Narben entstehen. Die Hautveränderungen sind vor allem im Rückenbereich, seltener an Hals oder Extremitäten zu finden. Die Erkrankung manifestiert sich typischerweise in den ersten beiden Lebensjahren und wurde bisher in Nordamerika, Brasilien und England beobachtet. Die Diagnostik setzt sich aus den klinischen Hautläsionen sowie den typischen histopathologischen Veränderungen von Hautbiopsien zusammen. Zusätzlich liefert der Abstammungsnachweis betroffener Pferde Hinweise auf die Erkrankung, wenn Vorfahren enthalten sind, die als HERDA-Träger bekannt sind. Seit kurzem existiert auch ein genetisches Testverfahren, welches mittels Haaranalyse heterozygote Träger und homozygote erkrankte Pferde leicht erkennen lässt. Dieser Fallbericht beschreibt zum ersten Mal das Auftreten von HERDA bei 2 Quarter Horses in Österreich.

Introduction

Hereditary equine regional dermal asthenia (HERDA) is a congenital skin disease with an autosomal recessive mode of inheritance that affects the collagen fibres of Quarter horses (WHITE et al., 2004). Along with cutaneous asthenia (Ehlers-Danlos Syndrome [EDS], dematosparaxis and cutis hyperelastica) it belongs to a group of inherited, congenital dysplasias of the connective tissue that are characterized by loose, hyperextensible and abnormally fragile skin (SCOTT, 1988; SCOTT and MILLER, 2003). In human medicine EDS is well recognized (LAWRENCE, 2005; PROSKE et al., 2006) and was described in several species including cats (SEQUEIRA et al., 1999; BENITAH et al., 2004), dogs (POULSEN et al., 1985; PACIELLO et al., 2003), rabbits (SINKE et al., 1997; IGLAUER et al., 1999), cattle (HANSET and LAPIERE, 1974; WITZIG et al., 1984) and sheep (WEEREN-KEVERLING BUISMAN and KOEMAN, 1986; HALDEREN and GREEN, 1988). Even minor trauma can result in poorly healing wounds in affected animals (HARDY et al., 1988; BROUNTS et al., 2001; WHITE et al., 2004). In Quarter horses skin lesions can be single or multiple and are most commonly seen at the dorsum but also at the neck or the legs (STANNARD, 2000). Onset of clinical signs are typically noticed at a mean age of 1-2 years (HARDY et al., 1988; BORGES et al., 2005; WHITE et al., 2007; RENDLE et al., 2008), although reports of HERDA occurring in a newborn foal and a 4 year old horse have been published (WHITE et al., 2004).

The disease was first described in 2 horses in 1978 (LERNER and MCCRACKEN, 1978). Since then there are numerous reports of HERDA in Quarter horses (BROUNTS et al., 2001; WHITE et al., 2004; BORGES et al., 2005; WHITE et al., 2007; RENDLE et al., 2008). Similar case reports exist in an Arabian-crossbred horse, a thoroughbred, a Hanoverian and a Haflinger pony (GUN-

SON et al., 1984; SOLOMONS, 1984; WITZIG et al., 1984; SCOTT and MILLER, 2003) as well as in breeds with Quarter horse ancestry, such as Paints and Appaloosas.

This article documents the diagnosis of HERDA in 2 Quarter horses in Austria for the first time.

Case reports

2 Quarter horses were presented with chronic skin lesions at the Veterinary Medicine University (VMU) Vienna in the years 2004 and 2006.

Case 1

Horse 1, a 3 year old chestnut stallion, was presented at the VMU Vienna for breeding soundness examination. At the time of presentation old lacerations and scars on the dorsum were present, which first occurred at the age of 2 years.

The dermatological examination revealed irregular, sharply demarcated, hyperpigmented and alopecic skin areas with a diameter up to 40 cm at withers, saddle position and croup (Fig. 1). The surrounding skin and coat showed no abnormalities. The lesion margins were partly thickened. The affected skin and also the skin in the lateral neck areas could be easily raised and returned to its normal position very slowly.

A tentative diagnosis of HERDA was made due to the stallion's breed and the clinical presentation. At a later point skin biopsies were taken and histopathologic examination was in accordance with the diagnosis. Furthermore the pedigree of the stallion included ancestors known to be HERDA-carriers.

The breeding soundness examination revealed a scrotal hernia on the right side and a moderate hypoplasia of both testicles. Semen evaluation (density, total sperm count and percentage of morphologically normal spermatozoa) did not comply with the minimum requirements of fertile stallions of the same breed and age.

Due to the diagnosis of phenotypical visible hereditary diseases (scrotal hernia, hypoplasia of the testicles, HERDA) the allowance for insemination could not be granted (Viennese animal breeding legislation § 17). Therefore the stallion was commited to the breeding and embryo transfer station of the VMU Vienna. The horse was treated with care and excluded from any work to minimize the risk of further skin wounds.

7 months later the horse developed obvious supporting limb lameness in the right forelimb. Orthopaedic examination revealed horn rings typical of laminitis, wider at the heel than at the toe. The dorsal walls were concave with additional longitudinal grooves. The sole horn was weak at the tip of the frogs and within the level of the bearing surface, the white line was broadened and black coloured. The hoof tester was slightly positive medial to the frog apex of the right hoof. Radiographs of both distal limbs showed bilateral 3rd phalanx (coffin bone) deformation characteristic of laminitis: an atrophic apex of the coffin bone with obvious convexity of both dorsal cortex and solar surface. However, there was no obvious displacement of the coffin bone such as rotation or depression Significant dorsal subluxation of the 2nd phalanx was found in the coffin joint (Fig. 2). Based on these symptoms of the distal limbs the following diagnoses were made: atypical chronic laminitis of both forelimbs and a subsolar abscess in the right fore hoof. Drainage of the infection, regular trimming and occasional use of non-steroidal anti-inflammatory drugs (Phenylbutazone, Adler Apotheke, Wels, Austria), were performed successfully as therapy of the limb ailments.

Although the stallion was handled cautiously, new skin lesions appeared on the dorsum (Fig. 3). Even mild trauma resulted in slowly healing wounds that got secondarily infected and resulted in scar formations. The condition of the skin worsened gradually and had a negative impact on the stallion´s life quality. Therefore the horse was euthanised 17 months after first presentation at the age of 4 years and 5 months.

Case 2

Horse 2, an 1 year old brown stallion, was presented at the VMU Vienna with chronic skin lesions on the dorsum and the neck that appeared at the age of 6 months. According to the owner trauma was not a trigger factor of the skin lesions.

The stallion showed multiple irregular, sharply demarcated areas with torn skin leading to full thickness ulceration, peripheral alopecia, hyperpigmentation, crusts and scarring up to 50 cm in diameter bilaterally at the caudal saddle area, the dorsum and the left side of the neck (Fig. 4a,b). The skin appeared thinner at palpation in the affected areas as compared to non lesional skin. Furthermore there were sero-sanguinous crusts with a diameter of 2 cm at the fetlock region of both front legs. The skin and coat of the surrounding areas showed no abnormalities. Skin folds could easily be raised all over the trunk when pinched and did not return to normal position for several minutes (Fig. 5a,b).

The skin lesions in combination with the stallion's breed and pedigree (as in case 1 the pedigree of this stallion included ancestors known to be HERDA-carriers) were suggestive of HERDA. Due to the extent of the skin lesions and the guarded prognosis the stallion was euthanised at the owner's request.

Histopathologic examination and necropsy

Multiple 8 mm punch biopsies (Stiefel Laboratorium, Offenbach, Germany) were collected from the affected skin areas and from clinically normal skin of the neck and the abdominal region from both horses for histopathologic examination. The skin biopsies of horse 2 were histopathologicly compared to skin samples of a healthy horse. Necropsy was performed on both animals.

The punch biopsies were stained with haematoxylin and eosin (H & E) and in case 2 additionally with Masson's trichome.

In both horses the epidermis, the superficial dermis and the adnexae were unremarkable except in fibrous scarring areas supposedly representing old lesions with a total loss of adnexae. The collagen bundles in the dermis appeared partly separated by clear spaces and showed orientational disarray (Fig. 6).

In the biopsy samples of horse 2 the upper and middle portion of the deep dermis showed a zone of apparent rarefaction compared to the biopsy samples of the healthy horse. In these areas the changes of the collagen bundles were most obvious and there was more intense red staining of the collagen fibrils in Masson's trichome stain (Fig. 7).

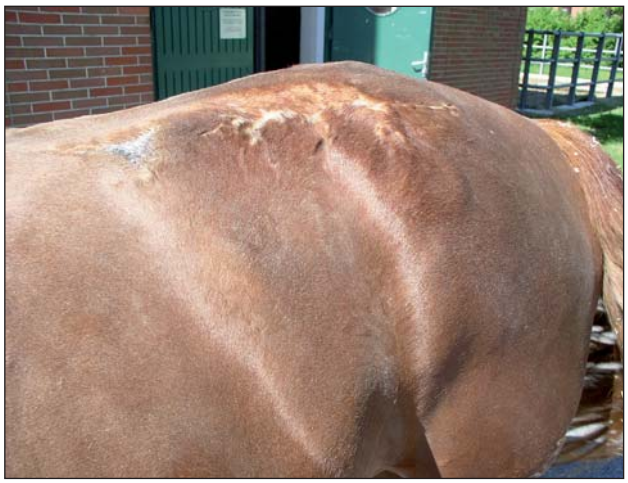

Fig. 1: Chronic skin lesions on the dorsum and croup of horse 1

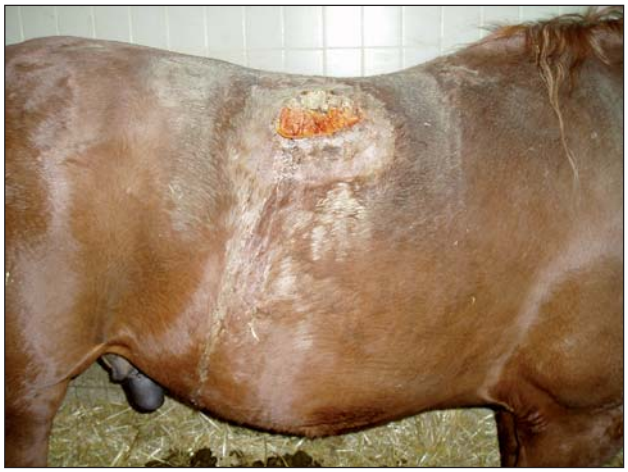

Fig. 3: Acute laceration at the saddle position without any previous trauma in horse 1

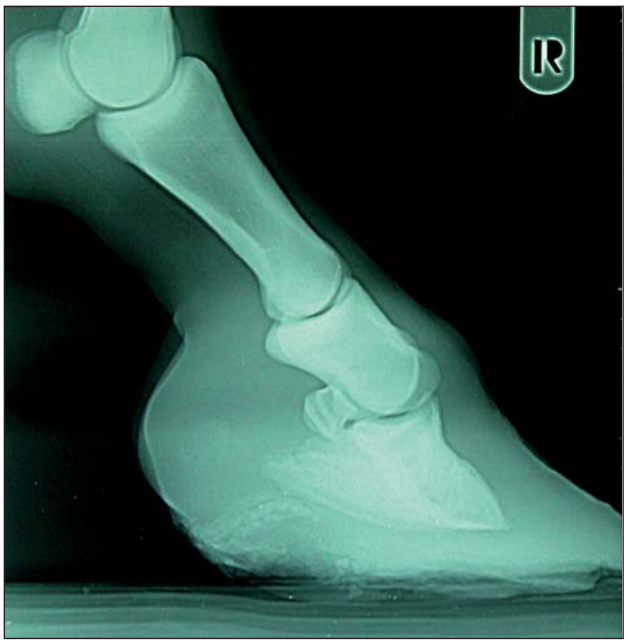

Fig. 2: Lateromedial radiograph of the right distal forelimb of horse 1 showing the concave shape of the dorsal wall, a deformed coffin bone with convex dorsal cortex and solar surface, and the subluxation of the coffin joint

Necropsy revealed changes characteristic of chronic laminitis in both front legs in horse 1 and no abnormalities in horse 2.

In summary, the histopathologic findings together with the clinical presentation and the pedigree are consistent with the diagnosis of HERDA in both horses.

Discussion

The skin lesions of both Quarter horse stallions described here are similar to the case reports form North America, Brazil and England (WHITE et al., 2004; BORGES et al., 2005; WHITE et al., 2007; RENDLE et al., 2008). As previously reported (HARDY et al., 1988; WHITE et al., 2004; BORGES et al., 2005; WHITE et al., 2007; RENDLE et al., 2008), both horses showed a hyperelastic and hyperextensible skin that was easily torn and resulted in scar formations (horse 1 at the age of 2 years, horse 2 at the age of 6 months). Typically the skin lesions in HERDA horses are found on the dorsum (STANNARD, 2000). Horse 2 also showed sero-sanguinous crusts at the distal parts of the front legs. Similar lesions were described in 10 % of the affected horses in a North American study (WHITE et al., 2004). The amount of collagen in skin wounds at the distal extremities has been compared to collagen in wounds affecting the thoracic region in 6 horses. An increased accumulation of collagen was noticed on the legs (SCHWARTZ et al., 2002). It is not proven whether or not these findings have any relevance with respect to lesion formation in HERDA.

When taking the history, some owners often think the clinical signs are due to trauma or saddle training (WHITE et al., 2004). There are though case reports of HERDA, where the clinical signs appear before the saddle training and without any history of trauma (WHITE et al., 2007; RENDLE et al., 2008). Skin lesions do not seem to occur as a consequence of surgery (e.g. castration) or parturition (WHITE et al., 2004, 2007). Although spontaneous occurring wounds are more often noticed in HERDA-affected Quarter horses, it is reasonable to minimize trauma in affected horses to decrease the risk of lacerating the skin (STANNARD, 2000).

In human medicine EDS is described as a heterogeneous group of inheritable connective tissue disorders,

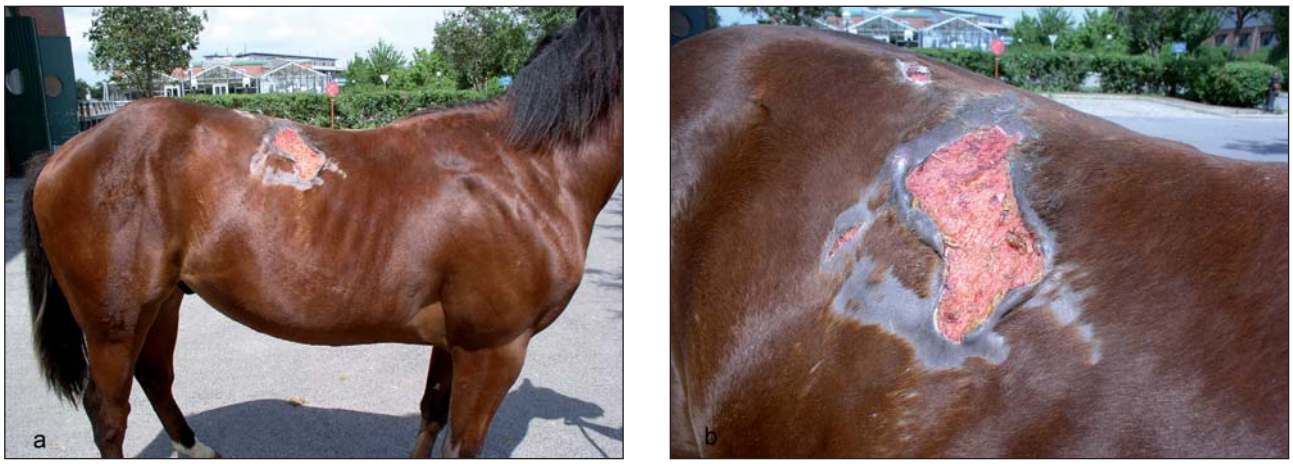

Fig. 4: Extensive laceration on the dorsum of horse 2 (a) and a close up of the lesion (b)

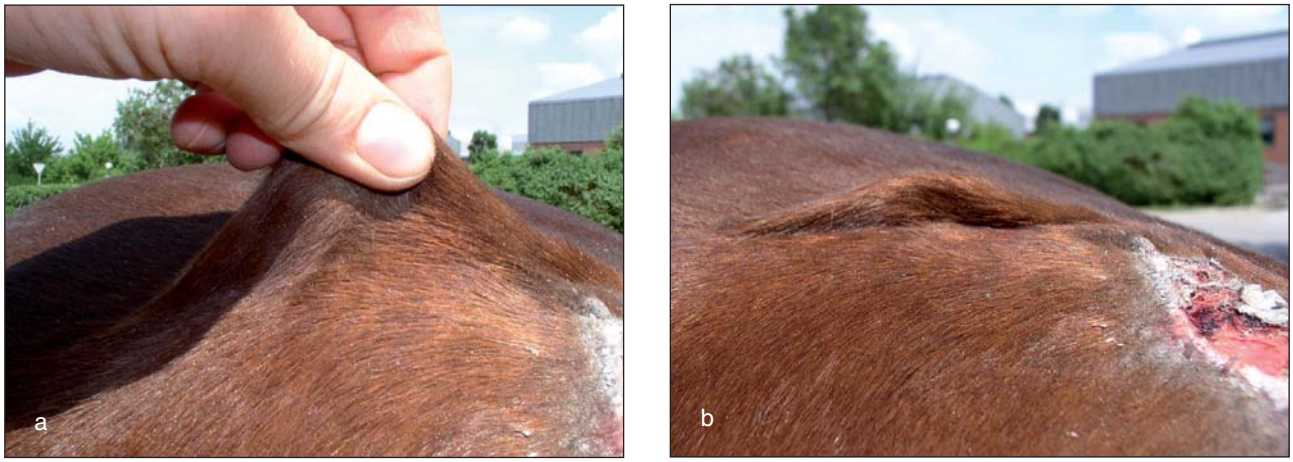

Fig. 5: A skin fold is easily raised on the dorsum of horse 2 (a); the raised skin did not return to its normal position for minutes (b).

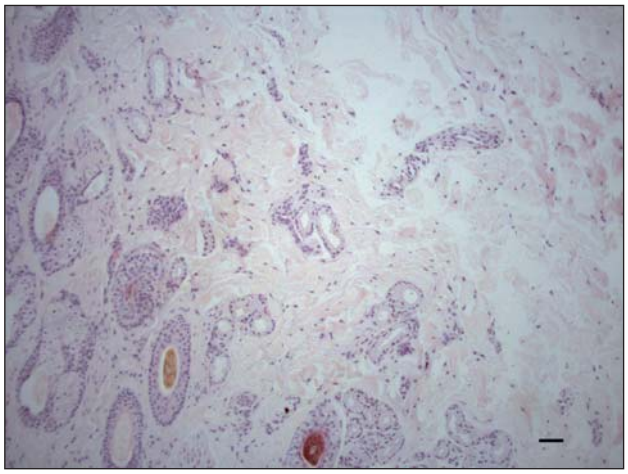

Fig. 6: Separation of the collagen bundles especially in the upper and middle portion of the deep dermis (right part of the picture); H&E, bar = 100 µm

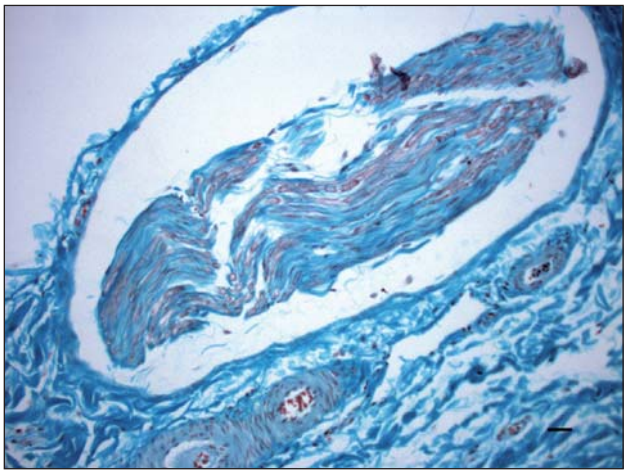

Fig. 7: Red staining of collagen fibres in Masson´s trichome stain; bar = 100 µm

which are caused by mutations of genes encoding for collagens. Due to altered gene, the mechanical properties of skin, joints, ligaments and blood vessels can be affected (LAWRENCE, 2005). Patients with EDS suffer from skin hyperextensibility, atrophic scarring, joint hypermobility and generalised tissue fragility (CALLEWAERT et al., 2008). Horse 1 suffered from recurrent laminitis of both forelimbs in his last year of life which could be controlled with restricted movement and anti-inflammatory drugs. Joint disorders have not been reported in other Quarter horses with HERDA (WHITE et al., 2004, 2007; BORGES et al., 2005; RENDLE et al., 2008). However, there are case reports of dogs with EDS-like syndrome showing lacerations and hyperextensible skin together with joint hypermobility (ANDERSON and BROWN, 1978; PACIELLO et al., 2003). The subluxation of both coffin joints visible in the radiographs might be due to a laxity and hypermobility of the collateral ligaments. It is unknown whether the changes found in the fore hooves of horse 1 were a coincidence of 2 different diseases, HERDA and laminitis. Another possibility is that the laminitis was a consequence of HERDA, with the change of the coffin bone tissue being due to an abnormal loading pattern of the bone as a result of a disturbed collagen quality of the supporting apparatus of the bone.

The histopathologic findings in the skin biopsies of both horses were in accordance with findings described in H&E staining of lesional skin in HERDA affected horses. There was a separation of the collagen fibres which also showed orientational disarray (WHITE et al., 2004; RENDLE et al., 2008). These changes were most pronounced in the deep dermis where there was an apparent rarefaction (BORGES et al., 2005; WHITE et al., 2007).

Massom's trichome stain, used in the biopsy specimens of horse 2, stains abnormal collagen fibres red compared to normal collagen fibres that stain blue or blue-green. This technique was successfully used in horses and cats with collagen defects to show the difference between normal and defective collagen fibres (HARDY et al., 1988; FERNANDEZ et al., 1998). However, there are case reports where the collagen fibres stained red in both healthy and affected horses which implicates that this examination technique might not be a reliable tool (BROUNTS et al., 2001; WHITE et al., 2004; BORGES et al., 2005).

A zonal dermal separation in the mid to deep dermis as described in some HERDA- cases (BROUNTS et al., 2001; WHITE et al., 2004; BORGES et al., 2005), was not evident in our biopsies. Some authors postulated that the separation is an artefact due to sampling technique or processing (WHITE et al., 2004).

HERDA has an autosomal recessive mode of inheritance (TRYON et al., 2005, 2009). Affected Quarter horses remain undetected until they develop typical clinical signs (WHITE et al., 2007). The severity of the skin lesions and the lack of successful therapy often results in euthanasia of the animals (TRYON et al., 2007). In the described cases the diagnosis was made based on the clinical presentation, pathohistologic changes and a suspicion based on the pedigree. As the disease has been linked to a mutation of equine cyclophilin B, a genetic test has now been made available. The test identifies both heterozygous carriers and homozygous affected horses (TRYON et al., 2007). Both stallions in this case report were genetically tested at a later point with blood samples that were taken and frozen before euthanasia. Recently the genetic test was further developed and allows now using hair or other tissue to extract DNA for diagnosis. Both horses in this study were homozygous for the genetic defect, confirming the diagnosis of HERDA. For further information about the genetic test the authors refer to the internet site of the University of California, Davis, USA (www.vgl.ucdavis.edu/servive/horse/index.html).

The pedigree analysis of both horses could be traced back to the stallions Poco Bueno and King, 2 suspected HERDA-carriers based on the pedigrees of their progeny (RASHMIR-RAVEN et al., 2004).

In conclusion, HERDA is a debilitating and difficult disease to handle. We suggest that horses with known HERDA-carriers in their pedigree should be routinely tested and all animals with a positive genetic test result should be excluded from breeding program, as this is the only way to eliminate this severe and often fatal disease.

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