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Consensus on equine tendon disease: Building on the 2007 Havemeyer symposium

Introduction

This conference was held from 24 to 26 September 2007 in Iceland to bring together a number of clinicians and scientists interested in equine tendon disease; this injury accounts for a large number of days out of training/competition and/or retirement. It is evident that there have been considerable advances in this field over the past 20 years and here we draw together conclusions from the discussion of the following 4 key areas in equine tendon disease where a consensus view was reached to guide future research of most relevance to the equine industry: 1) severity grading and staging; 2) genetics of tendon disease; 3) the design of clinical trials; and 4) rehabilitation programmes.

Terminology

'Tendinitis' is commonly used to describe a nontraumatic overstrain injury to a tendon. The term 'tendinosis' has been suggested in the human field because many pathological specimens from human patients show no evidence of traditional histological features of inflammation. This, however, does not rule out components of the inflammatory cascade having some role, and so more recently, the nonspecific terms 'tendinopathy' or 'tendon disease' have been adopted. The latter may be the most appropriate given that it is clear that while clinical tendon overstrain injuries occur suddenly, most appear to be associated with previously acquired cumulative degeneration; hence, the term 'tendon disease' has been used here.

Severity grading and staging of tendon disease

Severity

The severity of disease was thought to be most practically and objectively determined by ultrasonographic assessment of tendon cross-sectional area. The size of the hypoechoic lesion was thought to be less accurate because of the difficulty in accurately determining lesion size. Although only the central area of the injured tendon may be hypoechoic, damage to other parts of the tendon may still be present, and in more generalised lesions it is often not possible to delineate the lesion. Owing to the large natural variation in normal tendon cross-sectional area, injured tendon data (size and echogenicity) could be normalised to an internal 'standard', such as the contralateral tendon (although bilateral disease is common) or an area of the tendon that is rarely affected by disease in the same horse.

TABLE 1: Proposed criteria for severity grading of superficial digital flexor tendon injuries (consensus includes previously proposed definitions)

Proposed definitions	Havemeyer grade 1 (mild)	Havemeyer grade 2 (moderate)	Havemeyer grade 3 (severe)
BAPTEN protocol [18]	0–15% of tendon 'volume' affected	16–25% of tendon 'volume' affected	>25% of tendon 'volume' affected
Lesion size at MIZ	0<10%	10–40%	>40%
Maximum tendon CSA	<2 cm^2	2–5 cm^2	>5 cm^2

MIZ = maximum injury zone; CSA = cross-sectional area.
Percentage of tendon 'volume' affected = (summation of lesion CSA for 7 levels)/(summation of tendon CSA for 7 levels) × 100 which therefore incorporates both a measure of affected cross-sectional area and length of lesion.

Table 1 shows the proposed definitions of the severity of superficial digital flexor tendon injury based on ultrasonographic findings.

Staging

An accurate definition of the disease is vital for epidemiological and genetic studies. The identification of subclinical disease depends on whether the pathology is at a molecular level (hence undetectable by clinical methods) or due to previous pathology that is missed clinically. It was concluded that the diagnosis and staging of tendon disease can currently be best standardised within one or more of the following categories, which are summarised in Table 2: 1) clinical signs; 2) ultrasonographic changes; 3) magnetic resonance imaging (MRI) changes; and 4) histological analysis.

Clinical definition

Tendon disease can be identified clinically by the presence of heat, pain or swelling of the affected tendon on palpation, not all of which may be apparent, reflecting differences in the stage (acute vs. chronic), the tendon affected and the severity of the disease process.

Ultrasonographic definition

Ultrasonographic criteria of disease were originally correlated with broad histological parameters (e.g. [1,2]). However, more recently, more sophisticated computer-assisted analysis of the ultrasonographic image [3] shows potential for defining and staging the disease through providing a more accurate determination of the nature of the tissue within the tendon.

The ultrasonographic definition of disease relies on identifying an increase in cross-sectional area, changes in echogenicity (usually hypoechoic change in the acute stage and a heterogeneous pattern in chronic disease), fibre alignment pattern in longitudinal images, shape, position and margination. The correlation between clinical and ultrasonographic criteria are commonly, but not always, matched. It is rare for there to be clinical positive findings without ultrasound, but less rare for there to be ultrasonographic positive findings and no clinical evidence of disease.

Concern was raised over the sensitivity of ultrasound to monitor disease accurately. Ultrasonography was not found to be particularly useful for this in human patients and had been recently found to be insensitive for predicting clinical disease [4]. However, it was agreed that for horses to return to normal use, the ultrasonographic parameters should be a stable cross-sectional area, homogeneous echogenicity and good longitudinal pattern.

Magnetic resonance imaging definition

A more accurate and/or sensitive definition of disease may be provided by MRI, although to be practical, this should be performed using standing units. Different sequences are available for characterisation of the injury, providing detailed information about the nature of the tissue contained within the disrupted area [5–8] and the condition of the surrounding tendon fibres. Identification of the optimal sequences would speed up application of the technique, hence reducing cost and improving practical application. It was considered important that, just as with ultrasonography, the changes observed on MRI need to be correlated with defined pathology.

Magnetic resonance imaging provides detailed information about the nature of soft tissue injury and is exquisitely sensitive to the presence of haemorrhage. However, reliably identifying acute vs. chronic tendon injury with MRI can be challenging, depending on the MRI findings present. The presence of fluid in a tendon indicates a moderate to severe abnormality, depending on the nature and distribution and is often associated with fibre disruption. Fluid is best detected with T2-weighted or fat suppressed images. Combining different sequences can be helpful in ageing haemorrhage.

TABLE 2: Summary of the parameters used to define and stage tendon disease

Category	Criteria	Normal	Acute	Chronic
Clinical findings		No heat, pain or swelling	Heat, pain or swelling on palpation	Enlargement only
Ultrasonography	Cross-sectional area	Normal cross-sectional area 80–140 mm^2 for superficial digital flexor tendon at level 2B in Thoroughbreds; <20% difference from contralateral limb	Enlargement of tendon	Enlargement of tendon
	Echogenicity	Even and bright echogenicity	Reduction in echogenicity	Heterogeneous and variable echogenicity
	Longitudinal pattern	Prominent striated pattern (dependent on tendon)	Reduction in longitudinal fibre pattern	'Fibrotic' longitudinal fibre pattern
Magnetic resonance imaging		Homogeneous low signal intensity with normal size/shape	Enlargement of tendon with increased signal intensity	Enlargement of tendon with increased signal intensity; alternatively, enlarged with low signal intensity on all sequences
Histology	Vascular component	Sparse vasculature	Haemorrhage; increased vascularity	Neovascularisation; increased to normal vascularity
	Fascicle organisation	Prominent fascicular pattern with linearly arranged fascicles Crimp pattern to fascicles but reduced in older animals	Fibre disruption, giving rise to a variation in crimp signifying partial rupture	Disorganised fascicular pattern Accumulation of proteoglycan between fascicles Cyst formation; fat necrosis
	Cellular component	Sparse tenocyte population Acellular areas in old tendon	Tenocyte necrosis Inflammation and reparative cellular proliferation and fibroplasias; neutrophil and monocyte infiltration	Increased cellularity with cell rounding Acellular areas

Mild to moderate injury is not always associated with fluid accumulation in the tendon, but increased signal intensity on proton density or T1-weighted images with no increased signal intensity or a slight to mild concurrent signal increase on T2-weighted and short TI inversion recovery images may be seen. This signal pattern can also be present following healing of a severe injury and can remain for an extended period of time following the dissipation of fluid. In other cases, acute and chronic injury can appear identical on MRI and findings should be correlated with the clinical examination of the horse.

Histological definition

Histological analysis could provide the most accurate definition of pathological change, although it is usually only appropriate *post mortem* because of the trauma created by biopsy techniques. It is important to distinguish between histological changes in the lesion itself vs. those associated with ageing or pre-existing tendon disease. While some features overlap, there is evidence that these are distinct pathological processes. The processes thought to be associated with age-related degeneration are not always characterised by histological changes, occurring more at a subcellular and molecular level. In addition, while some histological changes, such as acellular areas [9], may be more related to ageing change and therefore of questionable clinical significance they could still represent predisposition to injury or re-injury.

Several different pathologies exist in different tendons and ligaments and at different stages of the disease. Soon after the clinical onset of the injury, the lesion will show fibre disruption, haemorrhage, tenocyte necrosis, inflammation and reparative cellular proliferation and fibroplasia, i.e. a wound healing profile. Chronic (often also termed 'degenerative') tendon disease, which can also occur in areas potentially well removed from the site of the initial lesion [10], are characterised by increased tenocyte numbers and cell rounding, neovascularisation, collagen fibril malalignment, interfascicular matrix accumulation and proteoglycan accumulation. Importantly, degenerative change lacks inflammatory cell infiltration and may arise as an end-stage of previous injury or because of alterations in loading. These pathological changes are consistent between different tendons and across species.

Histological changes are well described in human tendon, including degenerative lesions that have been identified in significant numbers of asymptomatic individuals and that have been determined to have greater severity when observed in specimens from acutely ruptured Achilles tendons [11,12]. Some of these changes, including alterations in crimp waveform and variation in cellular density, have been looked for and described in horses, while others, including abnormal tenocyte morphology, myxomatous or lipoid degeneration and collagen microtears, have not [1,2,9]. There has not been a recent study of asymptomatic and ruptured equine tendons to define more clearly the normal and pathological histological features of the matrix and cellular populations in digital tendons and ligaments of horses of different ages and breeds and varying exercise histories. The accuracy and detail of histological descriptions would be improved by the involvement of pathologists in such studies.

The participants considered there to be a need for a standardised histological scoring system. One of the best examples was that of Movin *et al*. [12], which is a modification of the protocol used by Astrom and Rausing [11]. The system has been further modified by Maffulli *et al*. [13,14]. The system uses a 4-point scoring, where 0 is normal, 1 slightly abnormal, 2 moderately abnormal and 3 markedly abnormal, and each of the following 8 parameters were assessed: fibre structure, fibre arrangement, roundness of nuclei, regional variations in cellularity, increased vascularity, decreased collagen staining, noncollagenous matrix accumulation and fibrosis/hyalinisation. Thus, 0 was normal and the most severe pathology 24. Scores between 1 and 8 were classified as slightly abnormal, between 9 and 16 as moderately abnormal and between 17 and 24 as markedly abnormal. This can be simplified into the assessment of the following 3 broad categories: 1) cellular changes, i.e. cellularity and cell morphology (rounding); 2) vascularity; and 3) organisation, i.e. collagen fibre alignment, interfascicular matrix infiltration and proteoglycan content (e.g. toluidine blue staining).

Because of the variation of pathology across the tendon, a sufficiently representative area is required to provide a confident picture of the tendon as a whole. It was recommended that ideally more than one section from each tendon sample should be evaluated, with at least 10 representative fields (×20) being scored from each section. As some fields may have

limited pathology, scores from each field should then be summed (rather than averaged) to give a total score for the tendon section.

Genetics of tendon disease

It is often difficult to determine the relative importance of genetics and the environment for tendon disease. Both genetic and environmental factors should be included in epidemiological 'models'. While some environmental factors are considered to be influenced by genetic components, this is likely to be less so in the horse, because the horse has no voluntary choice with respect to its environment. Furthermore, to date, there are very few diseases in the horse that have been shown to have significant genetic components.

Genetics has the potential to provide useful markers of susceptible individuals and disease mechanisms, which will be beneficial in the future to help prevent injury. Approaches at present may be either to establish a list of 30–40 candidate genes in the first instance or to progress immediately to whole-genome screening approaches. With the completion of the equine genome, such genome-wide screens become a reality, and progress may now become rapid owing to the recent commercial availability of a large equine single nucleotide polymorphism Chip. For this to be successful, however, it requires the accurate identification of 'clean' control horses, free of tendon disease.

The design of clinical trials and objective outcome parameters

There are many studies published in both the human and the equine literature on tendon disease that are underpowered. To assist in the design of future studies to compare outcomes between different treatments, a power calculation was performed to give the sizes of the groups required, assuming a P value of 0.05 (2-sided), 90% power and 1:1 treated : nontreated ratio (see Table 3). Thus, in a randomised controlled trial, for a 55% re-injury rate (data for conventional treatment in National Hunt racehorses [15]); in the 'nontreated' (i.e. conventionally managed) group and a clinically relevant improved prognosis of 30% re-injury rate for a new treatment, 2 groups of 88 horses would be needed to complete the study. To account for drop-outs (e.g. lost to follow-up, change of career and noncompliance; usually about 10%), there should be more than 100 horses per group.

With respect to the best outcome parameters, the consensus view was that there is no convincing single outcome parameter. It was agreed that the most objective parameters were considered to be similar to those considered by Dyson [15], namely:

- Proportion showing re-injury over a 2-year period after a return to full work. Analysis should only be performed on those horses that had returned to a reasonable level of performance (e.g. returned to full work) so that the healed tendon was subjected to the level of loading experienced by the normal use of the horse.
- Proportion still racing for 2 seasons after returning to full work.
- Proportion of horses racing more than once (return to racing, i.e. completing one race, was thought to be an unreliable indicator); ideally 3 or 5 races, although racing frequency differs between countries.
- Return to the previous level of performance without re-injury to both the treated and the contralateral limb (horses that have not previously raced should be analysed separately).
- Other outcome measures could be included for other disciplines, such as a mile time for Standardbreds, although this was considered less relevant because tendon injury may not influence maximal speed.

Kinetic and kinematic measures indicative of gait abnormality could also be used as an outcome parameter. While lameness is not a common feature of superficial digital flexor tendon disease after the initial inflammatory phase, there is increasing evidence that there are changes, both discrete and quantitative, that can be defined via both elaborate (e.g. force plate or video) or simple (e.g. accelerometer) methods; however, these must be well controlled for gait and speed without horse acceleration.

Rehabilitation protocols

There is a need for a standardised rehabilitation protocol. At present, programmes are empirical, but the variability of the disease makes scientific testing of these protocols impossible. The most damaging aspect of exercise was considered to be high-impact loading, and this may be more evident in some horses compared with others that exhibit asymmetric limb loading, especially when tired, which can induce subclinical tendon damage ('microklutziness') associated with minor differences in neuromuscular control of limb motion [16]. Rehabilitation should therefore occur in an environment that limits high-impact loads. The group proposed a graduated rehabilitation protocol for overstrain injury to the superficial digital flexor tendon in a racehorse based on current (and empirical) experience (see Table 4). Both walking and trotting exercise can be given in hand or with the use of a horse walker. The surfaces on which the horse is exercised become more important when the horse begins faster work (trotting and cantering); generally, softer surfaces are thought to be protective of superficial digital flexor tendon disease, most probably because of their effect at slowing the horse. Serial ultrasonographic examinations were considered important at a minimum of 3-monthly intervals to allow modification of the programme. The recommended programme was a generic one, which could be adapted (shortened or lengthened) depending on the following factors: 1) the

TABLE 3: Samples sizes for a randomised controlled trial to determine the efficacy of one treatment (with treatment) compared with another (without treatment)

		Percentage of re-injury without treatment						
		40	45	50	55	60	65	70
Percentage of re-injury with treatment	**10**	49	38	31	25	21	18	15
	20	119	80	58	44	35	28	23
	25	216	128	85	61	46	36	28
	30	496	230	134	88	63	47	36
	35	2008	523	240	138	90	63	47
	40	–	2092	538	244	140	90	63
	45	–	–	2134	544	244	138	88
	50	–	–	–	2134	538	240	134
	55	–	–	–	–	2092	523	230
	60	–	–	–	–	–	2008	496
	65	–	–	–	–	–	–	1882
	70	–	–	–	–	–	–	–

Shaded areas give an example of the group size needed to demonstrate an improved outcome of 55% to 30% between two treatments.

TABLE 4: Generic outline of a rehabilitation programme for a superficial digital flexor tendon injury in a racehorse

Weeks after injury	Duration and nature of exercise
1–2	Box rest
3–4	10 min walking
5–6	15 min walking
7–8	20 min walking
9–10	25 min walking
11–12	30 min walking
13–14	35 min walking*
15–16	40 min walking
17–20	40 min walking; 5 min trotting
21–24	35 min walking; 10 min trotting
25–28	30 min walking; 15 min trotting
29–32	25 min walking; 20 min trotting
33–40	45 min exercise daily with slow canter†
41–48	45 min exercise daily with fast work 3 times a week
From 48 weeks	Return to full competition/race training

*The stage at which underwater treadmill exercise can be introduced (reduces limb loading by 40%). †The stage at which turnout can be considered. Based on the loading levels on the limbs at different gaits: walk, 60% bodyweight; trot, 90–100% bodyweight; and canter, 130–140% bodyweight.

discipline, i.e. upper level event horses and dressage horses may need more time, while showjumpers can sometimes be rehabilitated more rapidly; 2) the severity of the disease; and 3) the structure affected, i.e. deep digital flexor tendon disease might follow the same programme, while desmitis of the accessory ligament of the deep digital flexor tendon and the suspensory ligament would complete the same programme within less time.

Recommendations for future research

The following were considered the most important directions for research into tendon disease in the horse in the immediate future.

- Biomechanics:
  - Evaluation of 'microklutziness' in the horse.
  - Relationship of biomechanics to risk of injury, both in terms of 'macrobiomechanics' (i.e. of the limb) and biomechanics at a tissue level.
- Experimental models of tendon disease in the horse:
  - Validation of surgical models in the horse [17] and sheep [10].
  - Development and validation of *in vitro* models to identify molecular processes of degradation.
- Diagnostic imaging (ultrasonography and MRI) of tendon injuries.
- More sensitive and specific correlation with histology.
- Genetics of tendon disease.
- The influence of genetics on tendon biology and risk of injury.
- Prevention of tendon disease:
  - Development of training regimes to prevent tendon disease in both young and mature horses.
  - Epidemiological studies to identify risk factors.
  - The influence of surface to tendon injury.
- Medication targets in tendons.
- Development of new drugs to reduce the apparent excessive remodelling in tendons.
- 'Biological' therapies:
  - Mechanism and evidence-based outcomes for platelet-rich plasma and other growth factor/cytokine-related therapies.
  - Mechanism and evidence-based outcomes for cell-based therapies.
- Clinical trials:
  - Need to be well controlled, with an adequate number of horses per group (see power calculation above).
  - Need for randomised controlled trials, although this may not be possible/practical in the equine industry.
- Rehabilitation programmes

Authors' declaration of interests

No conflicts of interest were declared.

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