



Relationship between the ultrasonographic findings of suspected superficial digital flexor tendon injury and the prevalence of subsequent severe superficial digital flexor tendon injuries in Thoroughbred horses: a retrospective study

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ABSTRACT. The onset of severe injury to the superficial digital flexor tendon (SDFT) is extremely difficult to predict from slight changes in ultrasonographic findings in cases with no apparent clinical signs. This study investigated the relationship between an increased cross-sectional area (CSA) or edema in the subcutaneous tissue around the tendon and the subsequent onset of severe SDFT injury in Thoroughbred racehorses. Horses were classified into three groups based on ultrasound diagnosis (USD) findings: Group A included cases with enlarged tendons; Group B included cases with tendons of normal size but with prominent edema in the peritendinous tissue; and Group C (control group) included cases with no abnormal USD findings. The incidence of subsequent severe tendon injury was significantly higher in the horses in Groups A (25.7%, 28/101) and B (28.3%, 65/212) than in those in Group C (4.9%, 2/41). There were no significant differences in the median period and the median number of races from the first examination to the subsequent tendon injury between Groups A (140 days, 1 race) and B (120 days, 1 race). The results of this study revealed that horses with increased CSA and peritendinous edema are likely to suffer a subsequent severe tendon injury. Also, these two USD findings, i.e., increased CSA and peritendinous edema, indicate the risk of onset of severe SDFT injury.

KEY WORDS: horse, hypoechoic lesions, suspected superficial digital flexor tendon, ultrasonographic finding

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Injury of the superficial digital flexor tendon (SDFT) is common in Thoroughbred horses [5]. Unfortunately, complete repair does not occur [3] because scar tissue that forms during the healing phase results in material properties inferior to the normal tendon tissue. This inadequate tissue healing causes a high risk of re-injury [8]. Up to 67% of horses experience relapse within 2 years of the original injury [3, 8]. Moreover, horses with SDFT injury can run only a few races; however, these horses can return to running activities after rehabilitation [15]. Numerous owners and trainers of performance horses regard tendon injuries as potentially more threatening to an equine athlete's future career than fractures [7].

The clinical signs of SDFT injury vary. These signs include swelling and thickening of the affected area, increased surface temperature, and painful responses to palpation at the site of injury. Ultrasonographic imaging has been used to confirm the diagnosis and objectively identify the injury in tendon tissue [11, 13]. Anechoic and hypoechoic lesions are the principal diagnostic findings for SDFT injury by conventional B-mode ultrasound diagnosis (USD) [11, 13]. However, in early stages of tendon injury, regions of alteration in echogenicity of the tendon matrix may not be apparent. Thus, in these cases, clinicians have difficulty determining the possibility of continued training and exercise because extremely few investigations have focused on early changes in USD findings associated with SDFT injury.

SDFT injuries are considered an accumulation of microdamage and degenerated tendon tissue [10, 17], usually observed postmortem in horses that exhibited no clinical signs of SDFT injury [2, 16]. Without active repair of the fatigue damage, the

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tendons will weaken and eventually rupture [6]. A variety of stages of tendon damage are visible even without visible anechoic and hypochoic lesions.

Some controversy has arisen regarding the ability of USD to detect subclinical or preclinical lesions. In cases with subtle signs, the only finding is often slight heat and subtle swelling at the palmar aspect of the metacarpal region. In these cases, clinicians are often limited to establishing a more detailed and earlier diagnosis of SDFT injury using ultrasonography [14]. Edema in subcutaneous tissues and a larger cross-sectional area (CSA) of the SDFT might be early signs of injury. A significant increase in CSA with training is often more attributable to injury than adaptation [4]. In contrast, Avella *et al.* [1] reported no changes in CSA or ultrasonographic appearance before the development of an injury with hypochoic lesions; however, the authors mentioned that because of the small number of cases included in their analysis, a larger study with more frequent examination could better identify these features.

We hypothesized that an increase in CSA of the SDFT or subcutaneous peritendinous edema detected using USD would predict the subsequent onset of severe tendon injury, even without obvious hypochoic lesions. The relationship between CSA outside the normal range [9] or edema in subcutaneous tissue and subsequent severe SDFT injury in Thoroughbred racehorses was analyzed.

MATERIALS AND METHODS

We conducted this retrospective investigation by reviewing the medical records of Thoroughbred horses registered with the Japan Racing Association (JRA) at the Miho Training Center between 2010 and 2012. Regardless of the presence or absence of clinical signs (swelling, increase in surface temperature, and painful response to palpation at the metacarpal region), we selected the cases that had undergone USD of the SDFT by JRA veterinarians. This USD was defined as the “first examination” in this study. We obtained information on the above clinical signs and sex, age, and weight. The racing histories of all horses retrieved from the initial examinations were reviewed to confirm their experience of steeplechase racing. Horses with more than one steeplechase racing experience were considered steeplechase horses.

Before an ultrasonographic examination, a horse was sedated with intravenous medetomidine (0.004 mg/kg; Domitor; ZENOAQ, Fukushima, Japan), as needed, and the hair over the palmar metacarpal area was clipped. All B-mode ultrasonographic examinations throughout this study used the same ultrasound system (HI VISION Preirus Avius; Hitachi Medical Corp., Tokyo, Japan) and the same linear-array transducer (EUP-L53; 5–10 MHz) with ultrasound gel and a standoff pad. Two or more veterinarians, some of whom had at least 15 years of experience in equine musculoskeletal ultrasonography, determined USD to eliminate operator-associated variation. The palmar metacarpal regions were categorized into the following seven zones (4 cm each) from proximal to distal: 1A, 1B, 2A, 2B, 3A, 3B, and 3C [14]. The images obtained from the regions and the seven zones showing the representative condition of injured tendons, including subcutaneous peritendinous edema, a hypochoic lesion, or increasing CSA, were recorded as electronic files.

We included only those cases with no previous history of SDFT injury and no hypochoic lesions in the tendon during the first examination in this investigation. We excluded cases in which horses had been retired within 1 month after the first examination because there would be an insufficient follow-up period required to obtain the prognosis of the cases. We classified the cases into three groups based on the most representative USD findings of the seven zones obtained at the first examination. The abnormal values of CSA in each zone were set according to previous studies [9] (Table 1). Group A included cases with abnormal CSA values, with or without peritendinous tissue edema. Group B included cases with CSA within the normal range but with obvious peritendinous tissue edema. Group C (control group) included cases with no abnormal USD findings. These cases were performed to confirm the diagnosis of wound or dermatitis in the metacarpal region without tendon injury. All of the ultrasonographic images obtained at the first examination were reviewed by two veterinarians (M.I. and K.S.) to classify the cases into three groups.

Figure 1 shows representative ultrasonographic images of each group.

We investigated the incidence of severe tendon injury with an obvious hypochoic lesion at subsequent USD in the three groups during the follow-up period. The follow-up period was up to the onset of subsequent tendon injury or one year from the first

Table 1. Cross-sectional area (CSA) value judged to be abnormal value in each zone

Zone	CSA
1A (0–4 cm)	1.24<
1B (4–7 cm)	1.20<
2A (7–10 cm)	1.23<
2B (10–14 cm)	1.24<
3A (14–18 cm)	1.25<
3B (18–23 cm)	1.30<
3C (23–28 cm)	1.46<

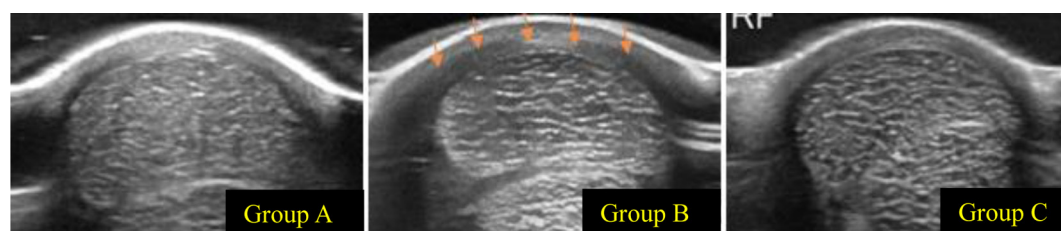


Fig. 1. Representative ultrasonographic images of each group.

examination. We compared the duration from the first examination to the onset of subsequent severe tendon injury and number of race starts during that period between onset horses in Group A and those in Group B. However, statistical analysis in the three groups was not possible because the data of onset horses in Group C were lacking.

Statistical analysis was performed using Kruskal–Wallis and Steel–Dwass tests for the median values of age and weight, respectively. Fisher’s exact test was used to analyze the percentage of males and steeplechase horses in each group. Based on the prognostic information of each case collected during the follow-up period, Fisher’s exact test was used for comparing differences in the incidence rates of subsequent severe SDFT injury after the first examination among Groups A, B, and C. Mann–Whitney test was used to compare the duration and number of races between onset horses in Group A and those in Group B. *P* values <0.05 were considered to be statistically significant.

RESULTS

Our evaluation included 354 horses. According to the inclusion criteria, 101 horses (28.5%) were classified into Group A, and 212 horses (59.9%) were classified into Group B. The remaining 41 horses (11.6%) were classified into Group C. The percentage of steeplechase horses in Groups A, B, and C were 16.8% (17/101), 17.5% (37/212), and 19.5% (8/41), respectively. There were no significant differences in the percentage of steeplechase horses among the three groups. Table 2 presents the median values for age, weight, CSA, and the percentage of males in each group. The prevalence of SDFT injury was not significantly associated with age, weight, and sex.

The incidence of subsequent severe SDFT injury after the first examination was 25.7% (28/101), 28.3% (65/212), and 4.9% (2/41) for Groups A, B, and C, respectively. Significant differences in the incidence of subsequent severe tendon injury were observed between horses in Group A and those in Group C (*P*=0.0045) and between horses in Group B and those in Group C (*P*=0.0006). However, no difference in the incidence of subsequent severe tendon injury was observed between horses in Group A and those in Group B (*P*=0.8913).

A subpopulation statistical analysis of subsequent severe tendon injury in Groups A and B revealed that the median duration (range) from the first examination to the onset of subsequent severe tendon injury was 140 (9–273) days in Group A and 120 (16–360) days in Group B. The median number (range) of race starts during the period from the first examination to the onset of subsequent severe tendon injury was 1 (0–10) in Group A and 1 (0–9) in Group B. Therefore, it was revealed that there was no difference between horses in Group A and those in Group B in terms of the median duration from the first examination to the onset of subsequent severe tendon injury and the median number of race starts.

DISCUSSION

This is the first study involving a relatively large dataset to analyze the correlation between suspicious injury findings obtained by USD, namely, CSA outside the normal range, or edema in the subcutaneous tissue around the SDFT, and subsequent occurrence of severe tendon injury in Thoroughbred racehorses. Our results showed that subsequent severe tendon injury incidence was significantly higher in the horses with an enlarged tendon or subcutaneous edema compared with the control horses. USD findings of an increased CSA value and subcutaneous edema, even if not accompanied by hypochoic findings, seem strongly related to the onset of subsequent severe injury. Approximately 60% of the horses with a subsequent severe tendon injury in Groups A and B could run only one or a few races after the first examination.

No significant differences in the incidence of subsequent tendon injury between Groups A and B. In addition, there was no significant difference in the number of races completed during the period from the first examination until the onset of injury between Groups A and B. These results suggest that both USD findings include increased CSA and peritendinous edema, indicating the same risk of onset.

Eventually, more than 25% of horses in Groups A and B developed subsequent severe tendon injury, although they presented with no obvious hypochoic lesions at first examination. Regarding the early detection of tendon injury, Reef [12] reported that an increased CSA, decreased echogenicity, and regularity of fiber alignment was an early indicator of injury. However, the edema in subcutaneous tissues around the SDFT has not been considered an early indicator of tendon injury. Particularly, peritendinous edema is associated with the same risk of developing subsequent severe SDFT injury as increased CSA, which may be caused by a change in the tendon tissue material itself. These results suggest that both increased CSA and the formation of edema that is not accompanied by hypochoic findings are associated with an increased risk of subsequent severe tendon injury.

Table 2. Age year, weight, and cross-sectional area (CSA, median (range)) and percentage of males in Groups A–C

	Age year median (range)	Weight kg median (range)	CSA cm ² median (range)	Male %
Group A	3 (2–10)	477 (406–548)	1.37 (1.24–2.36) ^a	66.3
Group B	3 (2–8)	474 (412–548)	1.07 (0.72–1.25) ^b	52.8
Group C	3 (2–8)	474 (436–532)	1.03 (0.83–1.25) ^b	58.5

a,b: Significant difference between different letters.

A complete understanding of the mechanisms of healing and appropriate treatment strategies for SDFT injuries has not been established. Two stimuli, i.e., microdamage accumulation and SDFT tissue degeneration, may cause processes leading to more serious injury [10, 17]. The accumulation of microdamage and the degeneration of tendon tissue are frequently observed in SDFTs that exhibited no clinical signs of injury [2, 16]. Our findings of an increased CSA or the formation of edema in subcutaneous tissue around SDFT may suggest an accumulation of microdamage and degeneration of tendon tissue above a certain level.

The limitations of this investigation include the possibility that the ultrasound examination of SDFT immediately after the beginning of degeneration or inflammation in tendon tissue did not detect anechoic or hypoechoic lesions. In these cases, a recheck should be performed within one week of the original assessment [13]; however, the horses included in this study were not re-examined. The data on ultrasound examinations conducted between the initial examination and the occurrence of the more severe injury would be essential in evaluating the condition of the tendon in detail. In future studies, the frequency of tendon ultrasonographic examinations should be increased. While assessing the regularity of tendon fiber alignment is an early indicator of tendon injury, we did not include a fiber alignment assessment. Many evaluators (i.e., different veterinarians at a racehorse hospital) had recorded the data we used, and the uniformity of such a score may not have been maintained. Veterinarian advices on ultrasonographic results might have affected the training menu after examination. The difference in the training menu might be associated with the incidence of subsequent severe SDFT injury. Additional investigation of the post-examination training menu may lead to a better understanding of the relationship between training and pathophysiology of subsequent severe tendon injury.

This investigation revealed that racehorses with an enlarged tendon (increased CSA) or the formation of edema in the subcutaneous tissue around the SDFT would suffer a subsequent severe tendon injury, even though USD revealed no hypoechoic lesions. Both USD findings, i.e., increased CSA and peritendinous edema, indicate the risk of onset of severe SDFT injury. Our results provide a scientific basis that enlarged tendons or the formation of edema observed on USD are important early indicators of SDFT injury. In cases with only mild clinical symptoms, such as heat and swelling at the palmar aspect of the metacarpal region, we recommend USD for early detection of injury and to prevent severity exacerbation. In addition, identifying these symptoms can help reduce the incidence of subsequent severe SDFT injury. We expect these data to be a valuable resource for further assessing the risks and screening methods available for the detection of early or impending tendon injuries.

CONFLICT OF INTEREST. All authors declare no conflicts of interest associated with this manuscript.

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REFERENCES

1. Avella, C. S., Ely, E. R., Verheyen, K. L., Price, J. S., Wood, J. L. and Smith, R. K. 2009. Ultrasonographic assessment of the superficial digital flexor tendons of National Hunt racehorses in training over two racing seasons. *Equine Vet. J.* **41**: 449–454. [Medline] [CrossRef]
2. Birch, H. L., Bailey, A. J. and Goodship, A. E. 1998. Macroscopic ‘degeneration’ of equine superficial digital flexor tendon is accompanied by a change in extracellular matrix composition. *Equine Vet. J.* **30**: 534–539. [Medline] [CrossRef]
3. Dyson, S. J. 2004. Medical management of superficial digital flexor tendonitis: a comparative study in 219 horses (1992–2000). *Equine Vet. J.* **36**: 415–419. [Medline] [CrossRef]
4. Gillis, C. L., Meagher, D. M., Pool, R. R., Stover, S. M., Craychee, T. J. and Willits, N. 1993. Ultrasonographically detected changes in equine superficial digital flexor tendons during the first months of race training. *Am. J. Vet. Res.* **54**: 1797–1802. [Medline]
5. Kasashima, Y., Takahashi, T., Smith, R. K., Goodship, A. E., Kuwano, A., Ueno, T. and Hirano, S. 2004. Prevalence of superficial digital flexor tendonitis and suspensory desmitis in Japanese Thoroughbred flat racehorses in 1999. *Equine Vet. J.* **36**: 346–350. [Medline] [CrossRef]
6. Ker, R. F. 2002. The implications of the adaptable fatigue quality of tendons for their construction, repair and function. *Comp. Biochem. Physiol. A Mol. Integr. Physiol.* **133**: 987–1000. [Medline] [CrossRef]
7. Lam, K. H., Parkin, T. D., Riggs, C. M. and Morgan, K. L. 2007. Descriptive analysis of retirement of Thoroughbred racehorses due to tendon injuries at the Hong Kong Jockey Club (1992–2004). *Equine Vet. J.* **39**: 143–148. [Medline] [CrossRef]
8. Marr, C. M., Love, S., Boyd, J. S. and McKellar, Q. 1993. Factors affecting the clinical outcome of injuries to the superficial digital flexor tendon in National Hunt and point-to-point racehorses. *Vet. Rec.* **132**: 476–479. [Medline] [CrossRef]
9. Oikawa, M. and Kasashima, Y. 2002. The Japanese experience with tendonitis in racehorses. *J. Equine Sci.* **13**: 41–56. [CrossRef]
10. Patterson-Kane, J. C. and Firth, E. C. 2009. The pathobiology of exercise-induced superficial digital flexor tendon injury in Thoroughbred racehorses. *Vet. J.* **181**: 79–89. [Medline] [CrossRef]
11. Reef, V. B. 1998. *Equine Diagnostic Ultrasound*, 1st ed., W.B. Saunders, Philadelphia.
12. Reef, V. B. 2001. Superficial digital flexor tendon healing: ultrasonographic evaluation of therapies. *Vet. Clin. North Am. Equine Pract.* **17**: 159–178, vii–viii. [Medline] [CrossRef]
13. Ross, M. W., Genovese, R. L., Dyson, S. J. and Jorgensen, J. S. 2011. Superficial digital flexor tendonitis. pp. 706–726. In: *Diagnosis and Management of Lameness in the Horse*, 2nd ed. (Ross, M. W. and Dyson, S. J. eds.), W.B. Saunders, St. Louis.
14. Smith, R. K. W. and Cauvin, E. R. J. 2014. Ultrasonography of the metacarpus and metatarsus. pp. 73–105. In: *Atlas of Equine Ultrasonography*, 1st ed. (Kidd, J.A., Lu, K.G. and Frazer, M.L. eds.), John Wiley & Sons, Hoboken.
15. Tamura, N., Kodaira, K., Yoshihara, E., Mae, N., Yamazaki, Y., Mita, H., Kuroda, T., Fukuda, K., Tomita, A. and Kasashima, Y. 2018. A retrospective cohort study investigating risk factors for the failure of Thoroughbred racehorses to return to racing after superficial digital flexor tendon injury. *Vet. J.* **235**: 42–46. [Medline] [CrossRef]
16. Webbon, P. M. 1977. A post mortem study of equine digital flexor tendons. *Equine Vet. J.* **9**: 61–67. [Medline] [CrossRef]
17. Wilson, A. M. and Goodship, A. E. 1994. Exercise-induced hyperthermia as a possible mechanism for tendon degeneration. *J. Biomech.* **27**: 899–905. [Medline] [CrossRef]