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Ischemic Degeneration of the Posterior Tibial Tendon in Rabbits

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The pathophysiology of rupture of the posterior tibial tendon has not been extensively investigated. The purpose of this study was to evaluate the possible role of ischemia in the degeneration of the posterior tibial tendon in an animal model. The animal subjects, consisted of forty New Zealand rabbits, was divided into 4 groups. Both or either one of the intrinsic and extrinsic nutritional supplies to a segment of the posterior tibial tendon was abolished and the tendons were harvested for examination three weeks later. The results showed that ischemia can lead to degeneration and rupture of the posterior tibial tendon. Intratendinous vascular flow is the main source of nutrition for the posterior tibial tendon. Nutrition obtained by diffusion from the synovial fluid itself is not sufficient to maintain the integrity of this tendon. Tension sustained in the posterior tibial tendon may aggravate the ischemic insults to the tendon substance. (J Rehab Med Assoc ROC 2001; 29(2): 77 – 82)

Key words: ischemia, degeneration, posterior tibial tendon, animal study

INTRODUCTION

Rupture of the posterior tibial tendon is common in elderly individuals. Degenerative changes, pre-existing tenosynovitis, constriction beneath the flexor retinaculum, and the anatomical course of the tendon have all been implicated as causes of rupture of the posterior tibial tendon [1-4]. Although numerous authors [5-8] have discussed various etiologies of this disorder, the pathophysiology has not been extensively investigated.

The potential vulnerability of the posterior tibial tendon to injury due to poor vascular supply was perhaps first suggested by Harris in 1942 [9]. Relative avascularity in a tendon can cause focal cell death in affected areas, with resultant degenerative changes and rupture. Frey et al. [10] reported that a zone of hypovascularity could be identified in the posterior tibial tendon posterior and distal to the medial malleolus by injecting cadaveric limbs with an India ink-gelatin mixture. They suggested that relative avascularity and resulting degenerative changes might be the predisposing factors in rupture of the posterior tibial tendon.

The normal nutritional supply of a tendon consists of a dual system. One is from the vessel that enters the tendon (intrinsic supply). The other is from synovial diffusion (extrinsic supply). The purpose of this study was to investigate the possible role of ischemia played in


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the degeneration of the posterior tibial tendon using an animal model.

**MATERIALS AND METHODS**

Forty New Zealand rabbits weighing from 3 to 4 kilograms were divided into 4 groups. General anesthesia with atalant was administered to the rabbits, and then the leg skin was prepared and disinfected. In group 1, the posterior tibial tendon was dissected out and a 2-centimeter segment of the tendon behind the medial malleolus was wrapped around with an adhesive membrane and both ends ligated tightly with silk (Fig. 1) thereby totally obliterating both the intrinsic and extrinsic nutritional supplies to this segment of the tendon. In group 2, a segment of the tendon was wrapped around with an adhesive membrane to hinder the extrinsic nutritional supply, but the intrinsic blood flow was preserved. In group 3, a segment of the tendon was resected by two separated stab wounds from both ends of the posterior tibial tendon behind the medial malleolus to interrupt the intrinsic blood supply. However, the extrinsic nutritional mechanism was not disturbed. In group 4, a segment of the tendon was ligated at both ends to interrupt the intrinsic blood supply. The difference between group 3 and 4 was that the divided segment of the tendon in group 3 sustained no tension. In each group, the experiment was performed in one leg and the other leg was used for sham operation. Three weeks later, the tendons were harvested for examinations, including gross evaluation and microscopic examination with H&E stain.

**RESULTS**

Grossly, the normal posterior tibial tendon of the

![Fig. 1](image-url)  
*Fig. 1.* A segment of the posterior tibial tendon was wrapped around with an adhesive membrane and both ends ligated tightly with silk in group 1. A segment of tendon was wrapped around with an adhesive membrane in group 2. A segment of tendon was divided at both ends through two separate stab wounds on the rabbit's leg in group 3. A segment of tendon was ligated at both ends in group 4.
rabbits appeared white and shiny. The consistency was firm and it exhibited a high tensile strength. The tendons in group 1 became necrotic and ruptured and had lost their shiny appearance. Group 2 tendons had an appearance similar to the control tendons. The group 3 tendons became less shiny and thinner than that of the sham groups. The group 4 tendons looked less shiny and mildly swollen (Fig. 2).

Under microscopic examination, the tenocytes were totally absent from the tendon substance in group 1 and collagen fibers became necrotic. In group 2, the collagen bundles were uniform in size and had a parallel arrangement. Elongated tenocytes scattered among the collagen bundles were noted (Fig. 3).

Shrinkage of the collagen bundles was apparent in group 3. The cellularity appeared relatively increased and the tenocytes were substantially enlarged. Edematous change was found in the collagen substance in group 4. The tenocytes were mildly swollen and deranged (Fig. 4). The degenerative change of the tendons was more significant in group 4 than group 3, particularly in the central portion of the ligated segment.

Kannus and Jozsa reported a characteristic histopathological patterns in biopsy specimens from spontaneously ruptured tendons in 891 patients. Most (97%) of the

![Fig. 2](image1.png)  
**Fig. 2.** The tendon from group 1 became necrotic and ruptured. Group 2 tendons had a similar appearance to the control tendons. The group 3 tendons became less shiny and thinner than that of the sham groups. The group 4 tendons looked less shiny and mildly swollen. Black stitches were noted at both ends of the harvested tendon.

![Fig. 3](image2.png)  
**Fig. 3.** Upper: The tenocytes were totally absent from the tendon substance in group 1 and collagen fibers became necrotic. Lower: In group 2, the collagen bundles were uniform in size and had a parallel arrangement. Elongated tenocytes scattered between the collagen bundles were noted. (HE stain, 200X)
pathological changes were degenerative and included hypoxic degenerative tendinopathy, mucoid degeneration, tendolipomatosis, and calcified tendinopathy, either alone or in combination [11].

This observation confirms the findings of Arner et al. [12] and Konn and Everth [13] that spontaneous rupture of a tendon, almost without exception, is preceded by degenerative changes — that is, degenerative or calcifying tendinopathy, or mucoid degeneration. Hypoxic degenerative tendinopathy was the most frequent finding in ruptured tendons, both those that had a single type of degeneration and those that had multiple types. Using light microscopy, Arner et al. [12] found edema and disintegration of the tendon, characterized by fragmentation of collagen fibers, as well as absence or decreased numbers of tenocytes.

The basic mechanism of the hypoxic degenerative changes in tendons has not been vigorously established, although decreased arterial blood flow has been proposed most frequently. Hastad et al. [14] used measurement of the uptake of the injected isotope Na to show that the blood flow in the Achilles tendon decreases after the third decade of life. These observations are consistent with the histological findings of Arner et al. [12] and Kannus et al. [11]. In both of these studies, the ruptured tendons and their paratenons often exhibited narrowing and obliteration of the lumina of the medium-caliber arteries due to hypertrophy of the media. Occasionally, thrombosis or arteritis was also found.

In areas of decreased blood supply, the survival of cells depends on the ability of tissue fluids to diffuse through the tendon. In his study of tendon healing, Lundborg [15-17] placed a segment of sutured rabbit flexor tendon in the synovial cavity of the knee joint as a free body. The subsequent lack of development of adhesions around the tendon segment and the bridging of the anastomotic gap led him to conclude that the sutured tendons healed by the intrinsic ability of their tenocytes to produce collagen. Menon et al. [18] further demonstrated that healing of an irradiated nonviable tendon was brought about by the cells present in the synovial fluid after the killing of the tenocytes in the tendon itself by radiation. This finding suggests that these tissue areas, in analogy to joint cartilage, are nourished by diffusion from the synovial fluid [19]. However, Smith [20] showed that, even in a specimen from a young, healthy individual, diffusion cannot support the metabolism of the tendon if the tendon is more than one to two centimeters from the nutritional source.

In this study of a rabbit model, when the extrinsic and intrinsic supplies were abolished simultaneously, the tendon lost all supplies and then became necrotic and eventually ruptured as in group 1. In group 2, the intrinsic supply remained intact though the extrinsic supply was blocked. We believed the intrinsic supply be the key nutritional supply based on the finding that the integrity of the tendon was preserved. In group 3 and 4, the intrinsic supply was deprived and the tendon showed degen-
operative changes. This implied that extrinsic supply alone could not maintain the metabolic need of that tendon. In group 4, the tendon sustained a tension not experienced in group 3, the degenerative findings were more severe. It was thus postulated that tension would have aggravated the ischemic changes.

In conclusion, ischemia plays a major role in the degeneration and rupture of the posterior tibial tendon in rabbits. The intrinsic supply is the key nutritional supply to the tendon. Tension on the tendon aggravates the impact of ischemia.

REFERENCES

以動物實驗觀察因缺血造成之脛後肌肌腱退化

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脛後肌肌腱結裂的病態生理現象至今仍未有廣泛的研究。本文的目的乃利用兔子動物模型來探討缺
血在脛後肌肌腱退化中可能扮演之角色。我們利用40 隻兔鼠分白兔組之分為四組，將一段脛後肌肌腱
的內在性與外在性供應途徑阻斷其一或同時阻斷，三星期後將此段肌腱取出，做病理學之觀察。實
驗結果顯示，缺血的確可以造成兔子脛後肌肌腱的退化甚至斷裂。而肌腱內在性的血循環乃是脛後肌
腱主要的營養供應途徑，從外側滑膜囊液滲透進入肌腱的養份並不足以維持脛後肌肌腱的完整性。脛後
肌肌腱所承受的張力會加重缺血對肌腱所造成的傷害。（中華復健醫誌 2001; 29(2): 77 - 82）

關鍵詞：缺血(ischemia)，退化(degeneration)，脛後肌肌腱(posterior tibial tendon)，動物實驗(animal study)