

Simultaneous Bilateral Quadriceps Tendons Rupture in a Patient with Polyneuropathy

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A 66 year old man with a history of sensory motor polyneuropathy of unknown etiology presented to the emergency department with bilateral suprapatellar knee pain and swelling after a fall. He was unable to walk or actively extend his knees after the incident. Radiographs of the knees showed abnormal soft tissue contour in the suprapatellar region and abnormal calcific opacities several centimeters cephalad to the patella in both knees

[Figure 1]. Bedside ultrasound using a high frequency linear probe was performed [1]. The high resolution images showed irregular thickening and retraction of the quadriceps tendons which were attached to hyperechoic foci well above the patella; in addition, anechoic fluid collections were identified. These findings confirmed the clinical and radiographic diagnosis of bilateral quadriceps tendon rupture with avulsion of the superior pole of the patella, and acute hematoma in the suprapatellar bursa [Figure 2]. One day after the accident, the patient underwent surgery with primary repair of both quadriceps tendons. His legs were then immobilized in full extension by tutor casts for 6 weeks. The injury was suspected to be associated with the polyneuropathy as the patient had no other identifiable risk factors.

Simultaneous bilateral quadriceps tendon rupture is a rare injury, generally occurring in men over the age of 50 who are diabetic, obese, or have age-related changes in their tendons [1]. Other factors leading to tendon rupture mostly in younger individuals include local steroid injections, use of anabolic steroids, history of chronic tendonitis, chronic renal failure, hyperparathyroidism, rheumatoid arthritis, systemic lupus, and gout [1]. A similar case of bilateral quadriceps tendons rupture after statin use has been described [1]. These predisposing conditions cause tendon degeneration by altering collagen synthesis, resulting in sclerosis or fibrosis in the tendon, fatty degeneration, decreased tendon collagen composition, necrosis, or calcification [2].

The quadriceps tendon is inherently a very strong structure that is extremely resistant to heavy load [3]. The tendon can rupture spontaneously or as a result of trauma. The most common cause of simultaneous bilateral quadriceps tendon rupture appears to be either a fall [4] or a sudden violent eccentric contraction of the quadriceps mechanism against the body weight with the knee slightly flexed and the feet in a fixed position [2,3]. The commonest site of rupture is the osseotendinous junction [4].

Early diagnosis in quadriceps tendon rupture is important, because a delay in treatment can result in considerable morbidity. Tendon retraction, fibrosis, and atrophy reduce the possibility of successful operative repair [5].

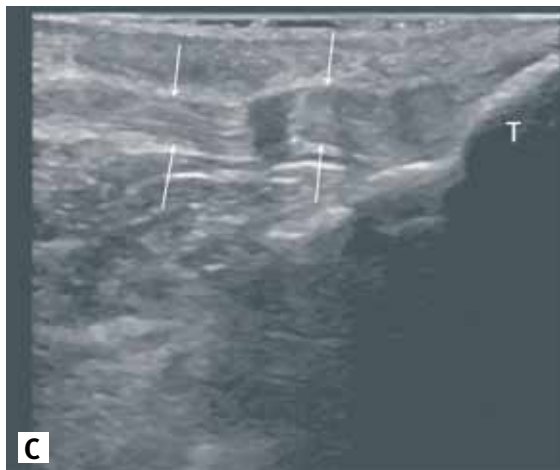
Plain knee radiographs [Figure 1] can be suggestive of underlying conditions, such as the calcified opacities found in our patient, suggestive of chronic tendinopathy

Figure 1. Lateral radiographs of the knees

[A] Right knee, with depression in the soft tissue contour in the suprapatellar region and a calcified opacity superior to the patella. **[B]** Left knee, with similar findings



Figure 2. Sagittal ultrasound images of the knees



[A] Area of the quadriceps tendon of the right knee. The quadriceps tendon (between thin arrows) extends only to a hyperechoic focus (thick arrow), but not to the patella (P). There is a hematoma cephalad to the patella (*)

[B] Area of the quadriceps tendon of the left knee, with a similar picture but less obvious hematoma formation

[C] Right patellar tendon. There is a wavy appearance to the tendon (between arrows) proximal to its insertion on the tibia (T), reflecting quadriceps tendon tear and caudal displacement of the patella, with resulting relaxation of the patellar tendon

[3]. Diagnosis can be confirmed by ultrasound [Figure 2], which is an inexpensive, sensitive, simple and reliable method for diagnosing tendon ruptures, whether partial or complete, at the bedside.

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References

1. Nesselroade RD, Nickels LC, Ultrasound diagnosis of bilateral quadriceps tendon rupture after statin use. *West J Emerg Med* 2010; 11 [4]: 306-9.
2. Chiu M, Forman ES, Bilateral quadriceps tendon rupture: a rare finding in a healthy man after minimal trauma. *Orthopedics* 2010; 10: 203-5.
3. Arumilli B, Adeyemo F, Samaraji R, Bilateral simultaneous complete quadriceps rupture following symptomatic tendinopathy: a case report. *J Med Case Reports* 2009; 3: 9031.
4. Senevirathna S, Rhada S, Rajeev A, Bilateral simultaneous rupture of the quadriceps tendon in a patient with psoriasis: a case report and review of the literature. *J Med Case Reports* 2011; 5: 331.
5. Chung KL, Wong TT, Yuen MK, Kam CW, Sonography of quadriceps tendon ruptures. *Hong Kong J Emerg Med* 2004; 11: 169-72.

Capsule

Ultraviolet radiation-induced inflammation promotes angiotropism and metastasis in melanoma

Intermittent intense ultraviolet (UV) exposure represents an important etiological factor in the development of malignant melanoma. The ability of UV radiation to cause tumor-initiating DNA mutations in melanocytes is now firmly established, but how the microenvironmental effects of UV radiation influence melanoma pathogenesis is not fully understood. Bald et al. report that repetitive UV exposure of primary cutaneous melanomas in a genetically engineered mouse model promotes metastatic progression, independent of its tumor-initiating effects. UV irradiation enhanced the expansion of tumor cells along abluminal blood vessel surfaces and increased the number of lung metastases. This effect depended on the recruitment and activation of neutrophils, initiated by the release of high mobility group box 1 (HMGB1) from UV-damaged epidermal keratinocytes and driven by Toll-like receptor 4 (TLR4). The UV-induced neutrophilic inflammatory response stimulated angiogenesis and promoted the ability of melanoma cells to migrate towards endothelial cells and

use selective motility cues on their surfaces. These results not only reveal how UV irradiation of epidermal keratinocytes is sensed by the innate immune system, but also show that the resulting inflammatory response catalyses reciprocal melanoma-endothelial cell interactions leading to perivascular invasion, a phenomenon originally described as angiotropism in human melanomas by histopathologists. Angiotropism represents a hitherto underappreciated mechanism of metastasis that also increases the likelihood of intravasation and hematogenous dissemination. Consistent with these findings, ulcerated primary human melanomas with abundant neutrophils and reactive angiogenesis frequently show angiotropism and a high risk for metastases. This study indicates that targeting the inflammation-induced phenotypic plasticity of melanoma cells and their association with endothelial cells represents rational strategies to specifically interfere with metastatic progression.

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“Learning is weightless, a treasure you can always carry easily”

Chinese proverb