Acute compartment syndrome may be a result of any trauma to the limb. The trauma is usually a result of an open or closed fracture of the bones, or a crush injury to the limb. Other causes include haematoma, gun shot or stab wounds, animal or insect bites, post-ischaemic swelling, vascular damage, electrical injuries, burns, prolonged tourniquet times, etc. Other causes of compartment syndrome are genetic, iatrogenic, or acquired coagulopathies, infection, nephrotic syndrome or any cause of decreased tissue osmolality and capillary permeability.

Chronic compartment syndrome is most typically an exercise induced condition characterised by a relative inadequacy of musculofascial compartment size producing chronic or recurring pain and/or disability. It is seen in athletes, who often have recurring leg pain that starts after they have been exercising for some time. There is no history of trauma. The pain is localised to the involved muscles or the entire compartment and the symptoms are very similar to acute compartment syndrome. The symptoms often settle with rest, however if the athlete returns to his sport after laying off, the symptoms usually recur. The treatment of confirmed chronic compartment syndrome is a surgical fasciotomy of the affected compartments.

Essentially, any cause of increased compartment pressure can result in a compartment syndrome. Muscle rupture has been implicated in causing increased compartment pressures in athletes after severe exercise. The diagnosis of acute compartment syndrome due to rupture

of the body of gastrocnemius has been reported in athletes.^{7,8} This, however, is the first reported case of acute compartment syndrome caused by a gastrocnemius muscle rupture in a non-athlete.

Conclusion

Soft tissue injuries and muscle tears occur frequently in athletes. Most injuries result from direct trauma. Indirect trauma resulting in muscle tears and ruptures can cause acute compartment syndrome in athletes. It is also important to keep in mind the possibility of similar injuries in a non-athlete as well. More research is needed to define optimal management patterns and potential strategies for injury prevention.

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Atraumatic bilateral Achilles tendon rupture: an association of systemic steroid treatment

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Abstract

A case of bilateral Achilles tendon rupture associated with steroid use is reported. This case illustrates the importance of taking a thorough drug history in cases of tendon rupture. In lower limb tendon rupture all patients, especially those on steroids, should be warned of the increased risk of contralateral injury.

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Keywords: Achilles tendon; steroid use

Case report

A 67 year old man presented to the accident and emergency department with sudden onset of left sided calf pain that occurred while crossing a road. He was unable to weight bear on his left leg. This previously fit male had developed severe pneumonia needing intensive

care treatment two months previously, after which he had been started on oral prednisolone. His respiratory symptoms were well controlled at the time of presentation. There was no history of any tendon or joint pathology before the steroid treatment.

Examination revealed a palpable gap in his left Achilles tendon 2 cm proximal to the insertion on the calcaneum. A diagnosis of ruptured Achilles tendon was made and the patient was placed in an equinus plaster of Paris below knee cast. The next day repair of the tendon under local anaesthetic was performed utilising an open technique; at surgery the tendon appeared degenerate. The equinus cast was reapplied with a plan to change to the mid-equinus position in two weeks and then to a Samson boot for mobilisation of the ankle within four weeks.

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Five days later, the patient developed mild pain in his right calf on mobilising. A further two days later while walking at home with crutches, he developed severe pain in his right calf and presented with inability to weight bear on the right leg. Clinical examination confirmed rupture of the right Achilles tendon. The patient was admitted to hospital and placed in a below knee equinus plaster cast maintained for six weeks. A conservative approach was adopted for the rupture on the right side.

The patient was reviewed at three months and was able to fully weight bear on both legs with full range of movement at the ankle joints.

Discussion

The Achilles tendon is the largest and thickest tendon in humans. It is formed from the aponeuroses of gastrocnemius and soleus and inserts into the middle third of the posterior surface of the calcaneum, separated from the superior part of the tuberosity by a bursa. The two muscles are the principle plantarflexors of the foot at the ankle joint.

Achilles tendon rupture usually follows sudden forced movement at the ankle joint. It usually occurs in men between 30 and 50 years of age. There are usually no prodromal symptoms and histological examination reveals a normal tendon. Rupture can also occur after trivial loading such as during gentle locomotion, the so called atraumatic ruptures. Atraumatic rupture is incompletely understood and often occurs in an older age group, with prodromal symptoms as in this case. Histology usually reveals inflammatory changes consistent with a preceding tendinitis.¹ In this latter group, there is a high association with steroid use.

Tendon ruptures at the ankle have been described in association with rheumatoid arthritis,² gout, systemic lupus erythematosus,³ and chronic renal failure and renal transplantation.⁴ In addition, Achilles tendinopathies including rupture have been associated with quinolone antibiotic usage.⁵ In a study of 10 atraumatic cases five were associated with respiratory disease treated with systemic steroids.⁶ The mechanism of action is unclear but steroids may act by suppressing the repair of partially ruptured or degenerated tendons such that complete rupture may occur after relatively minor trauma. The dose or duration of steroid treatment does not appear to affect susceptibility to atraumatic tendon rupture.

The diagnosis of Achilles tendon rupture is usually clinical, although ultrasonography and

magnetic resonance imaging may be helpful in certain scenarios such as in the detection of tendon or paratenon inflammation, where appropriate treatment may prevent rupture.⁷ Treatment of the ruptured tendon(s) can be either operative or conservative; neither has proved superior, as demonstrated by the similar recovery time and functional outcome shown in our case. Surgery does facilitate early mobilisation but wound sepsis is a risk, given the poor local vascularity.⁸ Repair should be with strong permanent or slowly absorbable sutures, and may be effected percutaneously. Non-operative management, however, carries a greater risk of re-rupture.

In this case, there was no history of tendon or joint pathology before starting steroid treatment. The patient developed atraumatic bilateral Achilles tendon rupture after two months of steroid treatment. In addition, the rupture of the right Achilles tendon may have been caused by the added load on that leg after repair of the left tendon.

On assessment of the patient with a suspected Achilles tendon rupture in the accident and emergency department it is vital to obtain a thorough drug history. In most cases those on steroids will need to continue their treatment, but other medications such as quinolone antibiotics could be stopped or substituted. The patient should be advised regarding the increased risk of rupture of the contralateral tendon and encouraged to take extra care to decrease this risk.

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