

# Acute compartment syndrome of the foot in a soccer player: a case report

Michelle A. Laframboise, BKin (Hons), DC<sup>\*,†</sup>

Brad Muir, HBSc (Kin), DC, FRCCSS(C)<sup>\*,§</sup>

**Objective:** *To present the diagnostic and clinical features including management of acute compartment syndrome (ACS) of the foot and to create a sense of emergency amongst clinicians of this rare and dangerous condition.*

**Clinical Features:** *A 28-year old male soccer player on acetylsalicylic acid (ASA) and verapamil presented with severe swelling, paresthesia, and pain in the left ankle after an acute grade three-inversion ankle sprain. A diagnosis of foot compartment syndrome was made.*

**Intervention and Outcome:** *A fasciotomy was not performed and subsequent neurological sequelae occurred. We hypothesize that the edema caused by the ankle sprain was excessive due to the use of ASA and verapamil, resulting in increased compartmental pressure and neurological signs in the foot.*

**Summary:** *Although rare, it is extremely important to be aware of the clinical features of ACS of the foot to obtain an appropriate diagnosis and manage this medical emergency promptly.*

(JCCA 2011; 55(4):302-312)

**KEY WORDS:** acute compartment syndrome, inversion ankle sprains, soccer, aspirin, verapamil

## Introduction

Inversion ankle sprains causing disruption to the anterior talofibular ligament are one of the most common injuries seen in the athletic population.<sup>1</sup> Approximately 25% of all

**Objectif :** *présenter le diagnostic et les caractéristiques cliniques, notamment la gestion du syndrome des loges aigu du pied, et sensibiliser les cliniciens à cet état pathologique rare et dangereux.*

**Caractéristiques cliniques :** *un joueur de soccer de 28 ans consommant de l'acide acétylsalicylique et du vérapamil souffrait d'une grave enflure, de paresthésie et de douleurs dans la cheville gauche suite à une entorse d'inversion de la cheville de catégorie trois. On lui a diagnostiqué un syndrome des loges du pied.*

**Intervention et résultat :** *la fasciotomie n'a pas été exécutée, et des séquelles neurologiques s'ensuivirent. Nous supposons que l'œdème causé par l'entorse à la cheville était grave en raison de l'utilisation de l'acide acétylsalicylique et du vérapamil, qui a eu pour effet d'accroître la pression et les signes neurologiques dans le pied.*

**Sommaire :** *bien que rare, il est très important de connaître les caractéristiques cliniques du syndrome des loges aigu afin d'émettre un diagnostic précis et de gérer cette urgence médicale rapidement.*

(JCCA 2011; 55(4):302-312)

**MOTS CLÉS :** syndrome des loges aigu, entorses d'inversion de la cheville, soccer, aspirine, vérapamil

musculoskeletal injuries leading to medical treatment are acute ankle sprains and ruptures of the lateral ligaments of the ankle, most commonly caused by an inversion mechanism of injury.<sup>2</sup> Further, the most common injury occur-

\* Canadian Memorial Chiropractic College, 6100 Leslie Street, Toronto, Canada.

† Division of Graduate Studies, Sports Sciences, Canadian Memorial Chiropractic College, 6100 Leslie Street, Toronto, Ontario, Canada.

§ Assistant Professor, Division of Undergraduate Studies, Canadian Memorial Chiropractic College, 6100 Leslie Street, Toronto, Ontario, Canada.

Corresponding author: Dr. Michelle A. Laframboise, mlaframboise@cmcc.ca, T: (416) 482-2340 ext. 242; F: (416) 482-2560.

© JCCA 2011

ring in the sport of soccer is injury to the ankle region resulting in ankle sprains.<sup>3</sup> Ankle sprains are common in soccer players due to frequent contact with other players, repetitive jumping, running, and frequent abrupt changes in direction of motion also known as cutting movements. Cutting movements cause an excessive amount of angular and rotational strain on the ankle joint.<sup>4</sup> Initially, a common treatment plan for an acute inversion ankle sprain is to protect, rest, ice, compress, and elevate<sup>5</sup> (PRICE). This simple protocol is well documented in current literature to decrease swelling thereby decreasing time to heal and decreasing the time it takes for an athlete to return to play. However, this may not be the proper protocol when there is an increase in intracompartmental pressure (ICP) leading to acute compartment syndrome (ACS).<sup>6,7,8</sup> ACS of the foot is extremely rare after an inversion ankle sprain and may be complicated with the use of platelet inhibitors and calcium channel blockers.

This article presents a case of ACS of the foot following a grade three inversion ankle sprain in a 28-year-old male soccer player on a daily regimen of ASA (162 mg) and Verapamil (320 mg) pharmacotherapy. The patient consented to release all information in regard to his case for publication.

### Case report

A 28-year old male, recreational soccer player, presented to a chiropractor with a primary complaint of left lateral ankle pain and swelling surrounding and up to 6 cm proximal to the lateral malleolus following a high-energy deceleration injury into ankle inversion and plantar flexion. The patient attributed this injury to attempting to block a kick of the opposing player while playing soccer. Presentation to the chiropractor was fifteen minutes after initial trauma. The patient reported immediate sharp pain and swelling of the lateral aspect of the ankle. Initially, the pain intensity was 6/10. Medical history revealed this patient previously suffered two cryptogenic (of undetermined origin) ischemic strokes at age 17 and was prescribed 162 mg of ASA for platelet inhibition and 360 mg of verapamil to increase vasodilation. Hospitalization occurred for approximately one week after each stroke and pharmacotherapy was prescribed to decrease the chance of recurrent stroke. The patient was also on 40 mg of omeprazole used as a proton pump inhibitor to decrease hydrochloric acid formation in the stomach. No nutritive

icals were reported. The patient reported no other history of significant injury, trauma, or surgeries, and systems review was unremarkable.

On immediate physical examination, observation revealed that the distal 1/3 of the anterolateral aspect of the left leg and left foot was extremely swollen. There was pain on palpation of the distal 1/3 of the left extensor digitorum longus, extensor hallucis longus, the left lateral malleolus, and 6 cm above the lateral malleolus. Range of motion revealed limitations in dorsiflexion and plantarflexion of 10 degrees total in the left ankle. There was an inability to perform inversion and eversion due to extreme pain and swelling. Neurological examination revealed paresthesia on the dorsum of the foot, the 2<sup>nd</sup>, 3<sup>rd</sup>, 4<sup>th</sup> digits, and one inch inferior to the medial malleolus. Motor examination revealed a significant decrease in strength in all ranges of motion. Deep tendon reflexes were within normal limits (L4 and S1).

The chiropractor proceeded with radiographical evaluation of the left ankle and left tibia and fibula. Radiological examination revealed a small flake fracture of the distal aspect of the left fibula and severe soft tissue swelling (see Figure 1 and 2). The chiropractor provided the patient with crutches and a strict protocol of rest, ice, compression, and elevation for a grade three-inversion ankle sprain. Approximately nine hours after release from the chiropractor the patient experienced extreme swelling, hypersensitivity, and shooting pain in the dorsum of the left foot. The patient proceeded to the emergency department due to extreme constant sharp, electric like pain out of proportion of injury and rapidly deteriorating neurological signs.

Upon examination in the emergency room, vital signs revealed a temperature of 36.1, pulse rate 118 bpm, respiratory rate 20 bpm, and blood pressure was 134/86. The left foot was severely swollen, tender, and extremely painful and weak on dorsiflexion and plantarflexion of the toes and ankle. The character of the pain was reported as sharp and electric like. Pain intensity was reported as 10/10. Observation revealed a large one-inch blister forming on the lateral aspect of the left ankle (see Figure 3). Severe bruising and swelling of the left leg and foot was evident (see Figure 4 and 5). Further, there was severe pitting edema on the dorsum of the left foot and 1/3 of the distal aspect of the leg. The toes were slightly pale and capillary refill was approximately 2 seconds on left and

Figure 1 AP and medial oblique radiograph of the left ankle. Arrows indicate an avulsion fracture of the distal fibula (fragmentation).



<2 seconds on right. Sensory examination revealed continued paresthesia in the same distribution. The dorsalis pedis and posterior tibial pulses were not palpable due to extreme swelling. (See Table 1 and 2).

The patient was admitted to the hospital for observation and consultation by an orthopedic surgeon due to rapidly progressing pain and swelling in left foot and ankle and a differential diagnosis of ACS of the foot. The orthopedic surgeon suspected the patient might have sustained a subtalar dislocation with spontaneous reduction although no dislocation was visualized on radiographic imaging of left ankle and foot.

Due to the past history of cryptogenic ischemic strokes the patient was sent for complete doppler examination of left leg. The visualized portions of the common femoral, superficial femoral, popliteal, and calf veins were free of echogenic thrombus, with normal compression, augmentation, and phasicity (the cyclic rise and fall of venous

flow with normal respiration). There was no evidence of deep vein thrombosis in the left leg. Further, doppler ultrasound showed intact dorsalis pedis and posterior tibial pulses of the left foot.

A diagnostic ultrasound of the left ankle revealed a large hematoma on the lateral malleolus measuring  $3.7 \times 3.3 \times .69$  cm, the left anterior talofibular ligament (ATFL) was not intact, and bony fragments were found surrounding the distal aspect of the fibula (see Figure 6, 7 and 8).

Continuous inpatient monitoring occurred for 6 days with repeated clinical assessment every 2 hours. The ICP was not measured. The patient was placed in an open plaster cast with the foot raised to the level of the heart for 6 consecutive days. No change in medication was provided. Morphine 5–7 mg was prescribed intravenously every 15 minutes PRN (as needed), 10 mg oxycodone was administered orally every 6 hours PRN for pain control, and



Figure 2 Lateral radiograph of the left ankle. The arrow indicates severe soft tissue swelling of the left foot and ankle.

25 mg dimenhydrinate intravenously every 4 hours PRN for nausea relief.

The patient's left leg was immobilized and placed in a below-knee airform walker and released from the hospital after a 6-day duration. A fasciotomy was not performed. This patient was reviewed in the Outpatient Fracture Clinic 14 days after release from the hospital where another radiological ankle series was performed and read as unremarkable.

The patient received three weeks of extensive chiropractic treatment after discharge from the hospital, once the airform walker was removed and ambulation was near pre-injury state. Active Release Technique® was provided

Table 1 Blood Chemistry Results

Procedure	Value	Reference Range	Units
Urea	<b>8.3 H</b>	[2.0–8.0]	MMOL/L
Creatinine	<b>114 H</b>	[44–110]	UMOL/L
Anion Gap	<b>20 H</b>	[6–15]	MMOL/L
Potassium	<b>3.1 L</b>	[3.3–4.5]	MMOL/L
Carbon dioxide	<b>20 L</b>	[22–29]	MMOL/L
Glucose random	5.8	[3.8–6.1]	MMOL/L
Sodium	144	[133–145]	MMOL/L
Chloride	104	[95–107]	MMOL/L

*H* – Value higher than reference range

*L* – Value lower than reference range

to the anterior tibialis, extensor hallucis longus, extensor digitorum longus, extensor retinaculum, flexor hallucis longus, tibialis posterior, flexor digitorum longus, and achilles tendon. No mobilizations or manipulations of the ankle were performed. Range of motion exercises, rehabilitation, and proprioception exercises including single leg heel raises and theraband® concentric and eccentric exercises were prescribed. Rehabilitation exercises were completed at home for three months duration.

Upon one-year follow-up, weakness in dorsiflexion of the great toe on the left could be elicited. Motor examination of the extensor hallucis longus was 4/5. Weakness of the extensor hallucis longus may have been due to severe swelling of the foot leading to hypoxia and focal tissue necrosis of the deep fibular nerve or axonotmesis directly from the mechanism of injury. All other motor testing was within normal limits. The patient could actively dorsiflex the foot to 15 degrees and plantarflex to 45 degrees, normal ranges are 20 degrees and 50 degrees respectively. Sensory deficits of the left foot included paresthesia of the dorsum of the left foot, 2<sup>nd</sup>, 3<sup>rd</sup>, and 4<sup>th</sup> digits, and one inch inferior to the medial malleolus. Axonotmesis, a peripheral nerve injury occurred to the superficial fibular nerve causing sensory disruption in the dorsum of the foot and digits and to the tibial nerve causing sensory disruption one inch inferior to the medial malleolus. Posterior tibial and dorsalis pedis pulses were palpable. No contractures of the toes or deformities of the affected leg or foot were noted. The patient was not engaged in any intervention

Table 2 Hematology Results

Procedure	Value	Reference Range	Units
Leukocyte count	<b>15.0 H</b>	[4.0–11.0]	10E9/L
Red blood cell distribution	<b>0.147 H</b>	[0.115–0.140]	
Neutrophils	<b>10.90 H</b>	[2.0–7.5]	10E9/L
Hemoglobin	<b>129 L</b>	[130–180]	g/L
Hematocrit	<b>0.379 L</b>	0.390–0.540]	L/L
Red Blood Cell Count	4.52	[4.50–6.50]	10E12/L
Mean corpuscular volume	84	[78–98]	fL
Volume distribution of erythrocytes	339	[320–360]	g/L
Platelets	385	[150–400]	10E9/L
Lymphocytes	3.00	[1.0–4.0]	10E9/L
Monocytes	0.90	[0.1–1.2]	10E9/L
Eosinophils	0.10	[0–0.4]	10E9/L
Basophils	0.05	[0–0.2]	10E9/L
International Normalized Ratio Blood Test	1.15	[0.84–1.16]	
Partial Thromboplastin Time	30.3	[26.0–38.0]	seconds

**H** – Value higher than reference range

**L** – Value lower than reference range

or rehabilitation at one-year post injury. This patient successfully returned to recreational sport participation with a left ankle brace for stability at one-year post injury.

## Discussion

Ankles are one of the most traumatized sites in the body with respect to sports injuries and account for 10–30% of all sports injuries.<sup>9</sup> Seventy-six (76%) of all injuries that occur in soccer are related to the ankle joint with sprains accounting for over 80% with 77% involving the lateral



Figure 3 Photograph of subject's left ankle 3 days post injury. The arrow indicates a large blister on lateral aspect of ankle.



Figure 4 Photograph of subject's left ankle 5 days post injury. The arrow indicates severe bruising on the lateral aspect of left leg.

aspect of the ankle joint and 73% involving rupture to the anterior talofibular ligament specifically.<sup>9,10,11</sup>

We present a case of a patient who was given a presumptive diagnosis of severe grade three-inversion ankle sprain after a thorough history of the mechanism of in-



Figure 5 Photograph of subject's left ankle 7 days post injury. The arrow indicates severe swelling surrounding ankle and dorsum of left foot.

jury and complete physical examination of the ankle and foot. Plain film radiographs confirmed the diagnosis. The patient may have sustained a subtalar dislocation with spontaneous relocation; however, there were no findings present clinically or radiographically of such an event.<sup>12,13</sup> Deteriorating neurological signs over a nine-hour period lead the surgeon to suspect ACS of the foot.

In this case ACS was a complication of a severe grade three-inversion ankle sprain. ACS typically occurs after a traumatic event such as a fracture, soft tissue injury, or vascular trauma.<sup>6,8,14,15,16,17</sup> It can be a devastating medical emergency that occurs from an increase in ICP within a closed osteofascial space. The circulation and viability of the tissues within the compartment are compromised due to an initial traumatic or haemorrhagic injury.<sup>8</sup> Hypoperfusion occurs to the tissues in response to the increased ICP leading to tissue ischemia and necrosis of muscles and nerves in the compartment.<sup>7,8,14,18</sup> Specifically, ACS occurs when the perfusion pressure of one of the osteofascial compartments falls below the tissue pressure.<sup>7,8,14,18</sup> Increased volume within a tissue compartment is usually caused by bleeding due to injury but can be caused by a coagulopathy, a disorder of blood coagulation.<sup>7,8,19</sup> This patient presented with severe pain and allodynia in the

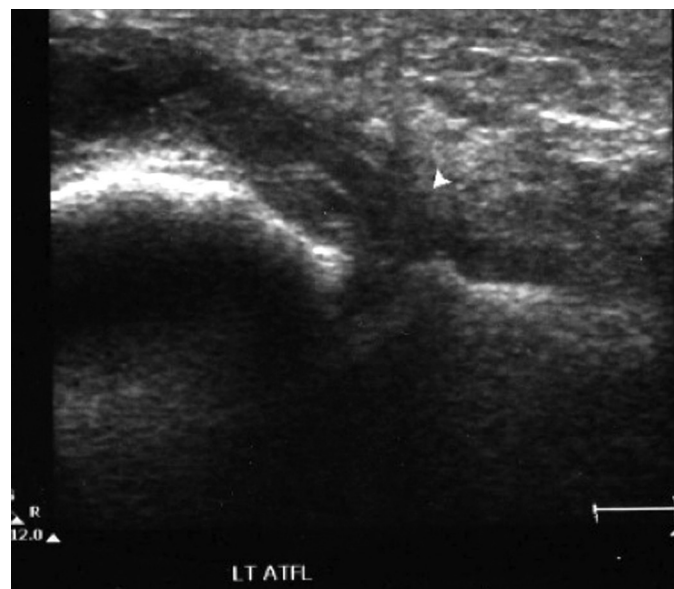


Figure 6 Diagnostic ultrasound of lateral aspect of left ankle. The arrowhead indicates the left ATFL is not intact.

foot due to hypoperfusion of the tissue causing tissue ischemia. Further, the paresthesia that followed was due to an increase in volume within the foot compartment leading to an increased ICP causing necrosis of nerves and soft tissues within the compartment.

The ischemia caused by ACS causes perifascicular and intrafascicular edema.<sup>8</sup> The end result of ACS without surgical intervention includes severe neurological deficits, ischemic contracture, gangrene, and tissue necrosis leading to amputation.<sup>6,7</sup> Therefore, early diagnosis is extremely important and requires a high index of suspicion during evaluation of the patient.<sup>7,14,15</sup> The initial symptomatology of this case may have contributed to confusion resulting in an accurate but non-comprehensive diagnosis of acute grade three-inversion ankle sprain. The initial symptomatology and a low index of suspicion also may have resulted in the delayed diagnosis of ACS of the foot. This case emphasizes the importance of a high index of suspicion when assessing an ankle sprain with pain out of proportion from the initial injury with concomitant paresthesia in the foot.

ACS occurring in the four compartments of the lower leg has been well reported within the current literature.<sup>8,17,18,20</sup> There are currently nine specific compart-



Figure 7 Diagnostic ultrasound of lateral aspect of left ankle. The 2 x's show a large hematoma over the left lateral malleolus and a complete avulsion of AFTL.

ments within the foot that have been described: medial, lateral, superficial, adductor, four interosseal, and calcaneal<sup>7,16,17,21,22</sup> (see Table 3 and Figure 9).

The signs and symptoms of ACS of the foot are subtle and hard to distinguish from the primary foot injury. Particular attention to the mechanism of injury, the amount of energy involved with the injury, the presence of trauma, and coagulopathies are important.<sup>7,16</sup>

The clinical diagnosis of ACS is made based on a constellation of physical signs and symptoms; increased pain out of proportion of original injury, pallor, paresthesia, paralysis, and pressure<sup>7,15,16</sup> (see Table 4). ACS may present as overt pain that is particularly accentuated by active and passive range of motion of the ankle joint, forefoot, or toes, sensory deficits in the specific compartment and a significant amount of swelling.<sup>14,18</sup> Pulselessness is uncommon and only seen in the late stages of ACS.<sup>7</sup> Pulselessness is usually caused by a vascular injury because the compartment pressure does not reach systolic blood pressure allowing blood flow.<sup>8,17</sup> If pulses are present palpation of the dorsal artery of the foot or the posterior tibial artery may be falsely reassuring as pulses can persist with a compartment pressure of >80 mmHg.<sup>15</sup> The examination of pulses is unreliable in the diagnosis

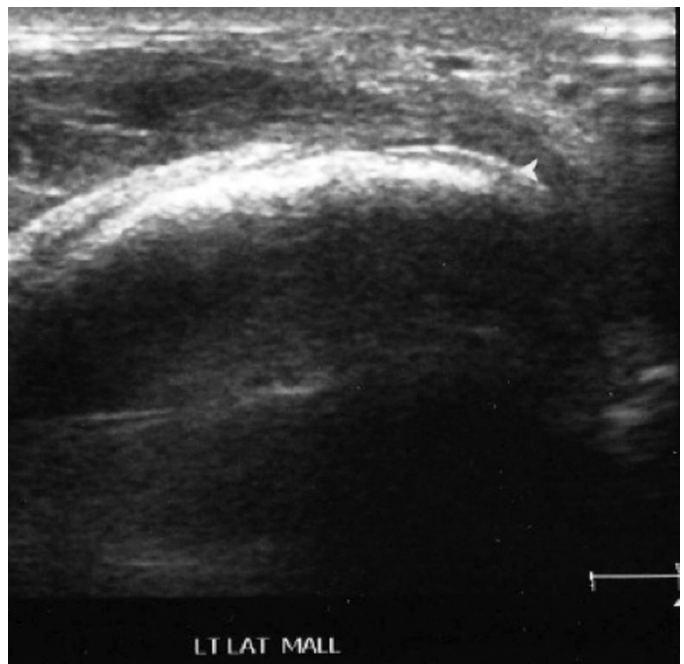


Figure 8 Diagnostic ultrasound of lateral aspect of left ankle. The arrowhead shows fragmentation of the left lateral malleolus.

of ACS since ICP does not reach systolic pressure.<sup>17</sup> Necrosis of the soft tissues of the foot may appear with compartment pressures ranging from 30–60 mmHg.<sup>15</sup>

Severe and spontaneous pain has been identified as the earliest and most sensitive clinical sign that manifests with ACS of the foot.<sup>23,24,25</sup> No evidence based recommendations can be made as to how to manage ACS, however, it has been suggested that serial examination of motor and sensation of the foot should be done at least every hour, since muscle necrosis can occur within 4 hours.<sup>26</sup>

Most often the clinical diagnosis of ACS is confirmed by measurement of the ICP. In many cases, the direct measurement of ICP is not warranted if the classic signs and symptoms of ACS are clinically evident and the patient is conscious.<sup>4,6</sup> ICP monitoring may not show any differences in outcome or time to surgery when compared to serial monitoring.<sup>27</sup> In our patient the ICP was not measured due to convincing signs and symptoms of ACS at approximately nine hours post-injury.

The use of platelet inhibitors (ASA) and calcium channel blockers (verapamil) are extremely common today.<sup>28</sup>

Table 3 *Compartments of the foot*<sup>7,16,17,22,23</sup>

Compartments	Muscles	Vessels	Nerves
Medial	1. Flexor hallucis brevis 2. Abductor hallucis	n/a	n/a
Lateral	1. Abductor digiti quinti 2. Flexor digiti minimi	n/a	n/a
Superficial	1. Flexor digitorum brevis 2. Lumbricals (4) 3. Flexor digitorum longus tendons	n/a	1. +/- Medial plantar nerve
Interosseus (×4)	1. Interossei	n/a	n/a
Adductor	1. Adductor	n/a	n/a
Calcaneal	1. Quadratus plantae	1. Posterior tibial artery 2. Posterior tibial vein 3. Lateral plantar artery 4. Lateral plantar vein	1. Posterior tibial nerve 2. Lateral plantar nerve 3. +/- Medial plantar nerve

Figure 9 *The compartments of the foot*<sup>33</sup> (Reprinted with permission from Mubarak SJ and Hargens AR: *Compartment Syndromes and Volkman's Contracture*, p. 45. Philadelphia, W. B. Saunders, 1981 and Bonutti PM and Bell GR *Compartment syndrome of the foot. A case report. JBJS.* 1986; 68:1449–1451)

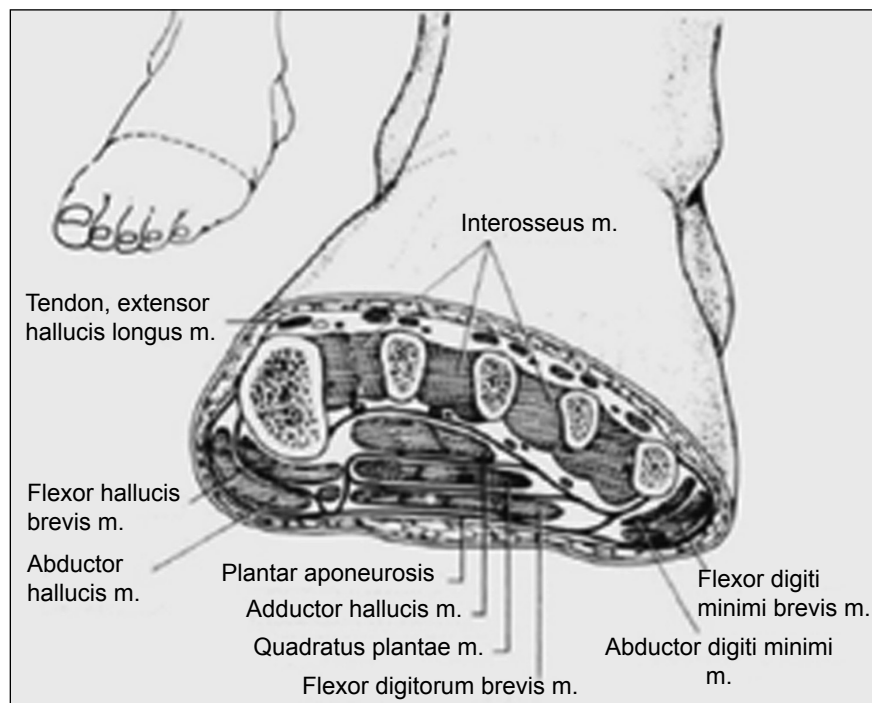




Table 4 *Signs and Symptoms of ACS*<sup>7,8,15,16</sup>

ACS of the foot
Severe pain that is out of proportion to the apparent injury
Pain provoked by passive range of motion of the foot
Severe swelling in the specific compartment
Progression of pain and swelling over a short period of time
Paraesthesia
Pallor of the surrounding cutaneous skin
Pulselessness (only occurs in the later stages)
Paralysis of the affected foot (only occurs in the later stages)

However, the frequency of these medications causing severe peripheral edema causing ACS seems particularly rare. ASA is used as a platelet inhibitor and verapamil is traditionally used as a vasodilator in the treatment of hypertension, angina pectoris, and cardiac arrhythmias and is an L-type calcium channel blocker of the Phenylalkylamine class. Verapamil may cause a decrease in arteriolar resistance with no change in venous circulation. This difference causes a change in the peripheral resistance causing an increase in hydrostatic pressure in the precapillary circulation.<sup>29</sup> When this occurs the vessels permit a fluid shift into the interstitial compartment.<sup>29</sup> Further, ASA causes anti-platelet aggregation leading to a decreased clotting time. These two distinct components lead to an increase in the peripheral perfusion to the injured area, which may cause the likelihood of ACS to increase exponentially. We hypothesize that the grade three-inversion ankle sprain combined with these medications may have increased the amount of swelling into the foot resulting in severe pain and paresthesia and ultimately leading to an increased ICP.

Upon review of the literature there are only three other case reports that are similar.<sup>30–32</sup> Beall et al. described a case of an 18 year-old male football player with ACS of the anterolateral aspect of the leg associated with the intake of platelet inhibitors used for an inherited platelet defect.<sup>30</sup> Compartment pressure measurements of the anterolateral compartment of the leg was taken with a slit

catheter and revealed a pressure of 40 mmHg. Decompression of the anterolateral compartment of the leg was performed by a fasciotomy. No neurological sequelae followed due to the immediate decompression.<sup>30</sup>

Secondly, Rancan et al. described a case of a 57-year old male with ACS of the anterior aspect of the leg caused by intramuscular hemorrhage of the anterior tibialis muscle following a low energy, non-contact injury.<sup>31</sup> The patient was on a daily regimen of platelet inhibitors (75 mg ASA daily). The clinical diagnosis of ACS was confirmed in this case with a handheld ICP monitoring system. The ICP was measured as 95 mmHg and immediate surgical decompression of the anterior and lateral compartments were performed. A six-week follow-up was performed and no neurological deficits were found and there was full function of the knee and ankle joints.<sup>31</sup>

A third case report by Dhawan et al. described a case of 35-year old male who sustained a severe inversion ankle sprain while playing basketball.<sup>32</sup> The patient was initially prescribed a treatment at home of rest, ice, elevation, and compression. Examination of the foot 26 hours after injury revealed a swollen foot and ankle with severe blistering. Posterior tibial and dorsalis pedis pulses were not palpable; capillary refill was brisk in all toes. No fracture was diagnosed on plain film radiographs. The patient's dorsal compartment pressures were measured at 120 mmHg, confirming a diagnosis of ACS of the foot and a disruption of the anterior tibial artery. A seventy-seven month follow-up was performed and no neurological deficits were found.<sup>32</sup>

For patients who sustain a high-energy deceleration injury resulting in pain, severe swelling, and paresthesia ACS should be suspected. ICP measurement may be considered if the patient is unconscious and the signs and symptoms of ACS are not evident.<sup>6,7</sup> Patients with a measured ICP of 30 mmHg are recommended to undergo an immediate fasciotomy to relieve the pressure in the tissue compartment.<sup>6</sup> Normal ICP in the foot is approximately 4.7–6 mmHg and in the lower leg 0–8 mmHg.<sup>6,8</sup> In patients presenting with ACS, pain and paresthesia first appears at an ICP between 15–30 mmHg. Therefore, many surgeons will perform fasciotomies of the affected compartment at 30 mmHg.<sup>6,8</sup> Irreversible changes occur to soft tissues and neurological structures with an ICP of 30 mmHg for durations of 4 hours or longer.<sup>6,8,27</sup> The highest compartment pressures are often found 12–36

hours post injury.<sup>27</sup> In this patient a fasciotomy was considered but not performed resulting in sensory deficit at a one-year follow-up. A fasciotomy was not performed due to a recent outbreak of methicillin-resistant staphylococcus aureus in the hospital that may have resulted in a serious life threatening infection. Decompression surgery in this case would have occurred if the patient's neurological signs and symptoms deteriorated further from initial presentation.

## Summary

ACS is often not suspected in common ankle injuries. We report a case of a 28-year old recreational soccer player who was eventually diagnosed with ACS of the foot. Although ACS is usually diagnosed with an ICP measurement, this case represents a diagnosis of ACS of the foot without ICP measurement. This case highlights the necessity for all primary health care practitioners responsible for diagnosing and treating ankle sprains to be aware of the potential complication of ACS of the lower limb or foot. A delayed diagnosis and inappropriate management may lead to devastating neurological sequelae and even amputation of the affected limb, especially in patients presenting with early signs and symptoms and those on antiplatelet therapy or calcium channel blockers<sup>6</sup>. ACS may have been exacerbated by the use of ASA and Verapamil pharmacotherapy in this case.

## References

- Garrick JG. The frequency of injury, mechanism of injury, and epidemiology of ankle sprains. *Am J Sports Med.* 1977; 5:241–242.
- Pihlajamäki H, Hietaniemi K, Paavola M, Visuri T, Mattila VM. Surgical versus functional treatment for acute ruptures of the lateral ligament complex of the ankle in young men: A randomized controlled trial. *J Bone Joint Surg Am.* 2010; 92:2367–74.
- Schmikli SL, de Vries WR, Inklaar H, Backx FJG. Injury prevention target groups in soccer: Injury characteristics and incidence rates in male junior and senior players. *J Sci Med Sport.* 2010; doi:10.1016/j.jsams.2010.10.688
- Waterman BR, Owens BD, Davey S, Zacchilli MA, Belmont PJ. The epidemiology of ankle sprains in the United States. *J Bone Joint Surg Am.* 2010; 92:2279–84.
- Lynch SA, Renstrom PA. Treatment of acute lateral ankle ligament rupture in the athlete. Conservative versus surgical treatment. *Sports Med.* 1999; 27:61–71.
- Kostler W, Strohm PC, Sudkamp NP. Acute compartment syndrome of the limb. *Injury, Int. J Care Injured.* 2004; 35:1221–1227.
- Malik AA, Khan WSA, Chaudhry A, Ihsan M, Cullen NP. Acute compartment syndrome – a life and limb threatening surgical emergency. *J Preop Practice.* 2009; 19(5):137–142.
- Tiwari, A, Haq AI, Myint F, Hamilton G. Review: Acute compartment syndrome. *Br J Surg.* 2002; 89:397–412.
- Tik-Pui Fong D, Hong Y, Chan L, Shu-Hang Yung P, Chan K. A systematic review on ankle injury and ankle sprain in sports. *Sports Med.* 2007; 37(1):73–94.
- Garrick JG, Requa RK. The epidemiology of foot and ankle injuries in sports. *Clin Sports Med.* 1988; 7(1): 29–36.
- Gerber JP, Williams GN, Scoville CR, Arciero RA, Taylor DC. Persistent disability associated with ankle sprains: a prospective examination of an athletic population. *Foot Ankle Int.* 1998; 19(10):653–60.
- DeLee JC, Curtis R. Subtalar dislocation of the foot. *J Bone Joint Surg Am.* 1982; 64:433–437.
- Zimmer TJ, Johnson KA. Subtalar dislocations. *Clin Orthop.* 1989; 238:190–4.
- Kosir R, Moore FA, Selby JH, Cocanour CS, Kozar RA, Gonzalez EA, Todd SR. Acute lower extremity compartment syndrome (ALECS) screening protocol in critically ill trauma patients. *J Trauma.* 2007; 63:268–275.
- Elliott KGB, Johnstone AJ. Diagnosing acute compartment syndrome. *J Bone Joint Surg [Br].* 2003; 85:625–632.
- Fulkerson E, Razi A, Tejwani N. Review: Acute compartment syndrome of the foot. *Foot & Ankle International.* 2003; 24(2):180–187.
- Frink M, Hildebrand F, Krettek C, Brand J, Hankemeier S. Compartment syndrome of the lower leg and foot. *Clin Orthop Relat Res.* 2010; 468:940–950.
- Shadgan B, Menon M, O'Brien P, Reid D. Diagnostic techniques in acute compartment syndrome of the leg. *J Orthop Trauma.* 2008; 22:581–587.
- Roberge RJ, McLane M. Compartment syndrome after simple venouuncture in an anticoagulated patient. *J Emerg Med.* 1999; 17:647–649.
- Kashuk JL, Moore EE, Pinski S, Johnson JL, Moore JB, Morgan S, Cothren CC, Smith, W. Lower extremity compartment syndrome in the acute care surgery paradigm: safety lessons learned. *Patient Safety in Surgery.* 2009; 3(11):1–6.
- Perry MD, Manoli A. Foot compartment syndrome. *Ortho Clin North America.* 2001; 32(1):103–111.
- Richter J, Schulze W, Klaas A, Clasbrummel B, Muhr G. Compartment syndrome of the foot: an experimental approach to pressure measurement and release. *Arch Orthop Trauma Surg.* 2008; 128:199–204.
- Myerson M. Diagnosis and treatment of compartment syndrome of the foot. *Orthopedics.* 1990;13:711–717.

- 24 Myerson MS. Management of compartment syndromes of the foot. *Clin Orthop Relat Res.* 1991; 239–248.
- 25 Ulmer T. The clinical diagnosis of compartment syndrome of the lower leg: are clinical findings predictive of the disorder? *J Orthop Trauma.* 2002; 16:572–577.
- 26 Blaisdell FW. The Pathophysiology of skeletal muscle ischemia and the reperfusion syndrome: a review. *Cardiovascular Surgery.* 2002; 10(6):620–630.
- 27 Al Dadah OQ, Darrah C, Cooper A, Donell ST, Patel AD. Continuous compartment pressure monitoring vs. clinical monitoring in tibial diaphyseal fractures. *Injury.* 2008; 39:1204–1209.
- 28 Kirshner HS, Biller J, Callaban AS. Long-Term Therapy to Prevent Stroke. *J Am Board Fam Pract.* 2005; 18:528–40.
- 29 Sica DA. Calcium channel blocker-related peripheral edema: Can it be resolved? *J Clin Hypertens.* 2002; 5:291–297.
- 30 Beall S, Garner J, Oxley D. Anterolateral compartment syndrome related to drug-induced bleeding: A case report. *Am J Sports Med.* 2003; 11:454–455.
- 31 Rancan M, Esser MP, Kossmann T, Cameron P, Fitzgerald M. Acute compartment syndrome following low energy non-contact injury. *J Surg.* 2004; 11:1023–1025.
- 32 Dhawan A, Doukas WC. Acute compartment syndrome of the foot following an inversion injury of the ankle with disruption of the anterior tibial artery. *J Bone Joint Surg Am.* 2003; 85:528–532.
- 33 Bonutti PM, Bell GR. Compartment syndrome of the foot. A care report. *J Bone Joint Surg Am.* 1986; 68:1449–1451.

## Canadian Chiropractic Research Foundation



Creating a culture of research