DIFFERENTIAL MERITS OF MRI PULSE SEQUENCES IN EVALUATION OF ANKLE INJURIES

Thesis
Submitted for Partial Fulfillment of M.D. Degree in Radiodiagnosis

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Mohamed Salah Haggag
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<td>Turbo Spin Echo</td>
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INTRODUCTION AND

AIM OF THE WORK
INTRODUCTION

Foot and ankle structures bear massive amounts of force during athletic activities and are naturally susceptible to a vast and ever-expanding array of injuries (Zoga and Schweitzer 2003).

The ankle is one of the most frequently injured joints. In order to a better understanding of these lesions, a classification based on the anatomic origin are outlined. The spectrum of injuries has been classified in: (1) osseous lesions, (2) ligamentous injuries, (3) tendinous lesions, (4) miscellaneous injuries (Narváez et al. 2003).

It is easiest to organize the approach to analyzing pathology at the ankle by considering compartmental anatomy. The compartments can simply be divided into the anterior, posterior, lateral, and medial soft tissue compartments. The signal characteristics of the marrow and contour detail of the joints are also described. Last, the sinus tarsi, plantar fascia, and subcutaneous soft tissues should be surveyed (Leffler and Disler 2002).

MR imaging has become the modality of choice in the evaluation of most of these lesions (Narváez et al. 2003).

Magnetic resonance imaging is playing an increasingly important role in evaluation of the injured foot and ankle. Magnetic resonance imaging allows accurate detection of bony abnormalities, such as stress fractures, and soft-tissue abnormalities, including ligament tears, tendon tears, and tendinopathy. The interpreter of magnetic resonance images should
systematically review the images, noting normal structures and accounting for changes in soft-tissue and bony signal \textit{(Riley 2007)}.

Magnetic resonance (MR) imaging with its multiplanar capability and superb soft tissue contrast is quickly becoming the method of choice for evaluating chronic foot and ankle pain and further defining the extent of tendon and ligament injuries \textit{(Dunfee et al. 2002)}.

MR imaging continues to become more widely available with a growing number of systems and shorter scan times, while technologic improvements allow for better anatomic detail and an increased sensitivity for pathology. Often the exact location and nature of an injury is governed by the principle of failure at the weakest point along a musculo-tendo-osseous axis. This point of failure then varies with patient age and physical condition. Adolescents and young adults are most susceptible to bony growth plate or apophyseal injury, whereas tendinous and musculotendinous injuries are more prevalent in the middle aged \textit{(Zoga and Schweitzer 2003)}.

When imaging the foot and ankle after an injury, we employ pathology-sensitive and anatomy-specific MR sequences in multiple imaging planes. In most cases, a pathology-sensitive sequence in the form of a T2-weighted sequence with fat suppression or short tau inversion recovery (STIR) is obtained in different planes and anatomicT1-weighted sequences are performed. It is important for one bone marrow-specific sequence, usually T1 weighted, to be obtained without fat suppression \textit{(Zoga and Schweitzer 2003)}.
Routine ankle MR imaging is performed in the axial, coronal, and sagittal planes parallel to the table top. Marrow abnormalities are best evaluated with fat suppression with short tau inversion recovery (STIR) sequences. Cartilage abnormalities can be visualized with two-dimensional or three-dimensional (3D) gradient-echo sequences. Any pathology would cause local inflammatory response, which in turn cause edema and water retention in the tissue. Thus the area of inflammation or infection would appear dark on T1-weighted images (WI) and very bright on short tau inversion recovery (STIR) images (Rosenberg et al.2000).
AIM OF THE WORK

The purpose of this study is to highlight the most accurate and beneficial pulse sequences for the evaluation of bone and soft tissue injuries of the ankle region.
REVIEW OF THE LITERATURE
Review of the literature

Gross anatomy of the ankle joint.

- Osseous structures.
- Retinaculae.
- Ligaments.
- Tendons.
- Sectional MRI imaging anatomy (axial images).
- Sectional MRI imaging anatomy (sagittal images).
- Sectional MRI imaging anatomy (coronal images).

Pathology of post traumatic ankle joint.

- Osseous injuries.
- Non osseous injuries.
  - Tendon Injury.
  - Ligament Injury.
  - Miscellaneous Injuries

MRI findings of post traumatic ankle joint.

- Osseous injuries.
- Non osseous lesions.
  - MRI appearance of tendon injuries.
  - MR appearance of ligamentous injuries.
  - Miscellaneous injuries.
GROSS ANATOMY
OF THE ANKLE
JOINT
Gross Anatomy Of The Ankle Joint

The Talocrural Joint (Ankle Joint)

The ankle joint is of uni-axial type; the lower end of the tibia and its malleolus, the malleolus of the fibula and the inferior transverse tibiofibular ligament together form a deep recess in which the body of the talus is embraced (El Refaiy et al. 2008).

The line of the joint can be gauged from the anterior margin of the lower end of the tibia which can be felt through the skin when the overlying tendons are relaxed. Although anatomically this joint appears to be a simple hinge joint and is usually styled “uni-axial”, it must be emphasized that the axis of rotation is dynamic, taking up a series of different positions during dorsi-flexion - planter flexion changes (El Refaiy et al. 2008).

The articular surfaces are covered with hyaline cartilage. The trochlear surface of the talus, which is convex from before backwards and gently concave from side to side, is wider infront than behind, and the inferior articular surface of the tibia is reciprocally shaped. The articular surface for the medial malleolus is restricted to the upper part of the medial surface of the talus. It is fairly flat and comma-shaped, being deeper infront than behind. The articular surface on the lateral side of the talus is triangular in
outline and concave from above downwards; that on the lateral malleolus is reciprocally curved. Posteriorly, the edge between the trochlear and fibular articular surfaces of the talus is beveled to form a flattened triangular area which articulates with the inferior transverse tibio-fibular ligament (El Refaiy et al. 2008).

The bones are connected by a fibrous capsule, and by deltoid, anterior and posterior talofibular and calcaneo fibular ligaments (Dunfee et al. 2002).

The ankle joint is formed of: - (Fig 1-A & B)

- Bony structures.
- Ligaments and tendons.
- Muscles (El Refaiy et al. 2008).

**Bony Structures:-**

- The Tibia:
  
  - The lower end of the tibia has the medial malleolus and the fibular notch for the inferior tibiofibular joint laterally.
  
  - Its inferior surface is flattened and articulates with the talus in the ankle  (El Refaiy et al. 2008).
Bones of the Ankle Joint

(AP view)

Figure 1-A

1. Fibula
2. Tibia
3. Distal tibiofibular joint
4. Malleolar fossa
5. Lateral malleolus
6. Ankle joint
7. Medial malleolus
8. Talus

(El Refaiy et al. 2008).
Bones of the Ankle Joint

(Lateral view)

Figure 1-B
1. Fibula
2. Tibia
3. Ankle joint
4. Promontory of tibia
5. Trochlear surface of talus
6. Talus
7. Posterior tubercle of talus
8. Calcaneus
9. Sustentaculum tali
10. Tarsal tunnel
11. Navicular
12. Cuneiforms
13. Cuboid

(El Refaiy et al. 2008).
- **The Fibula:**
  - The fibula is mainly a site of origin of muscles and has no weight bearing function.
  - Proximal and distal tibiofibular joints unite it with the tibia and it articulates with the talus in the ankle joint.
  - The lateral malleolus is more distal than the medial malleolus.
  - The calcaneofibular ligament is attached to its tip.
  - The fibula is proportionally thicker in children than in adults (*El Refaiy et al. 2008*).

- **The Tarsus:**
  - The tarsus consists of seven bones: two large (calcaneus and talus) and five smaller (navicular, cuboid and three cuneiform bones) (*El Refaiy et al. 2008*).

- **The Calcaneus:**
  - Talar articulations: Anterior, middle & posterior facets.
  - Weight-bearing, springboard for locomotion.
  - Anterior process articulates with cuboid.
  - Sustentaculum tali: Medial protuberance, middle facet.
  - Tuberosity: Achilles tendon insertion, posterior subtalar facet.
  - Plantar surface: Anterior, medial & lateral tubercles (Fig. 2). (*Manaster et al. 2006*).
Figure 2. Drawings illustrate the anatomy of the calcaneus, including the anterior process of the calcaneus (a), anterior facet of the talus (A), anterior facet of the cuboid bone (C), groove for the flexor hallucis longus tendon (Fhl), lateral process (Lp), middle facet of the talus (M), medial process (Me), posterior facet (P), peroneus longus groove (Pl), sustentaculum tali (S), sulcus calcanei (SC), posterior tuberosity (T), and trochlear process (Tp) (Badillo et al. 2011)

- The Talus: -

It affords plantar flexion & dorsiflexion of ankle and it is the keystone of medial longitudinal arch. Its proximal body
(trochlea) articulates with tibia. The talar articular surface with tibia (trochlea) is broader anteriorly than posteriorly. The body articulates with medial & lateral malleoli. Posterior process is a groove between medial & lateral tubercles for flexor hallucis longus tendon. Talus has 3 inferior facets articulate with calcaneus. Head articulates with navicular bone, spring ligament & sustentaculum tali. No muscle attachments, 2/3 covered by cartilage, dominant blood supply enters neck (Manaster et al. 2006).

- **Sinus tarsi**
  - Lateral, funnel shaped space between talar neck & calcaneus.
  - Base is tarsal canal, between posterior subtalar joint & sustentaculum tali.
  - Traversed by: Medial, lateral & intermediate roots of inferior extensor retinaculum, cervical & talocalcaneal interosseous ligaments, fat, neurovascular anastomosis.
  - Talocalcaneal interosseous ligament: Most medial, extends from talar sulcus to calcaneus between posterior & middle calcaneal facets, taut in eversion.
  - Cervical ligament: Anterior & lateral, extends from talar neck to calcaneus, taut in inversion (Manaster et al. 2006).
• **Retinacula**
  - Focal thickening of deep fascia.
  - Prevents bowstringing, binds tendons down (*Manaster et al. 2007*).

• **Superior extensor retinaculum**
  - A few cm above ankle joint.
  - Attaches to anterior fibula laterally, tibia medially.
  - Proximally continues with fascia cruris.
  - Distally attaches to inferior extensor retinaculum.
  - Binds down anterior compartment muscles (*Manaster et al. 2007*).

• **Inferior extensor retinaculum**
  - At ankle joint, Y shaped, stem laterally, proximal & distal bands medially.
  - Stem attaches laterally to upper calcaneus:
    - *Loops around extensor tendons.*
    - *Roots extend into sinus tarsi.*
  - Proximal medial band has deep & superficial layers, loop around extensor hallucis longus tendon & occasionally tibialis anterior.
  - Distal medial band superficial to extensor hallucis longus & tibialis anterior tendons and attaches to plantar aponeurosis.
  - Dorsalis pedis vessels, deep peroneal nerve: Deep to all layers of inferior extensor retinaculum.
• **Flexor retinaculum**
  - Attaches to medial malleolus.
  - Proximally continuous with deep fascia of leg.
  - Distally continuous with plantar aponeurosis.
  - Abductor hallucis partly attached to it.
  - Binds deep flexor tendons to tibial & calcaneal grooves.
  - Lateral border of tarsal tunnel (*Manaster et al. 2007*).

• **Superior peroneal retinaculum**
  - Origin: Lateral malleolus, insertions vary, most commonly to deep fascia of leg & calcaneus.
  - Binds peroneal tendons into retro fibular groove (*Manaster et al. 2007*).

• **Inferior peroneal retinaculum**
  - Continuous with inferior extensor retinaculum.
  - Inserts on lateral calcaneus, peroneal tubercle (trochlea).
  - Binds peroneus brevis, peroneus longus tendons to calcaneus.
**The Fibrous Capsule:**

Surrounds the joint; it is thin infront and behind and attached above to the borders of the articular surfaces of the tibia and malleoli and below, to the talus close to the margins of the trochlear surface except infront where it is attached to the dorsum of the neck of the talus at some distance infront of its superior articular surface. It is supported from each side by strong collateral ligaments. The posterior part of the capsule consists principally of transverse fibres. It blends with the inferior transverse ligament and is somewhat thickened laterally where it reaches as far as malleolar fossa of the fibula. A synovial membrane lines the fibrous capsule, and the joint cavity ascends for a short distance between the tibia and fibula (William *et al.* 1989).

**Ligaments**

*Syndesmotic tibiofibular complex*

- Bind the fibrous distal tibiofibular joint.
- Composed of 1) anterior tibiofibular, 2) posterior tibiofibular, 3) inferior transverse, 4) interosseous tibiofibular.
- Optimally visualized on axial, coronal MR images; intermediate to low in signal.
- Anterior, posterior tibiofibular: may be heterogeneous due to fat between fascicles; oblique course, extend to level of talar dome. 
- Inferior transverse: thick heterogeneous signal; band like, extends distal to tibial posterior surface; tibial insertion almost at medial malleolus. 
- Posterior tibiofibular ligament may stimulate an intra articular body on sagittal images (Manaster et al. 2007).

- **Lateral collateral ligaments:**
  
  Bind talus & calcaneus to fibula. Composed of 1) anterior talofibular ligament, 2) posterior talofibular ligament 3) calcaneofibular ligament. Anterior talofibular & posterior talofibular ligaments optimally visualized on axial images. Posterior talofibular and calcaneofibular well seen on coronal images. Ligaments reflect thickening of the capsule; therefore delineated by joint fluid on fluid sensitive images. Highlighted by fat, obliteration of fat is indicative of disease (Manaster et al. 2007).
  
  - Insert on fibula at malleolar fossa. 
  - Talus is oblong shaped at level of lateral collateral ligaments. 
  - Posterior talofibular ligament is fan shaped & striated at its insertion to fibula. 
  - Anterior talofibular ligament should be straight, with smooth undersurface.
- Calcaneofibular ligament is usually seen on axial images performed in mild plantar flexion (Manaster et al. 2007).

- **Deltoid ligament:**
  - Subdivided into superficial & deep bands, many variations.
  - Superficial subdivided into anterior tibiotalar, posterior tibiotalar, tibionavicular, tibiospring and tibiocalcaneal.
  - Deep subdivided into anterior tibiotalar and posterior tibiotalar.
  - Deep tibiotalar often striated.
  - Superficial components originate from medial malleolus as continuous band, differentiation based on insertion sites.
  - Tibiospring band continuous with superomedial component of spring ligament (Manaster et al. 2007).

**Muscles Producing Movements:**

- **Dorsiflexion:** Tibialis anterior assisted by extensor digitorum longus, extensor hallucis longus and peroneus tertius.
- **Planter flexion:** Gastrocnemius and soleus assisted to a lesser degree by plantaris, tibialis posterior, flexor hallucis longus and flexor digitorum longus (El Refaiy et al. 2008).
Figure 3-A: Ligaments and Tendons of the Ankle (Medial Aspect) (Netter 1997).

Figure 3-B: Ligaments and Tendons of the Ankle (Lateral Aspect) (Netter 1997).
**Tendons**

- **Anterior (extensor) compartment:**
  - **Tibialis anterior tendon:**
    - Most medial & largest tendon in anterior compartment.
    - Inserts on medial cuneiform, base of 1st metatarsal.
    - Dorsiflexes ankle, inverts foot, tightens plantar aponeurosis.
- Supports medial longitudinal arch during walking \cite{Manaster et al. 2007}.

\textbf{Extensor hallucis longus tendon:}
- Inserts on dorsal base of 1st distal phalanx.
- Extends 1st phalanges, dorsiflexes foot \cite{Manaster et al. 2007}.

\textbf{Extensor digitorum longus tendon:}
- Divides into four slips on dorsum of foot.
- Slips receive tendinous contributions from extensor digitorum brevis, lumbrical & interosseous muscles.
- Each slip divides into 3: Central one inserts on dorsal base of middle phalanx & 2 collateral ones which reunite & insert on bases of 2nd-5th distal phalanges.
- Dorsiflexes ankle, extends toes, tightens plantar aponeurosis \cite{Manaster et al. 2007}.

\textbf{Peroneus tertius tendon:}
- Typically part of extensor digitorum longus tendon.
- Inserts on dorsal base of 5th metatarsal \cite{Manaster et al. 2007}.

- \textit{Lateral compartment}

\textbf{Peroneus longus tendon:}
- Posterolateral to peroneus brevis tendon in retrofibular groove, deep to superior peroneal retinaculum.
-Proximally has common tendon sheath with peroneus brevis.
-Second tendon sheath at sole of foot.
-Descends behind peroneal tubercle, deep to inferior peroneal retinaculum.
-Curves under cuboid deep to long plantar ligament.
-Inserts on plantar base of 1st metatarsal, medial cuneiform.
-Plantarflexes ankle, everts foot, supports longitudinal & transverse arches during walking.
-Os peroneum always present, ossified in about 20% of individuals (*Manaster et al. 2007*).

❖ Peroneus brevis tendon:

- Anteromedial to peroneus longus tendon in retrofibular groove, deep to superior peroneal retinaculum.
- Descends anterior to peroneal tubercle of calcaneus, deep to inferior peroneal retinaculum.
- Inserts into base of 5th metatarsal.
- Everts foot, limits foot inversion (*Manaster et al. 2007*).
**Superficial posterior compartment**

- **Achilles tendon:**
  - Largest & strongest tendon in body.
  - Conjoined tendon of medial & lateral gastrocnemius & soleus muscles.
  - Approximately 15 cm long.
  - Lacks tendon sheath, enclosed by paratenon.
  - Inserts on posterior calcaneal tuberosity.
  - Retrocalcaneal bursa between distal tendon & calcaneal tuberosity.
  - Main plantarflexor of ankle & foot (*Manaster et al. 2007*).

- **Plantaris tendon**
  - Vestigial, slender tendon, medial to Achilles tendon.
  - Inserts on or medial to Achilles tendon (*Manaster et al. 2007*).

- **Deep posterior (flexor) compartment**

- **Tibialis posterior tendon:**
  - Crosses flexor digitorum longus tendon above ankle joint to become most posteromedial tendon.
  - Shares tibial groove with flexor digitorum longus tendon.
  - Inserts on navicular tuberosity, cuneiforms, sustentaculum tali, bases of 2nd-4th metatarsals.
-Main invertor of foot, aids in plantar flexion.
-Supports medial longitudinal arch (Manaster et al. 2007).

Held in groove by flexor retinaculum. Except Achilles largest posterior tendon, approximately 2-3 times size of adjacent flexor digitorum on axial images. May have normal minimal fluid in tendon sheath. Often increased signal at navicular attachment due to magic angle effect, fibro cartilage and os naviculare. No tendon sheath distally; distal peritendinous signal is abnormal. Distal insertion slips can be seen on axial images (Manaster et al 2007).

- **Flexor digitorum longus tendon:**
  - Lateral to tibialis posterior tendon in tibial groove.
  - Crosses flexor hallucis longus tendon at master knot of Henry.
  - Divides into 4 slips which give origin to lumbricals.
  - Slips pass through openings in corresponding tendons of flexor digitorum brevis.
  - Slips insert on bases of 2nd-5th distal phalanges.
  - Flexes distal phalanges, assists in plantar flexion of ankle.
  - When foot on ground: Maintains pads of toes on ground.
  - When foot off ground: Plantar flexes 2nd-5th phalanges, aids in maintaining longitudinal arches (Manaster et al. 2007).
Flexor hallucis longus tendon:

- Passes 3 fibro-osseous tunnels: 1) between medial & lateral talar tubercles, 2) under sustentaculum tali, 3) between 1st medial & lateral sesamoids.
- Crosses & sends slip to flexor digitorum longus at master knot of Henry.
- Inserts on base of 1st distal phalanx.
- When foot on ground: Maintains pad of 1st toe on ground.
- When foot off ground: Plantar flexes 1st phalanges, aids in maintaining medial longitudinal arch.
- Weak plantar flexor of ankle.
- Innervated by tibial nerve (Manaster et al. 2007).

- The Synovial Sheaths in the Talocrural Region:

  The tendons crossing the talocrural joint are all to some degree deflected from a straight course, and are hence held down by retinacula and enclosed in synovial sheaths. Anteriorly the sheath for tibialis anterior extends from the proximal margin of the superior extensor retinaculum to the interval between the diverging limbs of the inferior retinaculum; the sheath for extensor digitorum longus and peroneus tertius and another for extensor hallucis longus start just above the level of the malleoli; the former reaching the higher (Fig. 4) (William et al. 1989).
The sheath of extensor hallucis longus is prolonged to the base of the first metatarsal bone, while that of extensor digitorum longus reaches only to the level of the base of the fifth metatarsal bone. Medial to the ankle the sheath for tibialis posterior extends for about 4 cm above the malleolus; below, it ends just proximal to the attachment of the tendon to the tuberosity of the navicular. The sheath for the flexor hallucis longus reaches the level of the malleolus, while that for flexor digitorum longus goes slightly higher; the former is continued to the base of the first metatarsal bone, the latter ends at the navicular. Lateral to the ankle a sheath, which is proximally single but double below, encloses peroneus longus and brevis. It extends for about 4 cm proximal to the tip of the malleolus, and downwards and forwards for about the same distance (William et al.1989).
Figure 4-A: The mucous sheaths of the tendons around the ankle (Lateral aspect) *(Netter 1997).*

Figure 4-B: The mucous sheaths of the tendons around the ankle (Medial aspect) *(Netter 1997).*
- The Tarsal Tunnel:

The tarsal tunnel is defined as a space between the flexor retinaculum (roof) and the talus and calcaneus (floor). The tarsal tunnel syndrome is a clinical diagnosis based on parathesias, pain, and weakness attributed to the posterior tibial nerve (*El Refaiy et al. 2008*).

-Sinus Tarsi:

The sinus tarsi is composed of fat, branches of the posterior tibial and peroneal arteries and their associated nerves, and five ligaments. The sinus tarsi ligaments include the lateral, intermediate, and medial roots of the inferior extensor retinaculum, the interosseous talocalcaneal ligament, and the cervical ligament (*El Refaiy et al. 2008*).

-Plantar fascia

On sagittal and coronal MR images, the normal plantar fascia appears as a thin, hypointense structure extending anteriorly from the calcaneal tuberosity. The plantar fascia has a normal thickness of 3.22 mm ± 0.53 and flares slightly at the calcaneal insertion (*El Refaiy et al. 2008*).
**Sectional MRI Imaging Anatomy**

*Axial Images:*

In the axial plane, the low signal intensity bands of the anterior and posterior inferior tibiofibular ligaments are demonstrated at the level of the tibial plafond. The inferior extensor retinaculum is identified anterior to and at its attachment to the medial malleolus and represents the upper limb of this Y-shaped band of the deep fascia. On axial images through the tibiotalar joint, the tendons of the tibialis anterior, extensor hallucis longus, extensor digitorum longus and peroneus longus muscle occupy the anterior compartments in a medial-to-lateral direction (*Stoller and Ferkel 1997*).

The peroneus brevis muscle and tendon and the more lateral peroneus longus tendon are located posterior to the lateral malleolus. The tendons of the tibialis posterior, flexor digitorum longus and the flexor hallucis longus can be identified posteriorly, running from a medial position posterior to the medial malleolus to a lateral position posterior to the tibial plafond and talar dome. Posterior and medial to the greater saphenous vein, the anterior tibionavicular fibers of the deltoid ligament blend with the low signal cortex of the anterior surface of the medial malleolus (*Stoller and Ferkel 1997*).
The Achilles tendon is identified in cross section as a thick structure of low signal intensity with a convex posterior surface and a flattened anterior surface. The posterior Achilles tendon is formed by the convergence of the gastrocnemius, plantaris and soleus muscles. The soleus muscle group that is present at the level of the distal tibia is not seen at the tibiotalar joint level. Sections through the level of the distal lateral malleolus demonstrate the anterior and posterior talofibular ligaments. Medially, the tibionavicular and tibio-calcaneal parts of the deltoid ligament are also shown at this level. The peroneal retinaculum can be seen coursing medial and posterior to the lateral malleolus. The inter-osseous talocalcaneal ligament is posterolateral to either the anterior talus or the talar head. The plantar calcaneo-navicular ligament or spring ligament is located inferior to the lateral malleolus between the lateral talus and tibialis posterior tendon (*Stoller and Ferkel 1997*).

The calcaneofibular ligament is optimally seen with the foot in 40° of plantar flexion, and on neutral axial images it can be seen lateral to the posterior inferior talus, anterior and medial to the peroneus brevis tendon. The sural nerve, intermediate in signal intensity, is located posteromedial to the peroneus brevis muscle. The tibial nerve is medial to the flexor hallucis longus tendon and continues distally as the medial and lateral plantar nerves. The
flexor retinaculum is superficial to the tendons of the deep muscles on the medial side of the ankle. In the foot, the tendons of the flexor hallucis brevis and longus muscles are seen posterior to the first metatarsal and cuneiform. The longitudinally oriented quadratus plantaris and abductor hallucis muscles are medial to the calcaneus and cuboid. The peroneus longus tendon - a fourth-layer muscle of the sole of the foot - enters the foot by passing posterior to the lateral malleolus and can be seen obliquely crossing the foot to its insertion onto the base of the first metatarsal and medial cuneiform bone *(Stoller and Ferkel 1997)*.

The anterior neurovascular bundle, composed of the anterior tibial artery and vein and deep peroneal nerve, is located posterior to the extensor tendons, whereas the posterior neurovascular bundle, composed of the posterior tibial artery, vein, and tibial nerve, is located posterior to the flexor digitorum and flexor hallucis longus tendons *(Haygood 1997)*.
MRI Axial Plane of the Ankle
(From superior to inferior)

Figure 5: MRI Axial Plane of the Ankle (Stoller and Ferkel, 1997)

Figure 6: MRI Axial Plane of the Ankle (Stoller and Ferkel, 1997)
Figure 7: MRI Axial Plane of the Ankle (Stoller and Ferkel, 1997)

Figure 8: MRI Axial Plane of the Ankle (Stoller and Ferkel, 1997)
Figure 9: MRI Axial Plane of the Ankle *(Stoller and Ferkel 1997)*

Figure 10: MRI Axial Plane of the Ankle *(Stoller and Ferkel 1997)*
Sagittal Images:

- Medial Sagittal Images:

  In the plane of the medial malleolus, the tibialis posterior and flexor digitorum longus tendons are directly posterior to the medial malleolus. The tibialis posterior tendon enters the foot by passing deep to the flexor retinaculum and superior to the sustentaculum tali to its insertion on the tuberosity of the navicular bone (Stoller and Ferkel 1997).

  The flexor digitorum longus tendon also enters the foot after passing posterior to the medial malleolus and deep to the flexor retinaculum. This tendon is divided into four segments after crossing the flexor hallucis longus tendon, which contributes slips to the medial two divisions. These segments insert onto the bases of the distal phalanges. The quadratus plantaris muscle inserts at the division of the flexor hallucis into four tendons. Distally, each tendon is an origin for the lumbrical muscles (Stoller and Ferkel 1997).

  The deltoid ligament, composed of the tibio-calcaneal, tibionavicular, and anterior and posterior tibiotalar ligaments, appears as a wide band of low signal intensity radiating from the distal tibia (i.e., medial malleolus) to the tuberosity of the navicular bone and the sustentaculum tali. The flexor hallucis longus tendon is located posterior to the tibialis posterior tendon.
and the flexor digitorum longus. It passes posterior to the medial malleolus, deep to the flexor retinaculum. The low signal intensity tendon hugs the posterior talar process and inferior surface of the sustentaculum tali proximal to its insertion onto the base of the distal phalanx of the great toe (*Stoller and Ferkel 1997*).

The plantar flexor digitorum brevis (a first-layer muscle of the sole of the foot) and the quadratus plantaris (a second-layer muscle of the sole of the foot) are displayed on medial sagittal images. The adductor hallucis (a first-layer muscle) inserts onto the medial proximal phalanx of the first toe and is seen between the first and second metatarsals on medial sagittal images. The tibialis anterior tendon crosses the dorsal surface of the talus before it inserts on the medial cuneiform bone and the bone of the first metatarsal (*Stoller and Ferkel 1997*).

- **Mid-plane Sagittal Images:**

  The middle subtalar joint, the tarsal sinus, and the posterior subtalar joint are demonstrated on sagittal images medial to the mid-Sagittal plane. The anterior subtalar joint is shown in the plane of the cuboid and calcaneo-cuboid joint. The peroneus longus, which extends anteriorly along the lateral inferior surface of the calcaneus and is inferior to the peroneal tubercle, enters the foot at the lateral inferior margin of the cuboid. The extensor
hallucis longus tendon is identified along the dorsum of the foot and inserts onto the distal phalanx of the first toe. The interosseous talocalcaneal ligament, with its associated high signal intensity fat, is bordered anteriorly by the anterior process of the calcaneus and posteriorly by the lateral process of the talus. On T1-weighted sequences, the high signal intensity pre-Achilles fat pad is located directly anterior to the low spin intensity Achilles tendon (Stoller and Ferkel 1997).

- **Lateral Sagittal Images:**

  In the plane of the fibula, the peroneus brevis and longus tendons pass posterior to the lateral malleolus. The peroneus brevis lies anterior to the peroneus longus tendon and is in direct contact with the lateral malleolus. The peroneus brevis can be followed to its insertion on the base of the fifth metatarsal bone. The peroneus longus tendon disappears inferior and medial to the peroneus brevis tendon and enters the cuboid sulcus; therefore, it appears shorter than the peroneus brevis tendon on lateral sagittal images. (Stoller and Ferkel 1997).
GROSS ANATOMY OF THE ANKLE JOINT

**MRI Sagittal Plane of the Ankle**
*(From medial to lateral)*

- Tibialis anterior tendon
- Extensor hallucis longus tendon
- Middle subtalar joint
- Navicular
- Tibialis posterior tendon
- Flexor digitorum longus tendon
- Talus
- Achilles tendon
- Flexor hallucis longus tendon
- Sustentaculum tali
- Calcaneus
- Quadratus plantae
- Abductor hallucis longus muscle

Figure 11: MRI Sagittal Plane of the Ankle *(Stoller and Ferkel 1997)*

- Tibialis anterior tendon
- Extensor hallucis longus tendon
- Intertosseous talocalcaneal ligament
- Navicular
- Middle subtalar joint
- Intermediate cuneiform
- Tibia
- Flexor hallucis longus tendon
- Achilles tendon
- Talus
- Posterior subtalar joint
- Calcaneus
- Quadratus plantae muscle

Figure 12: MRI Sagittal Plane of the Ankle *(Stoller and Ferkel 1997)*
GROSS ANATOMY OF THE ANKLE JOINT

Figure 13: MRI Sagittal Plane of the Ankle (Stoller and Ferkel 1997)

Figure 14: MRI Sagittal Plane of the Ankle (Stoller and Ferkel 1997)
**Coronal images**

- **Posterior Coronal Images:**

  The thick, low signal intensity Achilles tendon is clearly displayed on posterior coronal images. Its attachment to the calcaneal tuberosity can also be observed on these images. The soleus muscle, with its inverted-V-shaped origin from the soleal line of the tibia and posterior fibula, contributes to the calcaneal tendon (or Achilles tendon), along with the gastrocnemius and plantaris. The peroneus brevis and flexor hallucis longus muscles are identified lateral to the soleus muscle, and the peroneal tendons are located inferior to the lateral malleolus. The flexor digitorum longus muscle and tendon cross superficially, in a medial-to-lateral direction, to the tibialis posterior in the distal calf. The tibialis posterior tendon is located medial to the posterior malleolus. The posterior talofibular and inferior tibiofibular ligaments are shown on coronal images at the level of the posterior malleolus and posterior process of the talus. The plantar aponeurosis is superficial to the flexor digitorum brevis muscle, whereas the quadratus plantae muscle lies deep to this muscle (*Stoller and Ferkel 1997*).

- **Mid plane Coronal Images:**

  The calcaneofibular ligament is best imaged at the level of the posterior subtalar joint and lateral malleolus. The lateral
process of the talus can be seen in the same sections as the anterior lateral malleolus. The middle subtalar joint is formed by the sustentaculum tali and the inferior medial talar surface. This is the best plane for evaluating talocalcaneal coalitions. The peroneus brevis and longus tendons course laterally, superior and inferior, respectively, to the peroneal groove of the calcaneus (Stoller and Ferkel 1997).

- **Anterior Coronal Images:**

  The tibiotalar and tibio-calcanean fibers of the deltoid ligament extend obliquely to the talus and vertically to the sustentaculum tali, respectively. The tibialis posterior tendon is medial to the deltoid ligament and superior to the sustentaculum tali, and can be used as a landmark. The flexor digitorum longus tendon enters the foot, having crossed superficially in a medial-to-lateral direction to both the tibialis posterior and the flexor hallucis longus tendons, which are parallel. The flexor digitorum longus tendon is located medial to the sustentaculum tali. The anterior compartment tendons (the tibialis anterior, the extensor hallucis longus, and the extensor digitorum longus) are displayed on the anterior surface of the distal tibia, medially and laterally. The anterior tibiotalar fibers of the deltoid ligament are also seen in the plane of the anterior tibia (Stoller and Ferkel 1997).
MRI Coronal Plane of the Ankle
(From posterior to anterior)

Figure 15: MRI Coronal Plane of the Ankle (Stoller and Ferkel 1997)

Figure 16: MRI Coronal Plane of the Ankle (Stoller and Ferkel 1997)
Figure 17: MRI Coronal Plane of the Ankle (Stoller and Ferkel 1997)

Figure 18: MRI Coronal Plane of the Ankle (Stoller and Ferkel 1997)
GROSS ANATOMY OF THE ANKLE JOINT

Figure: 19: MRI Coronal Plane of the Ankle *(Stoller and Ferkel 1997)*
PATHOLOGY OF POST-TRAUMATIC ANKLE JOINT
Pathology Of Post-Traumatic Ankle Joint

Injuries to the ankle result in many different combination of bone and ligament injury. The position of the foot influences the location of the initial stage of injury: Supination of the foot tightens the lateral structures, which are injured first; pronation tightens the medial structures, which then will be injured first. The injury pattern then moves sequentially around the ankle in the same direction as the deforming force. Abduction or external rotation, posterior displacement of the talus, vertical loading, or combinations of these forces causes fractures of the medial malleolus. In external rotation or abduction, the posterior tibiofibular ligament is under tension and can either rupture or, more commonly, avulse the posterolateral corner of the tibia. Syndesmotic disruption occurs from external rotation or abduction forces (Giesster et al. 1996).

I- Osseous injuries:

Osseous injuries include bone contusions, and fractures. The fractures can be further subdivided into acute fractures, osteochondral fractures, and stress fractures (Bencardino et al. 1999).

II- Non Osseous Injuries:

These injuries include:

A- Tendon Injury:

1) Achilles tendon injury.
2) Posterior tibial tendon injury
3) Peroneal tendon injuries.
4) Flexor hallucis and flexor digitorum longus injuries.
5) Extensor tendon injuries.

B- Ligament Injury:
   1) Lateral ankle sprain.
   2) Medial sprain and Syndesmotic injury.

C- Miscellaneous Injury:
   A. Impingement.
   B. Peripheral nerve entrapment.
   C. Compartment syndromes.
   D. sinus tarsi syndrome.
   E. Muscle injuries.

(El Refaiy et al. 2008).

I) Osseous Injuries:

Bone contusions:

The detection of such marrow contusions in patients is important, a delay in resumption of normal sport activities should be considered in the presence of such lesions to avoid progression of any weakening of the mechanical properties of bone related to the presumed disruption (Deutsch et al. 1992).
Fractures:

A- Acute Fractures:

Three main classification systems for acute ankle fractures exist. Ankle fractures may be described by, the number of malleoli involved [unimalleolar, bimalleolar, or trimalleolar); by the location of the fibular fracture relative to the ankle mortise [Weber Classification]; or by the mechanism of the injury [Lauge-Hansen classification] (Greenspan 2004).

1- Tibia and Fibula:

The ankle can be thought of as a ring in which bones as well as ligaments play an equally important role in the maintenance of joint stability. If the ring is broken in one place the ring remains stable (Fig. 20). When it is broken in two places, the ring is unstable and may dislocate (Smithuis 2010).

Figure 20: Stability of ankle (Smithuis 2010).
**Normal flexibility of the ankle.**

The ankle joint has to be flexible in order to deal with the enormous forces applied exerted on the talus within the ankle fork. The medial side of the joint is quite rigid because the medial malleolus - unlike the lateral malleolus - is attached to the tibia and the medial collateral ligaments are very strong (*Smithuis 2010*).

On the lateral side there is a flexible support by the fibula, syndesmosis and lateral collateral ligaments. This lateral complex allows the talus to move laterally and dorsally in exorotation during forward motion and subsequently pushes it back into its normal position. The fibula has no weight-bearing function, but merely serves as a flexible lateral support. The syndesmosis is the fibrous connection between the fibula and tibia formed by the anterior and posterior tibiofibular ligaments - located at the level of the tibial plafond (French for ceiling) - and the interosseous ligament, which is the thickened lower portion of the interosseous membrane. The anterior and posterior tibiofibular ligaments are often referred to as anterior and posterior syndesmosis (*Smithuis 2010*).

**Position of the foot.**

There are two positions of the foot in which the flexible ankle joint becomes a rigid and vulnerable system: extreme supination and pronation. In these positions forces applied to the talus within the ankle mortise can
result in fractures of the malleoli and rupture of the ligaments (Smithuis 2010).

In 80% of ankle fractures the foot is in supination (Fig. 21). The injury starts on the lateral side, since that is where the maximum tension is. In 20% of fractures the foot is in pronation with maximum tension on the medial side. The injury starts on the medial side with either a rupture of the medial collateral ligaments or an avulsion of the medial malleolus (Smithuis 2010).

Figure 21: Position of foot (Smithuis 2010).
Pull-off or push-off fractures.

The shape of a fracture indicates which forces were involved. An oblique or vertically oriented fracture indicates 'push-off'. A transverse or horizontal fracture is the result of a 'pull-off' (Fig. 22).

Figure 22: On the left image the lateral malleolus is pushed off by exorotation of the talus. On the right image the medial malleolus is pulled off by the medial collateral ligament due to pronation of the foot (Smithuis 2010).

Weber and Lauge-Hansen the two most commonly applied classification systems for ankle fractures (Smithuis 2010).
The Weber system

This system focuses on the integrity of the syndesmosis. It owes its popularity mainly to its simplicity (Smithuis 2010).

- Type A occurs below the syndesmosis, which is intact.
- Type B is a trans syndesmotic fracture with usually partial - and less commonly, total - rupture of the syndesmosis.
- Type C occurs above the level of the syndesmosis with usually a total rupture of the syndesmosis, and consequently instability of the ankle mortise.

(Smithuis 2010).

The Lauge-Hansen system

This system focuses on the trauma mechanism. Based on the findings on the radiographs you deduce what the trauma mechanism must have been. It stages the severity of the injury, which allows you to predict the ligamentous injury and instability (Fig. 23) (Smithuis 2010).

This system is based on:

- Position of the foot at the moment of injury, either in supination (80%) or in proration (20%).
- Direction of the force on the foot within the ankle mortise, which is either exorotation (80%) or adduction (20%) (Smithuis 2010).
Figure 23: Weber and Lauge-Hansen classification (Smithuis 2010).

**Classification:**

*Weber A - Lauge Hansen SA (Supination Adduction)*

This is the most simple ankle fracture. The diagnosis as well as the treatment usually poses no problems. It occurs in about 20-25% of all
ankle fractures. The foot is fixed on the ground in supination when an adduction force is applied to the talus. The first injury will occur on the lateral side, which is under tension (*Smithuis 2010*).

**Stage 1**

Supination results in a tear of the lateral collateral ligament or an avulsion fracture of the lateral malleolus below the level of the tibial plafond, i.e below the level of the syndesmosis (Fig. 24) (*Smithuis 2010*).  

**Stage 2**

More talar tilt results in the medial malleolus being *pushed off* in a vertical or oblique way. This second stage is very uncommon and is unstable (*Smithuis 2010*).

---

Figure 24: Weber A - Lauge Hansen SA (Supination Adduction) (*Smithuis 2010*).
Before we continue with the Weber B and C fractures, it is important to understand that most malleolar fractures have a ligamentous counterpart and vice versa (Table 1). The Tillaux fracture is an avulsion fracture of the tibia where the anterior syndesmosis attaches. It is an uncommon finding (Smithuis 2010).

<table>
<thead>
<tr>
<th>Ligamentous rupture</th>
<th>Fracture equivalent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medial collateral band</td>
<td>Transverse medial fracture</td>
</tr>
<tr>
<td>Lateral collateral band</td>
<td>Transverse lateral fracture</td>
</tr>
<tr>
<td>Anterior syndesmosis</td>
<td>Tillaux fracture</td>
</tr>
<tr>
<td>Posterior syndesmosis</td>
<td>Posterior malleolus fracture</td>
</tr>
</tbody>
</table>

Table 1: ligamentous rupture and fracture equivalent (Smithuis 2010).

**Weber B - Lauge Hansen SE (supination exorotation).**

This is the most common type and occurs in about 60-70% of all ankle fractures (Okanobo et al. 2012)

The foot is fixed on the ground in supination and an exorotation force is applied to the talus due to an endorotation of the lower leg (Fig. 25) (Smithuis 2010).

**Stage 1**

The first injury will occur on the lateral side, which is under maximum tension. As the talus exorotates, the anterior tibiofibular ligament ruptures first.
Stage 2

Since the foot is in supination, the lateral malleolus is held tightly in place by the lateral collateral ligaments and cannot move away without breaking. As a result more rotation of the talus will fracture the fibula in an oblique or spiral fashion because the lateral malleolus is pushed off from anterior to posterior. The fracture starts at or only a few cms above the level of the ankle joint and extends proximally.

Figure 25: Weber B - Lauge Hansen SE (supination exorotation) (Smithuis 2010).
Stage 3

Posterior displacement of the lateral malleolus fragment by the talus results in rupture of the posterior tibiofibular ligament or avulsion of the malleolus tertius.

Stage 4

More posterior movement of the talus will result in extreme tension on the medial side and the deltoid ligament will either rupture or pull off the medial malleolus in the transverse plane.

(Smithuis 2010).

The sequence of events in a Weber B fracture or Lauge-Hansen supination exorotation injury happens in a clockwise sequence (Fig. 26):

1. Rupture of the anterior tibiofibular ligament
2. Oblique fracture of the distal fibula
3. Avulsion of the posterior malleolus or rupture of the posterior tibiofibular ligament
4. Avulsion of the medial malleolus or rupture of the medial collateral ligament

(Smithuis 2010).
Immediately after the injury the injured parts may again align, which can make it difficult to detect the fractures and ligamentous ruptures (*Smithuis 2010*).

![Image](image.png)

**Figure 26:** In Weber B or supination exorotation injury the events take place in a clockwise manner (*Smithuis 2010*).

**Weber C - Lauge Hansen (PER) pronation exorotation.**

This is seen in approximately 20% of ankle fractures. The foot is fixed on the ground in pronation when an exorotation force is applied to the talus (Fig. 27) (*Smithuis 2010*).
**Stage 1**

The first injury will occur on the medial side, which is under maximum tension. It will lead to rupture of the medial collateral ligament or avulsion of the medial malleolus.

**Stage 2**

The talus rotates externally and moves laterally because it is free from its medial attachment. Due to the pronation, the lateral side is not under tension and the fibula can move away from the tibia. This causes rupture of the anterior syndesmotic ligament.

**Stage 3**

The fibula will be twisted distally, while proximally it is fixed in position. Finally the interosseous membrane will rupture up to the point where the fibular shaft fractures above the level of the syndesmosis. The fibular fracture may or may not be visible on the ankle X-rays.

**Stage 4**

Finally the posterior syndesmotic ligament ruptures, or there is an avulsion of the posterior malleolus, also known as the malleolus tertius.
The sequence of events in a Weber C fracture or Lauge-Hansen pronation exorotation injury also happens in a clockwise sequence (Fig. 28):

1. Avulsion fracture of the medial malleolus or medial collateral band rupture.
2. Rupture of the anterior tibiofibular ligament.
3. High transverse fracture of the fibula.
4. Avulsion of the posterior malleolus or rupture of the posterior tibiofibular ligament.

After the event the pieces may align again and be difficult to detect on the radiographs (Smithuis 2010).
Figure 28: The sequence of events in a Weber C fracture or Lauge-Hansen pronation exorotation injury also happens in a clockwise sequence (Smithuis 2010).
Common ankle fractures involving the tibia:

✓ Pylon (pilon) fracture.
  o Split or comminuted fracture of the distal tibial plafond.
  o Typically involves the anterior lip of the tibia.
  o Axial loading fracture of the tibia
    ▪ Jump from height or motor vehicle accident.
    ▪ Talus is driven up through the plafond (*Borrelli and Ellis 2002*).

✓ Isolated posterior lip (Malleolus) fracture.
  o Could occur by hyper plantar flexion mechanism.
  o Exclude Weber B and Maisonneuve mechanisms
    ▪ Supination external rotation but only if posterior lip of the tibia fractures just after tear of anteroinferior tibiofibular ligament with no oblique fibula fracture.
    ▪ Exclude proximal fibula fracture, medial malleolar fractures and disrupted syndesmosis (*Novelline et al. 2004*).

✓ Tillaux fracture.
  o Foot external rotation mechanism.
  o Avulsion fracture of the anterior tubercle of the tibia.
    ▪ Tension in the anterior tibiofibular ligament.
    ▪ If avulsion fracture of the fibula at attachment of the anterior tibiofibular ligament instead of anterior
tibial tubercle, then called Wagstaff-Lefort fracture (rare) (Novelline et al 2004).

✓ Tri-plane fracture.
  o Children and young adults before closure of the growth plate.
  o Plantar flexion with external rotation mechanism.
  o Three fracture planes;
    ▪ Transverse fracture of the epiphyseal plane.
    ▪ Sagittal fracture of the epiphysis.
    ▪ Coronal distal metaphyseal fracture.

(Novelline et al. 2004).

✓ Medial malleolar fractures may also part of fibular fracture mechanism (Borrelli and Ellis 2002).

Ankle Fractures – Fibula.

The distal fibula forms the lateral malleolus of the ankle: fracture of the distal fibula may contribute to instability of the ankle (Boutis et al. 2001).
- Classic imaging appearance: fracture lucency with variably displaced distal fibular fragment.
- Dupytren's fracture is an eponym for:
  - Fracture of the fibula up to 10 cm proximal to the tibiofibular syndesmosis.
  - Tear of syndesmotic and interosseous ligaments.
  - Lateral displacement of the talus.
  - May have fracture of posterior malleolus (Pinzur 2001).

2- Talar fractures and dislocations:

The talus is a unique and important functional unit of the hind foot. It supports the body weight and distributes the forces to the foot. Articular cartilage covers 60% of the talar surface, and there are no direct muscle or tendon attachments. Therefore the blood supply is vulnerable. The main blood supply enters the talus via the tarsal canal as a branch of the posterior tibial artery. This artery supplies the inferior neck and most of the body. Branches of the dorsalis pedis artery enter the superior aspect of the talar neck and supply the dorsal portion of the neck and the head of the talus. The peroneal artery supplies a portion of the lateral talus (Berquist et al. 2001).

1- Talar neck fractures:

Most commonly talar neck fractures. Talus can be dislocated with or without fracture (Novelline et al. 2004).
Hawkins classification of talus fractures:

- Helps predict osteonecrosis of the talar dome.
- Vessels enter talar neck on its undersurface.
- Type I – Non displaced fracture of the talar neck. Osteonecrosis approximately 10%.
- Type II – Mildly displaced neck fracture and displaced subtalar joint. Osteonecrosis approaches 40%.
- Type III – displaced neck fracture and dislocation of talar body from subtalar and tibiotalar joints. Osteonecrosis about 90% (Novelline et al. 2004).

**II-Talar body, head and process fractures:**

Fractures of the talar body and posterior and lateral processes are uncommon in adults and rare in children. Most of talar body fractures are due to significant falls or motor vehicle accidents that lead to axial compression of the talus between the tibial plafond and calcaneus. Fractures of the lateral process usually occur with the foot dorsiflexed and inverted. The calcaneus causes shearing of the lateral process. Described six basic fracture patterns, these included simple compression fractures, vertical fractures in the coronal and sagittal planes, posterior tubercle fractures, lateral tubercle fractures, and comminuted crush fractures. In addition, chip or avulsion fractures may also occur. Fractures of the head generally involve the talonavicular joint. Many body fractures, especially due to shearing
forces, are displaced and associated with sublaxation (Berquist 1989).

**III-Talar dome fractures:**

Osteochondral fractures of the talar dome differ from other chip or avulsion fractures in that they are more difficult to detect and prognosis is potentially worse than a non-articular chip fracture. Talar dome fractures are the most common talar fractures. This injury is much more common in adults. The etiology of the lesion is somewhat controversial. Suggested mechanisms include ischemic necrosis, congenital disorders, spontaneous necrosis, and trauma. They can occur after any type of injury to the ankle, including “simple sprains” (El Refaiy et al. 2008).

**3- Fractures of the calcaneus:**

Fractures of the calcaneus have been divided into intraarticular and extraarticular fractures based on the involvement of the posterior facet of the subtalar joint (Rubino et al. 2009.).

Intraarticular fractures of the calcaneus represent about 75% of all calcaneal fractures in adults (Schepers et al. 2009).

Sanders classified intraarticular fractures of the calcaneus into four types on the basis of fracture line location at the posterior facet (Badillo et al. 2011).
Nondisplaced fractures (displacement <2 mm) are classified as type I regardless of fracture lines. Types II–IV are displaced fractures with an increasing number of fracture lines and fragments. Type II fractures consist of two articular pieces from a single intraarticular fracture line and are divided into three subtypes on the basis of whether the fracture line location is lateral (IIA), central (IIB) or medial (IIC). Medial fractures are harder to evaluate and manage surgically. Type III fractures consist of three articular pieces from two fracture lines and are subdivided into types IIIAB, IIIB, and IIIBC. Fractures with more than three intraarticular fracture lines are considered comminuted and are classified as type IV (Badillo et al. 2011).

Extraarticular fractures account for about 25% of calcaneal fractures and include all fractures that do not involve the posterior facet of the subtalar joint. Generally, extraarticular calcaneal fractures fall into one of three categories depending on whether the involvement of the calcaneus is anterior, middle, or posterior. Type A fractures involve the anterior process of the calcaneus. Fractures that involve the mid calcaneus or body, including the trochlear process, sustentaculum tali, and lateral process, are type B fractures. Type C extraarticular calcaneal fractures involve the posterior calcaneus, including the posterior tuberosity and the medial tubercle (Badillo et al. 2011).
**Osteochondral injuries Pathogenesis:**

They are generally believed to be the result of shearing, rotatory, or tangentially aligned impaction forces. They can produce damage to the subchondral bone with preservation of the overlying articular cartilage. Alternatively, fragments consisting of cartilage alone (purely chondral fragments) or cartilage and underlying subchondral bone (osteochondral fragments) can result. The most common site of osteochondral injury at the ankle region is the talar dome. They implicate either the lateral or medial side of the talar dome (*El Refaiy et al. 2008*).

1. **Lateral talar dome lesions:**

   They develop when strong inversion force to a dorsiflexed foot is applied. The lateral dome margin impacted and compressed against the medial articular surface of the fibula, creating shearing the compressing components that if of sufficient strength would displace the fragment. The middle third of the lateral border of talar dome is frequently involved (*El Refaiy et al. 2008*).

2. **Medial talar dome lesions:**

   They develop when strong inversion force is applied a plantar flexed foot with lateral rotation of the tibia on the talus, allowing the posteromedial aspect of the talar dome to impact on the posteromedial lip of the tibia. The posterior third of the medial talar border is
frequently involved. They are deeper cup shaped than lateral lesions \textit{(El Refaiy et al. 2008)}.

There are four stages for osteochondral fractures based on the classification system of \textit{Berndt and Harty}.

<table>
<thead>
<tr>
<th>Stage 1</th>
<th>Is a small area of compression of the subchondral bone.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 2</td>
<td>Is a partially attached osteochondral fragment.</td>
</tr>
<tr>
<td>Stage 3</td>
<td>Is a completely detached but non-displaced fragment.</td>
</tr>
<tr>
<td>Stage 4</td>
<td>Is a displaced detached fragment.</td>
</tr>
</tbody>
</table>

Table 2: Classification system of \textit{Berndt and Harty} of osteochondral fracture \textit{(El Refaiy et al. 2008)}.

Healing of the osteochondral fracture is dependant on the stability of the fragment and the degree of intact overlying articular cartilage. Healing starts when hemorrhage at site of the defect develops into a fibrin clot that may eventually be modulated into fibrovascular repair tissue that gradually increases in cellularity and eventually revascularizes the segment. If there is failure of healing, the zone between the fragment and the cartilage or the subchondral bone, evolves into a dense fibrous tissue and dense avascular eburnated bone. This leads to lack of subchondral support, subchondral cyst formation, secondary articular deformity and subsequent degenerative changes \textit{(El Refaiy et al. 2008)}. 
C- Stress Fractures:-

They are micro-fractures or linear fractures occur either in the form of fatigue or insufficiency types. Fatigue type results from abnormal repeated stress on normal bone *(El Refaiy et al 2008)*.

Stress fractures occur when normal bone is subjected to repetitive stress. Although no individual stress is capable of producing a fracture, over time bone fatigue and failure result. Stress is the force or absolute load applied to a bone that may arise from weight-bearing or muscular action. The force may be applied as an axial, bending, or torsional load *(Spitz and Newberg 2003)*.

Bone is a dynamic tissue that requires stress for normal development, and it undergoes constant remodeling in response to changing environmental forces. Initially, osseous remodeling manifests as osteoclastic activity and resorption of lamellar bone. This is subsequently replaced by denser, stronger osteonal bone. In repetitive stress overload, however, the accelerated remodeling results in an imbalance between bone resorption and bone replacement, leading to weakening of the bone *(Boden et al. 2001)*.

Continued stress results in further imbalance, leading to bone fatigue, injury, and fracture. Osseous stress injury is not an all or none phenomenon, but a physiologic continuum ranging from
normal osseous remodeling, to accelerated remodeling with fatigue and early injury, to frank stress fracture (Boden et al. 2001).

Both resorption and replacement of bone characterize the early changes of stress injury to bone. This is manifest by local hyperemia and edema. Because of its high sensitivity for the detection of edema, MR imaging is an excellent modality for the detection of early osseous stress injury. Subsequently, MR imaging clearly depicts the more advanced findings of cortical bone breakdown and frank stress fracture. It is this differentiation between the changes of early stress injury to bone, and later stress fracture, that has predictive value in estimating the duration of disability, helping to guide therapy (Spitz and Newberg 2003).

Important external factors predisposing to stress fractures of the lower extremity are training errors, excessively hard training surfaces, and inadequate shoes; of the intrinsic biomechanical factors, the most important appear to be forefoot varus, subtalar varus, tibia vara, unequal length of the lower extremities, pes cavus, and muscular insufficiency (Schils et al. 1992).
II] Non-Osseous Injury:

A- Tendon Injury:

Tendon injuries commonly occur as a result of overuse and in the athletes, they typically occur in sports with repetitive cutting maneuvers, running or jogging. The injuries occur as a continuum from chronic degeneration to partial tears to full thickness tears. Although multiple tendons course through the ankle, only few are routinely pathologically affected, these are primarily flexor tendons, the extensor are rarely affected. The most commonly affected tendon is the Achilles tendon, followed by tibialis posterior and peroneus brevis tendon (Bencardina et al. 1999).

The tendon is an elastic structure which primary function is to transmit motion passively from a contracting muscle to a bone or fascia at its insertion. The tendon is formed of collagen, elastin and reticulin fibers; materials that give its strength, flexibility and bulk. The collagen fibers, which dominate the composition of the tendons. This arrangement account is for the ability of the tendon to withstand heavy loads more than twice that of its associated muscle (Frey et al. 1988).

After tendon injury, the tendon and tendon sheath follow a standard sequence of pathological changes.
- The first stage of injury is disruption of well-ordered parallel arrangement of collagen fibers.
- The second stage of injury i.e. with increased degree of damage, there is an increase in the fibroblasts, endothelial cells and blood vessels.

- The final stage of injury is fibrosis; the tendon fibres are twisted and interlaced with new collagen in a haphazard configuration. *(Deutsch et al. 1992).*

- Tenosynovitis refers to the inflammation of the investing soft tissues of the tendon whereas tendonitis refers to an injury or symptomatic degeneration of the tendon itself *(El Refaiy et al. 2008).*

- Eventually, the tendon and its sheath become bound together in an inflammatory mass and the normal gliding between motion that occurs between tendon and synovium becomes retracted and ultimately non-existing *(Deutsch et al. 1992).*

**Clinical Classification:**

- **First degree** is low grade inflammatory process. There are swelling, oedema and some discomfort on use of the affected tendon. But there is no loss of strength or restriction of motion.

- **Second degree** is incomplete disruption.

- **Third degree** is complete rupture with total or near total discontinuity and major loss of function *(Deutsch et al. 1992).*
1- Achilles Tendon Injury:

- Achilles tendon injuries have become more common over the last few decades as the number of runners in the population has increased. Athletes in running sports have a high incidence of Achilles tendon overuse injuries and approximately 75% of complete Achilles tendon tears and most partial tears are related to sports activities that include repetitive jumping and sprinting movements (El Refaiy et al. 2008).

- Degeneration of the tendon characteristically occur 2cm to 6cm proximal to the calcaneal insertion, this has been related to both relative ischaemia in the portion of the tendon because of watershed phenomenon and an intrinsic weakness in the tendon at this location as the fibres of the gastrocnemius and soleus tendon internally forming the common tendon (Bencardino et al. 1999).

- The Achilles tendon rupture is usually diagnosed clinically and ancillary studies are obtained to determine the extent of injury and treatment planning (El Refaiy et al. 2008).

- Clinically patients presents with pain, local swelling and inability to raise up on their toes on the affected side. Also radiographic findings suggestive of Achilles tendon injury or pathology include abnormal thickening of the tendon or obscuration of the pre-Achilles fat [Kager's fat pad] (Larry and mark 1995).
2- Posterior Tibial Tendon Injuries:

- Posterior tibial [PT] tendon injuries typically occur in sports with rapid changes in direction, as it is one of the main stabilizers of the hind foot and is responsible for inversion and plantar flexion (Bencardino et al. 1999).

- The most common PT tendon injury in athlete is acute tenosynovitis caused by overuse, the PT tendon sheath contain normally physiologic fluid and should not be mistaken for tenosynovitis (El Refaiy et al. 2008).

- PT tendon injury typically occurs as the tendon passes posterior to the medial malleolus. It is most susceptible to injury at this location because of frictional forces (Bencardino et al. 1999).

- The PT tendon is normally 1.5 to 2 times the size of the adjacent flexor digitorum longus [FDL] tendon. The presence of an accessory navicular bone may predispose the PT tendon to injury, fibres of the PT tendon generally insert onto both the navicular bone and the accessory bone. It is this abnormal insertion of the PT tendon that has been theorized to predispose patients to tendon tear (El Refaiy et al. 2008).

- The patients present with medial ankle pain, local tenderness and swelling, by local examination may reveal non palpable tendon, rupture of the PT tendon can lead to progressive flat foot deformity (El Refaiy et al. 2008).
3- *Peroneal Tendon Injuries:*

- Peroneal tendon subluxations and dislocations are uncommon and were originally described in ballet dancers. In more recent years skiing has become the most common sport associated with peroneal tendon injury (*Safran et al. 1999*).

- The peroneus brevis and longus tendons are contained within a common tendon sheath. The peroneus brevis tendon lies just anterior to the peroneus longus tendon and posterior to the lateral maleollus and is generally injured first. This is caused by pressure and friction on peroneus brevis tendon by adjacent peroneus longus tendon (*Bencardino al. 1999*).

- The patients clinically presents acutely with an ankle sprain or have symptoms of chronic instability and if the tendon is torn the patient will be unable to effectively evert the foot (*Deutsch et al. 1992*).

4- *Flexor Hallucis and Flexor Digitorum Longus injuries:*

- Injury to the flexor hallucis longus [FHL] tendon typically occurs in soccer players and runners. The FHL tendon runs posterolateral to the PT and FDL tendons in the medial aspect of the ankle. The FHL sheath communicates with the ankle joint in 20% of patients so fluid within the tendon sheath is not necessarily pathology (*Bencardino et al.1999*).
The intersection of the FDL and FHL tendons forms the "master Knot of Henry" which is in close proximity to the medial plantar nerve, one of the terminal branches of the PT nerve. The tendons may become entrapped at this location, referred to as jogger's foot (Masciocchi et al. 2000).

The function of FHL tendon is believed to be non-essential and surgical repair to alleviate pain, even in athletes, is recommended only when the tendon ends are easily amenable to surgery (El Refaiy et al. 2008).

5- Extensor Tendon Injuries:
- Tears of the extensor tendons are rare. When they do occur in athletes, downhill runners, skiers, and soccer plays, the anterior tibial tendon is the most common extensor tendon injured (Bencardino et al. 1999).

Rupture of the tibialis anterior tendon can occur between the extensor retinaculum and insertion onto the medial first cuneiform and adjacent 1st metatarsal. Weakness of dorsiflexion, localized tenderness and drop foot gait are observed on clinical evaluation (El Refaiy et al. 2008).
B- Ligament Injury.

Ligamentous injuries of the ankle are extremely common. Ligament injury caused by excessive range of motion at the joint, in the absence of fracture or dislocation, is a sprain. The ankle sprain is a common ankle injury that can lead to subsequent re-injury resulting in instability and permanent disability (Marchi et al. 1999).

In many sports, the ankle is weight bearing, with the foot plantar flexed weather the athlete is running, performing cutting maneuvers, or landing following a jump. It is the unpredictability of jump landings in basketball that contributes to the high incidence of ankle sprains (Lassiter et al. 1989).

The diagnosis of an acute ankle sprain is based primarily on the clinical examination. Conventional radiographs are performed to exclude fracture and often show only soft tissue swelling or minimal malalignment. The common sports-related ligament injury in the foot involves the plantar capsular ligament of the first metatarsophalangeal joint (MTP; turf toe) (El Refaiy et al. 2008).

1- Lateral Ankle Sprain:

Lateral ankle sprains are common injury. The lateral ankle sprain has been reported to represent up to 45% of basketball injuries and 31% of soccer injuries (Lynch et al. 1989).
In addition, it has been reported that 17% to 25% of sports injury time-loss is directly related to ankle sprains (*Safran et al. 1999*).

Most (up to 85%) sprains are caused by inversion forces during plantar flexion with damage to the lateral ligaments. This is caused by three main factors. First, the bony stability of the ankle mortise is diminished during plantar flexion because of the shape of the talar dome. Second, the lateral malleolus extends more distally than the medial malleolus providing more bony obstruction to eversion than inversion, some believe that the deltoid ligament is stronger than the lateral ligament complex (*Safran et al. 1999*).

The sequence of lateral ligament complex tears is well established involving the ATFL first, then the CFL and finally the PTFL (*Safran et al. 1999*).

O'Donohue's classification of lateral ankle sprains is widely accepted and the sprain is graded on a scale of 1 through 3 (*Safran et al. 1999*).

<table>
<thead>
<tr>
<th>Grade 1</th>
<th>Sprains represent microscopic injury of the ATFL without instability or stretching.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grade 2</td>
<td>Sprains represent a partial tear of the ATFL with or without a partial tear of the CFL with mild laxity and instability</td>
</tr>
<tr>
<td>Grade 3</td>
<td>Injury entails complete disruption of both the ATFL and CFL with gross ankle laxity and instability.</td>
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</tbody>
</table>

(*Safran et al. 1999*)
Grades 1 and 2 are typically treated conservatively with excellent results, whereas treatment of grade 3 injuries remains controversial with both surgical and non-surgical treatments having been successful (Liu et al. 1994).

Radiologic evaluation of ankle sprains typically begins with conventional radiographs to exclude malalignment, fractures, or osteochondral injury (El Refaiy et al. 2008).

The ankle mortise should be symmetric about the talus on all three views. Widening of the mortise or a tibiofibular distance at the level of the anterior tibial tubercle greater than 5 mm on either the AP or mortise views is abnormal (El Refaiy et al. 2008).

On the lateral view, the crescent-shaped inferior border of the tibial plafond and the talar dome should remain parallel and less than 5 mm. Stress radiographs may be performed to evaluate instability of the ankle mortise (Masciocchi et al. 2000).

2- Medial Sprains and Syndesmotic injury:

Although less common than lateral ligament complex tears, deltoid ligament and Syndesmotic ankle injuries do occur constituting 5% and 10% of ankle sprains, respectively (Liu et al. 1994).
They are generally the result of an eversion injury or an external rotational force caused by either a direct blow to the lateral aspect of the ankle or to the lateral aspect of the player's knee with the foot planted firmly on the ground in external rotation (*El Refaiy et al. 2008*).

As with lateral ankle sprains, the diagnosis are made clinically and radiographs are performed to exclude osseous injury. If medial widening of the mortise or tibiofibular diastases is seen, implying a tear of the deltoid ligament or syndesmosis, a high fibular fracture, first described by Maisonneuve in 1840 should be excluded clinically or radiographically (*El Refaiy et al. 2008*).

**III- Miscellaneous Injuries:**

**A- Impingement.**

Ankle impingement syndromes are common in athletes and may be separated into osseous impingement and soft tissue impingement (*Masciocchi et al. 2000*).

(i) *Osseous Impingement:*

In the ankle is often caused by a bony excrescence, an osteophyte, or an accessory ossicle that subsequently presses against the tibia during exaggerated dorsiflexion or plantar-flexion. Soccer players may develop an exostosis on the dorsal aspect of the talonavicular joint causing anterior impingement during
dorsiflexion. The diagnosis is often suggested on conventional radiographs (*El Refaiy et al. 2008*).

Os trigonum syndrome is seen in runners, soccer players, and football players and is a cause of posterior impingement. Os trigonum syndrome presents with posterolateral ankle pain caused by impingement of the calcaneus on the posterior tibial plafond. The os trigonum may be trapped between the posterior lip of the tibia and calcaneus resulting in disruption of the cartilaginous synchondrosis between the ossicle and the lateral talar tubercle leading to contusion or compression fracture of the os trigonum (*Cooper et al. 1999*).

**(ii) Soft Tissue Impingement:**

Posterior impingement syndrome may be caused by the extension of the posterior intermalleolar ligament into the posterior tibiotalar joint. This ligament, a normal variant, arises between the tibiofibular and talofibular ligaments and crosses posterior to the ankle parallel to the ankle mortise. It is hypothesized that during maximal plantar flexion, while an weight bearing, the intermalleolar ligament extends intra-articularly and becomes thickened and frayed. This may progress resulting in posterior impingement of the tibiotalar joint presenting with locking and pain (*Cooper et al. 1999*).
Anterolateral impingement occurs following injury to the tibiofibular and anterior talofibular ligaments with thickening of the ligaments and scarring of the joint capsule as a sequelae of recurrent ankle sprains. The thickened band of fibrous tissue, termed a meniscoid lesion, becomes entrapped between the talus and lateral malleolus during dorsi-flexion. Talar impingement occurs when the distal fascicle of the ATFL becomes in contact with the anterolateral talar dome during dorsiflexion causing pressure and friction (Safran et al. 1991).

**B- Peripheral Nerve Entrapment:**

Peripheral nerve entrapment is a cause of ankle pain and parasthesias in athletes. Deep peroneal nerve entrapment, medial plantar nerve entrapment, and tarsal tunnel syndrome are the most common entrapment neuropathies. Deep peroneal nerve entrapment, also referred to as anterior tarsal tunnel syndrome, is generally seen in runners, skiers, and soccer players. It is generally associated with extensor hallucis longus (EHL) tenosynovitis or a dorsal osteophyte of the tibiotalar joint leading to irritation or impingement of the deep peroneal nerve. Although plain films may demonstrate the presence of the dorsal osteophyte, MR imaging is the method of choice to evaluate for EHL tenosynovitis (Masciocchi et al. 2000).
C- Compartment Syndromes:

Compartment syndromes are rare. The most common location is the leg; however, the foot is also one of the more common locations (El Refaiy et al. 2008).

Compartment syndromes are classified as either acute or chronic. In both acute and chronic compartment syndromes there is increased pressure within a muscular compartment confined by the surrounding fascia. The increased pressure reduces tissue perfusion producing relative ischemia and tissue necrosis (Eisele and Sammarco 1993).

Acute compartment syndrome occurs following a single traumatic event. In the athlete, acute compartment syndrome usually occurs in contact sports, such as hockey, rugby, and football accompanied, by an acute fracture or muscle rupture. Acute compartment syndrome is generally a clinical diagnosis and surgical treatment is urgent (El Refaiy et al. 2008).

Chronic compartment syndrome or chronic exertional compartment syndrome is often a difficult diagnosis. Chronic exertional compartment syndrome follows repetitive axial loading and can be seen in long distance runners. The diagnosis has also been associated with fractures of the forefoot and hind-foot. The diagnosis of chronic exertional compartment syndrome often follows an extensive work-up and radiologic imaging, including
conventional radiographs, MR imaging, and nuclear medicine scintigraphy, generally focusing on excluding fractures and tumors (El Refaiy et al. 2008).
MRI FINDINGS OF POST-TRAUMATIC ANKLE JOINT
MRI Findings of post-traumatic ankle Joint

I) Osseous Injuries:-

The presence of marrow edema is nonspecific but, in the setting of trauma, implies the presence of acute trabecular injury. Bone bruises or contusions are characterized by high T2 signal on fat-suppressed images and low T1 signal in the absence of fracture. Fracture lines are typically of low signal on all imaging sequences with adjacent high T2-weighted signal on fat-suppressed sequences representing surrounding edema and hemorrhage (Dunfee et al. 2002).

1] Bone Contusions:

Bone bruise indicates the traumatic origin of these bone marrow changes. It was defined as region of T2-hyperintensity in the absence of frank osseous fracture or subchondral cysts (Vanhoenacker et al. 2007).

During the acute stage, bone contusions (bone bruises) manifest at MR imaging as reticular areas of hypointensity on T1-weighted images and hyperintensity on T2-weighted and fat-suppressed images. They are related to micro fractures of the trabecular bone and edema or hemorrhage within the bone marrow. Bone contusions normally resolve within 8–12 weeks. In most cases, radiographic findings are negative. The clinical significance of bone contusions detected with MR imaging is unknown, but it is
generally accepted that continued stress placed on a contused bone may lead to complete fracture (Rosenberg et al. 2000).

2] Ankle Fractures:

In patients with normal plain films and high clinical suspicion of injury, MR imaging or bone scintigraphy may be used to assess the presence or absence of fracture. MR imaging is preferred as bone scintigraphy lacks specificity (Bencardino et al. 2000).

Stress fractures:

MR imaging allows depiction of abnormalities weeks before the development of radiographic abnormalities and has comparable sensitivity and superior specificity compared with radionuclide techniques for the detection of osseous abnormalities. MR imaging has the additional advantage of demonstrating concomitant soft tissue injury (Spitz and Newberg 2003).

Early MR imaging findings in osseous stress injury begin with periosteal, muscle, or bone marrow edema that is only appreciated on the STIR or fat-suppressed T2-weighted sequence. As injury becomes more severe, findings include marrow edema identified on both T2- and T1-weighted images and signal abnormalities in the cortical bone (Fig. 29). Frank stress fractures are diagnosed when identifying band-like areas of low signal in the intramedullary space that may be continuous with the cortex. The most common pattern of a fatigue-type fracture is a fracture line that is low signal on all
pulse sequences, surrounded by a larger, ill-defined zone of edema (Spitz and Newberg 2003).

The fracture line is continuous with the cortex and extends into the intramedullary space oriented perpendicular to the cortex and the major weight bearing trabeculae (Boden et al. 2001).

**Figure 29.** Stress fracture. Sagittal T1-weighted MR image demonstrates a transverse, non displaced fracture of the calcaneus (arrow) with surrounding bone marrow edema (Rosenberg et al. 2000).

MR imaging findings and a MR imaging classification of osseous stress injury (Fig. 30). Grade 1 injuries (mild) demonstrate periosteal edema, without focal bone marrow abnormality. Grade 2 injuries demonstrate more severe periosteal edema with bone marrow edema detected on T2-weighted images only. Grade 3 injuries demonstrate moderate to severe edema of both the
periosteum and marrow on both T1- and T2-weighted images. Grade 4 injuries demonstrate a low signal fracture line on all sequences, with changes of severe marrow edema on both T1- and T2-weighted sequences (*Spitz and Newberg 2003*).

![Figure 30: Calcaneal stress fracture. Sagittal fast STIR (TR/TE, 2750/43; inversion time 160 milliseconds) MR image demonstrates an oblique linear area of high signal intensity within the calcaneus (wavy arrows). No cortical fracture is present (*Dunfee et al. 2002*).](image)

**Osteochondral Fractures:**

Osteochondral fractures originate from single or multiple traumatic events, leading to partial or complete detachment of the osteochondral fragment with or without associated osteonecrosis. The term osteochondral lesion (or transchondral fracture) is preferred to the term osteochondritis dissicans because it better describes the traumatic nature of these lesions. Transchondral
fracture refers to those lesions that exclusively involve the articular cartilage with no associated subchondral bone lesion (Flick and Gould 1985).

The signal intensity of the interface between normal bone and osteochondral fragment has received attention in MR imaging literature. Hypo-intensity of the interface with T2 weighted pulse sequences indicates healing and stability, while hyper-intensity may indicate fluid interposed between the fragment and the donor site and therefore instability (El Refaiy et al. 2008).

A potential pitfall is hyper-intensity at the interface related to healing granulation tissue. In such cases intra-articular injection of gadolinium based contrast material may be helpful. Contrast material interposed between the fragment and the donor site indicates lack of healing and instability. Conversely if no contrast material is seen at the interface, healing and stability of the fragment with an intact cartilage are expected (Mesgarzadeh et al. 1987).

The signal intensity of the fragment itself is also significant. Low signal intensity in all pulse sequences indicate necrosis, whereas hyper-intensity on T1 weight images indicate viable bone marrow. Viability can be further assessed by means of intravenous injection of gadolinium based contrast material with fat suppressed, T1 weighted pulse sequences. Enhancement of the bone marrow of
the fragment indicates viability, whereas lack of enhancement indicates non-viable tissue (*Nelson et al. 1990*).

Berndt and Harty have classified osteochondral lesions into four stages based on the integrity of the articular cartilage and condition of the subchondral fragment. Stage I: Lesions involve the subchondral bone, with preserved integrity of the overlying articular cartilage (Fig. 31). Stage II: Lesions consist of a partially detached fragment of articular cartilage and subchondral bone (Fig. 32). Stage III: Lesions are characterized by a completely detached fragment that is still located within the defect produced by the fracture (Fig. 33 & 35). Stage IV: Lesions consist of a completely detached osteochondral fragment located in the joint recess away from the fracture site (Fig. 34) (*Rosenberg et al. 2000*).

Non-fat-suppressed T1-weighted sequences and gradient echo or (spoiled gradient echo) sequences are most sensitive for osteochondral injuries, and the ankle mortise should be evaluated in coronal and sagittal projections (*Zoga and Schweitzer 2003*).
Figure 31. Stage I osteochondral lesion. Coronal T1-weighted MR image shows a subchondral area of decreased signal intensity in the medial talar dome. (Rosenberg et al. 2000)

Figure 32. Stage II osteochondral lesion. Coronal T1-weighted MR image reveals a partially detached osteocartilaginous fragment in the lateral talar dome (arrow). (Rosenberg et al. 2000)
Figure 33. Stage III osteochondral lesion. Coronal T1-weighted MR image reveals an osteochondral fragment that is completely detached from the talus (arrow) but is still located within its crater (Rosenberg et al. 2000)

Figure 34. Stage IV osteochondral lesion. Coronal T1-weighted MR image demonstrates a crater in the medial talar dome (white arrow). Note also the separate, nonviable bone fragment displaced away from the donor site (black arrow) (Rosenberg et al. 2000)
Osteonecrosis:

MR imaging is valuable in assessing the presence, size and fragment viability of post-traumatic osteonecrosis (Fig. 36) (El Refaiy et al. 2008).

Areas of inhomogeneous signal intensity surrounded by a hypointense band, sometimes with second band of high signal intensity on T2 weighted images (double line sign) are characteristic findings in osteonecrosis of the femoral head before subchondral fracture and collapse occur, these findings can also be seen in post traumatic osteonecrosis of the talus (Mitchell and Kressel 1988).
**Figure 36.** Avascular necrosis of the talus. Sagittal STIR MR image demonstrates serpentine areas of increased signal intensity in the talus (arrows). *(Rosenberg et al. 2000)*

### II] Non Osseous Lesions:

1] **MRI Appearance OF Tendon Injuries:**

MR imaging is the primary modality used to assess tendon injury. Normal tendons are of low signal intensity on all pulse sequences because of the high collagen concentration and low water content. Short TE sequences help to define better the anatomy because of the inherent higher signal-to noise ratio and differentiating fat from hemorrhage. Abnormally increased signal intensity on short TE sequences denotes tendinosis *(Dunfee et al. 2002)*.

Abnormal T2 signal within the tendon probably represents a more severe injury or partial tear. T2-weighted sequences are
primarily used to visualize edema, hemorrhage, fluid, and fibrosis. Gradient echo, STIR, and fat-suppressed T2-weighted sequences are very sensitive for noting abnormal water content within the tendon or adjacent soft tissues and bone in the setting of trauma (Dunfee et al. 2002).

With the exception of the Achilles tendon, which is well visualized in both the sagittal and axial planes, the tendons of the ankle and foot are evaluated best on the axial images. Each tendon should be carefully traced to verify normal morphology and continuity (Manaster et al. 2007).

Tenosynovitis is diagnosed by the presence of a large amount of fluid within the tendon sheath with morphologically normal tendons. The commonly used surgical grading system of tendon tears classifies tendon injuries as 1 to 3. Type 1 is degeneration and partial intrasubstance tear denoted by fusiform enlargement of the tendon. Type 2 is a high-grade partial tear with focal attenuation of the tendon. A type 3 tear is a complete tear with discontinuity of the tendon and retraction (Dunfee et al. 2002).

Exceptions to low signal of normal tendons:

Magic angle effect, ossicles and fibro cartilage in tendons (Manaster et al. 2007).

Tendon injuries can be grouped into several categories according to MRI findings:-
a) Tendinosis:

MR characteristics include fusiform shape and focal areas of increase tendon girth associated with increase signal intensity within the tendon on T1 weighted and proton density weighted images. T2 signal intensity alterations are noted when significant inter-substance degeneration is present (Nikken et al. 2005).

b) Tenosynovitis and peritendinosis:

They are caused by inflammation or mechanical irritation of the tendon sheath and peri-tendon respectively. MR images reveal fluid accumulation, synovial proliferation or scarring within the tendon sheath or adjacent soft tissues. Stenosis tenosynovitis occurs when synovial proliferation and fibrosis surround the tendon, causing entrapment and even rupture. It manifests as areas of intermediate to low signal intensity in the soft tissues around the tendon with all MR images sequences (Nikken et al. 2005).

c) Partial rupture:

Manifest on T1 WIs and proton WIs and occasionally on T2 WIs as an area within the substance of tendon having signal intensity similar to that seen in advanced peritendinosis (Nikken et al. 2005).

d) Complete rupture:

It is depicted as complete disruption of the tendon fibers. MR imaging is useful in the detection of dislocation and sublaxation of
the peroneal and posterior tibial tendons and in assessment of tendon disease (Nikken et al. 2005).

**Posterior compartment:**

**Achilles tendon Injuries:**

Tendon best seen on sagittal images. Uniform in diameter on sagittal images. Concave or flat anterior surface on axial images. Occasional shifting bulge on anterior surface reflects spiraling soleus and gastrocnemius tendon fibers. Punctuate increased signal on T1, PD, due to infolding paratenon vessels and connective tissue. Paratenon: ring of intermediate signal surrounding medial, posterior, and lateral tendon margins. Kager fat pad anterior to tendon, typically traversed by vessels. Minimal amount of fluid in retrocalcaneal bursa is normal. Small tendon medial to Achilles is plantaris (Manaster et al. 2007).

With Achilles paratendinitis, the tendon maintains its normal size and shape with abnormal semi circumferential T2 hyper intensity peripherally at its paratenon (Fig. 37). The Achilles is enlarged in a fusiform configuration on sagittal images with an abnormal anterior convexity but without focal signal abnormality on both T1-weighted and T2-weighted sequences (Zoga and Schweitzer 2003).
Figure 37. T2-weighted fat-suppressed image through the ankle demonstrating abnormal fluid signal at the Achilles paratendon (arrows). Note the loss of the normal anterior concave border of the Achilles, in this case of paratendinitis (Zoga and Schweitzer 2003).

At MR imaging, partial Achilles tendon tears demonstrate heterogeneous signal intensity and thickening of the tendon without complete disruption (Fig. 38, 39 & 40). Differentiation between partial tear and severe chronic Achilles tendinosis may be difficult apart from clinical history. However acute partial tears are often associated with subcutaneous edema, hemorrhage within the Kager Fat Pad and intra-tendinous hemorrhage at MR imaging, whereas chronic tendinosis doesn't usually demonstrate increased subcutaneous signal intensity on T2 weighted images (Rosenberg et al. 2000).
Figure 38. Chronic tendinosis of the Achilles tendon. Sagittal T1-weighted MR image shows fusiform thickening of the Achilles tendon without evidence of increased intrasubstance signal intensity (arrows) (Rosenberg et al. 2000).

Figure 39. Sagittal FS PD FSE MR shows hyper intense signal in distal Achilles tendon. partial tear of the distal Achilles tendon (insertional tear) (Stoller et al. 2004).
**Figure 40.** Insertional partial tear of the Achilles tendon. Sagittal STIR MR image shows increased signal intensity at the insertion site of the Achilles tendon (white arrowheads) associated with retrocalcaneal bursitis (black arrowhead). A prominent posterosuperior calcaneal tuberosity (Haglund deformity) and edematous bone marrow (*) are also noted. *(Rosenberg et al. 2000).*

Complete Achilles tendon rupture manifest as discontinuity with fraying and retraction of the torn edges of the tendon (Fig. 41). In acute rupture, the tendon gap demonstrates intermediate signal intensity on T1 weighted images and high signal intensity on T2 weighted images, findings that are consistent with edema and hemorrhage, whereas in chronic ruptures, scar or fat may replace the tendon. Partial rupture occurs in approximately 2% of surgically treated Achilles tendon rupture *(Rosenberg et al. 2000).*
Figure 41. Complete tear of the Achilles tendon. Sagittal T2-weighted MR image depicts complete disruption and retraction of the torn edges of the Achilles tendon (arrows) with a fluid-filled gap (*) (Rosenberg et al. 2000).

*Posterior tibial tendon injuries:*

At MR imaging, tenosynovitis appear as fluid within the tendon sheath. The tendon demonstrates normal signal intensity and morphologic characteristics, although nodular or diffuse thickening in chronic tenosynovitis and scarring to the peritenon may be encountered tendinosis manifests as mild to severe heterogeneity and thickening of the tendon (Rosenberg et al. 2000).
Chronic posterior tibial tendon rupture typically develops in women during the 5th and 6th decades of life and is usually associated with flat foot deformity. The tear is commonly noted behind the medial malleolus, where the tendon is subjected to significant amount of friction, acute partial or complete rupture of the posterior tibial tendon in young athletic individuals is less common and is usually seen at the insertion of the tendon on the navicular bone (El Refaiy et al. 2008).

MR imaging classification of chronic posterior tibial tendon ruptures divides these injuries into three types: (Stoller et al. 2004).

**Type I partial tear:**
This consists of an incomplete tear with fusiform enlargement, intrasubstance degeneration and longitudinal splits (Fig. 42 & 43). On axial MR images, the diameter of the tendon may be five to ten times that of the adjacent flexor digitorum longus tendon. High signal intensity foci representing longitudinal splits are noted within the substance of the tendon on T1 weighted and proton density weighted images, thus diagnostic overlap exists between sever tendinosis and partial type I tears because both demonstrate fusiform thickening of the tendon with intrasubstance signal intensity alteration (Ouzounian and Myerson 1992).
Figure 42. Type I tear of the posterior tibial tendon. On an axial T2-weighted MR image, the markedly thickened tendon (straight arrow) has a diameter 10 times that of the adjacent flexor digitorum longus tendon (curved arrow). Heterogeneous intrasubstance signal intensity representing longitudinal splits is also noted (Rosenberg et al. 2000).

**Type II partial tear:**

Attenuated section of tendon at level of medial malleolus with sub-tendons (Fig. 44) (Stoller et al. 2004).
Figure 43. Advanced type I tear of the posterior tibial tendon. Axial T1-weighted MR image shows marked tendon thickening as well as high-signal-intensity foci (arrow) representing longitudinal splits (Rosenberg et al. 2000).

Figure 44. Type II tear of the posterior tibial tendon. Axial proton-density-weighted MR image shows an attenuated posterior tibial tendon (open arrow) with a caliber equal to that of the adjacent flexor digitorum tendon (solid arrow) (Rosenberg et al. 2000).
**Type III tear:**

Complete tendinous discontinuity with low to intermediate signal intensity fluid-filled gap (Fig. 45) (*Stoller et al. 2004*).

Axial plane key in evaluating changes of tendon Morphology. Coronal and sagittal planes used for secondary confirmation of tendon pathology (*Stoller et al. 2004*).

A number of soft tissue and bone abnormalities are encountered at MR imaging in patient with posterior tibial tendon injuries. These include:

- Fluid within the tendon sheath.
- Fluid within the medial or lateral bursae, sinus tarsi syndrome.
- Periostitis at the insertion of the flexor retinaculum on the tibia, hind foot valgus, subtalar and talonavicular malalignment, and accessory navicular bone (*Sonin et al. 2010*).

The mechanism of injury is usually related to sever dorsiflexion associated with a torn flexor retinaculum, allowing the tendon to slide out of its groove (*Sonin et al. 2010*).

On Axial MR images sublaxation or complete dislocation of the posterior tibial tendon is easily identified. The tendon is seen medial or anterior to the medial malleolus (*Bencardion et al. 2000*).
Figure 45. Type III tear of the posterior tibial tendon. Axial T2-weighted MR image demonstrates absence of the posterior tibial tendon. The tibial retromalleolar groove has been replaced by synovial fluid and debris (arrow) (Rosenberg et al. 2000).

Figure 46. Dislocation of the posterior tibial tendon. Axial proton-density-weighted MR image shows the posterior tibial tendon anterior and medial to the tibial malleolus (black arrow). The flexor digitorum longus tendon is medially displaced within the retromalleolar groove (white arrow) (Rosenberg et al. 2000).
Peroneal tendon injuries:

Peroneal tendon injuries are frequently encountered and include peritendinosis, tenosynovitis, tendinosis, rupture and dislocation (El Refaiy et al. 2008).

MR imaging characteristics of peritendinosis and tenosynovitis, include scarring around the tendons and fluid within the common tendon sheath respectively (Fig. 47). Acute and chronic ruptures of the peroneal tendons occur in young athletes due to overuse and may be related to degenerative wear and tear in older, more sedentary patients (El Refaiy et al. 2008).

Calcaneal fractures typically predispose to partial tears, dislocation and entrapment of the peroneal tendons. Longitudinal tears of the peroneus brevis often begin at the fibular groove with extension toward the insertion site at the base of the fifth metatarsal. The split tendon fibers often reside on either side of the peroneus longus tendon (Fig. 49) (Dunfee et al. 2002).

On axial MR images longitudinal intrasubstance tears of the peroneus brevis tendon have a distinct appearance. The tendon assumes a C shaped configuration that partially envelops the peroneus longus tendon (Fig. 48). Partial or full substance splits within the tendon and intrasubstance high-signal intensity foci are noted on both T1 and T2 weighted images (Dunfee et al. 2002).
Figure 47. Peroneal tenosynovitis. Axial T2-weighted MR image shows a large amount of fluid within the common peroneal tendon sheath (arrow). The morphologic features of the tendons remain unchanged (Rosenberg et al. 2000).

Figure 48. Partial tear of the peroneus brevis tendon. Axial T2-weighted (TR/TE, 4000/96) MR image with fat saturation demonstrates the C-shaped configuration of the torn peroneus brevis tendon (pb) wrapping around the adjacent peroneus longus tendon (pl). L = lateral malleolus; T = tibia; A = Achilles tendon (Dunfee et al. 2002).
Acute or chronic tears of the peroneus longus tendon may be associated with peroneus brevis tendon tears at the level of the medial malleolus (*Khoury et al. 1996*).

Isolated tears of the peroneus longus tendon are more frequently seen at the level of the peroneal tubercle or cuboid tunnel (*Rademaker et al. 2000*).

Dislocation of the peroneal tendons is often clinically misdiagnosed as an ankle sprain. A flake like fracture of the distal fibular metaphysis may be present on conventional radiography, indicating an avulsed or stripped peroneal retinaculum. The mechanism of acute dislocation is a violent conduction of the peroneal muscles with secondary detachment of the peroneal tendon out of the retro-malleolar groove (*El Refaiy et al. 2008*).

MR imaging allows direct assessment of the position of the tendons relative to the fibular retro-malleolar groove (*El Refaiy et al. 2008*).

Dislocation is best demonstrated on axial images, which shows the tendon to be located anterior and lateral to the distal fibula. The tendons are often found with in a "pouch" formed on stripped-off superior peroneal retinaculum (Fig. 50). Avulsion off the distal fibula and mid-substance tears of the superior peroneal retinaculum are less frequently encountered (*Schweitzer W.E., et al. 1993*).
Figure 49. Longitudinal tear of the peroneus brevis tendon. Axial proton-density (TR/TE, 2500/10) MR image depicts split fibers of the peroneus brevis tendon (pb) on either side of the peroneus longus tendon (pl). L = lateral malleolus (Dunfee et al. 2002).

Figure 50. Dislocation of peroneal tendons. Axial proton-density-weighted MR image shows the dislocated peroneus brevis and longus tendons (arrowhead) within a "pouch" formed by the stripped-off superior peroneal retinaculum (arrows) (Rosenberg et al. 2000)
**Flexor hallucis and flexor digitorum longus injuries:-**

Injuries of the flexor hallucis longus tendon are best visualized on axial & sagittal MR imaging. *(Karasick and Schweizer 1996).*

The FHL sheath communicates with the ankle joint in 20% of patients so fluid within the tendon sheath is not necessarily pathologic, However synovial fluid surrounding an otherwise intact tendon is characteristic of chronic tenosynovitis, particularly if only a small amount of fluid is noted within the ankle joint. Isolated distal rupture of the flexor hallucis longus tendon is rare condition resulting from acute dorsiflexion laceration injuries *(Fig. 51) (Dunfee et al. 2002).*

The intersection of the FDL and the FHL tendons forms the “master knot of Henry,” which is in close proximity to the medial plantar nerve, one of the terminal branches of the posterior tibial nerve. The tendons may become entrapped at this location, referred to as jogger’s foot. The function of the FHL tendon is believed to be nonessential and surgical repair to alleviate pain, even in athletes, is recommended only when the tendon ends are easily amenable to surgery *(Dunfee et al. 2002).*
Figure 51. Flexor hallucis longus tenosynovitis. (a) Sagittal STIR MR image demonstrates abundant fluid (*) within the sheath of the flexor hallucis longus tendon (straight arrow). Edematous changes of the os trigonum, synchondrosis, and posterior talus (curved arrow) are also seen. (b) Axial T2-weighted MR image obtained in a different patient shows fluid and debris within the flexor hallucis longus tendon sheath (arrow), a finding that is consistent with tenosynovitis. Note the absence of joint fluid (Rosenberg et al. 2000)
Extensor tendon injuries

Tears of the extensor tendons are rare. When they do occur in athletes, they are generally seen in downhill runners, skiers, and soccer players. The anterior tibial tendon is the most common extensor tendon injured. MR imaging and clinical examination are the best methods in assessing anterior tibial tendon pathology (El Refaiy et al. 2008).

2] MR appearance of ligamentous injuries:

Lateral ankle sprains represent 16%–21% of all sports-related traumatic lesions. The anterior talofibular ligament is the weakest ligament and therefore the most frequently torn. There is usually a predictable pattern of injury involving the anterior talofibular ligament followed by the calcaneofibular ligament and the posterior talofibular ligament. Anatomic classification of ankle sprains is based on the number of affected ligaments. First-degree sprain is characterized by a partial or complete tear of the anterior talofibular ligament. In second degree sprain, both the anterior talofibular and calcaneofibular ligaments are either partially or completely torn. Third-degree sprain consists of injuries to the anterior talofibular, calcaneofibular, and posterior talofibular ligaments (Rosenberg et al. 2000).

The MR imaging criteria for the diagnosis of acute rupture of the lateral collateral ligament include morphologic and signal
MRI Findings Of Post Traumatic Ankle Joint

intensity alterations within and around the ligament. Injuries of the anterior talofibular ligament are easily seen on routine axial ankle MR images. Discontinuity, detachment, thickening, thinning, or irregularity of the ligament may be encountered. Heterogeneity with increased intraligamentous signal intensity on fat-suppressed or T2-weighted images is indicative of intrasubstance edema or hemorrhage. Obliteration of the fat planes around the ligament, extravasation of joint fluid into the adjacent soft tissues, and talar contusions may also be seen. Chronic tear often manifests as thickening, thinning, elongation, and wavy or irregular contour of the ligament (Fig. 52). There is usually no significant residual marrow or soft-tissue edema or hemorrhage. Decreased signal intensity in the fat abutting the ligaments with all pulse sequences is indicative of scarring or synovial proliferation (Rosenberg et al. 2000).

Edema anterior to one or more of these structures on fluid-sensitive sequences is indicative of ligamentous sprain. An enlarged ligament without surrounding edema suggests a chronic injury or scarring. When there is ligamentous disruption, fluid signal violates the normal anatomic course of the ligament, most often at its talar insertion (Zoga and Schweitzer 2003).

Lateral ankle ligament tears evolve rapidly, and a subacute tear may appear identical to a ligament sprain on MR imaging, so
secondary signs of pathology are helpful. Fluid dissecting around the distal fibula on coronal T2-weighted or STIR sequences is a strong indicator of ligamentous disruption. If the fluid tracks cephalad, the anterior talofibular ligament is likely torn, whereas caudal extension suggests a calcaneofibular ligament disruption (Zoga and Schweitzer 2003).

Injuries of the calcaneofibular ligament may be detected on routine axial ankle MR images but are more consistently visualized on coronal T1-weighted images. On sequential coronal images, the normal calcaneofibular ligament is seen in cross-section as a low-signal-intensity, homogeneous, oval structure surrounded by fat. The injured ligament is frequently thickened and heterogeneous, and the surrounding fat planes are often obliterated. Fluid within the peroneal tendon sheath can be a secondary sign of calcaneofibular ligament injury (Fig. 53 & 54) (Rosenberg et al. 2000).

Although less common than lateral ligament complex tears, deltoid ligament and syndesmotic ankle injuries do occur constituting 5% and 10% of ankle sprains, respectively (Fig. 55) (Dunfee et al. 2002).

MR imaging findings suggest that contusions of the deltoid ligament, particularly of its tibiotalar component, are frequently associated with inversion sprains. These contusions manifest as loss of the regular striations that are normally seen in the deltoid
ligament. Thus, contrary to what one would expect, the ligament demonstrates homogeneous intermediate signal intensity, a finding that is consistent with injury. Reactive fluid within the tendon sheath of the posterior tibial tendon is also frequently noted (Rosenberg et al. 2000).

The term “high ankle sprain” is often used to communicate a tibiofibular syndesmotic injury. This entity can be difficult to identify on MR because of normal fenestrations within the ligaments and the obliquity of their anatomic course (Zoga and Schweitzer 2003).

Axial MR sequences with high anatomic resolution (proton density or T1-weighted non–fat suppressed) are usually the most useful in evaluation of the syndesmotic ligaments (Zoga and Schweitzer 2003).

Helpful secondary signs of syndesmotic ligament disruption include an increased syndesmotic recess height and strain of the adjacent flexor hallucis longus muscle belly immediately posterior to the syndesmosis. Tears of one or both syndesmotic ligaments can occur in isolation without other ligamentous injury, but often the anterior syndesmotic ligament is ruptured while the posterior syndesmotic ligament remains intact. Ossification at the syndesmosis suggests a chronic or remote ligament injury (Zoga and Schweitzer 2003).
The accuracy of MR imaging in detecting injuries of the lateral collateral ligament has not yet been clearly established. The accuracy of 3D fast imaging with steady state precision in detecting acute tears of the anterior talofibular and calcaneofibular ligaments is reported to be 94.4%. MR arthrography has been shown to have an accuracy of 100% and 82% in detecting chronic anterior talofibular and calcaneofibular ligament tears, respectively, whereas conventional MR imaging has demonstrated an accuracy of 59% in diagnosing chronic lateral collateral ligament tears (*Rosenberg et al. 2000*).

Figure 52. Chronic tear of the anterior talofibular ligament. Axial T1-weighted MR image demonstrates waviness and irregularity of the anterior talofibular ligament (arrows) (*Rosenberg et al. 2000*)
Figure 53. Injury of the calcaneofibular ligament. Sequential coronal T1-weighted MR images demonstrate increased signal intensity and thickening of the calcaneofibular ligament (*) between the peroneal tendons (p) and the lateral wall of the calcaneus (c) (Rosenberg et al. 2000).

Figure 54. Chronic tear of the calcaneofibular ligament. Axial T2-weighted MR image demonstrates marked thickening and waviness of the calcaneofibular ligament (arrows) (Rosenberg et al. 2000)
Figure 55. Injury of the deltoid ligament. Coronal (a) and axial (b) T1-weighted MR images show indistinctness and swelling of the deltoid ligament as well as loss of the normal pattern of fatty striation (*), findings that are consistent with extensive partial tear. Some fibers of the tibionavicular ligament are still present (arrow in b) (Rosenberg et al. 2000).
III) Miscellaneous injuries:

1- MR Appearance of Soft Tissue Impingement Syndromes:

A- Anterolateral impingement syndrome:

It is a common cause of chronic lateral ankle pain. Injuries to the anterior talofibular and tibiofibular ligaments and an accessory fascicle of the anterior talofibular ligament have been implicated as causes of anterolateral impingement syndrome (Liu et al. 1994).

MR imaging typically depicts a "meniscoid" mass within the lateral gutter of the ankle that demonstrates low signal intensity with all pulse sequences. This soft tissue structure is best visualized on axial or coronal images when joint fluid is present within the gutter. Accurate diagnosis necessitates distinguishing this mass from the adjacent anterior talofibular ligament (Fig. 56) (Jordan et al. 2000).

B- Posterior impingement syndrome:

This is caused by the extension of the posterior intermalleolar ligament into the posterior tibio-talar joint (Cooper and Wolin 1999).

MR imaging has been reported to demonstrate a thickened hypointense band residing between the posterior tibio-fibular and talo-fibular ligament. This finding on MR imaging in the absence of other structure causes for posterior impingement syndromes, implies that the intermalleolar ligament is most likely cause (El Refaiy et al. 2008).
Figure 56. Anterolateral impingement syndrome. Sagittal STIR MR image shows a low-signal-intensity "meniscoid" mass (black arrow) related to redundant synovial tissue. Anterior tibial and talar "kissing" osteophytes are also noted (white arrows) (Rosenberg et al. 2000).
2- Sinus tarsi syndrome:

The sinus tarsi is a lateral space between the calcaneus and talus containing fat, branches of the posterior tibial and peroneal arteries and their associated nerves, and five ligaments. The sinus tarsi ligaments include the lateral, intermediate, and medial roots of the inferior extensor retinaculum, the interosseous talo calcaneal ligament, and the cervical ligament. The sinus tarsi syndrome usually reflects minor subtalar instability and is commonly seen in combination with other findings. Typically, there is lateral pain and a history of prior inversion injury. The MRI manifestations can range from edema to fibrosis to synovitis, with or without associated cystic changes (Leffler and Disler 2002).

Prior to the advent of MR imaging, arthrography of the subtalar joint and relief of pain following injection of a local anesthetic or steroid were the only techniques for diagnosing this syndrome. The MR imaging characteristics of sinus tarsi syndrome include the obliteration of fat in the sinus tarsi space. The space itself is replaced by either fluid or scar tissue, and the ligaments may be disrupted (Fig. 57) (Rosenberg et al. 2000).
Figure 57. Sinus tarsi syndrome in a patient with rheumatoid arthritis. Sagittal T1-weighted MR image shows obliteration of fat by an area of fluid-like signal intensity in the subtalar joint (*) (Rosenberg et al. 2000).

3) Tarsal Tunnel Syndrome:

Tarsal tunnel syndrome is characterized by pain and parasthesias in the plantar aspect of the foot and toes. This syndrome is most frequently unilateral, as opposed to carpal tunnel syndrome, which is typically bilateral. Nerve entrapment or compression can occur at the level of the posterior tibial nerve or its branches (medial calcaneal nerve, lateral plantar nerve, medial plantar nerve), producing different symptoms depending on the site of compression.
Intrinsic and extrinsic causes of posterior tibial nerve compression have been identified. Intrinsic lesions that often produce tarsal tunnel syndrome include accessory muscles, ganglion cysts (Fig. 58), neurogenic tumors, varicose veins, lipomas, synovial hypertrophy, and scar tissue. Foot deformities, hypertrophic and accessory muscles, accessory ossicle (os-trigonum), and excessive pronation during participation in some sports are just a few of the extrinsic causes of this syndrome. In about 50% of cases, the cause of tarsal tunnel syndrome cannot be identified. Relief of symptoms following retinacular release is frequently seen in these idiopathic cases (Rosenberg et al. 2000).

Figure 58. Tarsal tunnel syndrome secondary to ganglion cyst. Axial T2-weighted MR image reveals a ganglion cyst (*) interposed between the flexor digitorum longus (d) and flexor hallucis longus (h) tendons and abutting the adjacent neurovascular bundle (arrow) (Delfaut et al. 2003).
4) **Superficial Peroneal Nerve:**

The SPN is a branch of the common peroneal nerve. The SPN pierces the deep fascia of the leg about 12.5 cm above the tip of the lateral malleolus. Before it divides into its terminal branches roughly 6 cm above the tip of the lateral malleolus, the SPN sends out collateral motor branches to the peroneal brevis and peroneal longus muscles. It provides sensory innervations to the dorsolateral aspect of the foot and ankle (Fig. 59) (*Delfaut et al. 2003*).

The SPN is tethered as it pierces the deep fascia of the leg. It may be overstretched during inversion or plantar flexion injuries. This overstretching can be responsible for tension neuropathy, perineural fibrosis, and chronic ankle pain. If the SPN pierces the deep fascia more distally, it has limited ability to stretch or to move laterally or medially. This may be a predisposing factor to nerve injury in some individuals (*Delfaut et al. 2003*).
5) **Deep Peroneal Nerve:**

The DPN courses under the extensor retinaculum (Fig 57) between the extensor digitorum longus and extensor hallucis longus tendons and lateral to the anterior tibial artery. It divides about 1.3 cm above the ankle joint into a medial sensory branch for the first interspace and a lateral motor branch for the extensor digitorum brevis muscle (Fig. 60) (*Delfaut et al. 2003*).

There are two main compression sites of the DPN. The nerve may be compressed at the anterior tarsal tunnel, resulting in anterior tarsal tunnel syndrome. This syndrome is secondary to DPN compression at the inferior extensor retinaculum where the extensor
hallucis longus tendon crosses over it. Both sensory and motor DPN branches are involved. The pain is located at the dorsomedial aspect of the foot and is worst at rest. Weakness of the extensor digitorum brevis muscle may be evident. The DPN may also be compressed at the dorsum of the foot (Delfaut et al. 2003).

The sensory component of the DPN is located in a tight tunnel beneath the extensor hallucis brevis tendon and the deep fascia at the level of the first and second tarsometatarsal joints. Contusion, soft-tissue swelling, tight footwear or a high longitudinal arch, and naviculo-cuneiform or cunei-metatarsal osteophytes can cause acute or chronic nerve compression at this site. Sports-related injuries to the DPN have been described in skiers with tight boots and dancers in Pointe position, as well as in soccer players secondary to multiple blows to the dorsum of the foot(Delfaut et al. 2003).

Figure 60. Normal anatomy of the anterior tarsal tunnel. Shows the anterior tibial tendon (large arrowhead), extensor hallucis longus muscle and tendon (thick arrow), inferior extensor retinaculum (small arrowheads), dorsalis pedis artery, and lateral and medial terminal branches of the DPN (thin arrows) tenosynovitis of the extensor digitorum longus tendon (arrowhead) (Delfaut et al. 2003).
6) MRI Appearance of Plantar Fasciitis:

Plantar fasciitis is most likely related to repetitive trauma and mechanical stress, which produce micro tears and inflammation of the fascia and perifascial soft tissues. Plantar fasciitis is common in runners and obese patients. Inflammation of the plantar fascia can produce heel pain even in the absence of a traumatic event. Patients with plantar fasciitis present with pain at the origin of the plantar fascia. The pain is exacerbated by dorsiflexion of the toes and is more severe in the morning (Rosenberg et al. 2000).

When inflammatory changes take place, it becomes thickened (up to 7–8 mm) and demonstrates intermediate signal intensity on T1-weighted and proton-density–weighted images and hyperintensity on T2-weighted images. These changes are most prominent in the proximal portion of the plantar fascia at or near its insertion on the calcaneus. Signal intensity changes may also be present in the subcutaneous fat, in the deep soft tissues, and in the calcaneus near the fascial insertion. Thickening is often fusiform, in contrast with plantar fibromatosis, which demonstrates focal, nodular thickening. Discontinuity of the fibers of the plantar fascia, often with focal edema and hemorrhage, is noted when a tear of the fascia is present (Rosenberg et al. 2000).
PATIENTS AND METHODS
PATIENTS AND METHODS

MRI examinations

Patients were examined with closed superconductive 1.5 T Philips magnet using sense ankle foot coil.

Time of Study:

The study was performed starting from 2008 to 2012.

Subjects:

All patients (40) included in our study were suffering from trauma to ankle (old or recent). They were (12) women and (28) men. Their age ranged from 17 to 55 years with an average of 36 years. The left ankle was incriminated in 21 cases, the right one in 19 cases.

All the patients presented by ankle pain, sprain and swelling, and limited movement. 5 cases presented with pain since long time which increased on standing, walking or working.
Procedures:

1. All the patients underwent plain X ray of the painful ankle joint.
2. The entire patients were examined after explaining the procedure to them and they should not move their ankle during the examination.
3. All the patients were examined by sense foot-ankle coil, after lying supine with the foot about 20 degrees planter flexion (for better visualization of the calcaneo-fibular ligament and peroneal tendons). Pads were applied to support and fix the ankle position.
4. The examinations were done by taking different planes (axial, coronal and sagittal) at different pulse sequences.
   i. The axial plane to visualize anterior tibiofibular, anterior talofibular as well as flexors and extensor tendons.
   ii. The coronal plane for visualization medial collateral ligament, CFL, talar dome marrow abnormalities, cartilage and sinus tarsi ligaments.
   iii. The sagittal plane can visualize the Achilles tendon, talus, calcaneus and the tarsal tunnel as well as sinus tarsi ligaments.
   • On starting the examination, the machine takes three planes (coronal, axial and sagittal scout).
The Protocol Used In Our Examinations Is As Follows:

- T1 WIs, T2 WIs and fat suppression sequences.

**Routine protocol of examination**

<table>
<thead>
<tr>
<th>Pulse sequence</th>
<th>TR (msec)</th>
<th>TE (msec)</th>
<th>Gap (mm)</th>
<th>Slice thickness (mm)</th>
<th>FOV (mm)</th>
<th>Matrix</th>
</tr>
</thead>
<tbody>
<tr>
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<td>685</td>
<td>23</td>
<td>3</td>
<td>3</td>
<td>150-180</td>
<td>256X224</td>
</tr>
<tr>
<td>SE T2 WEIGHTED</td>
<td>3000</td>
<td>100</td>
<td>3</td>
<td>3</td>
<td>150-180</td>
<td>256X224</td>
</tr>
<tr>
<td>SE PD SPIR WEIGHTED</td>
<td>2000</td>
<td>25</td>
<td>3</td>
<td>3</td>
<td>150-180</td>
<td>256X224</td>
</tr>
<tr>
<td>SE STIR WEIGHTED</td>
<td>2650</td>
<td>30</td>
<td>3</td>
<td>3</td>
<td>150-180</td>
<td>256X224</td>
</tr>
</tbody>
</table>

Note SE= Spin echo, TE= echo time, TR= Repetition time, FOV=Field of view
PHYSICAL

CONSIDERATION
Physical consideration

Pulse sequences:

Variable pulse sequences are available each have its strengths and weakness that must be considered in the design of any imaging protocol (Mirowitz 1991).

The number of pulse sequences and combinations (‘hybrid techniques’) is almost infinitive: in musculoskeletal MR, the most commonly used sequences include conventional spin echo (SE) for T1-weighting, turbo SE (TSE) sequences for T2-weighting and gradient echo (GRE) sequences (Vanhoenacker et al. 2007).

SE T1-WI is used for anatomic detail, and as an adjunct in the evaluation of the osseous structures (Vanhoenacker et al. 2007).

The T1 relaxation time (longitudinal) is used to describe the return of protons back to equilibrium after application and removal of the radiofrequency pulse. T2 relaxation time (transverse) is used to describe the associated loss of coherence or phase between individual protons immediately after the application of the radiofrequency pulse. A variety of radiofrequency pulse sequences can be used to enhance the differences in T1 and T2, thus providing the necessary image contrast (Greenspan 2004).

Spin echo (SE) short repetition times (TR) (800 msec or less) and short echo delay times (TE) (40 msec or less) pulse sequences (or T1) provide good anatomic detail. Long TR (2000 msec or more) and long TE (60 msec or more) pulse sequences (or T2), however, provide good contrast, sufficient for evaluation of pathologic processes. Intermediate TR (1000
msec or more) and short TE (30 msec or less) sequences are known as proton or spin density images. They represent a mixture of T1 and T2 weighting, and although they provide good anatomic details, the tissue contrast is somewhat impaired. IR sequences can be combined with multiplanar imaging to shorten scan time (Greenspan 2004).

TSE sequence has replaced conventional SE for T2-weighting (due to its relatively long acquisition times). However, because of image blurring, TSE sequences are not recommended for proton density imaging. Blurring can be reduced by increasing TE, decreasing inter-echo time, echo train length (ETL), and by increasing matrix. TSE sequences are less susceptible to field inhomogeneity than SE sequences. Therefore, when metallic artifacts are present, such as in post-surgical patients, TSE sequences are preferred over SE and GRE (Vanhoenacker et al. 2007).

The use of an intermediate TE in FS T2-weighted images has an additional value in demonstrating underlying cartilage lesions (Vanhoenacker et al. 2007).

When using short TE in T1-weighted or PD images, one should take the magic angle phenomenon into account, a source of false positive MR findings (Vanhoenacker et al. 2007).
**Physical consideration**

**Magic angle effect:**

- All tendons, except for Achilles, are susceptible to magic angle effect as they curve around ankle joint.
- Magic angle effect results in increased tendon signal on low TE sequences.
- Magic angle effect can be avoided by 1) Plantar flexion foot, patient supine 2) Imaging patient prone 3) Correlating tendon’s signal alterations on high TE sequences (*Manaster et al. 2007*).

Furthermore, a pulse sequence is always a compromise between acquisition time, contrast, detail or signal-to-noise ratio (SNR). SNR is highest in TSE and decreases respectively in SE and GRE sequences (*Vanhoenacker et al. 2007*).

Both T2-WI with (spectral) FS and STIR images are most sensitive to bone marrow and soft tissue edema or joint effusion (*Vanhoenacker et al. 2007*).

Fat suppression technique is commonly used in MRI to detect adipose tissue or suppress the signal from adipose tissue. There are three methods to achieve this goal: frequency-selective (chemical) fat saturation, inversion–recovery imaging, and opposed-phase imaging (*Greenspan 2004*).
### Table 3: Fat Suppression Techniques (Greenspan 2004).

<table>
<thead>
<tr>
<th>Methods</th>
<th>Advantages</th>
<th>Disadvantages</th>
</tr>
</thead>
</table>
| Frequency-selective (chemical) fat saturation | ▪ Lipid-specific  
▪ Signal in non fat tissue unaffected  
▪ Excellent imaging of small anatomic detail  
▪ Can be used with any imaging sequence | ▪ Occasionally inadequate fat suppression.  
▪ Water signal may be suppressed.  
▪ Inhomogeneities in areas of sharp variations in anatomic structures.  
▪ Increased imaging time |
| Inversion recovery (STIR)        | ▪ Excellent contrast resolution  
▪ Very good for tumor detection  
▪ Can be used with low-field-strength magnets | ▪ Low signal-to-noise ratio  
▪ Tissue with a short T1 and long T1 may produce the same signal intensity  
▪ Signal from mucoid tissue, hemorrhage, and proteinaceous fluid may be suppressed |
| Opposed-phase                    | ▪ Ability to demonstrate small amounts of lipid tissue  
▪ Simple, fast, and available on every MR imaging system | ▪ Fat signal only partially suppressed  
▪ Suppresses water signal  
▪ Difficult to detect small tumors imbedded in fat  
▪ In post gadolinium studies, contrast material may be undetected |

MRI examination showed intraosseous areas, hyperintense on T2-weighted/STIR images and (to a lesser degree) hypo-intense on T1-images, in acutely injured joints with no abnormalities on plain radiographs. The use of an intermediate TE in FS T2-weighted images has an additional value in demonstrating underlying cartilage lesions (Vanhoenacker et al. 2007).
RESULTS
This study was performed on 40 patients of different post-traumatic status we had 28 males and 12 females ranging age from 17 to 55 years with mean of 35 years old.

Most of the patients were complaining of different types of trauma, and some had recent and some had old trauma reaching up to months.

The following are the findings found in the examined cases and also the net results are seen:

The number of patients having osseous injuries are 23 patients representing 57.5% of the patients.

Non osseous injuries are divided into tendinous, ligamentous and miscellaneous injuries.

The numbers of patients having ligamentous injuries are 17 patients representing 42.5% of the cases.

The numbers of patients having tendinous injuries are 24 patients representing 60% of the cases.
The numbers of patients having miscellaneous injuries are 31 patients representing 77.5% of the cases.

The numbers of patients having Flexor hallucis longus and flexor digitorum longus injury are 15 patients representing 37.5% of the cases.

The numbers of patients having effusion are 24 patients representing 60% of the patients.

The number of patients having lateral ankle sprain are 15 patients representing 37.5% of the patients.

The number of patients having medial ankle sprain are 3 patients representing 7.5% of the patients.

The number of patients having posterior tibial tendon injury are 4 patients representing 10% of the patients.

The numbers of patients having Achilles tendon injuries are 7 patients representing 17.5% of the patients.
The numbers of patients having peroneal tendon injuries are 4 patients representing 10% of the patients.

The number of patients having peripheral nerve entrapment are 4 patients representing 10% of the patients.

The number of patients having sinus tarsi syndrome are 2 patients representing 5% of the patients.

The previous findings are shown in the table below:
### Table 4: Percent of different types of ankle injuries.

<table>
<thead>
<tr>
<th>Factor</th>
<th>N</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Osseous injuries</td>
<td>23</td>
<td>57.5</td>
</tr>
<tr>
<td>Flexor hallucis &amp; flexor digitorum</td>
<td>15</td>
<td>37.5</td>
</tr>
<tr>
<td>Effusion</td>
<td>24</td>
<td>60</td>
</tr>
<tr>
<td>Lateral ankle sprain &amp; sinus tarsi syndrome</td>
<td>15</td>
<td>37.5</td>
</tr>
<tr>
<td>Medial sprain</td>
<td>3</td>
<td>7.5</td>
</tr>
<tr>
<td>Posterior tibial tendon</td>
<td>4</td>
<td>10</td>
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<tr>
<td>Achilles tendon</td>
<td>7</td>
<td>17.5</td>
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<tr>
<td>Peroneal tendon</td>
<td>4</td>
<td>10</td>
</tr>
<tr>
<td>Peripheral nerve entrapment</td>
<td>4</td>
<td>10</td>
</tr>
<tr>
<td>Sinus tarsi</td>
<td>2</td>
<td>5</td>
</tr>
</tbody>
</table>

I) Osseous injuries are 23 patients further divided into 15 patients had bone contusion, 7 cases had acute fracture, and 5 cases had osteochondral fracture.

<table>
<thead>
<tr>
<th>Bone Contusion</th>
<th>Acute Fracture</th>
<th>Osteochondral Fracture</th>
</tr>
</thead>
<tbody>
<tr>
<td>11</td>
<td>7</td>
<td>5</td>
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</table>
Table 5: Osseous fractures.

<table>
<thead>
<tr>
<th>Case</th>
<th>Fracture</th>
<th>Lateral collateral injury</th>
<th>Medial collateral injury</th>
<th>Tendon injury</th>
<th>Effusion</th>
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<td>1</td>
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<td>+</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
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<tr>
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<td>Calcaneus</td>
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<td>-</td>
<td>+</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>3</td>
<td>Tibia</td>
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<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>4</td>
<td>Tibia</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
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<tr>
<td>5</td>
<td>Tibia</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>6</td>
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<td>-</td>
<td>+</td>
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<tr>
<td>7</td>
<td>Tibia, fibula&amp; talus</td>
<td>+</td>
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<td>-</td>
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</tr>
</tbody>
</table>

II) flexor hallucis longus and flexor digitorum longus were 15 cases all of them were tenosynovitis

III) Lateral ankle sprain and sinus tarsi syndrome are totally 16 patients, 15 cases lateral ankle sprain and 2 cases sinus tarsi syndrome.

<table>
<thead>
<tr>
<th>Lateral Ankle Sprain</th>
<th>Sinus Tarsi Syndrome</th>
</tr>
</thead>
<tbody>
<tr>
<td>15</td>
<td>2</td>
</tr>
</tbody>
</table>

Lateral collateral sprain are totally 15 patients, 8 cases are grade I and 7 cases are grade II.

<table>
<thead>
<tr>
<th>Grade I</th>
<th>Grade II</th>
</tr>
</thead>
<tbody>
<tr>
<td>8</td>
<td>7</td>
</tr>
</tbody>
</table>
Table 6: Grade I lateral collateral sprain.

<table>
<thead>
<tr>
<th>Grade I Cases</th>
<th>Medial collateral injury</th>
<th>Tendon Injury</th>
<th>Fracture</th>
<th>Osteochondral injury</th>
<th>effusion</th>
<th>Subcutaneous edema</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>2</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>3</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>4</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>5</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>6</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>7</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>8</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
</tr>
</tbody>
</table>

Table 7: Grade II lateral collateral sprain.

<table>
<thead>
<tr>
<th>Grade II Cases</th>
<th>Medial collateral injury</th>
<th>Tendon Injury</th>
<th>Fracture</th>
<th>Osteochondral injury</th>
<th>effusion</th>
<th>Subcutaneous edema</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>3</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>4</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>5</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>6</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>7</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>+</td>
</tr>
</tbody>
</table>
IV) Medial collateral sprain are 3 cases.

Table 8: Medial collateral sprain.

<table>
<thead>
<tr>
<th>Cases</th>
<th>Lateral collateral injury</th>
<th>Tendon Injury</th>
<th>Fracture</th>
<th>Osteochondral injury</th>
<th>effusion</th>
<th>Subcutaneous edema</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>3</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>-</td>
</tr>
</tbody>
</table>

V) Posterior tibial tendon injury are 4 patients, 1 case type I partial tear, 3 cases are tenosynovitis.

<table>
<thead>
<tr>
<th>Type I partial tear</th>
<th>Tenosynovitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>3</td>
</tr>
</tbody>
</table>

VI) Achilles Tendon injuries are 7 cases, 3 cases are tendinosis, 2 cases are partial tear and 2 cases are complete tear.

<table>
<thead>
<tr>
<th>Tendinosis</th>
<th>Partial tear</th>
<th>Complete tear</th>
</tr>
</thead>
<tbody>
<tr>
<td>3</td>
<td>2</td>
<td>2</td>
</tr>
</tbody>
</table>
Table 9: Achilles tendon injury.

<table>
<thead>
<tr>
<th>Cases</th>
<th>Achilles tendon</th>
<th>ligaments injury</th>
<th>Tendon Injury</th>
<th>Osseous injury</th>
<th>effusion</th>
<th>Subcutaneous edema</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Tendinosis</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2</td>
<td>Tendinosis</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>3</td>
<td>Tendinosis</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>4</td>
<td>Partial tear</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>5</td>
<td>Partial tear</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>6</td>
<td>Complete tear</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>7</td>
<td>Complete tear</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
</tr>
</tbody>
</table>

VII) Peroneal tendon injuries were 4 cases, 2 of them were tenosynovitis and the other 2 were partial tear.

<table>
<thead>
<tr>
<th>Tenosynovitis</th>
<th>Partial Tear</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>2</td>
</tr>
</tbody>
</table>

VIII) Peripheral nerve entrapment was 4 cases, 3 cases are tarsal tunnel syndrome and one case lateral plantar nerve compromise.

XI) Subcutaneous edema was 10 cases.

X) Miscellaneous injuries are 31 cases, 24 cases are joint effusion, 10 cases are subcutaneous edema, 3 cases Haglund deformity, one case fibrous tarsal coalition and one case vascular malformation.
Table 10: Sensitivity of MRI pulse sequences in osseous injury.

<table>
<thead>
<tr>
<th>Cases</th>
<th>Bone contusion (15)</th>
<th>Fractures (7)</th>
<th>Osteochondral lesion (5)</th>
</tr>
</thead>
<tbody>
<tr>
<td>T1</td>
<td>7</td>
<td>7</td>
<td>5</td>
</tr>
<tr>
<td>T2</td>
<td>8</td>
<td>7</td>
<td>5</td>
</tr>
<tr>
<td>FS</td>
<td>15</td>
<td>5</td>
<td>5</td>
</tr>
</tbody>
</table>

Table 11: Sensitivity of MRI pulse sequences in grade I lateral collateral sprain.

<table>
<thead>
<tr>
<th>Cases</th>
<th>Grade I lateral collateral sprain</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>8</td>
</tr>
<tr>
<td>T1</td>
<td>-</td>
</tr>
<tr>
<td>T2</td>
<td>4</td>
</tr>
<tr>
<td>FS</td>
<td>8</td>
</tr>
</tbody>
</table>

Table 12: Sensitivity of MRI pulse sequences in grade II lateral collateral sprain.

<table>
<thead>
<tr>
<th>Cases</th>
<th>Grade II lateral collateral sprain</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>7</td>
</tr>
<tr>
<td>T1</td>
<td>2</td>
</tr>
<tr>
<td>T2</td>
<td>4</td>
</tr>
<tr>
<td>FS</td>
<td>7</td>
</tr>
</tbody>
</table>
### Results

Table 13: Sensitivity of MRI pulse sequences in medial collateral sprain.

<table>
<thead>
<tr>
<th></th>
<th>Medial collateral sprain</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cases</td>
<td>3</td>
</tr>
<tr>
<td>T1</td>
<td>-</td>
</tr>
<tr>
<td>T2</td>
<td>1</td>
</tr>
<tr>
<td>FS</td>
<td>3</td>
</tr>
</tbody>
</table>

Table 14: Sensitivity of MRI pulse sequences in Achilles tendon injury.

<table>
<thead>
<tr>
<th></th>
<th>Achilles Tendon injury</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cases</td>
<td>Tendinos (3)</td>
</tr>
<tr>
<td>T1</td>
<td>3</td>
</tr>
<tr>
<td>T2</td>
<td>3</td>
</tr>
<tr>
<td>FS</td>
<td>3</td>
</tr>
</tbody>
</table>

Table 15: Sensitivity of MRI pulse sequences in Sinus Tarsi syndrome.

<table>
<thead>
<tr>
<th></th>
<th>Sinus tarsi</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cases</td>
<td>3</td>
</tr>
<tr>
<td>T1</td>
<td>1</td>
</tr>
<tr>
<td>T2</td>
<td>1</td>
</tr>
<tr>
<td>FS</td>
<td>3</td>
</tr>
</tbody>
</table>
CASE
PRESENTATION
CASE 1

Male patient 40 years old had fallen from height.

Complaint:
Pain and swelling along the ankle joint and inability to walk.

Diagnosis:
- Comminuted fracture distal tibia involving the distal articular surface.

Findings:
- Comminuted fracture of the distal end of tibia seen involving the posterior and medial cortices of the shaft as well as the articular surface. The fractures lines display low signal in T1 WI and T2 WI, while in STIR images edema masks the fracture lines.
- PD with fat suppression sequences showed fracture lines better than STIR because of less bright edema that mask fracture lines.
Case Presentation

Figure 61 (A): Sagittal T1 WI

Figure 61 (B): Sagittal STIR.
Figure 61(C): axial T1 WI

Figure 61 (D): Axial T2 WI

Figure 61 (E): axial PD SPIR
CASE 2

Male patient 37 years old exposed to trauma to the posterior aspect of the ankle.

**Complaint:**
Sever Pain and swelling at the posterior aspect of the left ankle joint with inability to walk.

**Diagnosis:**
- Type 2 complete rupture of the Achilles tendon.

**Findings:**
- Loss of continuity of Achilles tendon with complete tear at a point 3cm above the os calcis. The proximal tendon fibers are seen retracted with both ends are seen frayed and swollen. A 2 cm gap is seen between the two ends where mixed fat and edema signals are seen interposed at T1 WI and better appreciated at T2 WI.
- The STIR sequence delineates the complete tear better than T1 and T2 WIs.
- PD with fat suppression better delineates the intrinsic bright signal within the retracted fibers of the tendon.
Case Presentation

Figure 62 (A): axial T1 WI.

Figure 62 (B): axial T2 WI.

Figure 62 (C): axial STIR.
Figure 62 (D): Sagittal T1 WI.

Figure 62 (E): Sagittal T2 WI.

Figure 62 (F): Sagittal PD SPIR WI.
CASE 3

Male patient 48 years old was walking and fall on his foot.

Complaint:
Sever Pain and swelling around ankle joint with pain on walk or move his ankle joint.

Diagnosis:
- Acute osteochondral lesion of the talus with reactive marrow edema of the talus and calcaneus.

Findings:
- Osteochondral lesion is seen involving the inferior surface of the talar bone measuring 1x0.5 cm, displaying low signal in T1 WI.
- The STIR sequence better delineates the lesion and showing reactive marrow edema involving the body of the talus and calcaneus.
- The gap between the osteochondral lesion and the talus appears hypointense in STIR sequence denoting stability of the lesion.
Figure 63 (A): Coronal T1 WI.

Figure 63 (B): Coronal STIR.
CASE 4

Female patient 35 years old exposed to inversion injury during playing tennis had inversion type of trauma.

**Complaint:**
Pain and swelling around the right ankle joint with inability to walk.

**Diagnosis:**
- Grade II lateral collateral ligament sprain.

**Findings:**
- In T1 WI, the ATFL appears intact however in T2 WI it is surrounded by hyperintense signal.
- In Fat suppression sequence, the ATFL is torn with interrupted fibers continuity and surrounded by fluid signal.
Case Presentation

Figure 64 (A): Axial T1 WI

Figure 64 (B): Axial T2 WI

Figure 64 (C): Axial PD SPIR
CASE 5

Male patient 33 years old exposed to inversion injury during walking.

**Complaint:**
Pain and swelling along the lateral aspect of left ankle joint with painful movement on walking.

**Diagnosis:**
- Grade I lateral collateral ligament sprain.

**Findings:**
- Ill definition of the ATFL in the T1 WI that better delineated, stretched and surrounded with Fluid signal in the PD fat suppression.
Figure 65(A): Coronal T1 WI.

Figure 65(A): Coronal PD SPIR WI.
CASE 6

Male patient 28 years old was walking and fall on her foot.

Complaint:
Sever Pain and swelling around ankle joint with inability to walk or move her ankle joint.

Diagnosis:
- Tenosynovitis of the deep flexor tendons.

Findings:
- The flexor tendons around ankle joint appear normal in T1 WI and surrounded by fluid signals in T2 WI. The fluid signals appear exaggerated in fat suppression sequence.
Case Presentation

Figure 66 (A): Axial T1 WI.

Figure 66 (B): Axial T2 WI.

Figure 66 (C): Axial PD Fat suppression.
Male patient 60 years old with repeated trauma to back of the ankle.

**Complaint:**
Sever Pain and swelling around right ankle joint with pain on walk.

**Diagnosis:**
- Focal tendinosis of the distal 3cm of the Achilles tendon with threatened partial thickness tear.

**Findings:**
- Fusiform swelling of the distal 3cm of the Achilles tendon with anterior convex border in axial view.
- T1 WI showed intermediate signal. The altered signals become more conspicuous in T2 WI.
- The fat suppression sequences showed altered signal more evidently and also demonstrates that intrinsic bright signal partially interrupting the anterior and posterior peritendinous outline so raising the grade from focal tendinosis to threatened partial thickness tear. The PD SPIR also demonstrate intrinsic bright signal within the fibers itself.
Case Presentation

Figure 67 (A): Axial T1 WI.

Figure 67 (B): Axial T2WI.

Figure 67 (C): Axial PD SPIR.
Figure 67 (D): Sagittal T1WI.

Figure 67 (E): Sagittal STIR.
CASE 8

Male patient 35 years old was subjected to repeated trauma to right ankle.

**Complaint:**

Pain and swelling around right ankle joint with pain on walk.

**Diagnosis:**

Focal tendinosis of Achilles tendon.

**Findings:**

- Focal thickening of the distal most insertional fibers of the Achilles tendon with convex anterior border in axial images. It showed intrinsic bright signal in the T1 WI that become less evident in the T2 WI however the Fat suppression sequence makes the altered signal more conspicuous yet no complete tear or sizeable peri-tendinous fluid signal noted.
Figure 68 (A): Axial T1 WI.

Figure 68 (B): Axial T2 WI.

Figure 68 (C): Axial STIR.
Case Presentation

Figure 68 (D): Sagittal T1 WI.

Figure 68 (E): Sagittal T2 WI.

Figure 68 (F): Sagittal STIR.
Case 9

Female patient 45 years old was subjected to repeated trauma to left ankle.

Complaint:

Pain and swelling around left heel with inability to step of her foot.

Diagnosis:

Haglund’s deformity (Focal insertional tendinosis of the Achilles tendon, abnormal marrow signal of the calcaneus and pre Achilles and retrocalcaneal bursitis).

Findings:

- Focal thickening of the distal most insertional fibers of the Achilles tendon and showing intrinsic bright signals in the T1 and T2 WI that become more evident in the STIR sequence.
- Marrow edema signal of the calcaneal tuberosity is seen. It displays low signal in T1 WI that become less conspicuous in T2 WI and STIR sequence make the bone marrow edema flaring up as increased signal.
- The pre Achilles and retro calcaneal bursitis appears hypointense in T1 WI that appears bright in T2 WI. The size and signal of the bursitis is mostly appreciated in STIR sequence.
Case Presentation

Figure 69 (A): Sagittal T1 WI.

Figure 69 (B): Sagittal T2 WI.

Figure 69 (C): Sagittal STIR.
CASE 10

Male patient 32 years old was subjected to trauma one year ago and diagnosed as grade II ATFL injury.

Complaint:
Pain along the lateral aspect of the ankle.

Diagnosis:
Sinus tarsi syndrome.

Findings:
- Effacement of sinus tarsi fat with Poorly defined margins of interosseous and cervical Ligaments on T1 WI.
- Fluid signal intensity of the sinus tarsi with increased signal intensity of the sinus tarsi ligaments and hyperintense bone marrow edema of the roof of the sinus tarsi are well appreciated at fat suppression sequences.
Case Presentation

Figure 70 (A&B): Sagittal T1 WI & STIR.

Figure 70 (C&D): Coronal T1 WI & STIR.
DISCUSSION
Discussion

Ankle joint is frequently subjected to trauma either acute or chronic. The trauma usually occurs in athletes. Before the invention of the recent radiological modalities the diagnosis of the ankle injuries depends on the clinical examination (Berquist et al. 1990). After the development of the different radiological modalities as plain x ray, Arthrography, CT and MRI the evaluation became indispensable in any and every ankle to reach the most accurate diagnosis and so the proper treatment either conservative or surgical (Berquist et al. 2001).

Many ankle injuries were undiagnosed by conventional radiology and needs further evaluation to diagnose ligamentous, tendinous and muscle injuries as well as osseous lesions as stress fractures and avascular necrosis (Berquist et al. 2001).

The ultrasound examination role in ankle joint injuries has a limited role due to the inability of the sound waves to penetrate the bone. The bones of the lower extremity are deep except for the subcutaneous portion of the tibia allowing easier the examination of the soft tissue structures as the Achilles tendon, however ultrasound has some limitations as it is operator dependant technique, lacking proper contrast resolution and complex anatomy of the ankle region.
makes the examination difficult. CT provides excellent bone details; it may be valuable in imaging of the subtalar joint. CT is capable of evaluating the bones and joints to establish the diagnosis of bone injury however, ligamentous, tendinous, stress fracture and marrow changes could not be diagnosed by CT (Berquist et al. 2001).

MR imaging is the modality of choice for optimal detection of most soft-tissue disorders of the tendons, ligaments, and other soft-tissue structures of the ankle. This modality is also valuable in the early detection and assessment of a variety of osseous abnormalities seen in this anatomic location (El Refaiy et al. 2008).

Is this work we study the cases to highlight the most accurate and beneficial pulse sequences for the evaluation of bone and soft tissue injuries of the ankle region.

We classify the cases into 4 groups according to the tissue injured:

- **Group I** osseous injuries.
- **Group II** tendinous injuries.
- **Group III** ligamentous injuries.
- **Group IV** miscellaneous injuries.
In group I

23 patients had osseous injury, 11 patients had bone contusion, 7 cases had acute fracture, and 5 cases had osteochondral fracture due to overlap.

In cases with bone fractures, associated lateral collateral sprain was noticed in 3 cases while medial collateral sprain occurred in 2 cases. Associated tendon injuries was noticed in 4 cases while effusion was associated with three cases.

In bone contusion, T1 WI is less sensitive in demonstrating marrow edema than T2 WI and PD sequence while fat suppression is the best sequence.

*Vanhoenacker et al. 2007* found that MRI examination showed bone bruise as intraosseous areas, hyperintense on T2-weighted/STIR images and (to a lesser degree) hypo-intense on T1-images, in acutely injured joints with no abnormalities on plain radiographs. The use of an intermediate TE in FS T2-weighted images has an additional value in demonstrating underlying cartilage lesions.

Both T2-WI with FS and STIR images are most sensitive to bone marrow and soft tissue edema or joint effusion (*Vanhoenacker et al. 2007*).
Although bone marrow edema can be observed on T1-weighted magnetic resonance (MR) images, more sensitive sequences, such as short tau inversion-recovery (STIR), and T2-weighted fat-suppressed turbo SE sequences, are commonly used for detecting bone marrow abnormalities (*Schmid et al. 2002*).

STIR images and T1-weighted contrast-enhanced fat-suppressed MR images demonstrate almost identical imaging patterns, and diagnoses determined with these findings show little difference (*Schmid et al. 2002*).

We found that fat suppression sequences are less sensitive in demonstrating the acute fractures than T1 and T2 WI because marked edema partially masks the fracture line.

Osteochondral lesion are equally appreciated at all sequences however T2 and fat suppression sequences are the best sequences in showing the interface between normal bone and osteochondral fragment. The coronal plane is the best one in demonstrating the talus injury.

(*Rosenberg et al. 2000*) found that the ideal pulse sequence for identifying osteochondral lesions is still a subject of controversy, but recent reports based on knee imaging seem to favor thin-section 3D Fourier transform spoiled gradient-echo techniques with fat saturation and, more recently, fat-saturated fast spin echo.
techniques. Some authors have also recommended intraarticular injection of gadolinium based contrast material for improved visualization of osteochondral lesions.

Sonin et al. 2010 found that FS sequences overestimate size of lesion by including adjacent marrow edema in lesion dimensions. Coronal FS PD FSE images are the best to define morphology of overlying chondral surface.

**In Group II**

We found that tenosynovitis are best appreciated in T2 and fat suppression sequences while the later may be over estimating the fluid signal.

Tenosynovitis will be seen as edema around the tendon seen as high signal on T2 weighted images. Using conventional MRI it is not possible to differentiate the synovial thickening from free fluid. If intravenous Gadolinium DTPA is given, the increased signal that occurs within most areas of synovitis will assist (Vanhoenacker et al. 2007).

Fat suppressed T2-weighted images are very effective in demonstrating paratenonitis. MRI cannot identify the distinction between synovitis and fluid within a tendon sheath without the use of intravenous contrast (Vanhoenacker et al. 2007).
Partial rupture manifests on T1-weighted and proton-density-weighted images and occasionally on T2-weighted images as an area within the substance of the tendon having a signal intensity similar to that seen in advanced tendinosis (Vanhoenacker et al. 2007).

In agreement with (Dunfee et al. 2002) STIR, and fat-suppressed T2-weighted sequences are very sensitive for noting abnormal water content within the tendon or adjacent soft tissues and bone in the setting of trauma.

7 patients having Achilles tendon injuries (3 cases tendinosis, 2 case partial tear and 2 case complete tear) are associated with one case of ligamentous injury, one case of other tendinous injury and 4 cases of osseous injury.

Vanhoenacker et al 2007 found that Fat suppressed T2-weighted imaging useful for assessment of paratenonitis. In one case of Achilles tendinosis, the fat suppression sequence demonstrates extension of the intra tendinous abnormal signal to the paratenon, finding that change the diagnosis to partial tendon tear.

The fat suppression sequences is the best one in demonstrating the partial tear while in complete tear all sequences can detect it. In agreement with (Sonin et al. 2010) the fat suppression sequence is the best in demonstrating the tear and the gap and PD FS is better.
than STIR in demonstrating the fraying in the tendon fibers and showed the intrinsic bright signal within.

**In Group III**

In our study 15 patients have lateral collateral sprain representing 37.5% of the ankle sprains.

Lateral ankle sprains represent 21% of all sports-related traumatic lesions *(Rosenberg et al. 2000)*. *Sonin et al. 2010* found that 85% of ankle sprains occur laterally.

Lateral ankle sprains are the most common athletic injury. The lateral ankle sprain has been reported to represent up to 45% of basketball injuries and 31% of soccer injuries *(Lassiter et al. 1989 & Dunfee et al. 2002)*. In addition, it has been reported that 17% to 25% of sports injury time-loss is directly related to ankle sprains *(Safran et al. 1999 & Dunfee et al. 2002)*. Most (up to 85%) sprains are caused by inversion forces during plantar flexion with damage to the lateral ligaments *(Dunfee et al. 2002)*.

We found 8 cases of grade I lateral collateral sprain and associated with one case of medial collateral injury. Tendon injuries were elicited in 5 cases while and osseous injury in 2 cases.
In our study 7 cases grade II lateral collateral sprain are detected. Tendon injuries could be identified in four cases and osseous injury in 2 cases.

We found 3 cases of medial collateral sprain representing 7.5% of the ankle sprains and are associated with one case of lateral collateral sprain, one case of tendon injury and two cases of osseous injury.

*Sonin et al. 2010* found that deltoid ligament sprain represents 10 to 15% of all ankle sprains and less frequent than lateral injury.

We found that the fat suppression sequences are the best sequence in demonstrating the ligamentous injury in agreement with *Sonin et al. 2010*. Two cases of the grade II lateral collateral sprain were diagnosed as grade I in T1 and T2 WI however the usage of the fat suppression sequences is changing the diagnosis to grade II as they showed torn ATFL.

Axial T2WI using FS PD or T2 FSE best to visualize status of ligament morphology and adjacent hyperintense edema (*Sonin et al. 2010*).

**IN GROUP IV**

In this group we found two cases of sinus tarsi syndrome and four cases of peripheral nerve entrapment.
DISCUSSION

Impingement syndromes of the ankle are usually a clinical diagnosis, and complex radiologic evaluation is not always necessary (Robinson and White 2002).

We found that diagnosis of the sinus tarsi syndrome best appreciated in T1 WI as obliterated fat planes and in fat suppression sequences as fluid signal within sinus tarsi, edema of sinus roof and intrinsic bright signal in the sinus tarsi ligaments.

Stoller et al. 2004 found that the best sequences in the diagnosis of the sinus tarsi are sagittal T1 WI and FS PD FSE.

The best sequences in case of tarsal tunnel syndrome are the axial, coronal and sagittal T1 and FS PD FSE (Stoller et al. 2004).
SUMMARY AND

CONCLUSION
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Foot and ankle structures bear massive amounts of force during athletic activities and are naturally susceptible to a vast and ever-expanding array of injuries.

The ankle is one of the most frequently injured joints. In order to a better understanding of these lesions, a classification based on the anatomic origin is outlined. In this study, the spectrum of injuries has been classified into: (1) osseous lesions, (2) ligamentous injuries, (3) tendinous lesions, (4) miscellaneous injuries.

It is easiest to organize the approach to analyze pathology at the ankle by considering compartmental anatomy. The compartments can simply be divided into the anterior, posterior, lateral, and medial soft tissue compartments.

MR imaging has become the modality of choice in the evaluation of most of these lesions.

Magnetic resonance imaging is playing an increasingly important role in evaluation of the injured foot and ankle. Magnetic resonance imaging allows accurate detection of bony abnormalities, such as stress fractures, and soft-tissue abnormalities, including ligament tears, tendon tears, and tendinopathy. The interpreter of magnetic resonance images should systematically review the
images, noting normal structures and accounting for changes in soft-tissue and bony signal.

After ankle trauma, the patients present with pain so in order to shorten the examination time, we should choose the most beneficial and informative MRI sequences.

When imaging the foot and ankle after an injury, we employ pathology-sensitive and anatomy-specific MR sequences in multiple imaging planes. In most cases, a pathology-sensitive sequence in the form of a T2-weighted sequence with fat suppression or short tau inversion recovery (STIR) is obtained in different planes and anatomic T1-weighted sequences are performed. It is important for one bone marrow-specific sequence, usually T1 weighted, to be obtained without fat suppression.

Short TR-TE T1-weighted images provide the general anatomic information and clearly depict abnormalities related to the marrow space and fat planes.

T2 weighted images utilize relatively long TR and TE and contribute high specificity regarding tendinous pathology. They have poor signal to noise ratio but are essential for soft tissue edema, fluid collection and for characterizing signal intensity alterations within ankle tendons.
The addition of fat suppression technique increased the sensitivity in detection of small amount of fluid contained in small tendinous and ligamentous tear. In comparison to the conventional spin echo imaging, use of fat suppression has been reported to increase the sensitivity for detecting partial tears from 67% to 92% (Mirowitz 2003). The use of an intermediate TE in FS T2-weighted images has an additional value in demonstrating underlying cartilage lesions.

So according to the clinical suspicion and examination we suggest the following sequences:

- In osseous injuries: T1 WI and fat suppression sequences in any plane.
- In osteochondral lesion of the talus: coronal T1 WI, T2 WI and fat suppression sequences.
- In ligamentous injuries: axial T1 WI, T2 WI and fat suppression and coronal fat suppression sequences.
- In tendinous injuries (except Achilles tendon): axial T1 WI, T2 WI and fat suppression sequences.
- In Achilles tendon injury: sagittal T1 WI and fat suppression sequences.
- In sinus tarsi syndrome: sagittal T1 WI and fat suppression sequences.
- In tarsal tunnel syndrome: T1 WI and fat suppression in any plane.
References

References


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References


الملخص العربي

تتعرض مكونات الكاحل والقدم لقدر هائل من القوة أثناء الممارسات الرياضية وهم طبيعيًا معرضين لمجموعة واسعة من الإصابات.

يعتبر الكاحل واحد من أكثر المفاصل إصابة. ولقد قسمت هذه الأمراض في هذه الدراسة على أساس الناحية التشريحية لكي يسهل فهمها. فتقتسم الإصابات إلى 1) إصابات عظمية. 2)إصابات الأربطة. 3)إصابات الأوتار. 4) إصابات متفرقة.

يجب أن نأخذ التشريح الجزئي للكاحل في الاعتبار لتسهيل تشخيص امراض الكاحل. تنقسم الأنسجة الرخوة للكاحل ببساطة إلى جزء أمامي، خلفي، جانبي للخارج، جانبي للداخل.

لم تصبح التصوير بالرنين المغناطيسي الطريقة المثلى في تقييم معظم هذه الآفات.

ويلعب التصوير بالرنين المغناطيسي دور مهم ومتزايّد في تقييم إصابات القدم والكاحل فهو يسمح بالتحديد الدقيق للتغيرات غير الطبيعية في العظام مثل الكسور الناتجة عن الإجهاد والضغط.

تغريت الأنسجة الرخوة وتشمل تمزقات الأربطة والأوتار والتهابات الأوتار. فالمض عبارة عن تحليل صور الرنين المغناطيسي يجب عليه مناظرتها بصورة نظامية ويثبتون البصريات الطبيعية ويصف التغريت في إشارات الأنسجة الرخوة والعظم.

عند تصوير إصابات الكاحل والقدم نستخدم نبضات الرنين المغناطيسي الحساسة للمرض و دقيقة التشريح في مستويات مختلفة. في معظم الحالات النبضات التي تنتج هى النبضات الحساسة للمرض المحملة على الزمن الثاني مع إكمال الدهون و مشتقاتها و تلك التشريحية المحملة على الزمن الأول. ومن الضروري عمل النبضات المحملة على الزمن الأول بدون إكمال الدهون التي تكون خاصة بلب العظام.