Management of neuropathic fracture of ankle and foot

Essay

Submitted for fulfillment of master degree in orthopedic

By

Mohamed Abd-elfatah Osman M.B, B.ch. Menofia University

Supervisors

Prof. Dr. Bahaa Ali Kornah

Professor of orthopedic surgery

El-Azhar University

Dr. Ismail Ahmed Yasseen

Assistant Professor of orthopedic surgery

El-Azhar University

Faculty of medicine

El-Azhar University

2013

Contents

Contents	Page
Acknowledgement	I
List of abbreviations	II
List of figures	III
Introduction	1
Aim of the work	3
Etiology	4
Epidemiology of neuropathic fracture of ankle and foot	6
Anatomy	7
Biomechanics of ankle	16
Pathophysiology	18
Pathogenesis	77
Classification	Y £
Clinical picture	27
Investigation	30
Treatment	36
Summary and conclusion	٥,
References	0 7
Arabic summary	1

Acknowledgement

First of all, all thanks to ALLAH for the inestimable blessing upon his slaves.

I would like to express my deepest gratitude and appreciation to Prof. DR. Bahaa Ali Kornah, professor of orthopedic, faculty of medicine, El-Azhar University, for his valuable supervision and generous support.

I would like to express my special thanks to DR. Ismail Ahmed Yassen, Associated. Professor orthopedic, faculty of medicine, El-Azhar University, for his support, and guidance.

I would like to express my special thanks to Orthopedic department, Al-Hussin hospital, for wonderful support.

I would like to express my deep thanks to my parents and family for their support, care and encouragement.

List of abbreviations

ACA	Acute charcot arthropathy	
ATFL	Anterior talofibular ligament	
CFL	Calcaneofibular ligament	
CN	Charcot neuroarthropathy	
CPPD	Calcium pyrophosphate dihydrate	
EBG	Electric bone growth	
ESR	Erythrocyte sedimentation rate	
FTA-ABS	fluorescent treponemal antibody absorbed, a blood serum screening	
FTCL	fibulotalocalcaneal	
HIV	Human immunodeficiency virus	
IER	inferior extensor retinacula	
IL	interleukin	
LTC	lateral talocalcaneal	
PN	Peripheral neuropathy	
PTFL	Posterior talofibular ligament	
RPR	rapid plasma reagin	
TENS	Transcutaneous electrical nerve stimulation	
TNF	Tumor necrosis factor	
WBC	White blood cells	
WHO	World health organization	
1CTP	serum collagen COOH-terminal telopeptide of type 1 collagen	

List of figures

Figure	Title	Page
Figure 1	Anatomy of the lateral ankle	8
Figure 2	Medial ankle view 8	
Figure 3	Bones of the foot, dorsal and 15 planter views.	
Figure 4	the vertical axis of ankle joint. 16	
Figure 5	Ratio of total contact area to joint area in the subtalar joint.	17
Figure 6	Radiograph of rocker bottom Charcot foot.	21
Figure 7	severely deformed Charcot foot.	22
Figure 8	charcot staging radiologically by sella.	25
Figure 9	Patterns of diabetic osteoarthropathy based on anatomic sites of involvement.	26
Figure 10	The Semmes-Weinstein monofilament is pressed against the skin until the filament bends.	28
Figure 11	The total contact, laminated, (a) open and (b) closed.	40
Figure 12	lateral radiograph of ankle after tibiotalocalcaneal arthrodesis	47
Figure 1 ^r	(a) AP view. (b) Lateral postoperative X-ray with external circular frames of the ankle.	47

Introduction

Peripheral neuropathy (PN) is a term used to describe damage to nerves of the peripheral nervous system, which leads to symptoms such as pain, numbness, tingling, burning, and weakness. Peripheral neuropathy most commonly affects the peripheral limbs, namely hands, arms, feet and legs. (Baron et

One of the most important functions of peripheral nerve cells is to alert a person to tissue injury and noxious stimuli or events in their environment. (Baron et al., 2010).

Normally, pain is a signal of imminent or actual harm to the body that initiates protective reflexes to prevent or minimize that danger. When tissue damage occurs, the resulting pain prompts special attention to the affected area and the person responds either by removing the source of danger (e.g., pulling a hand away from a hot object) or by initiating treatment quickly. (Michael, 2007).

The pain which is felt in response to a harmful stimulus is known as nociceptive pain. It is caused by stimulation of certain pain receptors and is generally described as sharp, aching, or throbbing. It is also the type of pain felt in some chronic, painful conditions (e.g., arthritis). (Botez et al., 2010).

Symptoms of peripheral neuropathy include weakness, sensory loss, abnormal balance, and autonomic dysfunction. Weakness in peripheral nerve disease is often distal and more severe in the legs than the arms. Deep and superficial muscles that are innervated by the peroneal nerve, such as the tibialis anterior and peroneus brevis and longus muscles, often cause more symptoms than do the plantar flexion muscles innervated by the tibial nerve, such as the gastrocnemius. As a result,

tripping on carpet or curb and spraining one's ankle are frequent symptoms of neuropathy. (Michael, 2007).

Clinical and experimental observations indicate that bone growth, repair and remodelling may be under the influence of the nervous system. (Dysart et al., 1989).

In humans, neural injuries are known to affect fracture healing. Brain damage appears to stimulate callus formation and fracture healing while spinal injuries and paralysis may cause pathologic fractures and excessive callus formation. (Mabilleau et al., 2010).

Aim of the work

The aim of this work is to review the etiology of naturopathic fracture of ankle and foot, the pathology, the staging, the classification, the diagnosis and the method of treatment either it is conservative or surgical.

Aetiology

Neuroarthropathy can result from various disorders which have the potential to cause a peripheral neuropathy (Table1). With the decline in numbers of patients with tertiary syphilis since Charcot's time and the concomitant rise in prevalence of diabetes mellitus, the latter disease has now become the primary condition associated with Charcot joints. (Frykberg et al., 2000).

Table1: Diseases With Potential for Causing Neuropathic Osteoarthropathy. (Frykberg, 2005).

Disorder	Prediliction site
Diabetes mellitus	Foot and ankle
Tabes dorsalis	Knee, shoulder, hip, ankle, and
	spine
Leprosy (Hansen's disease)	Foot, ankle, and hand
Syringomyelia	Shoulder, elbow, and cervical
	spine
Spina bifida	Hip and knee
Meningomyelocele	Foot and ankle
Congenital insensitivity to	Ankle and foot
pain	
Chronic alcoholism	foot
Peripheral nerve injury	Ankle and knee
Sciatic nerve severance	Ankle and knee
Spinal cord injury	Variable
Hysterical insensitivity to	Variable
pain	
Myelodysplasia	Variable
Multiple sclerosis	Variable
Riley-day syndrome	Variable
Intra-articular injections	Variable
Paraplegia	Variable

Neuropathic joints were first described in 1868 by Jean Martin Charcot. Initially, he was describing the neuroarthropathy associated with high-grade syphilis. However, this description has come to describe the neuroarthropathy associated with uncontrolled diabetes mellitus, first described by Jordan in 1936. (Samuel et al., 2011).

Charcot arthropathy of the foot and ankle is a syndrome consisting of fractures and dislocations in patients with peripheral neuropathy. (Trepman et al., 2004).

Epidemiology of neuropathic fracture of ankle and foot

The incidence of Charcot arthropathy, defined as the number of new cases over a set period of time divided by the study population initially without the disease, was first reported by to occur more often in diabetic neuropathy patients than in those with other neurological disorders. (Dane et al., 2009).

Neuropathic arthropathy is prevalent in 0.8 to 7.5 percent of diabetic patient with neuropathy; 9 to 36 percent of these affected patients have bilateral involvement. The higher prevalence occurs in referral based practices. Most patient with neuropathic arthropathy have had poorly controlled diabetes mellitus for 15 to 20 years. (fayed, 2006).

The tarsometatarsal (lisfranc's) joint is the most common site for arthropathy, with initial involvement usually occurring on the medial column of the foot. The distribution of naturopathic arthropathy is 70 percent at the midfoot and 15 percent at the forefoot or rearfoot; it is usually contained in one area. Nearly 50 percent of patient with neuropathy had an associated plantar ulcer. (fayde, 2006).

Both type 1 diabetes mellitus and type 2 diabetes mellitus patients appear to be equally at risk, although the former seem to present at a slightly earlier age. There is no sex predilection. (Vella et al., 2008).

Anatomy

The ankle complex comprises 3 articulations: the talocrural joint, the subtalar joint, and the distal tibiofibular syndesmosis. These 3 joints work in concert to allow coordinated movement of the rearfoot. Rearfoot motion is often defined as occurring in the cardinal planes as follows: sagittal-plane motion (plantar flexion-dorsiflexion), frontal-plane motion (inversion-eversion), and transverse-plane motion (internal rotation-external rotation). (Huson, 1987)

The motor and sensory supplies to the ankle complex stem from the lumbar and sacral plexus. The motor supply to the muscles comes from the tibial, deep peroneal, and superficial peroneal nerves. The sensory supply comes from these 3 mixed nerves and 2 sensory nerves: the sural and saphenous nerves. The lateral ligaments and joint capsule of the talocrural and subtalar joints have been shown to be extensively innervated by mechanoreceptors that contribute to proprioception. (Michelson et al., 1995).

The arteries of the midfoot and forefoot are (1) arches on the two aspects give rise to metatarsal arteries, which in turn give rise to digital arteries; (2) the dorsal arteries are exhausted before reaching the distal ends of the digits, so the plantar digital arteries send branches dorsally to supply the distal dorsal aspects of the digits, including the nail beds; and (3) perforating branches extend between the metatarsals forming anastomosis between the arches of each side. (Sinkjaer et al., 1988).

The talocrural joint receives ligamentous support from a joint capsule and several ligaments, including the anterior talofibular ligament (ATFL), posterior talofibular ligament (PTFL), calcaneofibular ligament (CFL), and deltoid ligament. The

ATFL, PTFL, and CFL support the lateral aspect of the ankle (Figure 1), while the deltoid ligament (Figure 2), provides medial support. (Stormont et al., 1985).

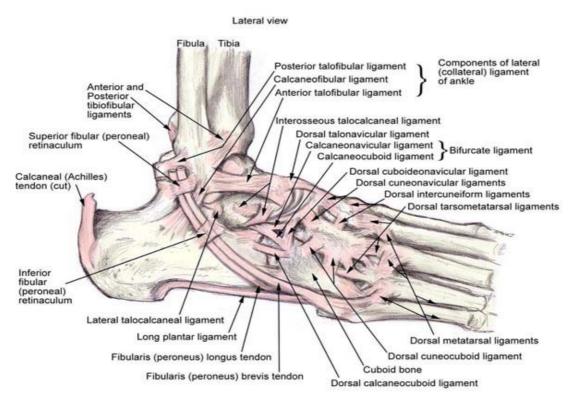


Figure 1: Anatomy of the lateral ankle ligamentous complex and related structures. (Cass et al., 1985).

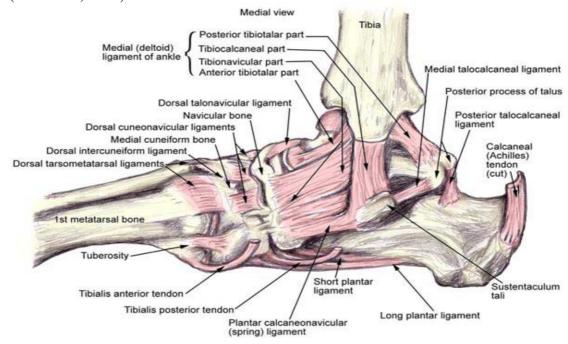


Figure 2: Medial ankle view showing the ligamentous anatomy of the deltoid ligament and related structures. (Cass et al., 1985).

It is slightly anterior to the frontal plane as it passes through the tibia but slightly posterior to the frontal plane as it passes through the fibula. Isolated movement of the talocrural joint is primarily in the sagittal plane, but small amounts of transverseand frontal-plane motion also occur about the oblique axis of rotation. (Lundberg et al., 1989).

Closed kinetic chain dorsiflexion occurs when the tibia moves anteriorly on the fixed talus during weight bearing. The concept of triplanar motion at the talocrural motion is important in understanding the stability of the talocrural joint. (Lundberg et al., 1989).

The ATFL lies on the dorsolateral aspect of the foot and courses from the lateral malleolus anteriorly and medially toward the talus at an angle of approximately 45° from the frontal plane. The ATFL is an average of 7.2 mm wide and 24.8 mm long. (Stormont et al., 1985).

The ATFL demonstrates lower maximal load and energy to failure values under tensile stress as compared with the PTFL, CFL, anterior inferior tibiofibular ligament, and deltoid ligament. (Attarian et al., 1985).

The CFL courses from the lateral malleolus posteriorly and inferiorly to the lateral aspect of the calcaneus at a mean angle of 133° from the long axis of the fibula. (Burks et al., 1994).

The CFL restricts excessive supination of both the talocrural and subtalar joints. In vitro experiments have demonstrated that the CFL restricts excessive inversion and internal rotation of the rearfoot and is most taut when the ankle is dorsiflexed. (Stormont et al., 1985).

The PTFL runs from the lateral malleolus posteriorly to the posterolateral aspect of the talus. The PTFL has broad insertions

on both the talus and fibula and provides restraint to both inversion and internal rotation of the loaded talocrural joint. (Burks et al., 1994).

The subtalar joint is formed by the articulations between the talus and the calcaneus and, like the talucrural joint; it converts torque between the lower leg (internal and external rotation) and the foot (pronation and supination). The subtalar joint allows the motions of pronation and supination and consists of an intricate structure with 2 separate joint cavities. The posterior subtalar joint is formed between the inferior posterior facet of the talus and the superior posterior facet of the calcaneus. (Rockar, 1995)

The anterior subtalar, or talocalcaneonavicular, joint is formed from the head of the talus, the anterior-superior facets, the sustentaculum tali of the calcaneus, and the concave proximal surface of the tarsal navicular. This articulation is similar to a ball-and-socket joint, with the talar head being the ball and the anterior calcaneal and proximal navicular surfaces forming the socket in conjunction with the spring ligament. (Perry, 1983).

The anterior and posterior subtalar joints have separate ligamentous joint capsules and are separated from each other by the sinus tarsi and canalis tarsi. The anterior joint lies farther medial and has a higher center of rotation than the posterior joint, but the 2 joints share a common axis of rotation. (Viladot et al., 1984).

This discrepancy results in an oblique axis of rotation of the subtalar joint, which averages a 42° upward tilt and a 23° medial angulations from the perpendicular axes of the foot Great variations have been identified in the position of the axis of rotation across individuals. (Inman, 1976).

The ligamentous support of the subtalar joint is extensive and not well understood. Marked discrepancies exist in the literature