



Ain Shams University
Faculty of Medicine
Dep. Of Orthopaedic Surgery

Stress Injuries of the Femur

Essay

Submitted For Partial Fulfillment of the Requirement of
the Master Degree in Orthopaedic Surgery

By:

Ahmed M. Ahmed Abd El-Aziz
M.B;B.CH.

Supervised by:

Prof. Dr. Ali Ibrahim Hussein
Prof. of Orthopaedic Surgery
Faculty of Medicine, Ain Shams University

Dr. Nabil Abd El-Monaem Ghaly
Ass. Prof. of Orthopaedic Surgery
Faculty of Medicine, Ain Shams University

65#Ü26#ÜTøD&5

{65+25õ5 , 7E" B áD // 'î 15 B{5ç8-{MÜ5/ {ö

(."ÜÑ2A5 2Q, 2M 2ø{52òY 2B2L2DE°! B5

ACKNOWLEDGEMENT

First of All, Thanks To Allah

I am so fortunate to carry this work under the guidance of **Prof. Dr. Ali Ibrahim Hussien** and **Ass. Prof. Dr. Nabel Abd El-Monaen Ghale**. Great thanks to both who offered me a lot of their knowledge, time and patience. And to orthopaedic department for giving this chance to me.

Thanks a lot to my father and my mother who supported me along my life, and special thanks to my wife who have the favour of my success.

Finally, I gift this work to my son and my daughter.

Ahmed M. Abd El-Aziz

CONTENTS

Subjects	Page
List of Figures	I
List of Tables	III
Chapter (I): Introduction	
1 - Introduction.....	1
2 - Aim of the study.....	3
Chapter (II): Structure of the femur	
1 - Histology of the bone.....	4
2 - Chemistry of bone.....	8
3 - Physiology of the bone.....	9
4 - Anatomy of the femur.....	11
5 - Biomechanics of the femur.....	16
Chapter (III): Stress injuries of the femur	
1 - Definition of stress injury.....	19
2 - Mechanisms of stress injury.....	21
3 - Risk Factors.....	20
4 - Effect of Age, Sex and Race.....	29
5 - Causes of stress injury.....	31
6 - Grades of fatigue injury.....	32
7 - Anatomical Distribution.....	34
8 - Incidence of fatigue injury of the femur....	37
9 - Bilateral fatigue injuries.....	39
10 - Complication of stress injury.....	40
Chapter (IV): Clinical presentation	
1 - History Taking.....	42
2 - Physical Examination.....	40
3 - Differential Diagnosis.....	47
Chapter (V): Investigations	
1 - Imaging Studies.....	51
2 - Laboratory studies.....	53

Cont. CONTENTS

Chapter (VI): Treatment of stress injury

1- Prevention.....	54
2- Physiological Dependency.....	55
3- Phases of the treatment.....	57
4- Medication.....	62
5- Surgical intervention.....	65
6- Treatment's Plan.....	68
7- Prognosis.....	69

Chapter (VII): Summary..... 70

Chapter (VIII): References..... 73

Chapter (IX): Arabic Summary

LIST OF FIGURES

No.	Title	Page
1	Show different types of bone cells	5
2	Section of bone tissue	5
3	Coronal section of the femur	7
4	The histology of bone	7
5	Calcium and phosphate homeostasis	10
6	Anterior view of femur	12
7	Posterior view of the femur	13
8	A. Coronal, B. Transverse sections of the femur	15
9	Femur's axes	18
10	Types of forces	20
11	Mechanical stress and bone	21
12	Ischemic mechanisms of stress injuries	23
13	Cyclic etiology of stress injuries formation	24
14	Microdamage to blood vessels	24
15	Bilateral coax vara	25
16	Coronal section of the femur show normal and osteoporosis in the femur	26
17	The carve of bone density	26
18	Forces on femur	28
19	MRI of fatigue injury	32
20	Grade II on MRI	32

Cont. LIST OF FIGURES

No.	Title	Page
21	Coronal MRI indicating a transverse supracondylar femoral fracture	33
22	MRI scan positive for a compression inferior femoral neck fracture	34
23	X-ray film showing a tension-side femoral neck fatigue fracture	35
24	Stress distribution in the femoral neck	35
25	Bone scan of the femur	36
26	MRI with marrow oedema in the medial femoral condyle	36
27	MRI with endosteal oedema of knee	39
28	Types of neck's stress fractures	40
29	Blood supply of the head of the femur	40
30	Displaced stress fracture	41
31	CT of the head of the femur	41
32	The continuum of pain	43
33	X-ray, bone scan, MRI of the neck of the femur	52
34	R.E.S.T. acronym	55
35	A 3-phase progression of stress injury rehabilitation	60
36	Three-phase cycle	61
37	Early operation	65
38	Delayed operation	66
39	X-ray after Antegrade intramedullary nail	67
40	Supracondylar fixation	67

LIST OF TABLES

No.	Table	Page
1	Bone mineral homeostasis	9
2	Grades for the femoral fatigue injuries	33
3	Percentage of incidence of femoral fatigue injuries	37
4	Grades and numbers of condyle fatigue injuries	38
5	Bilateral injuries of the femur	39
6	Site of pain in relation to the site of injury	43
7	Radiological findings	52
8	Rehabilitation goals	56
9	(A) Calcium carbonate (B)calcium citrate	62
10	(A) Ibuprofen, (B) Naproxen, and (C) Ketoprofen	63, 64
11	Avascular necrosis of the femoral head	66
12	Pathology and treatment	69

1 - Introduction

Stress injuries are partial or complete breaks in the continuity of bone that result from rhythmic, repeated, sub-threshold tensile or compressive loads (1). Not only in athletes and in military trainees but also in healthy people who have recently started a new or intensive physical activity (2, 3).

Bone constantly remodels itself to more efficiently endure external forces (4, 5). Most of the remodeling in the long bone takes place in the outer cortex (6). Remodeling involves the resorption of existing bone by osteoclast and the formation of new bone by osteoblast (7). Tension forces create a relative electropositivity on the convex, or tension side, of the bone. This increase in positive charge is conducive to osteoclast resorption (8, 9). Thus, repeated distraction forces at a focal point of bone lead to stress injuries.

Although the femur is longest, strongest and heaviest bone in the body, 70-80% of the stress injuries occur in the femur (10), at various sites including the head, neck (most common), shaft, supracondylar, condylar (next common) regions (11, 12). And bilateral injuries of the two femora also occurs (13).

Stress injuries have been classified into two types: **fatigue** and **insufficiency**. The fatigue injuries are caused by an abnormal stress to a normally elastic bone. Insufficiency fractures arise from the application of a normal stress on a mineral deficient or abnormally inelastic bone (1).

Fatigue injuries were graded on based MRI finding as: **grade I** (endosteal marrow oedema); **grade II** (periosteal and endosteal marrow oedema); **grade III** (muscle, periosteal and endosteal marrow oedema); **grade IV** (fracture line); **grade V** (callus in cortical bone) (14).

Usually the patients have a history of exercise induced pain in the hip, groin, thigh or knee, which is less severe at the beginning of exercise (15, 16), and occur mostly on 3rd week of the beginning of exercise (17), increase with continuing stress and when advanced becomes constant.

There are no specific findings on physical examination, there are negative finding on plain radiography (13, 10).

MRI can differentiate between stress reaction and stress fracture, and can distinguish between stress fracture and bone tumor or infection conditions (13).

The management of stress injuries depend on physiologic healing process of bone and include three phases, **phase I** allow time for the maturing of the periosteum and maturing of osteocytes, **phase II** include general condition and strengthening specific to the injured extremity, **phase III** allow for gradual remodeling of the bone and return to the original level of activity (15).

2- Aim of the study

The aim of the study is to review the literatures dealing with the mechanisms of stress injuries of the femur, to assess the anatomical distribution and incidence of femoral stress injuries, what are the risk factors, how to decrease the incidence, what are the proper diagnosis and management.

Chapter I

Introduction

1-Histology of the Bone

Bone is an extremely well organized tissue, from the modulation of hydroxyapatite crystal. At the microscopic level, the bone cells are:

I. Osteoprogenitor cells

Undifferentiated or osteoprogenitor cells are small cells with a single nucleus, few organelles with irregular form, remain in undifferentiated state until stimulated to proliferate or differentiate into osteoblast. They usually reside in the canals of bones, the endosteum and the periosteum (16).

II. Osteoblasts

The bone forming cells, it lines the surface of bone. At the light microscopic level, the active shows intense staining with basophilic stains and appears to be polarized, with the nucleus located at the end of the cell, away from the bone surface. The cytoplasm of the cell is occupied by three major components: the nucleus, the Golgi apparatus, and the rough endoplasmic reticulum (figure 1).

Alkaline phosphatase is distributed over the outer surface of the osteoblast cell membrane. It produces type I collagen, is responsive to parathyroid hormone, and produces osteocalcin when stimulated by 1,25-dihydroxy vitamin D (17).

III. Osteocytes

Once an osteoblast becomes surrounded by bone matrix, which then becomes mineralized, the cell is characterized by a higher nucleus-cytoplasm ratio, and contains fewer organelles. It arranged concentrically around the central lumen of an osteon and between lamellae. Osteoblast and osteocytes have extensive cell processes that project through the canaliculi, this produce communication between adjacent osteocytes and the central canals of osteon (figure 2) (17).

IV. Osteoclasts

Osteoclasts are the major resorptive cells of bone and are characterized by their large size [20-100 micrometer in diameter] and their multiple nuclei (**figure 1**). It is derived from pluripotent cells of the bone marrow, which are the hematopoietic precursors. It produces acid phosphatase.

Osteoclasts bind to the bone surface through cell attachment proteins called **integrins**, and they resorb bone by isolating an area of bone under the region of cell attachment. The osteoclasts then lower the PH of the local environment by production of hydrogen ions through the carbonic anhydrase system. The lowered PH increases the solubility of the hydroxyapatite crystal, and the organic components of the matrix are removed by acidic proteolytic digestion (11).

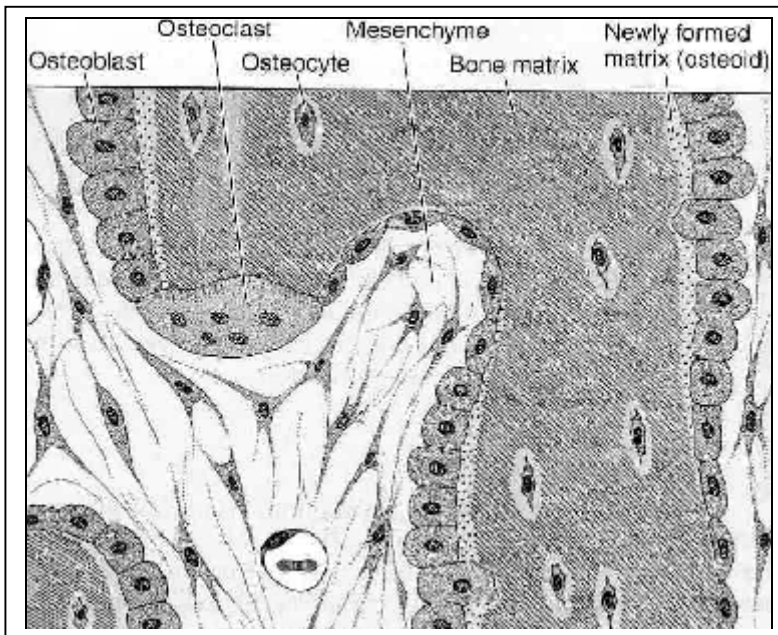


Figure 1:

Show different type of bone cells, osteoblast, osteocytes, and osteoclast (11).

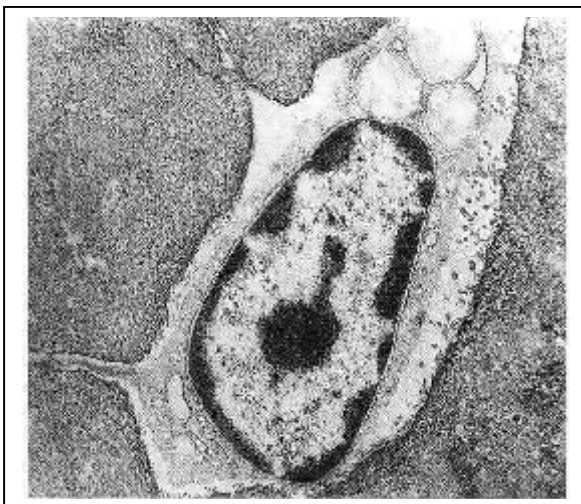


Figure 2:

Section of bone tissue showing an osteocytes with its cytoplasmic processes surrounded by matrix (11).

The bone consists of two types:

A- Woven bone is considered immature bone, or primitive bone, and normally is found in the embryo and the newborn, in fracture callus, and in the metaphyseal region of growing bone, it is coarse-fibered and contains no uniform orientation of the collagen fibers, it has more cells per unit volume than does lamellar bone, its mineral content varies, and its cells are randomly arranged

B- Lamellar bone begins to form one month after birth. By one year of age, it is actively replacing woven bone, as the latter is resorbed. By age four, most normal bone is lamellar bone. Lamellar bone thus is a more mature bone. It is found in several structural and functional systems: trabecular lamellae, and osteon with concentric lamellae.

Woven and lamellar bones are structurally organized into trabecular (spongy or cancellous) bone and cortical (dense or compact) bone (14).

Trabecular bone is found primarily at the metaphysis and epiphysis of long bone (figure 3).

Cortical bone composes the diaphysis in long bones (figure 3). **Cortical bone is subject to bending and torsional forces, as well as to compressive forces.**

Haversian bone is the most complex type of cortical bone. It is composed of vascular channels circumferentially surrounded by lamellar bone. This complex arrangement of bone around the vascular channels is called the osteon (figure 4).

The osteon is an irregular, branching, and anastomosing cylinder composed of a more or less centrally placed neurovascular canal surrounded by cell-permeated layers of bone matrix. Osteons are usually oriented in the long axis of the long bone and are the major structural units of cortical bone (14).

The central canal of an osteon called **the Haversian canal** contains cells, vessels, and occasionally nerves. Most vessels in the Haversian canals have the ultra-structural features of capillaries. The basement membrane of capillary walls may function as a rate-limiting or selective ion-limiting, transport barrier. The presence of this barrier is particularly important in calcium and phosphorus ion transport to and from bone, and is also important in explaining **the response of bone to mechanical loads** (14).