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Lipid Profileand it'sAssociation with Bone Mineral Density Among Postmenopausal Sudanese Women (a study in Khartoum State)

مستويات الدهون وعلاقتها بكثافه العظام لدى النساء السودانيات بعد سن اليأس في ولاية الخرطوم

A dissertation submitted in partial fulfillment for the requirement of M.Sc. degree in Medical Laboratory Science – Clinical chemistry

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Dedication

I dedicate this work to those who were the causes of mysuccess after God Allah.

To whom I saw the way of my life and from whom I drew my strength and my self-esteem, to the endless struggle, to the great who taught me the meaning of persistence and that no things Impossible with faith power and proper planning

(my greatest father).

To whom she has supported me in her prayer, shared me my Joys and sorrows, to the source of tenderness and the finest women in the world

(mydearest Mother).

To those who are closer to me than my heart and soul

(my loved sisters)

To my little beautiful babies

(Talien and Asia)

To my very great family and my friend.

To all the mothers especially the ones I studied with.

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Abstract

Background: Low bone mass is a major health problem in postmenopausalwomen.The aim of this study to investigate the association between lipid profile and bone mineral density (BMD) in postmenopausal women in Khartoum, Sudan .

Materials and Methods: This wasacross-sectional study, performed on 98 postmenopausal women from Khartoum-Sudan, between March 2019 to December2019. Bone mineral density was measured by Dual-energy X-ray absorptiometry(BMD in g/cm2).Serumtotal cholesterol, triglyceride and HDL-C levels were measured by enzymatic method usingspectrophotometer,LDL-C calculated using the Friedewald formula.Weight and height were measured byHY-RGZ160 160.Data were analyzed by using SPSS program version 16.

Results: According to DEXA result, 37.9 % of women were normalpenia, 47.9% were osteopenia and 11.2% were osteoporosis

According to BMD groups (normalpenia, osteopenia, osteoporosis) there were insignificant change in means of serum total cholesterol (166.5 ± 31.4 mg/dL), (177.9 ± 45.3 mg/dL), (182.5 ± 46.3 mg/dL),serum triglyceride (134.2 ± 40 mg/dL), (120.8 ± 38.2 mg/dL), (110 ± 26.6 mg/dL), serum HDL-c(58.1 ±16.5 mg/dL), (57 ± 12.8 mg/dL), (64±10.7 mg/dL), serum LDL-c (79.9 ± 30.3 mg/dL) (94.4 ± 40.9 mg/dL), (96.9 ± 48.1 mg/dL) *p-value* >0.05 respectively.

Conclusion:

The data of the study suggest thatSudanese postmenopausal women had normal lipid profile level. Meanwhile, no association observed between lipid profile and bone mineral density.

المستخلص

خلفية الدراسة: انخفاض كتلة العظم مشكله صحيه كبيره لدى النساء بعد سن اليأس. أجريت هذه الدراسه لبحث العلاقة بين مستوى الدهون وكثافة العظم في النساء بعد سن اليأس في ولاية الخرطوم- السودان.

المواد والطرق: دراسة وصفيه أجريت اثناء الفترة من مايو الى ديسمبر2019 في ثمانيه وتسعون من النساء السودانيات بعد سن اليأس. تتضمن الدراسة قياس الكثافه المدنيه العظام عن طريق امتصاص الاشعه السينيه المزدوجة الطاقه(كجم/200. تم قياس مستويات الكولسترول الكلي، الدهون الثلاثيه،والدهون مرتفعه الكثافه باستعمال طريقه الكشف الكيميائي باستخدام الاسبكتروفوتومتر. وحساب الدهون منخفضة الكثافة عن طريق معادله فرايدولد. وقد قيس الوزن والطول عن طريق ميزان قياس الوزن والطول 160 HY-RGZ160

النتائج: وفقاً لنتجه جهاز قياس كثافه العظم فإن 37.7% من النساء لديهن كثافه عظم طبيعية 47.9%تم تشخيصهن بترقق العظام،11.1% مصابات بهشاشه العظام. أظهرت الدراسة حسب الكثافة المعدنية للعظام (طبيعي ،ترقق العظام ،هشاشه العظام) لا يوجد تغيير في متوسطات الكوليسترول الكلي(المتوسط والانحراف المعياري31.4 ±166.5)، (المتوسط والانحراف المعياري45.3±177.9)، (المتوسط والانحراف المعياري46.3±182.51)، الدهون الثلاثية (المتوسط والانحراف المعياري 40±134.2)، (المتوسط والانحراف المعياري 38.2± 120.8)، (المتوسط والانحراف المعياري 26.6±110)، الدهون مرتفعة الكثافة (المتوسط والانحراف المعياري 16.5 ±58.1)، (المتوسط والانحراف المعياري 12.8±57)، (المتوسط والانحراف المعياري 10.7±64)، الدهون منخفضة الكثافة (المتوسط والانحراف المعياري 30.3±79.9)، (المتوسط والانحراف المعياري 40.9±94.4)، (المتوسط والانحراف المعياري84.1±96.9) مع قيم احتمال < 0.05 بالتوالي.

الخلاصة: تشير بيانات الدراسه إلى ان النساء السودانيات بعد سن اليأس لديهن مستوى دهون طبيعي. في الوقت نفسه لم يلاحظ أي ارتباط بين مستوى الدهون وكثافة العظم.

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List of Abbreviations

Abbreviations	Full terms
ALP	Alkaline phosphatase
BMD	Bone mineral density
BMI	Body mass index
DEXA	Dual energy x ray absorbmetry
HDL-C	High density lipoprotein
ISCD	International society for clinical densitometry
LDL-C	Low density lipoprotein
LRP	receptor related protein
ONJ	Osteonecrosis of the jaw
Pdxa	Peripheral Dual Energy X ray Absorbmetry
SD	Standard Deviation
SPSS	Statistical Package for the Social Science
SXA	Single Energy X ray Absorbmetry
USPSTF	United states preventative services task force
WHO	World health organization

Chapter One

Introduction, rationale, objective

1.Introduction, Rationale, and Objectives

1.1 Introduction

Low bone mineral density are currently estimated to be a major health threat for almost 44 million US men and women aged 50 and older, or 55% of the population in that age range**(Nieves.,2005).**

Low bone mineral density is a disease which affect the population of so called "third age", or the elderly, while it more affect females as a result of the fact that they have a 30% lower bone mass than men, and that among them there is a rapid process of losing bone mass after entering the menopause and the occurrence of ovarian insufficiency**(Bijelic et al.,2016).**

The emergence of low bone mineral density involves number of factors (genetic and environmental factors), as well as other pathological entities that can lead to rapid loss of bone mass. Bone mineral density is decreased due to the breakdown of bone without compensatory, subsequent remodeling **(Oleson and Morina., 2017).**

Low bone density is measured by a bone mineral density test that measure the amount of mineral by square centimetre and can be performed by number by radiological Densitometry procedure, most often dual energy x ray absorbemtry (DEXA). the most common areas evaluated are the lumber spine or proximal hip and distal radius**(Oleson and Morina., 2017).**

Dislipidemia has been associated with low bone mass in some studies, Some studies have shown that subjects thelow bone density have a higher risk ofcardiovascular events resulting fromatherosclerosis than those with

osteopenia **(Ghadiri et al.,2016).**The mechanism of this relation may be directly related with the lipid biosynthetic pathway, which determines cholesterol levels and contributes to the activity of the osteoclast **(Bauer et al.,2003**). These findings proposed the probable association between serum lipid profile and BMD especially among patients with increased risk oflow bone mineral density rather than healthy persons **(Sivas et al.,2009).**

1.2 Rationale:

Low bone mass is a major health problem in postmenopausal women, 1 in 2 women over the age of 50 will fracture at some point in their lifetime
Significant mortality and morbidity are associated with osteoporotic fracture.
Low bone density has no clinical manifestations until there is a fracture, low bone mineral density results in a decreased quality of life, increased disability-adjusted life span,
Lackofresearchinourcountryisaggravatingthesituationduetolackoffundigforresearchingeneralandforthistypeofresearchinparticular,alongwiththelackofhumancapacitytrainedinconductinggoodqualityresearchareimportantreasonsforthescarcityofresearch.

Estrogen deficiency reduces the amount of components that maintain bone .density and strengthIt also lead to obesity, as obesity in the abdominal region causes elevated levels of cholesterol, fat associate‹Theseriskfactorsarepartofmetabolicsyndrome.thebloodin(triglycerides) .andbloodvessels‹dwithanincreasedriskofdevelopingheartdisease

1.3 Objectives:

1.3.1 Genral objective:

To investigate the association between lipid profile and bone mineral density among postmenopausal women in khartoum state.

1.3.2 specific objectives:

1-To estimate lipid profile levels in in Sudanese postmenopausal women

2- To compare between means serum level lipid profile and bone mineral density(DEXA score).

3- To measure weight, higherand calculate body mass index.

4-To correlate between lipid profile and body mass index.

5- To correlate between lipid profile and age.

Chapter two

Literature review

2. Literature Review

2.1 Bone

Bone provides mechanical support and protection to soft organs, enables movement, hosts hematopoietic tissue, and serves as storage of calcium, phosphate, and magnesium ions. From one third it consists of protein matrix, and from two thirds of the bone mineral ***(Fialovz and Vejrazka., 2017).***The bone protein matrix contains mostly type I collagen (90 %) together with other proteins such as osteocalcin,osteonectin, osteopontin, etc. (10 %) ***(Fialova and Vejrazka., 2017).***The bone mineral is composed from small crystals of hydroxyapatite Ca10(PO4)6(OH)2. Other compounds, such as calcium carbonate, calcium fluoride and magnesium phosphate, are present as well. The metabolic activity of bone tissue is provided by bone cells. Osteoblasts form osteoid (the bone protein matrix), into which mineral salts are deposited. Other cell types include osteoclasts, whose main function is bone resorption, and osteocytes, which complement activity of osteoclasts by providing fine tuning of bone resorption***(Fialova and Vejrazka., 2017).***

2.1.1 Functions of Bone

Mechanically They provide a frame to keep the body supported, and an attachment point for skeletal muscles, tendons, ligaments and joints, which function together to generate and transfer forces so that individual body parts or the whole body can be manipulated in three dimensional space (the interaction between bone and muscle is studied in biomechanics). Bones protect internal organs, such as the skull protecting the brain or the ribs protecting the heart and lungs ***(Vincent K.,2013)*** .

bone does have a significant degree of elasticity, contributed chiefly by collagen. The macroscopic yield strength of cancellous bone has been

investigated using high resolution computer models ***(LevreroF and Margetts L .,2016).***

Synthetic Function The cancellous part of bones contain bone marrow, Bone marrow produces blood cells in a process called hematopoiesis***(FernándezKS and Alarcón DE.,2013).*** bone marrow is also one of the major sites where defective or aged red blood cells are destroyed.

Metabolic functions Mineral storage— bones act as reserves of minerals important for the body, most notably calcium and phosphorus ***(Walker and Kristin.,2017).***

Fat storage-marrow adipose tissue(MAT) acts as a storage reserve of fatty acids ***(Styner et al.,2017)***

Acid-base balance — bone buffers the blood against excessive pH changes by absorbing or releasing alkaline salts.

Detoxification—bone tissues can also store heavy metals and other foreign elements, removing them from the blood and reducing their effects on other tissues.

Endocrine organ—bone controls phosphate metabolism by releasing fibroblast growth factor – 23 (FGF-23), which acts on kidneys to reduce phosphate reabsorption. Bone cells also release a hormone called osteocalcin, which contributes to the regulation of blood sugar (glucose) and fat deposition. Osteocalcin increases both the insulin secretion and sensitivity, in addition to boosting the number of insulin-producing cells and reducing stores of fat ***(Lee NA.,2007)***.

2.1.2 Bone cells

2.1.2.1 Osteoblasts

are mono nucleate bone forming cells ,they are located on the surface of osteon seams and make a protein mixture known as osteoid, which mineralizes to become bone,is primarily composed of Type I collagen, Osteoblasts also manufacture hormones such as prostaglandins to act on the bone itself.The osteoblast creates and repairs new bone by actually building around itself. First, the osteoblast puts up collagen fibers. These collagen fibers are used as a framework for the osteoblasts‘ work. The osteoblast then deposits calcium phosphate which is hardened by hydroxide and bicarbonate ions. The brand new bone created by the osteoblast is called osteoid ***(Washington.,2006).***

2.1.2.2Bone Lining Cells

Bone lining cells are quiescent flat shaped osteoblasts that cover the bone surfaces, where neither bone desorption nor bone formation occurs.The secretory activity of bone lining cells depends on the bone physiological status, whereby these cells can reacquire their secretory activity, enhancing their size and adopting a cuboidal appearance ***(Rubin et al.,1995).*** Bone lining cells functions ar not completely understood, but it has been shown tha these cells prevent the direct interaction between osteoclast and bone matrix, when bone resorption should not occur, and also participate in osteoclast differentiation, producing osteoprotegerin (OPG) and the receptor activator of nuclear factor kappa-B ligand (RANKL) ***(Skorzynska et al., 2009).***

the bone lining cells, together with other bone cells, are an important component of the BMU, an anatomical structure that is present during the bone remodeling cycle ***(Korper et al., 2002).***

2.1.2.3 Osteocyte :

Osteocytes are mostly inactive osteoblasts, Osteocytes originate from osteoblasts that have migrated into and become trapped and surrounded by bone

matrix that they themselves produced**.** Osteocytes have many processes that reach out to meet osteoblasts and other osteocytes probably for the purposes of communication***(Natalie A.,2014).***

2.1.2.4 Osteoclast:

Is very large multinucleate cells that are responsible for the breakdown of bones by the process of bone resorption. bone is constantly remodeled by the resorption of osteoclasts and created by osteoblasts.Because the osteoclasts are derived from a monocyte stem-cell lineage, they are equipped with phagocytic-like mechanisms similar to circulating macrophages Osteoclasts mature and/or migrate to discrete bone surfaces. Upon arrival, active enzymes, such as tartrate resistant acid phosphatase, are secreted against the mineral substrate. the reabsorption of bone by osteoclasts also plays a role in calcium homeostasis.

2.1.3 bone remodeling

is the process by which old bone is replaced by new bone. The normal bone remodeling process consists of five phases: the resting phase,activation, resorption, reversal, and formation .

In the activation phase of remodeling, osteoclasts are recruited to the surface of the bone ***(Gallagher and Harsha., 2013).***

In the resorption phase, osteoclasts generate an acidic microenvironment between the cell and the surface of the bone dissolving or resorbing the mineral content of the bone ***(Gallagher and Harsha., 2013)***.

In the reversal phase osteoclasts undergo apoptosis and osteoblasts are recruited to the bone surface***(Gallagher and Harsha., 2013)***.

In the formation phase, osteoblasts then deposit collagen; this is mineralized to form new bone ***(Gallagher and Harsha., 2013).***

In normal process of bone turn over the rate of bone resorption and bone formation is equal in which by acidification osteoclasts remove bone and by secreting osteoid into the resorption cavity osteoblasts build bone. The rate of bone turnover is increased due to elongation of the life span of osteoclasts and reduction in the lifespan of osteoblasts ***(Shakoor and Putra., 2015).***

2.1.4 Bone Mineral Density (BMD):

A bone mineral density test (BMD), a non-invasive and painless test, is the best way to determine bone health. BMD tests can identify osteoporosis, determine the risk for fractures and monitor the response to an osteoporosis treatment. Different BMD tests may measure the hip, spine, wrist, finger, shinbone or heel. The National Osteoporosis Foundation recommends BMD testing for the following individuals:

All women aged 65 and older regardless of risk factors.

Younger postmenopausal women with one or more risk factors.

Postmenopausal women who present with fractures (to confirm the diagnosis and determine disease severity).

Estrogen deficient women at clinical risk for osteoporosis.

Individuals with vertebral abnormalities.

Individuals receiving, or planning to receive, long-term glucocorticoids (steroid) therapy.Individuals with primary hyperparathyroidism.

Individuals being monitored to assess the response or efficacy of an approved osteoporosis drug therapy.Mechanical properties of bone are determined by various factors, such as size and shape of individual bone, cortical thickness, porosity and the orientation of collagen fibers, and, to a greater extent, degree of mineralization or bone mineral density (BMD), which is defined as “the mass of

inorganic (mineral) matter per unit volume.". BMD must be analyzed in bone's three levels of biological organization: in bone material (BMD material), in a bone's trabecular and cortical tissue compartments (BMD compartment), and in the entire bone (BMD total) ***(Cole RE ., 2008)*** .

in healthy individuals, BMD changes occur under circumstances such as ageing, adaptation, healing Pathologically, BMD can be modified under many different conditions, affecting bone's behavior under physical stress.

Common bone mineral density tests:

Dual Energy X-ray Absorptiometry (DEXA).Peripheral Dual Energy X-ray Absorptiometry (pDXA). Single Energy X-ray Absorptiometry (SXA) Peripheral Quantitative Computed Tomography (pQCT).Radiographic Absorptiometry (RA). Quantitative Computed Tomography (QCT).

Quantitative Ultrasound(QUS).

2.1.5 DualenergyX-ray absorptiometry (DEXA);

it is the actual expression of the bone in absolute terms of grams of mineral (primarily, as g/cm2 of calcium) per square centimeter of the scanned bone. BMD measurements of the hip and spine are used to establish or confirm the diagnosis of osteoporosis to predict future fracture risk and monitor patients. The difference between the patient's BMD and mean BMD of young females aged in the range of 20-29 years (divided by the standard deviation (SD) of the reference population) yields the T-score; comparing the BMD of a particular age, sex, and ethnicity-matched adult reference population is called the Z-score. As defined by the World Health Organization (WHO), osteoporosis is present when BMD is 2.5 SD or more below the average value for young healthy women (a T-score of <-2.5 SD). A second, higher threshold describes "low bone mass" or osteopenia as a T-score that lies between -1 and -2.5 SD. "Severe" or "established" osteoporosis denotes osteoporosis that has been defined in the

presence of one or more documented fragilres ***(Kanis JA .,2007).*** T-score criteria are applied for the BMD measured by means of central DXA at the femoral hip and lumbar spine for postmenopausal women and men aged 50 years and older. For premenopausal women, men less than 50 years of age, and children, the BMD diagnostic classification as defined by the WHO should not be applied. The International Society for Clinical Densitometry (ISCD) recommends using ethnic- or race-adjusted Z-scores: Z-scores of −2.0 or lower are defined as "low bone mineral density for chronological age" or "below the expected range for age" and those above −2.0 are defined as "within the expected range for age" (***Schousboe et al.,2013***). The United States Preventive Services Task Force (USPSTF) recommends the testing of all women aged 65 years and above and younger women whose fracture risk is equal to or greater than that of a 65-year-old white woman who exhibits no additional risk factors ***(Nelson et al.,2010)***. Indications for measuring BMD are listed at ***(Papaioannou et al.,2013).***

2.1.6 Osteopenia

Osteopenia is a clinical term used to describe a decrease in bone mineral density (BMD) below normal reference values, yet not low enough to meet the diagnostic criteria to be considered osteoporotic. BMD is diagnosed via(DEXA) bone scan***(Varacallo M.,2018)***

The diagnostic difference delineating osteopenia from osteoporosis as defined by the World Health Organization (WHO) is a t score between 1 to 2.5. Values less than 2.5 are diagnostic for osteoporosis. Decreasing BMD values are reflective of an underlying disruption in the micro architecture of bone and osteopenia, and osteoporosis are considered quantitative, not qualitative,

disorders of bone mineralization ***(Varacallo M.,2018)***

Overall, females have a fourfold higher overall prevalence of osteopenia compared to males. However, males are more likely to demonstrate secondary causes of decreased bone mass. While secondary osteopenia and osteoporosis can develop at any age, the incidence of osteopenia in select subgroups demonstrates predictable patterns and trends. In the United States, 54% of postmenopausal 2.1.p2.2.2.c, and additional 30% are already considered osteoporotic. By age 80, this relative trend predictably shifts in favor of osteoporosis as 27% of women are osteopenic, and 70% are osteoporotic ***(Varacallo M.,2018)*** .

2.1.6.2 Pathophysiology of osteopenia

Osteopenia occurs secondary to uncoupling of osteoclast osteoblast activity, resulting in a quantitative decrease in bone mass. Peak bone mass is typically achieved by males and females just prior to, or earlyon in the third decade of life. Beyond age 30, bone resorption gradually becomes favored as dynamic bone remodelling continues into later decades of life ***(Varacallo M.,2018***) .

2.1.6.2 Histopathology of osteopenia

Histologic specimens demonstrate markedly thinned trabeculae, decreased osteon size, and enlarged haversian and marrow spaces (***Varacallo M.,2018)*** .

2.1.6.3 Toxicokineticsof osteopenia

Bisphosphonates are the most commonly prescribed medication class for treatment. Adverse side effects are well documented in the literature as prolonged use has been linked to two major clinical side effects: osteonecrosis of the jaw (ONJ) and the atypical sub trochanteric femur fracture. ONJ is rare and is associated with intravenous forms and not oral forms of the medication. Treatment entails immediately stopping the offending agent. Atypical femur fractures also are rare but have significant associated morbidity, and clinicians

are cautioned against the chronic, uninterrupted bisphosphonate use beyond 3 to 5 years or in situations when patients report mild thigh discomfort while undergoing treatment ***(Varacallo M.,2018)***.

2.1.6.4 Treatment and Management of osteopenia

The core treatment options for osteopenic patients involve early education on how to achieve and maintain healthy bone mass levels and extensive education and counselling on the relevant social, environmental, and lifestyle risk factors that compromise bone health ***(Varacallo M.,2018).***

2.1.7 Osteoporosis

Is a disease that is characterized by low bone mass, deterioration of bone tissue, and disruption of bone microarchitecture, it can lead to compromised bone strength and an increase in the risk of fractures ***(JAMA., 2001).*** Osteoporosis is the most common bone disease presenting a major public health problem. It is more common in Caucasians, women, and older people. Osteoporosis affects an enormous number of people, of both sexes and all races, and its prevalence will increase as the population ages. It is a silent disease until fractures occur, which causes important secondary health problems and even death***(Cosman et al.,2014).*** Bones that commonly break include the vertebra in the spine, the bones of the forearm, and the hip ***(Golob AL and Laya MB ., 2015)***. Osteoporosis may be due to lower-than normal maximum bone mass and greater than- normal bone loss. Bone loss increases after menopause due to lower levels of estrogen .

Osteoporosis has generally been divided into two categories: primary and secondary osteoporosis. Primary osteoporosis is age related, affects 95 % of women and about 80 % of men, and is related to estrogen loss in women and a testosterone deficiency in men; other factors include low calcium and vitamin D

intake as well as hyperparathyroidism. In contrast, secondary osteoporosis stems from other conditions including hormonal imbalances, diseases, and medications that predispose to bone loss. It may arise at any age and affects both men and women ***(Oleson and Morine., 2017).***Osteoporosis may also occur due to a number of diseases or treatments, including alcoholism, anorexia, hyperthyroidism, kidney disease, and surgical removal of the ovaries. According to the World Health Organization osteoporosis is considered a silent epidemic of the century, causing great economic and social impacts***(Moreira et al., 2017)***.

Literal meaning of osteoporosis is “porous bone”. It is a multifactorial skeletal disorder which leads to low bone mineral density and is the major cause of fracture incidences, disabilities, reduced mobility and poor quality of life ***(Hirani et al., 2014)***.

2.1.7.1 Signs and symptoms of osteoporosis

Osteoporosis itself has no symptoms; its main consequence is the increased risk of bone fractures.

Fractures are a common symptom of osteoporosis and can result in disability ***(Jameson et al.,2018).***Acute and chronic pain in the elderly is often attributed to fractures from osteoporosis and can lead to further disability and early mortality ***(Old JL and Calvert M., 2004)***.

The most common osteoporotic fractures are of the wrist, spine, shoulder and hip. The symptoms of a vertebral collapse ("compression fracture") are sudden back pain, often with radicular pain (shooting pain due to nerve root compression) and rarely with spinal cord compression or cauda equina syndrome. Multiple vertebral fractures lead to a stooped posture, loss of height, and chronic pain with resultant reduction in mobility ***(Kim DH and Vaccaro AR.,2006)***.

Fracture risk calculators assess the risk of fracture based upon several criteria, including bone mineral density, age, smoking, alcohol usage, weight, and gender. Recognized calculators include FRAX ***(Susan Ott.,2009).*** and Dubbo.

2.1.7.2Risk factors of osteoporosis

2.1.7.2.1 Non modifiable:

Age(in both men and women) and female sex; estrogen deficiency following *Bone density peaks at about 30 years of age. Women lose bone mass more rapidly than men* ***(Waugh et al.,2009).***menopause or surgical removal of the ovaries is correlated with a rapid reduction in bone mineral density, while in men, a decrease in testosterone levels has a comparable (but less pronounced) effect ***(Sinnesael et al.,2015)***

Ethnicity while osteoporosis occurs in people from all ethnic groups, European or Asian ancestry predisposes for osteoporosis ***(Melton LJ .,2003).***

Heredity those with a family history of fracture or osteoporosis are at an increased risk; the heritability of the fracture, as well as low bone mineral density, is relatively high, ranging from 25 to 80%. At least 30 genes are associated with the development of osteoporosis ***(Raisz L .,2005).***Those who have already had a fracture are at least twice as likely to have another fracture compared to someone of the same age and sex (***Markides et al.,2007).***

Build a small stature is also a non modifiable risk factor associated with the development of osteoporosis ***(Guglielmo et al., 2009).***

2.1.7.2.2 Potentially modifiable:

Excessive alcohol Although small amounts of alcohol are probably beneficial (bone density increases with increasing alcohol intake), chronic heavy drinking (alcohol intake greater than three units/day) probably increases fracture risk despite any beneficial effects on bone density ***(Nieves JW.,2005)***.

Vitamin D deficiency*(Gielen et al.,2011).* Low circulating Vitamin D is common among the elderly worldwide. Mild vitamin D insufficiency is associated with increased parathyroid hormone (PTH) production, PTH increases bone re sorption, leading to bone loss. A positive association exists between serum 1,25-dihydroxycholecalciferol levels and bone mineral density, while PTH is negatively associated with bone mineral density ***(WHO.,2003).***

Tobacco smoking Tobacco smoking has been proposed to inhibit the activity of osteoblasts, and is an independent risk factor for osteoporosis ***(Wong et al.,2007).*** Smoking also results in increased breakdown of exogenous estrogen, lower body weight and earlier menopause, all of which contribute to lower bone mineral density ***(WHO.,2003).***

Malnutrition Nutrition has an important and complex role in maintenance of good bone. Identified risk factors include low dietary calcium and/or phosphorus, magnesium, zinc, boron, iron, fluoride, copper, vitamins A, K, E and C (and D where skin exposure to sunlight provides an inadequate supply). Excess sodium is a risk factor. High blood acidity may be diet-related, and is a known antagonist of bone ***(Ilich JZ, and Kerstetter JE .,2000).***

Some have identified low protein intake as associated with lower peak bone mass during adolescence and lower bone mineral density in elderly populations. Conversely, some have identified low protein intake as a positive factor, protein is among the causes of dietary acidity. Imbalance of omega-6 to omega-3 polyunsaturated fats is yet another identified risk factor***(Weiss et al.,2005)***

High dietary protein from animal sources research has found an association between diets high in animal protein and increased urinary calcium, and have been linked to an increase in fractures ***(Feskanich et al.,1996).***However, the relevance of this observation to bone density is unclear, since higher protein diets tend to increase absorption of calcium from the diet and are associated

with higher bone density ***(Kerstetter et al.,2011).*** Indeed, it has recently been argued that low protein diets cause poor bone health ***(Bonjour JP .,2005).*** No interventional trials have been performed on dietary protein in the prevention and treatment of osteoporosis ***(Insogna et al.,2003).***

Underweight/inactive bone remodeling occurs in response to physical stress, so physical inactivity can lead to significant bone loss. Weight bearing exercise can increase peak bone mass achieved in adolescence, and a highly significant correlation between bone strength and muscle strength has been determined ***(Michalk et al.,1996)***

Heavy metals a strong association between cadmium and lead with bone disease has been established. Low-level exposure to cadmium is associated with an increased loss of bone mineral density readily in both genders, leading to pain and increased risk of fractures, especially in the elderly and in females. Higher cadmium exposure results in osteomalacia (softening of the bone) ***(Staessen et al.,1999).***

Soft drink some studies indicate soft drinks (many of which contain phosphoric acid) may increase risk of osteoporosis, at least in women***(Tucker et al.,2006).***

2.1.7.3 Treatment and Follow up Considerations of low BMD

Treatment duration varies depending on the class of medication utilized. Agents such as teriparatide and hormonal based therapy require immediate follow up treatment with another agent upon stopping the medication, otherwise ,bone mass is rapidly lost. Clinicians also must remain cautious against the prolonged use of uninterrupted bis phosphonate therapy beyond a 3to 5year period. Patients should also be made aware of these potentially morbid adverse events, and they should be counseled to seek immediate care if they are experiencing any symptoms of thigh discomfort ***(Varacallo M.,2018)*** .

Any patient on bisphosphonates for any given time period and presenting with

mild thigh discomfort should have the following treatment workup:

Educate on the risks of and immediately stop all weightbearing activity. Obtain fulllength femur and hip radiographs ***(Varacallo M.,2018)*** .

Thigh pain may be indicative of an impending pathologic, atypical femur fracture ***(Varacallo M.,2018)*** .

Attention should be directed to the subtrochanteric and diaphyseal regions of the femur,particularly the lateral cortex which often demonstrates evidence of periosteal reaction ***(Varacallo M.,2018)*** **.**

Immediately discontinue bisphosphonate use.Refer to an orthopedic surgeon for prophylactic surgical fixation ***(Varacallo M.,2018)***.

2.2Lipids and lipoprotein:

2.2.1 Lipids

Are any heterogeneous group of fat and fat like substances characterized by being water insoluble and soluble in nonpolar solvent such as alcohols, ether, chloroform, benzene and etc . the lipids have importance role in source of energy , serving as hormones , fat soluble vitamins , aiding in digestion and acting as structural components af cells membrane **(*carl et al., 2008*)** .

2.2.1.1.classifications of lipids

2.2.1.1.1 fatty acids

are simply linear chains of CMH bonds that terminate with a carboxyl group (MCOOH).1 In plasma, only a relatively small amount of fatty acids exists in the free or unesterified form, most of which is bound to albumin. The majority of plasma fatty acids are instead found as aconstituent of triglycerides or phospholipids (Michael, 2010). fatty acids are catabolized by enzymatic oxidation in mitochondria and produce energy by serious ofreactions known by B-oxidation ***(carl et al., 2008)*** .

2.2.1.1.2 Cholesterol

is an unsaturated steroid alcohol containing four rings, and it has a single C--H side chain tail similar to a fatty acid in its physical properties. The only hydrophilic part of cholesterol is the hydroxyl group in the first-ring. Cholesterol is, therefore, also an amphipathic lipid and is found on the surface of lipid layers along with phospholipids***(Michael.,2010).*** Cholesterol is also important in membrane structure and is the precursor of steroidhormonesand bileacids ***(William.,2012).*** Cholesterol is endogenous with 90% synthesized by liver and intestine.Cholesterol is also unique in that, unlike other lipids, it is not readily catabolized by most cells and, therefore, does not serve as a source of fuel. Cholesterol can, however, be converted in the liver to primary bile acids, such as cholic acid and chenodeoxycholic acid, which promote fat absorption in the intestine by acting as detergents. A small amount of cholesterol can also be converted by some tissue, such as the adrenal gland, testis, and ovary, to steroid hormones such as glucocorticoids, mineralocorticoids, and estrogens. Finally, asmall amount of cholesterol, after first being converted to 7-dehydrocholesterol, can also be transformed tovitaminD3 in the skin by irradiation from sunlight ***(Michael et al., 2010).***

2.2.1.1.3 Triglyceride :

Is an organic compound consisting of up to three molecules of long chain fatty acids esterified to glycerol. Triacylglycerol constitute 95% of tissue storage fat and predominant form of glycerol ester found in plasma ***(carl et al.,2008)*** . Triglyceride is present in dietary fat, and can be synthesized in the liver and adipose tissue to provide a source of stored energy; this can be mobilized when required, for example during starvation. Although the majority of fatty acids١ in the body are saturated, certain unsaturatedfatty acids are important as precursors of prostaglandins and in the esterification of cholesterol. Triglycerides containing both saturated and unsaturated fatty acids are important components

of cell membranes ***(William.,2012)***. Triglycerides, containing cis unsaturated fatty acids (plant source), typically form oils at room temperature, whereas triglycerides containing mostly saturated fatty acids(animal sources) are usually solid at room temperature. Triglycerides are no charged groups or polar hydrophilic groups, making it very hydrophobic and virtually water insoluble. Because it has no charge, triglyceride is classified as a neutral lipid ***(Michael et al., 2010).***

2.2.1.1.4 Phospholipids:

Are compounds similar to the triglycerides but with one fatty acid residue replaced by phosphate and a nitrogenous base ***(William., 2012).*** Several types of phospholipid head groups, such as choline, inositol, serine, and ethanolamine, which are all hydrophilic in nature. The various types of phospholipids are named based on the type of phospholipid head group present. Phosphatidylcholine for example, has a choline head group and is the most common phospholipid found on lipoproteins and in cell membranes. The two fatty acids in phospholipids are normally 14 to 24 carbon atoms long, with one fatty acid commonly saturated and the other unsaturated. Because phospholipids contain both hydrophobic fatty acid CMH chains and a hydrophilic head group, they are by definition amphipathic lipid molecules and, as such, are found on the surface of lipid layer ***(Michael et al., 2010).*** Because the lipids are not water soluble, For that transported in the plasma in association with proteins. Albumin is the principal carrier of free fatty acids (FFAs); the other lipids circulate in complexes known as lipoproteins. These consist of a non-polar core of triglyceride and cholesteryl esters surrounded by a surface layer of phospholipids, cholesterol and proteins known as Apo lipoproteins

(William.,2012).

2.2.2 Lipoproteins:

Are lipids proteins complex in which lipids are transport in the blood. Lipoproteins particle consist of spherical hydrophobic core of triglyceride or cholesterol ester surround by monolayer of phospholipids, cholesterols and apolipoprotins.The lipids synthesis in liver and intestines and carry by macromolecules (lipoproteins)***(carl et al.,2008)*** .

2.2.2.1Classification of lipoproteins

Lipoproteins are classified on the basis of their densities as demonstrated by their ultracentrifugations separation ***(William.,2012)***.

2.2.2.1.1 Chylomicrons:

Are produced by the intestine, where they are packaged with absorbed dietary lipids. Containing apo B48 ,A. Once they enter the circulation, triglycerides and cholesteryl esters in chylomicrons are rapidly hydrolyzedby lipases and, within a few hours, they are transformed into chylomicron remnant particles, which are recognized by proteoglycans and remnant receptors(apo E) in the liver, facilitating their uptake.The principal role of chylomicrons is the delivery of dietary lipids to hepatic and peripheral cells ***(Michael., 2010***).

2.2.2.1.2 Very low density lipoproteins (VLDL) :

Are formed from triglycerides synthesized in the liver either de novoor by reesterification of free fatty acids ***(William., 2012).*** VLDL is produced by the liver and contains apo B-100, apo E, and apo Cs; like chylomicrons, they are also rich in triglycerides.18,19 They are the major carriers of endogenous (hepatic-derived) triglycerides and transfer triglycerides from the liver to peripheral tissue. Like chylomicrons. they also reflect light and account for most of the turbidity observed in fasting hyperlipidemic plasma specimens, although they do not form a creamy top layer like chylomicrons, because they are smaller and less buoyant ***(Michael., 2010).***

2.2.2.1.3 Low density lipoproteins (LDL) :

LDLs are the principal carriers of cholesterol, mainly in the form of cholesteryl esters. LDL are derived from VLDL, via IDL. They are removed by the liver and other tissues by a receptor-dependent process involving the recognition of apo B-100 by the LDL receptor. The LDL particles are hydrolysed by lysosomal enzymes, releasing free cholesterol which (i) inhibits HMG-CoA reductase, the rate-limiting step in cholesterol synthesis, (ii) inhibits LDL receptor synthesis and (iii) stimulates cholesterol esterification by augmenting the activity of the enzyme acyl CoA: cholesterol acyl transferase (ACAT) **(*William.,2012).*** Recently, there has been great interest in measuring LDL subfractions, because small, dense, LDL particles have been shown to be more proatherogenic and may be a better marker for coronary heart disease risk***(Michael., 2010)***.

2.2.2.1.4 Lipoproteins (a):

Lipoproteins (a) particles are LDL-like particles that contain one molecule of apo (a) linked to apo B-100 by a disulfide bond. Lp(a) particles are heterogeneous in both size and density. Elevated levels of Lp(a) are thought to confer increased risk for premature coronary heart disease and stroke. Because the kringle domains of Lp(a) have a high level of homology with plasminogen, a protein that promotes clot lysis, it has been proposed that Lp(a) may compete with plasminogen for binding sites, thereby promoting clotting, a key contributor to both myocardial infarction and stroke ***(Michael., 2010).***

2.2.2.1.5 High density lipoproteins:

HDL, the smallest and most dense lipoprotein particle, is synthesized by both the liver and intestine. HDL comprising phospholipid, cholesterol, apo E and apo A. Uptake of cholesterol is stimulated by ATP-binding cassette protein A1 (ABCA1). Nascent HDL is disc shaped, in the circulation, it acquires apo C and apo A from other lipoproteins and from extrahepatic tissues, and in doing so

assumes spherical conformation. The free cholesterol is esterified by the enzyme lecithin-cholesterol acyltransferase (LCAT), which is present in nascent HDL and activated by its cofactor, apo A-I. This increases the density of the HDL particles, which are thus converted from HDL3 to HDL2 ***(William., 2012)***.

The ability of HDL to remove cholesterol from cells, called reverse cholesterol transport, is one of the main mechanisms proposed to explain the antiatherogenic property of HDL. When discoidal HDL has acquired additional lipid, cholesteryl esters and triglycerides form a core region between its phospholipid bilayer, which transforms discoidal HDL into spherical HDL. HDL is highly heterogeneous separable into as many as 13 or 14 different subfractions. There are two major types of spherical HDL based on density differences: HDL2 and HDL3. HDL2 particles are larger in size and richer in lipid than HDL3 and may reflect better efficiency in delivering lipids to the liver ***(Michael., 2010).***

2.3 Menopause

The term "menopause" comes from two Greek words that mean "month" and "to end". In other words, it translates as "the end of the monthlies" ***(Huda., 2008)***. Menopause is defined as the permanent cessation of menstruation resulting from the loss of ovarian follicular activity ***(Helena., 2014)*** It is the result of irreversible changes in the hormonal and reproductive functions of the ovaries, Hormonal fluctuations affect more than a woman's reproductive system .During menopause a woman's body slowly produces less of the hormones estrogen and progesterone. This often happens between ages 45 and 55, A woman has reached menopause when she has not had a period for 12 months in a row ***(Suri and Suri., 2016)***.

2.3.1 Stages of menopause

2.3.1.1 Perimenopause or (menopause transition)

Perimenopause can begin 8 to 10 years before menopause, when the ovaries gradually produce less estrogen. It usually starts in women at 40 but can start in the 30 as well, Perimenopause lasts up until menopause, the point when the ovaries stop releasing eggs in the last 1-2 years of Perimenopause, the drop in estrogen accelerates. At this stage, many women can experience menopause symptoms.

2.3.1.2 Menopause

Menopause is point when a woman no longer has menstrual periods. At this stage,the ovaries have stopped releasing eggs and producing most of their estrogen. Menopause is diagnosed when a women has gone without a period for 12 consecutive months ***(Cleveland., 2017).***

2.3.1.3 Post menopause

These are years after menopause, During this stage menopausal symptoms such as hot flashes can ease for many women, But as a result of a lower level of estrogen. Postmenopausal women are at increased risk for a number of health conditions such as osteoporosis and heart diseases. Medication such as hormone therapy and/or healthy life styles changes may reduce the risk of some of these conditions ***(Cleveland, 2017).***

2.3.2 Symptoms of menopause

Menopause symptoms begin gradually while the ovaries are still functioning and a woman is still having menstrual periods. Some of symptoms are common such as:

Changes in period: time between periods or flow may be different***(Questions.,2013)***.Hot flashes (“hot flushes”): getting warm in the face, neck and chest with and without sweating **(Questions.,2013)**.Night sweats that

may lead to problems sleeping and feeling tired, stressed or tense**(Questions.,2013**).Vaginal changes:the vagina may become dry and thin, and sex may be painful**(Questions.,2013**).Thinning of bones: which may lead to loss of height and bone breaks (osteoporosis) **(Questions.,2013**).

2.3.3 Causes of menopause

The menopause happens when the ovaries stop responding to certain hormones from the brain, and so eggs stop maturing regularly. There is a drop in the levels of estrogen and progesterone (the two female sex hormones produced by the ovaries).It is this fall in hormone levels that causes symptoms of menopause **(Huda.,2008)**.

2.4 Body mass index

Is a number calculated from a person weight and height. BMI is a fairly reliable indicator of body fatness for most people. By WHO criteria based on the international classification on adults, a person with a BMI between 18.5 and 25 kg/m2 is considered as healthy. a person with a BMI over 25 kg/m2 but less than 30 kg/m2 is considered overweight and a person with a BMI over 30 is considered obese***(Donoghue.,1985).*** BMI and weight are important factors that change both bone mineral density and lipids. Relationship between bone mineral density and lipids is manipulated by many various factors such as age, lifestyle, physical activity, consumption of dairy products and amount of fat mass that all of them should be considered***(Donoghue.,1985).***

2.7 Association betweenlipid and lowbone density

A ‹role for atherogenic lipids and lipoproteins in the pathogenesis of bone loss ‹lipoproteins on bone cells/including direct effects of these bioactive lipids .c differentiationinhibiting osteoblastic differentiation and promoting osteoclasti It also addresses recent evidence that suggests that bioactive lipids blunt the

effects of bone anabolic agents such as teriparatide and bone morphogenetic Systemic and intracellular oxidant stress and inflammation are .proteins)lipoproteins/ed in mediating the effects of bioactive lipidsimplicat**Tintut Y and Demerit L.,2014)**

Chapter three

Material and Method

3.Material and method

3.1 Study design

Descriptive cross sectional study .

3.2 Study Area and period

This study carried out in Khartoum state, during the period of March to December2019.

3.3 Inclusion criteria

postmenopausal women's.

3.4 Exclusion criteria

Patients were excluded if they were taking drugs such as statins, vitamin D, bisphosphonates, glucocorticoids and high dose of levothyroxine.

Also patients with renal failure, chronic infections such as tuberculosis, malignancy, osteomalacia, and thyrotoxicosis were excluded from the study. History of smoking, alcohol abuse and low trauma fracture was recorded.

3.5 Ethical considerations:

Study was approved from local ethical committee of the Sudan University of Science and Technology and then approved by Ethical Commite of Ministry of Health,verbal informed consent was obtained from all participants after informed by aims of the study.

3.6 Data collection and Samples:

Direct interviewing of patients using standardized questionnaires to collect the data, concerning determinant of plasma lipid profile levels.

Samples were collected by using dry, plastic syringes ,tourniquet was used to make the vein more prominent, blood samples (5ml) was collected in lithium heparin containers from each volunteer under septic condition. then they were

centrifuges at 4000 rpm to obtain the plasma samples, and stored in-20° until the analysed. Weight and height were measured without the subjects' wearing shoes and wear hight cloths. Body mass index (BMI) was calculated as weight/height2(m)

3.7 Estimation of plasma total cholesterol

3.7.1 Principle of method:

Ester cholesterol hydrolyzed in present of cholesterol esterone to free fatty acid and free cholesterol which oxidized by atmospheric oxygen in presence of cholesterol oxidase to cholestene_3,1 and hydrogen peroxide,which converted by peroxidase to H2O and oxygen then oxygen accepted by para amino phenazone in presence of phenol to producequinoninmine pink colour measured by spectphotometry**(Bishop.,2010).**

3.7.2 Procedure: Appendix 2

3.8 Estimation of triglycerides:

3.8.1 Principle of method:

Triglycerides hydrolyzed enzymatically in the presence of lipase to 3fatty acid and glycerol,which phosphorylated in the presence of ATP and glycerol kinase to glycerol_3_phosphate that oxidized in presence of glycerol_3_phosphate oxidase to dihydroxyacetone phosphate and hydrogen peroxide which converted by peroxidase to H2O and O2 then oxygen accepted by para_amino phenazone in presence of phenol to produce quinoninmine pink colour measured by spectrophotometry**(Bishop et al.,2010).**

3.8.2 Procedure: Appendix 3

3.9 Estimation of high density lipoprotein cholesterol(HDL_c):

3.9.1 Principle of method:

VLDL,chylomicrone and LDL in the sample precipitate with phosphotungstate and magnesium ions, after centrifugation the supernatant contain high density lipoprotein which measured by cholesterol oxidase method spectrophotometric ally.

3.9.2 Procedure: Appendix 4

3.10 Calculation of low density lipoprotein (LDL-c)

Calculated from **Fried -wald's equation:**

LDL-c = total Cholesterol – HDL-c triglyceride/5. If triglyceride concentration is below 400 mg/dl ***(Bishop et al .,2010).***

3.11 BMI calculation

BMI obtained by calculation according to formula: Weight(kg) ÷ Height (m2)**(Bishop et al.,2010).**

3.12 BMD measurement

Dual-energy X-ray absorptiometry was used to measure bone mineral density (CM200) (BMD in g/cm2).

3.13 Quality control

To ensure adequate quality control, to verify the performance of measurementby use control serumnormal and pathological .

3.14 Statistical analysis:

The data was analyzed using statistical package of social science (SPSS) version 16. Computer program using frequency and percentage, and one way a nova test. result was expressed as (mean ±SD) for variables of groups and significance different was considering as (p-value <0.05).

Chapter four

Results

4. Result

The study was conducted on 98 subjects postmenopausalwomen, all participants had no history of smoking or alcohol abuse, and participant average DEXA score is (1.3 ± 0.83) average BMI is (31.3 ± 6.2)kg/m2 to evaluate the level of lipid profile.

Table(4.1)Show frequency and percentage of bone mineral density and body mass index among study group.

Frequency analysis of BMD and BMI

Table (4.2) Show frequency and percentage of lipid profile

Frequency analysis of total cholesterol, triglyceride, high density lipoprotein and low density lipoprotein.

Table (4.3) Show frequencies of study groups according to lipid profile Level.

Show insignificant different of total cholesterol, triglyceride, high density lipoprotein and low density lipoprotein in bone mineral density.

Table (4.4) Comparison means of total cholesterol, triglyceride, HDL-c and LDL-c to reference values.

Show significant decrease in means of cholesterol, TG, HDL-c and LDL.c when compared to reference values.

Table (4.5) Comparison between lipid profile according to bone mineral density among study group.

Show insignificant different of total cholesterol, triglyceride, high density lipoprotein and low density lipoprotein in bone mineral density.

Table (4.6) comparison between lipid profile and body mass index among study population.

Show insignificant different of total cholesterol, triglyceride, high density lipoprotein and low density lipoprotein in body mass index.

Table(4.1): Frequency and percentage of bone mineral density and body mass index among study group

Variable	Classification	Frequency	Percentage %
Bone mineral density	Normalpenia (T- score < -1)	39	39.7%
	Osteopenia (T-score -1 to -2.50)	48	48.9%
	Osteoporosis (T-score >-2.50)	11	11.2%
Body mass index	Underweight (< 19 kg/m2)	3	3.1%
	Normal (20 - 24.9 kg/m2)	13	13.3%
	Overweight (25 - 29.9 kg/m2)	26	26.5%
	Obese(>30 kg/m2)	56	57.1%
	Total	98	100%

Table (4.2) :Frequency and percentage of lipid profile among study group.

		Frequency	Percentage%
Total cholesterol	Low risk level(<200 mg/dL)	76	77.7%
	Border line risk(200 -240 mg/dL)	18	18.4%
	High risk (>240mg/dL)	4	4.1%
Triglyceride	Low risk (>150mg/dL)	74	75.5%
	Border line risk (150-200mg/dL)	19	19.3%
	High risk (<200 mg/dL)	5	5.1%
HDL-c	Low risk (>60 mg/dL)	49	50%
	Border line risk (35-60mg/dL)	19	44.9%
	High risk (<35mg/dL)	5	5.1%
LDL-c	Low risk (>130mg/dL)	84	85.7%
	Border line risk (130-160mg/dL)	9	9.2%
	High risk (>160mg/dL)	5	5.1%
	Total	98	100%

HDL-c high density lipoprotein

LDL-c low density lipoprotein

Table (4.3) Show frequencies of lipid profile according to BMD group in study population.

Lipid classification		**Normalpenia**	**osteopenia**	**osteoporosis**	**p.value**
Total cholesterol	Low risk	34.7% (n=34)	34.7% (n=34)	8.2% (n=8)	0.3
	Border line risk	5.1% (n=5)	11.2% (n=11)	2.1% (n=2)	
	High risk	P% (n=0)	3.1% (n=3)	1.1% (n=1)	
HDL-c	Low risk	20.4% (n=20)	22.4% (n=22)	7.1% (n=7)	0.6
	Border line risk	16.3% (n=16)	24.5% (n=24)	4.1% (n=4)	
	High risk	3.1% (n=3)	2% (n=2)	0% (n=0)	
LDL-c	Low risk	36.7% (n=36)	39.8% n=(39)	9.2% (n=9)	0.4
	Borderline risk	3.1% (n=3)	5.1% (n=5)	1% (n=1)	
	High risk	0% (n=0)	4.1% (n=4)	1% (n=1)	
Triglyceride	Low risk	25% (n=25)	36.7% (n=36)	9.1% (n=9)	0.3
	Border line risk	10.2% (n=10)	7.1% (n=7)	1% (n=1)	
	High risk	2% (n=2)	2% (n=2)	1% (n=1)	

*One way ANOVA was used to compare between means

*p value considered insignificant at level >0.05.

Table (4.4) Comparison between means of plasma total cholesterol, triglyceride, HDL-c and LDL-c in study to reference values.

Lipid (mg/dL)	Mean ±SD	P value
Cholesterol case Test value	173.9±40.5 220	0.000
Triglyceride case Test value	124.9±38.4 175	0.000
HDL-c case Test value	28.2±14.5 47.5	0.000
LDL-c case Test value	89.1±38.2 145	0.000

One sample T test used to compare between means.

P value considered Insignificant at level> 0.05

Table(4.5) comparison between lipid profile according to bone mineral density among study group.

Lipids(mg/dL)	Bone density	Mean±SD	p-value
Cholesterol	Normal	166.5±31.4	0.327
	Osteopenia	177.9±45.3	
	osteoporosis	182.5±46.3	
Triglyceride	Normal	134.2±40	0.107
	Osteopenia	120.8±38.2	
	osteoporosis	110±26.6	
HDL-C	Normal	58.1±16.5	0.336
	Osteopenia	57±12.8	
	osteoporosis	64±10.7	
LDLC	Normal	79.9±30.3	0.175
	Osteopenia	94.4±40.9	
	osteoporosis	96±48.1	

One way ANOVA test was used to compared between means.

p-value considered insignificant at level >0.05

Table (4.6) comparison between means of lipid profile and body mass index among study population

Lipid profile (mg/dL)	Body mass index(kg/m2)	Mean±SD	p-value
Total Cholesterol	Underweight(<19 kg/m2)	178.0±51.2	0.8
	Normal weight (20-24.9 kg/m2)	179.2±57.5	
	Over weight (25-29.9kg/m2)	168.3±38.1	
	Obese(>30kg/m2)	175±37.3	
Triglyceride	Underweight	100±30.8	0.5
	Normal weight	130.1±43	
	Over weight	129.5±40	
	Obese	122.9±37.1	
HDL-c	Underweight	41.3±5.6	0.1
	normal weight	60.6±12.5	
	over weight	59.6±15	
	Obese	57.9±14.1	
LDL-c	Underweight	110.3±48.5	0.6
	normal weight	92.3±52.4	
	over weight	82.4±35.1	
	Obese	89.8±35.9	

One way ANOVA test was used to comparison between means

p-value considered insignificant at level >0.05

Chapter five

Discussion, conclusion, RecommenDation.

5. Discussion, Conclusion and Recommendations

5.1 Discussion

Low bone mineral density is a disease which affect the population of so called "third age", or the elderly, while it more affect females,and that among them there is a rapid process of losing bone mass after entering the menopause and the occurrence of ovarian insufficiency and is a multifactorial disease that occurs due to the effects of genetic and environmental factors***(Bijelicetal.,2016)*.** According to DEXA result, 37.9% of postmenopausal women were normalpenia BMD, 47.9% were diagnosed with osteopenia and 11.2% osteoporosis BMD, this result agree with **(Maria et al.,2015)**. At the women menopause calcium storage in bones decreases, as result of lack of estrogen produced by ovary and consequently the demolitions and calcium salt come out the bone, and the women begin suffer from low bone density. Also With estrogen deficiency, the osteoclasts live longer and are therefore able to resorb more bone. In response to the increased bone resorption, there is increased bone formation and a high-turnover state develops which leads to bone loss***(Bijelicetal.,2016)*.**

Concerning to BMI result, 57.1% of postmenopausal women were obese, 26.5% Overweight, 13.3% normal BMI and 3.1% with underweight, this result agree with**(Bijelic et al.,2016)**. Estrogen was responsible for controlling body weight, reduced estrogen caused reduction in metabolic rate and also cause the body to use starches and blood sugar less effectively, and it will increase fat storage and make it difficult to lose the weight. Muscle mass loss and lack of exercise also makes gaining extra weight more and faster**(Bijelicetal.,2016).**

The study showed significant decrease in means of lipid profile(cholesterol, triglyceride, HDL-c and LDL-c) when compared with the reference value,This

is may be due to the different eating patterns, sudan is one of developing countries where people not consuming more fats, oils and animal product.

The finding of this study was showed that the mean value of cholesterol, triglyceride, HDL-c and LDL-c were statistically insignificant when compared with bone mineral density in postmenopausal women ,this result is in agreement by **(Salmon et al .,2005 ;Ghadiri et al.,2018)** they found no relationship between lipid parameters and bone mineral density**(Ghadirietal.,2018).**

The study also showed no correlation between means of lipid profile and BMI, this result agree by ***(Lejka ME and Edham HA.,2012***), the increase of body mass in menopause and different distribution of adipose tissue is the result of changes in estrogen and androgen level in circulation, but also is a result of changesinlipidandcarbohydratemetabolism,reductionofenergeticneedsandphysicalactivity***(LejkaMEandEdhamHA.,2012).***

5.2 Conclusion

Lowbonemineraldensityareverycommonamongpostmenopausalwomeninsudan.

My study conclude that serum lipid profile at normal level in Sudanese postmenopausal women withlow bone mineral density. Body mass index increase with age in sudanesepostmenopausal women with low bone density, while no association between lipid profile and bone mineral density.

5.3 Recommendations

Every women above 30 should take calcium and vitamin D supplement and fortified foods.

Screening should be done after menopause to pervent from consequence.

Create awareness about low bone density and its consequences and encourage people for taking steps for prevention and treatment before reaching severe conditions.

Exercise, walking daily for 30 minutes and weight bearing exercises on a treadmill are helpful in prevention and treatment.

Estimation of other parameters and its comparing to BMD.

Estimation of vitamin D level, thyroid function tests, and other parameters and bone markers.

Further research with cohort study

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(Appendix 1)

Sudan University of Science and Technology

College of Graduate Studies

Lipid Profileand at association with Bone Mineral Density Among Postmenopausal Sudanese Women
(a study in Khartoum State)

Quationare No ( )

Participant Name: …… Address: …….

Age: ……. Contact Phone Number: ……....

Weight: …….. Higther: ……..

Drink tea and coffee: ……. Smoke and alcohol abuse: .....

Are you in menopause? YES( ) NO( )

Do you take Statin and or other antiatherogenic drugs? YES( ) NO( )

Do you take any medicine for low bone mineral density? YES( ) NO( )

Do you take Vitamin D and Ca Supplements? YES( ) NO( )

Do you have any endocrine disorder? YES( ) NO( )

Do you currently take hormone replacement therapy? YES( ) NO( )

History of fractures?

When?.....Which bone? .....

DEXA result:

Investigation:

Total cholesterol:.....mg/dl.

Triglyceride: ..... mg/dl.

LDL-C : ..... mg/dl.

HDL-C : ..... mg/dl.