

INTERNATIONAL JOURNAL OF CURRENT RESEARCH IN CHEMISTRY AND PHARMACEUTICAL SCIENCES

(p-ISSN: 2348-5213; e-ISSN: 2348-5221)
www.ijrcps.com



Research Article

THE CHANGE IN THE LIPID PROFILE IN PATIENTS WITH OSTEOPOROSIS

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Abstract

Background: Osteoporosis is a silent disease of the bones that makes them weaken and prone to fracture. In osteoporosis, the cortex becomes thinner and more brittle, while the inner trabecular bone develops larger holes. Mature adult bone is continually being remodelled. **Objectives:** To investigate the level of lipid profile in patients with osteoporosis. **Subjects and methods:** The present study was designed to investigate changes in levels of lipid profile in the sera of patients with osteoporosis. Lipid profile were measured by colorimetric method by use kits from linear company. **Results :**The results show that there was a positive significant difference ($P < 0.05$) in HDL level compared with control group, while there was a negative significant difference ($P < 0.05$) in total cholesterol, LDL while there was positive correlation between bone mineral density with HDL compared with control group. **Conclusions:** From the relationship of bone mineral density with lipid profile, bone mineral density positively associated with HDL while negatively associated with (triglyceride, LDL, total cholesterol and VLDL), this may lead us to consider (triglyceride, LDL, cholesterol and VLDL as risk factors while HDL consider as protective factor.

Keywords: Osteoporosis, and lipid profile.

Introduction

Osteoporosis is a silent disease of the bones that makes them weaken and prone to fracture. The disease is silent because there are no symptoms, this can occur even after a minor injury, such as a fall. The most common fractures occur at the spine, wrist and hip. Spine and hip fractures in particular may lead to chronic pain^[1].

In osteoporosis, the cortex becomes thinner and more brittle, while the inner trabecular bone develops larger holes. Mature adult bone is continually being remodeled. Specialized cells called osteoclasts absorb old bone and other cells called osteoblast secrete new, strong, bone. In this way, bone retains its strength and density. Normally in the adult skeleton, about 3% of 'cortical' bone the outer hard part – and 25% of 'trabecular' bone – the inner, honeycomb par is remodeled each year^[2].

Many factors will increase your risk of developing osteoporosis and suffering a fracture. Some of these risk factors can be changed, while others cannot, risk factors

include: Older age, non-Hispanic white and Asian ethnic background, small bone structure, family history of osteoporosis or osteoporosis-related fracture in a parent or sibling, previous fracture following a low-level trauma, especially after age 50, sex hormone deficiency, particularly estrogen deficiency, anorexia nervosa, low dietary intake or absorption of calcium and vitamin D, medications: glucocorticoid medications, and certain diseases can affect bone, such as endocrine disorders (hyperthyroidism ,hyperparathyroidism, Cushing's disease, etc.) and inflammatory arthritis^[3].

Subjects

This study was conducted during the period from September 2011 to December 2011. This study includes ten patients with osteoporosis admitted to AL-Yarmouk Hospital and ten subjects with matched age, sex and BMI, were included in this study as control group. Blood samples were taken from patients after having thoroughly examined.

Samples collection

From each patient and control, five ml of venous blood were aspirated from a suitable vein. Samples were collected between (8-9 A.M.) after 10 hours fasting. The blood, transferred to a plain tube to measure the levels of lipid profile. The non heparinized blood in the plain tubes were left to clot and then centrifuged at 4000 rpm for 5 minutes to separate the serum and dispensed into tightly closed Eppendorf tubes in 1.0 ml and stored at -20 C° until assayed. Lipid profile measured by Kits from Linear Chemicals-France.

Statistical analysis

Statistical analysis was performed by statisticians with the SPSS 15.01 Statistical Package for Social

Sciences and also Excel 2003. Data analysis was done using chi-square test for tables with frequencies, while independent sample t-test was used for tables with means and standard Deviation. P-value of 0.05 was used as the level of significance. Correlation coefficient used to find the correlation between studied markers by using Pearson correlation.

Results**Anthropometrics parameters: (Age, height, weight, BMI, waist and hip):**

Value of age, height, weight, BMI, waist circumference, and hip circumference were compared between the patient group and control group using analysis of variance t-test of significant as in table (1)

Table (1) Comparison between studied groups for (age, height, weight, BMI, waist circumference hip circumference and neck circumference)

Parameters	Patients Mean±SD No.=10	Control Mean±SD No.=10	p –value
Bone mineral densety	-2.35±1.07	0.72± 0.28	0.01*
Age(Years)	58.22± 3.11	53.5 ± 8.21	0.266
Height (cm)	157.11± 10.96	156.75 ± 9.03	0.472
Weight (Kg)	78.11 ± 13.53	75.625±10.32	0.342
BMI= Kg/m2	33.88 ± 6.39	30.78 ± 3.35	0.130
Waist circumference (cm)	108 ± 10.24	111.37±12.10	0.265
Hip circumference (cm)	114.55±10.44	105.5±11.78	0.05*
Neck circumference (cm)	38.77± 9.87	37.7 ± 2.18	0.166

*= significant difference p 0.05

Comparison between groups for lipid profile.

Table (2) Comparison between groups for lipid profile.

Parameters	Patients Mean±SD (mmole/l) No.=10	Control Mean±SD (mmole/l) No.=10	p –value
Total cholesterol mmole/l	6.56± 1.40	4.25± 0.62	<0.05
LDL(mmole/l)	5.24± 0.73	3.06±1.38	<0.05*
Triglyceride (mmole/l)	2.01± 0.76	1.96± 0.81	0.452
VLDL(mmole/l)	0.88± 0.34	0.83± 0.28	0.407
HDL(mmole/l)	1.06± 0.22	2.35± 0.14	<0.05*

*= significant difference p 0.05

Relation between BMD and other parameters**1- Relation between BMD and age, height, weight, BMI, waist circumference hip circumference and neck circumference)****Table (3)** The correlation (r) between BMD and age, height, weight, BMI, waist circumference hip circumference and neck circumference for patients group.

Parameters	(r)	p -value
Age	-0.712	0.266
Height	-0.548	0.472
Weight	-0.587	0.342
BMI	-0.548	0.130
Waist circumference	-0.296	0.265
Hip circumference	-0.575	0.05*
Neck circumference	-0.104	0.166
Fat percent	-0.428	0.266

* = significant difference p 0.05

2- Relation between BMD and lipid profile**Table (4)** The correlation (r) between BMD and lipid profile

Parameters	(r)	p –value
Total cholesterol	-0.508	<0.05
LDL	-0.558	<0.05*
Triglyceride	-0.301	0.452
VLDL	-0.304	0.407
HDL	0.591	<0.05*

* = significant difference p 0.05

Discussion

As shown in table (3) there were significantly negative correlation between bone mineral density (BMD) with hip circumference and negative correlation with age, high, BMI, waist and neck circumference and this result agree with Rohit Gopinath *et al.*, who reporter the that the BMD decreases by advancing age and has a negative relation by BMI^[4].

Body mass index (BMI) is a better marker of bone mineral density in the weight-bearing sites than in the non-weight-bearing sites, implying a mechanical effect of weight on bone mineral density(BMD), low body weight is known to be associated with an increased risk for osteoporosis. Advancing age and low body weight are associated with an increased risk for osteoporosis and such a higher prevalence of osteoporosis may due to interplay of various

mechanisms that generally occur in postmenopausal women hormonal factors strongly determine the rate of bone resorption; lack of estrogen (as a result of menopause) increases bone resorption as well as decrease the deposition of new bone that normally takes place in weight-bearing bones. Another explanation could be due to the lack of estrogen replacement for women at menopause, which virtually help maintain bone density and reduce the risk of development of osteoporosis. [5]

Bone dissolves and is absorbed faster than the formation of new bone leading to thinner bones because of sudden decrease in estrogens as a result of menopause and also as a natural part of aging. In addition to estrogen, calcium metabolism plays a significant role in bone turnover, and deficiency of calcium and vitamin D may lead to impaired bone deposition.[6]

The results shown in table (2) that there were significant elevation in total cholesterol and LDL level

in patients group, and table (4) show the negative correlation between the total cholesterol LDL-cholesterol, triglycerides and BMD.

The levels of serum total cholesterol (TC) and low-density lipoprotein cholesterol (LDL-C) were inversely associated with BMD in both pre- and postmenopausal women. A number of studies have suggested a positive relationship between BMD and triglyceride levels found no association between serum triglycerides and BMD in menopausal women, relation between triglyceride and BMD literature concerning relationships of LDL cholesterol levels and BMD is contradictory some reports showing a negative association between BMD and HDL cholesterol even though in a limited number of postmenopausal osteoporotic women^[7].

In spite of that several studies show the association between different components of lipid profile and BMD, a constant relation has not been found Cui and coworker found that serum level of total cholesterol and LDL significantly different in various bone conditions. These measured lipids were significantly higher in osteopenic patients than normal individuals and also higher in osteoporotic than osteopenic women^[7]. This upward trend is in agreement with other finding that showed negative relationship between total cholesterol and LDL and BMD. Despite many studies conducted to reveal the relation between lipid profile and BMD, pathophysiology and molecular mechanisms of this relation have not been fully investigated. Recent studies emphasize on important role of Osteoprotegerin (OPG) and receptor activator of nuclear factor κ B (NF- κ B) ligand (RANKL) in bone metabolism. OPG is a soluble glycoprotein that belongs to the tumor necrosis factor (TNF) receptor super family. OPG acts as a decoy receptor of the RANKL, which is a critical regulator of osteoclastogenesis and is known to restrain osteoclastogenesis by binding to RANKL and preventing RANKL from binding to the receptor activator of NF- κ B on osteoclasts.^[8]

Conclusion

- There was a significant increase in the levels of total cholesterol, LDL- cholesterol, while there was decrease in the level of HDL- cholesterol in patients with osteoporosis.
- From the relationship of bone mineral density with lipid profile, bone mineral density positively associated with HDL while negatively associated with (triglyceride, LDL- cholesterol, total cholesterol and VLDL), this lead to consider; triglyceride, LDL- cholesterol, cholesterol and VLDL as risk factors while HDL consider as protective factor.

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